While outlining the advantages conferred by vivipary upon the embryo, Greene (*op. cit.*) plausibly argues that the thermo-regulation by basking in cool climates at high altitudes (above 6000 ft) confers an advantage "for embryos to undergo development in the female rather than in an egg abandoned in the environment".

Another equally important factor appears to be the relative fecundity of the concerned forms. *L. himalayanum* has been observed to produce on an average lesser number of all types of eggs (Table I) than *A. tuberculata,* although both show

TABLE I

Monthly incidence of ovarian and oviducal eggs in Agama and Lygosoma

Month	*Agama tuberculata*		*Lygosoma himalayanum*	
	Ovarian Eggs	Oviducal Eggs	Ovarian Eggs	Oviducal Eggs
April	+/36 (26–40)	..	+*	+/2
May	+/32 (22–34)	+/6	+/16 (12–18)	+/4
June	+/24 (22–26)	+/7	+/18 (12–20)	Emy/6
July	+/22 (18–24)	+/7	+/18 (12–22)	Emy/4
August	+/20 (18–24)	..	+/20 (14–24)	..
September	+/24 (18–26)	..	+/20 (16–24)	
October	+/28 (26–32)	..	+/22 (16–26)	..

* Only one specimen obtained for this month.

a similar breeding period (May to July) and the latter retains oviducal eggs for two months (from middle of May to middle of July ; no lizard seemed to bear oviducal eggs after July) as against *L. himalayanum,* which retains the oviducal eggs and/or near term embryos for two months and a half (early May to mid July). This works to over 25% longer gestation period in *L. himalayanum.* These facts add the factor of fecundity to the eco-habit differences in the two lizards without, however, reducing the importance of temperature as one of the other factors involved. A longer gestation and reduced fecundity would inevitably render the scincid lizard a comparative disadvantage that would exclude from the company of oviparous species which are potentially better reproducers. With increased tendency towards the accomplishment of vivipary, the ability to produce the greater number of eggs, thus appears to have got progressively reduced ; and with progressive lowering in fecundity, the surest coverage against any risk of ovular dystrophy or failure of fertilized eggs to develop in adverse environmental conditions would be, for the female, to bear the eggs within her body during the embryonal development—a stage identifiable as *ovovivipary.* Any further intimacy between the embryo and the maternal tissue must ultimately have led to vivipary. Severity of climatic conditions, reduced thermoperiod, low fecundity and high altitude together with other intrinsic factors like hormonal interplay and extrinsic factors like different types of rays that obtain at high altitudes might have collectively contributed to the evolution of vivipary—envisaged as a multifactorial hypothesis as hinted by Tinkle (1967).

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PHYSIOLOGICAL STUDIES ON THE EFFECTS OF NUTRITIONAL IMBALANCE ON THE CENTRAL NERVOUS SYSTEM: HORMONE-LIKE ACTION OF THE BLOOD OF THIAMINE-DEFICIENT CHICKEN, *GALLUS DOMESTICUS*

It is well established that the deficiency of B_1-vitamin (Thiamine) causes beri-beri in man and signs of nervous symptoms in birds referred to as Poly-neuritis[1,2]. Thiamine as thiamine-pyrophosphate is known to influence the oxidative decarbo-

lation of pyruvate in pigeon and man[3,4]. It has been suggested that accumulation of pyruvate in thiamine-deficient vertebrates may be responsible for various signs of poly-neuritis[4,5]. It is presumed that *injury to medulla* is responsible for the pathogenic symptoms of B_1-avitaminosis. The medullary nervous symptoms during thiamine-deficiency have been reported to be due to toxic product accumulation[5,6].

Earlier investigations of Nayeemunnisa[7,8] and other workers[9] indicated changes in the levels of RNA, activity levels of certain oxidative enzymes and acetylcholinesterase in the different regions of the brain of chicken, *Gallus domesticus*, during thiamine-deficiency. The present investigation aims to see whether the neural disturbances result in blood-borne factors and if so whether these factors affect the cardiac rhythmicity.

Three days old, male white leghorn chicken, *Gallus domesticus*, ranging in weight from 12–14 grams were reared in the laboratory in electrically-heated cages at 37 ± 2° C. The controls were fed on standard chicken feed (Mysore Feeds, Bangalore, India). The experimental birds were fed on polished rice for 25–30 days as described by Peters[5] to induce thiamine-deficiency. Water was made available *ad libitum* to both the groups.

The control chicken were given mild ether anaesthesia and dissected with sterilized instruments to expose the heart. The heart and the exposed body cavity was flooded with avian Ringer[10,11] at the body temperature. The heart was then carefully connected with the help of a wire hook and thread to the writing pointer of the kymographic drum (Patent Inco, India. No. M.E. 1880 220 Volts A.C. rotated at 2·5 mm/second) after *in vitro* soaking by blood obtained from other control chicken. These recordings of the cardiac contractions constituted observations for the control animals. The preparation was washed in fresh avian Ringer[11]. Blood from the thiamine-deficient chicken, showing signs of opisthotonus was extracted from the wing vein with a sterilized syringe, was added directly on the heart of the control chicken and the heart beat under the influence of the blood of avitaminous birds was recorded kymographically under identical conditions.

The kymographic recordings were fixed in turpentine : varnish (2 : 1) and photographed. In general the heart beat is decreased on *in vitro* administration of blood from thiamine deficient chicken. The per cent decrease of heart beat in control birds is — 26·4, — 50·0 and — 28·2 in the burst frequency (33 and 24·3 per minute for controls and experimentals respectively) amplitude (1·5 and 0·75 mm for controls and test birds respectively) and duration (2·2 and 1·6 seconds for controls and experimentals respectively) whereas inter-burst interval was prolonged (per cent increase is 92·3 and average values being 0·13 and 0·2 mm for controls and test animals respectively) on administration of blood from B_1-avitaminosis birds (Table I).

TABLE I

The heart-beat parameters of Gallus domesticus

Characteristics measured	*Controls	**Experimentals	Percentage change
Burst frequency (Per minute)	33·0±3·0	24·3±2·7	−26·4
Burst amplitude (mm)	1·5±0·21	0·75±0·23	−50·0
Burst duration (Seconds)	2·2±0·01	1·58±0·016	−28·2
Inster-burst Interval (mm)	0·13±0·02	0·25±0·01	+92·3

Values are Mean of 4 observations±S.D.
(+) and (−) signs indicating increase and decrease respectively over controls.
Values are significant at $P > 0·01$.
*Control chicken heart soaked with the blood of normally active chicken.
**Control chicken heart soaked with the blood of thiamine deficient chicken.

These changes produced in the heart beat by the blood of thiamine deficient chicken, *Gallus domesticus* clearly indicate the presence of one or more hormone-like factors which can produce *in vitro* effects. Generally, thiamine deficiency results in the profound changes in metabolic rate of the tissues[3-5]. The fact that the blood of avitaminosis birds is capable of depressing the heart beat of normal chicken, clearly suggests the presence of an inhibiting factor in the blood of thiamine-deficient birds, produced as a consequence of metabolic alterations.

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PEROXIDASE ACTIVITY IN PAPAYA PLANT INFECTED WITH PAPAYA DISTORTION MOSAIC VIRUS

VIRAL infection induces several metabolic changes in plants[1,2]. Changes in peroxidase activity following infection have also been reported. Loebenstein and Linsey[3], Bhargava *et al.*[4], and Wood and Barbara[5] reported an increase in peroxidase activity in vein clearing virus infected sweet potato, sugarcane plants infected with sugarcane mosaic virus and tobacco plants infected with cucumber mosaic virus respectively. In tobacco mosaic virus infected tobacco leaves, however, peroxidase activity decreased below that of healthy leaves both during early and later stages of the development of the disease[6]. Present investigation was undertaken to examine the effect of papaya distortion mosaic virus on peroxidase activity in papaya leaf.

To determine the effect of papaya distortion mosaic virus on peroxidase activity, papaya plants grown in the glass house were inoculated at 4–5 leaf stage with sap from infected papaya plants using carborandum as an abrasive. Healthy plants of the same age served as control. Peroxidase activity was determined at 0, 3, 6, 9, 12 and 15 days after inoculation. During sampling 6 discs of 1 cm diameter each were taken from healthy as well as infected leaves, care being taken that sampling was always done from the third leaf. The discs were weighed and homogenized in 2·0 ml of chilled distilled water and filtered through cheese cloth. The filtrate was centrifuged in a refrigerated centrifuge at 1,000 rpm for one hour. The enzyme activity in the clear supernatant was measured colorimetrically according to the method of Loebenstein and Linsey[3] as described by Wood and Barbara[5].

The results presented in Table I show that the peroxidase activity in the papaya leaves increased with papaya distortion mosaic virus infection, the increase being upto a maximum of 39·5% after 9 days of inoculation.

TABLE I

Peroxidase activity in healthy and virus infected papaya leaves

Days after inoculation	Enzyme activity (S^{-1} g^{-1} fresh wt.)		
	Control	Inoculated	Percentage increase
0	27·1	27·1	0
3	21·3	26·7	25·3
6	22·0	25·7	16·9
9	13·4	18·7	39·5
12	23·2	31·3	34·9
15	38·9	37·5	0

Peroxidase is known to catalyse the oxidation of phenolic substances and aromatic amines to quinones in the presence of hydrogen peroxide[7], though its exact physiological role, even in normal metabolism, is not clearly understood. In many cases increase in peroxidase activity in plants has been correlated with either disease resistance[8,9] or susceptibility and symptom response to virus[3,10]. Loebenstein and Linsey[3] reported that increase in peroxidase activity following inoculation commences with the appearance of symptoms in the infected plants. In our study, increase in peroxidase activity was found after 3 days, when no symptoms had appeared though viral activity was present. Maximum increase was, however, found after 9 days of inoculation. In this connection, it may be mentioned that viral activity was also more pronounced between 9 and 12 days after infection, showing a correlation between