by decapitation at the different times of the day indicated in the Table; the eyes were removed and fixed in a mixture containing alcohol, formaline and acetic acid, stained and mounted in glass slides as described in previous work[2]. Mitosis were counted with the use of a microscope with a reticulum (8 × 8 mm) ocular (× 8) and oil immersion lens (× 100). 50 (125 × 125 μ) fields were counted in each cornea. All phases of mitosis were included but an arbitrary elimination was done of those cells in early prophase and later telophase. All animals were received and kept in the department's animal quarters for 1 week prior to their use in the experiments, on a commercial diet ad libitum and a natural regimen of day light and temperature.

In 2 control experiments, groups of 5 rats each were sacrificed at 15.00, 19.00, 23.00, 03.00, 07.00 amd 10.00 (Table). In a third experiment hepatectomy was carried out in a group of 75 normal rats, between 09.00 and 11.00. The sacrifice of each group of 5 rats started at 19.00 of the same day in which hepatectomy was performed; the subsequent groups were sacrificed at 4 h intervals up to 52 h post hepatectomy. The results obtained both with normal and partially hepatectomized rats are presented in the Table.

Circadian distribution of mitosis in the cornea of normal and partially hepatectomized rats

Time of day	Mitosis/50 high power microscopic fields ± S.E.[a]			
	Control[b]	Control II	70% hepatectomized	
19.00	16 ± 6.9	9.4 ± 5.1	47 ± 8.3	(8 $P < 0.02$)[c]
23.00	26 ± 3.4	30 ± 2.6	104 ± 30	(12 $P < 0.02$)
03.00	101 ± 20	114 ± 9.5	121 ± 32	(16 $P > 0.05$)
07.00	228 ± 28	224 ± 21.8	191 ± 46	(20 $P > 0.05$)
10.00	172 ± 6.9	176 ± 14	148 ± 42	(24 $P > 0.05$)
15.00	108 ± 2	125 ± 13.6	197 ± 21	(28 $P < 0.005$)
19.00			16 ± 4	(32 $P > 0.05$)
23.00			148 ± 37	(36 $P < 0.02$)
03.00			194 ± 22	(40 $P < 0.02$)
07.00			396 ± 87	(44 $P > 0.05$)
10.00			246 ± 13	(48 $P < 0.01$)
15,00			132 ± 25	(52 $P > 0.05$)

Time of day, the hour in which the rats were sacrificed; [a] S.E., standard error of the mean values; [b] two separate control experiments carried out with 1 month interval; [c] hours post partial hepatectomy and P values when compared to normal controls.

The results obtained with the 2 control groups indicate that no significant day to day variations occur in the number of cells in mitosis when comparing control animals sacrificed at comparable time periods of the day.

The results also show that the general distribution of mitosis throughout the day is practically the same in both the normal and in hepatectomized animals, with a period of minimal occurrence of mitosis at 19.00 and a peak at 07.00. While the general circadian distribution of mitosis is practically the same in both groups, statistically significant higher levels of mitosis were found in several of these groups of partially hepatectomized rats, when compared to their normal controls. The P values for the groups sacrificed at the different hours post partial hepatectomy were as follows: 8 h, $P < 0.02$; 12 h, $P < 0.02$; 16 h, $P > 0.05$; 20 h, $P > 0.05$; 24 h, $P > 0.05$; 28 h, $P < 0.005$; 32 h, $P > 0.05$; 36 h, $P < 0.02$; 40 h, $P < 0.02$; 44 h, $P > 0.05$; 48 h, $P < 0.01$ and 52 h, $P > 0.05$, when compared with their respective (hour of the day at which the control animals were sacrificed) normal controls.

In summary, these results indicate that the mechanism(s) which synchronize the occurrence of mitosis in the cornea of normal animals is still active in hepatectomized rats as: (1) the periods of the day in which maximal and minimal numbers of cells in mitosis occur in the corneas of partially hepatectomized rats and in control rats is similar, as also is the overall distribution of mitosis; (2) that increased mitotic activity is found particularly in the second day following partial hepatectomy[5].

Zusammenfassung. Die Resultate zeigen, dass der oder die Mechanismen, welche die Mitosis in der Cornea von normalen Tieren synchronisieren, nach partieller Hepatektomie immer noch aktiv sind.

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Pancreozymin/Cholecystokinin a Physiological Mediator of Gastric Secretory Inhibition of Duodenal Origin

The most highly purified form of pancreozymin/cholecystokinin (PZ/CCK)[1] will stimulate gastric acid secretion and potentiate the stimulatory action of methacholine on gastric acid. On the other hand PZ/CCK antagonizes gastric acid secretion which has been stimulated by feeding or by i.v. gastrin pentapeptide[2,3].

If this is a physiological property of PZ/CCK then endogenously released hormone should manifest the same actions. We have demonstrated that this is the case in conscious dogs with Heidenhain gastric pouches. Four of these had duodenal fistulae in addition and 3 had Thiry Vella loops of duodenum. Gastric secretion was stimulated with a continuous i.v. infusion of either methacholine or gastrin pentapeptide (Ayerst Laboratories). In every case when methacholine was the gastric acid stimulant, the

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The effect of i.v. secretin and acid in a Thiry Vella loop, with and without mucosal anesthesia, on acid secretion from a Heidenhain pouch using either methacholine or gastrin pentapeptide as acid stimulants.

Stimulus	Acid		Acid + Oxaine		Secretin	
	Before	After	Before	After	Before	After
Methacholine 4 γ/min	3.1 ± 0.9	5.4[a] ± 0.57	3.4 ± 1.2	2.8 ± 1.08	1.05 ± 0.32	0.45[a] ± 0.39
Gastrin pentapeptide 2 γ/min	2.7 ± 0.6	1.6[a] ± 0.3	4.0 ± 1.6	4.3 ± 1.4	1.11 ± 0.43	0.37[a] ± 0.20

HCl is expressed as mEq/10 min ± the standard error. [a] paired differences significant at the 95% level or higher.

introduction of N/10 HCl into the duodenum or Thiry Vella loop increased gastric acid secretion (Table). On the other hand, when gastrin pentapeptide was the stimulus acid secretion was diminished. In 4 animals the gall bladder was cannulated for pressure recording. N/10 acid contracted the gall bladder whether gastric acid secretion was increased or decreased.

Acidification of the duodenum releases secretin as well as PZ/CCK into the circulation, but secretin cannot be responsible for the observed increased stimulation of gastric acid when i.v. methacholine is the background stimulus, since i.v. secretin inhibits methacholine stimulated gastric acid (Table).

A likely explanation for our results is that PZ/CCK is the mediator of the well known gastric secretory inhibition produced when the duodenum is acidified[4]. Further support for this resides in the fact that if the duodenum is topically anesthetized acidification of the duodenum will no longer effectively stimulate either pancreatic enzyme production[5] or gall bladder contraction[6]; neither will it potentiate gastric acid secretion stimulated by methacholine nor inhibit gastric secretion stimulated by feeding[6].

Both CCK/PZ and duodenal acid inhibit basal motility in transplanted fundic pouches.

A difficulty in the way of accepting any hormone as the mediator of gastric secretory inhibition of duodenal origin is the reported failure to obtain inhibition of secretion from denervated gastric pouches[6]. This matter is still controversial, but we (Table) and others[4] have demonstrated inhibition of secretion from Heidenhain pouches by acidification of the duodenum[7].

Résumé. La Pancréozymine-cholécystokinine (PZ) augmente la sécrétion de l'acide gastrique stimulée par la méthacholine, mais diminue la réponse acide à la gastrine pentapeptide. L'acidification de l'anse Thiry-Vella du duodénum fait augmenter pareillement la sécrétion acide de la poche d'Heidenhain stimulée par la méthacholine et la diminue quand le stimulant est de la gastrine pentopeptide. Il est bien connu que l'acidification du duodénum libère le PZ de la muqueuse duodénale. Nos résultats suggèrent que le PZ est l'agent médiateur d'abaissement de l'acidité gastrique après acidification du duodénum.

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Etude comparative des besoins minima en oxygène chez différentes espèces de Téléostéens

La différence fondamentale des échanges respiratoires observée chez plusieurs espèces de Téléostéens[1] nous a conduit à préciser leurs besoins minima en oxygène.

L'étude comparative a été faite chez les Cichlidés, *Aequidens latifrons*, les Cyprinidés, *Carassius auratus* et les Cyprinodontidés, *Xiphophorus helleri*, en utilisant un appareil enregistreur automatique qui permet d'effectuer des expériences de longue durée.

Méthode d'étude. Une chambre respiratoire de 150 cm³ de volume a été utilisée pour étudier 3 groupes expérimentaux: *A. latifrons* 2–8 g, *C. auratus* 2–10 g, *X. helleri* 0,9–1,5 g. Elle fait partie d'un appareillage automatique enregistrant simultanément la consommation d'oxygène et l'activité des poissons[2,3].

Une macroélectrode de type Clark, branchée dans la chambre respiratoire, mesure les variations de la pression partielle d'oxygène dans le milieu. Celles-ci sont enregistrées par une table enregistreuse Beckman. La température d'expérience est de 25 °C.

L'eau de la chambre respiratoire est préalablement saturée d'air. Le passage d'un courant d'azote provoque la diminution de l'oxygène jusqu'à une pression de 50 mmHg. C'est à partir de cette tension que s'effectue l'enregistrement de la baisse de la teneur en oxygène.

Pour éviter la réoxygénation à partir de l'air, l'eau est recouverte d'une couche d'huile de paraffine. Dès que le poisson présente des signes de gênes respiratoires, l'eau

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