ecdysone occurs. In contrast, the imaginal ecdysis in Pieris can take place when the ecdysone levels is 8 times lower than its maximum value. It is apparent that the amount of hormone required for imaginal ecdysis is low and considerably less than the highest concentration attained. This results brings to mind experiments performed in the larvae of Manduca sexta[10], where infusions of small doses of ecdysone were more effective in eliciting a normal physiological response than a single large dose of the hormone. The prothoracic glands may secrete an excess of hormone which is not necessary for the imaginal moult but may have some other function than moulting. The critical period for the pupal stage (72 h) coincides with the 1st peak of α-ecdysone described in Pieris by Lafont et al.[7] who used a gas liquid chromatography and mass spectrometry method. We have not found this distinct peak in our result.

The loss of prothoracic glands parallels the loss of the large peak in ecdysone levels, but there is a persistant low level which varies between 0.1 and 0.3 μg/ml. Life of ecdysone in vivo is about one or few h[10–13]. The constant low level of ecdysone could be due to a synthesis by an organ other than the prothoracic glands, or to the liberation of stored ecdysone from tissues. In some insects, isolated abdomens have been shown to synthetize ecdysone[14–17], as do oenocytes[18] and ovaries[19–22].

Abdomens isolated at 72 h developed more slowly and with greater variability than the control animals. In Samia cynthia[23] and Galleria mellonella[24], injection of physiological large amount of ecdysone accelerated development. In Locusta migratoria, partial removal of the ventral glands during stages IV and V increased the length of these stages, and implantation decreased it[25]. There seems to be a relationship between rate of development and amount of ecdysone, and this could explain in part the lengthening of the pupal stage for isolated abdomens.

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Effects of constant light exposure and blindness on the oxidative metabolism of selected brain areas in male rats

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31 January 1977

Summary. Male rats were housed in continuous illumination or blinded when 21 day-old and killed 69 days later. The continuous illumination exposure increased the weights of testes and sex accessory organs and reduced the pineal gland weight. Blindness decreased weights of testes, sex accessory organs and anterior pituitary. The oxygen consumption rate of the hypothalamus was higher in the blinded animals than in the controls and lower in the continuously illuminated rats. No one of such groups showed significant changes in the oxygen consumption by either the amygdala or the hippocampus.

Some data show evidence that the oxidative metabolism of the hypothalamus and several areas of the limbic system can be influenced by the pituitary-gonadal axis activity. Thus, the oxygen consumption rates of the hypothalamus, the amygdala and, to a lesser extent, the hippocampus, has been reported to be changed by the oestrus cycle phases[1,2] and after experimental manipulations such as castration[1,3,4], hypophysectomy[5] and in vivo treatment with gonadal steroids and gonadotropins[3,4,6]. Data from in vitro works suggest that such effects are mainly due to changes in the pituitary gonadotropin output, which in turn would act directly on the above-mentioned brain areas[1,4,7]. This point constitutes one of the main supports for the gonadotropins 'short feed-back' theory[8].

On the other hand, the exposure of rats to constant illumination has been said to result in an enhanced gonadotropin secretion, and both the blindness or the exposure to constant darkness had the opposite effect (see Fraschini et al.[9] and Reiter[10] for review).

The present experiment was planned in order to see whether changes in light manipulations, with the associated changes in gonadotropin secretion mentioned above, could also affect the hypothalamus, the amygdala or the hippocampus oxygen consumption rates.

Material and methods. 21 day-old male Wistar rats were placed under different experimental conditions. All the animals were maintained in a temperature-controlled room (20 ± 3 °C) and given tap water and commercial chow ad libitum. Animals were divided into the 3 groups shown in table 1. Rats of group 1 were maintained under 14 h daily of artificial illumination, and those of group 2 were under constant illumination during the experiment. Rats of group 3 were blinded and maintained under constant darkness. When the animals were 90 days old, they were killed by decapitation and their endocrine organs dissected out and weighed. The amygdala, hypothalamus and hippocampus were dissected out. The oxygen consumption was determined by Warburg manometry method[11] in vessels in 12–15 ml capacity containing

Table 1. Effects of continuous light exposure or blindness on endocrine organs weights of male rats

Groups	B. wt (g)	Testicular wt (g)	Sex accessory organs wt (mg)	Ant. pituitary wt (mg)	Pineal wt (mg)
Control	212.6 ± 5.54 (23)	2.13 ± 0.08 (23)	319.12 ± 14.82 (19)	6.29 ± 0.18 (23)	1.32 ± 0.09 (19)
Constant light	226.8 ± 2.82 (16)	2.66 ± 0.07 (16)**	408.56 ± 20.12 (12)	6.58 ± 0.26 (12)	0.85 ± 0.07 (15)**
Blindness	221.9 ± 6.48 (14)	1.70 ± 0.04 (10)**	148.30 ± 15.82 (10)**	5.60 ± 0.22 (10)*	1.21 ± 0.07 (14)

Data expressed as means ± SEM. Number of measurements in parenthesis. *$p < 0.05$ vs control; **$p < 0.01$ vs control.

Table 2. Effects of continuous light exposure or blindness on oxidative metabolism of Amygdala, Hypothalamus and Hippocampus of male rats

Groups	Amygdala	Hypothalamus	Hippocampus
Control	1.080 ± 0.088 (17)	1.099 ± 0.051 (16)	1.110 ± 0.048 (12)
Constant light	1.059 ± 0.121 (8)	0.799 ± 0.039 (8) **	0.977 ± 0.022 (8)
Blindness	0.973 ± 0.098 (12)	1.358 ± 0.141 (10)*	0.994 ± 0.078 (12)

Data expressed as means ± SEM of µl O_2/mg wet tissue/h. Number of measurements in paranthesis. *$p < 0.05$ vs control; **$p < 0.01$ vs control.

3 ml of Krebs-Ringer phosphate buffer pH 7.4 and 7.7 mM glucose. The central wall of the vessel contained 0.2 ml of saturated NaOH solution. The vessels were gassed for 5 min with 100% O_2. After 10 min to allow for the equilibrium of the system, the study was executed at 37 °C and 120 beats per min for 1 h. The results are expressed as µl O_2/mg wt tissue/h. The analysis of the data was carried out using Student's t-test.

Results and discussion. Table 1 shows the effects of blindness or constant light exposure on the weights of organs. No significant differences in b.wts were observed at the p-value of 0.01 or lower. Exposure to constant illumination resulted in a significant enlargement of testes and sex accesory organs, suggesting an enhanced gonadotropins secretion, and decreased pineal gland weights, which is in agreement with other reports[12]. The low testicular, sex accessory organs and anterior pituitary weights found in the blinded animals, suggesting a decreased pituitary gonadotropin output, are in agreement with data reported by other workers[13,14].

Continuous light exposure decreased the oxygen consumption by the hypothalamus (table 2). Such an effect is similar to that reported for castration[1,15] and in vitro addition of gonadotropins to hypothalamic tissue[7]. On the other hand, blindness enhanced the hypothalamic oxygen consumption rate, as has been found after hypophysectomy[5].

Such parallelisms between the present results and those of the experimental conditions existing in the literature suggest that the changes in the hypothalamic oxidative metabolism observed in the present experiment could be secondary to the changes in gonadotropin secretion induced by the different lighting conditions. Neither the amygdala or the hipocampus oxygen consumption showed significant differences from the control group. However, the amygdala oxygen consumption has been reported to decrease after hypophysectomy and to increase after castration or in vitro addition of gonadotropins[4]. From the analogy of such experimental conditions and those of blindness and exposure to constant illumination respectively, as mentioned above, some changes could be expected in the amygdala also. A possible explanation for such a discrepancy could be different sensitivities to the gonadotropins of the amygdala and the hypothalamus. Thus blindness, which decreases but does not completely suppress the gonadotropin secretion[14], would increase the hypothalamic oxygen consumption without changes in that of the amygdala. The exposure to constant illumination, which enhances the gonadotropin secretion probably without reaching the highest plasma levels found after castration[16], would only change the oxidative metabolism of the more sensitive hypothalamus.

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