Apical potassium (BK) channels and enhanced potassium secretion in human colon

Sir,

The recent review\(^1\) suggested that K\(^+\) secretion by the colon was important in K\(^+\) homeostasis. This is, I think, questionable.

Other than in diarrhoea, faecal fluid has little capacity for extra K\(^+\),. In humans, K\(^+\) concentration in faecal fluid averages about 80 mmol/l and Na\(^+\) 25 mmol/l.\(^2\) Faecal fluid is about 100–200 ml/24 h so that if all Na\(^+\) was replaced by K\(^+\), this would only amount to an increased excretion of ≤5 mmol/24 h, which is a small fraction of the daily dietary intake. Only when there is considerable increase in delivery of inorganic and organic anions to the colon (as due to diarrhoea or drugs) can colonic K\(^+\) loss rise substantially.

The colonic K\(^+\) secretory mechanism is conserved across the species indicating its significant Darwinian survival value. In nature, K\(^+\) is in plentiful supply while, in contrast, Na\(^+\) is not readily available to land animals living away from the sea. They are constantly threatened by Na\(^+\) deficiency; hence, the efficient Na\(^+\) conservation mechanisms in sweat glands, kidney and gut.

In Na\(^+\)-depleted states, K\(^+\) is substituted for Na\(^+\) in the faeces and this is so even when the colon load is considerably increased experimentally by feeding cation exchange resins.\(^2\) Colonic K\(^+\) secretion minimizes Na\(^+\) loss; its importance is in Na\(^+\) homeostasis.

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References


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Response to letter from Dr Charles Edmonds Re: Apical potassium (BK) channels and enhanced potassium secretion in human colon

Sir,

We should like to thank Dr Edmonds for his interest in our recent review.\(^1\) Healthy individuals do indeed have modest losses of K\(^+\) in faecal fluid (~9 mmol/day, which is ~10% of the dietary intake, the remainder being lost in the urine\(^2\)\) and their colonic capacity for K\(^+\) secretion is relatively small.\(^2\) We also appreciate the importance of increased colonic K\(^+\) secretion in situations where dietary Na\(^+\) intake is restricted or the body is threatened by excessive Na\(^+\) losses.

The aim of our review was to emphasize that we now know considerably more about the mechanism of K\(^+\) secretion in human colon and specifically, the role of apical high conductance potassium (BK) channels. Furthermore, it is clear that the capacity of the human colon for K\(^+\) secretion may increase in certain diseases and during the excessive use of cyclic adenosine monophosphate (cAMP)-mediated laxatives. These clinical scenarios are associated with increased expression and/or activity of apical BK channels. End-stage renal disease (ESRD), treated by continuous ambulatory peritoneal dialysis, is the prime example of the role of increased colonic K\(^+\) secretion in maintaining K\(^+\) homeostasis. These patients produce little or no urine, have near-normal dietary K\(^+\) intakes (~80 mmol/day), but lose only ~35 mmol/day by dialysis. Yet they remain normokalaemic, which leaves the colon as the only feasible route for K\(^+\) excretion. The concept...