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PHOTOPERIODIC MODULATION OF LEYDIG CELL FUNCTION IN THE BANK VOLE (CLETHRIONOMYS GLAREOLUS)

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JUVENILE VOLES BORN AND REARED IN A LONG PHOTOPERIOD (I8L:6D) UNTIL WEANING (I8-22 DAYS OF AGE) WERE SUBJECTED EITHER TO THE SAME LONG (GROUP L) OR TO A SHORT PHOTOPERIOD (6L:I8D, GROUP S) FOR 0,3,7,I4 OR 42-56 DAYS.THE TEMPORAL CHANGES IN TESTICULAR WEIGHT,LEYDIG CELL NUMBERS AND IN THE SPECIFIC BINDING OF [^{125}I]-HCG WERE STUDIED,ALSO THE LEYDIG CELL ULTRASTRUCTURE AS WELL AS THE TESTICULAR IN VITRO METABOLISM OF [4-^{14}C]-I7α-HYDROXYPROGESTERONE WERE INVESTIGATED IN VOLES SUBJECTED TO DIFFERENTIAL PHOTOPERIODS FOR 42-56 DAYS.
LIGHT DEPRIVATION INHIBITED TESTICULAR WEIGHT-GAIN IN JUVENILE BANK VOLES AND SUBSEQUENTLY INDUCED A MARKED DECREASE IN TESTICULAR WEIGHT.THERE WAS AN OVERALL INCREASE ($P<0.001$) IN LH RECEPTORS PER TESTIS DURING TESTICULAR MATURATION IN GROUP L WHEREAS A SIGNIFICANT ($P<0.001$) REDUCTION WAS NOTED IN GROUP S.SHORT PHOTOPERIOD ARRESTED THE INDUCTION OF LH RECEPTORS AS WELL AS THE INCREASE IN LEYDIG CELL NUMBERS ASSOSIATED WITH NORMAL TESTICULAR MATURATION.THESE CHANGES WERE FIRST EVIDENT AFTER ONE WEEK ($P<0.001$) OF DIFFERENTIAL PHOTOPERIOD TREATMENT.GROUP S TESTES HAD ALSO A REDUCED CAPACITY TO CONVERT THE RADIOLABELLED SUBSTRATE TO 17α-HYDROXY,20α-DIHYDROPROGESTERONE AND ANDROGENS.IN GROUP S,WHEN COMPARED TO GROUP L,THERE WAS A MARKED REDUCTION IN THE CYTOPLASMIC AS WELL AS THE NUCLEAR VOLUME OF LEYDIG CELLS AND IN THE ORGANELLES INVOLVED IN STEROIDOGENESIS.IT IS CONCLUDED THAT PHOTOPERIOD IS AN IMPORTANT REGULATOR OF LEYDIG CELL FUNCTION IN THIS SPECIES.

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GLICOCORTICOID AND ANDROGEN STEROIDS IN OBESE PATIENTS

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The aim of this work was studying adrenocortical function in obese patients. We examined a group of 18 patients, aged between 16 and 71: 15 admitted into our Institute and 3 in Hospital Day; all of them were more than 20% beyond ideal weight according by Lorentz's formula. We tested plasmatic cortisole and androgen steroids. An hypercortisolism was found in 44% of cases and it was, on an average, 33% beyond the normal upper limit (range from 5 to 20 µg/100 ml. according to R.I.A. Service of Ferrara Hospital), values near upper limits (15 to 20 µg) in another 33% of cases. This hypercortisolism seems to be indipendent from age. Dexamethasone low dose supression test (practised on a sample group of obese patients) and maintenance of circadian rhythm seem to show a right preservation of feed-back mechanism still controlling both obesity and hypercortisolism. To investigate adrenocortical androgen production, we tested some patients, as a sample, for Δ4-androstenedione and moreover, testosterone. Δ4-androstenedione increased in 70% of cases (with an average increase of 34%), testosterone in 50% of cases (average increase of 25%): this androgen production was supressed by low dose dexamethasone. It is suggestive to notice that both Δ4-androstenedione and cortisole incresed in the same patients and almost with the same percentage, slightly lower was testosterone increase.
From analysis of data, presence of obesity, hypercortisolism and hyperandrogenism can suggest that weight increase coul be associated to an adrenocortical hyperfunction. This hyperfunction appears to be likely ascribed to glucocorticoid and androgenic fractions.
It is possible that also mineralocorticoid fraction could be affected if our preliminary data concerning aldosterone in 3 obese patients (that results increased and dinamically alterated in clino-orthostatic position) will be confirmed in future.