the distribution of lanternfish populations suggests that they are maintained and integrated by patterns of circulation (*1*). Although more rigorous physical analysis is required to either confirm the existence of the proposed westward current at 40°S to 45°S or provide a more parsimonious explanation for the observed distribution of isohalines, the existence of counterclockwise circulation in Pacific subantarctic waters would provide a mechanism for the maintenance of endemic species as well as the eastward attrition of a significant number of more widely distributed subantarctic and transitional region species. The fact that additional widespread subantarctic as well as transitional-region lanternfish species do occur in this region might be attributable to differences in physiological tolerances or vertical distribution.

It would be worthwhile to closely examine other pelagic taxa for similar patterns of distribution in Pacific subantarctic waters. The appearance of such patterns in planktonic protists, for example, might affect paleoceanographic conclusions based on the distribution of protistan microfossils in subantarctic sediments. On the other hand, it seems possible that myctophid fossils might be common enough in pelagic sediments to at least provide an additional reference for paleoceanographic studies. Not only are the lanternfish very abundant and the most speciose group of oceanic fish, they are frequently present in exposed Cenozoic and Pleistocene fossil deposits and are known to occur (especially otoliths) in pelagic sediments. The approximately 200 Recent species, the distributions of which appear to dramatically reflect hydrology, represent a remarkable radiation of low-level carnivores in the World Ocean pelagic environments. It seems probable that the Myctophidae could become increasingly important for deciphering the evolution of oceanic ecosystems.

Should the existence of the proposed westward current at 40°S to 45°S be confirmed by more rigorous physical analysis, it could appropriately be named the Deacon Current, in honor of Sir George Deacon, who first suggested its possible existence and who gave oceanography a model of the Southern Ocean which has withstood the demanding test of time.

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Gamma-Aminobutyric Acid Effects on Pituitary Gonadotropin Secretion

Abstract. *Gamma-aminobutyric acid (GABA) injected into the third ventricle of male rats promotes the release of pituitary luteinizing hormone (LH) but not follicle-stimulating hormone. When GABA was injected directly into the pituitary it was ineffective in promoting LH release. This evidence suggests that GABA may play a role in controlling the discharge of hypothalamic luteinizing hormone releasing factor.*

It is generally accepted that neurohormonal substances (releasing and inhibiting factors) elaborated by the hypothalamus control hormone secretion from the anterior pituitary gland. However, the neural pathways and neurotransmitters that regulate neurohormone secretion have not been completely established. Recently, considerable interest has focused on putative neurotransmitters that may alter the secretion of these hypothalamic neurohormones. For example, certain biogenic amines appear to promote secretion of the pituitary gonadotropins, luteinizing hormone (LH) and follicle-stimulating hormone (FSH), and this effect is presumably mediated by a hypothalamic releasing factor (*1*).

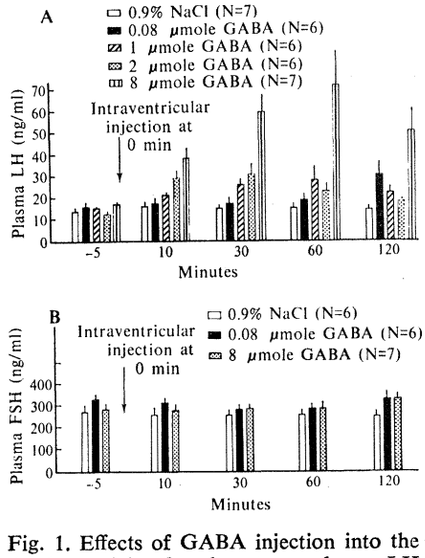

Fig. 1. Effects of GABA injection into the third ventricle of male rats on plasma LH (A) and FSH (B). Numbers of animals per group are in parentheses, and vertical lines are standard errors of the mean.

For several years certain amino acids within the central nervous system have been found to produce alterations in neuronal function, possibly acting as neurotransmitters (*2*). Gamma-aminobutyric acid (GABA), for example, is found in high quantities in the rat brain and may account for transmission at a large number of synapses (*3*). This acid has also been detected within nerve terminals in various brain regions, with high concentrations found in the diencephalon (*3*), an area known to participate in the physiologic control of reproductive function. Until now, however, there has been no evidence for the participation of GABA in the regulation of pituitary gonadotropin secretion. This preliminary report provides evidence that GABA can alter pituitary secretion of LH and that this effect is mediated by the hypothalamus.

Male Sprague-Dawley rats weighing 275 to 350 g were anesthetized with sodium pentobarbital (35 to 40 mg/kg). Throughout each experiment, the animals were ventilated through an endotracheal tube by means of a rodent respirator. The ventral diencephalon and pituitary were exposed by using a parapharyngeal approach (*4*). Test solutions were then injected into the cerebrospinal fluid of the third ventricle or into the anterior pituitary. A glass microcannula was used for all injections. GABA was dissolved in 0.9 per-

cent saline and the *p*H was adjusted to 7.2. A small quantity of the dye lissamine green was added to each solution to act as a marker providing proof of placement of cannulas, and in order to follow the dispersion of the solution after injection. The volume of solution injected was always 2.0 μl. For control purposes, 2 μl of 0.9 percent saline containing lissamine green was injected. Blood samples were removed from a cannulated femoral artery before the test solutions were administered and at designated times thereafter during the 2-hour observation period. Peripheral levels of LH and FSH were measured by radioimmunoassay (*5*). The data were analyzed by using a two-way analysis of variance and Duncan's multiple range test for statistical differences between means (*6*).

The injection of 1, 2, or 8 μmole of GABA into the third ventricle markedly stimulated the release of LH (Fig. 1A). The plasma LH variations were highly significant with time and treatment ($P < .001$). The rise in LH appeared to be related to the dose of GABA. The highest dose, 8 μmole, caused a dramatic rise in LH, with peripheral levels doubling within 10 minutes after treatment, attaining concentrations within 30 minutes that were four- to fivefold greater than resting levels. Smaller but significant elevations in LH were observed with lesser amounts of GABA (1 and 2 μmole). Further evaluation of these data by Duncan's multiple range test indicated that all treatment means were significantly greater than that from animals given 0.9 percent NaCl ($P < .05$). The intraventricular injection of the lowest dose of GABA, 0.08 μmole, or 0.9 percent saline, did not alter LH levels significantly. Plasma FSH levels in these same animals were not altered by any dose of GABA injected into the third ventricle (Fig. 1B). Levels of FSH remained constant during the 2-hour observation period.

In contrast to the effect when injected intraventricularly, there was no significant difference in plasma LH levels following the injection of 8 μmole of GABA or 0.9 percent saline into the anterior pituitary (Fig. 2). Evaluation of these data by means of a two-way analysis of variance revealed a slight but significant ($P < .05$) variation with time for both groups. This suggests that volume injections per se into the anterior pituitary may cause a slight release of pituitary LH. The levels of FSH, however, were not influenced by these same manipulations (data not shown). These results indicate that the ability of GABA to promote LH release is not due to a direct effect on the anterior pituitary gland, but requires elements within the central nervous system. However, the possibility that intraventricular injections are a better means of distributing GABA throughout the anterior pituitary, via the capillary plexuses, than intrapituitary injections has not been ruled out in this study.

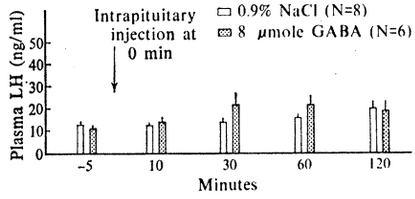

Fig. 2. Effects of GABA injection into the anterior pituitary on the plasma LH levels.

The observation that GABA injections into the third ventricle can promote release of LH and not FSH appears somewhat contrary to the concept that one hypothalamic LH releasing factor (LRF) exists for releasing both pituitary LH and FSH (*7*). One possible mechanism for a differential release of the gonadotropins can occur as a result of the concentration of LRF entering hypophyseal portal blood. For example, our previous data showed that the infusion of synthetic LRF into a hypophyseal portal vessel can stimulate both LH and FSH release, but the doses of LRF required for FSH release are much higher than those needed for the release of LH (*8*). Complete resolution of this problem will have to wait for future investigations.

The importance of GABA as a transmitter substance in crustaceans is generally accepted, while evidence for a physiological role in the central nervous system in mammals is increasing. Early studies have associated GABA with a role as an inhibitory neurotransmitter, although such evidence is not complete for the mammalian nervous system (*9*). In addition to its neurotransmitter role, GABA also appears to subserve a variety of metabolic functions within the brain. Products of GABA metabolism include intermediates in the tricarboxylic acid cycle, and other brain compounds, some of which possess neurophysiological properties (*9*). The present study suggests that GABA may be involved in the neural control of pituitary LH release. However, the site or mechanism of GABA action within the brain cannot be determined. GABA, for example, may be acting as a neurotransmitter directly affecting diencephalic elements or modulating the action of other synaptic transmitters that control the secretion of the neurohormone. The possibility also arises that GABA effects on LH release are related to changes in brain metabolites. The present findings emphasize a possible role for GABA in the control of reproductive function, adding another compound to the increasing list of putative neurotransmitters that may act neurophysiologically to regulate pituitary LH secretion.

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