

growth retardation of Pygmies may be due to excessive exposure to infections with resultant elevation of immunoglobulins, in spite of absent gross nutritional deficiency secondary to infection.

Dulloo AG, et al. *Eur J Clin Nutr* 1996;50(6):371-380.

Editor's comment: This study is very nicely executed, and extensive data are presented to raise much skepticism about the

previous hypothesis that an increase in IGF-1, which is normally associated with the adolescent growth spurt, does not occur in Pygmies. The data not only negate the former hypotheses but also reveal a new biochemical alteration (elevated immunoglobulins) that, in my opinion, may or may not be associated with growth retardation. This article was kindly brought to my attention by Dr. Guy Van Vliet.

Robert M. Blizzard, MD

Nonadipose Tissue Production of Leptin: Leptin as a Novel Placenta-Derived Hormone in Humans

The authors report that leptin, the appetite-regulating polypeptide hormone secreted by adipocytes, also is synthesized and secreted by human placental chorionic villi and amnion cells. They demonstrated that plasma leptin concentrations were higher in pregnant women than in nonpregnant women of comparable body mass index (BMI), a finding made by other investigators. Whereas in nonpregnant women plasma leptin concentrations and BMI were directly related, this relationship was not demonstrable in pregnant subjects. Leptin values

declined rapidly after birth. Umbilical vein leptin concentrations were slightly higher than umbilical artery values. Leptin was detectable in amniotic fluid. Plasma leptin values were elevated in patients with hydatidiform moles and choriocarcinoma and fell rapidly with surgical removal or chemotherapeutic ablation of the tumor.

Expression of *ob*, the leptin gene, was demonstrated in placental chorionic villi and amnion. Immunoreactive leptin was present in trophoblasts of first trimester chorionic villi and syncytiotrophoblasts and amnion cells in the third trimester. The investigators suggest that placental leptin may be of physiologic importance in modulating the metabolic relationship between mother and fetus. Plasma leptin values may serve as a marker of tumors of placental origin.

Masuzaki H, et al. *Nat Med* 1997;3:1029-1033.

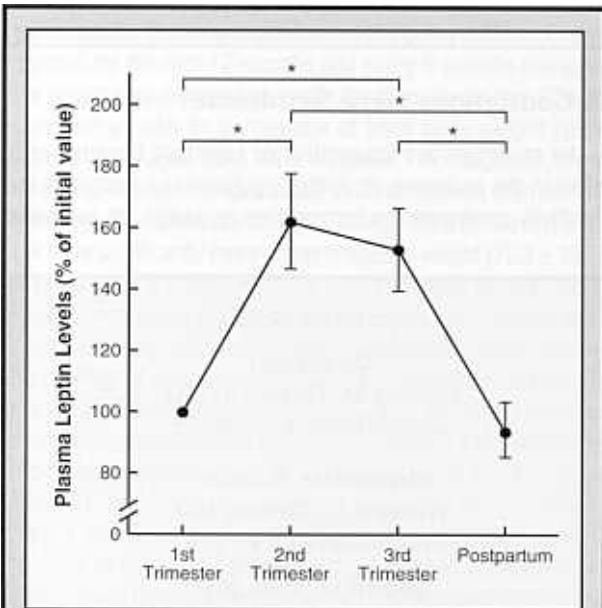
Editor's comment: This paper reports that leptin may be secreted by nonadipose placental cells, namely, placental trophoblasts and syncytiotrophoblasts, and secreted into amniotic fluid and the maternal circulation. Thus, leptin is another of the many protein hormones that are secreted not only by their primary tissue but also by the placenta. Placental leptin may modulate metabolic homeostasis in the pregnant woman and fetus. One wonders if, in the pregnancy complicated by placental insufficiency and intrauterine growth retardation, leptin deficiency may permanently alter hypothalamic appetite regulatory mechanisms that influence the postnatal growth of that individual.

Chehab points out that the elevated leptin concentrations in pregnant women imply an element of leptin resistance because appetite is not suppressed by pregnancy. Although these investigators did not note correlation between BMI and serum leptin concentrations in pregnant women, Hartmann et al reported that the 2 were highly correlated.

Allen W. Root, MD

Chehab FF. *Nat Med* 1997;3:952-953.

Hartmann BW, et al. *N Engl J Med* 1997;337:863.



Elevated plasma leptin levels during pregnancy. Time course of plasma leptin levels determined consecutively during pregnancy and postpartum ($n=40$). The plasma leptin levels in the first trimester are defined as 100%.

* $P<0.005$ versus the values in the first trimester or postpartum.

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