I. INTRODUCTION

Penicillium marneffei (*P. marneffei*) is the only dimorphic species of the genus *Penicillium*. It is the etiological agent of penicilliosis marneffei, the disease involving infection of reticuloendothelial system, and rarely noted before the epidemic of the acquired immune deficiency syndrome (AIDS). *P. marneffei* infection has become more prevalent in the endemic areas, including Southeast Asia and Southern part of China. Penicilliosis marneffei is the third most common opportunistic infection after extrapulmonary tuberculosis and cryptococcosis in AIDS patients in northern Thailand (Supparatpinyo *et al.*, 1994). Molecular studies of this fungus have been more frequent since 2000. The several genes involving to the fungal morphogenesis (Borneman *et al.*, 2000; 2001; 2002; Boyce *et al.*, 2001; 2003; Zuber *et al.*, 2002; 2003; Todd *et al.*, 2003) and immunogenic proteins (Cao *et al.*, 1998a; Pongpom, 2004) were reported. However, the studies in adaptation of this fungus during stress in molecular level are still unclear.

The fungus grows as a multicellular saprobic mycelium at 25 ^{o}C and as a fission yeast at 37 ^{o}C. Dimorphism is an adaptive mechanism, whereby the fungus developing its saprobic existence in nature is able to adjust to the 37 ^{o}C temperature of its casual homeothermic host. During *in vitro* growth, phase transitions are triggered when the incubation temperature is shifted from 25 ^{o}C to 37 ^{o}C or in the reverse direction (Vanittanakom and Sirisanthana, 1997; Andrianopoulos, 2002). The mycelial to yeast phase transition is of particular interested because the conversion to the yeast form is thought to be important in the pathogenicity of dimorphic pathogenic fungi such as *Paracoccidioides brasiliensis*, *Histoplasma capsulatum*, *Blastomyces dermatitidis* and *Penicillium marneffei*. These fungi have the unique ability to colonize host tissues in parallel with the development of mold to yeast morphological transition. As mentioned earlier, *P. marneffei* is the only fungus among the numerous species of the genus *Penicillium* that possesses the dimorphic property (Cooper and McGinnis, 1997; Cooper and Haycocks, 2000). Therefore, adaptation process is

likely to be necessary to ensure survival of the fungus in the warm-blooded human host.

All living organisms are likely to respond to the environmental stress by rapidly producing increased amounts of heat shock proteins (Hsps). Expression of most Hsps is induced by heat and other stresses including pH, osmolarity, oxygen radicals, metabolic disruption and viral infection (Lindquist, 1986; Morimoto, 1993). Most Hsps function as chaperones; they participate in folding, assembly and disassembly of protein complexes. They also assist in translocation of proteins from one compartment to another. Although a number of Hsps have been identified, Hsp70 is one of the most abundant reports. Entry of a pathogen into a warm-blooded host is usually accompanied by a shift-up in temperature. It is therefore expected that heat shock genes may play an important role in their pathogenesis. It is assumed that heat shock genes are upregulated upon infection in order to prevent misfolding and aggregation of damaged proteins. These processes are probably essential for survival of the pathogen within the host. In our circumstances, none of studies was performed to demonstrate the correlation between *hsp70* expression and heat adaptation in *P. marneffei*. Therefore this study is straight forward to investigate the expression of *hsp70* in different stages of this fungus and stress conditions.

A previous study by Pongpom (2004), a cDNA library of yeast phase in this fungus was constructed. Clones containing part of *hsp70* were isolated by antibody screening method using anti-*Histoplasma capsulatum* Hsp70 monoclonal antibody. Analysis of DNA sequences obtained from one of these clones revealed that it was gene encoding Hsp70. In this study we would like to obtain the full length sequence of *hsp70* and characterize the gene at both mRNA and genome level. Additionally, the expression pattern of *hsp70* was investigated during phase transition and heat shock conditions.