course, is that pteridine is not a cofactor for synthesis of carotenoids and the action of PDAP on carotenoid dehydrogenation is independent of pteridines.

Both photosynthetic phosphorylation and CO_2 fixation in bacteria are inhibited by PDAP (*4*, *5*). Pteridines can stimulate these processes (*8*). It seems probable that the effect of this pteridine inhibitor on other photosynthetic processes in bacteria (H_2 evolution, chlorophyll synthesis, CO_2 fixation, photophosphorylation, and so forth) is not related to the effect described above on carotenoid biosynthesis—unless the carotenoid pigments function more directly in photosynthetic electron transport than is presently envisaged.

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Calcitonin from Ultimobranchial Glands of Dogfish and Chickens

Abstract. *Acid extracts of thyroid glands from a small shark* Squalus suckleyi *and domestic fowl* Gallus domestica *contained no detectable calcitonin activity, while very potent hypocalcemic responses were obtained in rats with similar extracts from the ultimobranchial glands of these two species. The calcitonin concentration was 4 to 40 times that present in hog thyroid, which, as in most other mammals, contains ultimobranchial tissue. The evidence suggests that calcitonin is a fundamental calcium-regulating hormone present in all higher vertebrates and that it is an ultimobranchial rather than a thyroid hormone. It also indicates an important and hitherto unrecognized function for the ultimobranchial glands.*

In 1962 Copp *et al.* (*1*) presented evidence for a hypocalcemic hormone released when thyroid and parathyroid glands were perfused with high-calcium blood. They named the hormone calcitonin, since it was apparently involved in regulating the level or "tone" of calcium in body fluids. Originally thought to be of parathyroid origin, it was soon found that the hormone is present in (*2*), and released by (*3*), cells present in the mammalian thyroid. For this reason, the name thyrocalcitonin has been suggested for the hormone (*2*). Using immunofluorescent techniques, Bussolati and Pearse (*4*) showed that calcitonin is not present in the regular thyroid cells, but is present in the parafollicular C cells (also called "light" cells). Pearse and Carvalheira (*5*) also demonstrated by histological specific staining reactions that these are probably ultimobranchial cells. In other classes of vertebrates (fish, amphibians, reptiles, and birds) these exist as separate glands, but in mammals they become imbedded in the thyroid and inferior parathyroid (see *6*).

Inspired by these observations, we have investigated the hypocalcemic activity of acid extracts of thyroid and ultimobranchial glands in chickens and dogfish, in which the two glands are separate and distinct. In the embryo, the thyroid arises from the thyroglossal duct in the anterior pharynx in the midline. The ultimobranchial gland (or body) arises from the ventral aspect of the last branchial pouch. It is present in all gnathostomes, and has hitherto been assigned no specific function.

In the Pacific Coast dogfish (*Squalus suckleyi*), a small shark weighing 2 to 4 kg, the ultimobranchial gland is located just above the pericardium on the left side in the triangle formed by the basibranchial and ceratobranchial cartilages and the coracobranchial muscle (*7*). The glands, which weighed 10 to 20 mg, were dissected out of fish caught 2 to 4 hours earlier, and were immediately frozen with dry ice. The chicken ultimobranchial gland is located in the chest near the bifurcation of the common carotid and axillary artery. The thyroid makes a good landmark, since the two parathyroids and the ultimobranchial glands are strung out in sequence below it. The glands were obtained from reject birds at a local poultry processing plant and were removed and frozen within 2 hours of the death of the bird. Extracts were prepared by the method of Hirsch *et al.* (*2*). The chicken glands were first defatted by extracting eight times with ten volumes of acetone. The dried material was then homogenized with ten volumes of 0.1*N* HCl and extracted at room temperature for 1 hour. It was then centrifuged for 10 minutes in a clinical centrifuge to remove cell debris. The dogfish glands contained little or no fat, and the glands were extracted directly. Thyroid tissue similarly treated served as control.

Extracts were brought to *p*H 4 by addition of 0.1*N* NaOH, and suitable dilutions were made with 0.9 percent NaCl so that the final volume injected was 0.5 ml. This was administered intraperitoneally to 44- to 49-day-old rats weighing 180 to 200 g. Samples of tail blood (0.2 ml) were collected at 0, 1, 3, and 6 hours after injection and were analyzed for calcium by the method of Copp (*8*). The response was assessed by the assay method of Copp and Kuczerpa (*9*) and is expressed as the area (in milligrams of calcium per 100 milliliters of plasma, per hour) between the plasma calcium curve after injecting extract and the plasma calcium level of the control. This evaluates both the intensity and the duration of the hormone effect. Five or six rats were used at each dose level.

The dose (per 100 g of the rat's body weight) is expressed in terms of the fresh weight of gland used to prepare the extract. The response was compared to that obtained in similar rats injected with a standard preparation of calcitonin from beef thyroid. This preparation contained 0.11 MRC units per milligram. These units

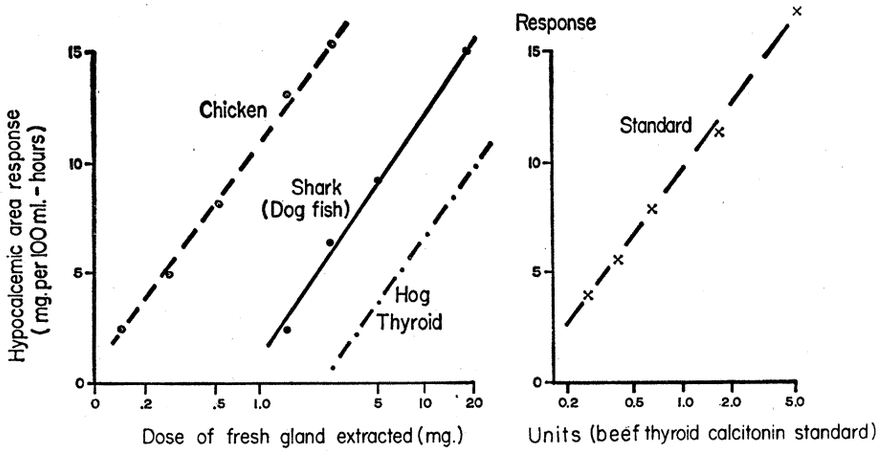

Fig. 1. Relation of hypocalcemic response to log dose of acid extracts of ultimobranchial glands of chicken *Gallus domestica* and a small shark *Squalus suckleyi*. Each point represents four to six rats. For comparison, the response to a standard preparation of calcitonin from beef thyroid and the estimated response to extracts of fresh hog thyroid (*10*) are indicated.

refer to an international standard (Thyroid Calcitonin–Standard A) prepared and made available by the Division of Biological Standards, National Institute for Medical Research, Medical Research Council, London. The relationship between response and the logarithm of the dose is shown in Fig. 1. The slopes for calcitonin from these three different sources are essentially parallel, suggesting similar biological activity. From these curves, the calcitonin extracted per gram of chicken ultimobranchial was estimated to be 130 MRC units as compared to 15 units per gram of dogfish ultimobranchial, and 2.6 to 4.3 units per gram of hog thyroid (*10*). The low value for the latter is not surprising, since ultimobranchial cells make up only 1 to 2 percent of the mass of hog thyroid.

The fall in plasma calcium level 1 hour after the injection of extract from one-tenth of a chicken ultimobranchial (0.5 mg) was 1.65 ± 0.30 mg per 100 ml; the fall after administration of extract from one-fourth of a dogfish ultimobranchial (4 mg) was 2.92 ± 0.19 mg per 100 ml. There was no detectable hypocalcemic effect from injection of extracts from 200 to 500 mg of chicken or dogfish thyroid.

The absence of detectable calcitonin in the thyroids of these animals, together with the very high level in the ultimobranchial glands, strongly suggests that in birds and elasmobranch fishes calcitonin is an ultimobranchial rather than a thyroid hormone. This is consistent with the view that calcitonin is produced by the C cells of the mammalian thyroid (*4*), and that these are of ultimobranchial origin (*5*). It is also interesting that the other calcium-regulating glands, the parathyroids, arise from a very similar embryological anlage in more anterior branchial pouches. The presence of calcitonin in the most primitive (elasmobranchs) and advanced (birds and mammals) vertebrates possessing ultimobranchial tissue suggests that this must be one of the fundamental vertebrate hormones. Indeed, phylogenetically the ultimobranchials and calcitonin appear to precede the other two important factors in calcium regulation, bone and parathyroid hormone. In mammals, calcitonin appears to act primarily on bone to inhibit osteolysis. It will be interesting to determine the action of this hormone in elasmobranchs in which there is no boney skeleton.

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Photoperiodic Control of Hamster Testis

Abstract. *The response of the testes of juvenile and adult hamsters to various photoperiods was examined. The testes of juvenile animals reached maturity regardless of the light cycle on which the animals were raised. However, the testes of adult hamsters required at least 12.5 hours of light per day to maintain spermatogenesis and prevent degeneration. This is one of the few demonstrations of a response suitable for study in investigations of the photoperiodic control of testicular function in a laboratory mammal.*

Seasonal breeding patterns in the field have long been observed and are well documented for most mammalian species (*1*). To insure production of the young at the time of year most conducive to survival, accurate synchronization of reproductive activities with the environment is essential. Most attempts to explain the observed correlation of reproductive activity with season invoke day length as the controlling factor. For, of all the environmental cues which indicate seasonal change, the duration of light per day is the most reliable.

The controlling role of light has been demonstrated by extensive experimental work on the timing of estrus in several mammalian species (*2*). However, only a few studies have considered photoperiodic influences on the male. Bissonnette (*3*) subjected male goats, which normally breed on the short days of autumn, to a short photoperiod in the spring. As a result of this treatment mature sperm were produced at a time when the seminal epithelium is normally quiescent. Ortavant *et al.* (*4*) showed that testis weight and sperm production in the ram was maximum on a photoperiod of LD 8:16 (*5*). Photoperiods shorter or longer than 8 hours resulted in loss of testicular weight, as well as in decrease in spermatogenesis. Several studies have