regimen (4% of their b.wt food daily) and trained to respond for food by pushing a panel switch with their snouts in a modified metabolism cage serving as a Skinner box. Each response was rewarded with 10 g of a commercial granular food delivered by a solenoid-driven feeder. The session was ended after the pig had completed 80 responses or after 30 min. Sessions were run daily, 5 days a week. After stabilization in this procedure for several weeks, pigs were given in their pen unrestricted access to the same food as that used as a reward in conditioning experiments. They were then submitted to 15 min CRF daily sessions until they did not eat any of the food obtained by the few responses emitted during the session. After this criterion had been fulfilled for at least 2 consecutive sessions, pigs were injected i.m. with 1 mg/kg diazepam, in commercially available vials (Valium Roche) $^1/_2$ h before the beginning of the session. 3 pigs were given a further 'disinhibition' session during which 3 different types of stimulus, a high pitched tone, the sound of a buzzer and a flashing light, spaced 1 min apart, were presented in a random order. The whole sequence was presented 3 times at 2 min intervals during the course of the 15 min CRF session. Number of panel presses and the amount of food eaten were recorded during each session.

Results. Under the restricted regimen, pigs emitted 80 responses in 13–15 min and ate all the delivered food. After 2 or 3 days under ad libitum feeding, they typically indulged in a few panel presses during the CRF session and did not pay attention to the delivered food. Such pigs were then eating in their pen about 8% of their b.wt food and gaining 1–1.5 kg/day.

The table presents the number of responses and the amount of food eaten in the last control session preceding the drug session, the drug session and the disinhibition session. Diazepam-treated pigs emitted significantly more responses ($p = 0.031$ by a one-tailed Walsh test[8]) and ate most of the food rewards obtained. There was no evidence of change in either instrumental or consummatory behaviour during the disinhibition session with respect to the control session.

Discussion. Morgan[4] recently pointed out the methodological difficulties encountered in experiments on satiation, contending that in most studies there was insufficient proof that satiation was effectively reached, i.e. that subjects did not eat in the experimental situation. In the present study, great care was taken to ensure that the pigs met the 'no eating' criterion of satiation. Panel pressing continued to some extent but was very different from the organized behavioural pattern governing the instrumental responding of normal subjects. Ad libitum fed pigs tended to act in an agitated way, grunting, defecating and attempting to escape the experimental situation. The same behaviour was seen throughout the disinhibition test whatever the stimulus presented. Diazepam-induced responding and eating were limited mainly to the first 5–10 min of the session, subjects tending to display agitated behaviour during the last part of the session. This was certainly not due to a waning of the effect of diazepam since in cats, varying the interval between drug administration and test from 15 min to 12 h, did not modify the eating-stimulant effect of another benzodiazepine, oxazepam[2].

Similarities between satiation and extinction have been suggested mainly on the basis of the frustration-like effects produced by exposing sated subjects to the experimental situation, the dissociation between consummatory and instrumental responses and the possibility of disinhibition of satiation by external stimuli[4,7]. The present experiment demonstrates that a dose of diazepam which does not affect fully extinguished responding of pigs[6] is still able to increase resistance to satiation, in spite of the fact that the animals were not eating in the experimental situation. Moreover the treatment did not suppress the agitated behaviour which reappeared a few minutes after the beginning of the session and did not prevent the earlier disappearance of consummatory responses as compared with instrumental responses. Finally, presentation of novel stimuli in a disinhibition test, did not cause either lever pressing or eating to be resumed.

These results therefore cast some doubt on the analogy drawn between extinction and satiation at least in pigs and show that the effects of benzodiazepines cannot be dealt with in terms of disinhibition. Benzodiazepines appear to have enhancing effects both on instrumental and on consummatory behaviour, which can be interpreted either as the result of a direct action on hunger and satiety mechanisms[2,3], or as the result of a breakdown in a decision process confronting satiety interoceptive feedbacks with exteroceptive cues[9].

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Filamentous bodies in human glomerulonephritis

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Summary. Filamentous bodies have been identified in the glomerular cells of human kidney biopsies. These structures have a close morphological resemblance to ciliary rootlets, although the cells of the glomerular tuft only occasionally bear cilia. Their significance could be, as for cilia, of a cellular disdifferentiation of a pathological cellular proliferation.

In recent years, there have been occasional reports of cytoplasmic filamentous structures in non-ciliated cells from rabbit, rat and man, which were associated by most authors with ciliary rootlets. It is our purpose to report the existence of similar structures in the glomerular cells of human kidney.

Material and methods. Fragments of kidney biopsies from 21 patients with different types of glomerulonephritis were fixed in 4% glutaraldehyde in cacodilate buffer, postfixed in 1% osmium tetroxide in the same buffer, dehydrated in graded ethanol and included in Epon. Ultrathin sections cut on a LKB-Ultratome III

were stained with uranyl acetate and lead citrate and observed in a Philips EM 300.

Results. Filamentous bodies were observed in the glomerular cells of 13 biopsies out of the 21 investigated. They consisted of bundles of 2 to 6 filaments 8 nm wide, varying accordingly in thickness from 30 nm to 180 nm. They had a transverse periodicity of dense bands of 90 nm and sometimes a subperiodicity or a branching pattern. They were observed in the vicinity of the Golgi complex or of the centriole, sometimes arising from the latter. Filamentous bodies were found in the endothelial, mesangial and epithelial cells of the glomerular tuft, though more often in the first. They were not observed in Bowman's capsule epithelium.

Discussion. The structures reported here have been described, other than ciliated epithelia, in spermatocytes[1], spermatocytic seminoma[2], human tubular epithelium of normal kidney[3], rabbit lymphatic endothelial cells of the lung[4], rabbit and rat kidney glomerulus and non-specified human glomerular cells from diseased kidney[5]. In human kidney, cilia have been reported in normal tubular epithelium[6] and in Bowman's capsule cells[7], and occasionally in mesangial, endothelial and epithelial cells of the glomerular tuft[8]. Whether these structures are a constant feature or a occasional finding is not clear, as random ultrathin sectioning is not a suitable method to derive such information. Cilia have also been reported to be a constant feature of glomerular cells in 13 to 16 week human metanephros[9].

Although the filamentous structures we describe have a close morphological resemblance with ciliary rootlets, they were not found by us in Bowman's capsule ciliated cells. The cellular proliferation present in glomerulonephritis could be associated with filamentous bodies induction which could have the same significance as ciliogenesis in spindle-inhibited cells[10]. Both situations could correspond to a disdifferentiation of cells normally ciliated in the embryo.

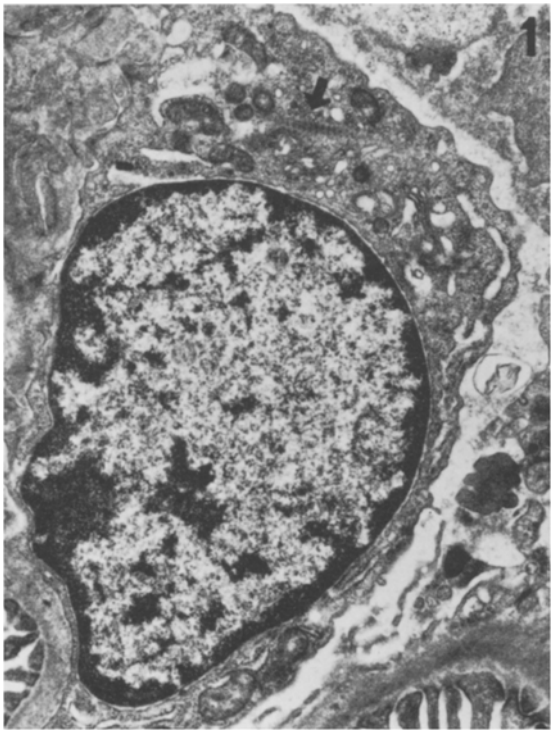

Fig. 1. Endothelial glomerular cell with a filamentous body lying next to the Golgi complex. ×10,500.

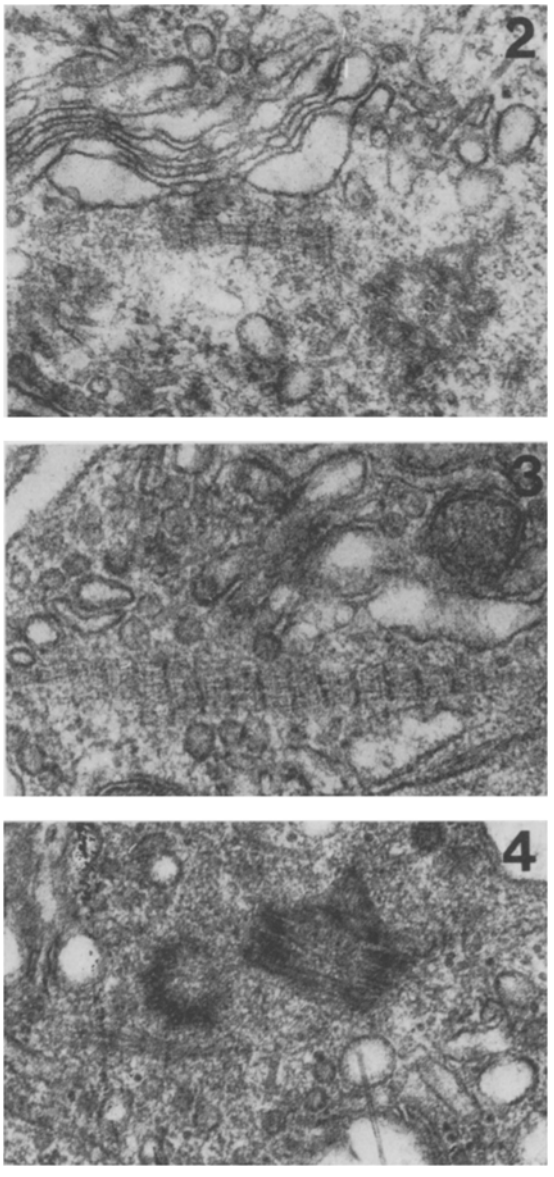

Fig. 2–4. Filamentous bodies showing their subperiodicity and connections with the Golgi complex and centriole. ×55,000.

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