Errata

 $\overline{}$, we are the property of $\overline{}$

Seebohm G, Sanguinetti MC & Pusch M (2003). Tight coupling of rubidium conductance and inactivation in human KCNQ1 potassium channels. *J Physiol* **552**, 369–378.

On page 371, Equation 2 should have appeared as:

$$
I_{\infty} = \frac{p_I}{p_I + p_{O_2}} = \frac{\lambda}{\lambda + \mu} = \frac{a_f/a_s - \tau_f/\tau_s}{1 - \tau_f/\tau_s}
$$
\n⁽²⁾

Pollock B, Gross J, Dirks M, Timmermann L & Schnitzler A (2004). The cerebral oscillatory network of voluntary tremor. *J Physiol* **554**, 871–878.

On page 874, it was stated in the second paragraph that the distribution of phase differences between S1/M1 and EMG showed peaks at certain points. These stated values should have read -170 ± 7.5 deg, -78.2 ± 5.4 deg, 69.5 ± 4.2 deg and 159.1 ± 6.5 deg (mean \pm s.e.m.)

Lyall V, Alam RI Malik SA Phan THT, Vinnikova AK, Heck GL & DeSimone JA (2004). Basolateral Na+-H⁺ echanger-1 in rat taste receptor cells is involved in neural adaptation to acidic stimuli. *J Physiol* **556**, 159–173.

On page 169, part of the third paragraph was obscured by Fig. 12. The text should have read:

In polarized TRCs, stimulating the apical membrane with acidic stimuli induced sustained decreases in pHi (Lyall *et al*. 2001, 2002*a*,*b*). Thus both strong acids and weak organic acids gain entry into TRCs across the apical cell membrane and induce a decrease in pH_i. Weak organic acids permeate the apical membrane as neutral molecules, and strong acids via an H⁺ entry pathway that is both amiloride- and Ca²+-insensitive, but is activated by cAMP (Lyall *et al*. 2001, 2002*a*,*b*; DeSimone *et al*. 2001*b*). During acid stimulation, a decrease in TRC pH_i, rather than a decrease in pH₀, is the stimulus intensity variable that correlates specifically with increased CT taste nerve activity. Since inhibiting acid-induced TRC acidification also inhibits the acid-evoked CT response (Lyall *et al*. 2001, 2002*b*), it indicates that a decrease in TRC pHi is the proximate stimulus for sour taste.

Martinez V, Wang L, Rivier J, Grigoriadis D & Tache Y (2004). Central CRF, urocortins and stress increase colonic transit via ´ CRF1 receptors while activation of CRF2 receptors delays gastric transit in mice. *J Physiol* **556**, 221–234.

On page 228, part of the second paragraph was obscured by Fig. 3. The text should have read:

Effects of I.C.V. CRF receptor antagonists on restraint stress-induced defecation. In mice maintained in non-stressful conditions, pellet output was low (2.0 ± 0.7 pellets h⁻¹, *n*=7). Restraint stress for 1 h increased defecation to 10.4 ± 1.3 pellets h⁻¹ (*ⁿ* ⁼ 10, *^P* < 0.05; Fig. 6*A*). The peak defecatory response occurred during the first 15 min of stress (5.7 [±] 0.6 pellets h−¹ *^P* < 0.05 *versus* non-stress: 0.3 ± 0.2 pellets h⁻¹), thereafter values decreased, although at 30 min, values were still significantly elevated (Fig. 6*B*). NBI-35965 at 50 or 100 μ g reduced stress-induced defecation to 4.8 \pm 1.0 and 4.0 \pm 1.5 pellets h⁻¹, respectively (*n* = 9 and 5; both $P < 0.05$ *versus* vehicle + stress; $F_{4,33} = 10.025$, $P < 0.001$) while astressin₂-B (10 μ g, i.c.v.), did not modify the colonic motor response to restraint stress (10.0 [±] 0.7 pellets h−1, *ⁿ* ⁼ 5; Fig. 6). None of the CRF receptor antagonists tested by themselves (NBI-35965, $n = 7$; astressin₂-B, $n = 4$), had a significant effect on pellet output in non-stressed mice.

Eskurza I, Monahan KD, Robinson JA & Douglas RS (2004). Effect of acute and chronic ascorbic acid on flow-mediated dilation with sedentary and physically active human ageing. *J Physiol* **556**, 315–324.

On page, 319, Table 2 should have appeared as follows:

