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INNATE GROWTH PATTERNS. Eleanor Colle, Division of Endocrinology, McGill University-Montreal Childrens Hospital Research Institute, Montreal, H3Y 2X3

Innate growth patterns are constrained by the genetic program of the organism. This program determines not only the time-dependent changes in mass but also the development of the internal and external form characteristic of the species. The two processes occur in concert throughout the growing stages of the organism. Certain instructions of the program are carried out literally. Other instructions permit the development of capabilities which may interact with the environment to increase the range of activities of the organism. However, the degree of flexibility and the range of possible variations are determined by the program itself. In complex organisms, different organ systems grow at different rates. Cellular proliferation is associated with cellular differentiation, cellular migration, secretion of extracellular products and, at certain times, with cell death. The mechanisms by which signals for these events are generated and then exchanged between cells is better characterized for some organ systems that for others. Circulating hormones represent one type of signal. However, it seems likely that local signals which both initiate and arrest cell proliferation must play a central role in the execution of the program. Developmental anomalies provide insight into the nature of the communication.

B10

THE COMPLEXITY OF HUMORAL GROWTH REGULATION. J.L. Van den Brande* and J.S. Sussenbach, University Hospital for Children and Youth "Het Wilhelmina Kinderziekenhuis" and Laboratory for Physiological Chemistry, State University of Utrecht, The Netherlands.

Growth hormone (GH), acting through the somatomedins (SM) IGF-I and II, the thyroid hormones and the adrenal and gonadal steroids are well-defined and the most important humoral growth regulatory factors. In recent years this clear concept has been disturbed by a number of facts: the heterogeneity of GH and the possible occurrence of metabolic fragments, the possible direct growth stimulating effects of GH, the uncertainty on the precise molecular structure of the SM's as synthesized and in circulation, the occurrence of inhibitors of SM's, and finally the contribution of locally synthesized SM's and the interaction of SM's with other growth factors at the target.
We have focused on the nucleotide sequence of SM-cDNA's and on the molecular nature of SM's in plasma and extracts from Cohn fraction IV. Using recombinant DNA techniques we have demonstrated that IGF-I and II are synthesized as precursors. Detailed studies of native plasma by molecular size and charge show heterogeneity of the 150 kd fraction associated with SM-activity. Isofocusing of the SM's by charge reveils the presence of multiple GH-dependent SM's, the significance of which needs further study. A further unravelling of the genetic organization and processing of SM-precursors, their exact nature in plasma and the specificity of their effects is necessary for understanding their role in growth regulation.

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NUTRITIONAL ASPECTS OF FETAL GROWTH. J.A. Brasel*, Harbor-UCLA Medical Center, 1000 West Carson Street, Torrance, CA 90509.

Epidemiological, food intervention, morphological and animal studies support the view that maternal nutrition influences fetal growth, even in circumstances where no serious evidence of maternal malnutrition exists. When gestational age is held constant, maternal pregravid weight and weight gain during pregnancy, indices of past and present maternal nutritional status, respectively, are two of the most important determinants of birth weight. Prenatal dietary supplements can raise birth weight in women at risk of producing low birth weight infants, depending in part on the nutritional state of the woman. In heavier women there is unlikely to be any rise; in starved women a deficit of 300-400 g can be made up by restoring a normal diet. Not surprisingly, severe malnutrition produces its most marked effects when present in the last half to third of pregnancy when fetal growth is most rapid. The mechanism(s) responsible for the fetal growth failure is not clear. It appears to be more complicated than a simple competition for nutrients between mother and infant. It is