The physiological responses to the use of MDMA whilst clubbing: posterior pituitary function and fluid balance

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It is estimated that there are half a million users of 3,4-methylenedioxymeth-amphetamine (MDMA, “ecstasy”) every week in the UK, use being predominantly associated with clubbing. However the metabolic effects of MDMA are not well described. MDMA given to subjects in a laboratory setting stimulated vasopressin (Forsling et al. 2001) and a study has now been performed “in the field” on 51 self nominating drug users (18 women, 33 men) with a mean age of 25 years. All recruits were experienced clubbers who on average had been clubbing for 6.6 yrs, the majority (49/51) smoked cigarettes and all had used ‘ecstasy’ previously.

The study was performed with the consent of the local ethics committee and all participants gave written consent. Subjects attended the study centre before going clubbing and returned at the end of the evening. Pre-and post clubbing measurements of body weight, pulse rate and sitting and standing blood pressure were performed and urine and blood samples obtained to determine parameters of fluid balance and plasma hormone concentrations and to confirm drug use.

Of the 31 subjects whose urine tested positive for a psychoactive substance, 21 clubbers screened positive for MDMA. Eight clubbers had blood alcohol levels ranging from 23–106 mg/dl, of which only one used in combination with MDMA. Two clubbers had tested above the legal limit for driving (80 mg/dl). The other main substance used was cannabis The average, resting pre-clubbing pulse rate was 78 (range 60–120) BPM rising to 99.3 (range 140–170) BPM upon return to the club site. There were 18 post-clubbing subjects who had BPM>100. Five post clubbers (range 140–170) BPM upon return to the club site. There were 18 post-clubbing subjects who had BPM>100. Five post clubbers

The role of calcitonin gene-related peptide (CGRP) in stress induced suppression of LH pulses in the rat

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CGRP is involved in a variety of stress responses. Central administration of CGRP causes fear related behaviour, activates the sympathetic system and the hypothalamo-pituitary-adrenal (HPA) axis, specially enhancing corticosterone release. CRH antiserum blocks the CGRP-induced rise in corticosterone (Kovacs et al. 1995). CRH, which is central to the HPA axis, plays a pivotal role in stress-induced suppression of pulsatile LH secretion. The aims of the present study were to test the hypothesis that CGRP suppresses LH pulses and to determine whether CRH mediates this response.

Ovariectomized (ketamine, 100 mg kg⁻¹, ip) Wistar rats were implanted with intracerebroventricular (icv) and intravenous (iv) cannulae (ketamine, 100 mg kg⁻¹, ip). Blood samples (25 μl) were collected every 5 min for 6 h and assayed for LH. After 2 h of sampling CGRP (75, 400, or 1200 pmol) or artificial cerebrospinal fluid (4μl) were administered by icv injection (n = 6–12). In addition, CGRP (400 pmol) was co-administered icv with the CRP antagonist (CGRP₈–₃₇, 1 nmol, n = 8) or CRH antagonist (α-helical CRF₉–₄₁, 26 nmol, n = 11). Finally rats were given 2 nmol CGRP₈–₃₇ and 10 min later 0.5 U kg⁻¹ insulin was given iv to induce hypoglycaemia; a stress known to suppress LH (Cates & O’Byrne, 2000). Animals were humanely killed.


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All procedures accord with current UK legislation.