

and might theoretically mediate stress-induced GH suppression in this species. Even though sufficient catecholamine disruption had occurred to disturb normal rhythmic GH secretion, suppression of GH by stress was only slightly affected in treated animals (76·6–89·0% of controls, depending on the method of assessment) and failed to reach statistical significance. The physiological function of noradrenergic afferents to the median eminence in GH regulation, therefore, seems to be primarily that of regulating the rhythmicity of the GH pulsatile rhythm. Noradrenergic afferents may participate slightly in the stress response but do not appear to be essential for it.

Although systemic 6-OHDA also disrupts noradrenergic innervation of the other circumventricular organs, a significant function for these structures in GH regulation has not yet been established. It seems likely, in view of the central role of the median eminence in pituitary regulation, that the effects of 6-OHDA are due to disruption of median eminence noradrenergic afferents.

Because a histochemical fluorescence method has been used in this study, the extent of noradrenergic denervation cannot be quantified. Evidence that denervation is functionally effective, so far as GH regulation is concerned, is that the time course of the disturbance of GH regulation correlates with that of the histochemically observed depletion in fluorescence in the median eminence (Day and Willoughby 1980). Furthermore, in this model, there appears to be no functional impairment of dopaminergic neurones, as judged by normal basal levels of prolactin after 6-OHDA (Day and Willoughby 1980). It is known that systemically administered 6-OHDA causes degeneration of peripheral autonomic noradrenergic nerves, but does not impair adrenal medullary function. Moreover, maintenance of normal blood pressure immediately following such treatment depends on a compensatory increase in adrenal function (Kostrzewa and Jacobowitz 1974). As a consequence, if adrenaline and noradrenaline in the systemic circulation participate in stress suppression of GH by an action at the median eminence (outside the blood brain barrier), it might be expected that GH concentrations would be suppressed following systemic 6-OHDA treatment. Even though this is not the case, it may be possible for a further stress-induced rise in circulating adrenaline to effect stress-induced suppression of GH, so obscuring any change in GH regulation produced by 6-OHDA. To the present time, however, amines and peptides of peripheral origin have not been thought to participate in pituitary regulation in physiological circumstances.

This study also indicates that the Porton strain of rat is relatively resistant to stress-induced GH suppression. It is known that GH secretion in the Sprague Dawley rat may remain fully suppressed for 5 h after a stress identical to the one used here (Terry *et al.* 1976). We have evidence that the DA Agouti strain also is extremely stress-sensitive, for when simply caged in isolation these rats fail to thrive and do not secrete GH (M. F. Menadue and J. O. Willoughby, unpublished data). Although certain neuroendocrine responses to stress may be important adaptive mechanisms, the variation in GH stress responses across rat strains and across many mammalian species (Martin 1976) makes it unlikely that the GH response to stress is an important aspect of stress physiology.

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