F-test[7] showed that the slopes of the 2 regression lines were significantly different ($p < 0.05$).

A dose-effect relationship was sought by comparing the effects of 3 doses of isaxonine administered over 14 days after the lesion, as shown in the figure, B. Covariance analysis demonstrated that the slope of the dose-effect regression line was significantly different from a line parallel to the abscissa ($p < 0.01$).

Recovery of sensory and motor functions in rats was investigated on groups of 20 animals tested independently by 2 observers. Sensory recovery was appreciated by the reappearence of a behavioural response to electrical stimulation of the plantar surface of the digits on the lesioned side (2 shocks of 1 msec, interval 1 msec, 180 V). The motor response was tested by searching for the reapparance in the lesioned leg of a gripping reaction obtained by drawing the 4 legs backward on the table[8]. The figure, C and D, shows that recovery of sensory and motor responses had occurred in all animals of the control group by day 42. In the treated group, recovery of the sensory and motor responses were observed in all animals by days 21 and 28, respectively.

Results confirm in vivo the positive effect of isaxonine on nerve elongation, observed previously in vitro. They show significant enhancement of the rate of sciatic fibres regeneration associated with consistent increase in rate of sensory and motor recovery.

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Plasma lipid concentration and lipoprotein distribution in exercising and nonexercising rats fed a high sucrose diet

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Summary. Voluntary physical activity in rats fed a high sucrose diet reduces the plasma triglyceride level but has no major influence on the lipoprotein distribution.

There are several studies on the effect of physical activity on plasma lipid concentration[2-9], while little information seems to be available on the effect of physical activity on plasma lipoprotein distribution. In a group of very active men, Wood et al.[10] observed that the level of high density lipoprotein cholesterol was higher and the level of low density lipoprotein cholesterol lower than in a control group of relatively inactive men. These findings are significant in light of epidemiological studies suggesting that the level of high density lipoproteins is associated with reduced risk and the level of low density lipoproteins with enhanced risk of developing coronary heart disease[11-17]. More direct evidence for a causal effect of physical activity on the plasma lipoproteins was obtained by Lopez-S et al.[18], who showed that α-lipoproteins increased and β-lipoproteins decreased in men following 7 weeks of severe exercise.

We recently reported that the level of voluntary physical activity in rats is an inheritable phenomenon[19]. Surprisingly, in genetically active rats there was a pronounced age-related increase in the plasma cholesterol and triglyceride concentration; while no such increase was observed in genetically passive rats. However, nonexercising subgroups of both types of rats had a similar plasma lipid level as the corresponding exercising group. The rats were fed a stock diet and the lipoprotein distribution was not investigated.

We recently observed that a high sucrose diet increased the plasma lipid level and appreciably affected the lipoprotein distribution[20]. In the present study we have investigated whether physical activity might influence plasma lipid concentration and the lipoprotein distribution in genetically active rats fed this high sucrose diet.

Material and methods. Genetically active female Wistar rats

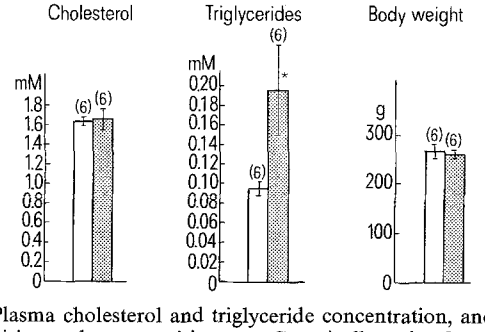

Fig. 1. Plasma cholesterol and triglyceride concentration, and b.wt of exercising and nonexercising rats. Genetically active female rats were divided into exercising (open columns) and nonexercising (striped columns) subgroups as described in text. Both groups were fed the same purified high sucrose diet for 3 months. Mean values ±SEM are shown with number of rats in each group. *$p < 0.05$ vs exercising rats (Wilcoxon's test for 2 samples[21]).

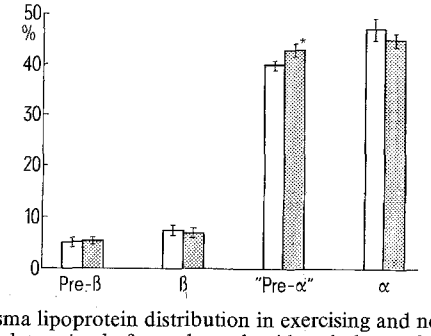

Fig. 2. Plasma lipoprotein distribution in exercising and nonexercising rats as determined after polyacrylamide gel-electrophoresis and densitometric scanning. Lipoprotein distribution is presented as percent of total weight of the transferred densitogram. Total weight of densitogram was 0.401±0.021 g and 0.394±0.026 g for the exercising and nonexercising group, respectively. Symbols are as in figure 1. *$p < 0.05$ vs exercising rats.

of a local strain were used in this study. Selection and maintenance of the rats were as previously described[19]. The rats were housed individually in a wheelrunning apparatus to which was attached a small living cage. Exercising rats had free access between the cage and the drum. The mean voluntary wheelrunning activity of this group by the end of the experiment was 15.0±0.6 km/24 h. In the nonexercising group, the admittance to the drum was closed with a metal plate. The rats were fed the purified high sucrose formula diet A, previously reported[20], and water ad libitum. At the start of the experiment, the exercising and nonexercising groups were selected so as to provide equal mean levels of running activity. Blood samples were obtained after 3 months for determination of plasma cholesterol and triglyceride concentration, and for carrying out lipoprotein electrophoresis. Blood sampling and the analytical methods were as previously described[19,20].

Results and discussion. The concentration of plasma cholesterol was remarkably similar in exercising and nonexercising groups (figure 1). In contrast to this, the mean level of plasma triglycerides was reduced by more than 50% in the exercising group as compared with the nonexercising group. B.wts were not different in the 2 groups.

The percentage of pre-β and β-lipoproteins were quite similar in exercising and nonexercising rats (figure 2). The 'pre-α fraction', which consists of several diffuse bands between the β- and α-bands, was nearly as large as the α-fraction. There was a slightly increased percentage of 'pre-α' lipoproteins in the nonexercising group as compared with the exercising group. α-lipoprotein percentages were not significantly different in the 2 groups.

These results suggest that the level of voluntary physical activity in rats does not greatly influence the plasma lipoprotein distribution. Our observation is in contrast to the finding of Lopez-S et al.[18], who reported that α-lipoproteins increased and β-lipoproteins decreased in response to physical activity in young men. This effect was, however, small. Moreover, maximal exercise was performed. The observation that physical activity did not influence the cholesterol level is in agreement with previous studies[5-8], as is also the reduction by physical activity of plasma triglycerides concentration[2-4,7,9]. It should, however, be pointed out that in a similar study, in which the rats were fed a stock diet, no effect of voluntary physical activity on the plasma triglyceride level was observed[19]. Thus, the triglyceride response to physical activity seems to depend upon the diet used. It would appear that diet is more important than physical activity, both for the regulation of plasma lipid concentration and lipoprotein distribution.

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Cholecystokinin-like peptides in avian brain and gut

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Summary. Extracts of turkey brain and jejunum contain a factor closely resembling the COOH-terminal octapeptide of porcine cholecystokinin (CCK). Turkey antral extracts contain factors distinguishable in immunochemical and gel filtration properties from the mammalian forms of gastrin and CCK.

In mammals, gastrin and cholecystokinin (CCK) occur in both gut endocrine cells and central or peripheral neurones, and so may function not only as gut hormones but also as neurotransmitters or neuromodulators[2-5]. Porcine gastrin and CCK share a common COOH-terminal pentapeptide, and on this evidence are thought to have evolved from a common ancestor[6,7]. However, the phylogenetic origins of the dual distribution in brain and gut remain poorly understood. In the present study the comparative aspects of this problem have been extended to include an immunochemical analysis of gastrin- and CCK-like peptides in the brain and gut of a bird, the turkey *(Meleagris gallopavo).*

Methods. Young turkeys (0.5–4.0 kg) were anaesthetized with urethane, and brain, proximal jejunum and antrum removed and boiled for 3 min in water or acetic acid (0.5 M). The tissue extracts (0.1 g/ml) were homogenized, centrifuged (2000×g, 10 min) and supernatants stored at −20 °C prior to analysis by radioimmunoassay (RIA) and gel filtration on Sephadex G50. The antiserum (L48) used in routine RIA was raised against synthetic COOH-terminal octapeptide of porcine CCK (CCK8) coupled to bovine serum albumin by carbodiimide[8], and was employed with