Diabetic ketoacidosis (DKA) is a major complication of diabetes and is associated with significant mortality, morbidity and costs. In Australia and New Zealand, the incidence of intensive care unit admissions of adults with DKA increased five-fold between 2000 and 2013, to 5.3 per 100,000 people, with an in-hospital mortality of 1.4%. In Europe and North America, a similar incidence and even higher mortality have been reported. It is likely that optimal early management of hyperglycaemia is important for patients with DKA admitted to the ICU. Correction of hyperglycaemia, hyperosmolarity and modulation of lipolysis and ketogenesis are typically considered to be simultaneous physiological goals. However, the rapid correction of hyperglycaemia may also increase the risk of hypoglycaemia and hypokalaemia, induce hypo-osmolarity and increase the risk of cerebral oedema, especially in children. These pathophysiological considerations leave clinicians with uncertainty about how to best manage blood glucose levels (BGLs) in the first 24 hours of ICU admission.

Current DKA-specific guidelines recommend blood glucose reduction by 54 mg/dL/hour. They also recommend glucose infusion when the blood glucose level falls below 198 mg/dL or even below 252 mg/dL, with avoidance of a glucose level < 180 mg/dL (to convert to mmol/L, multiply mg/dL by 0.0555). However, such guidelines are not based on empirical observations of benefit in adults, and their threshold values are not evidence-based.

We used data from a high-quality, bi-national database of ICU admissions in Australia and New Zealand. We tested the hypothesis that, compared with intensive early glycaemic control (highest blood glucose, ≤ 180 mg/dL), a less intensive approach (highest blood glucose, > 180 mg/dL) might be associated with significant differences in hyperglycaemia, hypoglycaemia, hypokalaemia, hypo-osmolarity and mortality. In a nested cohort of patients from these databases, we also studied the relevant emergency department (ED) treatment in detail to obtain information on typical immediate management of BGL for these patients before ICU admission.

ABSTRACT

Objectives: To determine the impact of the intensity of early correction of hyperglycaemia on outcomes in patients with diabetic ketoacidosis (DKA) admitted to the intensive care unit.

Methods: We studied adult patients with DKA admitted to 171 ICUs in Australia and New Zealand from 2000 to 2013. We used their blood glucose levels (BGLs) in the first 24 hours after ICU admission to determine whether intensive early correction of hyperglycaemia to ≤ 180 mg/dL was independently associated with hypoglycaemia, hypokalaemia, hypo-osmolarity or mortality, compared with partial early correction to > 180 mg/dL as recommended by DKA-specific guidelines.

Results: Among 8553 patients, intensive early correction of BGL was applied to 605 patients (7.1%). A greater proportion of these patients experienced hypoglycaemia (20.2% vs 9.1%; P < 0.001) and/or hypo-osmolarity (29.4% vs 22.0%; P < 0.001), but not hypokalaemia (16.7% vs 15.6%; P = 0.47). Overall, 11 patients (1.8%) in the intensive correction group and 112 patients (1.4%) in the partial correction group died (P = 0.42). However, after adjustment for illness severity, partial early correction of BGL was independently associated with a lower risk of hypoglycaemia (OR, 0.38; 95% CI, 0.30–0.48; P < 0.001), lower risk of hypo-osmolarity (OR, 0.80; 95% CI, 0.65–0.98; P < 0.03) and lower risk of death (OR, 0.44; 95% CI, 0.22–0.86; P = 0.02).

Conclusions: In a large cohort of patients with DKA, partial early correction of BGL according to DKA-specific guidelines, when compared with intensive early correction of BGL, was independently associated with a lower risk of hypoglycaemia, hypo-osmolarity and death.

Methods

This study was approved by the Alfred Hospital human research ethics committee and was performed in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki and its later amendments. Individual hospitals contributed data to the nested cohort with a waiver of informed consent.

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Selection of study patients
We identified all adult patients (> 16 years) in the Australian and New Zealand Intensive Care Society (ANZICS) Adult Patient Database (APD) who were admitted between 1 January 2000 and 31 December 2013 with a primary ICU admission diagnosis of DKA. In the ANZICS APD, a diagnosis of DKA is registered according to the Acute Physiology and Chronic Health Evaluation (APACHE) II and/or III diagnosis codes. The ANZICS APD is one of four clinical quality registries run by the ANZICS Centre for Outcome and Resource Evaluation for the purposes of benchmarking and monitoring intensive care practices and outcomes in Australia and New Zealand. We excluded readmissions, patients with missing data on mortality and/or diabetic status, and patients without BGL data. Year-by-year changes in the epidemiology for this cohort were recently published.2

Operational definitions
The ANZICS APD records the highest and lowest BGL measurements early in the ICU admission (in the first 24 hours). We categorised patients based on whether every early ICU BGL value was \( \leq 180 \text{ mg/dL} \) (intensive correction group) or whether at least one such ICU value was > 180 mg/dL (partial correction group), as recommended by DKA-specific guidelines.6-8

In addition, to understand whether outcomes in patients with BGLs in the DKA-specific guidelines range changed according to the degree of correction, we divided patients into four strata (highest BGL of 181–360 mg/dL, 361–540 mg/dL, 541–720 mg/dL and > 720 mg/dL) and compared all study outcomes across progressive levels of hyperglycaemia with those in the intensive early correction group.

Definition of outcomes
We defined hypoglycaemia as a BGL in the first 24 hours below 72 mg/dL. We defined hypokalaemia as a plasma potassium level below 3.3 mmol/L.6 We defined hyposmolarity as a calculated plasma osmolarity below 285 mmol/L (see Appendix, online at cicm.org.au/Resources/Publications/Journal).16

Nested cohort
The methodological details for selection and analysis of the nested cohort of patients with DKA are shown in the Appendix.

Statistical analysis
We performed statistical analyses using SAS, version 9.4 (SAS Institute). We used a logistic regression model to assess the association between a highest BGL of > 180 mg/dL and hypoglycaemia, hypokalaemia, hypo-osmolarity and in-hospital mortality. Calculations were made after adjusting for the following pre-defined variables: patient illness severity, admission year, propensity for having a highest BGL > 180 mg/dL during the first 24 hours of ICU admission, urea level, insulin-treated diabetes and plasma creatinine. Details of the propensity score development are shown in the Appendix.

To create a measure of illness severity that was independent of BGL, we developed a patient risk-of-death score in accordance with the Australian and New Zealand Risk of Death (ANZROD) methodology17 with the glucose component removed. The ANZROD model is an updated mortality-prediction model derived from components from the APACHE II and III scores. It has been validated for use in Australian and New Zealand ICUs and has superior calibration and discrimination compared with the APACHE III model.17

We compared the risk of hypoglycaemia, hypokalaemia, hypo-osmolarity and mortality across progressive levels of hyperglycaemia with intensive BGL correction. To do so, we used hierarchical multivariable logistic regression, adjusting for illness severity (ANZROD model with glucose component removed), and year of admission with patients nested within site and site treated as a random effect. We used locally weighted scatterplot smoothing (LOWESS) analysis to display the crude relationship between highest BGL in the ICU and predicted mortality risk. A two-sided \( P < 0.05 \) was considered statistically significant.

Results
Patient characteristics
We searched 1 259 982 ICU admissions recorded in the ANZICS APD between January 2000 and December 2013. We identified 8553 adult patients (median age, 35 years [IQR, 23–51 years]) admitted to an ICU for DKA and with available data on BGL, pre-morbid diabetes diagnosis and hospital mortality. Of these, 45.8% were men and 72.6% were treated with insulin before hospital admission. Patients were admitted to the ICU a median of 4.0 hours (IQR, 1.8–6.5 hours) after hospital arrival, with most patients (87.2%) being transferred directly from the ED.

Overall, 605 of 8553 patients (7.1%) received intensive early correction of hyperglycaemia, with a similar prevalence between 2000 and 2013 (see Appendix, Figure 1). Compared with patients who received partial early correction of BGLs, these patients were younger, had less chronic cardiovascular disease, and had lower APACHE III scores. Their predicted mortality risk was also significantly lower, irrespective of the mortality prediction model (Table 1).
Blood glucose, osmolarity and acid–base status

A comparison of distributions of BGLs, electrolyte levels and acid–base status in the two groups are shown in Table 2. BGLs, osmolarity and potassium levels were significantly lower in the intensive early correction group.

Outcomes

Overall, 122 patients (20.2%) in the intensive early correction group and 722 (9.1%) in the partial early correction group developed hypoglycaemia ($P < 0.001$). In addition, 101 patients (16.7%) in the intensive early correction group, and 1239 patients (15.6%) in the partial early correction group developed hypokalaemia ($P = 0.47$). Hypo-osmolarity occurred in 178 patients (29.4%) in the intensive early correction group and in 1745 patients (22.0%) in the partial early correction group ($P < 0.001$).

Median ICU and hospital lengths of stay were shorter in the intensive early correction group. Similar proportions of patients developed acute renal failure or were mechanically ventilated. A total of 11 patients (1.8%) in the intensive early correction group and 112 patients (1.4%) in the partial early correction group died in hospital ($P = 0.42$) (Table 3).

In the propensity-adjusted analysis (Table 4), partial early correction of hyperglycaemia was associated with lower odds of hypoglycaemia, hypo-osmolarity and mortality. Moreover, the odds ratio (OR) for hypoglycaemia, with adjustment for the modified risk of death (with glucose component removed), year of admission, and site, was significantly decreased for patients in the partial early correction group across all higher BGL strata ($P < 0.001$).

Finally, the stratum with moderate correction of BGL showed the strongest association with lower mortality (OR,
**Table 2. Glycaemic and acid–base status in the full cohort of patients with diabetic ketoacidosis, and by degree of early glycaemic control**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Total (n = 8553)</th>
<th>Intensive correction (n = 605)</th>
<th>Partial correction (n = 7948)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Median glucose level, mg/dL (IQR)*</td>
<td></td>
<td></td>
<td></td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Highest</td>
<td>356 (268–513)</td>
<td>146 (122–166)</td>
<td>373 (286–535)</td>
<td></td>
</tr>
<tr>
<td>Lowest</td>
<td>133 (95–197)</td>
<td>99 (79–126)</td>
<td>137 (97–193)</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Sodium level, mmol/L (IQR)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Highest</td>
<td>139 (136–142)</td>
<td>138 (136–141)</td>
<td>139 (136–142)</td>
<td>0.02</td>
</tr>
<tr>
<td>Lowest</td>
<td>134 (131–138)</td>
<td>135 (133–138)</td>
<td>134 (131–138)</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Potassium level, mmol/L (IQR)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Highest</td>
<td>4.5 (4.1–5.0)</td>
<td>4.2 (3.8–4.6)</td>
<td>4.5 (4.1–5.0)</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Lowest</td>
<td>3.8 (3.4–4.1)</td>
<td>3.7 (3.4–4.0)</td>
<td>3.8 (3.4–4.1)</td>
<td>0.006</td>
</tr>
<tr>
<td>Urea level, mmol/L (IQR)</td>
<td></td>
<td></td>
<td></td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Highest</td>
<td>7.0 (4.3–12.3)</td>
<td>5.3 (3.3–9.2)</td>
<td>7.1 (4.4–12.5)</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Osmolarity, mmol/L (IQR)†</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Highest</td>
<td>315 (304–334)</td>
<td>299 (293–308)</td>
<td>317 (306–335)</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Lowest</td>
<td>292 (285–302)</td>
<td>289 (284–298)</td>
<td>292 (285–303)</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Bicarbonate level, mmol/L</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean highest (SD)</td>
<td>19.8 (5.7)</td>
<td>20.8 (5.6)</td>
<td>19.7 (5.7)</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Low</td>
<td>12.2 (7–18)</td>
<td>16 (10.8–21)</td>
<td>12 (7–18)</td>
<td></td>
</tr>
<tr>
<td>Base excess, mmol/L (IQR)‡</td>
<td></td>
<td></td>
<td></td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Highest</td>
<td>–14.4 (–20.8 to –7.5)</td>
<td>–10.8 (–17.3 to –5.44)</td>
<td>–14.6 (–21 to –7.7)</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Lowest</td>
<td>30 (23–36)</td>
<td>32 (25–37)</td>
<td>30 (23–36)</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Worst arterial PacO2, mmHg (IQR)</td>
<td></td>
<td></td>
<td></td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Highest</td>
<td>7.25 (7.13–7.34)</td>
<td>7.30 (7.20–7.37)</td>
<td>7.25 (7.12–7.33)</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Lowest</td>
<td>93 (58–141)</td>
<td>72 (49–112)</td>
<td>95 (60–142)</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Respiratory rate, bpm (IQR)</td>
<td></td>
<td></td>
<td></td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Highest</td>
<td>24 (20–28)</td>
<td>22 (20–26)</td>
<td>24 (20–28)</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Lowest</td>
<td>14 (12–16)</td>
<td>14 (12–16)</td>
<td>14 (12–16)</td>
<td>&lt; 0.001</td>
</tr>
</tbody>
</table>

IQR = interquartile range. SD = standard deviation. PacO2 = partial pressure of arterial carbon dioxide. bpm = beats per minute. * SI conversion factor: to convert glucose to mmol/L, multiply values by 0.0555. † Plasma osmolarity was calculated as: 2 (plasma sodium + plasma potassium) + blood glucose + plasma urea. ‡ Base excess was calculated as: 0.0279 × PacO2 × 10(pH –6.1) + 13.8012 × pH – 124.82088.

**Table 3. Outcomes of the full cohort of patients with diabetic ketoacidosis and subgroups, by degree of early glycaemic correction**

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Total (n = 8553)</th>
<th>Intensive correction (n = 605)</th>
<th>Partial correction (n = 7948)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypoglycaemia, n (%)*</td>
<td>844 (9.9%)</td>
<td>122 (20.2%)</td>
<td>722 (9.1%)</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Hypokalaemia, n (%)†</td>
<td>1340 (15.7%)</td>
<td>101 (16.7%)</td>
<td>1239 (16.5%)</td>
<td>0.47</td>
</tr>
<tr>
<td>Hypo-osmolarity, n (%)‡</td>
<td>1923 (22.5%)</td>
<td>178 (29.4%)</td>
<td>1745 (22.0%)</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Median ICU length of stay, hours (IQR)</td>
<td>43.0 (24.7–67.2)</td>
<td>38.1 (22.5–56.8)</td>
<td>43.2 (25.0–67.6)</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>ICU mortality, n (%)</td>
<td>60 (0.7%)</td>
<td>4 (0.7%)</td>
<td>56 (0.7%)</td>
<td>0.90</td>
</tr>
<tr>
<td>Acute renal failure, n (%)§</td>
<td>454 (5.3%)</td>
<td>30 (5.0%)</td>
<td>424 (5.3%)</td>
<td>0.69</td>
</tr>
<tr>
<td>Mechanical ventilation, n (%)</td>
<td>589 (6.9%)</td>
<td>43 (7.1%)</td>
<td>546 (6.9%)</td>
<td>0.82</td>
</tr>
<tr>
<td>Median hospital length of stay, days (IQR)</td>
<td>4.0 (2.6–7.5)</td>
<td>3.8 (2.2–6.7)</td>
<td>4.0 (2.6–7.5)</td>
<td>0.002</td>
</tr>
<tr>
<td>Treatment limitation or palliative care, n (%)</td>
<td>86 (1.0%)</td>
<td>3 (0.5%)</td>
<td>83 (1.0%)</td>
<td>0.19</td>
</tr>
<tr>
<td>Hospital outcome, n (%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Death</td>
<td>123 (1.4%)</td>
<td>11 (1.8%)</td>
<td>112 (1.4%)</td>
<td>0.42</td>
</tr>
<tr>
<td>Discharged, home</td>
<td>7962 (93.1%)</td>
<td>559 (92.4%)</td>
<td>7403 (93.1%)</td>
<td>0.49</td>
</tr>
<tr>
<td>Discharged, rehabilitation</td>
<td>190 (2.2%)</td>
<td>1.3% (8%)</td>
<td>2.3% (182%)</td>
<td>0.12</td>
</tr>
<tr>
<td>Discharged, other hospital</td>
<td>3.3% (278%)</td>
<td>4.5% (27%)</td>
<td>3.2% (251%)</td>
<td>0.08</td>
</tr>
</tbody>
</table>

ICU = intensive care unit. IQR = interquartile range. * Glucose level < 72 mg/dL; SI conversion factor: to convert glucose to mmol/L, multiply values by 0.0555. † Potassium level < 3.3 mmol/L. ‡ Osmolarity < 285 mmol/L. § Urine output < 410 mL per 24 hours, or plasma creatinine ≥ 133 µmol/L in patients not receiving long-term dialysis.
0.36; 95% CI, 0.17–0.75; P = 0.001) (Table 5). In particular, more limited correction strata, even up to a BGL of > 720 mg/dL, showed favourable point estimates for most study outcomes and no association with harm (Table 5). None of the higher-BGL strata were associated with increased mortality, compared with the moderate-correction stratum (Appendix, Tables 2 and 3).

The unadjusted nature of the relationship between BGL and mortality was further shown in the LOWESS analysis showing the lowest predicted mortality with glucose values between 180 and 360 mg/dL (Appendix, Figure 2).

Analysis of the nested cohort
We selected 219 patients for a nested cohort and analysed their baseline characteristics, detailed aspects of DKA treatment delivered in the ED (for which data were available for 197 patients [90.0%]), the biochemical response to such treatment, and the outcomes. The results of that analysis are shown in Appendix Figure 3 and Appendix Tables 4–7. The patients in the nested cohort were representative of the full studied cohort of patients with DKA and showed that their immediate ED treatment was in high compliance with current guidelines during the study period.

Discussion
Key findings
We studied a large cohort of adult patients with DKA admitted to ICUs in Australia and New Zealand. We found that, after adjustment for illness severity and compared with early management according to DKA-specific guidelines, more intensive early correction of hyperglycaemia was associated with a higher risk of hypoglycaemia, hypo-osmolarity and death. We also found that moderate correction of blood glucose according to DKA-specific guidelines was associated with the lowest risk of hospital mortality. Finally, we found
that even permissive early management of hyperglycaemia to values > 720 mg/dL showed no association with harm.

Relationship to previous studies

To our knowledge, this is the first study to explore the independent association between early correction of hyperglycaemia and the risk of hypoglycaemia, hypokalaemia, hypo-osmolarity and mortality in adult patients with DKA admitted to the ICU. Previously, the association between early management of hyperglycaemia and osmolarity in DKA patients had been explored in a case–control paediatric study, in which early insulin administration was independently associated with five-fold higher odds of developing cerebral oedema. In a small, retrospective observational study, a greater early fall in serum tonicity was also associated with cerebral oedema. In response to these concerns, low-dose insulin therapy has been advocated for children. A retrospective observational study of 67 children confirmed a slower reduction of BGL and tonicity with low-dose insulin infusion. A subsequent trial in children randomised to low-dose insulin (0.05 units/kg/h) v standard-dose insulin (0.1 units/kg/h) did not confirm these findings. However, hypokalaemia and hypoglycaemia were more common in the standard-dose group, and one patient in the standard-dose group developed cerebral oedema.

In adults, a small study found that early hypo-osmolar rehydration (220 mosmol/kg water) without simultaneous insulin administration reduced BGL by 18 mg/dL/h, with a decline in stress hormone levels, suggesting that rehydration alone may aid DKA resolution. In another cohort, an insulin bolus followed by a low-dose insulin infusion (1 unit/h) was combined with fluid replacement of 1 L/h during the first 4 hours. Similarly to our nested cohort, patients’ BGLs decreased by 57.6 mg/dL/h, without electrolyte disorders. However, none of the above investigations had the statistical power to assess the independent relationship between glycaemic control and hypoglycaemia, hypokalaemia, hypo-osmolarity or mortality, and none compared early glycaemic control according to ICU-specific or DKA-specific guidelines.

Study implications

Our study has public health implications, because its findings apply to thousands of patients with DKA who are admitted to an ICU, and also because they may be relevant to many more patients with DKA presenting to hospitals worldwide. For example, if data from Australia and New Zealand apply to other countries, patients with DKA admitted to an ICU should account for almost 400 000 ICU presentations per year, worldwide. If data from Denmark, the United States and Italy also apply, all hospital admissions for DKA should number about 2 million worldwide every year. Our findings imply that more intensive early management of hyperglycaemia to a glucose level considered standard-of-care for critically ill patients without DKA is likely to be unnecessary in patients with DKA, and that it is likely that current DKA-specific guidelines are safer. This appears to be true even in a clinical environment, where, as data from our
nested cohort show, early DKA treatment was in agreement with available guidelines and literature.

Our findings further imply that there may be possible harm to one patient in every 15 patients exposed to intensive early glucose correction, and they provide the first empirical support for the preferential use of DKA-specific early glycaemic targets.6–8

Strengths and limitations

Our study has several strengths. We assessed a previously unexplored, unknown relationship between early glycaemic management and complications and mortality in adult patients with DKA. We used a large database from 171 ICUs, from two countries, involving > 8000 patients, thus providing a degree of epidemiological robustness and external validity for applying our findings to other developed countries. Even in a cohort with a very low overall mortality, we found greater safety when early management was within DKA-specific glycaemic targets, an observation which has therapeutic implications. Our findings also mean it is unlikely that early intensive correction of hyperglycaemia has clinical benefits, and suggest that concerns about the risks of BGLs that are too high with partial correction strategies may be unwarranted.

Our study also has some limitations. First, it is an observational study, and can only describe associations, which cannot be taken to imply causation. However, assuming a 2% mortality with intensive glucose management, and a reduction to 1% with DKA-specific early glucose control, more than 5000 patients would have to be randomised to have a 90% power to detect such an effect at an alpha of 0.05. Given the logistic demands, the number of admissions, and the uncertain ethical acceptability of randomising patients to intensive early glycaemic control, such a study is unlikely ever to take place. Our findings also carry many of the characteristics that describe associations with a greater potential for causality, as described by Hill: carry many of the characteristics that describe associations such a study is unlikely ever to take place. Our findings also randomising patients to intensive early glycaemic control, of admissions, and the uncertain ethical acceptability of

Conclusions

In adult patients with DKA admitted to ICUs in Australia and New Zealand, compared with partial early correction of hyperglycaemia according to DKA-specific guidelines, intensive early glycaemic management was independently associated with an increased risk of hypoglycaemia, hypo-osmolality and hospital mortality. These findings support the preferential application of DKA-specific guidelines to the early management of DKA-patients admitted to ICU.

Acknowledgements

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Competing interests

None declared.

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