If a pair of female CA and a 0 day ovary were implanted into the males, the pattern of JH synthesis (Fig. 1) resembled that of normal females and was identical in timing to that of females with denervated CA (Fig. 3) (*7*). The final decline in the cycle of activity of implanted CA was achieved by implanting an ovary, and this occurred in concert with the completion of oocyte maturation. This inhibition of JH synthesis by the implanted ovary must occur by way of a humoral pathway.

Figure 4 shows ovaries implanted into males with and without CA. Ovaries implanted into males in the absence of female CA showed no growth. Even after 10 days the basal oocytes remained in the previtellogenic stage as at implantation (0.65 ± 0.001 mm in length) (Fig. 2). Hence the production of JH by male CA was insufficient for oocyte development. However, in the presence of female CA the oocytes increased in length to 1.40 ± 0.06 mm by day 5 and complete chorion deposition occurred between days 7 and 8. These observations indicate that the event leading to the decline in rates of JH synthesis after day 7 is the completion of vitellogenesis as signaled by chorion formation.

In *D. punctata*, an ovary usually contains six ovarioles (*13*). In ovaries implanted into males, only a fraction produced mature oocytes (Fig. 2). To compare the relative suitability of male and female environments for ovarian growth, oocyte lengths were measured in ovaries implanted into females ovariectomized and implanted on day 0 with one ovary and a pair of CA from day 0 females. On days 5 and 7 after the operation, the mean lengths of the oocytes were greater than those of ovaries grown in males; and more oocytes have developed (Fig. 2). In addition, the vitellin content was greater by a factor of 10 (*9*). Hence the male environment, although able to support oocyte growth in the presence of female CA, did not do so to the same extent as that which occurs in normal females.

The presence of ecdysteroids in mature cockroach ovaries and hemolymph (*14*) suggests that ecdysteroids may participate in regulating the rates of JH synthesis by the CA. To test this hypothesis, CA from day 0 females were implanted into day 0 males and males injected on days 6 and 7 with doses of ecdysterone (*15*). CA were assayed for JH synthesis on day 8. The rates of JH synthesis as a function of the total dose (two equal injections) of ecdysterone are shown in Fig. 3. These data show that the inhibitory effect of ecdysterone is dose-dependent. At 100 μg and 10 μg the rates of JH synthesis are low, 13.15 ± 2.04 and 18.45 ± 2.59 pmole per pair per hour, respectively. These low rates are similar to those measured at the end of the gonadotrophic cycle in normal females (*4*) and in males implanted with an ovary and female CA (13.40 ± 5.91 and 15.69 ± 4.92 pmole per pair per hour on days 9 and 10, respectively) (Fig. 1). The rates are significantly lower than those of the controls (Fig. 3).

Whether ecdysterone from the ovary limits the activity of the CA at the end of a normal gonadotrophic cycle either directly or indirectly remains to be determined. However, our results show that the ovary is acting humorally to limit JH synthesis, supporting the hypothesis that it is doing so by production of ecdysterone. Engelmann's observation, in another cockroach species, that either implanted prothoracic glands or injection of the hormone ecdysone inhibit both egg maturation and increase in volume of CA (*16*), is consistent with this hypothesis. Ecdysone from mosquito ovary has been shown to control production of vitellogenin (*17*); the fly ovary is thought to produce an oostatic hormone (*18*). The demonstration of an endocrine function for the cockroach ovary makes it likely that the endocrine functions of insect ovaries are more widespread than previously thought.

BARBARA STAY
Department of Zoology
University of Iowa, Iowa City 52242
THOMAS FRIEDEL, STEPHEN S. TOBE
ERIC C. MUNDALL
Department of Zoology, University of Toronto, Toronto, Ontario, Canada

References and Notes

1. F. Engelmann, *Physiology of Insect Reproduction* (Pergamon, Oxford, 1970), pp. 148–168.
2. W. J. Bell, *J. Insect Physiol.* **15**, 1279 (1969).
3. V. J. Brookes, *Dev. Biol.* **20**, 459 (1969); M. L. Pan, W. J. Bell, W. H. Telfer, *Science* **165**, 393 (1969); W. J. Bell and R. H. Barth, *Nature (London) New Biol.* **230**, 220 (1971); R. Wilhelm and M. Lüscher, *J. Insect Physiol.* **20**, 1887 (1974).
4. S. S. Tobe and B. Stay, *Gen. Comp. Endocrinol.* **31**, 138 (1977).
5. J. G. Kunkel and M. L. Pan, *Am. Zool.* **17**, 914 (1977); F. Engelmann, *Insect Biochem.* **8**, 149 (1978).
6. B. Stay and S. S. Tobe, *Gen. Comp. Endocrinol.* **34**, 376 (1978).
7. S. S. Tobe and B. Stay, *ibid.*, in press.
8. CA were inserted through an incision in the neck; ovaries were injected into the abdomen. Animals were anesthetized with cold and treated postoperatively with a few crystals of streptomycin.
9. E. C. Mundall, S. S. Tobe, B. Stay, *Nature (London)* **282**, 97 (1979).
10. S. S. Tobe, A. Musters, B. Stay, *Physiol. Entomol.* **4**, 79 (1979).
11. C_{16}JH is methyl (2*E*,6*E*)-(10*R*)-10,11-epoxy-3,7,11-trimethyl-2,6-dodecadienoate. For this in vitro radiochemical assay, individual pairs of CA were incubated for 3 hours at 28°C in medium 199 (Hanks salts; with glutamine, Hepes buffer, 25 m*M*, *p*H 7.2; Gibco) plus Ficoll (20 mg/ml) to which [methyl-^{14}C]-methionine (Amersham/Searle, final specific radioactivity 35 to 39 mCi/mmole) was added. [see (*4*) and S. S. Tobe and G. E. Pratt, *J. Exp. Biol.* **62**, 611 (1975)].
12. S. S. Tobe and B. Stay, *Nature (London)* **281**, 481 (1979).
13. H. R. Hagan, *Psyche* **48**, 1 (1941); L. M. Roth and E. R. Willis, *ibid.* **62**, 55 (1955).
14. D. Bullière, F. Bullière, M. de Reggi, *Wilhelm Roux' Arch. Dev. Biol.* **186**, 103 (1979); M. M. Nihout and J. K. Koeppe, *Am. Zool.* **18**, 626 (1978).
15. Ecdysterone from Sigma was injected in 5 μl of medium 199 containing 5 percent ethanol; controls were injected with ethanolic medium only.
16. F. Engelmann, *Z. Vgl. Physiol.* **41**, 456 (1959).
17. H. H. Hagedorn, J. D. O'Conner, M. S. Fuchs, B. Sage, D. A. Schlaeger, M. D. Bohm, *Proc. Natl. Acad. Sci. U.S.A.* **72**, 3255 (1975); M. S. Fuchs, D. A. Schlaeger, C. Shroyer [*J. Exp. Zool.* **207**, 153 (1979)] found that ecdysterone, not ecdysone, controls vitellogenin synthesis and assume that a blood meal results in the conversion of ecdysone to ecdysterone.
18. T. S. Adams, A. M. Hintz, J. G. Pomonis *J. Insect Physiol.* **14**, 983 (1968).
19. Supported by USPHS grant AI 15230 and the Natural Sciences and Engineering Research Council of Canada.

1 October 1979

Eyeblinks and Visual Suppression

Abstract. *A technique for bypassing the eyelids permits equivalent visual stimulation of the retina before, during, or after a blink. Sensitivity to these stimuli decreases during voluntary blinks. This indicates that the source of the deficit is neural rather than optical. Such a visual loss may help to explain the common experience that most blinks go unnoticed.*

Under normal conditions of vision, eyeblinks occur every few seconds (*1–3*), each blink producing an almost total interruption of light and pattern reaching the eye. It is a surprising feature of visual perception that the interruption of vision typically goes unnoticed, and the subjective visual world remains continuous and stable. The effect is even more surprising when one considers the magnitude and duration of this interruption. Consider the voluntary blink diagramed in Fig. 1A. As the upper lid drops over the pupil, it severely attenuates the light reaching the eye (*4*) and wipes out nearly all visual information in the form of contour and contrast. The time during which the pupil is completely obscured is relatively long: 110 msec in the example shown (*5, 6*). Yet the blink is scarcely noticed. By contrast, in the absence of a blink, a much briefer interruption of room lights has a pronounced visual effect (*3, 7*).

The observation that the perceptual effect of a blink is small with respect to the

0036-8075/80/0222-0900$00.50/0

changes that it actually produces on the retina suggests that the brain generates an inhibitory signal that accompanies the blink and diminishes the effect of the blink on the visual system. Such a neural inhibition has already been shown in the case of saccadic suppression, the reduction of visual sensitivity that accompanies saccadic eye movements (*8*).

If a neural inhibitory mechanism operates during blinks, its properties can be assessed by measuring visual sensitivity under special experimental conditions such that closing the eyes has no effect on the amount of light reaching the retina. Accordingly, we have bypassed the normal optical path through the pupil and optic media. We placed a fiber-optic bundle against the roof of the mouth and used it to deliver light to a region directly below the right eye, stimulating the retina through the back of the eyeball. Opaque goggles eliminated all other sources of light, and the dark-adapted subject saw a diffuse cloud of light located over a large portion of the upper temporal visual field, that is, the lower nasal retina.

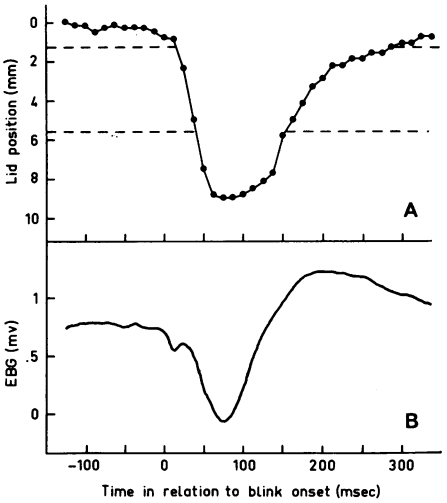

Fig. 1. Upper eyelid movement and electrical signal of a typical blink. (A) Curve based on a motion picture of the eye taken at 80 frames per second. It shows the position of the advancing edge of the upper lid, relative to its open resting position, as it executes a voluntary blink. The dashed lines represent the upper and lower margins of the pupil, also relative to the position of the upper lid at rest. Successive points represent samples of the blink taken at 12.5-msec intervals. (B) Corresponding electromyographic signal (EBG), recorded between electrodes placed above and below the eye. The EBG was displayed on a storage oscilloscope and photographed along with the blinking eye. Blink onset (time 0) is the time at which the trigger detected the blink. The sensitivity was set to detect the blink before the upper lid began to cover the pupil. For the blink shown here, the lid closed maximally about 140 msec after the time it began to move. The pupil was completely covered about 40 msec after detection of the blink and remained covered for 110 msec. Total blink duration was about 350 msec.

The stimulus event was a brief decrement of the otherwise steady, diffuse light. On a given trial, the decrement occurred at one of a number of possible times before, during, or after a voluntary blink. To detect the blink, we placed standard skin electrodes above and below the eye. A large electromyographic response wave appeared between these electrodes during the closing and opening of the lids. We call this wave the "electroblepharogram" (EBG) (Fig. 1B). The EBG was used to monitor blinks and was differentiated to activate trigger and delay circuits to present the stimulus decrement at preset times during or after a blink. To stimulate the retina just before a blink, the decrement was presented manually by the experimenter just after a signal to the subject to execute the blink. The exact time interval between the stimulus decrement and the blink onset was read from a digital clock. A sufficient number of intervals were sampled to analyze temporal changes in visual sensitivity associated with the blink.

Our experiments were conducted with two highly practiced adult male subjects, according to a constant stimulus method and a two-alternative forced-choice response paradigm; on each trial, the subject executed two voluntary blinks and judged which one was accompanied by the stimulus decrement. Across trials, the order of presentation was randomized, as were the principal stimulus variables: amplitude of decrement and time of decrement in relation to the onset of the blink. Such procedures are characterized by lack of bias.

For each time of occurrence of the decrement in relation to blink onset, the experiment yielded a curve relating the proportion of stimuli detected to the amplitude of the decrement. We corrected these proportions for chance success (50 percent), transformed them to a normal probability scale, and performed linear regression analyses to arrive at a threshold. We defined threshold as the particular size of decrement that would yield 75 percent correct detection (that is, halfway between chance and complete success).

Figure 2 shows log sensitivity (the log of the reciprocal of the threshold) for each of the designated times of occurrence of the decrement in relation to blink onset. During blinks there was a pronounced loss of visual sensitivity. For both subjects, the loss began before blink onset and reached a maximum before the lid covered the pupil. That maximum was about 0.4 log unit for subject L.A.R. and 0.5 or 0.7 for subject W.J.D., relative to the sensitivity of the steady eye (closed or open). Sensitivity did not fully recover until about 200 msec after blink onset.

The magnitude and time course of visual suppression during blinks are in close agreement with comparable measurements for voluntary saccades (*8*). Thus, the question arises whether saccades might be so closely associated with blinks that blink suppression can be explained by saccadic suppression. We have conducted preliminary observations of eye movements during blinks (*9*, *10*), with results in substantial agreement with those of Lawson (*6*), Ginsborg and Maurice (*10*), and Doane (*11*). They show no evidence of saccades, and no evidence of Bell's phenomenon (the upward rotation of the eyes when closed as in sleep). They support Doane's conclusion that a small inward rotation of the globe results from a lateral motion of the lower lid, and a small downward rotation results from the pressure of the upper lid. They also show that the extent and direction of these small movements depend on the primary position of the eyes in the head as the blink occurs. Thus, these eye movements are not of the type that could account for visual suppression during blinks.

A remaining question concerns the generality of blink suppression to spontaneous, and perhaps also to reflex blinks, in addition to voluntary ones (*1*-

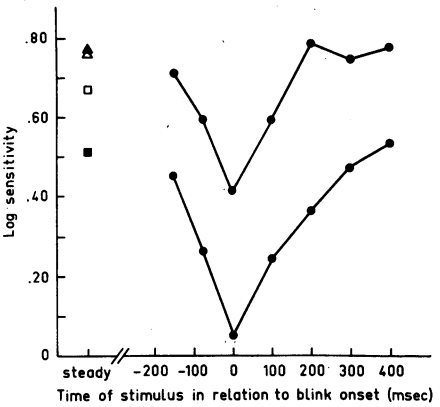

Fig. 2. Visual sensitivity to the decremental stimulus as a function of the temporal relation of the stimulus to the onset of a blink, for two subjects. At the left of each function, steady eyes-open (open symbols) and eyes-closed (closed symbols) control conditions for each subject are shown. Upper function and triangles, subject L.A.R.; lower function and squares, subject W.J.D. Stimulus duration was 20 msec for L.A.R. and 30 msec for W.J.D. Each plotted point was derived from a psychophysical function containing five or six values of decrement, each value presented approximately 40 times at each temporal relation to blink onset.

3). Others have concluded that whereas spontaneous blinks are highly variable and sometimes incomplete (*11*), they do not differ objectively from voluntary blinks (*2*). Our own preliminary observations confirm this conclusion (*12*) and support the extension of our findings to spontaneous blinks. Of still greater importance is the fact that we have already verified (*3, 7*) that voluntary blinks, like spontaneous ones, cause subjective interruptions of the visual scene that are much smaller than objective measurements would predict.

We conclude that the decrease in visual sensitivity that we measured cannot be attributed to optical factors. We attribute this decrease to a neural inhibitory mechanism in the brain. This mechanism, by decreasing the perceptual effect of the blink, contributes to the continuity of vision.

FRANCES C. VOLKMANN
Department of Psychology,
Smith College,
Northampton, Massachusetts 01063
LORRIN A. RIGGS
ROBERT K. MOORE
Hunter Laboratory of Psychology,
Brown University,
Providence, Rhode Island 02912

References and Notes

1. Blinking is a temporary closure of both eyes that is under both voluntary and involuntary control. It has been categorized into three types: (i) voluntary blinks, which can be executed on external or internal command; (ii) spontaneous or periodic blinks, which are involuntary, centrally programmed, and which constitute most of normal blinking; and (iii) reflex blinks, which are produced involuntarily in response to peripheral stimulation, such as a foreign body approaching or touching the eye (*2, 3*).
2. W. K. McEwen and E. K. Goodner, in *The Eye*, vol. 3, *Muscular Mechanisms*, H. Davson, Ed. (Academic Press, New York, 1969).
3. R. A. Moses, in *Adler's Physiology of the Eye*, R. A. Moses, Ed. (Mosby, St. Louis, 1975).
4. We have assessed the light attenuation due to the eyelids by measuring absolute visual thresholds to 0.2-sec flashes in fully dark-adapted eyes. Using full-field (Ganzfeld) conditions, the thresholds for eyes closed were 1.8 to 2.0 log units higher than for eyes open.
5. Investigators have correctly distinguished between blink duration, which is typically reported to be 300 to 400 msec, and blackout duration, the time during which the upper lid covers the pupil, which is reported to range from 40 to 200 msec [R. W. Lawson, *Nature (London)* **162**, 531 (1948); A. T. Slater-Hammel, *Res. Q. Am. Assoc. Health Phys. Educ. Recreat.* **24**, 363 (1953)].
6. R. W. Lawson, *Nature (London)* **161**, 154 (1948).
7. We have confirmed this observation and have found that subjects confidently match the visual effect of a blink with that of a much briefer and shallower decremental pulse in the illumination of a visual field.
8. See, for example, F. C. Volkmann, L. A. Riggs, R. K. Moore, and K. D. White [in *Eye Movements and the Higher Psychological Functions*, J. W. Senders, D. A. Fisher, R. A. Monty, Eds. (Erlbaum, Hillsdale, N.J., 1978), p. 35]. We view this neural inhibition as a corollary discharge such as that described by R. W. Sperry [*J. Comp. Physiol. Psychol.* **43**, 482 (1950)]. Under certain conditions, saccades may also elicit a retinal component of visual impairment such as optical smear or masking of contours. These factors are not relevant to the suppression during blinks in the present experiments.
9. Our observations involved two techniques. First, we used a double Purkinje image eye tracker to measure the time during which the pupil was actually obscured during a blink and to indicate whether any movements occurred before the lids cut off the input to the tracker and after the lids reopened [T. N. Cornsweet and H. D. Crane, *J. Opt. Soc. Am.* **63**, 921 (1973)]. A second technique, modeled after that of Ginsborg and Maurice (*10*), involved subjective observations of a small spot of light which could be made to move either horizontally or vertically across a large field. When an observer succeeded in blinking just as the spot crossed the fovea, the trace of the spot appeared to be deflected, and the subjective deflection could be used to estimate the speed, duration, and extent of the eye movement which produced it. We estimate that just before a normal blink there is an inward and downward motion of the eyes of about 0.3 deg arc.
10. B. L. Ginsborg, *Nature (London)* **179**, 412 (1952); ____ and D. M. Maurice, *Br. J. Ophthalmol.* **43**, 435 (1959).
11. M. G. Doane, *Invest. Ophthalmol. Visual Sci.* (Suppl.), p. 188 (1978).
12. In two subjects, neither the form of the blink as reflected in the EBG, nor the mean duration of pupil covering as measured on the eye tracker (*9*) was markedly different for voluntary and spontaneous blinks.

13. These experiments were conducted at Brown University, supported by grant EY 00744 from the National Eye Institute. We thank K. D. White, W. J. Donovan, K. Fuld, and J. Volkmann for their contributions. Preliminary reports of portions of this research were presented at the 1978 meeting of the Optical Society of America and at the 1979 meeting of the Association for Research in Vision and Ophthalmology.

17 September 1979; revised 6 December 1979

Sparing of the Brain in Neonatal Undernutrition: Amino Acid Transport and Incorporation into Brain and Muscle

Abstract. *Rates of tyrosine and lysine transport and incorporation into protein were measured in control and undernourished weanling rats. Undernutrition was induced by feeding lactating dams a low protein diet (12 percent casein) from birth to day 21. At weaning, body and brain weights of undernourished rats were 50 percent and 88 percent, respectively, of control values. Lysine and tyrosine transport rates into skeletal muscle were reduced by over 75 percent, more than twice the reduction seen in brain. Rates of amino acid incorporation into muscle protein were reduced by approximately 50 percent; the change in rate of incorporation into brain protein was not statistically significant. These data indicate that, in spite of marked retardation of amino acid transport into brain, the brain seems fully capable of maintaining normal rates of protein synthesis.*

Nutritional deprivation during critical periods of development in early life has profound and persistent effects on the body and brain (*1*). The fact that the growth impairment of the brain is smaller than that of the body as a whole has led to the concept of brain "sparing" (*2*). We have studied the mechanisms by which the brain is spared by measuring the transport of two amino acids, tyrosine and lysine, into brain and skeletal muscle and their incorporation into tissue protein. Both activities were sharply reduced in skeletal muscle of undernourished animals. In brain, the reduction of amino acid transport was about half that seen in muscle, yet the reduction in incorporation into brain protein was not statistically significant.

Female sperm-positive rats (*3*) were caged individually and housed in a temperature-controlled room with a 12 hour light and 12 hour dark (0900 to 2100 hours) diurnal cycle. The animals were fed a normal protein diet containing 25 percent casein (*4*) throughout gestation. At birth, litter size was made uniform by randomly distributing the rat pups among the lactating females, eight per litter. During lactation, experimental mothers were fed a low protein diet containing 12 percent casein (*4*), whereas control mothers continued on the 25 percent casein diet. The low protein intake reduces the volume of milk without altering its composition (*5*). Thus, throughout lactation the experimental pups were subjected to undernutrition by receiving suboptimal amounts of a diet of normal composition; during the third week of

Table 1. Body and brain weights of control and undernourished rats. The data are expressed as means ± standard error ($N = 30$).

Tissue	Weight (g)		Percentage decrease*
	Control	Experimental	
Body	60.6 ± 1.13	30.4 ± 0.93	49.8
Whole brain	1.438 ± 0.0166	1.267 ± 0.0117	11.9
Forebrain	1.128 ± 0.0121	1.009 ± 0.0105	10.5
Brainstem	0.128 ± 0.0049	0.115 ± 0.0030	10.3
Cerebellum	0.181 ± 0.0053	0.142 ± 0.0042	21.5

*$P < .001$, one-tailed t-test.

0036-8075/80/0222-0902$00.50/0

Eyeblinks and visual suppression
FC Volkmann, LA Riggs and RK Moore (February 22, 1980)
Science **207** (4433), 900-902. [doi: 10.1126/science.7355270]

Editor's Summary

Science (print ISSN 0036-8075; online ISSN 1095-9203) is published weekly, except the last week in December, by the American Association for the Advancement of Science, 1200 New York Avenue NW, Washington, DC 20005.