The relationship between log background luminance and the luminance of the spot necessary to meet a criterion response was examined for the (transient first 80 msec) of the response of X and Y cells. The criterion response was the spike rate produced by a stimulus luminance 0.8 log units above threshold at the 0.28 log cd/m² AL. Equal-response curves were then generated by solving the intensity-response functions for this criterion spike rate. Regression lines were then fitted to the computed equal-response data. Linear equations provided a satisfactory fit ($p < 0.001$) to data for both cell types. The figure shows the equal-response plots.

The change in retinal sensitivity over the observed range of adapting luminances was smaller than that predicted by Weber's Law ($\Delta I/I_a = K$): I_a refers to AL and ΔI the intensity difference between test target and AL necessary to produce a criterion response; K is a constant. It has been demonstrated previously for ganglion cells considered as a single group (for threshold responses), that the test spot luminance required to yield a constant response does not rise directly with I_a, but is proportional to I_a^n, where n is smaller than 1[15]. The relationship between ΔI and I_a is given in the figure for X cells and Y cells. The values for the slope of the relationship is 0.744 for Y cells and 0.743 for X cells. No statistical difference could be found between these values ($p > 0.15$).

The meaning of this function is that the intensity-response curve generated at a given AL is shifted along the background luminance continuum by the relation $I = f(I_a)^n$. Sakmann and Creutzfeldt[16] reported similar findings with the exponent equal to 0.7. This is confirmed here for both X and Y cells considered separately.

Increased resistance to satiation in diazepam-treated pigs

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Summary. Sated pigs, having a previous history of operant responding according to a CRF schedule, resumed responding when put back in the Skinner box after 1 mg/kg diazepam. This effect did not appear to be related to the disinhibition of an extinction process.

Benzodiazepines have been reported to increase resistance to satiation in cats or rats under conditions in which emotional factors are not present[2,3]. This effect has been attributed to an interference of the drug with hunger or satiety mechanisms. However, in an extensive review of resistance to satiation, Morgan[4] has recently suggested that satiation is highly related to extinction, so that the effects of benzodiazepines may be viewed as another example of drug-induced resistance to extinction[5]. Using pigs, we have shown that diazepam is effective in increasing responding only in the early stages of the extinction procedure, when there is still some residual responding, but is no longer effective when responding is fully suppressed[6]. In studies of benzodiazepine effects on satiation, satiation was operationally defined by giving the subjects free access to food during the course of the experiment[3] or, during a limited time, just before the drug test[2]. It was found, in both studies, that control animals ate in the experimental situation, i.e. that satiation was not effectively reached. If satiation is governed by the same active inhibitory process as that which controls extinction, benzodiazepines would be expected to have no effect on the behaviour of fully sated subjects which do not eat in the experimental situation. Moreover, satiation should be 'disinhibited' by appropriate external stimuli[7]. The present experiments were initiated to test this assumption using pigs as experimental subjects.

Methods. Apparatus and general training procedures have already been fully described[6]; 5 pigs, 3–5 months old and weighing 25–50 kg were first put on a restricted

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Number of responses and amount of food eaten in sated pigs submitted to a 15 min CRF session under different conditions

Subject	Control session		Drug session		Disinhibition session		
	Number of responses	Amount of food eaten	Number of responses	Amount of food eaten	Number of responses a	b	Amount of food eaten
109	2	0	21	77%	–	–	–
110	7	0	38	53%	–	–	–
82	5	0	115	75%	2	0	0
73	5	0	26	82%	3	0	0
79	0	0	80	54%	6	2	0

* Percent of food delivered. a: total number of responses emitted; b: responses emitted during the stimulus presentations.

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