γ-MSH POTENTIATES ACTH ANALGESIA. J.M. Walker*, H. Akil, S.J. Watson*, Mental Health Research Institute, University of Michigan, Ann Arbor, MI 48109, USA

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Introduction: β-endorphin derives from a 31K dalton glycoprotein termed pro-opiocortin. Other products of pro-opiocortin include one or more ACTH related peptides and a 16K dalton fragment which is under investigation. The discovery that these cells produce and package several biologically active peptides raises many new questions about the nature of synaptic transmission. We previously showed the ACTH and α-MSH are potent analgesics following PAG administration, suggesting homolgous actions of β-endorphin and the ACTH-like connterparts. We now suggest that γ-MSH 1-12, a portion of the N-terminal fragment of pro-opiocortin does not produce analgesia. However, it promotes the analgesic effects of ACTH 1-24.

Methods: Permanent indwelling cannulas aimed for the periaqueductal gray were implanted in 14 male rats (250-350 gr) under deep barbituate anesthesia. After one week of recovery the animals were given four tests to measure the analgesic effects of ACTH 1-24 and γ-MSH 1-12 alone and in combination. Analgesia was measured using tail-flick test. Half the animals received 17nm γ-MSH, the other half received 32nm.

Results and Discussion: As in a previous report ACTH 1-24 was found to produce a significant rise in tail-flick latency after micro-injection in the PAG (ANOVA). γ-MSH alone produced no statistically reliable change in tail-flick latency but when administered in combination with ACTH the combined effect was nearly twice that of ACTH alone. At 17nm similar effects were observed but as expected the degree of potentiation was smaller. It thus appears that one function of dual release may be to modulate the efficacy of the synaptic junction. If so the effectiveness of β-endorphin in modulating pain sensitivity might also depend on the rate of synthesis or release of other pro-opiocortin products.

ALTERATIONS IN STRESS-INDUCED ANALGESIA: EFFECTS ON STRESSFUL OR ANALGESIC PROCESSES. R.J. Bodnar, M.M. Wallace, D. Badillo-Martinez*, J. Kordower*, D. Simone*, A. Kirchgessner*, N. Nicotera* and K.P. Merrigan*, Department of Psychology, Queens College, C.U.N.Y., Flushing, NY 11367, USA

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Aim of Investigation: Acute exposure to a wide range of stressors results in a transient analgesia that can be eliminated or attenuated depending on the stressor employed. However, stress-induced analgesia can be reduced by changes in either the analgesic mechanisms subserving the response or the organism's perception of the stressful consequences of the stressor. This study investigated both possibilities for the analgesic response to cold-water swims (CWS), which is attenuated by hypophysectomy (HYPOX), neonatal monosodium glutamate (MSG) and D-phenylalanine (D-PHEN).

Methods and Results: Matched groups of control, HYPOX, neonatal MSG and D-PHEN-treated rats were tested both before and after CWS (2°C for 3.5 min) for pain thresholds, hypothermia and activity levels. CWS significantly increased pain thresholds, lowered core and skin temperatures, and increased activity levels over pre-swim levels in control rats. Both HYPOX and D-PHEN rats displayed significant attenuations in pain thresholds relative to controls while maintaining post-CWS hypothermic and hypermotile responses. By contrast, neonatal MSG reduced both the analgesic and hypothermic alterations induced by CWS.

Conclusions: It appears that when HYPOX and D-PHEN reduce CWS analgesia, they act upon analgesic pathways since other stressful consequences of CWS are unaffected. By contrast, the attenuation of CWS-induced analgesic and hypothermic alterations by MSG suggest that this manipulation may be changing the organism's perception of stress rather than an analgesic system. Alterations for other stressors will be discussed.

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