Physiology, Div. of Endocrin., Duke Univ. Med. Centr., Durham, North Carolina): ISOLATION AND CHARACTERIZATION OF A SERUM INHIBITOR OF CARTILAGE METABOLISM. Endocrinology 95:1600, 1974.

N-butanol extraction of rat, pig or human serum was found to remove a potent inhibitor of in vitro embryonic chicken cartilage metabolism. This inhibitor was active at less than 1/100 of its serum concentration and therefore interfered with in vitro somatomedin assays which were run with 1 to 10 per cent serum in the medium. The butanol extractable inhibitor (BEI) affected [^{3}H]uridine incorporation into both RNA and an acid soluble cartilage fraction, $^{35}SO_4$ incorporation into chondromucoprotein and [^{3}H]thymidine incorporation into DNA. Studies of the solubility and thin layer chromatographic properties of BEI, revealed that the inhibitor was probably a glucocorticoid. This hypothesis was confirmed when it was found that cortisol, corticosterone and dexamethasone in concentrations as low as 2.5×10^{-10}M inhibited cartilage metabolism in the same manner as BEI.

Ash, Patricia; and Francis, M. J. O. (Nuffield Dept. of Orthopaedic Surgery, Univ. of Oxford, Nuffield Orthopaedic Centre, Headington, Oxford, England): RESPONSE OF ISOLATED RABBIT ARTICULAR AND EPIPHYSEAL CHONDROCYTES TO RAT LIVER SOMATOMEDIN. J. Endocrinol. 66:71, 1975.

Isolated rat liver, when perfused with medium containing bovine growth hormone, produced somatomedin-like activity (liver somatomedin).

Liver somatomedin is useful in studies of the hormonal control of the cartilage plate in vitro, since unlike serum it is not contaminated with other hormones or growth factors (apart from growth hormone). Chondrocytes isolated from various regions of the growth cartilage responded differently to liver somatomedin; proliferative chondrocytes, like those isolated from the articular cartilage, showed increased [^{3}H]thymidine uptake in response to liver somatomedin, whereas hypertrophic chondrocytes did not respond. It is suggested that there is a reduction in the response to somatomedin by growth plate chondrocytes as they pass from the proliferative to the hypertrophic state.

Thyroxine, thought to be involved in the processes of hypertrophy and new bone formation, did not directly affect [^{3}H]thymidine uptake by proliferative chondrocytes, but inhibited stimulation of their activity by liver somatomedin.

Measurement of [^{3}H]thymidine uptake by isolated articular chondrocytes may provide a useful assay for both liver and serum somatomedin. The graded response of chondrocytes to increasing concentrations of liver somatomedin paralleled the response to increasing levels of serum somatomedin.

Liberti, J. P. (Dept. of Biochemistry, Med. Coll. of Virginia, Virginia Commonwealth Univ., Richmond, Virginia): PURIFICATION OF BOVINE SOMATOMEDIN[1,2]. Biochem. Biophys. Res. Commun. 67:1226, 1975.

A procedure for the purification of bovine somatomedin (SM[4]) is presented. The purification scheme utilizes ultrafiltration through membranes of nominal mol. wt. cutoffs, molecular sieve chromatography and finally isoelectric focusing. Two peaks of SM activity, measured by the in vitro stimulation of ^{35}S-Na_2SO_4 and ^{3}H-thymidine uptake by costal cartilage, were present after focusing; an acidic component having a pI of 6.0-6.7 and a basic component having a pI in the range of 7.8-8.3. The acidic component comprised 2 per cent of the initial activity and was 120,000-fold purified: the basic component comprised 10 per cent of the initial activity and was 350,000-fold purified relative to the starting material. These components are similar in molecular size and pI to SM-A and SM-C isolated from human plasma.

Tato, Luciano; Du Caju, Marc V. L.; Prevot, Claude; and Rappaport, Raphael (Unité de Recherche sur les Maladies du Métabolisme chez l'Enfant, Hôpital des Enfants Malades, Paris, France): EARLY VARIATIONS OF PLASMA SOMATOMEDIN ACTIVITY IN THE NEWBORN. J. Clin. Endocrinol. Metab. 40:531, 1975.

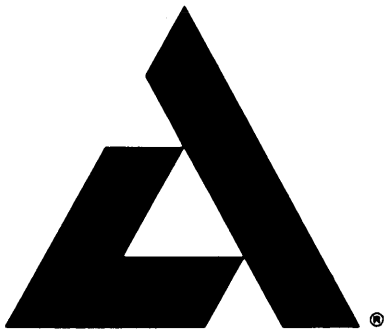
In neonates, plasma somatomedin as measured by the porcine cartilage assay was very low during the first day of life. A striking increase was observed on days 4 and 5, with a return to lower values at a later age. These findings indicate an early capacity to generate somatomedin activity in newborns.

BOOK REVIEW

DIABETIC RETINOPATHY: CLINICAL EVALUATION, PROGNOSIS AND TREATMENT WITH PHOTOCOAGULATION, *by S. Riaskoff, M.D. Dutch Guilders 50.–, 64 pages, with 39 figures (of primarily color fundus photos), 1 table. The Hague, Dr. W. Junk bv Publishers, 1976.*

This monograph has been written with the apparent intent of making diabetic retinopathy easier to understand and, at the various stages, to be better able to prognosticate the visual outcome. Supposedly it has been written for the ophthalmologist but does not appear likely to make him any more aware of how to treat, when to treat, and why to treat. Moreover, this reviewer would disagree with the amount of treatment in some of the illustrations (usually not enough to cover the pathology), the time of treatment (too early in some cases), or treatment at all (in some cases, where extensive fibrous proliferation was so great that the treatment probably was really no treatment or too risky, and other approaches such as scleral buckling surgery or vitrectomy, or both, might better have been considered).

Some of the comments as to efficacy and preference of technic are based on the personal prejudices of the author, but then we are all somewhat guilty of that. The list of references does not include reference to *Management of Diabetic Retinopathy* by Okun, Johnston, and Boniuk; if Dr. Riaskoff had read it, he might have found less incentive to write this "simple" monograph. The intent is fine, but is does not appear the issue is made any clearer for either the ophthalmologist or the internist. ISAAC BONIUK, M.D.



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