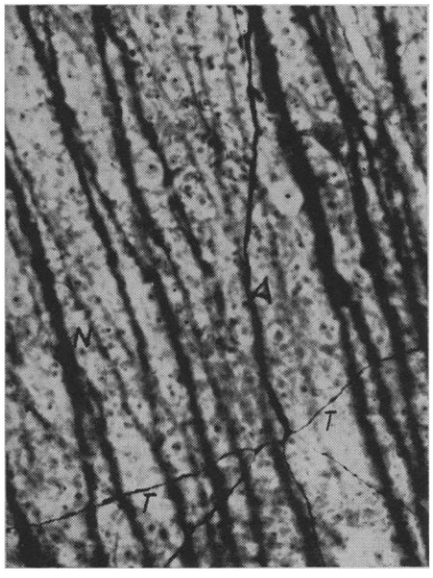

Fig. 2. Dog's retina. *A*, axon of one associational cell of the ganglion cell layer; *t*, terminal branches; *n*, optic nerve fibers. Unretouched photomicrograph.

its division into terminal branches; and he supposed that the axon was joined to the optic nerve fibers leading to the centers. His description does not exactly match that of the cells we have described. He states that the axon puts out branches over its entire length. In our stainings the salient characteristic of the type of cells described is the lack of collaterals in the segment between the "recurrent" ones and the sudden division into several branches. The reason for this interpretation probably is that he had seen only the segment of the axon close to the cell body where it puts out the "recurrent" collaterals. The sections made from retinas stained by means of the Golgi method—even by Cajal's "enroulement" technique—do not permit us to follow the axon and its terminal branches over the whole extent of their distribution in the retina.

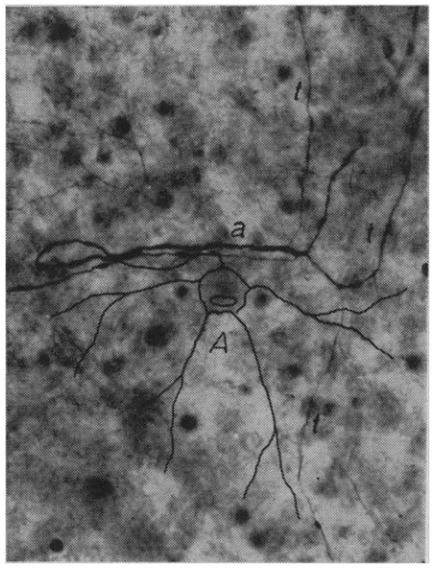

Fig. 3. Human retina. *A*, Associational cell of the ganglion cell layer; *a*, axon; *t*, terminal branches. The body and dendrites, being out of focus, have been drawn.

The area of the retina covered by the axon and terminal branches of these cells is very large (Fig. 1). We observed the cells in the peripheral part of the retina. At the moment it is practically impossible to ascertain how numerous they are. As far as we can judge from the stainings we have studied, we can only guess that they are present in a proportion of less than 1 percent of the giant ganglion cells.

When studying a retina we can see in practically all of it, by means of our technique. A great number of fibers of different thicknesses cross the bundles of the optic nerve fibers in several directions. There is no doubt that some of the fibers are the terminal branches of these new cells, but there also exist others, besides the centrifugal fibers, whose origin has not yet been identified. Hence a more complex system of intraretinal association than had been hitherto supposed may exist.

In the retina several types of cells have been regarded (*9*) as elements of transverse intraretinal association. Such cells are found (i) at the level of the outer plexiform layer, and (ii) at the level of the inner plexiform layer. Cells in the outer layer are of two main types: the outer, small, horizontal cells, without a cylinder axis, forming a plexus that spreads all over the retina, and the inner, large, horizontal cells whose axon terminals end at some distance from the body but still within the outer plexiform layer. Cells in the inner layer are the several types of amacrine cells.

From a morphological point of view the cells we are describing look as if they were playing an associative role at the level of the second neuron: the ganglion cells. Through their dendrites they might be activated by excitation from the visual cells which connect to the bipolars in their "dendritic field," and by the impulses from their axons they might modify the responses of the ganglion cells located at some distance. It is not going to be simple to record the electrical responses of these cells because of the smaller number found in the retina and their similarity to the regular ganglion cells, but their presence must be borne in mind when the electrical responses obtained at the level of the ganglion cells or the optic nerve fibers are being interpreted.

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Anopheles balabacensis balabacensis Identified as Vector of Simian Malaria in Malaysia

Abstract. *The mosquito* Anopheles balabacensis balabacensis *has been identified as a natural vector of at least two species of simian malaria in the monsoon forests of the northern Malay States. This mosquito is also a serious vector of human malaria from Viet Nam to northern Malaya. This is the first report of a mosquito which transmits both human and simian malaria in nature.*

Natural vectors of simian malaria are known only from Malaysia, and all are members of the *Anopheles leucosphyrus* group of mosquitoes. Wharton and Eyles (*1*) identified *A. hackeri* as a vector of *Plasmodium knowlesi*, and this mosquito has since been found naturally infected with *P. cynomolgi*, *P. coatneyi*, and *P. fieldi*. In 1962 Wharton *et al.* (*2*) reported that *A. leucosphyrus* is a vector of *P. inui*, and Eyles *et al.* (*3*) found *A. balabacensis introlatus* naturally infected with *P. cynomolgi*. We now record the presence of both *P. cynomolgi* and *P. inui* in *A. b. balabacensis* in Malaysia.

The *A. leucosphyrus* group of mosquitoes is broadly distributed through

South Central and Southeast Asia. *Anopheles balabacensis balabacensis* is distributed from Indochina through Thailand and into the island of Borneo; it is considered to be a serious vector of human malaria in the inland hills of Viet Nam, Cambodia, Thailand, and northern Malaya (*4*).

Field studies of human and simian malaria were conducted in the monsoon forests of the state of Perlis in the northern Malay States in December 1964 and January 1965. The relative attraction of man and monkeys for anophelines in the area was determined by trapping mosquitoes in monkey-baited net traps on platforms in the forest and in human-baited net traps on the ground. Mosquitoes were collected every 2 hours from 6 p.m. until 6 a.m. and taken to a nearby field laboratory each morning, where they were identified and dissected. The salivary glands were dissected in serum-saline, and the sporozoites from positive specimens were inoculated intravenously into malaria-free rhesus monkeys.

Of 42 *A. b. balabacensis* dissected, five had sporozoites in their salivary glands. Sporozoites from three of the mosquitoes were inoculated into one monkey (R464), and sporozoites from a fourth were inoculated into another (R466); the material from the fifth infected mosquito was preserved for morphological studies. The two monkeys became positive for malaria 10 and 14 days after inoculation. The parasite in one (R464) reached a peak density of 685,000 per cubic millimeter on the 6th day of patency and was identified as *P. cynomolgi*. The second monkey (R466) developed a *P. inui* infection with a maximum parasitemia of 113,000 per cubic millimeter on the 11th day of patency.

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DNA-Dependent Synthesis of RNA Is Not Implicated in Growth Response of Chick Comb to Androgens

Abstract. *Local application of actinomycin D does not detectably modify growth response of the chick comb to androgens. The mode of action of androgens on chick comb (a connective-tissue organ) appears to differ from their action on epithelial-tissue organs such as seminal vesicles and prostate.*

Much recent experimental evidence indicates that estrogens (*1–4*) and androgens (*5*) exert their growth-promoting action on specific target tissues initially by stimulating synthesis of RNA. Kinetic studies show that this activation of RNA synthesis precedes in time the phase of increased synthesis of protein (enzyme) that leads to proliferation of the target tissue (*2*). It has also been demonstrated that inhibition by actinomycin D of DNA-dependent synthesis of RNA blocks the ultimate physiological and cytological manifestations of the effects of some of these steroid hormones (*3*). These observations have led to the postulate that stimulation of synthesis of RNA in the target tissue is an important and determining event in the mode of action of these hormones.

Available data on the action of androgens on prostate, seminal vesicles, and muscles seem to accord with this model (*6*). Another system has been known since the birth of endocrinology to depend on androgens. Berthold in 1849 reported regression of the rooster comb on castration and its reappearance after implantation of testes (*7*). Subsequent studies have shown that the weight and size of the comb are linearly related to the amount of androgen hormone directly applied to the comb of capon or chick (*8*); the response is so exactly proportional

Table 1. Effect of androgens on the growth of chick comb, with and without topically applied actinomycin D(AMD). The number of birds in each test follows (in parentheses) the test designation. AST, androsterone; TSP, testosterone propionate.

Treatment	Comb (mean ± S.E.)	
	Length + height (mm)	Weight/body wt (mg/g)
	Experiment 1	
(*a*, 6) 0.05 ml ethanol	11.67 ± 0.192	0.193 ± 0.041
(*b*, 5) 3 μg AST*	15.80 ± 0.970	0.643 ± 0.104
(*c*, 6) 0.25 μg AMD†, 3 μg AST*	15.50 ± 0.955	0.716 ± 0.112
	Experiment 2	
(*d*, 10) 0.05 ml ethanol	10.80 ± 0.291	0.180 ± 0.017
(*e*, 9) 1.14 μg TSP*	14.40 ± 0.821	0.640 ± 0.032
(*f*, 5) 0.3 μg AMD†, 1.14 μg TSP*	13.80 ± 0.313	0.895 ± 0.117
	Statistical significance	
Length + height: $a \sim b$, $P < .01$	Comb wt/body wt:	$a \sim b$, $P < .001$
$a \sim c$, $< .01$		$a \sim c$, $< .01$
$b \sim c$, $< .90$		$b \sim c$, $< .90$
$d \sim e$, $< .001$		$d \sim e$, $< .001$
$d \sim f$, $< .001$		$d \sim f$, $< .001$
$e \sim f$, $< .700$		$e \sim f$, $< .01$

* Applied daily. † Applied twice daily.

Table 2. Effect of actinomycin D (AMD, topically applied twice daily) on the growth of chick comb under the influence of endogenous androgns. The number of birds tested follows (in parentheses) the test designation.

Application	Comb (mean ± S.E.)		
	Length + height (mm)		Weight/body wt (mg/g)
	Day 15	Day 28	
	Experiment 1		
(*a*, 8) 0.05 ml saline	10.75 ± 0.36	21.10 ± 1.29	0.77 ± .09
(*b*, 8) 0.3 μg AMD	11.27 ± 0.83	21.87 ± 1.23	1.06 ± 0.17
	Experiment 2		
(*c*, 14) 0.1 ml saline		22.93 ± 1.35	0.63 ± .132
(*d*, 14) 0.2 μg AMD		21.30 ± 0.995	.54 ± .134
	Statistical significance		
Length + height: $a \sim b$, $P < .70$		Comb wt/body wt:	$a \sim$ b, $P < .20$
$c \sim d$, $< .40$			$c \sim$ d, $< .70$