## Errata

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:

$$I_{\infty} = \frac{p_I}{p_I + p_{O_2}} = \frac{\lambda}{\lambda + \mu} = \frac{a_f/a_s - \tau_f/\tau_s}{1 - \tau_f/\tau_s}$$
(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  $-170 \pm 7.5 \text{ deg}$ ,  $-78.2 \pm 5.4 \text{ deg}$ ,  $69.5 \pm 4.2 \text{ deg}$  and  $159.1 \pm 6.5 \text{ deg}$  (mean  $\pm$  s.e.m.)

Lyall V, Alam RI Malik SA Phan THT, Vinnikova AK, Heck GL & DeSimone JA (2004). Basolateral Na<sup>+</sup>-H<sup>+</sup> 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<sub>i</sub>. Weak organic acids permeate the apical membrane as neutral molecules, and strong acids via an H<sup>+</sup> entry pathway that is both amiloride- and Ca<sup>2+</sup>-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<sub>i</sub>, rather than a decrease in pH<sub>o</sub>, 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 pH<sub>i</sub> is the proximate stimulus for sour taste.

Martinez V, Wang L, Rivier J, Grigoriadis D & Taché Y (2004). Central CRF, urocortins and stress increase colonic transit via CRF<sub>1</sub> receptors while activation of CRF<sub>2</sub> 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  $(2.0 \pm 0.7 \text{ pellets h}^{-1}, n=7)$ . Restraint stress for 1 h increased defecation to  $10.4 \pm 1.3 \text{ pellets h}^{-1}$  (n=10, P < 0.05; Fig. 6A). The peak defecatory response occurred during the first 15 min of stress  $(5.7 \pm 0.6 \text{ pellets h}^{-1} P < 0.05 \text{ versus non-stress}: 0.3 \pm 0.2 \text{ pellets h}^{-1})$ , thereafter values decreased, although at 30 min, values were still significantly elevated (Fig. 6B). NBI-35965 at 50 or 100  $\mu$ g reduced stress-induced defecation to  $4.8 \pm 1.0$  and  $4.0 \pm 1.5$  pellets h<sup>-1</sup>, respectively  $(n = 9 \text{ and } 5; \text{ both } P < 0.05 \text{ versus vehicle + stress}; F_{4,33} = 10.025, P < 0.001)$  while astressin<sub>2</sub>-B (10  $\mu$ g, I.C.V.), did not modify the colonic motor response to restraint stress ( $10.0 \pm 0.7 \text{ pellets h}^{-1}$ , n = 5; Fig. 6). None of the CRF receptor antagonists tested by themselves (NBI-35965, n = 7; astressin<sub>2</sub>-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:

Table 2.	Brachial artery parameters and hyperaemic flow
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Parameter		BA diameter (mm)	FMD (∆mm)	FMD (%)	HF (% increase)	EID (%)
Young sedentary	Baseline	4.1±0.1	0.33±0.02	8.1±0.5	314±21	20.7±0.9
	Acute	4.1±0.1	0.34±0.02	8.2±0.6	302±26	20.8±1.0
	Chronic	4.1±0.1	0.31±0.02	7.8±0.3	330±26	21.3±1.0
Older sedentary	Baseline	4.3±0.1	0.20±0.01*	4.6±0.2*	324±31	20.8±0.8
	Acute	4.3±0.1	0.29±0.02‡	6.7±0.3*‡	291±24	20.6±0.8
	Chronic	4.3±0.1	0.22±0.03*	5.4±0.4*	325±18	18.8±1.1
Older trained	Baseline	4.0±0.1	$0.28{\pm}0.02{\dagger}$	7.0±0.6†	273±17	21.7±2.2
	Acute	4.1±0.1	$0.30{\pm}0.01$	7.3±0.3	306±23	21.4±1.7
	Chronic	4.0±0.1	$0.28{\pm}0.01{\dagger}$	7.0±0.4†	305±38	22.5±1.9