No. B-13.1.1 Study on Physiological Effects of Heat Stress

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Total Buget for FY1990-FY1992 25,361,000 Yen

Abstract Global warming has potential impacts on human health during extreme high temperature in summer. The epidemiological results showed that hyperthermia caused by heat stress induced many heat stroke patients in Nanjing, China.

Since heat stress caused an increase in morbidity and mortality, oxygen radical damages in hyperthermia were studied. Since lipid peroxidation was greatly induced in liver, hyperthermia greatly developed hypertrophy and vacuolized degeneration in hepatic cells. Protective enzymes, such as glutathione peroxidase activities were induced in hyperthermia. Heat stress also affected central nervous systems and changed the sleeping behaviour.

Key Words Health Risk, Heat Stress, Heat Wave, Radical Damage, Central Nervous System

INTRODUCTION According to various scinarios of global warming, heat stress may have a profound impacts on human health in hot regions(1-3). Heat stress in hot summer affects directly morbidity, mortality and human health(4,5). Therefore as a result of global warming, the heat related disorders will increase in many regions.

To estimate the mechanism of adverse effects of heat stress on human health, experimental study using model animals is necessary.

Research Objective Since there is an urgent need for risk evaluation of global warming, epidemiological and experimental research works on pathogenesis of heat related disorders were carried out.

Subjects and Methods To evaluate the health risk of extreme heat stress in hot summer, cooperative epidemiological work were carried out in Nanjing. In Nanjing area, extreme heat stress in hot summer has frequently occured at heat waves.

To study the biochemical and physiological impacts of heat

stress on human health, animals were kept at 25 ± 0.5 °C, 30 ± 0.5 °C, and 35 ± 0.5 °C at 40 ± 10 % relative humidity. After exposure to various temperature, tissue was prepared for histochemical and biochemical analysis.

Result and Discussion A severe heat wave occured in July 1988 in Nanjing, China. As shown in Fig. 1, the daily mean temperature was above 30 °C during 18 successive days (mean temperature was 32.0 ± 0.95 °C during the heat wave).

This heat wave produced heat shock in about 4,500 persons, an incidence of 188 per 100,000. Of the 4,500 victims, 411 suffered severe heat stroke, 65.9 % of whom were elderly, and 124 died as shown in Fig. 1.

Exposure to various environmental temperature affected to central nervous system. Heat stress changed sleeping pattern and decreased episodes of non-REM sleep in 24 hr.

After exposure to 35 °C, hyperthermia was observed and rectal temperature in rats increased from 36.7 ± 0.20 °C to 37.7 ± 0.30 °C. Hyperthermia induced serious histochemical changes in liver. Hepatic cells around hepatic vein greatly developed hypertrophy and vacuolized degeneration.

A significant increase in lipid peroxidation was observed in hyperthermia. As shown in Fig.2, animals in hyperthermia induced production of TBARS in liver. Induction of lipid peroxidation was observed successively during heat stress. Free radicals can damage cellular redox state, intracellular structure, and some membrane bound enzymes.

In rat, glutathione peroxidase activities were greatly induced in hyperthermia as shown in Fig. 2. It was obvious that heat stress inducible glutathione peroxidase was selenium glutathione peroxidase. Other protective enzymes for oxygen radicals were not affected.

In guinea pig, hepatic mitochondrial electron transport system, such as cytochrome c oxidase and succinate-cytochrome c reductase system were simultaneously inhibited in hyperthermia.

From the biochemical studies on oxygen radical damage, liver may be one of the most sensitive target organs of heat stress(6).

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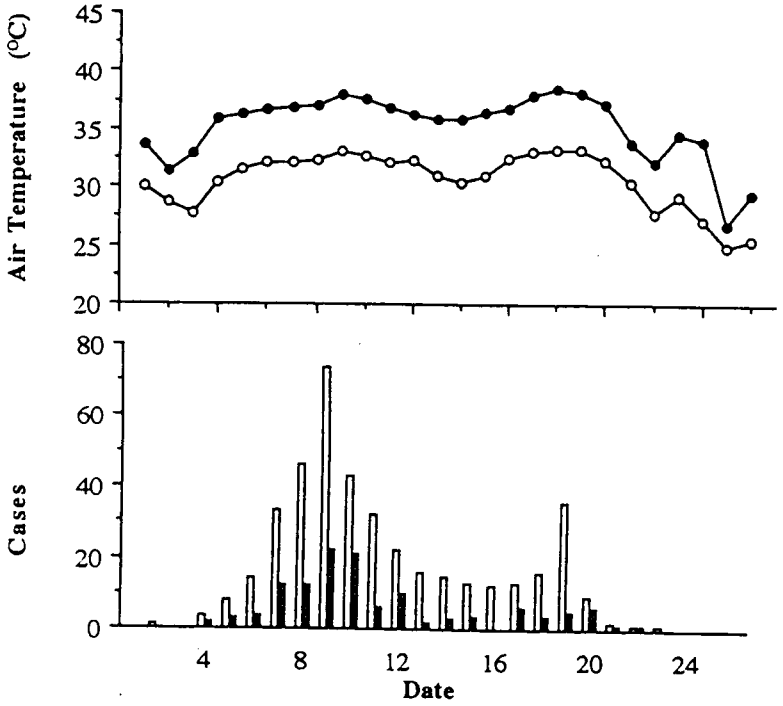

Fig.1 Maximum(●), mean temperature(○), severe heat stroke(□) and heat stroke death(▩) during heat wave in Nanjing at July 1988.

Fig.2