Although the utriculi majores of the males were filled with uric acid, they were not stretched to their fullest extent. The amount of uric acid in the gland depends on the frequency of mating, since these glands are virtually emptied during copulation.

Mating appears to be an important means of excreting uric acid in males of *B. germanica*. However, not all male cockroaches possess these uricose (from uric + -ose, full of) glands. Only males of 7 of 39 species examined had uric acid in their accessory sex glands (*6*).

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6. A survey of the occurrence of uricose glands, together with a discussion of their possible significance, is in preparation.

28 August 1964

Reptilian Thermoregulation: Evaluation of Field Studies

Abstract. *Inanimate objects may exhibit distributions of temperature similar to those of reptiles in the field. The uncontrolled field methods of collecting body temperatures of reptiles have resulted in the accumulation of much inconclusive data. A return to more comprehensive study is called for.*

In 1944 Cowles and Bogert showed that many reptiles regulate their body temperature by behavioral means (*1*). This demonstration stimulated both the accumulation of records of body temperature from many reptiles and discussion of the interpretation of the records. Since that time the method of Cowles and Bogert has been progressively simplified (*2*).

Many workers have discarded the several categories of thermal responses proposed by Cowles and Bogert in favor of determining the body temperatures of reptiles surprised in the field. These records are often presented without interpretation and only to indicate the body temperatures that occur during the normal life of an animal. A problem arises when the field records are used to try to elucidate thermoregulatory mechanisms when no true regulation has been observed.

The method now in use by many workers is quite simple. Reptiles are shot, noosed, or run down, and their body temperatures are recorded with a small, sensitive thermometer. The air temperature is usually taken, but there is no standardization in the way this is done. In some cases body temperatures below an arbitrary level are ignored because they lie in the so-called "basking range" of the animal. The activity of the animal prior to measurement is rarely known, but we are usually assured that temperature regulation is occurring by reference to the work of Cowles and Bogert. Concern about this method led to the development of a simple demonstration that data on reptile temperatures must be collected and interpreted with great care.

In order to determine whether the distribution of temperatures of inanimate objects would be distinguishable from those obtained from reptiles under similar conditions, I substituted water-filled metal cans for reptiles. Thirteen cylindrical metal containers (beer cans), 11.5 by 6.8 cm in diameter, with colored patterns on the outside, were filled with tap water at air temperature. Eleven were placed so as to receive direct sunlight throughout a full day. Two were kept in full shade. Temperatures 1 cm from the top of the can were recorded hourly from 1030 until 1830 P.S.T. with a hypodermic thermistor probe 0.3 mm in diameter. Accurate temperatures can be recorded in 0.2 second with this small probe. Air temperature next to the cans was recorded with a shaded probe. The recording process could not substantially alter the temperature of the can. Evaporation was too low to change the can's temperature measurably. It was sunny throughout the day.

Cans in the shade remained within 1°C of the air temperature. The distribution of temperature recorded from cans in direct sunlight is remarkably like that of a heliothermic reptile (Fig. 1). A sharp break, in this case at 35°C, is typical of records obtained from reptiles. The break has been interpreted to mean that an animal actively seeks shade at that temperature. In view of the data obtained from cans, such an interpretation would need to be justified.

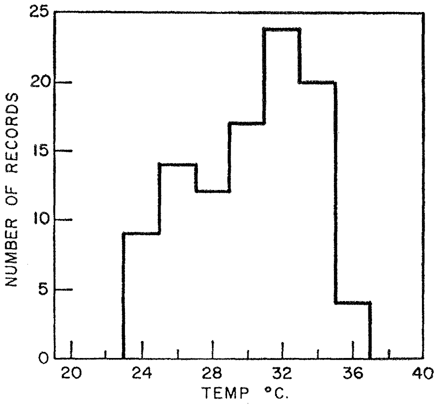

Fig. 1. Temperature distribution of cans in direct sunlight (July 1963). The skew reflects both the skew in ambient temperature and the relatively constant radiative heat input from the sun through the middle of the day (mean 30.3°C; standard deviation 3.14; $N = 97$).

Arranging the data into a scatter diagram is informative (Fig. 2). The records were treated two ways. The lower diagram shows all the points obtained. Can temperature is loosely correlated with air temperature ($r = +.41$;

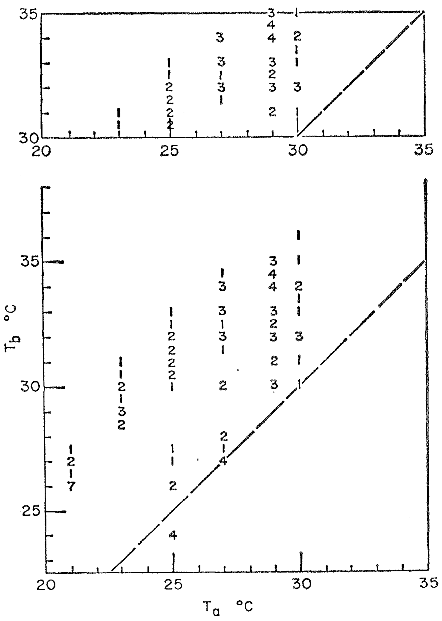

Fig. 2. Scatter diagram showing the relation of can temperature (T_b) to air temperature (T_a) in intervals of 0.5°C. In the upper graph all can temperatures below 30°C are eliminated. Dashed line, isothermal line; numerals, number of records at each interval.

$P < .005$). Following the precedent of others, all can temperatures below an arbitrary level, in this case 30°C, were ignored (Fig. 2, upper portion). The product-moment correlation coefficient obtained is $-.09$; $P < .05$. This indicates that the can temperature is independent of the air temperature. The range of correlation approximates that reported among nine species of lizards (*3*). A consistent deviation of body temperature from air temperature cannot be regarded as *prima facie* evidence of regulation, nor can the highest temperatures obtained from a reptile in the field be considered equivalent to a regulated level.

The relation of can temperature to air temperature is indistinguishable from that of heliothermic reptiles. A can exposed to direct sunlight becomes hotter than the surrounding air in the same manner as a reptile. A can, or a lizard, reaches an equilibrium between heat gain and heat loss that results in the maintenance of an elevated body temperature. Although reptiles can regulate their temperature behaviorally, they often reach an equable body temperature and remain at that level passively without need of active regulation (*4*). Therefore, body temperatures randomly collected in the field need not reflect regulation.

If Cowles and Bogert's approach is adequate to demonstrate regulation, at what point in the simplification of procedure was the demonstration of regulation eliminated? Only two of their categories, the maximum voluntary tolerance and minimum voluntary tolerance, contain behavior which alters the heat load upon the animal. The other categories, composed of lethal temperatures and activity and basking ranges, although convenient, are not directly related to active regulation. The elimination of the categories of maximum and minimum voluntary tolerances also eliminated the regulatory elements in Cowles and Bogert's method.

A study of behavioral temperature regulation requires either that the regulatory behavior be witnessed and body temperature immediately measured or that a control be used so that the body temperature of the regulating animal can be compared to the temperature of a model. The cans used in this demonstration would not be adequate as a control because they differ from reptiles in size, shape, reflectivity, and thermal conductance. A simple and useful control might be to tether an animal in the direct sunlight and check its temperature periodically. A deviation of the control temperature from that of an animal collected randomly would be partly attributable to regulation. However, more significant results would come from direct analysis of the regulating behavior.

Further field work must adhere strictly to the methods proposed by Cowles and Bogert. It seems unlikely that the random collection of body temperatures will contribute to further knowledge of temperature regulation among reptiles. The experimental studies of Ruibal (*5*), Lee (*4*), Bartholomew and Tucker (*6*), and Heath (*7*) offer more fruitful approaches.

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8. Thanks are due the many biologists whose concern over these matters led to the formulation of this critique, particularly K. S. Norris and B. H. Brattstrom. Special thanks are due R. B. Cowles, who laid the original groundwork for the study of behavioral temperature regulation. Prepared in part under NIH postdoctoral fellowship at University of California, Los Angeles.

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Homocystinuria due to Cystathionine Synthetase Deficiency: The Mode of Inheritance

Abstract. *Deficiency of cystathionine synthetase activity results in the clinical syndrome of homocystinuria. In both parents of a patient with homocystinuria, the hepatic cystathionine synthetase activity was 40 percent of that in unrelated control patients. These findings demonstrate that the metabolic error is inherited and suggest that the parents, although clinically normal, represent the heterozygous state. A second case of homocystinuria also is shown to be associated with cystathionine synthetase deficiency.*

The excretion of homocystine in the urine, homocystinuria, is associated with various clinical features, including mental retardation, dislocation of the ocular lenses, sparse blond hair, genu valgum, convulsive tendencies, failure to thrive, thromboembolic episodes, and fatty change of the liver (*1–5*). In most reported cases, the patients have had, in addition to the high urinary content of homocystine, elevated concentrations of methionine and homocystine in the plasma. We demonstrated previously that the liver of a child with this syndrome lacks activity of cystathionine synthetase and proposed that this enzyme deficiency causes the disorder (*5*). Cystathionine synthetase catalyzes the condensation of homocysteine and serine to cystathionine, this being an intermediate step in the conversion of methionine to cysteine. Absence of the enzyme activity explains the biochemical manifestations of the syndrome.

Recently, Gerritsen and Waisman (*6*) reported that the brain of a homocystinuric patient contained no detectable cystathionine; in children without homocystinuria, cystathionine was present. Although their earlier studies led them to conclude "that the cystathionine synthetase path was not blocked" in this case of homosystinuria (*3*), Gerritsen and Waisman consider their most recent study to be consistent with the absence of hepatic cystathionine synthetase. We think that all their findings in this one case are compatible with a deficiency of this enzyme.

This report concerns the yet unanswered question of whether the error of metabolism is hereditary; if so, what is the mode of inheritance? We also examine the question of whether the enzyme deficiency is invariably associated with mental retardation. This inquiry was prompted by the finding of Barber and Spaeth (*4*) that a paternal cousin of a homocystinuric patient (*4, 5*) excretes homocystine in her urine, but is neither mentally retarded nor otherwise symptomatic.

We assayed hepatic cystathionine synthetase activity in relatives of a homocystinuric patient, C.T. The re-