The results given in the following paragraph support the conclusion that at least one other sex pheromone is present in *Cadra* females and is necessary to elicit a normal sex stimulatory response in *Cadra* males.

Another compound purified by GLC, isolated from tips of nonmated *Cadra* females, elicits no apparent behavioral activity in *Cadra* males. Yet this compound, combined in the natural ratio with the behaviorally active sex pheromone, causes a definite increase in orientation of males. As compared with extracts of *Cadra* females, the combined compounds effect the same degree of orientation and copulatory behavior in *Cadra* males observed. This is the first demonstration in a lepidopteran species of a second compound isolated from females that is inactive by itself, but is synergistic with a sex pheromone. This inactive compound may be a factor in isolating closely related species.

Attractancy tests were conducted as described in Table 2 and also as follows. About 5:00 p.m., 3 hours after nonmated males (2 days old) were released into the room, Stikem-coated traps were charged with test material on filter paper and an air stream was directed into each trap. Lights were then turned off, and captures were recorded next morning. The attracting property (as distinguished from the sexual stimulatory property) of compound **1** to *Plodia* males is the same as that of the active pheromones isolated from *Plodia* and *Cadra* females (Table 2), and is similar to but slightly less effective than that of crude extracts and live *Plodia* females. Initial results with single and combined fractions of a thin-layer chromatogram of an extract of *Plodia* females indicate the presence of a compound (or compounds) that slightly synergizes attraction to the behaviorally active sex pheromone. Compound **1** is as attractive to *Cadra* males as the active compound from *Cadra* females (Table 2). However, either compound is considerably less attractive to male *Cadra* than crude extracts of female *Cadra* containing about equal amounts of the behaviorally active sex pheromone.

We emphasize the distinction between our bioassays for sexual stimulatory response and for attraction. This distinction has unfortunately been ignored in many studies.

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Synaptic Adjustment after Deafferentation of the Superior Colliculus of the Rat

Abstract. *Eyes were removed from rats shortly after birth, when there are few formed synapses in the colliculus. It was found that synaptogenesis continues to give a near-normal ratio of terminals containing either spheroidal or flattened vesicles. After eye removal in adult rats, however, reinvasion of synaptic sites vacated by degenerate optic terminals occurs, with an incomplete return toward a normal proportion of synaptic types.*

Previous work has indicated that removal of afferent nerve fibers to a region of the central nervous system can cause compensatory sprouting of other pathways projecting to the region (*1*). The object of this study has been to investigate the degree of synaptic adjustment in the upper layers of the superior colliculus of the rat after removal of their major input—the input from the retina. Three experimental situations have been studied. In the first, one or both eyes were removed from adult animals, in which the normal synaptic patterns of the superior colliculus are well established. In the second situation, one or both eyes were removed from animals within the first postnatal day. At this time very few synapses of any kind have been formed in the colliculus. In the third case, eyes were removed at intermediate ages: 10, 14, and 24 days postnatal. At these times the full synaptic pattern has not yet been established, the main formation being within the 2-week period after the eyes are opened at 14 days postnatal.

Most of the animals were allowed to survive for 4 to 5 months; some of the enucleated adults were killed after 14 postoperative days. Of the animals with unilateral enucleation at birth, a few had second lesions as adults, either of the cortex or remaining eye, to see how much synaptic reorganization might be due to compensatory sprouting of the corticotectal or uncrossed retinotectal pathways. Comparison material for light microscopy from each series was obtained and stained with Fink and Heimer, neurofibrillar, and Nissl methods. All animals were perfused with paraformaldehyde and glutaraldehyde in phosphate buffer (*2*).

Attention is particularly directed to four main features: (i) synaptic vesicle morphology, (ii) serial synapses, (iii) morphology of synaptic contacts, and (iv) occurrence of synapses where the presynaptic element is dendritic. From previous work (*3*), in which buffered aldehyde fixatives of appropriate osmolarity were used, two main populations of terminals can be recognized; they contain either spheroidal synaptic vesicles (S terminals) or predominantly flattened vesicles (F terminals) (see Fig. 1a). With regard to serial synapses (where one terminal is presynaptic to another), the postsynaptic element in the superior colliculus always contains flattened vesicles, but the presynaptic element may be either an S or an F terminal. A synaptic density may have a pronounced postsynaptic thickening (asymmetric contact), or this may be absent (symmetric contact). Presynaptic dendritic profiles occur in the normal superior colliculus; they form F terminals with symmetric contacts.

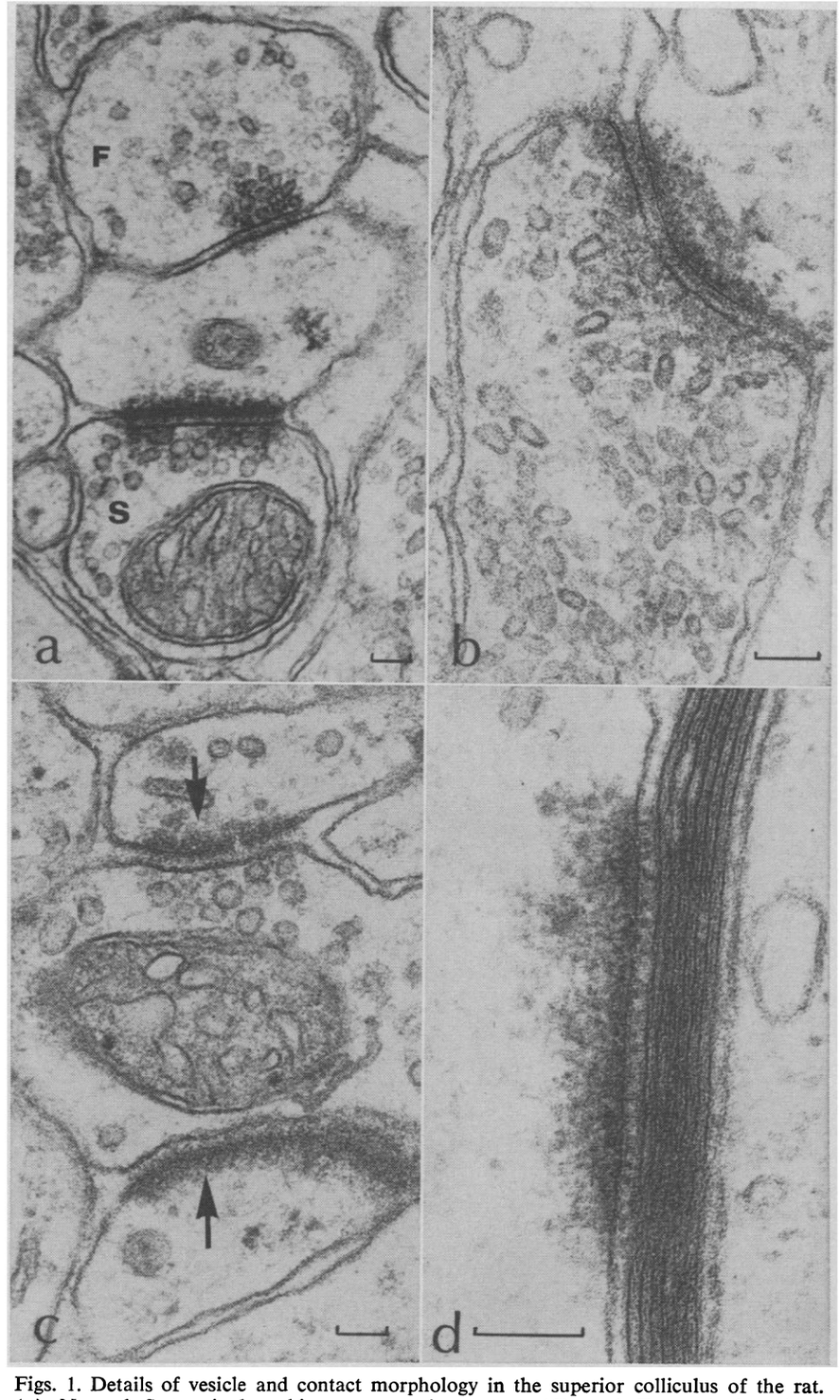
Counts were taken of synaptic contact features from electron micrographs or by direct observation of electron microscope sections. The results are summarized in Table 1. Each of the experimental groups contained at least three animals, and, for each animal, counts were taken from several blocks of tissue. Each count was from a column extending from stratum zonale to the superficial region of the stratum

opticum. The tissue blocks were generally taken from the same central region of the colliculus. However, in some normal and experimental animals, tissue was taken from each quadrant. In the normal animals, there was no significant difference between the counts taken from each quadrant; in experimental animals, the only difference detected could be accounted for by the larger number of uncrossed optic terminals in the anteromedial region. Analyses of variance for unequal sample sizes were made, and the means were compared by the Newman-Keuls method. Each of the percentages of S to F contacts differs significantly at the .05 level from each of the others; the experimental groups in column 2 are significantly different ($P < .05$) from the normal. Of the F terminals forming asymmetric contacts, the adult enucleation group and the group enucleated at 10 to 24 days of age differ significantly ($P < .05$) from each other and from the other two groups.

In the normal tissue, the percentages shown in Table 1 remain quite consistent from one animal to another, with the exception of the proportion of F terminals forming asymmetric contacts. This can vary from 0 to 4 percent in individual counts, each of about 100 F contacts. The optic terminals (recognized by comparison with their early degenerative changes in operated material) account for about 75 percent of the S contacts. They commonly make asymmetric contacts and contain large pale mitochondria. This last feature, found also in optic terminals in the lateral geniculate nucleus (*4*), is not shared by any other terminal of the superior colliculus.

By 14 days after enucleation in an adult, there is little indication that any normal optic endings remain, except for a very small number of uncrossed optic terminals lying in the anteromedial region. At this survival time, there are many degenerating terminals present, as well as a considerable number of asymmetric postsynaptic sites with no presynaptic terminals contacting them. In addition, cases have been observed of occupation of an asymmetric postsynaptic site partly by an F terminal and partly by a degenerate terminal, an astrocytic process, or an S terminal (see Fig. 2). This and the high F asymmetric population (Table 1 and Fig. 1b) found after 14 days strongly suggests that F terminals (and perhaps S terminals also) can take over synaptic sites formerly occupied by optic terminals either directly or after a temporary glial occupation of the site (*3*). From these findings it would appear that the low ratio of S to F contacts is due largely to removal of the optic input but is due also, in part, to a population of F terminals invading asymmetric contact sites. We could find no supporting evidence for an alternative hypothesis that spheroidal vesicles may become flattened.

With survival times of 5 months after enucleation in adult rats, some degenerating terminals persist, as do some unoccupied postsynaptic sites still with the presynaptic membrane opposed. The ratio of S to F contacts is increased



Figs. 1. Details of vesicle and contact morphology in the superior colliculus of the rat. (a) Normal S terminal making asymmetric contact (*S*) and F terminal making symmetric contact (*F*). (b) An F terminal making asymmetric contact. (c) An S terminal making two asymmetric contacts (arrows); only one contact has vesicles aggregated to it. (d) Myelin lamellae making asymmetric contact. Scale indicates 0.1 micrometer.

Table 1. Effects of enucleation at different ages on synaptic patterns in the superior colliculus.

Animal group	No. of terminal contacts counted	Terminal contacts (%S/%F)	S terminals forming serial synapses (%)	F terminals forming serial synapses (%)	F terminals forming asymmetric contacts (%)
Normal adult	3151	61/39	10	3	2
Enucleation as adult					
Survival for 14 days	1097	26/74	4	2	12
Long survival	2061	36/64	3	3	9
Enucleation at 10 to 24 days old with long survival	1186	45/55	2	3	16
Enucleation at birth with long survival	1929	56/44	2	2	5

compared with the ratio after 14 days' survival, but it is still below that for a normal animal. With area and shrinkage taken into account, this appears to be due to a slight increase in the S population rather than a reduction in the F population. The proportion of F terminals with asymmetric contacts remains unchanged compared with the short survival time, as does the proportion of S contacts forming serial synapses.

In animals 4 months old which had undergone eye removal at birth, the ratio of S to F contacts is only slightly lower than in a normal animal, the number of F terminals making asymmetric contacts is slightly higher than normal, and the proportion of serial synapses formed by S contacts is lower than normal.

When a second lesion is made in these animals, either of the remaining eye or of the visual cortex, light microscopy of the resulting degeneration shows an extension from the normal area of projection of these pathways. The uncrossed retinotectal pathway spreads across the entire colliculus instead of being confined to the small anteromedial region, and the corticotectal pathway extends to the surface of the colliculus instead of occupying the deeper half of the stratum griseum superficiale. With the electron microscope these degenerating uncrossed retinotectal and corticotectal terminals can be identified; the two pathways together account for no more than 15 percent of the S contacts in the central region of the colliculus. The extension of the uncrossed pathway has not been observed in enucleated adult rats after a long-term survival (*5*).

In the animals with eyes removed at 10, 14, and 24 postnatal days which were allowed to survive for 4 months, the ratio of S to F contacts fell to a value between that of animals operated as adults and at birth.

Dendrodendritic synapses were found in all animals—normal and operated at any age. In the operated animals, they may, however, make asymmetric synaptic contacts. Two further features common to all operated animals but extremely rare in normal animals are (i) the occurrence of terminals in which one of several asymmetric contacts made has no vesicles oriented toward it (Fig. 1c) and (ii) the occurrence of myelinated axons that make contacts, show cleft filaments, and have an asymmetric "post-synaptic" density (Fig. 1d). The first finding is particularly obvious with the S terminal population in long-survival animals operated at 14 days or older, but some of the F terminals that make asymmetric contacts also have no vesicles specifically oriented to the contact. Several reports (*6*) have implicated the aggregation of vesicles adjacent to the synaptic membrane with the transmission process, and this last finding would by comparison imply a failure of transmission. The finding of myelinated axons contacting asymmetric sites is occasional and accounts for less than 1 percent of all contacts with the exception of those animals operated at 14 and 24 days postnatal, where it amounts to between 1 and 2 percent of all contacts. These two findings, as well as the observation that the features of an asymmetric contact may be maintained for long periods of time after a lesion with only remnants of the presynaptic membrane remaining, indicate that the maintenance of an asymmetric contact site does not necessarily depend on the transmission process.

This study shows that adjustment of remaining synaptic patterns occurs after removal of a group of terminals within a region. Such adjustment takes the form of a partial restoration to the normal value of the ratio of S to F contacts. This value is most closely approached when the lesion is made at birth, at a time when few synapses are yet formed.

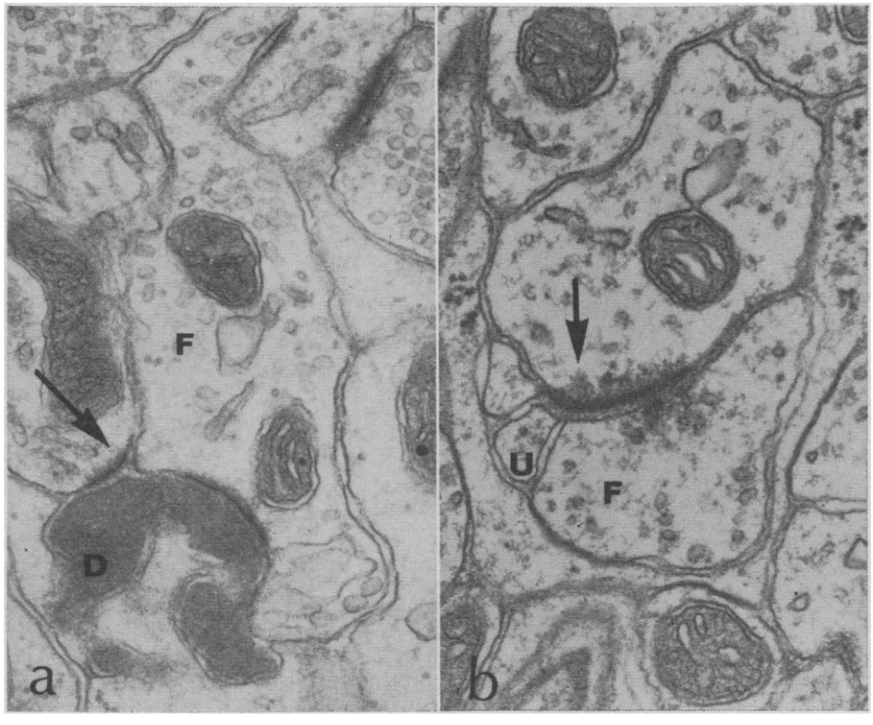


Fig. 2. Partial occupation of postsynaptic asymmetric contacts. (a) The contact (arrow) has a degenerate terminal (*D*) and an F terminal (*F*) adjacent to it. (b) The contact (arrow) is shared by an F terminal (*F*) and an unidentified profile (*U*). Magnification × 50,000.

In such animals this restoration is not due solely to a sprouting of the uncrossed optic or corticotectal pathways, because this process accounts for no more than 15 percent of the S contacts. It is suggested that the near-normal ratio occurring after eye removal at birth may result from a regulated development such that the number of F contacts formed closely parallels the number of S contacts formed in a fixed ratio, even though the S terminals may be of different type and proportion from normal. The results from lesions at times when synaptogenesis is still incomplete (10 to 24 days postnatal) indicate a ratio intermediate between that produced by lesions in newborn and adult animals. This suggests that, once a group of S terminals is removed, the F contacts that had developed secondarily to it remain and are not available to complement S contacts formed later, and thus the normal ratio cannot be restored.

Although the significance of such ratios is unclear, different regions of the brain have been shown to have characteristic but different ratios of S to F contacts (*7, 8*). The ratios may reflect a balance of extrinsic and intrinsic contacts or of excitatory and inhibitory contacts (*9*).

In addition to this developmental regulation, a second, reorganizational, process can occur. Synaptic sites that have already been formed may become available for reinnervation by other terminals, once their normal presynaptic component has been removed. The most obvious example of this process is the increased number of F terminals making asymmetric contacts, a feature that appears within 2 weeks after the lesion is made and persists for long periods. It is possible that the slower increase in the number of S contacts after a lesion in an adult is also the result of invasion of unoccupied sites.

Considerable numbers of F terminals making asymmetric contacts have also been reported 2 weeks after specific deafferentation in the trigeminal system (*7*), as well as in normal cerebellum (*10*). The cerebellum differs from the colliculus in that there is considerable synaptic reorganization during development (*11*). In the superior colliculus it appears that, during normal development, F terminals are unable to "induce" asymmetric contacts. It would be of value to know whether this is generally true throughout the nervous system and whether the presence of F terminals making asymmetric contacts indicates the previous removal of an S terminal from these sites, either as a result of damage to S axons or as a part of a normal developmental process. We suggest that the 2 percent of F terminals associated with asymmetric contacts found in the normal adult colliculus are due to developmental accidents to S terminals. The patchy occurrence of these F asymmetric terminals in normal tissue would tend to support this suggestion.

The study does not include such problems as changes in cell populations or cell morphology after long-term deafferentation, or the possibilities of reorganization within the S population, or different reactions of the various subdivisions of the S and F population. With more extensive investigation, these factors may provide considerable information as to the reorganization possible in neuronal systems.

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Eye Lens Color: Formation and Function

Abstract. *Aromatic amino acids are photooxidized by near-ultraviolet light to colored products that are bound very tightly to protein amino groups. The resulting colored proteins absorb near-ultraviolet light more strongly and are rendered more hydrophobic than the untreated compounds, and they fluoresce at 440 nanometers when excited at 360 nanometers. Coloration in the lenses of diurnally active animals (including man) may be caused by this reaction, and senile cataracts may result. Such changes in many other proteins (as in the skin and retina) could lead to more serious consequences.*

In the eyes of most diurnally active animals (for example, geckos, snakes, tree shrews, squirrels, monkeys, and man) the lenses are various shades of yellow, amber, and brown, whereas in many nocturnally active animals (including rats, rabbits, guinea pigs, and most fishes) they are colorless (*1*). I report here a possible chemical basis for lens coloration and indicate what function coloration may have.

Many aromatic compounds, such as the amino acids phenylalanine, tryptophan, and tyrosine, become colored when they are exposed to a sufficient dosage of near-ultraviolet light aerobically or in a vacuum. This reaction, which causes these compounds to become yellow, brown, or black, respectively, appears to be a photooxidation of the aromatic rings to quinones. The idea that these reactions are oxidation reactions is supported by the finding that reduced glutathione or ascorbic acid (at 0.001*M* excess) markedly inhibits the formation of the yellow or brown photoproducts of tryptophan and *p*-aminobenzoic acid (PABA) when 0.006*M* or 0.007*M* solutions of these compounds are irradiated for 18 hours with 4000 μw/cm^2 of light (wavelength, 340 to 380 nm) at 20°C. (An Ultra-Violet Products PCQ 008L photochemical lamp was used for all of the irradiation experiments described.)

When the lenses of dogfish (*Mustelus canis*) eyes were incubated for 2 days under the same irradiation conditions in elasmobranch Ringer solutions containing 0.1 percent tryptophan or PABA, the normally uncolored lenses became yellow or brown but they remained transparent. The degree of absorption of light at 365 nm by the normal control lenses, lenses treated with photooxidized PABA [see (*2*)], and lenses treated with photooxidized tryptophan is shown in Table 1.

Transmitted light at 365 nm was measured with an Ultra-Violet Prod-