found ecological effects. That this is currently happening is indicated by the sea otter–abalone controversy in California. A decrease in sport and commercial abalone fisheries has been reported following the influx of sea otters into areas of previously unoccupied habitat (*24*). Surveys conducted in 1967 by the California Department of Fish and Game revealed that throughout the sea otters' range preferred sea otter forage items were reduced in number and restricted to protected habitat as compared with habitat outside the range (*25*). Also, an increased diversity in sea otter forage items has been reported in areas long inhabited by sea otters. This is apparently the result of reduced availability of preferred sea otter forage items (*24*).

The sea otter may also be important in restoring kelp beds (and associated species of animals) in southern California. Sea otters in California completely remove large sea urchins (*Strongylocentrotus franciscanus*) from areas by predation, permitting luxuriant development of the *Nereocystis-Pterygophora* (brown algae) association (*4*). Recent increases in sea urchin populations are correlated with kelp bed reduction (*5*). Although kelp bed reductions are obviously related to phenomena more recent than the disappearance of sea otters (*26*), the reestablishment of sea otters should decrease invertebrate populations and increase vegetational biomass.

The sea otter is an important species in determining structures and dynamic relations within nearshore communities, and so fits Paine's (*27*) concept of a keystone species. Many changes have resulted from the near extinction of the sea otters in these communities during the 18th and 19th centuries. In modern biological studies of nearshore marine communities along the Pacific coast of North America the species' ecological importance has not been considered in sufficient detail. We believe that the sea otter is an evolutionary component essential to the integrity and stability of the ecosystem.

JAMES A. ESTES
Arizona Cooperative Wildlife Research Unit, University of Arizona, Tucson 85721

JOHN F. PALMISANO
College of Fisheries, University of Washington, Seattle 98195

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Puromycin: A Questionable Drug for Studying the Mechanism of Thyroid Calorigenesis in vivo

Abstract. *Puromycin fails to alter minimal oxygen consumption of rats treated with thyroxine, provided the rectal temperatures of these rats are maintained at 37.8° to 38.1°C. The previously reported puromycin-induced decline in basal metabolic rate of thyroxine-treated rats may have been due to the hypothermia produced by this drug.*

Thyroid hormone–induced alteration of the rate of protein synthesis is a familiar hypothesis proposed to explain the elevated consumption rate of O_2 observed after administration of thyroid hormones (*1, 2*). This hypothesis is based on the results of experiments that measured basal metabolic rate (BMR) before and after the use of inhibitors of protein synthesis (such as puromycin) in vivo (*1, 2*).

Because of the importance of this hypothesis, we tried to confirm the original findings (*2*) by using a new parameter—minimal oxygen consumption (MOC) (*3–6*). Among the 70 endocrine and nonendocrine factors studied, the MOC appears to measure changes in thyroid state more specifically than the BMR (*6*). Unlike various BMR methods, MOC is measured in sleeping or anesthetized rats, at their thermoneutral temperature (*3, 6*). Thermoneutrality is defined as the highest test chamber temperature that maintains a normal rectal temperature (37.8° to 38.1°C) (*3, 6*). Oxygen consumption was detected volumetrically with a precision-bore glass tube (*6*); a servosystem corrected for extraneous variations in ambient temperature and pressure (*4*). The MOC was expressed in

Table 1. Effect of puromycin on MOC (milliliters of O_2 consumed per minute per 100 g of fat-free body weight). Each value represents the mean ± standard deviation of five rats.

Dose of thyroxine	Minimal oxygen consumption	
	Before puromycin	After puromycin*
0	1.50 ± 0.10	1.52 ± 0.10
0.1 mg	1.73 ± 0.11	1.77 ± 0.07

* Puromycin (100 mg/kg) was administered in two divided doses, 15 and 60 minutes before MOC determination [see (2)].

Table 2. Effect of puromycin on BMR (milliliters of O_2 consumed per minute per 100 g of fat-free body weight) at different ambient temperatures. Each value represents the mean ± standard deviation of seven to eight rats; T_4, thyroxine.

Treatment	(a) Ambient temperature (22°C)		(b) Thermoneutrality*	
	BMR	Rectal temperature	BMR	Rectal temperature
T_4, 0; before puromycin	4.88 ± 0.76	37.8 ± 0.2	3.34 ± 0.16	37.8 ± 0.2
T_4, 0; after puromycin	2.84 ± 0.24†	33.5 ± 0.2	3.27 ± 0.19	37.9 ± 0.1
P	< .001	< .001	> .5	> .5
T_4, 1 mg; before puromycin	6.44 ± 0.76	38.0 ± 0.3	5.54 ± 0.36	37.9 ± 0.05
T_4, 1 mg; after puromycin	4.76 ± 0.40†	36.0 ± 0.2	6.09 ± 0.53	38.1 ± 0.1
P	< .001	< .001	> .5	> .5

* Defined in text. † Q_{10} corrected values given in text.

milliliters of O_2 at standard temperature and pressure per 100 g of fat-free body weight (*5*) per minute; surface area corrections were not used for reasons given elsewhere (*6*). All rats were fed a low iodine diet (*3, 6*). The effect of puromycin on MOC of young adult rats receiving thyroxine (T_4) (0.1 mg kg^{-1} day^{-1} for eight consecutive days) was determined.

Contrary to our expectations, puromycin failed to decrease the MOC of anesthetized hyperthyroid and euthyroid rats (Table 1). In order to resolve the contradiction between these results and previous findings (*2*), we duplicated the original procedure. Therefore, the BMR was measured at an ambient temperature of 22°C in unanesthetized 4-week-old rats previously treated with 1 mg of T_4 per kilogram per day for ten consecutive days. Under these conditions, the original findings appear to be confirmed (Table 2, column a); puromycin depresses the BMR. However, puromycin significantly lowers rectal temperature in all rats, but rectal temperature was not reported in earlier publications (*1, 2*). When rectal temperatures are maintained within the normal range of 37.8° to 38.1°C (*3, 6*), by controlling the temperature of the test chamber, puromycin has no effect on the BMR of hyperthyroid and euthyroid rats (Table 2, column b).

Depression of the BMR by puromycin was observed in our experiments only in those rats whose rectal temperatures were permitted to fall to subnormal values. We suggest that a similar decline in rectal temperature might have occurred in the original experiments (*2*) carried out at a subthermoneutral ambient temperature (22°C). Valid comparisons between most reaction rates including O_2 consumption rates at different reaction temperatures requires a Q_{10} correction for these temperature differences. In order to make this correction, we used 2.3 as the Q_{10} of the major exothermic reactions of the rat (*6, 7*). Rectal temperature was used as an approximate index of core temperature (*5, 8*). After Q_{10} correction of the BMR of rats with subnormal rectal temperatures (Table 2, column a), there is no significant decrease in the BMR following puromycin treatment (Q_{10} corrected oxygen consumption of euthyroid, 4.06 ± 0.38; hyperthyroid, 5.61 ± 0.43).

While the earlier hypothesis concerning a possible relation between the rate of protein synthesis and the calorigenic effect of thyroid hormones ultimately may be correct, the finding of the present experiments, that puromycin lowers rectal temperature, appears to cast some doubt on the validity of the experimental conditions originally used to test this hypothesis.

GLENDA E. BILDER
W. DONNER DENCKLA
Roche Institute of Molecular Biology, Nutley, New Jersey 07110

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Genetic Recombination in the Dinoflagellate Crypthecodinium cohnii

Abstract. *Genetic recombination in dinoflagellates has been detected with the use of chemically induced carotene-deficient mutants of* Crypthecodinium cohnii.

We report the first unequivocal demonstration of genetic recombination in dinoflagellates with carotene-deficient mutants (*1, 2*) of *Crypthecodinium cohnii* (Seligo) Chatton in Grassé, 1952 (*3*), a nonphotosynthetic marine species. Because dinoflagellates possess chromosomes which remain condensed throughout their cell cycle (*4*) and contain large amounts of DNA per cell (*2, 5*), speculation as to their chromosome structure and nuclear organization has received increasing attention (*2, 6*). The occurrence of sexual reproduction in dinoflagellates has been a subject of conjecture (*7*). Until recently only incomplete cytological evidence existed for the presence of a sexual cycle.

Using *N*-methyl-*N'*-nitro-*N*-nitrosoguanidine, we have produced (*1, 2*) mutant clones that are deficient in β- and γ-carotenes—the only pigments present in wild-type cells (*1*)—at frequencies (*2*) consistent with the vegetative cell being haploid. At least two phenotypes of carotene-deficient mutants have been isolated (*1*); albino clones (pig-1, pig-3, pig-4, pig-5, and pig-13) lack measurable carotenes, and cream-colored clones (pig-2 and pig-10) contain about one-tenth the carotenes of the wild type (*8*).

In media depleted of nitrogen and phophorus (*9*) we have observed that pairs of motile cells sometimes fuse while swimming. The cytological studies of von Stosch (*10*), in which he observed in *Gymnodinium pseudopalustre* Schiller and *Woloszynskia*