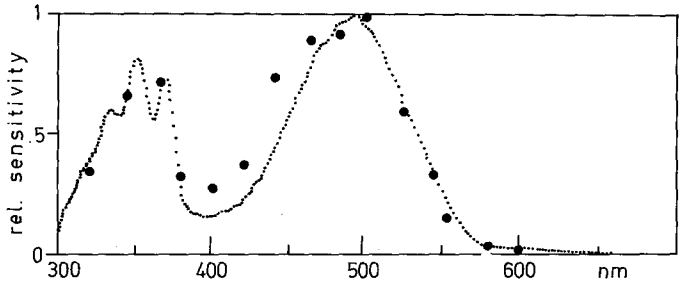

Fig. 1. Relative spectral sensitivity measured with the ERG of the fly *Calliphora erythrocephala* chalky with FIS (fine dots). Larger dots: measurement of the spectral sensitivity of the retinula cells R1 – R6 (from [5])

well-known one at 493 nm and 3 UV maxima at 369, 350, and 332 nm.

The UV maxima are stable in position under all the conditions we have so far tested the highest peak being 350 nm, while the others are significantly lower. It can not yet be said if there is a fourth UV peak below 332 nm. These results prove that the UV sensitivity cannot be caused by a rhodopsin of 350 nm maximum absorption. Therefore, they support the antenna pigment hypothesis. Refering to [6] and [7] we are led to the conclusion that the UV sensitivity is caused by a polyene. In this case it is possible to take the longest wavelength of the peaks as an indicator of the number of conjugated bonds of the form C–C=C, directing us to n around 5. The spacing of the peaks with 19 and 18 nm is also strong support for this view. Yet one has to be careful, because the solvent of a polyene, the interaction with other molecules, and the steric configuration modify the spectra. Therefore we must perform additional measurements especially dealing with the range from 200 to 350 nm, to look for further fine structures before a final conclusion will be made.

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Homing by Path Integration in a Mammal

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Homing by integration of (idiothetic and visual) azimuth information over the pathway taken by the animal was hitherto known only in a number of invertebrate species (see [1] for a review of the capability and [2, 3] for an analysis of the mechanism). As to vertebrates, well worked out general theories and specific hypotheses about inertial navigation stand in contrast to scant experimental evidence, by far insufficient to support them (for reviews cf. [4] and [5]). This report will show that gerbils (*Meriones unguiculatus*) do, in fact, home by integrating azimuthal information from rotatory accelerometers over an idiothetic variable which is proportional to the progress of the animal along its path.

We make use of the strong motivation of the female desert mouse to carry its sucklings back home if they are displaced, on a circular arena ($\varnothing = 130$ cm, i.e., about ten times the animal's length), to a shallow cup at various angles and distances from their nest. The female soon ventures out from the nest in search of them and, upon finding one, snaps it up at a special fold at the young's midriff. The mouse returns at once, its helpfully stiffening charge athwart its snout, on a rather straight course *to the starting point of the excursion.* It is able to do this in complete darkness, with the cup taken to another place before every excursion, after arbitrarily varying detours of the mouse on its way out, and with the nest shifted to arbitrary places after the animal has left it. Even if the new location of the nest is close to the original site, the returning female, heedless of the noise and smell of the nearby litter, frantically scurries about the place where the nest had been. Under our experimental conditions, which do not quite bring forth the full precision of the animal's performance, the runs home scatter about their mean directions with standard deviations of 10–20° yielding "home components" [6] of 0.94 to 0.98. All the following experiments are done under the above conditions, with the round nest entrance fitted to any one of 48 identically formed holes around the circumference of the arena.

(A) If, with the cup at the center, the arena is rotated quickly to various degrees and directions while the female stays at the cup, the mouse returns nearly to the place in (Newtonian) space where the nest had been before the rotation. If only the cup is briskly rotated relative to the arena while the female is sitting on it, homing is about as good as before. If the cup with the mouse on it is smoothly accelerated to no more than $0.24°/s^2$ and then decelerated just as smoothly to a full stop after 23.5 s and a total turn of 37°, however, the nest is missed by very precisely this amount in the direction of rotation.

(B) If the cup with the mouse on it is shifted *sideways,* the animal runs on a parallel to the line connecting the nest and the point where the mouse had been before the shift, thus missing home by the amount of the sideways displacement. This happens even if care is taken to displace the mouse in a normal snoutward direction and within the temporal range of the animal's normal movements. If the *entire arena* is translatorily displaced relative to the room this way, the mouse homes relative to the arena as normal, irrespective of the direction of linear acceleration and irrespective of whether it is sitting or running at the time.

The clearcut conclusions are that the gerbil uses (a) a message about its rotatory acceleration relative to Newtonian space, twice temporally integrated, as azimuthal infor-

mation, yet (b) an idiothetic message about the translatory displacement of the animal *relative to its substrate* as the path integration variable. A likely candidate for the former is, of course, the afference of the semicircular canals caused by a cupula deflection, which in the frequency range of the animal's motions integrates acceleration mechanically once over time, but fails to do so at very low frequencies, such as those applied in the last-mentioned experiment under (A). From the results under (B) it follows that linear accelerometers such as the otoliths are excluded as a source; complications which would arise in conjunction with their static (postural) function are thus obviated. It remains to be studied whether proprioceptive measurements or "efference copies" of the number and amplitude of the steps, or the energy spent to produce them, provide the necessary translatory information. The principle or also the particular mechanism of homing thus revealed may–as (unpublished) pilot experiments on geese appear to corroborate—turn out to be an important general feature of vertebrate navigation.

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Myocardial Uptake of Thallous Ion by Facilitated Diffusion

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In the group of heavy metals, thallium shows some interesting pecularities. In particular, the process of poisoning on the microscopical level is not yet understood, with the exception of a sulfhydryl blocking which has been shown in vitro. On the macroscopical level, thallium causes heavy neurogenic symptoms of a toxic neuropathy as an epithelial and nerval poison. The element exists as a monovalent and a trivalent cation in vitro; in vivo, only the monovalent form is found. Antidotes such as BAL and other chelate agents are of no use in the therapy.

In medical diagnostics ^{201}thallium has been used for about ten years as Tl^+Cl^- to perform myocardial perfusion scintigraphy under stress and rest conditions to detect ischemia or infarction in the heart muscle. This was first suggested by Kawana et al. [1]. The thallous ion is taken up by myocardial tissue in a potassium-like manner. Gehring and Hammond suggested the activation of the sodium- and potassium-activated adenosine-triphosphatase enzyme (Na-K-ATPase) by both potassium and thallium for the incorporation of these elements [2]. We find this assumption should be revised. The experiments of Ku and collaborators [3] showed that Tl^+ inhibits and Li^+ stimulates cardiac Na-K-ATPase activity in vitro.

We suggest that the specific behavior of Tl^+ can cause multiple bondings in the Na-K-ATPase due, for instance, to low-lying empty d-orbitals. This causes an inferior desorption of thallous ion from the transport enzyme. Electrosorption is not observed with potassium, whereas thallium is strongly bound. For this reason active transport of thallous ion by the Na-K-ATPase seems to be unlikely. To get physiological support for our electrochemical and quantum chemical theories, we performed an experimental series in patients treated with digoxin. Inhibition of cardiac Na-K-ATPase is the mechanism of the positive inotropic action of digitalis in heart [4–6]. Similar experiments were performed using drugs such as digoxin, strophantin, procain, and valinomycin in dogs [7] and in *Streptococcus lactis* 7962 [8, 9]. Contradicting results arose from these animal experiments.

The 15 patients examined were separated into two groups, 7 digitalized with digoxin and 8 non-digitalized. The digoxin blood level was determined shortly before i.v. injection of thallium. The average level was 2.1 µg/l (normal range 0.8–1.8 µg/l RIA). Data were stored on-line in a computer system. In the anterior view the whole heart was taken as region of interest. A mean countrate of addresses was calculated. The background uptake was determined by setting a region of interest in the mediastinal and lung area; here again a mean countrate was calculated by computer. The evaluation of mean countrates of the regions of heart and background was done by the plotsystem program "ROI Ratio Dig/Nondig". The resulting region of interest ratios were found to be similar in both digitalized and non-digitalized patient groups following a one-term linear function.

As shown by our experiments, digoxin does not inhibit uptake of thallium into myocardial tissue. Thus, the Na-K-ATPase cannot be connected with the uptake of thallous ion as the main agent. We suggest this depends on facilitated diffusion of thallous ion through the cell membrane as a monovalent cation. This hypothesis is supported by the finding [10] that there is a dependence of myocardial uptake of Tl^+ from oxygen. Therefore, thallium must be incorporated by an active process that is not active transport.

The poisoning of thallium on microscopical level seems to depend on the inhibition of Na-K-ATPase and facilitated diffusion of thallous ion into the cell. Further experiments and quantum chemical calculations are to be performed.

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