Ever wondered why sweat is salty?

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WHY is sweat salty? The simplest answer is that keeping us cool may require the evaporation of substantial amounts of water and the only way the body has of moving large volumes of water is by creating salt gradients.

Most of the salt can be recycled, as in the kidneys, and as it is from human sweat when aldosterone, secreted in response to a fall in blood volume, reduces sweat sodium concentration, potentially to very low levels indeed. Of course, it might be more straightforward if we simply lost water to keep cool, as most other mammals do through panting. But it would not be helpful for verbal communication – for example, when hunting in packs or in battle – if we had to pant and shout simultaneously. The fact is, we sweat, as do horses.

We, like our cells, live surrounded by salt; most of the planet is covered by strong saline, an environment mimicked in evolution by our extracellular fluid (ECF). Insufficient salt threatens ECF volume, but excess can be rapidly lethal because increased concentration in ECF causes brain cell shrinkage. Salt is among the few substances we actually taste (rather than smell) and unique among materials sufficiently valuable to have been widely used as currency in that it is genuinely precious and physiologically vital – unlike gold.

Are salt tablets needed? Probably not, if our daily routine, even in hot climates, is not particularly athletic. The response of our sweat glands to aldosterone should keep us safe, particularly bearing in mind the fact that most humans have a dietary sodium intake, even without added salt, which is far above the nutritional maintenance requirement.

A bit of sweat simply means there is less excretion of excess sodium demanded of the kidneys. But if sweat rates become extremely high, for example, for soldiers, tennis players or endurance athletes in high environmental temperatures, and especially when these combine with high humidity, loss of sodium in sweat can become significant, despite the effect of aldosterone. In which case, there is the risk of reduction of ECF volume and, therefore, of plasma.

By opting instead for upper respiratory evaporation and pure water loss as the main cooling mechanism, most mammals have simplified the problem of maintaining fluid balance.

It should be possible, at any ambient temperature and humidity, and for any thermal load – both endogenous through activity and exogenous through solar exposure – to calculate exactly how much water needs to be evaporated to maintain normal body temperature, through the cooling effect of the latent heat of evaporation.

The main benefit of air conditioning is not to cool the air but to dry it so that sweat evaporates efficiently from a virtually dry skin surface, instead of failing to evaporate and uselessly wetting both skin and clothing. It is also why a dog in an unshaded car on a hot day with plenty of water but closed windows is doomed – the contents of the dish will evaporate and humidify the air so that panting becomes increasingly inefficient and demanding, until the muscular effort generates more heat than can be dissipated. The dog needs ventilation and water.

Volume or concentration

Which is the greater need – salt or water? Tricky question; whether you are chasing your prey or escaping your predator in hot surroundings, the issue is which poses the greater threat to athletic performance – a fall in plasma volume or a rise in plasma sodium concentration?

A reduced plasma volume stimulates progressive vasoconstriction in “non-essential” capillary beds to protect central nervous system perfusion, but a small reduction in muscle perfusion may cause a literally vital fall in running speed. Since most animals, unlike us, drink water rather than flavoured fluids, their main regulation of water balance is through accurate changes in thirst, which serve to stabilise the body’s index of adequate hydration – plasma sodium concentration. Anti-diuretic hormone and renal water conservation provide a back-up and urine is normally concentrated – unlike ours, which is diluted by the need to excrete the excess water consumed for taste or habit as cola, skinny latte or best bitter.

These days, human marathon runners have access to drinks stations where they can replenish their body water. Of course, were you a lion or a zebra, drinking stations would be a luxury you could ill afford. The predator has a choice – find water. The prey does not. This brings us to two questions that are seldom asked.

Firstly, why are horses so poorly designed, relying on sweating to keep cool and losing prodigious amounts of salt as a result? Alternatively, here’s a question worthy of Eric Cantona – are horses like seabirds?

Those who believe that mammals function like humans that never learned to stand upright readily appreciate that equine sweating is an appalling design failure. Here is a species developed for sustained speed, yet doomed to squander body sodium whenever it engages in serious exertion.

But granted that horses have had to cope with environments where salt may be as scarce as water, would they have evolved successfully in that way? Would they have persisted with such spectacularly defective sweat glands – glands unlike almost any other – that seem resistant to the sodiumconserving effect of aldosterone?

There, I believe, lies the decisive clue. Long ago, I was told of some preliminary work that remained unpublished, suggesting that sweat sodium in horses did not respond to aldosterone. What tends to be forgotten is that while horses can not afford to disrupt their respiratory pattern to pant, they still have enormous water losses from the respiratory tract during exertion. These would almost certainly cause incapacitating hypernatraemia were it not for appropriate sodium excretion via the sweat glands. Equine sweating, therefore, on a temporary basis, fulfils the same function as the salt glands of seabirds and marine reptiles: the excretion of excess sodium. It is a brilliant adaptation because other mammals have the ability to defend themselves against hypernatraemia through “dehydration natriuresis”, but horses, in the wild, would scarcely have time to urinate their way to safety. Of course, unlike seabirds, the sodium “excess” is temporary and the lost sodium needs to be replaced once body water has been sufficiently replenished.

However, is it true that equine sweat glands are remarkable, not just as sweat glands, but among epithelia generally, in resisting the sodium-conserving effect of aldosterone, which is usually second in importance only to its role in facilitating the excretion of the enormous dietary potassium loads imposed on herbivores? A paper from 2002[1] showed that aldosterone infusions that caused massive reductions in both urinary and faecal sodium loss left sweat sodium unaltered. Clearly horses, like dogs, would have needed a designer with extraordinary genius to create such brilliant adaptations to the demands of thermoregulation. Let alone camels.

References

- 1. Jansson A et al (2002). Plasma aldosterone concentration and cardiovascular response to low sodium intake in horses in training, *J Appl Physiol* **92**: 135-141.