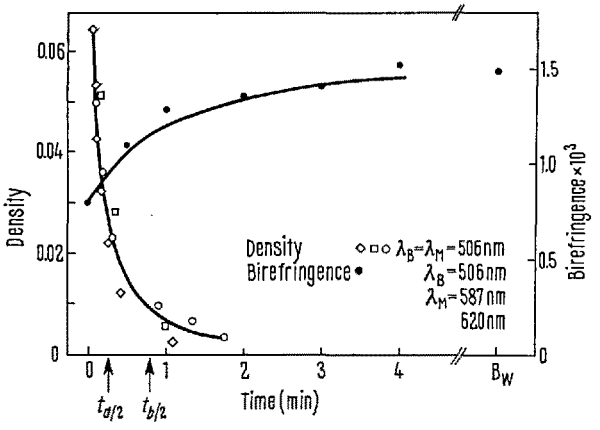
quantitative brackets given by SCHMIDT[3]. The change occurs only during light exposure. Data obtained with other measuring wavelengths show that the results for the earlier, fast (dichroic) component have the same photo-sensitivity as rhodopsin and agree spectrally with its extinction coefficient. The retardance spectrum of the slower (birefringence) function is independent of the measuring wavelength.

Measurements with $\lambda = 470$ nm revealed dichroism attributable to a photoproduct, i.e. the third component mentioned at the beginning. It disappeared with a half-time of several minutes[6]. Hence the reaction with the time-constant of 46 sec is unlikely to be a property of rhodopsin or of any of its photoproducts, although additional measurements show that the photolysis of rhodopsin causes it. The chain of events following the absorption of light by rhodopsin provides a clock for timing the occurrence of events connected with it: as the disruption of the rhodopsin complex in the rod proceeds very slowly and is unlikely to have passed within a few minutes of photolysis the stage at which both metarhodopsin I and II are in equilibrium, the rod material is probably altered soon after photic absorption. The aforementioned changes in the birefringence of nerve fibres during the passage of an action potential[1] are faster than those reported here; but those in the rods are much larger, owing to the favourable observing conditions. It remains to be seen if they relate to the visual process, and what happens following a flash. Further details will be published elsewhere.



Comparison between the time courses of photolysis (left scale) and a change in birefringence (right scale) of isolated frog rods as a function of exposure time (abscissa) to light of 3.03×10^{18} q_{506} nm/m²/s. The half-times of the 2 changes are indicated by the arrows and marked $t_{d/2}$ (density change) and $t_{b/2}$ (change in birefringence) respectively. The wavelength of the bleaching light is given by λ_B, that of the measuring light by λ_M. Birefringence is expressed as Retardance/path length.

Zusammenfassung. In distalen Stäbchen der Froschretina entwickelt sich nach Belichtung eine Änderung der Doppelbrechung als Ausdruck des photochemischen Exitationsprozesses. Letzterer wird in Analogie mit der nach Transmission eines Aktionspotentials auftretenden Änderung der Transmission des Nerven gesetzt.

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Chronic Effects of Nicotine on Gastric Secretion in Vagotomized Rats

Tobacco smoking has been incriminated as an etiologic agent in peptic ulcer formation in man[1]. Furthermore, the mortality from duodenal ulcer disease is greater for smokers than for non-smokers[2]. We have recently shown that nicotine when given chronically (2 weeks) to rats, produces an increase in basal gastric acid and pepsin outputs[3]. The precise mechanism of this nicotine effect is not clear but may involve central stimulation. Since central stimuli usually act via the vagus nerve, we investigated the effect of chronic nicotine exposure on gastric secretion in vagotomized rats.

Materials and methods. 24 male Sprague-Dawley rats[4] weighing 326.6 ± 3.6 g were used. They were housed as described previously[5] and randomly divided into 2 groups of 12 rats each; one group was vagotomized, whereas the second was sham operated. Bi-lateral abdominal vagotomy was performed under pentobarbital[6] anesthesia (50 mg/kg) by LAMBERT's method[7]. Control rats were sham operated; sham operation consisted of an upper abdominal laparotomy under pentobarbital anesthesia. From the first post-operative day, 6 vagotomized and 6 sham operated rats were injected s.c. daily for 14 days with nicotine (2000 µg base/2.0 ml/kg); control vagotomized and sham operated rats received nicotine control saline (2.0 ml 0.85 g/100 ml w/v NaCl/kg). All rats survived the operation and after 14 days of injections they were isolated from food for 40 h as described previously[5]. Basal, unstimulated gastric secretion was collected following pylorus ligation. Preparation of nicotine and control injectables, details of animal care and housing, and techniques of gastric juice collection and analysis have been presented previously[3,5].

Results. Gastric secretory data are presented in the Figure. In sham operated rats chronic nicotine injections significantly increased basal gastric juice volume ($P < 0.025$), acid output ($P < 0.05$) and pepsin output ($P < 0.01$). Following vagotomy however, there were no differences between chronic nicotine-, or chronic nicotine control-injected rats.

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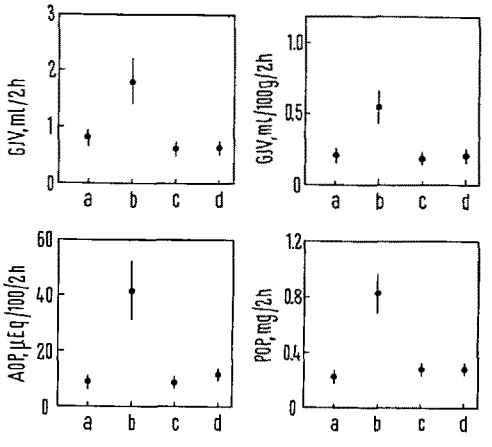
In both groups of vagotomized rats the stomachs were dilated and contained food residue in spite of the 40 h isolation period. This food retention is presumably responsible for the gastric secretion obtained from these rats. Food retention, and gastric dilatation are expected following vagotomy due to absence of vagal motor stimulation. Body weight gain over the 14 days of injections was reduced following vagotomy ($P < 0.05$) as compared to the sham operated rats.

Discussion. The results presented here show that abdominal vagotomy in rats prevents nicotine-induced gastric secretory stimulation. Chronic oral or parenteral nicotine administration has been shown previously to increase gastric juice volume and acid and pepsin outputs in rats[3,8]. The cause of this nicotine effect is not known but several possible mechanisms may be operating induction of histidine decarboxylase, histamine release, or central vagal activation. We have previously shown[9] that nicotine induces histidine decarboxylase activity and releases histamine in the rat stomach suggesting that these effects are responsible for the gastric secretory stimulation. Support for this concept is provided by KIM and SHORE[10] who reported that vagotomy inhibited or blocked depletion of gastric histamine by reserpine. However, recently GLICK et al.[11] have shown that vagotomy and pyloroplasty in rats are followed by increased histidine decarboxylase activity in both fasted and fed animals.



Gastric juice volume (GJV) and volume/100 g, acid output (AOP) and pepsin output (POP) in sham-operated and vagotomized rats. Each group is the mean ± S.E.M. of 6 rats. Chronic NaCl (2.0 ml 0.85 g/100 ml w/v NaCl/kg) and nicotine (2000 µg base/2.0 ml/kg) were given daily s.c. for 14 days commencing on the first post-operative day. Gastric secretion was collected in 2 h pylorus-ligated rats. There are no differences for any parameter between the vagotomized rats injected with NaCl or nicotine. Significant differences in the sham-operated rats are as follows: GJV and GJV/100 g ($P < 0.025$), AOP ($P < 0.05$) and POP ($P < 0.01$). a) Sham operation: chronic NaCl. b) Sham operation: chronic nicotine. c) Vagotomy: chronic NaCl. d) Vagotomy: chronic nicotine.

Gastric secretion in the rat is depressed by vagotomy[7]. Failure to show this here in the control rats is probably directly dependent upon the food retained in the stomach due to delayed gastric emptying, a sequalae of vagotomy[12]. Certainly, gastric histamine formation (of similar magnitude to that induced by gastrin or 2 deoxyglucose) has been shown in the rat following gastric distention[13]. The absence of nicotine-induced gastric secretory stimulation following vagotomy is not proof of a primary central vagal activation since the vagus has a 'permissive' effect on gastric secretion; vagotomy reduces the responsiveness of the parietal and chief cells to both histamine and gastrin, and reduces the release of gastrin from the gastrin antrum[14,15,16].

In view of the deleterious effects of tobacco smoking on peptic ulcer disease in man[1,2], and the frequency with which vagotomy is performed as part of surgical treatment, the results presented here are of great potential interest[17].

Résumé. La sécrétion gastrique a été mesurée dans des rats mâles avec ligature pylorique de deux heures, après 14 jours d'injections sous-cutanées de nicotine ou de NaCl. Les résultats de l'administration de nicotine furent une augmentation du volume du suc gastrique, de la production d'acide et de la production de pepsine. La vagotomie abdominale bilatérale a prévenu la stimulation gastrique secreteuse, provoquée par la nicotine.

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Effects of Acute Hypothermia on the Chick ERG

In the previous paper, OOKAWA and TATEISHI[1] reported the relation between gradually decreased body temperature and the electroretinogram (ERG) in the developing chick. Further investigation on the chick's ERG was made under the condition of dark adaptation during rapidly decreased body temperature using the ice-pack, as herein reported.

Method. 8 male White Leghorn chicks (Goto-201 line), age 12–16 days after hatching, were used. For the purpose of rapid cooling, the ice-pack was placed on the back at the rectal temperature of 39 to 40 °C. Body temperature was checked by the thermister placed in the rectum. Under local anesthesia (Benoxil, Osaka), both eyelids, the nictiating membrane and upper edge of the