several respects. It is the first evidence that an influenza virus antigenically and genetically related to avian viruses can be associated with severe disease in wild mammals. As with influenza in swine, concurrent respiratory infection with an organism such as mycoplasma and adverse environmental conditions may have triggered the expression of a latent virus. The striking similarities between the harbor seal epizootic and large-scale seal mortalities in the past suggest that these events may be linked by common biological and environmental factors that we are only beginning to recognize.

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16. Field officers of the U.S. National Marine Fisheries Service (NMFS), U.S. National Park Service, U.S. Department of Agriculture, Massachusetts Department of Natural Resources, Maine State Department of Marine Research, Canadian Fisheries and Oceans (CFO), Sealand of Cape Cod, and the International Fund for Animal Welfare coordinated the collection of seals. R. Prescott, Cape Cod Museum of Natural History, and C. Skinder, New England Aquarium, gave technical assistance. M. Rosenfeld, Manomet Bird Observatory (Massachusetts), provided field descriptions. R. A. Del Guidice, Frederick Cancer Research Center, Frederick, Md., and J. G. Tully, National Institutes of Health, Bethesda, Md., helped to characterize the mycoplasma. S. Marsh and C. Rodd prepared the figure and M. Hundleby critically reviewed the manuscript. This study was supported by grants AI02649 and AI16841 from the National Institute of Allergy and Infectious Diseases, by the NMFS, CFO (01SUFP802-0-2186), Arco Foundation, and by Natural Sciences and Engineering Research Council of Canada (A6130).

13 October 1981

Computer-Detected Patterns of Electroencephalographic Delta Activity During and After Extended Sleep

Abstract. *Delta (0.5 to 3 hertz) waves are the electroencephalographic hallmark of human sleep. We measured their rate of production during and following an extended night of sleep. On the extended night, we confirmed previous observations of a linear decline in delta wave production across the first four periods of non-rapid-eye-movement (non-REM) sleep. An asymptote was reached in the fifth non-REM period, perhaps signifying that sleep processes reached completion. On the day after the extended night, subjects were allowed to remain awake 3.6 hours less than normal. During the next sleep session, amplitude and number of delta waves in non-REM periods 1 and 3 were significantly reduced. These findings illustrate the value of computer analysis of electroencephalographic waveforms in sleep. Systematic measurement of the amount and distribution of these waveforms as a function of preceding waking duration should provide clues to the kinetics of the metabolic processes underlying sleep.*

Stage 4 sleep, characterized by dense, high-voltage delta (0.5 to 3 Hz) electroencephalographic (EEG) waves (*1, 2*) is of interest because (i) the amount depends on the duration of waking that has preceded sleep (*3*); (ii) delta sleep diminishes steeply across the night (*1, 4*) as though some substrate were being consumed (*5, 6*); (iii) stage 4 sleep occurring in daytime naps reduces the amount of stage 4 in the subsequent night's sleep, whereas rapid-eye-movement (REM) sleep during naps does not affect subsequent REM (*5*); (iv) delta sleep remains at normal levels or higher when total sleep time is restricted for long periods, whereas REM sleep is sacrificed (*7*); and (v) stage 4 is the sleep stage most strongly affected by age over the human lifespan (*6, 8*). These facts led us to suggest that delta waves are an electrophysiological correlate of those unknown metabolic processes by which sleep reverses the effects of waking on the brain (*6*).

Despite the intensive research on sleep during the past 25 years, and despite growing recognition of the importance of delta sleep, some of its basic characteristics have not yet been fully established. We do not know whether a longer period of wakefulness increases stage 4 sleep by increasing the density, duration, or amplitude of delta waves, each of which affects the visual scoring of this stage (*2, 9*). The effects of variations in duration of wakefulness on the distribution of delta waves across the night's sleep are also undetermined. These questions can now be investigated effectively with relatively simple computer techniques (*10*). We applied such methods in a study of EEG patterns during and after extended sleep. The visually scored data have been reported (*11*). Here, we present new observations on the trends in delta production across a night of extended sleep and on the effects of a reduced time awake on the number, amplitude, and distribution of delta waves during subsequent non-REM sleep.

Eye movements and EEG were recorded in 19 medical and 2 undergraduate male students (median age, 24.1 years; range 20.3 to 29.3 years) on three consecutive nights. The subjects were in bed for 8 hours on the adaptation night, 12 hours on the extended night, and 8 hours on the recovery night, beginning at 11 p.m. each night (*12*). They averaged 428, 641, and 427 minutes of sleep, respectively, on the three nights (*13*). Thus, the duration of waking in the 24-hour period preceding the extended night was 1012 minutes and that in the 24 hours preceding the recovery night, 799 minutes.

Table 1 gives durations of non-REM

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periods and measures of delta sleep for the 18 subjects who completed six non-REM periods on the extended night and four on the recovery night. Durations of non-REM periods are not constant, but change systematically across the night (Table 1) (*6, 9*). The results for the extended night are typical for young adults: non-REM durations were equal in the first three non-REM periods and declined sharply from period 3 to period 5. Non-REM periods 5 and 6 were equal in duration. The decline across periods 3 to 5 is found from early childhood to old age and is one of the most consistent of the temporal phenomena of human sleep (*6, 9*).

On the extended night, visually scored delta sleep (stages 3 and 4) was maximal in non-REM period 1 and declined across periods 1 to 4 (Table 1). A steep decline of stage 3 and 4 EEG across sleep has been firmly established (*1, 4, 6*) and the results for the first four non-REM periods on the extended night are typical. We combined stage 3 (moderate density of high-voltage delta) with stage 4 (high density of delta) in this report because the combined scores should more closely parallel the computer measures, which include all of the delta waves in each non-REM period.

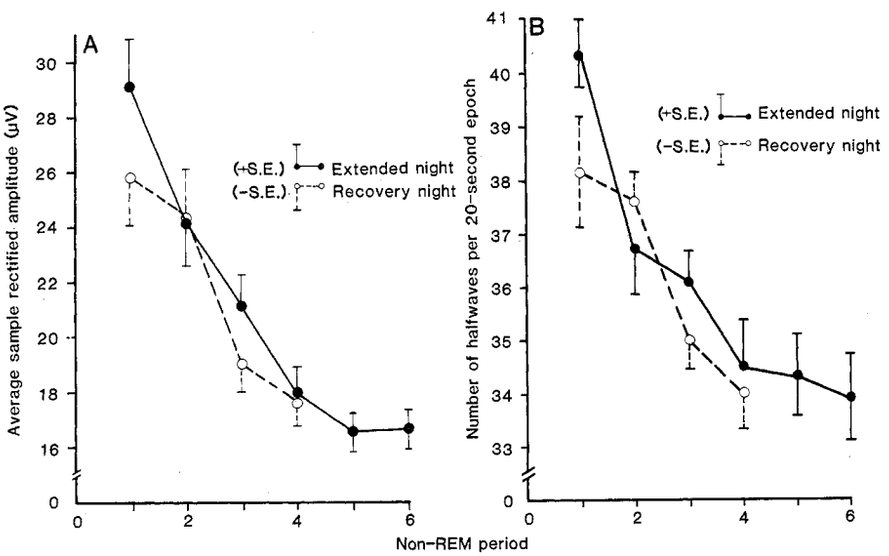

Fig. 1. Mean [± standard error (S.E.)] sample amplitude and number of halfwaves in the 0.5- to 3-Hz (delta) frequency band for the average 20-second epoch of non-REM sleep plotted by consecutive non-REM periods on the extended and recovery nights. The rate of delta wave production seems to reach an asymptote in the fifth non-REM period on the extended night. The reduced waking duration preceding the recovery night was associated with a diminished rate of delta production in the first and third non-REM periods.

Table 1. Visually scored and computer measures of delta waves in successive non-REM periods on extended and recovery nights (N = 18); N.S., not significant.

Period	Night	Visually scored		Computer measures			
		Non-REM duration (min)	Stages 3 + 4 (min)	Halfwaves per 20 seconds (No.)*	Time in bend per 20 seconds (sec)*	Mean sample amplitude (μV)†	Mean frequency (Hz)
1	Extended	70.4	43.0	40.4	13.9	29.1	1.45
	Recovery	64.5	32.4‡	38.2‡	12.9‡	25.8‖	1.49
2	Extended	73.3	23.8	36.7	12.8	24.2	1.45
	Recovery	70.8	28.2	37.6	12.9	24.4	1.46
3	Extended	71.2	15.9	36.1	12.1	21.1	1.50
	Recovery	74.9	10.5	35.0‡	11.2§	19.0‡	1.58‡
4	Extended	60.0	6.3	34.5	10.8	17.9	1.61
	Recovery	61.4	8.0	34.0	10.5	17.6	1.62
5	Extended	52.7	3.4	34.3	10.3	16.5	1.68
6	Extended	51.7	3.8	33.9	10.2	16.6	1.67
F_{linear}¶	(*15*)						
	Extended (d.f. = 1,104)	33.7	202.0	157.8	290.6	269.3	107.9
	Recovery (d.f. = 1,69)	N.S.	47.5	22.6	35.5	24.6	19.1

*Totals for each non-REM period can be derived by multiplying each of these values by (3 × non-REM duration). †Integrated amplitude divided by time in band (*10*); t-test comparison of extended and recovery night values: ‡$P < .05$. §$P < .01$. ‖$P < .001$. ¶All F values for linear trend are significant at $P < .001$.

Our previous studies revealed systematic, linear declines of amplitude, duration, and number of delta waves across non-REM periods 1 to 4 and an increase in their average frequency (*10*). The increased sleep time on the extended night permitted us to extend this analysis to include periods 1 to 6. We were interested, in terms of a metabolic model of sleep presented elsewhere (*6*), in whether delta wave production would continue to decline after period 4 or would reach an asymptote. Figure 1 and Table 1 suggest that a plateau was reached in period 5; thus, all computer measures of 0.5- to 3-Hz waves were at identical levels in non-REM periods 5 and 6. This flattening of the delta curves produced a statistically significant (analysis of variance, $P < .001$) quadratic component for each of the computer measures listed in Table 1 except for mean frequency, which showed a significant ($P < .01$) cubic component. The apparent asymptote in rate of delta production suggests that the metabolic processes reflected by 0.5- to 3-Hz EEG activity are complete by non-REM period 5. This interpretation is supported by the fact that the spontaneous sleep of human adults is normally made up of four or five non-REM periods (*6, 9, 14*).

Previous work by others (*3*) led us to expect that visually scored EEG stages 3 and 4 would be reduced on the recovery night, before which subjects were awake 3.6 hours less than before the extended night. This result was observed, but a significant reduction in visually scored delta (stage 4) was observed only in the first non-REM period (Table 1). Computer analysis of 0.5- to 3-Hz waves corroborated this result and, in addition, revealed significantly reduced delta in period 3 (Table 1 and Fig. 1).

In the first non-REM period, the most marked effect of the experimental conditions was on the amplitude of delta wave activity. In period 3, the most pronounced effect was on the duration of delta wave activity. The failure to find any experimental effects in the second non-REM period is surprising, since this period normally contains a substantial amount of delta activity.

Delta waves are perhaps the most important EEG feature distinguishing sleep from waking. An adequate empirical de-

scription of sleep, therefore, requires that one determine the quantitative functions relating the amount and distribution of delta waves during sleep to the duration of prior waking. Such data are also of theoretical interest. We have proposed that it is non-REM sleep which reverses the effects of waking on the brain and that this process is most intense during the high density, high amplitude delta phase (*6*). According to this view, the rate of delta production during sleep increases with increased waking duration because the brain activity of waking produces the "substrate" for sleep. If this hypothesis is correct, the relations between duration of waking and delta activity during subsequent sleep should reflect the kinetics of the metabolic processes of sleep.

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13. Alternatively, one could consider the sleep: wake ratio as the independent variable because we could not distinguish between effects produced by a longer period of sleep from those produced by a shorter duration of waking in the preceding 24 hours. The sleep:wake ratio preceding the extended night was 0.423 (428/1012) and that preceding the recovery night was 0.802 (641/799).
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18 August 1980; revised 28 July 1981

Maternally Inherited Sex Ratio in the Parasitoid Wasp *Nasonia vitripennis*

Abstract. *Facultative control of the sex ratio has been reported in the wasp* Nasonia vitripennis. *In a newly wild-caught strain, females produced few or no male offspring and did not show the usual alterations of sex ratio in response to external conditions. The aberrant trait is inherited through females.*

Werren *et al.* (*1*) described a paternally inherited factor that causes the production of all-male broods in the wasp *Nasonia vitripennis*. I now report its opposite: maternally inherited factor which results in the production of all-female broods. These are the first non-Mendelian sex ratio factors reported in the Hymenoptera. They are of particular interest because of the diversity of sex ratio phenomena already known in Hymenoptera and because Hymenoptera are frequently used in sex ratio studies (*2–5*).

Nasonia vitripennis is a gregarious wasp with facultative control over the sex ratio. Females alter sex ratio in response to host availability (*3*), female density (*4*), and whether they are the first or second female to parasitize a host (*5*). This control is due to the mechanism of sex determination; unfertilized eggs become haploid males and fertilized eggs, diploid females. By controlling sperm access to eggs, a female may control the sex ratio of her progeny. In July 1980, a new lab stock (sc/h) was initiated by crossing wild-caught *Nasonia* to a standard lab stock (ScDr) (*6*). The new stock produced 3.1 percent males. By contrast, normal stocks produce 50 percent males under the same mass culture conditions (*7*).

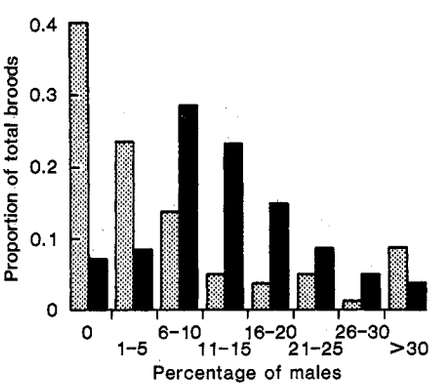

Fig. 1. Frequency histogram of the sex ratio of individual females of the two strains used in experiments. Stippled bars represent sc/h males × sc/h females (N = 81) and solid bars represent ScDr males × ScDr females (N = 81). The two distributions are significantly different (χ^2 = 46.60, d.f. = 6, $P << .001$; categories 21 to 25 and 26 to 30 were lumped for the analysis).

Because solitary females normally produce lower sex ratios (proportion of males) than are produced in mass culture (*4*), sc/h females were first tested to determine what sex ratio they exhibited in isolation. Males and females from the experimental stock were mass mated and then each female was isolated with a single fly host for 24 hours (*8*). Controls were ScDr females treated in exactly the same fashion, but mated to ScDr males. After the offspring had pupated, host puparia were opened, and the number and sex of the offspring were recorded. Although the mean number of offspring per female did not differ between groups (*9*), the sex ratios did (Fig. 1). The proportion of male offspring produced by isolated sc/h females was lower than that produced by the isolated control females (*10*). In a second experiment, when reciprocal and control crosses were made with the sc/h and ScDr stocks, sc/h females gave low sex ratios and ScDr females gave normal sex ratios, no matter which male they mated with (*11*).

In order to determine how the sex ratio trait is inherited, lines were initiated from two reciprocal crosses. Line 1 was begun with females from the cross ScDr ♂ × sc/h ♀, and line 2 with females from the cross sc/h ♂ × ScDr ♀. For each of five generations, females from each line were backcrossed to sc/h males, and then each female was given a host. Both lines were initiated with females composed of equal complements

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