Table 1. Repeated photoreversibility of root adhesion and bioelectric potential. Roots were irradiated with red (R), far-red (FR), and then red light for 4 minutes each, and the development of a bioelectric potential as well as the percentage of the roots adhering to glass were measured.

Treatment	Change in bioelectric potential* (mv)	Adhesion† (%)
R	+ 1.20, + 0.28, + 2.00	100, 90, 90, 90
R–FR	− 0.88, − 0.25, − 0.70	10, 0, 30, 20
R–FR–R	+ 0.35, + 0.23, + 0.15	90, 90, 80, 90

* Results of three experiments. † Results of four experiments.

reaching a plateau after about 150 seconds, and then resuming the previous rate of change. It is not known what the plateau represents, but it occurred quite regularly at the time of completion of the Tanada effect. It may be a manifestation of the termination of photoconversion of one form of phytochrome and the beginning of that of another form (*6*). The bioelectric potential slopes and time courses were, however, generally similar to those of the Tanada effect. Thus, red light (which produced adhesion to a negatively charged surface) produced a positive bioelectric potential, and both effects were reversed by far-red light. The repeated photoreversibility of both phenomena are shown in Table 1. The general decrease in amplitude of bioelectric potential change with time probably shows that the root tip was closely confined in a small volume of solution and may have used up the available oxygen as time elapsed. Nonetheless, the sign of the generated bioelectric potential was repeatedly associated with quality of light.

Because the development of a bioelectric potential, distinctive as to sign, is correlated so well with a phytochrome-mediated electrotropic response, it is concluded that this is one of the first events, if not the first, following photoreception and photoconversion of the phytochrome holochrome, and possibly is itself another manifestation of the same event. This last could be true if the holochrome were structurally part of the plasmalemma and, as light altered its own configuration, it in turn altered the configuration of the membrane, changing permeability characteristics and inducing a localized electrochemical gradient, manifested as a bioelectric potential. Such a chain of events might explain the sudden increase in electrolyte efflux from pinnae of *Albizzia julibrissin* exposed to red light (*7*).

Since the induction of a bioelectric potential must occur across a cell membrane due to electrochemical gradients across that membrane (*8*), these data support the view (*7*, *9*) that the primary phytochrome events are membrane bound.

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Anaerobic Brain Function: Effects of Stagnant and Anoxic Anoxia on Persistence of Breathing in Reptiles

Abstract. *Turtles tolerate anoxic anoxia about 14 times longer than stagnant anoxia. In snakes and crocodiles this difference is much less marked. Apparently, the remarkable anaerobic viability of turtles is dependent on blood circulation. Analyses of plasma indicate that loss of brain function in anoxic crocodiles is not caused by systemic acidosis or hypoglycemia. It is suggested that the ability of the central nervous system of the turtle to function without oxygen is due to a comparatively high rate of anaerobic uptake or metabolism of glucose.*

Lack of oxygen may result from stopping the circulation (stagnant anoxia) or from breathing gas mixtures which contain no O_2 (anoxic anoxia). Turtles as a group are much more resistant to anoxic anoxia than are the squamata and the crocodilia (*1*). In pure N_2, most turtles continue to breathe spontaneously at least ten times longer than other reptiles. Other behavioral indicators of central nervous system (CNS) function, such as corneal reflex, are

Table 1. Time (minutes, mean ± standard deviation) from first to last breath under conditions of acute anoxia. Numbers in parentheses are numbers of animals used.

Species	Stagnant anoxia	Anoxic anoxia
Caiman crocodilus	26.4 ± 2.2 (8)	32.0 ± 3.1 (8)
Natrix fasciata	39.3 ± 3.5 (20)	62.2 ± 11.1 (20)
Chrysemys picta	72.4 ± 8.0 (8)	1104 ± 170 (8)
Pseudemys concinna	65.2 ± 6.4 (20)	900 ± 77 (20)

Table 2. Analyses of heparinized blood and plasma from N_2-breathing crocodiles and turtles; mean values. Two animals were used under each condition; duplicate analyses were made of each sample.

Measurement	Duration of N_2-breathing				
	Caiman crocodilus		*Chrysemys picta*		
	0 min	30 min	0 min	30 min	720 min
*p*H	7.54	7.58	7.78	7.86	7.12
Glucose (mmole/liter)	7.8	10.6	5.4	8.1	53.3
Lactate (meq/liter)	5.4	8.5	1.1	4.2	22.7

similarly persistent in anoxic turtles. I now report that turtles are less able to withstand stagnant anoxia.

Snakes (*Natrix fasciata*) and turtles (*Pseudemys concinna*) were collected within an 80-km radius of Gainesville, Florida, during March and April 1963. Crocodiles (*Caiman crocodilus*) and turtles of another species (*Chrysemys picta*) were obtained from animal dealers during November 1966. All animals weighed between 150 and 250 g. All were kept at 22° ± 2°C, given water but no food, and used between 1 and 2 weeks after their arrival in the laboratory. Stagnant anoxia was produced by removing the animals' hearts under local (lidocaine) anesthesia. Incisions through which the hearts could be reached were made under local anesthesia 24 hours before cardiotomy, and were sealed to prevent pneumothorax. Animals which were to be subjected to anoxic anoxia were also incised 24 hours prior to their experiments as described above, but instead of having their hearts removed, they were placed in a continuously flushed, pure N_2 (oxygen tension < 0.4 torr) atmosphere (*1*). All animals breathed this atmosphere freely. Body temperatures of all animals during experiments were 22.0° ± 0.2°C. In experiments where N_2 was breathed, time from the first breath of N_2 to the last breath was measured; in cardiotomy experiments, time from the first breath after removal of the heart to the last breath was measured (Table 1). An electrocardiogram (EKG) was recorded from each N_2-breathing snake or crocodile about 20 minutes after the last breath, and from each N_2-breathing turtle about 120 minutes after the last breath. In all cases these EKC's had characteristics previously correlated with effective cardiac activity, showing that the deterioration of CNS function was not secondary to failure of the heart.

Blood samples taken anaerobically by cardiac puncture from additional N_2-breathing turtles and crocodiles were analyzed for *p*H, lactic acid, and glucose (*2*) (Table 2). These animals received the same treatment as did those in which persistence of breathing was measured. Each was used for a single sample only, in which as much blood as possible (6 to 8 ml) was obtained.

Anaerobic CNS failure is probably due to lack of metabolic energy. In a previous experiment (*3*), turtles (*Sternothaerus minor*) were injected with iodoacetic acid, which diminishes energy production from anaerobic glycolysis. In air, these turtles maintained nearly normal rates of O_2 uptake and survived indefinitely, but in N_2 their survival time averaged less than 20 minutes, compared to more than 12 hours for controls. Anaerobic glycolysis thus seems primarily responsible for maintaining the activity of the CNS under anoxic conditions. The data of Table 1 show that the comparatively long survival of turtles under conditions of anoxic anoxia is dependent on blood circulation. Since CNS failure in turtles occurs so quickly when anoxia is the result of lack of perfusion, and so slowly when O_2-free blood is present, it follows that the long survival of turtles breathing pure N_2 depends on the transfer of substances (presumably glucose and lactic acid) between the blood and the CNS. Snakes and crocodiles are evidently unable to make such significant use of these transfers. Table 2 suggests that the differences between turtles and other reptiles are not related to concentrations of hydrogen ions, glucose, or lactate ions in the blood.

To supply energy at the same rate as aerobic metabolism does, anaerobic glycolysis would require a more than tenfold increase in glucose transport into the cells and in activity of the Embden-Meyerhof pathway. Thus the failure of snakes and crocodiles to maintain CNS function under conditions of anoxic anoxia may be due to insufficient glycolytic capacity of the brain cells, to inability of these cells to take up glucose from the blood at a rate sufficient to meet anaerobic needs, or to buildup of a toxic intracellular concentration of lactic acid.

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Tyrosine Aminotransferase: Enzyme Induction Independent of Adenosine 3′,5′-Monophosphate

Abstract. *The importance of adenyl cyclase and adenosine 3′,5′-monophosphate in the induction of tyrosine aminotransferase by adrenocorticosteroids has been tested in HTC cells derived from a rat hepatoma and grown in tissue culture. Adrenocorticosteroids cause a 10- to 15-fold increase in the rate of synthesis of tyrosine aminotransferase in these cells. Under various experimental conditions, with or without glucocorticoids, neither adenyl cyclase nor cyclic adenosine monophosphate could be detected in HTC cells. In addition, neither the cyclic nucleotide nor* N^6,$O^{2'}$*-dibutyryl cyclic adenosine monophosphate caused increased activity of the transaminase in HTC cells. We conclude that induction of tyrosine aminotransferase by glucocorticoids is not mediated by the adenyl cyclase–cyclic adenosine monophosphate system.*

Increased synthesis of the hepatic enzyme tyrosine aminotransferase (TAT) (E.C. 2.6.1.5) occurs when adrenocortical hormones are administered to intact rats (*1*), perfused through isolated livers (*2*), or added to fetal liver explants in organ culture (*3*). Activity of the enzyme is increased in liver by insulin (*4*) and glucagon (*4*) through increased synthesis of enzyme and in fetal liver explants by epinephrine (*5*), presumably through the same mechanism. The mechanism of induction by these nonsteroid hormones apparently differs from steroid hormones since (i) combinations of a steroid and a nonsteroid hormone cause an additive response but combinations of two different nonsteroid hormones do not (*5*); (ii) the kinetics and magnitude of the responses to the two groups of hormones differ (*4*); and (iii) induction of the enzyme in fetal rats occurs in response to insulin and glucagon but not hydrocortisone (*6*).

Certain physiological actions of chemically diverse hormones, including insulin, glucagon, and epinephrine, appear to be mediated through alterations