DISCUSSIONS

A number of light microscopic cytological investigations of the basic organization of the insect brain, such as the grasshopper (*Locusta*) or honey bee (*Apis*), were referred to by Mobb (1985). In fact, early studies on the brain tended to be restricted to examinations of the cells and tracts of particular regions, with most centering on the ganglia and connectives of the nerve cord, and not usually including the details of more general characteristics.

This investigation provided the brain anatomy of the blowfly, *Chrysomya megacephala.* Three planes of hematoxylin-eosin sections of the adult male brain of *C. megacephala* revealed the brain compartments of this fly species. The most obvious vertical plane exhibited more information on the brain neuropils than the others. This orientation was appropriate for describing various features of the nervous system, since structurally homologous areas occupied similar positions.

Based on the electron microscope study, three types of the blow fly neuron described here may be compared with the three of five conventional neurons of the housefly, *Musca domestica* (Sohal *et al*., 1972): The Type 1 blowfly neuron was small and had dense patches of chromatin in the nucleus, while Type 2, the smallest neuron, had both a diffused and dense form of chromatin. These two types coincided with the Type II and Type I neuron of the housefly, respectively; and Type 3 of the blowfly, which was bigger than the others with a primarily diffused form of chromatin, agreed with the Type III neuron of the housefly. Distended extracellular spaces, lined by glial cells, as described in the housefly brain were observed in the

blowfly. In addition, a large number of tracheoles, which determined in the blowfly brain, were not discussed in the housefly.

The functional distinctions between all three types of *C. megacephala* neuron were obscured, while the nutritive and supportive functions of the glial cells were documented (Sohal *et al*., 1972). It is well-known that the fly's brain has no vascular system, so substances only have access to the nerve cell by diffusion from the hemolymph through various layers including the glial sheath.

When the nerve cells were examined in senescent flies, it became apparent that nerve cell loss occurs during aging. This data disagreed with the observation of Sohal and Sharma (1972). The difference in nerve cell count methodology, variation in sampling, tissue processing technique, pathological histories of the organisms, and other factors should be considered as a result of discrepancy. This study, decreased number of Type 1 and Type 3 neurons in senile flies, except the Type 2 neuron, were agreed with more recent study (Rutten *et al*., 2003). Rutten and colleagues stated that only specific types of neurons had an age-related loss of cells. From the molecular and cellular markers of age-related alterations in the brain, which varied significantly between different brain regions and different types of neuron, it was assumed that some types of aging brain showed no accumulation of unrepaired nuclear DNA damage. Since cells with the greatest decline in nuclear DNA damage repaired capacity, the highest amount of nuclear DNA damage was lost during aging. However, many of the previous studies claimed that neuron loss does not appear to be an important contribution to age-related functional decline in animals (Rapp and Gallagher, 1996; Morrison and Hof, 1997). More investigations into this point are needed. The age-dependent brain degenerations were noticeable in senescent blowfly

as well as the housefly. The behavioral changes such as sluggish movement or inability to fly were monitored in old flies of both blowfly and housefly species. Sohal and Sharma (1972) suggested that the mechanism responsible for the deteriorative changes in neurons, including an involvement of brain degeneration in the behavioral changes of senile flies, is still unknown. Investigations carried out in recent years have summarized that three main mechanisms are responsible for the brain degenerative process in both mammals and invertebrates. Of these three mechanisms, the first is based on the existence of error associated with the pathways accountable for cell energy metabolism (Nomura, 1996). The second mechanism is the consequence of programmed cell death (Agid, 1995; Pavon *et al*., 1998; Jellinger, 2001), and finally, the formation of free radicals for different reasons (Sohal and Weindruch, 1996; Yan *et al*., 1998, 2000; Calabrese *et al*., 2001; Sugaya, 2001). These causes occur during aging and ultimately lead to neuronal damage.

The behavioral changes and brain degeneration involvement is still unclear. Behavior is the result of interactions between sensory input, reflexes and centrally generated neural patterns including the role of endocrine system (Chapman, 1998). Chapman also implied that the pattern of behavior during aging may occur spontaneously without any external sensory triggering. Thus, the nervous system must be capable of spontaneously generating organized patterns of behavior from within (endogenously).