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## Gonadal hormones and heart rate as an emotional response

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## SUMMARY

Animal experiments may give information on the physiology of hormones under stress conditions. The model for the investigation of acute emotional stress in animals that has been chosen permits the study of heart rate in freely moving laboratory rats as a sensitive psychophysiological parameter. This paradigm is usually described as passive avoidance behaviour. It uses the animals' innate preference for dark over light. After initial adaptation trials for learning rats to step from a lit platform into a dark chamber a single electric foot shock is given in this chamber. After 24 h the animals are reintroduced into the same dark chamber where heart rate is measured during one minute and compared to heart rates of unshocked controls. A few hours later the animals' tendency to reenter the dark compartment where they had been shocked is measured and expressed as latency time to enter, i.e. avoidance behaviour. Heart rate reactions during the forced exposure to the shock compartment consist of a relative bradycardia in comparison to the stimulated heart rates of freely exploring control animals. Based on findings by others it may be inferred that this bradycardia is mediated vagally through a mechanism independent of baroreceptor reflex influences, although blood pressure is known to increase during passive avoidance behaviour and forced exposure. This reaction pattern may be related with or identical to the circulatory component of the orienting response.

The hypothesis formulated for the present research holds that the condition of oestrus in the rat would decrease the responsiveness of heart rate as a physiological stress parameter, oestradiol being the most probable hormone of interest.

In the first set of experiments (Chapter III), heart rate was studied during actual avoidance behaviour in castrated female rats treated with oestradiol and progesterone according to a scheme that is known to induce sexual receptiveness. These experiments were unsuccessful due to a combination of unexpected difficulties. Firstly, avoidance latencies were very unequal which hampered proper comparison within groups between short and long avoiding individuals. Secondly, no significant bradycardia was obtained in comparison to the last preshock entering trial under the conditions used. Passive avoidance latency times were not influenced by any of the hormone treatment modalities used, i.e. oil-diluent, oestradiol benzoate, or progesterone, either alone or in combination, either low or high dose administrations, notwithstanding the effectiveness reflected in vaginal cytology.

Next (Chapter IV), we measured heart rate responses during forced exposure to the shock compartment preceding behavioural testing. Now, intact female animals were used while their hormonal cycles were monitored by daily vaginal cytology sampling after experimentation. During di-oestrus a significant bradycardia was found relative to non-shocked controls. Rats in oestrus had less stimulated heart rates in the control (non-shock) situation and lacked stress bradycardia. Thus, the condition of oestrus seemed to have an influence on the psychophysiology of both environmental modalities. No effect of oestrus on subsequent passive avoidance behaviour was found. Intact male rats were tested in the same way. They showed slightly higher levels of stress bradycardia relative to their controls, which by themselves were quite comparable to di-oestrus female controls. Their avoidance latencies were not different from either group of females. Experiments with ovariectomized females and spayed males pointed to a specific effect of female gonadal hormones, but not testosterone, on stress bradycardia while no effects on avoidance behaviour after forced exposure were found.

Behavioural rating of immobility during forced exposure revealed no differences between oestrus and di-oestrus or with intact males. Separate analysis of heart rates for immobility versus movement did not indicate that immobility was a cause of lower heart rates. Interestingly, the intensity of bradycardia was positively correlated to the total time of immobility during forced exposure in the males, but not in the females in either hormonal status. This points to an effect independent of the presence of ovarian hormones. It could be related to the actual presence of testosterone or to organizational differences between males and females induced perinatally, i.e. to sexual dimorphism. When intact male rats were matched to age instead of body weight of the females, stress bradycardia had disappeared in these considerably older males. This also points to a sexual dimorphic difference between males and females while in this situation an activational effect of testosterone seems improbable.

The final set of experiments (Chapter V) was performed in order to analyze the differential influences of oestradiol and progesterone in females and their activational effectiveness in adult males. Moderately high amounts of oestradiol benzoate given during three subsequent days could restore heart rate responses in ovariectomized females to those seen during oestrus. Low physiological doses of the hormone could not replicate this effect, however, neither was oestradiol able to induce an attenuation of bradycardia in spayed males in moderate or even in pharmacologically high amounts. Interestingly, progesterone in a moderately high dose induced a dichotomy of heart rate responses in stressed females; a number of these displayed strong bradycardia while the others showed no bradycardia at all in comparison to the controls. In the control group no dichotomy occurred. No differences in subsequent avoidance behaviour were found in any of these groups.

Oestradiol is considered to be of primary importance for the reduction of heart rate responsiveness to acute emotional stress in adult female rats. It is effective as an activational substance while adult males are insensitive pointing to sexual dimorphism by an organizational influence of testosterone, probably in the

period around birth. This is also suggested by the disappearance of stress bradycardia in older male rats age-matched to the females used. Strikingly, oestradiol alone is ineffective in low physiological amounts. In analogy to the induction of sexual behavioural receptivity the hypothesis is formulated that both these effects, the behavioural and the psychophysiological, are mediated via the same brain structure, namely the ventromedial nucleus of the hypothalamus. It is more difficult to interpret the bimodal effect of progesterone, but it could still support this hypothesis if one considers its time dependent facilitatory-inhibitory effects on sexual behaviour.

In conclusion, the state of oestrus may not only imply soliciting behaviour in order to find a mate and behavioural receptivity (lordosis behaviour). It may also imply a decrease of arousability and anxiety as reflected by heart rate stimulation or inhibition, dependent on the external cues. This may be understood as a facilitatory condition for reproduction thus serving the existential need of the species.