



Corrigendum

The authors wish to apologise for errors in the paper 'Early increases in renal kallikrein secretion on administration of potassium or ATP-sensitive potassium channel blockers in rats' by Fujita *et al.*, British Journal of Pharmacology (1999), 128, 1275–1283. Figures 1, 2, 3 and 4 and corresponding text show incorrect values. The corrected figures and text appear below.

Results

Effect of sodium and potassium gluconate on urinary KK excretion

Figure 1 shows that the increase in urinary KK excretion during the 30 min infusion of potassium gluconate solution (21.7 ± 5.3 mU 30 min^{-1} 100 g b.wt.^{-1}) was larger than that of sodium gluconate solution (3.1 ± 3.8 mU 30 min^{-1} 100 g b.wt.^{-1}).

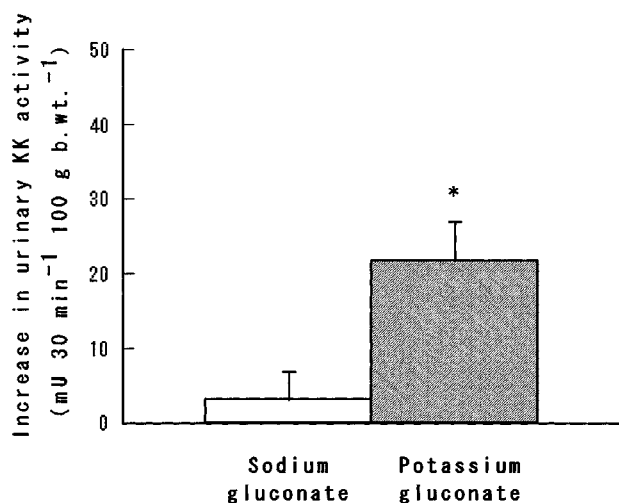


Figure 1 Increase in urinary KK excretion during the 30 min infusion of sodium and potassium gluconate. Values are expressed as mean \pm s.e. mean; $n = 5$ for the sodium gluconate group and $n = 6$ for the potassium gluconate group. After a 45 min intravenous infusion of saline, either sodium gluconate (30 mM) or potassium gluconate (30 mM) was substituted and infused for a further 30 min. *Significantly different from the sodium gluconate value, $P < 0.05$.

Effect of potassium chloride on urinary KK excretion and urine volume, and urinary excretion of sodium, potassium and chloride

In order to clarify whether other potassium solutions increase urinary KK excretion and whether urinary KK excretion was increased by potassium loading as result of the washout phenomenon, the effect of potassium chloride on urinary KK excretion, urine volume and urinary excretion of electrolytes were examined. Urinary KK excretion tended to increase immediately after infusion of the 75 mM potassium solution and significantly during the 30 min infusion of potassium (50.8 ± 3.1 mU 15 min^{-1} 100 g b.wt.^{-1}) in comparison with saline (40.6 ± 2.0 mU 15 min^{-1} 100 g b.wt.^{-1}). The increase in urinary KK excretion was maintained during the potassium infusion (Figure 2a). Urine volume and urinary excretion of sodium, potassium and chloride increased gradually during both the infusion of potassium and saline (Figure 2b–e).

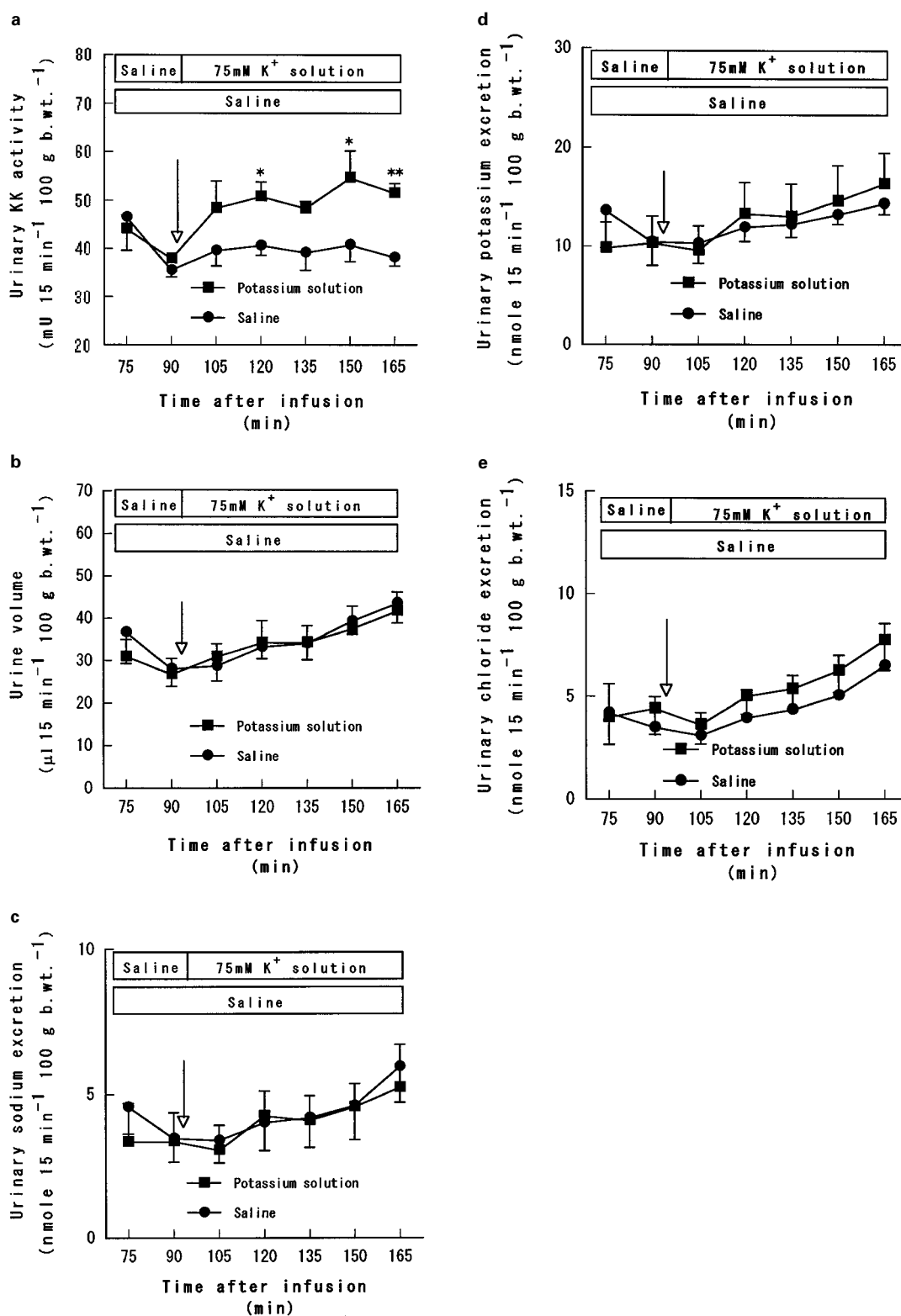


Figure 2 Changes with time in urinary KK excretion (a); urine volume (b); urinary excretion of sodium (c); potassium (d); and chloride (e) after infusion of (mm) K^+ 75, Na^+ 75 and Cl^- 150 solution or saline. Values are expressed as mean \pm s.e.mean; $n=5$ for each group. After intravenous infusion of saline for 90 min, either (mm) K^+ 75, Na^+ 75 and Cl^- 150 solution was substituted (indicated by the arrow in the figure) or saline was continued. The infusion was performed for a further 75 min. *,**Significantly different from corresponding control value, $P<0.05$, $P<0.01$.

Effects of PNU-37883A on urinary KK excretion and urine volume, and urinary excretion of sodium, potassium and chloride

Figure 3 shows changes in urinary KK excretion (a), urine volume (b), urinary excretion of sodium (c), potassium (d), and

chloride (e) after the administration of PNU-37883A at a dose of 10 mg kg^{-1} . Urinary KK excretion started to increase immediately after the administration of PNU-37883A ($23.9 \pm 2.7 \text{ mU } 15 \text{ min}^{-1} 100 \text{ g b.wt.}^{-1}$), peaked 30 min later ($26.2 \pm 1.8 \text{ mU } 15 \text{ min}^{-1} 100 \text{ g b.wt.}^{-1}$), and then decreased to its original level within the next 30 min ($17.3 \pm 4.3 \text{ mU}$

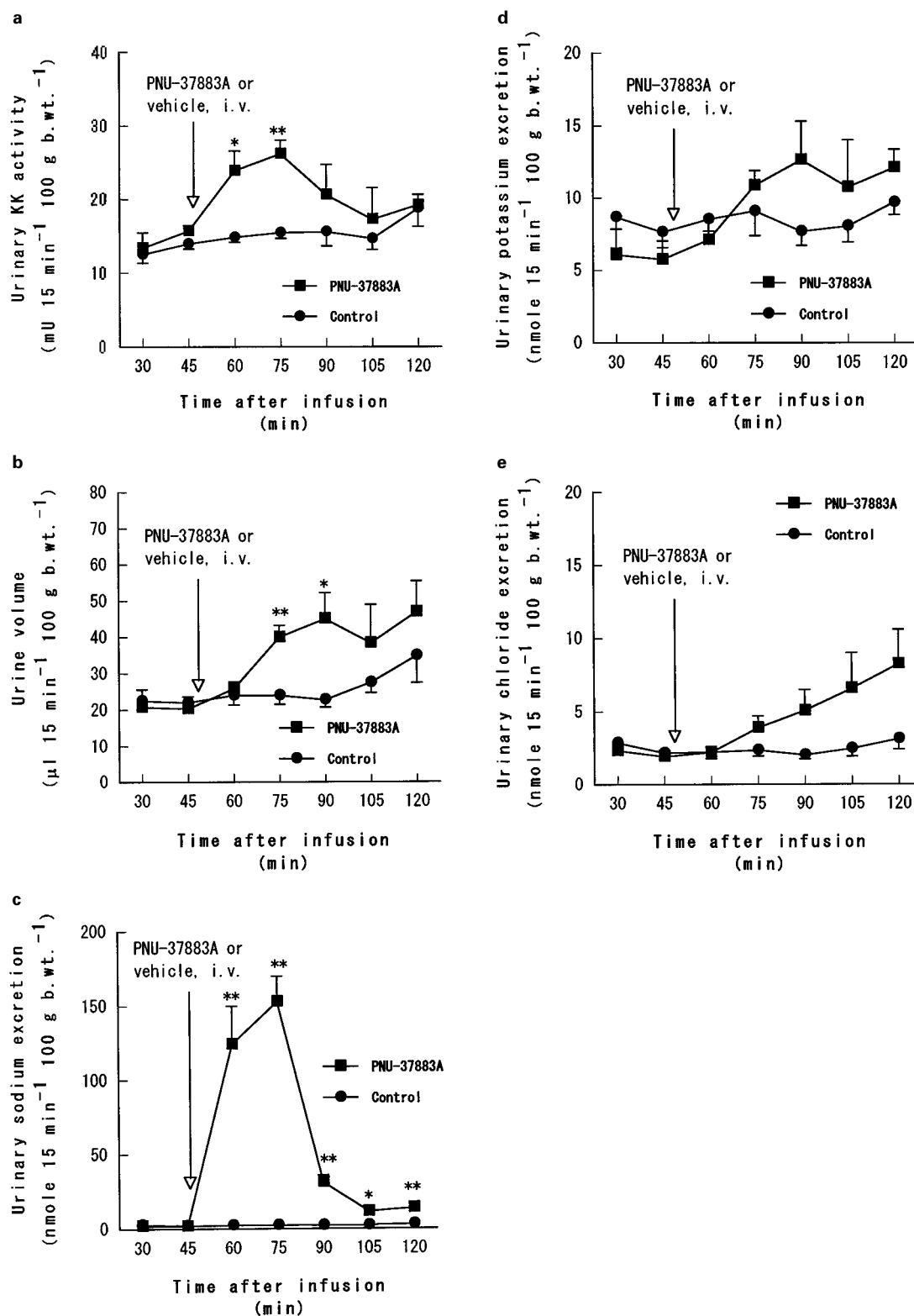


Figure 3 Changes with time in urinary KK excretion (a); urine volume (b); urinary excretion of sodium (c); potassium (d); and chloride (e) after the administration of either PNU-37883A or vehicle. Values are expressed as mean \pm s.e. mean; $n=6$ for PNU-37883A and $n=5$ for vehicle. Either PNU-37883A (10 mg kg^{-1}) or vehicle was administered 45 min after the start of infusion. *, **Significantly different from corresponding vehicle value, $P < 0.05$, $P < 0.01$.

15 min⁻¹ 100 g b.wt.⁻¹). In the control group, the urinary activity of KK corresponding to the maximum level resulting from PNU-37883A administration was 15.4 ± 0.8 mU 15 min⁻¹ 100 g b.wt.⁻¹. Urine volume increased 15 to 30 min after the injection of PNU-37883A (40.3 ± 3.3 vs 24.0 ± 2.4 (control) μ l 15 min⁻¹ 100 g b.wt.⁻¹) and peaked 30 min later (45.1 ± 7.2 vs 22.8 ± 2.0 (control) μ l 15 min⁻¹ 100 g b.wt.⁻¹). Urinary sodium excretion markedly increased immediately after the administration of PNU-37883A; whereas, changes in the urinary excretion of potassium and chloride after the administration of PNU-37883A were not significantly different from control.

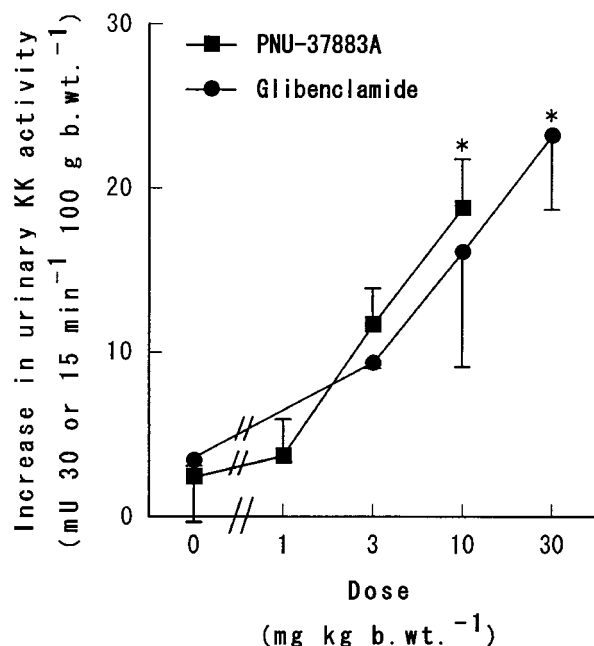


Figure 4 Increases in urinary KK excretion after administration of either PNU-37883A or vehicle and either administration of glibenclamide or vehicle 30 min and 15 min after either the administration. Values are expressed as mean \pm s.e.mean; $n=5$ for each vehicle, $n=4$ for PNU-37883A at a dose of 1 mg kg⁻¹ or 3 mg kg⁻¹, $n=6$ for PNU-37883A at a dose of 10 mg kg⁻¹, $n=3$ for glibenclamide at a dose of 3 mg kg⁻¹ or 10 mg kg⁻¹, $n=5$ for glibenclamide at a dose of 30 mg kg⁻¹. PNU-37883A, glibenclamide or vehicle was administered 45 min after the start of infusion.