and 2572 μm^3 after 2 weeks at 4 °C). Surviving cells enlarged after 3 weeks at 4 °C (3473 μm^3), but here the situation was still more complicated because of K^+ losses from cells damaged and dead at this time. Single cell data are necessary to clarify these points.

As the LC3 cells spend the majority of their life at 4 °C, changes in the properties of membrane lipids and fluidity may influence the activity of Na^+K^+-ATPase and K^+ transport in the cold. In view of the importance of K^+ for protein synthesis and mitosis[1,2,9-11], the maintenance of an adequate K^+ concentration may play an important role in the quick regrowth of the LC3 cells rewarmed to 36 °C[2].

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The effect of lidocaine on the secretion induced by cholera toxin in the cat small intestine[1]

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Summary. The intraluminal administration of lidocaine, a local anaesthetic agent, inhibits the net loss of fluid into the intestinal lumen produced by cholera toxin in the cat. It is suggested that the activation of a nervous reflex is involved in the pathogenesis of cholera.

The profuse fluid loss in cholera is, according to current concepts, the result of a direct cellular action of the cholera toxin on the intestinal epithelium[3]. Cyclic AMP is believed to be involved in this process since the concentration of this compound in the enterocytes and in most other cells is increased after cholera toxin exposure[4,5]. It has been reported that the intestinal contents in cholera contain vasoactive intestinal polypeptide (VIP) at high concentrations[6]. Since VIP has been demonstrated to be localized only in nervous tissue in the gut[7], it was considered of interest to investigate to what extent a cholera induced secretion could be decreased by a local anaesthetic agent, indicating a possible nervous reflex involvement in the pathogenesis of cholera.

Methods. The experiments were performed on cats deprived of food for 24 h with free access to water. The animals were anaesthetized with chloralose (50 mg/kg b.wt). Venous outflow from a denervated segment of the jejunum weighing 10–15 g was recorded by an optical drop recorder unit operating an ordinate writer. Arterial blood pressure was recorded from the left femoral artery by a pressure transducer. Net intestinal transport of fluid was measured continuously with the method described in detail by Jodal et al.[8]. This technique implied the perfusion of the intestinal lumen with an isotonic electrolyte solution at a constant rate of 1 ml/min in a recirculating system. Changes in net fluid transport were registered by a volume transducer connected to the recirculating system via a T-tube. All recordings were made on a Grass polygraph.

The solution used to perfuse the lumen of the small intestine contained (mmole/l): NaCl 122; KCl 3.5; $NaHCO_3$ 25; KH_2PO_4 1.2; $MgCl_2 \cdot 6\,H_2O$ 1.2; $CaCl_2$ 2.5; mannitol 30. The osmolarity of the solution was 310–315 mOsm/kg H_2O. Lidocaine (Xylocain®; generously supplied by Astra AB, Södertälje, Sweden) was added in amounts of 1 or 2 g/l electrolyte solution. The lidocaine solution was perfused through the intestinal segment for 90 min.

About 400 mg of a crude cholera toxin (kindly supplied by Dr Jan Holmgren, Department of Medical Microbiology, University of Göteborg) was dissolved in 5–10 ml physiological saline and exposed to the intestinal segment for 30 min. The toxin was washed away with 50–100 ml warm saline.

Results. The results of 6 technically successful cat experiments are summarized in the table. It is evident that exposing small intestinal segments to cholera toxin induced a net secretion of fluid into the intestinal lumen. This occurred within 60–150 min. Changing the intestinal perfusion solution to one containing lidocaine inhibited net fluid loss into the lumen in all experiments. In 3 experiments it even produced a net fluid absorption from the lumen. It was possible to follow the net fluid transport after lidocaine exposure in 5 experiments. In all but one a net fluid loss into the lumen was again recorded within 1 h after terminating the lidocaine perfusion.

In control experiments it was shown that lidocaine itself in the concentrations used in this study did not affect net water uptake from a normal intestinal segment.

Arterial blood pressure, intestinal blood flow and intestinal fluid transport during control conditions and after cholera toxin exposure: before, during and after perfusing the intestinal segment with a lidocaine solution. – denotes a net fluid transport from tissue to lumen. Mean ± SE

	Control	Cholera		
		Before lidocaine	During lidocaine	After lidocaine
Arterial blood pressure mm Hg	113 ± 7	98 ± 8	86 ± 7	83 ± 5
Intestinal blood flow ml × min^{-1} × 100 g^{-1}	29 ± 5	34 ± 6	40 ± 5	46 ± 5
Intestinal fluid transport µl × min^{-1} × 100 cm^2 serosal $surface^{-1}$	242 ± 62	− 149 ± 40	61 ± 41	− 49 ± 32
Number of observations	6	6	6	5

Discussion. The results presented in the table strongly suggest that exposing a choleraic intestinal segment to lidocaine inhibits the net fluid loss and in some instances even induces a water absorption. This observation indicates a nervous involvement in the pathogenesis of cholera although one cannot entirely exclude the possibility that lidocaine in some way affects the direct cellular action of the cholera toxin. However, no experiments to test such a cellular effect seem to have been performed.

A nervous involvement in the pathogenesis of cholera was, as pointed out in the introduction, suggested by the finding of large amounts of VIP in the choleraic secretion. VIP has been proposed to be a neurotransmitter in the small intestine[9] involved in the elicitation of a nervous reflex producing a functional hyperemia in the gut[10]. This reflex hyperemia could be abolished by lidocaine or 5-HT blocking agents. In this context it is interesting to note that chlorpromazine, a serotonin receptor blocking agent, markedly inhibits cholera secretion in the mouse[11].

It is thus proposed that the activation of an intramural nervous reflex may be one mechanism of importance in explaining the fluid loss from the small intestine in cholera. Serotonin and VIP may possibly be involved in this nervous reflex.

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The excretion of urea by the larvae of *Spodoptera mauritia* Boisd. (Noctuidae: Lepidoptera) during development

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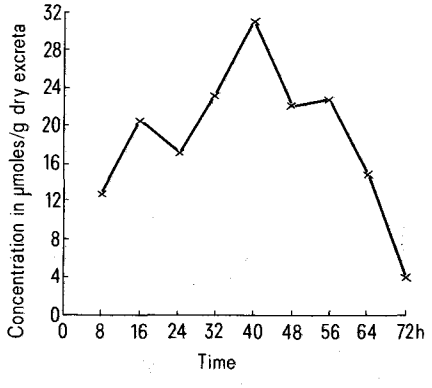
Summary. The concentration of urea in the excreta of the 6th instar larvae of *Spodoptera mauritia* varies from 4.017 ± 0.541 to 31.052 ± 1.193 µmoles/g dry excreta (mean ± SE). The observation confirms that urea excreted is of metabolic origin.

Though the presence of urea in the excreta of insects is well documented[1,2], no information is available on the nature of urea in the excreta during the development of insects. In this communication we show the occurrence and changes in the concentration of urea in the excreta of the last larval instar of *Spodoptera mauritia* during its development.

The 6th instar larvae were separated immediately after moulting from the colony reared in the laboratory. The larvae, fed on the grass, *Ischaemum asistatum*, were used for the experiments. The excretory pellets collected at regular intervals were dried to a constant weight at 60 °C and were analysed for urea[3].

The concentration of urea determined at regular intervals of larval development is illustrated in the figure. The concentration varies from 4.017 ± 0.541 to 31.052 ± 1.193 µmoles/g dry excreta (mean ± SE). Analysis of the food reveals that it contains urea only in trace amounts. The urea concentration in the excreta shows an initial increase at 16 h; after moulting, however, the level declines at 24 h. Later it increases rapidly and reaches the peak at 40 h. Thereafter the concentration falls sharply till 72 h, interrupted by a gap between 48 and 56 h. No excretory pellets were voided after 72 h.



Concentration of urea in the excreta of the 6th instar larvae of *S. mauritia* during development.

It has been demonstrated that the synthesis of urea is enhanced by the increased substrate availability[4,5]. The increase in the production and excretion of urea in the early stages of the larvae may be due to the intensive metabolism of nitrogenous compounds during this period when active feeding and rapid growth take place. The present observations also reveal that the concentration of urea in the excreta falls gradually to a low level during the period when the feeding activity and growth rate are low. Similar variations in the concentration of urea are reported for *Dysdercus fasciatus*[6]. Since the food contains only detectable amounts of urea, it is presumed that the large quantity of urea found in the excreta of this insect is of metabolic origin.

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