

mized rats (group 3) has once again produced a significant decrease ( $p=0.03$ ) in mast cell population in comparison to control rats, but the decrease is less marked than in case of group 2. 3. The mast cell population in adrenalectomized Shay rats (group 3) is significantly greater ( $p=0.001$  or less) than in Shay rats with adrenals intact (group 2).

**Discussion.** Pylorus ligation in normal rats acts like a stressor leading to degranulation of mast cells in gastric mucosa, thereby decreasing their number. This decrease is less pronounced in rats devoid of adrenals. This implies that the action of stressor on gastric mucosal mast cells is mediated through adrenal glands. In light of available literature<sup>1,11</sup>, it can be inferred that stress acts via hypothalamus and anterior pituitary on adrenal cortex which secretes glucocorticoids and the latter in turn degranulate the mast cells leading to a decrease in their number. In the absence of adrenals, the mast cell degranulation is comparatively less and hence many remain to take up the stain leading to an increase in their population. Degranulation of

mast cells lead to histamine liberation and this potent secretagogue can explain stress-induced gastric hypersecretion and peptic ulceration.

Decrease even in absence of adrenals in group 3 rats, can be attributed to extra-adrenal factors involved in stress syndrome<sup>8</sup>. It is logical to conclude from these observations that a stressful stimulus like pylorus ligation involves the adrenal glands and the gastric mucosal mast cells, in its action on gastric secretion.

Effect of pylorus ligation on gastric mucosal mast cell population (MCP) in normal and adrenalectomized albino rats

Group (10 rats in each group)	MCP $\pm$ SE	p-value
1 Control	255 $\pm$ 7	1-2: <0.001
2 Control-Shay	189 $\pm$ 9	1-3: 0.03
3 Adrenalectomy-Shay	233 $\pm$ 6	2-3: <0.001

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## The influence of the sex-hormone testosterone on body temperature and metabolism of the male Japanese quail (*Coturnix coturnix japonica*)

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**Summary.** Testosterone causes a significant body temperature decrease in male quails. Oxygen consumption/g b.wt remains the same, however.

Up to now, relatively little is known about the effect of sex-hormones on the body temperature and metabolism of birds. Observations point to sex specific differences in body temperatures<sup>1,2</sup> as well as decrease in the metabolism of castrates<sup>3,4</sup>. In this experiment, we investigated the qualitative influence of the male sex hormone, testosterone, on the physiological parameters stated above.

**Materials and methods.** The test animals (the Japanese quail, *Coturnix coturnix japonica*) were 3-4 months old. They were kept at 20°C when exposed to long days (15 h light:9 h dark) as also when exposed to short days (9 h light:15 h dark). At this constant temperature, oxygen consumption was measured (measuring instruments: Beckman G2 oxygen analyser, Hartmann u. Braun Uras 2T and Magnos 2T; testing time: in darkness between 16.00 h and 08.00 h). Body temperature was measured cloacally with a digital thermometer designed by Testotherm KG. 'Testoviron' and 'Testoviron-Depot' (Schering AG/Berlin) were used as testosterone-substitutes.

**Results.** 1. Body temperature: There are highly significant sex specific differences concerning body temperature (male:  $41.8 \pm 0.20^\circ\text{C}$ , female:  $42.1 \pm 0.15^\circ\text{C}$ ). Castrated male quails (K) kept under long day conditions display a

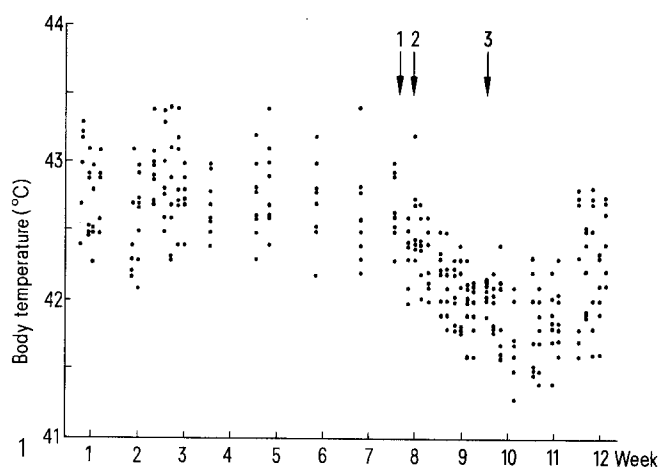


Fig. 1. Body temperature of castrated male quails before and after testosterone-substitution. The dots show single values measured ( $n=8$ ). The arrows show when Testoviron injections were given. Arrow 1: 1 mg Testoviron, arrow 2: 25 mg Testoviron-Depot and arrow 3: 12.5 mg Testoviron-Depot. The injections were given i.m. in the breast muscle.

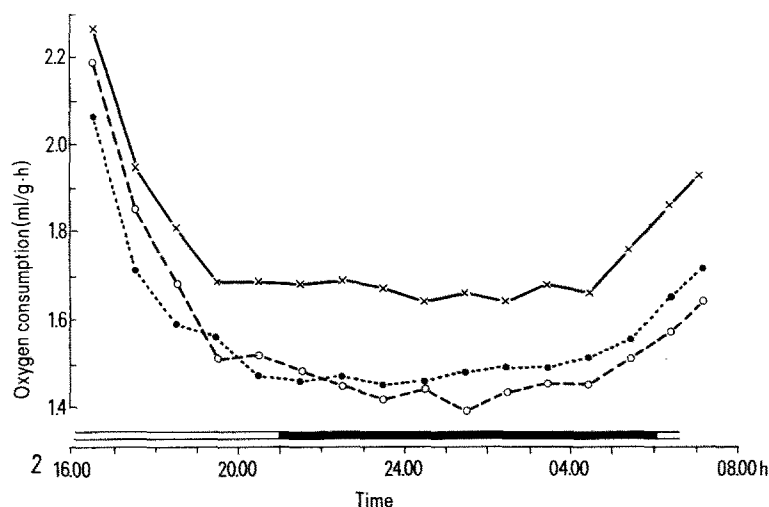


Fig. 2. Nocturnal oxygen consumption of different test-groups:  $\times$  = normal males under long day conditions ( $n=19$ ),  $\bullet$  = castrated males under long day conditions ( $n=12$ ),  $\circ$  = castrates treated with Testoviron ( $n=18$ ). Each single value gives the respective hourly mean of all animals tested. The bar in black and white shows the LD-cycle under laboratory conditions.

highly significant higher body temperature ( $42.8 \pm 0.18^\circ\text{C}$ ) than normal male quails; under short day conditions (testicles inactive), these differences do not occur. Both test groups show approximately equally high body temperatures (average values: males  $43.1^\circ\text{C}$ , castrates  $43.3^\circ\text{C}$ ) above those measured under long day conditions. Through Testoviron or Testoviron-Depot, the body temperature of castrates held under long day conditions can be lowered to the level of control animals (active testes). Body temperature rises again, when the effect of the given preparation diminishes (figure 1).

2. Metabolism: All groups tested showed an oxygen consumption which figured approximately 35% above the expected value<sup>5</sup>. This supports Blem's results<sup>6</sup>. In relation to g b.wt, castrates' metabolism is reduced about 11% compared with that of the normal males ( $1.52 \pm 0.13$  respectively  $1.71 \pm 0.11$  ml  $\text{O}_2/\text{g} \cdot \text{h}$ ; figure 2). This difference is highly significant.

The average weight of castrates (123 g) lies, however, approximately 7% above that of control animals (115 g). If

this weight specific effect is taken into account, no difference between the metabolism of castrates and males can be seen. Compare also with Pohl<sup>7</sup> here.

During the time in which the testosterone preparation is effective (low body temperature), metabolism of the castrates does not change (before:  $1.52 \pm 0.13$ ; after:  $1.51 \pm 0.07$  ml  $\text{O}_2/\text{g} \cdot \text{h}$ ). Therefore, the changed body temperature is probably due to different thermal conductance (insulation), caused for example by an accumulation of reserve fat. Accordingly further experiments have been undertaken.

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## Effects of isoproterenol and dopamine on the myocardial hexose monophosphate shunt<sup>1</sup>

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**Summary.** Long-term exposure of rats to isoproterenol and dopamine resulted in an increase of glucose-6-phosphate dehydrogenase activity and a greater availability of 5-phosphoribosyl-1-pyrophosphate in the myocardium. These results are interpreted to indicate an enhanced flow through the hexose monophosphate shunt.

The stimulatory effect of catecholamines on myocardial glycogenolysis is well established<sup>3,4</sup>, their long-term influence on the hexose monophosphate shunt (HMS), however, has not yet been sufficiently elucidated. The HMS is important for cardiac metabolism mainly because it supplies ribose-5-phosphate for production of 5-phosphoribosyl-1-pyrophosphate (PRPP). This is an essential substrate necessary for the conversion of the purine bases adenine and hypoxanthine to the corresponding mononucleotides as well as for the biosynthesis of purine and pyrimidine nucleotides<sup>5</sup>. Therefore, the HMS was evaluat-

ed in rat hearts under the influence of isoproterenol and dopamine. Compared with other methods<sup>6,7</sup> our procedure for assessing the HMS is based on the in vitro-measurement of the activity of glucose-6-phosphate dehydrogenase (G-6-P-DH, EC 1.1.1.49), the first and rate-limiting enzyme of the HMS<sup>8</sup>, and the in vivo-determination of the available PRPP, which is supplied by the HMS via ribose-5-phosphate.

**Material and methods.** All experiments were done on female Sprague-Dawley rats (200–220 g) fed a diet of Altromin<sup>®</sup> with free access to water. Isoproterenol (pur-