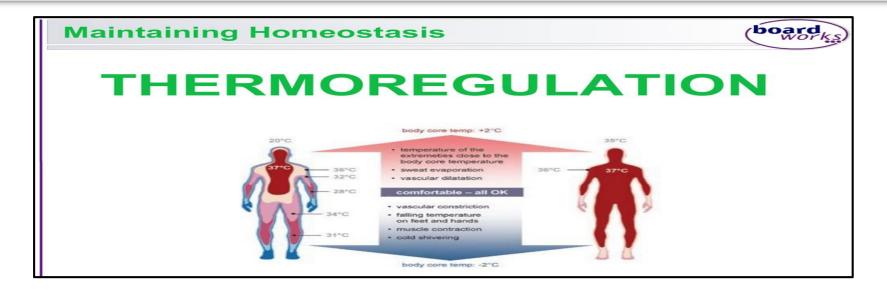
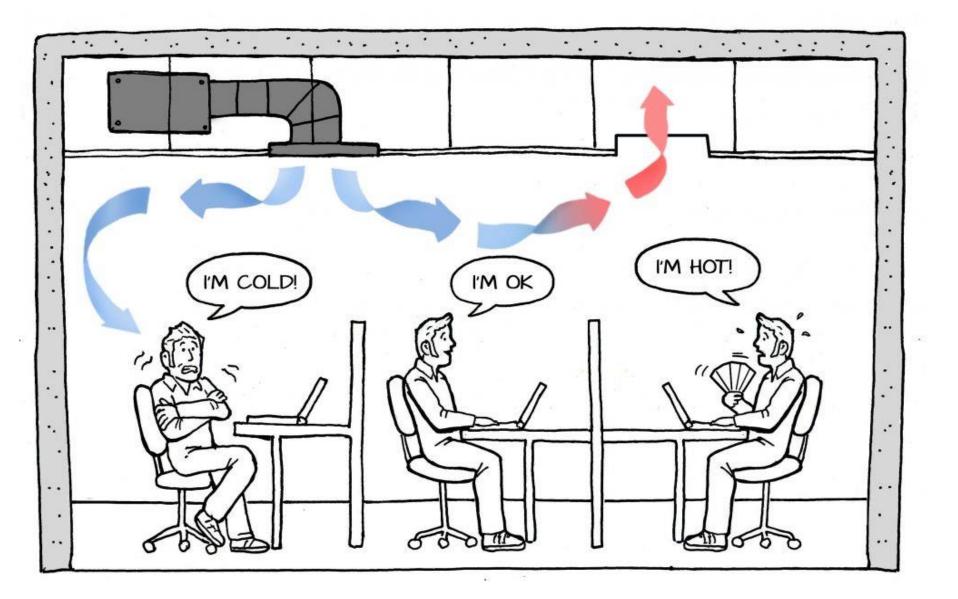
MSS MODULE PHYSIOLOGY LECTURE 2 THERMOREGULATION II

BY Dr. Fatma Farrag Ali Associate Professor of Medical Physiology 2023-2024

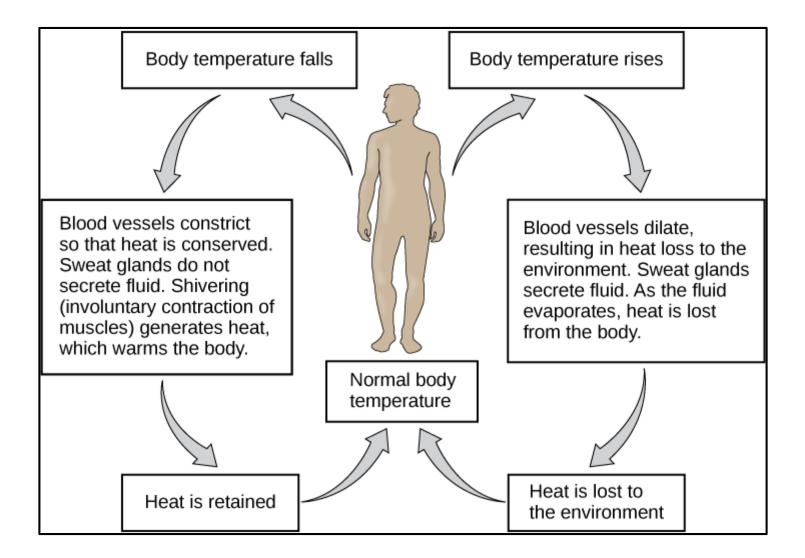




EXPOSURE TO COMFORTABLE TEMPERATURE (24-32°C)

- This is the range of environmental temperature at which the body temperature is kept constant through skin vasomotor changes only (which control heat transfer across the skin by R &CD).
- Normally, this range varies from about 24 to 32-34°C in the nude (= **unclothed**) **person**. Within this range, thermostasis is maintained as follows: 1) As the environmental temperature rises above 24°C, the heat gained by the body gradually increases, and to maintain body temperature constant, heat loss should also increase. The heat loss center is stimulated and this leads to inhibition of sympathetic tone to the cutaneous blood vessels resulting in their V.D. which increases the skin blood flow. This raises the skin temperature leading to increased heat loss through (R& CD), Thus the body temperature will be kept constant. However, such mechanism is sufficient to maintain the body temperature constant only till the environmental temperature rises to **32-34°C** (at which there is maximum cutaneous V.D.), and beyond that temperature the body temperature is maintained constant through other mechanisms of heat loss specially **sweat secretion**.

2) As the environmental temperature drops below 32-34°C, the heat loss by the body gradually increases, and to maintain the body temperature constant, such heat loss should decreased. The heat gain center is stimulated and this increases the sympathetic tone to the cutaneous **blood** vessels leading to their **V.C.** which **decreases the skin blood flow**. This reduces the skin temperature leading to decreased heat loss through (R& CD), thus the body temperature will be kept constant. However, such mechanism is sufficient to maintain the body temperature constant only till the environmental temperature drops to **about 24°C (at which there is maximum cutaneous V.C.)**, and below that temperature the body temperature is maintained constant by the mechanisms that increase heat production e.g. shivering.



Thermoregulatory Mechanisms on Exposure to Cold

Drop of the body temperature below the standard set point due to exposure to cold environment stimulates both peripheral and central thermoreceptors, which, in turn, stimulate **the heat gain center**. This center maintains thermostasis by :

Anti-drop response:

- (A) Increasing heat production.
- (B) Decreasing heat loss.

(A) Increase Heat Production:

Increasing the rate of heat production is the main thermoregulatory mechanism on exposure to cold.

Mechanisms:

(1) <u>Increased muscle tone and shivering:</u>

Mechanism: These are initiated by impulses discharged from a center located in the dorsomedial part of the posterior hypothalamus called the primary motor center for shivering. This center discharges stimulating signals downwards via certain extra-pyramidal tracts (specially the reticulospinal tracts) to anterior horn cells (AHCs) of spinal cord. These signals produce:

- Increased muscle tone all over the body. When it exceeds a certain level; shivering begins
- Shivering: synchronous contraction and relaxation of small antagonistic muscle groups. No work is done, so all energy is transformed into heat. It is useful as early response but not to maintain body temperature during long exposure to cold.
- Some people also increase heat production on cold exposure by doing voluntary muscular contraction e.g. foot stamping and hand clapping.

(2) Increasing secretion of thermogenic hormones (chemical thermogenesis):

Stimulation of **heat gain center** leads to secretion of the following hormones which increase the cellular metabolism leading to more heat production.

a) Catecholamines:

The **heat gain center** discharges impulses to the spinal cord which stimulate the **sympathetic preganglionic cholinergic fibers** that supply **the adrenal medullae** leading to secretion of **catecholamines (mainly adrenaline)**. These hormones increase the cellular metabolism and leads to oxidation of various food stuffs (which releases energy in the form of heat; **calorigenic effect**).

b) Thyroxine and glucocorticoids:

Excitation of **heat gain center** leads to secretion of both thyrotropin and corticotropin releasing hormones from the hypothalamus \rightarrow stimulation of secretion of anterior pituitary trophic hormones:

- ➤ Thyrotropin releasing hormone (TRH) → ↑ thyroid stimulating hormone (TSH) → ↑ thyroxine → ↑ heat production.
- ➤ Corticotropin releasing hormone(CRH) → ↑ adrenocorticotropic hormone (ACTH) → ↑ glucocorticoids → stimulation of organic metabolism → ↑ heat production.

N.B.

Thyroxine plays a **minor role as an immediate mechanism** of increased heat production (since the thyroid gland requires several weeks to increase its secretion sufficiently). On the other hand, **during chronic exposure to cold**, thyroxine becomes essential for **acclimatization** to cold.

(3) Increased sensations of appetite and food Intake: *Mechanism:*

Impulses discharged from the heat gain center \rightarrow increased appetite \rightarrow increased food intake \rightarrow increases heat production by its SDA. Protein is the preferable food in cold climates due to its high SDA.

(4) Brown Fat:

- □ It is a special type of fat present between and around the scapulae.
- □ It is a source of a considerable heat production, particularly in **infants** (absent in human adults).
- □ It has a high rate of metabolism→ increase heat production (thus infants tolerate cold better than adults).
- It is similar to electric blanket as it is richly supplied by sympathetic nerve fibers.
- □ It is also important for acclimatization to cold.

(B) Decrease Heat loss:

Decrease heat loss by non-evaporative mechanisms.

The body tries to decrease non-evaporative heat loss by :

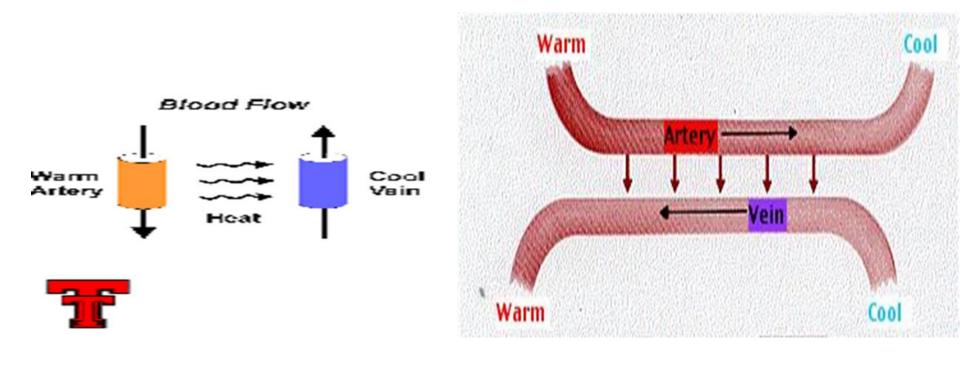
1)Vasoconstriction (V.C.) of skin blood vessels: this occurs as a result of stimulation of sympathetic centers in posterior hypothalamus \rightarrow decrease skin blood flow \rightarrow decrease skin temperature \rightarrow decrease temperature gradient between skin and environment \rightarrow decrease heat loss. Maximal V.C. occurs at 24°C.

2) Counter current heat exchange mechanism: Heat is directly transferred from the warm arterial blood to the cold venous blood as it returns to the heart, thus heat is returned back to the body core).

Accordingly, the body core (rectal) temperature will be kept constant although the skin temperature is decreased.

3) Behavioral responses to reduce the heat loss occur as **putting on heavy clothes and curling** to decrease body surface area exposed to the environment.

4) Horripilation: Erection of skin hair in animals as a result of contraction of piloerector muscles as a result of increased sympathetic discharge from the heat gain center. In humans, it only produce goose pimples (because the skin hair is poor and thin). So in cold weather people usually supplement this layer of hair by wearing clothes.



N.B.

Maximal vasoconstriction occurs at 24°C.

When the environmental temperature is <24 °C, the main physiological mechanism of body temperature regulation is through increase heat production.

Acclimatization to cold

This occurs on **prolonged exposure to cold**, and is produced mainly through increasing the **hormonal (chemical) thermogenesis particularly by increased secretion of thyroxine** (this may explain the much higher incidence of toxic goiter in persons living in cold climates). The presence of **brown fat** also helps acclimatization to cold due to its high rate of metabolism (thus **infants tolerate cold better than adults**).

Thermoregulatory Mechanisms on Exposure to Heat

- Rise of the body temperature above the standard set point due to exposure to hot environment (>32-34 °C) stimulates mainly the central thermoreceptors which in turn stimulate the heat loss center.
- This center maintains thermostasis by:

Anti-rise response:

- (a) Decreasing heat production.
- (b) Increasing heat loss.

(A) Mechanisms of decreasing heat production:

- Anorexia (= loss of appetite).
- □ Apathy and inertia.
- Inhibition of chemical thermogenesis: by inhibiting secretion of the thermogenic hormones (catecholamines & thyroid hormones).

(B) Mechanisms of increasing heat loss:

Increasing heat loss is the main thermoregulatory mechanism during exposure to heat, and is produced through the following **3 effects**:

(1) Cutaneous V.D.:

This occurs as a result of inhibition of sympathetic centers in posterior hypothalamus by impulses discharged from the heat loss center \rightarrow increase skin blood flow \rightarrow increase skin temperature \rightarrow increase heat loss by R and CD (so the skin is called the radiator system of the body). Accordingly, the body core (rectal) temperature will be kept constant although the skin temperature is increased.

Such mechanism is sufficient to maintain the body temperature constant only till the environmental temperature rises to 32-34 °C.

(2) Sweat secretion:

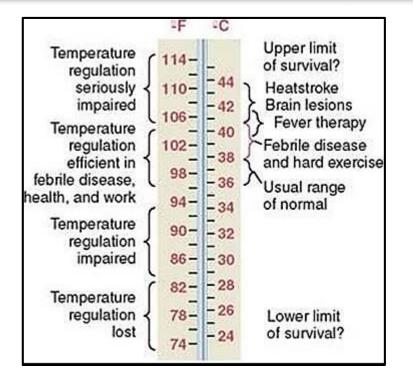
- This occurs whenever the non-evaporative heat loss mechanisms fail to maintain the body temperature constant. Therefore, it starts when the environmental temperature exceeds 32-34 °C.
- If the environmental temperature is > that of the body, the body gains heat through non-evaporative methods and so, the evaporative methods becomes the only way for heat loss.
- The cooling effects of sweating is dependent on its evaporation which is inversely proportionate to humidity.

(3) Panting:

- This is shallow rapid breathing that occurs in animals having no sweat glands (e.g. dogs) on exposure to hot environment.
- It occurs by impulses discharged from the heat loss center to a special panting center that is closely related to the pneumotaxic respiratory center in pons.



ABNORMALITIES OF THERMOREGULATION



Fever (Pyrexia)

Definition:

Elevation of body temperature due to the **resetting** of the **set point** (37.1 °C) for body temperature (hypothalamic thermostat) to a higher level.



Pathogenesis:

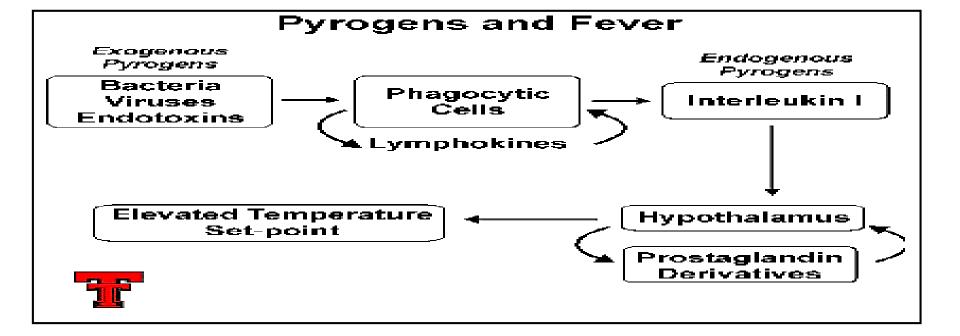
Febrile conditions:

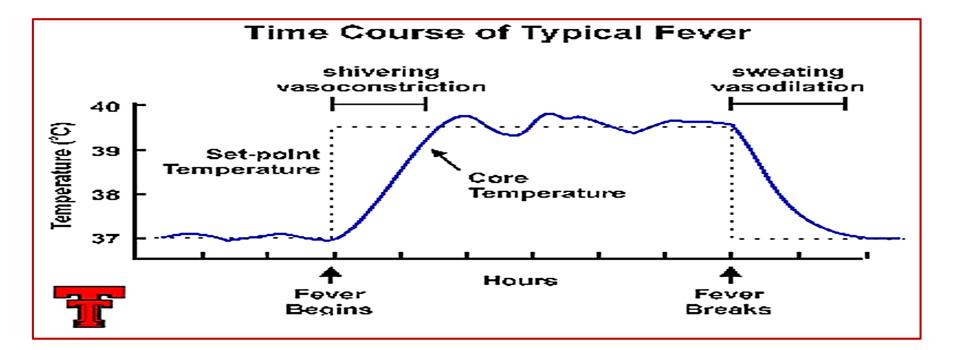
- Proteins, breakdown products of proteins, and certain other substances, especially lipopolysaccharide toxins released from bacterial cell membranes, can cause the set-point of the hypothalamic thermostat to rise (resetting). Substances that cause this effect are called pyrogens.
- When bacteria or breakdown products of bacteria are present in the tissues or in the blood, they are phagocytized by the blood leukocytes and by tissue macrophages. All these cells digest the bacterial products and then release the substance interleukin-1—also called leukocyte pyrogen or endogenous pyrogen—into the body fluids. The interleukin-1, on reaching the hypothalamus, immediately activates the processes to produce fever in only 8 to 10 minutes.
- Several experiments have suggested that interleukin-1 causes fever by first inducing the formation of one of the prostaglandins (PGs), mainly prostaglandin E2 (PGE2) which acts in the hypothalamus to elicit the fever reaction. This may be the explanation for the manner in which aspirin reduces fever, because aspirin impedes the formation of prostaglandins from arachidonic acid. Drugs such as aspirin that reduce fever are called antipyretics.

- The set-point of the hypothalamic temperature-regulating center becomes higher than normal while the thermoreceptors signal that the actual body temperature is below the new set point. Accordingly, all the mechanisms for raising the body temperature are brought into play, including heat conservation and increased heat production (anti-drop effect).
- **Decreased heat loss:** Vasoconstriction of skin blood vessels and contraction of piloerector muscle \rightarrow goose skin \rightarrow decrease non-evaporative heat loss + curling in bed and covering the body with blankets.
- Increased heat gain: By shivering.

This produces a chilly (cold) sensation.

- Within a few hours after the set-point has been increased, the body temperature also approaches this level. So, the net effect is a rise in the body temperature to a new set point which is maintained so long as the pyrogen exists.
- When recovery occurs: The pyrogen disappears and the set point returns to its normal level. The patient feels hot and anti-rise measures occurs → sweating and vasodilatation of skin blood vessels (Crisis or flush)→ increase evaporative and non- evaporative heat loss mechanisms → drop of body temperature to normal.





HEAT STROKE

- Heat stroke is the most severe form of heat exhaustion and is a life-threatening emergency.
- It is a condition that develops rapidly and requires immediate medical treatment.
- It occurs when the rate of heat production exceeds that of heat loss.
- ✓ Heat strokes occurs in hot humid atmosphere.
- ✓ All mechanisms of heat loss are not functioning.
- ✓ There is excessive sweating but sweat doesn't evaporate (only drips from the body).
- ✓ The body temperature is increased with dehydration and salt loss and may be circulatory shock (due to excessive sweating).
- ✓ Depression of thermoregulatory center may also occurs. It leads to more increase in body temperature →→ more depression of thermoregulatory center and a vicious circle develops leading to serious increase in body temperature, (+ve feedback mechanism).
 - **If body temperature exceeds 43°C**, it is **fatal** due to denaturation of cellular proteins and permanent damage of the CNS (degeneration of cells allover the body especially in brain).



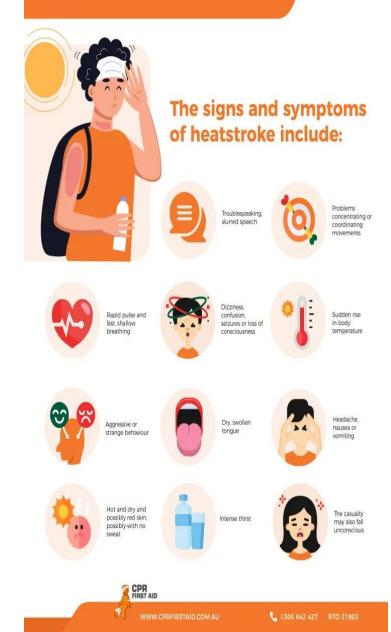
What are the symptoms of heat stroke?

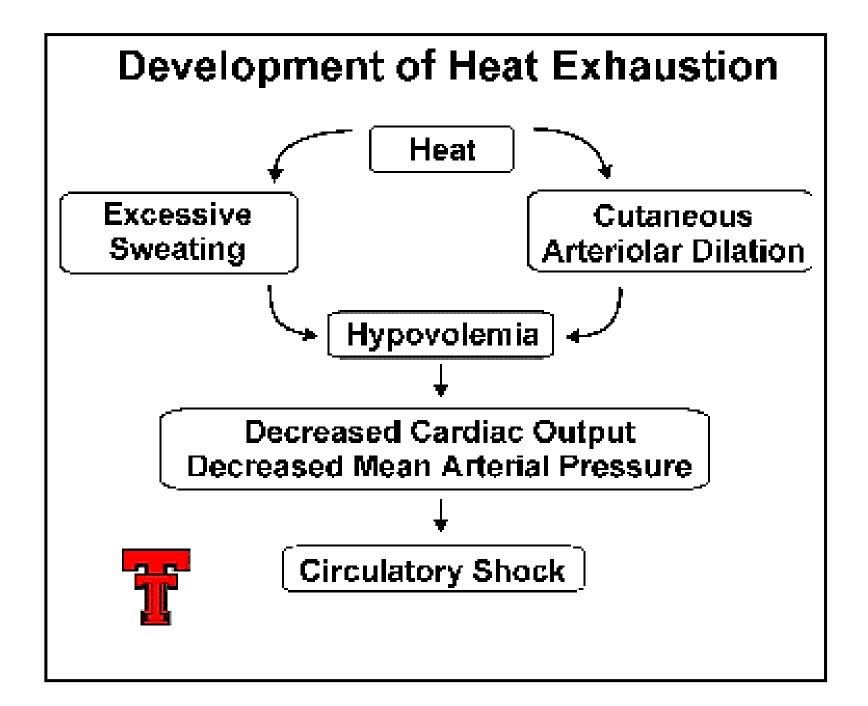
The following are the most common symptoms of heat stroke. However, each individual may experience symptoms differently.

Symptoms may include:

- Headache.
- Blurring of vision.
- Disorientation or confusion.
- Hot flushed skin.
- A high body temperature.
- Rapid heartbeat.
- Hallucinations.
- Dehydration and salt loss
 →circulatory shock.
- Loss of consciousness.

SYMPTOMS OF HEATSTROKE





<u> Treatment :</u>

There are some immediate first-aid measures including the following:

- Rapid cooling of the body by placing the person in a cold water bath OR Sponging with alcohol (which rapidly evaporates).
- Intravenous (IV) fluids are often necessary to compensate for fluid or electrolyte loss.

Sunstroke

- It is similar to heat stroke but it is produced by prolonged direct exposure to sun rays.
- In addition to the manifestations of the heat stroke, the falling sun rays on the back of the neck and skull have a damaging effects on the brain and spinal cord by the local heating effect they produce.



Frostbite:

- It occurs when the body is exposed to very low temperature.
- Freezing of certain surface areas e.g. earlobes & digits of hands and feet → ice crystals in cells→ permanent damage of affected parts →gangrene→ loss of affected parts.

Hypothermia

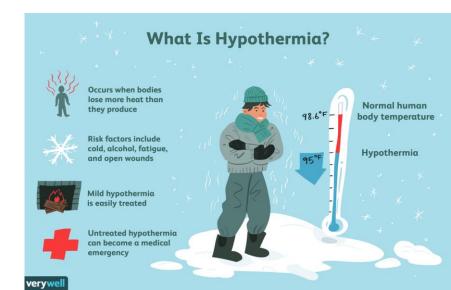
✓ Definition:

Hypothermia is a decrease in body temperature.

✓ Causes:

Conditions in which heat loss is greater than heat gain;

- Prolonged exposure to cold.
- Alcohol intoxication.



✓ Effects:

- Most tissues tolerate hypothermia and \downarrow their rate of metabolism and physiological processes slow down (\downarrow HR, RR and ABP).
- It usually produces no ill effects and the person returns to normal condition if re-warmed within a certain period with external heat.
- O However, the thermoregulatory center is depressed in severe cases → more hypothermia (+ ve feedback mechanism) → sleepiness, coma and death.
- ✓ Artificial (induced) hypothermia has been extensively used in surgery: Patient is given strong sedative → depress hypothalamic thermostat and cooling the patient with ice and cooling blankets until temperature is decreased → slows metabolism → ↑body survival.

