defect in the mesosalpinx is then repaired using several 10-0 nylon sutures. The remaining 2 animals underwent double transection surgery without reversing the orientation of the segment. These oviducts served as surgical controls. Unoperated contralateral oviducts in all 4 animals served as additional controls. 9–12 months after surgery, animals were bred to males of proven fertility and examined 2 weeks later at laparotomy. The numbers of corpora lutea and uterine implantations were recorded. Following delivery and return to estrus, animals were induced to ovulate with an i.v. injection of 100 IU hCG. 12–14 h later, a laparotomy was performed and the reproductive tract was exteriorized and examined. A normal complement of fresh ovulation points (3–8) was observed in the ovaries of all 4 animals. 3–6 individual rabbit ova in cumulus obtained from donor animals previously injected with 100 IU hCG and supravitally stained with methylene blue were placed on the fimbria of operated oviducts and their in vivo transport within the ampulla examined with an operating microscope (Zeiss OPMI 6). The oviducts were then excised, placed in oxygenated Krebs solution at 37 °C, cut open and pinned flat, endosalpinx up. 3–6 additional stained ova in cumulus were placed at various locations along the ampulla and their transport similarly examined in vitro.

*Results and discussion.* Microsurgical reversal of a 1-cm segment of mid ampulla prevented pregnancy. No implantations occurred in those uterine horns associated with oviducts that had undergone segmental reversal, whereas normal pregnancy occurred in all others, including those whose oviducts had undergone double transection without reversal (table). Ova in cumulus placed on the fimbrial surface of oviducts that had undergone ampullary segmental reversal were transported in normal fashion through the ampulla until reaching the interface between normal ampulla and the reversed segment. At this point, net forward movement ceased. Ova failed to enter and pass beyond the reversed segment despite observation times in excess of several hours. When mechanically displaced into the reversed segment, ova were transported toward the ovary, but again became arrested when they reached the anastomosis between normal tube and the reversed segment. In contrast, transampullary movement of ova was normal in double transected controls. Ova were transported across both anastomosis sites to the ampullary isthmic junction. In vitro ovum transport results were the same and confirmed that cilia within reversed segments of ampulla continued to beat in a coordinated manner in the ovarian direction, counter to that in the rest of the oviduct, and thereby effectively impeded transport of ova across the reversed segment and into the uterus.

It appears that in the rabbit, so long as the structural integrity of the cells within a reversed segment of ampulla is maintained, the originally programmed direction and coordination of ciliary beating is maintained and is not altered or reprogrammed by adjacent ciliated cells in normal segments of ampulla.

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## Abdominal vagotomy attenuates drinking induced by intravenous infusion of various osmotic loads in the rat

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*Summary.* Male rats were subjected to sham surgery or total abdominal vagotomy and then subsequently received a chronic i.v. cannula. Vagotomy attenuated the dipsogenic effects of infusions of hypertonic saline and sucrose that were observed in the control rats.

A controversy exists about whether central osmoreceptors or sodium receptors mediate the behavioral effects of bodily water imbalances[1,2]. With respect to receptors located in the viscera, recent electrophysiological research has provided evidence that hepatic sodium- and osmo-sensitive cells both activate neurons in the ventrobasal thalamus of rats[3]. It has been suggested that these visceral receptors project to the brain via afferent vagal fibers[4]. It is consistent with this view that behavioral investigations have demonstrated that abdominal vagotomy attenuates the dipsogenic effect of the i.p. injection of hypertonic saline[5,6]. The present experiment was done to determine whether this vagally-mediated visceral drinking system is sensitive to nonionic perturbations of plasma osmolality, as well as to changes in the sodium ion concentration.

*Methods.* The subjects were 13 male Long-Evans pigmented rats obtained from Simonsen Laboratories (Gilroy, CA). Throughout the experiment these rats received Purina Lab chow and tap water ad libitum, except during the drinking tests when food was absent.

Prior to denervation or control surgery, all rats were fasted for about 12 h. The subjects were anesthetized with pentobarbital sodium (50 mg/kg b.wt) and then underwent either bilateral subdiaphragmatic vagotomy including transection of the hepatic branch of the vagus or a sham surgery procedure. The details of these surgical procedures are provided elsewhere[7]. During the subsequent 6-12-week recovery period, these rats received palatable foods as a supplement to their regular diet to facilitate the maintenance of normal body weight gain.

Following recovery from the trauma of the 1st surgical intervention, each rat underwent surgery, receiving a Silastic cannula in the hepatic-portal vein. The details of this cannulation method are described elsewhere[7]. During

Water intake and latency following hepatic-portal infusions in vagotomized (VAGX) and sham operated (SHAM) rats

| | Vehicle | 0.38 M NaCl | 0.75 M NaCl | 0.75 M sucrose | 0.75 M glucose | 0.75 M urea |
|---|---|---|---|---|---|---|
| Intake (g): | | | | | | |
| VAGX | 0.41±0.13 | 0.95±0.43 | 1.12±0.45 | 1.07±0.57 | 0.24±0.17 | 0.63±0.29 |
| SHAM | 0.64±0.19 | 1.41±0.30* | 1.90±0.22** | 1.65±0.29** | 0.93±0.27 | 0.73±0.24 |
| Latency (min): | | | | | | |
| VAGX | 7.0±5.8 | 7.5±5.7 | 3.3±2.4 | 6.3±5.9 | 21.8±5.8 | 13.9±6.7 |
| SHAM | 10.5±4.4 | 2.4±1.1 | 1.6±0.8* | 3.3±1.9** | 5.0±2.8 | 5.6±3.5 |

* $p < 0.05$, ** $p < 0.01$ for comparison with vehicle infusion condition.

the subsequent recovery period of 4–7 days, the rats received palatable foods in addition to their regular diet. Each day, the cannula of each subject was injected with heparinized physiological saline to maintain patency.

Each rat was tested with a single infusion of buffered Ringer's solution (vehicle) or 0.38 M NaCl, 0.75 M NaCl, 0.75 M sucrose, 0.75 M D-glucose, or 0.75 M urea dissolved in the vehicle. The order of administration of these substances was different for each subject.

Only a single injection was given each day, during the latter half of the light-dark cycle. Infusions (0.5% b.wt) were done in the home cage at a rate of 0.3 ml/min. Immediately after the completion of an infusion, a water bottle was attached to the cage and both latency to drink and 0.5-h water intake measured. After the conclusion of the experiment, all rats were electrophysiologically verified to have sustained extensive vagal denervation (vagotomy group) or to exhibit no evidence of vagal damage (sham-vagotomy group). In addition, each cannula was verified as functional. The details of these verification methods are given elsewhere[7]. Statistical analysis was done with the Wilcoxon matched-pairs signed-ranks test. On the basis of previous research[8,9], the hypertonic saline and sucrose solutions were expected effectively to elicit drinking, urea was expected perhaps to produce a slight dipsogenic effect, due to its action on brain osmosensitive cells[10], whereas glucose was not expected to exert dipsogenic properties in comparison to infusion of vehicle alone. The p-values for these comparisons were based upon directional 1-tail tests, with p-values of less than 0.05 considered to be significant.

*Results.* Prior to surgery, the mean (±SEM) body weight of the vagotomy group (N=5) was 268±20 g and at the time of testing it was 347±20 g; the mean body weights of the sham-vagotomy group (N=8) were 242±15 g and 331±13 g, respectively. The sham-operated rats drank significantly more water following hepatic-portal infusion of 0.38 M NaCl ($p < 0.04$), 0.75 M NaCl ($p < 0.005$), and 0.75 M sucrose ($p < 0.005$) than after vehicle alone. The latency to drink for the sham-operated rats was significantly shorter only following infusion of 0.75 M NaCl ($p < 0.04$) and 0.75 M sucrose ($p < 0.005$) relative to control infusion. In the vagotomy group, none of the experimental infusions significantly increased intake or decreased latency in comparison to infusion of the vehicle alone. The water intake and latency data are presented in the table.

*Discussion.* In the present experiment, i.v. infusion of either hypertonic saline or sucrose in sham-operated rats significantly increased water consumption relative to vehicle infusion, whereas infusion of equiosmotic glucose or urea did not. Similarly, infusion of either 0.75 M NaCl and 0.75 M sucrose in sham-operated rats significantly decreased the latency to drink in comparison with vehicle infusion, but 0.38 M NaCl, 0.75 M urea, and 0.75 M glucose did not. Following abdominal vagotomy, hypertonic saline and sucrose infusions induced only non-significant increases in water intake relative to vehicle infusion. Furthermore, vagotomized rats exhibited decreased latencies only following infusion of 0.75 M NaCl and 0.75 M sucrose, but in neither instance did the difference reach statistical significance, being relatively minor for sucrose infusion. Latency would be expected to be a particularly sensitive measure of the loss of function of a visceral drinking mechanism because it reflects the initial responsiveness to a regulatory challenge. The existence of brain osmosensitive cells important in the regulation of drinking[11,12], which presumably continue to function after vagal denervation, would ensure eventual, although perhaps attenuated, drinking after osmotic challenge. Abdominal vagotomy produced a comparable attenuation of drinking induced by equiosmotic infusions of hypertonic sucrose and saline, suggesting that the observed deficit is not merely due to the loss of a sodium-sensitive drinking mechanism. These results may indicate that there is a vagally mediated drinking system that is sensitive to osmotic fluctuations, not just to changes in plasma sodium concentration. Alternatively, however, the impairment in osmotically-induced drinking observed after abdominal vagotomy may result indirectly from any of the numerous behavioral and physiological changes produced by vagal denervation, rather than as a direct consequence of loss of vagal afferent input to the brain. Furthermore, the results of the present experiment do not provide any information concerning the location of vagally mediated osmosensitive cells. Insofar as previous research has located both osmo- and sodium-sensitive cells in the hepatic-portal circulation[3,4], comparison of the effects of abdominal vagotomy on drinking induced by hepatic-portal vs systemic infusions of osmotic loads could be expected to provide valuable information concerning the location of the hypothetical vagally mediated osmosensitive cells modulating drinking behavior.


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