HOPSU-HAVU and GLENNER[2]. This enzyme seems to be excreted into parotid saliva from the salivary gland in a higher concentration than the other aminopeptidases, since the hydrolysis of the substrate was found to be relatively higher in parotid salivary fluid[1]. This may indicate a selective mechanism for the enzyme secretion from the salivary gland. Physiological significance of the presence of this enzyme in the salivary gland and in saliva remains unknown. Since the amino-acid sequence glycyl-proline is predominant in collagen, the enzyme may act on collagen metabolism. The possibility of implication of this enzyme for collagen metabolism has been suggested by HOPSU-HAVU, RINTOLA and GLENNER[7,8]. Since collagen is a main protein in the oral tissues including dentine, physiological and pathological role of this enzyme remains to be elucidated.

Hydrolysis of amino-acid β-naphthylamides by aminopeptidases in bovine parotid gland

Amino-acid β-naphthylamide	Aminopeptidase activity	
	Microsomal fraction nmoles/min/mg protein	Soluble fraction nmoles/min/mg protein
Ala	22	7.9
Arg	4.1	7.0
Asn	1.1	1.2
α-L-Asp	0.0	0.0
Gln	1.2	2.2
α-L-Glu	0.1	0.5
D-Glu	0.0	0.0
γ-Glu	0.0	0.0
Gly	3.6	0.8
Gly-Phe	1.3	0.4
Gly-Pro	31	4.3
Ile	0.5	0.0
Leu	14	3.7
Lys	–	3.7
Met	11	4.8
Norleu	6.7	3.5
Norval	6.3	3.2
Phe	–	5.4
Pro	0.6	0.8
Ser	0.7	0.7
Val	0.6	0.0

Zusammenfassung. Die Aktivität der Aminopeptidasen in Ohrspeicheldrüsen wurde gemessen. Glycyl-Prolin β-naphthylamid, Alanin β-naphthylamid, Leucin β-naphthylamid, Methionin β-naphthylamid, und Arginin β-naphthylamid wurden von der Mikrosomenfraktion und der löslichen Fraktion schnell gespalten. Das Glycyl-Prolin β-naphthylamid spaltende Enzym war in Ohrspeicheldrüsen in relativ grösserer Menge vorhanden. Die Aufspaltung von Glycyl-Prolin β-naphthylamid in Glycyl-Prolin und β-Naphthylamin wurde papierchromatographisch nachgewiesen.

H. OYA, I. NAGATSU[9],
M. HARADA and T. NAGATSU

Department of Biochemistry, School of Dentistry, Aichi-Gakuin University, Nagoya, and Department of Anatomy and Physiology, Aichi Prefectural College of Nursing, Nagoya (Japan), 25 August 1969.

[7] V. K. HOPSU-HAVU, P. RINTOLA and G. G. GLENNER, Acta chem. scand. *22*, 299 (1968).

[8] The authors are grateful to Dr. G. G. GLENNER (National Institutes of Health, Bethesda) for the gift of amino-acid β-naphthylamides and to Dr. S. SAKAKIBARA and Dr. K. TAKADA (Institute for Protein Research, Osaka) for the synthesis of glycyl-L-prolyl β-naphthylamide. The valuable technical assistance of Miss Y. NISHIKAWA and Miss Y. SHIBAHARA is gratefully acknowledged.

[9] Department of Anatomy and Physiology, Aichi Prefectural College of Nursing, Nagoya (Japan).

Effect of Adrenalectomy on the Response of Rat Liver Polyribosomes to Whole-Body Gamma Radiation

Polyribosomes from regenerating rat livers[1,2] and from intact livers of both rats and guinea-pigs exposed to whole-body X-irradiation are characterized by a marked shift in their pattern of distribution[3,4]. After either of the above treatments, the liver C-ribosome profile on a linear sucrose density gradient shows a marked decrease in monomers and smaller oligomers and a concomitant increase in heavier aggregates. In contrast, whole-body exposure of rats with regenerating livers to either gamma or neutron radiation within a few hours after surgery initially prevents the characteristic shift in the polyribosome pattern normally observed 24 h after partial hepatectomy. However, when a longer time interval is allowed between surgery and killing (36 h), a heavy aggregate profile, characteristic of 36 h regenerating liver from unirradiated animals, is obtained[2,5].

In this communication we report experiments with adrenalectomized rats which were performed in an attempt the better to understand the varying response to ionizing radiation obtained with intact and regenerating rat liver. Unfortunately the data presented deal only with the effect of adrenalectomy and radiation on rats with intact liver because the combined stress of adrenalectomy, partial hepatectomy and radiation employed in these studies was incompatible with survival.

Material and methods. Male Badger rats (Badger Research Corp., Madison, Wisconsin, USA) weighing 240–260 g were used. The rats were bilaterally adrenalectomized and given ad libitum access to a 1% NaCl drinking solution. 24 and 48 h after surgery, some rats were injected s.c. with one adrenal gland, which had been prepared for injection by homogenizing in 0.5 ml of 0.85% NaCl.

One hour after the second adrenal gland injection the rats were subjected to whole-body radiation. Irradiation at 60 R/min was delivered by a 2000 Ci ^{137}Cesium source emitting 662 KeV gamma rays. All rats were fasted for 20 h prior to killing (by decapitation) and their liver polyribosomes prepared and analyzed as previously reported[2].

[1] P. CAMMERANO, G. GIUDICE and B. LUKES, Biochem. biophys. Res. Commun. *19*, 487 (1965).

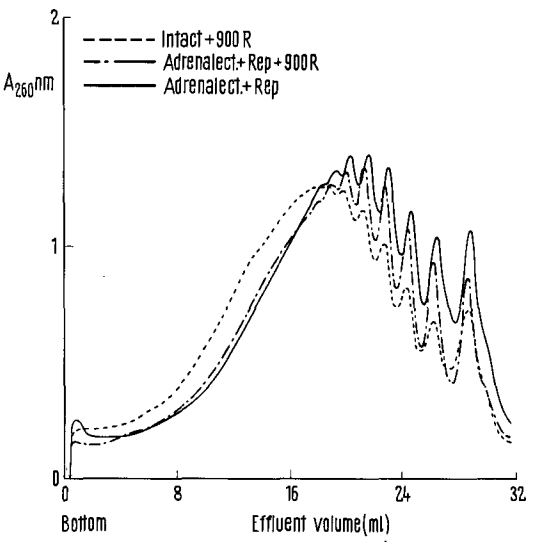
[2] M. B. YATVIN and T. P. LATHROP, Biochem. biophys. Res. Commun. *25*, 535 (1966).

Results and discussion. As expected from previous reports[3,4], the curves in the Figure and the data in the Table show a marked shift in liver polyribosome species of intact rats 20 h after whole-body gamma radiation. In contrast, no polyribosome response was obtained in liver of adrenalectomized, irradiated rats, even when exposed to doses up to 10,800 R (Table).

Furthermore, 3 days after adrenalectomy, polyribosome distributions were similar in livers of adrenalectomized only and unirradiated intact rats (Table).

Rats were killed 3 days after adrenalectomy in order to allow them some time to recover from the operation. Longer time intervals between adrenal extirpation and radiation were avoided in an attempt to obviate any influence hypertrophy or hyperplasia of accessory adrenal tissue might have had on the interpretation of data.

Though it had no apparent effect on polyribosomal patterns, replacement therapy was continued in the expectation that it would aid in maintaining the animal in a more 'normal' physiological state. Thus the failure to obtain a radiation shift in the pattern of ribosome distribution in adrenalectomized corticoïd supplemented rats is probably not attributable to any non-specific debilitating effects of adrenalectomy. Furthermore, the polyribosome yield per gram of wet weight of liver was unaltered by any of the treatments employed. Therefore, it would appear that failure to obtain a polyribosome response is the result of the inability of adrenalectomized rats to pour out adrenal corticoïds as could intact rats under the stress of radiation.



The C-ribosome patterns of liver from irradiated intact rats (900 R); adrenalectomized rats given adrenal replacement therapy and 900 R and non-irradiated adrenalectomized rats given adrenal replacement therapy. The livers were obtained 20 h after either irradiation and fasting or 3 days after adrenalectomy.

Treatment	% Heavy aggregates
Intact	40.8 ± 2.1
Adrenalectomized	41.7 ± 3.4
Adrenalectomized + rep.[a]	42.8 ± 2.2
Adrenalectomized + rep. + 900 *R*	40.8 ± 1.9
Adrenalectomized + rep. + 3600 R	44.8 ± 3.2
Adrenalectomized + rep. + 10,800 R	38.5 ± 2.8
Adrenalectomized + cortisone[b]	65.7 ± 5.3
Intact + 900 R	59.1 ± 3.6
Intact + 1500 R	57.4 ± 2.9

[a] Adrenal replacement therapy (1 gland/day). [b] Cortisone acetate 10 mg/100 g body weight. Percent of aggregates = 100 × ratio of heavy polyribosomes to total number of liver polyribosomes arbitrarily defining heavy polyribosomes as the area under the distribution curve beyond the first 6 peaks (18 ml). Each average value (± S.E.) is derived from 4 individual liver patterns.

The observation of a marked shift in polyribosome distribution 24 h after administering 10 mg per 100 g body weight of cortisone acetate to adrenalectomized rats (Table) supports the above interpretation. In contrast, BAEYENS and GOUTIER (personal communication) found that the livers of their adrenalectomized rats are capable of responding to radiation as early as one day after adrenal removal. However, this apparent difference in the liver polyribosome response of adrenalectomized rats to radiation could well be the result of strain differences in the rats used in their studies, since accessory adrenals are common in some strains of white rats after adrenalectomy[6,7].

The problem of how to rationalize the different early responses of normal and regenerating livers in animals still remains, however. Perhaps as BAEYENS and GOUTIER[3] have suggested in attempting to explain our results with regenerating liver[2,5], the difference might be due to the presence of m-RNA populations with different turnover rates in normal and in regenerating liver. Based on the current study, it is not possible to determine whether the explanation for this different radiation response of normal and regenerating liver is due to a differential effect of radiation on m-RNA metabolism, or is the result of an altered endocrine balance in the partially hepatectomized rat, which prevents the shift in polyribosome distribution. Nevertheless, in the intact rat the present results indicate that the action of irradiation on liver polyribosomes is mediated, at least in part, by the adrenal gland[8].

Résumé. L'adrénalectomie supprime le net déplacement, causé par l'irradiation, vers les espéces de polyribosomes plus lourdes observées chez les rats normaux irradiés. Ces résults font supposer que l'action de l'irradiation sur les polyribosomes du foie est réglée au moins en partie, par la glande surrénale.

M. B. YATVIN[9]

Radiobiology Research Laboratory,
Department of Radiology and Pathology,
University of Wisconsin Medical School,
Madison (Wisconsin 53706, USA), 12 September 1969.

[3] W. BAEYENS and R. GOUTIER, Archs. int. Physiol. Biochim. *76*, 785 (1968).
[4] E. J. HIDVEGI, J. HOLLAND, E. BÖTÖNI, P. LONAI, F. ANTONI and V. VARTERESZ, Biochem. J. *109*, 495 (1968).
[5] M. B. YATVIN, J. L. MITCHEN, A. L. WILEY JR. and H. H. VOGEL JR., Biochim. biophys. Acta *169*, 536 (1968).
[6] H. L. JAFFÉ, Arch. Path. Lab. Med. *3*, 414 (1927).
[7] J. M. LASCANO-GONZALES, C. r. Soc. biol. *116*, 451 (1934).
[8] This work was supported in part by Public Health Service Research Grant No. CA08318 and Center Grant No. CA06295 from the National Institutes of Health. We thank Miss SHARON K. CURTIS for excellent technical assistance.
[9] Recipient of U.S. Public Health Service Research Career Development Award No. CA38655 from the National Cancer Institute and on special leave from August 1969–July 1970 at the Département de Radiobiologie, Centre d'Etude de l'Energie Nucléaire, Mol (Belgium).