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# ABSTRACTS

*(All are verbatim summaries)*

*Elders, M. Joycelyn; Wingfield, Barbara S.; McNatt, M. Loretta; Clarke, James S.; and Hughes, Edwin R.* (Depts. of Pediatrics, Univ. of Arkansas Med. Centr., Little Rock, and Univ. of South Alabama Sch. of Med., Mobile, Alabama): GLUCOCORTICOID THERAPY IN CHILDREN. Am. J. Dis. Child. *129*:1939, 1975.

Glucocorticoids cause growth retardation in children. We have studied the effect of these hormones on serum somatomedin (Sm) levels in seven children with nephrosis.

Intravenous administration of methylprednisolone sodium succinate, 2.2 mg./kg., causes a rapid fall in serum Sm activity. The activity remains suppressed during continuous therapy, but re-

turns toward normal when medication is omitted during the course of alternate-day therapy. We conclude that one reason for growth retardation secondary to continuous glucocorticoid therapy is suppression of Sm generation. A direct effect of these hormones on the cartilage cell or induction of an Sm inhibitor cannot be excluded by the reported experiments.

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*Marshall, Robert N.; Underwood, Louis E.; Voina, Sandra J.; Foushee, Doretha B.; and Van Wyk, Judson J.* (Dept. of Pediatrics, Univ. of North Carolina Sch. of Med., Chapel Hill, North Carolina): CHARACTERIZATION OF THE INSULIN AND SOMATOMEDIN-C RECEPTORS IN HUMAN PLACENTAL CELL MEMBRANES. J. Clin. Endocrinol. Metab. *39*:283, 1974.

Human placentas have been used to further investigate the binding of insulin and somatomedin to cell membrane receptors. Conditions for optimal binding of $^{125}$I-insulin were defined for both particulate membrane preparations and membranes solubilized in Triton X-100. Human placental membranes are a rich source of high affinity insulin receptors. As in previous studies with rat liver and fat cell preparations, only somatomedin and proinsulin were effective competitors for binding to the insulin receptor. These findings were used to define the optimal conditions for a competitive binding assay for insulin and insulin-like peptides. This assay had threshold sensitivities of 10 $\mu$U./ml. for insulin and less than 0.1 U./ml. for somatomedin. Incubation at 4° C for 15-18 hr. essentially eliminated proteolytic degradation of the labeled hormone and led to strikingly better binding than was observed at higher temperatures.

A highly purified preparation of somatomedin-C labeled with radioactive iodine was found to bind preferentially to a highly specific placental receptor different from the insulin receptor. Using identical membrane preparations and incubation conditions, the binding of $^{131}$I-somatomedin-C was 100 times more sensitive to competition by unlabeled somatomedin than when $^{125}$I-insulin was used as the label. Furthermore, neither insulin nor a wide variety of other hormones tested competed with $^{131}$I-somatomedin-C for binding except at high molar concentrations. A competitive binding assay utilizing labeled somatomedin-C has proven useful in monitoring the purification of somatomedin and measuring somatomedin levels in unextracted plasma. With this technique, significant differences were found between plasma somatomedin-C levels in hypopituitary, normal and acromegalic subjects.

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*Phillips, Lawrence S.; Herington, Adrian C.; and Daughaday, William H.* (Dept. of Med., Metab. Div., Washington Univ. Sch. of Med., St. Louis, Mo. 63110): STEROID HORMONE EFFECTS ON SOMATOMEDIN. I. SOMATOMEDIN ACTION IN VITRO. Endocrinology *97*:780, 1975.

The effects of cortisol, estradiol, and testosterone on somatomedin action on cartilage incubated in vitro have been examined. The addition of hormones in the absence of serum had no effect on the incorporation of sulfate by cartilage from hypophysectomized rats, embryonic chicks, or normal young pigs. Normal human serum provided a source of somatomedin which stimulated the incorporation of sulfate by cartilage in a dose-response relationship; the potency of serum with and without added steroid hormone was determined after formal parallel-line analysis. Moderately supraphysiologic levels of cortisol, 17 $\beta$-estradiol, and testosterone generally had little effect on somatomedin action in these test systems. Very high levels of serum cortisol (100-1000 $\mu$g./100 ml.) inhibited somatomedin action on pig cartilage, but had little effect on rat or chick cartilage. A 20 ng./100 ml. increase in serum estradiol had no effect on somatomedin action on chick cartilage, but appeared to enhance somatomedin action on pig cartilage. A 5 $\mu$g./100 ml. increase in serum testosterone did not affect somatomedin action on either chick or pig cartilage.

These studies suggest that the alteration of somatomedin action is not a major mechanism in the effect of steroid hormones on growth. In addition, since modest increases in serum levels of cortisol, estradiol, and testosterone had little effect on somatomedin action in our assay systems, these systems should be satisfactory for the study of hormone effects on somatomedin generation.

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*Cohen, Kenneth L.; Short, Patricia A.; and Nissley, S. Peter* (Metab. Branch, National Cancer Inst., NIH, Bethesda, Maryland): GROWTH HORMONE-DEPENDENT SERUM STIMULATION OF DNA SYNTHESIS IN CHICK EMBRYO FIBROBLASTS IN CULTURE. Endocrinology *96*:193, 1975.

We have investigated the role of GH in the serum requirement for the multiplication of chick embryo fibroblasts (CEFs) in culture. Serum from hypophysectomized (hypox) rats is much less effective than normal serum in stimulating the incorporation of [$^3$H-methyl]thymidine into DNA. More importantly, bovine GH (bGH) treatment of the hypox rat restores 60 per cent or more of the activity in the $^3$H-thymidine incorporation assay. Bovine GH is inactive when tested directly in the assay. Mixing experiments show that the decreased activity of hypox serum is not due to the presence of an inhibitor in the hypox serum. The GH dependent factor is nondialysable and stable to boiling at pH 5.5. Boiling the normal, hypox, and bGH treated hypox rat sera results not only in enhancement of the activity but also a more linear dose response curve in the $^3$H-thymidine incorporation assay. The $^3$H-thymidine incorporation data reflect DNA synthesis because measurements of cell numbers show the CEFs multiply less well in boiled hypox rat serum than in boiled normal rat serum, and bGH treatment of the hypox rat restores approximately half of the multiplication stimulating activity of normal boiled rat serum. CEFs in culture may provide a satisfactory in vitro system for the study of the mechanism of action of the growth hormone dependent anabolic factors found in serum.

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*Yalow, Rosalyn S.; Hall, Kersten; and Luft, Rolf* (Solomon A. Berson Lab., Veterans Administration Hosp., Bronx, New York, and Dept. of Med., Mt. Sinai Sch. of Med., City Univ. of New York, and the Dept. of Endocrin. and Metab., Karolinska Sjukjuset, Stockholm, Sweden): RADIOIMMUNOASSAY OF SOMATOMEDIN B: APPLICATION TO CLINICAL AND PHYSIOLOGIC STUDIES. J. Clin. Invest. *55*:127, January 1975.

A radioimmunoassay has been developed for Somatomedin B, a growth hormone-dependent factor that stimulates DNA synthesis in human glia-like cells. The sensitivity permits detection of this factor in human plasma diluted 1:20,000 and in monkey plasma diluted 1:5,000. It is not measurable in nonprimate plasma diluted 1:20. The concentration in growth hormone-deficient adult patients is equivalent to 6.6±0.5 $\mu$g./ml. of a highly purified somatomedin preparation. In acromegaly the concentration is

19.3 ± 2.3 μg./ml. and falls after definitive therapy that results in a decrease in plasma growth hormone. In unextracted human plasma the immunoreactive Somatomedin B is associated with a plasma protein at least as large as γ-globulin and with an electrophoretic mobility on paper resembling the α-globulins. The level of Somatomedin B in the bound form in human plasma under steady-state conditions may depend on the rate of production of the peptide and/or the concentration of the plasma-binding protein. At present there is no information concerning which of these is modulated by growth hormone. Immunoreactive Somatomedin B is found predominantly in Cohn plasma fractions III and IV, largely dissociated from the plasma-binding protein. The disappearance curves of labeled purified Somatomedin B and of immunoreactive Somatomedin B from acromegalic plasma administered intravenously to a dog were superposable; the terminal portion of the disappearance curve having a half time of almost an hour.

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*Megyesi, Klara; Kahn, C. Ronald; Roth, Jesse; Neville, David M., Jr.; Nissley, S. Peter; Humbel, Rene E.; and Froesch, E. Rudolf* (Diabetes Branch, NIAMDD, NIH, Bethesda, Maryland, Section of Biophysical Chemistry Lab. of Neurochemistry, NIMH, Metab. Branch, NCI, NIH, Bethesda, Maryland, Dept of Med. and Biochemistry, Univ. of Zurich, Switzerland): THE NSILA-S RECEPTOR IN LIVER PLASMA MEMBRANES. CHARACTERIZATION AND COMPARISON WITH THE INSULIN RECEPTOR. J. Biol. Chem. *250*:8990, December 1975.

NSILA-s (nonsuppressible insulin-like activity, soluble in acid ethanol) is a serum peptide that has insulin-like and growth-promoting activities. We have demonstrated previously that liver plasma membranes possess separate receptors for NSILA-s and insulin and have characterized the insulin receptor in detail. In the present study we have characterized the properties and specificity of the NSILA-s receptor and compared them to those of the insulin receptor in the same tissue. Both $^{125}$I-NSILA-s and $^{125}$I-insulin bind rapidly and reversibly to their receptors in liver membranes; maximal NSILA-s binding occurs at 20° while maximal insulin binding is seen at 1-4°. The pH optimum for NSILA-s binding is broad (6.0 to 8.0), in contrast to the very sharp pH optimum (7.5 to 8.0) for insulin binding. Both receptors exhibit a high degree of specificity. With the insulin receptor, NSILA-s and insulin analogues compete for binding in proportion to their insulin-like potency: insulin > proinsulin > NSILA-s. With the NSILA-s receptor, NSILA-s is most potent and the order is reversed: NSILA-s >> proinsulin > insulin. Furthermore, six preparations of NSILA-s which varied 70-fold in biological activity competed for $^{125}$I-NSILA-s binding in order of their potencies. NSILA-s which had been inactivated biologically by reduction and aminoethylation and growth hormone were less than 1/100,000 as potent as the most purified NSILA-s preparation. Purified preparations of fibroblast growth factor, epidermal growth factor, nerve growth factor, and somatomedins B and C were less than 1 per cent as effective as NSILA-s in competing for the $^{125}$I-NSILA-s suggesting that these factors act through other receptors. In contrast, somatomedin A was 10 per cent as active as NSILA-s and multiplication-stimulating activity was fully as active as NSILA-s in competing for the NSILA-s receptor. Analysis of the data suggests that there are approximately 50 times more insulin receptors than NSILA-s receptors per liver cell, while the apparent affinity of NSILA-s receptors is somewhat higher than that of the insulin receptor.

*Jacobs, L. S.; Sneid, D. S.; Garland, J. T.; Laron, Z.; and Daughaday, W. H.* (Metab. Div., Dept. of Med., Washington Univ. Sch. of Med., St. Louis, Missouri, Section of Endocrin. and Metab., Rush Presbyterian St. Luke's Med. Centr, Chicago, Illinois, and Inst. of Pediatric and Adolescent Endocrin., Beilinson Hosp., Petah Tikvah, Israel): RECEPTOR-ACTIVE GROWTH HORMONE IN LARON DWARFISM. J. Clin. Endocrinol. Metab. *42*:403, 1976.

The hepatic radioreceptor assay for hGH has been applied to the detection of hGH in the sera of patients with high growth hormone dwarfism (Laron dwarfism). Substantial quantities of receptor-active hGH were found in the sera of all seven patients studied. In one patient, arginine infusion elicited a prompt increase in both immunoactive and receptor-active hGH. These observations suggest that circulating hGH in Laron dwarfism is biologically active and support the concept that the disease may be caused by a generalized defect in hGH receptors.

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*Kastrup, K. W.; Andersen, H.; and Hanssen, K. F.* (Childrens Hosp., Fuglebakken, and Steno Memorial Hospital, Copenhagen, Denmark): INCREASED IMMUNOREACTIVE PLASMA AND URINARY GROWTH HORMONE IN GROWTH RETARDATION WITH DEFECTIVE GENERATION OF SOMATOMEDIN A (LARON'S SYNDROME). Acta Paediatr. Scand. *64*:613, 1975.

In a boy four years old with clinical hypopituitary dwarfism, high plasma and urinary levels of immunoreactive growth hormone were found. Somatomedin A levels in serum were low and failed to respond after short-term treatment with human growth hormone. The parents were first cousins. In the arginine and insulin tolerance tests the initially high immunoreactive growth hormone levels were later followed by a decrease to high normal values. Insulinopenic response was present during the arginine and glucose tolerance tests. As a growth hormone molecule defect is not found in these patients and no growth or other metabolic response to exogenous HGH can be demonstrated, it is concluded that a defective somatomedin generation may be present, probably in conjunction with a generalized receptor defect and deficient feedback system with abnormal release of HGH. The lack of somatomedin A is responsible for the severe growth retardation and the disturbance in carbohydrate metabolism is probably caused by sustained high growth hormone levels.

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*Fryklund, Linda; Uthne, Knut; Sievertsson, Hans; and Westermark, Bengt* (The Recip Polypeptide Lab. S-104 25, Stockholm, Sweden, and the Wallenberg Lab., Uppsala, Sweden): ISOLATION AND CHARACTERIZATION OF POLYPEPTIDES FROM HUMAN PLASMA ENHANCING THE GROWTH OF HUMAN NORMAL CELLS IN CULTURE. Biochem. Biophys. Res. Commun. *61*:950, 1974.

The isolation in pure form and the chemical characterization is first described here of four polypeptides from human plasma which stimulate [$^3$H]thymidine uptake into glial cells in culture and increase the number of human embryonic lung fibroblasts. The polypeptides have a molecular weight of 5,000. Although they differ in charge the amino acid compositions are essentially identical and each peptide has four disulphide bridges. The amino and carboxyl terminal residues are aspartic acid and threonine, respectively. The peptides are tentatively designated Somatomedin B.

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*Eisenbarth, George S.; and Lebovitz, Harold E.* (Dept. of Med. and

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 $2.5 \times 10^{-10}$M inhibited cartilage metabolism in the same manner as BEI.

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*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.

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*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.

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*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.