

Thus, the participation of secretin or enterogastron, which is assumed to be released by duodenal souring (around pH 1.5–2.0) or presence of fat in the duodenum¹³, appeared to be dismissible from the inhibitory factors. The distention of the duodenum by the secreted juice, as well as the ligation procedure itself, may influence the gastric secretory conditions, by mechanisms so far unknown. However, in dogs distention of the duodenum per se was reported to increase the gastric secretion, because of the act of the so-called 'intestinal phase'¹⁴. In addition, 2 of 10 animals subjected to A ligation had almost no juice in the duodenum. Thus, the participation of distention of the duodenum to the inhibitory mechanisms may be negligible. Further study concerning the detailed mechanisms seems to be worthwhile.

Résumé. La ligature de l'intestin, particulièrement du duodénum, gêne la sécrétion gastrique chez le Rat à pylore lié avec ou sans fistule fine.

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¹³ S. ANDERSSON, in *Alimentary Canal* (Eds. C. E. CODE and W. HEIDEL; American Physiological Society, Washington 1967).

¹⁴ W. SIRCUS, Q. J. exp. Physiol. 38, 91 (1953).

Do Chickens have Gastrin-Like Compounds?

It has been reported that in chickens gastrin is located in the mitochondrial fraction of the duodenum¹. Recently, using BLAIR's extraction procedure for gastrin², we found no gastrinlike activity in any parts of the chicken's upper gastrointestinal tract, bioassaying it either in an anaesthetized rat³ or in conscious gastric fistula chickens⁴. However, in the experiments with anaesthetized rats, a very low gastrin-like activity in duodenal extracts could not be completely excluded³. It was therefore of interest to reinvestigate the problem radioimmunologically.

From 10 chickens venous blood samples were withdrawn after 18 h of starvation and after subsequent refeeding for 30 min. Serum was obtained by spinning down the clot formed within a few minutes. Another group of 10 chickens was killed by cutting the throat. The upper gastrointestinal tract was dissected into oesophagus crop, glandular stomach, gizzard, and duodenum (the first 10 cm distally from the gizzard). The mucosa was scraped from the organs, homogenized in 4 volumes of ice-cold 1/15 M phosphate buffer (pH 7.0), and boiled for 5 min. The pancreas in total was treated in the same way. The boiled homogenates were filled up to the original volume with distilled water and were then frozen. The frozen material was taken by car to one of the authors (H. K.) for the radioimmunoassay of gastrin. The experimental procedure was in principle that described by MCGUIGAN⁵ as modified by FEURLE et al.⁶ using synthetic human gastrin 2–17 (SHG 2–17) as antigen. The investigator identified the samples only by numbers.

The following concentrations of immunoreactive gastrin-like material (IGM) were found:

Serum	a) after starvation	0 pg/ml
	b) after refeeding	0 pg/ml
Oesophagus		0 pg/g
Crop		0 pg/g
Glandular stomach		0 pg/g
Gizzard		0 pg/g
Duodenum		19 ± 8 pg/g
Pancreas		0 pg/g

The only organ cross-reacting with antibodies against SHG 2–17 was the duodenum. Although the assay was done under blind conditions, none of the duodenal extracts was found to be free of IGM. These results are in agreement with those of BLAIR et al.¹. However, the

concentration of IGM we found was much less than the gastrin-like biological activity found by these authors. This may be due to the fact that chicken's gastrin cross-reacts only very poorly with antibodies against SHG 2–17. If so, it must differ chemically from human gastrin. This hypothesis is supported by the fact that dilution curves of the duodenal extracts are much flatter than those obtained with human gastrin. Presumably, if only 1% or less of the IGM in the duodenal mucosa was detectable by this method, even then the concentration is very much lower than that found in the mammalian antrum. This finding, together with the poor responsiveness of chickens to pentagastrin⁴, and the fact that no rise in serum IGM in response to feeding could be observed, makes it unlikely that this IGM is the main source for the humoral control of gastric acid secretion in chickens. To speculate: There is either another big pool of gastrin in chickens hitherto undetected or the chicken represents a species in which the humoral control of gastric acid secretion by gastrin-like compounds is not a very important factor.

Zusammenfassung. Bei Hühnern wurden in Extrakten der Schleimhaut des Duodenums geringe Mengen einer Substanz nachgewiesen, die radioimmunologisch mit Antikörpern gegen synthetisches Humangastrin 2–17 reagiert. Serum, Oesophagus, Kropf, Drüsenmagen, Hornmagen und Pankreas enthielten keinerlei Aktivität. Die gastrinähnliche Substanz spielt vermutlich für die Steuerung der Magensekretion bei Hühnern keine wesentliche Rolle.

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¹ E. L. BLAIR, H. S. A. SHERRATT and D. D. WOOD, *Biochem. J.* 104, 54P (1967).

² E. L. BLAIR, A. A. HARPER, H. J. LAKE, J. D. REED and T. SCRATCHERD, *J. Physiol., Lond.* 156, 11P (1961).

³ H.-J. RUOFF and K.-Fr. SEWING, *Naunyn-Schmiedeberg's Arch. Pharmak.* 265, 301 (1970).

⁴ H.-J. RUOFF and K.-Fr. SEWING, *Naunyn-Schmiedeberg's Arch. Pharmak.* 267, 170 (1970).

⁵ J. E. MCGUIGAN, *Gastroenterology* 54, 1005 (1968).

⁶ G. FEURLE, H. KETTERER, H. D. BECKER and W. CREUTZFELD, *Scand. J. Gastroenterol.* 7, 177 (1972).

⁷ Supported by the Deutsche Forschungsgemeinschaft.