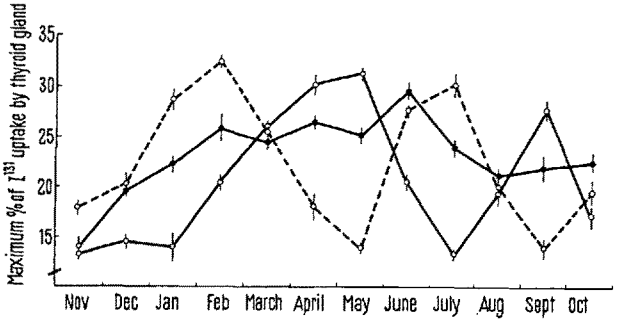
Effects of Varied Photoperiods on Rhythmic Activity of Thyroid Gland in a Teleost, *Mystus vittatus* (Bloch)

Cyclic changes in thyroidal activity have been reported in several species of teleosts[1-6]. Though the cyclic nature of its activity is well established, the reasons for the changes are not clear. Thyroidal iodine accumulation depends on the availability of environmental iodine[7-9], temperature[10,11], photoperiods[12] and seasons of the year[13,14]. Hypertrophy and hyperplasia of thyroid have been reported in *Astyanax mexicanus* subjected to darkness[15]. BARRINGTON and MATTY[16] have reported an increase in cell height in minnows kept in darkness, with rising water temperature. BERG et al.[11] have stated that thyroidal I^{131} uptake varied with season, being four times as great in January as in July. But this quantitative variation was neither correlated with the seasonal changes in thyroxin production, nor with thyroidal epithelial cell height, nor with photoperiods. HOAR and ROBERTSON[12] have recorded that thyroid activity was slightly greater in goldfish maintained under shorter photoperiods.

Thyroid activity in *Mystus vittatus* reported here was judged by its I^{131} uptake. Eight hundred and sixty-four mature specimens of both sexes were divided into 3 equal groups, one was kept in natural photoperiods, the second was subjected to continuous illumination and the third was exposed to total darkness. The first group kept in natural photoperiods was also treated as control for the other 2 groups. Under the above photoperiodic conditions the thyroid activity was evaluated for 12 months. Since the aquaria were not kept in temperature-controlled conditions, their temperature varied in accordance with the natural environmental fluctuations; but a uniformity in temperature was maintained in all 3 groups as they were subjected to similar environmental temperature variations. The effect of temperature on thyroid gland was not considered here because, at any period during the course of experiments, the temperature was the same for all groups. The chemical contents including iodine concentration of aquarium water were constant throughout the year. In every month 24 fishes from each group were given a tracer dose of 5 μc of I^{131} and utmost care was taken to check the loss of solution during and after tracer injections. Four fishes from each group were killed at regular intervals of 1, 2, 3, 4, 5 and 6 days after the administration of tracer dose. The region of the lower jaw containing thyroid follicles was cut and its I^{131} uptake were evaluated with scintillation counter. The maximum accumulation of radioiodine was observed from 18–24 h of post final treatment. This fish exhibited seasonal cyclical changes in thyroid activity. In its natural cycle at normal photoperiods, there were 2 peaks of activity, each of which was alternated by a quiescent phase. At the start of the experiments in November, the activity was low. The first peak was observed in April through May (Figure). This was followed by a decline which was at its ebb during July (Figure). The next peak was encountered in September, succeeded again by a quiescent phase. The pattern of thyroid activity in the second group exposed to continuous light was similar to that observed in specimens subjected to natural photoperiods. But in continuous illumination the periods of maximum and minimum activity were advanced by 2 months. Thus January through February and June through July were the months of optimum activity; and May and September of minimum (Figure). In spite of these alterations in activity, the basic inherent capacity of the thyroid for the cyclical changes was maintained under continuous illumination. The third group exposed to total darkness showed an increased state of activity throughout the year as compared to the other 2 groups (Figure). It is interesting to note that under constant darkness the distinct seasonal changes in thyroidal activity are lost. It appears that darkness reacted as a continuous stress which might be responsible for the abolition of the inherent rhythm for cyclic changes. SWIFT[3] has suggested that the cyclical changes in the thyroid gland may be due to the variations in the external environment or due to its inherent capacity for such a rhythmic activity.

In *M. vittatus* the external factors such as photoperiods do influence and modify the inherent rhythm of thyroidal activity to a greater or lesser extent[17].



Seasonal variations in the activity of thyroid of *M. vittatus* under varied photoperiods. o——o normal photoperiod, o- - -o continuous light, •——• total darkness.

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[17] I am grateful to Prof. A. J. MATTY, Department of Biological Sciences, University of Aston in Birmingham, for his helpful suggestions, and to Dr. A. G. SATHYANESAN, Department of Zoology, Banaras Hindu University, for his help and encouragement in the preparation of the manuscript.

Résumé. La glande tyroide du *Msytus vittatus* sous photoperiodes normales montre des variations cycliques saisonnières: Au cours d'un an il y eut 2 phases d'activité tyroidale augmentée et chacune fut séparée par 1 phase de repos. Sous illumination prolongée le type de variations resta identique; sauf les phases de repos furent enregistrées 2 mois plus tôt que la normale. Ce plan rythmique d'activités disparut dans une obscurité totale et l'activité tyroidale demeura a un niveau élevé toute l'année.

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STUDIORUM PROGRESSUS

Epidermal Homeostasis - a Numerical Model. Kinetics of Epidermal Cells

With the possible exception of the nervous tissue, the maintenance of functional capacity of adult organs involves a continuous process of cell renewal. In the majority of cases this process depends on the persistence of low level mitotic activity, but it may involve cellular transformation as when antigenic stimulation induces plasma cell formation. An important aspect of the problem is that, in general, mitotic activity keeps pace with cell destruction in such a manner that there tends to be maintained a rather constant ratio between the mass of a given organ and the total body mass. This regulation appears to be the rule, although in some instances physiological cyclical activity or altered functional demand obscures this basic relation, as in the case of the uterus during menses or pregnancy.

The manner in which mitotic activity keeps pace with cellular destruction is not understood and may involve a variety of mechanisms. In the case of the liver or kidneys, regeneration experiments suggest that functional load is the determining factor. A similar conclusion may apply to the regulation of the mass of red cells, since either bleeding or injection of additional red cells is compensated for, the mass of red cells returning to the pre-experimental level[1].

A kinetic model of growth regulation developed by WEISS and KAVANAU interprets cellular homeostasis in terms of production of inhibitory or repressive substances which are able to diffuse across cell boundaries[2]. BULLOUGH has likewise postulated an inhibitor-based negative feedback to account for the maintenance of the normal epidermis[3], and has subsequently claimed to have isolated the repressor substance. IVERSEN incorporated these ideas in an electronic analogue and demonstrated that an inhibitor-based negative feedback system does indeed reproduce the kinetic behavior of epidermal cells under a variety of conditions[4].

Suggestive as these ideas are, there is no satisfactory evidence to substantiate them. IVERSEN's analogue simulation does not per se prove the existence of a molecular repressor mechanism, since the postulated model is descriptive of a number of different physical systems. BULLOUGH's evidence is rather unsatisfactory, mainly because the postulated repressor does not repress unless epinephrine is present in the system. The lowest dose of epinephrine employed in vivo[5] is so large (10 μg/mouse) as to exceed thousands of times the normal blood content of this amine, (less than 1 μg/l) suggesting that mitotic inhibition may have resulted from the combined effects of adrenocortical activation and intense prolonged cutaneous vasoconstriction. The in vitro experiments are similarly inconclusive because they were conducted in an atmosphere of pure oxygen[5,6] which favors the oxidation of epinephrine to adrenochrome, a known mitotic poison. This consideration is more than suggestive, since addition of reduced glutathione or ascorbic acid which by themselves do not affect mitosis, effectively prevents or abolishes the presumed antimitotic effects of epinephrine[7].

This short appraisal of current views highlights our surprisingly unsatisfactory grasp of factors involved in epidermal homeostasis. An attempt to re-evaluate the problem de novo has resulted in a basically simple kinetic formulation.

Epidermal nutrition. In contrast to other tissues, the epidermis does not possess a blood supply of its own, depending for its non-gaseous needs on the movement of substances across the basement membrane. Thus the nutritive flux into the epidermis could be governed both by the extent of exchange of materials between blood and tissue fluid in the dermis and by the magnitude of the subsequent transport of metabolites across the dermal-epidermal junction.

Since transendothelial exchange is flow limited both in the case of small[8] and large[9] molecules, it is subject to hemodynamic regulation. However, since at low blood flows local metabolic regulation may take precedence over central constrictor influences[10], it is unlikely that under normal conditions insufficient transendothelial exchange can last long enough to materially affect the nutritive traffic across the basement membrane.

Thus, assuming an adequate transendothelial movement of metabolites in the dermis, epidermal nutrition becomes a function of the magnitude of metabolite flux across the basement membrane. This process is usually thought to be due to diffusion, but under certain conditions it is possible to distinguish the presence of another component of transport, which is mediated by the bulk flow of water. This component may readily be recognized in scotch tape stripped skin, where the surface becomes

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