Benha University
Faculty of Science
Department of Zoology

Adaptation Physiology (318 Z)

Semester: second (2015/2016)
Level: third & fourth levels
Sepc: Zoology & Chemistry
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Exam time: 2:00 hours

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Group (B): (1 hour, 24 marks)

1. Compare between the followings: (12 marks)

a) Carbohydrate metabolism in liver during feeding and fasting.

Carbohydrate metabolism in liver during feeding

After a meal containing carbohydrate, the liver becomes a net consumer of glucose, retaining roughly 60 of every 100 g of glucose presented by the portal system. This increased use is not a result of stimulated glucose transport into the hepatocyte, because this process is normally rapid and the glucose transporter, GLUT-, is insulin insensitive. Rather, hepatic glucose metabolism is increased by the following mechanisms.

1. Increased phosphorylation of glucose: Elevated levels of glucose within the hepatocyte (as a result of elevated extracellular levels) allow glucokinase to phosphorylate glucose to glucose 6-phosphate. (Recall that glucokinase is not subject to product inhibition.) This contrasts with the postabsorptive (fasted) state in which hepatic glucose levels are lower and glucokinase is largely dormant because of its low affinity (high Km) for glucose.
2. Increased glycogen synthesis: The conversion of glucose 6-phosphate to glycogen is favored by the activation of glycogen synthase—both by dephosphorylation and by increased availability of glucose 6-phosphate, its allosteric effector.
3. Increased activity of the hexose monophosphate pathway (HMP): The increased availability of glucose 6-phosphate in the absorptive state, combined with the active use of NADPH in hepatic lipogenesis, stimulate the HMP. This pathway typically accounts for 5 10% of the glucose metabolized by the liver.
4. Increased glycolysis: In liver, glycolytic metabolism of glucose is significant only during the absorptive period following a carbohydrate- rich meal. The conversion of glucose to acetyl CoA is stimulated by the elevated insulin to glucagon ratio that results in increased activity (and amount) of the regulated enzymes of glycolysis, for example, pyruvate kinase (see p. 102). Pyruvate dehydrogenase (PDH), which converts pyruvate to acetyl CoA, is active (dephosphorylated) because pyruvate inhibits PDH kinase. Acetyl CoA is used as either a building block for fatty acid synthesis, or it provides energy by oxidation in the tricarboxylic acid (TCA) cycle.

5. Decreased gluconeogenesis: Whereas glycolysis is stimulated in the absorptive state, gluconeogenesis is decreased. Pyruvate carboxylase, which catalyzes the first step in gluconeogenesis, is largely inactive due to low levels of acetyl CoA—an allosteric effector essential for enzyme activity. The high insulin to glucagon ratio also favors inactivation of other gluconeogenic enzymes, such as fructose 1, 6-bisphosphatase.

Carbohydrate metabolism in liver during fasting

The liver first uses glycogen degradation and then gluconeogenesis to maintain blood glucose levels to sustain energy metabolism of the brain and other glucose-requiring tissues in the fasted (postabsorptive) state. [Note: Recall the presence of glucose 6-phosphatase in the liver allows the production of free glucose both from glycogenolysis and from gluconeogenesis.

1. Increased glycogen degradation: Figure 24.10 shows the sources of blood glucose after ingestion of 100 g of glucose. During the brief absorptive period, ingested glucose is the major source of blood glucose. Several hours later, blood glucose levels have declined sufficiently to cause increased secretion of glucagon and decreased release of insulin. The increased glucagon to insulin ratio causes a rapid mobilization of liver glycogen stores (which contain about 80 g of glycogen in the fed state) due to phosphorylation (activation) of glycogen phosphorylase. Note that liver glycogen is nearly exhausted after 10–18 hours of fasting; therefore, hepatic glycogenolysis is a transient response to early fasting.
2. Increased gluconeogenesis: The synthesis of glucose and its release into the circulation are vital hepatic functions during fasting. The carbon skeletons for gluconeogenesis are derived primarily from glucogenic amino acids and lactate from muscle, and glycerol from adipose. Gluconeogenesis, favored by activation of fructose 1, 6-bisphosphatase (due to a drop in its inhibitor, fructose 2,6-bisphosphate) and by induction of phosphoenolpyruvate (PEP) carboxykinase by glucagon, begins 4–6 hours after the last meal and becomes fully active as stores of liver glycogen are depleted. Gluconeogenesis plays an essential role in maintaining blood glucose during both over night and prolonged fasting. [Note: Whereas acetyl CoA cannot be used as a substrate for gluconeogenesis, the acetyl CoA produced by hepatic oxidation of fatty acids supplied from lipolysis in adipose is an allosteric activator of pyruvate carboxylase (and an allosteric inhibitor of pyruvate dehydro -

genase), and thus pushes pyruvate to gluconeogenesis.

b) Physiological problems of weightlessness during short and long period.

The physiologic problems of weightlessness have not proved to be of much significance, as long as the period of weightlessness is not too long. Most of the problems that do occur are related to three effects of the weightlessness:
(1) Motion sickness during the first few days of travel, (2) translocation of fluids within the body because of failure of gravity to cause normal hydrostatic pressures, and (3) diminished physical activity because no strength of muscle contraction is required to oppose the force of gravity. Almost 50 per cent of astronauts experience motion sickness, with nausea and sometimes vomiting, during the first 2 to 5 days of space travel. This probably results from an unfamiliar pattern of motion signals arriving in the equilibrium centers of the brain, and at the same time lack of gravitational signals.

The observed effects of prolonged stay in space are the following: (1) decrease in blood volume, (2) decrease in red blood cell mass, (3) decrease in muscle strength and work capacity, (4) decrease in maximum cardiac output, and (5) loss of calcium and phosphate from the bones, as well as loss of bone mass. Most of these same effects also occur in people who lie in bed for an extended period of time. For this reason, exercise programs are carried out by astronauts during prolonged space missions. In previous space laboratory expeditions in which the exercise program had been less vigorous, the astronauts had severely decreased work capacities for the first few days after returning to earth. They also had a tendency to faint (and still do, to some extent) when they stood up during the first day or so after return to gravity because of diminished blood volume and diminished responses of the arterial pressure control mechanisms.

c) Acute and chronic oxygen poisoning in deep sea.

Acute Oxygen Poisoning

The extremely high tissue Po_2 that occurs when oxygen is breathed at very high alveolar oxygen pressure can be detrimental to many of the body's tissues. For instance, breathing oxygen at 4 atmospheres pressure of oxygen (Po_2 = 3040 mm Hg) will cause brain seizures followed by coma in most people within 30 to 60 minutes. The seizures often occur without warning and, for obvious reasons, are likely to be lethal to divers submerged beneath the sea. Other symptoms encountered in acute oxygen poisoning include nausea, muscle twitchings, dizziness, disturbances of vision, irritability, and disorientation. Exercise greatly increases the diver's susceptibility to oxygen toxicity, causing symptoms to appear much earlier and with far greater severity than in the resting person.

Chronic Oxygen Poisoning

A person can be exposed to only 1 atmosphere pressure of oxygen almost indefinitely without developing the acute oxygen toxicity of the nervous system just described. However, after only about 12 hours of 1 atmosphere oxygen exposure, lung passageway congestion, pulmonary edema, and atelectasis caused by damage to the linings of the bronchi and alveoli begin to develop. The reason for this effect in the lungs but not in other tissues is that the air spaces of the lungs are directly exposed to the high oxygen pressure, but oxygen is delivered to the other body tissues at almost normal Po_2 because of the hemoglobin-oxygen buffer system.

2. Explain the following: **(12 marks)**

a) Increased diffusing capacity after acclimatization.

It will be recalled that the normal diffusing capacity for oxygen through the pulmonary membrane is about 21 ml/mm Hg/min, and this diffusing capacity can increase as much as threefold during exercise. A similar increase in diffusing capacity occurs at high altitude. Part of the increase results from increased pulmonary capillary blood volume, which expands the capillaries and increases the surface area through which oxygen can diffuse into the blood. Another part results from an increase

in lung air volume, which expands the surface area of the alveolar-capillary interface still more. A final part results from an increase in pulmonary arterial blood pressure; this forces blood into greater numbers of alveolar capillaries than normally—especially in the upper parts of the lungs, which are poorly perfused under usual conditions.

b) Natural acclimatization of native human being living at high altitudes.

Many of natives are born at these altitudes and live there all their lives. In all aspects of acclimatization, the natives are superior to even the best acclimatized lowlanders, even though the lowlanders might also have lived at high altitudes for 10 or more years. Acclimatization of the natives begins in infancy. The chest size, especially, is greatly increased, whereas the body size is somewhat decreased, giving a high ratio of ventilator capacity to body mass. In addition, their hearts, which from birth onward pump extra amount of cardiac output, are considerably larger than the hearts of lowlanders. Delivery of oxygen by the blood to the tissues is also highly facilitated in these natives. The arterial oxygen Po_2 in the natives at high altitude is only 40 mm Hg, but because of the greater quantity of hemoglobin, the quantity of oxygen in their arterial blood is greater than that in the blood of the natives at the lower altitude. Note also that the venous Po_2 in the high altitude natives is only 15 mm Hg less than the venous Po_2 for the lowlanders, despite the very low arterial Po_2, indicating that oxygen transport to the tissues is exceedingly effective in the naturally acclimatized high-altitude natives.

Naturally acclimatized native persons can achieve a daily work output even at high altitude almost equal to that of a lowlander at sea level, but even well-acclimatized lowlanders can almost never achieve this result.

c) Using helium instead of nitrogen in deep dives.

In very deep dives, especially during saturation diving, helium is usually used in the gas mixture instead of nitrogen for three reasons:
(1) It has only about one fifth the narcotic effect of nitrogen.
(2) Only about one half as much volume of helium dissolves in the body tissues as nitrogen, and the volume that does dissolve diffuses out of the

tissues during decompression several times as rapidly as does nitrogen, thus reducing the problem of decompression sickness.
(3) The low density of helium (one seventh the density of nitrogen) keeps the airway resistance for breathing at a minimum, which is very important because highly compressed nitrogen is so dense that airway resistance can become extreme, sometimes making the work of breathing beyond endurance