

Multiple Actions of Melanotropic Peptides in the Teleost *Oreochromis mossambicus* (Tilapia)

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In teleosts, two melanotropic peptides regulate the skin melanophores in an antagonistic manner: MCH (melanin-concentrating hormone), and α -MSH (α -melanocyte stimulating hormone). In addition to their roles in background adaptation, other functions have been postulated for these peptides in both teleosts and mammals. Effects of the peptides have been documented on behavior, immune function, and the neuroendocrine stress response.¹ Accordingly, influences other than changes in background color have been demonstrated to stimulate or inhibit α -MSH and/or MCH secretion.² Recently, we have shown the remarkable steroidogenic potency of the secretory signal from the neurointermediate lobe of tilapia.³ The presence of α -MSH in this signal could be shown to be essential for this effect, and in particular diacetyl- α -MSH has a strong corticotropic potency.⁴ FIGURE 1 demonstrates that communication between the melanotropes and the interrenal cortisol producing cells is bidirectional: cortisol, when administered *in vitro* at a concentration measured previously in this species during the initial phase of the stress response, significantly inhibits the sensitivity of the pars intermedia cells to CRH (corticotropin-releasing hormone), without affecting the unstimulated α -MSH release. Areas under the curves (arbitrary units) were 1198 ± 311 for the controls, 93 ± 59 after cortisol treatment [mean \pm standard error of the mean (SEM), $p < 0.001$; $n = 6$ for both groups]. A control experiment indicated that the cortisol effect could not be attributed to depletion of the melanotropes, since two consecutive CRH pulses, administered at $t = 4$ h and $t = 8$ h without cortisol pretreatment, elicited statistically indistinguishable responses: 873 ± 125 versus 845 ± 248 ($n = 4$; area under the curve). Direct feedback of corticosteroids on melanotropes has to date not been demonstrated in other vertebrate classes, although indirect mechanisms of action have been suggested.⁵ The present results suggest that the melanotropes might be an integral part of the pituitary-interrenal axis in teleosts. The release of the second melanotropic peptide, MCH, has also been demonstrated to be under the negative influence of corticosteroids.¹ However, the authors could not demonstrate a direct *in vitro* action of MCH on interrenal cortisol release. TABLE 1 illustrates that, in tilapia, immunoadsorption of endogenously released MCH *in vitro* reversibly affects the release of α -MSH. MCH, as other hypothalamic neuropeptides in teleosts, is released in the proximity of the melanotropes, and the release of these neuropeptides

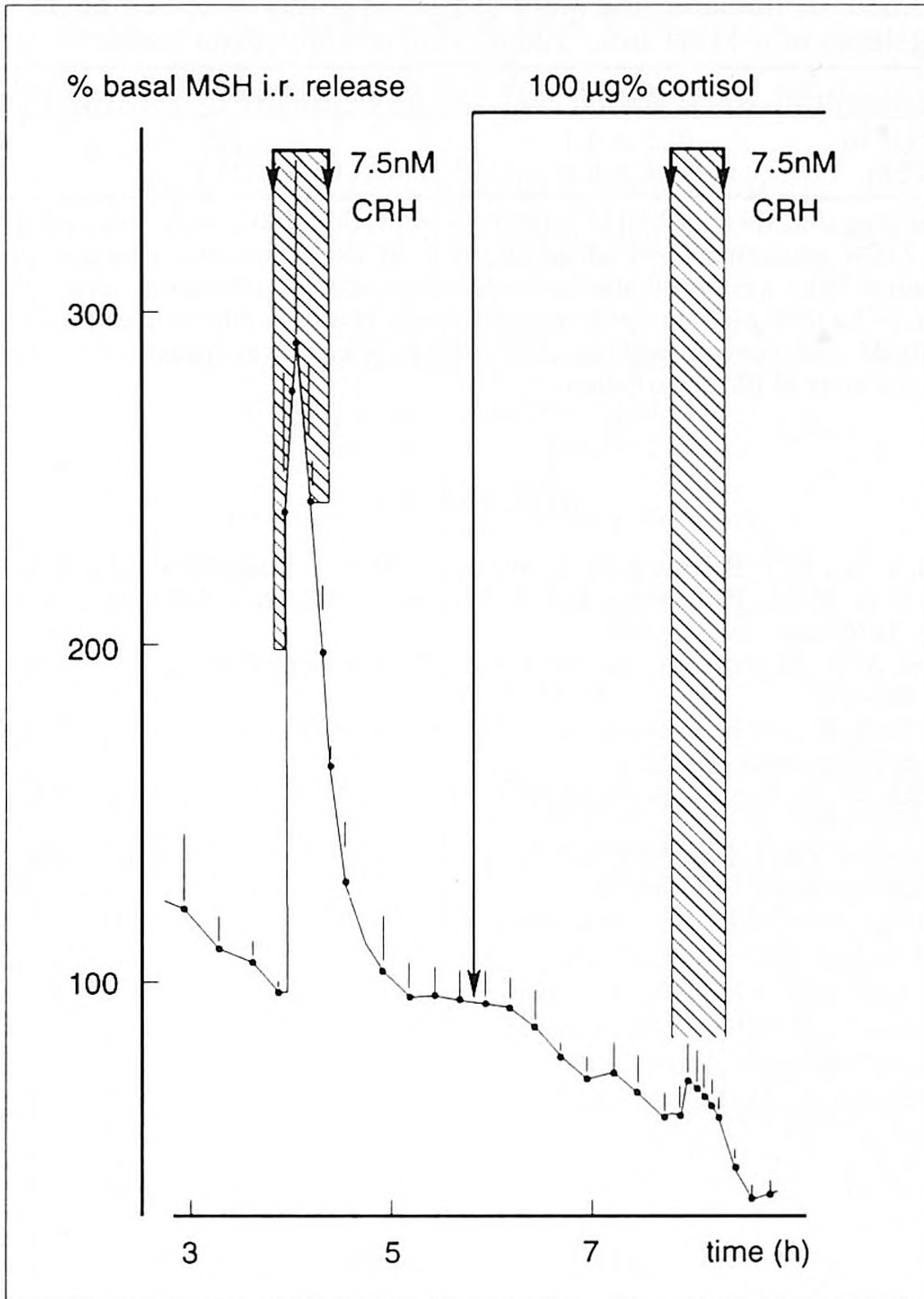


FIGURE 1. *In vitro* effect of cortisol on the release of α -MSH from the neurointermediate lobe in *Oreochromis mossambicus* (tilapia). Results are expressed as percent of basal release, which is defined as the average release rate during three fractions just prior to the first CRH (oCRH; Peninsula) pulse. α -MSH is measured in the superfusion² fractions by means of a MSH radioimmunoassay.² Differences between the pulses were assessed by subjecting the data to the Mann-Whitney *U* test (see text). $\mu\text{g}\%$ (w/v).

persists when the neurointermediate lobe is incubated *in vitro*. This experiment indicates that the α -MSH release is under tonic inhibitory control by MCH. This has been shown for rainbow trout previously,⁶ and therefore appears to be a general phenomenon in teleosts.

Taken together, the results presented suggest that MCH might regulate interrenal cortisol secretion via regulation of the melanotropes, which are under feedback control by corticosteroids.

TABLE 1. Effect of Immunoabsorption of Endogenously Released MCH on the *In Vitro* Release of α -MSH from Tilapia Neurointermediate Lobes^a

	Controls (<i>n</i> = 4)	MCH Adsorbed (<i>n</i> = 4)	
Treatment (15 h)	10.1 ± 3.1	25.4 ± 2.6	<i>p</i> < 0.01
Recovery (2 h)	61.4 ± 9.8	66.0 ± 11.3	NS

^a Release in pg α -MSH min⁻¹ NIL⁻¹. Immunoabsorption: NILs were cultured in medium² with 0.1% MCH antiserum (v/v) added (a gift from Dr. Kawauchi, Kitasato University, Japan). Control NILs were incubated in the presence of 0.1% preimmune serum. Recovery: At *t* = 15 h, NILs from both groups were individually placed in microsperfusion chambers² and superfused with control medium. The α -MSH release was measured between 2 and 2.5 h after the start of the superfusion.

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