172.12 EFFECTS OF COMPLETE AND PARTIAL SCN LESIONS ON ULTRADIAN AND CIRCADIAN LOCOMOTOR ACTIVITY RHYTHMS IN LEW/ZTM RATS.

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Both circadian and ultradian rhythms in locomotor activity have been observed in laboratory rats. However, while the neural pacemaker involved in the regulation of the circadian activity rhythm (as well as many other rodent circadian rhythms) appears to be located in the suprachiasmatic nuclei (SCN), the neural basis for the generation of behavioral ultradian rhythms remains unclear. Complete and partial lesions of the SCN were used to determine if the same neural substitute may underlie both circadian and ultradian rhythms in the wheel-running activity of LEW/Ztm rats. This inbred strain of laboratory rats exhibits very precise and reproducible ultradian rhythms with periods of 4 and 4.8 h in locomotor activity (Büttner & Wollnik, Behav. Genet., 14:137, 1984) as well as in heart rate and body temperature.

Male and female rats of this strain were maintained under light-dark entrainment (LD 12:12) and under free-running conditions (blinded animals). They received complete or partial SCN lesions between days 35 and 50 after initially being housed in running-wheel cages. Wheel-running activity was recorded for a total of 90 days using an on-line microcomputer system. To detect rhythmic components, activity records were subject to chi-square periodogram and harmonic spectral analyses.

While sham lesions had little or no effect on the wheel-running activity pattern, complete SCN lesions resulted in a complete loss of ultradian and circadian rhythms in both male and female animals. The absence of ultradian and circadian rhythms was observed in blinded, free-running animals as well as in those maintained under LD 12:12 conditions. Under both free-running or entrained conditions, ultradian and circadian rhythms were still present after partial SCN lesions, with their amplitude dependent on the size of the lesion. Periodogram analysis for any given animal revealed that the period of the ultradian rhythm was always a submultiple of the entrained or free-running circadian period. Furthermore, a close relationship between ultradian and circadian rhythms was suggested by a high correlation between the amplitudes of their spectral estimates.

The present results indicate that the SCN contributes to the control of both ultradian and circadian rhythms in wheel-running behavior of LEW/Ztm rats. However, it is not clear if ultradian rhythms are caused by an ultradian pacemaker within the SCN or if they are the result of desynchronization among a population of circadian oscillators. Further examination of the LEW/Ztm strain might be useful in developing a better understanding of the origin and function of behavioral ultradian rhythms.

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