labelled molecules which had regained superhelical turns during the chase were not more numerous than those which had replicated. We therefore found no evidence supporting a mechanism whereby superhelical turns would be introduced in polyoma DNA independently from replication.

The effects of puromycin on polyoma DNA biosynthesis which we have just described could be due to the inhibition of the synthesis of several proteins. At least one of these, however, should be a protein which affects the configuration of the viral DNA, and possibly forms a complex with it. Whether this protein would either act by neutralizing the charges of the DNA[13] or have enzymatic activity (twistase ?) is not clear at present.

Experiments undertaken with the aim of identifying the puromycin-sensitive factor(s) with the product(s) of recognized genes of polyoma virus using temperature-sensitive mutants are in progress.

We thank Mrs Monique D'Allaire and Mr Robert Vigneault for technical assistance. This work was supported by a grant from the Medical Research Council of Canada.

PIERRE BOURGAUX
DANIELLE BOURGAUX-RAMOISY
Département de Microbiologie,
Centre Hospitalier Universitaire,
Université de Sherbrooke,
Sherbrooke, Quebec

Received July 9; revised October 5, 1971.

1 Green, M. H., Miller, H. I., and Hendler, S., *Proc. US Nat. Acad. Sci.*, **68**, 1032 (1971).
2 Bourgaux, P., Bourgaux-Ramoisy, D., and Seiler, P., *J. Mol. Biol.*, **59**, 195 (1971).
3 Stanners, C. P., *Virology*, **21**, 464 (1963).
4 Bourgaux, P., Bourgaux-Ramoisy, D., and Dulbecco, R., *Proc. US Nat. Acad. Sci.*, **64**, 701 (1969).
5 Sambrook, J., and Shatkin, A. J., *J. Virol.*, **4**, 719 (1969).
6 Branton, P. E., Cheevers, W. P., and Sheinin, R., *Virology*, **42**, 979 (1970).
7 Kit, S., and Nakajima, K., *J. Virol.*, **7**, 87 (1971).
8 Kang, H. S., Eshbach, T. B., White, D. A., and Levine, A. J., *J. Virol.*, **7**, 112 (1971).
9 Vinograd, J., Lebowitz, J., Radloff, R., Watson, R., and Laipis, P., *Proc. US Nat. Acad. Sci.*, **53**, 1104 (1965).
10 Radloff, R., Bauer, W., and Vinograd, J., *Proc. US Nat. Acad. Sci.*, **57**, 1514 (1967).
11 Hudson, B., Upholt, B., Devinny, J., and Vinograd, J., *Proc. US Nat. Acad. Sci.*, **62**, 813 (1969).
12 Eason, R., and Vinograd, J., *J. Virol.*, **7**, 1 (1971).
13 Murakami, W. T., Fine, R., Harrington, M. R., and Ben Sassan, Z., *J. Mol. Biol.*, **36**, 153 (1968).
14 Fried, M., and Pitts, J. D., *Virology*, **34**, 761 (1968).

Somatomedin: Proposed Designation for Sulphation Factor

THE action of growth hormone (GH) on skeletal tissue was proposed to be mediated through a secondary substance, described by the operational term "sulphation factor"[1]. The observations underlying this hypothesis have been amply confirmed and extended. A GH-dependent plasma factor stimulates in cartilage not only the incorporation of sulphate into chondroitin sulphate, but also the incorporation of thymidine into DNA[2], proline into the hydroxyproline of collagen[3] and uridine into RNA[4]. GH has substantially no *in vitro* effect on cartilage metabolism.

In plasma sulphation factor (SF) circulates associated with the large molecular components but extracts of considerably smaller size with sulphation factor activity have been prepared by denaturating plasma protein by boiling[4] or by extraction with acid ethanol[5]. Van Wyk *et al.*[5] purified their acid ethanol extracts by gel filtration, ion exchange chromatography and electrophoresis. After an approximate 25,000-fold purification, the biological activity could be attributed to a neutral peptide with a molecular weight of about 8,000 (ref. 6).

The spectrum of biological action of SF has been extended by experiments with partially purified SF. Hall and Uthne[7] demonstrated that injections of SF active extracts induced widening of the tibial epiphyseal cartilage. The biological actions of SF are not limited to cartilage. Salmon and DuVall[8] showed that partially purified SF had insulin-like actions on isolated rat diaphragm and Hall and Uthne[7] have shown that similar SF preparations stimulate conversion of ^{14}C-glucose to $^{14}CO_2$ by rat adipose tissue. The insulin-like activity cannot be neutralized by anti-insulin serum and follows SF activity through multiple fractionation procedures[5], suggesting that SF is identical with or very similar to the smaller molecular weight component of the non-suppressible insulin-like activity (NSILA-S) extensively studied by Jakob, Hauri and Froesch[9]. NSILA-S stimulates thymidine incorporation in rat costal cartilage[10]. Recently Salmon and Hosse[11] reported that a serum fraction with SF activity stimulated HeLa cell growth.

Although the mechanism of production and ultimate physiological role of SF remain to be defined, its importance in the growth and anabolic responses of both skeletal and certain non-skeletal tissues can be in no doubt. After consideration of many alternatives to the operational terms "sulphation factor" or "thymidine factor", we propose the more general term, "somatomedin"; the prefix, "somato", connotes both a hormonal relationship to somatotropin and, also, to the soma which is the target tissue of this agent. "Medin" is included in the name to indicate that it is an intermediary in somatotropin action.

WILLIAM H. DAUGHADAY
Department of Medicine,
Washington University School of Medicine,
St Louis, Missouri
KERSTIN HALL
Department of Endocrinology and Metabolism,
Karolinska sjukhuset, Stockholm
MAURICE S. RABEN
Department of Medicine,
Tufts University School of Medicine,
Boston, Massachusetts
WILLIAM D. SALMON, JUN.
Veterans Administration Hospital,
Nashville, Tennessee
J. LEO VAN DEN BRANDE
Department of Pediatrics,
School of Medicine, University of Rotterdam,
Rotterdam
JUDSON J. VAN WYK
Department of Pediatrics,
University of North Carolina, School of Medicine,
Chapel Hill, North Carolina

Received September 6, 1971.

1 Salmon, W. D., and Daughaday, W. H., *J. Lab. Clin. Med.*, **49**, 825 (1957).
2 Daughaday, W. H., and Reeder, C., *J. Lab. Clin. Med.*, **68**, 357 (1967).
3 Daughaday, W. H., and Mariz, I. K., *J. Lab. Clin. Med.*, **59**, 741 (1962).
4 Salmon, jun., W. D., and DuVall, M. R., *Endocrinology*, **86**, 721 (1970).
5 Van Wyk, J. J., Hall, K., Van den Brande, J. L., and Weaver, R. P., *J. Clin. Endocrinol. Metab.*, **32**, 389 (1971).
6 Van Wyk, J. J., Hall, K., Van den Brande, J. L., Weaver, R. P., Uthne, K., Hintz, R. L., Harrison, J. H., and Mathewson, P., *Proc. Second Intern. Symp. Growth Hormone* (edit. by Pecile, A., and Mueller, E. (in the press).
7 Hall, K., and Uthne, K., *Proc. Second Intern. Symp. Growth Hormone* (edit. by Pecile, A., and Mueller, E.) (in the press).
8 Salmon, jun., W. D., and DuVall, M. R., *Endocrinology*, **87**, 1168 (1970).
9 Jakob, A., Hauri, C., and Froesch, E. R., *J. Clin. Invest.*, **47**, 2678 (1968).
10 Raben, M. S., Murakawa, S., and Matute, M., *Proc. Second Intern. Symp. Growth Hormone* (edit. by Pecile, A., and Mueller, E.) (in the press).
11 Salmon, W. D., and Hosse, B. R., *Proc. Soc. Exp. Biol. Med.*, **136**, 805 (1971).