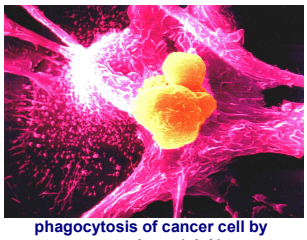
5.

Seminar: Structure and function of connective tissue proper and adipose tissue.

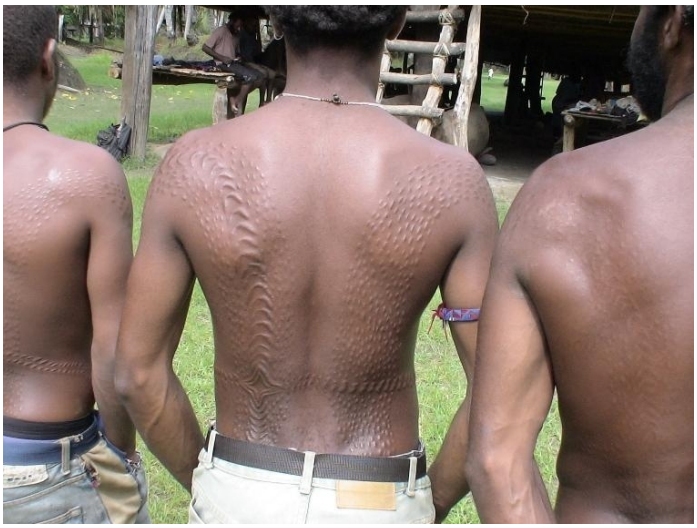
Practical class: Connective tissue proper and adipose tissue.



phagocytosis of cancer cell by macrophage (pink)

- **loose connective tissue – mesentery, mast cells, elastic fibers (slide # 9),**
- **dense connective tissue – tendon (slide # 7),**
- **unilocular (yellow) adipose tissue – hypodermis or synovial membrane of joint capsule (slide # 38),**
- **multilocular (brown) adipose tissue (slide # 110),**
- **reticular fibers - spleen (slide # 113),**
- **leptin, the hormone of satiety, secreted by adipocytes (text # 22).**
- **"Crocodile people" - photo # 24**

photo no. 24.



Ornamental keloids on the skin of inhabitants of New Guinea "Crocodile people". From the special correspondent of the Department of Histology & Embryology, Prof. dr hab. Marek Kamiński.

Text no. 22

LEPTIN, THE HORMONE OF SATIETY, SECRETED BY ADIPOCYTES

Adipocytes secrete the protein hormone (mol. mass 16 kD) termed leptin. **The latter appears in the blood plasma at the concentration of 5-50 nmoles and its concentration depends upon the overall mass of adipose tissue of the body. Leptin binds to the specific receptors of the cellular membrane of hypothalamic neurons and choroid plexus cells. The role of the leptin is not clear since it was discovered few years ago. One of the hypothesis assumes that leptin is a kind of "adipostatic signal" that carries the information on the adipose tissue mass to the brain. Accordingly, the increase of leptin level triggers the nerve and hormonal mechanisms yielding in the reduction of adipose tissue mass (negative feedback). Pivotal role in that mechanism seems to be played** by neuropeptide Y **(neurotransmitter) that is very potent stimulator of apetite. Leptin inhibits secretion of neuropeptide Y and thereby reduces the apetite.**

Both the discovery of leptin and our knowledge of adipose tissue homeostasis have emerged as a result of research of mutations in *obese (ob)* gene of the mice. Homozygous *oblob* mice fail to produce leptin and occur to be obese. Moreover, *ob/ob* mice cured with the leptin lost the weight and became slim. The results of those experiments were the source of a presumption that the human obesity could result in the mutation in the gene *ob.* However, the analysis of DNA obtained from obese people failed to confirm such an assumption.

Mouse obesity can also be a result of the mutation in *diabetes (db)* gene that codes for the leptin receptor. The similar mutation in human *db* gene of patients suffering of obesity was so far not found.

The starvation of mice can lead to the decrease of leptin level. On the other hand, the starvation results in malfunction of endocrine glands (reduction of secretion of LH and thyroxine as well as the increase of secretion of corticosterone and ACTH); the injection of leptin restores, at least partially, the normal function of endocrine glands disturbed by starvation. Therefore, leptin may be suspected to be one of the factors participating in the adaptation of starving organisms.

Leptin and its receptors constitute one of the systems controlling the adipose tissue homeostasis. Although the mechanisms causing the obesity continue to be an enigma, some pharmaceutical companies make contemporarily attempts to apply leptih as a drug for treatment of obese people.