ELECTROMAGNETIC RADIATION FROM SMARTPHONE AFFECTED HEADACHE AND SLEEP QUALITY OF HIGH SCHOOL STUDENTS IN CHIANG MAI PROVINCE

WANNA CHONGCHITPAISAN

DOCTOR OF PHILOSOPHY
IN COMMUNITY MEDICINE

GRADUATE SCHOOL
CHIANG MAI UNIVERSITY
JANUARY 2018

ELECTROMAGNETIC RADIATION FROM SMARTPHONE AFFECTED HEADACHE AND SLEEP QUALITY OF HIGH SCHOOL STUDENTS IN CHIANG MAI PROVINCE

WANNA CHONGCHITPAISAN

A THESIS SUBMITTED TO CHIANG MAI UNIVERSITY IN PARTIAL FULFILLMENT OF THE REQUIREMENTS FOR THE DEGREE OF DOCTOR OF PHILOSOPHY IN COMMUNITY MEDICINE

GRADUATE SCHOOL, CHIANG MAI UNIVERSITY
JANUARY 2018

ELECTROMAGNETIC RADIATION FROM SMARTPHONE AFFECTED HEADACHE AND SLEEP QUALITY OFHIGH SCHOOLSTUDENTSIN CHIANG MAI PROVINCE

WANNA CHONGCHITPAISAN

THIS THESIS HAS BEEN APPROVED TO BE A PARTIAL FULFILLMENT
OFTHE REQUIREMENTS FOR THE DEGREE
OFDOCTOR OF PHILOSOPHY
IN COMMUNITY MEDICINE

Examination Committee:

..... Chairman
(Prof. Dr. Wichai Aekplakorn, M.D.)

..... Member
(Prof. Dr. Phongtape Wiwatanadate, M.D.)

..... Member
(Asst. Prof. Dr. Nipapon Siripon)

..... Member
(Lect. Surat Tanprawate, M.D.)

..... Member
(Dr. Narupon Chattrapiban)

..... Member
(Assoc. Prof. Dr. Ratana Sapbamrer)

Advisory Committee:

..... Advisor
(Prof. Dr. Phongtape Wiwatanadate, M.D.)

..... Co-advisor
(Asst.Prof.Dr.Nipapon Siripon)

..... Co-advisor
(Lect. Surat Tanprawate, M.D.)

..... Co-advisor
(Lect.Assawin Narkpongphun, M.D.)

15 January 2018

To

my family, teachers and all colleagues who helped and taught me the importance of studying and working to my full potential and be happy in life.

ACKNOWLEDGEMENT

I would really thank everybody who encouraged me and helped me in my thesis project. First, I am deeply and profoundly grateful to my supervisor, Professor Phongtape Wiwatanadate, M.D., Ph.D., always took the time to guide, work with me and provided thoughtful feedback. I would like to thank Assistant Professor Nipapon Siripon, Ph.D. for her assistance on generating the application for smartphone output power measurement. I would also like to thank Surat Tanpravate, M.D. and Atsawin Narkpongphun, M.D. who helped me gain a better understanding of headache and adolescent included provided thoughtful feedback and constant encouragement. I want to thank Komsak Meksamoot, Ph.D. who helped me for developing proposal. I would also like to recognize Mr. Nathaphop Chaichaya who helped me in my data management. I would like to thank Professor Wichai Eakplakorn, M.D., Ph.D. and Narupon Chattrapiban, Ph.D. the examiner committee. I would also like to thank the faculty, staff and colleagues at Community Medicine Department for always being supportive of my efforts here. I would like to thank Associate Professor Penprapa Siviroj, Dr.PH. who support throughout the period of my study. I would also like to thank all teachers and students in Watatnothai Payapschool who participated in this study, for willingness to share their information. I special thank my boss, Adul Bandhukul, M.D. who was extremely kind for helping me achieve my dream and putting me on a journey for many more. I am also grateful to Faculty of Medicine Chiang Mai University and Department of Medical service fund, for granting me the funding to complete this degree.

I would like to thank my entire family for always being there and encouraging me throughout this process. I would especially like to thank all mylovely daughter, Pakanaporn, Ornkamon and Monrada Chongchitpaisanfor helping me. Finally, I would like to thank my mother, Mrs. Wilai Loumongkonnimit, who has always stood by me with love and supported me in all my endeavors.

Wanna Chongchitpaisan

หัวข้อดุษฎีนิพนธ์	การแผ่รังสีแม่เหล็กไฟฟ้าจากสมาร์ทโฟนมีผลต่ออาการปวดศีรษะและคุณภาพการนอนหลับของเด็กนักเรียนมัธยมศึกษาตอนปลายในจังหวัดเชียงใหม่
ผู้เขียน	นางวรรณา จงจิตรไพศาล
ปริญญา	ปรัชญาดุษฎีบัณฑิต (เวชศาสตร์ชุมชน)
คณะกรรมการที่ปรึกษา	ศ.ดร.นพ.พงศ์เทพ วิวรรธนะเดช อาจารย์ที่ปรึกษาหลัก ผศ.ดร.นิภาภณช์ ศิริพล อาจารย์ที่ปรึกษาร่วม อ.นพ.สุรัตน์ ตันประเวช อาจารย์ที่ปรึกษาร่วม อ.นพ.อัศวิน นาคพงศ์พันธุ์ อาจารย์ที่ปรึกษาร่วม

บทคัดย่อ

สมาร์ทโฟนเป็นเครื่องมือสื่อสารที่มีความทันสมัย สามารถตอบสนองความต้องการได้อย่างหลากหลาย โดยประเทศไทยพบประชากรกลุ่มอายุ 15-24 ปี เป็นกลุ่มที่มีการใช้คอมพิวเตอร์อินเตอร์เน็ตและโทรศัพท์เคลื่อนที่มากที่สุด ซึ่งสมาร์ทโฟนนับเป็นแหล่งแพร่พลังงานแม่เหล็กไฟฟ้าที่มีการใช้ใกล้กับบริเวณศีรษะและระบบประสาทมากที่สุด ซึ่งระบบประสาทในมนุษย์เป็นเนื้อเยื่อทางไฟฟ้าและมีภาวะที่ไม่เสถียร การสัมผัสพลังงานแม่เหล็กไฟฟ้าที่มีความเข้มต่ำก็สามารถกระตุ้นหรือเหนี่ยวนำให้เกิดการเปลี่ยนแปลงหน้าที่ของระบบประสาทที่เกี่ยวข้องกับอาการปวดศีรษะและปัญหาการนอนหลับ วัตถุประสงค์ในการศึกษานี้จึงต้องการศึกษาความสัมพันธ์ระหว่างการแผ่รังสีแม่เหล็กไฟฟ้าจากสมาร์ทโฟนที่มีผลกระทบต่ออาการปวดศีรษะและคุณภาพการนอนหลับ

ในการศึกษานี้ทำการสุ่มตัวอย่างนักเรียนมัธยมศึกษาตอนปลายในโรงเรียนประจำจังหวัดเชียงใหม่ 996 คน ด้วยวิธีการ random sampling เพื่อทำการสำรวจและคัดเลือกนักเรียนจำนวน 200 คน ตามเกณฑ์การคัดเข้าและคัดออกและทำการศึกษาในรูปแบบ prospective time series designs กลุ่มตัวอย่างได้ทำการบันทึกอาการปวดศีรษะ การนอนหลับ และกิจกรรมที่เกี่ยวข้องด้วยแบบบันทึกประจำวันที่จัดทำเป็น Application ร่วมกับการจัดทำ Application เพื่อบันทึกข้อมูล smartphone output power ที่ทำการวัดจากเครื่อง smartphone ทำการวิเคราะห์ข้อมูลด้วย generalized estimation

equations และ binary logistic regression ผลการศึกษาในระยะที่ 1 พบอาการปวดศีรษะที่เกี่ยวเนื่องจากการใช้โทรศัพท์เคลื่อนที่ (Mobile Phone Associated Headache: MPAH) ร้อยละ 77.7 (95% CI: 74.8-80.3) ซึ่งเป็นอาการปวดศีรษะที่ไม่สามารถจัดเข้ากลุ่มได้ (undetermined headache) มีลักษณะอาการปวดศีรษะเฉพาะแตกต่างจาก primary headache พบปัจจัยการสนทนาทางโทรศัพท์เคลื่อนที่ (MP) และอาการร้อนรอบหูมีผลต่อ MPAH และพบการใช้ MP ในระยะเวลานานก่อนนอนมีผลต่อคุณภาพการนอนหลับ โดยการศึกษาในระยะที่ 2 พบว่า smartphone output power (SOP) มีค่าต่ำสุดเท่ากับ 9 $x10^{-9}$ สูงสุดอยู่ในค่า 1.55mW ค่าเฉลี่ยเท่ากับ 0.001mW ขณะที่พบว่า SOP ในช่วง $\leq$ 1.79 และ 1.80-1.99$x10^{-5}$mW มีความสัมพันธ์ต่อการเกิดอาการปวดศีรษะชนิดไมเกรน OR_{adj}2.02; 95% CI: 1.17-3.49 และ OR_{adj}3.25; 95% CI: 1.65-6.42 โดยอาการปวดศีรษะชนิดที่ไม่สามารถจัดเข้ากลุ่มมีการตอบสนองต่อ SOP ในช่วง 1.80-1.99$x10^{-5}$mW โดยเทียบกับช่วง $\geq$ 2 $x10^{-5}$mW พบ OR_{adj}2.32; 95% CI: 1.23-4.34 โดยเทียบกับ SOP ในช่วง $\geq$2 $x10^{-5}$mW และพบอาการปวดศีรษะมีการตอบสนองต่อผลหน่วงของ SOP ในรูปแบบ dose-response ขณะที่ไมเกรนตอบสนองในรูปแบบ reverse dose-response โดยพบลักษณะของ SOP มีลักษณะเข้าได้กับเกณฑ์การเป็นตัวกระตุ้น (trigger) ต่ออาการปวดศีรษะไมเกรนนอกจากนี้พบปัญหาการนอนหลับยากตอบสนองต่อ SOP ที่ใช้ก่อนนอนในช่วง $\leq$1.79 x 10^{-5}mW เทียบกับช่วง $\geq$2 x 10^{-5}mW พบ OR_{adj}2.19; 95% CI: 1.01 - 4.71 โดย SOP ในช่วง $\leq$1.79 และ $\geq$2 $x10^{-5}$mW เมื่อเทียบกับช่วง 1.80 - 1.99 x 10^{-5}mW มีความสัมพันธ์กับการนอนไม่มีประสิทธิภาพ OR_{adj}4.54; 95% CI: 3.33-6.20 และ OR_{adj}3.81; 95% CI: 2.59-5.60 พบการใช้ SOP ก่อนนอนในช่วง 1.80-1.99$x10^{-5}$mW เมื่อเทียบกับช่วง $\leq$1.79 $x10^{-5}$mW มีความสัมพันธ์กับอาการง่วงนอนตอนเช้า OR_{adj}1.78; 95% CI: 1.21-2.61 ขณะที่ระยะเวลาการนอนไม่เพียงพอและการตื่นนอนระหว่างคืนตอบสนองต่อการใช้ SOP ก่อนนอนในช่วง $\geq$2 x 10^{-5}mW โดยพบ OR_{adj}1.78; 95% CI: 1.21-2.61 และ OR_{adj}1.26; 95% CI: 1.01-1.57 ตามลำดับ ขณะที่การใช้ SOP ในช่วง $\geq$2 x 10^{-5}mW สัมพันธ์กับคุณภาพการนอนโดยรวม OR_{adj}1.30; 95% CI: 1.03-1.64 และการใช้ SOP เวลาหลังเที่ยงคืนในช่วง 1.80 - 1.99$x10^{-5}$mW พบมีความสัมพันธ์กับคุณภาพการนอนโดยรวม OR_{adj}1.66; 95% CI: 1.15-2.40 และพบว่าผลหน่วงของ SOP มีความสัมพันธ์เกือบทุกด้านกับคุณภาพการนอนหลับโดยผลการศึกษาที่พบความสัมพันธ์ระหว่าง SOP และระบบประสาทเป็นความสัมพันธ์ที่ไม่เป็นเส้นตรง เรียกว่า window effect

สรุป อาการปวดศีรษะและปัญหาคุณภาพการนอนหลับในกลุ่มวัยรุ่นมีแนวโน้มเพิ่มขึ้นสอดคล้องกับการเพิ่มขึ้นของโทรศัพท์เคลื่อนที่ โดยพบอาการปวดศีรษะที่เกี่ยวข้องกับการใช้โทรศัพท์เคลื่อนที่ (MPAH) ซึ่งเป็นกลุ่มที่ไม่สามารถจัดเข้ากลุ่มได้ (undetermined headache) มีลักษณะเฉพาะเข้าได้กับเกณฑ์ secondary headache พบการแผ่รังสีแม่เหล็กไฟฟ้าจากสมาร์ทโฟน

มี ความสัมพันธ์ต่ออาการปวดศีรษะและคุณภาพการนอนในรูปแบบ window effects และพบผลหน่วงของ smartphone output power ต่ออาการปวดศีรษะและปัญหาการนอนหลับพบ smartphone output power ที่มีลักษณะเข้าได้กับเกณฑ์การเป็นตัวกระตุ้น สุดท้ายผลการศึกษาพบว่า smartphone output power มีแนวโน้มในระดับต่ำความสัมพันธ์กับระบบประสาท ซึ่งอาจจะเกี่ยวข้องกับความถี่ของแม่เหล็กไฟฟ้าที่ควรทำการศึกษาต่อไปในอนาคต

Dissertation Title	Electromagnetic Radiation from Smartphone Affected Headache and Sleep Quality of High School Students in Chiang Mai Province	
Author	Mrs.Wanna Chongchitpaisan	
Degree	Doctor of Philosophy (Community Medicine)	
Advisory Committee	Prof. Dr. Phongtape Wiwatanadate, M.D.	Advisor
	Asst. Prof. Dr. Nipapon Siripon	Co-advisor
	Lect. Surat Tanpravate, M.D.	Co-advisor
	Lect. Atsawin Narkpongphun, M.D.	Co-advisor

ABSTRACT

Smartphones are modernized devices and designed to respond multiple needs. In Thailand, the people whose ages between 15-24 years have mostly used computers, internet, and mobile phones. Smartphones are sources of electromagnetic energy used closely to the heads and nervous systems, which are electrical-linked to each other but unstable. Furthermore, the exposure of low-intensity electromagnetic energy will excite or lead to changes in nervous system function related to headache and sleep disturbance. The objective of the study is to investigate the relationship between the effects of smartphones radiation on headache and quality of sleep.

The study has been designed cross-sectional using stratified random sampling by selecting 996 high school students of the provincial high schools in Chiang Mai. Therefore, 200 subjects have been selected from stage 1, based on sets of inclusion and exclusion criteria for participating in the prospective time series study. The subjects have been recorded daily headache, sleep, symptoms, and activities by application. The application has also been developed to collect smartphone output power level on Android and IOS operating systems. These data have been analyzed by binary logistic regression and generalized estimation equations. The result of the 1st phase shows the

symptom of Mobile Phone Associated Headache (MPAH) is 77.7% (95% CI: 74.8, 80.3), which defined the undetermined headache group who has specific characteristic pain that differentiates them from primary headache. The study results demonstrate that talking mode of mobile phone and ear burning sensation have strong associations with MPAH (OR_{adj}1.71; 95%CI: 1.16-2.51 and OR_{adj}2.43; 95%CI: 1.58-3.72). Long duration time of mobile phone use at night also relates to sleep quality (OR_{adj}1.60: 95% CI; 1.09-2.34). The result of the 2nd phase has found the minimum of smartphone output power (SOP) use among samples to be at 9 $x10^{-9}$mW, with maximum and mean of 1.55mW and0.001mW. Therefore, the result shows that the SOP in the range of ≤1.79 and 1.80 - 1.99$x10^{-5}$mW will affect migraine (OR_{adj}2.02; 95% CI: 1.17-3.49 and OR_{adj}3.25; 95% CI: 1.65-6.42). The undetermined headache has relatively been found with SOP in the range of1.80-1.99$x10^{-5}$mW (OR_{adj}2.32; 95% CI: 1.23-4.34).The SOP also has had the delay effect on headache in a dose-response correlation while migraine has reverse dose-response correlation. Additionally, smartphone electromagnetic radiation effects have triggered to the criteria that induce headache, especially migraines.

The SOP in the range of ≤1.79 x 10^{-5}mW correlates to sleep difficultly (OR_{adj}2.19; 95% CI: 1.01-4.71), in the range of ≤1.79 and ≥2 x 10^{-5}mW, and correlates to inefficiency sleeping (OR_{adj}4.54; 95% CI: 3.33-6.20 and OR_{adj}3.81; 95% CI: 2.59-5.60), also the range of 1.80 - 1.99$x10^{-5}$mW correlates to morning sleepiness (OR_{adj}1.78; 95% CI: 1.21-2.61). Sleep loss and wake up at night have related to SOP in the range of ≥2 $x10^{-5}$mW (OR_{adj}1.78; 95% CI: 1.21-2.61 and OR_{adj}1.26; 95% CI: 1.01-1.57 respectively). The SOP which the range of ≥ 2 $x10^{-5}$mW has correlated to poor sleep (OR_{adj}1.30; 95% CI: 1.03-1.64) and the nocturnal SOP in the range of 1.80-1.99 x 10^{-5}mW also affected poor sleeping (OR_{adj}1.66; 95% CI: 1.15-2.40). The delay effect of SOP has correlated with every domain of sleep quality. Thus, the result has shown non-linear correlation which has been called the window effect response.

Conclusion: According to the rapid changes of modernized technology and growth of smartphone use in tandem with the higher frequency in the symptom of MPAH and sleep problem in adolescence, the results have shown that MPAH characteristic classified as secondary headache. Electromagnetic radiation of smartphone correlates to headache and sleep problems in window effect. The delay effect of SOP also correlates to headache

and sleep problems. The SOP was classified as a trigger for migraine. Finally, the results have shown the trend of low output power correlated with the nervous system. That might be the frequency of electromagnetic radiation which should be investigated in the future study.

CONTENTS

	Page
Acknowledgement	d
Abstract in Thai	e
Abstract in English	h
List of Tables	m
List of Figures	q
List of Figures Abbreviation	r
List of Symbols	u
Glossary	v
Statement of originality	bb
Chapter 1 Introduction	1
1.1 Background	1
1.2 Rationale	2
1.2.1 Headaches and smartphone	2
1.2.2 Sleep and smartphone	3
1.3 Aims to study	4
1.4 Theory and literature review	5
1.4.1 Theory and literature review for electromagnetic radiation from Smartphone	5
1.4.2 Electromagnetic radiation emissions from mobile phones and biological impacts	13
1.4.3 Biological impacts on the nervous system	15
1.4.4 Theory and literature review for headache	17
1.4.5 Theory and literature review for sleep problem	38
1.5 Scope of the study	57
1.6 The benefit of the study	58
Chapter 2 Methodology	59
2.1 Part 1: Mobile phone use and health problem	59
2.2 Part 2: Electromagnetic radiation from smartphone and health problem	66

Page

Chapter 3 Result 74

3.1 Mobile phone use and health problem 74

3.1.1 The characteristic of MP using, prevalent of headache among high school students 74

3.1.2 The characteristic of MP using, prevalent of sleep quality among high school students 87

3.2 Electromagnetic radiation from smartphone and health problems. 97

3.2.1 The correlation between smartphone output power and headache among high school students 97

3.2.2 The correlation of smartphone output power and sleep sleep quality among high school students 114

Chapter 4 Discussion and suggestion 123

4.1 Discussion 123

4.2 Limited and strong point of study 159

4.3 Suggestion 161

4.4 The future study 163

References 164

List of publication 196

Appendix 197

Appendix A 198

Appendix B 241

Appendix C 251

Appendix D 256

Biography 266

LIST OF TABLES

		Page
Table 1.1	Special peak average SAR categorized according to phone use and frequency.	9
Table 1.2	Public SAR standard values and functions according to international organizations.	9
Table 1.3	Steps for categorizing migraine headaches.	32
Table 1.4	Steps for classifying tension-type headaches (TTHs).	33
Table 3.1	Demographic characteristics of participants presented as percentage unlessspecified otherwise.	75
Table 3.2	Characteristic of MP use of participants presented as percentage.	77
Table 3.3	The headache symptom and detail of headache and their 95% confidence intervals unless specified otherwise.	79
Table 3.4	The headache symptom and their 95% confidence intervals by demographic characteristics.	80
Table 3.5	The headache symptom and their 95% confidence intervals By pain score and impact of headache.	82
Table 3.6	The headache symptom and their 95% confidence intervals by characteristics of MP use.	83
Table 3.7	The mobile phone associated headache (MPAH) by characteristics of headache.	85
Table 3.8	Odds ratios (ORs) of MPAH and their 95% confidence intervals for each factor adjusted for all other factors using logistic regression.	87
Table 3.9	Characteristic of sleep of participants presented as percentage by grade level.	88
Table 3.10	PSQI score of participants compared by demographic data presented as mean and standard deviation unless specified otherwise.	89
Table 3.11	PSQI score of participants compared by risk behavior presented as mean and standard deviation unless specified otherwise.	90

		Page
Table 3.12	Prevalence of sleep quality component presented as percentage and their 95% confidence intervals by characteristic of MP use	91
Table 3.13	Prevalence of sleep quality in quantity domain and their 95% confidence intervals by characteristic of MP use.	93
Table 3.14	PSQI scores compared by impact of sleep.	95
Table 3.15	Odds ratios (ORs) of sleep quality component and their 95% confidence intervals for each factor adjusted for all other factors using logistic regression.	96
Table 3.16	Odds ratios (ORs) of doze and their 95% confidence intervals for each factor adjusted for all other factors using logistic regression.	97
Table 3.17	Demographic characteristics of participants presented as percentage unless specified otherwise	99
Table 3.18	Characteristics of headache of participants presented as percentage.	100
Table 3.19	Smartphone output power (SOP) by cycle time and daily dose.	100
Table 3.20	Smartphone output power group by cycle time and dailydose.	103
Table 3.21	Odds ratios (ORs) of headache event and their 95% confidence intervals for each factor and Lag dose adjusted for all other factors using GEE.	104
Table 3.22	Odds ratios (ORs) of headache event and their 95% confidence intervals for each factor and daily dose adjusted for all other factors using GEE (AR1, QIC=8397.22, QICC=8366.53).	105
Table 3.23	Odds ratios (ORs) of duration time of headache and their 95% confidence intervals for each factor and daily dose adjusted for all other factors using GEE (AR1, QIC=3473.75, QICC=3458.362).	105
Table 3.24	Odds ratios (ORs) of frequent headache and their 95% confidence intervals for each factor and daily dose adjusted for all other factors using GEE (AR1, QIC=10672.19 QICC=10572.75).	106
Table 3.25	Odds ratios (ORs) of pain scores and their 95% confidenceintervals for each factor and daily dose adjusted for all other factors using GEE (AR1, QIC=19288.47 QICC=19144.93).	106

Page

Table 3.26 Odds ratios (ORs) of headache type and their 95% confidence intervals for each factor and Lag dose adjusted for all other factors using GEE. 107

Table 3.27 Odds ratios (ORs) TTH type and their 95% confidence intervals for each factors and daily dose adjusted for all other factors using GEE (AR1, QIC=6959.62, QICC=6935.18). 108

Table 3.28 Odds ratios (ORs) migraine type and their 95% confidence intervals for each factors and daily dose adjusted for all other factors using GEE (AR1, QIC=1314.86, QICC=1309.60). 108

Table 3.29 Odds ratios (ORs) undetermined headache and their 95% confidence Intervals for each factor and daily dose adjusted for all other factors using GEE (AR1, QIC=3303.33, QICC=3289.53). 109

Table 3.30 Odds ratios (ORs) of time cycle headache and their 95% confidence Intervals for each factor and Lag dose adjusted for all other factors using GEE. 110

Table 3.31 Odds ratios (ORs) nocturnal headache (24-6.00AM) and their 95% confidence intervals for each factor and daily dose adjusted for all other factors using GEE (AR1, QIC=899.92, QICC=895.28). 111

Table 3.32 Odds ratios (ORs) morning headache (6-12.00AM) and their 95% confidence intervals for each factor and daily dose adjusted for all other factors using GEE (AR1, QIC=3863.46, QICC=3847.87). 111

Table 3.33 Odds ratios (ORs) daytime headache (12-18.00AM) and their 95% confidence intervals for each factor and daily dose adjusted for all other factors using GEE (AR1, QIC=3581.55, QICC=3566.90). 112

Table 3.34 Odds ratios (ORs) evening headache (18-24.00AM) and their 95% confidence intervals for each factor and daily dose adjusted for all other factors using GEE (AR1, QIC=3969.12, QICC=3950.87). 113

Table 3.35 Time cycle headache by headache type. 113

Table 3.36 Characteristic sleep of participants presented as percentage. 114

Table 3.37 Characteristic sleep quality of participants presented as percentage. 115

Page

Table 3.38 Odds ratios (ORs) of sleep quality and their 95% confidence intervals for each factor and Lag dose adjusted for all other factors using GEE. 117

Table 3.39 Odds ratios (ORs) of difficult sleep and their 95% confidence Intervals for each factor and daily dose adjusted for all other factors using GEE(AR1, QIC=6404.39, QICC=6336.22). 117

Table 3.40 Odds ratios (ORs) of sleep loss and their 95% confidence intervals for each factor and daily dose adjusted for all other factors using GEE (Exchangeable,QIC=15141.01, QICC=15054.85). 118

Table 3.41 Odds ratios (ORs) of inefficiency sleep and their 95% confidence intervals for each factor and Daily dose adjusted for all other factors using GEE (exchange, QIC=6297.93, QICC=6275.83). 119

Table 3.42 Odds ratios (ORs) of sleep problem and their 95% confidence intervals for eachfactor and daily dose adjusted for all other factors using GEE (exchange, QIC=13194.68, QICC=13085.02). 119

Table 3.43 Odds ratios (ORs) of morning sleepy and their 95% confidence intervals for each factor and daily dose adjusted for all other factors using GEE (exchange,QIC=12203.98, QICC=12125.19). 120

Table 3.44 Odds ratios (ORs) of poor sleep and their 95% confidence intervals for each factor and daily dose adjusted for all other factors using GEE (exchange,QIC=13610.26, QICC=13535.37). 121

Table 3.45 Odds ratios (ORs) of doze and their 95% confidence intervals for each factor and daily dose adjusted for all other factors sing GEE (AR1,exchange, QIC=11349.42, QICC=11268.01). 122

LIST OF FIGURES

		Page
Figure 1.1	Visual chart of knowledge on electromagnetic waves	5
Figure 1.2	Electromagnetic spectrum categorized by wavelength and frequency	6
Figure 1.3	Wireless cellular communication(adapted from google)	7
Figure 1.4	Pain suppression system	20
Figure 1.5	Areas with headaches from various causes	21
Figure 1.6	Sleep architecture	41
Figure 1.7	Reticular formation	43
Figure 1.8	Ascending reticular activating system, monoaminergicnerves and cholinergic neurons	44
Figure 1.9	Cholinergic, aminergic, orexinergic and VLPO nerves	45
Figure 1.10	Flip –flop switch control	46
Figure 1.11	Theoretical conceptual framework	58
Figure 2.1	Flow of sampling divided by class and gender	60
Figure 2.2	The signals measurement of spectrum analyzer	70
Figure 3.1	The sampling procedure and sample response flow chart	75
Figure 3.2	Factors contributing to poor sleep presented as odds ratio adjusted for all factors using multiple logistic regression.	96
Figure 3.3	The sampling procedure and sample response flow chart.	98
Figure 3.4	Smartphone output power100 groups	102
Figure 3.5	Proportion of headache by 100 groups of smartphone output power	102

LIST OF ABBREVIATIONS

ANOVA	Analysis of Variance
ADL	*Activities of Daily Living*
ARAS	Ascending Reticular Activating System
BMI	*Body Mass Index*
BS	BaseStation
CBBs	Calcium-channel blockers
CI	Confidence interval
CSE	CumulativeSpecific Energy
DALY	*Disability-Adjusted Life Years*
DNA	Deoxyribonucleic Acid
EEG	Electroencephalography
EHT	Environmental Health and Toxicology
ESS	Epworth Sleepiness Scale
FDMA	Frequency Division Multiple Access
GABA	Gamma-Aminobutyric Acid
GEE	Generalized Estimating Equations
GLM	Generalized Linear Models
GHz	Gigahertz
HAD	Hospital Anxiety and Depression
HAMP	Headache Associated Mobile Phone
HIT6	Headache Impact Test
ICHD	*International Classification of Headache Disorders*
ICHD-3 beta	*International Classification of Headache Disorders* into Edition 3
ICSD	International Classification Sleep Disorder
IHS	International Headache Society
kHz	Kilohertz
LC	*Locus Coeruleus*
LDT	Laterodorsal Tegmental Nucleus

LED	*Light Emitting Diode*
LEV	Levetiracetam
MHz	Megahertz
MIDAS	Migraine Disability Assessment
MP	*Mobile Phone*
MTSO	Mobile Telephone Switching Office
NAR	*Non-Allergic Rhinitis*
NREM	*Non-Rapid Eye Movement*
OR	*Odds Ratio*
PedMIDAS	Pediatric Migraine Disability Assessment
PAG	Periaqueductalgray
PET	*Positron Emission Tomography*
PSG	Polysomnography
PSIQ	Pittsburgh Sleep Quality Index
PTIEs	Potentially traumatic interpersonal events
QIC	Quasi-likelihood under Independence Model Criterion
QICC	Corrected Quasi-likelihood under Independence Model Criterion
RAS	*Reticular Activating System*
rCBF	regional Cerebral Blood Flow
REM	*Rapid Eye Movement*
RF	*Radio Frequenc*ies
RF	*Reticular Formation*
RR	*Risk Ratio*
SAR	Specific Absorption Rate
SCN	Suprachiasmatic nucleus
SER	SmartphoneElectromagnetic Radiation
SOP	Smartphone Output Power
SWS	Slow-wave sleep
TDMA	Time Division Multiple Access
TTH	Tension-type headaches
TMN	Tuberomammillary nucleus
T-Test	Student's t-test

UMTS	Universal Mobile Telecommunications System
UV	Ultraviolet
VLPO	Ventrolateralpreoptic neurons
V/m	Volts per meter
WHO	World Health Organization
YLD	Years of Life Lost due to Disability

LIST OF SYMBOLS

μ	MOR: opioid receptors
δ	Delta: opioid receptors.
κ	KOR: opioid receptors.
$\sum_{n=1}^{n}$	Summation

GLOSSARY

ANOVA	Analysis of Variance: Statistical models used to analyze the mean differences
ADL	Activities of Daily Living: People's daily self care activities Assessment
ARAS	Ascending Reticular Activating System: a set of connected nuclei in the brains of vertebrates
BMI	Body Mass Index: value derived from the mass (weight) and height of an individual
BS	Base Station: a land station in the land mobile service.
CBBs	Calcium-channel blockers:Calcium blockers drug
CI	Confidence interval: a type of interval estimate of a population parameter that is computed from the observed data
CSE	Cumulative Specific Energy: Total cumulative energy Absorption
DALY	Disability-Adjusted Life Years: a measure of overall disease Burden
DNA	Deoxyribonucleic Acid: Molecule that carries the genetic Instructions
EEG	lectroencephalography: an electrophysiological monitoring method to record electrical activity of the brain.
EHT	Environmental Health and Toxicology: multidisciplinary field of science concerned with the study of the harmful effects of various chemical, biological and physical agents on living organisms.
ESS	Epworth Sleepiness Scale: the questionnaire for measuring daytime sleepiness

FDMA	Frequency Division Multiple Access: a channel access method used in multiple-access protocols as a channelization protocol that allocation of one or several frequency bands, or channels
GABA	Gamma-Aminobutyric Acid: the chief inhibitory neurotransmitter in the mammalian central nervous system.
GEE	Generalized Estimating Equations: statistics estimate the parameters of a generalized linear model with a possible unknown correlation between outcomes
GLM	Generalized Linear Models: statistics estimate the flexible generalization of ordinary linear regression that allows for response variables that have error distribution models other than a normal distribution
GHz	Gigahertz: The derived unit of frequency in the International System of Units hertz
HAD	Hospital Anxiety and Depression: Anxiety and Depression assessment questionnaire
HAMP	Headache Associated Mobile Phone: Headache attack during or after using mobile phone
HIT6	Headache Impact Test: Headache Impact assessment questionnaire
ICHD	International Classification of Headache Disorders:a detailed hierarchical classification of all headache related disorders
ICHD-3 beta	International Classification of Headache Disorders into Edition 3: detailed hierarchical classification of all headache related disorders Edition 3
ICSD	International Classification Sleep Disorder: a primary diagnostic, epidemiological and coding resource in the field of sleep and sleep medicine

HIS	International Headache Society: the world's leading membershiporganization for those with a professional commitment to helpingpeople affected by headache
kHz	Kilohertz: the derived unit of frequency in the International System of Units equal to one thousand hertz
LC	Locus Coeruleus: a nucleus in the pons of the brainstem involved with physiological responses to stress and panic.
LDT	Laterodorsal Tegmental Nucleus: a nucleus situated in the brainstem, spanning the midbrain tegmentum and the pontine tegmentumsends cholinergic (acetylcholine) projections to many subcortical and cortical structures.
LED	Light Emitting Diode: two-lead semiconductor light source.
LEV	Levetiracetam: a medication used to treat epilepsy
MHz	Megahertz: the derived unit of frequency in the International System of Units equal to one million hertz
MIDAS	Migraine Disability Assessment: questionnaire of migraine disability assessment
MP	Mobile Phone: a wireless handheld device that allows users to make calls and send text messages, among other features
MTSO	Mobile Telephone Switching Office: the mobile equivalent to a Public Switched Telephone Network Central Office
NAR	Non-Allergic Rhinitis: condition causing profuse chronic watery rhinorrhoea
NREM	Non-Rapid Eye Movement: collectively, sleep stages 1–3, previously known as stages 1–4

OR	Odds Ratio: a measure of magnitude of association between exposure and outcome
PedMIDAS	Pediatric Migraine Disability Assessment: questionnaire of pediatric migraine disability assessment
PAG	Periaqueductal gray:the primary control center for descending pain modulation
PET	Positron Emission Tomography: a nuclear medicine functional imaging technique
PSG	Polysomnography: a multi-parametric test used in the study of sleep
PSIQ	Pittsburgh Sleep Quality Index: Sleep quality assessment
PTIEs	Potentially traumatic interpersonal events: a mental disorder that can develop after a person is exposed to a traumatic event, such as sexual assault, warfare, traffic collisions, or other threats on a person's life
QIC	Quasi-likelihood under Independence Model Criterion: criteriato choose between two correlation structures for goodness of fit
QICC	Corrected Quasi-likelihood under Independence Model Criterion: criteria to choose between two sets of model terms for goodness of fit
RAS	Reticular Activating System: a set of connected nuclei in the brains of vertebrates that is responsible for regulating wakefulness and sleep-wake transitions.
rCBF	regional Cerebral Blood Flow: the amount of blood flow to a specific region of the brain
REM	Rapid Eye Movement: a unique phase of sleep in mammals and birds, characterized by random/rapid movement of the eyes
RF	Radio Frequencies: any of the electromagnetic wave frequencies that lie in the range extending from around 3 kHz to 300 GHz

RF	Reticular Formation: Set of interconnected nuclei that are located throughout the brainstem.
RR	Risk Ratio: an intuitive way to compare the risks for the two Groups
SAR	Specific Absorption Rate: measure of the rate at which energy is absorbed by the human body when exposed to a radio frequency
SCN	Suprachiasmatic nucleus: a tiny region of the brain in the hypothalamus, situated directly above the optic chiasm. It is responsible for controlling circadian rhythms.
SER	Smartphone Electromagnetic Radiation: Electromagnetic Radiation from Smartphone
SOP	Smartphone Output Power: output power that measured from smartphone
SWS	Slow-wave sleep: referred to as deep sleep, consists of Stage three of non-rapid eye movement sleep
TDMA	Time Division Multiple Access: a channel access method for shared-medium networks in the same frequency channel by dividing the signal into different time slots.
TTH	Tension-type headaches: The primary headache which pain radiate from the lower back of the head, the neck, eyes, or other muscle groups in the body
TMN	Tuberomammillary nucleus: histaminergic nucleus located within the posterior third of the hypothalamuswhich is involved with the control of arousal, learning , memory, sleep and energy balance.
T-Test	Student's t-test : any statistical hypothesis test in which the test statistic follows a Student's *t*-distribution under the null hypothesis
UMTS	Universal Mobile Telecommunications System: a third generation mobile cellular system

UV	Ultraviolet: an electromagnetic radiation with a wavelength from 10 nm to 400 nm
VLPO	Ventrolateral preoptic neurons: a small cluster of neuronssituated in the anterior hypothalamus has a key role in sleep
V/m	Volts per meter: unit of electrical intensity
WHO	World Health Organization: a specialized agency of the United Nations that is concerned with international public health
YLD	Years of Life Lost due to Disability: the number of years disabled weighted by level of disability caused by a disability or disease

ข้อความแห่งการริเริ่ม

1. สมาร์ทโฟนเป็นเทคโนโลยีที่ทันสมัยตอบสนองความต้องการที่หลากหลายต่อกลุ่มวัยรุ่นทำให้มีการใช้เพิ่มขึ้นอย่างรวดเร็ว การศึกษาพบว่าการแผ่รังสีแม่เหล็กไฟฟ้าจากสมาร์ทโฟนมีผลกระทบต่ออาการปวดศีรษะและการนอนหลับยาก

2. การศึกษาพบว่าอาการปวดศีรษะจากการใช้โทรศัพท์เคลื่อนที่ควรถูกจัดเป็น secondary headache และ output power จากสมาร์ทโฟนมีลักษณะเป็น trigger ต่อไมเกรน

3. การวัดการสัมผัสการแผ่รังสีแม่เหล็กไฟฟ้าจากสมาร์ทโฟนในรูปแบบของ Specific Absorption rate เป็นการคำนวณที่ยุ่งยาก ซึ่งคำนวณจากค่า output power สูงที่สุดของสมาร์ทโฟน การวัด output power จากสมาร์ทโฟน จึงเป็นการประเมินการสัมผัสระดับบุคคลที่ใช้ในการศึกษาทางระบาดวิทยา ดังนั้นการวัด output power ที่มีการวัดและบันทึกอย่างต่อเนื่องโดยการใช้ application ที่ติดตั้งเพิ่มในสมาร์ทโฟนจึงเป็นวิธีการใหม่ที่ทำให้สามารถทำการศึกษาทางระบาดวิทยารูปแบบ time series ในมนุษย์

STATEMENTS OF ORIGINALITY

1. The study focus on smartphone, which is a modernize technology to meet many needs of adolescents. As a result, adolescents have high rates of mobile phone possession and use. The study found the effect of electromagnetic radiation from smartphoneon headache and difficult sleep.

2. The study found the mobile phone associated headache should be classified into secondary headache and smartphone output power was classified as a trigger for migraine.

3. Measurement of exposure to electromagnetic radiation with specific absorption rates (SAR) in human heads is difficult and vague. Specific absorption rate is calculated by using maximum output power of smartphones. Smartphone output power is assessment of individual exposure to electromagnetic energy that is important for epidemiological study. Application which was installed in smartphone, use for continuous record smartphone output power is the new method for time series design of epidemiology study.

CHAPTER 1

Introduction

1.1 Background

The development of the industrial and economical work usually needs technological development, especially communication. Currently, mobile phones are popularly used to meet people's various needs. The social, cultural, economic, political, and educational changes in every part of the global society create borderless world which is linked systematically and internationally. Mobile phones will encourage small business growth[1] by creating electronic commerce, educational system development and increasing in perception[2] leads to thinking processes improvement which will help us to solve problems accurately and quickly.[3] Furthermore, mobile phones will save people for emergency situations,[4] enhance family relationship[5,6] and, the most important, provide many forms of entertainment which will increase the demands quickly. The communication hardware industry has been found as one of the top four technological markets in the world with continually rising trends. Mobile phone sales in 2016 were expected to be as high as 2.6 billion or 1.4 devices/person.[7] In 2014, mobile phones use were more than 50% of all electrical appliances in the world.[8] Moreover, telephone expenses throughout the world were increased to 105.9 billion in 2011 with the expectations of reaching 712 billion in 2017.[9]

In Thailand, the overall values of the communication markets in 2011 grew by 9.2% and the smartphone market was forecasted to increase by 24.9%.[10] Students aged six years old and more possessed smart phone (72%) with trends of computer, internet and mobile phone usage increased from 36.7% in 2005 to 66.4% in 2012. The population aged between 15-24 years old is the group who has used the highest computer, internet and mobile phone usage.[11] The significant data has shown Thai adolescents have a long duration time talking on mobile phone, making them the first rank in Asia with a mean of 60.7 minutes per day.[12]

Mobile phones function as wireless cellular communication systems.[13,14] Currently, mobile phones have been developed and improved the functional capacity into what we called "smartphone".[15,16] Smartphones use electromagnetic wave transmission, microwave frequencies, from the antennas to destination's signal numbers via control channels to the base station (BS). Then the base station will send the requested signals to the mobile telephone switching office (MTSO) in order to search and connect with the destination unit. However, if smartphone are unable to detect signal channels during communication, smartphone will display "no service" during conversations. If smartphone move from one station's service radius to others, the destination smartphone will switch to use the signal channels of the base station in the new cell or "hand over" in order to be consistent.[13,14] Assessment of the levels of contact with electromagnetic radiation and the amount of smartphone energy absorbed by tissues are assessments of smartphone output levels,[17-19] based on the time smartphone are in use.[18]

While electromagnetic radiation emitted by smartphone is non-ionizing, it has sufficient energy to excite electrons and cause two biological effects including thermal and non-thermal.[20-21] Smartphones are sources of electromagnetic energy located closest to users' heads.[22-24] Although the emitted electromagnetic energy is below the maximum standard value that can affect human nerves as the nerves are electrical parts of human bodies and far from being in a state of equilibrium.[21-22, 25-26] Furthermore, contact with low-intensity electromagnetic radiation can also excite or lead to changes in nervous system functions,[23,25-27] this will lead to either biological effects or symptoms of nervous system related to headache[22] and sleep problems. Most studies of the health impact of using mobile phones have found fatigue (42-45%), memorial problem (15-40.6%), sleep (38.8%), hearing (23.1%), concentration (34.3-52%), heat around the ears (28.3-50%) and headache (16.1-65%) to the most frequently encountered symptoms.[28-31]

1.2 Rationale

1.2.1 Headache and smartphone

Headache is a type of pain referred to the surface of the head from deep structures in the head caused by changes in the skull such as blood vessel and nerve excitement. Excitement outside the skull such as meningitis, lower spinal fluid pressure, migraine, consumption of alcohol beverages, and nose and eye irritation.[32] Headache is the most

severe symptom found among the group of neurological disorders and problems throughout the world[33] with some changes according to the area. Headache can be found in children more than adults. The prevalence of headache among preschool-aged children has been found at 3-7%, 37-51% of elementary school children, and 57-82% of high school students.[34] Migraine prevalence has been found at 7.7% and tension-type headache has been found at 52%.[33] Headache among adolescents has been found to have higher prevalence from 47-82% in 2006-2012[34-39] to 83-94% in 2013-2015[40-43] with effects on daily life, education, and quality of life.[36, 41, 44-46] Therefore, the managements of controlling headache are important for taking care of persons with headaches or adapt to the co-morbidities of headache such as depression, which has been found three times more than migraines population, etc.[47] Headache is related to many factors including pathology and psychology. Thus, the study is also interested in electromagnetic energy from mobile phones causing headache.

Adolescents have some of the highest mobile phone possession rates,[11] while mobile phones are sources of electromagnetic energy emissions used close to the head.[22, 24] Studies followed the groups that use mobile phones have been found patients with migraines and dizziness to have higher standardized hospitalization ratios by 10-20%.[48] Furthermore, the study conducted by Chu et al. concluded that headache from mobile phones was secondary headache.[49] Electromagnetic radiation from smartphone excite or lead to chemical and biological changes in cells,[27] causing presenting symptoms and behaviors which are due to changes in nervous system function.[28]

1.2.2 Sleep problems and smartphone

Good sleep is similar to food, drinking water, and fresh air which is necessary for physical growth, behavioral, emotional, and learning development among adolescents.[50-53] Sleep is a state with changes in mobility, movement, and perception that is different from unconsciousness or lack of feeling because sleeping state can return to normal. Sleep is a physiological process with functions at various levels of central and peripheral nerves with hormone secretion.[54]

Sleep problems in adolescents occur throughout the world. The National Sleep Institute in the United States has found prevalence of sleeping problem in children and adolescents at 25% and 40%.[55, 56] The study conducted in multiple countries by Mindell

in 2008 found prevalence of sleeping problem at 25-40%[55] and sleeping problem prevalence increased from 16.9-54.2% in 2000-2013[51, 55, 57-59] to 58.7-66% in 2014-2016.[53, 60] The thesis focuses on adolescents whose sleep patterns depends on many factors. Furthermore, personal factors including gender, grade level, family, school, social culture, environment, and sleep hygiene[51-53, 57, 61-62] have affected to sleep architecture combined with declining slow wave sleep in NREM sleep. These lead to changes in homeostatic and circadian regulation of sleep.[63]

Therefore, sleep problems among adolescents have influenced on perception and learning ability, which result in low academic performance,[64-66] substance abuse, consumption of caffeine, alcohol beverages leading to more potential accidents.[67-69] In addition, sleep problems can cause drowsiness during daytime (35-40%), health effects, and chronic diseases.[64, 70-72] Sleep problems are one of the risk factors of death (RR: 1.12; 95% CI 1.06-1.18)[73] that also have affected to psychological problems, stress, and depression.[74-75] Thus, adequate sleep is essential for a good health and quality of life.[53] Adolescents are currently growing up in the era of modern technology[76] with electrical devices and electronic media mostly found in adolescents' bedrooms (75%).[77] The uses of electronic media and mobile phone for conversation before sleeping have related to sleep problems (77%).[64, 77-84] Therefore, the uses of these technologies are an environmental factor that will influence sleep.

Health impacts from the electromagnetic energy emitted from smartphone remain inconclusive. Usage of mobile phones increases on occurrence of direct contact of brain with electromagnetic radiation, especially for children and adolescents in the "digital era"[76] whose possession of smartphones become continually rising trends. For this means, children and adolescents are at high risk and studies of health impacts aiming at creating precautionary protection are necessary.[85]

1.3 Aim:

The study aimed to investigate the relationship between the effects of smartphone radiation on headache and sleep quality among high school students. Secondary objectives are the followings.

1.3.1 To study the characteristics of mobile phone use, headache symptom, and sleep quality among high school students.

1.3.2 To study the correlation between smartphone output power and headaches among high school students.

1.3.3 To study the correlation between smartphone output power and sleep quality among high school students.

1.4 Theory and literature review

1.4.1 Theory and literature review for electromagnetic radiation from smartphone.

1) Electromagnetic waves

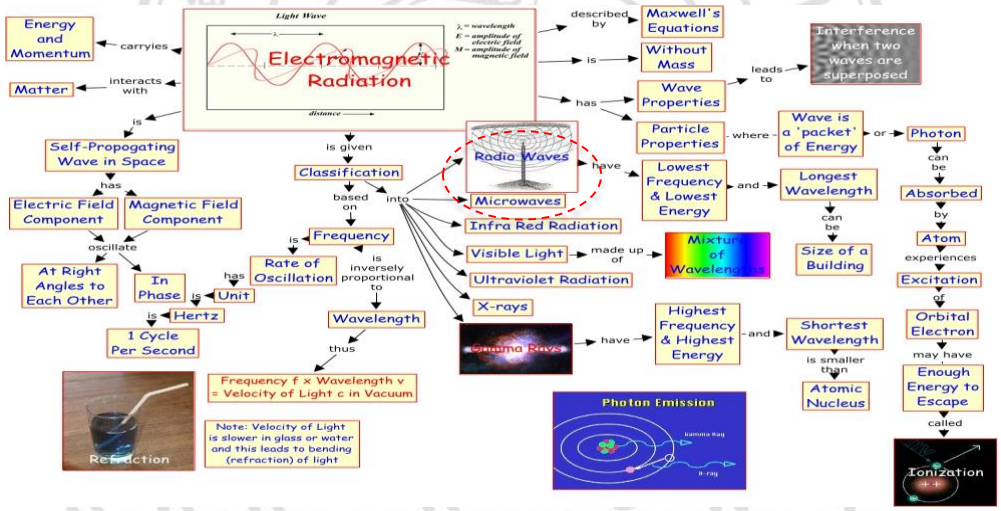


Figure 1.1 Visual chart of knowledge on electromagnetic waves**.**
Source**:** IHMC Camp tool, website *http://cmapspaceexpihmcus/rid=1162335457928_93187048_ 8202/25%20 Electromagnetic Radiationcmap*; [cited 2014].

Electromagnetic energy propagates in the form of electromagnetic fields compare to hypothetical lines indicating areas showing the intensity of energy consisting of the line of force of electric and magnetic or electric and magnetic fields.[86-88] The Maxwell equation explained the electromagnetic fields. For the theory, the movement of electric charges produces magnetic fields. Furthermore, the theory predicts that when magnetic

field change, electric field is also changed.[20, 89] Electromagnetic radiations will be created from an oscillation of charges. The radiation consists of wave of electric and magnetic wave that oscillate perpendicularly to one another. It can transverse the atmosphere while transmitting energy.[20] Electromagnetic waves are categorized by their spectrum which is known as energy power of each frequency. The frequency is employed to describe the manner of the electromagnetic signal in term of how often the signal oscillates per second. Additionally, the unit of frequency called Hertz[20, 88] The electromagnetic radiation used with smartphone are microwaves frequency of the electromagnetic spectrum with frequency ranging from 1-10 GHz as a specific group of radio frequencies (RF) ranging from 3 kHz to 300 GHz.[86, 90]

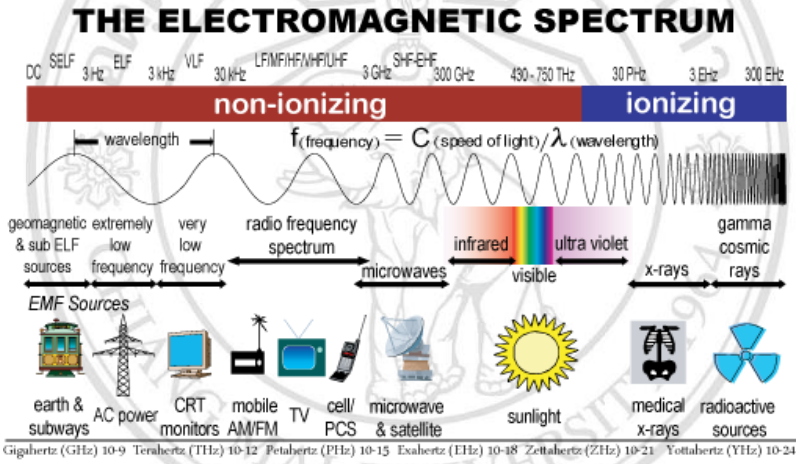


Figure 1.2 Electromagnetic spectrum categorized by wavelength and frequency. Source: Study.com, website: *http://studycom/academy/lesson/what-are-gamma-rays-definition-examples-quizhtml,* [cited 2014].

2) Wireless telecommunication systems

Smartphone is a communication device developed to have more working capacity that communication equipment is called "smartphones".[15-16] Smartphones have different characteristics from ordinary phones. Also, it is similar to portable computers function on mobile phones[15] with system software[16, 91-93] capable of connecting the main capabilities of mobile phones and support applications or applied programs,[94] install additional programs to increase phone capabilities, depend on the phone's operating system. Furthermore, smartphones can connect with other devices while support multimedia files in various forms of mobile phone applications such as messages, images

and motion pictures, etc.[16] Therefore, smartphones have met the online users' social needs in the digital age with higher usage trends.[91, 93]

Smartphone as the communication equipment connects or sends the signal to the destination through the communication network with main components[95,96] consisting of the mobile phone called mobile units, the base station (BS) and the mobile telecommunications switching office (MTSO) with the following functions:

2.1) Smartphones transmit microwaves from the antenna in smartphones signals to the destination number via control channels to the BS and the BS sends requested signals to the MTSO to search and connect with the destination unit. However, if smartphones cannot find signal channels during communication, smartphones will show a "no service" message.[13, 20, 96]

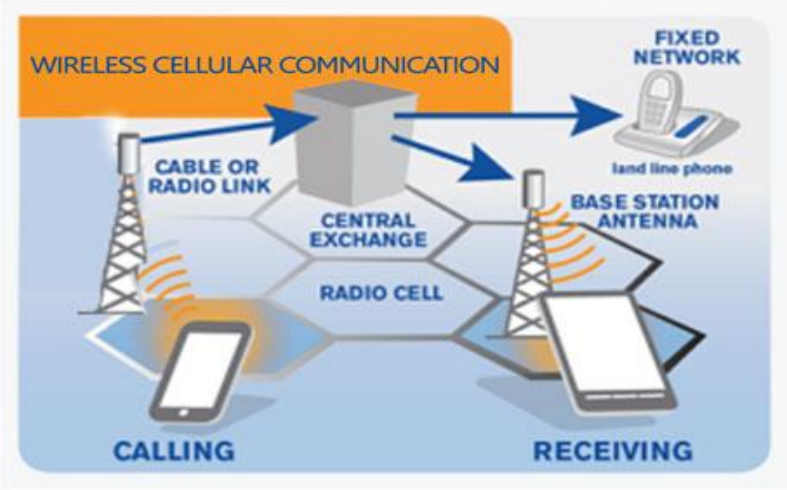


Figure 1.3 Wireless cellular communication. Source adapted from google

2.2) Receiving smartphones send a response signal to the BS and transmit to the MTSO and create a communication channel that is a communication circuit for the caller and recipient. During conversations, the BS and the MTSO will connect signal channel for receiving and sending information.[13, 20, 96]

2.3) During conversations, if smartphones move from the original BS to other cell areas, the mobile phone will automatically switch to use the signal channels of the new BS without interrupt the conversation or dropping the call. This is called a "hand over".[19, 23] Base station coverage areas with sufficient signal transmitter-receivers during movement are needed to prevent communication problems.[13, 20, 96]

The wireless communication system consists of GSM (Global System for Mobile Communications),[20, 95, 96] for example, Frequency Division Multiple Access (FDMA) The TDMA and Universal Mobile Telecommunications System (UMTS).[20, 95, 97]

3) Measurement of electromagnetic energy from smartphones enters the human body.

3.1) Specific absorption rate (SAR)

Measurement of exposure toward electromagnetic energy with health impacts is difficult and vague. Most quantified exposure levels is popularly described the energy defined by FCC in the form of SAR in human heads.[17, 89, 99] The study conducted by Cadis et al.[100] calculated Cumulative Specific Energy (CSE) with units in Jules per kilogram (J/kg) by calculating the total sum of SAR with cumulative time of mobile phone use. Therefore, cumulative SAR distribution in brain tissues is the level of cumulative exposure time to mobile phone use in a lifetime with chronic health impacts such as tumors.[101]

Specific Absorption Rate is a measurement of dose rates for electromagnetic energy absorption (dW) per body mass (dm) at the specified volume (dV) of density (ρ)[102] or equal to the rate of electromagnetic energy emitted from antennas to the head or other parts of the body.[102, 103] SAR is usually calculated in the frequency ranges of 100 MHz and up.[18] SAR related to increase in temperature with units being absorbed energy/mass such as Watt per gram (w/1g) or Watt per gram (W/10g).[102] SAR at 4W/kg increases temperature by 1ºc.[104]

Gogineni calculated SAR in human heads with different values based on brain area. The occipital region has been found to have the highest special peak SAR (1g) at 1.86mW/kg, 0.668mW/kg at the parietal area and 0.251mW/kg at the frontal area. Use of telephones in talking mode has more output power than the standby mode with the differences based on electromagnetic wave frequency on Table 1.1. Use of telephones in talking mode has higher SAR than every frequency.[20]

Table 1.1 Special peak average SAR categorized according to mode of phone use and frequency[20]

Frequency (MHz)	Talking Mode(200mW)		Standby Mode(20mW)	
	Spatial Peak Average (1g)(W/kg)	Spatial peak Average (10gW/kg)	Spatial Peak Average (1g)(W/kg)	Spatial peak Average (10g)(W/kg)
900	0.0825	0.0476	0.00825	0.00476
1800	0.177	0.141	0.0177	0.0141
2200	0.311	0.229	0.0311	0.0229

Source: Adapted from Gogneni, thesis 2012, p. 51-52.

In the area of standard values of limiting exposure to electromagnetic fields, international organizations in the area of electromagnetic fields are aware of health impacts and, therefore, specified standards and regulations to limit exposure to electromagnetic waves, create safety and comply with precautionary protection principles by limiting electromagnetic radiation from various devices not exceed standard criteria. Each organization has different standard values according toTable 1.2.[22] Furthermore, the American National Standards Institute/Institute of Electrical and Electronics Engineers (ANSI/IEEE) defined time to expose at a mean of 30 minutes while other organizations specified exposure times at only six minutes.[22] Member countries without regulatory standards use limitations in line with the International Commission on Non-Ionizing Radiation Protection (ICNIRP) guideline.

Table 1.2 Public SAR standard values and functions according to international Organizations[22]

Standard	Local SAR in head (W/kg)	
	Public	Occupational
ARPANSA	2	10
Safety Code	1.6	8
ICNIRP	2	10
FCC	1.6	8
NRPB	10	10
ANSI/IEEE	1.6	8

Source: Lu Y, Huang Y 2012, p. 1.

3.2) Smartphone output power

Specific absorption rates in head tissues of smartphone users relate to the process of causing heat from electromagnetic energy to tissues.[38, 101] Specific absorption rates are calculated by using maximum output power of smartphones. Smartphone output power directly relates to exposure.[100] Therefore, smartphone output power is an important indicator in assessing electromagnetic energy outside the body related to exposure levels and the amount of energy absorbed in tissues,[17-18] which depends on the duration of smartphone use.[18] Therefore, time of repeated exposure is an important variable for estimating exposure to electromagnetic energy[17] and related to the amount of energy absorbed in tissues.[17, 19] Assessment of individual exposure to electromagnetic energy is important for epidemiological study to be reference data and generalize to the population at risk.[99]

The smartphones have different output power in accordance with the production standards regulated and safety systems. The BS has a power control system to minimize the output power of mobile phones with good signal reception[101, 105-106] by modifying the system to control signal strength in the area[105] depending on network efficiency or capacity. Therefore, output power from smartphones changes according to conversation time.[18, 107]

3.2.1) Factors related to output power from smartphone

(1) The power control system modifies mobile phone output power based on signal strength in the area.[108] Power control involves 15 steps. Each step reduces output power by two decibels (dB) per time according to the European Telecommunications Standards Institute.[17,19, 99,105, 108]

- GSM 900 from 33 to 5dBm
- DSC 1800 from 30 to 2dBm

For conversations near the base station (BS), output power from smartphone is low (GSM 13dBm; 0.02W and 5dBm; 0.003W) while maximum output power is 2W.[105-106] The power of control system modifies output power from smartphones down to 1mW during conversations.[99] Therefore, smartphone output power based on duration of smartphone use and power control systems.

(2) Hand-overs are changes in the signal channels of the BS because of movement during conversations.[19] For hand overs, smartphones will use

maximum output power for starting connection. Therefore, handovers increase mean output power of smartphone.[17, 106, 108]

(3) The density of base stations in rural areas is low because the distance between base stations is so long as the output power strength in rural will be higher than urban.[17, 19, 99, 105, 108]

(4) Telecommunication system, for example 1800 and UMTS presented by figure 4, The UMTS is the lowest output power and out of fashion.[19, 99, 101, 107-108]

The potential relevant factor of the smartphone output power consisted of the power control system depending on operator networks, wave frequency and signal strength related to base station density, distance from the base station and population density. Furthermore, output power in each country is different.[17] The rural areas in the United Kingdom, output power has been found at 2W nearly the entire time when smartphones are used while output power at 2W less than 70% of time used in Sweden.

3.2.2) Output power measuring method

(1) Measuring instruments

(1.1) Output power is measured by using a test mobile system (TEMS). TEMS is a computer program modified to measure smartphone power emissions (TXPOWER) and record information of mobile phone use between the base stations to provide data and cumulative distribution.[106]

(1.2) SYNEHA2 (System Network and Handset Analyzer Version2) is a standard tool for measuring real time output power[19,109] but it required many tools to measure the large samples.[19]

(2) Software or commands in the system

(2.1) The Telia software[99] of the GSM network is a software used by operator network to record distribution of output power levels connected to smartphones and mobile traffic recording,[17] especially in the GSM system.

(2.2) Software in smartphone is linked to computers and used to monitor networks, assessed signal strength and sent information on smartphones output power to linked computers.[105]

(2.3) Software modified phones (SMP) is a smartphone with modified software or command sets of recording data of addresses and can be used normally. Command sets record dates, times and length of each conversation in addition

to record frequency and output power levels.[19, 108] The method is convenient and easy for exposing the assessment of electromagnetic energy. The study conducted by Kelsh (2011) found data from SMP related to Gold Standard SYNEHA.[19, 101] Later on, hardware modified phones were developed with additional smartphone device modifications enabling phones to record changes of angles in smartphone use and power control levels to provide data for more accurate SAR analysis.[110]

3.2.3) Output power calculation

Most studies of output level measurements calculated output power in many ways according to research objectives. Most of the aforementioned studies have been aimed to study the factors influencing mobile phone output levels with the following calculations:

(1) In the area of mean output power per telephone conversation, Vrijheid et al.[108] calculated the mean power per telephone conversation and compared to each group of time spent on telephone conversation, in addition to compare output power to studied factors.[18-19, 106]

(2) Time percent of maximum output per call was calculated in terms of percent of time with maximum output of each call found by Vrijheid et al.[108] and Wiart et al.[106] This resulted in hand-overs and comparison between areas or activities between telephone use.[17]

(3) Level of crossing rate is the level of signals exceeding specified levels per time unit and average duration fading, also a consideration of mean time with power higher than specifications. Therefore, mentioned measurement relates to communication engineering found in the study conducted by Vrijheid et al.[108]

Epidemiological studies relate to smartphone use mostly assessing exposure roughly by using questionnaires and interviews relate to time and frequency of smartphone use. Lack of exposure assessment indicates the quantity of electromagnetic energy in individual exposure. Accurate exposure assessment is important epidemiological studies to study the relationship between quantity of exposure and health impacts (dose-response) in addition to expose assessment accurately to prevent bias and misclassification.[110] Measurement of specific absorption rate is an assessment under the maximum exposure in order to guarantee safety in contacting electromagnetic energy emitted from smartphone use. Furthermore, SAR assessment must use expertise, complex

and time-consuming methods that cannot be measured or calculated by using normal instruments or laboratory examinations.[111] In real situations, normal use of smartphone does not result in maximum exposure with output power at all times. Therefore, exposure assessment with electromagnetic energy from mobile phones must be made from output power of mobile phones according to time when mobile phones used and factors related to mobile phone output power in the areas of networks, wave frequency, phone model, signal strength and handover, all are data on the truth quantity of exposure with electromagnetic energy.[19]

4) Research on output power from smartphone

4.1) Lonn et al.[99] found rural areas to have maximum output power at 2W with a frequency of 900 MHz or 50% of smartphone time at the lowest power. Urban areas used maximum output at 25% and minimum output at 22% and output power was lower in daytime.

4.2) d'Amor et al.[18] measured electric field at 2 volts per meter (V/m) and found output power at 2mW while electric fields changed to 0.001 volts per meter (V/M) can increased output power by more than 100 times.

4.3) Hillert et al.[17] found that high mobile phone output power was more frequent in rural areas whereas the other factors (length of call, moving/stationary, indoor/outdoor were less importance.

4.4) Nikolay et al.[105] found smartphone user hand and head positions to increased smartphone output power by 12-14dBm in men and 10-12dBm in women because head and hand positions cause signal strength to change.

4.5) Kelsh et al.[19] revealed that phone technology and, to a lesser extent, degree of urbanization were two times stronger influences on smartphone signal.

1.4.2 Electromagnetic radiation emissions from smartphone and biological impacts.

Electromagnetic radiation is divided into two groups consisting of ionizing and non-ionizing radiation with different processes causing impacts on the body as follows:

1) Ionizing radiation such as x-ray or gamma radiation has sufficient energy to pull electrons from atoms or connecting molecules, causing biological tissue damaged including changes in DNA.

2) Non-ionizing radiation such as electromagnetic radiation from radio frequencies, microwave frequencies, UV lights, etc., have insufficient energy to pull electrons from atoms and molecules while have sufficient energy to excite electrons.

Microwave Frequency Radiation (MFR) used with smartphones are non-ionizing radiation with two biological effects, namely, thermal effects and non-thermal effects.[20, 21]

2.1) Thermal effects are caused by increasing heat, especially exposure to high quantity of MFR radiation. Energy has absorbed into biological tissue, a component of water, will be converted into thermal energy then thermal will increase[112] quickly and reach over one degree Celsius because biological heat control systems cannot reduce internal body heat to a balanced level, causing tissue damage during MFR exposure. Heat distribution is not directly related to SAR distribution[44] but also involves heat increasing in the biological tissues.[27]

2.2) Non-thermal effects are caused by exposure with electromagnetic energy which is an insufficient energy for inducing temperature control systems in the body to increase heat or temperature.[113] The body humans have internal control which function under electrical processes that are interfered from electromagnetic waves.[21] Electromagnetic waves lead to changes of cells at the molecular level or tissue level causing chemical and biological changes in cells,[27] including structure and functions in cells.[27] Furthermore, skin tissues have a circuit characteristic. When encountering high frequency electricity, skin tissues of nerves in the brain will be excited.[27] However, non-thermal effects continue to have unclear data and arguments.[21-23, 27] Non-thermal effects usually have exposed with electromagnetic energy of intensity below 10 $\mu W/cm^2$ with long time expose.[114]

Smartphone are sources of electromagnetic energy used most closely to the head, even though electromagnetic emissions are below the highest specified standard value.[22-24] Even with nervous systems in the human, which are electrical tissues and unstable, contact with low-intensity electromagnetic energy can excite or induce changes to nervous system functions.[25-26]

1.4.3 Biological impacts on the nervous system

Nervous systems are electrical organs. Therefore, exposure of electromagnetic energy usually leads to nervous system changes[63] with the following potential neurological impacts on the nervous system:

1) Impacts on the Blood-Brain Barrier (BBB). BBB is a part separating blood circulation from fluids outside brain cells[115] and functions to prevent the spread of hazardous molecules in the central nervous system. Most studies have found absorption of large amounts of high-intensity electro-magnetic energy by experimenting in experimental animals at intensity levels of 3-30W/cm^2 have created heat and increased brain temperature by 43°c. Higher heat is a significant factor causing BBB to allow increasing in absorption,[116] leads to cerebral edema, abnormal pressure in the brain, and brain damage.[25, 116] However, Frey et al. found higher fluorescent in the brains of experimental rats injected with dyes in the blood by using low-intensity electromagnetic energy at the frequency of 1,200 MHz and an intensity of 0.2mW/cm^2. The present study results differed from an experiment carried out by Merritt et al.[25,116] and Okonigene[114] which proposed that the thermal effects might be caused by hot spots in the brain, even though total energy from mobile phones was not high. Frey indicated that BBB deterioration might be related to headache.[25]

2) Effects on the cellular structure of the brain, MFR will trigger changes to central nervous system structure. This is usually found from exposure to high-intensity energy over extended periods. Reports of swelling and wounds of the head and cell death in pig brains which have been in contact with MFR at a frequency of 3,000MHz and an intensity of 25mW/cm^2 for three hours related to the causes of cancer,[116] effects on anti-oxidant concentrations in cells, anti-oxidant molecules, atoms, or ions with unpaired valence electrons.[24] When antioxidant metabolism processes are disturbed, the amount of anti-oxidants become excessive, resulting in oxidative stress will lead to cellular structure changes, and cause differences in genes, finally to mutation and apoptosis.[24]

3) Effects on electroencephalography (EEG). EEG is a method used for measuring brain's electrical changes directly from the skull[117, 118] by examining responses of the brain to various triggers, using the method of recording electrical signals shown as brainwaves.[118] Studies on EEG changes have found inconsistent experiments in experimental animals reporting contact with MFR at an intensity of 7mW/cm^2 for 200

hours. In experimental rabbits, they have found desynchronized EEG after using phenobarbital.[57] Most studies have found changes with higher alpha (8-12Hz) waves in the occipital area after contacting with electromagnetic energy in the first minute.[117,63] In 2010, Croft found EEG response only in 2G systems without discovering in 3G systems. Leung et al. found changes to both systems. Lustenberger et al. found more slow-waves in the brain during contact with MFR, which related to the final stage of the sleep cycle. Furthermore, Bak et al. and Maganioti et al.[63] found electrical changes with the perceptive and memory nerve cells after contact with MFR from mobile phones.[63] Significant research findings revealed reactions to signals from the epileptic foci in the brains of patients with seizures by Tombini et al. and Vechhio et al.[24] Furthermore, the use of low frequencies such as flash lights (15Hz) could also excite symptoms among patients with epilepsy caused by frequencies matching excited brainwaves.[21] However, other studies revealed no changes in EEG that was due to contact with electromagnetic energy.

4) Concerning the effects on cell membranes, cell membranes are strong layers of electrodes held by large and complex molecules. The molecules will create vibrations only in the areas of conflict between the positive and negative charges, causing electrode oscillation with a frequency of 10^{11}-10^{12} times/minute and a speed in cell membranes of 105-106 centimeters/minute. The experimental contact of MFR on the cell membranes usually shows the allowance of cell membranes to increases absorption of sodium and potassium ions. Liburdy (1985) studied the rabbits which were in contact with MFR at 2,450MHz and a density of 400mW/g and found Na to increase then returned to normal in 60 minutes.[119] However, experiments using dyes in living subjects returned to normal in two hours.[24]

5) Regarding the effects on control systems inside the cells, most studies have been interested in the effects of electromagnetic energy to gene displays, especially the protein changes in gene coding. However, most studies remain unclear. Interestingly, electro-magnetic energy has impacts on calcium concentrations which send information inside the cells. Chronic contact with electromagnetic energy have separated calcium ions from cell membranes into the cytosol, which is the liquid inside the cells and relates to gather of chromatin that has effects on genes.[24]

6) Concerning the effects of neurotransmitter functions. Neurotransmitters are chemicals with the duty to lead, expand, and control information along electrical

signals from one nerve cell to another.[63] Each neurotransmitter has different biochemical mechanisms, based on the type of neurotransmitter in the brain. Most studies have been interested in changes of neurotransmitters resulting from contact with high intensity MFR such as catecholamines, serotonin, and acetylcholine. Responses of nervous systems from MFR contact are different depending on the time of each contact, number of contacts, and part of the contacted brain.[116, 120] MFR has been found to have effects on neurotransmitters, creating changes in calcium ion concentrations and disturbance to neurotransmitter secretions and receptors, etc.[116]

The studies of the biological effects of microwave frequency radiation (MFR) are usually interested in the effects which induce higher temperatures in human heads, impact on the central nervous system,[100] while MFR from smartphones are insufficient electromagnetic energy for increasing body temperature.[22] The brain is a system which controls body functions with a system similar to electricity.[63, 69] It is also in unstable condition. Electromagnetic energy may cause non-thermal effects by changing biochemical reactions that will cause changes in molecular or cellular dispersion and shape, ion transmission, and changes to protein creation by showing pathologies of various diseases; such as increasing absorption of BBB or changes caused by endogenous opioid stimulation, etc., These are factors of symptoms and behaviors that are problems of nervous systems caused by smartphones use.[28] Medicine in the Soviet Union and Poland designated symptoms caused by exposure with microwaves as "Microwave Sickness Syndrome", consisting of weakness, skin symptoms, rashes, headache, insomnia, changes to blood pressure, tumors, impotence and memory deterioration. Headache, in particular, were most encountered.[24] Furthermore, studies of dopamine-opiates related to headache[25] have been shown that exposure with MFR at low intensity can prevent apomorphine secretion in the opiate system.[121] Furthermore, headache has related to MFR contact with the dopamine-opiate system of the brain and able to pass through the cell membrane. Both issues are connected to headache.[21]

1.4.4 Theory and literature review for headache

Headache is the most prevalent among neurological abnormalities and constitutes a global health issue in addition to be classified as the seventh most incapacitating disease. The study conducted by Lewis in 2007 found headaches at 57-82%.[34] According to the

Global Year against Headaches 2012 project, headache prevalence was found at 50%.[33] The systematic review conducted by Wöber-Bingöl in 2013 found headache prevalence at a mean of 54.4% (95% CI 43.1-65.8).[37] Straube and colleagues in 2013 found headache prevalence at 66-71%.[122] Sweden found headache prevalence at 64.9% with migraines and possible migraines encountered at 24.9%. The tension type headache (TTH) and possible TTH were encountered at 37.6%. Unclassified headaches were encountered at 31.2%.[123] Taiwan (2010) found very high prevalence at 86.6%.[46] The study conducted in 2014 by Wöber-Bingöl and colleagues found headache prevalence at 89.3%.[124] Differences in headache prevalence has relied on the characteristics of the studied groups such as age, gender, ethnicity, economic status, genetic factors, food consumption characteristics, area geographic characteristics, air, and diagnosis of headache, which may be caused by factors or additional stimuli in daily life,[125-127] including methods for studying and analyzing headache.[128] Headache has been found in children more than adults. Prevalence of headache was found at 3-8% in preschool-aged children, 37-51% in elementary school children, and 57-82% in high school students.[34] The Global Campaign to Reduce the Burden of Headache Worldwide (2007) found migraine and TTH prevalence at 11% and 42%.[129] In the meantime, the World Health Organization (WHO) has reported from the Atlas of Headache Disorders and Resources in the world of 2011 found headache among adults (aged 18–65 years) in Asian countries to have the highest prevalence (63.9%). European countries had the highest percent for migraines and TTH (14.9% and 80%).[130] While studies in adolescents in 2014 found that migraines had increased to 19.3-39.3% and TTH has been found at 17.9-37.9%.[40,131-132] Headache in pre-adolescence has been found in males rather than females while, in early adolescence, were found in more girls than boys.[34,133] Headache in adults has been found at 47-50% while migraines were found at 10-18%.[33,47] The WHO found the world's adult population (1.7-4%) affected by headache more than or equal to 15 days in every month.[47] Students would have headache which led to cessation of studying, unable to participate in activities (20.7-68.0%), reduced capabilities, suffering, and lack of quality of life.[44, 46-47, 125, 134] Burdens from headaches can be considered a disability of 1.3 years from years lived with disability (YLD) and rank 19th among disability-adjusted life years (DALY).[35, 134] Therefore, management of risk factors controlling, lifestyles, and environment in order to prevent headaches[35] and enable persons with headache to adjust to the illness and co-

morbidities of headache such as depression encountered in three times the population with migraines, etc.[47]

Headache is a pain which has sent from the deep parts in the brain to the brain surface. Therefore, an understanding of the body's pain mechanism is required in order to explain headache mechanisms.

1) Pain mechanism

The pain mechanisms in the body occur when tissues are destroyed, it also serves as a preventive system to excite or remind the body to perceive and stay away from pain stimuli.[32] However, many persons have displayed pain symptoms without tissue loss or pathologies causing pain, which usually psychological causes.[135] Pain is divided into two types consisting of fast and slow pain. Fast pain is felt within 0.1 seconds while slow pain is felt in one second and more after stimulation. Pain receptors are free nerve endings with functions to receive pain. Nerve endings are found in the skin and other tissues throughout the body. Deep tissues will have few nerve endings, thereby causing slow pain.

There are three main types of pain receptor stimuli[32] as follows:

1) Mechanical pain stimuli respond to mechanical damage such as being cut by sharp objects, etc.

2) Thermal pain stimuli cause the skin to respond to extreme temperatures, especially extreme heat. Pain is perceived when the skin receives heat at more than 45°c.

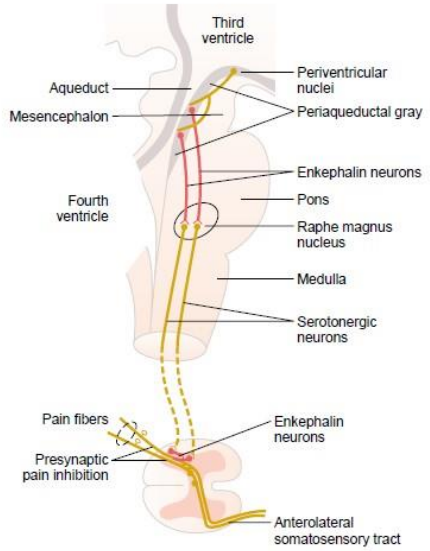
3) Chemical pain stimuli are chemicals caused by secretion in the body such as bradykinin, serotonin, histamine, potassium ions, acid, acetylcholine, and proteolyticenzymes. Furthermore, prostaglandins and Substance P increase nerve ending sensitivity without directly stimulating nerve endings.

Mechanical and thermal stimuli cause fast pain and deliver signals from receptors via A-delta afferent fiber into spinal cord and to the brainstem and the thalamus, while chemical pain stimuli cause slow pain with C-fiber taking signals from receptors into spinal marrow and to the brainstem and the thalamus.[32]

1.1) The brain's dopamine-opiate system

The dopamine-opiate system is used by the body to suppress pain with opioid receptors. Opioid receptors are naturally secreted neurotransmitters in the brain with effects similar to opium. Opioid receptors are neuropeptides with the key

components consisting of beta-endorphins, met-enkephalin, leu-ekephalin, and dynorphone. Both types of enkephalins are found at the brainstem and spinal cord. Beta-endorphins are found in the hypothalamus and the pituitary gland, while dynorphins are found in the brainstem and spinal cord at much lower amounts compare to encephalin.[136]



The pain suppression system functions by sending pain control signals from the cortex and the hypothalamus. Most nerve fibers came from the periventricular nuclei and the periaqueductal gray, which can stimulate enkephalin secretion and send encephalin to the raphe magnus nucleus, there it triggers and releases encephalin. Nerve fibers in this area will send signals to the dorsal area of spinal cord in order to stimulate serotonin and enkephalin secretion at spinal cord. Enkephalin suppresses pain nerve fiber entry with the ability to block initial pain signals in the spinal cord.[32]

Figure 1.4 Pain suppression system.
Source: Arthur C. The Nervous System: textbook, 2006 p.602.

1.2) Headache according to pain stimuli

Headache is pain caused by deep structures in the brain. Slow pain has been caused from the following stimulation inside and outside of the skull:

1.2.1) In the area of headache from stimulation in the skull,[32] most brain tissues are not sensitive to pain. However, venous sinus stimulation or stimulation of blood vessels in the dura area, especially at the base of the brain, by stretching or damaging during accidents, will cause blood vessels to be stretched, pressed and cause significant headaches, etc.

1.2.2) Headache in the skull or referred pain[32]

(1) Nerve stimulation above the tentorium such as inflammation or stimulation of nerves in the brain, especially the trigeminal nerve, causes feelings of pain transmitted from the face, etc.

(2) Occipital nerve stimulation under the tentorium enters the central nervous system through glossopharyngeal nerve, vagal nerve, and cervical nerves at the upper brain, behind and under the ears, causing headaches to be referred to the occipital area.

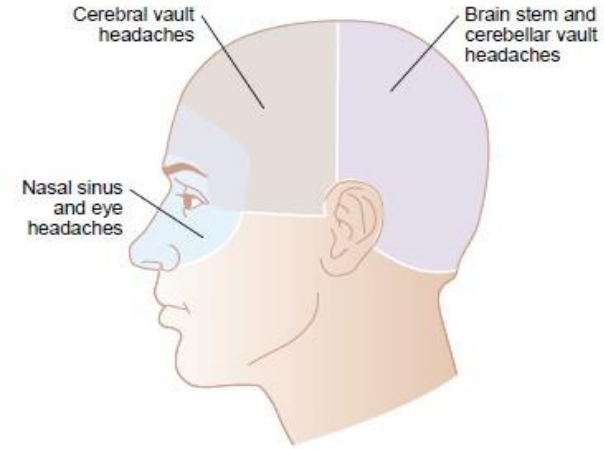


Figure 1.5 Areas with headache from various causes.
Source: Arthur C. The Nervous System: textbook, 2006 p. 606.

2) Types of headache

2.1) Types of headache from causes in the skull[32]

2.1.1) Headache from meningitis include the dura and the sinus area, which is especially sensitive. This inflammation causes severe headaches covering the entire head.

2.1.2) Headache caused by low spinal fluid pressure. Reductions of spinal fluid by only 20 millimeters from the spinal cavity causes severe headaches in the skull, causing changes to brain weight and changing the shape of the dura surface and ultimately causing headache.

2.1.3) Migraine headache are a special form of headache caused by vascular abnormalities. Mechanisms for this type of headache are not clearly known and usually begin with dizziness, blurred vision, and other symptoms which usually begin and continue up to 30 minutes to one hour after headache. Many theories which explained migraine headache are stress, meninges pressure, vascular contraction, and genetic defects, etc. Furthermore, migraine incidence has been found in families with history of migraines (65-90%) with the prevalence of female two times higher than male.

2.1.4) Headache caused by alcohol consumption are usually caused after liquor consumption because alcohol is toxic to the tissues in the body, causing irritation to meninges and pain in the skull.

2.1.5) Regarding headache caused by constipation, persons with headache from constipation are among patients whose sensory nerve channels relate to pain at the spinal cord have been cut, caused by toxin absorption, or changes to blood circulation, causing loss of intestinal fluid.

2.2) Types of headache caused by factors outside the skull[32]

2.2.1) Headache caused by stress results in muscle contraction, especially the muscles which connected to the skull and the neck muscles attached to the occiput. Stress is a frequent cause of headache.

2.2.2) Headache caused by nasal irritations and parts of the nose. Nasal mucous and cavities are sensitive to pain but not very severe. Most infections or irritations in the nose structure cause headache behind the eyes. In cases of frontal sinus infections, patients will have headache at the surface of the forehead and the skull. Pain from lower sinuses such as the maxillary sinus, occur at the face.

2.2.3) Headache caused by eye abnormalities causes difficulty in focusing eyesight and ciliary muscle contraction with retro-orbital headaches.

2.2.4) Eye irritation from excessive amounts of UV light by looking at the sun or the light caused by welding for 2-3 seconds can cause headache for 24-48 hours. Receiving radiation can cause photo-chemicals injury of the conjunctiva. Pain occurs in the skin of the head or the retro-orbital area. Heavy focus of welding or sunlight on the retinas will cause burns at the retinas and headache.

The International Headache Society (IHS) classified headache and determined an international classification of headache disorders (ICHD).[137] In 2013, the International headache society modified the criteria up to edition 3 and classified headaches into three main groups as follows:

1) Primary headache was not caused by diseases or complications without being able to identify the true causes of headache. The etiology of primary headache related to blood circulation, neurotransmitters, and cranial nerves. Primary headache was divided into the following groups:

1.1) Migraine was the third most prevalent headache[137] and the seventh most encountered abnormality of the global level. Migraines are headache with significant impacts on population disability[82] and usually found among early adolescents and adults whose ages between 35-45 years. Migraine prevalence among children is at 4-10%[34] which causes from the stimulation deep in the brain lead to inflammation and pain around nerves and blood vessels in the brain. Migraines are divided into two main groups consisting of:[137]

1.1.1) Migraines with aura

1.1.2) Migraines without aura

Migraines are usually repetitive, frequent, and determined by the following symptoms before occurring of headaches: [47]

1) Moderate severity
2) Nausea
3) One-sided and rhythmic pain
4) Stimulated by physical movement
5) Long duration of symptoms, hours to 2-3 days
6) Symptom frequency from once per year to once per week Migraines in children trend to shorter duration and clear gastrointestinal symptoms. Migraines are hypothesized into two stages in the incident process:

Stage 1 is caused by artery contraction, causing lower blood circulation in the skull, leading to ischemia that usually begins from the rear and spreads to the front of the skull, thereby results in auras.[137]

Stage 2 is caused by artery dilatation outside the skull then leads to rhythmic pain. Ergotamine has been found to cause vascular contraction and temporarily reduced the rhythmic arterial throbbing, thereby reduced headache. Furthermore, the use of pressure at the carotid artery on the same side as migraines has also been found to reduce pain.

1.3) Tension-type headache is the most frequently encountered type of headache in the headache group and found in 30-78%[137] of the population. TTH has usually found more frequent in girls entering adolescence than boys with a ratio of 2:1. TTH has significant social and economic impacts. TTH's causes related to stress or muscle and bone problems in the neck. TTH has pressing pain or cause tightness as

though patients are being squeezed around the head with occasional pain at the neck. TTH usually lasts for 2-3 days or several days[47] and TTH is different from migraine headache.

TTH occurs in four stages according to muscular contraction as follows:

Stage 1: During stressful situations, peripheral muscles of the skull will contract.

Stage 2: Muscle contraction over extended periods of time reduces blood flow of the supporting muscles.

Stage 3: Ischemia

Stage 4: Ischemic muscles lead to headache from muscle contractions.

Infrequent TTH is usually caused by nerve ending stimulation, while chronic headache is usually caused by central nerves. Muscle pains are usually found at the head, face, neck, and back as presenting symptoms of headaches detected by feeling while turning.

1.4) Cluster headache occurs on a single side of the head with eye pains, red eyes, tears, and obstructed nasal passages. Few patients have been found with drooping eyelids (0.1%). Cluster headache has been found in patients aged 20 years and above, and in women at a ratio of 1:6 compare to men.[47]

2) Secondary headache is caused by other diseases. Headache severity depends on the causes such as:[137]

2.1) Accidents, injuries, or surgery in the head or neck areas

2.2) Abnormalities of the blood vessels in the brain or the spinal cord; such as stroke, vasculitis, and cerebral hemorrhaging, etc

2.1) Abnormalities in the brain such as higher intracranial pressure, brain tumors, cerebral cancer, and epilepsy, etc

2.2) Substances in the body and lack of substances; such as food, nutrients, narcotic substances, cocaine, liquor, or chronic overdoses of pain medication for headaches such as ergotamine, triptan, paracetamol and various pain relieving medications. Chronic headache from medication overdoses has been found more than 5% of the population and more in women than men.[47] Causes also include abstinence from medications or caffeine.

2.3) Infections such as infections of the cell membranes, brain tissue, and brain abscess, and infections that spread throughout the body, etc.

2.4) Impaired homeostasis issues; such as lack of oxygen, high altitude, diving into deep water, heart disease, hypertension, pre-eclampsia or eclampsia, dialysis, and fasting, etc.

2.5) Headache or facial pain occurs from abnormalities of the skull, neck, eyes, ears, nose, sinuses, teeth, mouth, or other parts of the body such as sinusitis, teeth, and gum abnormalities, dislocation of the jaw, cavities, strabismus, and glaucoma, etc.

2.6) Psychiatric disorders

3) Neuralgias and other headache such as trigeminal neuralgia, etc., which is usually chronic, one-sided headaches with moderate severity and facial pain caused by blood vessels pressing nerves.

Headache is divided based on acute and chronic types of symptoms. Chronic migraines occur at the frequency of more than or equal to 15 days per month for more than three months. Children with migraines usually have migraines once a week or day (3-15%), while chronic TTHs occur at the frequency of more than 15 days per month for at least three months. Children with at least one headache per month have been found at 9-33%.[138] Most causes of chronic headache are from medication overdose.[137]

Therefore, most headache has been caused by nerves which received pain signals from head and neck, stimulated by various causes, and the nerves that received pain while send feelings to the center of the brain with responses shown as headache. Furthermore, headache related to chronic nervous system abnormalities has usually the following co-morbidities:[139]

1) Headache from cerebral abnormalities including ataxia, nystagmus, and vibration.

2) Patients with intracranial pressure abnormalities are found with swollen discs, vomiting at night and in the morning, and large head size. 3) Patients with local nervous system abnormalities will begin to have double vision, local seizures, changes in personality, and lower educational achievements.

3) Headache factors and triggers in children[34, 140-141]

Headache factors and triggers in children are as follows:

3.1) The medical migraines records of the parents relate to headache in children.[142]

3.2) Gender and age factors.[53] Higher frequency has been found in females than males.[35, 142] Headache etiology usually relates to estrogen and progesterone[141] preceding menstruation, causing headaches and migraines. Furthermore, gender and age also relate to sleep quality according to age and gender, especially males being found with these causes more than females.[55]

3.3) Exercises and sports are also considered as one of the factors. Headache is more frequently found among the groups that exercise (OR 1.17; 95% CI 1.00-1.37).[140]

3.4) Coughing, sneezing, blowing air with force, and sexual intercourse.

3.5) Constipation and dehydration, based on the definition offered by the World Gastroenterology Organization Practice Guidelines.[143] Children with migraines are found to relate to constipation, which is usually a result of stress, depression, and anxiety.[144]

3.6) Sleep problems and sleep deprivation (OR 2.03; 95% CI 1.6-2.5)[140] are considered as the problems of the quality of sleep[145] and the factors of triggering migraines.[42, 44, 142] Sleep walking prevalence has been found in the children with migraines at 30-55%. Sleep is able to ease headache, while headache is a cause of sleep disturbance.[42]

3.7) Food and ingredients such as monosodium glutamate and chocolate, etc. Furthermore, patients who do not eat regularly will have more headache than patients who eat regularly (OR 1.2; 95% CI 1.08-1.33). [140]

3.8) Beverages such as alcoholic beverages have dilated blood vessels[71,141] with histamine and prostaglandin secretion that can stimulating migraines.[141] Beverages with caffeine will cause headaches (60%) with minor severity.[146] Furthermore, abstinence from caffeine has caused headache based on ICHD-3 beta diagnostic criteria.

3.9) Environmental factors such as odors, loud noises, bright lights, and hot air, etc.

3.10) Co-morbidities of headache in ordinary children and adolescents.[42, 147] Headaches, especially migraines, are usually accompanied with other symptoms of illness such as hypertension, stroke, heart disease, asthma and obesity.

3.10.1) Headache among children with hypertension is found at a prevalence of 1-2%.[148] Use of beta-blockers or vasoconstrictor agents has been found to reduce migraine severity at 45%.[149] Furthermore, the use of dipyridamole, as a treatment of cerebral thrombosis triggers headaches[150] and cerebral thrombosis is a cause of headache specified in ICHD II criteria.[149]

3.10.2) Muscle or joint pain causes headache (OR 1.9; 95% CI 1.6-2.1).[140]

3.10.3) Research findings from many studies have not confirmed the definite relationships between cardiovascular disease and headache. Most of the data found migraine to be one of the risk factors of cardiovascular disease.[151] In 2004, headache caused by heart disease specified as a code for diagnosis in ICHD-II with a clear diagnostic criteria.[152] Furthermore, medications used to treat coronary thrombosis were found to trigger headaches, such as nitrates, nicorandil, sildenafil, tadalafil, and vardenafil related to headache.[153]

3.10.4) Headache found in epilepsy patients is defined as secondary headache in ICHD-3 beta headache diagnostic criteria. Migraine and tension-type headache prevalence in patients with epilepsy have been found at 82.1% and 9.2%.[154-155] Epilepsy in children is a risk factor of migraines at 4.5 times higher than TTH.[154] Furthermore, use of levetiracetam (LEV) has caused dizziness and headaches (1-1.4%).[156]

3.10.5) Eyesight problems and headaches will have higher prevalence among patients with headaches, especially astigmatism in children who have received corrections for eyesight abnormalities, causing headache symptoms to improve with 38% of patients without repeated headaches.[157] Eyesight abnormality problems usually relate to frontal headaches OR 1.429; 95% CI 1.130-0.806 and are astigmatism and farsightedness (63.6% and 27.27%).[158]

3.10.6) Headache with other health problems such as asthma, ear infections, sinusitis, and mucositis has been found in children with headache (41.6%).[42, 147] Children with headache will have health problems including difficulty breathing, ear infections, and high fevers at 13.6 times. Children with headache and at least one health problem have been found the prevalence at 41.6%.[159]

3.10.7) Obesity has related to increase headache[44] OR 2.62; 95% CI: 1.07-6.45.[141] Obese persons with body mass index (BMI) >30 will have five times higher

prevalence of headache among persons with normal weight.[93] With increased in migraine severity among the groups of BMI ≥35 (OR1.9).[160-161]

3.10.8) Headache from psychiatric problems has been determined according to ICHD-3 beta criteria such as anxiety, depression, being bullied, and panic. Patients with depression and anxiety will have 1.87–3 times higher risk of migraines than the general population.[35, 42, 134, 140] Use of anti-depressants is able to reduce tension-type and migraine headache frequency. Moreover, children with significant psychiatric problems relate to headache such as attention-deficit or hyperactivity disorder usually have headache with Tourette syndrome,[162] a neurodevelopmental disorder has been found in 1% of school-aged children. Migraine frequency in children with Tourette syndrome has four times higher. Headache and migraine prevalence in children with Tourette syndrome has been found to be 55% and 25%.[42] Children with Tourette syndrome and ADHD usually have difficulty in learning (22.7%)[163] with psychological abnormalities, following traumatic events such as violence, abuse, rape, abandonment, and domestic violence. All of the aforementioned factors have correlated with higher prevalence and frequency of chronic headache.[164]

4) Headache diagnosis

Headache diagnosis in order to categorize or classify headaches, especially primary headache, is important for an appropriate and accurate treatment, along with overdose reduction.[137] Doctors usually diagnose headache based on backgrounds, physical examinations, and laboratory blood tests. However, if patients have chronic pains, severe pains, or other abnormal symptoms such as nausea, vomiting, or limb weakness, etc., doctors will carry out further examinations such as eye examinations, nerve system examinations and x-ray computer, or MRI scans, including CSF tests when patients are suspected to have meningitis, etc. Criteria for diagnosing headache according to ICHD-3 beta are as follows.[137]

4.1) Diagnostic criteria for migraines without auras:[137]

A. At least five symptoms out of B-D

B. Headache duration of 4-72 hours

C. Headache characterized at least two out of following four:

1. Pain locating on one side
2. Rhythmic pain according to vital signs (throbbing)

3. Moderate to severe pain
4. Symptom exacerbated by movement such as walking or walking up the stairs

D. At least one of the following symptoms during headaches:

1. Nausea and/or vomiting
2. Fear of lights and sounds

E. No symptoms in other diagnostic criteria in ICHD-3 beta.

Migraines in children and adolescents have usually manifested as pain on two sides with exhibition in the front temporal area. Migraine headache without aura usually related to menstruation.

4.2) Diagnostic criteria for migraines with aura:[137]

A. At least five symptoms out of B-D

B. Auras returned to normal with one or more of the following symptoms:

1. Seeing flashing lights

2. Sensory disorders, such as feelings of being punctured by needles or numbness while moving slowly, on at least one side of the body, such as face or tongue

3. Speech or language disorders found in few patients

4. Abnormal weakness, especially paraplegia which is highly associated with migraines

5. Brainstem abnormalities such as dysarthria, dizziness, vertigo, tinnitus, hypacusis, diplopia, ataxia, and reduced awareness

6. Sight abnormalities such as scintillations or scotomata, including blindness found by visual field testing

C. At least one of the four pain characteristics:

1. At least one or more of the aura symptoms
2. Aura duration of 5–60 minutes
3. At least one aura symptom located on the same side
4. Aura symptom followed by headache within 60 minutes

D. Symptoms cannot be grouped into other symptoms in ICHD-3 beta diagnostic criteria and other symptoms caused by temporary ischemia.

However, when patients have auras, patients must have maximum aura duration of 3-60 minutes. Weakness may occur for as long as 72 hours. Auras related to visibility most frequently encountered in over 90% of patients, followed by sensory impairments including numbness. The least frequently encountered aura is speech abnormalities found as language impairments, though it is difficult to categorize it

4.3) Tension-type headache diagnostic criteria:

A. Occasional headache has occurred at least ten times with a mean of no more than one day per month or less than twelve times per year. In most cases, headache occurs at least 1–14 days per month for more than three months. It is more than or equal to 12 times or less than 180 days per year, according to B-D.

B. Symptom duration of 30 minutes to seven days.

C. At least two of the following characteristics:

1. Pain location on 2 sides
2. Pain on both sides
3. Pain's characteristics similar to being pressed or squeezed, and not in rhythm with vital signs (throbbing)
4. Low to moderate severity
5. No symptom exacerbation from activities in daily life such as walking or walking up the stairs

D. Both of the following symptoms:

1. No nausea or vomiting
2. Any of the following symptoms: Fear of lights, fear of sounds

E. Unable to specify into other criteria in ICHD-3 beta.

Migraine and TTHs should be recorded by using headache diaries to categorize or distinguish both types of headache. Chronic TTH include pain for at least 15 days per month.

4.4) Diagnostic criteria for cluster headaches:

A. At least five presenting symptoms according to criteria in B-D.

B. Pain around or above the eye sockets or severe or extremely severe pain at one temple for 15-180 minutes.

C. Any or both of the following symptoms:

1. Having symptoms or at least one presenting symptom on the same side as the headache:

1.1 Red eyes and/or tears

1.2 Obstructed nasal passages or mucus flows

1.3 Swollen eyelids

1.4 Sweating in the forehead and facial areas

1.5 Red forehead and face

1.6 Ringing ears

1.7 Small pupils or droopy eyelids

1.8 Discomfort or unease

2. Discomfort or unease

D. Frequent headache with symptoms day after day, before reaching eight times per day with symptoms will occur almost all the time.

E. Unable to specify into other criteria in ICHD-3

4.5) Diagnostic criteria for ordinary secondary headache:[137]

A. Headache according to criteria in C.

B. Abnormalities confirmed with documents based on academic principles must be a cause of diagnosed headache.

C. Causal relationship must be shown as at least two of the following:

a. Headache occurring in temporal relation to time of beginning headaches and factors has believed to be the cause of abnormality.

b. One of the two following causes:

1.1 Headache increase with statistical significance when encountered with factors has believed to be the cause

1.2 Headache improvement with statistical significance when the solution of the factors has believed to be the cause are made

1.3 Headache with specific characteristics for the causes

c. Academic information of causes

D. Unable to specify into other criteria in ICHD-3 beta.

However, most headaches have no detected pathologies. Therefore, the key

of headache diagnosis has used the history of headache or headache diaries to provide details on headache and enable doctors to diagnose accurately. In 2011, the World Health Organization (WHO) organized The Lifting the Burden Project[165-166] to lift headache burdens and enabled headache diagnosing criteria to be the same in every country. Hence, the World Health Organization prepared steps for diagnosis and classification of headaches according to Table 1.3 and 1.4.

Table 1.3 Steps for categorizing migraine headaches

Group	Headache Characteristics	First Scoring	Second coring	Total Score
	1. Headache lasted less than 15 days/month.			
B	2. Headache duration of 4 – 72 hours (no less than four hours or no more than 72 hours).	1		1
C	3. Bad-Very bad feelings = 1	3-6 together $\geq$2 = 1		1
	4. Throbbing/Rhythmic pain = 1	3-6 together <2 = 0		0
	5. Pain on one side = 1			
	6. Worsened symptoms during activities = 1			
D	7. Nausea = 1	7 – 8 is D1	D1+ D2 $\geq$1=1	1
	8. Vomiting = 1	D1 together $\geq$ 1 = 1	D1+ D2 =0 = 0	0
		D1 together = 0 = 0		
	9. Fear of lights = 1	9 – 10 is D2		
	10.Fear of sounds = 1	D1 together = 2 = 1		
		D2 together $\leq$1 = 0		
			B+ C+ D = 3	Migraine
			B+ C+ D =2	Probable migraine
			B+ C+ D<2	Go to probable migraine

Table 1.4 Steps for classifying TTHs

Group	Pain Characteristics	First Scoring	Second Scoring	Total Score
	1. Headache duration of no more than one day/month or <12 times per year.			
B	Headache duration of 30 minutes – seven days (no less than 30 minutes or no more than seven days).	1		1
C	3. Not bad and bad feelings = 1 4. Tight and tense pain = 1 5. Pain on both sides = 1 6. Symptoms do not worsen from activities = 1	3-6 together ≥2 = 1 3-6 together <2 = 0		1 0
D	7. Nausea = 1. 8. Vomiting = 1. 9. Fear of lights = 1. 10.Fear of sounds = 1.	7 – 8 is D1 D1 together >1 = 1. D1 together = 0 = 0. 9 – 10 is D2 D1 together = 2 = 0. D2 together ≤ 1 = 1	D1+ D2 =2 =1 D1+ D2 <2 = 0	1 0
			B+ C+ D = 3	TTH
			B+ C+ D =2	Probable TTH
			B+ C+ D < 2	Unable to classify

5) Headache assessment

Headache assessments use recorded headache background data to show and categorize pain characteristics, and diagnose headache causes,[141] in addition to compare with diagnostic criteria or handbooks that are not only interviews.[167] Headache assessment usually uses two processes as follows:

5.1) Ask the patients if the patients have headache. If patients have headache, ask the patients about details.

5.2) Conduct the interview if the patients have answered that patients have headaches and details of headache are known.

Data on recording forms consisted of:[141]

1. Demographic data, age, gender, level of education, nationality and culture indicating relationship between the study group and headache.[167]

2. Questions relate to classification diagnosis such as:

2.1 Age when patients have begun to have headache, in order to differentiate chronic and acute headache.

2.2 Headache frequency and duration, as temporary symptom models to use as a baseline for treatment assessment.

2.3 Headache severity and headache levels, measured on an 11-level scale, are widely used and shown to be accurate in children and adults.[168] Where 0 indicates no pain and 10 indicates the most pain. Patients should be asked about details for the past three months.

2.4 Time when patients have begun to have symptoms, to show when headache is stimulated such as when sleeping or waking.

2.5 Symptoms occurring before headaches.

2.6 Characteristics and symptoms during headache such as: 1) Type of pain such as slow pain, tight pain, or dull pain. 2) Related symptoms such as nausea, vomiting, fear of lights, and fear of sounds. 3) Psychological, physiological, food, beverage, and environmental triggers of headache.

2.7 Measure headache impacts[167]

Therefore, headache diagnosis depends on history and details of headache by using headache diaries to perform preliminary headache impact assessments. Repetitions, chronic pain, and treatment efficiency are also assessed in addition to assess physical and emotional capabilities and satisfaction. Expected outcomes from measurement include two important areas, consisting of frequency and duration, and severity. Headache characteristics and impacts from headache, by measuring loss of capabilities and quality of life, are also included.

6) Headache impact assessment

Assessment of impacts from headache is aimed at considering headache severity and usually made in the psychological and behavioral aspects as follow:[145]

6.1) In the area of impacts on psychological, emotional and social function, headache has caused the distress and disturbance to the psychosocial development[145] and perception. Headache effects on learning achievements causing children to become aggressive, resulting in impact on family relationship.

6.2) Behavioral impacts on abilities at school and avoidance of activities cause children to prefer being alone and get addicted to games, [145] and finally cause children to miss school. Children with migraines (31%) have missed school in the past three months for at least one day.[145]

Most impact assessments have been made in the past month and consisted of the following: 1) The Migraine Disability Assessment (MIDAS) is used in age group of equal to or more than 20 years, in order to assess the impacts in the areas of working, doing household chores, off-working time including family time, society, and free-time activities. 2) The Pediatric Migraine Disability Assessment (PedMIDAS) is used to assess impacts in children of the age 14–19 years, by assessing capabilities at school and participation in activities. 3) HIT6 assessed impacts and severity of headaches, by assessing capabilities at school and participation in activities and daily activities.[169] Headaches have multiple causes including pathological and psychological causes. Furthermore, many studies are interesting in studying the electromagnetic energy from mobile phones, causing headache. However, no clear data on the process in which electromagnetic energy from mobile phones causes headache has been found, so headache from mobile phones is not designated based on ICHD-3 beta criteria. Many studies on headache caused by mobile phones have encountered as follows:

7) Research findings on headache caused by mobile phones.

7.1) Yoong, Heron, Oftedal et al., Punamaki et al., (cited in[170]), Khan,[29] Praveen et al.,[30] Nathan et al.,[31] Szyjkowska[28] found excessive use of mobile phones to cause health problems including headache (16.1-65%), fatigue (42-45%), memory problems (15-40.6%), sleep problems (38.8%), hearing problems (23.07%), lack of concentration (34.3-52%), and feelings of heat around the ears (28.3-50%). Moreover,

patients (26%)[28] would have headache continuously for six hours after ending the telephone conversations.

7.2) Joachim et al.[48] followed-up on mobile phone users in 2003 who began using mobile phones in 1982-1995, and found patients who were hospitalized for migraines, dizziness and vertigo to have 10-20% higher standardized hospitalization ratios.

7.3) Kumar et al.[171] surveyed perceived risks from mobile phone use, and found the samples (62%) acknowledges the health risks. Most of the samples agreed that telephones were the cause of headache, loss of concentration, and sleep problems.

7.4) Heinrich et al.[172] surveyed children of the age 8-12 years and adolescents of the age 13-17 years, along with recording symptoms while using the devices in order to measure electromagnetic waves emitted from mobile phone as radio frequencies. He found headaches, dizziness, and fatigue to have few correlations with mobile phones.

7.5) Milde-Busch et al.[173] surveyed the use of electronic devices and headaches. The study has been found that listening to music for a long period of time correlated with headache. However, no correlation was encountered when headaches were categorized.

7.6) Augner and Hacker[174] surveyed groups with abnormally high mobile phone use. The result found the relationship between chronic stress, low emotional severity, depression, and young females.

7.7) Chia et al.[175] studied headache prevalence among mobile phone users in Singapore, in the communities of the age 12-70 years. Headache prevalence had increased according to duration of telephone use (minute/day) with statistical significance. Headache prevalence had decreased by more than 20% in the groups who used hand-free devices.

7.8) Chu et al.[49] studied the clinical characteristics of headache relate to mobile phone use with a cross-sectional study design among university students. The result found the prevalence of headache from mobile phones at 18.9%. Sweden and Norway found prevalence of headache from mobile phones at 8.4-13%. Saudi Arabia found prevalence of headache from mobile phones at 22.4% and France found prevalence

of headache from mobile phones at 10-20%. Chu et al. concluded headache from mobile phones is a secondary headache.

7.9) Chongchitpaisan and Bandhukhul[43] found prevalence of mobile phone use at 89.3%, headache prevalence at 81.1%, and migraine headaches at 4.4%. Persons with migraine headaches would have higher mean of mobile phone use. Moreover, students who used hand-free phones would have lower headache prevalence with statistical significance.

7.10) Xavier et al.[131] found excessive use of electronic devices to post a risk for ordinary headache and migraines (OR 1.21; 95% CI 1.02-2.03 and OR 1.86; 95% CI 1.01-3.67).

Furthermore, headache prevalence among adolescents was found to have higher likelihood with a prevalence of 47-82% in 2006-2012, which increased to 83.1-94% in 2013-2015. This concurred with higher mobile phone use. Moreover, according to data from the literature review, electromagnetic energy from smartphones was found to have the following significant processes and effects on headache:

Electromagnetic energy from smartphones effects on neurotransmitters in the pain suppression system. Stimulation of the dopamine-opiate system resulted in decreasing amount of the neurotransmitters in the system and prevented pain suppression functions.[28, 113]

Electromagnetic energy from smartphone use causes heat in the skin surrounding the ears[44] of the same side of headache.[49] Increased temperature from electromagnetic energy may have effects on blood vessels in the aforementioned area, thereby causing vasodilation and resulting in headache.[47]

Headache prevalence among adolescents would have higher likelihood with prevalence of 47-82% in 2006-2012,[35, 38-39, 46] increased to 83.1-94% in 2013-2015.[37, 133-134] This is consistent with mobile phone use among adolescents who have higher trends of using mobile phones. Therefore, a worldwide concern regarding the aforementioned problem has risen. The World Health Organization prepared research plans to assess health risks from using mobile phones with emphasis on children and adolescents. While headache can be prevented, and chronic headache risks and burdens from the diseases can be reduced by controlling factors and stimuli among adolescents.[176]

Most studies of health impacts from electromagnetic radiation emitted by smartphone have usually found in the reports of headache and sleep problems. Sleep is a process of energy restoration and repairing of the body, helping the brain and the deteriorated parts of the body to restore efficiency and readiness for the next day.[67] Therefore, sleep is a basic biological process similar to drinking, eating, and breathing air for physical, behavioral, emotional, and intellectual development, especially among adolescents.[50-53, 65] In a nutshell, sleep problems among adolescents cause effects on perception and learning ability, resulting in low academic performance[58, 62, 64-66, 77] and chronic diseases,[64, 70-72, 74] in addition to be one of the risk factors of death (RR 1.12; 95% CI 1.06-1.18).[73]

1.4.5 Theory and literature review for sleep problem

Good sleep is like food, water, and air that is essential for physical growth as well as behavioral, emotional, and learning development among adolescents.[51-52, 61] Sleep is a condition of changes in movement levels and perception level that is different from unconsciousness or lack of feeling, because sleep can return to a completely normal state. Sleep is a physiological process which cooperates with functions at various levels of central and peripheral nerves. Sleep stores energy to metabolize in the center of nervous system, together with hormone secretion.[54] Therefore, sleep is defined as a natural state of living creatures based on normal systems that can recur and return to a completely normal state.[54] Sleep problems in adolescents occur in many countries worldwide. The National Sleep Foundation in the United States found the prevalence of sleep problems among children and adolescents to occur at rates of 25% and 40% in 2006.[55, 56] With insomnia being the most frequently encountered problem. However, most of the patients did not receive proper diagnosis and treatment. The United States (2011) reported sleep loss in adolescence at 75%.[79] Lebanon, Belgium, Italy, Israel, and India were found to have sleep loss problems.[53 ,62, 82-83, 177] Adolescents in Hong Kong and Korea were found to have a mean sleep time of 7.74 and 6.30 hours,[77, 178] Israel <7.5 hours,[177] while Australia reported sleep durations of 8.5-9.1 hours.[60] A study conducted in multiple countries by Mindell in 2008, and found the prevalence of sleep problems to be 25-40%.[55] Furthermore, the prevalence of sleep problems had increased from 16.9-54.2%[51, 57-59, 179] in 2000-2013 to 58.7-66% in 2013-2016.[53, 60] Therefore, adolescents were found to have more substance abuse, caffeine consumption, alcohol consumption, and more

accidents,[64, 78, 180] which thereby resulting in daytime drowsiness and fatigue at 35-40%.[64] Furthermore, sleep problems influenced psychological problems, stress, and depression.[64] In a nutshell, good sleep was important for good health and quality of life.[181]

1) Sleep architecture

Human bodies have Circadian Rhythms to adjust sleep wake cycles with many important neurotransmitters and hormones in the body, including the influence of light during light-dark cycles, controlling sleep and waking. Sleep cycles are explained according to electroencephalographic characteristics by Loomis.[68] Standards for stages of sleep were introduced in 1968 by Rechtschaffen and Kales called R&K rules according to EEG changes. Sleep was divided into two stages as follow.[54, 68, 182]

1.1) Non-Rapid Eye Movement sleep (NREM).[56, 68] Sleep in this stage is highly important and relates to the digestive system and growth hormone secretion. NREM has a distinct point from lower physical function, slower brain waves, and higher waves. NREM is divided into four stages[54, 182] beginning from Stage 1 to REM and returning to Stage 1.

Stage 1 Light sleep: This stage is a feels confused, half asleep and easily awaken. This period may have muscle spasms called "hypnicmyoclonia" which usually follows symptoms similar to vertigo. Brain waves and muscle functions will be slow. Eye movements will be slow too.[54, 183]

Stage 2 True sleep: This stage is not considered as fully asleep yet. Eye movement will stop. Brain waves will be slower with periodical increases in speed, called sleep spindles,[182] combined with continual muscle contraction and relaxation, lower heart rates and lower body temperature. Stages 1 and 2 are in light sleep range and sleepers are easily awakened.[81,182]

Stage 3 Electroencephalography (EEG) will have delta wave characteristics.

Stage 4 This stage is where sleepers are fast asleep and all EEG are delta waves. Stages 3-4 are collectively known as slow wave sleep. Sleepers will be fast asleep and sleep more deeply, considered the most difficult to awaken. There is no eye movement or other physical movement. Sleepers will have low blood pressure and heart rates, caused by withdrawal of the sympathetic nervous system and stimulation of the parasympathetic nervous system. When sleepers are awaken from this stage, they feel confused for a short while after waking up.[34, 54, 182]

1.2) Rapid Eye Movement sleep (REM) occurs within 90 minutes after sleeping. Sleeping in this stage is controlled by the pons and the midbrain.[184] In this stage, sleep stimulation nerves are fully stimulated. The body will stop sending some brain nerve waves, preventing muscles from moving and reducing response to the environment[34] while having intense brain wave function. Breathing is faster, deeper, and irregular. The eyes move rapidly in every direction. This stage has functions of important neurotransmitters such as serotonin, norepinephrine, and histamine. Dreams occur during REM-sleep and sleepers who wake from this period of sleep will be able to remember their dreams.

NREM sleep cycles alternate with REM sleep in an Ultradian Cycle. Each cycle takes approximately 90-110 minutes. NREM sleep occurs 4-6 times during 6-8 hours of normal sleep, depending on sleep duration, age, medications, physiological, and psychological health.[68, 138] Multiple Ultradian Cycles each night are collectively known as "sleep architecture".[34] When a person begins to sleep, the person will enter NREM sleep. Stages 3 and 4 (slow-wave sleep) are concentrated in the early periods of sleep while REM sleep is concentrated in later periods of sleep.[56] In adults, NREM sleep accounts for 75-90% of all sleep time at 3-5% in Stage 1, 50-60% in Stage 2 and 20% in Stages 3 and 4. REM sleep accounts for 10-25% of sleep.[68] Balanced REM and NREM sleep are important for resting, and help with learning, memorization, emotional development, and capabilities. Adults need to have a sleeping period of 7-9 hours, adolescents need a mean sleeping period of 9.5 hours, and infants need approximately 16 hours of sleep per day.[185]

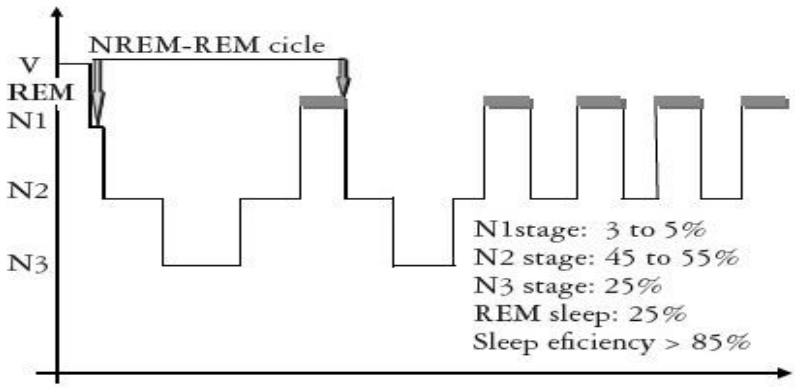


Figure 1.6 Sleep architecture[54]

Source: Hasan, Alóe 2011, p. 54 (4)

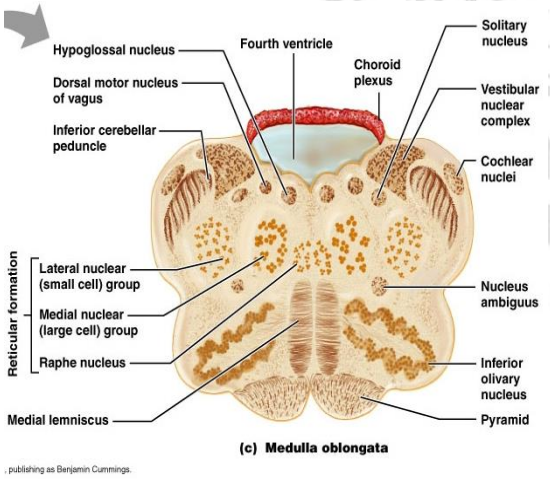
2) Sleep mechanisms

Physiological mechanisms of sleeping and waking are the effects of reducing sensory input which related to wakefulness, including brain anatomy structures and different nerve functions in the wakefulness system. Those signals have been sent from the brainstem to the thalamus and the hypothalamus, and the front of the base of the brain to the cerebral cortex. These signals will stimulate sensory input such as feelings of warmth and comfort, etc. This reduces reticular activating system (RAS) functions at the forebrain, including important neurotransmitters of sleep such as serotonin and gamma-aminobutyric acid (GABA). Therefore, the sleep mechanism is composed of two systems, namely, wakefulness and sleep status.[182]

2 .1) Wakefulness is composed of the following:

2.1.1) Reticular formation: Anatomic structure related to waking.

Reticular formation (RF) (Figure 1.7) has glutamatergic neurons working with aminergic nuclei, serotonin, noradrenaline, dopamine, histamine, and cholinergic receptors in pons and orexin, which is in the nerve cells that send signals into the lateral hypothalamic nuclei. The lateral hypothalamic nuclei functions to trigger wakefulness. The RF functions the most while waking, and will be rapidly reduced with the suppression from GABAergic during NREM and REM sleep.[54]



RF is a neural anatomical formation composed of cells in the brain stem, extending to the central part of the brain and the thalamus. RF functions as a wire leading nerve signals and senses from various parts of the body to the brain stem and interprets into feelings of wakefulness and interest in surrounding issues, enabling awareness and consciousness to be maintained.[54]

Figure 1.7 Reticular formation. Source: Reticular Formation Picture. Website:*http://www.apsubiology.org/anatomy/2010/2010_Exam_Reviews/Exam_4_Review/CH_12_Cerebellum-Brain_Stem.htm: Picture. 2014*.

Therefore, the RF is an area where the "wake-on" switch is turned. Awareness and consciousness are preserved by the reticular formation. The reticular formation stops working when the person is asleep, a manner similar to turning a switch off.[54]

2.1.2) The Ascending Reticular Activating System (ARAS)

Ascending neurons in the wakefulness system as a group of nerves and neurotransmitters working together, this was not just one anatomical structure. These nerve groups begin from the upper brain stem, to the pons, and extend to the midbrain and the diencephalon, before diverging into two routes as follows:[182]

(1) Nerves enter the thalamus will have cholinergic neurotransmitters, consisting of Pedunculopontine Tegmental Nucleus (PPT) and Laterodorsal Tegmental Nucleus (LDT). Cholinergic neurotransmitter will be released at higher rates during REM sleep and the waking stage, decreased during NREM sleep, and stopped in the NREM stage.[141] Therefore, cholinergic cells are called "REM-On". This stage will be found with EEG desynchronization and lower muscle tension during REM sleep.

(2) Nerves enter the lateral hypothalamic nuclei, the basal forebrain, and the cerebral cortex, the monoaminergic group consist of the following:

(2.1) Noradrenergic neurons sent from the locus coeruleus.

(2.2) Serotoninergic neurons send from the dorsal and the medianraphe nuclei.

(2.3) Dopaminergic neurons send from ventral periaqueductal grey (vPAG).

(2.4) Histaminergic neurons send from the tuberomammillary nucleus (TMN).

(2.5) Melanin hormones and orexin send from the lateral side of the hypothalamus.

(2.6) Acetylcholine or GABA sends from basal forebrain nuclei.

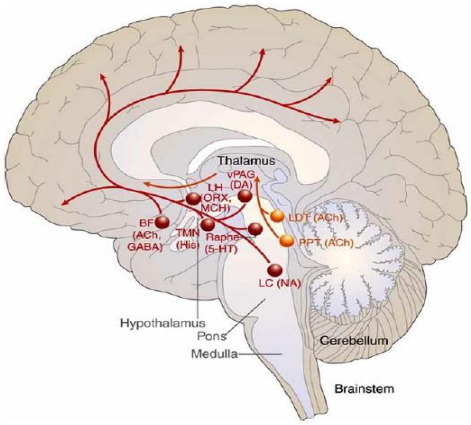


Figure 1.8 Ascending reticular activating system, monoaminergic nerves and cholinergic neurons.

Source: Adapted from Saper 2005, p. 1258 [99].

Aminergic functions will be released most quickly during waking stage[182] by stimulating thalamocortical circuits,[54] become slower during NREM sleep, before stopping during REM sleep. Therefore, aminergic neurons are called "REM-off". The aminergic nervous system suppresses GABAergic cells of VLPO,[54] which have functions similar to orexin but opposite to cholinergic neurons of the base of the forebrain. It will be released at the highest rates during REM sleep and when awake,[182] before stopping in NREM sleep.[54] This is similar to melatonin functions which will be released at a high rate then preserve the balance state during REM sleep.[54, 182] Having lesions in this area will cause narcolepsy and sleeping problems.[54, 182]

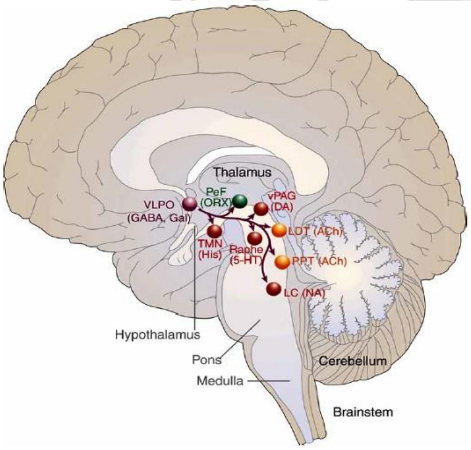
2.1.3) Posterior hypothalamus

The orexin/hypocretinergic system is located at the back and the sides of the hypothalamus. It is triggered by the limbic system at the base of the forebrain and the suprachiasmatic nucleus, which function is to preserve waking stability and important behaviors such as seeking for food or fighting, etc. If a person is sleep-deprived, increased amounts of orexin or hypocretin will be stimulated. Orexin or hypocretin will stimulate the ascending reticular activating system (ARAS) and thalamocortical circuits, and then the signal will be sent to stimulate the cerebral cortex and the limbic system along with cholinergic nuclei in pons. Orexin or hypocretin functions the most during the waking stage and wears off during NREM and REM sleep. Orexin or hypocretin will also stimulate the aminergic system, which then suppresses VLPO indirectly in order to

prevent sleep. At the same time, melanin and GABAergic hormones in VLPO will suppress orexin or hypocretin, causing orexin or hypocretin functions to reduce and eventually disappear during sleep.[54]

2 .2) Sleep status

Ventrolateralpreoptic neurons (VLPO) and sleep status. VLPOs are located at the front of the hypothalamus. Most of the nerves (approximately 80%) are in the GABAergic and Galaninergic nerve group, which their functions are to suppress the wakefulness system.[54] VLPOs are stimulated by the sleep-inducing neurotransmitters such as adenosine and prostaglandin, accumulated at the base of the brain during wakefulness. Sleep-inducing neurotransmitters have high secretion rate during the beginning of NREM sleep and REM sleep.[182]



VLPOs are suppressed by aminergic nerves, consisting of noradrenaline, serotonin, histamine, and acetylcholine which stimulate wakefulness. While orexin suppresses GABA and galanin in VLPOs. Furthermore, suprachiasmatic nuclei can also send signals to suppress VLPO, creating control rhythmic cycles.

Figure 1.9 Cholinergic, aminergic, orexinergic, and VLPO nerve.

Source: Adapted from Saper 2005, p. 1258 [99].

Cholinergic and aminergic neurotransmitters work with orexin in order to promote and maintain awareness. During sleep, these circuits are blocked by VLPO nerves.[182] (Figure 1.9)

The sleeping process is composed of sleeping at night and waking up during the day. This process is controlled by two internal factors consisting of sleep homeostasis and circadian rhythms[56, 187] as follows:

2.3) Circadian process

Circadian process means sleep-wake rhythms related to light and dark, according to daytime and night time in a circadian rhythm that is closely consistent with our regular sleeping hours.[68] Sleep-wake mechanisms have a cycle controller for sleeping

and waking in one day, sleep structure is also controlled**.** The controller is in the suprachiasmatic nucleus (SCN) in the hypothalamus called the "Master Clock". The circadian cycle depends on RF in the brainstem, which functions to control consciousness and sleep with signals from the lights which suppress melatonin secretion. Without light, melatonin secretion will be stimulated and helps the body to have a circadian cycle of 24 hours. Furthermore, SCN also related to body temperature changes, appetite, and sleep-wake cycle duration.[54] SCN produces the waking signals and maintains the balance, by sending signals to suppress VLPOs. When there is no sunlight, SCN signals are reduced. Reduction of SCN will result in higher VLPOs, which will stimulate GABAergic neurons and send feedback to SCN and orexin, in order to begin sleeping process and enter NREM.

Therefore, light suppresses melatonin secretion. While there is no light, the body will enter sleep.[54] Cases where there is light from televisions or computer screens make sleep more difficult. Changes in the places related to time zone changes, which cause early darkness or slower light, will effects on melatonin secretion.[56] Therefore, taking melatonin helps with sleep duration and sleep quality by adjusting the biological clock or melatonin secretion.[68]

2.4) Sleep homeostasis process

The sleep homeostasis process[56] is a balancing mechanism for sleep, the process of controlling sleeping, and waking balance, by causing drowsiness after waking and reducing drowsiness after sleeping or day-time naps. Sleep nourishes the body, returns our body to the normal stage, and maintains balance when sleep is disturbed. Furthermore, sleep deprivation will trigger more sleep, in order to compensate for lost of sleep. Therefore, sleep control is explained with sleep balancing mechanisms and stimulation of the circadian cycle in order to induce sleep. Balance maintenance is done by adenosine accumulation.[54] A product of metabolism, at the base of the forebrain, while we are awake or sleep-deprived.[54] Higher adenosine levels will cause the base of the forebrain to stop VLPO suppression, resulting in lower cholinergic neuron, together with decreased SCN stimulation, resulting in NREM sleep. When adenosine is reduced, the base of the forebrain will send signals to stimulate orexin and suppress VLPO, in order to stimulate waking up. Caffeine, an anti-adenosine substance, stimulates the base of the forebrain and affects sleep.[54]

2.5) Control of sleeping and waking systems

Sleeping and waking status are cyclic theories, and suppression of one another between VLPOs and the wakefulness of hypocretinergic-aminergic system. There are bi-stable conditions, called the "Flip-Flop switch"[182] or the "Sleep Switch".[54] Creating sleeping and waking states. When VLPOs are stimulated during sleep, other systems will be suppressed. When nerves are stimulated during we are awake, VLPOs will be suppressed. Thus, sleeping and waking cycles are controlled by stimulation models that are balanced and stable.[182]

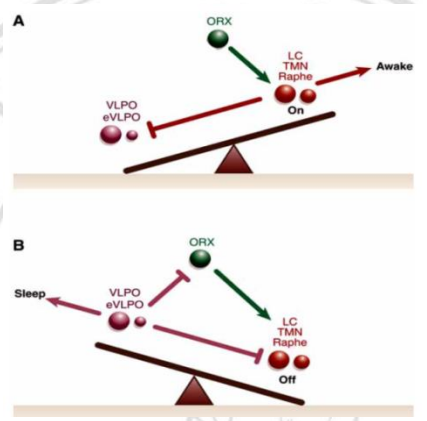


Figure 1.10 Flip-flop switch control[182]

Source: Adapted from Saper 2005, p. 1259 [99].

Adolescents should have proper sleeping standards, according to ages, by specifying regular sleep-wake rhythms.[52, 62] Adolescents' sleep models depend on biological changes of the body, minds, emotions, behaviors from growth into adulthood, and factors in the area of personal data including gender, grade level, family, school, and social culture. Family lifestyles, which will be required to adapt to social and environmental change, can influence sleep hygiene,[51-53, 57, 62, 179] affect the sleep architecture, and change the homeostatic and circadian regulation of sleep.[52, 56]

3) Sleep disorders

The International Classification Sleep Disorder (ICSD) criteria divided sleep disorders into eight groups as follows (AASM 2005):[146, 150-151, 182]

3.1) Insomnia is the symptom of difficulty in becoming drowsy, continuing sleep, waking early, and having poor sleep quality.

3.2) Sleep-related breathing disorders are sleep problems relate to abnormal breathing, including problems from snoring and resistance of the upper respiratory tract, respiratory obstruction, and rapid breathing in obese persons. All of which are the causes of chronic sleep obstruction.

3.3) Hypersomnia is the problem resulting from excessive sleep, not caused by problem of the circadian rhythms.

3.4) Circadian rhythm sleep disorders are sleep disorders caused by abnormal circadian rhythms, such as working in shifts or changing places, time zones, or medications, resulting in disturbance of the 24 hours biological time set system.

3.5) Parasomnias are disorders relate to the sleep process caused by central nervous system stimulation. Disorders occur while we are waking from REM sleep or, in some patient, from NREM sleep. Patients are found to have nightmares, delusions, fear, bed wetting, grinding teeth, and sleepwalking.

3.6) Sleep-related movement disorders are sleep with movement disorders such as restless leg syndrome and nervous system disorders, caused by leg movement to reduce discomfort in legs.

3.7) Isolated symptoms are separate sleep disorders with clearly different characteristics from normal sleep without corrections.

3.8) Other sleep disorders such as the following:

3.8.1) Narcolepsy: Patients with narcolepsy are patients who oversleep during the day, accompanied by REM sleep disorder.

3.8.2) Psychiatric disorders: Patients with psychiatric problems such as depression, etc.

3.8.3) Alcohol abuse-related disorders: Patients with alcohol abuse-related disorders have problems with sleeping and waking, lead to more sleeping problems.

4) Factors causing insomnia

Children and adolescents sleeps usually have the following physiological, psychological, and social problems:

4.1) Gender and age were factors relate to sleep quality. Insomnia prevalence differs with age and gender, with males being found to have higher prevalence than females.[55]

4.2) Brain development, controlling waking and sleeping.[188]

4.3) Physical diseases and sleep problems. Physical health problems are relate to sleep quality (r=0.51; p <0.05).[4] The following health problems relate to sleep are frequently encountered.

4.4) Endocrine Diseases and Metabolism Disorders

4.4.1) Obesity is a metabolism disorder usually relates to sleep problems.[189] Prevalence of sleep apnea is found in males with BMI more than 39 (40%) and pre-menopausal women at 3%. Most importantly, weight loss has improved sleep apnea problems[189] and the obese population (BMI $\geq$40) has poor sleep efficiency for 10.5 days or more than the population with normal weight (OR1.7; 95%CI 1.5-1.8).[190-191]

4.4.2) Diabetes patients with glycemic control problems highly correlated with day-time sleep (r=0.239). Diabetes has related to sleep quality (r=0.325; p<0.05).[192] Prevalence of patients with type 2 diabetes mellitus and obesity who have moderate to severe sleep apnea have been found at 70%.[189, 193] Furthermore, abnormal sleep structure, poor sleep quality, sleep difficulty, frequent waking, reduced sleep efficiency, lower sleep duration and drowsiness are encountered more frequently in the diabetes patients.[193]

4.5) Multiple sclerosis usually occur in adults who have sleep problems (45.3%).[194]

4.6) Heart failure[(189)] has been found to be a risk factor for obstructive sleep apnea, causing significant drowsiness during daytime (OR 1.5).[195]

4.7) Gastroesophageal reflux disorder[189, 191] causes difficulty in the beginning of sleep. This is usually in obese persons. Furthermore, persons with gastroesophageal reflux disorder are in a moderate risk for daytime drowsiness.[195]

4.8) Respiratory disorders and sleeping problems.[191] There are usually caused by lack of oxygen and carbon dioxide accumulation. These usually occur in acute and chronic forms such as asthma or emphysema, etc.

4.8.1) Patients with asthma usually have difficult breaths during sleep, especially before waking. Patients with asthma (74%) have been found that they need to wake up at night at least once a week.[196] Furthermore, patients have sleep difficulty and wake up frequently after going to sleep, in addition to have decreased sleep efficiency. Asthma poses hundreds of times higher risk of presenting symptoms at 4:00-5:00 a.m.[196]

4.8.2) Severe chronic obstructive pulmonary disease usually results in worse gas exchange during sleep and increases carbon dioxide accumulation, resulting in poor quality of sleep. Patients with chronic obstructive pulmonary disease have been reported to insomnia at 48.1% (OR 2.4).[197]

4.8.3) Allergic rhinitis (AR)[196] and non-allergic rhinitis (NAR) are usually triggered by allergens in the environment, resulting in stimulation of the immune system and causing inflammation. Symptoms usually occur at night during sleep. Allergic rhinitis has created obstruction for ≥5 nights per month. Udaka et al. in Japan found patients with allergic rhinitis to have sleep apnea (OR 5.22) and drowsiness during the day (OR 2.17).[198]

4.9) In the area of neurological diseases with sleep problems, adolescents with chronic neurological disorders are found to have sleep walking (57%) and drowsiness during the day (>50%). Neurological diseases are more frequently found in boys than older girls.[199]

4.10) Acute pain relates to sleeping problems will find the delta waves in patients' EEG. Delta waves are types of brain waves indicating shallow sleep, found in patients with medium brain injuries. Beta and gamma waves have indicated higher level of awareness (p<0.04). Furthermore, beta frequencies during NREM sleep are found more frequently, thereby indicating that pain relates to electroencephalography of sleeping problems.[200] In China, adolescents are found to have chronic pain and sleeping problems (19.1%).[201] Moreover, sleep quality has been improved in patients after knee surgery, who had less pain.[202]

4.11) Physical illness or discomfort such as pain, chronic coughing, sleep difficulty, and waking up to urinate frequently,etc.[188]

4.12) Headache will cause sleep problem. Children with migraines are found to have insomnia (55.2%) more often than children who have tension-type headaches (OR 3.45; 95% CI: 1.45-8.22).[142] Children with headaches are usually found to have sleep difficulty, insufficient sleep, frequently waking during the night, nightmares, and day time fatigue.[42]

4.13) Psychiatric problems, psychological problems, and anxiety are related to sleep disorders in both quantity and quality means, including decreased ability of works during day time. Augner (2011) found sleep quality to be related to depression (r=−0.57)

and anxiety (r=−0.54, p<0.01).[74] Depression creates risk for sleep disturbance in children and adolescents (OR 2.47-3.90). According to epidemiological studies, patients with depression have suffered from insomnia (90%),[75] which usually caused by problems regarding relationship between parents and family problems, relationship between teachers and school, separation anxiety, and loneliness (OR 2.52; 95% CI: 1.15-5.49).[51, 74] Furthermore, psychiatric problems such as Tourette syndrome, etc. require medications that cause sleep disturbance.[163]

4.14) Physiological abnormalities of patients who are more sensitive than normal, such as patients with high awareness causing them to be easily excited, etc.[188]

4.15) Sleep problems caused directly by sleep diseases such as abnormal limb movement during sleep, periodical sleep apnea, and sleep walking, etc.[188]

4.16) Use of some medications or substances, such as caffeine beverages, alcoholic beverages, and nicotine causes sleep disturbance.[189] Use of alcohol and marijuana has increased sleep problems in females more than males.[203] Sleep apnea is usually found in alcoholics, while problems of persistent insomnia are found even after the patient stopped alcohol consumption.[166] Furthermore, insomnia has caused severe drowsiness during daytime in females (OR 1.5), alcoholic (OR 1.4), overdosed on sleep medications (OR 2.5), and used pain relief medications (OR 3.4).[195]

4.17) Environmental changes such as bright lights, loud noises, hot temperatures, changes in sleeping spaces, traveling across time zones, and working on late night shifts have disturbed the normal circadian cycle.[188]

4.18) Improper sleeping behaviors and hygiene from poor sleeping behaviors over a long period of time, which usually begins in childhood, cause loss of sleep balance and result in inadequate sleep, daytime fatigue, lack of motivation, and depression.[188]

5) Sleep hygiene as follow:[56]

5.1) Bedroom environment, darkness, appropriate temperature, and avoidance of other activities in the bedroom such as using computers and lying down to watch television.

5.2) Set bed times and regular waking hours with flexibility of no more than one hour.

5.3) Enjoyable and relaxing activities. Avoid emotionally stimulating activities before sleeping.

5.4) If a person is unable to sleep after more than 30 minutes in bed, the person should get up to do other activities until he or she feels sleepy.

5.5) Children should be supported to sleep by themselves by sending children to bed when children begin to feel sleepy.

5.6) Avoid daytime napping near sleeping hours at night.

5.7) Playing sports or exercising regularly helps promote good sleep quality.

5.8) Hunger is a barrier to sleep. Eating snacks before sleeping can help to improve sleep.

5.9) Avoid food or beverages containing caffeine.

5.10) Organize studying hours not to be excessive.

6) Sleep quality

Sleep quality means a person's perception of his sufficiency and satisfaction of sleep. Sleep quality is complex[204] for concrete measurement and not only sleep quantity.[205] The ordinary population have been disturbed sleep qualities at 15-35%.[204] Sleep quality is composed of the quantity aspect of sleep and the qualitative aspect of sleep.

6.1) Quantity aspect of sleep

6.1.1) Sleep latency is the period when the person determines to sleep until the person is able to sleep. Easy sleep takes less than 15 minutes. Normal sleep takes no more than 30 minutes. Sleep latency exceeding 30 minutes indicates difficulty in initiating sleep.[204]

6.1.2) Sleep duration is the period from entering sleep until the person wakes up, this doesn't include waking periods during sleep.[206] Normal sleep duration differs from age such as 16-20 hours per day for infants, from birth to 3months. Sleep duration is the period alternated with waking periods, it is related to hunger and being full. Sleep duration is not related to daytime and night time. School-aged children need 10-11 hours to sleep. Adolescents need 9 hours, and adults need 8 hours.[150]

6.1.3) Number of arousal during sleep causes inconsistent sleep. Being aroused once means occurrence of awareness during sleep more than 15 seconds. Being

aroused more than three times per night or sleep difficulty after waking leads to inconsistent sleep. Persons with consistent sleep duration will have good sleep qualities.

6.1.4) Sleep efficiency is the ratio between actual sleeping duration each night to hours that the people will spend on bed. Normal sleep efficiency is more than 75% and will result in good sleep quality. It can be calculated as following:[207-208] One day, people require eight hours of sleep, 30 minutes to sleep, and 30 minutes to wake up in the morning before schedule. If a person wakes up during the night time for an hour, sleep efficiency will be 6/8 hours or 75%.

Measurement of sleep quantity in all four aspects clearly indicates good sleep models, predict sleep quality, and specify satisfaction in sleep.

6.2) Qualitative aspect of sleep

6.2.1) Sleep quality is a subjective aspect of sleep, i.e. “good”, “bad”, “sufficient”, “insufficient”, “deep” or “shallow” sleep, including feelings of being “fresh” or “fatigued” after waking. Satisfaction towards sleep results in good sleep quality.[206]

6.2.2) Impact on activities in daily living from poor sleep quality[206] can cause sleepiness, yawns, lack of enthusiasm, fatigue, or lack of concentration. Sleep quality is relate to the function or activity during day time.[205] Activities during the day, after waking up, are variables to judge sleep quality.

7) Effects from sleeping problems among children and adolescents on intrinsic and family.[56]

Emotion: Insecure emotions, irritation, being easily angered, and lack of emotional control, leading to more anxiety.

Behavior: Impaired focus, repeated thoughts and actions, aggression, stubbornness, resistance, risk behaviors, and sleeping during day time.

Learning: Impaired concentration, creativity, and management such as decision-making, reduced problem-solving, and lack of academic capacity.

Body: Fatigue, negative effects of the heart, the immune system, metabolism, and the endocrine system, exacerbated illnesses, and likelihood for accidents.

Family: Effects on parents’ sleep, fatigue during daytime, fluctuating emotions, conflicts with children, internal family stress, and disagreement with parents.

8) Sleep quality assessment

Most ordinary sleep quality assessment methods use self-assessment forms to show sleep conditions. Subjective behavioral and psychological assessments use many types of measuring forms. The Pittsburgh Sleep Quality Index: PSQI is a popular sleep quality measuring instrument, used worldwide with accuracy scores and accepted confidence scores. The form is translated in many languages including Thai. The form has limitations in reporting real time. Reporters may be unable to report sleep time accurately and sleep hours for the entire night, but the form is able to assess quality of sleep. Furthermore, PSQI is a sleep data collection form indicating quantity and quality of sleep. Thus, the form can be applied to every aged group. Sleepiness or naps during the day must also be assessed.[55-56] The Epworth Sleepiness Scale (ESS) is usually used as the form for measuring chance of sleepiness during the day, by using short questions in order to assess sleeping problems. ESS was used in 1991 by Murray Johns in Australia, as a measuring form related to sleep disorders.[209] The scale had eight questions on past events, a sensitivity of 93.5% and specificity of 100% in diagnosing excessive sleepiness.[210]

At the moment, sleep behaviors can be measured objectively by using wrist actigraphy instruments attached to the wrist. It has the capacity to analyze movement during sleep in one night. Furthermore, measurements of physical function during sleep can be done in the laboratories by using polysomnography (PSG) to measure EEG, EOG, EMG and ECG, all of which are gold standard of sleep measurements that are both expensive and require expertise in handling instruments.

Therefore, subjective forms for measuring sleep quality are continuously used, without the data regarding sleep architecture. However, sleep quantity and quality can be measured with easy management with inexpensive prices and broad survey ranges.[211]

9) Components in assessing sleep disorders

Sleep quality components have different degrees of significance in each person.[204] Therefore, self-assessments are important in measuring sleep quality[56, 211] using the following data:

9.1) Sleep history, recorded using sleep tables in order to show sleeping disorders, including the time when the person goes to bed until the person is asleep, with capacity to indicate difficultly or ease in sleeping[55] by recording the following data:

9.1.1) Normal bed times, regular bed time schedules, durations, places, sleepiness before going to bed, and refusal to go to bed including evening activities and regular activities before going to bed such as watching TV, using computers, reading books, and doing homework, etc.[55]

9.1.2) Sleeping hours at night. Since the person goes to sleep, sleeping behaviors at night, number of times and length of time when the person wakes up during the night, respiratory disturbance, frequent urination, night time behaviors[55] or problems caused by the bedroom environment, familiarity, lights, sounds and temperature.[56]

9.2) Behaviors during the day, waking hours, and daytime naps[55] are assessed in the following areas:

9.2.1) Daytime weakness or fatigue

9.2.2) Capacity during day time, ability in school, and socialization.

9.2.3) Relationship between family and incidents in life with severe effects on our minds.[55]

9.2.4) Food, caffeine beverages, history of medications, and current use of medications.

9.2.5) Use of technology and education.

9.3) Sleeping models, data on sleep, sleep duration from the time when the person goes to sleep, number and length of waking during sleep,[56] total sleep duration, and nap duration. It's usually used diary records of two weeks.[55]

9.4) Family history related to sleeping problems.[56]

9.5) History of frequently encountered psychiatric disorders among children and adolescents, with sleeping disorders consist of the following:[55, 212]

9.5.1) Children or adolescents with ADHD are usually found to take longer time to go to sleep after going to bed with shorter sleep durations at night. The prevalence is at 25-50%.

9.5.2) Children or adolescents with autism have sleeping problems at a prevalence of 44–83%.

9.5.3) Children or adolescents with emotional disorders such as depression and anxiety.[74]

9.6) Sleep hygiene[55]

9.7) Physical examinations[56] in the areas of development, growth, ear, neck, nose, oral, and nervous system examinations.

Sleep quality assessment forms for children will assess sleep, sleepiness, substance abuse, use of sleep medications, anxiety, and depression by assessing the past two weeks. [206]

10) Research of electromagnetic energy from smartphones and sleep.

Adolescents are currently growing up in the age of modern technology[11] with changes in the activities of daily living (ADL) according to technology. Electrical appliances and electronic media can be found in adolescents' bedrooms (75%).[77] Use of electronic media and talking on smartphone before going to sleep have related to the sleeping problems (77%).[64, 77-82] Lights from computer and smartphone screens are light-emitting diodes (LED) and short-wavelengths in the blue range. Contact with light-emitting diodes in the evening for five hours was encountered. In the meantime, Wood et al. found that contact for as long as two hours[212] could suppress melatonin in the body, thereby causing reductions in slow eye movements and low frequencies (1-7 Hz) EEG in frontal brain regions, led to less sleepiness, higher wakefulness, and problems for sleep quality.[212-214] Furthermore, excessive use of entertainment media causes adolescents to sleep late with shorter sleep durations, although it has not been correlated with weekend period because adolescents are able to wake up later than usual.[83] Moreover, excessive use of entertainment media has induced separation from society, causing depression and suicidality.[178] Adolescents currently have likelihoods to increase sleepiness during the day and more tea or coffee consumption, indicating insufficient sleep duration[64, 78] and creating concerns in the world for health problems caused by modern technology. The following studies on electromagnetic energy from smartphones and the effects of sleep problems have yielded the following findings:

10.1) Jarupat found mean ears temperature higher during periods with continual smartphone use. Moreover, melatonin levels in saliva among telephone users were lower than among the groups which did not use mobile phones (p <0.05). [215]

10.2) Huber et al. experimented by using patch antennas and dipoles to send electromagnetic energy from mobile phones among healthy adults. Alpha brain waves were found in the period of entering NREM sleep and spindle waves were found during NREM sleep, with higher frequency and no statistically significant differences. This occurred because electromagnetic energy was low. Electromagnetic energy caused

intense electrical stimulation to the thalamus and induced spindle wave vibrations, which might cause sleep problems due to exposure over long periods of time. [216]

10.3) Achermann and Rigel experimented in healthy adults aged 20-26 years who came into contact with electromagnetic energy at SAR 0.2 and 5W/kg, only on the left side for 30 minutes. Alpha brain waves, spindle frequency during NREM sleep in Stage 2, and slow Wave Sleep during NREM sleep in Stages 3-4 would have higher frequency. Furthermore, sleep latency was longer based on electromagnetic energy intensity. Moreover, electromagnetic energy led to less local blood circulation, especially the areas below the inferior temporal cortex with blood circulation increasing according to longer distance in the prefrontal cortex. [217]

10.4) Perentos et al. experimented with a cross-over model by having contact with electromagnetic energy from GSM mobile phones for 15 minutes. However, no changes to the Alpha Band were found. This might have been caused by contact at different amounts. [218]

10.5) Supe reported the contact with telephones before going to sleep could increase the frequency in the spindle range. [219]

10.6) Kesari et al. experimented in lab rats aged 3-5 years, by allowing contact with electromagnetic energy at a microwave frequency of 2.45 GHz with SAR of 0.14W/kg for a period of two hours in 45 days and found melatonin reductions. [220]

10.7) Thoméeet al. surveyed and monitored the population in the age of 20-24 years in Sweden. Reports of sleep disorders and depression among many of the group who used mobile phones were found, with most of the problems encountered in groups who reported stress from using mobile phones. [221]

10.8) Gradisar et al. found that American adolescents possessed and used mobile phones (72%) and electronic devices before sleeping, causing difficulty in sleeping and sleepiness after waking. [222]

10.9) Nathan and Zeitzer surveyed sleep during daytime by using ESS forms and mobile phones. The study was found ESS scores to be correlated with women who felt the need to access mobile phones at all times and usually used mobile phones at night. [31]

10.10) Cao et al. experimented with contact with electromagnetic energy at a radio frequency. He found reductions of melatonin and anti-oxidant enzymes such as glutathione peroxidase and superoxide dismutase, etc. [223]

According to the literature review, electromagnetic waves would have two important processes influencing quality of sleep. Changes of brain waves during NREM sleep in Stage 1 cause longer NREM sleep and lengthening sleep latency. Rigel et al.[219] found that sleep latency depended on electromagnetic energy intensity, with potential effects on the hypocretinergic-aminergic nerves controlling NREM sleep or the ARAS which controlled both NREM and REM sleep by causing disturbance in secreting neurotransmitters related to sleep.

Reductions in melatonin levels relate to heat from electromagnetic energy stimulate the pineal gland, which function to secrete melatonin. Furthermore, the light from mobile phones is Light-Emitting Diodes (LED) and in the blue range, causing increased in melatonin suppression in the body and problems in sleeping.

Nowadays, health impacts, including headaches and sleep problems from electromagnetic energy in smartphones, remain inconclusive. While the rate of smartphone use increases rapidly. Smartphone use worldwide is expected to increase to 1.4 devices/person.[7] Smartphones are usually used close to the heads, causing the brains to come into contact with electromagnetic emissions and disrupt nervous system's functions, resulting in headaches and sleep problems, especially among children and adolescents or "digital age" people[76] who have the highest smartphone uses with continuously increasing trends. Therefore, studies of health impacts from electromagnetic energy in smartphones are aimed at creating preventive guidelines based on precautionary protection principles.[85]

1.5 Scope of the study

The study has conducted among high School Students in Wattanothai School in Chiang Mai Province, who have the same characteristics as high school students nationwide. The study is divided into two phases on October-December 2015. The objectives of the study are to explore the characteristics and the prevalence of mobile

phone use, headache and sleep problem among high school students, the relationship between smartphone output power, and headache and sleep quality among high school students.

Therefore, to study the effects of exposure to electromagnetic radiation from smartphone use on headaches and sleep quality among high school students has designed at Chiang Mai's provincial high school with a conceptual framework is shown in Figure 1.11.

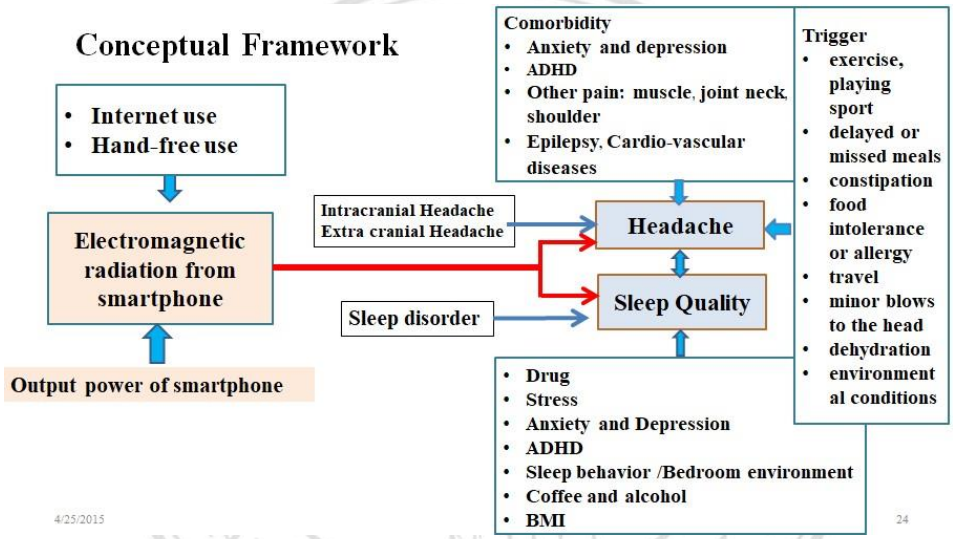


Figure 1.11 Theoretical conceptual framework

1.6 The benefit of the study

The findings of the study can be used as a data to create a system for monitoring and preventing, based on a precautionary protection principle. The findings can also provide data for the preparation of measures or guidelines to promote and support safe smartphone technology use among high school students and other population groups.

The smartphone is a modernize technology which has been designed to meet divesre needs. The previous study found that electromagnetic radiation from smartphone affected nervous system. The technology is worthless without properly using, the researcher has aimed to investigate the correlation between electromagnetic radiation from smartphone and nervous system.

CHAPTER 2

Methodology

The objectives of the study are exploring the characteristics and the prevalence of mobile phone use, headache and sleep problem among high school students in addition to study the relationship between smartphone output power, headache and sleep quality among high school students. The research has been designed in the following two stages: Stage 1 involved to study the characteristics and prevalence of mobile phone use, headache and sleep quality among high school students. Stage 2 is subjected to study the correlations between smartphone emissions; headache and sleep quality among high school students, therefore the subjects have been selected from stage 1 based on set inclusion and exclusion criteria for participating in the time series study.

2.1 Part 1: Mobile phone use and health problem

Objective 1: To study the characteristics of mobile phone use, headache and sleep quality among high school students.

To explore the characteristics of mobile phone use, headache and sleep quality in a situation with mobile phone technology changes in smartphones from telephone conversations to text messages, images and motion pictures from communication to entertainment. The data has involved in the research included studies of headache and sleeping characteristics among adolescents in the current digital era. At this stage, the data has included demographic data, data on illness and health-promoted behaviors in order to select students for entry into the second stage of the study, which is a prospective time series study.

2.1.1 Population and sample group

1) Population study

The population was all high school students (grade 10-12) at a provincial school in Chiang Mai (1,422).

2) Sample

The sample size has been calculated by determining headache from mobile phones prevalence in South Korea at 10%[49] according to the sixth equation[224-225] as specified.

- Two-sided significance level (1-alpha): 99
- Power (1-beta, % chance of detecting): 90
- Population (N) = 1,422
- Proportion (p) =0.1
- Error (d) =0.01
- Z (confidence level) =1.96

$$n = \frac{Np(1-p)z^2_{1-\frac{\alpha}{2}}}{d^2(N-1)+p(1-p)z^2_{1-\frac{\alpha}{2}}} \quad (1)$$

$$= \frac{1{,}422(0.1)\,(1\text{-}0.10)\,1.96^2}{0.01^2(1{,}422\text{-}1)+(0.1)\,(1\text{-}0.10)\,1.96^2}$$

= 1008 (add5%) = 1,058 students

3) Sampling

3.1) Stratified random sampling[224, 226-227] has been used because of age and gender factors have been related to headache and sleep quality. Therefore, the samples are divided by class, which represented age and gender divisions to control confounder variables, gender and age as shown in Figure 2.1.

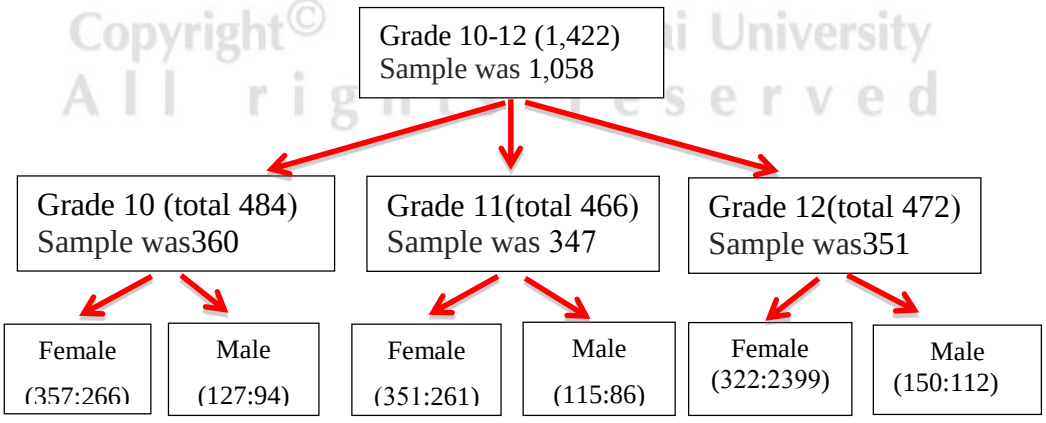


Figure 2.1 Flow of sampling divided by class and gender

3.2) Systematic random sampling[224] is a sampling in which all of the samples have equal opportunity for being selected by determining distance between the samples and selecting based on the ratio of classes and gender groups in each grade before sampling by using computers.

3.3) The inclusion criteria has consisted of Grade 10-12 high school students at a provincial school in Chiang Mai.

2.1.2 Research design

The study is based on a cross-sectional research design[224, 226, 228] aimed at determining the prevalence of diseases or problems by collecting data of interest prospectively over a period or point of time.

2.1.3 Data measurement

1) Questions about headache type and prevalence sleep quality and mobile phone use characteristics have been contained in the questionnaires created by the researcher based on the literature review and tested for content validity by three qualified experts. The questionnaires have been tested in 30 students in order to have reliable results by calculating internal consistency from cronbach's alpha that is 0.715. The questionnaires have consisted of the following:

1.1) Demographic data such as gender, age, weight, height, class, family income and family residence.

1.2) History of illness, chronic diseases and medication adherence.

1.3) Health-related behaviors such as smoking, alcohol consumption, food consumption and exercise.

1.4) Headache factors such as headache characteristics and quality, headache duration and frequency, and headache triggers.

2) Questions about mobile phone usage characteristics have been modified from the study of Chu et al., 2011[49] concerning the age when a person becomes a smartphone owner and user, number of smartphones used, smartphone brands, smartphone characteristics, methods for using smartphones, methods for holding smartphones during conversations, daily smartphone conversation frequency, duration of each smartphone conversation, hand-free device use, speaker phone use during

conversations, burning sensations around the ears and face and smartphone use before sleeping.

3) The Pittsburg Sleep Quality Index (PSIQ) has been translated into Thai by Chanamanee which has been used to assess sleep quality internal consistency, which has a Cronbach's alpha of 0.78.[207] The sleep quality assessment form consisted of seven components. Each component has scores based on selected responses with scores of 0-3 points and a total posible scoring range of 0-21 points. Overall scores of less than or equal to five points have meant good sleep quality. Overall scores of more than five points have also meant poor sleep quality.[204]

4) An assessment scale has been used to provide baseline data for comparisons and to exclude the samples based on exclusion criteria as follows.

4.1) The Headache Impact Test (HIT6) has been an assessment form used in the headache clinic, Maharaj Nakorn Chiang Mai Hospital. The test has been used to assess headache severity and impacts over the four weeks preceding the study with six questions. Each question has a scoring range of 6-12 points with total scores divided into the following groups:

Scores ≤ 49 points indicated no impacts from headaches.

Scores of 50-55 points indicated slight impacts.

Scores of 56-59 points indicated impacts from headaches causing students to stop learning.

Scores of >59 points indicated severe headaches with impacts on learning, family and activity participation.

4.2) The Epworth Sleepiness Scale (ESS) has been used to assess daytime dozing. The form has been translated into Thai by Sookying[229] and was not tested for content validity and reliability. The form has assessed sleepiness over the four weeks preceding the study. Scores are divided into 0-3 points for each question with a total of eight questions or 24 points.

Scores of 2-10 points indicated normal sleepiness

Scores of 11-15 points indicated minor sleepiness[184]

Scores of more than 15 points indicated high sleepiness

4.3) The Hospital Anxiety and Depression form (HAD) has been used to assess depression and anxiety. The form has been translated into Thai by Ninchaikowit and colleagues.[230]

The sensitivity of the anxiety assessment form is 100% with specificity at 86%. The anxiety assessment form was related to clinical diagnosis at 88.33%. Internal consistency has a cronbach's alpha of 0.86.

The sensitivity of the depression assessment form is 85.71% with specificity at 91.3%. The anxiety assessment form was related to clinical diagnosis at 90%. Internal consistency has a cronbach's alpha of 0.83.

The HAD contains 14 questions with seven odd-numbered questions on anxiety and seven even-numbered questions on depression. Likert scale ranking has scoring values of 0-3 points per question divided into anxiety and depressions cores with a score range of 0-21 points.

Scores of 0-7 points	indicated the subjects are normal.
Scores of 8-10 points	indicated groups with high anxiety or depression.
Scores of 11-21 points	indicated groups with anxiety and depression considered to be psychiatric abnormalities.[195]

4.4) The sleep hygiene assessment form has been translated into Thai by Chanamanee which accuracy is cronbach's alpha accuracy.[207] The sleep hygiene assessment form contains 21 questions with 13 positive questions and 8 negative questions. Each question has responses to be selected with scoring from 0-4 points as follows:

Positive Questions	Answer	Definition	Scoring
	Never	No behaviors/1 week	0 point
	Sometimes	1-2 behaviors/week	1 point
	Frequently	3-4 behaviors/week	2 points
	Regularly	More than 5 behaviors/week	3 points

Negative Questions	Never	No behaviors/1 week	3 points
	Sometimes	1-2 behaviors/week	2 points
	Frequently	3-4 behaviors/week	1 point
	Regularly	More than 5 behaviors/week	0 point

The total scoring range is 0-63 points. High scores mean better sleep promoted behaviors than low scores.

2.1.4. Data collection

In the present study, the researcher collected data by coordinating with research assistants to collect data according to the following steps:

1) The researcher has used a letter of introduction from the Faculty of Graduate Studies, Chiang Mai University, to contact a school in Chiang Mai to ask for permission to conduct the study, explain objectives, research procedures and instruments in addition to notify and schedule the date for conducting the research in the area.

2) The researcher has instructed the students and teachers who are research assistants to complete the questionnaires and complete questionnaires via electronic mail. The researcher has also explained the meaning of each question in the questionnaire.

2.1.5 Data and statistical analysis

1) Descriptive Statistics

1.1) Data has been collected on demographics, health, headache, characteristics of sleep and mobile phone use, all of which have been analyzed by using frequency distribution and percent.

1.2) Data on headache characteristics has been used to classify headache types categorized based on ICHD 3 beta. Scores are classified based on the WHO guidelines for categorizing headache types by symptom clusters. The first headache type classified as migraines and groups are divided by scores where 3=migraines, 2=potential migraines and scores of less than 2=uncategorized patients. The group with scores of less than and equal to 2 points (potential migraine and uncategorized groups will be re-calculated in the TTH group) has been calculated for TTH. The scores are then divided by scores where 3=TTH, 2=potential TTH and scores of less than 2, which classified as undetermined headache. Groups with scores of 2 points in the prevous

migraine group that are not categorized as TTH but are categorized as students in the potential migraine group.[122-123]

1.3) After conducting analysis to divide headache types, headache has been collected into the following three groups.

1.3.1) The migraines with potential migraines group are classified to the migraines group.

1.3.2) The TTH with potential TTH group are classified to the TTH group.

1.3.3) Groups that could not be categorized will be classified to undetermined headache group.

The researcher has analyzed for frequency distribution and percent. The confidence interval (CI) is 95% and percent of symptom has been compared for each variable.

1.4) The impacts of headaches have been obtained by calculating overall scores and analyzing mean maximum, minimum and standard deviation.

1.5) Data on sleep latency, sleep duration, sleep efficiency, sleeping problems, sedative use, impacts of sleep problems on activities/education and sleepiness after waking has been analyzed with frequency distribution and percent. Then each domain of sleep quality has been calculated into PSQI scores. Analysis has been carried out to determine mean, maximum, minimum and standard deviation. The groups are then divided by frequency distribution and percent. In addition, the researcher has calculated prevalence and a 95% Confidence Interval (95% CI) in order to compare the prevalence for each variable.

1.6) The scores of data on dozing, anxiety and depression have been combined, calculated and divided based on anxiety and depression. Analysis has been carried out to determine mean maximum, minimum and standard deviation.

2) Inferential statistics

2.1) The methods of calculation has been used to compare frequency and mean differences consist of Chi-square, t-test, ANOVA and *p*-value <0.05.

2.2) To calculate the risk factors on magnitude of smartphone use, demographic data causing headache, headache related to smartphone use and sleep quality have been assessed. Multiple logistic regression has been used to calculate odds

ratios (OR) and a 95% confidence interval (95% CI) obtained with p-value <0.05. Factors are controlled by adjusting related variables such as demographic data, health data and confounding factors.

2.2 Part 2: Electromagnetic radiation from smartphone and health problem

Objective 2: To study determine the effects of smartphones on headache among high school students.

Objective 3: To study determine the effects of smartphones on sleep quality among high school students.

The study is an analytic epidemiological prospective study, or a time series study, conducted in a group with exposure events and specific outcomes in the form of data sets with time orders.[189] This is periodic measurement and data showing natural phenomenon generally shown based on the order of data. Furthermore, values can be predicted to benefit planning and correction.

2.2.1 Population and sample group

The population is composed of the students who participated in stage 1 of the study. The 200 samples are composed of grade 10-12 high school students who were selected from Stage 1 based on the inclusion and exclusion criteria.

1) Inclusion criteria: The subjects have owned at least one smartphone. Parents have no history of migraines. Weight to height is not more than the 95th percentile based on standard weight criteria showing obesity. The subjects have not worked after 8:00 p.m. The subjects have no daily health-related behaviors including liquor, coffee or tea consumption and smoking. The subjects have no disease or health problems diagnosed by doctors and undergoing treatment. Questionnaires have been used with parents or guardians and students on the following topics: hypertension, diabetes mellitus, heart disease, severe trauma or brain or neck surgery, seizures, sinusitis, dialysis, hypothyroidism, asthma, ear infection and oral diseases such as dislocated jaws, jaw deviation, toothache, ataxia, vertigo, tremor, diplopia, localized seizures, meningitis, brain abscess, systemic infections, facial muscle pain, rashes, allergies, eyesight problems (myopia, presbyopia, astigmatism, squint-eyed and uncorrected eyesight). Also, questionnaires have been used to determine regular medications as treatment for hypertension, blood vessels in the brain, occlusions, seizures, heart disease, pain relief,

allergy, medications for reducing mucus, narcotic substances, psychiatric problems such as attention deficiency, current treatment for Tourette syndrome or tics (abnormalities in nervous system development), phobia or inability to eliminate fear from thoughts and encounter with psychological traumatic events such as violence, torture, rape, abandonment, etc. No sleep disorders are encountered to use questionnaires with parents or guardians on the following topics: hypersomnia, narcolepsy, sleep apnea, sleep walking, bed wetting and restless leg syndrome.

2) Exclusion criteria

2.1) Depression or anxiety was determined by HAD with anxiety or depression scores of 11 points and up

2.2) Sleep hygiene scores of less than or equal to 20 points

2.3) Severe illnesses or injuries

2.2.2 Research design

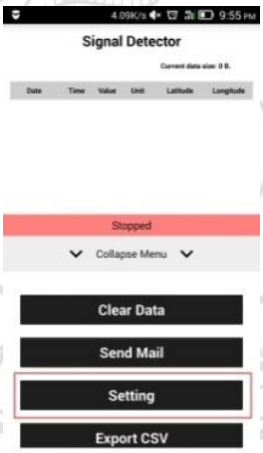
The study is an analytic epidemiological prospective study, or a time series study, conducted in a group with exposure events and specific outcomes in the form of data sets with time orders.[189] This is a periodic measurement and data showing natural phenomenon generally shown based on the order of data. Furthermore, values can be predicted to benefit planning and correction.

2.2.3 Data measurement

1) The application has been developed to collect smartphone output power on Android and IOS operating systems. Smartphones measured output power from smartphone antenna to the nearest area and the application has requested access to the aforementioned smartphone output power via the program's framework by setting to save every five minutes and transmitting saved data by email to a researcher. The students are allowed to delete the data in their computers after they had sent an email. New data could then be saved as shown in the following steps:

1.1) Install the signal detector program.

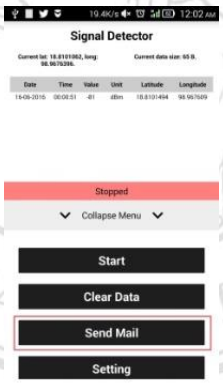
1.2) Click on “setting” to set the tester’s code for the person who will use the program in the test



1.3) Enter the tester’s code

1.4) Click on the “start” button to begin saving data

1.5) Click on the “stop” and “send email” buttons to send data. After data has been sent, click on the “clear data” button. Then press “start” again.



2) The measurement error

The thesis has measured the error of output power from smartphones by spectrum analyzers which considered as gold standard. The spectrum analyzers measure the magnitude of signals within the full frequency range of the instrument. The display of a spectrum analyzer is shown on the frequency tab on the horizontal axis. The amplitude is displayed on the vertical axis (see Figure 2.2).[231]

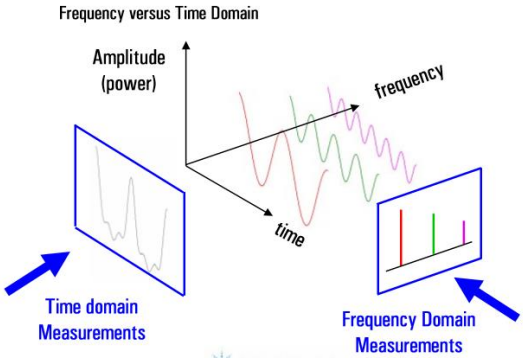


Figure 2.2 The signals measurement of spectrum analyzer.
Source: AgilentTechnology
https://www.keysight.com/upload/cmc_upload/All/Spectrum-Analysis-Back-to-Basics-Jan-2012-rev7-File2.pdf

The diferrent brands of smartphone have different output power, therefore, the same error figure has been used to adjust the output power to the same brand of smartphone. The measurement error in the study has been conducted in a normal room not a chamber room, therefore, the error adjustment might not be accurate.

3) The data obtained will be calculated by using the following parameters:

3.1) The mean of smartphone output power has been collected from the measurements taken at five-minute intervals for 15 minutes.[18-19,106,108]

3.2) Smartphone output power has been collected at four periods of time consisting of 12:01-6:00 a.m., 6:01 a.m.-12:00 p.m., 12:01-6:00 p.m. and 6:01 p.m.-12:00 a.m.

3.3) The mean of daily dosage data has been collected by applying the average time of exposure equation in OET Bulletin 56 of the Federal Communications Commission[90] as follows:

$$(2mW/cm^2) \times (3min) + (0mW/cm^2) \times (3min) = (1mW/cm^2) \times (6min) \quad (2)$$

By adjusting intensity into mean smartphone output power in the following new equation:

$$\text{Daily Dose} = \sum_{n=1}^{n}(\text{Average Output Power})n \times (\text{Duration Time})n \quad (3)$$

This equation “n” is the number of minutes measuring the average smartphones output power. The duration is the time of measuring smartphone power each time. The study has taken measurements every 15 minutes.

4) Diaries

4.1) The headache diary has been created based on the literature review and tested for content validity by three experts. The questions in the diary consist of time when headache has begun and finished, symptoms prior to headache, symptoms occurring with headache, headache triggers, headache severity, headache solutions, characteristics of pain, area of headache, headache side, and telephone conversations by the internet and hand-free or speaker phone use.

4.2) The sleep diary has been created based on the literature review and modified from the sleep diary of the National Sleep Foundation. The sleep diary has been tested for content validity by three experts. The questions in the diary consist of sleeping and waking times, latency of sleep from when the students slept, number of night time awakenings, total sleeping time at night, feelings upon waking up in the morning, sleep disturbances, barriers to medication adherence, activities and events from the morning to the evening before sleeping such as times and amounts of tea/coffee consumption, anxiety and depression, daytime dozing and activities before sleeping.

The smartphone usage has been included in the headache diary.

(1) Telephone conversations by using internet use such as line, etc.

(2) Hand-free device and speaker phone use.

2.2.4 Data collection

Stage 2 of the study has involved to collect data from students who participated in stage 1 of the project and have been selected based on set criteria. The students have been trained and tested by recording data in headache and sleep diaries. Data collection has been prepared as a smartphone application for the students participating in the study. Data has regularly been recorded every day over a period of two months (60 days) as follows:

1) Every episode of headache has been recorded when it began and finished in addition to all headache details. In case there is no headache occurring among the students, data has been recorded before sleeping.

2) Data on sleep has been recorded as follows:

2.1) Daytime activities have been recorded before sleeping.

2.2) Data on sleep has been recorded including sleeping and waking times, numbers of awakening, duration of sleep for the entire night, sedative use, and feelings after waking in the morning and after arriving at school.

3) Data on headache and sleep has been sent by email. The teachers who are research assistants have checked the data online in the morning and messaged students who do not make records via Line every day. The researcher corrected and scoring recorded daily sleep data to sleep latency, sleep duration, sleep efficiency, sleeping problems, sedative use, impacts of sleep problems on activities/education and sleepiness after waking. Then each domain of sleep quality has been calculated into PSQI scores. Sleep loss prevalence was calculated from number of students who had sleep loss <8 hours, sleep latency >20 min, sleep efficiency ≥85% and poor sleep (sleep quality score <5) by the total number of observations who completed a sleep diary.

4) Excluded data

4.1) Incidents in life with severe psychological impacts.

4.2) Severe illness and admission for hospital treatment.

2.2.5 Data and statistical analysis

1) Descriptive statistics

1.1) Demographic data, health data, headaches, headache severity, types of headache categorized by ICHD 3 beta, sleep quality data, smartphone usage data, anxiety and depression have been analyzed by using frequency distribution and percent.

1.2) Smartphone output radiation is the mean maximum and minimum and mean daily output power, which are continuous data collected in a time series.[189]

2) Inferential statistics

2.1) To compare frequency and mean differences will be calculated by using chi-square, t-test and analysis of variance (ANOVA).

2.2) The relationship between electromagnetic energy from smart-phone and dose-response assessments with headache has been measured for headache severity, pain frequency, duration of pain (continuous data), and event of headache (yes or no). In the sleep quality domain, PSQI and daytime dozing have been measured in continuous scores and divided into category data. Data analysis has been conducted by

using Generalized Estimating Equations (GEE), which is an extension of the Generalized Linear Models (GLM) used for dependent variables with correlated responses because data has been collected from the same person. Data in the same cluster (within-subject) is correlated. However, data between clusters (between subjects) has to be independent. In the analysis, therefore, the correlational structure has set for dependent variables by considering from Quasi Likelihood under Independence Model Criterion (QIC) and low QIC scores has been selected, which indicate that correlational structure is good and correct Quasi-likelihood under Independence Model Criterion (QICC) has been used to compare the models under one correlational structure. Low QICC scores indicate that a model is fit.[232-233]

2.2.6 Human ethical approval

The study was certified by the Institutional Review Board of the Research Ethics Committee, Faculty of Medicine, Chiang Mai University on 21 September 2014, and has renewed on October 2017.

The main objectives of the study are subject to explore the relationship of smartphone output power on headache and sleep quality among high school students. The methodology has been conducted in 2 phase for selecting the subjects and control confounder by inclusion and exclusion criteria. The subjects has been asked for recording headache and sleep daily and sent smartphone output power by email. The study has used GEE for analyzing the correlation of smartphone output power and headache and sleep quality.

CHAPTER 3

Result

3.1 Mobile phone (MP) use and health problem

Objective 1: To study the characteristic of mobile phone using, headache symptom and prevalence of sleep quality among high school students

3.1.1 The characteristic of mobile phone using, headache symptom among high school students

Use of smartphone, in fact a MP, with mid-range personal computer capacity and various application features[15-16 ,234] has increased rapidly because this device can serve a diversity of needs particularly among teenagers who grow up in the era of modern technology. It was estimated by the year 2016 that 26,000 million units of MP would be sold worldwide or about 1.4 MP/person.[7] In Thailand, the MP market is expected to grow at 24.9% annually.[10] Roughly 72% of Thai school children aged 6 years old and above reportedly owned MP (2012) and trends to increase among the number of young MP users.[11] The similar study in Singapore (2008) found the rate at 44.8%,[175] and that in Rayong Province (2014) 89.3%.[43] Furthermore, the study in 2011 revealed Thai teenagers ranked first in Asia for the length of time talking via MP average 60.7 minutes per day.[12] Previous studies also found the growing extent of headache prevalence and sleep problem associated with MP use in teenagers. Prevalence of headache increased[122] from 47-82% in 2006-2012[34, 35-39] to 83.1-94% in 2013-2015[40-42, 44] and sleep problem from 16.9-54.2% in 2000-2013[51, 55, 57-59] increased to 58.7-66% in 2013-2016.[53, 60] The investigation on the characterisitcs of MP use which give rise to headache and sleep problem in teenage MP users can provide useful findings for identifying measures to prevent the health impacts from MP use and to assure the safe use of MP.

1) The characteristic of mobile phone using and headache

Out of the total 1,422 high school students who are the population of the study, 1,058 students have been randomly selected, and 996 (94.1%) responded to the survey for the analysis (Fig. 3.1).

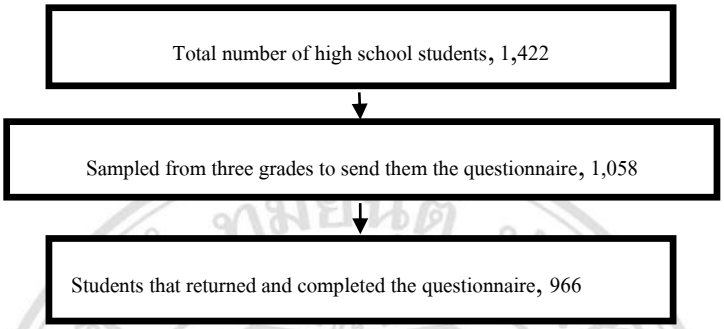


Figure 3.1 The sampling procedures and sample response flow chart

Table 3.1 Demographic characteristics of participants presented as percentage unless specified otherwise

Characteristic	**Grade 10** **N (%)**	**Grade 11** **N (%)**	**Grade 12** **N (%)**	**Total** **N (%)**
Gender				
Male	76 (23.0)	69 (21.6)	107 (30.9)	252 (25.3)
Female	254 (77.0)	251 (78.4)	239 (69.1)	744 (74.7)
Age, mean± SD	16.60±0.59	17.66±0.64	18.58±0.55	17.63±1.01
Range, min max	16-20	16-20	16-20	16-20
BMI, mean± SD	20.32±4.12	20.63±4.44)	20.92±3.79)	20.63±4.01
Overweight and obesity	38(11.7)	32(11.0)	30(8.7)	100 (10.1)
Underlying disease	69 (20.9)	75 (23.4)	75 (21.7)	219 (22.0)
Medicine using	12 (3.6)	6 (1.9)	5 (1.4)	23 (2.3)
Vision problem(no correction)	67 (20.3)	57 (17.8)	97 (28.0)	221 (22.2)
Head or neck injury	1 (0.3)	1 (0.3)	1 (0.3)	1 (0.3)
Potentially traumatic interpersonal events (PTIEs)	10 (3.0)	8 (2.5)	11 (3.2)	29 (2.9)
Phobia	21 (6.4)	14 (4.4)	14 (4.0)	49 (4.9)
Anxiety	6.6±4.4	6.8±4.5	4.5±4.8	6.8±4.6
Depression	3.5±3.0	3.7±2.7	2.7±2.6	3.5±2.8

1.1) Demographic characteristics

Most students are female 74.7% (744) with mean age 17.6±1.0 years old (range 16-20) (Table 3.1). The mean body mass index is 20.6±4.0 (85^{th} percentile, BMI in female 26.5-31.5, male 27-30.5).[161] The students with underlying disease, regular use of medicines, and vision problem have accounted for 20.5%, 2.3% and 22.2% respectively. The mean scores of anxiety and depression are 6.8±4.6 and 3.5±2.8 respectively.

1.2) Characteristic of MP use

The study has found that almost all of the students or 99.8% have owned MPs, with 99.9% being MP and 69.3% with Android and other brand of MP device (Table 3.2). The mean age when starting using MP of these students is 12.2±2.0 years old this has shown the tendency of the young to start using MP at earlier age of life. The researcher has assessed MP use in three natures, conversation, social media (such as Facebook, LINE or Skype), and entertainment, and at three levels namely the use at less than 50% meaning seldom use and more than and equal to 50% representing regular use. The research has found that 80.62% of the students used MP for social media and 46.3% of the students used talking mode reported holding MP close to the ear. The use of hand-free or speaker phone is similarly assessed at three levels: not use, rarely use if less than 50% and usually use if more than or equal to 50%. The research has found that most of the students have seldom used of hand-free and speaker phone. Most students have short time durations and low frequency talking on MP. The students, 37.7%, have been also reported getting burning sensation around their ears during conversations on MPs.

Table 3.2 Characteristic of mobile phone use of participants presented as percentage

Characteristic of MP use	**N (%)**
MP owner	994 (99.8)
Smartphone	993 (99.9)
MP brand	
Apple	281 (30.7)
Other	634 (69.3)
Burning sensation	
No	614 (62.3)
Yes	371 (37.7)
Mode of MP used ≥50%	
Conversation	492 (49.5)
Social media (line/Facebook)	801 (80.6)
Entertainment	788 (79.3)
MP holding	
Close to ear	631 (63.5)
Transpose holding	171 (17.2)
Far from ear	191 (19.2)
Hand free using	
≥ 50 % (usually)	251 (25.3)
< 50% (seldom)	448 (45.1)
No used	294 (29.6)
On Speaker phone	
≥ 50 % (usually)	184 (18.5)
< 50% (seldom)	459 (46.2)
No used	350 (35.2)
Duration time used (min/time)	
<10 min	707 (71.1)
≥10 min	287 (28.9)
Frequency used(times/day)	
<5	809 (81.4)
≥5	185 (18.6)

2) The headache symptom

2.1) headache symptom

The headache symptom in one year is 92.6% (Table 3.3). When excluding co-morbidity disease of headache, the migraine and probable migraine are about 1.1% and 11.1% respectively. By type of headache, the highest is TTH and potential TTH with 22.0% and 45.4% respectively whereas the MP associated headache (MPAH) symptom is 77.7%. MPAH is defined as headache attack during or after MP use.

Table 3.3 The headache symptom and detail of headache and the 95% confidence intervals unless specified otherwise

Headache	**Number**	**%**	**95 %CI**	
			Lower	**Upper**
Headache	922	92.6	90.8	94.1
Migraine	10	1.1	0.5	2.0
Probable migraine	102	11.1	9.1	13.3
TTH	203	22.0	19.4	24.8
Probable TTH	419	45.4	42.2	48.7
Undetermined headache	172	18.7	16.2	21.3
Headache with underlying	16	1.7	1.0	2.8
MP associated Headache	716	77.7	74.8	80.3
<10 times/year	288	40.2	36.6	43.9
≥10 times/year	428	59.8	56.1	63.4
Impact activity/learning	114	13.7	11.4	16.2
Scores pain of headache (mean± SD)		3.44±1.885		

2.2) The headache symptom across demographic groups

The migraine and TTH types of primary headache symptom have been found to be highest in the groups of females with underlying diseases, tea drinking habits, and triggers at statistically significant difference level (p<0.05) (Table 3.4). Meanwhile, the MPAH symptom is higher in the groups of young students (16-17) without trigger nor tea and coffee drinking habit, with abnormal vision and abnormal anxiety, at statistically significant difference level (p<0.05).

2.3) The headaches symptom across affected groups

The migraine headache is high in the groups of students having pain scores more than 5 as well as those getting impact on routine activity or learning ability, having bad sleep quality and with regular use of medicines, at statistically significant difference level ($p<0.05$) (Table 3.5). The TTH is high among students who have low score pain, with no impact of headache on daily life and learning, and without regular use of medicines, with statistically significant difference ($p<0.05$). The MPAH symptom similar to TTH is high among students who have low score pain with no impact of headache, with good sleep quality, and without regular use of medicines, at statistically significant difference level ($p<0.05$).

2.4) The headache symptom across groups of MP use characteristics

The research has found that only the TTH which is primary headache is high among students who have seldom used of hand free and with ear burning sensation during conversation by MP, at statistically significant difference level ($p<0.05$) (Table 3.6). The MPAH symptom is high among students usually used MP for talking, with regular use of hand free and speaker phone, and also with burning sensation, which is significantly different ($p<0.05$).

Table 3.4 The headache symptom and their 95% confidence intervals by demographic characteristics

Demographic	Total	Migraine (total=112)		p-value	TTH (total=622)		p-value	Total	MPAH (total=716)		p-value
		Prevalence	95% CI		Prevalence	95% CI			Prevalence	95% CI	
Age				0.68			0.12				<0.01
16-17	403	51 (12.7)	9.6-16.3		261 (64.8)	59.9-69.4		400	353 (88.3)	84.7-91.2	
≥18	519	61 (11.8)	9.1-14.8		361 (69.6)	65.4-73.5		511	363 (71.0)	66.9-74.9	
Gender				0.03			0.05				0.56
Male	222	18 (8.1)	4.9-12.5		138 (62.2)	55.4-68.6		220	176 (80.0)	74.1-85.1	
Female	700	94 (13.4)	11.0-16.2		484 (69.1)	65.6-72.5		691	540 (78.1)	74.9-81.2	
BMI				0.61			0.73				0.10
Normal	818	101 (12.3)	10.2-14.8		554 (67.7)	64.4-70.9		808	628 (77.7)	74.7-80.5	
Overweigh	95	10 (10.5)	5.2-18.5		66 (69.5)	59.2-78.5		94	80 (85.1)	76.3-91.6	
Underlying disease				<0.01			0.04				0.55
No	714	70 (9.8)	7.7-12.2		502 (70.3)	66.8-73.6		706	552 (78.2)	75.0-81.2	
Yes	190	41 (21.6)	16.0-28.1		119 (62.6)	55.3-69.5		187	150 (80.2)	73.8-85.7	
Medicine use				<0.01			0.42				<0.01
No	626	58 (9.3)	7.1-11.8		417 (66.6)	62.8-70.3		619	514 (83.0)	79.8-85.9	
Yes	296	54 (18.2)	14.0-23.1		205 (69.3)	63.7-74.5		292	202 (69.2)	63.5-74.4	
Vision				0.70			0.95				<0.01
Normal	711	88 (12.4)	10.0-15.0		480 (67.5)	63.9-70.9		703	533 (75.8)	72.5-78.9	
Abnormal	211	24 (11.4)	7.4-16.5		142 (67.3)	60.5-73.6		208	183 (88.0)	82.8-92.1	

Table 3.4 (continue)

Demographic	Total N	Migraine (total=112) prevalence	95% CI	p-value	TTH (total=622) prevalence	95% CI	p-value	Total N	MPAH (total=716) prevalence	95% CI	p-value
Trigger				<0.01			<0.01				<0.01
No	94	1 (1.1)	0.03-5.8		18 (19.1)	11.8-26.6		94	92 (97.9)	92.5-99.7	
Yes	828	111 (13.4)	11.1-16.0		604 (72.9)	69.8-75.9		817	624 (76.4)	73.3-79.3	
Tea				<0.01			0.07				<0.01
No	292	21 (7.2)	4.5-10.8		185 (63.4)	57.5-68.9		290	256 (88.3)	84.0-91.7	
Yes	630	91 (14.4)	11.8-17.4		437 (69.4)	65.6-72.9		621	460 (74.1)	70.4-77.5	
Coffee				0.13			0.06				0.01
No	657	73 (11.1)	8.8-13.8		431 (65.6)	61.8-69.2		647	524 (81.0)	77.7-83.9	
Yes	265	39 (14.7)	10.7-19.6		191 (72.1)	66.3-77.4		264	192 (72.7)	66.9-78.0	
Anxiety				0.10			0.92				0.03
Normal	495	52 (10.5)	7.9-13.5		341 (68.9)	64.6-72.9		491	364 (74.1)	70.0-78.0	
Risk-abnormal	337	48 (14.2)	10.7-18.4		231 (68.5)	63.3-73.5		335	270 (80.6)	75.9-84.7	
Depression				0.62			0.26				0.42
Normal	754	92 (12.2)	1.0-14.8		514 (68.2)	64.7-71.5		748	577 (77.1)	74.0-80.1	
Risk-abnormal	78	8 (10.3)	4.5-19.2		58 (74.4)	63.2-83.6		78	57 (73.1)	61.8-82.5	

Table 3.5 The headache symptom and their 95% confidence intervals by pain scores and impact of headache

Pain scores and impact of headache	**Total N**	**Migraine (total=112)**		**p-value**	**TTH (total=622)**		**p-value**	**Total N**	**MPAH (total=716)**		**p-value**
		Prevalence	**95% CI**		**Prevalence**	**95% CI**			**Prevalence**	**95% CI**	
Pain scores				<0.01			<0.01				0.04
≤5	785	8.4	6.6-10.6		69.6	66.2-72.8		776	79.8	76.8-82.5	
>5	137	33.6	25.7-42.1		55.5	46.7-64.0		135	71.9	63.5-79.2	
Impact of headache to activity/learning				<0.01			<0.01				0.05
No	882	11.5	9.4-13.7		68.6	65.4- 71.6		871	78.1	75.2-80.8	
Yes	40	27.5	14.6-43.9		42.5	27.0-59.1		40	90.0	76.3-97.2	
PSQI				0.03			0.27				<0.01
Bad sleep	471	14.6	11.6-18.2		69.9	65.5-74.0		466	73.4	69.1-77.4	
Good sleep	438	9.8	7.2-13.0		66.4	61.8-70.9		432	83.8	80.0-87.1	
Medicine use				<0.01			0.42				<0.01
No	626	9.3	7.1-11.8		66.6	62.8-70.3		619	83.0	79.8-86.0	
Yes	296	18.2	14.0-23.1		69.3	63.7-74.5		292	69.2	63.5-74.4	

Table 3.6 The headache symptom and their 95% confidence intervals by characteristics of MP use

Characteristic of MP use	Total N	Migraine (total=112) Prevalence	95% CI	p-value	TTH (total=622) Prevalence	95% CI	p-value	Total N	MPAH (total=716) Prevalence	95% CI	p-value
Mode of MP use ≥50%				0.76			0.12				<0.01
Talking	460	58 (12.6)	9.7-16.0		299 (65.0)	60.4-69.4		454	385 (61.7)	81.2-88.0	
Non talking	394	47 (11.9)	8.9-15.5		276 (70.1)	65.3-74.5		329	239 (38.3)	67.9-77.0	
MP holding				0.63			0.07				0.86
Near to ear	588	73 (12.4)	9.9-15.4		381 (64.8)	60.8-68.7		580	453 (78.1)	74.5-81.4	
Far from ear	179	18 (10.1)	6.1-15.4		129 (72.1)	64.9- 78.5		179	141 (78.8)	72.0-84.5	
Transpose	152	20 (13.2)	8.2-19.6		110 (72.4)	64.5- 79.3		151	121 (80.1)	72.9-86.2	
Hand free use				0.70			0.02				0.03
No	265	35 (13.2)	9.4-17.9		181 (68.3)	62.3-73.9		263	196 (74.5)	68.8-79.7	
<50	421	51 (12.1)	9.2-15.6		299 (71.0)	66.4-75.3		417	325 (77.9)	73.6-81.8	
≥50	233	25 (10.7)	7.1-15.4		140 (60.1)	53.5-66.4		230	194 (84.3)	19.0-88.8	
Speaker phone use				0.58			0.12				0.02
No	315	40 (12.7)	9.2-16.9		217 (68.9)	63.5-74.0		311	240 (77.2)	72.1-81.7	
<50	430	54 (12.6)	9.6-16.1		297 (69.1)	64.5-73.4		427	326 (76.3)	72.0-80.3	
≥50	174	17 (9.8)	5.8-15.2		106 (60.9)	53.2-68.2		172	149 (86.6)	80.6- 91.3	

Table 3.6 (continue)

Characteristic of MP use	Total N	Migraine (total=112) Prevalence	95% CI	p-value	TTH (total=622) Prevalence	95% CI	p-value	Total N	MPAH (total=716) Prevalence	95% CI	p-value
Duration of MP use/time (min)				0.70			0.41				0.84
≤30	807	97 (12.0)	9.9-14.5		540 (66.9)	63.5-70.2		798	628 (78.7)	75.7-81.5	
≥30	113	15 (13.3)	7.6-20.9		80 (70.8)	61.5-79.0		113	88 (77.9)	69.1-85.1	
Frequency of MP use/day (time)				0.67			0.35				0.37
<10	871	107 (12.3)	10.2-14.7		584 (67.0)	63.8-70.2		862	680 (78.9)	76.0-81.6	
≥10	49	5 (10.2)	3.4-22.2		36 (73.5)	58.9-85.1		49	36 (73.5)	58.9-85.1	
Burning sensation				0.38			0.04				<0.01
No	557	64 (11.5)	9.9-14.4		389 (69.8)	65.8-73.6		195	409 (73.6)	69.7-77.2	
Yes	357	48 (13.4)	10.1-17.4		226 (63.3)	58.1-68.3		355	307 (86.5)	82.5-89.9	

3) The characteristic of mobile phone use affects to headache symptom (MPAH)

The characteristics of headache have been defined based on ICHD-III criteria regarding to frequency and attack duration, pain characteristic, pain form, time of pain attack, side and area of pain (Table 3.7). The research has found that higher MPAH symptom occurred among students with short time pain, pulsing and tightening, unstable increased or decreased of pain, and undetermined form of pain, pain mostly in the morning, one side headache and pain at occipital and frontal areas, $p<0.05$. The higher MPAH symptom also occurs among students with low severity of pain scores had significantly, $p<0.05$.

Table 3.7 The mobile phone associated headache (MPAH) symptom by characteristics of headache

Characteristic of headache	Total N	MPAH <10 times/year	MPAH ≥10 times/year	MPAH Total	p-value
Frequency pain/year					0.26
<5 times/month	796	243 (30.5)	378 (47.5)	621 (78.0)	
≥5 times/month	115	45 (39.1)	50 (43.5)	95 (82.6)	
Duration pain					<0.01
Short time (second)	478	149 (31.2)	252 (52.7)	401 (83.9)	
5min - 4 hrs.	394	126 (32.0)	158 (40.1)	284 (72.1)	
>4 hrs.	39	13 (33.3)	18 (46.2)	31 (79.5)	
Characteristic of pain					0.03
Pulsing	515	151 (29.3)	262 (50.9)	413 (80.2)	
Tightening	166	62 (37.3)	74 (44.6)	136 (81.9)	
Dull and other	230	75 (32.6)	92 (40.0)	167 (72.6)	
Model of pain					<0.01
Short period	207	45 (45.0)	55 (55.0)	100 (48.8)	
Instable pain	377	142 (45.4)	171 (54.6)	313 (84.4)	
Continuous pain	129	59 (49.0)	49 (51.0)	96 (75.6)	
Undetermined pain	209	54 (26.1)	153 (73.9)	207 (99.5)	
Period of time pain					<0.01
Morning after wake up	215	86 (40.0)	98 (45.6)	184 (85.6)	
During day and after school	164	62 (37.8)	72 (43.9)	134 (81.7)	
Undetermined time and other	532	140 (26.3)	258 (48.5)	398 (74.8)	

Table 3.7 (continue)

Characteristic of headache	Total	MPAH <10 times/year	MPAH ≥10 times/year	MPAH Total	p-value
Size of pain					0.03
One size	272	98 (36.0)	129 (47.4)	227 (83.4)	
Two size	261	73 (28.0)	126 (48.3)	199 (76.3)	
Transpose	236	71 (30.1)	103 (43.6)	174 (73.7)	
Other	142	46 (32.4)	70 (49.3)	116 (81.7)	
Area pain					<0.01
Occipital	163	67 (41.1)	75 (46.0)	142 (87.1)	
Parietal	499	152 (30.5)	236 (47.3)	388 (77.8)	
Frontal	137	39 (28.5)	77 (56.2)	116 (84.7)	
Other	112	30 (26.8)	40 (35.7)	70 (62.5)	

4) The factor of MP using and mobile phone associated headache (MPAH)

To control the confounding effects, the research has conducted a statistical test to evaluate the relationship between various factors and found no interaction effect on them. Additional computation has been adjusted the effects of such potential confounders as demographic characteristics, use of medical stimulants, health risk behavior, and characteristics of MP use. The adjusted odds ratio indicates that the students using MP in talking mode regularly are 1.7 times (95% CI: 1.16-2.51) more likely to experience MPAH than those doing otherwise (Table 3.8). Similar to our findings, ear burning sensation has a strong link with MPAH (OR_{adj}2.43; 95% CI: 1.58-3.72) while age is the highest risk factor as MP users at younger age are 4.07 times more likely than those at older age to get MPAH (OR_{adj}4.07; 95% CI: 2.64-6.26) and the vision problem also has significant effect on MPAH (OR_{adj}2.22; 95% CI: 1.33-3.69). Meanwhile on the contrary, the students with regular use of medicines, risk behavior toward health and poor sleep quality are less likely to have MPAH as reflected by the adjusted odds ratios of these factors at 0.58 (95% CI: 0.39-0.86), 0.33 (95% CI: 0.16-0.69) and 0.53 (95% CI: 0.35-0.79) with $p<0.05$, respectively.

Table 3.8 Odds ratio (OR) of MPAH and their 95% confidence intervals for each factor adjusted for all other factors using logistic regression

Factor	Total	MPAH %	Crude OR	Adjusted OR	95%CI		p-value
					Lower	Upper	
Typical used of MP mode							
Talking/ Non-talking	454/391	84.8/ 72.6	2.1	1.71	1.16	2.51	<0.01
Burning sensation							
Yes/ No	355/ 556	86.5/ 73.6	2.3	2.43	1.58	3.72	<0.01
Age							
16-17/ ≥18	511/ 400	88.3/ 71.0	3.1	4.07	2.64	6.26	<0.01
Vision							
Abnormal / Normal	208/ 703	88.0/ 75.8	2.34	2.22	1.33	3.69	<0.01
Drug use							
Yes / No	292/ 619	69.2/ 83.0	0.46	0.58	0.39	0.86	<0.01
Risk behavior							
Yes/ No	755/ 156	75.4/ 94.2	0.19	0.33	0.16	0.69	<0.01
PSQI							
Bad/ Good sleep quality	466/ 432	73.4/ 83.8	0.53	0.53	0.35	0.79	<0.01

Adjusted by age, gender, BMI, underlying disease, drug use, PTIEs, phobia, risk behavior, type of headache, anxiety, depression , MP mode, MP holding, hand free use, speaker phone, duration of MP use, frequency of MP use.

3.1.2 The characteristic of mobile phone using, prevalent of sleep quality among high school students

1) The characteristic of mobile phone using and sleep quality

1.1) Characteristic of sleep quality

The sleep characteristics of teenagers, the result has been found that most students have delayed sleep and waked up which showed sleep after 10 pm and waked up after 5 a.m., sleep loss (Table 3.9). The difficult sleep, inefficient sleep, wakes up during night and morning sleepiness has shown 4.9, 6.1, 18.8 and 22.4 respectively. The PSQI (Pittsburg Sleep Question Index), sleep hygiene and doze mean scores are 4.8±2.9, 34 3±8.1 and 3.1±2.8, in normal range. The prevalence of poor sleep quality is 50.5 and sleep characteristics are significantly difference during grade level, $p<0.05$.

Table 3.9 Characteristic of sleep of participants presented as percent by grade level.

Sleep characteristics	Grade level			Total	95% CI
	Level 10	Level 11	Level 12		Lower-Upper
>10.00 p.m.	158(48.9)*	185(58.4)*	199(58.9)*	542(55.4)	52.2-58.6
>5.00 a.m.	227(70.3)**	221(69.7)**	276(81.7)**	724(74.0)	71.2-76.8
Wake up at night	69(21.0)	65(20.5)	51(15.0)	185(18.8)	16.4-21.4
Latency sleep >30 min	7(2.2)*	23(7.3)*	18(5.3)*	48(4.9)	3.6-6.5
Sleep loss <8	208(64.4)*	233(73.5)*	221(65.4)*	662(67.7)	64.7-70.6
Efficiency sleep <75	27(8.4)*	10(3.2)*	23(6.8)*	60(6.1)	4.7-7.8
Morning sleepiness	57(17.4)**	97(30.6)**	66(19.5)**	220(22.4)	19.8-25.1
Poor Sleep	152(46.5)**	191(60.3)**	153(45.1)**	496(50.5)	47.3-53.6
Impact activity and learning	216 (66.1)**	240(75.7)**	178(52.5)**	634(64.5)	61.4-67.5
Doze, mean: SD	304(3.1±2.7)	295(3.7±3.0)	258(2.5±2.6)	857(3.1±2.8)	
PSQI scores, mean: SD	327(4.8±2.9)**	317(5.5±2.7)**	339(4.3±2.8)**	983(4.8±2.9)	
Sleep hygiene scores, mean: SD	306(34.1±8.6)**	301(35.6±6.3)**	258(32.8±9.0)**	865(34 3±8.1)	
Depression scores, mean: SD	302(3.5±3.0)	301(3.7±: 2.7)	295(3.5±2.6)	898(3.5±2.8)	
Anxiety scores, mean: SD	303(6.6±:4.41)	301(6.8±4.5)	295(7.2 ±4.8)	899(6.8±4.6)	

***p*-value <0.05 ** *p*-value <0.01**

1.2) PSQI score across demographic groups

The higher PSQI scores are found in female those with underlying disease, drug use, history of phobia and potentially traumatic interpersonal events (PTIEs) (Table 3.10). The higher PSQI scores are also found in students having migraine headache and correlate with anxiety and depression scores, therefore, the students with risk behavior, which are significantly difference (p<0.05) (Table 3.11).

Table 3.10 PSQI score of participants compared by demographic data presented as mean and standard deviation unless specified otherwise

Demographic data	Total	PSQI scores		p-value
	N	Mean	SD	
Age				0.40
16-17	423	4.91	2.88	
≥18	560	4.75	2.86	
BMI				0.76
Normal	878	4.83	2.82	
Overweigh	100	4.74	3.22	
Vision				0.16
Normal	766	4.75	2.78	
Abnormal	217	5.06	3.16	
Phobia				<0.01
No	934	4.75	2.84	
Yes	49	6.06	3.03	
Headache type				<0.01
Migraine	112	5.60	2.91	
TTH	620	5.01	2.79	
Unidentified	161	4.14	3.00	
Gender				<0.01
Male	246	4.24	2.83	
Female	737	5.01	2.85	
Underlying disease				<0.01
No	912	4.73	2.81	
Yes	69	5.91	3.43	
Drug use				<0.01
No	676	4.48	2.88	
Yes	307	5.55	2.69	
PIETs				<0.01
No	954	4.74	2.84	
Yes	29	7.24	2.87	
Sleep behavior	864	*r*= .006		0.86
Anxiety	897	*r*= .162		<0.01
Depression	896	*r*= .162		<0.01

Table 3.11 PSQI score of participants compared by risk behavior presented as mean and standard deviation unless specified otherwise

Risk behavior	Total	PSQI scores		p-value
	N	Mean	SD	
Tea drink				<0.01
No	308	4.06	3.00	
Yes	675	5.16	2.74	
Energy drink				<0.01
No	788	4.60	2.79	
Yes	195	5.69	3.02	
Coffee drink				<0.01
No	699	4.53	2.84	
Yes	284	5.53	2.80	
Diet				<0.01
No	429	3.90	2.83	
Yes	554	5.53	2.69	

2) The characteristic of mobile phone using and prevalence of poor sleep quality

The PSQI is composed of quantity and quality sleep domain. The component of the quantity sleep domains, the higher prevalence of difficult to sleep, sleep loss, inefficient sleep has shown among students without hand free use, burning sensation around ear, long time duration time of MP use more than and equal to 30 min and frequency more than and equal to 10 times (Table 3.12). The higher prevalence of sleep problem (waking at night) has been shown among students with MP talking mode use which is significantly difference (p<0.05).

The component of the quality sleep domains, the higher prevalence of feeling morning sleepiness has been found among students with long time duration of MP use and ear burning sensation from MP use (Table 3.13). Therefore, the higher prevalence of poor sleep (PSQI≥5) has found that among students with MP talking mode, long time duration time of MP use, Android system and MP use at night. Finally, the result has shown higher PSQI score among students with drug use, impact activity and learning included correlated with doze score (r=0.39), which is significantly difference (p<0.05)

Table 3.12 Prevalence of sleep quantity domain presented as percent and their 95% confidence intervals by characteristic of MP use

Characteristic of MP use	Total N	Sleep latency ≥30 min (47)		p-value	Sleep duration ≤8 hrs. (661)		p-value	Efficiency sleep <75% (55)		p-value
		Prevalence	95% CI		Prevalence	95% CI		Prevalence	95% CI	
Mode of MP use ≥50%				0.47			0.07			0.10
Talking	478	21 (4.4)	2.7-6.6		317 (66.3)	61.9-70.5		35 (7.3)	5.2-10.0	
Non talking	424	23 (5.4)	3.4-8.0		305 (71.9)	67.4-76.2		20 (4.7)	2.9-7.2	
MP holding				0.24			0.30			0.25
Near to ear	616	34 (5.5)	3.9-7.6		417 (67.7)	63.8-71.4		43 (7.0)	5.1-9.3	
Transpose	169	4 (2.4)	0.6-5.9		108 (63.9)	56.2-71.1		6 (3.6)	1.3-7.6	
Far from ear	190	9 (4.7)	2.1-8.8		136 (71.6)	64.6-77.9		11 (5.8)	2.9-10.1	
Hand-free use				0.05			0.70			0.41
No	290	20 (6.9)	4.3-10.5		194 (66.9)	61.2-72.3		15 (5.2)	2.9-8.4	
Yes	685	27 (3.9)	2.6- 5.7		467 (68.2)	64.5-71.7		45 (6.6)	4.8-8.7	
Speaker phone use				0.82						0.35
No	347	16 (4.6)	2.7-7.4		237 (68.3)	63.1-73.2		18 (5.2)	3.1-8.1	
Yes	628	31 (4.9)	3.4-6.9		424 (67.5)	63.7-71.2		42 (6.7)	4.9-8.9	
Duration of MP use/time (min)				0.12			0.02			0.05
<30	859	38 (4.4)	3.1-6.0		571 (66.5)	63.2-69.6		48 (5.6)	4.1-7.3	
≥30	117	9 (7.7)	3.6-14.1		90 (76.9)	68.2-84.2		12 (10.3)	5.4-17.2	

Table 3.12 (continued)

Characteristic of MP use	Total N	Sleep latency ≥30 min (47)		p-value	Sleep duration ≤8 hrs. (661)		p-value	Efficiency sleep <75% (55)		p-value
		Prevalence	95%CI		Prevalence	95%CI		Prevalence	95%CI	
Frequency of MP use/day (time)				0.23			0.71			0.01
<10	929	43 (4.6)	3.4-6.2		628 (67.6)	64.5-70.6		53 (5.7)	4.3-7.4	
≥10	47	4 (8.5)	2.4-20.4		33 (70.2)	55.1-82.7		7 (14.9)	6.2-28.3	
MP use at night				0.70			0.01			0.01
No	268	15 (5.6)	3.2-9.1		178 (66.4)	60.4-72.0		8 (3.0)	1.3-5.8	
Yes	532	28 (5.3)	3.5-7.5		364 (68.4)	64.3-72.4		41 (7.7)	5.6-10.3	
Device system				0.79			0.12			0.86
IOS	279	13 (4.7)	2.5-7.8		182 (65.2)	59.3-70.8		16 (5.7)	3.3-9.1	
Android and other	630	32 (5.1)	3.5-7.1		444 (70.5)	66.7-74.0		38 (6.0)	4.3-8.2	
Burning sensation				0.09			0.04			0.06
No	608	35 (5.8)	4.0-7.9		397 (65.3)	61.4-69.1		31 (5.1)	3.5-7.2	
Yes	359	12 (3.3)	1.7-5.8		257 (71.6)	66.6-76.2		29 (8.1)	5.5-11.4	

Table 3.13 Prevalence of sleep quality domain and their 95% confidence intervals by characteristic of MP use

Characteristic of MP use	Total N	Sleep problem (618)		p-value	Total N	Morning sleepiness		p-value	Poor sleep		p-value
		Prevalence	95% CI			Prevalence	95% CI		Prevalence	95% CI	
Mode of MP use ≥50%				<0.01				0.13			<0.01
Talking	490	280 (57.1)	52.6-61.6		480	102 (21.3)	17.7-25.2		222 (46.3)	41.7-50.8	
Non talking	428	295 (68.9)	64.3-73.3		427	109 (25.5)	21.5-29.9		246 (57.6)	52.8-62.3	
MP holding				0.03				0.37			0.31
Near to ear	629	374 (59.5)	55.5-63.3		619	130 (21.0)	17.9-24.4		311 (50.2)	46.2-54.3	
Transpose	171	111 (64.9)	57.3-72.0		171	44 (25.7)	19.4- 33.0		80 (46.8)	39.1-54.6	
Far from ear	191	133 (69.6)	62.6-76.0		190	45 (23.7)	17.8-30.4		104 (54.7)	47.4-62.0	
Hand-free use				0.26				0.36			0.26
No	292	190 (65.1)	59.3-70.5		293	60 (20.5)	16.0-25.6		156 (53.2)	47.4-59.1	
Yes	699	428 (61.2)	57.5-64.9		687	159 (23.1)	20.0-26.5		339 (49.3)	45.5-53.2	
Speaker phone use				0.81				0.85			0.87
No	350	220 (62.9)	57.6-67.9		350	77 (22.0)	17.8-26.7		178 (50.9)	45.5-56.2	
Yes	641	398 (62.1)	58.2-65.9		630	142 (22.5)	19.3-26.0		317 (50.3)	46.3-54.3	
Duration of MP use/time (min)				0.14				0.01			<0.01
<30	874	538 (61.6)	58.2-64.8		863	181 (21.0)	18.3-23.8		421 (48.8)	45.4-52.2	
≥30	118	81 (68.6)	59.5-76.9		118	38 (32.2)	23.9-41.4		74 (62.7)	53.3-71.4	

Table 3.13 (continue)

Characteristic of MP use	Total	Sleep problem (618)		p-value	Total	Morning sleepiness		p-value	Poor sleep		p-value
		Prevalence	95%CI			Prevalence	95%CI		Prevalence	95%CI	
Frequency of MP use/day(time)				0.86				0.13			0.41
<10	943	589 (62.5)	59.3-65.6		933	204(21.9)	19.3-24.7		468(50.2)	46.9-53.4	
≥10	49	30(61.2)	46.2-74.8		48	15(31.3)	18.7-46.3		27(56.3)	41.2-70.5	
MP use at night				0.11				<0.01			<0.01
No	270	160(59.3)	53.1-65.2		269	53(19.7)	15.1-25.0		117(43.5)	37.5-49.6	
Yes	533	351(65.9)	61.7-69.9		535	121(22.6)	19.1-26.4		295(55.1)	50.8-59.4	
Some	230	157(68.3)	61.8-74.1		231	35(15.2)	10.8-20.4		117(50.6)	44.0-57.3	
Frequent	303	194(64.0)	58.3-69.4		304	86(28.3)	23.3-33.7		178(58.6)	52.8-64.1	
Device system				0.32				0.19			<0.01
IOS	281	174(61.9)	56.0-67.6		280	57(20.4)	15.8-25.6		117(41.8)	35.9-47.8	
Android and other	632	413(65.3)	61.5-69.1		634	154(24.3)	21.0-27.8		355(56.0)	52.0-60.0	
Burning sensation				0.35				0.03			0.22
No	612	391(63.9)	60.0-67.7		613	124(20.2)	17.1-23.6		301(49.1)	45.1-53.1	
Yes	371	226(60.9)	55.7-65.9		359	94(26.2)	21.7-31.1		191(53.2)	47.9-58.5	

3) The PSQI and impact of sleep

The result has shown higher PSQI score among students with medicine use, impact activity and learning included correlated with doze score (r=0.39), which is significantly difference (p<0.05) (Table 3.14).

Table 3.14 PSQI scores compared by impact of sleep

Impact of Sleep	**Total N**	PSQI scores mean ±SD	**p-value**
Impact activity			0.01
No	177	5.3±2.0	
Yes	268	7.3±2.5	
Medicine use			0.01
No	947	4.7±2.8	
Yes	36	8.5±3.1	
Impact learning			0.01
No	180	4.7±2.0	
Yes	401	6.7±2.5	
Doze scores	856	r =0.386**	0.01

** ***p*-value <0.01**

4) The factor of mobile phone using and poor sleep quality

To control the confounding effects, the research has conducted a statistical test to evaluate the relationship between various factors and found no interaction effect on them. Additional computation has been adjusted the effects of such potential confounders as demographic characteristics, use of medical, health risk behavior, and characteristics of MP use. The factors of MP used have associated to PSQI in quantity domain, sleep loss is frequently MP use at night OR_{adj}1.88: 95% CI; 1.21-2.93 and inefficient sleep is long duration time of MP talking OR_{adj}2.76: 95% CI; 1.39-5.48 (table 3.15). The factors of MP have associated to PSQI in quality domain, morning sleepiness is frequently MP use at night OR_{adj}1.86: 95% CI; 1.16-2.99. While the factor of long duration time of MP talking and device system associated to poor sleep (PSQI scores ≥5) OR_{adj}1.60: 95%CI; 1.09-2.34 and OR_{adj}1.57: 95% CI; 1.08-2.27.

Table 3.15 Odds ratio (OR) of sleep quality component and their 95% confidence intervals for each factor adjusted for all other factors using logistic regression

Factor of MP use	Total N	PSQI %	Crude OR	Adjusted OR	95%CI		p-value
					Lower	Upper	
PSQI: Quantity domain							
Aspect sleep latency ≥30 min							
Hand free use: No/ Yes	290/ 685	6.9/3.9	1.81	2.40	1.190	4.820	0.01
Aspect duration of sleep <8 hr.							
MP use at night: frequent/some	302/ 230	73.5/61.7	1.72	1.88	1.21	2.93	<0.01
Aspect efficiency sleep < 75%							
Duration talking : >10/ <10	283/ 693	9.2/ 4.9	1.961	2.76	1.390	5.478	<0.01
PSQI : Quality domain							
Aspect sleepy after wake up							
MP use at night: frequent/no	304/ 269	28.3/19.7	1.608	1.86	1.160	2.992	0.01
PSQI: Poor sleep							
Device system: other/Apple	634/ 280	56.0/41.8	1.77	1.57	1.08	2.27	0.02
Duration of MP talking: >10/ <10	286/ 695	62.2/45.6	1.965	1.60	1.09	2.34	0.02

Adjusted by age, gender, BMI, underlying disease, drug use, PTIEs, phobia, risk behavior, type of headache, anxiety, depression , MP mode, MP holding, hand free use, speaker phone, duration of MP use, frequency of MP use, burning sensation.

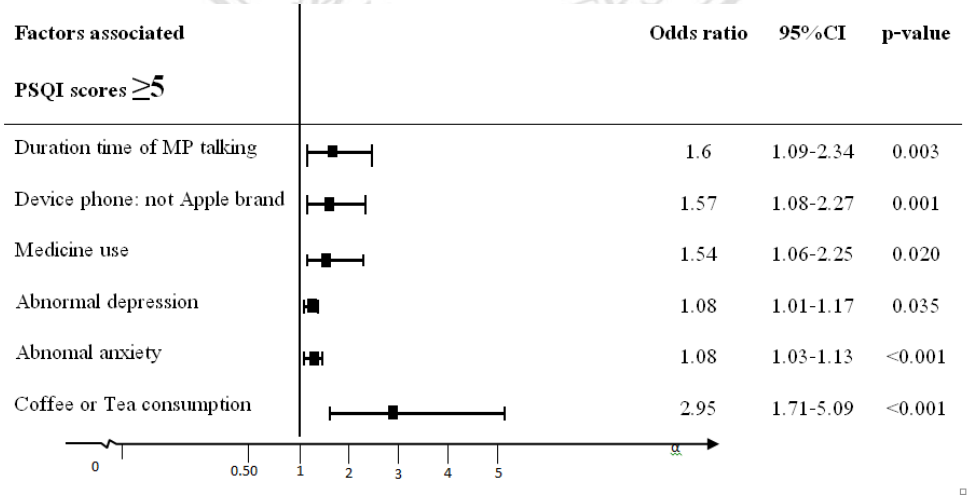


Figure 3.2 Factors contributing to poor sleep presented as odds ratio adjusted for age, gender, BMI, underlying disease, PTIEs, phobia, risk behavior, type of headache, MP mode, MP holding, hand free use, speaker phone, frequency of MP use, burning sensation, using multiple logistic regression

The factors of medicine use, anxiety and depression have associated with poor sleep OR_{adj}1.54: 95% CI; 1.06-2.25, OR_{adj}1.08: 95% CI; 1.01-1.17 and OR_{adj}1.08:95% CI; 1.03-1.13 respectively (Figure 3.1) In Addition, The result has shown factor of risk behavior has the strongest associated with poor sleep OR_{adj}2.95: 95% CI, 1.71-5.09.

Table 3.16 Odds ratio (OR) of doze and their 95% confidence intervals for each factor adjusted for all other factors using logistic regression

Factor	Total (N)	Doze %	Crude OR	Adjusted OR	95%CI		p-value
					Lower	Upper	
PSQI: Good sleep	420	1.20	1				
PSQI: Bad sleep	436	8.30	7.47	9.03	2.723	29.935	<0.01

Adjusted by age, gender, BMI, underlying disease, drug us, risk behavior, anxiety, depression, MP mode, MP holding, hand free use, speaker phone, duration of MP use, frequency of MP use.

Finally, the result has shown poor sleep associated with doze OR_{adj}9.03: 95% CI, 2.72-29.94, p<0.01 (Table 3.16).

3.2 Electromagnetic radiation from smartphone and health problems

Objective 2: To study the correlation between smartphone output power and headache among high school students

3.2.1 The correlation between smartphone output power and headache among high school students

Out of the total 996 high school students who are the population for questionnaire interviews in the first phase of the study, 200 students have been selected by inclusion and exclusion criteria as studied subjects.

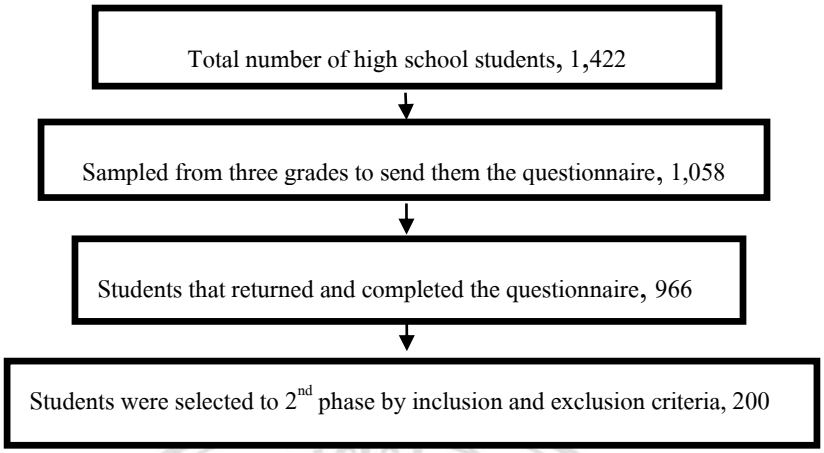


Figure 3.3 The sampling procedure and sample response flow chart

The 200 samples have been asked to record headache and sleep quality by a smartphone application to measure smartphone electromagnetic radiation (SER) in terms of smartphone output power (SOP) by another application daily sent via email daily. As the information has been sent daily for 60 days, there will be totally 12,000 observations. However, during the information collection process in the first week, 10 samples have withdrew from the study because they have loaded with homework and class reports, so they cannot afford the time for recording and sending the information; 15 samples do not have the devices as their parents have taken away their smartphones; 17 samples have failed to send information about SOP; and 13 samples are removed by the researcher from participating in the study because they failed to keep record at the determined time and the researcher cannot get a hold of them. To fill in the missing information, the researcher has extended the time for information collected from 60 days to 120 days. Finally 12,696 observations are obtained from totally 145 students and specifically from 84 samples have kept record and sent information for 90-120 days, 54 samples who have kept for 60-90 days, 7 samples have kept the records less than 60 days, and one who has recorded only 35 days. The data and information have been provided by the 145 samples for descriptive statistical analysis of demographic data as presented below.

1) Demographic characteristics of participants

The study has found that the majority of the samples are female, 17.4 years old on the average age (Table 3.17) with normal body mass index and normal eye sight as normal health condition is also a criterion for participants' selection. All of them are reported in

the percentage of smartphone uses and 13.4% indicate to have headache (Table 3.18) with average duration headache of 3.2± 3.7 min, average frequency headache of 1.7± 1.6 time/day, and average pain score of 3.2± 1.9. In most cases, the headache is of tension type or TTH, 74.1%, followed by migraine and unidentified types at 9.0% and 16.9% respectively. The headache has taken place in the morning, at noon, and in the evening for 32.1%, 30.2%, and 32.4% of the participants respectively, while only 5.3% have got headache at night time.

Table 3.17 Demographic characteristics of participants presented as percentage unless specified otherwise.

Demographic				**N (%)**
Gender				
Female				129 (89.0)
Male				16 (11.0)
BMI				
Abnormal				13 (9.0)
Normal				132 (91.0)
Vision problem				
Abnormal				26 (17.9)
Normal				119 (82.1)
Age	Mean ±SD	17.36±0.95	Max: 19.00	Min: 16.00

Table 3.18 Characteristics of headache of participants presented as percent.

Variable	N (%)
Headache	
Yes	1705 (13.4)
No	10991 (86.6)
Headache type	
Migraine	153 (9.0)
TTH	1264 (74.1)
Undetermined	288 (16.9)
Morning headache	
Yes	547 (32.1)
No	1158 (67.9)
Evening headache	
Yes	553 (32.4)
No	1152 (67.6)
Nocturnal headache	
Yes	90 (5.3)
No	1615 (94.7)

2) Smartphone output power (SOP)

Table 3.19 Smartphone output power (SOP) by cycle time and daily dose.

Variable	N	Minimum	Maximum	Mean	Standard deviation
Sum Nocturnal dose	12696	0	1.54703000	0.0010027961	0.01452703607
Sum Morning dose	12696	0	0.60338196	0.0011027388	0.00644747700
Sum Daytime dose	12696	0	0.36942911	0.0011809051	0.00618567980
Sum evening dose	12696	0	0.4080357	0.001072926	0.0065726714
Daily dose	12696	0.00000009	1.54872780	0.0020833594	0.01623557023

The data on smartphone output power has been adjusted considering the value of error measured from each device brand to normalize the value for all device brands. Unit

transformation has been made from dBm into mW for aggregating SOP value of each of the four time periods (6 hours), morning time 6:01-12:00 a.m., daytime 12:01-18:00 p.m., evening time 18:01-24:00 p.m. and nocturnal time 0:00-6:00 a.m. The SOP values of each day are aggregated into daily dose which is by average $2.08 \pm 16.2 \times 10^{-3}$mW (Table 3.19). Apparently, average smartphone output power is the highest during daytime 1.18×10^{-3}mW followed by morning time 1.1×10^{-3}mW while the lowest during nocturnal time1.0×10^{-3}mW. However, the maximum value of SOP occurs during nocturnaltime1.55mW. Furthermore, the obtained data has been managed for analysis in dose-response framework to understand the relationship between SOP and headache. Consequently the data has been arranged into appropriate groups in the same manner as those used for probit analysis (probit regression).[235-236] The observations on smartphone output power are set into 100 groups, and in each group they are arranged in the lowest value to the highest value order and each of them will be accompanied with the information on the number of samples exposed to it and the number of samples with headache. Any sample with exposure of SOP and headache in a group will not be recounted in the next group. The percent is then calculated for samples with exposure of SOP and headache in each group. All the results are presented in Figure 3.4. Figure 3.5 shows the correlation of SOP and proportional headache symptom in a reverse dose-response. The correlation of the proportional headache symptom and SOP in 15^{th}-20^{th} group has been decreasing. These data are useful for dividing groups of SOP for correlation analysis by GEE method.

Snapshots

	group	min_dailyd~e	max_dailyd~e	num_expose	num_headache	percent
1	1	9.45e-08	1.68e-06	7	1	14.29
2	2	1.68e-06	2.92e-06	14	4	28.57
3	3	2.94e-06	4.13e-06	12	3	25.00
4	4	4.14e-06	4.76e-06	8	1	12.50
5	5	4.77e-06	5.06e-06	8	1	12.50
6	6	5.06e-06	7.63e-06	21	5	23.81
7	7	7.65e-06	8.60e-06	10	1	10.00
8	8	8.61e-06	8.96e-06	10	0	0.00
9	9	8.96e-06	.0000108	21	5	23.81
10	10	.0000108	.000011	11	2	18.18
11	11	.000011	.0000116	11	0	0.00
12	12	.0000116	.000013	19	1	5.26
13	13	.000013	.0000139	13	0	0.00
14	14	.0000139	.0000152	20	4	20.00
15	15	.0000152	.0000169	22	2	9.09
16	16	.0000169	.0000196	23	1	4.35
17	17	.0000196	.0000208	16	1	6.25
18	18	.0000208	.0000212	9	1	11.11
19	19	.0000212	.0000237	24	2	8.33
20	20	.0000237	.0000249	20	3	15.00
21	21	.0000249	.0000286	28	0	0.00
22	22	.0000287	.0000332	25	3	12.00
23	23	.0000333	.0000358	22	0	0.00
24	24	.0000358	.0000387	27	4	14.81
25	25	.0000387	.000041	22	2	9.09
26	26	.000041	.000042	19	0	0.00
27	27	.0000421	.0000447	23	2	8.70
28	28	.0000447	.0000481	19	0	0.00
29	29	.0000481	.0000505	25	3	12.00
30	30	.0000505	.0000511	9	1	11.11
31	31	.0000511	.0000577	33	2	6.06
32	32	.0000578	.0000616	25	2	8.00
33	33	.0000616	.0000623	12	2	16.67
34	34	.0000624	.0000666	25	2	8.00

Figure 3.4 Smartphone output power100 groups

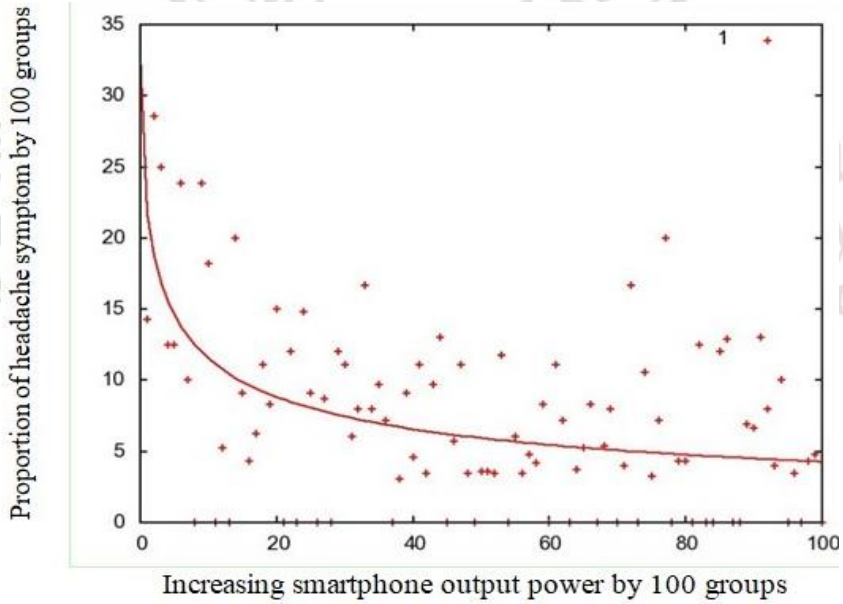


Figure 3.5 Proportion of headache by 100 groups of smartphone output power

The SOP observations are then divided into three ranged groups: equal and less than 1.79, 1.8-1.99, and equal and more than $2.0x10^{-5}$mW (Table 3.20). The most common range of SOP to which the samples exposed was found at $\geq 2.0x10^{-5}$mW, 80.6% of the observations, during all periods of the day but mostly during the evening. SOP in range of $1.8\text{-}1.99x10^{-5}$mW has appeared the least prevalent, only 2.4% of the observations, taking place mostly during nocturnal time. SOP in the range of $\leq 1.79x10^{-5}$mW, 31.4% of the observations, correspond to smartphone use in the morning. Previous studies indicated most teenagers, 62-72%, used advanced smartphone in the evening and at night, after 9:00 p.m. and during 00:00-3:00 a.m., and 34-55% of the use were for texting, social media[64, 82, 237-238], and 24% for playing games.[82]

Table 3.20 Smartphone output power group by cycle time and daily dose

Output power (x 10^{-5}mW)	daily dose N (%)	Morning N (%)	Daytime N (%)	Evening N (%)	Nocturnal N (%)
≤1.79	1943(15.3)	3597(31.4)	2479(20.1)	2303(18.8)	2648(20.9
1.8-1.99	186(1.5)	226(2.0)	120(1.0)	79(0.6)	301(2.4)
≥2.0	10567(83.2)	7646(66.7)	9710(78.9)	9896(80.6)	9747(76.8)

The study on the relationship between SOP and headache has involved the details concerning headache event, duration and pain score, frequency of headache in a day, type of headache, and time period of the day getting the headache.

2) Relationship between SOP and headache

In this section of study, headache has been investigated in terms of headache symptom, duration and pain score, and one-day frequency of headache. The results of the factors of age, anxiety, PSQI, internet use, and hand-free use have relationship with headache event (Table 3.22), long duration of headache (>4 hr.) (Table 3.23), frequency of headache (Table 3.24), and pain score (Table 3.25). Hand-free use and Internet use have the strongest association with headache (OR_{adj}3.22; 95% CI: 2.25-4.62 and OR_{adj}2.45; 95% CI: 1.94-3.10), long duration of headache (OR_{adj}3.03; 95% CI: 1.74- 5.27 and OR_{adj}2.06; 95% CI: 1.75-3.32), severe headache (OR_{adj}3.20; 95% CI: 2.21-4.63 and OR_{adj}2.29; 95% CI: 1.87-2.80) and frequency of headache (OR_{adj}3.17; 95% CI: 2.23-4.49 and OR_{adj}1.98; 95% CI: 1.57-2.49).The study has also found that smartphone output

power in the range of 1.80-1.99x10^{-5}mW can cause 1.84, 1.95, and 1.55 times greater likelihood risky for headache event, severe headache and frequent headache to occur, respectively. Meanwhile SOP in the range of ≤1.79x10^{-5}mW can pose 1.54 time greater risk for long duration of headache (95% CI: 1.08-2.19) to occur compare to the range of ≥2.00x 10^{-5}mW

The study on the impact of MFR on headache has assumed the exist lag (or delayed) effects of SOP on headache symptom. The temporal dimension has added in the study addressed two types of delayed effects taking place in different time intervals namely: 1) 6-hour lag after exposure implying there are three lag in a day, and 2) one-day or 24-hour lag after exposure meaning that there are six lag in a week. Lag_5 of daily smartphone output power have affected to the frequency of headache and pain score, OR_{adj}6.89 % CI: 1.64-28.98 and OR_{adj}7.59 % CI: 2.02-28.44, as dose-response relationship (Table 3.21).

Table 3.21 Odds ratio (OR) of headache symptom and their 95% confidence intervals for each factor and lag dose adjusted for all other factors using GEE

Parameter	Exp.(B)	95% CI for Exp.(B)		p-value	Correlation structure	QIC	QICC
		Lower	Upper				
Frequent pain							
Lag_5	7.576	2.018	28.444	0.003	Exchangeable	10721.426	10654.986
Lag_5	6.761	1.451	31.497	0.015	AR1	10730.914	10652.525
Pain score							
Lag_5	6.894	1.640	28.980	0.008	AR1	19480.688	19362.260

Adjusted by Age, BMI, Vision, Anxiety, Depression, PSQI, Internet use, Hand free use, and Brand device

Table 3.22 Odds ratio (OR) of headache symptom and their 95% confidence intervals for each factor and daily dose adjusted for all other factors using GEE (AR1,QIC=8397.22, QICC=8366.53)

Factor	Headache symptom		Crude	Adjusted	95% CI		p-
	Yes	No	OR	OR	Lower	Upper	value
Age, mean ±SD	17.1±0.9	17.4±1.0	0.75	1.33	1.19	1.49	<0.01
Anxiety score, mean ±SD	2.4±2.7	1.7±2.4	1.11	1.08	1.04	1.13	<0.01
PSQI score, mean ±SD	4.0±2.2	3.6±2.0	1.08	1.05	1.02	1.09	<0.01
Total	1705	10990					
Internet use: Yes/ No	1416: 11280	22.2: 12.3	1.98	2.45	1.94	3.10	<0.01
Hand-free use							
No/ Frequent	10477: 951	14.1: 7.8	2.38	3.22	2.25	4.62	<0.01
Sometime/ Frequent	1268: 951	12.0: 7.8	1.76	1.92	1.24	2.97	<0.01
Dose group ($x10^{-5}$mW)							
1.80-1.99/ ≥200	186: 10567	18.3: 13.1	2.01	1.84	1.20	2.81	<0.01

Adjusted by Age, BMI, Vision, Anxiety, Depression, PSQI, Internet use, Hand free use, Brand device, and SOP.

Table 3.23 Odds ratio (OR) of duration time of headache and their 95% confidence intervals for each factor and daily dose adjusted for all other factors using GEE (AR1, QIC=3473.75, QICC=3458.362)

Factor	Duration pain		Crude	Adjusted	95% CI		p-
	>4 hr.	<4 hr.	OR	OR	value	Upper	value
Age, mean ±SD	17.1±0.9	17.4±1.0	1.45	1.48	1.26	1.74	<0.01
Anxiety score, mean ±SD	2.6±2.8	1.8±2.4	1.10	1.10	1.05	1.15	<0.01
Total	460	12236					

Factor	Total	Duration pain >4 hr. %	Crude OR	Adjusted OR	95% CI		p- value
					Lower	Upper	
Internet use : Yes/ No	1416: 11280	6.5:3.3	2.06	2.41	1.75	3.32	<0.01
Hand-free use							
No: Frequent	10477: 951	3.9: 1.7	2.33	3.03	1.74	5.27	<0.01
Dose group ($x10^{-5}$mW)							
≤1.79/ ≥2.00	1943:10567	4.9: 3.4	1.60	1.54	1.08	2.19	0.02

Adjusted by Age, BMI, Vision, Anxiety, Depression, PSQI, Internet use, Hand free use, Brand device, and SOP.

Table 3.24 Odds ratio (OR) of frequent headache and their 95% confidence intervals for each factor and daily dose adjusted for all other factors using GEE (AR1, QIC=10672.19 QICC=10572.75)

Factor	Total	Correlation frequent pain (*r*)	Crude OR	Adjusted OR	95% CI		p-value
					Lower	Upper	
Age	12691	0.085**	0.702	0.69	0.621	0.774	<0.01
Anxiety score	12691	0.085**	1.12	1.11	1.08	1.14	<0.01
PSQI score	12691	0.042**	1.06	1.04	1.00	1.07	0.03
Lag_5 dose (mW)	12691	0.002	2.81	7.58	2.02	28.44	<0.01
Internet use: Yes/ No	1416/11280	0.4±0.9/ 0.2±0.8	1.76	1.98	1.57	2.49	<0.01
Hand-free use							
No/ Frequent	10477/ 951	0.2±0.8/ 0.1±0.5	2.64	3.17	2.23	4.49	<0.01
Sometime/ Frequent	1268/ 951	0.2±0.9/ 0.1±0.5	2.10	2.14	1.31	3.48	<0.01
Dose group($x10^{-5}$mW)							
1.80-1.99/ ≥2.00	186/ 10567	0.3±0.7/ 0.2±0.8	1.92	1.55	1.13	2.15	<0.01

** *p*-value=0.01

Adjusted by Age, BMI, Vision, Anxiety, Depression, PSQI, Internet use, Hand free use, Brand device, and SOP.

Table 3.25 Odds ratio (OR) of pain scores and their 95% confidence intervals for each factor and daily dose adjusted for all other factors using GEE (AR1, QIC=19288.47 QICC=19144.93).

Factor	Total	Correlation score pain (*r*)	Crude OR	Adjusted OR	95% CI		p-value
					Lower	Upper	
Age	12696	0.078**	0.78	0.78	0.71	0.86	<0.01
Anxiety score	12696	0.106**	1.12	1.08	1.05	1.12	<0.01
Depression score	12696	0.083**	1.10	1.04	1.01	1.07	0.03
PSQI score	12696	0.065**	1.08	1.06	1.03	1.09	<0.01
Lag_5 dose (mW)	12696	0.004	1.76	6.89	1.64	28.98	<0.01
Internet use: Yes / No	1416/ 11280	0.77±1.8/ 0.38±1.2	2.0	2.29	1.87	2.80	<0.01
Hand-free use							
No/ Frequent	10477/ 951	0.44±1.3/ 0.23±0.9	2.55	3.20	2.21	4.63	<0.01
Sometime/ Frequent	1268/ 951	0.40±1.3/ 0.23±0.9	2.02	2.11	1.40	3.17	<0.01
Dose group($x10^{-5}$mW)							
1.80-1.99/ ≥2.00	186/ 10567	0.63±1.6/ 0.41±1.3	2.26	1.95	1.42	2.69	<0.01

**** p-Value=0.01**

Adjusted by Age, BMI, Vision, Anxiety, Depression, PSQI, Internet use, Hand free use, Brand device, and SOP.

3) Relationship between smartphone output power and headache type

The study has found that the factors of age, anxiety, depression, PSQI, internet use, and hand-free use have correlated with tension type headache (TTH) (Table 3.27) while age, anxiety, internet use, and hand-free use have correlated with migraine (Table 3.28) and undetermined headache (Table 3.29). Apparently, hand-free use and internet use have the strongest association with TTH (OR_{adj}3.72; 95% CI: 2.49-5.56 and OR_{adj}2.15; 95% CI: 1.75-2.64), migraine (OR_{adj}3.96; 95% CI: 1.21-12.87and OR_{adj}2.06; 95% CI: 1.20-3.51) and undetermined headache (OR_{adj}1.92; 95% CI: 1.11-3.29 and OR_{adj}2.33; 95% CI: 1.71-3.19). The study has found that SOP in 1.80-1.99x10^{-5}mW range is the risk factor for undetermined headache, and those in the range of ≤1.79 and 1.80-1.99x10^{-5}mW are riskier than that the range of ≥2.00x10^{-5}mW for migraine to occur. However, TTH does not occur as a response to SOP at any ranges of exposure. Meanwhile, Lag_6 of daily SOP exposure has produced migraine effect in reverse dose-response manner (Table 3.26).

Table 3.26 Odds ratio (OR) of headache type and their 95% confidence intervals for each factor and lag dose adjusted for all other factors using GEE

Parameter	Exp.(B)	95% CI for Exp.(B)		p-value	Correlation structure	QIC	QICC
		Lower	Upper				
Migraine type							
Lag_6	1.106E-38	4.548E-69	2.690E-08	0.01	AR1	1346.608	1341.171
Lag_6	3.988E-46	8.918E-83	1.784E-09	0.02	Exchangeable	1347.436	1341.779

Adjusted by Age, BMI, Vision, Anxiety, Depression, PSQI, Internet use, Hand free use, Brand device.

Table 3.27 Odds ratio (OR) TTH type and their 95% confidence intervals for each factors and daily dose adjusted for all other factors using GEE (AR1, QIC=6959.62, QICC=6935.18).

Factor	TTH		Crude	Adjusted	95% CI		p-
	Yes	No	OR	OR	Lower	Upper	value
Age mean ±SD	17.2±0.9	17.4±1.0	1.26	1.26	1.12	1.42	<0.01
Anxiety score mean ±SD	2.3±2.6	1.8±2.4	1.11	1.08	1.04	1.12	<0.01
Depression score mean ±SD	1.8±2.5	1.4±2.2	1.10	1.05	1.02	1.08	<0.01
PSQI score mean ±SD	4.0±2.1	3.6±2.1	1.06	1.04	1.01	1.08	0.02
Total	1264	11432					
Internet use: Yes/ No	1416/ 11280	16.0/ 9.2	1.73	2.15	1.75	2.64	<0.01
Hand-free use							
No/ Frequent	10477/ 951	10.5/ 5.2	2.90	3.72	2.49	5.56	<0.01
Sometime/ Frequent	1268/ 951	8.8/ 5.2	2.25	2.43	1.55	3.80	<0.01

Adjusted by Age, BMI, Vision, Anxiety, Depression, PSQI, Internet use, Hand free use, Brand device, and SOP.

Table 3.28 Odds ratio (OR) of migraine type and their 95% confidence intervals for each factors and daily dose adjusted for all other factors using GEE (AR1, QIC=1314.86, QICC=1309.60)

Factor	Migraine headache		Crude	Adjusted	95% CI		p-
	Yes	No	OR	OR	Lower	Upper	value
Age, mean ±SD	16.9±0.9	17.4±1.0	1.80	1.79	1.38	2.31	<0.01
Anxiety score, mean ±SD	3.0±3.0	1.8±2.4	1.12	1.12	1.06	1.19	<0.01
Lag_6 dose (x 10^{-3}mW), mean ±SD	0.72±1.4	2.1±16.3	$6.3x10^{-44}$	$1.11x10^{-38}$	$4.55\ x10^{-69}$	$2.69x10^{-8}$	0.01
Total	153	12537					
Internet use: Yes/ No	1416/11280	2.0/ 1.1	1.89	2.06	1.20	3.51	<0.01
Hand-free use:							
No/ Frequent	10477/ 951	1.3/ 0.5	3.26	3.96	1.21	12.87	0.02
Dose group (x10^{-5}mW)							
≤1.79/ ≥2.00	1943/10567	2.1/ 1.0	2.00	2.02	1.17	3.49	0.01
1.80-1.99/ ≥2.00	186/ 10567	2.7/ 1.0	4.08	3.25	1.65	6.42	<0.01

Adjusted by Age, BMI, Vision, Anxiety, Depression, PSQI, Internet use, Hand free use, Brand device, and SOP.

Table 3.29 Odds ratio (OR) of undetermined headache and their 95% confidence intervals for each factor and daily dose adjusted for all other factors using GEE (AR1, QIC=3303.33, QICC=3289.53)

Factor	Undetermined headache		Crude	Adjust	95% CI		p-
	Yes	No	OR	OR	Lower	Upper	value
Age, mean ±SD	17±0.9	17.4±1.0	1.49	1.49	1.26	1.76	<0.01
Anxiety score, mean ±SD	2.8±2.9	1.8±2.4	1.12	1.12	1.07	1.17	<0.01
Total	441	12255					
Internet use: Yes/ No	1416/ 11280	6.2/ 3.1	2.07	2.33	1.71	3.19	<0.01
Hand-free use:							
No/ Frequent	10477/ 951	3.6/ 2.6	1.50	1.92	1.11	3.29	0.02
Dose group ($x10^{-5}$mW)							
1.80-1.99/ ≥2.00	186/ 10567	5.4/ 3.3	2.82	2.32	1.23	4.34	<0.01

Adjusted by Age, BMI, Vision, Anxiety, Depression, PSQI, Internet use, Hand free use, Brand device, and SOP.

4) Relationship between smartphone output power and time cycle headache

Age, anxiety, depression, PSQI, internet use, and brand device have been associated with nocturnal headache (Table 3.31), morning headache (Table 3.32), daytime headache (Table 3.33), and evening headache (Table 3.34). Internet use and brand device strongly related to nocturnal headache (OR_{adj}2.14; 95% CI: 1.07-4.25 and OR_{adj}2.33; 95% CI: 1.08-5.05),Not using hand-free and internet use will have strong association with morning headache (OR_{adj}2.62; 95% CI: 1.59-4.32and OR_{adj}1.91; 95% CI: 1.44-2.54), daytime headache (OR_{adj}3.01; 95% CI: 1.67-5.42 and OR_{adj}1.88; 95% CI: 1.39-2.55) and evening headache (OR_{adj}3.02; 95% CI: 1.67-5.49 and OR_{adj}2.62; 95% CI: 1.93-3.56).

Smartphone output power in the range of ≤$1.79x10^{-5}$mW is related to daytime and evening headache (OR_{adj}1.52; 95% CI: 1.10-2.11 and OR_{adj}2.60; 95% CI: 1.36-4.97. The relationship between morning headache and SOP (OR_{adj}194.11; 95% CI: 1.22-30821.27) will appear in the form of dose-response. Furthermore, the study has found daytime Lag_2 of SOP in the range of 1.80-1.99x0^{-5}mW to have strong association with nocturnal headache (OR_{adj}5.18; 95% CI: 3.44-7.81) compared the range of ≥$2.00x10^{-5}$mW. Lag_6 daily SOP has the relationship with nocturnal headache in the form of reverse dose-response.

On delay effect of time cycle smartphone output power, the study has found that nocturnal Lag_1of SOP in the range of ≥$2.00x10^{-5}$mW will have 2.35 times higher risk

for morning headache (95% CI:1.12-4.94) (Table 3.30);morning Lag_1of SOP in the range of 1.80-1.99x10^{-5}mW will have 1.79 times greater risk for daytime headache (95% CI: 1.03-3.12); while daytime Lag_1of SOP in the range of ≤1.79x10^{-5}m W and the range of ≥2.00x10^{-5}mW ranges have posed 1.76 and 1.70 times respectively greater risk for evening headache (95% CI: 1.36-4.97 and 95% CI: 1.38-4.86). The delay effect Lag_5 of daily SOP has 9.96 times greater risk for daytime headache to occur (95% CI: 1.28-77.43) and Lag_4 has 4.53 times greater risk for evening headache to take place (95% CI: 1.06-19.38) in the form of dose response relation.

Table 3.30 Odds ratio (OR) of time cycle headache and their 95% confidence intervals for each factor and lag dose adjusted for all other factors using GEE

Parameter	Exp.(B)	95% CI		p-value	correlation structure	QIC	QICC
		Lower	Upper				
Nocturnal headache							
Lag_1	7.415E-51	1.122E-100	0.490	0.05	Exchangeable	921.611	913.392
Lag_4	7.608E-38	9.118E-73	0.006	0.04	Exchangeable	921.819	914.458
Lag_6	7.335E-40	2.899E-82	1855.887	0.07			
Lag_6	1.032E-39	9.147E-74	1.165E-05	0.03	Exchangeable	921.492	914.215
Lag_2(12-18 p.m.)	5.184	3.441	7.809	<0.01	AR1	899.919	895.279
Morning headache							
Lag_1(24-6 am)	2.354	1.121	4.944	0.024	AR1	3860.341	3846.285
Daytime headache							
Lag_5	4.983	0.286	86.730	0.271			
Lag_5	9.960	1.281	77.432	0.028	Exchangeable	3603.135	3593.614
Lag_6	3.246E-13	7.472E-24	0.014	0.021	AR1	3618.691	3611.250
Lag_1 (6-12 p.m.)	1.792	1.029	3.123	0.039	AR1	3594.202	3581.771
Evening headache							
Lag_4	4.527	1.058	19.375	0.042	Exchangeable	3987.845	3976.577
Lag_1	2.218	1.037	4.745	0.040			
(12-18 p.m.)	2.086	1.051	4.142	0.036	AR1	3971.210	3953.953

Adjusted by Age, BMI, Vision, Anxiety, Depression, PSQI, Internet use, Hand free use, Brand device, and SOP.

Table 3.31 Odds ratio (OR) of nocturnal headache (24:00-6:00 a.m.) and their 95% confidence intervals for each factor and daily dose adjusted for all other factors using GEE (AR1, QIC=899.92, QICC=895.28)

Factor	Nocturnal headache		Crude OR	Adjusted OR	95% CI		p-value
	Yes	No			Lower	Upper	
Age, mean ±SD	16.9±0.8	17.4±1.0	1.76	1.68	1.1	2.4	<0.01
Lag_6 dose(x 10^{-3}mW), mean ±SD	0.8±1.7	2.1±16.0	$1.84x10^{-35}$	$1.03x10^{-39}$	$9.15x10^{-74}$	$1.17x10^{-5}$	0.03
	90	12600					
Internet use: Yes/ No	1416/11280	1.5/0.6	2.13	2.14	1.07	4.25	0.03
Brand device							
Other / Apple	9170/ 3526	0.8 / 0.4	2.40	2.33	1.08	5.05	0.03
Daytime dose group (x10^{-5}mW)							
1.80-1.99 /≥2.00	79/ 9896	5.1/ 0.7	6.97	5.18	3.44	7.81	<0.01

Adjusted by Age, BMI, Vision, Anxiety, Depression, PSQI, Internet use, Hand free use, Brand device, and SOP.

Table 3.32 Odds ratio (OR) of morning headache (6:00-12:00 a.m.) and their 95% confidence intervals for each factor and daily dose adjusted for all other factors using GEE (AR1, QIC=3863.46, QICC=3847.87)

Factor	Morning headache		Crude OR	Adjusted OR	95% CI		p-value
	Yes	No			Lower	Upper	
Age, mean ±SD	17.1±0.9	17.4±0.1	1.34	1.33	1.14	1.56	<0.01
Anxiety score, mean ±SD	2.4±2.7	1.8±2.4	1.10	1.10	1.05	1.14	<0.01
Morning dose (x10^{-3}mW), mean ±SD	1.3±5.0	1.1±7.0	14.10	194.11	1.22	30821.27	0.04
Total	441	12255					
Internet use: Yes/ No	1416/11280	6.4/ 4.1	1.59	1.91	1.44	2.54	<0.01
Hand-free use:							
No/ Frequent	10477/ 951	4.6/ 2.9	2.11	2.62	1.59	4.32	<0.01
Nocturnal dose group (x10^{-5}mW)							
≥2.00/ 1.80-1.99	7646/ 226	4.5/ 2.2	2.40	2.35	1.12	4.94	.024

Adjusted by Age, BMI, Vision, Anxiety, Depression, PSQI, Internet use, Hand free use, Brand device, and SOP.

Table 3.33 Odds ratio (OR) of daytime headache (12:00 a.m.-18:00 p.m.) and their 95% confidence intervals for each factor and daily dose adjusted for all other factors using GEE (AR1, QIC=3581.55, QICC=3566.90)

Factor	Daytime headache		Crude	Adjusted	95% CI		p-
	Yes	No	OR	OR	Lower	Upper	value
Age, mean ±SD	17.1±0.9	17.4±1.0	1.34	1.33	1.13	1.55	<0.01
Anxiety score, mean ±SD	2.4±2.7	1.8±2.4	1.09	1.08	1.04	1.14	<0.01
PSQI score, mean ±SD	4.2±2.1	3.6±2.1	1.11	1.09	1.04	1.15	<0.01
Lag_5 dose (x 10^{-3}mW), mean ±SD	2.1±14.0	2.0±16.0	3.44	9.96	1.28	77.43	0.03
Total	515	12176					
Internet use: Yes/ No	1416/ 11280	6.4/ 3.8	1.66	1.88	1.39	2.55	<0.01
Hand-free use							
No/ Frequent	10477/ 951	4.2/ 2.2	2.48	3.01	1.67	5.42	<0.01
Sometime/ Frequent	1268/ 951	4.0/ 2.2	1.96	2.07	1.07	3.99	0.03
Daytime dose group (x10^{-5}mW)							
≤1.79/ ≥2.00	2303/ 9896	4.8/ 3.9	1.46	1.52	1.10	2.11	0.01
Morning Dose group (x10-^{5}mW)							
1.80-1.99/ ≥2.00	120 /9710	5.8/ 4.0	1.58	1.79	1.03	3.12	0.04

Adjusted by Age, BMI, Vision, Anxiety, Depression, PSQI, Internet use, Hand free use, Brand device, and SOP.

Table 3.34 Odds ratio (OR) of evening headache (18:00 p.m.-24:00 a.m.) and their 95% confidence intervals for each factor and daily dose adjusted for all other factors using GEE (AR1, QIC=3969.12, QICC=3950.87)

Factor	Evening headache		Crude	Adjusted	95% CI		p-
	Yes	No	OR	OR	Lower	Upper	value
Age, mean ±SD	17.2±0.9	17.4±1.0	1.18	1.17	1.01	1.35	0.03
Depression score, mean ±SD	2.5±2.6	1.8±2.4	1.11	1.07	1.00	1.13	0.04
Lag_4 dose (x 10^{-3}mW), mean ±SD	2.4±11.0	2.1±16.0	2.95	4.53	1.06	19.38	0.04
Total	553	12139					
Internet use: Yes/ No	1416/ 11280	7.9/ 3.9	2.21	2.62	1.93	3.56	<0.01
Hand-free use							
No/ Frequent	10477/ 951	4.6/ 2.3	2.25	3.02	1.67	5.49	<0.01
Sometime/ Frequent	1268/ 951	3.9/ 2.3	1.91	2.04	1.05	3.97	0.04
Evening dose group (x10^{-5}mW)							
≤1.79/ 1.80-1.99	2648/ 301	4.8/ 2.0	2.48	2.60	1.36	4.97	<0.01
≥2.00/ 1.80-1.99	9747/ 301	4.3/ 2.0	2.36	2.59	1.38	4.86	<0.01
Daytime dose group (x10^{-5}mW)							
≤1.79/ 1.80-1.99	2303/ 79	4.1/ 2.5	1.76	2.60	1.36	4.97	0.04
≥2.00/ 1.80-1.99	9896 79	4.4/ 2.5	1.70	2.59	1.38	4.86	0.04

Adjusted by Age, BMI, Vision, Anxiety, Depression, PSQI, Internet use, Hand free use, Brand device, and SOP.

5) Time cycle headache and headache type

Table 3.35 Time cycle headache by headache type

Time cycle headache	Headache type			Total
	Migraine N (%)	TTH N (%)	Unidentified N (%)	N (%)
Morning headache	61(11.2)	382(69.8)	104(19.0)	547(32.1)
Daytime headache	60(11.7)	360(69.9)	95(18.4)	515(30.2)
Evening headache	21 (3.8)	460(83.2)	72(13.0)	553(32.4)
Nocturnal headache	11(12.2)	62(68.9)	17(18.9)	90(5.3)

TTH symptom is found mostly in all time periods compare to other headache type and as evening headache (83.2%). The migraine is the most symptoms as nocturnal headache (12.2%) (Table 3.35).

The study has concluded that headache symptom has associated with the factors of age, anxiety, depression, PSQI, internet use and hand-free use and dose of output power in range of 1.80-1.99x10^{-5}mW. On dose-response, smartphone output power has been found to have delay effects on headache symptom in both dose-response and reverse dose-response natures.

Objective 3: To study the correlation of smartphone output power and sleep quality of high school students.

3.2.2 The correlation of smartphone output power and sleep quality of high school students.

1) Sleep characteristics of participants

Table 3.36 Characteristic sleep of participants presented as percentage.

Variable	N	Minimum	Maximum	Mean ±SD
Time to bed	12696	5.00 p.m.	8.13 p.m.	24.26±2.0
Time wake up	12696	1.00 a.m.	23.35p.m.	8.04±2.27
PSQI score	12696	0	12	3.7±2.1
Duration time sleep	12696	0.30 hours	16 hours	7.4± 1.7
Efficiency sleep	12696	22.9%	100%	95.4 ± 7.1

The study on the relationship between SOP and sleep quality has found that most participating high school students go to bed and wake up late and their average sleep quality score is 3.7±2.1 (Table 3.36). Sleep problems in terms of difficult falling asleep (>20 min) and lack of habitual sleep efficiency have been found only 8.2% and 8.5% of the participants, respectively (Table 3.37). Meanwhile, 52.9% have sleep loss (<8hrs.), 29.7% have problems about frequent waking up during sleep, 23.9% have problems about feeling sleepy after getting up in the morning, 32.1% have poor sleep, 52.1% have poor sleep hygiene, 48.3% drinking 1 to 5 cups of coffee a day, and 27.9% using MP before going to bed.

Table 3.37 Characteristic sleep quality of participants presented as percentage.

Sleep quality	**N (%)**
Difficult sleep (min)	
>20	1036 (8.2)
≤20	11660 (91.8)
Sleep loss (hrs.)	
<8	6717 (52.9)
≥8	5979 (47.1)
Efficiency sleep	
<85%	1078 (8.5)
≥85%	11618 (91.5)
Wake up at night	
Yes	3777 (29.7)
No	8919 (70.3)
Morning sleepiness	
Sleepy	3037 (23.9)
Fresh	9659 (76.1)
PSQI	
Poor sleep	4071 (32.1)
Good sleep	8625 (67.9)
Doze	
Yes	9313 (73.4)
No	3383 (26.6)
Bad hygiene sleep	
Yes	6613 (52.1)
No	6083 (47.9)
Coffee or tea drink in day	
0	6547 (51.6)
1-5	6137 (48.3)
>5	12 (0.1)
MP USE @NIGHT	
Yes	3542 (27.9)
No	9154 (72.1)

The study on the relationship between SOP and sleep quality has considered sleep quality in two aspects: quantity and quality with the findings presented below

2) Relationship between smartphone output power and sleep quality in quantitative aspect.

Difficult getting asleep has associated with internet use, hand-free use (Table 3.39). Anxiety, depression, headache had relationship with sleep loss (Table 3.40). Headache, coffee drink, hand-free use and bad sleep hygiene have contributed to sleep problems (Table 3.42), and the factor of coffee drinking will have strong association with sleep problems OR_{adj}2.92; 95% CI: 1.31-6.54.

Daily dose, evening SOP in range of ≤1.79x10^{-5}mW and nocturnal SOP in range of 1.80-1.99x10^{-5}mW have1.71, 2.19 and 1.90 times respectively stronger relationship with difficult sleep while daily dose, evening and nocturnal smartphone output power in range of ≥2.00x10^{-5}mW have 1.32, 1.34 and 1.41 times respectively stronger relationship with sleep loss than their effects in range of ≤1.79 x10^{-5}mW (Table 3.40). The inefficient sleep has responded to daily SOP in range of ≤1.79 and ≥2.00x10^{-5}mW which is 4.54 and 3.81 times respectively stronger response than what happens in range of 1.80-1.99x10^{-5}mW (Table 3.41). Additionally, daily and nocturnal SOP in range of ≥2.00x10^{-5}mW appear to have 1.26 and 1.6 times respectively stronger relationship with sleep problems compare to their effects in the other two ranges while daily SOP in in range of ≤1.79 and ≥2.00x 10^{-5}mW has been found to have 4.54 and 3.81 times respectively stronger relationship with inefficient sleep than that in range of 1.80-1.99x10^{-5}mW range.

Morning Lag_1 in range of ≤1.79x10^{-5}mW and ≥2.00x10^{-5}mW have 3.41 and 2.32 times (95% CI: 1.50-7.75 and 95% CI: 1.11-4.86) respectively stronger link age with difficult sleep. Lag_6 daily dose has found the relationship with difficult sleep in the nature of reverse dose-response. Meanwhile, morning Lag_2 and daytime Lag_1 in range of ≥2.00x10^{-5}mW range are at 1.60 and 1.36 times respectively relationship with sleep loss. The relationship between Lag_4 daily dose and sleep loss has been found in the form of reverse dose-response. Morning Lag_2 in in range of ≥2.00x10^{-5}mW will appear to have 1.69 times strong relationship with sleep problems compares to in range of 1.80-1.99x10^{-5}mW. (Table 3.38)

Table 3.38 Odds ratio (OR) of sleep quality and their 95% confidence intervals for each factor and Lag dose adjusted for all other factors using GEE

Parameter	Exp.(B)	95% CI		p-value	Correlation structure	QIC	QICC
		Lower	Upper				
Latency sleep							
Lag_1	1.329E-15	1.564E-28	0.011	0.02	AR1	6449.132	6410.726
Lag_5	1.076E-16	1.870E-29	0.001	0.01	AR1	6446.205	6408.567
Lag_6	1.044E-12	1.155E-21	0.001	0.01	AR1	6452.448	6415.747
Lag-2	2.324	1.112	4.857	0.03	AR1	6431.641	6364.360
(6-12 a.m.)	3.413	1.503	7.753	<0.01			
Sleep loss							
Lag_4	0.003	1.156E-05	0.885	0.05	Exchangeable	15260.212	15229.724
Lag_2 (6-12a.m.)	1.599	1.108	2.306	0.01	AR1	15068.060	14971.740
Lag_1 (12-18 p.m.)	1.355	1.036	1.771	0.03	AR1	15094.968	15003.199
Sleep problem							
Lag_2(6-12a.m.)	1.685	1.170	2.426	0.01	Exchangeable	13193.475	13082.964
Poor sleep							
Lag_5	0.001	9.935E-07	0.822	0.04	exchangeable	13683.315	13614.371
Lag_2 (6-12 a.m.)	1.484	1.046	2.106	0.03	exchangeable	13638.242	13544.390
Lag_1(12-18 p.m.)	1.479	1.009	2.167	0.05	exchangeable	13615.496	13537.590

Adjusted by Age, BMI, Vision, Anxiety, Depression, Bad hygiene sleep, Coffee drink, Headache, Internet use, Hand free use, Brand device, SOP.

Table 3.39 Odds ratio (OR) of sleep difficulty and their 95% confidence intervals for each factor and daily dose adjusted for all other factors using GEE (AR1, QIC=6404.39, QICC=6336.22).

Factor	Sleep difficulty (>20 min)		Crude OR	Adjusted OR	95% CI		p-value
	Yes	No			Lower	Upper	
Lag_6dose (x 10^{-3}mW), mean ±SD	1.0±3.0	2.2±17.0	9.90 x10^{-13}	1.04x10-^{12}	1.16x10^{-21}	1x10^{-3}	<0.01
Total	1036	11654					
Internet use: Yes/ No	1416/ 11280	9.5/ 8.0	1.13	1.21	1.00	1.47	0.05
Hand-free use No / Yes	10477/ 2219	8.5/ 6.4	1.25	1.31	1.05	1.64	0.02
Dose group (x10^{-5}mW) ≤1.79/ 1.80-1.99	1943/ 186	11.1/ 10.8	1.80	1.71	1.12	2.63	0.01

Table 3.39 (continue)

Factor	Sleep difficulty (>20 min)		Crude OR	Adjusted OR	95% CI		p-value
	Yes	No			Lower	Upper	
Evening dose group (x10^{-5}mW)							
≤1.79/ 1.80-1.99	2648/301	11.9/ 6.6	2.13	2.19	1.01	4.71	0.05
Nocturnal dose group (x10^{-5}mW)							
1.80-1.99/ ≥2.00	226/ 7646	15.0/ 7.6	1.93	1.90	1.20	3.03	<0.01
Morning dose group (x10^{-5}mW)							
≥2.00/ 1.80-1.99	9710/ 120	7.7/ 3.3	2.33	2.32	1.11	4.86	0.03
≤1.79/ 1.80-1.99	2479/ 120	10.6/ 3.3	3.43	3.41	1.50	7.75	<0.01

Adjusted by Age, BMI, Vision, Anxiety, Depression, Bad hygiene sleep, Coffee drink, Headache, Internet use, Hand free use, Brand device, SOP.

Table 3.40 Odds ratio (OR) of sleep loss and their 95% confidence intervals for each factor and daily dose adjusted for all other factors using GEE (Exchangeable, QIC=15141.01, QICC=15054.85)

Factor	Sleep loss <8 hr.		Crude OR	Adjusted OR	95% CI		p-value
	Yes	No			Lower	Upper	
Anxiety score, mean ±SD	2.1±2.6	1.5±2.2	1.05	1.06	1.02	1.11	<0.01
Depression score, mean ±SD	1.7±2.4	1.2±2.0	1.08	1.04	1.00	1.08	0.04
Lag_4 dose (x 10^{-3}mW), mean ±SD	1.7±7.0	2.4±22.0	0.003	0.003	1.16x10^{-5}	0.89	0.05
Total	6717	5979					
Headache: Yes/ No	1705/ 10991	57.0/ 52.3	1.21	1.16	1.03	1.31	0.02
Daily dose group (x10^{-5}mW)							
≥2.00/ ≤1.79	10567/ 1943	53.8/ 48.5	1.30	1.32	1.08	1.60	<0.01
Evening dose group (x10^{-5}mW)							
≥2.00/ ≤1.79	9747/ 2648	53.8/ 50.2	1.13	1.34	1.02	1.77	0.04
Nocturnal dose (x10^{-5}mW)							
≥2.00//≤1.79	7646/ 3597	55.6/ 47.7	1.27	1.41	1.09	1.82	<0.01
Morning dose group (x10^{-5}mW)							
≥200/ 180-199	9710/ 120	54.9/ 41.7	1.44	1.60	1.11	2.31	0.01
Daytime dose group (x10^{-5}mW)							
≥2.00//≤1.79	9896/ 2303	54.5/ 47.7	1.32	1.36	1.04	1.77	0.03

Adjusted by Age, BMI, Vision, Anxiety, Depression, Bad hygiene sleep, Coffee drink, Headache, Internet use, Hand free use, Brand device, SOP.

Table 3.41 Odds ratio (OR) of inefficient sleep and their 95% confidence intervals for each factor and daily dose adjusted for all other factors using GEE (exchange, QIC=6297.93, QICC=6275.83)

Factor	Total	inefficient sleep%	Crude OR	Adjusted OR	95% CI		p-value
					Lower	Upper	
Daily dose group (x 10^{-5}mW)							
≤1.79/ 1.80-1.99	1943/ 186	11.8/ 3.8	4.51	4.54	3.33	6.20	<0.01
≥2.00/ 1.80-1.99	10567/ 186	8.0/ 3.8	3.79	3.81	2.59	5.60	<0.01

Adjusted by Age, BMI, Vision, Anxiety, Depression, Bad hygiene sleep, Coffee drink, Headache, Internet use, Hand free use, Brand device, SOP.

Table 3.42 Odds ratio (OR) of sleep problem and their 95% confidence intervals for each factor and daily dose adjusted for all other factors using GEE (exchange, QIC=13194.68, QICC=13085.02)

Factor	Total	Sleep problem%	Crude OR	Adjusted OR	95% CI		p-value
					Lower	Upper	
Headache: Yes/ No	1705/ 10991	34.3/ 29.0	1.13	1.13	1.00	1.28	0.04
Coffee or tea drink(cup) : >5/ No	12/ 6547	50.0/ 31.8	2.81	2.92	1.31	6.54	0.01
Hand-free use: Sometime/ Frequent	1268/ 951	34.5/ 29.4	1.19	1.18	1.01	1.39	0.04
Bad hygiene sleep: Yes/ No	6613/6083	31.8/ 27.6	1.12	1.16	1.03	1.31	0.02
Daily dose group(x 10^{-5}mW)							
≥2.00/ ≤1.79	10567/ 1943	30.4/ 25.7	1.28	1.26	1.01	1.57	0.04
Nocturnal dose group (x 10^{-5}mW)							
≥2.00/ ≤1.79	7646/ 3597	30.7/ 24.3	1.62	1.60	1.22	2.11	<0.01
Morning dose group (x 10^{-5}mW)							
≥2.00/ 1.80-1.99	9710/ 120	29.4/ 13.3	1.69	1.69	1.17	2.43	<0.01

Adjusted by Age, BMI, Vision, Anxiety, Depression, Bad hygiene sleep, Coffee drink, Headache, Internet use, Hand free use, Brand device, SOP.

3) Relationship between smartphone output power and sleep quality in quality aspect and overall sleep quality.

Morning sleepiness has responded to an anxiety (Table3.43) while poor sleep as an overall indicator of sleep quality respond to the factors of age, BMI, anxiety, depression, and headache (Table 3.44). BMI will have strong association with poor sleep (OR_{adj}2.12; 95% CI: 1.38-3.23).

Daily, evening and nocturnal smartphone output power in1.80-1.99x10^{-5}mW range have 1.60, 1.78 and 2.25 times respectively stronger effects on morning sleepiness compare to the range of ≤1.79x10^{-5}mW while daily and nocturnal smartphone output power in the range of ≥2.00 and 1.80-1.99x10^{-5}mW have1.30 and 1.66 times respectively stronger relationship with poor sleep compare to the range of ≤1.79x10^{-5}mW. Furthermore, the study has found that morning Lag_2 and daytime Lag_1 in the range of 1.80-1.99 x10^{-5}mW and ≥200x10^{-5}mW have 1.48 times stronger relationship with poor sleep than those in the range of ≤179x10^{-5}mW while Lag_5 daily dose relates to poor sleep in the form of reverse dose-response.

Table 3.43 Odds ratio (OR) of morning sleepiness and their 95% confidence intervals for each factor and daily dose adjusted for all other factors using GEE (exchange, QIC=12203.98, QICC=12125.19).

Factor	Morning Sleepiness		Crude OR	Adjusted OR	95% CI		p-value
	Yes	No			Lower	Upper	
Anxiety score, mean ±SD	2.3±2.6	1.7±2.4	1.06	1.06	1.03	1.10	<0.01
Total	3037	9659					
Daily dose group (x 10^{-5}mW)							
1.80-1.99/ ≤1.79	186/ 1943	36.6/ 30.5	1.58	1.60	1.20	2.14	<0.01
Evening dose group (x 10^{-5}mW)							
1.80-1.99/ ≤1.79	301/ 2648	34.9/ 32.9	1.77	1.78	1.21	2.61	<0.01
Nocturnal dose group (x 10^{-5}mW)							
1.80-1.99/ ≤1.79	226/ 3597	49.1/ 31.8	2.67	2.25	1.09	4.62	0.03

Adjusted by Age, BMI, Vision, Anxiety, Depression, Bad hygiene sleep, Coffee drink, Headache, Internet use, Hand free use, Brand device, SOP.

Table 3.44 Odds ratio (OR) of poor sleep and their 95% confidence intervals for each factor and daily dose adjusted for all other factors using GEE (exchange, QIC=13610.26, QICC=13535.37).

Factor	Poor sleep		Crude	Adjusted	95% CI		p-
	Yes	No	OR	OR	Lower	Upper	value
Age, mean ±SD	17.3±1.0	17.4±1.0	1.20	1.15	1.00	1.32	0.04
Anxiety score, mean ±SD	2.3±2.7	1.6±2.3	1.07	1.06	1.03	1.09	<0.01
Depression score, mean ±SD	1.9±2.6	1.3±2.1	1.09	1.05	1.01	1.09	0.02
Lag_5 dose ($x10^{-3}$mW), mean ±SD	1.7±6.0	2.2±19.0	0.001	0.001	$9.94x10^{-7}$	0.82	0.04
Total	4071	8620					
BMI: Abnormal/ Normal	1232/ 11464	48.5/ 30.3	2.25	2.12	1.38	3.23	<0.01
Headache : Yes/ No	1705/ 10991	38.8/ 31.0	1.29	1.21	1.06	1.39	0.01
Daily dose group (x 10^{-5}mW)							
≥2.00/ ≤1.79	10567/1943	32.3/ 30.5	1.32	1.30	1.03	1.64	0.03
Nocturnal Dose group (x 10^{-5}mW)							
180-199/ ≤179	226/ 3597	49.1/ 31.8	1.97	1.66	1.15	2.40	0.01
Morning dose group (x 10^{-5}mW)							
≥200/ ≤179	9710/ 2479	32.6/ 30.4	1.52	1.48	1.05	2.11	0.03
Daytime dose group (x 10^{-5}mW)							
180-199/ ≤179	79/ 2303	35.4/ 30.0	1.51	1.48	1.01	2.17	0.05

Adjusted by Age, BMI, Vision, Anxiety, Depression, Bad hygiene sleep, Coffee drink, Headache, Internet use, Hand free use, Brand device, SOP.

4) Relationship between smartphone output power and doze consequential to sleep problems

Anxieties, PSQI, bad sleep hygiene and coffee or tea drinking have the relationship with doze (Table3.45) but no respond from SOP.

Table 3.45 Odds ratio (OR) of doze and their 95% confidence intervals for each factor and daily dose adjusted for all other factors using GEE (AR1, exchange, QIC=11349.42, QICC=11268.01)

Factor	Doze		Crude OR	Adjusted OR	95% CI		p-value
	Yes	No			Lower	Upper	
Anxiety score, mean ±SD	2.0±2.5	1.3±2.0	1.16	1.05	1.01	1.10	0.02
PSQI score, mean ±SD	4.1±2.0	2.6±2.0	1.53	1.34	1.27	1.42	<0.01
Total	9313	3383					
Bad hygiene sleep: Yes/ No	6613/ 6083	77.4/ 69.0	1.50	1.18	1.03	1.36	0.02
Coffee or tea drink: <5/ No (cup)	6137/ 6547	83.9/ 63.5	3.23	1.77	1.41	2.23	<0.01

Adjusted by Age, BMI, Vision, Anxiety, Depression, Bad hygiene sleep, Coffee drink, Headache, Internet use, Hand free use, Brand device, SOP.

Sleep problems have associated with the factors of age, anxiety, depression, headache, and coffee drinking similar to findings in other studies. However, the study also provides the evidence not using hand-free device has a connection with sleep problems thus a piece of information supports the notion that smartphone electromagnetic radiation will affect to have difficult sleep, inefficient sleep, morning sleepiness, and poor sleep quality in the nature of window effect. Furthermore, delayed effect of SOP has been found to establish in the relationship with difficult sleep, sleep loss, sleep problems, morning sleepiness, and poor sleep quality in the form of window effect, dose response and reverse dose response.

CHAPTER 4

Discussion and suggestion

4.1 Discussion

Part 1: The mobile phone uses and health problem

Objective1: To study the characteristic of mobile phone using, headache symptom, and sleep quality among high school students.

4.1.1 The characteristic of mobile phone using and the headache symptom among high school students

1) The headache and characteristics of MP using

The results show that 92.6% of high school students (95% CI 90.8-94.1) have headaches at least once a year, which are divided into 12.3% of migraine and potential migraine group, 68.6% of TTH and potential TTH group, and 18.6% of undefined headache group. Nevertheless, the percentages of the current study, comparing to 54.4% (95% CI 43.1–65.8) of the past 25-years report, the systematic review of Wöber-Bingöl in 2013, which studied about headache in children and teenagers, are higher. However, the result of the study of Wöber-Bingöl et al. in 2014 were 89.3%[124] which was less than the percentage results in this study. In addition, the studies of Lewis (2007), the results were 57-82%.[34] Then, Straube et al., 2013 the results were 66-71%.[122] In Sweden, teenagers' ages between 12-18 years old, the percentage of the results are 64.9%, which are divided into 24.9% in migraine and potential migraine group, 37.6% in TTH and potential TTH group, and 31.2% of undefined headache group.[123] Also, Taiwan, in 2010, the results were 86.6%.[46]

In conclusion, the differences of the prevalence of headache among the various studies depend on the characteristics of the investigated samples, which include genders, ages, genetic factors, styles of living, geographic and climatic conditions of the studied area, and the diagnosis of headache itself.[125-127] The prevalence of headache in the teenagers trends to increase, which probably results from increasing the varieties of

stimuli or factors in daily lives, especially the new model released of mobile phone which are capable of serving a diversity of needs.[1]

The increasing values of MP use among children and teenagers in Thailand are well-manifested by the prevalence of mobile phone owner in this study, which is 99.8% of all subjects higher than 44.8%[175] of the study in Singapore, 2000 and 89.3 %[11] of the study in Rayong Province of Thailand, 2014.

Furthermore, the MPAH symptom (defined as MPAH ≥10 times/year) among Korean teenagers reported in Chu et al. study in the year 2011, was 18.9%[49] was much lower than 59.8% of this study. Thus, the increasing values of MP use might contribute to higher prevalence of headache in teenagers. According to the characteristics of MP use studied routinely, the results reveal 80.6% use for social media purposes and only 49.5% for verbal communication. Most students use MP at most 5 times a day with less than 10 minutes each, far different from Synovate survey (2011) which showed the average time of a teen talking on MP to be 60.7min/day.[12] The information reflect the change in the characteristics of MP use following the advancement of MP features.

2) The mobile phone associated headache (MPAH) symptom across demographic data and MP use

The study found that the MPAH symptom increases in the groups of younger students whose ages are between 16-17 years old. As the occurrence of MPAH is high risk factor, the younger-aged students have 4.1 times higher than the elder ones (greater or equal to 18) (95% CI: 2.64-6.26). The tendency that children start using MP in the earlier years of life is also the indicative of children's use of MP longer time and will have greater health effects on MP users in the young groups. The MPAH symptom will be higher in the groups of students who have visual problems, anxiety, and those who have a statistically significant correlation with MPAH. According to OR_{adj}2.22; 95% CI: 1.33-3.69 and OR_{adj}1.58; 95% CI: 1.07-2.35, the study shows 58.9% of the students having vision problems use MP generally for talking. Anxiety and visual problem may be a trigger of MPAH.

Furthermore, the MPAH symptom will be higher among the students who usually use talking mode of MP and have burning sensation at a significantly different level (p<0.05). The study has also shown that talking mode of MP use has 2.2 time higher risk than other modes. The findings are consistent with the Gogineni (2010), who found that talking mode had more power emitting source of an MP than the standby mode. The following shows SAR value comparison between talking mode (0.245, 0.548 and 0.963) and standby mode (0.0245, 0.0548 and 0.0963) at 900, 1800 and 2200 MHz frequencies in the GSM technology with output power of 200mW and 20mW respectively.[20] The study demonstrates the way in which electromagnetic radiation from MP may bring about headaches. The electromagnetic energy is a non-ionizing radiation that can produce heat.[20-21] The frequency of 900 and 1,800 MHz can increase the heat on the skin to 37.037 and 37.057 degree Celsius, respectively, and may lead the subject into experiencing burning sensations around the ears.[239-241] The present study has found no interaction between the effect of ear burning sensation on the relationship between typically using MP for talking and MPAH from, its stratified analysis. In the group of students' experiences no ear burning sensation, the study has found that they use MP typically for talking at 1.95 times (95% CI: 1.25-3.05), and are more likely to get MPAH compared to the use of MP in other modes. Furthermore, the study has found that the use of MP typically for talking is about 1.67 times (95% CI: 1.27-2.19) which is more likely to cause burning sensation, compare to the use of MP for other purposes. Therefore, talking on MP is a source of ear burning sensation and MPAH without controlling the confounding factors

Chu et al, 2011 found that MPAH linked to time talking but the present study has found no difference of the MPAH symptom on different duration of conversation using MP.[49] The result has related to the study of Lonn et al. and Gogineni,[20] they found that mobile phone, once pressed for function, used the peak power of 1W at 1800MHz frequency and then 2W at 900MHz frequency to establish the connection, and after that the power was controlled down to the level of as low as 1mW during conversation with a good signal quality, by the operator's network.[20, 99] The study shows that very short calls have been made at a higher than average output power.[108] Nevertheless, the output power also varies with operator's network, wave frequency, quality of the signal or density of the base station, the distance of MP from base station, and the movement of

user, while talking on MP, which necessitates a hand over or the change to a new signal channel.[17, 19, 99, 106-107] Furthermore, the study has found that most students (71.1%) have talked on MP less than 10 minutes per time and the effect of the duration group on MPAH symptom is manifested by OR 2.70 (95% CI: 1.83- 4.00; $p<0.05$). The data has ensured that duration of MP talking does not relate to MPAH, but MP talking itself relates to MPAH.

The association between use of hand-free device and MPAH has found in other studies, but not in the present study. Most students in the study, 71.1% and 81.4%, have spent short time, less than 10 minutes a time, with low frequency, less than 5 times per day, of MP use for talking. Hence, the result shows that the students generally use MP for social media purposes without hand-free devices. Thus, the study has found no association between use of hand-free device and MPAH. However, the study shows that the TTH will be higher among students who do not use hand-free devices, and also be accompanied by ear burning sensation. The result creates a point to consider whether talking on MP without using hand-free device can trigger TTH or not.

Other risk factors of MPAH at statistically significant level, $p<0.05$, include the uses of medicines for regular treatments, health risk behaviors, and poor sleep quality with OR_{adj}0.58; 95% CI: 0.39-0.86, OR_{adj}0.33; 95% CI: 0.16-0.69 and OR_{adj}0.53; 95% CI: 0.35-0.79, respectively. The target groups of students with regularly use of medicines, health risk behaviors, and poor sleep quality has less prone to MPAH than the opposite counterparts. The study has shown that less severe pain (mean 3.3 and 3.9 of non-MPAH) of MPAH resulted to no need for the use of medicine. The health risk behavior, in terms of tea and coffee drinking, will affect sleep quality and tea and coffee drinking will appear to relieve MPAH. With respect to sleep quality factor, the mean score is 4.8±2.9, indicating the normal level. The study has found that 54% of the students who have good sleep quality also use MP typically in talking mode. Thus, the study has found that uses of medicines, health risk behaviors, and sleep quality factors may not affect MPAH. The results indicate the difference between MPAH and primary headache in the forms of both migraine and TTH.

3) The characteristics of MPAH

Regarding the MPAH prevalence in terms of pain characteristics, the study has found MPAH has specific pain characteristics, including short time pain, pulsing and tightening, instable form of pain, pain often occurring in the morning, one side headache, and pain in the occipital and frontal areas which is consistent with the study of Gogineni in 2010 that found occipital area to have the spatial peak-average SAR (1g) at 1.86mW/kg.[20] The study has also found that the MPAH with different pain characteristics have low scores of pain severity and no impact from the pain, thus making the target groups of students with MPAH no need to take medication and no poor sleep quality problem. Similar to the South Korea study, the pain with specific characteristics identified in the present study could not be classified as primary headache,[49] neither does migraine or TTH.

The MPAH can be classified as secondary headache, as far as the MPAH meets at least two of the following criteria[137]: 1) There is a temporal relationship between the onset of headache symptom and the exposure of the factors believed to cause the disorder. The present study has found 53.2% of the students with MPAH report to the experiences of headache after having own mobile phones. 2) The characteristic of headache specifies to the causal factor. The study has found that the MPAH occurred during or after MP use has specific pain characteristics, different from the indicative of migraine or TTH. Hence, it cannot be classified as primary headache. 3) The headache becomes worse, statistically significant, with higher exposure to the causal factor. The study has found higher MPAH prevalence among students usually (more than 50%) use mobile phone for talking than the students seldom or sometime (less than or equal to 50%) use mobile phone for talking ($p<0.05$), relate to the exposure of more electromagnetic radiation. 4) Headache will be resolved, at a statistically significant level, with the well-managed of the causal factors. The criteria will be confirmed in the future. 5) The existing scientific information about the causes. Previous studies supported the content that the electromagnetic radiation from mobile phone induced the change in biological reaction, change of protein in the brain, and made nervous system problem, especially headache symptoms.[21, 24-25, 63]

Conclusion

Almost students have headache as well as high MPAH symptom in tandem with often use of MP. The present investigations on the relationship between the characteristics of MP use and MPAH have conducted in the context of rapid advancement in MP applications which is the reason for the change in the characteristics of MP use. However, the study results demonstrate that talking mode of MP is the strongest source emitting highest intensity of electromagnetic radiation from the device, and thus the use of MP in talking mode has the link with ear burning sensation and MPAH. The younger ages and visual problems also have high effects on MPAH. The characteristics of MP use and other factors that can trigger MPAH are expected to change with the advancement of the state-of-the art of MP in applications. The results have shown the difference between the characteristic of primary headache and that of MPAH, which has specific characteristics, including attack in the morning, short period pain, pulsing and tightening, indefinite form of pain, pain on one side, pain in the occipital and frontal areas, and ear burning sensation. The study results lead to the suggestions that using MP talking shall be made with hand-free device, to enable placing MP far away from the head, and that the ages at which children start having own and using MP will be higher, for health safety reasons and to prevent chronic headache.[176] The findings from this study can be informative and useful for further investigations in the future, on the relationship between electromagnetic radiation from MP and headache.

4.1.2 The characteristic of mobile phone using and prevalent of sleep quality among high school students

1) The prevalent of sleep quality

In the present study, PSQI has been found to have a mean of 4.8±2.9. Sleep problem prevalence (PSQI>5) has been found at 50.5%. In the study, the prevalence has been found to be higher than studies conducted before 2008. According to the literature review, prevalence was found to be 25-40%,[55] 39.61% in China,[51] and 66.10% in Australia.[60] However, the findings have concurred with a study conducted in Brazil and Lebanon, which assessed PSQI and found the prevalence to be at 54.7% and 58.79%,[53] respectively. The study has found sleep difficulty and frequently waking are at 4.0% and 17.9%. The study has different from Finland where sleep difficulty and frequently

waking are at rates of 30% and 60%.[242] In the meantime, sleep loss has been found to be consistent with studies conducted in the United States where sleep loss has been found at 61%.[79] In the study, mean sleeping time has encountered at a mean of 6.88±1.28 hours, which is less than the findings from a study conducted in Taiwan where mean sleeping time has been found at 7.35±1.23 hours,[243] and less than the findings from a study conducted in Greece where mean sleeping time has been found at 7.28±1.165 hours.[57] In the present study, adolescents have dozed during the day (4.8%), which was lower than the findings from a study conducted in the United States where dozing has been found at 22%[79] or 30% in China,[51] 70% in Greece.[55] To reduce dozing problems in the study may be caused by the fact that adolescents will often consume tea and coffee (68.1%), thereby preventing adolescents from experiencing daytime drowsiness. Furthermore, sleeping time also depends on different cultural and environmental factors in each country.[244] However, sleep problems tend to increase rapidly regarding to technological advances, particularly for mobile phones which have spread quickly.[64] The mobile phone use in adolescents had been found at 44.8% in Singapore (2006),[175] 64% in the United States(2011),[64] 78–84% in Malaysia (2014),[171] 89.34% in Rayong Province, Thailand (2014)[43] and 99.8% in the present study (2015). The study has shown that the sleep problems with MP use are more than half of the total prevalence. Many previous studies show that electronic media use and mobile phone talking before going to sleep have related to sleep problems (77%) and reduced sleep quality.[64, 77-78, 80, 82-84]

2) The sleep quality components across demographic data and MP use

The result shows the sleep of the risk behavior groups, in the area of tea or coffee consumption and use of medications have the strongest association with poor sleep quality OR_{adj}2.95: 95% CI, 1.71-5.09. Medications are used when the body is in discomfort, therefore, discomfort is a cause of poor sleep quality. Furthermore, the higher PSQI scores are from the students with history of phobia and potentially traumatic interpersonal events (PTIEs), also with students who have migraine headache and correlate to anxiety and depression score, with significant difference (p<0.05). The variables have weak effects on poor sleep quality and are adjusted by analysis. The researcher has assessed daytime dozing, which has impacted on sleep problems, and

found poor sleep quality to have the strongest effect to dozing during the day (OR_{adj}9.03: 95% CI, 2.72-29.94).

The study result shows that no use of hand-free devices in MP created 2.4 times higher risk for sleep difficulty among students who use hand-free devices. According to the sub-analysis in the MP with non-talking mode groups, no hand-free devices use consider as a risk factor for sleep difficulty OR_{adj}2.48 (95% CI, 1.13-5.43) compared to hand-free use. Therefore, sleep difficulty should be caused by non-talking mode MP uses. The finding is consistent with the findings where mobile phone use at night has risk for sleep loss (OR_{adj}1.82: 95% CI, 1.10-3.01). MP use at night is not usually aimed at conversations, meaning students who use MP tend to do so for a long time and causes students to sleep late. The students (55.4%) slept after 10:00 p.m., causing sleep loss with a mean sleeping time of only 6.88±1.28 hours. Thus, MP uses at night are found to pose a risk for dozing in the morning after waking up, (OR_{adj}1.70: 95% CI, 1.08-2.66). MP uses at night for purposes other than conversation have related to MP lights which disturb sleep. The study conducted by Alexandru et al. found extended TV viewing and game-playing among students related to sleep difficulty, [64, 81] the study can also predict shorter sleeping times.[245] Lights from mobile phones, televisions, and gaming consoles have been found to suppress melatonin secretion and disturbed the sleep-wake cycle, resulting in longer time until we fall asleep.[64]

MP conversation duration of more than ten minutes has been found to pose risks for sleep deficiency (OR_{adj}2.26: 95% CI, 1.17-4.36). Extended MP talking mode has high exposure rate to electromagnetic radiation from MP. The finding concurs with a study conducted by Gogineni, who found the talking mode to have higher output power (200mW) than standby mode (20Mw).[20] Data from an experiment conducted by Huber et al. found that exposure to an electromagnetic radio frequency from MP for 30 minutes led to increase blood circulation at the dorsolateral prefrontal cortex, creating changes to alpha brainwave frequencies before sleeping and increased spindle brainwave frequency in Stage 2 of sleep.[246-248] Furthermore, latency sleep time will increase more electromagnetic energy intensity that indicates a relationship of dose-response.[248] Furthermore, Burch et al. Wood et al. and Jarupat et al. found the groups that used mobile phones for more than 25 minutes per day to have lower

6-hydroxymelatonin sulfate (6-OHMS), a melatonin metabolism that is excreted in urine and saliva,[249-251] with even decreasing amount in the groups with high MP use.[249] A recent review article has supported the fact that MP uses before sleep will change melatonin and cortisol secretions, which are the hormones in the sleep-wake cycle[219, 252] that triggered the nervous system in the parts related to sleep.[219]

PSQI is overall sleep quality. Duration of MP conversation is more than ten minutes and non-Apple brands of MP have been found to pose a risk for poor sleep quality (OR_{adj}1.601: 95% CI, 1.10-2.34 and OR_{adj}1.57:95 CI, 1.08-2.271, respectively). Each of MP device brands has difference antenna, causing Specific Absorption Rates to be different.[20, 253] On the contrary, SAR in Apple devices have been found more than other brands. Furthermore, the effects of Apple brand devices in rural areas on poor sleep are manifested by OR_{adj}2.79: 95% CI, 1.27-6.41; p<0.05, compared to urban areas. Therefore, brands of mobile phone do not have influence on sleep quality, but might be the environmental factors involved in the use of MP, such as urban areas with strong signals, density of base stations, and service networks might release lower MP output power.[17, 107-108, 253]

The study result also shows non-conversation and long conversations MP uses at night will affect quantity of sleep. Furthermore, long MP conversations will have poor sleep quality risks. Previous studies have found that adolescents who have MP conversations before sleeps (23.6–62%) relate to daytime dozing.[64, 82] Adolescents usually have conversations at 00:00–3:00 a.m. causing sleep loss, inefficient sleep, and fatigue with weakness during the day. Long MP uses at night more than once a week will have 5.1 times higher risk for fatigue and weakness during the daytime.[237] However, modern MP causes changes in MP modes from MP conversations to online social media uses. The study shows short durations and frequency uses in MP conversations while online social media purposes are the highest uses (80.6%) with more time requirements and exposed to lights from MP, causing effects on sleep quality.

Conclusion

The sleep problems with MP use is more than half of the total prevalence. The modernized MP use affects characteristics of sleep, sleep quality, and impact of learning

of the teenagers. The hand free uses, MP talking modes, calls duration, frequency of MP use, device systems, and MP uses at night related to PSQI both quantity and quality domain. Poor sleep quality will cause dozing in the daytime. MP is a source of electromagnetic radiation which is used close to the head, while human nervous systems are unstable electrical tissues.[64] Therefore, disturbance or stimulation of changes in brain waves and biological sleeping systems in the body can occur. Nevertheless, confirmation of relationships between electromagnetic radiation from MP and sleep quality require further progressive studies.

The study of characteristics of MP using, prevalent of headache, and sleep quality in high school students had some limitations, because the study is self-reported study. The information obtained could be either underestimated or overestimated due to recall bias. The cross-sectional design prevents accurate and clear explanation of temporal cause-effect relationship. In addition, the lack of study coverage dealing with different MP operation networks and wave frequencies are pertinent to the transmission and radiation emitting power of the mobile phones. The important points of the study include the sample sizes, which are large enough for reliable analytical results, and the selection of the high school students in the provincial schools, as the study subjects can represent the population of high school students nationwide.

Part 2: Electromagnetic radiation from smartphone and health problem

Objective 2: To study the correlation between smartphone output power and headache among high school students

4.1.3 The correlation between smartphone output power and headache among high school students

1) Headache

The present study shows the current headache to be 13.4%, predominantly in the types of TTH, migraine, and undetermined headache with rate of 74.1%, 9.0% and 16.9% respectively. The result is consistent with the findings from the first phase of study, involving the entire student population, that the prevalence of headache in the three types was 68.6%, 12.3%, and 18.6% respectively which is, however, different from studies conducted elsewhere. A study in Sweden using daily headache record revealed migraine 24.9%, TTH 37.6%, and undetermined 31.2%.[123] In low income

countries, the headache prevalence is 54.4%.[37] The 2002-2012 survey in USA revealed the prevalence of migraine headache to be 12.2%.[254] Headache prevalence varies across geographic areas, cultures, and the sample groups, as well as the criteria for classifying types of headache.[125-127] The present study shows low pain scores of headache, similar to the study result of Larsson that found 55.0% of teenagers reporting low pain scores of headache,[123] and consistent with the characteristics of headache associated with MP use in the study by Chu[49] as well as the results of the study in the first phase. With the division of a day into four time periods, the present study finds most students experienced headache before 00:00 a.m. However a study in Belgium in 2007 reported that teenagers used MP during 00:00-03:00 a.m. for conversation (17.3%) and texting messages (20.3%).[237]

2) Information on smartphone output power

The characteristics of smartphone usage among sample students are considered on the basis of smartphone output power (SOP) which can be measured real time by the device. The researcher has made an application which is able to record SOP and send them through email. SOP values in this study are thus lower than the values of smartphone electromagnetic radiation in other studies, which was measured by external metering devices, and might be increased by the radiation from other sources. The previous study of Frei et al.[255] found mean personal exposure of electromagnetic field intensity over one week from the different sources, excluding own mobile phone, was 0.013mW/cm.^{2} Bolte and Eikelboom et al.[256] which the exposure was measured over 24hours at 0.0180mW/cm^2 and 0.00289mW/cm^2 of Beekhuizen et al. In the present study, maximum and mean SOP was 1.55 and 0.001mW. The maximum and mean SOP were correspond to the study of Kelsh et al.,[19] and Gosselin MC (2010), which found the average output power between 0.001-0.63mW.[109] The results were lower than the study of Lonn.[99] which was found at 25-63mW. In the present study, maximum SOP has been found at a nocturnal time (00:00-6:00 a.m.) but most subjects under study hardly used smartphone at night, resulting in the average SOP of $1.0x10^{-3}$mW. Since students shut down their smartphone from time to time, this makes the minimum SOP in each period of the day equal to zero. The students generally use the devices in the morning, daytime, and evening before bedtime (18.00-24.00 p.m.) with the average SOP of 1.1, 1.2 and $1.1x10^{-3}$mW respectively, the average is the highest at night. However, the daily

smartphone output power of $2.0x10^{-3}$mW suggests that some students use the device quite heavily at night. The first phase of the present study using questionnaire has found that 66.5% of students use telephone before going to bed, and MP has affected to the quality of sleep. The study by Bulck in 2007 found 62-72% prevalence of adolescence use MP at night, after 9:00 p.m.,[237] 55.6% for texting messages, 58% for talking,[237] and 24% for playing games, and found that they used MP and internet late at night up until 00:00-03:00 a.m.[237]

3) Factors correlated to headache

The present study has found the correlation between the factors of ages, anxiety, depression, and PSQI and headache in terms of headache events, durations, frequencies of uses, severities, and types including migraine, TTH, and unidentified.

Anxiety is a risk factor of morning and daytime headache, OR_{adj}1.10 and 1.08 (95% CI: 1.04-1.14). Depression is a risk factor of the occurrence of evening headache, OR_{adj}1.08-2.41; 95% CI: 1.04-1.14. PSQI is a risk factor of daytime headache, OR_{adj}1.09-2.41; 95% CI: 1.04-1.15. Meanwhile, both ages and internet uses are risk factors of headache at any period of times in a day.

Age has been found to have relationship between headache symptom, time duration of headache, frequency of headache, severity of headache, and all types of headache. Compared to a year younger student, a year older likely faces a relatively high severe level of headache symptom, duration, frequency, severity, and types of headache. The result is contrast with most of the previous studies, which found headache symptom varies to age,[38, 122] particularly the migraine type and tension type of headache, though the positive correlation appeared weaker in the 30-39 age group.[35] The results from the present study are in line with the first phase investigation, on factors associated with headache from mobile phone use, that young age has implication for MPAH. Previous surveys revealed as high as 31% of children of the age 8-10 own and use MP.[258] A study in Korea (2013) found the average age of children first owning and/or using MP decreased from 12.5 years old in 2008 to 8.4 years old in 2011. The results were implied the tendency of children own and use MP at a younger age.[258] The tendency result in a longer accumulated time of owning and using MP during their childhood, has supported the theory that young-aged students are more likely to get

headache when use MP, compared to the old-aged students who have more regular activities and other extracurricular activities in school, hence having less time for smartphone use. The present study has found that students at the average age of 17.37 years old correspond to smartphone output power in the range of $\geq$ 2.0 x10^{-5}mW, compared to those with average age of 17.88 years old who correspond to SOP in the range of 1.8-1.99x10^{-5}mW (p<0.01). The current study has also found that the factor of age associated with headache in all four periods of the day, and that the information supports the observation to ensure that young students use smartphones all time periods. The higher scores of anxiety, depression, and PSQI are the greater bearing on the frequency and severity of headache and TTH. The previous study on headache indicated that depression had 1.9 times more likely to trigger headache.[140] The study by Fuh et al. revealed that higher depression score was a predictor of moderately severe and severe headache,[42, 46] and with the finding that anxiety would increase the risk of headache to occur. Both anxiety and depression are likely to generate more frequent and more severe headache.[42,259] Empirically, anxiety and depression have been found to be 4.1 and 1.7 times, respectively, higher risk than other factors that cause headache,[2 60] as well the anxiety and depression are risk factors of TTH with OR_{adj}1.04-1.06; 95% CI: 1.00-1.08.[42, 259, 261] Also, the anxiety and depression have associated with migraine type, which is different from the result of the present study that suggests the association with TTH which generally arises from psychological, social stress, and muscle tension.[261] Furthermore, the study has found the anxiety can cause only morning and daytime headache not evening and nocturnal headache. According to, the students always have attended the classes, have activities, and interact with numerous people in the morning and daytime, so the students are able to reduce their anxiety. While evening time, night time and bedtime are the most relaxing hours, thus there is no anxiety to lead to headache in particular periods of time. There is information confirming that sleeping can relieve headache, while sleep problem can trigger headache,[42] and sleep quality and headache has reciprocal connection. There are also evidences that suggest sleep problems makes it 2.03 times more likely for headache to occur (95% CI: 1.6-2.5).[42, 140, 260, 262]

High scores of PSQI are found to link with migraine[260, 262] and sleep quality has been found to be a factor highly explanatory for frequent headache attacks.[262] This is

also supported by the present finding that poor sleep quality has association with headache attack and daytime headache (OR_{adj}1.04-1.09). The sleep has related to headache, particularly migraine, is triggered to hypothalamus which is linked with limbic system, retino-hypothalamic tract, and brainstem aminergic nuclei,[260] as well as with periaqueductal gray (PAG) matter. Provoking the orexin once causes the "rapid-eye-movement sleep-off", the norexin will trigger ventrolateral part of the PAG matter to suppress antinociceptive activity in trigeminal nucleus caudalis, resulting in migraine headache.[259] The abnormal brain functions, sleep related biological mechanism, and the migraine headache type have reciprocally connected. The study has been found that migraine can be affected by the neurotransmitters, serotonin, dopamine, and melatonin[260] and the anti-inflammatory system, provoked by migraine, will affect the level of melatonin.[42] Other findings in the study on the relationship between poor sleep quality, frequency, and severity of headache confirm the result from the first phase of the study, that poor sleep quality is not a protective factor of MPAH. In other study found that 48-74% and 26-72% of migraine and TTH cases have associated with lack of sleep. The report showed that after the patients adjusted their sleeping pattern, their headache were relieved to a better condition.[264]

Internet use is a risk factor for headache event, duration, severity, frequency, and all types of headache (OR_{adj}1.98-2.41; 95% CI: 1.20-3.51). Talking on smartphone in both internet and cellular modes often involves holding the device close to the head, and the electromagnetic radiation from smartphone to which the users are exposed to induce the change in biological reaction, change of protein in the brain, and causes nervous system problem, especially headache symptoms.[22-23, 25-27] Thus, brain is the organ closest to MP when the device is in use, especially by holding it next to the ear for talking and listening. Electromagnetic radiation from talking mode is nine times more intense than standby mode.[265] The study by Gogineni in 2010 concluded that the talking mode had more intense power emitting source of an MP than the standby mode which reported the SAR values of talking mode (0.245, 0.548, and 0.963) and standby mode (0.0245, 0.0548, and 0.0963) at 900, 1800, and 2200 frequencies in the GSM technology with output power of 200Mw and 20mW, respectively.[20] Furthermore, the recent study has found higher mean of radiated power during voice over internet protocol, which has been assessed at 1.9mW, than the mean of radiated power during

voice over Circuit Switch calls, which has been assessed at 0.55mW.[266] The information supports the finding from the first phase study that MP use in talking mode is a risk factor of MPAH and the finding from the present study that internet use for talking can bring about headache in all time periods of the day, which in turn indicates that students use internet for talking in all periods of the day. There is also a finding from the present study that not using hand-free device while talking on smartphone produces strongest effect in causing migraines and TTH (OR_{adj}3.96; 95% CI:1.21-12.87 and OR_{adj}3.72; 95% CI: 2.49-5.56). This practice had association with headache event, duration, severity, and frequency of headache (OR_{adj}3.03-3.22; 95% CI: 1.74-5.27). Use of hand-free device for talking allows some distance between the telephone set and user's head, resulting in lower exposure to electromagnetic radiation.[239] Not using hand-free device was also found to link with morning, daytime, and evening headache because these time periods are when most students use smartphone. The results are manifested by the mean SOP in the morning, daytime, and evening dose, 1.07-1.18x10^{-3}mW, while nocturnal dose has appeared to be the least, 1.00x10^{-3}mW. The results indicate that using hand-free device has related to SOP and headache.

Brand of the device has appeared to have a bearing on nocturnal headache. From the analysis of smartphone use in the nighttime (00:00-6:00 a.m.), the result has been found among late night users that students using brand devices other than Apple, mostly, 83.6%, use smartphone output power in the range of ≥2.00x10^{-5}mW, compared to the 58.9% figure of Apple brand device users. The result implies that users of smartphone other than Apple brand use the device heavily at night, thus contributing to the linkage between brand device and nocturnal headache. It is important to note that brand device is a representative of area where the device is used and the brand device used popularly in rural area, which has less density of the base station, will have an effect on sleep quality. The theory is in line with the findings from previous studies, that the factors governing smartphone output power has included the control system of the operator's network, the wave frequency, the strength of the signal, which depends on the signal density of the base station, the distance of MP from the base station, and population density.[17-19, 99, 107]

4) Smartphone output power (SOP) and headache

Smartphone Output Power in this study reflects the Smartphone Electromagnetic Radiation from the phone use during a time period as defined for a day. It is the important measurement of power emission from smartphone for assessing the levels of electromagnetic energy to which human body has exposed to and absorbed into tissues,[17-18, 99] which vary with the amount of time using the mobile phone.[18] The duration of time of repeated or continued mobile phone use is thus an important variable for the assessment of exposure to electromagnetic energy.[17] The assessment of the exposure to electromagnetic radiation at an individual level is a crucial issue in epidemiological study, as the findings from the inference study process can be applied to the pertinent population.[99]

The present findings revealed that headache symptom, pain score and frequency of headache, undetermined headache type respond to smartphone output power in the range of 1.80-1.99x10^{-5}mW, duration of headache responds to smartphone output power in the range of ≤1.79x10^{-5}mW, and migraine responds to SOP in the range of ≤1.79x10^{-5}mW and 1.80-1.99x10^{-5}mW. The responses are of power effect type. Furthermore, the analysis on the part of headache event in four periods of the day indicated that morning headache responds to morning SOP in dose-response nature, while daytime headache responds to daytime SOP in the range of ≤1.79x10^{-5}mW, and evening headache responds to evening SOP in the range of ≤1.79x10^{-5}mW and ≥2.00x10^{-5}mW in the nature of power effect.

The effect of SOP on headache has been found to be non-linear. There appeared to be no statistical evidence of maximum SOP having a bearing on headache. However, it was found that SOP in the range of ≤1.79x10^{-5}mW and 1.80-1.99x10^{-5}mW have the linkage with headache event, frequency of headache, and severity of headache, but the relationship has not found for SOP in the range of ≥2.00x10^{-5}mW. By the types of headache, both migraine and undetermined type headache have appeared to be associated with SOP in the range of ≤1.79 and 1.80-1.99x10^{-5}mW, respectively. The undetermined headache in the 1st stage of study was the group of mobile phone associated headache symptom which was different in clinical features, from primary headache, and it should be classified as secondary headache. Many previous studies

found MP had negative impacts on human health and the headache was found to be the most common problem.[22, 24] The present study has found the non-linear relationship between SOP and headache, which agrees with the experimental studies on the exposure of MRF which found the response to take place only at specific values or in specific range but no response beyond the upper and lower thresholds, is called window effect.[111, 267-269] Similarly, Frey et al. conducted a study by getting experimental animals to expose to RFR at 1200MHz frequency and 2.4mW/cm^2 and 0.2mW/cm^2 intensity for 30 min, and found the dye could penetrate through BBB. Meanwhile, Merritt et al. investigated the exposure to RFR at 1200MHz frequency and 2-75mW/cm^2 concentration, and found no difference in BBB permeability of fluorescein-albumin. The results can be concluded that BBB responds to RFR only at 2.4mW/cm^2 and 0.2mW/cm^2 intensity.[268] Bawin et al. found electromagnetic radiation at 6 and 16Hz frequencies to be the frequency windows which allow the maximum reduction of response to calcium efflux from brain tissue,[270] as well as the change in EEG pattern that was found from exposure to MFR only at 8 and 16Hz frequencies, not at any other frequencies. Meanwhile, Dutta et al. found AChE to increase in intensity at SAR equal to 0.05 and 0.02W/kg.[116] Furthermore, numerous studies found genetic alteration of the brain tissues resulting from exposure to Microwave Frequency Radiation (MFR) and found the response to take place between 1 and 10μW/m^2 intensity, which was shaped by the window effect, since no response could be found outside this range of intensity.[271] Moreover, migraine has been found to have effects on biological system, not only the nervous systems but also other systems of human body which have regulatory function to ensure system equilibrium.[272] The result can explain why the response takes the form of nonlinearity.[111] Response as found in different studies have not taken place at a definite time, as it is a low level response depending on the sensitivity of the exposed individual.[273] Therefore, headache from MP use is characterized by low severity, which is typical for the so called mobile phone associated headache (MPAH).

Mechanism of headache response to MFR is often driven by the dysfunction of endogenous pain control system, which is a helper in adjusting the pain or the response level by sending signal to the trigemino-cervical complex that regulates the function by neurotransmitter. Exposure to RFR of 1600-3000MHz at 10-30mW/cm^2 8h/day for

7 days has been found to decrease the concentration of both serotonin and 5-hydroxyindolacetic acid, which is the product of serotonin metabolism[268] (Snyder, refer in[273]). Furthermore, acetylcholine in AChR type, located at neuronal nAChR in thalamas, raphe magnus nucleus, and spinal cord have important role in inhibiting pain. Baranski, who conducted a study on experimental rats, which were put to be exposed to RFR at 3000MHz frequency and 25mW/cm^2 intensity, found that would reduce the functioning of AChE at diencephalon.[268] Moreover, headache is often associated with the functioning of the dopamine-opiate system, that human body uses for inhibiting pain sent by the opioid receptor and thus causing the reduction in number of neurotransmitters in the system and consequently the failure of the system to inhibit pain.[275-276] Exposure to low intensity MFR or microwave energy has been found to inhibit apomorphine in the opiate system.[25] Lai et al. gave narcotic antagonist, naltrexone (a medication to reduce activity of dopamine-opiate) to experimental rats every day before exposure (pretreatment) to radio frequency radiation (RFR) at 2450MHz and SAR of 0.6W/kg for 20 minutes and found no change in response of apomorphine. Meanwhile, Frey and Wesler examined the functioning of dopamine system and found that the pain response period was reduced when the experimental rats were given apomorphine before exposure to RFR (1200 MHz at 0.2mW/cm^2 for 15 min).[121, 268] These evidences supported the theory that exposure to smartphone output power can reduce the number of neurotransmitters which trigger headache, especially the migraine type. In the related study, Ungureanu et al. found that using smartphone to talk for 5-10 minutes and 10-15 minutes resulted in rapid temperature increase by 2-3.5°C in tissues of the face and ear areas, which were the trigeminal area.[240-241] The increased temperature will be diffused to neighboring areas, causing the dilation of intracranial-extra cerebral blood vessels which carry blood to dura mater, which in turn produces an effect on the surrounding outer layer of blood vessels of sensory nerves in trigeminal area, and these are the key mechanisms of migraine headache.[277] The afore-cited study lends a support to the present findings that not using hand-free device has a bearing on headache event, duration, frequency, severity, and types of headache, particularly migraine.

The present study has found frequency and high pain score of headache, as well as daytime and evening headache, respond to the delayed effects after exposing to daily dose of smartphone output power about 6 to 7 days in the form of dose-responses. While migraine of which the nervous system has adjusted processes to delay effects after exposing to daily dose of smartphone output power for 7 days in the form of a reverse dose-response. As the repeated exposure to MFR for a length period of time results in the accumulation of response and the long time response, which is consistent with the findings of Merritt[268] The study was conducted in experimental rats with expose to RFR at 2375MHz frequency and 50 and 500μW/cm^2 intensity, 7 hours a day for 30 days, and found the epinephrine response in the brain to increase on the 20th day after the exposure and get back to normal on the 30th day. While the result of 500μW/cm^2 intensity revealed the increase in norepinephrine, dopamine, and serotonin on the 5th day after exposure which, however, reduced after the continued or repeated exposure.[268] The delayed effect from previous exposure and the biological response from repeated exposure to RFR depends on the stressed or the exposed factors and the exposed area. Oscar et al. got experimental rats to expose to MFR 2800MHz and SARs 0.2 and 3W/kg for 5 to 60 min, and found blood flows increased in rats' brain in the 6th minutes post-exposure, which increased the most in pineal gland, hypothalamus, and temporal cortex. The increased blood flow, which is the response, will vary across regions of the brain, probably due to the uneven diffusion of RFR and different parts of the brain are sensitive differently to RFR.[278] Biological responding to prolonged and repeated exposure to RFR depends on the dose of the exposing RFR. Exposure to RFR at SAR 0.6W/kg for 10 minutes was found to increase activity of cholinergic receptors in the brain, while the repeated exposure for 10 days appeared to bring about the reduced activity of cholinergic receptors in frontal cortex and hippocampus areas, but cholinergic activity in hippocampus has been found to increase from the repeated 45 min exposure in 10 days. The change has been found to depend on endogenous-opioids and indicated that the response of nervous system to RFR exposure is driven by the induction of endogenous-opioids system.[25, 268] Meanwhile, no change in activity of cholinergic receptors is proven when the lab animals have been given pre-treatment narcotic antagonist.[268] Results from various studies demonstrate that duration of exposure, repetition of exposure, frequency and intensity of electromanetic energybring

about different biological impacts and responses, and that the physiological changes in different parts of the brain will stop and everything will get back to normal after stop the exposure.[279] However, there is no clear explanation on the mechanism for the occurrence of delayed response to electromanetic exposure which might be due to the adaptive process of the nervous system to return to equilibrium or due to allostatic load. Yet, the repeated exposure is synonymous with the continued accumulation and thus causes the nervous systems to respond to the accumulated effect. For this reason, headache in general responds to delayed effect in the form of dose-response, while migraine and nocturnal headache respond to delayed effect in a reverse dose-response pattern.

Characteristically, migraine occurs as the result of brain nervous system using adaptive process as a response mechanism to cope with repeated stress, and the adaptive process involves the continuative and cumulative alteration in cells and system which leads to either the restoration of equilibrium (allostasis) or allostatic load, if the adaptive process takes place abnormally.[2 72] The migraine maladaptive process results in the system's failure to habituate to repeat the stressor of the same kind[280] and this activates increased in response to other mediators, manifested as central sensitization leading to lower resistance to pain, and sensitivity in response to the same stressor.[2 81] Certain responses reflect protective adaptive process, for examples, allodynia, phonophobia, photophobia, osmophobia, andallodynia, which pains are triggered by the stressors at a normal level.[2 8 2-2 85] Children attacked by migraine have been sensitive to hot temperature at trigeminal area, indicating that greater numbers of nerve cells are being stimulated.[285-287] Thus, the finding from the first phase of this study, headache severity using MP,[4 9] can explain that migraine has respond to low output level and delayed effect in the form of reverse dose-response, and is an output effect.

From the study on headache in different periods of the day, the researcher has found that nocturnal headache have no response to SOP during the evening, before bedtime. The finding indicates that using smartphone before going to bed does not stimulate the brain, in order to result in nocturnal headache (00:00-6:00 a.m.).

Meanwhile, daytime smartphone use (12:00-18:00 p.m.) with power effect (1.80-1.99 $x10^{-5}$mW) and perhaps in combination with night time smartphone use can bring about nocturnal headache. Furthermore, using daily dose of SOP for 7 days has been found to have nocturnal headache consequence in a reverse dose-response form, which is likely to be the adaptive process of the nervous system, particularly in migraine type headache. Use of SOP in the morning (6:00-12:00 a.m.) and at night (00:00-6:00 a.m.) in the form of high power effect ($\geq 2.00x10^{-5}$mW) was found to link with morning headache and indicates that students having morning headache will include those who use smartphone heavily after midnight. Severe morning headache is not the consequence of only exposure to high dose of SOP but also sleep deprivation.

Meanwhile, use of SOP during daytime and in the morning ($\leq 1.79x10^{-5}$mW and 1.80-1.99$x10^{-5}$mW, respectively) in the form of power effect has been found to induce daytime headache. Furthermore, use of daily dose of SOP for 6 days apparently caused daytime headache in the form of dose-response.

Use of SOP in the evening ($\leq 1.79x10^{-5}$mW and $\geq 2.00x10^{-5}$mW, the minimum and maximum output level, respectively) has associated with evening headache. Moreover, the exposure to daytime SOP for 6 hours ($\leq 1.79x10^{-5}$mW and $\geq 2.00x10^{-5}$mW) in the form of power effect and daily dose of SOP for 5 days have given rise to evening headache in the form of dose-response. The researcher has observed that SOP in the range of $\leq 1.79x10^{-5}$mW, which is the lowest level that can trigger headache, probably due to the sensitivity of each individual[273] or the response of the nervous system to the frequency of Smartphone Electromagnetic Radiation in this range.[288]

Nocturnal headache in the present study is different from hypnic headache, as it does not wake one up from sleep but it just occurs at night (00:00-6:00 a.m.) and has been found in only 5.3% of the participants. However, nocturnal headache, mostly, can be classified as migraine type (12.2%). Nocturnal headache has been found to respond to the delayed effect of daily dose of SOP in the form of a reverse dose-response, just like migraine headache which has some other kinds of protective response for example photophobia. There is an adaptive process in the nervous system that migraine develops.[272] The researcher's findings ensure that both migraine and nocturnal headache will have specific responses to SOP. Furthermore, many of the recent studies of pain

potentiated the activation by light[289] and the result has shown increasing in cortical excitability during migraine attacks and visual hyper-excitability occurred. The pathway between the eye and the brain may produce pain. The pain pathway has begun with the intrinsically light cells that will transform the light absorbed by the eye into a painful stimulus.[290] Noseda et al. found that light stimulations activated migraine by dura-sensitive thalamic neurons that receive photic signals from the retinal ganglion cells and transmit signal to cortical areas involved nociceptive. The retinothalamic-cortical pathway has provided exacerbation of migraine headache by light.[291-293] The information ensures that nocturnal headache in the study is migraine which has been activated by output power and the light from smartphone.

The findings from the present study point out that MFR from smartphone is likely to be the trigger of MPAH and a trigger of headache, particularly the migraine type. To be considered as trigger,[263] the factor or stressor must contain the following conditions: 1) Trigger must be able to reach blood vessels or many receptors in the nervous system, in order to cause the biochemical reactions. Previous studies revealed that MFR induced the response from various neurotransmitters such as cholinergic receptors and endogenous-opioids,[252-268] 2) Trigger must have reasonable linkage with the receptors; 3) There is a definite amount of exposure to make the response observable. Study findings revealed that headache and migraine response to smartphone output power in the window effect pattern ($1.80\text{-}1.99\times10^{-5}$mW and $\leq1.79\times10^{-5}$mW) which are the specific levels for the occurrence of response; 4) There might be co-factor for triggering effect like use of MP for a long time conversation: 5) Triggers on the central nervous system effects must be able to penetrate or damage blood-brain barrier. The existence of empirical evidences have proven that MFR causes alteration in blood-brain barrier.[24, 268, 294-295]

5) The mechanisms of headache response to smartphone electro-magnetic radiation (SER).

Smartphone output power, which is smartphone electromagnetic radiation, is a chemical pain stimuli, causing alteration of neurotransmitters in human body and resulting in slow pain,[32] as smartphone is an electromagnetic emission source closest to the head. Although the radiation is lower than the standards for maximum permissible

levels of exposure.[22-24] Meanwhile, the nervous system in human is non-equilibrium electrical organ controlled by electrochemical process and subjected to oscillation due to the electrical process and bio-electro-chemical interaction. The nervous system and the oscillatory activities can be interfered by the incoming radiation. The evidences of change in the intensity of electrical waves in brain exposed to SER[296] and that the electromagnetic energy with low intensity can stimulate or induce the functional alteration in the nervous systems[25-26] and affect the biological system of the brain. Lu and Huang suggested that the brain was like the antenna of electromagnetic waves which will be shaken or kneaded whenever there is signal transmission, and would cause of headache.[22] SER has been found to affect the areas of thalamas, raphe magnus nucleus, and spinal cord which decrease neurotransmitter such as acetylcholine, neuronal (nAChR), and has important role in inhibiting pain.[268] Furthermore, SER has been found to affect the function of dopamine-opiate system resulting in lower numbers of cells of the neurotransmitter, to the extent that it cannot perform the function of pain suppresser.[275] Moreover, use of smartphone will have thermal effects on the facial area and will thus enlarge the arteries that supply blood to the dura mater and stimulate the trigeminal nerves, which are responsible for sensation. All of these are the main mechanisms of migraine headache.[277] The response takes place in the nature of non-linearity with delayed effect in the form of a reverse dose-response. It is responsive to repeated exposures for a long period of time, cumulative effects and the adaptive process of human body that can cause allostatic load and the responses sensitive to the stressor.

The present study can be concluded that SOP which is smartphone electromagnetic radiation has a relationship with headache in the form of window effect. The result shows that migraine and undetermined headache response to SOP. The first stage of the early study found that undetermined headache is in the mobile phone associated headache (MPAH) group. The result has confirmed that SOP induces headache in MPAH group, which should be classified into a secondary headache, while migraine was triggered by SOP.

Objective 3: To study the correlation between smartphone output power and sleep quality among high school students

4.1.4 The correlation between smartphone output power and sleep quality among high school students

1) The prevalence of sleep quality

The prevalence of sleep quality in this stage is the object of repeated measures. The prevalence of sleep disorders is 32.1% with a mean sleep quality score of 3.66±2.07. The prevalence and mean score are lower than the study in Stage 1 (50.5% prevalence and 4.8±2.9 mean score). This is concurrent with a previous study conducted in 2008 (25-40%)[55] but lower than the prevalence found after 2008, which was 52.7% in Ethiopia[297] (2012), 66.1% in Australia[60] (2013), 39.6% in China[51] (2014), and 58.7% in Lebanon[53] (2016), respectively. Meanwhile, the prevalence of the problems of sleep difficulty and frequently waking are 8.2% and 29.7%, respectively. The result is different from the findings of the study in Stage 1 and concurs with the studies conducted in Norway (2014) where prevalence rates were 10% and 4%,[298] in China (2015) where the prevalence rates were 19% and 15.6%,[80] and America (2011) where the prevalence rates were 42% and 35%.[79] Moreover, the prevalence rates of sleep difficulty in Ethiopia[297] (2012) and Japan[81] (2006) were 36.7% and 27.9%, respectively. In the present study, the mean sleeping time is 7.4±1.7 hours with a prevalence of sleep loss (<8 hours) at 52.9%, whereas the mean sleep time is7.35±1.23 hours in Taiwan,[243] 7:22 ± 2:36 hours in Israel,[177] 7.28 ± 1.16 hours in Greece[57] and 7.39±1.23 hours in Lebanon.[53] At the same time, the prevalence of sleep loss (<7 hours) is 61% in the United States.[79] In the study, poor sleep hygiene is 52.1%, coffee drinking of 1–5 cups/day is 48.3%, and smartphone use before sleep was 27.9%. These findings are lower than the findings in the previous study where MP use before sleep was 23.6-62%.[64, 82] The findings concur with the study on the factors related to sleep quality in Stage 1, which found smartphone use before sleep to lead to sleep loss (OR_{adj}1.82: 95% CI: 1.10-3.01). Furthermore, smartphone use before going to sleep frequently for entertained conversations and social media use requires significant time and reduces sleep duration. The consequences are inefficient sleep and daytime sleepiness. In the present study, the prevalence of daytime sleepiness is as high as 73.4%, which concurs with the findings of other studies, 70% in Greece,[57] 22% in America[79] and 90.4% in

China.[51] Daytime sleepiness resulted in a 48.3% increase in students' tea or coffee intake.

2) Factors correlated with the sleep quality components

According to the findings, anxiety and depression create risks for sleep loss (OR_{adj}1.04-1.06; 95% CI: 1.02-1.1), and potentially poor sleep (OR_{adj}1.05-1.06; 95% CI: 1.01-1.1), while anxiety creates risks for morning sleepiness, (OR_{adj}1.06; 95% CI: 1.03-1.1) . Previous studies have found anxiety and depression to be the common co-morbidities encountered with sleep problems in adolescents quantitatively or qualitatively. The study by Augner found quality of sleep correlate to depression r=-0.57 and anxiety r=-0.54, p<0.01.[74] Furthermore, stress[299] and depression create risks concerning sleep quality (OR 2.47-3.90; 95% CI: 1.88-8.06).[51, 74] This is a frequently encountered, as a result of relationships with parents, family problems, and relationships with teachers and school, in addition to abandonment, anxiety, and loneliness (OR 2.52, CI: 1.15- 5.49151).[51] Depressed patients also experience to sleep difficulty through insomnia at a rate of 90%,[75] while adolescents display symptoms of daytime sleepiness will reduce sleep quality into five and two times more likely to be at risk for stress and anxiety, respectively. Meanwhile, adolescents experience physical changes resulting from growth that leads to susceptibility to depression. The thalamocortical circuit is triggered, in addition to the serotonergic, noradrenergic, cholinergic and GABAnergic systems. As a result, the sleep regulation system and electrical brain waves are disrupted through the supression of the sleep spindle before the occurrence of depression.[300] Furthermore, depression has been found to be linked to the circadian rhythm, which played a role in mood regulation, led to delay circadian phase and sleep impacts. Hence, the better sleep quality is, the improvement of mood issues are.[64] Additionally, abnormal mood regulatory system within the brain is also linked with increased REM sleep.[301]

Headache is correlated to sleep loss (OR_{adj}1.2; 95% CI: 1.03-1.3) and risks for sleep problems (OR_{adj}1.1; 95% CI: 1.0-1.3) and potentially leading to poor sleep (OR_{adj}1.2; 95% CI: 1.1-1.4). Headaches have been found to disrupt sleep. Although sleep can help to relieve headaches, sleep problems and trigger headaches.[42] According to the findings, migraines increase insomnia risk by 3.5 times, compare to TTH.[142]

Hence, headaches and sleep share a reciprocal connection. Previous studies have found that adolescents with headaches have lower sleep qualities[260] with higher mean of PSQI scores among migraines groups.[262] Furthermore, sleep quality has been the factor closely linked to the frequency of headaches.[262] Adolescents with headaches frequently experiences sleep difficulty, inefficient sleep, frequent nighttime awakenings, nightmares, and daytime fatigue. In terms of process, the hypothalamus, which is connected to the limbic system, retinohypothalamic tract, and brain-stem aminergic nuclei are triggered and migraines occur. Concurrently, the hypothalamus is connected to the periaqueductal gray (PAG) matter. It triggers orexin to result in "rapid-eye-movement sleep-off", while orexin triggers the ventrolateral part of the PAG matter, which suppresses anti nociceptive activity in the trigeminal nucleus caudalis and leads to migraines.[260] Hence, the abnormal function of the brain and biological mechanisms are correlated to sleep and migraines.[260] Furthermore, the neurotransmitters changes; serotonin, dopamine, and melatonin occur,[260] and the anti-inflammatory system triggered by migraines will affect melatonin,[42] lead to reduce REM sleep,[42, 273] thereby cause abnormal sleep-wake rhythms and drowsiness while awake.[42,273]

Poor sleep hygiene has been found the correlation with poor sleep quality (OR_{adj}1.2: 95% CI: 1.03-1.3). Inappropriate sleep behaviors and sleep hygiene, in addition to increased arousal before sleep such as consumption of coffee or tea, use of modern technology such as smartphone, videogames, TVs, computers in the bedroom and irregular sleep time, affect bedtime and sleep quality[55, 308] as well as daytime dysfunction.[308] The findings of the study support that behavior modification to achieve regular good sleep behaviors, sleeping at regular times based on the circadian rhythm, sleeping without hunger, consumption of non-caffeine or non-alcohol, and reduced anxiety, arousal, and fear. Enhancing sleep comfort and achieving good sleep hygiene can lead to good quality of sleep and morning learning ability.[308-310] Age (low age) has been found to be correlated with poor sleep quality (OR_{adj}1.2: 95% CI:1.0-1.3). Most studies found age correlates with sleep. In other words, a one-year increase in age decreases nighttime sleep by an average of 14 minutes. The development of adolescents causes changes in sleep structure, especially in the current era of technology. Older adolescents go to bed and wake up later times with an increased likelihood for engaging in activities at night, thus becoming classified as "owls". Moreover, the aforementioned

adolescents have regular nighttime study and reading obligations increased in social and entertainment activities. At this stage, parents pay less attention to the sleep of adolescents,[52, 62, 80, 311] causing adolescents response to sleep difficulty, inefficient sleep, poor sleep quality, and drowsiness.[80, 312] The prevalence of insomnia varies by age group.[55] People whose ages between 9-17 years have reduced slow wave (delta) in REM sleep. People whose ages between 11-12 years have decreased slow wave (delta) in REM sleep by 66%. Furthermore, female adolescents have developed quickly during adolescents' periods. As a result, a correlation between low age and reduced sleep quality was in the majority of female subject to the sample group.[312] As in the present study, the majority of the sample group is composed of females. Younger students have experienced more sleeping problems. The findings differ from most studies. One study on students with a mean age of 17.4 years who use smartphone output power in the range of $\geq 2.0 \times 10^{-5}$mW compared with students with a mean age of 17.9 years who used smartphone output power in range of $1.8\text{-}1.99 \times 10^{-5}$mW ($p<0.01$), found that younger students have increasingly used smartphone. Increasing in using smartphone leads to have higher exposure to electromagnetic radiation and screen light from smartphone. High exposure of smartphone light screen in the evening can shorten the circadian cycle due to the decreased light sensitivity resulted from melatonin secretion. This leads to disruption of the circadian rhythm or sleeps regulatory system, and eventually resulted in sleep difficulty. According to the findings, 150-500 lux of light from 11:00 p.m.-0:00 a.m. and 3:00-4:00 a.m. suppress melatonin response.[52]

Body mass index (BMI) was found to be correlated with poor sleep (OR_{adj}2.1: 95% CI: 1.4-3.2). Pathological studies and experiments conducted in the past found that reduced sleep time and quality is related to obesity.[72] A study by Penn State found that obesity decreases sleep quality and causes stress by 47%, leading to ineffective sleep (OR 1.7, CI 1.5-1.8).[189-190] According to the findings, high BMI is correlated with three times high riskier than less than eight-hours sleep in adolescent men.[64] Obesity is frequently associated with respiratory system abnormalities due to increased chest and abdominal mass, with decreased flexibility of the chest wall, resulting in suppressed diaphragm function. Obese people have been found to have increase in sleep latency and decreased sleep effectiveness.[189] Sleep loss and inadequate sleep changes the body's metabolism and endocrine system, increasing cortisol concentration in the evening

along with raised level of ghrelin and reduced level of leptin,[72, 313] in addition to the increased stimulation of the sympathetic nervous system. As a result, the neurological hormone related to hunger and fullness became stimulated leading to increase hunger and reduce fullness. Increased in HOMA-IR, an insulin resistant occur,[314] leads to increase in eating and decrease exercise. The study has been found an inverse dose-response relationship,[64, 315] that one hour of less sleep increases waistline by up to 80%. Nevertheless, the present study has been found the quality of sleep is the mediate impacts caused by the use of modern technology on obesity. Hence, appropriate use of technology during adolescence can increase sleep quality and reduce obesity.[317]

In the present study, internet use has been correlated with sleep difficulty (OR_{adj}1.2: 95% CI: 1.0-1.5) and non-use of hand-free devices correlated with sleep difficulty and sleep problems (OR_{adj}1.3: 95% CI: 1.1-1.6) and (OR_{adj}1.2: 95% CI: 1.01-1.4). Adolescents have used smartphone in the evening and at night, around 9:00 p.m., up to 62-72% overall.[237] Of these, 34-55% involved testing, chatting, and playing electronic media and 24% involved playing computer games.[41] A strong correlation of sleep disturbance led to sleep difficulty and reduced sleep time,[64] by a mean of 5.43 hours, and daytime napping, by a mean of two hours.[237, 318-319] Students have been found their needs to carry smartphone at all times,[40] Furthermore, smartphone and internet use from 0:00–3:00 a.m. has increased the risks of daytime fatigue and drowsiness up to 4 times,[2 37, 2 38] while internet addiction increases the risk by five times[84] (95% CI: 2.7-10.2). In chatting, smartphone are frequently placed against the head. Consequently, the brain is the most frequently exposed to electromagnetic radiation, resulting in the brain activity and function impacts. The electromagnetic radiation from smartphone can disrupt brain function, particularly brain waves and sleep structure. This behavior can increase in brain wave frequency in the sleep spindle with reduced REM sleep. This generally occurs temporarily and not throughout the entire night. Short-term contact, however, is sufficiently effective to trigger changes in brain function during the early stages of sleep.[219] Response to sleep brain wave changes has also found to have a dose-response correlation with the level of electromagnetic radiation exposure.[248] Furthermore, the talk mode has caused starting time of sleep to occur later than other modes and led to brain wave frequency in the frontal region ranging from 1-4 Hz, which indicates a higher brain wave at the start of sleeping time. As a result, the power of

electromagnetic radiation in talking mode might be higher than standby mode by up to nine times.[265] This concurs with the findings of the present study stating that non-use of hand-free devices creates risk for difficult sleep and frequently waking up at night. The use of hand-free devices must be combined with chatting in smartphone, result in increased distance between the smartphone and the head during conversations, in order to reduce exposure to electromagnetic radiation. Hence, the findings confirm that use of hand-free device is correlated with sleep. Additionally, smartphone use for text, entertainment, or conversations at night disrupt the function of the parts of the nervous system which is responsible for regulating waking and sleeping. Melatonin suppression has affected the circadian rhythm with reduced REM sleep.[64, 212] As a result, it is increased risk for sleep difficulty, frequently waking during night, and reduced sleep time and quality.[81, 237]

3) Factors correlated with daytime sleepiness

Daytime sleepiness is an assessment of impacts resulting from sleep quality problems which is hard to accurately assess. Daytime sleepiness is correlated with the increased level of the mediate involved in inflammation and increased cytokines, a medium that regulates sleep and affects drowsiness and fatigue.

Poor sleep hygiene has potentially caused daytime sleepiness (OR_{adj}1.2: 95% CI: 1.03-1.4). According to the findings, poor sleep hygiene involving sleeping late, drinking coffee and smoking before bedtime, and napping during the evening on a regular basis, can cause frequent waking up. This can lead to inefficient sleep and reduced sleep time and sleep quality. It is also correlated with daytime sleepiness.[310, 320] A correlation was found between sleep hygiene and sleep quality (r=0.40-0.05, p<0.01).[321] Napping in the evening can result in the ability to perform activities at night without feeling drowsy leading to reduced sleep time at night. A study by Dewald and colleagues found that inadequate sleep, reduced sleep quality, and drowsiness could result from disturbances to the pre-frontal cortex function, which played a part in regulating sleep.[322]

Anxiety was found to be correlated with daytime sleepiness (OR_{adj} 1.1: 95% CI: 1.01-1.1) to a very small degree. Nevertheless, anxiety has affected the amount and the

quality of sleep, as well as causing nightmares, which is a cause of suicidal idea.[323-325] Triggered biological systems associated with stress can cause fatigue.[324] Furthermore, daytime sleepiness is frequently encountered in evening cronotypes[326] and highly depressed individuals.[327] Correlations are frequently found between depression and increased sleepiness at younger ages. According to the findings, depression is a metabolic factor more frequently associated with sleepiness than sleep disturbance and poor sleep quality.[328] Most studies found sleep quality problems associated with daytime sleepiness. Corresponding to the present study, the result found PSQI has correlated to daytime sleepiness (OR_{adj}1.3: 95% CI: 1.3-1.4). The study on factors related to sleep quality in Stage 1 found PSQI to create risk for daytime sleepiness with the highest degree of correlation (OR_{adj}9.0: 95% CI: 2.7-29.9). Adolescents with sleep quality problems, whether insomnia, inadequate sleep (<6.5 hours), inefficient sleep (70%), or poor sleep quality have correlations with daytime sleepiness, emotional response problems, behaviors, and learning abilities.[306, 312, 329-331] Nighttime sleep schedule changes are also related.[331] Adolescents enjoy the "night owl" lifestyle and have increased engagement in nighttime activities because adolescents are experiencing development that causes biological changes involving sleep-way processes regulated by the internal clock and secretion of hormones for maintaining sleep balance. As a result, their circadian rhythms delayed lead to reduced sleep time at night and poor sleep quality.[306]

Caffeine has been found to be correlated with sleepiness (OR_{adj}1.8: 95% CI: 1.4-2.2). Caffeine is also associated with frequently waking and sleep disturbance; it can also lead to poor sleep quality, which causes daytime sleepiness. Caffeine disrupts adenosine function along with the parts of the nervous system that promote sleep. This results in sleep disturbance,[306] suppressed slow wave and REM sleep, and reduce sleep in stages 3 and 4. Furthermore, the findings demonstrate how caffeine suppresses the function of adenosine regulating the sleep balance process[54, 303] and reduces melatonin in the urine. This reduces sleep quality and causes daytime sleepiness.[307] However, discontinuation of regular caffeine intake has led to caffeine shortage symptoms, even in regular small doses discontinuation for short periods and led to sleepiness.[307]

4) Smartphone output power (SOP) and sleep quality

In the present study, SOP use in the evening and nocturnally is pre-bedtime use and usage during sleep time. While use during daytime and morning is Lag_1 and Lag_2 at 6-hour lags (or delayed effect) and daily lags (24 hours). The study has found response in sleep quality in each domain correlate with SOP in the forms of power effect and dose-response. Sleep difficulty, inefficient sleep, and morning sleepiness patterns have power effect response to SOP (≤1.79 and 1.80-1.99 $x10^{-5}$mW). Inefficient sleep is a calculation of the total sleep time and duration on the bed. Therefore, there are responses to smartphone output power in two ranging (≤1.79 and ≥ 2.0 $x10^{-5}$mW). Meanwhile, sleep loss and sleep problems have responded to smartphone output power, periodically ranging to the highest value of SOP (≥ 2.0$x10^{-5}$mW). Poor sleep is overall sleep quality and calculation of the sum of every sleeping domain. Therefore, there are responses to smartphone output power in power effect and the highest value range of SOP. This result has confirmed that increased and longer SOP use result in reduced sleep time. The majority of systematic review findings indicated that the use of smartphone and new technology before sleep cause late sleeping time. Adolescents have used smartphone in the evening and at night after the lights are turned off at 9:00 p.m. up to 62-72%.[237] Furthermore, smartphone and Internet use from 0:00-3:00 a.m.[237] has been found to be as high as 34-55%, consist of text messages, chatting, and 24% online activities like video games.[82] These are correlated with sleep disturbance (OR1.79; 95% CI:1.39-2.31) and reduce sleep time and quality (OR 1.53; 95% CI: 1.11-2.10).[64, 317,332-334] In the present study, the mean sleeping time is 7.4±1.7 hours with the presence of sleep loss (<8 hour) at 52.9%. Similarly, sleep loss (<7 hours) in America is 61%,[79] while mean sleeping time is 7.35±1.23 hours in Taiwan[243] and 7.28±1.17 hours in Greece.[57] Furthermore, the study findings at Stage 1 has found the prevalence of sleep loss to be 67.7% with a mean sleep time of 6.88±1.28 hours. The data indicates a decreasing trend of mean sleeping time. Hence, frequently waking and insufficient sleep time frequently cause sleepiness during the waking times. Sleep loss, sleep problems, and morning sleepiness respond to SOP use in the group with the greatest number by dose-response.

The study findings show that SOP creates risks for sleep difficulty and inefficient sleep, morning sleepiness, as well as poor sleep in the form of power effect. The information provided has indicated that SOP influences the parts of the nervous system that are responsible for sleep regulation. Similar to many studies, smartphone use increases the risk for sleep difficulty by 2.85 times (95% CI: 1.58-5.13). [64, 81, 335-336] Moreover, the findings concur with a previous laboratory study finding which states that electromagnetic radiation can alter brain waves as measured by EEG. The information clearly illustrates that brain cells have unstable electrical characteristics and function to regulate and control physical functions.[26, 265] The result can also be measured by electrical waves sorted in frequencies corresponding to the brain function characteristics. The majority of studies found increased brain wave response to occur in the alpha frequency.

Furthermore, previous laboratory studies have found increased intra-cortical stimulation and decreased suppression during exposure to MFR.[279] Subsequent to the first ten minutes of exposure, increased brain wave changes are induced at the alpha frequency from 11.5-12.25 Hz during the initial stage of non-REM sleep.[247-248, 337-341] Increased slow sleep spindles are found, while REM sleep time shortens.[246, 338-341] Reduced slow-wave sleep (SWS), which is deep sleep, occurs.[340] The increase in alpha brain wave is found in the temporal region exposed to electromagnetic radiation from smartphone the most. Furthermore, the alpha frequency in the frontal area decreases. The alpha frequency in the frontal region functions to maintain sleep.[342] The different responses of the brain regions to MFR have resulted from the uneven distribution of electromagnetic radiation on the brain.[278] Repeated exposures also produce the same results, after one hour of discontinued exposure, the physiological characteristics of the brain revert to their normal state.[279] However, no effect has been found on sleep structure.[288,340] Meanwhile, exposure of MFR for up to eight hours increases sleep latency and REM sleep latency without change, regardless of exposure size.[343] The findings concur with a study in lab rats that are exposed to MRF for a period of one month.[344]

Nevertheless, the time course of response changes depends on each study. Additionally, the response is a low degree which depends on the sensitivity of each

individual.[273] Furthermore, the study has been found that electromagnetic waves with frequencies similar to the brain wave frequencies increases the response in the brain.[344] Changes in brain waves during the sleep stage have dose-response correlations with the MFR power. A study by Regel and colleagues found brain wave power at spindle frequencies to increase by 7.7, 10 and 13.6% after exposed to electromagnetic radiation (SAR 10g) at 0.2, 1 and 5 W/kg.[248] This differs from the findings of the present study that found that prolonged sleep latency has power effect response to SOP (the range of $\leq 179 \times 10^{-5}$mW and $1.80\text{-}1.99 \times 10^{-5}$mW). The power might be consistent with brain waves. Similarly, a study conducted by Schmid and colleagues, which administered exposure of EMF at a frequency of 14 Hz, found increased spindle frequency in the NREM range, while EMF exposure at 217-Hz frequency revealed an increase but without statistical significance.[288] Additionally, a study by Regel and colleagues that administered exposure to MFR through GSM 900 signals with SAR10 g at 0.2 and 5 W/kg resulted in sleep duration of 19.4±2.4 min and 20.7±2.8 min without differences.[265]

Furthermore, on talking mode use, sleep latency was greater than the standby mode with increased brain wave power at a frequency of 1-4 Hz in the frontal region (1-4 Hz EEG frontal power).[265] This might be due to the fact that the electromagnetic strength is nine times higher than the standby mode.[265] Furthermore, a study conducted by Gogineni found talking mode to have 20 times higher output power (200mW) than the standby mode (20Mw).[20] Additionally, it is hypothesized that the talking mode is similar to electromagnetic low frequency at 8 Hz and 217 Hz frequencies, which can affect the onset of sleep. It is possible that sleep onset responds to the talking mode due to the 8 Hz frequency. However, the data remains unclear in humans.[265] The electromagnetic wave responds to exposure from smartphone electromagnetic radiation (SMR) stimulates the cortical neurons. This leads to the reticular nucleus of the thalamus in the subcortical region to emit signals to the cortex. The return signals of the corticothalamic then cause synchronization, which leads to the occurrence of sleep spindles. As a result, REM sleep decreases and brain wave power within a frequency range of 11.5-12.25 Hz increases.[247, 278, 338-339] Furthermore, MFR has been found to increase the regional cerebral blood flow in the dorsolateral prefrontal cortex, the same region exposed to MFR (ipsilateral). [246, 341] This alters the electrical characteristics of the

brain cells[2 48, 2 6 5, 3 45-346] causing more stimulation (excitable) with increased regional cerebral blood flow. Additionally, a study by Kesari and colleagues found exposure to 2.45-GHz of MFR to decrease calcium ion efflux from the pinealocytes, led to decreased melatonin. Numerous experimental studies have found that MFR affects the concentration of calcium ion, which has an important function in the nervous system and changes in neurotransmitters.[116]

Alpha brain wave is an indicator of the starting stage of sleep, while alpha brain wave in the frontal region functions in the process for maintaining sleep.[342] Therefore, response to increased alpha brain waves resulting from smartphone use provides confirmation to the present study that sleep difficulty and morning sleepiness have power effect response to SOP, while sleep loss have dose-response to SOP. This indicates that extended smartphone use before sleep decreases sleep duration. Furthermore, several previous studies conducted by Burch and colleagues, Wood and colleagues, and Jarupat and colleagues found groups that used telephones for more than 25 minutes per day to have decreased 6-hydroxymelatonin sulfate (6-OHMS), which wasthe metabolized melatonin and excreted into urine and saliva.[249-251] According to the findings, the secretion of melatonin from the pineal gland was sensitive to electromagnetic waves.[252]

Meanwhile, poor sleep has power effect response to SOP in two ranges (the range of $\geq$2.00 and 180-199x10^{-5}mW) because poor sleep is the sum of every domain regarding sleep quality and it can be resulted from exposure to the light of smartphone displays, which are light-emitting diodes (LED) backlit screens. These screens are lit in the back by short wavelength LEDs (460 nm). Hence, they are sensitive to photoreceptors and can stimulate the melanopsin in the retinal ganglion cells.[347] This transmits signals to the suprachiasmatic nuclei, which then transmits signals to the pineal gland in order to suppress the secretion of melatonin. Meanwhile, signals are emitted from the visual photoreceptor system to stimulate the ascending arousal system, resulting in increased wakefulness. Signals have sent to suppress the ventrolateral preoptic nucleus and the noradrenergic locus coeruleus systems.[348] This leads to disruption of the internal clock or sleep balance system due to delayed circadian rhythm, resulting in sleep difficulty. The study has been found that 150-500 lux of light from

11:00 p.m.-0:00 a.m. and 3:00-4:00 a.m. suppresses melatonin response and[52] lead to sleep difficulty.[11, 49] According to the findings, photoreceptors in the retinal ganglion cells are not sensitive to short wave lengths (short-wavelength~ 420 nm).[347] Hence, previous studies found that staring into the screen of an electronic device for a period ≥1 hour increased the risk of sleep difficulty by 3.4 times[349] and exposed to an LED backlit screen <100 lux for two to four hours suppresses melatonin secretion in the evening and affected the circadian rhythm, thereby led to reduce REM sleep.[64, 212] Furthermore, EEG is found at the frequencies of 1-7 Hz (slow-wave activity) in the frontal brain region, while NREM decreases. This results in sleep difficulty and sleep disturbance.[3 47, 350] Therefore, smartphone use, whether for texting, entertainment, or chatting at night, can disrupt the function of the parts of the nervous system that are responsible for regulating sleep-wake cycle, lead to higher risk of sleep difficulty, frequent waking up during night, and reduce sleep time and quality, eventually causing daytime sleepiness.[81,237] Furthermore, sleep quality in nearly every domain has been found to respond to lag (or delayed) effects of SOP. Heavily smartphone use in the morning from 6:00 a.m.-12:00 p.m. has resulted in delayed effects (6 hours) on sleep difficulty, sleep loss, sleep problems, morning sleepiness, and poor sleep quality. Daytime use of smartphone with an output power (12:00-6:00 p.m.) ranging from 1.80-1.99x10^{-5}mW resulted in delayed effects on poor sleep quality. On the other hand, daily dose of SOP has delayed effects (24 hours) on sleep difficulty, sleep loss, and poor sleep (Lag 6, Lag 4 and Lag 5, respectively) in the form of a reverse dose-response. The findings of the present study are consistent with the study by Lowden and colleagues in which an experiment was conducted to administer repeated MFR exposures for periods of four hours. The study found increased in brain wave activity at the frequencies of 0.5-1.5 and 5.75-10.5 Hz (delta, theta and alpha frequency bands) at 30 minutes, one hour and two hours of sleep in Stage 2. That no change was found on the power of electrical brain waves during slow wave sleep or in the third hour of sleep in stage 2. This shows that prolonged MFR exposure has acute and continuous impacts, involving electrical brain waves in the alpha frequencies.[273, 340] It might not immediately disappear after the smartphone is turned off or discontinuing the exposure. Instead, the changes gradually subside and disappear within ten minutes.[351] Moreover, a study in lab rats has exposed to MFR through GSM signals from smartphone with SAR values of 0.12, 1.2,

12 and 120mW/kg for two hours found different levels of albumin to leak out from the arteries in the rats at two hours, seven days, and fourteen days after recovery at the rate of 50%, 25% and 29%, respectively. The increasing of leakage of albumin at 14th days might be resulted from new openings in the blood brain barrier[352] as U-curve response or delayed effects of daily dose of SOP to sleep quality in the form of a reverse-dose response. Furthermore, the findings concur with a study conducted by Berneret and colleagues that found changes in protein synthesis from MFR exposure with SAR value of 2mW/kg for eight hours. Which no change was found from exposure at 30 minutes and one hour. However, changes encountered could revert to normal after two hours of exposure. The changes occurred due to prolonged exposure.[353]

Therefore, brain system response to MFR exposure might be the result of simultaneous stimulation and suppression. The study by Noor and colleagues found low-level of MFR exposure through smartphone to trigger stimulation and suppression of amino acids in the cerebellum of adult and young lab rats, after one hour of exposure. Additionally, in young animals, neurochemical suppression have been found in one month exposure but reverted to normal after one to two months after exposure, with increasing of stimulation by the fourth months after exposure. The adult animals have fluctuated changes throughout the entire experiment which reverted to normal after two to four months after exposure, followed by suppression of neurochemicals by the fourth months after exposure. Thus, both stimulation and suppression have affected brain system response to a MFR exposure at the same time. The stimulation in one system might suppress another system and that short-term exposure might cause both acute and long-term effects with a period of recovery to normal due to the adjustment of the brain.[354] The aforementioned data indicates that the nervous system can adapt and re-balance itself after exposure to MFR. However, repeated exposure eventually leads to stress that causes the balancing process to fail. This can lead to abnormalities in the system.[355]

5) The mechanism of sleep response to smartphone electromagnetic radiation (SER).

Smartphone electromagnetic radiation disturbs the cortical neurons causing the reticular nucleus of the thalamus in the sub cortical region to send signals to the cortex

lead to changes in electrical brain waves. As a result, the brain wave power in the alpha frequency band and sleep spindle in the non-REM sleep stage increase, while slow-wave sleep, which is deep sleep and REM sleep, decrease. This leads to sleep difficulty and frequently waking. At the same time, adolescence is in a stage of growth with physiological and brain changes. This causes brain waves in the alpha (8-12 Hz) and beta bands (13-30 Hz) to increase with oscillation of delta (0–3 Hz) and theta bands (4-7 Hz) to decrease according to changes in brain wave synchronization.[356] Hence, adolescents have increased sensitivity to electrical brain wave response to SER exposure.

Adolescents live in the digital age,[76] who aged group ranks in the top three of the highest possession and use of technology[11] and frequently spends time at night. As a result, adolescents are exposed to SER and smartphone screen lighting, which is short wavelength light (450 nm) that sends signals to the suprachiasmatic nuclei. Signals are then transmitted to the pineal gland to suppress the secretion of melatonin. Meanwhile, signals are transmitted from the visual photoreceptor system to stimulate the ascending arousal system in order to increase wakefulness. While signals are sent to suppress the ventrolateral preoptic nucleus system and the noradrenergic locus coeruleus[348] in order to prevent sleep. Adolescents have growth and biological changes in the sleep-wake process controlled by the internal clock and hormone secretion for a balance sleep. As a result, the circadian rhythm is delayed in adolescence and adolescents enjoy spending time at night as “night owls” in increased nighttime activities.[306] In addition to extensive use, there is potential impact from sensitivity to exposure to smartphone screen light, resulting in sleep quality problems.

The previous study result showed the environmental variables influences on headache, for example weather and smog.[357, 358] The study has not been measured environmental variable, which is the limitation of the study. However, the study is a panel study, meaning the outcomes and exposures are followed in the same sample group. The same samples in same environment, considered as controlling individual and environmental confounder.

This study has found the curious result of slight SOP value in the range of $\leq$1.79, 1.8-1.99x10^{-5}mW. Also, the study has also been found the correlation with the nervous system that concurred with the average power consumption of a human cell, at

$1x10^{-9}$mW. While the human is exposed to the cosmic microwave background radiation of $3x10^{-3}$mW from an early stage of the universe in Big Bang cosmology.[359] The value of SOP in this study, which can induce the nervous system, might be the trend of low output power and not the exact value. Furthermore, the result might be the response of the nervous system to the electromagnetic frequency. It should be an interesting study in the future.

Conclusion

Smartphone output power, which is smartphone electromagnetic radiation, has a non-linear correlation with neurological impacts and the non-linear response is a close connection between environmental and living factors.[111] The smartphone output power has affected to headache, sleep difficulty, inefficient sleep, morning sleepiness, and poor sleep quality in the range of ≤1.79 and 1.8-1.99$x10^{-5}$mW which has been called the window effect responses. The result shows that migraine and undetermined headache response to SOP. In the first stage, undetermined headache is in the same group with mobile phone associated headache. The result has confirmed that SOP has induced headache in MPAH group which should be classified into secondary headache. The result has also found headache response to delayed effect of SOP in the form of dose-response except migraine that's with a specific response. Additionally, smartphone electromagnetic radiation has effects that fit the criteria for triggers that induce headaches, especially migraines. However, nervous system has accumulated and delayed effect response on repeated exposure which can recovery and re-balance itself after exposure to MFR, but data of time to recovery was unclear. Finally, the result shows the trend of low output power that correlated with the nervous system.

4.2 Limitation and strength of the study

4.2.1 The Limitation of the study:

1) The study of headache and quality of sleep has not been conducted by a diagnosis of a doctor, but conducted by diagnosing headache and divided into each type of headache, so the disadvantage of the study is lacking of answer's accuracy. The researcher dealt with the problems by defining the importance of the represented subjects and how they should give accurate answers, so that the study can be used for

the future study all over the country or even all over the world. The researcher has also explained the meaning of each question in order to provide the accurate answers related to the objective of the study. When the researcher has entered the second phase, LINE application has been used to collect data. LINE group has been created in one group for one classroom so that the results of daily track records have easily been kept. The researcher has also met the subjects twice a week, then every 30 days, for paying the subjects and encourages the subjects to continue collecting daily data.

2) A potential limitation of the study is the unequal distribution of males and females.

3) When entered second phase, the subjects have final examinations and summer holidays and some students had problems of recording data. In this phase, because of the reasons mentioned above, 55 of the subjects were excluded from the study. The 21 of the excluded students were in grade 12, 15 were in grade 11, and 19 were in grade 10. The remaining subjects were lengthened their data collecting time as a result. Some of the subjects have less than 60 days data, and some have more than 60 days data. The remaining subject was 145 people and 12,696 data for analyzing in total.

4) The smartphone's output power data has not been collected properly as power intensity and absorption rate, which is different from other studies. The researcher found the window reaction or non-linearity relationship prior to the study conducted by Panagopoulos et al. The study explained that the specific absorption rate calculated was in the linear relationship.

5) Measuring smartphone's output power has been researched by using the data in the smartphone, not measuring the electromagnetic radiation from outside, so it would not be interfered by outside exposure, but in reality the subjects always exposed to the outside electromagnetic radiation which can lead to misclassification of exposure. Therefore, the researcher will concern the exposure of electromagnetic radiation from outside and inside the smartphone or in the environment should be used in the future study.

4.2.2 The strength of the study:

1) The study of the relationship between exposure of smartphone's output power and headache is the pioneer study in human, which is performed in an observational

study, meaning the researcher has studied the environment factor as it really is. The study has been designed in prospective time series which can identify the cause and the effect of each other. Moreover, time series can limit the confounder which 12,696 data required 12,696 subjects and a lot of confounder will be found and hard to control. In the study, the researcher has only used 145 subjects, so the researcher would have better control of confounder and avoid recall bias by collecting data day by day.

2) The study has been a panel study, meaning the outcomes and exposures have been followed in the same sample group. The same samples in same environment have considered as controlling individual and environmental confounder.

3) The study has large sample size, which possibly made an analysis even the effect of slight SOP in the range of $\leq$1.79, 1.8-1.99 $x10^{-5}$mW on nervous system.

4) The sampling subjects in the study have included every classroom in each year, so the ratio of each classroom of all boys and girls will be calculated to achieve the objective of study.

5) The tools used in the study have relied on the technology, by creating an application which is used to answer the questions and collect data. The input data from the subjects can be checked anytime, so the subjects who do not fill in the information can be tracked down easily. The researcher also conveniently communicates to every subject through LINE group. An application was created in order to measure smartphone's output power, by using the information collected by the smartphone itself. The application was designed to open, save, and send data through email, then clear the data from its own memory in order to start collecting new data. The email is specific design for each person, so all the subjects have to give the information by clicking the email button. Moreover, the application used for answering and collecting data will decrease the mistakes which can occur in the analysis phase.

6) The data that the researcher got is the data about everyday sleep. Every parameter of sleep has been collected so the information was the truth. Plus data of output power collected by the smartphone itself, as a result the researcher can assure that there is no bias from the subjects.

4.3 Suggestion

4.3.1 How to use smartphone safely?

1) From the study, the researcher found that talking on smartphone with no hand-free device is more likely to be a risk factor for headache and sleep problem because smartphone is a source of electromagnetic energy used closed to the head. So the safe way to use a smartphone is to position it far away from our head.

2) Hand-free, which comes with every purchased phone, should clearly suggest the way to use it together with the smartphone, in order to achieve highest safety.

3) When the phone is connected to the other end is the time that phone creates highest amount of radiation, so using speaker and not position the phone onto our ear are ways to avoid harming ourselves.

4) Talking on the phone while driving, walking, or moving around means the subjects are we're leaving the area of one base station and move

5) Into another base station, which will increases the MFR. Talking on the phone during driving or moving also increases the risk of accident.

4.3.2 Health care system

1) The study has found that SOP is a trigger of migraine and associated with headache and sleep problem. Thus, the headache and sleep clinic should improve health care for the health problem about smartphone use of the patients, suggesting safety smartphone use, preventing chronic headache, and improving sleep quality.

2) The safety use of smartphone campaign and poster should be conducted in high school and university in order to prevent vulnerable group about health impact.

4.3.3 Policy advocacy

1) Currently, there is no epidemiological evidence which can guarantee the maximum radiation exposure level that is safe for smartphone use to prevent overexposure, but the limit of exposure usually conflict with the minimum energy required for smartphone to function normally. In the study, the researcher found trend of low output power (1.79, 1.88-1.99 $x10^{-5}$mW) which is lower than the standard, 2 . 0 W/Kg in 10 g of tissue, calculated by ICNIRP,[22, 86, 360] but it can disturb human nervous system and change the wave form of our brain. So safe usage of smartphone should be

clearly indicated. For example, use of smartphone in children under the age of 14,[361] the school should create a rule in order to limit the use of smartphone such as limit the area which Wi-Fi can be used or limited the period of time in which smartphone can be used. As mentioned above, we can limit smartphone use, promote smartphone use for academic purpose[362] and explain the disadvantages of using smartphone for a long time only for entertainment purpose.

2) Nowadays the telephone charges, Wi-Fi, or internet are facilitated as a package which cause children and adolescences to use the smartphone days and nights. So the charges for smartphone should be appropriately adjusted. Change of signaling should also be done by using medium or low antenna and increase the number of the antenna instead.

4.4 The future study

4.2.1 Further study should include measurement the frequency of electromagnetic and electromagnetic field in the area, so all of electromagnetic exposure can be taken into account.

4.2.2 In the study, the researcher did not include output power while using smartphone mode, period of time, and frequency into the study. If they were taken into account, suggested that the properly use of smartphone should be written, such as how long talking mode should be used or how long other modes should be used, so that it will not affect to the people's health.

4.2.3 Further study should include subjects with underlying diseases, because stimulation from electromagnetic can worsen the underlying disease.

REFERENCES

(1) Han Ei, C. The Effects of Mobile Phones in Social and Economic Development: The Case of Female Microentrepreneurs in Chennai, India. Dissertation of Phd Degree in Communication Arts and Sciences, Michigan State University. 2012.

(2) David O, J. A study of The Impact of Mobile Phones as Learning Tools for Youth in Southern Baptist Churches. Dissertation of Doctoral Degree in Church and Family Ministry, Southwestern Baptist Theological Seminary. 2012.

(3) Tangtermwong C. A Current Trend of a Social Network on Mobile phones. Journal of Liberal Arts And Social Science. 2013;2(1).

(4) Cormier M. Acquisition, Modalities of Usage, and Domestication of Mobile Phones in The Cultural Context of Haiti: The Case of High School Students. Thesis of Master Degree in Communication, Faculty of Arts, University of Ottawa. 2007.

(5) Chen Y-F. The Mobile Phone and Socialization: The Consequences of Mobile Phone use in Transitions from Family to School Life of U.S. College Students. Dissertation for Doctor of Philosophy Graduate Program in University of New Jersey. 2007.

(6) Huiqi Z. Socioscope: Human Relationship and Behavior Analysis in Mobile Social Networks. Dissertations of Doctoral Degree in University of North Texas. 2010.

(7) Canalys. Mobile Device Market to Reach 2.6 Billion Units by 2016, Website: *www.canalys.com; Press release 2013,* 25 June 2015.

(8) Pansilpakom N. Study of Strategic Position of Electrocal and Electric Industrial. Institute of Electrocal and Electric. Office of Industrial Economic. Ministry of Industry.2014.

(9) Global Mobile Statistics 2014 Section F: M-Commerce, M-Payment, NFC, M-Ticketing, M-Coupons, M-Gambling and M-Entertainment, Website: *Mobithinking.com 2014*, 2014.

(10) National Science and Technology Development Agency. Thailand ICT Market 2010 and Outlook 2011. 2010.

(11) Electronic Ttransectors Development Agency. Thailand Internet User Profile 2013. 2013.

(12) Manager 360° magazine week. Thai Teenager was The First Ranked in Asia for The Length of Time Talking and Social Network, website:*http://wwwmanagercoth/mgrWeekly/ViewNewsaspx?2010*, April 2015.

(13) Madhow U. Introduction to Communication Systems. University of California, Santa Barbara. 2014.

(14) Stüber G L. Principles of Mobile Communication. Edition S, editor. New York, Boston, Dordrecht, London, Moscow: Kluwer Academic Publishers; 2002.

(15) Rodthong S. Behavioral Intention in Downloading Mobile Application among Smartphone Users. 2013.

(16) Prakobtham B. Smart Phone Trend in 2010. Executive Journal.2010;8(17):197-202. Website: *http://wwwbuacth/knowledgecenter/executive_journal /302/ pdf/aw33pdf*.

(17) Hillert L, Ahlbom A, Neasham D et al. Call-Related Factors Influencing Output Power from Mobile Phones. J Expo Sci Environ Epidemiol. 2006; 16 (6): 507-14.

(18) d'Amore G, Anglesio L, Alberto et al. Human Exposure Evaluation to Mobile Phone Electromagnetic Emissions. The Regional Committee for Communication in Piedmont (CoReCom Piemonte).

(19) Kelsh M A., Shum M, Sheppard A R., et al. Measured Radiofrequency Exposure During Various Mobile-phoneuse Scenarios. J Expo Sci Environ Epidemiol. 2011;21:343-54.

(20) Gogneni S. Computational Study of Electromagnetic Wave Effect Induced by Mobile Phones on Brain Tissues and Its Biological Implicztions. Master of Science in Mechanical Engineering. The University of Texas at San Antonio. 2012.

(21) Hyland, G J. Physics and Biology of Mobile Telephony. The Lance. 2000; 356: 1833.

(22) Lu Y, Huang Y. Biological Effects of Mobile Phone Radiation. Microwave and Millimeter Wave Technology (ICMMT), International Conference on 5-8 May 2012. IEEE.2012;2.doi: 10.1109/ICMMT.2012.6230101.

(23) Sudan M. Cell Phone Exposures and Headaches, Hearing Loss, and Behavioral Problems in Children. Dissertation for Doctor of Philosophy in Epidemiology, University of California. 2012.

(24) Shckorbatov Y. The Main Approaches of Studying the Mechanisms of Action of Artificial Electromagnetic Fields on Cell. J Electr Electron Syst Electromagnetic Wave Theory. 2014; 3(2).

(25) Frey A H. Headaches from Cellular Telephones: Are They Real and What Are The Implications? Environ Health Perspect.1998; 6(3): 101-3.

(26) Weinberger Z., Richter E. D. Cellular Telephones and Effects on The Brain: The Head as an Antenna and Brain Tissue as a Radio Receiver. Med Hypotheses. 2002:1-3.

(27) Cleary S F. Biological Effects and Health Implications of Microwave Radiation. Symposium Proceedings. Department of Biophysics, Virginia Commonwealth University. 1970.

(28) Szjlkowska A, Gadzicka E, Szymczak W et al. The Risk of Subjective Symptoms in Mobile Phone Users in Poland an Epidemiological Study. Int J Occup Med Environ Health. 2014; 27(2):293-303.

(29) Khan M M. Adverse Effects of Excessive Mobile Phone Use. Int J Occup Med Environ Health. 2008;21(4):289-93.

(30) Kumar P, Dongre K, Ghai G. Prevalence of Subjective Symptoms in Mobile Users Aged More than 18 Years in Nagpur. Indian J. Physiother. Occup.Therapy.2013;7(2) 263.

(31) Nathan N, Zeitzer J. A Survey Study of The Association between Mobile Phone Use and Daytime Sleepiness in California High School Students. BMC Public Health. 2013;13(840).

(32) Guyton A C. The Nervous System: A. General Principles and Sensory Physiology. Textbook of Medical physiology: Eleventh Edition. Elsevier Inc. Philadelphia, Pennsylvania. 2006.

(33) International Association for the Study of Pain. Gobal Year Against Headache: Epidemiology of Headache. Website: *file:///C:/Users/wanna%20occ/Desktop/1-Epidemiology%20(1)pdf 2012,* 2013.

(34) Lewis D W. Headaches in Children and Adolescents. Curr Probl Pediatr Adolesc Health Care. 2007.

(35) Jensen R, Stovner L J. Epidemiology and comorbidity of headache. Lancet Neurol.2008;7(4):295.

(36) Kathleen R, Merikangas P. Contributions of Epidemiology to Our Understanding of Migraine Review Articles. Headache. 2013;53:230-46.

(37) Wöber-Bingöl Ç. Epidemiology of Migraine and Headache in Children and Adolescents. Curr Pain Headache Rep. 2013;17(341). doi10.1007/s11916-013-0341-z.

(38) RhoY-I, Chung H-J, Lee K-H et al. Prevalence and Clinical Characteristics of Primary Headache samong School Children in South Korea: A Nationwide Survey. Headache. 2012;52: 592-99.

(39) Lucchetti G, Peres M. The Prevalence of Migraine and Probable Migraine ina Brazilian Favela: Results of a Community Surveyhead_1899 971. Headache. 2011;51:971-9.

(40) Lehmann S, Milde-Busch A, Straube A et al. How Specific are Risk Factors for Headache in Adolescents Results from a Cross-sectionalStudy in Germany. Neuropediatrics. 2013;44: 46-54.

(41) Lima AS, de Araújo RC, Gomes MR et al. Prevalence of Headache and Its Interference in The Activities of Daily Living in Female Adolescent Students. Rev Paul Pediatr. 2014;32(2):256-61.

(42) Bellini B, Arruda M, Cescut A et al. Headache and Comorbidity in Children and Adolescents. J Headache Pain. 2013;14:79.

(43) Chongchitpaisan W,Bandhukul A. The Characteristics of Headache and Mobile Phone Used in Secondary and High School Students in Rayong Province

Thailand, 2014. Asian Regional Conference for Headache: ARCH Chiangmai 2015. 2015.

(44) Casucci G., Terlizzi R., Cevoli S. et al. Headache in School Age. Neurol Sci. 2014;35(1):31-5.

(45) Allena T, Steiner T. J., Sances G. et al. Impact of Headache Disorders in Italy and The Public-Health and Policy Implications: Apopulation-Based Study Within The Eurolight Project. J Headache Pain. 2015;16:(100). doi:10.1186/s10194-015-0584-7.

(46) Fuh J-L, Wang S-J, Lu S-R et al. Headache Disability among Adolescents: A Student Population-Based Study. Headache. 2010;50:210-8.

(47) World Health Organization. Headache Disorders. Fact sheet N°277, Website: *http://www.who.int/mediacentre/factsheets/fs277/en/;2012*,Janyuary 2015.

(48) Schu J, Waldemar G, Olsen J H. et al. Risks for Central Nervous System Diseases among Mobile Phone Subscribers: A Danish Retrospective Cohort Study. PLoS ONE. 2009; 4(2):4389.

(49) Chu M K, Song H G, Kim C et al. Clinical Features of Headache Associated with Mobile Phone Use: A Cross-Sectional Study in University Students. BMC Neurology. 2011;11(115).

(50) Yang C-K, Kim J K., PatelS R et al. Age-Related Changes in Sleep/Wake Patterns among Korean Teenagers. Pediatrcs. 2005;115(1).

(51) Guo L, Deng J, He Y et al. Prevalence and Correlates of Sleep Disturbance and Depressive Symptoms among Chinese Adolescents: A Cross-Sectional Survey Study. BMJ. 2014;4.

(52) Colrain I M, Baker F C. Changes in Sleep as a Function of Adolescent Development. Neuropsychol Rev. 2011;21:5-21.

(53) Kabrita C S, Hajjar-Muça T A. Sex-Specific Sleep Patterns among University Students in Lebanon: Impact on Depression and Academic Performance. Nature and Science of Sleep. 2016;8:189–96.

(54) Hasan R, Aióe F. Updates on The Sleep-Wake Cycle. Sleep Sci 2011;4(2): 6-60.

(55) MindellJ A and MeltzerL J. Behavioural Sleep Disorders in Children and Adolescents. (Review Article) Ann Acad Med Singapore. 2008;37:722-8.

(56) Hongsahnguansri S. Sleep Disturbances in Children and Adolescents. Child and Adolescent Psychiatric Society of Thailand. 2005.

(57) Lazaratou H, Dikeos D G., Anagnostopoulos D C. et al. Sleep Problems in Adolescence: A study of Senior High School Students in Greece. Eur Child Adolesc Psychiatry. 2005;14(4):237–43.doi: 10.1007/s00787-005-0460-0.

(58) Pagel J F., Forister N, Kwiatkowki C. Adolescent Sleep Disturbance and School Performance: The Confounding Variable of Socioeconomics. Journal of Clinical Sleep Medicine. 2007;3(1).

(59) National Sleep Foundation. Sleep in America Poll 2006. 2006.

(60) Short M A., Gradisar M, Gill J et al. Identifying Adolescent Sleep Problems. PLOS ONE. 2013;8(9). doi:10.1371/journal.pone.0075301.

(61) Liu X, Liu L, Owens J A. et al. Sleep Patterns and Sleep Problems among School Children in the United States and China. Pediatrics. 2005;115 (Supplement 1).

(62) Ravi G, Manjeet S, Vishal C et al. Sleep Patterns of Urban School-Going Adolescents. Indian Pediatrics. 2008;45:183-9.

(63) Lai H. Neurological Effects of Non-Ionizing Electromagnetic Fields. The BioInitiative Working Group, USA. 2014.

(64) Owens J. Insufficient Sleep in Adolescents and Young Adults: An Update on Causes and Consequences. Pediatrics. 2014;134(3):921-32. doi:10.1542/peds.2014-1696.

(65) Gaultney J F. The Prevalence of Sleep Disorders in College Students: Impact on Academic Performance. J Am Coll Health . 2010;59(2).

(66) Azad M C, Fraser K, Rumana N et al. Sleep Disturbances among Medical Students: A Global Perspective. J Clin Sleep Med. 2015;11(1):69-74.

(67) Sage C, Carpenter D O. et al. Public Health Implications of Wireless Technologies. Pathophysiology. 2009;16(2-3):233-46.

(68) Roebuck A, Monasterio V, Gederi E et al. A Review of Signals Used in Sleep Analysis. Physiol Meas. 2014;35(1):1-57.

(69) Ram S, Seirawan H, Kumar K S. et al. Prevalence and Impact of Sleep Disorders and Sleep Habits in The United States. Sleep Breath. 2010; 14:(63).

(70) Ferrie J E, Kumari M, Salo M et al. Sleep Epidemiology- A rapidly Growing Field. Int J Epidemiol . 2011;40:1431-7doi:10.093/ije/dyr203.

(71) Kersted T., Nilsson P.M. Sleep as Restitution: An Introduction. J Intern Med. 2003;254:6-12.

(72) Beccutia G, Pannain S. Sleep and Obesity. Curr Opin Clin Nutr Metab Care. 2011;14(4):402-12.

(73) Cappuccio F P., D'Elia L, Strazzullo P et al. Sleep Duration and All-Cause Mortality: A Systematic Review and Meta-Analysis of Prospective Studies. Sleep. 2010;33(5):585-92.

(74) Augner C. Associations of Subjective Sleep Quality with Depression Score, Anxiety, Physical Symptoms and Sleep Onset Latency in Students. Cent Eur J Public Health 2011;19(2):115.

(75) Spiegelhalder K, Regen W, Nanovska S et al. Comorbid Sleep Disorders in Neuropsychiatric Disorders Across The Life Cycle. Current Psychiatry Reports. 2013;15(6):364.

(76) Prensky M. Digital Natives, Digital Immigrants. On the Horizon MCB University Press. 2001; 9(5).

(77) Buxton O M., Chang A-M, Spilsbury J C et al. Sleep in The Modern Family: Protective Family Routines for Child and Adolescent Sleep. Sleep Health. 2015;1(1):15-27.

(78) Landtblom A-M, Engström M. The Sleepy Teenager–Diagnostic Challenges. Frontiers in Neurology. 2014;5(140). doi:10.3389/fneur.2014.00140.

(79) National Sleep Foundation. Sleep in America Poll: Communication Technology in Bedroom. wwwSleepfoundationorg. 2011.

(80) Jiang X, Hardy L L., Baur L A. et al. Sleep Duration, Schedule and Quality among Urban Chinese Children and Adolescents: Associations with Routine After-School Activities. PLOS ONE.2015. doi:10.1371/journal.pone.0115326.

(81) Alexandru G , Michekazu S , Shimako H et al. Epidemiological Aspects of Self-Reported Sleep Onset Latency in Japanese Junior High School Children. J Sleep Res. 2006;15,:266-75.

(82) Bruni O, Sette S, Fontanesi L et al. Technology Use and Sleep Quality in Preadolescence and Adolescence. J Clin Sleep Med.2015;11(12):1433-41.

(83) Van den Bulck J. Television Viewing, Computer Game Playing, and Internet Use and Self-Reported Time to Bed and Time out of Bed in Secondary-School Children. Sleep. 2004;27,(1):101-4.

(84) Choi K, Son H, Park M et al. Internet Overuse and Excessive Daytime Sleepiness in Adolescents. Psychiatry Clin Neurosci. 2009;63:455-62.

(85) Martuzzi M, Tickner J A. The Precautionary Principle: Protecting Public Health, The Environment and The Future of our Children. World Health Organization. 2004.

(86) International Commission on Non-Ionizing Radiation Protection (ICNIRP). Guidelines on Limits of Exposure to Static Magnetic Fields. Health Physics. 2009; 96 (4).

(87) National Institute of Environmental Health Sciences. EMF Electric and Magnetic Fields Use of Electric Power. The National Institute of Environmental Health Sciences, Department of Energy (NIEHS/DOE) EMF Rapid Program. 2002.

(88) National Institute of Environmental Health Sciences (U.S.). Electric Magnetic Fields Associated with The Use of Electric Power EMF. 2002.

(89) International Commission on Non-Ionizing Radiation Protection (ICNIRP). ICNIRP Guidelines for Limiting Exposure to Time-Varying Electric, Magnetic and Electromagnetic Fields (Up to 300 GHz). Health Physics. 1998;74 (4):494-522.

(90) Robert F. Cleveland J, Jerry L. et al. OET Bulletin 56 4th Edition. Federal Communications Commission. 1999.

(91) Palachaipiromsin S. Usages Trend of Mobile Application. Executive Journal. http://wwwbuacth/knowledgecenter/executive_journal/oct_dec_11/pdf/aw018pdf ; 2010 [Cited 2014 July].

(92) Zhang X. Smart Phone Application- A New Path To Power: Long Island University 2013.

(93) Nath S D, Saha A K, Hossain A. Identification and Measurement of the Factors Affecting Satisfaction Level of Smart Phone Users: Empirical Evidence from Bangladesh. Int. J. Bus. Manag. 2015;10(4).

(94) Lee N. The 411: Feature Phones vs. Smartphones.Website : *http://www.cnet.com/news/the-411-feature-phones-vs-smartphones/: CNET; 2010*,2 July 2015.

(95) Noidonprai P. Data Communication and Network. 2013.

(96) Peasai T. Wireless Network System . Hand out subject wireless communication and network. website: *http://wwwstouacth/website/subbj/fileUpload/99313-9-1pdf*, 2015.

(97) Moulder et al. Cell Phones and Cancer: What Is the Evidence for a Connection. Radiat Res. 1999;151:513-31.

(98) Otto M, Mu¨hlendahl K E. Electromagnetic fields (EMF): Do They Play a Role in Children's Environmental Health (CEH). Int J Hyg Environ Health. 2007;210:635-44.

(99) Lonn S, Forssen U, Vecchia P et al. Output Power Levels from Mobile Phones in Different Geographical Areas; Implications for Exposure Assessment. J Occup Environ Med. 2004;61(9):769-72.

(100) Cardis E, Armstrong B K, Bowman J D et al. Risk of Brain Tumours in Relation to Estimated RF Dose from Mobile Phones: Results from Five Interphone Countries. J Occup Environ Med. 2011;68(9):631-40.

(101) Taki M, Wake K. Exposure Assessment and Dosimetry for Epidemiology on The Mobile Phone Use. IEEE. 2012.

(102) Institute of Electrical and Electronics Engineers (IEEE). IEEE Recommend Practice for Determinig the Peak Spatial-Avrage Specific Absorption Rate (SAR) in the Human Head from wireless comunication Device: Measurement Technique. 2013.

(103) The National Radiation protection Board (NRPB). Review of the Scientific Evidence for Limiting Exposure to Electromagnetic Field (0-300 GHzx. Document of thr NRPB. 2004;15(4).

(104) Hakim B M. Precise SAR Measurements in The Near–Field of RF Antenna Systems. Dissertation for Doctor of Philosophy in Faculty of the Graduate School of the University of Maryland. 2006.

(105) Nikolay T. Æ Gabriela L. A, Atanasova. An Investigation of The Effects from The User's Hand and Head over Received Level and Adaptive Power Control of a GSM Mobile Phone in Typical Operating Environment. Environmentalist. 2007;27:585-91.

(106) Wiart J, Dale C, Bosisio A V et al. Analysis of the Influence of the Power Control and Discontinuous Transmission on RF Exposure with GSM Mobile Phones. IEEE Transactions on Magnetics Compatibility 2000; 42(4).

(107) K¨uhn S, Kuster N. Field Evaluation of the Human Exposure From Multiband, Multisystem Mobile Phones. IEEE Transactions on Magnetics Compatibility. 2013;55(2).

(108) Vrijheid M, Mann S, Vecchia P et al. Determinants of Mobile Phone Output Power in a Multinational Study: Implications for Exposure Assessment. J Occup Environ Med. 2009;66:664-71.

(109) Gosselin M-C, K¨uhn S, Kuster N. Evaluation of the Output Power Control of Multi Communication System Mobile Phones. IEEE. 2010.

(110) Inyang I, Benke G, McKenzie R et al. Comparison of Measuring Instruments for Radiofrequency Radiation from Mobile Telephones in Epidemiological Studies: Implications for Exposure Assessment. J Expo Sci Environ Epidemiol.2008;18(2):134-41.

(111) Panagopoulos D J, Johansson O, Carlo G L. Evaluation of Specific Absorption Rate as a Dosimetric Quantity for Electromagnetic Fields Bioeffects. PloS ONE. 2013;8(6):62663.

(112) Karger C P. Mobile Phones and Health: A Literature Overview. Z Med Phys. 2005;15:73-85.

(113) Wessapan T, Rattanadecho P. Numerical Analysis of Specific Absorption Rate and Heat Transfer in Human Head Subjected to Mobile Phone Radiation: Effects of User Age and Radiated Power. J. Heat Transfer.2012;134.

(114) Okonigene R E. Sitting of GSM Base Station Antenna And Its Health Consequences. International Conference on Information Technology. 2010;613(8).

(115) Berg G, Schuz J, Samkange-Zeeb F et al. Assessment of Radio Frequency Exposure from Cellular Telephone Daily Use in an Epidemiological Study: German Validation Study of The International Case-Control Study of Cancers of The Brain—Interphone - Study. J Expo Sci Environ Epidemiol. 2005;15(3):217-24.

(116) Lai H. Neurological Effects of Radiofrequency Electromagnetic Radiation Relating to Wireless Communication Technology. wwwmapcruzincom. 1997.

(117) Cook C M. The Effects of a Weak, Extremely Low Frequency Pulsed Magnetic Fields Upon Human Brain Electrical Activity. The Dissertation for Doctor of Philosophy Graduate Program in Medical Biophysics Faculty of Graduate Studies The University of Western Ontario. 2005.

(118) Vittayakittipong M. Adult electroencephalography: Basic Knowledge for Nurses. Songkla Med J 2006;24(5):445-5.

(119) Liburdy R P, Vanek P F. Microwaves and The cell membrane. II. Temperature, Plasma, and Oxygen Mediate Microwave-Induced Membrane Permeability in The Erythrocyte. PubMed. 1985;102(2):190-205.

(120) Saroka K S. Quantitative Electroencephalographic Profiles of Individuals Exposed to Weak - Intensity Physiologically Patterned Electromagnetic Fields. Thesis for Master of Arts in Experimental Psychology, Laurentian University Sudbury, Ontario. 2011.

(121) Frey A H., Wesler L S. Dopamine Receptors and Microwave Energy Exposure. Electromagn Biol Med. 1983; 2(2-3):145-57.

(122) Andreas S, Florian H, Friedrich E et al. Headache in School Children: Prevalence and Risk Factors (Review Article). Dtsch Arztebl Int. 2013;110(48):811-18 doi: 10.3238/arztebl.2013.0811.

(123) Larsson B. Headache Prevalence and Characteristics among Adolescents in The General Population: Acomparison between Retrospect Questionnaire and Prospective Paper Diary Data. J Headache Pain2014;15:(80): doi http://www.thejournalofheadacheandpain.com/content/15/1/80.

(124) Wöber-Bingöl Ç, Wöber C, Uluduz D et al. The Global Burden of Headache in Children and Adolescents – Developing a Questionnaire and Methodology for a Global Study. J Headache Pain. 2014;15(86) doi: http://www.thejournalofheadacheandpain.com/content/15/1/86.

(125) Buse D C., Manack A N., Fanning K M. et al. Chronic Migraine Prevalence, Disability, and Sociodemographic Factors: Results From the American Migraine Prevalence and Prevention Study. Headache. 2012;52:1456-70 doi:10.111/j.526-4610.2012.02223.x.

(126) Vuković V, Plavec D, Pavelin S et al. Prevalence of Migraine, Probable Migraine and Tension-Type Headache in The Croatian Population. Neuroepidemiology. 2010;35:59-65.

(127) Mengistu G, Alemayehu S. Prevalence and Burden of Primary Headache Disorders among a Local Community in Addis Ababa, Ethiopia. J Headache Pain.2013;14:(30)doi:http://www.Thejournalofheadacheandpain.com/content/14/1/30.

(128) Steiner T J, StovnerL J, Katsarava Z et al. The Impact of Headache in Europe: Principal Results of The Eurolight Project. J Headache Pain. 2014;15(31).

(129) Stovner L, Hagen K, Jensen R et al. The Global Burden of Headache: A Documentation of Headache Prevalence and Disability Worldwide. Cephalalgia. 2007;27:193-210.

(130) World Health Organization. Atlas of Headache Disorders and Resources in The World 2011. 2011. 2011.

(131) Xavier MKA, Pitangui ACR, Silva GRR et al. Prevalence of Headache in Adolescents and Association with Use of Computer and Videogames. Ciência & Saúde Coletiva. 2015;20(11):3477-86,.

(132) El-Sherbiny N A., Masoud M, Shalaby M N. et al. Prevalence of Primary Headache Disorders in Fayoum Governorate, Egypt. J Headache Pain. 2015;16(85). doi:10.1186/s10194-015-0569-6.

(133) Ezeala-Adikaibe B A., Onyekonwu C, Okudo G. Prevalence of Primary Headaches in an Urban Slum in Enugu South East Nigeria: A Door-to-Door Survey. Headache. 2014;54:1601-10.

(134) Manandhar K, Risa A, Linde M et al. The Burden of Headache Disorders in Nepal: Estimates from a Population-Based Survey. The Journal of Headache and Pain. 2016);17(3). doi:10.1186/s10194-016-0594-0.

(135) Saab C Y. Chronic Pain and Brain Abnormalities. Elsevier Inc Elsevier 32 Jamestown Road, London NW1 7BY, UK. 2014.

(136) Koneru A. Endogenous Opioids: Their Physiological Role and Receptors. Global Journal of Pharmacology. 2009;3(3):149-53.

(137) International Headache Society (IHS). The International Classification of Headache Disorders, 3rd Edition (Beta Version). Headache Classification Committee of the International Headache Society. Cephalalgia. 2013;33(9).

(138) Pompili M, Di Cosimo D, Innamorati M et al. Psychiatric Comorbidity in Patients with Chronic Daily Headache and Migraine: A Selective Overview Including Personality Traits and Suicide Risk. J Headache Pain. 2009;10(4):283-90.

(139) Abu-Arafeh I, Macleod S. Serious Neurological Disorders in Children with Chronic Headache. Archives of Disease in Childhood. 2005;90(9):937-40.

(140) Walter S M. Lifestyle Behaviors and Illness Related Factors as Predictors of Recurrent Headache in U.S. Adolescents. Dissertation for Doctor of Philosophy in Nursing, West Virginia University. 2009.

(141) Lance J W., Goadsby P J. Machanism and Management of Headache 7th Edition. Butterworth-Heinemann: Oxford. 2005.

(142) Talebian A, Soltani B, Haji Rezaei M. Causes and Associated Factors of Headaches among 5 to 15-Year-old Children Referred to a Neurology Clinic in Kashan, Iran. Iran J Child Neurol. 2015; 9 (1).

(143) World Gastroenterology Organisation. Constipation. World Gastroenterology Organisation Practice Guidelines. 2007.

(144) Inaloo S, Dehghani S M, Hashemi S M et al. Comorbidity of Headache and Functional Constipation in Children: A Cross-Sectional Survey. Turk J Gastroenterol. 2014;25(3).

(145) O'Byrne K K. Psychosocial Factors in Pediatric Tension and Migraine Headache: A Meta-Analysis. Dissertation for Doctor of Philosophy in Counseling Psychology, Faculty of The University of Missouri-Kansas City. 2003.

(146) Martinez-Selles M, Datino T, Figueiras-Graillet L et al. Cardiovascular Safety of Anagrelide in Healthy Subjects: Effects of Caffeine and Food Intake on Pharmacokinetics and Adverse Reactions. Clinical Drug Investigation. 2013;33(1):45-54.

(147) Olesen J et al. Headaches 3rd Edition. Lippincott Williams and Wilkins. 2006:243-9.

(148) Varda MND, Gregoric A. A Diagnostic Approach for The Child with Hypertension. Pediatr Nephrol. 2005;20(4):499-506.

(149) Sacco S, Cerone D, Carolei A. Comorbid Neuropathologies in Migraine: An Update on Cerebrovascular and Cardiovascular Aspects. J Headache Pain. 2008;9(4):237-48.

(150) Halkes P H, Gijn J, Kappelle L J et al. European/Australasian Stroke Prevention in Reversible Ischaemia Trial Study G. Risk Indicators for Development of Headache During Dipyridamole Treatment after Cerebral Ischaemia of Arterial Origin. J Neurol Neurosurg Psychiatry. 2009;80(4): 437-9.

(151) Aslan G, Sade LE, Yetis B et al. Flow in The Left Anterior Descending Coronary Artery in Patients with Migraine Headache. Am. J. Cardiol. 2013;112(10):1540-4.

(152) Bini A, Evangelista A, Castellini P et al. Cardiac Cephalgia. J Headache Pain. 2009;10(1):3-9.

(153) Providencia R A. Headache and Cardiovascular Disease: Old Symptoms, New Proposals. Future Cardiol. 2010;6(5).

(154) Toldo I, Perissinotto E, Menegazzo F et al. Comorbidity between Headache and Epilepsy in a Pediatric Headache Center. J Headache Pain. 2010;11(3):235-40.

(155) Gameleira F T, Ataide L, Raposo M C. Relations between Epileptic Seizures and Headaches. Seizure. 2013;22(8):622-6.

(156) Shorvon S D, Rijckevorsel K van. A New Antiepileptic Drug. J Neurol Neurosurg Psychiatry. 2002;72:426-8.

(157) Gil-Gouveia R., Martins I.P. Headaches Associated with Refractive Errors: Myth or Reality. J Head and Face Pain. 2002;42(4):242-328.

(158) Marasini S, Khadka J, Sthapit PRK et al. Ocular Morbidity on Headache Ruled Out of Systemic Causes- A Prevalence Study Carried out at a Community Based Hospital in Nepal. J Optom. 2012;5(2):68-74.

(159) Lateef T M, Merikangas K R, He J et al. Headache in a National Sample of American Children: Prevalence and Comorbidity. J. Child Neurol. 2009;24(5):536-43.

(160) Bigal ME, Lipton RB. Modifiable Risk Factors for Migraine Progression. Headache. 2006;46(9):1334-43.

(161) Tienboon P. Body Mass Index in Pediatrics. Thai J Parenter Enteral Nutr. 2004;15(3).

(162) Siriratrekha T. Tourette's Disorder, Website: *http://wwwhappyhomeclinic*com/*sp07-tourettehtm.2008*, 2013.

(163) Du J-C, Chiu T-F, Lee K-M et al. Tourette Syndrome in Children: An Updated Review. Pediatr Neonatol. 2010;51(5):255-64.

(164) Stensland S O, Dyb G, Thoresen S et al. Potentially Traumatic Interpersonal Events, Psychological Distress and Recurrent Headache in a Populationbased Cohort of Adolescents: The HUNT Study. BMJ Open. 2013;3.

(165) World Health Organization. Diagnosis, Prevalence Estimation and Burden Measurement in Population Surveys of Headache: Presenting The HARDSHIP Questionnaire. J Headache Pain.2014, 15:3
doi: http://www.thejournalofheadacheandpain.com/content/15/1/3.

(166) Steiner T. J., HagenK, Jensen R et al. The Global Campaign, World Health Organization and Lifting The Burden: Collaboration in Action. J Headache Pain. 2011;12::273-4.

(167) Stovner L J, Jumah M A, Birbeck G L et al. The Methodology of Population Surveys of Headache Prevalence, Burden and Cost: Principles and Recommendations from The Global Campaign Against Headache. J Headache Pain. 2014;15(5).

(168) Huguet A, Stinson J, MacKay B et al. Bringing Psychosocial Support to Headache Sufferers Using Information and Communication Technology: Lessons learned from Asking Potential Users What They Want. Pain Res Manag. 2014;19 (1):1-8.

(169) Ware J E. HIT-6 Scoring Interpretation English Version Website:*www.headachetestcom. 2001*, 2013.

(170) Warr A M. Age Cohorts and Emotional Attachment to Mobile Phones. A Thesis for The Degree of Master of Arts, Faculty of The University of Alabama at Birmingham. 2013.

(171) Kumar L R, Chii K D , Way L C et al. Awareness of Mobile Phone Hazards among University Students in a Malaysian Medical School. Health. 2011; 3(7):406-15.

(172) Heinrich S, Thomasl S, Heumann C et al. Association between Exposure to Radiofrequency Electromagnetic Fields Assessed by Dosimetry and Acute Symptoms in Children and Adolescents: a Population based Cross-sectional Study. Environmental Health. 2010; 9(75).

(173) Milde-Busch A, von Kries R , Thomas S et al. The Association between Use of Electronic Media and Prevalence of Headache in Adolescents: Results from a Population-Based Cross-sectional Study. BMC Neurology. 2010;10(12).

(174) Augner C, Hacker G W. Associations between Problematic Mobile Phone Use and Psychological Parameters in Young Adults. Int J Public Health. 2012;57:437-41.

(175) Chia S-E, Chia H-P, Tan J-S. Prevalence of Headache among Handheld Cellular Telephone Users in Singapore: A Community Study. Environmental Health Perspectives. 2000;108.

(176) Anannit V, Lunliya T, Chaiyos K et al. Migraine in Junior High-School Students: A prospective 3-Academic-year Cohort Study. Brain Dev. 2010;Volume 32(10):855-62.

(177) Shochat T, Flint-Bretler O, Tzischinsky O. Sleep Patterns, Electronic Media Exposure and Daytime Sleep-Related Behaviours among Israeli Adolescents. Acta Pediatrica. 2010;99:1396-400.

(178) Seo J-H, Kim J H, Yang K I et al. Late Use of Electronic Media and Its Association with Sleep, Depression,and Suicidality among Korean Adolescents. Sleep Medicine. 2017;29:76-80.

(179) LeBourgeois M K, Giannotti F, Cortesi F et al. The Relationship Between Reported Sleep Quality and Sleep Hygiene in Italian and American Adolescents. Pediatrics. 2005;115(10):257-65. doi:10.1542/peds.2004-0815H.

(180) Bansal C.P, Badami S, Galagali P, et al. Adolescent Today. IAP Adolescent Health Academy. 2015;6(3).

(181) Hillman D R, Lack L C. Public Health Implications of Sleep Loss: The Community Burden. MJA. 2013;199:S7-S10.

(182) Schwartz JR.L., Roth T. Neurophysiology of Sleep and Wakefulness: Basic Science and Clinical Implications. Curr Neuropharmacol. 2008; 6:367-78.

(183) Lynn C. Wellage M S. Headache In Children: Data From The 1988 Child Health Supplement to The National Health Interview Survey. Dissertation of Doctor of Philosophy in The Graduate School of The Ohio State University.1997.

(184) Mohler E, Frei P, Frohlich J et al. Effects of Radiofrequency Electromagnetic Field Exposure on Sleep Quality. PLoS ONE.2013; 7(5) 37455.

(185) Mindell J A, Meltzer L J. Behavioural Sleep Disorders in Children and Adolescents. (Review Article) Ann Acad Med Singapore. 2008;37:722-8.

(186) Reticular Formation Picture. Website:*http://www.apsubiology.org/anatomy/2010/2010_Exam_Reviews/Exam_4_Review/CH_12_Cerebellum-Brain_Stem.htm: Picture. 2014*, 2014.

(187) National Sleep Foundation. Sleep-Wake Cycle: Its Physiology and Impact on Health. National Sleep Foundation. 2006.

(188) Limsrijalearn K. Insomnia. Website: http://wwwsimahidolacth/Th/department/psychiatrics/dept_article_detailasp?a_id=815; 2014 [cited2014 8 Febuary].

(189) Lee-Chiong TL. Sleep Medicine Essentials. Canada: John Wiley and Son, Inc., 2009.

(190) Wheaton A G, Perry G S, Chapman D P et al. Relationship between Body Mass Index and Perceived Insufficient Sleep among U.S. Adults: an Analysis of 2008 BRFSS data. BMC Public Health. 2011;11:295.

(191) Wordsworth. W. Sleep in Patients with Respiratory Disease and Respiratory Failure. London and New York: Taylor and Francis Group; 2004.

(192) Chasens E R, Korytkowski M, Sereika S M et al. Effect of Poor Sleep Quality and Excessive Daytime Sleepiness on Factors Associated with Diabetes Self-Management. Diabetes Educ. 2013;39(1):74-82.

(193) Seibert P S., Valerio J, DeHaas C. The Concomitant Relationship Shared by Sleep Disturbances and Type 2 Diabetes: Developing Telemedicine as a Viable Treatment Optio. Diabetes Sci Technol.2013; 7(6).

(194) Leonavicius R, Adomaitiene V. Fetures of Sleep Disturbances in Multiple Sclerosis Patients. Psychiatr Danub. 2014;26(3): 249-55.

(195) Ohayon M M. Determining The Level of Sleepiness in The American Population and Its Correlates. J Psychiatr Res. 2012;46(4):422-7.

(196) Smolensky M H, Di Milia L, Ohayon M M et al. Sleep Disorders, Medical Conditions, and Road Accident Risk. Accid Anal Prev. 2011;43(2):533-48.

(197) Ohayon M M. Chronic Obstructive Pulmonary Disease and Its Association with Sleep and Mental Disorders in The General Population. J Psychiatr Res. 2014;54:79-84.

(198) Kent D T, Soose R J. Environmental Factors That can Affect Sleep and Breathing: Allergies. Clin Chest Med. 2014 35(3):589-601.

(199) Ofovwe G E, Ofovwe C E, Okunola P. Prevalence and Pattern of Sleep Disorder among Children with Neurological Diseases in University of Benin Teaching Hospital, Benin City, Nigeria. Niger J Paediatr. 2012;39(1):14 - 7.

(200) Khoury S, Chouchou F, Amzica F et al. Rapid EEG Activity during Sleep Dominates in Mild Traumatic Brain Injury Patients with Acute Pain. J Neurotrauma. 2013;30(8):633-41.

(201) Siu Y-F, Chan S, Wong K-M et al. The Comorbidity of Chronic Pain and Sleep Disturbances in a Community Adolescent Sample: Prevalence and Association with Sociodemographic and Psychosocial Factors. Pain Med. 2012;13:1292-1303.

(202) Serhan M, Altinel E C, Altinel L et al. An Assessment of Sleep Quality in Patients Undergoing Total Knee Arthroplasty Before and After Surgery. Acta Orthop Traumatol Turc. 2014;48(1):50-4.

(203) Ogeil R P, Phillips J G, Rajaratnam S M et al. Risky Drug Use and Effects on Sleep Quality and Daytime Sleepiness. Human Psychopharmacology. 2015.

(204) Buysse D J., Reynold C F., Monk T H. et al. The Pittsburgh Sleep Quality Index: A New Instrument for Psychiatric Practice and Research. Psychiatry Res. 1988;28: 193-213.

(205) Harvey A G., Stinson K, Katriina L et al. The Subjective Meaning of Sleep Quality: A Comparison of Individuals with and without Insomnia. Sleep. 2008; 31(3).

(206) Lomelí H.A., Pérez-Olmos I., Talero-Gutiérrez C. et al. Sleep Evaluation Scales and Questionnaires: a Review. Actas Esp Psiquiatr. 2008;36(1):50-9.

(207) Chanamanee P,Taboonpong S, Intanon T. Sleep Quality and Related Factors among University Students in Southern Thailand. Songkla Med J. 2006;24(3):163-73.

(208) Cauter E V. Sleep Quality and Endocrine Markers of Sleep Quality, Website: *http://wwwmacsesucsfedu/research/allostatic/sleepphp.;1997*,March 2015.

(209) Murray W, Johns. A. New Method for Measuring Daytime Sleepiness The Epworth Sleepiness Scale. Sleep. 1991;14(6).

(210) Johns M W. Sensitivity and Specificity of The Multiple Sleep Latency Test (MSLT), The Maintenance of Wakefulness Test and The Epworth Sleepiness Scale: Failure of the MSLT as a Gold Standard. J Sleep Res. 2000;9:5-11.

(211) Yi H, Shin K, Shin C. Development of The Sleep Quality Scale. J Sleep Res 2006;15:309-16.

(212) Wood B, Rea M S., Plitnick B et al. Light Level and Duration of Exposure Determine The Impact of Self-Luminous Tablets on Melatonin Suppression. Applied Ergonomics. 2012:1-4. doi.org/10.1016/j.apergo. 2012.07.008.

(213) Cajochen C, Frey S, Anders D et al. Evening Exposure to a Light-Emitting Diodes (LED)-Backlit Computer Screenaffects Circadian Physiology and Cognitive Performance. J Appl Physiol. 2011;110:1432-8.

(214) Figueiro M G., Wood B, Plitnick B et al. The Impact of Light from Computer Monitors on Melatonin Levels in College Students. Biog. Amines. 2011;25(2):106-16.

(215) Jarupat S. The Studies on Efects of The Electrically Heated Bedding and The Cellular Phone Producing. Division of Human Life and Environmental Sciences. 2002.

(216) Roosli M. Radiofrequency Electromagnetic Field Exposure and Non-Specificsymptoms of Ill Health: A Systematic Review. Environmental Research. 2008;107:277-87.

(217) Achermann P, Regel S. Pulse-Modulated Radio Frequency Electromagnetic Fields Affect Human Brain Physiology. University of Zurich, Institute of Pharmacology and Toxicology.

(218) Perentos N, Croft R J, McKenzie R J. et al. Comparison of The Effects of Continuous and Pulsed Mobile Phone Like RF Exposure on The Human EEG. Australasian Physical & Engineering Sciences in Medicine. 2007;30(4).

(219) Supe S S. Mobile Phones and Sleep - A Review. Pol J Med Phys Eng. 2010;16(1):1-10.

(220) Kesari K K, Kumar S, Behari J. Pathophysiology of Microwave Radiation: Effect on Rat Brain. Appl Biochem Biotechnol. 2012;166:379-88.

(221) Thomée S, Härenstam A, Hagberg M. Mobile Phone Use and Stress, Sleep Disturbances, and Symptoms of Depression among Young Adults - A Prospective Cohort Study. BMC Public Health. 2011;11:(66).

(222) Gradisar M, Wolfson A R., Harvey A G. et al. The Sleep and Technology Use of Americans: Findings from The National Sleep Foundation's 2011 Sleep in America Poll. J Clin Sleep Med. 2013;9(12):1291-9.

(223) Cao H, Qin F, Liu X et al. Circadian Rhythmicity of Antioxidant Markers in Rats Exposed to 1.8 GHz Radiofrequency Fields. Int J Environ Res Public Health. 2015;12:2071-87.

(224) Patumanond J. Medical Epidemiology. PHRCG Publishers 231 Sugar Sam 5 Lanna Hospital Road Chiangmai 50300, Thailand. 1998.

(225) Wayne W. D. Biostatics: A Foundation of Analysis in The Health Sciences: 6th Edition. John Wiley and Sons, inc,. 1995.

(226) Bonita R, Beaglehole R, Kjellström T. Basic Epidemiology 2nd Edition. WHO Library Cataloguing in Publication Data.2006

(227) Thaikruea L. Basic in Epidemiology: Confounder and Bias.: Canna Print. Paseecharean, Bangkok. Asscoiation of Epidemiology; 2014.

(228) Szklo M, Nieto F. J. Epidemiology Beyond the Basic: 3rd edition. Jones and Bartlet Learning books. 2014. 190

(229) Sookying C. Excessive Daytime Sleepiness. Website: *http://www. vichaiyut.co.th/jul/28_02-2547/28_02-2547_P70-76.pdf ; 2014*,March 2015.

(230) Nilchaikovit T. et al. Thai HADS. J Psychiatr Assoc Thailand. 1996;41(1).

(231) Technologies A. Spectrum Analysis Back to Basics. Website : *http://www.keysightcom/upload/cmc_upload/All/Spectrum_Analysis_Back_to_ Basicspdf*, April 2016.

(232) Wiwattanadate P. Health Risk Assessment and Modeling. Chiang Mai Province2014.

(233) Stupica B. Generalized Estimating Equations (GEE): A Modern Love Story. Website : *www.kevin-e-ogradyumdedu/GEEppt2011*, 5 May 2017.

(234) Wikipedia. Mobile technology. Website : *https://enwikipediaorg/wiki/Mobile_technology. last edited 2017*,1 June 2017.

(235) Vincent K. Probit Analysis. Website:*file:///C:/Users/wanna%20occ/ Downloads/ProbitAnalysispdf*, 2017.

(236) Pograme M S. Probit regression (Dose-Response Analysis). Website: *https://wwwmedcalcorg/manual/probitregressionphp: access 2017*, 2017.

(237) Van den Bulck J. Adolescent Use of Mobile Phones for Calling and for Sending Text Messages After Lights Out: Results from a Prospective Cohort Study with a One-Year Follow-Up. Sleep. 2007;30(9):1220-3.

(238) Marjut W, Saarnic L, Rimpela A et al. Use of Information and Communication Technology (ICT) and Perceived Health in Adolescence: The role of Sleeping Habits and Waking-Time Tiredness. J. Adolesc.2006.

(239) Wessapan T, Srisawatdhisukul S, Rattanadecho P. Specific Absorption Rate and Temperature Distributions in Human Head Subjected to Mobile Phone Radiation at Different Frequencies. Int J Heat Mass Transf. 2012;55:347.

(240) Bhutani S, Bagai D. Electromagnetic Radiations-Sources, Effects, Safety Standards and Precautions. JECET 2014;3.(3):1203-11.

(241) Ungureanu L, Cristea L, Baritz M et.al. The Heating Level Analysis of Facial Tissue During The Exposure of Electromanetic Radiations Provided from Mobile Phone. The 4th IEEE International Conference on E-Health and Bioengineering-EHB 2013. 2013.

(242) Tynjala J, Kannas L, Levalahit E et al. Perceived Sleep Quality and Its Precursors in Adolescents. Health Promot Int.14(2):155-66.

(243) Huang Y-S, Wang C-H, Guilleminault C. An Epidemiologic Study of Sleep Problems among Adolescents in North Taiwan. Sleep Medicine. 2010;11(10):1035-42.

(244) Hense S, Barba G, Pohlabeln H et al. Factors That Influence Sleep Duration in European Children. SLEEP. 2011; 34(5):633-9.

(245) Nuutinen T, Ray C, Roos E. Do Computer Use, TV Viewing, and The Presenceof The Media in The Bedroom Predict School-Aged children's Sleep Habits in a Longitudinal Study. BMC Public Health. 2013;13(684):doi:10.1186/471-2458-13-684.

(246) Huber R., Treyer V., Borbely A.A. et al. Electromagnetic Fields, such as Those from Mobile Phones, Alterregional Cerebral Blood Flow and Sleep and Waking EEG. J Sleep Res. 2002;11:289-95.

(247) Huber R, Schuderer J, Graf T et al. Radio Frequency Electromagnetic Field Exposure in Humans: Estimation of SAR Distribution in the Brain, Effects on Sleep and Heart Rate. Bioelectromagnetics. 2003;24::262-76.

(248) Regel S J., Tinguely G, Adam M et al. Pulsed Radio-Frequency Electromagnetic Fields: Dose-Dependent Effects on Sleep, The Sleep EEG and Cognitive Performance. J Sleep Res. 2007;16:253-8.

(249) Burch J. B., Reif J. S., Noonan C. W. et al. Melatonin Metabolite Excretion among Cellular Telephone Users. International Journal of Radiation Biology. 2009;78(11):1029-36.

(250) Suchinda J, Atsuko K, Hiromi T et al. Effects of the 1900 MHz Electromagnetic Field Emitted from CellularPhone on Nocturnal Melatonin Secretion. J Physiol Anthropol. 2003;22(1):61-3.

(251) Wood A W, Stough C. Does Evening Exposure to Mobile Phone Radiation Affect Subsequent Melatonin Production International Journal of Radiation Biology. 2006;82,(2).

(252) Bogdan L, Grzegorz R, Arkadiusz G et al. Influence of Electric, Magnetic, and Electromagnetic Fields on The Circadian System: Current Stage of Knowledge. Biomed Res Int. 2014;2014:1-13.

(253) Amit K, Tanvir S, Yunfei L et al. Health Implications of Mobile Radiations &Role of SAR. International Journal of The Computer, The Internet and Management (IJCIM). 2012; Special Issue.

(254) Loder S, Sheikh H U., Loder E. The Prevalence, Burden, and Treatment of Severe, Frequent and Migraine Headaches in US Minority Populations: Statistics From National Survey Studies. Headache. 2015;55(214-228).

(255) Frei P, Mohler E, Burgi A et al. A prediction Model for Personal Radio Frequency Electromagnetic Field Exposure. The Science of The Total Environment. 2009;408(1):102-8.

(256) Bolte J F, Eikelboom T. Personal Radiofrequency Electromagnetic Field Measurements in The Netherlands: Exposure Level and Variability for Everyday Activities, Times of Day and Types of Area. Environ Int. 2012;48:133-42.

(257) Beekhuizen J, Vermeulen R, Kromhout H et al. Geospatial Modelling of Electromagnetic Fields from Mobile Phone Base Stations. The Science of The Total Environment. 2013;445-446:202-9.

(258) Byun Y-H, Ha M, Kwon H-J et al. Epidemiological Characteristics of Mobile Phone Ownership and Use in Korean Childrenand Adolescents. Environ Health Toxicol.(EHT). 2013;28:1-8.

(259) Antonaci F, Nappi G, Galli F et al. Migraine and Psychiatric Comorbidity: A Review of Clinical Findings. J Headache Pain. 2011;12(2):115-25.

(260) Dosi C, Riccioni A, Corte M et al. Comorbidities of Sleep Disorders in Childhood and Adolesce: Focus on Migraine. Nature and Science of Sleep. 2013;5: 77-85.

(261) Anttila P. Tension-Type Headache in Childhood and Adolescence. Lancet Neurol. 2006;5:268-74.

(262) Walters A. B, Hamer J D., Smitherman T A et al. Sleep Disturbance and Affective Comorbidity AmongEpisodic Migraineurs. Headache. 2014;54: 116-24.

(263) Martin V T., Behbehani M M. Toward a Rational Understanding of Migraine Trigger Factors. Medical Clinics of North. 2001; 85 (4).

(264) Rains J C., Poceta J. S. Sleep and Headache. Headache. 2010;12:1-15.

(265) Hunga C-S, Anderson C, Horne J A. Mobile Phone 'talk-mode' Signal Delays EEG-Determined Sleep Onset. Neuroscience Letters. 2007;421:82-6.

(266) Jovanovic D, Bragard G, Picard D et al. Mobile Telephones: A Comparison of Radiated Power between 3G VoIP Calls and 3G VoCS Calls. J Expo Sci Environ Epidemiol. 2015;25:80-3.

(267) Blackman C F. Evidence For Disruption By The Modulating Signal. The US Environmental Protection Agency. 2007.

(268) Lai H. Evidence For Effects on Neurology and Behavior. Prepared for The BioInitiative Working Group. 2007.

(269) Trošić I, Pavicic I, Marjanovic M. Non-Thermal Biomarkers of Exposure to Radiofrequency/Microwave Radiation. Biomarkers of RF/MW Exposure Arh Hig Rada Toksikol. 2012;1:67-73.

(270) Bawin S. M., Adey W. R. Sensitivity of Calcium Binding in Cerebral Tissue to Weak Environmental Electric Fields Oscillating at Low Frequency. Proc Natl Acad Sci USACell Biology. 1976;73(6):1999-2003.

(271) Pangopoulos D J, Chavdoula E, Margaritisdimitris L H. Bioeffects of Mobile Telephony Radiation in Relation to Its Intensity or Distance from The Antenna. Int J Radiat Biol. 2010;86(5):345-57.

(272) Borsook D, Nasim Maleki N, Becerra L et al. Understanding Migraine Through The Lens of Maladaptive Stress Responses: A Model Disease of Allostatic Load. Neuron. 2012;73: 219-34.

(273) Loughran S P., McKenzie R J., Jackson M L. et al. Individual Differences in The Effects of Mobile Phone Exposure on Human Sleep: Rethinking The Problem. Bioelectromagnetics. 2012;33:86-93.

(274) Hossmann K.-A., Hermann D.M. Effects of Electromagnetic Radiation of Mobile Phones on the Central Nervous System. Bioelectromagnetics. 2003;24:49-62.

(275) Hocking B, Westerman R. Neurological Effects of Radiofrequency Radiation. Occupational Medicine. 2003;53:123-7.

(276) Seppia C D, Ghione S, Luschi P et al. Pain Perception and Electromagnetic Fields. Neuroscience and Biobehavioral Reviews. 2007;31(4):619-42.

(277) Antoinette M, Duncker D J, Saxena P R. Migraine Headache is not Associated with Cerebral or Meningeal Vasodilatation—A 3T Magnetic Resonance Angiography Study. Brain. 2009;132:1-2.

(278) Fabrizio V, Claudio B, Florinda F et al. Mobile Phone Emission Modulates Interhemispheric Functional Coupling of EEG Alpha Rhythms. European J. Neurosci. 2007;25:1908-13. (oscar)

(279) Ferreri F, Curcio G, Pasqualetti P et al. Mobile Phone Emissions and Human Brain Excitability. Ann Neurol. 2006;60:188-96.

(280) Gioacchino T, Antonio R, Alessandro T. Functional Neuroimaging: The Adaptive Mechanisms in Migraine. The Journal of Headache and Pain. 2015;16. (277)

(281) Maurizio Z, Simona G, Anna F et al. Basal Cutaneous Pain Threshold in Headache Patients. J Headache Pain. 2011;12:303-10.

(282) Schwedt T J, Krauss M J, Frey K et al. Episodic and Chronic Migraineurs are Hypersensitive to Thermalstimuli between Migraine Attacks. Cephalalgia. 2011;31(1):6-12.

(283) Woolf C J. Central Sensitization: Implications for The Diagnosis and Treatment of Pain. Pain. 2011;152(3):S2-15.

(284) Carlo L, D'Amico D, Pierluigi B et al. Acute and Interictal Allodynia in Patients With Different Headache Forms: An Italian Pilot Study. Headache. 2008;48:272-7.

(285) Louter M A., Bosker J E., van Oosterhout W P J. et al. Cutaneous Allodynia as a Predictor of Migraine Chronification. Brain. 2013;136:3489-96.

(286) Aguggia M. Allodynia and Migraine. Neurol Sci. 2012; 33(Suppl 1):S9-S11.

(287) Ning C, Junran Z, Pian W et al. Functional Alterations of Pain Processing Pathway in Migraine Patients with Cutaneous Allodynia. Pain Medicine. 2015;16:1211-20.

(288) SchmiM R., LoughranS P., RegelS J. et al. Sleep EEG Alterations: Effects of Different Pulse-Modulated Radiofrequency Electromagnetic Fields. J Sleep Res. 2012;21:50-8.

(289) Boulloche N, Denuelle M, Payoux P et al. Photophobia in Migraine: a PET Study of Visual Cortex Hyperexcitability and Its Modulation by Pain. JNNP. 2010;81(9):974-84.

(290) Ali E N. Excitability and Photosensitivity in Neurological Disorders Using Pupillary Response to Sparse Multifocal Stimuli: Australian National University; 2016.

(291) Noseda R, Kainz V, Jakubowski M et al. A Neural Mechanism for Exacerbation of Headache by Light. Nat Neurosci. 2010;13(2):239-45.

(292) Noseda R, Kainz V, Jakubowski M et al. A Retinal-Thalamic Pathway for Photophobia During Migraine. Cephalalgia. 2009;29:2-3.

(293) Noseda R, Burstein R. Migraine Pathophysiology: Anatomy of The Trigeminovascular Pathway and Associated Neurological Symptoms, CSD, Sensitization and Modulation of Pain. Pain. 2013;154(1)

(294) Brenneman KM. Blood-Brain Barrier Permeability To TC-99M-DTPA From 825MHZ GSM Reactive Near-Field Cell Phone Exposure. 2009.

(295) Neetu S, Sumeet G, Gunjan S et al. A Systemic Review on CNS Effect of Electromagnetic Radiation 1800/900 MHz. Int J Pharm Sci. 2014;27(41): 228-33.

(296) Kramarenko A V. Central Clinic Hospital N5 Kharkov U Effects Of High-Frequency Electromegnatic Fields On Human EEG: A Brain Mapping Study. Intern J Neuroscience. 2003;113:1007-19.

(297) Seblewengel L., Patel S V., Tarekegn Y A. et al. The Epidemiology of Sleep Quality, Sleep Patterns, Consumption of Caffeinated Beverages, and Khat Use among Ethiopian College Students. Sleep Disorders. 2012;2012:1-11.

(298) Junker A, Bjørngaard J H, Gunnell D et al. Sleep Problems and Hospitalization for Self-Harm: A 15-Year Follow-Up of 9,000 Norwegian Adolescents. The Young-HUNT Study. Sleep. 2014;37(3).

(299) Mesquita G, Reimao R. Stress and Sleep Quality in High School Brazilian Adolescents. An Acad Bras Cienc. 2010;82(2).

(300) Lopez j, Hoffmann R, Armitage R. Reduced Sleep Spindle Activity in Early-Onset and Elevated Risk for Depression Running Head: Sleep Spindles and Adolescent MDD. J Am Acad Child Adolesc Psychiatry 2010; 49(9):934-43.

(301) Michal K., Gal S., Sadeh A. Sleep and Emotions: Bidirectional Links and Underlying Mechanisms. International Journal of Psychophysiology. 2013;89:218-28.

(302) Hindmarch U, Rigney N. Stanley P et al. A Naturalistic Investigation of The Effects of Day-Long Consumption of Tea, Coffee and Water on Alertness, Sleep Onset and Sleep Quality. 2000;149(3):203-16.

(303) Snel J, Lorist M M. Effects of Caffeine on Sleep and Cognition. Progress in Brain Research. 2011;190.

(304) James J E, Keane M A. Caffeine, Sleep and Wakefulness: Implications of New Understanding about Withdrawal Reversal. Hum Psychopharmacol Clin Exp. 2007;22:549-58.

(305) Carrier J, Paquet J, Fernandez-Bolanos M et al. Effects of Caffeine on Daytime Recovery Sleep: A Double Challenge to The Sleep-Wake Cycle in Aging. Sleep Medicine. 2009;10:1016-24.

(306) Hershner S D, Chervin R D. Causes and Consequences of Sleepiness among College Students. Nat Sci Sleep. 2014;6:73-84.

(307) Timothy R., Rotha T. Caffeine: Sleep and Daytime Sleepiness. Sleep Med Rev. 2008;12:153-62.

(308) Brick C A., Seely D L., Palermo T M. Association Between Sleep Hygiene and Sleep Quality inMedical Students. Behav Sleep Med. 2010;8(2):113-21.

(309) Brown F C., Buboltz W C. et al. Relationship of Sleep Hygiene Awareness, Sleep Hygiene Practices, and Sleep Quality in University Students. Sleep Hygiene Awareness. 2002;28.

(310) Gibson E S, Powles P, Thabane L et al. "Sleepiness" is Serious in Adolescence: Two Surveys of 3235 Canadian Students. BMC Public Health. 2006;6(116).

(311) Adeosun S O, Asa S O, Babalola O O et al. Effects of Night-Reading on Daytime Sleepiness, Sleep Quality and Academic Performance of Undergraduate Pharmacy Students in Nigeria. Sleep Biol Rhythms.2008;6: 91-4.

(312) Dewald J F., Meijer A M., Oort F J. et al. The Influence of Sleep Quality, Sleep Duration and Sleepiness on School Performance in Children and Adolescents: A Meta-Analytic Review. Sleep Med Rev. 2010;14:179-89.

(313) Leproult R, Cauter E V. Role of Sleep and Sleep Loss in Hormonal Release and Metabolism. Endocr Dev. 2010;17:11-21.

(314) Matthews K A., DahR E., Owens J F. et al. Sleep Duration and Insulin Resistance in Healthy Black and White Adolescents. Sleep. 2012;35(10): 1353-8.

(315) Wells JCK, Hallal PC, Reichert FF et al. Sleep Patterns and Television Viewing in Relation to Obesity and Blood Pressure: Evidence from an Adolescent Brazilian Birth Cohort. Int J Obes . 2008;32:1042-9.

(316) Eisenmann J C., Ekkekakis P, Holmes M. Sleep Duration and Overweight among Australian Children and Adolescents. Acta Pediatrica. 2006;95:956-63.

(317) Arora T, Hussain S, Hubert Lam K-B et al. Exploring the Complex Pathways among Specific Types of Technology, Self-Reported Sleep Duration and Body Mass Index in UK Adolescents. International Journal of Obesity. 2013;37:1254-60.

(318) Sara T, Annika H, Mats H. Mobile Phone Use and Stress, Sleep Disturbances, and Symptoms of Depression among Young Adults–Aprospective Cohort Study. BMC Public Health. 2011,;11(66).

(319) Urooj B, Keenjhar R, Memon M Q et al. Effects of Advanced Cell Phone Technology on the Duration of Sleep. New York Science Journal. 2015; 8(2):86-8.

(320) GiannottF, Cortesi F, Sebastiani T et al. Circadian Preference, Sleep and Daytime Behaviour in Adolescence. J Sleep Res. 2002;11:191-9.

(321) Monique K. LeBourgeois P, Giannott F et al. The Relationship Between Reported Sleep Quality and Sleep Hygiene in Italian and American Adolescents. Pediatrics. 2005 115(10):257-65.

(322) Dewald J F., Meijer A M, Oort F J. et al. The Influence of Sleep Quality, Sleep Duration and Sleepiness on School Performance in Children and Adolescents: A Meta-Analytic Review. Sleep Med Rev. 2010;14:179-89.

(323) Nadorff M R., Nazem S, Fiske A. Insomnia Symptoms, Nightmares, and Suicidal Ideation in a College StudentSample. Sleep. 2011;34(1).

(324) Susan L. Calhoun P, Alexandros N. et al. Prevalence and Risk Factors of Excessive Daytime Sleepiness in a Community Sample of Young Children: The Role of Obesity, Asthma, Anxiety/Depression, and Sleep. Sleep. 2011;34(4):503-7.

(325) Moore M, Kirchner H. L, Drotar D et al. Relationships among Sleepiness, Sleep Time, and Psychological Functioning in Adolescents. J Pediatr Psychol. 2009;34(10):1175–83.

(326) Alazar H., Bizu G., Vitool L et al. Circadian Rhythm Characteristics, Poor Sleep Quality, Daytime Sleepiness and Common Psychiatric Disorders among Thai College Students. Asia Pac Psychiatry. 2015;7(2):182-9.

(327) Chellappa S L, Araújo J F. Excessive Daytime Sleepiness in Patients with Depressive Disorder. Rev Bras Psiquiatr. 2006;28(2):126-9.

(328) Bixler E. O., Vgontzas A. N., Lin H.-M. et al. Excessive Daytime Sleepiness in a General Population Sample: The Role of Sleep Apnea, Age, Obesity, Diabetes, and Depression. J Clin Endocrinol Metab. 2005;90(8):4510-5.

(329) Franzen P L., Siegle G J., Buysse D J. Relationships between Affect, Vigilance, and Sleepiness Following Sleep Deprivation. J Sleep Res. 2008;17(1):34-41.

(330) Bartlett D. J., Marshall N. S., Grunstein R R. Sleep Health New South Wales: Chronic Sleep Restriction and Daytime Sleepiness. J Intern Med. 2008;38: 24-31.

(331) Kang J-H, Chen S-C. Effects of an Irregular Bedtime Schedule on Sleep Quality, Daytime Sleepiness, and Fatigue among University Students in Taiwan. BMC Public Health. 2009;9(248).

(332) Neralie C., Gradisar M. Electronic Media Use and Sleep in School-Aged Children and Adolescents: A Review. Sleep Med. 2010;11:735-42.

(333) Carter B, Rees P, Hale L et al. Association Between Portable Screen-Based Media Device Access or Use and Sleep Outcomes A Systematic Review and Meta-Analysis. JAMA Pediatr. 2016;170(12):1202-8.

(334) Mak Y W, Ting Wu C S, Shun Hui D W et al. Association between Screen Viewing Duration and Sleep Duration, Sleep Quality, and Excessive Daytime Sleepiness among Adolescents in Hong Kong. Int J Environ Res Public Health. 2014;11:11201-19.

(335) Arora T, Broglia E, Thomas G. N. Associations between Specific Technologies and Adolescent Sleep Quantity, Sleep Quality, and Parasomnias. Sleep Med. 2014;15:240-7.

(336) Hardell L, Carlberg M, Mild K H. Epidemiological Evidence for an Association between Use of Wireless Phones and Tumor Diseases. Pathophysiology. 2009;16:113–22.

(337) Croft R.J., Leung S., McKenzie R.J. et al. Effects of 2G and 3G Mobile Phones on Human Alpha Rhythms: Resting EEG in Adolescents, Young Adults, and The Elderly. Bioelectromagnetics. 2010;31:434-44.

(338) Loughrana S P., Wooda A W., Bartona J M. et al. The Effect of Electromagnetic Fields Emitted by Mobile Phones on Human Sleep. Neuroreport. 2005;16(17).doi: 10.1097/01.wnr.0000186593.79705.3c.

(339) Curcio G., Ferrara M., Moroni F. et al. Is The Brain Influenced by a Phone Call, an EEG Study of Resting Wakefulness. Neurosci. Res. 2005;53:265-70.

(340) Lowden A, kerstedt T, Ingre M et al. Sleep After Mobile Phone Exposure in Subjects with Mobile Phone-Related Symptoms. Bioelectromagnetics. 2010.

(341) Croft R.J., Hamblin D.L., Spong J. et al. The Effect of Mobile Phone Electromagnetic Fields on the Alpha Rhythm of Human Electro-encephalogram. Bioelectromagnetics. 2008;29:1-10.

(342) Pivik R T, Harman K. A Reconceptualization of EEG Alpha Activity as an Index of Arousal During Sleep: All Alpha Activity is not Equal. J Sleep Res. 1995;4:131-7.

(343) Danker-Hopfe H, Dorn H, Bahr A et al. Effects of Electromagnetic Fields Emitted by Mobile Phones (GSM900 and WCDMA/UMTS) on The Macrostructure of Sleep. J Sleep Res. 2011;20:73-81.

(344) Mohammed H S., Fahmy H M., Radwan N M. et al. Non-Thermal Continuous and Modulated Electromagnetic Radiation Fields Effects on Sleep EEG of Rats. Journal of Advanced Research. 2013;4:181-7.

(345) Aalto S, Haarala C, Bruck A et al. Mobile Phone Affects Cerebral Blood Flow in Humans. Journal of Cerebral Blood Flow & Metabolism. 2006;26:885-90.

(346) Huber R., Treyer V., Schuderer J. et al. Exposure to Pulse-Modulated Rradio Frequency Electromagnetic Fields Affects Regional Cerebral Blood Flow. European Journal of Neuroscience. 2005;21:1000-6.

(347) Kobialka S, Steiner R, Oelhafen P et al. Wavelength-Dependent Effects of Evening Light Exposure on Sleep Architecture and Sleep EEG Power Density in Men. Am J Physiol Regul Integr Comp Physiol. 2006;290:1421-8.

(348) Cajochen C. Alerting Effects of Light. Sleep Med Rev. 2007;11:453-64.

(349) Hale L and Guan S. Screen Time and Sleep among School-Aged Children and Adolescents: A Systematic Literature Review. Sleep Med Rev. 2015;21:50-8.

(350) Evers S, Bauer B, Grotemeyer et al. Event-Related Potentials (P300) in Primary Headache in Childhood and Adolescence pg. 322. J. Child Neurol. 1998;13,.

(351) Kramarenko A V. Effects of High-Frequency Electromagnetic Fields on Human EEG: A Brain Mapping Study. Intern J Neuroscience. 2003;113: 1007-19.

(352) Nittby H, Brun A, Eberhard J et al. Increased Blood–Brain Barrier Permeability in Mammalian Brain 7 Days After Exposure to The Radiation from a GSM-900 Mobile Phone. Pathophysiology. 2009;16:103-12.

(353) Christopher G., Verena H., Ulla S et al. Increased Protein Synthesis by Cells Exposed to a 1,800-MHz Radio-Frequency Mobile Phone Electromagnetic Weld, Detected by Proteome Pro Wling. Int Arch Occup Environ Health. 2010;83:691–702.

(354) Noor N.A., Ahmed N.A., Radwan N M. Variations in Amino Acid Neurotransmitters in Some Brain Areas of Adult and Young Male Albino Rats due to Exposure to Mobile Phone Radiation. European Review for Medical and Pharmacological Sciences. 2011;15:729-42.

(355) Chongchitpaisan W, Wiwatanadate. P. Electromagnetic Radiation from Smartphone and Its Biological Effect. Journal of medicine and Health Sciences. 2016;23(2):26-35.

(356) Kerstin K, Christine F, Uhlhaas P J. Brain Development During Adolescence. Dtsch Arztebl Int. 2013;110(25):425-31.

(357) Prince P B., Rapoport A M., Sheftell F D. et al. The Effect of Weather on Headache. Headache. 2004;44:596-602.

(358) Yang AC, Fuh J-L, Huang N E et al. Temporal Associations between Weather and Headache: Analysis by Empirical Mode Decomposition. PLoS ONE. 2011;6(1):14612.

(359) Wikipedia. Orders of Magnitude (Power). Last Edited 2018, [cited 2018].

(360) Hardell L, Sage C. Biological Effects from Electromagnetic Field Exposure and Public Exposure Standards. Biomedicine & Pharmacotherapy Biomedecine & Pharmacotherapie. 2008;62(2):104-9.

(361) Neetu S., Sumeet G., Gunjan S. et.al. A Systemic Review on CNS Effect of Electromagnetic Radiation 1800/900Mhz. Int J Pharm Sci. 2014;27(41): 228-33.

(362) Center PR. Internet Seen as Positive Influence on Education but Negative on Morality in Emerging and Developing Nations. 2015

APPENDIX

APPENDIX A

Model estimation

Table 1A Odds ratio (OR) of headache and their 95% confidence intervals for each factor and adjusted for all other factors using GEE (AR1, QIC=8397.221, QICC=8366.525)

Parameter	B	Exp.(B)	95% Wald Confidence Interval for Exp.(B)		p-value
			Lower	Upper	
(Intercept)	1.399	4.049	0.531	30.883	0.18
Age	-0.283	0.754	0.673	0.844	<0.01
Anxiety score	0.079	1.083	1.036	1.131	<0.01
Depression score	0.038	1.039	0.994	1.086	0.09
PSQI score	0.050	1.051	1.017	1.086	<0.01
WIFI yes	0.895	2.447	1.935	3.095	<0.01
WIFI no	0[a]	1			
Hand-free no	1.169	3.220	2.247	4.616	<0.01
Hand-free sometime	0.650	1.915	1.235	2.969	<0.01
Hand-free usually	0[a]	1			
SOP ($x10^{-5}$mW)					
≤1.79	0.125	1.133	0.877	1.464	0.34
1.8-1.99	0.609	1.838	1.201	2.814	0.01
≥2.0	0[a]	1			

Adjusted by Age, BMI, Vision, Anxiety, Depression, PSQI, Internet use, Hand free use, Brand device, and SOP

Logit (probability of headache symptom) = 1.399-0.283 (Age)+0.079 (Anxiety score)+0.038 (Depression score)+.050 (PSQI score)+0.895 (WIFI) +1.169 (No Hand-free)+0.650 (Sometime Hand-free) +0.125 (SOP ≤1.79 $x10^{-5}$mW)+0.609 (SOP 1.8-1.99 $x10^{-5}$mW)

Table 2A Odds ratio (OR) of duration pain and their 95% confidence intervals for each factor and adjusted for all other factors using GEE (AR1, QIC=3473.753, QICC=3458.362)

Parameter	B	Exp.(B)	95% Wald Confidence Interval for Exp.(B)		p-value
			Lower	Upper	
(Intercept)	2.136	8.468	0.508	141.175	0.14
Age	-0.391	0.676	0.574	0.796	<0.01
Anxiety score	0.094	1.099	1.052	1.148	<0.01
BMI abnormal	-0.403	0.668	0.416	1.072	0.09
BMI normal	0[a]	1			
WIFI yes	0.880	2.412	1.751	3.322	<0.01
WIFI no	0[a]	1			
Hand-free no	1.107	3.025	1.735	5.274	<0.01
Hand-free sometime	0.403	1.497	0.769	2.916	0.24
Hand-free usually	0[a]	1			
SOP ($x10^{-5}$mW)(daily dose)					
≤1.79	0.432	1.540	1.080	2.194	0.02
1.8-1.99	0.503	1.653	0.727	3.757	0.23
≥2.0	0[a]	1			
(Scale)	1				

Adjusted by Age, BMI, Vision, Anxiety, Depression, PSQI, Internet use, Hand free use, Brand device, and SOP

Logit (probability long duration pain (>4hr.) = 2.136-0.391 (Age)+0.094 (Anxiety score)-0.403 (BMI)+0.880 (WIFI)+1.107 (No Hand-free use)+0.403 (Sometime Hand-free)+0.432 (SOP ≤1.79x10^{-5}mW) + 0.503 (SOP 1.8-1.99x10^{-5}mW)

Table 3A Odds ratio (OR) of frequent headache and their 95% confidence intervals for each factor and adjusted for all other factors using GEE (AR1, QIC=10672.192, QICC=10572.754)

			95% Wald Confidence Interval for Exp.(B)		
Parameter	**B**	**Exp.(B)**	**Lower**	**Upper**	**p-value**
(Intercept)	3.327	27.843	3.899	198.814	<0.01
Age	-0.366	0.693	0.621	0.774	<0.01
Anxiety score	0.102	1.108	1.077	1.140	<0.01
PSQI score	0.035	1.035	1.003	1.068	0.03
BMI abnormal	-0.402	0.669	0.437	1.025	0.07
BMI normal	0[a]	1			
WIFI yes	0.682	1.978	1.570	2.492	<0.01
WIFI no	0[a]	1			
Hand-free no	1.153	3.167	2.233	4.493	<0.01
Hand-free sometime	0.759	2.136	1.314	3.475	<0.01
Hand-free usually	0[a]	1			
SOP ($x10^{-5}$mW)					
≤1.79	0.051	1.052	0.818	1.354	0.69
1.8-1.99	0.441	1.554	1.125	2.146	0.01
≥2.0	0[a]	1			
(Scale)	1				

Adjusted by Age, BMI, Vision, Anxiety, Depression, PSQI, Internet use, Hand free use, Brand device, and SOP

Logit (probability of frequent headache) = 3.327-0.366 (Age)+0.102 (Anxiety score)+0.035 (PSQI score)-0.402 (BMI)+0.682 (WIFI)+1.153 (No Hand-free) + 0.759 (Sometime Hand-free)+0.051 (SOP ≤1.79x10^{5}mW) +0.441 (SOP 1.8-1.99x10^{-5}mW)

Table 4A Odds ratio (OR) of frequent headache and their 95% confidence intervals for each factor and lag dose adjust for all other factors using GEE (Exchangeable, QIC=10721.426, QICC=10654.986)

			95% Wald Confidence Interval for Exp.(B)		
Parameter	**B**	**Exp.(B)**	**Lower**	**Upper**	**p-value**
(Intercept)	3.131	22.894	3.029	173.029	<0.01
Age	-0.351	0.704	0.627	0.791	<0.01
Anxiety score	0.102	1.108	1.081	1.135	<0.01
PSQI score	0.022	1.022	0.994	1.051	0.12
WIFI yes	0.654	1.923	1.558	2.373	0.000
WIFI no	0[a]	1			
Hand-free no	1.092	2.980	2.169	4.095	<0.01
Hand-free sometime	0.764	2.146	1.386	3.323	<0.01
Hand-free usually	0[a]	1			
Lag_5	2.025	7.576	2.018	28.444	<0.01
(Scale)	1				

Adjusted by Age, BMI, Vision, Anxiety, Depression, PSQI, Internet use, Hand free use, Brand device, and SOP

Logit (probability of frequent headache) = 3.131-0.351 (Age)+0.102 (Anxiety score)+0.022 (PSQI score)+0.654 (WIFI)+1.092 (No hand-free)+0.764 (Sometime hand-free)+2.025 (SOP Lag_5mW)

Table 5A Odds ratio (OR) of pain score and their 95% confidence intervals for each factor adjusted for all other factors using GEE (AR1, QIC=19288.465, QICC=19144.925)

			95% Wald Confidence Interval for Exp.(B)		
Parameter	**B**	**Exp.(B)**	**Lower**	**Upper**	**p-value**
(Intercept)	1.756	5.788	1.048	31.970	0.04
Age	-0.246	0.782	0.709	0.863	<0.01
Anxiety score	0.077	1.080	1.045	1.117	<0.01
Depression score	0.037	1.038	1.005	1.072	0.03
BMI abnormal	-0.284	0.753	0.551	1.029	0.08
BMI normal	0[a]	1			
PSQI score	0.054	1.055	1.025	1.086	<0.01
WIFI yes	0.828	2.289	1.868	2.804	<0.01
WIFI no	0[a]	1			
Hand-free no	1.163	3.200	2.211	4.631	<0.01
Hand-free sometime	0.745	2.106	1.401	3.165	<0.01
Hand-free usually	0[a]	1			
SOP ($x10^{-5}$mW)					
≤1.79	0.130	1.139	0.920	1.410	0.23
1.8-1.99	0.670	1.954	1.420	2.688	<0.01
≥2.0	0[a]	1			
(Scale)	1				

Adjusted by Age, BMI, Vision, Anxiety, Depression, PSQI, Internet use, Hand free use, Brand device, and SOP

Logit (probability of pain score)= 1.756-0.246 (Age)+0.077 (Anxiety score)+0.037 Depression-0.284 (BMI)+0.054 PSQI score+0.828 (WIFI)+1.163 (No hand-free)+0.745 (Sometime hand-free)+0.130 (SOP ≤$1.79x10^{-5}$mW) +0.670 (SOP ≤$1.8\text{-}1.99x10^{-5}$mW)

Table 6A Odds ratio (OR) of pain score and their 95% confidence intervals for each factor and lag dose adjusted for all other factors using GEE (AR1, QIC=19480.688, QICC=19362.260)

			95% Wald Confidence Interval for Exp.(B)		
Parameter	**B**	**Exp.(B)**	**Lower**	**Upper**	**p-value**
(Intercept)	1.625	5.079	0.823	31.331	0.08
Age	-0.236	0.790	0.711	0.877	<0.01
Anxiety score	0.077	1.080	1.044	1.116	<0.01
Depression score	0.033	1.034	1.000	1.069	0.05
PSQI score	0.051	1.052	1.022	1.083	<0.01
WIFI yes	0.841	2.318	1.896	2.835	<0.01
WIFI no	0[a]	1			
Hand-free no	1.143	3.137	2.194	4.485	<0.01
Hand-free sometime	0.725	2.064	1.375	3.100	<0.01
Hand-free usually	0[a]	1			
SOP Lag_5 (mW)	1.931	6.894	1.640	28.980	0.01
(Scale)	1				

Adjusted by Age, BMI, Vision, Anxiety, Depression, PSQI, Internet use, Hand free use, Brand device, and SOP

Logit (probability of pain score) = 1.625-0.236 (Age)+0.077 (Anxiety score)+0.033(Depression score)+0.051 PSQI score+0.841 (WIFI)+1.143 (No hand-free use)+0.725 (Sometime hand-free)+1.931(SOP Lag_5)

Table 7A Odds ratio (OR) of TTH and their 95% confidence intervals for each factor adjusted for all other factors using GEE (AR1, QIC=6959.624, QICC=6935.178)

			95% Wald Confidence Interval for Exp.(B)		
Parameter	**B**	**Exp.(B)**	**Lower**	**Upper**	**p-value**
(Intercept)	0.092	1.097	0.114	10.514	0.94
Age	-0.233	0.792	0.699	0.897	<0.01
Anxiety score	0.073	1.076	1.038	1.116	<0.01
Depression score	0.048	1.049	1.016	1.083	<0.01
BMI abnormal	-0.366	0.694	0.466	1.033	0.07
BMI normal	0[a]	1			
Vision abnormal	0.303	1.354	0.978	1.874	0.068
Vision normal	0[a]	1			
PSQI score	0.041	1.042	1.007	1.078	0.02
WIFI yes	0.764	2.147	1.749	2.636	<0.01
WIFI no	0[a]	1			
Hand-free no	1.314	3.722	2.491	5.561	<0.01
Hand-free sometime	0.886	2.427	1.548	3.803	<0.01
Hand-free usually	0[a]	1			
(Scale)	1				

Adjusted by Age, BMI, Vision, Anxiety, Depression, PSQI, Internet use, Hand free use, Brand device, and SOP

Logit (probability of TTH type)= 0.092-0.233 (Age)+0.073 (Anxiety score)+0.048 (Depression score)-0.366 (BMI)+0.303 (Vision)+0.041 (PSQI score)+0.764 (WIFI)+1.314(No Hand-free use)+0.886 (Sometime Hand-free)

Table 8A Odds ratio (OR) of undetermined headache and their 95% confidence intervals for each factor adjusted for all other factors using GEE (AR1, QIC=3326.067, QICC=3312.361)

			95% Wald Confidence Interval for Exp.(B)		
Parameter	**B**	**Exp.(B)**	**Lower**	**Upper**	**p-value**
(Intercept)	2.599	13.448	0.787	229.686	0.07
Age	-0.397	0.672	0.572	0.790	<0.01
Anxiety score	0.112	1.119	1.069	1.171	<0.01
WIFI yes	0.828	2.289	1.678	3.122	<0.01
WIFI no	0[a]	1			
Hand-free no	0.594	1.810	1.073	3.056	0.03
Hand-free sometime	0.122	1.130	0.579	2.205	0.72
Hand-free usually	0[a]	1			
SOP ($x10^{-5}$mW)					
≤1.79	0.270	1.310	0.910	1.885	0.15
1.8-1.99	0.834	2.302	1.237	4.285	0.01
≥2.0	0[a]	1			
(Scale)	1				

Adjusted by Age, BMI, Vision, Anxiety, Depression, PSQI, Internet use, Hand free use, Brand device, and SOP

Logit (probability of undetermined headache) = 2.599-0.397 (Age)+0.112 (Anxiety score)+0.828 (WIFI)+0.594 (No hand- free use)+0.122 (Sometime hand-free)+0.270 (SOP ≤1.79x10^{-5}mW) +0.834 (SOP 1.8-1.99x10^{-5}mW)

Table 9A Odds ratio (OR) of migraine headache and their 95% confidence intervals for each factor adjusted for all other factors using GEE (Independent, QIC=1314.86, QICC=1309.60)

			95% Wald Confidence Interval for Exp.(B)		
Parameter	**B**	**Exp.(B)**	**Lower**	**Upper**	**p-value**
(Intercept)	3.666	39.109	0.470	3251.316	0.10
Age	-0.581	0.559	0.432	0.723	<0.01
Anxiety score	0.117	1.124	1.061	1.192	<0.01
WIFI yes	0.721	2.056	1.204	3.509	0.01
WIFI no	0[a]	1			
Hand-free no	1.376	3.961	1.219	12.871	0.02
Hand-free sometime	1.012	2.750	0.718	10.531	0.14
Hand-free usually	0[a]	1			
SOP ($x10^{-5}$mW)					
≤1.79	0.705	2.023	1.174	3.488	0.01
1.8-1.99	1.179	3.252	1.648	6.419	<0.01
≥2.0	0[a]	1			
(Scale)	1				

Adjusted by Age, BMI, Vision, Anxiety, Depression, PSQI, Internet use, Hand free use, Brand device, and SOP

Logit (probability l of migraine headache) = 3.666-0.581 (Age)+0.117 (Anxiety score)+0.721 (WIFI)+1.376 (No hand-free use)+1.012 (Sometime hand-free)+0.705 (SOP ≤1.79x10^{-5}mW) +1.179 (SOP 1.8-1.99x10^{-5}mW)

Table 10A Odds ratio (OR) of migraine headache and their 95% confidence intervals for each factor and lag adjusted for all other factors using GEE (AR1, QIC=1346.608, QICC=1341.171)

			95% Wald Confidence Interval for Exp.(B)		
Parameter	**B**	**Exp.(B)**	**Lower**	**Upper**	**Sig.**
(Intercept)	4.276	71.935	0.597	8665.524	0.08
Age	-0.582	0.559	0.426	0.732	<0.01
Anxiety score	0.108	1.114	1.046	1.186	<0.01
WIFI yes	0.758	2.134	1.279	3.562	<0.01
WIFI no	0[a]	1			
Hand-free no	1.069	2.912	1.040	8.154	0.04
Hand-free sometime	0.662	1.938	0.558	6.738	0.29
Hand-free usually	0[a]	1			
SOP Lag_6 (mW)	-87.397	1.106E-38	4.548E-69	2.690E-08	0.01
(Scale)	1				

Adjusted by Age, BMI, Vision, Anxiety, Depression, PSQI, Internet use, Hand free use, Brand device, and SOP

Logit (probability of migraine headache) = 4.276-0.582 (Age)+0.108 (Anxiety score)+0.758 (WIFI)+1.069 (No Hand- free use)+0.662 (Sometime Hand-free)-87.397 (SOP Lag_6)

Table 11A Odds ratio (OR) of nocturnal headache and their 95% confidence intervals for each factor and lag time dose adjusted for all other factors using GEE (AR1, QIC=3581.548, QICC=3566.899)

			95% Wald Confidence Interval for Exp.(B)		
Parameter	**B**	**Exp.(B)**	**Lower**	**Upper**	**p-value**
(Intercept)	3.760	42.967	0.057	32473.916	0.27
Age	-0.533	0.587	0.405	0.850	0.01
PSQI score	0.108	1.114	0.993	1.249	0.07
Device system: IOS	-0.871	0.419	0.192	0.911	0.03
Device system: Android	0[a]	1			
WIFI yes	0.805	2.237	1.158	4.322	0.02
WIFI no	0[a]	1			
SOP ($x10^{-5}$mW) (daytime dose Lag2 12.00-18.00 p.m.)					
≤1.79	0.021	1.021	0.534	1.954	0.95
1.8-1.99	1.612	5.014	3.357	7.489	<0.01
≥2.0	0[a]	1			
(Scale)	1				

Adjusted by Age, BMI, Vision, Anxiety, Depression, PSQI, Internet use, Hand free use, Brand device, and SOP

Logit (probability of nocturnal headache) = 3.760-0.533 (Age)+0.108 (Anxiety score)-0.871 (Device system)+0.805 (WIFI)+0.021 (SOP (daytime dose) ≤$1.79x10^{-5}$mW)+1.612 (SOP (daytime dose -lag2) 1.8-$1.99x10^{-5}$mW)

Table 12A Odds ratio (OR) of nocturnal headache and their 95% confidence intervals for each factor and lag day dose adjusted for all other factors using GEE (Exchangeable, QIC=921.492, QICC=914.215)

			95% Wald Confidence Interval for Exp.(B)		
Parameter	**B**	**Exp.(B)**	**Lower**	**Upper**	**p-value**
(Intercept)	4.771	117.982	0.114	122198.230	0.18
Age	-0.558	0.572	0.382	0.857	0.01
Device system: IOS	-0.923	0.397	0.168	0.937	0.04
Device system: Android	0[a]	1			
WIFI: yes	0.743	2.103	1.082	4.087	0.03
WIFI : no	0[a]	1			
Lag_6 (mW)	-89.769	1.032E-39	9.147E-74	1.165E-05	0.03
(Scale)	1				

Adjusted by Age, BMI, Vision, Anxiety, Depression, PSQI, Internet use, Hand free use, Brand device, and SOP

Logit (probability of nocturnal headache) = 4.771-0.558 (Age)-0.923 (Device system)+0.743 (WIFI use)-89.769 (SOP Lag_6)

Table 13A Odds ratio (OR) of morning headache and their 95% confidence intervals for each factor adjusted for all other factors using GEE (AR1, QIC=3863.458, QICC=3847.869)

Adjusted by Age, BMI, Vision, Anxiety, Depression, PSQI, Internet use, Hand free use, Brand device, and SOP

			95% Wald Confidence Interval for Exp.(B)		
Parameter	**B**	**Exp.(B)**	**Lower**	**Upper**	**p-value**
(Intercept)	0.554	1.740	0.114	26.662	0.69
Age	-0.287	0.750	0.645	0.873	<0.01
Anxiety score	0.090	1.094	1.050	1.141	<0.01
PSQI score	0.039	1.040	0.988	1.094	0.14
Vision abnormal	0.216	1.241	0.847	1.817	0.27
Vision normal	0[a]	1			
WIFI yes	0.632	1.881	1.409	2.510	<0.01
WIFI no	0[a]	1			
Hand-free no	0.933	2.542	1.544	4.186	<0.01
Hand-free sometime	0.244	1.276	0.633	2.575	0.49
Hand-free usually	0[a]	1			
SOP dose 6.00-12.00 a.m.(mW)	5.268	194.114	1.223	30821.275	0.04
(Scale)	1				

Logit (probability of morning headache)= 0.554-0.287 (Age)+0.090 (Anxiety score)+0.039 (PSQI score)+0.216 (Vision)+0.632 (WIFI use)+0.933 (No Hand-free use)+0.244 (Sometime Hand-free)+5.268 (SOP (morning dose) mW)

Table 14A Odds ratio (OR) of morning headache and their 95% confidence intervals for each factor and lag adjusted for all other factors using GEE (Independent, QIC=3860.341, QICC=3846.285)

			95% Wald Confidence Interval for Exp.(B)		
Parameter	**B**	**Exp.(B)**	**Lower**	**Upper**	**p-value**
(Intercept)	-0.209	0.811	0.046	14.381	0.89
Age	-0.288	0.750	0.642	0.875	<0.01
Anxiety score	0.091	1.095	1.049	1.142	<0.01
PSQI score	0.040	1.041	0.987	1.098	0.14
WIFI yes	0.647	1.910	1.436	2.539	<0.01
WIFI no	0[a]	1			
Hand-free no	0.963	2.620	1.590	4.316	<0.01
Hand-free sometime	0.274	1.316	0.656	2.641	0.44
Hand-free usually	0[a]	1			
SOP ($x10^{-5}$mW) (nocturnal dose Lag_1 0.00-6.00 a.m.)					
≤1.79	0.675	1.965	0.935	4.128	0.07
≥2.0	0.856	2.354	1.121	4.944	0.02
1.8-1.99	0[a]	1			
(Scale)	1				

Adjusted by Age, BMI, Vision, Anxiety, Depression, PSQI, Internet use, Hand free use, Brand device, and SOP

Logit (probability of morning headache) = -0.209-0.288 (Age)+0.091 (Anxiety score)+0.040 (PSQI score)+0.647 (WIFI use)+0.963 (No hand-free use)+0.274 (Sometime hand-free)+0.675 (SOP (nocturnal dose) ≤$1.79x10^{-5}$mW)+0.856 (SOP (nocturnal dose) ≥$2.0x10^{-5}$mW)

Table 15A Odds ratio (OR) of daytime headache and their 95% confidence intervals for each factor adjusted for all other factors using GEE (AR1, QIC=3581.548, QICC=3566.899)

			95% Wald Confidence Interval for Exp.(B)		
Parameter	**B**	**Exp.(B)**	**Lower**	**Upper**	**p-value**
(Intercept)	0.359	1.431	0.080	25.736	0.81
Age	-0.302	0.739	0.628	0.870	<0.01
Anxiety score	0.080	1.084	1.035	1.135	<0.01
PSQI score	0.088	1.092	1.039	1.148	<0.01
BMI abnormal	-0.427	0.653	0.421	1.012	0.06
BMI normal	0[a]	1			
WIFI yes	0.633	1.884	1.394	2.545	<0.01
WIFI no	0[a]	1			
Hand-free no	1.102	3.009	1.671	5.419	<0.01
Hand-free sometime	0.727	2.070	1.074	3.991	0.03
Hand-free usually	0[a]	1			
SOP ($x10^{-5}$mW) (daytime dose 12.00-18.00 p.m.)					
≤1.79	0.421	1.523	1.100	2.110	0.01
1.8-1.99	-0.182	0.834	0.158	4.389	0.83
≥2.0	0[a]	1			
(Scale)	1				

Adjusted by Age, BMI, Vision, Anxiety, Depression, PSQI, Internet use, Hand free use, Brand device, and SOP

Logit (probability of daytime headache) = 0.359-0.302 (Age)+0.080 (Anxiety score)+0.088 (PSQI score)-0.427 (BMI)+0.633 (WIFI use)+1.102 (No Hand-free use)+0.727 (Sometime Hand-free)+0.421 (SOP (daytime dose) ≤$1.79x10^{-5}$mW) -0.182 (SOP (daytime dose) 1.8-$1.99x10^{-5}$mW)

Table 16A Odds ratio (OR) of daytime headache and their 95% confidence intervals for each factor and lag adjusted for all other factors using GEE (AR1, QIC=3594.202, QICC=3581.771)

			95% Wald Confidence Interval for Exp.(B)		
Parameter	**B**	**Exp.(B)**	**Lower**	**Upper**	**p-value**
(Intercept)	-0.052	0.949	0.051	17.661	0.97
Age	-0.275	0.759	0.643	0.897	<0.01
Anxiety score	0.075	1.078	1.027	1.132	<0.01
PSQI score	0.084	1.087	1.035	1.143	<0.01
WIFI yes	0.656	1.927	1.416	2.623	<0.01
WIFI no	0[a]	1			
Hand-free no	1.099	3.000	1.674	5.376	<0.01
Hand-free sometime	0.701	2.015	1.051	3.863	0.04
Hand-free usually	0[a]	1			
SOP ($x10^{-5}$mW) (morning dose Lag_1 6.00-12.00 a.m.)					
≤1.79	0.066	1.068	0.728	1.567	0.74
1.8-1.99	0.584	1.792	1.029	3.123	0.04
≥2.0	0[a]	1			
(Scale)	1				

Adjust by Age, BMI, Vision, Anxiety, Depression, PSQI, Internet use, Hand free use, Brand device, and SOP

Logit (probability of daytime headache) = -0.052-0.275 (Age)+0.075 (Anxiety score)+0.084 (PSQI score)+0.656 (WIFI use)+1.099 (No hand-free use)+0.701 (Sometime Hand-free)+0.066 (SOP Lag_1 (morning dose) ≤$1.79x10^{-5}$mW)+0.584 (SOP Lag_1 (morning dose)$1.8-1.99x10^{-5}$mW)

Table 17A Odds ratio (OR) of daytime headache and their 95% confidence intervals for each factor and lag adjusted for all other factors using GEE (Exchangeable, QIC=3603.135, QICC=3593.614)

			95% Wald Confidence Interval for Exp.(B)		
Parameter	**B**	**Exp.(B)**	**Lower**	**Upper**	**p-value**
(Intercept)	-0.040	0.960	0.046	19.874	0.98
Age	-0.273	0.761	0.642	0.902	<0.01
Anxiety score	0.095	1.100	1.046	1.156	<0.01
PSQI score	0.069	1.071	1.019	1.127	0.01
WIFI yes	0.591	1.806	1.343	2.428	<0.01
WIFI no	0[a]	1			
Hand-free no	1.101	3.007	1.684	5.370	<0.01
Hand-free sometime	0.758	2.134	1.118	4.073	0.02
Hand-free usually	0[a]	1			
SOP Lag_5 (mW)	2.299	9.960	1.281	77.432	0.03
(Scale)	1				

Adjusted by Age, BMI, Vision, Anxiety, Depression, PSQI, Internet use, Hand free use, Brand device, and SOP

Logit (probability of daytime headache) = -0.040-0.273 (Age)+0.095 (Anxiety score)+0.069 (PSQI score)+0.591 (WIFI use)+1.101 (No Hand-free use)+0.758 (Sometime Hand-free)+2.299 (SOP Lag_5mW)

Table 18A Odds ratio (OR) of evening headache and their 95% confidence intervals for each factor adjusted for all other factors using GEE (AR1, QIC=3969.122, QICC=3950.869)

			95% Wald Confidence Interval for Exp.(B)		
Parameter	**B**	**Exp.(B)**	**Lower**	**Upper**	**p-value**
(Intercept)	-2.728	0.065	0.005	0.828	0.04
Age	-0.154	0.857	0.744	0.988	0.03
Anxiety score	0.060	1.062	0.995	1.133	0.07
Depression score	0.063	1.065	1.003	1.131	0.039
WIFI yes	0.963	2.620	1.927	3.561	<0.01
WIFI no	0[a]	1			
Hand-free no	1.106	3.023	1.666	5.485	<0.01
Hand-free sometime	0.714	2.041	1.049	3.973	0.04
Hand-free usually	0[a]	1			
SOP ($x10^{-5}$mW) (evening dose 18.00-24.00 p.m.)					
≤1.79	0.953	2.595	1.356	4.965	<0.01
≥2.0	0.950	2.585	1.375	4.860	<0.01
1.8-1.99	0[a]	1			
(Scale)	1				

Adjusted by Age, BMI, Vision, Anxiety, Depression, PSQI, Internet use, Hand free use, Brand device, and SOP

Logit (probability of evening headache) = -2.728-0.154 (Age)+0.060 (Anxiety score)+0.063 (Depression score)+0.963 (WIFI use)+1.106 (No Hand-free use)+0.714 (Sometime Hand-free)+0.953 (SOP ≤1.79x10^{-5}mW)+0.950 (SOP ≥2.0x10^{-5}mW)

Table 19A Odds ratio (OR) of evening headache and their 95% confidence intervals for each factor and lag adjusted for all other factors using GEE (AR1, QIC=3971.210, QICC=3953.953)

			95% Wald Confidence Interval for Exp.(B)		
Parameter	**B**	**Exp.(B)**	**Lower**	**Upper**	**p-value**
(Intercept)	-2.431	0.088	0.007	1.167	0.07
Age	-0.160	0.852	0.739	0.983	0.03
Anxiety score	0.060	1.061	0.995	1.132	0.07
Depression score	0.062	1.064	1.002	1.130	0.04
WIFI yes	0.965	2.624	1.929	3.569	<0.01
WIFI no	0[a]	1			
Hand-free no	1.107	3.026	1.666	5.498	<0.01
Hand-free sometime	0.715	2.044	1.049	3.981	0.04
Hand-free usually	0[a]	1			
SOP ($x10^{-5}$mW) (daytime dose Lag_1 12.00-18.00 p.m.)					
≤1.79	0.797	2.218	1.037	4.745	0.04
≥2.0	0.735	2.086	1.051	4.142	0.04
1.8-1.99	0[a]	1			
(Scale)	1				

Adjusted by Age, BMI, Vision, Anxiety, Depression, PSQI, Internet use, Hand free use, Brand device, and SOP

Logit (probability of evening headache) = -2.431-0.160 (Age)+0.060 (Anxiety score)+0.062 (Depression score)+0.965 (WIFI use)+1.107 (No hand-free use)+0.715 (Sometime hand-free)+0.797 (SOP (daytime dose) ≤$1.79x10^{-5}$mW)+0.735 (SOP (daytime dose) ≥$2.0x10^{-5}$mW)

Table 20A Odds ratio (OR) of evening headache and their 95% confidence intervals for each factor and daily dose lag adjusted for all other factors using GEE (Exchangeable, QIC=3987.845, QICC=3976.577)

			95% Wald Confidence Interval for Exp.(B)		
Parameter	**B**	**Exp.(B)**	**Lower**	**Upper**	**p-value**
(Intercept)	-1.498	0.224	0.015	3.442	0.28
Age	-0.172	0.842	0.721	0.984	0.03
Depression score	0.104	1.110	1.066	1.156	<0.01
WIFI yes	0.920	2.509	1.896	3.319	<0.01
WIFI no	0[a]	1			
Hand-free no	1.187	3.278	1.752	6.132	<0.01
Hand-free sometime	0.849	2.336	1.199	4.550	0.01
Hand-free usually	0[a]	1			
Lag_4	1.510	4.527	1.058	19.375	0.04
(Scale)	1				

Adjusted by Age, BMI, Vision, Anxiety, Depression, PSQI, Internet use, Hand free use, Brand device, and SOP

Logit (probability of evening headache) = -1.498-0.172 (Age)+0.104 (Depression score)+0.920 (WIFI use)+1.187 (No Hand-free use)+0.849 (Sometime hand-free)+1.510 (SOP Lag_4mW)

Table 21B Odds ratio (OR) of difficult sleep and their 95% confidence intervals for each factor and daily dose adjusted for all other factors using GEE (Exchangeable, QIC=6436.365, QICC=6378.316)

			95% Wald Confidence Interval for Exp.(B)		
Parameter	**B**	**Exp.(B)**	**Lower**	**Upper**	**p-value**
(Intercept)	-3.008	0.049	0.024	0.100	<0.01
Anxiety score	0.049	1.051	0.998	1.105	0.06
WIFI yes	0.129	1.137	0.924	1.400	0.23
WIFI no	0[a]	1			
Hand-free: no	0.142	1.153	0.933	1.424	0.19
Hand-free: yes	0[a]	1			
SOP ($x10^{-5}$mW) (Daily dose group)					
≤1.79	0.538	1.713	1.116	2.630	0.01
≥2.0	0.383	1.466	0.758	2.836	0.26
1.8-1.99	0[a]	1			
(Scale)	1				

Adjusted by Age, BMI, Vision, Anxiety, Depression, Bad sleep hygiene, Coffee drink, Headache, Internet use, Hand free use, Brand device, SOP

Logit (probability of sleep difficulty) = -3.008+0.049 (Anxiety score)+0.129 (WIFI use)+0.142 (No Hand-free use)+0.538 (SOP ≤1.79x10^{-5}mW)+0.383 (SOP ≥2.0x10^{-5}mW)

Table 22B Odds ratio (OR) of difficult sleep and their 95% confidence intervals for each factor and evening dose adjusted for all other factors using GEE (AR1, QIC=6404.388, QICC=6336.220)

			95% Wald Confidence Interval for Exp.(B)		
Parameter	**B**	**Exp.(B)**	**Lower**	**Upper**	**p-value**
(Intercept)	-3.214	0.040	0.017	0.096	<0.01
Anxiety score	0.039	1.040	0.972	1.113	0.26
WIFI yes	0.190	1.209	0.995	1.468	0.06
WIFI no	0[a]	1			
Hand-free: no	0.271	1.311	1.049	1.638	0.02
Hand-free: yes	0[a]	1			
SOP ($x10^{-5}$mW) (18.00-24.00 p.m.)					
≤1.79	0.782	2.185	1.014	4.706	0.05
≥2.0	0.413	1.511	0.647	3.530	0.34
1.8-1.99	0[a]	1			
(Scale)	1				

Adjusted by Age, BMI, Vision, Anxiety, Depression, PSQI, Internet use, Hand free use, Brand device, and SOP

Logit (probability of sleep difficulty) = -3.214+0.039 (Anxiety score)+0.190 (WIFI use)+0.271 (No Hand-free use)+0.782 (SOP ≤1.79x10^{-5}mW)+0.413 (SOP ≥2.0x10^{-5}mW)

Table 23B Odds ratio (OR) of difficult sleep and their 95% confidence intervals for each factor and nocturnal dose adjusted for all other factors using GEE (AR1, QIC=6439.583, QICC=6367.825)

			95% Wald Confidence Interval for Exp.(B)		
Parameter	**B**	**Exp.(B)**	**Lower**	**Upper**	**p-value**
(Intercept)	-2.776	0.062	0.042	0.092	<0.01
Anxiety score	0.040	1.040	0.973	1.113	0.25
WIFI yes	0.191	1.211	0.999	1.467	0.05
WIFI no	0[a]	1			
Hand-free: no	0.255	1.291	1.034	1.612	0.02
Hand-free: yes	0[a]	1			
SOP ($x10^{-5}$mW) (00.00-6.00 a.m.)					
≤1.79	0.179	1.196	0.727	1.966	0.48
1.8-1.99	0.643	1.903	1.195	3.029	0.01
≥2.0	0[a]	1			
(Scale)	1				

Adjusted by Age, BMI, Vision, Anxiety, Depression, Bad hygiene sleep, Coffee drink, Headache, Internet use, Hand free use, Brand device, SOP

Logit (probability of sleep difficulty) = -2.776+0.040 (Anxiety score)+0.191 (WIFI use)+0.255 (No Hand-free use)+0.179 (SOP≤1.79x10^{-5}mW)+0.643 (SOP =1.8-1.99x10^{-5}mW)

Table 24B Odds ratio (OR) of difficult sleep and their 95% confidence intervals for each factor and morning dose adjusted for all other factors using GEE (AR1, QIC=6431.641, QICC=6364.360)

			95% Wald Confidence Interval for Exp.(B)		
Parameter	**B**	**Exp.(B)**	**Lower**	**Upper**	**p-value**
(Intercept)	-3.623	0.027	0.012	0.059	<0.01
Anxiety score	0.038	1.039	0.972	1.111	0.26
WIFI yes	0.193	1.213	1.002	1.469	0.05
WIFI no	0[a]	1			
Hand-free: no	0.261	1.298	1.042	1.618	0.02
Hand-free: yes	0[a]	1			
SOP ($x10^{-5}$mW) (6.00-12.00 a.m.)					
≥2.0	0.843	2.324	1.112	4.857	0.03
≤1.79	1.228	3.413	1.503	7.753	<0.01
1.8-1.99	0[a]	1			
(Scale)	1				

Adjusted by Age, BMI, Vision, Anxiety, Depression, Bad hygiene sleep, Coffee drink, Headache, Internet use, Hand free use, Brand device, SOP

Logit (probability of sleep difficulty)= -3.623+0.038 (Anxiety score)+0.193 (WIFI use)+0.261 (No hand-free use)+0.843 (SOP (morning dose) ≥2.0 $x10^{-5}$mW)+1.228 (SOP (morning dose) ≤1.79$x10^{-5}$mW)

Table 25B Odds ratio (OR) of sleep loss and their 95% confidence intervals for each factor and daily dose adjusted for all other factors using GEE (Exchangeable, QIC=15141.009, QICC=15054.851)

			95% Wald Confidence Interval for Exp.(B)		
Parameter	**B**	**Exp.(B)**	**Lower**	**Upper**	**p-value**
(Intercept)	-0.302	0.739	0.594	0.920	0.01
Anxiety score	0.038	1.039	1.006	1.073	0.02
Depression score	0.019	1.019	0.993	1.046	0.16
Vision: abnormal	0.291	1.338	0.923	1.939	0.12
Vision: normal	0[a]	1			
BMI: abnormal	0.407	1.502	0.903	2.499	0.12
BMI: normal	0[a]	1			
Headache: yes	0.068	1.071	0.965	1.188	0.19
Headache: no	0[a]	1			
SOP ($x10^{-5}$mW) (Daily dose group)					
≥2.0	0.273	1.315	1.083	1.595	0.01
1.8-1.99	-0.014	0.986	0.695	1.399	0.94
≤1.79	0[a]	1			
(Scale)	1				

Adjusted by Age, BMI, Vision, Anxiety, Depression, Bad hygiene sleep, Coffee drink, Headache, Internet use, Hand free use, Brand device, SOP

Logit (probability of sleep loss) = -0.302+0.038 (Anxiety score)+0.0019 (Depression score)+0.291 (Vision)+0.407 (BMI)+0.068 (Headache)+0.273 (SOP≥2.0$x10^{-5}$mW)-0.014 (SOP 1.8-1.99$x10^{-5}$mW)

Table 26B Odds ratio (OR) of sleep loss and their 95% confidence intervals for each factor and evening dose adjusted for all other factors using GEE (Independent, QIC=15102.325, QICC=14996.767)

			95% Wald Confidence Interval for Exp.(B)		
Parameter	**B**	**Exp.(B)**	**Lower**	**Upper**	**p-value**
(Intercept)	-0.422	0.656	0.501	0.859	<0.01
Anxiety score	0.080	1.083	1.033	1.135	<0.01
Depression score	0.040	1.040	0.997	1.086	0.07
Vision: abnormal	0.321	1.378	0.953	1.992	0.09
Vision: normal	0[a]	1			
BMI: abnormal	0.430	1.537	0.985	2.398	0.06
BMI: normal	0[a]	1			
Headache: yes	0.108	1.114	0.961	1.291	0.15
Headache: no	0[a]	1			
SOP ($x10^{-5}$mW) (18.00-24.00 p.m.))					
≥2.0	0.296	1.344	1.020	1.771	0.04
1.8-1.99	-0.046	0.955	0.548	1.664	0.87
≤1.79	0[a]	1			
(Scale)	1				

Adjusted by Age, BMI, Vision, Anxiety, Depression, Bad hygiene sleep, Coffee drink, Headache, Internet use, Hand free use, Brand device, SOP

Logit (probability of sleep loss) = -0.422+0.088 (Anxiety score)+0.040 (Depression score)+0.321 (Vision)+0.430 (BMI)+0.108(Headache)+0.296 (SOP (evening dose)≥$2.0x10^{-5}$mW)-0.046 (SOP (evening dose) $1.8\text{-}1.99x10^{-5}$mW)

Table 27B Odds ratio (OR) of sleep loss and their 95% confidence intervals for each factor and nocturnal dose adjusted for all other factors using GEE (Independent, QIC=15081.511, QICC=14971.075)

			95% Wald Confidence Interval for Exp.(B)		
Parameter	**B**	**Exp.(B)**	**Lower**	**Upper**	**p-value**
(Intercept)	-0.420	0.657	0.520	0.830	<0.01
Anxiety score	0.077	1.080	1.030	1.133	<0.01
Depression score	0.040	1.040	0.995	1.089	0.09
Vision: abnormal	0.295	1.344	0.929	1.943	0.12
Vision: normal	0[a]	1			
BMI: abnormal	0.399	1.491	0.932	2.386	0.09
BMI: normal	0[a]	1			
Headache: yes	0.106	1.112	0.961	1.285	0.15
Headache: no	0[a]	1			
SOP ($x10^{-5}$mW) (00.00-6.00 a.m.)					
≥2.0	0.344	1.411	1.093	1.823	0.01
1.8-1.99	0.126	1.134	0.608	2.114	0.69
≤1.79	0[a]	1			
(Scale)	1				

Adjusted by Age, BMI, Vision, Anxiety, Depression, Bad hygiene sleep, Coffee drink, Headache, Internet use, Hand free use, Brand device, SOP

Logit(probability of sleep loss) = -0.422+0.077 (Anxiety score)+0.040 (Depression score)+0.295 (Vision)+0.399 (BMI)+0.106 (Headache)+0.344 (SOP (nocturnal dose) ≥$2.0x10^{-5}$mW)+0.126 (SOP (nocturnal dose) 1.8-$1.99x10^{-5}$mW)

Table 28B Odds ratio (OR) of sleep loss and their 95% confidence intervals for each factor and morning dose adjusted for all other factors using GEE (AR1, QIC=15068.060, QICC=14971.740)

			95% Wald Confidence Interval for Exp.(B)		
Parameter	**B**	**Exp.(B)**	**Lower**	**Upper**	**p-value**
(Intercept)	-0.544	0.580	0.389	0.865	0.01
Anxiety score	0.065	1.067	1.025	1.111	<0.01
Depression score	0.035	1.036	1.000	1.074	0.05
Vision: abnormal	0.310	1.364	0.941	1.977	0.10
Vision: normal	0[a]	1			
BMI: abnormal	0.394	1.482	0.933	2.355	0.09
BMI: normal	0[a]	1			
Headache: yes	0.143	1.154	1.023	1.302	0.02
Headache: no	0[a]	1			
SOP ($x10^{-5}$mW) (6.00-12.00 a.m.)					
≥2.0	0.469	1.599	1.108	2.306	0.01
≤1.79	0.061	1.063	0.680	1.660	0.79
1.8-1.99	0[a]	1			
(Scale)	1				

Adjusted by Age, BMI, Vision, Anxiety, Depression, Bad hygiene sleep, Coffee drink, Headache, Internet use, Hand free use, Brand device, SOP

Logit (probability of sleep loss)= -0.544+0.065 (Anxiety score)+0.035 (Depression score)+0.310 (Vision)+0.394 (BMI)+0.143(Headache)+0.469 (SOP (morning dose) ≥$2.0x10^{-5}$mW)+0.061 (SOP (morning dose) ≤$1.79x10^{-5}$mW)

Table 29B Odds ratio (OR) of sleep loss and their 95% confidence intervals for each factor and daytime dose adjusted for all other factors using GEE (AR1, QIC=15094.968, QICC=15003.199)

			95% Wald Confidence Interval for Exp.(B)		
Parameter	B	Exp.(B)	Lower	Upper	p-value
(Intercept)	-0.403	0.668	0.516	0.866	<0.01
Anxiety score	0.062	1.064	1.024	1.107	<0.01
Depression score	0.037	1.038	1.002	1.075	0.04
Vision: abnormal	0.303	1.354	0.938	1.956	0.11
Vision: normal	0[a]	1			
BMI: abnormal	0.408	1.504	0.957	2.364	0.08
BMI: normal	0[a]	1			
Headache: yes	0.145	1.156	1.025	1.305	0.02
Headache: no	0[a]	1			
SOP ($x10^{-5}$mW) (12.00-18.00 p.m.)					
≥2.0	0.304	1.355	1.036	1.771	0.03
1.8-1.99	0.097	1.101	0.782	1.551	0.58
≤1.79	0[a]	1			
(Scale)	1				

Adjusted by Age, BMI, Vision, Anxiety, Depression, Bad hygiene sleep, Coffee drink, Headache, Internet use, Hand free use, Brand device, SOP

Logit (probability of sleep loss)= -0.403+0.062 (Anxiety score)+0.037 (Depression score)+0.303 (Vision)+0.408 (BMI)+0.145 (Headache) +0.304 (SOP (daytime dose) ≥$2.0x10^{-5}$mW) +0.097 (SOP (daytime dose) 1.8-$1.99x10^{-5}$mW)

Table 30B Odds ratio (OR) of sleep loss and their 95% confidence intervals for each factor and lag daily dose adjusted for all other factors using GEE (Exchangeable, QIC=15260.212, QICC=15229.724)

			95% Wald Confidence Interval for Exp.(B)		
Parameter	**B**	**Exp.(B)**	**Lower**	**Upper**	**p-value**
(Intercept)	0.030	1.030	0.885	1.200	0.70
Anxiety score	0.036	1.037	1.005	1.070	0.02
Depression score	0.019	1.020	0.994	1.046	0.14
Headache: yes	0.069	1.071	0.968	1.185	0.19
Headache: no	0[a]	1			
Lag_4 (mW)	-5.745	0.003	1.156E-05	0.885	0.05
(Scale)	1				

Adjusted by Age, BMI, Vision, Anxiety, Depression, Bad hygiene sleep, Coffee drink, Headache, Internet use, Hand free use, Brand device, SOP

Logit (probability of sleep loss) = 0.030+0.036 (Anxiety score)+0.019 (Depression score)+0.069 (Headache)-5.745 (SOP Lag_4)

Table 31B Odds ratio (OR) of inefficiency sleep and their 95% confidence intervals for each factor and daily dose adjusted for all other factors using GEE (Exchangeable, QIC=6297.934, QICC=6275.833)

			95% Wald Confidence Interval for Exp.(B)		
Parameter	**B**	**Exp.(B)**	**Lower**	**Upper**	**p-value**
(Intercept)	-3.601	0.027	0.016	0.045	<0.01
Headache: yes	0.067	1.069	0.900	1.269	0.45
Headache: no	0[a]	1			
Hand-free: no	-0.152	0.859	0.698	1.056	0.15
Hand-free: sometime	-0.278	0.757	0.551	1.040	0.09
Hand-free: usually	0[a]	1			
SOP ($x10^{-5}$mW) (Daily dose group)					
≤1.79	1.514	4.543	3.328	6.201	<0.01
≥2.0	1.337	3.807	2.590	5.595	<0.01
1.8-1.99	0[a]	1			
(Scale)	1				

Adjusted by Age, BMI, Vision, Anxiety, Depression, Bad hygiene sleep, Coffee drink, Headache, Internet use, Hand free use, Brand device, SOP

Logit (probability of inefficiency sleep) = -3.601+0.067 (Headache)-0.152 (No Hand-free)-0.278 (Sometime Hand-free)+1.514 (SOP ≤1.79x10^{-5}mW) +1.337 (SOP ≥2.0x10^{-5}mW)

Table 32B Odds ratio (OR) of sleep problem and their 95% confidence intervals for each factor and daily dose adjusted for all other factors using GEE (Exchangeable, QIC=13194.678, QICC=13085.015)

			95% Wald Confidence Interval for Exp.(B)		
Parameter	**B**	**Exp.(B)**	**Lower**	**Upper**	**p-value**
(Intercept)	-1.295	0.274	0.199	0.377	<0.01
Depression score	0.019	1.019	0.989	1.051	0.22
BMI: abnormal	0.480	1.617	0.864	3.027	0.13
BMI: normal	0[a]	1			
Headache: yes	0.126	1.134	1.004	1.281	0.04
Headache: no	0[a]	1			
Bad hygiene sleep: yes	0.149	1.161	1.030	1.309	0.02
Bad hygiene sleep: yes	0[a]	1			
Coffee drinking: >5 cup	1.072	2.923	1.307	6.536	0.01
Coffee drinking: 1-5 cup	-0.027	0.973	0.825	1.148	0.75
Coffee drinking: no	0[a]	1			
WIFI: yes	-0.083	0.920	0.768	1.101	0.36
WIFI: no	0[a]	1			
Hand-free: no	-0.018	0.983	0.844	1.143	0.82
Hand-free: sometime	0.169	1.184	1.011	1.387	0.04
Hand-free: usually	0[a]	1			
SOP ($x10^{-5}$mW) (Daily dose group)					
≥2.0	0.231	1.260	1.011	1.569	0.04
1.8-1.99	-0.002	0.998	0.670	1.487	0.99
≤1.79	0[a]	1			
(Scale)	1				

Adjusted by Age, BMI, Vision, Anxiety, Depression, Bad hygiene sleep, Coffee drink, Headache, Internet use, Hand free use, Brand device, SOP

Logit (probability of frequent wake up during night)= -1.295+0.019 (Depression score)+0.480 (BMI)+0.149 (Bad hygiene sleep)+0.126 (Headache)+1.072 (Coffee drinking >5 cup)-0.027 (Coffee drinking: 1-5 cup)-0.083 (WIFI use)-0.018 (no Hand-free)+0.169 (sometime Hand-free)+0.231 (SOP≥$2.0x10^{-5}$mW)-0.002 (SOP 1.8-$1.99x10^{-5}$mW)

Table 33B Odds ratio (OR) of sleep problem and their 95% confidence intervals for each factor and nocturnal dose adjusted for all other factors using GEE (Exchangeable, QIC=13188.560, QICC=13043.966)

			95% Wald Confidence Interval for Exp.(B)		
Parameter	**B**	**Exp.(B)**	**Lower**	**Upper**	**p-value**
(Intercept)	-1.431	0.239	0.170	0.336	<0.01
Depression score	0.020	1.020	0.989	1.052	0.21
BMI: abnormal	0.582	1.790	0.929	3.448	0.08
BMI: normal	0[a]	1			
Bad hygiene sleep: yes	0.151	1.163	1.033	1.310	0.01
Bad hygiene sleep: yes	0[a]	1			
Headache: yes	0.125	1.133	1.003	1.281	0.05
Headache: no	0[a]	1			
Coffee drinking: >5 cup	1.114	3.046	1.328	6.988	0.01
Coffee drinking: 1-5 cup	-0.029	0.972	0.826	1.143	0.73
Coffee drinking: no	0[a]	1			
WIFI: yes	-0.086	0.917	0.769	1.094	0.34
WIFI: no	0[a]	1			
Hand-free: no	-0.017	0.983	0.845	1.143	0.82
Hand-free: sometime	0.167	1.182	1.009	1.384	0.04
Hand-free: usually	0[a]	1			
SOP ($x10^{-5}$mW) (00.00-6.00 a.m.)					
≥2.0	0.472	1.603	1.219	2.108	<0.01
1.8-1.99	-0.040	0.961	0.316	2.920	0.94
≤1.79	0[a]	1	$		
(Scale)	1				

Adjusted by Age, BMI, Vision, Anxiety, Depression, Bad hygiene sleep, Coffee drink, Headache, Internet use, Hand free use, Brand device, SOP

Logit (probability of sleep problem) = -1.431+0.020 (Depression score)+ 0.582 (BMI)+ 0.151 (Bad hygiene sleep)+0.125 (Headache)+ 1.114 (Coffee drinking>5 cup)-0.029 (Coffee drinking: 1-5 cup)-0.086 (WIFI use)-0.017(No Hand-free)+0.167 (Sometime Hand-free)+ 0.472 (SOP (morning dose)≥2.0x10^{-5}mW)-0.040 (SOP (morning dose 1.8-1.99x10^{-5}mW)

Table 34B Odds ratio (OR) of sleep problem and their 95% confidence intervals for each factor and morning dose adjusted for all other factors using GEE (Exchangeable, QIC=13193.475, QICC=13082.964)

			95% Wald Confidence Interval for Exp.(B)		
Parameter	**B**	**Exp.(B)**	**Lower**	**Upper**	**p-value**
(Intercept)	-1.599	0.202	0.126	0.325	<0.01
Depression score	0.019	1.019	0.988	1.051	0.23
BMI: abnormal	0.479	1.615	0.857	3.045	0.14
BMI: normal	0[a]	1			
Bad hygiene sleep: yes	0.149	1.160	1.029	1.308	0.02
Bad hygiene sleep: yes	0[a]	1			
Headache: yes	0.125	1.134	1.004	1.280	0.04
Headache: no	0[a]	1			
Coffee drinking: >5 cup	1.065	2.901	1.297	6.489	0.01
Coffee drinking: 1-5 cup	-0.026	0.974	0.825	1.149	0.76
Coffee drinking: no	0[a]	1			
WIFI: yes	-0.082	0.921	0.770	1.102	0.37
WIFI: no	0[a]	1			
Hand-free: no	-0.016	0.984	0.846	1.145	0.84
Hand-free: sometime	0.171	1.187	1.014	1.390	0.03
Hand-free: usually	0[a]	1			
SOP ($x10^{-5}$mW) (6.00-12.00 a.m.)					
≥2.0	0.522	1.685	1.170	2.426	0.01
≤1.79	0.420	1.522	0.977	2.370	0.06
1.8-1.99	0[a]	1			
(Scale)	1				

Adjusted by Age, BMI, Vision, Anxiety, Depression, Bad hygiene sleep, Coffee drink, Headache, Internet use, Hand free use, Brand device, SOP

Logit (probability of frequent wake up during night)= -1.599+0.019(Depression score)+0.479 (BMI)+0.149 (Bad hygiene sleep)+0.125 (Headache)+1.065 (Coffee drinking>5 cup) -0.026 (Coffee drinking: 1-5 cup)-0.082 (WIFI use)-0.016(No Hand-free)+0.171 (Sometime Hand-free)+0.522 (SOP (morning dose)≥$2.0x10^{-5}$mW)+0.420 (SOP (morning dose)≤$1.79x10^{-5}$mW)

Table 35B Odds ratio (OR) of morning sleepiness and their 95% confidence intervals for each factor and daily dose adjusted for all other factors using GEE (Exchangeable, QIC=12203.977, QICC=12125.189)

			95% Wald Confidence Interval for Exp.(B)		
Parameter	**B**	**Exp.(B)**	**Lower**	**Upper**	**p-value**
(Intercept)	-1.481	0.227	0.164	0.315	<0.01
Anxiety score	0.060	1.062	1.026	1.098	<0.01
BMI: abnormal	0.333	1.395	0.837	2.325	0.20
BMI: normal	0[a]	1			
SOP ($x10^{-5}$mW) (daily dose group)					
≥2.0	0.232	1.261	0.959	1.659	0.09
1.8-1.99	0.469	1.598	1.195	2.138	<0.01
≤1.79	0[a]	1			
(Scale)	1				

Adjusted by Age, BMI, Vision, Anxiety, Depression, Bad hygiene sleep, Coffee drink, Headache, Internet use, Hand free use, Brand device, SOP

Logit (probability of morning sleepiness) = -1.481+0.060 (Anxiety score)+0.333 (BMI)+0.232 (SOP≥2.0$x10^{-5}$mW)+0.469 (SOP 1.8-1.99$x10^{-5}$mW)

Table 36B Odds ratio (OR) of morning sleepiness and their 95% confidence intervals for each factor and evening dose adjusted for all other factors using GEE (Exchangeable, QIC=12196.115, QICC=12117.695)

			95% Wald Confidence Interval for Exp.(B)		
Parameter	**B**	**Exp.(B)**	**Lower**	**Upper**	**p-value.**
(Intercept)	-1.404	0.246	0.185	0.327	<0.01
Anxiety score	0.059	1.061	1.026	1.098	<0.01
BMI: abnormal	0.328	1.388	0.848	2.272	0.19
BMI: normal	0[a]	1			
SOP ($x10^{-5}$mW) (18.00-24.00 p.m.)					
≥2.0	0.146	1.157	0.939	1.426	0.17
1.8-1.99	0.575	1.778	1.213	2.606	<0.01
≤1.79	0[a]	1			
(Scale)	1				

Adjusted by Age, BMI, Vision, Anxiety, Depression, Bad hygiene sleep, Coffee drink, Headache, Internet use, Hand free use, Brand device, SOP

Logit (probability of morning sleepiness) = -1.404+0.059 (Anxiety score)+0.328 (BMI)+0.146 (SOP (evening dose)≥2.0 x 10^{-5}mW)+0.0575 (SOP (evening dose) 1.8-1.99x10^{-5}mW)

Table 37B Odds ratio (OR) of morning sleepiness and their 95% confidence intervals for each factor and nocturnal dose adjusted for all other factors using GEE (Independent, QIC=12201.236, QICC=12067.701)

			95% Wald Confidence Interval for Exp.(B)		
Parameter	**B**	**Exp.(B)**	**Lower**	**Upper**	**p-value**
(Intercept)	-1.510	0.221	0.155	0.315	<0.01
Anxiety score	0.093	1.097	1.034	1.164	<0.01
BMI: abnormal	0.310	1.363	0.794	2.341	0.26
BMI: normal	0[a]	1			
SOP ($x10^{-5}$mW) (00.00-6.00 p.m.)					
≥2.0	0.186	1.204	0.805	1.802	0.37
1.8-1.99	0.809	2.245	1.090	4.624	0.03
≤1.79	0[a]	1			
(Scale)	1				

Adjusted by Age, BMI, Vision, Anxiety, Depression, Bad hygiene sleep, Coffee drink, Headache, Internet use, Hand free use, Brand device, SOP

Logit (probability of morning sleepiness) = -1.510+0.093 (Anxiety score)+0.310 (BMI)+0.186 (SOP (nocturnal dose) ≥2.0 x 10^{-5}mW)+0.809 (SOP (nocturnal dose) 1.8-1.99x10^{-5}mW)

Table 38B Odds ratio (OR) of poor sleep and their 95% confidence intervals for each factor and daily dose adjusted for all other factors using GEE (Exchangeable, QIC=13610.259, QICC=13535.367)

			95% Wald Confidence Interval for Exp.(B)		
Parameter	**B**	**Exp.(B)**	**Lower**	**Upper**	**p-value**
(Intercept)	0.929	2.532	0.244	26.315	0.44
Age	-0.126	0.882	0.769	1.010	0.07
Anxiety score	0.056	1.057	1.025	1.090	<0.01
Depression score	0.011	1.011	0.986	1.037	0.38
BMI: abnormal	0.749	2.115	1.384	3.231	<0.01
BMI: normal	0[a]	1			
Headache: yes	0.118	1.125	0.988	1.282	0.08
Headache: no	0[a]	1			
Coffee drinking: >5 cup	0.640	1.897	0.563	6.393	0.30
Coffee drinking: 1-5 cup	0.126	1.134	0.973	1.322	0.11
Coffee drinking: no	0[a]	1			
SOP ($x10^{-5}$mW) (daily dose group)					
≥2.0	0.264	1.302	1.034	1.639	0.03
1.8-1.99	0.314	1.368	0.908	2.062	0.13
≤1.79	0[a]	1			
(Scale)	1				

Adjusted by Age, BMI, Vision, Anxiety, Depression, Bad hygiene sleep, Coffee drink, Headache, Internet use, Hand free use, Brand device, SOP

Logit (probability of poor sleep) = 0.929-0.126 (Anxiety score) +0.056 (Anxiety score)+0.011 (Depression score)+0.749 (BMI)+0.118 (Headache)+0.640 (Coffee drinking>5 cup)+0.126 (Coffee drinking 1-5cup)+0.264 (SOP≥2.0$x10^{-5}$mW)+0.314 (SOP 1.8-1.99$x10^{-5}$mW)

Table 40B Odds ratio (OR) of poor sleep and their 95% confidence intervals for each factor and nocturnal dose adjusted for all other factors using GEE (AR1, QIC=13575.230, QICC=13482.849)

			95% Wald Confidence Interval for Exp.(B)		
Parameter	**B**	**Exp.(B)**	**Lower**	**Upper**	**p-value**
(Intercept)	1.258	3.518	0.331	37.391	0.29
Age	-0.142	0.868	0.756	0.996	0.04
Anxiety score	0.053	1.055	1.020	1.091	<0.01
Depression score	0.047	1.048	1.009	1.088	0.02
BMI: abnormal	0.726	2.067	1.332	3.208	<0.01
BMI: normal	0[a]	1			
Headache: yes	0.194	1.214	1.062	1.388	0.01
Headache: no	0[a]	1			
Coffee drinking: >5 cup	0.803	2.233	0.642	7.770	0.21
Coffee drinking: 1-5 cup	0.141	1.152	0.964	1.377	0.12
Coffee drinking: no	0[a]	1			
SOP ($x10^{-5}$mW) (nocturnal dose)					
≥2.0	0.129	1.137	0.871	1.485	0.35
1.8-1.99	0.507	1.660	1.146	2.404	0.01
≤1.79	0[a]	1			
(Scale)	1				

Adjusted by Age, BMI, Vision, Anxiety, Depression, Bad hygiene sleep, Coffee drink, Headache, Internet use, Hand free use, Brand device, SOP

Logit (probability of poor sleep) = 1.258-0.142(Age)+0.053 (Anxiety score)+0.047 (Depression score)+0.726 (BMI)+0.194 (Headache)+0.803 (Coffee drinking>5 cup)+0.141 (Coffee drinking 1-5cup)+0.129 (SOP (nocturnal dose)≥2.0x10^{-5}mW)+0.314 (SOP(nocturnal dose) 1.8-1.99x10^{-5}mW)

Table 41B Odds ratio (OR) of poor sleep and their 95% confidence intervals for each factor and morning dose adjusted for all other factors using GEE (Exchangeable, QIC=13638.242, QICC=13544.390)

			95% Wald Confidence Interval for Exp.(B)		
Parameter	**B**	**Exp.(B)**	**Lower**	**Upper**	**p-value**
(Intercept)	0.669	1.952	0.163	23.368	0.59
Age	-0.116	0.890	0.772	1.027	0.11
Anxiety score	0.055	1.057	1.025	1.090	<0.01
Depression score	0.011	1.011	0.986	1.037	0.38
BMI: abnormal	0.761	2.141	1.378	3.328	<0.01
BMI: normal	0[a]	1			
Headache: yes	0.116	1.123	0.986	1.280	0.08
Headache: no	0[a]	1			
Coffee drinking: >5 cup	0.646	1.909	0.573	6.358	0.29
Coffee drinking: 1-5 cup	0.127	1.136	0.974	1.324	0.10
Coffee drinking: no	0[a]	1			
SOP ($x10^{-5}$mW) (morning dose)					
≥2.0	0.395	1.484	1.046	2.106	0.03
1.8-1.99	0.293	1.341	0.726	2.477	0.35
≤1.79	0[a]	1			
(Scale)	1				

Adjusted by Age, BMI, Vision, Anxiety, Depression, Bad hygiene sleep, Coffee drink, Headache, Internet use, Hand free use, Brand device, SOP

Logit (probability of poor sleep) = 0.669-0.116 (Age)+0.055 (Anxiety score) +0.011 (Depression score)+0.761 (BMI)+0.116 (Headache)+0.646 (Coffee drinking>5 cup)+0.127 (Coffee drinking 1-5cup)+0.395 (SOP (morning dose)≥$2.0x10^{-5}$mW)+0.314 (SOP (morning dose) 1.8-$1.99x10^{-5}$mW)

Table 42B Odds ratio (OR) of poor sleep and their 95% confidence intervals for each factor and daytime dose adjusted for all other factors using GEE (Exchangeable, QIC=13615.496, QICC=13537.590)

			95% Wald Confidence Interval for Exp.(B)		
Parameter	**B**	**Exp.(B)**	**Lower**	**Upper**	**p-value**
(Intercept)	1.011	2.748	0.263	28.737	0.40
Age	-0.122	0.885	0.772	1.015	0.08
Anxiety score	0.056	1.057	1.026	1.090	<0.01
Depression score	0.011	1.011	0.986	1.037	0.39
BMI: abnormal	0.751	2.120	1.368	3.283	<0.01
BMI: normal	0[a]	1			
Headache: yes	0.118	1.125	0.987	1.283	0.08
Headache: no	0[a]	1			
Coffee drinking: >5 cup	0.635	1.886	0.556	6.400	0.31
Coffee drinking: 1-5 cup	0.128	1.137	0.974	1.326	0.10
Coffee drinking: no	0[a]	1			
SOP ($x10^{-5}$mW) (daytime dose)					
≥200	0.086	1.090	0.862	1.377	0.47
180-199	0.391	1.479	1.009	2.167	0.05
≤179	0[a]	1			
(Scale)	1				

Adjusted by Age, BMI, Vision, Anxiety, Depression, Bad hygiene sleep, Coffee drink, Headache, Internet use, Hand free use, Brand device, SOP

Logit (probability of poor sleep) = 0.669-0.116 (Age)+0.055 (Anxiety score)+0.011 (Depression score)+0.761 (BMI)+0.116 (Headache)+0.646 (Coffee drinking>5 cup)+0.127 (Coffee drinking 1-5cup)+0.395 (SOP (daytime dose)≥$2.0x10^{-5}$mW)+0.314 (SOP (daytime dose)1.8-$1.99x10^{-5}$mW)

Table 43B Odds ratio (OR) of poor sleep and their 95% confidence intervals for each factor and lag daily dose adjusted for all other factors using GEE (Exchangeable, QIC=13683.315, QICC=13614.371)

			95% Wald Confidence Interval for Exp.(B)		
Parameter	**B**	**Exp.(B)**	**Lower**	**Upper**	**p-value**
(Intercept)	1.116	3.054	.291	32.028	0.35
Age	-.120	.887	.774	1.017	0.09
Anxiety score	.058	1.059	1.028	1.092	<0.01
Depression score	.011	1.011	.986	1.037	0.39
BMI: abnormal	.730	2.076	1.337	3.221	<0.01
BMI: normal	0[a]	1			
Headache: yes	.120	1.127	.990	1.284	0.07
Headache: no	0[a]	1			
Lag_5 (mW)	-7.009	.001	9.935E-07	.822	0.04
(Scale)	1				

Adjusted by Age, BMI, Vision, Anxiety, Depression, Bad hygiene sleep, Coffee drink, Headache, Internet use, Hand free use, Brand device, SOP

Logit (probability of poor sleep)= 1.116-0.120 (Age)+0.058 (Anxiety score)+0.011 (Depression score)+0 .730 (BMI)+0.120 (Headache)-7.009 (Lag_5 SOP)

Table 44B Odds ratio (OR) of doze and their 95% confidence intervals for each factor adjusted for all other factors using GEE (AR1, QIC=12062.209, QICC=11910.695)

			95% Wald Confidence Interval for Exp.(B)		
Parameter	**B**	**Exp.(B)**	**Lower**	**Upper**	**p-value**
(Intercept)	-1.607	0.201	0.096	0.417	<0.01
Gender=1.00]	-0.680	0.507	0.247	1.040	0.06
Gender=2.00]	0[a]	1			
Anxiety score	0.014	1.014	0.959	1.072	0.63
Depression score	0.023	1.023	0.976	1.073	0.34
BMI: abnormal	0.249	1.283	0.664	2.477	0.46
BMI: normal	0[a]	1			
Headache: yes	0.086	1.090	0.938	1.266	0.26
Headache: no	0[a]	1			
Hand-free: no	-0.032	0.969	0.801	1.172	0.75
Hand-free: sometime	-0.212	0.809	0.637	1.027	0.08
Hand-free: usually	0[a]	1			
PSQI score	0.798	2.222	2.076	2.378	<0.01
(Scale)	1				

Adjusted by Age, BMI, Vision, Anxiety, Depression, Bad hygiene sleep, Coffee drink, Headache, Internet use, Hand free use, Brand device, SOP

Logit (probability of doze) = -1.607-0.680 (Gender)+0.014 Anxiety score) +0.023 (Depression score) +0.249 (BMI)+0.086 (Headache)-0.0327 (No hand-free) -0.212 (Sometime hand-free)+0.798 (PSQI score)

APPENDIX B

Questionnaire 1

ชุดที่ **1**

หเลขที่แบบสอบถาม ☐ / ☐☐ / ☐☐

ชั้น / ห้อง/ เลขที่

แบบสอบถามระยะที่ 1.1

กรุณาอ่านคำถามและรายละเอียดของแต่ละข้อให้เข้าใจและเลือกตอบในข้อที่ตรงกับท่านมากที่สุดโดยทำเครื่องหมาย รือ หน้าข้อที่ท่านเลือก

ส่วนที่ 1 ข้อมูลพื้นฐาน

- วัน/เดือน/ปีเกิด …../...../…..
- บ้านเลขที่……..
- เบอร์โทรศัพท์ที่ติดต่อได้……..
- เพศ 1. ☐ชาย 2. ☐หญิง
- น้ำหนัก.....กก. ส่วนสูง.....เซนติเมตร

1. ปัจจุบันท่านเรียนอยู่ชั้น
   1. ☐มัธยมศึกษาปีที่ 4 2.☐มัธยมศึกษาปีที่ 5 3.☐มัธยมศึกษาปีที่ 6
2. ปัจจุบันท่านรับจ้างทำงานนอกเวลาเรียนหรือไม่
   1. ☐ไม่ใช่ 2.☐ใช่ เลิกงานก่อน 20 .00 น 3. ☐ใช่เลิกงานหลัง 20 .00 น.
3. ท่านมีโรคที่ได้รับการวินิจฉัยโดยแพทย์และปัจจุบันทำการรักษาอยู่ **ตอบได้มากกว่า 1 ข้อ**
   1. ☐ ไม่มีโรคประจำตัว 2.☐ โรคหัวใจ 3.☐ โรคความดันโลหิตสูง
   4. ☐ โรคลมชัก 5. ☐ โรคหอบหืด 6.☐ ไซนัสอักเสบ
   7. ☐ ปัญหานอนไม่หลับ 8. ☐ เส้นเลือดสมองอุดตัน 9.☐ เลือดออกในสมอง
   10.☐ เยื่อหุ้มสมองอักเสบ 11.☐ไตวายต้องล้างไต 12☐ ไทรอยด์ฮอร์โมนต่ำ
   13.☐ ติดเชื้อช่องหู 14.☐ผื่นคัน 15.☐ โรคภูมิแพ้
   16.☐ ฟันผุปวดฟัน 17.☐ กรามหลุดขากรรไกรค้าง 18.☐ มีอาการไอนานติดต่อ 3 อาทิตย์
   19.☐ ปวดบริเวณใบหน้า 20.☐ ต้อหิน 21.☐ ตาอักเสบ

22.☐ โรคสมาธิสั้น 23.☐ โรคย้ำคิดย้ำทำ 24.☐ ปวดตามข้อ และตามร่างกาย

25.☐ มีอาการเหล่านี้ เดินเซ มือสั่น เห็นภาพซ้อน ชักเฉพาะที่ อย่างใดอย่างหนึ่งหรือมีอาการร่วมกัน

26.☐ กลุ่มอาการทูเร็ตต์หรือ ติ๊ก เช่น มีตาขยิบ หน้าขมุบขมิบ ส่งเสียงแปลกๆ ที่ควบคุมไม่ได้

27.☐ ท่านเคยได้รับหรือพบเหตุการณ์ ที่เจ็บปวดด้านจิตใจ เช่น การถูกกระทำรุนแรง กระทำทารุณข่มขืน ถูกทอดทิ้ง พบเห็นความรุนแรงในครอบครัว ถูกรังแก และไม่สามารถลืมได้

28.☐ ท่านรู้สึกกลัวเรื่องบางเรื่องเฉพาะ เช่น กลัวห้องแคบ

4. ปัจจุบันท่านมียาที่ใช้ประจำ ได้แก่

1.☐ ไม่มี 2. ☐มี ระบุ.....

4.2. ยาแก้ปวด

1.☐ Triptan 2. ☐ Paracetamol 3. ☐ Tylenol 4.☐ Ergotamine

5. ☐ มอร์ฟีน 6.☐อื่นๆ ระบุ.....

4.3. ยานอนหลับ

1.☐Ativan 2.☐Dormicum 3. ☐xanax 4.☐อื่นๆระบุ .....

4.4. ยาแก้แพ้ ลดน้ำมูก

1. ☐CPM 2.☐Atarax 3.☐zyrtec 4. ☐Actifed

5. ☐ อื่นๆ ระบุ.....

4.5. ยาที่ท่านใช้ประจำ ใช้บ่อยเท่าไร

1. ☐น้อยกว่า 10 ครั้ง /เดือน 2. ☐มากกว่า 10 ครั้ง/เดือน

5. ท่านมีปัญหาสุขภาพเหล่านี้ ตอบได้มากกว่า 1 ข้อ

1.☐ไม่มี

2. มีปัญหาการมองเห็น 2.1.☐สายตาสั้น 2.2.☐สายตายาว 2.3.☐สายตาเอียง 2.4. ☐ ตาเข

3. ปัญหาสายตาที่เป็น 3.1.☐แก้ไขแล้ว 3.2.☐ยังไม่ได้แก้ไข

4. ☐ เคยได้รับอุบัติเหตุ/บาดเจ็บ/ผ่าตัดทางศีรษะ

5. ☐ เคยได้รับอุบัติเหตุ/บาดเจ็บ/ ผ่าตัดบริเวณคอ

6.☐อื่นๆ ระบุ .....

6. ท่านมีพฤติกรรมดังต่อไปนี้หรือไม่ ตอบทุกข้อ

พฤติกรรม	ไม่ใช่	ใช่ นานครั้ง/ไม่ทุกวัน	ใช่ ทุกวัน
1. ท่านสูบบุหรี่			
2. ท่านดื่มสุรา			
3. ท่านดื่มกาแฟ			
4.ท่านดื่มชา/ชาขวด/ชานม			

5.ท่านดื่มเครื่องดื่มชูกำลัง			
6.ท่านอดอาหารรับประทาน**ไม่**ครบ 3 มื้อ		.....ครั้ง/เดือน	
7.ท่านรับประทานอาหาร**ไม่**ตรงเวลา		.....ครั้ง/เดือน	

ส่วนที่ 2 ข้อมูลเกี่ยวกับอาการปวดศีรษะ

7. ในชีวิตที่ผ่านมาท่านเคยมีอาการปวดศีรษะหรือไม่

   1. ☐ไม่ใช่ 2. ☐ใช่

   กรณีที่ท่านตอบข้อ 1. ไม่ใช่ ให้ข้ามไปตอบส่วนที่ 3 และ 4

8. ท่านมีอาการปวดศีรษะตั้งแต่อายุ.....ปี.หรือตั้งแต่ตอนเรียนอยู่ชั้น.....
9. ในระยะเวลา 1 ปีที่ผ่านมาท่านมีอาการปวดศีรษะประมาณ.....ครั้ง
10. ในระยะเวลา 1 เดือนที่ผ่านมา ท่านมีอาการปวดศีรษะประมาณ.....ครั้ง
11. ท่านมีอาการปวดศีรษะบ่อยเท่าไร (จำนวนวันต่อเดือน)

    1.☐1-5 วัน 2.☐5-10 วัน 3.☐10 – 15 วัน 4.☐ มากกว่า 15 วัน

    5.☐ ปีละ 10 ครั้ง 6.☐ น้อยกว่าปีละ 10 ครั้ง

12. อาการปวดศีรษะแต่ละครั้งนานเท่าใด

    1.☐ระยะสั้นๆ เป็นวินาที 2.☐15 นาที – 4 ชั่วโมง

    3.☐ มากกว่า 4 – 72 ชั่วโมง 4.☐ มากกว่า 72 ชั่วโมง (3 วัน)

    5.☐ อื่นๆ ระบุ นาน .....นาที.....ชั่วโมง.....วัน

13. อาการปวดศีรษะทำให้ท่านรู้สึกอย่างไร

    1.☐ไม่รู้สึกแย่ 2.☐รู้สึกแย่บ้าง 3.☐ รู้สึกแย่ 4.☐รู้สึกแย่มาก

14. จงให้คะแนนระดับความรุนแรงของอาการปวดศีรษะที่มักเกิดขึ้นกับท่าน

    (วงกลมที่ตัวเลขตามระดับความปวดของท่าน 0 = ไม่ปวด 10 = ปวดมากที่สุด)

    0 1 2 3 4 5 6 7 8 9 10

15. ลักษณะอาการปวดศีรษะเป็นอย่างไร

    1.☐ปวดตุ้บ ๆ เป็นจังหวะ 2.☐ปวดจี๊ดๆเหมือนเข็มแทง

    3.☐ปวดตื้อๆ หนักๆ เหมือนของทับหรือกด 4.☐ปวดตึงๆ แน่นๆ เหมือนถูกบีบ รัดรอบหัว

    5.☐ปวดทันทีทันใดเหมือนถูกค้อนทุบหัว 6. ☐ปวดแสบๆ ร้อนๆ

    7.☐ปวดลักษณะอื่น ระบุ.....

16. อาการปวดศีรษะที่เกิดขึ้นมักเกิดขึ้นข้างใด

    1.☐ข้างซ้ายข้างเดียว 2.☐ข้างขวา ข้างเดียว

    3.☐ปวดพร้อมกัน 2 ข้าง 4.☐ ปวดสลับข้างกัน ซ้ายหรือ ขวา

    5.☐ปวดตรงกลาง 6. ☐ปวดย้ายตำแหน่งไปทั่วๆ

17. ท่านมีอาการปวดศีรษะมากขึ้นเมื่อออกกำลัง เดินขึ้นบันได หรือทำกิจกรรม

1.☐ใช่ 2.☐ไม่ใช่

18. ท่านมักมีอาการเหล่านี้ก่อนเกิดอาการปวดศีรษะ (เกิดก่อนประมาณ 5 - 60 นาที) **ตอบได้มากกว่า 1 ข้อ**

1. ☐ไม่มีอาการ 2. ☐ ตาพร่า 3.☐มองเห็นแสงหรือเห็นภาพผิดปกติ
4. ☐เวียนศีรษะ 5. ☐พูดลำบาก 6.☐อ่อนแรงแขนหรือขา ข้างด้างหนึ่ง
7. ☐หูแว่ว 8. ☐เดินเซ 9. ☐ความรู้สึกผิดปกติ เช่น อาการชา/รู้สึกซ่า
10.☐เห็นภาพซ้อน 11.☐อื่นๆ ระบุ.....

19. ท่านมีอาการเหล่านี้ร่วมด้วยขณะปวดศีรษะหรือไม่ **ตอบได้มากกว่า 1 ข้อ**

1. ☐ไม่มีอาการ 2. ☐ คลื่นไส้ 3. ☐อาเจียน 4. ☐ กลัวแสง 5. ☐กลัวเสียง
6. ☐ คัดจมูก 7. ☐น้ำมูกไหล 8. ☐เหงื่อออก 9. ☐ตาบวม 10. ☐ตาแดง
11. ☐หนังตาตก 12. ☐น้ำตาไหล 13. ☐หูอื้อ

20. ท่านมักมีอาการปวดศีรษะบริเวณใด

1. ☐ท้ายทอย 2. ☐ขมับ 3. ☐กลางศีรษะ 4. ☐หน้าผาก
5. ☐เบ้าตา 6. ☐ใบหน้า 7. ☐จมูก/โพรงจมูก 8. ☐ใบหู/รูหู
9.☐ช่องปาก/ฟัน/เหงือก 10. ☐ลำคอ 11. ☐ ต้นคอด้านหลัง 12. ☐ไหล่/บ่า

21. ท่านมักมีอาการปวดศีรษะเวลาใด

1.☐ ตื่นนอนตอนเช้า 2.☐ สายๆ 3.☐ บ่ายๆ 4.☐ เย็นหลังเลิกเรียน
5.☐ ระหว่างวันทั้งวัน 6.☐ ขณะหลับ 7.☐ ไม่เป็นเวลา

22. รูปแบบอาการปวดศีรษะของท่านเป็นอย่างไร

1.☐ อาการปวดจะมากขึ้นเรื่อยๆ 2.☐ อาการปวดจะเป็นมากขึ้นและลดลงสลับกัน
3.☐ อาการปวดจะปวดต่อเนื่องเท่าเดิมไม่หาย
4.☐ อาการปวดจะเป็นมากขึ้นและลดลงสลับกันหลังจากนั้นจะปวดต่อเนื่องเท่าเดิมไม่หาย
5.☐ อาการปวดระยะสั้นๆ และมีอาการเป็นระยะๆ

23. ท่านคิดว่าสิ่งที่กระตุ้นให้ท่านมีอาการปวดศีรษะ ได้แก่ (ตอบได้มากกว่า 1 ข้อ)

1.☐ไม่มี 2.☐ ความเครียด ความวิตกกังวล 3.☐ ใช้สายตามาก เช่น ทำงานคอมพิวเตอร์
4.☐ แสงจ้า 5.☐เสียงดัง 6. ☐กลิ่นฉุน 7. ☐การออกกำลัง
8.☐อากาศร้อน 9.☐ อากาศเย็น 10.☐การใช้โทรศัพท์ มือถือ 11.☐การ ไอ จาม เบ่ง
12.☐นอนน้อย 13.☐ นอนมาก 14.☐ นอนไม่หลับ 15.☐เคี้ยวอาหาร
16.☐แปรงฟัน 17.☐ความหิว 18.☐ ทานอาหารไม่ตรงเวลา 19.☐อดอาหาร
20.☐กาแฟ 21.☐ชานม/ชาขวด 22.☐ เครื่องดื่มแอลกอฮอล์ 23.☐ของหมักดอง

24.☐สูบบุหรี่/ได้กลิ่นบุหรี่ 25.☐ช่วงมีประจำเดือน 26.☐เป็นหวัด

27.☐ปวดกล้ามเนื้อตามร่างกาย 28.☐คำน้ำลึก 29.☐มีเพศสัมพันธ์

30.☐ขึ้นที่สูงอย่างรวดเร็ว เช่น เครื่องบิน ขึ้นดอยโดยรถยนต์ 31.☐ใช้สารเสพติด

31.☐ท้องผูก (อยากถ่ายแต่ถ่ายไม่ออก ถ่ายไม่สุด อุจาระแข็งแห้งต้องออกแรงเบ่งมาก)

32.☐อาหารได้แก่..... 33.☐ผลไม้ ได้แก่.....

24. ท่านมักจะมีวิธีการ ทำให้อาการปวดศีรษะทุเลาลง อย่างไร

1.☐ไม่มี 2.☐ พักผ่อน นอนหลับ 3.☐ยาแก้ปวด ระบุ.....

4.☐ การนวด 5.☐อื่นๆ ระบุ

ส่วนที่ 3 ข้อมูลการนอนหลับ

25. ในระยะ 4 สัปดาห์ที่ผ่านมาท่านรู้สึกเกี่ยวกับการนอนของท่านอย่างไร **(ตอบทุกข้อ)**

โปรดตอบคำถามทุกข้อ หากท่านไม่แน่ใจให้เลือกคำตอบที่ใกล้เคียงที่สุด

1. ท่านเข้านอนเวลา.....น.
2. ท่านสามารถนอนหลับหลังจากเข้านอนนาน.....นาที
3. ท่านมักจะตื่นตอนเช้าเวลา.....น.
4. ท่านมีระยะเวลาการนอนจริงในตอนกลางคืน จำนวน .....ชั่วโมง(จำนวนเวลานอนหลับที่หักเวลาตื่นตอนกลางคืนทุกครั้ง)

ในระยะเวลา4 สัปดาห์ที่ผ่านมา	ไม่เลย	< 1 ครั้ง/สัปดาห์	1-2 ครั้ง/สัปดาห์	≥ 3 ครั้ง/สัปดาห์
1. ท่านมักจะมีปัญหาการนอนหลับที่เกิดจากเหตุการณ์ดังนี้ บ่อยเท่าใด 1.1 นอนไม่หลับเมื่อเข้านอนนานกว่า 30 นาที				
1.2 ตื่นขึ้นกลางดึกหรือตื่นเช้าเกินไป				
1.3 ต้องลุกเข้าห้องน้ำ				
1.4 หายใจไม่สะดวก				
1.5 ไอหรือกรนเสียงดัง				
1.6 รู้สึกหนาว				
1.7 รู้สึกร้อน				
1.8 ฝันร้าย				
1.9 ปวด				
1.10 อื่นๆระบุ.....				
2. ท่านใช้ยาที่ช่วยให้นอนหลับบ่อยเท่าใด				
3. ท่านมีปัญหาจากอาการง่วงนอนขณะทำกิจวัตรประจำวัน เช่น ทานอาหาร การร่วมกิจกรรม บ่อยเท่าใด				

ในระยะเวลา4 สัปดาห์ที่ผ่านมา	ไม่เลย	< 1 ครั้ง/สัปดาห์	1-2 ครั้ง/สัปดาห์	≥ 3 ครั้ง/สัปดาห์
4. อาการง่วงนอนเป็นปัญหาต่อการเรียน บ่อยเท่าใด				
5. ท่านมีปัญหาปัสสาวะรดที่นอน				
6. ท่านนอนกรนเสียงดัง				
7. ท่านมีอาการแขน ขากระตุกขณะหลับ				
8. ท่านมีอาการนอนละเมอพูด				
9. ท่านมีอาการนอนละเมอเดิน				
10. ท่านมีอาการนอนกัดฟัน				
11. ท่านมักตกใจตื่นจากฝันร้าย				
12. ท่านมีอาการหยุดหายใจขณะหลับ				
13. ใน 4 สัปดาห์ที่ผ่านมาท่านรู้สึกว่าคุณภาพการนอนหลับโดยรวมเป็นอย่างไร				

ส่วนที่ 4 โทรศัพท์มือถือ

26. ปัจจุบันท่านมีโทรศัพท์เป็นของตนเองหรือไม่

1.☐ใช่ จำนวน.....เครื่อง 2.☐ไม่ใช่

27. ท่านมีโทรศัพท์ เป็นของตนเองตั้งแต่เมื่อไร

เริ่มใช้เมื่ออายุ.....ปี หรือเริ่มใช้เมื่อเรียนอยู่ชั้น.....

28. ปัจจุบันโทรศัพท์มือถือของท่านเป็น แบบสมาร์ทโฟน

1.☐ใช่ 2.☐ไม่ใช่

29. ท่านใช้บริการอะไรจากโทรศัพท์มือถือและใช้มากน้อยเพียงใดถ้าคิดว่าการใช้ทั้งหมด เป็น 100

(ตอบได้มากกว่า 1 ข้อ)

บริการที่ใช้จากโทรศัพท์	มากกว่า 50	ประมาณ 50	น้อยกว่า 50	น้อยกว่า 25
1. ใช้สนทนา โทรเข้าและโทรออก				
2. ใช้ไลน์/สไกป์/ เฟสบุค				
3. ใช้ดูหนัง/ฟังเพลง/ใช้ถ่ายรูป				

30. ท่านถือโทรศัพท์ขณะทำการสนทนา อย่างไร

1. ☐ส่วนใหญ่ถือแนบหูข้างซ้าย 2.☐ส่วนใหญ่ถือแนบหูข้างขวา 3.☐ส่วนใหญ่ถือสลับข้างไปมา

31. ท่านใช้หูฟัง (hand free) ขณะสนทนาทางโทรศัพท์หรือไม่

1.☐ใช้หูฟังขณะการสนทนาทุกครั้ง 2.☐ไม่เคยใช้หูฟังในการสนทนา

3.☐ ใช้หูฟังขณะสนทนาบ่อยๆใช้เท่ากับหรือมากกว่า 5 ครั้งในการสนทนา 10 ครั้ง

4.☐ ใช้หูฟังขณะสนทนาบางครั้ง ใช้น้อยกว่า 5 ครั้งในการสนทนา 10 ครั้ง

32. ท่านมักเปิดลำโพง (speaker phone) ขณะสนทนาทางโทรศัพท์หรือไม่

1.☐เปิดลำโพงขณะการสนทนาทุกครั้ง 2.☐ไม่เคยเปิดลำโพงขณะสนทนา

3.☐เปิดลำโพงขณะสนทนาบ่อยๆใช้เท่ากับหรือมากกว่า 5 ครั้งในการสนทนา10 ครั้ง

4.☐เปิดลำโพงขณะสนทนาบางครั้ง ใช้น้อยกว่า 5 ครั้งในการสนทนา10 ครั้ง

33. ปัจจุบันท่านใช้โทรศัพท์ยี่ห้อใดในการสนทนา ทั้งโทรเข้า โทรออก และปริมาณเท่าไร

(กรณีมีมากกว่า 1 เครื่องให้ใส่ข้อมูลทุกเครื่อง)

ยี่ห้อโทรศัพท์	ระยะเวลาการสนทนาแต่ละครั้งนาน (นาที)			ความถี่บ่อยในการสนทนา (ครั้ง/วัน)		
	น้อยกว่า 10 นาที	10-30 นาที	มากกว่า 30 นาที	1- 5	5- 10	มากกว่า10
1.ยี่ห้อโทรศัพท์ระบุ..... 2.ยี่ห้อโทรศัพท์ระบุ..... 3. ยี่ห้อโทรศัพท์ระบุ.....						

a. เฉลี่ยระยะเวลาในสนทนาจากโทรศัพท์มือถือ/ครั้ง นาน.....นาที หรือ.....ชั่วโมง

b. เฉลี่ยจำนวนครั้งในการสนทนาจากโทรศัพท์มือถือ/วัน.....ครั้ง

34. ท่านคิดว่าท่านมีอาการปวดศีรษะหลังการสนทนาจากโทรศัพท์มือถือหรือไม่

1.☐ใช่ 2. ☐ไม่ใช่

35. ***<u>ในระยะเวลา 1 ปีที่ผ่านมา</u>***ท่านมีอาการปวดศีรษะขณะหรือหลังสนทนาโทรศัพท์มือถือ บ่อยเท่าไร

1.☐มีอาการปวดศีรษะน้อยกว่า 10ครั้งขณะหรือหลังสนทนาโทรศัพท์มือถือ

2.☐มีอาการปวดศีรษะเท่ากับหรือมากกว่า 10 ครั้งขณะหรือหลังสนทนาโทรศัพท์มือถือ

3.☐มีอาการปวดศีรษะทุกครั้งขณะหรือหลังสนทนาโทรศัพท์มือถือ

4.☐ไม่เคยมีอาการปวดศีรษะขณะหรือหลังสนทนาโทรศัพท์มือถือ

36. ***<u>ในระยะเวลา 1 เดือนที่ผ่านมา</u>***ท่านมีอาการปวดศีรษะขณะหรือหลังสนทนาโทรศัพท์มือถือ บ่อยเท่าไร

1.☐มีอาการปวดศีรษะน้อยกว่า 10 ครั้งขณะหรือหลังสนทนาโทรศัพท์มือถือ

2.☐มีอาการปวดศีรษะเท่ากับหรือมากกว่า 10 ครั้ง

3.☐มีอาการปวดศีรษะทุกครั้งที่มีการใช้โทรศัพท์มือถือ

4.☐ไม่เคยมีอาการปวดศีรษะขณะหรือหลังสนทนาโทรศัพท์มือถือ

37. ท่านรู้สึกร้อนบริเวณใบหูหลังหูบริเวณหน้าหรือศีรษะขณะหรือหลังการสนทนาจากโทรศัพท์มือถือหรือไม่

1.☐ใช่ 2.☐ไม่ใช่

ข้อมูลของท่านจะทำให้ช่วยหาสาเหตุอาการปวดศีรษะแลปัญหาการนอนหลับ และปรับปรุงระบบดูแลสุขภาพ เพื่อพัฒนาคุณภาพการเรียนรู้

Questionnaire 2

ชุดที่ **2.2**

เลขที่แบบสอบถาม ☐/☐☐/☐☐

ชั้น/ห้อง/เลขที่

กรุณาอ่านคำถามและรายละเอียดของแต่ละข้อให้เข้าใจและเลือกตอบในข้อที่ตรงกับท่านมากที่สุด โดยทำเครื่องหมาย หรือ หน้าข้อที่ท่านเลือก

- เบอร์โทรศัพท์ที่ติดต่อได้……..
- เพศ 1. ☐ชาย 2. ☐หญิง
- ปัจจุบันท่านเรียนอยู่ระดับ

1. ☐มัธยมศึกษาปีที่ 4 2.☐มัธยมศึกษาปีที่ 5 3.☐มัธยมศึกษาปีที่ 6

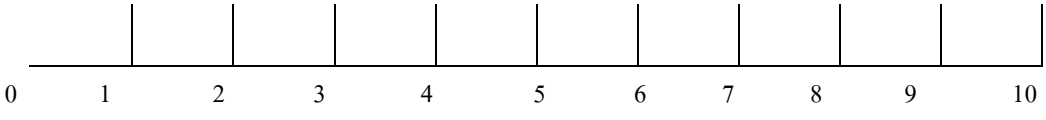
ส่วนที่ 1 ข้อมูลเกี่ยวกับอาการปวดศีรษะ HIT 6

1. **<u>ในช่วง 1 เดือนที่ผ่านมา</u>** ท่านมีอาการปวดศีรษะ ดังต่อไปนี้หรือไม่

อาการปวดศีรษะ	**ไม่เคย**	**นานๆ ครั้ง** 1-2 วัน/สัปดาห์	**บางครั้ง** 3–4 วัน/สัปดาห์	**บ่อยๆ** 5 วัน/สัปดาห์	**ทุกวัน** 6-7 วัน/สัปดาห์
1. ท่านมีอาการปวดศีรษะระดับรุนแรง บ่อยเท่าใด					
2. ท่านมีอาการปวดศีรษะจนไม่สามารถทำกิจวัตร ประจำวันหรือการเรียน บ่อยเท่าใด					
3. ท่านมีอาการปวดศีรษะมากจนอยากล้มตัวลงนอนบ่อยเท่าใด					
4. ท่านมีอาการปวดศีรษะจนรู้สึกเหนื่อยล้าเกินไป จนไม่อยากเรียนหนังสือบ่อยเท่าใด					
5. ท่านมีอาการปวดศีรษะจนทำให้รู้สึกเบื่อหน่าย หงุดหงิด รำคาญ บ่อยเท่าใด					
6. ท่านมีอาการปวดศีรษะจนไม่มีสมาธิในการทำกิจกรรมและการเรียน บ่อยเท่าใด					

2. ท่านคิดว่าอาการปวดศีรษะ ใน 4 สัปดาห์ที่ผ่านมามีความรุนแรงระดับใด

(วงกลมที่ตัวเลขตามระดับความปวดของท่าน 0 = ไม่ปวด 10 = ปวดมากที่สุด)



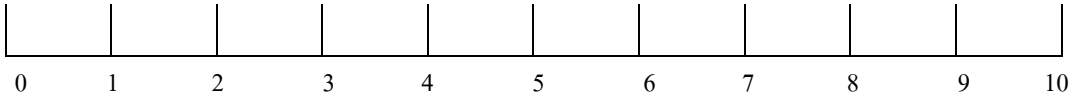
ส่วนที่ 2 ความวิตกกังวลและภาวะซึมเศร้า ใช้ HADS คะแนน ≥11

3. **<u>ในช่วง 1 สัปดาห์</u>**ที่ผ่านมา ท่านมีอารมณ์ ความรู้สึก ดังกล่าวหรือไม่ อย่างไร**(ตอบคำถามทุกข้อ)**

อารมณ์ความรู้สึก	บ่อยๆครั้ง ≥ 4 ครั้ง	บางครั้ง 2-3 ครั้ง	นานๆ ครั้ง 1 ครั้ง	ไม่เป็นไรเลย
1. ฉันรู้สึกตึงเครียด	3	2	1	0
2. ฉันมีความคิดวิตกกังวล	3	2	1	0
3. ฉันรู้สึกแจ่มใสเบิกบาน	0	1	2	3
4. ฉันรู้สึกว่าตัวเองคิดอะไรทำอะไรเชื่องช้า ลงกว่าเดิม	3	2	1	0
5. ฉันรู้สึกไม่สบายใจจนทำให้ปั่นป่วนในท้อง	3	2	1	0
6. ฉันรู้สึกผวาหรือตกใจขึ้นมาอย่างกะทันหัน	3	2	1	0
7. ฉันรู้สึกเพลิดเพลินกับการอ่านหนังสือฟังวิทยุดูทีวีหรือกิจกรรมอื่นๆที่ เคยเพลิดเพลินได้	0	1	2	3
8. ฉันรู้สึกเพลิดเพลินใจกับสิ่งต่างๆที่ฉันเคยชอบได้ 2				
1. ☐เหมือนเดิม	2. ☐ไม่มากเท่าเดิม	3. ☐มีเพียงเล็กน้อย	4. ☐เกือบไม่มีเลย	
9. ฉันมีความรู้สึกกลัวคล้ายกับว่ากำลังจะมีเรื่องไม่ดีเกิดขึ้น 3				
1.☐มีความรู้สึกและรุนแรง	2. ☐มีความรู้สึกแต่ไม่รุนแรง	3. ☐มีเล็กน้อยไม่กังวล	4. ☐ไม่มีเลย	
10. ฉันสามารถหัวเราะและมีอารมณ์ขันในเรื่องต่างๆได้ 4				
1.☐เหมือนเดิม	2. ☐ลดลงมีไม่มากนัก	3. ☐มีเล็กน้อย	4. ☐ไม่สามารถทำได้	
11. ฉันสามารถทำตัวตามสบายและรู้สึกผ่อนคลาย 7				
1.☐เหมือนเดิม	2. ☐ลดลงมีไม่มากนัก	3. ☐ไม่บ่อยนัก	4. ☐ไม่สามารถทำได้	
12. ฉันปล่อยเนื้อปล่อยตัว ไม่สนใจตนเอง 10				
1.☐.ใช่	2. ☐ไม่ใส่ใจเท่าที่ควร	3. ☐ใส่ใจน้อยกว่าเดิม	4. ☐ใส่ใจเหมือนเดิม	
13. ฉันรู้สึกกระสับกระส่ายเหมือนกับ จะอยู่นิ่งๆไม่ได้ 11				
1.☐เป็นมากทีเดียว	2. ☐เป็นค่อนข้างมาก	3. ☐ไม่มากนัก	4. ☐ไม่เป็นเลย	
14. ฉันมองสิ่งต่างๆในอนาคตด้วยความเบิกบานใจ 12				
1.☐มากเท่าที่เคยเป็น	2. ☐ค่อนข้างน้อยกว่าเดิม	3. ☐น้อยกว่าเดิม	4. ☐เกือบไม่มีเลย	

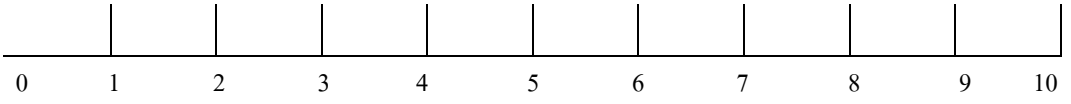
4. ท่านคิดว่าท่านมีความวิตกกังวล อยู่ระดับใด

(วงกลมที่ตัวเลขตามระดับความปวดของท่าน 0 = ไม่ปวด 10 = ปวดมากที่สุด)



5. ท่านคิดว่าท่านมีภาวะซึมเศร้า อยู่ระดับใด

(วงกลมที่ตัวเลขตามระดับความปวดของท่าน 0 = ไม่ปวด 10 = ปวดมากที่สุด)



ส่วนที่ 3 ข้อมูลพฤติกรรมการนอนและการง่วงนอนกลางวัน

6. ในชีวิตปกติท่านมีพฤติกรรมการนอน ดังนี้

โปรดตอบคำถามทุกข้อ หากท่านไม่แน่ใจให้เลือกคำตอบที่ท่านคิดว่าใกล้เคียงที่สุด

พฤติกรรมการนอน	**ไม่เลย**	**บางครั้ง** 1-2 ครั้ง/ สัปดาห์	**ประจำ** 3–4 ครั้ง/ สัปดาห์	**บ่อยๆ** 5 ครั้ง/ สัปดาห์
1. ท่านเข้านอนตรงเวลา	0	1	2	3
2. เข้านอนเมื่อรู้สึกง่วง	3	2	1	0
3. จัดเวลาการนอนได้อย่างน้อยคืนละ 8 ชั่วโมง	0	1	2	3
4. ตื่นนอนเป็นเวลา	0	1	2	3
5. ลุกจากเตียงทันทีเมื่อรู้สึกตัว	3	2	1	0
6. ท่านนอนกลางวันมากกว่า 1 ชั่วโมง	3	2	1	0
7. เมื่อนอนไม่หลับท่านลุกขึ้นหางานเบาๆทำ	0	1	2	3
8. ท่านรับประทานอาหารจำนวนมาก ในมื้อค่ำก่อนนอน	3	2	1	0
9. ท่านดื่มเครื่องดื่มอุ่นๆก่อนนอน	0	1	2	3
10. ออกกำลังกายอย่างสม่ำเสมอ	0	1	2	3
11. สูบบุหรี่ก่อนนอน	3	2	1	0
12. ดื่มแอลกอฮอล์หลังรับประทานอาหารเย็น	3	2	1	0
13. ดื่มเครื่องดื่มที่มีคาเฟอีนหลังเที่ยงวัน	3	2	1	0
14. ดื่มเครื่องดื่มชูกำลังหลังเที่ยงวัน	3	2	1	0
15. นอนในห้องที่มีอากาศถ่ายเทสะดวก	0	1	2	3
16. ท่านนอนในห้องที่ ไม่มีเสียงรบกวน	0	1	2	3
17. ท่านนอนในห้องที่ ไม่มีแสงรบกวน	0	1	2	3
18. นอนบนเครื่องที่ท่านรู้สึกสบาย	0	1	2	3
19. ทำกิจกรรมที่ทำให้ผ่อนคลายก่อนนอน	0	1	2	3
20. คิดถึงปัญหาต่างๆขณะเข้านอนเข้านอน	3	2	1	0
21. ทำกิจกรรมตื่นเต้นเร้าใจก่อนนอน เช่น ดูทีวี เล่นเกมส์คอมพิวเตอร์	3	2	1	0
22. ท่านมักคุยโทรศัพท์ก่อนเข้านอน				

7. **<u>ใน 1 เดือนที่ผ่านมา</u>** ท่านมีอาการโงกหลับ ดังนี้ (อาการโงกหลับ หรือสัปหงก การเคลิ้ม หรืองีบหลับ)**โปรดตอบคำถามทุกข้อ**

อาการโงกหลับ	**ไม่เลย**	**บางครั้ง** 1-2 ครั้ง/ สัปดาห์	**ประจำ** 3–4 ครั้ง/ สัปดาห์	**บ่อยๆ** 5 ครั้ง/ สัปดาห์
1. ท่านโงกหลับโดยไม่ตั้งใจทั้งกลางวันและตอนเย็น บ่อยเท่าใด				
2. ท่านโงกหลับขณะที่นั่งอยู่ตามสบาย บ่อยเท่าใด				
3. ท่านโงกหลับขณะที่นั่งดูโทรทัศน์หรืออ่านหนังสือ บ่อยเท่าใด				
4. ท่านโงกหลับขณะที่กำลังนั่งคุยอยู่ บ่อยเท่าใด				
5. ท่านเคยมีปัญหาง่วงนอนในช่วงเวลากลางวันหรือตอนเย็น บ่อยเท่าใด				
6. ท่านเคยมีประสบการณ์ว่าการโงกหลับในเวลากลางวันเป็นปัญหา บ่อยเท่าใด				

8. **<u>ใน 1 เดือนที่ผ่านมา</u>**ท่านคิดว่าการนอนไม่หลับของท่านเกิดจากสาเหตุอะไร **(ตอบได้มากกว่า 1 ข้อ)**

1. ☐ไม่มี/ไม่ทราบ 2. ☐ต้องการนอนกับพ่อแม่ 3.☐กลัวนอนคนเดียว 4.☐กลัวความมืด
5. ☐หิวหรืออิ่ม 6.☐นอนกลางวัน 7.☐ทะเลาะหรือมีปัญหากับเพื่อน/แฟน
8. ☐มีปัญหากับคนในครอบครัว 9.☐เรื่องเรียน การทำรายงาน
10.☐เล่นเกมส์จนดึก 11. ☐ไม่สบาย 12.☐เที่ยวกลางคืน
13.☐คุยโทรศัพท์จนดึก 14.☐อื่นๆระบุ.....

9. **<u>ใน 1 เดือนที่ผ่านมา</u>**ท่านมีวิธีการแก้ปัญหาการนอนไม่หลับอย่างไร **(ตอบได้มากกว่า 1 ข้อ)**

☐1.นอนเฉยๆ จนหลับ ☐2.อ่านหนังสือ ☐3.ฟังเพลงเบาๆ ☐4.สวดมนต์
☐5. นวดผ่อนคลาย ☐6.ลุกขึ้นมาดูทีวี ☐7. ทานยาช่วยให้หลับ
☐8. อื่นๆระบุ.....

APPENDIX C

Headache and Sleep daily

แบบบันทึกอาการปวดศีรษะ **ให้บันทึก**ทุกครั้งที่เกิดอาการปวดศีรษะ โดยเติมข้อมูลในช่องทุกช่องให้สมบูรณ์ เลือกตัวเลขจากช่องข้อมูลในการตอบคำถาม โดยพิจารณาให้ตรงหัวข้อ ก ข ค ง และ**กรณีไม่ปวดศีรษะให้บันทึกในเวลา 20.00น. โดยบันทึก ข้อ 7-9 เท่านั้น**

<table>
<tr><td rowspan="2">วันที่</td><td colspan="6">รายละเอียดอาการปวดศีรษะ</td></tr>
<tr><td>เวลาปวด</td><td>(ก) อาการก่อนอาการปวดศีรษะ</td><td>(ข) อาการเกิดร่วมกับอาการปวดศีรษะ</td><td>(ค) ผลกระทบอาการปวด</td><td>(ง)สิ่งที่กระตุ้นอาการปวดศีรษะ</td><td>(จ) การแก้ไขการปวดครั้งนี้</td></tr>
<tr><td>1.1 ปวดครั้งที่ 1</td><td>เริ่ม.....น.
หาย.....น.</td><td></td><td></td><td></td><td></td><td></td></tr>
<tr><td>2.1 ความรุนแรงของอาการปวดครั้งนี้</td><td colspan="6">0 1 2 3 4 5 6 7 8 9 10</td></tr>
<tr><td>1.2 ปวดครั้งที่ 2</td><td>เริ่ม.....น.
หาย.....น.</td><td></td><td></td><td></td><td></td><td></td></tr>
<tr><td>2.2 ความรุนแรงของอาการปวดครั้งนี้</td><td colspan="6">0 1 2 3 4 5 6 7 8 9 10</td></tr>
<tr><td colspan="7">3. การปวดครั้งนี้ท่านมีอาการร้อนบริเวณหูหรือใบหน้าด้านที่ใช้โทรศัพท์ 1.□ใช่ 2.□ไม่ใช่</td></tr>
<tr><td>4.ลักษณะอาการปวดในครั้งนี้</td><td colspan="6">1.□ปวดตุ้บๆ 2.□ปวดตื้อๆ ตึงๆ เหมือนถูกทับ 3.□ปวดจี๊ดๆ เหมือนถูกเข็มแทง
4.□ ปวดแน่นๆ เหมือนถูกบีบ รัด</td></tr>
<tr><td>5.บริเวณที่ปวดในครั้งนี้</td><td colspan="6">1.□ ท้ายทอย 2.□ ขมับ 3.□กลางศีรษะ 4.□ เบ้าตา 5.□ หน้าผาก 6.□ จมูก
7.□ ต้นคอด้านหลัง 8.□ ไหล่/บ่า</td></tr>
<tr><td>6.ปวดข้างใดในครั้งนี้</td><td colspan="6">1.□ ปวดข้างที่ใช้โทรศัพท์ 2. □ปวดข้างที่ไม่ใช้โทรศัพท์
3.□ ปวด 2 ข้างพร้อมกัน 4.□ ปวด 2 ข้างสลับกัน</td></tr>
<tr><td>7.วันนี้ท่านสนทนาโทรศัพท์แนบหู <u>โดยใช้ wifi</u> เช่น line</td><td colspan="6">1.□ไม่ใช่ 2.□ใช่ เวลา.....ครั้งละ.....นาที
(ให้ลงเวลาที่โทรทาง wifi และ จำนวนนาทีที่โทรทุกครั้ง)</td></tr>
<tr><td>8.วันนี้ท่านใช้หูฟังในการสนทนาทางโทรศัพท์มือถือหรือใช้ speaker phone</td><td colspan="6">1.□ ไม่ใช้ 2.□ ใช้< 50 % ของการสนทนา 3. □ใช้ 50 %ของการสนทนา
4.□ ใช้มากกว่า 50 % หรือใช้ทุกครั้ง</td></tr>
<tr><td colspan="7">9. จำนวนครั้งของอาการปวดศีรษะรวมในวันนี้ 1.□ จำนวน.....ครั้ง 2.□ไม่ปวดเลย</td></tr>
</table>

ข้อมูลในการตอบคำถาม (ทุกข้อเลือกได้มากกว่า 1 ข้อ)	
(ก) อาการก่อนเกิดปวดศีรษะ	**(ง) กระตุ้นอาการปวดศีรษะ**
1 ไม่มีอาการ	1 ไม่แน่ใจ
2 เห็นภาพซ้อน	2 ไม่สบาย เป็นหวัด
3 รู้สึกซ่า หรือชา	3 ใช้คอมพิวเตอร์นาน…….ชม.
4 เดินเซ	4 เสียงดัง แสงจ้า กลิ่นเหม็น ฉุน
5 พูดลำบาก	5 อากาศร้อน/เย็นเกินไป
6 อ่อนแรง ครึ่งซีก	6 การนอน(น้อย/มากไป)
7 ซึม หรือไม่รู้สึกตัว	7 ทานอาหารไม่ตรงเวลา/อดอาหาร
(ข) อาการเกิดร่วมปวดศีรษะ	8 เครื่องดื่มกาแฟ
1 ไม่มีอาการ	9 เครื่องดื่มชา/ชาขวด/ชานม
2 คลื่นไส้	10 เครื่องดื่มแอลกอฮอล์
3 อาเจียน	11 ประจำเดือน
4 กลัวแสง	12 มีเพศสัมพันธ์
5 กลัวเสียง	13 ใช้สารเสพติด
6 คัดจมูก น้ำมูกไหล	14 หลังสนทนาทางโทรศัพท์มือถือ
ตาแดง ตาบวม น้ำตาไหล	ไม่ใช้หูฟัง
7อื่นๆระบุ.....	15 ท้องผูก (ถ่ายไม่ออก/ ถ่ายไม่สุด/
(ค) ผลกระทบอาการปวด	อุจาระแข็งแห้งต้องเบ่งมาก)
1 =ปวดเล็กน้อยไม่มีผลกระทบ	**(จ) การแก้ไขอาการปวด**
2 =ปวดปานกลางกระทบการทำกิจกรรม	1 ไม่ทำอะไร
3=ปวดมากจนต้องนอนพัก	2 นอน
4=ปวดมากจนหยุดเรียน	3 ฟังเพลง
	4 การนวด
	5 ทานยา6. อื่นๆ ระบุ.....

แบบบันทึกการนอน ให้ท่านบันทึกส่วนนี้ให้สมบูรณ์<u>ในตอนเช้า</u>หลังตื่นนอน		
	วันจันทร์ที่	วันอังคารที่
1 เมื่อคืนท่านเข้านอนเวลา	.....น	.....น
2.ท่านตื่นนอนตอนเช้าเวลา	.....น	.....น
3 เมื่อคืนท่านเข้านอนแล้วหลับเป็นอย่างไร	1.☐ หลับง่าย < 15 นาที 2.☐ ใช้เวลา 15 - 20 นาที 3.☐ หลับยาก > 20 นาที	1.☐ หลับง่าย < 15 นาที 2.☐ ใช้เวลา 15 - 20 นาที 3.☐ หลับยาก > 20 นาที
4 เมื่อคืนท่านตื่นนอนตอนกลางคืนหรือไม่	1.☐ ไม่ใช่ 2.☐ ใช่ จำนวนครั้งที่ตื่น.....ครั้ง นานครั้งละ.....นาที	1.☐ ไม่ใช่ 2.☐ ใช่ จำนวนครั้งที่ตื่น.....ครั้ง นานครั้งละ.....นาที
5. โดยรวมแล้วเมื่อคืนท่านหลับได้	.....ชั่วโมง	.....ชั่วโมง
6 คุณรู้สึกอย่างไรเมื่อตื่นนอนตอนเช้าวันนี้	1.☐ สดชื่น 2.☐ ค่อยข้างสดชื่น 3.☐ ง่วงนอน เหนื่อยล้า	1.☐ สดชื่น 2.☐ ค่อยข้างสดชื่น 3.☐ ง่วงนอน เหนื่อยล้า
7. เมื่อคืนการนอนหลับของท่านถูกรบกวนจาก (ตอบได้มากกว่า 1 ข้อ)	1.☐ เปลี่ยนที่นอน 2.☐ แสงสว่าง 3.☐ เสียงดัง 4.☐ ร้อนหรือเย็น 5.☐ อื่นๆ ระบุ.....	1.☐ เปลี่ยนที่นอน 2.☐ แสงสว่าง 3.☐ เสียงดัง 4.☐ ร้อนหรือเย็น 5.☐ อื่นๆ ระบุ.....
8. เมื่อคืนคุณใช้ยานอนหลับ	1.☐ ไม่ใช่ 2.☐ ใช่ ระบุ	1.☐ ไม่ใช่ 2.☐ ใช่ ระบุ

ให้ท่านบันทึกส่วนนี้ให้สมบูรณ์ใน<u>ตอนค่ำ ก่อนเข้านอน</u>		
	วันจันทร์ที่	วันอังคารที่
1.ท่านดื่มกาแฟ/ชา	1.☐ เช้า จำนวน.....แก้ว 2.☐ กลางวัน .....แก้ว 3.☐ เย็น .....แก้ว	1.☐ เช้า จำนวน.....แก้ว 2.☐ กลางวัน .....แก้ว 3.☐ เย็น .....แก้ว
2. วันนี้ท่านรู้สึกวิตกกังวลและซึมเศร้าระดับใด (วันนี้ท่านพบเหตุการณ์ที่ทำให้วิตกกังวลหรือซึมเศร้า)	☐ 1วิตกกังวล X☐ 2ซึมเศร้าO 0 1 2 3 4 5 6 7 8 9 10 ให้ใช้สัญลักษณ์ลงตัวเลขตามระดับที่รู้สึก	

3.วันนี้ท่านเคลิ้มหลับขณะทำกิจกรรมดังนี้ (ตอบได้มากกว่า 1 ข้อ) 1.☐ กำลังนั่งและอ่านหนังสือ 2.☐ กำลังชมรายการทีวี 3.☐ นั่งเฉยๆขณะเรียนหรือในโรงหนัง 4.☐นั่งอยู่ในรถโดยสารนาน 1 ชม. 5.☐ เอนนอนกลางวัน 6.☐ นั่งเฉยๆหลังอาหารมื้อเที่ยง 7.☐ นั่งและพูดคุยกันอยู่กับบางคน 8.☐ นั่งในรถขณะรถติด 2-3 นาที		
4. ก่อนเข้านอน 2-3 ชั่วโมง ท่านได้ทำสิ่งเหล่านี้ (ตอบได้มากกว่า 1 ข้อ)	1.☐ ไม่ใช่ 2.☐ เครื่องดื่มแอลกอฮอล์ 3.☐ กาแฟ 4.☐ ชา/ชาขวด/ชานม 5.☐อาหารจนอิ่มมาก 6.☐โทรศัพท์ทาง wifi เช่น line นาน..... นาที	1.☐ ไม่ใช่ 2.☐ เครื่องดื่มแอลกอฮอล์ 3.☐ กาแฟ 4.☐ ชา/ชาขวด/ชานม 5.☐อาหารจนอิ่มมาก 6.☐โทรศัพท์ทาง wifi เช่น line นาน..... นาที
6. ก่อนเข้านอนท่าน ทำกิจกรรม ดังนี้ (ตอบได้มากกว่า 1 ข้อ)	1.☐ ไม่ใช่ 2.☐ ดูทีวีน่ากลัว ตื่นเต้น 3.☐ เล่นเกมส์คอมพิวเตอร์ 4.☐ ทำงานคอมพิวเตอร์ 5.☐ อ่านหนังสือ 6.☐ ฟังเพลงเบาๆ	1.☐ ไม่ใช่ 2.☐ ดูทีวีน่ากลัว ตื่นเต้น 3.☐ เล่นเกมส์คอมพิวเตอร์ 4.☐ ทำงานคอมพิวเตอร์ 5.☐ อ่านหนังสือ 6.☐ ฟังเพลงเบาๆ

Appendix D

Publication

The current issue and full text archive of this journal is available on Emerald Insight at:
www.emeraldinsight.com/2586-940X.htm

Cutting-edge technology and nocturnal headaches in adolescent smart phone users in Chiang Mai, Thailand

A time series study

Received 23 January 2019
Revised 31 March 2019
Accepted 9 April 2019

Wanna Chongchitpaisan, Phongtape Wiwatanadate, Surat Tanprawate and Assawin Narkpongphun
Faculty of Medicine, Chiang Mai University, Chiang Mai, Thailand, and
Nipapon Siripon
Faculty of Engineering, Chiang Mai University, Chiang Mai, Thailand

Abstract

Purpose – Nocturnal headaches among adolescents were reported to be increased with the development of modern technology. The purpose of this paper is to investigate the smartphone electromagnetic radiation related to nocturnal headaches among high school students.

Design/methodology/approach – The time series study of all 12,969 records from 145 high school students Chiang Mai Province was selected from the population in the first phase by setting criteria. The samples completed a headache diary utilizing a smartphone application. The smartphone output power (SOP) was measured and recorded by the smartphone application and transmitted by e-mail to a researcher. The smartphone use, sleep quality, anxiety and depression also were assessed. Data were analyzed using Generalized Estimating Equation adjusting for demographic data, smartphone use, and sleep quality and otherwise.

Findings – The resulted showed the prevalence of repeated headaches to be 13.4 percent, nocturnal headache only 5.3 percent and the strongest effect of day time SOP at a $1.80–1.99\times10^{-5}$ mW range on nocturnal headaches (OR_{adj}5.18; 95% CI: 3.44–7.81). Meanwhile, Lag_6 of daily SOP exposure produced a nocturnal headache effect in a reverse dose-response manner. Furthermore, the nocturnal headache also had the strongest association with age, internet use and device brand (OR_{adj}2.33; 95% CI: 1.08–5.05, OR_{adj}2.14; 95% CI: 1.07–4.2 and OR_{adj}1.68; 95% CI: 1.1–2.4).

Originality/value – The electromagnetic radiation from a smartphone is the environmental variables influences on headache. The results suggested that there should be limited times for smartphone use and older age to start using a smartphone to prevent headache attacks at night.

Keywords Cutting-edge technology, Nocturnal headache, Smartphone output power

Paper type Research paper

Introduction

Nocturnal headaches are headache symptoms that occur at night. A previous study found headaches at night were usually associated with sleep-disorders that were linked to a bidirectional way that shared some pathophysiological mechanisms[1, 2]. The prevalence of

The authors gratefully acknowledge the Faculty of Medicine, Chiang Mai University, for financial support for this study (Research Grant ID: COM-2558-05516), and also the teachers and students in Wattanothai Payap School for their engagement and help with the data collection required for this study.

Journal of Health Research
Emerald Publishing Limited
2586-940X
DOI 10.1108/JHR-01-2019-0013

frequent nocturnal headaches at more than one per week ranged at around 8.4 percent in the general population[3]. Adolescents tend to have a delayed circadian preference and are "night owls." This change occurs in association with puberty; more physically mature adolescents have a preference for later bedtimes and may have a lower homeostatic sleep drive, consequently experiencing increased night time activities[4]. Furthermore, adolescents living in the digital age are in the group that ranks in the top three of the highest possession and use of smartphones in Thailand[5], with frequent usage at night.

Smartphones are modern mobile phones which have an electromagnetic emission source often held closest to the head and that can affect the human nervous system. Human nerves can be described as the electrical parts of human bodies and, as such, they are far from being in a state of equilibrium[6]. Previous studies found adolescents reporting that being awakened by a smartphone during the night at least once a month was associated with an increase in ill health symptoms such as headache and physical ill-being[7–9]. Furthermore, many of the recent pain-related studies reported activation by light[10] with results showing increases in cortical excitability during migraine attacks and visual hyper-excitability. The pain pathway begins with the intrinsic light cells that transform the light absorbed by the eye into a painful stimulus[11].

However, the effect of electromagnetic radiation from a smartphone on nocturnal headaches remains unclear. In addition, a previous study has shown that adolescents who attempted to reduce mobile phone during the day were more likely to stay up later at night using their mobile phones and being distracted by it[7]. This study attempts to assess the effect of smartphone electromagnetic radiation on nocturnal headache, the results of which will provide data for recommending safe smartphone use and reducing headaches.

Materials and methods

Study design and participants

This prospective time series study was conducted among high school students in Chiang Mai Province during October–December 2015. The samples were composed of grades 10–12 high school students who were selected from the first phase of study based on the set criteria. The inclusion criteria were that subjects should have no daily health-related behaviors, including liquor, coffee or tea consumption and smoking, have no disease or health problems diagnosed by doctors and should not be undergoing treatment.

Data collection and headache measurement

The students recorded data every day over a period of two to four months (60–120 days) in the daily questionnaires which were sent to the researcher from a smartphone application. The questions in the headache diary consisted of the time when a headache has begun and stopped, characteristics of pain and details of telephone conversations by the internet and hands-free or speakerphone use. The time cycle headache was classified by the time of headache in four time periods (6 h), morning time 6:01–12:00 a.m., daytime 12:01–18:00 p.m., evening time 18:01–24:00 p.m. and nocturnal time 0:00–6:00 a.m. The Pittsburg Sleep Quality Index, anxiety, depression and smartphone use were also assessed by diary questionnaires. The daily questionnaires were tested amongst 30 students in order to have reliable results by calculating internal consistency from Cronbach's α, that is, 0.775. The researcher trained the students about daily recording in a smartphone application through a three-day recording practice.

The smartphone output power measurement

The smartphone output power (SOP) was measured from the smartphone antenna and the application requested access to SOP via the program's framework by setting to save every 5 min

and transmitting saved data by e-mail to the researcher every day. The mean of SOP was collected from the measurements taken at 5-min intervals for 15 min. The mean of daily dosage data was collected by applying the average time of exposure equation in the OET Bulletin 56 of the Federal Communications Commission[12] as follows:

$$\text{Daily Dose} = \sum_{n=1}^{n} (\text{Average Output Power})n \; x(\text{Duration Time})n,$$

where n is the number of minutes measuring the average smartphone's output power. The duration is the time of measuring smartphone power each time. The study took measurements every 15 min. The SOP was continuous data with non-normal distribution and divided into three groups of range.

Statistical analysis

The sample size was calculated based on a 10 percent prevalence of headaches by mobile phone use[12]. A total of 996 high school students made up the population for questionnaire interviews in the first phase of the study, and 200 students were selected by inclusion and exclusion criteria such as not being obese, having no daily health-related behaviors including liquor, coffee or tea consumption and smoking and having no disease or health problems diagnosed by doctors and undergoing treatment. To fill in the missing information, the researcher has extended the time for information collection from 60 to 120 days. In sum, 145 students completed data comprising 12,969 records which were coded and analyzed using Statistical Package for Social Science software version 20 to obtain the frequency, arithmetic mean and standard deviation. Relationships between SOP and nocturnal headaches, odds ratio (OR) and their 95% confidence intervals (95% CI) were investigated with p-value of < 0.05 considered to be statistically significant. The Generalized Estimating Equation (GEE) was also run to control the confounding effects of such factors as demographic data, coffee or tea drinking, anxiety, depression, smartphone use and sleep quality. The GEE was used for data in the same cluster. In the analysis, therefore, the correlational structure was set and considered by the low score of Quasi-Likelihood under Independence Model Criterion. The Corrected Quasi-likelihood under Independence Model Criterion (QICC) has been used to compare the models under one correlational structure. A lower QICC score will correspond to a model of a better fit.

Ethical considerations

The study was approved by the Ethics Committee for Human Research, Faculty of Medicine at Chiang Mai University (COM 2558-03316). Informed consent was obtained from all participants.

Results

The 200 samples were asked to record headaches daily in a smartphone application. The SOP measured by another application was sent via a daily e-mail. Finally, 12,696 observations were obtained from a total of 145 students. The study found that the majority of the samples were female, 17.4 years old on average with a normal health condition. The headaches occurred in the morning, day time and in the evening accounting for 32.1, 30.2 and 32.4 percent of the participants, respectively, while only 5.3 percent reported a headache at night (Table I).

The data on SOP has been adjusted considering the value of error measured from each device brand to normalize the value for all device brands. The SOP values were aggregated into four time periods (6 h), morning time 6.01–12.00 a.m., day time 12.01–18.00 p.m., evening time 18.01–24.00 p.m. and nocturnal time 0.01–6.00 a.m. The SOP values of each day were aggregated into a daily dose which was on average $2.08 \pm 16.2 \times 10^{-3}$ mW (Table II).

Time cycles headache	*n* (%)
Morning headache	
Yes	547 (32.1)
No	1,158 (67.9)
Daytime headache	
Yes	515 (30.2)
No	1,190 (69.8)
Evening headache	
Yes	553 (32.4)
No	1,152 (67.6)
Nocturnal headache	
Yes	90 (5.3)
No	1,615 (94.7)

Table I.
Time cycles headache of participants presented as a percent

Variables	*n*	Min.	Max.	Mean	SD
Sum nocturnal dose	12,696	0	1.54703000	0.0010027961	0.01452703607
Sum morning dose	12,696	0	0.60338196	0.0011027388	0.00644747700
Sum daytime dose	12,696	0	0.36942911	0.0011809051	0.00618567980
Sum evening dose	12,696	0	0.4080357	0.001072926	0.0065726714
Daily dose	12,696	0.00000009	1.54872780	0.0020833594	0.01623557023

Table II.
Smartphone output power (SOP) by time cycles and a daily dose of SOP

Apparently, the average SOP was the highest during day time, 1.18×10^{-3} mW, followed by morning time 1.1×10^{-3} mW, while the lowest is during night time, 1.0×10^{-3} mW. However, the maximum value of SOP occurred during night time at 1.55 mW. The SOP value observations were then divided into three ranged groups: ⩽1.79, 1.8–1.99 and ⩾2.0×10^{-5} mW (Table III). The SOP in the 1.8–1.99×10^{-5} mW range appeared to be the least prevalent, with only 2.4 percent of the observations, taking place mostly during night time.

The researcher conducted a statistical test to evaluate the confounding effects and the relationship between various factors and found no interaction effect existed among them. Additional computation was made to adjust the effects of such potential confounders as demographic characteristics, and smartphone use. Autoregression 1 (AR1) was set as the correlational structure due to its lowest QIC (Table IV). The results revealed that younger aged users, internet use, and the brand of the device were associated with nocturnal headache (OR_{adj}1.68; 95% CI: 1.10–2.40, OR_{adj}2.14; 95% CI: 1.07–4.25 and OR_{adj}2.33; 95% CI: 1.08–5.05). Not using hands-free and internet use had a strong association with morning headache (OR_{adj}2.62; 95% CI: 1.59–4.32 and OR_{adj}1.91; 95% CI: 1.44–2.54), day time headache (OR_{adj}3.01; 95% CI: 1.67–5.42 and OR_{adj}1.88; 95% CI: 1.39–2.55) and evening headache (OR_{adj}3.02; 95% CI: 1.67–5.49 and OR_{adj}2.62; 95% CI: 1.93–3.56). While the OR of nocturnal headache and lag were adjusted for all other factors using GEE, the exchangeable was set as the correlational structure

Output power ($\times 10^{-5}$ mW)	Daily dose *n* (%)	Morning *n* (%)	Daytime *n* (%)	Evening *n* (%)	Nocturnal *n* (%)
⩽1.79	1,943 (15.3)	3,597 (31.4)	2,479 (20.1)	2,303 (18.8)	2,648 (20.9
1.8–1.99	186 (1.5)	226 (2.0)	120 (1.0)	79 (0.6)	301 (2.4)
⩾2.0	10,567 (83.2)	7,646 (66.7)	9,710 (78.9)	9,896 (80.6)	9,747 (76.8)

Table III.
Smartphone output power group by time cycles and daily dose

due to its lowest QIC (Table V). This study found daytime lag_2 of SOP in $1.80–1.99 \times 10^{-5}$ mW range to have a stronger association with nocturnal headache (OR_{adj}5.18; 95% CI: 3.44–7.81) compared to $\geq 2.00 \times 10^{-5}$ mW. Meanwhile, lag_6 daily SOP had a relationship with nocturnal headache in the form of a reverse dose-response, while the SOP in $\leq 1.79 \times 10^{-5}$ mW range is related to daytime and evening headache (OR_{adj}1.52; 95% CI: 1.10–2.11 and OR_{adj}2.60; 95% CI: 1.36–4.97). The relationship between morning headache and SOP (OR_{adj}194.11; 95% CI: 1.22–30821.27) will appear in the form of a dose-response.

Discussion

This study showed the prevalence of nocturnal headache to be only 5.3 percent, which was consistent with findings in Brazil in 2009, i.e. 8.4 percent[3]. The nocturnal headache in the present study is different from hypnic headache, as it does not wake one up from sleep, but it just occurs at night (00.01–6.00 a.m.).

The study showed that a young student compared to a student a year older was likely to face a relatively greater degree of nocturnal headache. This result was in contrast with most previous studies which found headache prevalence to vary positively with age[13]. The results from the present study are in line with those found from the first phase investigation on factors associated with a headache from mobile phone use that younger age groups had implications for mobile phone associated headache. Previous surveys revealed as high

Factor	Nocturnal headache Yes	No	Crude OR	Adjusted OR	95% CI Lower	Upper	*p*-value
Age mean ± SD	16.9 ± 0.8	17.4 ± 1.0	1.76	1.68	1.1	2.4	< 0.01
Lag_6 dose ($\times 10^{-3}$ mW)							
Mean ± SD	0.8 ± 1.7	2.1 ± 16.0	1.84×10^{-35}	1.03×10^{-39}	9.15×10^{-74}	1.17×10^{-5}	0.03
Total	90	12,600					
Internet use							
Yes/no	1,416/11,280	1.5/0.6	2.13	2.14	1.07	4.25	0.03
Brand device							
Other/Apple	9,170/3,526	0.8/0.4	2.40	2.33	1.08	5.05	0.03
Daytime dose group ($\times 10^{-5}$ mW)							
1.80–1.99/≥2.00	79/9,896	5.1/0.7	6.97	5.18	3.44	7.81	< 0.01

Note: Adjusted by age, BMI, vision, anxiety, depression, PSQI, internet use, hand free use, brand device and SOP

Table IV. Odds ratios (OR) of nocturnal headache (24–6.00 a.m.) and their 95% confidence intervals for each factor and the daily dose adjusted for all other factors using GEE (AR1, QIC = 899.92, QICC = 895.28)

Parameter	Exp. (*B*)	95% CI Lower	Upper	*p*-value	Correlation structure	QIC	QICC
Nocturnal headache							
Lag_1	7.415E-51	1.122E-100	0.490	0.05	Exchangeable	921.611	913.392
Lag_4	7.608E-38	9.118E-73	0.006	0.04	Exchangeable	921.819	914.458
Lag_6	7.335E-40	2.899E-82	1,855.887	0.07			
Lag_6	1.032E-39	9.147E-74	1.165E-05	0.03	Exchangeable	921.492	914.215
Lag_2 (12–18 p.m.)	5.184	3.441	7.809	< 0.01	AR1	899.919	895.279

Note: Adjust by age, BMI, vision, anxiety, depression, PSQI, internet use, hand free use, brand device and SOP

Table V. Odds ratio (OR) of nocturnal headache and their 95% confidence intervals for lag dose adjusted for all other factors using GEE

as 31 percent of children between the age of eight and ten own and use mobile phones[14]. A study in Korea in the year 2013 found the average age of children first owning and/or using mobile phone decreased from 12.5 years old in 2008 to 8.4 years old in 2011. The results implied the tendency of children to own or use mobile phones at a younger age[14].

Internet use is a risk factor for nocturnal headaches (OR_{adj}2.14; 95% CI: 1.07–4.25). Talking on smartphones in both internet and cellular modes often involves holding the device close to the head, and the electromagnetic radiation from smartphone to which the users are exposed induces a change in biological reaction including a change of protein in the brain and causes nervous system problems, especially headache symptoms[15]. Electromagnetic radiation from talking mode is nine times more intense than the standby mode[16]. A recent study has found a higher mean of radiated power during voice over internet protocol, which has been assessed at 1.9 mW, than the mean of radiated power during voice over circuit switch calls, which has been assessed at 0.55 mW[17]. The results indicate that talking on smartphones without hands-free devices can give rise to a headache.

The brand of the device has appeared to have a bearing on the nocturnal headache. From the analysis of smartphone use during night time, it was found among late night users that students using device brands other than Apple, most (83.6 percent) used SOP in the $\geqslant 2.00 \times 10^{-5}$ mW range, compared to the 58.9 percent figure of Apple brand device users. The result implies that users of smartphones other than Apple brand use the device heavily at night, thus contributing to the link between the device brand and nocturnal headache. It is important to note that device brand is a representative of areas where the device is used, and the device brand used popularly in rural areas, which has less density of the base station, will have an effect on sleep quality. The theory is in line with the findings from previous studies that the factors governing SOP include the control system of the operator's network, the wave frequency, the strength of the signal which depends on the signal density of the base station, the distance of the mobile phone from the base station and population density[18, 19].

The SOP, which was measured and stored in the device, can be viewed with the use of an application. SOP values in this study are thus lower than the values of smartphone electromagnetic radiation in other studies which used the external metering device and might be affected upward by the radiation from other sources. This value concurred with the average power consumption of a human cell, at 1×10^{-9} mW[20]. In this study, the maximum SOP was 1.55 mW which occurs during night time. Previous studies indicated most teenagers (62–72 percent) used advanced smartphones in the evening and at night, after 9.00 p.m., and during midnight to 3.00 a.m., with 34–55 percent of the use for texting and social media, and 24 percent for playing games[21].

The use of SOP in the morning (6:00–12:00 a.m.) and at night (00:00–6:00 a.m.) in the form of high power effect ($\geqslant 2.00 \times 10^{-5}$ mW) was found to link with morning headache and indicates that students with a morning headache will include those who use a smartphone heavily after midnight. Severe morning headache is not only the consequence of exposure to high doses of SOP but also due to sleep deprivation. Meanwhile, the use of SOP during the day time and in the morning ($\leqslant 1.79 \times 10^{-5}$ and $1.80–1.99 \times 10^{-5}$ mW, respectively) in the form of power effect has been found to induce a daytime headache. Furthermore, the use of a daily dose of SOP for six days apparently caused daytime headache in the form of a dose-response. The use of SOP in the evening ($\leqslant 1.79 \times 10^{-5}$ and $\geqslant 2.00 \times 10^{-5}$ mW) has been associated with an evening headache. Moreover, the exposure to daytime SOP for 6 h ($\leqslant 1.79 \times 10^{-5}$ and $\geqslant 2.00 \times 10^{-5}$ mW) in the form of power effect and a daily dose of SOP for five days has given rise to an evening headache in the form of a dose-response. The researcher has observed that SOP in $\leqslant 1.79 \times 10^{-5}$ mW range, which is the lowest level that can trigger a headache, still has a strong effect probably due to the sensitivity of each individual or the response of the nervous system to the frequency of smartphone electromagnetic radiation in this range.

The nocturnal headache in the present study is different from the hypnic headache as it does not wake one up from sleep but it only occurs at night and has been found in only 5.3 percent of the participants. From the study on headache in different periods of the day, a nocturnal headache, mostly, can be classified as a migraine type, i.e. 12.2 percent (Table VI). The researcher has found that nocturnal headache is not a response to SOP during the evening, before bedtime. The finding indicates that using a smartphone before going to bed does not stimulate the brain, to result in a nocturnal headache. Meanwhile, daytime smartphone use with SOP at $1.80–1.99 \times 10^{-5}$ mW range and perhaps in combination with night time smartphone use can bring about a nocturnal headache. Furthermore, using a daily dose of SOP for seven days or delayed effect has been found to have nocturnal headache consequence in a reverse dose-response form, which is likely to be the adaptive process of the nervous system, particularly in a migraine type headache. The results from one study showed other kinds of protective response, for example, photophobia[22]. The researchers' findings ensure that both migraine and nocturnal headache will have specific responses to SOP. Noseda *et al.* found that light stimulations activated migraine by dura sensitive thalamic neurons that receive photic signals from the retinal ganglion cells and transmit signals to cortical areas and nociceptive. The retino-thalamic-cortical pathway provided exacerbation of migraine headache by light[23–25]. The light of smartphone displays is light-emitting diodes (LED) for backlit screens. These screens are lit in the back by short wavelength LEDs (460 nm)[26] that are sensitive to photoreceptors and can stimulate the retino-thalamic-cortical pathway. The information ensures that nocturnal headache in the study has been activated by output power and the light from smartphones.

Measuring smartphone's output power by using the data in the smartphone, not measuring from outside, can lead to misclassification of exposure. This study was a panel study, meaning the outcomes and exposures have been followed in the same sample groups which have been considered as controlling individual and environmental confounders. The tool of this study relied on the technology by creating a smartphone application which used recorded data every day and avoided recall bias. Finally, this study had a large sample size which can make even an analysis of the effect of slight SOP on the nervous system possible.

Conclusion

SOP, which is smartphone electromagnetic radiation, had a non-linear correlation with headaches during different periods of the day. Nocturnal headaches have been found to respond to the delayed effect of the daily dose of SOP in the form of a reverse dose-response, just like migraine headaches which have some other kinds of protective response, for example, photophobia. The information ensures that the nocturnal headache in this study is a migraine which has been activated by output power and the light from smartphones. Finally, for younger students, internet use has been a risk factor of the nocturnal headache. It is recommended that a limited time for smartphone use and delaying the use of smartphones to older age groups should be implemented in order to prevent migraine attacks at night.

Time cycle headache	Headache type Migraine *n* (%)	TTH *n* (%)	Undetermined *n* (%)	Total *n* (%)
Morning headache	61 (11.2)	382 (69.8)	104 (19.0)	547 (32.1)
Daytime headache	60 (11.7)	360 (69.9)	95 (18.4)	515 (30.2)
Evening headache	21 (3.8)	460 (83.2)	72 (13.0)	553 (32.4)
Nocturnal headache	11 (12.2)	62 (68.9)	17 (18.9)	90 (5.3)

Table VI. Time cycles headache classified by headache type

References

1. Pankaj BK. Primary nocturnal headache. Ann Neurosci. 2009; 16(1): 37-9.
2. Lin YK, Lin GY, Lee JT, Lee MS, Tsai CK, Hsu YW, *et al.* Associations between sleep quality and migraine frequency a cross-sectional case-control study. Medicine (Baltimore). 2016 Apr; 95(17): 1-7.
3. Lucchesi LM1, Speciali JG, Santos-Silva R, Taddei JA, Tufik S, Bittencourt LR. Nocturnal awakening with headache and its relationship with sleep disorders in a population-based sample of adult inhabitants of São Paulo City, Brazil. Cephalalgia. 2010; 30(12): 1477-85.
4. Tarokha L, Jared M, Saletinc JM, Carskadonc MA. Sleep in adolescence: physiology, cognition and mental health. Neurosci Biobehav Rev. 2016; 70: 182-8.
5. Electronic Transactions Development Agency. Thailand internet user profile 2013. Bangkok: Electronic Transistors Development Agency; 2013.
6. Lu Y, Huang Y. Biological effects of mobile phone radiation, *IEEE International Conference on Microwave and Millimeter Wave Technology (ICMMT)*, Shenzhen, China, May 5-8. 2012; 2, doi: 10.1109/ICMMT.2012.6230101
7. Kumar P, Dongre K, Ghail G. Prevalence of subjective symptoms in mobile users aged more than 18 years in Nagpur. Indian J Physiother Occup Ther. 2013; 7(2): 260-4.
8. Schoeni A, Roser K, Röösli M. Symptoms and cognitive functions in adolescents in relation to mobile phone use during night. PLOS One. 2015; 10(7): 1-15.
9. Nathan N, Zeitzer J. A survey study of the association between mobile phone use and daytime sleepiness in California high school students. BMC Public Health. 2013; 13(840): 1-5.
10. Boulloche N, Denuelle M, Payoux P, Fabre N, Trotter Y, Geraud G. Photophobia in migraine: a PET study of visual cortex hyperexcitability and its modulation by pain. JNNP. 2010; 81(9): 974-84.
11. Ali EN. Excitability and photosensitivity in neurological disorders using pupsillary response to sparse multifocal stimuli. Canberra: Australian National University; 2016.
12. Chu MK, Song HG, Kim C, Lee BC. Clinical features of headache associated with mobile phone use: a cross-sectional study in university students. BMC Neurol. 2011; 11(115): 1-7.
13. Rho Y-I, Chung H-J, Lee K-H, Eun B-L, Eun S-H, Nam S-O, *et al.* Prevalence and clinical characteristics of primary headachesamong school children in South Korea: a nationwide survey. Headache. 2012 Apr; 52(4): 592-9.
14. Byun YH, Ha M, Kwon HJ, Choi KH, Burm E, Choi Y, *et al.* Epidemiological characteristics of mobilephone ownership and use in Korean children and adolescents. Environ Health Toxicol. 2013 Dec; 28: 1-8.
15. Sudan M. Cell phone exposures and headaches, hearing loss, and behavioral problems in children. University of California; ProQuest 789 East Eisenhower Parkway. 2012.
16. Hunga C-S, Anderson C, Horne JA. Mobile phone 'talk-mode' signal delays EEG determined sleep onset. Neuroscience Letters. 2007; 421: 82-6.
17. Jovanovic D, Bragard G, Picard D, Chauvin S. Mobile telephones: a comparison of radiated power between 3G VoIP calls and 3G VoCS calls. J Expo Sci Environ Epidemiol. 2015; 25: 80-3.
18. Hillert L, Ahlbom A, Neasham D, Feychting M, Järup L, Navin R, *et al.* Call-related factors influencing output power from mobile phones. J Expo Sci Environ Epidemiol. 2006; 16(6): 507-14.
19. Kelsh MA, Shum M, Sheppard AR, McNeely M, Kuster N, Lau E, *et al.* Measured radiofrequency exposure during various mobile-phone use scenarios. J Expo Sci Environ Epidemiol. 2011; 21: 343-54.
20. Wikipedia. Orders of magnitude (power). [cited 2018]. Available from: https://en.wikipedia.org/wiki/Orders_of_magnitude_(power)
21. Bruni O, Sette S, Fontanesi L, Baiocco R, Laghi F, Baumgartner E. Technology use and sleep quality in preadolescence and adolescence. J Clin Sleep Med. 2015; 11(12): 1433-41.

22. Borsook D, Maleki N, Becerra L, McEwen B. Understanding migraine through the lens of maladaptive stress responses: a model disease of allostatic load. Neuron. 2012; 73: 219-34.
23. Noseda R, Bernstein CA, Nir RR, Lee AJ, Fulton AB, Bertisch SM, *et al.* A retinal-thalamic pathway for photophobia during migraine. Cephalalgia. 2009; 29: 2-3.
24. Noseda R, Burstein R. Migraine pathophysiology: anatomy of the trigeminovascular pathway and associated neurological symptoms, CSD, sensitization and modulation of pain. Pain. 2013; 154(1): 1-21.
25. Noseda R, Kainz V, Jakubowski M, Gooley JJ, Saper CB, Digre K, *et al.* A neural mechanism for exacerbation of headache by light. Nat Neurosci. 2010; 13(2): 239-45.
26. Münch M, Kobialka S, Steiner R, Oelhafen P, Wirz-Justice A, Cajochen C. Wavelength-dependent effects of evening light exposure on sleep architecture and sleep EEG power density in men. Am J Physiol Regul Integr Comp Physiol. 2006; 290: 1421-8.

Corresponding author
Phongtape Wiwatanadate can be contacted at: pwiwatanadate@gmail.com

Mail

Re: Acceptance Notification-1000112557

PW **Phongtape Wiwatanadate <pwiwatanadate@gmail.com>**
8/8/2019 2:58 PM

To: Wanna Apple

ไม่ทราบได้ดำเนินการหรือยัง ส่งมาให้เพื่อ make sure ครับ

พงศ์เทพ

On Wed, Jul 31, 2019 at 3:08 PM journal@sciepub.com <journal@sciepub.com> wrote:

Dear Phongtape Wiwatanadate,

Thanks for your contribution to ***American Journal of Public Health Research***

We are pleased to inform you that your paper has been accepted for publication.
Paper ID: 1000112557
Paper Title: New Trend of Headaches among High School Students affected by Smartphone Electromagnetic Pollution Exposures: A Time Series Study

Please sign in SciEP Online Manuscript Tracking System http://mts.sciepub.com/ and make sure the following steps are completed **before August 10, 2019.**
1. Sign your name on the Copyright Transfer Form
2. Pay the Publication Fee and send me the payment receipt. **The fixed expense of your paper is USD $290**
3. Whether your article needs to be modified or not (according to the review comments), please send the final paper (both word and PDF version) to Emma Taylor: journal@sciepub.com / sciepjournal@hotmail.com.
4. We'll prepare the proof for you to check based on the final paper you sent after we receive the fee, so please send the final paper before, or soon after you make the payment. If we do not receive the final paper within three days after receiving the payment, we will use the original draft you submitted.

For more detailed information, please refer to the "Acceptance Notification" at SciEP Online Manuscript Tracking System.

Thank you for your cooperation and support.
Please feel free to contact me if you have any questions.

I'll look forward to hearing from you soon.

Best regards,
Philip Smith

Emma Taylor: journal@sciepub.com / sciepjournal@hotmail.com
Philip Smith: billing@sciepub.com / sciepubpayment@yahoo.com
Online Manuscript Tracking System (http://mts.sciepub.com/author/index)
Science and Education Publishing (www.sciepub.com)

Biography

Author's Name	Mrs. Wanna Chongchitpaisan
Date/Year of Birth	3 November 1965
Place of Birth	Samutprakarn Province, Thailand

Education

1988	Bachelor of Nursing Science Bachelor of Nursing Science Program Boromrajonani College of Nursing
1994	Bachelor of Public Health B.P.H Bachelor of Public Health program in Occupational Health and Safety Sukhothai Thammathirat Open University
2003	Master of Science Occupational Medicine Chulalongkorn University
2009	Medical Preparedness & Response National Radiation Emergency Medical Center Seoul Korea
2008	Environmental and Occupational Medicine National Taiwan University Hospital
2007	Occupational Medicine UOEH in Japan
2006	Asian Asbestos Conference2006 Bangkok Thailand
1999	Certificate of Nursing Specialty in occupational health Burapa University
1997	Certificate of basic health nursing 60 hours Occupational Health Department

Publications

Chngchitpaisan W, Sitisarankul P, Bandhukul A. Occupational health practice for healthcare workers compared to hospital accreditation criteria in Thai governmental hospital, 2004. Journal of medicine and Health Sciences. 2004; 13(3): 235-47.

Chngchitpaisan W, Wiwatanadate P, Electromagnetic radiation from smartphone and its biological effect. Journal of medicine and Health Sciences. 2016; 23(2):26-35.

Experience

Nurse in charge of the Internal Medicine ward 1988-1998

2005-NOW Head of occupation health nurse of academic nurse section in nursing cluster Nopparatrajathanee Hospital, Department of Medicine Ministry of Public Health

Survey of using Antidote in hospital, Thailand 2005

Health status and Injury of healthcare workers in Nopparatrajathanee Hospital 2006

Occupational health service in factories around hospital by ISO 18000 criteria 2007

Occupational health for Diabetes workers in factories 2007

Occupational medicine practice for physicians from occupational medicine training program 2007

Occupational health service for healthcare workers in the hospitals of Department of Medicine 2007

Job stress of the healthcare workers in Nopparatrajathanee Hospital 2010

Occupational disease clinic practice in Thailand 2009-2010

Documentation

2011 Editorial board for Textbook of Occupational medicine First edition

2011 Co-writer of Occupational Health for Healthcare worker in Textbook of Occupational medicine

2011	Co-writer of Screening test in occupational Medicine: Pulmonary function test, Vision test and Hearing test in Textbook of Occupational medicine Co writer of Guideline for Occupational Hazard Prevention for Healthcare worker

LIST OF PUBLICATION

1. Chongchitpaisan W, Wiwatanadate P, Tanprawate S, Narkpongphun A, Siripon N. Cutting-edge technology and nocturnal headaches in adolescent smart phone users in Chiang Mai, Thailand: A time series study. JHR. https://doi.org/10.1108/JHR-01-2019-0013. 2019.

2. Chongchitpaisan W, Wiwatanadate P, Tanprawate S, Narkpongphun A, Siripon N. New Trend of Headaches among High School Students Affected by Smartphone Electromagnetic Pollution Exposures: A Time Series Study. American Journal of Public Health Research. 2019: 7; no. x : XX-XX. doi: xxxxxxxx.