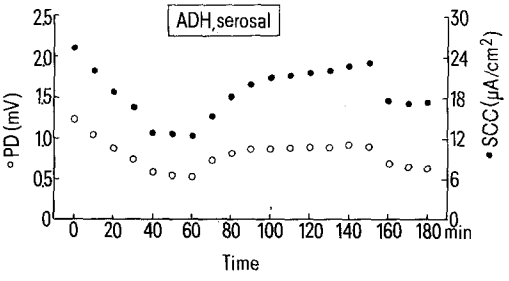
be localized on the inner or serosal rather than the outer or mucosal membrane of several epithelia[8], it is feasible to presume that ADH would be more accessible to adenyl cyclase when it is placed in the serosal bathing medium. Hence, the reason for stimulation of the electrical characteristics when ADH was placed in the serosal bathing medium and not in the mucosal bathing medium. This increased enzyme activity would lead to increased intracellular concentrations of cyclic AMP which would be the final mediator for increasing Na^+ permeability of the mucosal membrane. The increased intracellular Na^+ concentrations would then stimulate greater Na^+ pump activity which would be reflected in an increase in the unidirectional J_{MS} of Na^+ and SCC, which is shown in the data (figure and table).

ADH stimulates only the J_{MS} of Na^+, therefore, it is possible that it can effect its stimulation of acting directly on the Na^+ pump located in the serosal membrane. If ADH directly stimulates a Na^+ pump located in the serosal membrane, this could lower intracellular Na^+ concentration and therefore, indirectly could increase the permeability of the mucosal membrane to Na^+ as suggested by Biber[9] and Lewis and Diamond[10]. The data however, does not allow me to distinguish between the alternatives of ADH acting through adenyl cyclase or by its direct action on a Na^+ pump.



A typical experiment showing the stimulation of transmural potential difference and short-circuit current by the addition of ADH to the serosal bathing medium.

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Do free-living songbirds habituate to species-specific alarm-calls?[1]

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Summary. Free-living birds adapt to constantly repeated species-specific alarm-calls, despite the variable environmental situation.

When chaffinches (*Fringilla coelebs* L.) are played back copies of their 'pink' alarm calls in the wild, they respond to these with the 'pink' alarm call, males sometimes with 'huid' or 'rülsch' calls mixed with the 'pink' calls. Occasionally single aerial enemy or flight calls are intermingled with these[3,4]. The birds approach the source of the sound, changing their position frequently, erecting the head feathers and jerking their tails. It has also been observed that some birds answer but remain perfectly still; others show a high degree of locomotory activity but do not call[5]. These and other behaviour patterns associated with alarm can also be released in the laboratory. If chaffinches are played back the 'pink' alarm call repeatedly over a long period of time under almost constant laboratory conditions, however, after some time an almost complete reduction of response can be recorded[6]. This behaviour is based on a learning process termed 'long-term habituation'[7-9].

These findings raise the question whether such habituation processes are also possible under the unfore seen and continuous changes of the natural environment. In order to test this, 15 breeding chaffinch pairs were played the 'pink' alarm call every morning in the center of their territories. The acoustical response of the birds was recorded on a tape recorder, the non-acoustical responses for 5 min following stimulus presentation by written notes. If the responsiveness of a pair, measured by the number of alarm calls per day, is compared from day to day, it emerges that the animal respond less and less strongly to the stimulus on succeeding days (figures 1 and 2).

This reduction of response, however, is not continual but can be reversed in correlation to occurrences taking place during the experimental period. The first of these are associated with breeding biology: nest building, the start of

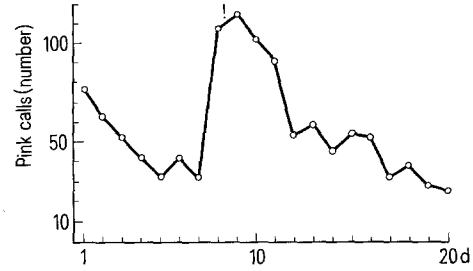

Fig. 1. Example for habituation to species-specific alarm calls in the chaffinch *Fringilla coelebs* in the wild: the 'pink' alarm call model which is presented once daily results in a gradual reduction of response. Ordinate: number of alarm calls given by a pair in the 5 min following stimulation. Abscissa: sequential days (d). The rapid increase in response on days 8 and 9 is synchronous with the hatching of the young (!).

breeding and the hatching of the young. In almost all cases, these appeared to result in increasing the responses again, which had been reduced to a lower level previously (figure 1). These phenomena can be explained by a change in response readiness which is to be considered as a result of changes in endogenous and exogenous stimuli[10]. From an ecological point of view, this is a highly adaptive mechanism: the animals increase their alarm behaviour, or readiness to give alarm calls, when critical stages in reproduction are reached, thus improving the survival chances of the young.

Another group of occurrences which can be correlated with a rise of the alarm response frequency is the appearance of natural enemies (figure 2). This can also be explained by an increase in response readiness and is a highly adaptive mechanism from an ecological point of view.

Following on from such an increase in response as a result of breeding biology or the appearance of an enemy, comes a rapid decrease and further habituation during the next few days. This raises the question as to whether the learning process continues from the level it had previously reached, or whether the learning process must start from the beginning again, e.g. the stored information has been eradicated by the newly received stimuli. The results indicate that the previously learned niveau is returned to after the appearance of an enemy. The question must remain open, however, in the case of changes observed as a result of breeding biology: is the disturbance one of long duration, and thus delays the onset of the old niveau, or does it make the stored information completely inaccessible so that the learning process must begin again?

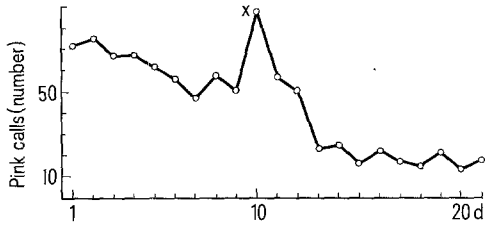

Fig. 2. Same as figure 1. The increase in response on day 10 is correlated with the previous appearance of a squirrel *(Sciurus vulgaris)* which remained near the nesting tree of the birds (×).

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Hageman factor activation and tight junction disruption in mice challenged with attenuated endotoxin[1]

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Summary. Endotoxin treated with chromium chloride is less toxic to mice than the parent molecule, but can disrupt intestinal permeability barriers and has an enhanced ability to activate Hageman factor.

Treatment of endotoxin with chromium chloride ($CrCl_3$) reduces its toxicity when injected i.p. into mice[2,3]. These endotoxin-chromium complexes are of interest because certain useful biological effects of the toxin may remain intact after treatment and because comparison of changes in host responses to treated and untreated endotoxin preparations may illuminate mechanisms responsible for pathologic effects of the toxin.

In this report we compared Hageman factor (HF) activation in mice challenged with untreated and $CrCl_3$-treated endotoxin. HF is a common initiator for kinin generation, intrinsic clotting, and plasma fibrinolytic systems[4]. Activation of HF apparently occurs in humans undergoing gram-negative shock[5] or following injection of volunteers with purified endotoxin[6].

We have recently associated disruption of tight junctional barriers between adjacent epithelial cells of the intestine with endotoxin-induced inflammation[7]. This phenomenon could lead to a self-sustaining endotoxemia[8] if host defenses against endotoxin from the intestine are impaired. We reported evidence for this event in mice made neutropenic by radiation prior to challenge with endotoxin attenuated with ferric chloride[9]. For these reasons, the effects of $CrCl_3$-treated endotoxin on tight junctional structure and survival of irradiated mice treated with oral antibiotics will be determined.

Male B6CBF1 mice were inoculated i.p. with a lethal dose of *Salmonella typhosa* lipopolysaccharide W (Difco). In some experiments, mice were decontaminated with oral bacitracin-neomycin mixtures as previously described[9]. Unirradiated mice were given 0.8 mg of the endotoxin, but mice irradiated 7 days previously with 1000 rads ^{60}Co (40 rad/min) were given only 0.3 mg of the toxin. Endotoxin was treated with chromium by adding 5 µl stock $CrCl_3$ (0.3 g/ml) to 1 ml of endotoxin (10 mg/ml pyrogen-free saline)[2]. This mixture was allowed to stand at 25 °C for 30 min and then was diluted 10 times with physiologic saline.

Blood for analysis was collected by retroorbital bleeding into plastic tubes containing 3% sodium citrate. Bleeding was initiated by insertion and immediate withdrawal of glass capillary tubes containing the anticoagulant. The citrated plasma prepared from this blood was assayed for HF activity using factor-deficient serum in a modification of the procedure of Hardisty and Macpherson[10]. Segments of ileum were removed from mice killed by cervical dislocation and fixed by immersion in cold 2.5% glutaraldehyde buffered with 0.1 M sodium cacodylate (pH 7.2). Samples