LETTERS TO THE EDITOR

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Iron deficiency anaemia, *helicobacter pylori* infection and delayed pubertal growth

We read with great interest the paper by Choe and colleagues which investigated a possible relationship between *Helicobacter pylori* infection, iron deficiency anaemia (IDA), and subnormal growth at puberty.[1] The authors concluded that *H pylori* infection and related IDA, rather than bacterial infection alone, might cause delayed pubertal growth. However, we believe that these results need some consideration.

H pylori infection may cause IDA in different ways: (a) the bacterium can cause a decrease in the gastric juice of the concentration of ascorbic acid, which is the best promoter of non-heme iron absorption[2]; (b) *H pylori* may increase iron demand because iron is an essential bacterial growth factor; (c) *H pylori* contains a 19.6 kDA protein resembling ferritin with a binding activity for heme iron in erythrocytes[3]; (d) acute or chronic blood loss and IDA are associated with typical *H pylori* related gastroduodenal lesions. In Choe's study, the treatment of *H pylori* infection with antibiotics (but also with proton pump inhibitors) was probably linked to a more rapid response to oral iron replacement because of the effects of proton pump inhibitors in healing some of the lesions.

However, IDA, failure to thrive, and delayed pubertal growth are important features of subclinical and silent coeliac disease. Therefore, coeliac disease should be suspected in children or adolescents with these signs, especially when IDA is resistant to oral iron replacement.

In a recent study,[4] IDA seemed to be the most frequent extra-intestinal marker of coeliac disease in both children and adults, followed by short stature for children. Thus, it would be advisable to screen paediatric and juvenile population with IDA, failure to thrive, or delayed pubertal growth for coeliac related antibodies (anti-endomysial, anti-transglutaminase, antigliadin) in order to identify coeliac patients who need lifelong gluten withdrawal for recovery of iron, and improved metabolism and growth.

Finally, it is reasonable that *H pylori* infection may make iron deficiency worse in coeliac patients and subsequently impair growth in children who need a large amount of this essential element.

L CUOCO
G CAMMAROTA
R A JORIZZO
R CIANCI
G GASBARRINI
Università Cattolica del Sacro Cuore, Policlinico "A.Gemelli", Istituto di Medicina Interna e Geriatria, Largo A.Gemelli, 8, 00168 Roma, Italy
gcammarota@libero.it

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Iron status and development

EDITOR,—You have recently published a response by Dr Morley to Dr Stevens concerning our paper on iron status and development.[1] We wish to raise two matters not discussed in Dr Morley's letter.

Firstly, when the paper was published, there was prominent media coverage, erroneously concluding that our paper rendered formula unnecessary in the later part of the first year, and that cow's milk was just as satisfactory. We emphasise that the reason for avoiding cow's milk in the first year (in babies not breast fed) is based on several considerations, including suboptimal nutrient content or bioavailability (for example, for vitamins D and C, iron, and copper), unnecessarily high protein content, and, possibly, increased risk of subclinical gastrointestinal bleeding. Our paper does not provide evidence that should change present policy on cow's milk feeding.[2]

Secondly, the haemoglobin data we included, which were not central to the focus of the paper, were from one centre only and should not be taken as typical of those for the UK population. A more complete presentation of iron status in children in the trial is now published.[3]

The current evidence on the importance of iron in infancy is much debated and frequently cited in policy documents. We wish the interpretation of our study to be as clear as possible.

ALAN LUCAS
ATUL SINGHAL
MRC Childhood Nutrition Research Centre, Institute of Child Health, 30 Guildford Street, London WC1N 1EH, UK

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Coeliac screening just as important!

EDITOR,—The paper by Noble *et al* on capillary dried spot testing for thyroid stimulating hormone measurement is a welcome advance for children with Down's syndrome (DS).[1] In reducing the number of venepunctures this patient group needs, we hope that those professionals caring for children with DS do not omit screening for coeliac disease (CD). This condition is equally prevalent and can be as difficult to diagnose as hypothyroidism in DS, as it too is often asymptomatic. Screening for CD should be done by the measurement of antigliadin (AGA) and antiendomysial (EmA) antibodies. Their use as a screening tool is well described in DS with reported prevalence between 3.9% and 16.9%.[2–4] Diagnosing CD has important consequences with regard to preventing long term complications and maximising growth potential.[4] We would like to highlight that community based testing is also feasible for CD.

A study at our centre investigating the prevalence of coeliac disease in type I diabetics utilised patient self sampling for screening blood samples.[5] Blood was drawn into a lithium heparin capillary tube (Monovette, Sarsdedt Ltd, Germany) or onto filter paper. The in house assays used for AGA and EmA were performed on 10–20 ml of serum or plasma; thus capillary samples were more than adequate. This method could easily be incorporated into the "at school" testing described by the authors.

Annual screening for hypothyroidism is recommended.[1] How often screening should be performed for CD is still a matter of debate. With their proposal to establish a Scottish register of school aged children with Down's syndrome, Noble *et al* provide the opportunity to perform a Scottish-wide population study for the prevalence of coeliac disease in Down's syndrome and, more importantly, to identify those children who may benefit from early detection. Community based screening with capillary samples would make that a very realistic prospect.

R K RUSSELL
P M GILLETT
Department of Paediatric Gastroenterology, Royal Hospital for Sick Children, 9 Sciennes Road, Edinburgh EH9 1LF, UK
email: richardkrussell71@hotmail.com

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Consider absolute risks in SIDS prevention

EDITOR,—The demonstration by Blair *et al* of an association between poor postnatal growth and an increased risk of sudden infant death syndrome (SIDS),[1] is a useful addition to our understanding of the aetiology of this condition. It is unfortunate that the conclusion in the abstract that "Poor postnatal weight gain was independently associated with an increased risk of SIDS and could be identified at the routine six week assessment" goes beyond the data presented.

It can be estimated from the data in this study that the overall risk of SIDS was 0.77/1000 live births. The risk in babies with birth weights greater than the 15th centile, the group in whom the relation with postnatal growth was detected, was 0.68/1000. Given the reported odds ratio of 1.75 associated with being in the slowest growing 16% who might be identified at six weeks, the data suggest that the absolute risk of SIDS among this group would be about 1.1/1000. Even a programme targeted at infants below the 2nd centile for growth at six weeks, would identify a group whose absolute risk of SIDS was about 4.2/1000—that is, for every infant who might benefit from the intervention, there would be 240 who would not. Even if it were accepted that this level of risk was sufficient to trigger an intervention, the nature of the intervention remains unclear. To the best of our knowledge, with the exception of the "Back to Sleep" campaign, there is no convincing evidence of the effectiveness of any intervention aimed at preventing SIDS. None of the intervention programmes described in the accompanying commentary[2] have been evaluated in appropriately controlled studies.

The discussion in the paper is rather more circumspect than the conclusion in the abstract, but it is the latter which, reinforced by the accompanying commentary, is likely to have disproportionate impact on those readers who do not read the whole paper. In the commentary Dr Carpenter acknowledges that the low rates of SIDS make such interventions difficult to justify but then suggests that "Targeting such infants for weight monitoring at home . . . will also identify numerous other problems at a remedial stage". This is a suggestion for which there is currently little evidence.

There are clear dangers in recommending population interventions on the basis of relative risks without taking account of absolute risks or of the need for interventions to be of proven effectiveness.

STUART LOGAN
HELEN BEDFORD
Department of Paediatric Epidemiology, Institute of Child Health, 30 Guilford Street, London WC1N 1EH, UK
email: slogan@ich.ucl.ac.uk

DAVID ELLIMAN
St George's Hospital, Blackshaw Road, London SW17 0QT, UK

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Drs Blair, Fleming, and Platt comment:

Poor postnatal weight gain was a significant factor in the multivariate analysis despite controlling for low birth weight, prematurity, neonatal problems, poor socioeconomic status, and many other potential confounding factors, and remained significant when further highly predictive covariates of SIDS such as infants put down prone, infants found with head covered, and tobacco exposure were added to the model. In this sense postnatal weight gain was independently associated with an increased risk of SIDS. We also found the difference in weight gain between the SIDS and control infants measured from birthweight to the last known weight was equally apparent if measured from birth to the six week assessment. We therefore stand by our conclusion in the abstract that poor postnatal weight gain "was independently associated with an increased risk of SIDS" and also that poor postnatal weight gain "could be identified at the routine six week assessment".

We find it difficult to understand how this conclusion advocates an intervention campaign on the basis of these two statements and disagree with Logan and colleagues that our discussion on this matter was circumspect. We point out clearly that poor weight gain itself is not a sensitive marker and that it "should be seen as a thread in a web of factors that render an infant vulnerable to SIDS and is both a consequence of adverse health and social conditions".

We agree that absolute risks must be used for targeted prevention campaigns but do not advocate such a campaign based on our solitary finding. Preliminary analysis of risk scoring on the first two years of our dataset, tested on the third year show that 42% of SIDS families can be identified from 8% of the population using just a few prenatal factors. Incorporating postnatal factors such as weight gain may improve the specificity and sensitivity of such a scoring system.

To our knowledge the "Back to Sleep" campaign initiated in October 1991 has not been evaluated in an appropriately controlled study yet the SIDS rate in England and Wales more than halved from 1.7 deaths per 1000 live births in 1990 to 0.77 deaths in 1992.[1] Findings from our study have helped build on the advice regarding safe sleeping practices; parents are now advised not only to avoid placing infants in the prone position but also the side position, bed sharing under certain circumstances, sharing a sofa to sleep, and to avoid head covering by placing the feet of the infant at the foot of the cot. In 1998 the SIDS rate fell a further 25% in England and Wales to 0.45 deaths per 1000 livebirths.[2] There is no direct evidence that these recommendations and fall in SIDS rates are linked although the dramatic fall in the prevalence of parents placing their infant in the prone position to sleep (57% before the campaign[3] to 3% after the campaign[4]) suggest many parents take up such recommendations.

A targeted population intervention would perhaps be inappropriate given the reduced number of SIDS deaths in this country but risk scoring systems could be used to identify and study "high risk" families to both increase our understanding of the risks associated with the infant sleeping environment and improve the advice we give parents in the hope that the number of SIDS deaths reduces still further.

PETER S BLAIR
PETER J FLEMING
FSID Research Unit, Department of Child Health, Royal Hospital for Children, St Michael's Hill, Bristol BS2 8BJ, UK
email: p.s.blair@bris.ac.uk

MARTIN WARD PLATT
Newcastle Neonatal Service, Ward 35, Royal Victoria Infirmary, Newcastle-upon-Tyne NE1 4LP, UK

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Are we requesting too many DMSA scans?

EDITOR,—The recent article by Christian *et al* highlights the value of clinical features in assessing the risk of renal scarring and therefore the need for dimerceptosuccinic acid (DMSA) scan after urinary tract infection (UTI).[1] We recently performed a case note study to assess the recording of fever, malaise, recurrent UTI, and urine culture results in children investigated with DMSA scan after UTI. Between April 1996 and October 1997 there were 171 DMSA scans in our hospital that fitted these criteria; 30 case notes could not be traced. There were 105 girls (74%) and 36 boys. Age when UTI was diagnosed ranged from 9 days to 15.3 years (mean 4.2 years, SD 3.2).

Urine culture results were: UTI ($>10^5$ cfu/ml) in 82 cases (58%), contaminant ($<10^5$ cfu/ml) in 27 cases (19%), no growth in 21 cases (15%), and no urine culture in 11 cases (8%). There were 17 (12%) cases of definite or probable renal scar, none of which followed a sterile or contaminated urine culture. Of the 141 case notes, there was no mention of fever in 48 (34%), and no mention of malaise in 76 (54%). In 69 case notes reviewed there was no mention of previous history of UTI in 14 (20%) cases. Of those with a history of fever, 19% (10/53) had an abnormal DMSA scan compared to 10% (4/40) in those without fever. Eighteen per cent (9/50) of those unwell at the time of UTI had an abnormal scan compared to 13% (2/13) of those not ill.

These data suggest that in a substantial proportion of cases, the decision to request a DMSA scan is apparently not influenced by salient clinical features and urine culture results. In this series, it would appear that those children with sterile or contaminated urine cultures should not have had a DMSA scan. This would have saved the cost and burden of 48 scans, 34% of this series, over an 18 month period. It is unlikely that these findings are peculiar to our district.

A H SACKEY
Leighton Hospital, Middlewich Road, Crewe CW1 4QJ, UK
email: ahsackey@lineone.net

1 Christian MT, McColl JH, MacKenzie JR, Beattie TJ. Risk assessment of renal cortical scarring with urinary tract infection by clinical features and ultrasonography. *Arch Dis Child* 2000;**82**:376-80.