Variation in hospital mortality in an Australian neonatal intensive care unit network

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ABSTRACT
Background Studding centre-to-centre (CTC) variation in mortality rates is important because differences in quality of care can be made permitting changes in practice to improve outcomes. However, comparisons between hospitals can be misleading unless there is adjustment for population characteristics and severity of illness.

Objective We sought to report the risk-adjusted CTC variation in mortality among preterm infants born <32 weeks and admitted to all eight tertiary neonatal intensive care units (NICUs) in the New South Wales and the Australian Capital Territory Neonatal Network (NICUS), Australia.

Methods We analysed routinely collected prospective data for births between 2007 and 2014. Adjusted mortality rates for each NICU were produced using a multiple logistic regression model. Output from this model was used to construct funnel plots.

Results A total of 7212 live born infants <32 weeks gestation were admitted consecutively to network NICUs during the study period. NICUs differed in their patient populations and severity of illness. The overall unadjusted hospital mortality rate for the network was 7.9% (n=572 deaths). This varied from 5.3% in hospital E to 10.4% in hospital C. Adjusted mortality rates showed little CTC variation. No hospital reached the +99.8% control limit level on adjusted funnel plots.

Conclusion Characteristics of infants admitted to NICUs differ, and comparing unadjusted mortality rates should be avoided. Logistic regression-derived risk-adjusted mortality rates plotted on funnel plots provide a powerful visual graphical tool for presenting quality performance data.

What this study adds?

Logistic regression derived risk-adjusted mortality rates plotted on funnel plots provide a powerful visual graphical tool for presenting quality performance data.

Centre-to-centre variation is readily identified, permitting hospitals to appraise their practices and start timely intervention.

BACKGROUND
Advances in perinatal and neonatal care have significantly reduced neonatal mortality rates, especially among preterm and low birth weight infants.1 However, there is significant centre-to-centre (CTC) variation in mortality rates among neonatal intensive care units (NICUs).2–12 Studying CTC variation in mortality rates is important because differences in quality of care can be made which permit changes in practice to improve outcomes.13 However, comparisons between hospitals can be misleading unless there is adjustment for differences in population characteristics and severity of illness.

Our aim was to report the risk-adjusted CTC variation in mortality rates for infants born at <32 weeks and admitted to all eight tertiary NICUs in the New South Wales (NSW) and the Australian Capital Territory (ACT) Neonatal Network (NICUS), Australia. Furthermore, we would like to show the benefits of adjusting CTC variation for population characteristics. NSW is the most populous state of Australia,14 and the ACT, home to the Government of the Commonwealth, is within NSW.

METHODS
We analysed prospectively collected data from a geographically defined area. All infants who were delivered before 32 weeks of gestational age and admitted to one of the eight tertiary perinatal centres in NSW and the ACT between 1 January 2007 and 31 December 2014 were included in the study. Full descriptions of the NSW and ACT neonatal service organisation and networking, medical and nursing staffing of the collaborating NICUs are available elsewhere.15–18 Geographically, the ACT lies within NSW and the state and territory combined have a total population of 7904500 with approximately 106 007 live births per year.19

DATA SOURCE
Data for this study were obtained from The Neonatal Intensive Care Units’ Data Collection, which is an ongoing prospective state and territory wide audit of infants admitted to all eight perinatal centres and two children’s hospitals in NSW and the ACT for one of the following reasons: (1) gestation 22+0 to 31+6 weeks; (2) birth weight ≤1500 g; (3) assisted ventilation (mechanical ventilation, continuous positive airway pressure, high flow humidified
Table 1 Maternal characteristics of the study group stratified by admitting hospital A to H

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>A (n=1225)</th>
<th>B (n=1000)</th>
<th>C (n=963)</th>
<th>D (n=889)</th>
<th>E (n=856)</th>
<th>F (n=848)</th>
<th>G (n=785)</th>
<th>H (n=646)</th>
<th>All (n=7212)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maternal age, years</td>
<td>29.0 (24.0–33.0)</td>
<td>30.0 (26.0–35.0)</td>
<td>29.0 (25.0–34.0)</td>
<td>32.0 (28.0–36.0)</td>
<td>32.0 (27.0–36.0)</td>
<td>32.0 (28.0–36.0)</td>
<td>29.0 (24.0–33.0)</td>
<td>30.0 (26.0–34.0)</td>
<td>30.0 (26.0–35.0)</td>
</tr>
<tr>
<td>Indigenous Australian</td>
<td>129 (10.5)</td>
<td>34 (3.4)</td>
<td>39 (4.0)</td>
<td>38 (4.3)</td>
<td>43 (5.0)</td>
<td>24 (2.8)</td>
<td>70 (8.9)</td>
<td>49 (7.6)</td>
<td>426 (5.9)</td>
</tr>
<tr>
<td>Assisted conception</td>
<td>105 (8.6)</td>
<td>156 (15.6)</td>
<td>104 (10.8)</td>
<td>136 (15.3)</td>
<td>129 (15.1)</td>
<td>141 (16.6)</td>
<td>77 (8.8)</td>
<td>73 (11.3)</td>
<td>921 (12.8)</td>
</tr>
<tr>
<td>Multiple pregnancy</td>
<td>338 (27.6)</td>
<td>271 (27.1)</td>
<td>276 (28.7)</td>
<td>279 (31.4)</td>
<td>251 (29.3)</td>
<td>244 (28.8)</td>
<td>185 (23.6)</td>
<td>205 (31.7)</td>
<td>2049 (28.4)</td>
</tr>
<tr>
<td>Hypertension in pregnancy</td>
<td>231 (18.9)</td>
<td>216 (21.6)</td>
<td>143 (14.8)</td>
<td>202 (22.7)</td>
<td>165 (19.3)</td>
<td>153 (18.0)</td>
<td>157 (20.0)</td>
<td>121 (18.7)</td>
<td>1388 (19.2)</td>
</tr>
<tr>
<td>Chorioamnionitis</td>
<td>161 (13.1)</td>
<td>293 (29.3)</td>
<td>229 (23.8)</td>
<td>152 (17.1)</td>
<td>297 (34.7)</td>
<td>232 (27.4)</td>
<td>111 (14.1)</td>
<td>219 (34.0)</td>
<td>1694 (23.5)</td>
</tr>
<tr>
<td>Intrauterine growth restriction</td>
<td>145 (11.8)</td>
<td>158 (15.8)</td>
<td>123 (12.8)</td>
<td>166 (18.7)</td>
<td>143 (16.7)</td>
<td>124 (14.6)</td>
<td>100 (12.7)</td>
<td>84 (13.0)</td>
<td>1043 (14.5)</td>
</tr>
<tr>
<td>Any antenatal steroids</td>
<td>1119 (91.3)</td>
<td>849 (84.9)</td>
<td>885 (91.9)</td>
<td>822 (92.5)</td>
<td>805 (94.0)</td>
<td>780 (92.0)</td>
<td>712 (90.7)</td>
<td>588 (91.0)</td>
<td>6560 (91.0)</td>
</tr>
<tr>
<td>Vaginal breech delivery</td>
<td>82 (6.7)</td>
<td>84 (8.4)</td>
<td>63 (6.5)</td>
<td>54 (6.1)</td>
<td>23 (2.6)</td>
<td>34 (4.0)</td>
<td>36 (4.6)</td>
<td>39 (6.0)</td>
<td>420 (5.8)</td>
</tr>
<tr>
<td>Caesarean section</td>
<td>681 (55.6)</td>
<td>594 (59.4)</td>
<td>561 (58.3)</td>
<td>578 (65.0)</td>
<td>601 (70.2)</td>
<td>551 (65.0)</td>
<td>517 (65.9)</td>
<td>385 (59.6)</td>
<td>4468 (62.0)</td>
</tr>
</tbody>
</table>

Data are presented as median (IQR) for maternal age and n (%) for all other variables. Chorioamnionitis includes clinically suspected as well as pathologically proven cases.

Outcome measure
The study outcome was hospital mortality (death before discharge home). The principal causes of death were coded using the International Classification of Diseases and Related Health Problems, 10th Revision, Australian Modification.22

Because some conditions, seizures for example, are rarely the principal cause of death, we used a composite neonatal adverse outcome indicator (NAOI). NAOI has been validated previously.23

Statistical analysis
Statistical analyses were performed using SPSS (IBM, IBM SPSS Statistics for Windows, V22.0.0.0. Released 2013).

We performed a stepwise multiple logistic regression elimination analysis to establish the independent risk factors associated with mortality, after controlling for significant confounding factors (eg, ethnicity) in addition to other predictors (eg, gestational age) identified in other studies. In this multivariate model, we controlled for antenatal and perinatal variables but not intermediate variables (eg, intraventricular haemorrhage, retinopathy of prematurity, necrotising enterocolitis, chronic lung disease), as these may be related, directly or indirectly, to the quality of the management and might thus act as intermediate confounders through which the effect of ‘hospital’ is mediated.24

We used the multiple logistic regression to estimate the probability of hospital mortality after adjusting for potential confounding factors. Calibration was determined by means of Hosmer-Lemeshow goodness-of-fit χ² test (high p value suggests better classification).25 The ability of the logistic regression model to discriminate between those who died in hospital and those who survived until discharge was summarised using the C-statistic. A C-statistic of 0.5 indicates that the model discriminates no better than chance alone, whereas a value of 1.0 indicates perfect discrimination.26 27

The probabilities of death within an NICU were summed to give the hospital’s expected rate of death. Risk-adjusted standardised mortality ratios (SMRs) for each hospital were then produced by dividing the observed number of hospital deaths by the expected number produced by the logistic regression model. The 95% CIs for SMR were calculated by using Miettinen’s modification as described by Rothman et al.28 The SMRs were used to calculate the adjusted mortality rates for each hospital.

We used funnel plots to provide visual indication to differentiate between two sources of variation, namely common-cause and special-cause variation, of risk-adjusted hospital mortality among NICUs.29 These plots test whether the rate of mortality of an NICU differs significantly from the network average rate, assuming only random sampling variation rather than bias influences the NICU’s rate. The overall network mortality rate is represented by a solid horizontal line while the ±95% (2σ) and ±99.8% (3σ) control limits are represented by the curved dotted lines. Assuming differences arise from random sampling variation alone, the chance of the hospital being within the limits is 95% for the inner funnel and 99.8% for the outer funnel.

The level of statistical significance for all analyses was set at p<0.05 using two-tailed comparisons. The significance level was not changed when multiple comparisons were performed.

**RESULTS**
A total of 7212 live born infants <32 weeks gestation were admitted consecutively to one of the eight tertiary perinatal centres during the study period. Maternal and neonatal characteristics stratified by admitting hospital are shown in tables 1 and 2.

The proportion of pregnancies and deliveries involving maternal hypertension, multiple birth, caesarean section and Apgar score <7 at 5 min varied between hospitals. Antenatal steroid was administered to 84.9% to 94.0% of neonates (table 1). Overall, 10.4% of the study population was born in non-tertiary hospitals (outborn). Median gestation at birth and birth weight were similar between hospitals ranging from 27 to 31 completed weeks and 910.0 to 1559.0 g, respectively. Use...
Table 2  Neonatal characteristics of the study group stratified by admitting hospital A to H

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>A (n=1225)</th>
<th>B (n=1000)</th>
<th>C (n=893)</th>
<th>D (n=856)</th>
<th>E (n=848)</th>
<th>F (n=889)</th>
<th>G (n=785)</th>
<th>H (n=646)</th>
<th>All (n=7212)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Onsite surgical support</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
<td>Yes</td>
<td>No</td>
<td>-</td>
</tr>
<tr>
<td>Born in non-tertiary hospital</td>
<td>155 (12.7)</td>
<td>89 (8.9)</td>
<td>97 (10.1)</td>
<td>78 (9.1)</td>
<td>74 (9.4)</td>
<td>61 (9.4)</td>
<td>74 (11.7)</td>
<td>74 (11.7)</td>
<td>747 (10.4)</td>
</tr>
<tr>
<td>Male gender</td>
<td>687 (56.1)</td>
<td>531 (53.1)</td>
<td>501 (52.0)</td>
<td>481 (56.1)</td>
<td>460 (53.7)</td>
<td>478 (56.4)</td>
<td>409 (52.1)</td>
<td>345 (53.4)</td>
<td>3892 (54.0)</td>
</tr>
<tr>
<td>Gestational age, week*</td>
<td>29.0 (27.0–31.0)</td>
<td>29.0 (27.0–30.0)</td>
<td>29.0 (27.0–31.0)</td>
<td>29.0 (27.0–30.0)</td>
<td>29.0 (27.0–30.0)</td>
<td>29.0 (27.0–30.0)</td>
<td>29.0 (27.0–30.0)</td>
<td>29.0 (27.0–31.0)</td>
<td>29.0 (27.0–31.0)</td>
</tr>
<tr>
<td>Birth weight, g*</td>
<td>1270 (960.0–1550.0)</td>
<td>1200.0 (910.0–1494.0)</td>
<td>1240.0 (938.0–1536.0)</td>
<td>1235.0 (940.0–1547.0)</td>
<td>1200.0 (960.0–1520.0)</td>
<td>1220.0 (930.0–1502.0)</td>
<td>1260.0 (960.0–1559.0)</td>
<td>1290.0 (980.0–1550.0)</td>
<td>1240.0 (950.0–1510.0)</td>
</tr>
<tr>
<td>Birth weight&lt;10th percentile</td>
<td>82 (6.7)</td>
<td>79 (10.0)</td>
<td>72 (7.5)</td>
<td>69 (6.8)</td>
<td>72 (8.5)</td>
<td>69 (8.2)</td>
<td>57 (7.3)</td>
<td>50 (7.8)</td>
<td>561 (7.8)</td>
</tr>
<tr>
<td>Head circumference&lt;10th percentile</td>
<td>47 (3.9)</td>
<td>36 (2.8)</td>
<td>34 (4.0)</td>
<td>47 (5.5)</td>
<td>33 (4.1)</td>
<td>31 (3.8)</td>
<td>25 (3.4)</td>
<td>22 (3.4)</td>
<td>265 (3.9)</td>
</tr>
<tr>
<td>Apgar score&lt;7 at 5 min</td>
<td>642 (52.4)</td>
<td>741 (74.1)</td>
<td>509 (52.9)</td>
<td>534 (60.1)</td>
<td>580 (73.9)</td>
<td>516 (60.8)</td>
<td>409 (52.1)</td>
<td>345 (53.4)</td>
<td>4467 (61.9)</td>
</tr>
<tr>
<td>Postnatal steroids</td>
<td>32 (2.6)</td>
<td>66 (6.6)</td>
<td>62 (6.4)</td>
<td>62 (7.2)</td>
<td>56 (6.8)</td>
<td>69 (8.1)</td>
<td>59 (7.5)</td>
<td>44 (6.8)</td>
<td>468 (6.5)</td>
</tr>
</tbody>
</table>

Validation of the logistic regression model

The logistic regression model used to calculate expected mortality showed in Table 3 showed an adequate overall discrimination with C statistic (95% CI 0.86 [0.83 to 0.89]) and good calibration with Hosmer–Lemeshow (χ²=5.82, P=0.560).

Predictors of mortality

The most common causes of death are listed in Table 3.

Cases of death

Figures 1A, B, C and D show the observed and expected mortality and SMR stratified by admitting hospital, all postnatal steroids adjusted for.

Table 3 shows the risk-adjusted mortality rates for all admissions stratified by gestational age at birth. No hospital exceeded the +95% control limit for gestational age at birth. Hospital C approached the +95% control limit for gestational age at birth between 27–28 weeks of gestation (figure 2C). Hospital B and E showed better outcome (ie, expected mortality, SMR and risk-adjusted mortality varied from 5.3% in hospital E to 10.4% in hospital C. Similarly, the overall hospital mortality rate was 7.9% (n=572 deaths), and SMR was 1.02 (95% CI 0.94 to 1.11) (table 3). Hospital mortality rate varied from 5.3% in hospital E to 10.4% in hospital C. The observed and expected mortality and SMR stratified by admitting hospital are shown in Table 3.

Validation of the logistic regression model

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The study showed that there is little CTC variability in ongoing benchmarking of performance is critical for implementation of improvement initiatives across NICUs. The study showed that there is little CTC variability in ongoing benchmarking of performance is critical for implementation of improvement initiatives across NICUs. The study showed that there is little CTC variability in ongoing benchmarking of performance is critical for implementation of improvement initiatives across NICUs.
Process variation is of two types: common (chance) and special (assignable) causes of variation. Common-cause variation, which is shown as data within the control limits of the chart, is intrinsic to the process. Reduction of common-cause variation requires

### Table 3  Neonatal mortality of the study group stratified by admitting hospital A to H

<table>
<thead>
<tr>
<th>Hospital</th>
<th>Observed mortality (n=1225)</th>
<th>Expected mortality (n=1000)</th>
<th>Standardised mortality ratio (95% CI)</th>
<th>Risk-adjusted mortality (n=7212)</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>107 (8.7)</td>
<td>94 (7.7)</td>
<td>1.14 (0.94–1.37)</td>
<td>110 (9.0)</td>
</tr>
<tr>
<td>B</td>
<td>69 (6.9)</td>
<td>89 (8.9)</td>
<td>0.77 (0.61–0.97)</td>
<td>61 (6.1)</td>
</tr>
<tr>
<td>C</td>
<td>100 (10.4)</td>
<td>83 (8.6)</td>
<td>1.21 (0.98–1.46)</td>
<td>92 (9.6)</td>
</tr>
<tr>
<td>D</td>
<td>71 (8.0)</td>
<td>70 (7.8)</td>
<td>1.06 (0.84–1.32)</td>
<td>72 (8.1)</td>
</tr>
<tr>
<td>E</td>
<td>45 (5.3)</td>
<td>58 (6.7)</td>
<td>0.78 (0.57–1.03)</td>
<td>53 (6.2)</td>
</tr>
<tr>
<td>F</td>
<td>69 (8.1)</td>
<td>63 (7.4)</td>
<td>1.10 (0.86–1.38)</td>
<td>74 (8.7)</td>
</tr>
<tr>
<td>G</td>
<td>63 (8.0)</td>
<td>56 (7.2)</td>
<td>1.12 (0.87–1.43)</td>
<td>69 (8.8)</td>
</tr>
<tr>
<td>H</td>
<td>48 (7.4)</td>
<td>47 (7.3)</td>
<td>1.02 (0.76–1.34)</td>
<td>52 (8.1)</td>
</tr>
<tr>
<td>All</td>
<td>572 (7.9)</td>
<td>560 (7.8)</td>
<td>1.02 (0.94–1.11)</td>
<td>584 (8.1)</td>
</tr>
</tbody>
</table>

Data are presented as n (%) (95% CI). Expected hospital mortality was calculated using a multiple logistic regression model adjusted for ethnicity, assisted conception, multiple gestation, hypertensive disease of pregnancy, antenatally diagnosed malformation, choioamnionitis, any antenatal steroids, outborn, method of birth, gender, gestational age, birth weight percentiles, Apgar score <7 at 5 min and postnatal steroid.

### Figure 1  Funnel plot showing observed mortality rates in neonatal intensive care units in New South Wales and Australian Capital Territory between 2007 and 2014 for infants born at 23 to 31 (A), 23 to 26 (B), 27 to 28 (C) and 29 to 31 (D) weeks of gestation. Average, 95% and 99.8% lower and upper control limits are shown.
fundamental changes in the underlying process. Special-cause variation, which is shown as out-of-control signals in the chart (ie, data point outside the ±99.8% limit), is the result of factors extrinsic to the process, and its reduction therefore requires identification of and action on the special causes. Opportunities for improvement in infrastructure and clinical practice could be drawn from hospitals operating below the lower −99.8% limit. In our study, no hospital showed special-cause variation.

Detailed framework for improvement using process control is discussed elsewhere. Another advantage of using funnel plots is to use the +95% (2σ) control limits to encourage further investigation. Hospitals exceeding this limit should start early quality improvements to make sure the outcome does not become out of control. Similarly, opportunities for improvement in infrastructure and clinical practice could be drawn from hospitals operating below the lower −99.8% limit.

CTC variation similar to that which we observed has been reported from other countries including the USA and UK, Canada, Germany, Finland, Switzerland and Japan. The causes of this variation are difficult to ascertain from this study and deserve further consideration. However, others have suggested that variation could be related to illness severity, different attitudes and thresholds to limiting or withdrawing intensive care measures or different management styles among NICUs.

This study has substantial strengths. It is one of the larger NICU network reports using validated data with standardised published definitions. All data were coded prospectively, enhancing reliability and accuracy. Our statistical approach is robust as it took case mix and NICU volume into consideration. Our data lacked certain information pertinent to neonatal outcomes, such as fetal compromise and sociocultural factors. Consequently, there is likely to be some residual confounding despite the high discrimination and goodness of fit of the logistic regression model. Delivery room deaths were not included in our database so we could not detect interhospital variation or determine causes for these deaths. Another limitation is that the maternal record is entered multiple times for ‘multiple and higher order infants’. Therefore, mothers of more than one premature neonate within the study frame appear more than once in table 1 and the number of mothers and neonates is equal (721). As the data are de-identified, we are unable to account for this. But we believe, particularly given relatively low parity in Australia, that the number of mothers included more than once would be small in the 7-year study period.

CONCLUSION AND POLICY IMPLICATIONS

Our data showed trends of risk-adjusted CTC mortality variation with a large NICU network. Centre-specific factors other than differences in baseline demographics and NICU volume may be involved. There is an urgent need to delineate these factors. The approach presented in our paper should form the basis for action on neonatal mortality variation and could easily be employed in real or near real time to detect variation early to facilitate timely interventions. In addition, there is a need to study variation in the main morbidities and neurodevelopmental outcomes between these hospitals.

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Arch Dis Child Fetal Neonatal Ed: first published as 10.1136/archdischild-2017-313222 on 26 October 2017. Downloaded from http://fn.bmj.com/ on October 31, 2023 by guest. Protected by
Acknowledgements The authors thank the Directors, the New South Wales (NSW) and the Australian Capital Territory (ACT) Neonatal Network (NICUS) members and the audit officers of all tertiary units in supporting this collaborative study: NICUS, Dr Jennifer Bowen (Chairperson), Barbara Bajuk (Coordinator), Sara Sedgley (Research Officer); Canberra Hospital, Dr Hazel Carlisle (Director), Judith Smith; John Hunter Children’s Hospital, Dr Paul Craven (Director), Lynne Cruden, Alissa Argomand; Royal Prince Alfred Hospital, Dr Ingrid Rieger (Director), Dr Girvan Malcolm, Shelley Reid; Liverpool Hospital, Dr Jacqueline Stack (Director), Dr Ian Callander, Kathryn Medlin, Kaye Marcin; Nepean Hospital, Dr Vijay Shingde (Director), Mei Fong Chin, Kerrie Bonser; The Children’s Hospital at Westmead, Prof Nadia Badawi (Co-Director), Dr Robert Halliday (Co-Director), Caroline Karskens; Royal North Shore Hospital, Dr Mary Paradisis, Prof Martin Kluckow, Claire Jacobs; Sydney Children’s Hospital, Dr Andrew Numa (Director), Dr Gary Williams, Janelle Young; Westmead Hospital, Dr Melissa Luig (Director), Jane Baird; and Royal Hospital for Women, Prof Kei Lui (Director), Diane Cameron. The authors also thank the babies and their families, the nursing and midwifery, obstetric and medical records staff of the obstetric and children’s hospitals in NSW and the ACT.

Contributors ME-L conceptualised and designed the study, carried out the statistical analyses, drafted the initial manuscript and led the writing. GN contributed to the statistical analysis and interpretation. BB retrieved and cleaned the data and contributed to statistical interpretation. KG contributed to the statistical interpretation. DH contributed to the study design and statistical interpretation and led the writing of manuscript. All authors critically reviewed and approved the final manuscript as submitted. All authors had full access to all of the data (including statistical reports and tables) in the study and can take responsibility for the integrity of the data and the accuracy of the data analysis.

Competing interests None declared.

Ethics approval The Australian National University (No 2015/029), ACT Health Human Research Ethics Committee (No EHFL.11.09/1031) and Hunter New England Research Ethics and Governance Unit has approved this study.

Provenance and peer review Not commissioned; externally peer reviewed.

Data sharing statement All authors had full access to all of the data (including statistical reports and tables) in the study and can take responsibility for the integrity of the data and the accuracy of the data analysis. No other data from the study are available.

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