isolation and clinical signifance.[2,3] Aerosol inhalation of the microorganism is therefore an important step in the development of oropharyngeal and tracheobronchial tree colonisation. Underlying lung disease, advancing age as well as concomitant immunosuppression are thought to further determine subsequent development of respiratory tract disease and in part correlate with an associated mortality of 31% in *P. multocida* associated pneumonia.[2,3]

P. multocida is a commensal of swine respiratory and gastrointestinal tracts and survival of *P. multocida* at scalding water tank temperatures is thought to explain contamination of most pigs during slaughter.[3] It is therefore speculated that *P. multocida* colonises pig trotters and in this case physically agitating the organism during soaking and cooking allowed fragmentation of biological material originating from porcine saliva or faeces. Transmission is likely to have been facilitated by uncovered boiling of the pig trotters which would not have killed *P. multocida* but rather promoted further airborne spread of droplets containing this unusual and virulent pathogen.

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letters to the editor

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'With a pinch of salt' revisited

Editor – I read with interest but some concern the recent lesson of the month (*Clin Med* February 2010 pp 86–7). The report highlights a case of severe hyponatraemia which the authors attribute to excessive sweating, poor fluid consumption and low salt intake in a hot environment. There is little evidence to suggest that a low salt intake would contribute to this event and the authors have not explored alternative likely explanations. During evolution, mankind has survived with very little salt in the diet. Even in modern times, this evidence is detectable in the Yanomano and Xingu Indians living in the humid and hot environment of the Amazon jungle.[1] Their average salt intake, when measured by 24-hour urine collections, varies between 1 and 10 mmol/day. These levels, however, are almost unseen in the western world due to the high salt intake we are exposed to, even when adhering to a low salt diet. Under conditions of exercise in a hot environment, a low salt intake does not impair the ability to exercise, and it does not cause changes in plasma sodium, potassium, osmolality or sweat rate, although the salt content of sweat is reduced on a low salt intake diet.[2] Gangopadhyay *et al* misquote the evidence in athletes and the military where the high morbidity from hyponatraemia is due to overhydration (ie too much water) rather than a low salt intake. The case presented here is clearly a case of diuretic abuse, surreptitious vomiting or laxative abuse, as we described in the past in a different scenario.[3] While hyponatraemia may possibly have been caused by water intoxication, it would not have caused plasma potassium to fall so low, or the renin–angiotensin system to be so stimulated. The authors do not seem to have considered screening for diuretics. Diuretic abuse would explain hyponatraemia, alkalotic hypokalaemia and activation of the renin–angiotensin–aldosterone system as described here. A moderate reduction in salt intake (up to 3 g per day) does not raise cause for concern and should be recommended to everyone to prevent cardiovascular disease and other common conditions like kidney stones and osteoporosis.[4]

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