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PROCEEDINGS OF THE NUTRITION SOCIETY

ABSTRACTS OF COMMUNICATIONS

The Three Hundred and Sixtieth Meeting of the Nutrition Society was held at the Bristol Polytechnic, Coldharbour Lane, Bristol, on Wednesday, 8 *April,* 1981 *when the following papers were read.*

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The effects of chronic treatment with the putative anorexogenic peptide (Glu-His-Gly), on body-weight and food consumption in rats. By D. M. ANDERSON, *Department of Pharmacology, Scientific Development Group, Organon Laboratories Limited, Newhouse, Lanarkshire*

It has been reported (Reichelt *et al.* 1978) that patients suffering from anorexia nervosa excrete in their urine, a factor which when administered to mice inhibits food intake and reduces body-weight. The active principle in this urine extract has been shown to be pyro Glu-His-Gly OH. However, other workers have been unable to repeat these findings in rats (Knoll, 1979; Nance *et al.* 1979) or in Mice (Bjorkman *et al.* 1980). In order to investigate this matter further the tripeptide was synthesized and tested in adult Wistar rats.

Four groups of ten rats each (average weights 230–240 g) were housed in individual cages at an ambient temperature of $25\pm1°$ and allowed to acclimatize to their surroundings. Two groups of rats were given daily subcutaneous injections of vehicle alone, while the other two groups received the tripeptide, 0·04 mg/kg and 0·02 mg/kg per d respectively. All animals were treated for 28 d and food intake and body-weight were measured daily. To solubilize the tripeptide, 1 mg was dissolved in 0·05 ml 0·05 M-HCl and made up to 50 ml in distilled water. The pH of this solution was then adjusted to 5 using 0·05 M-NaOH.

During the first 7 d of treatment, the tripeptide was without effect on food intake but from day 7 onwards the rats receiving the higher dose consistently ate less food (average 1–3 g/d) than the other three groups. The effect of the tripeptide on body-weight was more marked. The control groups of rats gained 129 ± 6 and 135 ± 7 g respectively during the 25 d of treatment while the rats receiving 0·02 or 0·04 mg/kg tripeptide gained 119 ± 8 and 103 ± 5 g respectively over the same period. The effect of the higher dose of peptide was significant ($P<0·05$) and was similar in magnitude to that observed during treatment with oral fenfluramine (10–15 mg/kg per d). After treatment was stopped in all groups, the rats treated with 0·04 mg/kg per d remained 10–15 g lighter than the other three groups during the next 6 months.

Thus in rats, the putative anorexogenic tripeptide, pyro Glu-His-Gly-OH, can produce a significant and sustained decrease in body-weight.

I should like to thank Dr Greven, Department of Chemistry, Organon International B.V., Oss, The Netherlands, for synthesizing the tripeptide.

Bjorkman, S., Karlsson, J. A., Sievertsson, H., Lewander, T. & Bowers, C. Y. (1980). *Acta Pharm. Suec.* **17**, 130.
Knoll, J. (1979). *Physiol. Behav.* **23**, 497.
Nance, D. M., Coy, D. H. & Kastin, A. J. (1979). *Biochem. Behav.* **11**, 733.
Reichelt, K. L., Foss, I., Trygstad, O., Edminson, P. D., Johansen, J. H. & Boler, J. B. (1978). *Neuroscience* **3**, 1207.

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Sex and trenbolone acetate (TBA) anabolic activity. By ANGELA TOONG and B. N. PERRY, *ARC Meat Research Institute, Bristol* and A. LOPEZ and J. VENTANAS, *University of Leon, Spain*

In cattle TBA (3-oxo-17β-hydroxy-4,9,11-estratriene acetate) is efficacious when used alone or with oestrogens (Heitzman, 1979). However, its loci or mode of action are unknown. In rats TBA-induced anabolism is sexually dimorphic; intact females but not castrated males respond (Vernon & Buttery, 1978).

Growth in rats treated daily with TBA subcutaneously (80 μg/100 *g body-weight*)

(Mean values with standard errors, litters equally represented)

	n	Age on test (d)	Mean initial weight (g)	Mean weight gain (g)	Significance of differences in weight gains
O—vehicle	6	45–61	167·3±6·0	61·8±2·4	
O—TBA	6	45–61	166·7±6·5	83·0±4·2	$P<0.001$
L—vehicle	9	58–71	186·1±2·8	54·8±3·5	
L—TBA	5	58–71	186·6±2·2	82·4±3·9	$P<0.001$
A—vehicle	8	29–52	91·8±5·4	110·3±2·6	
A—TBA	7	29–52	92·6±4·2	113·4±6·0	N.S.
I—vehicle	9	45–61	189·9±4·0	96·1±2·7	
I—TBA	8	45–61	191·0±4·6	94·9±3·8	N.S.

That ovariectomized (O) and LHRH-autoimmune (L) female rats also respond to TBA (see Table) suggests TBA does not simply restrict oestrogen–receptor complex action, unless it modifies adrenal steroidogenesis or peripheral enzymic oestrogen production from adrenal secretions. Such effects are unknown to us, although antagonism in muscle of glucocorticoid receptors has been implicated in TBA action (Buttery *et al.* 1978). However, TBA neither stimulates growth in castrated nor in intact males (I) or androgenized females (A) (see Table). Therefore, such a mechanism would require sexual dimorphism in muscle glucocorticoid receptors. We know of no such results but bone exhibits sexual dimorphism in sex-steroid receptors during development in mice. Male tibiae show substantial but female tibiae little oestradiol specific-binding up to 21 d of age (2150 *v.* 150 disintegrations/min ^3H per mg cytosol protein). A similar sex difference in testosterone specific-binding exists later (3100 *v.* 100 disintegrations/min ^3H per mg cytosol protein at 42 d of age).

Buttery, P. J., Vernon, B. G. & Pearson, J. T. (1978). *Proc. Nutr. Soc.* **37**, 311.
Heitzman, R. J. (1979). *J. Steroid Biochem.* **11**, 927.
Vernon, B. G. & Buttery, P. J. (1978). *Anim. Prod.* **26**, 1.

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