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.

I. FEINBERG
G. FEIN
Veterans Administration Medical Center, San Francisco, California 94121 and *Department of Psychiatry, University of California, San Francisco 94143*
T. C. FLOYD
Veterans Administration Medical Center, San Francisco

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12. The data for four subjects were lost on the adaptation night as a result of a power outage. The results given for this night are therefore based on 14 of the 18 subjects.
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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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*).

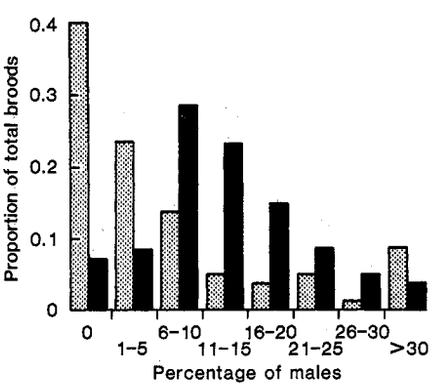

Fig. 1. Frequency histogram of the sex ratio of individual females of the two strains used in experiments. Stippled bars represent sc/h males × sc/h females (N = 81) and solid bars represent ScDr males × ScDr females (N = 81). The two distributions are significantly different (χ^2 = 46.60, d.f. = 6,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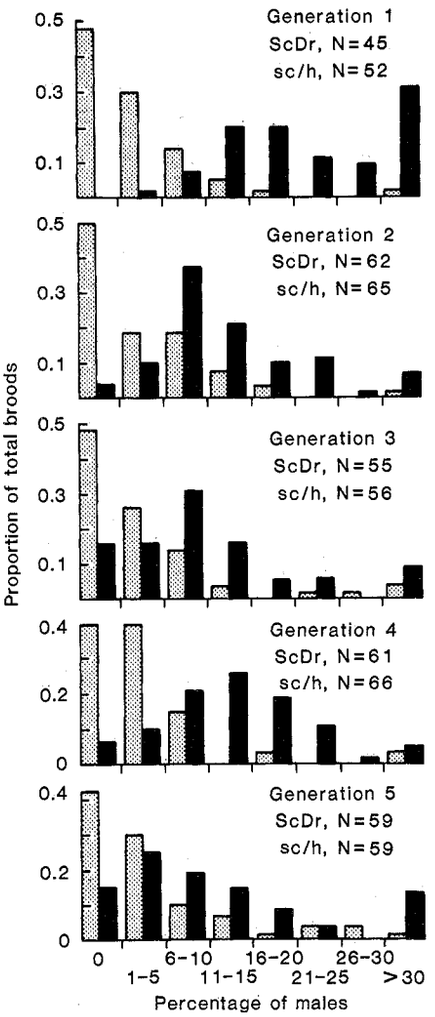

Fig. 2. Results of five generations of backcrossing females from two separate lines to sc/h males (see text for details). Stippled bars represent females with an sc/h maternal background (parental cross for generation 1 females was ScDr males × sc/h females). Solid bars represent females with an ScDr background (parental cross was sc/h males × ScDr females).

of ScDr and sc/h genes (50 percent each); they differed in maternal (cytoplasmic) background. Backcrossing to sc/h males increased the proportion of sc/h genes by roughly one-half with each generation, to ~ 97 percent sc/h in the fifth generation (*12*). In each generation, females were drawn at random from the ten broods in each line with the lowest sex ratios—thus biasing selection in favor of any genes for low sex ratio. If the trait is inherited through nuclear genes, this should result in both lines producing the low sex ratio within several generations, regardless of the genetic details. However, if the trait is maternally inherited, only line 1 should exhibit the trait, because only line 1 females have the sc/h maternal background.

Line 1 females exhibited the low sex ratio trait in all generations, whereas line 2 females never did (Fig. 2). Within each generation, the sex ratios of the two lines differed significantly ($P < .001$, Kruskal-Wallis one-way analysis of variance, d.f. = 1). Within each line, there were no significant shifts in sex ratio among generations, with the sole exception of generation 1 to generation 2 for line 2 (*13*). These data strongly suggest that the low sex ratio trait is maternally inherited and hence is extrachromosomal in nature.

Similar factors are known or suspected in a few other organisms (*14, 15*). Unlike autosomal genes, which are inherited through both sexes, extrachromosomal factors ("genes") are generally inherited through one sex only. Such extrachromosomal factors experience intrinsic selection to skew the sex ratio toward the sex through which they are inherited, whereas autosomal genes do not. This results in "conflict" over the sex ratio between extrachromosomal and autosomal factors (*14*). How the extrachromosomal factor in sc/h females causes the sex ratio skew is unknown. In some *Drosophila* species, a spiroplasma and an associated virus skew the sex ratio toward females through mortality of male offspring (*15*). Although the observed low number of sc/h males could be due to mortality, preliminary data suggest that this may not be the case. Virgin ScDr and sc/h females, which produce only males, were isolated with single hosts for 24 hours. After the females were removed, the hosts were opened, and all eggs were counted. Ten days later, the hosts were reexamined, and surviving offspring were counted. Twenty virgin ScDr females produced 487 eggs, 391 of which developed to pupae (80.3 percent); 28 virgin sc/h females produced 739 eggs, 577 of which developed (78.1 percent) (*16*). This indicates that the low sex ratios observed were not due to excess mortality of sc/h males.

Fertilization of eggs is required to produce female offspring in *Nasonia*; it occurs just before oviposition (*17*). It is possible that the sex ratio factor alters the reproductive morphology of sc/h females to prevent control over sperm access to eggs. Alternatively, the factor may actually mediate the behavior of sc/h females.

SAMUEL W. SKINNER
Department of Biology, University of Utah, Salt Lake City 84112

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7. Stocks are maintained by allowing 15 inseminated females to parasitize 10 cm^3 of *Sarcophaga* pupae until the females die. Generation time at 27°C is 2 weeks. Hosts are obtained from Carolina Biological Supply Company.
8. Two replicates were performed; data are combined since there were no significant differences between replicates. *Sarcophaga bullata* were used as hosts in all experiments.
9. The mean number of offspring [± standard deviation (S.D.)] was 27.1 ± 11.1 per ScDr female ($N = 81$) and 24.4 ± 8.9 per sc/h female ($N = 83$) (Student's $t = 1.71$; $.1 > P > .05$).
10. The mean sex ratio of sc/h females was constant (~ 3 percent male) whereas that for ScDr females altered (~ 50 percent male in mass culture and ~ 10 percent male in isolation).
11. Mean sex ratios (± S.D.) of the reciprocal crosses were: for sc/h males × ScDr females, 11.9 ± 8.06 percent ($N = 29$) and for ScDr males × sc/h females, 3.1 ± 4.39 percent ($N = 38$).
12. This assumes perfectly random assortment.
13. The Kruskal-Wallis test for generation 1 versus generation 2 ($H = 27.60$, $P << .001$, d.f. = 1, $N = 107$). Females from the high end of the generation 1 distribution were used to start a new stock that has continued to produce unusually high sex ratios. Thus, the high sex ratios of generation 1 may represent nuclear genes subsequently selected out of the experimental line.
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Estrogen Receptors at Implantation Sites of Rat Endometrium

Martel and Psychoyos (*1*) have suggested that the increased concentrations of estradiol and progesterone nuclear receptors that we found at implantation sites in the endometrium of 6-day pregnant rats (*2*) were due to an artefact caused by binding of the hormone by trypan blue. We do not agree with this interpretation, and our reasons are as follows.

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