60 YEARS OF NEUROENDOCRINOLOGY

TRH, the first hypophysiotropic releasing hormone isolated: control of the pituitary–thyroid axis

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The authors and journal apologise for an error in the above paper, which appeared in volume 226 part 2, pages T85–T100. The error relates to the artwork of Figure 2 on page T87, where ‘aMSH’ and ‘NPY/AgRP’ were transposed in the key. The correct Figure 2 is published in full below:

Figure 2

Elements involved in HPT regulation. At the level of the paraventricular hypothalamic nucleus (PVN), *Trh* mRNA is transcribed, its expression is regulated by multiple effectors, processed TRH is released from terminals localized at the median eminence (ME) in juxtaposition with tanycytes that contain deiodinase 2 (D2) and pyroglutamyl peptidase II (PPII). In response to nutrient status, arcuate neurons synthesizing POMC/CART or

NPY/AgRP project to the PVN and activate or inhibit (respectively) TRH neurons. Released TRH may be degraded by PPII before reaching portal vessels that transport it to the pituitary where it controls synthesis of TSH and glycosylation of both TSH subunits (a and b) to form bioactive TSH. At the thyroid, TSH stimulates synthesis and release of T_4 that is modified at target tissues by deiodinases (e.g. D1 and D2).