

## RELATIONS BETWEEN PROLACTIN AND CALCITONIN

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According to some reports, administration of exogenous calcitonin (injected intravenously or into the third ventricle of the brain) causes a marked fall of the peripheral blood prolactin level [4]. If this effect of calcitonin on the lactotrophic function of the pituitary is direct, the possibility that prolactin may have a reverse effect on calcitonin secretion must also be postulated. Some indirect considerations could evidently be interpreted as supporting this assumption. We know that the same thyrotrophin releasing hormone (TRH) which excites the thyrotrophic function of the pituitary [5] is an activator of adeno-hypophyseal lactotrophic function, and potentiation or weakening of one of these functions ought therefore to be reflected in the other. For instance, in women with galactorrhea, symptoms of hypothyroidism are frequently observed [2], and this evidence that increased secretion of prolactin is accompanied by weakening of thyrotrophic function. In the same way in thyrotoxicosis, when the pituitary thyrotrophic function ceases to respond to TRH, the patients' blood prolactin level is raised [1]. Meanwhile in thyrotoxicosis considerable stimulation of calcitonin secretion is probable, for the state of the parafollicular cells (calcitoninocytes) of the thyroid gland is evidence of their intensive activation [3].

However, direct information on the possible effect of prolactin on calcitonin production and secretion is restricted to the brief statement that administration of prolactin can raise the blood calcitonin concentration [6]. In the present investigation these hypotheses were tested and an attempt made to determine the true character of relations between the prolactin and calcitonin levels in the intact organism.

## EXPERIMENTAL METHOD

Experiments were carried out on sexually mature male Wistar rats into which prolactin (lactin, from the Kaunas Endocrine Preparations Factory) was injected intraperitoneally in a dose of 3.5 Units/200 g body weight daily for 5 days. At the end of this period blood samples were taken from the heart of the experimental and control intact animals (of the same weight and age), under ether anesthesia, and these were tested for their calcitonin concentration by radioimmunoassay, using the Calcitonin  $^{125}\text{I}$  RIA Kit from the "Immuno Nuclear Corporation" (Italy).

## EXPERIMENTAL RESULTS

Injection of prolactin caused the peripheral blood calcitonin concentration to rise on average from  $138.0 \pm 5.1$  to  $705 \pm 11.4$  pg/ml, which was more than 5 times higher than the level of this hormone in the control animals. These results confirm that prolactin stimulates production and secretion of calcitonin. Consequently, whereas an excess of exogenous calcitonin inhibits prolactin secretion by the anterior lobe of the pituitary, the effect of an excess of prolactin is manifested very clearly as an increase in production and secretion of calcitonin. Thus *in vivo* relations between the lactotrophic function of the adeno-hypophysis and the calcitonin concentration in the circulation constitute a negative feedback system, which maintains homeostatic equilibrium between the two factors, and should this equilibrium be disturbed, it helps to restore homeostasis.

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Since it has been shown that injury to the median eminence or blocking of dopamine receptors by haloperidol prevents the effect of calcitonin on prolactin secretion [4] it can be tentatively suggested that an important role in the mechanism of negative feedback between prolactin secretion and the blood calcitonin concentration is played by dopamine, and that this connection is closed in the hypothalamus (at the level of the median eminence).

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#### CHANGES IN PLOIDY CLASSES OF LEFT ATRIAL MYOCYTES AFTER LIGATION OF THE LEFT CORONARY ARTERY IN RATS

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Stimulation of DNA synthesis in myocytes of the left atrium after infarction have been clearly demonstrated [5]. A recent cytofluorometric study of atrial myocytes revealed a considerable increase in the mean number of binuclear cells 11 days after ligation of the left coronary artery [9].

The aim of this investigation was to study the effect of this procedure at different times after the operation.

#### EXPERIMENTAL METHOD

The left coronary artery was ligated in male Wistar rats weighing 120-140 g at the point of its origin from the aorta [10]. Some rats of the same weight were left intact as controls, and on some animals a mock operation was performed — the pericardium was removed. The animals were killed with ether after 10, 20, 50, 90, and 150 days. The auricle of the left atrium was fixed in 10% formalin solution in buffer at pH 7.0 and dissociated with alkali into single cells [2]. Feulgen's reaction was carried out on films of isolated cells. The DNA content in the nuclei was determined on a Vickers M-86 scanning microdensitometer. The number of mono- and binuclear monocytes (in %) was determined accurately by examining a further 2000-3000 cells from each animal.

#### EXPERIMENTAL RESULTS

Intact control rats weighing 150 g had diploid ( $2c$ ) myocytes in the left atrium 10 days after the beginning of the experiment, although binuclear cells were found in all films, mainly with diploid nuclei ( $2c \times 2$ ), together with a fraction of a per cent of multinuclear and other myocytes with high levels of ploidy (Table 1; Fig. 1a). After 40 or 80 days the number of binuclear myocytes ( $2c \times 2$ ) in intact rats weighing about 300 g rose to 23-24% (Fig. 1b), in agreement with data in the literature [9]. In three of four animals which survived 10 days after ligation of the left coronary artery, the composition of the myocytes

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