Mutations in the hereditary haemochromatosis gene HFE in professional endurance athletes

J L Chicharro, J Hoyos, F Gómez-Gallego, J G Villa, F Bandrés, P Celaya, F Jiménez, J M Alonso, A Córdova, A Lucía

Background: Hereditary haemochromatosis, a disease that affects iron metabolism, progresses with a greater or lesser tendency to induce iron overload, possibly leading to severe organ dysfunction. Most elite endurance athletes take iron supplements during their active sporting life, which could aggravate this condition.

Objective: To determine the prevalence and discuss potential clinical implications of mutations of HFE (the gene responsible for hereditary haemochromatosis) in endurance athletes.

Methods: Basal concentrations of iron, ferritin, and transferrin and transferrin saturation were determined in the period before competition in 65 highly trained athletes. Possible mutations in the HFE gene were evaluated in each subject by extracting genomic DNA from peripheral blood. The restriction enzymes XmnI and BclI were used to detect the mutations 845G\rightarrow A (C282Y) and 187C\rightarrow G (H63D).

Results: Our findings indicate a high prevalence of HFE gene mutations in this population (49.2%) compared with sedentary controls (33.5%). No association was detected in the athletes between mutations and blood iron markers.

Conclusions: The findings support the need to assess regularly iron stores in elite endurance athletes.

H
ereditary haemochromatosis is an autosomal recessive disease in which the body’s iron stores are increased, with serious negative effects on the function of several organs (liver cirrhosis, diabetes mellitus, heart failure). The clinical consequences of iron overload in these patients can be prevented by early diagnosis and appropriate treatment.

The HFE gene plays a major role in hereditary haemochromatosis. It occurs in the short arm of chromosome 6. Most patients with manifest hereditary haemochromatosis are homozygous for the C282Y mutation, and a small proportion are heterozygous for both the C282Y and H63D mutation of the HFE gene. Only a few affected cases show C282Y homozygotic or H63D homozygotic. Subjects with these two genotypes generally display fewer markers of iron overload than C282Y homozygotes. Although these patients have a very low or no risk of developing cirrhosis or other complications, some authors suggest that they should be treated similarly to C282Y homozygotes. Moreover, despite the compound C282Y/H63D heterozygous defect expressing the disease with low penetrance (0.44–1.5%), it has been identified as an independent risk factor for death from cardiovascular causes or myocardial infarction.

There is considerable evidence to suggest that mutations in the HFE gene, even H63D heterozygosity, affect blood iron indices, and subjects with one or more mutations show higher blood iron concentrations and transferrin saturation than subjects without mutations. Deugnier et al recently explored factors that induce the increased iron reserves observed in elite cyclists, and suggested that increased serum ferritin was mainly related to the excessive iron supplements taken by these athletes rather than to mutations in the HFE gene (C282Y and H63D).

Despite the tremendous advances made in the area of genetic mutations in disease since 1996, genetic evaluations such as screening the general population are not recommended. Phenotypic analysis based on transferrin saturation still seems to be the best and most economical diagnostic method to either preclude or warrant a more in depth analysis of the disease in a particular subject. Thus, if transferrin saturation is >45%, a genetic analysis of HFE mutations and serum ferritin determination would be called for, to better estimate the subject’s iron stores.

This study was designed to evaluate the prevalence of HFE mutations in elite endurance athletes. The results were compared with those of a control group matched by region of origin.

MATERIALS AND METHODS

Subjects
Sixty five elite, male athletes (50 professional road cyclists and 15 Olympic class endurance runners) from Spain were enrolled in the study. Written consent was obtained from each subject according to the guidelines of the Universidad Complutense, Madrid.

The mean (SD) age, height, mass, and maximum oxygen consumption (VO₂\text{\text{MAX}}) of the athletes were: 26 (3) years, 178 (5) cm, 66.7 (6.1) kg, and 71.8 (7) ml/kg/min respectively. The subjects were previously confirmed to be healthy by a medical examination including electrocardiography and cardiac ultrasonography. No subject had a familial or personal history of endocrine or metabolic disease. No exogenous substances had ever been detected in anti-doping checks performed in the subjects by the corresponding official organisations. Most (90%) of the athletes took iron supplements (not including the C282Y/H63D heterozygous subject (see the Results section)) at a mean dose of 105 mg Fe on alternate days for at least six months of the year. Doses and treatment regimens were similar in each subject.

A control group (n = 134) composed of random sedentary men from Spain also entered the study.

Measurement of blood iron markers in the athletes
Fasting blood samples were collected from all the athletes after at least three rest days during the period before competition. When available, serum ferritin concentrations...
one minute, 55 °C for 45 seconds, 72 °C for one minute, and a denaturation at 95 °C for five minutes; 35 cycles at 95 °C for mutation. The digested fragments were visualised by electrophoresis on 2% agarose gels stained with ethidium bromide.

Genomic DNA was extracted from peripheral blood using a standard phenol/chloroform procedure followed by alcohol precipitation. DNA amplification was performed using polymerase chain reaction (PCR) with specific primers for the two HFE gene mutations as described previously. The PCR conditions for both mutations were as follows: initial denaturation at 95 °C for five minutes; 35 cycles at 95 °C for one minute, 55 °C for 45 seconds, 72 °C for one minute, and a final extension at 72 °C for five minutes. The PCR products were then subjected to enzymic digestion for two hours, with restriction endonucleases cleaving the DNA at specific points such that the presence or absence of the mutations could be detected. The restriction enzymes used were SmaBI for the 845G→A (C282Y) mutation and BclI for the 187C→G (H63D) mutation. The digested fragments were visualised by electrophoresis on 2% agarose gels stained with ethidium bromide (fig 1).

Serum ferritin concentrations were measured by chemiluminescence immunoassay (N-lateX ferritin kit; Dade Behring Marburg GmbH). The coefficients of interassay and intra-assay variability averaged 1.2% and 1.8% for serum iron, 2.3% and 2.7% for transferrin concentration, and 1.2–3.1% and 1.0–4.6% for serum ferritin.

Serum ranges considered normal were 13–32 mmol/l for iron, 24–336 g/l for transferrin, 24–45% for transferrin saturation, and 20–300 μg/l for ferritin.

Study of C282Y and H63D mutations in all subjects

Genomic DNA was extracted from peripheral blood using a standard phenol/chloroform procedure followed by alcohol precipitation. The different variables were also compared in the athlete group in terms of the presence or absence of mutations (table 3). No differences were observed among the subgroups.


disturbing iron metabolism. Although there appeared to be no relation in the athlete group between the presence and absence of mutation with respect to the blood iron markers, it is observed that 61.1% of subjects, yet is similar to rates recently observed by Deugnier et al., who also warned of a higher prevalence of H63D mutations in French cyclists compared with healthy controls. It remains to be seen if these mutations afford any metabolic advantage to these athletes during exertion. We detected no significant differences in VO2MAX between subjects with or without the mutation, thus we cannot confirm this hypothesis.

The most important finding of our study was the high proportion of endurance athletes with a mutation in the HFE gene (49.2%), 29 (44.6%) of whom carried an H63D mutation and three (4.6%) a C282Y mutation. The prevalence of H63D heterozygosity occurred in 41.5% of the athletes and 24.6% of the controls (p = 0.01). No other significant difference was found between groups. No homozygote for the C282Y mutation was detected in athletes or controls.

Table 2 shows biometric variables, maximum aerobic capacity, and blood iron markers in subgroups of athletes established according to the type of HFE mutation carried. No significant differences in any of the variables (p>0.05) were detected among subgroups.

The different variables were also compared in the athlete group in terms of the presence or absence of mutations (table 3). No differences were observed among the subgroups.

DISCUSSION

The results were compared using a standard phenol/chloroform procedure followed by alcohol precipitation and the standard colorimetric method (Roche/Hitachi 714; Roche Diagnostics Corporation, Indianapolis, Indiana, USA). Serum iron concentrations were measured using a standard colorimetric method (Roche/Hitachi 714; Roche Diagnostics Corporation, Indianapolis, Indiana, USA). Serum ferritin saturation was calculated from these data as follows:

Transferrin saturation (%) = (serum iron concentration (mol/l)/(2 × transferrin concentration (mol/l))) × 100.

Serum ferritin concentrations were measured by chemiluminescence immunoassay (N-lateX ferritin kit; Dade Behring Marburg GmbH). The coefficients of interassay and intra-assay variability averaged 1.2% and 1.8% for serum iron, 2.3% and 2.7% for transferrin concentration, and 1.2–3.1% and 1.0–4.6% for serum ferritin.

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Statistical analysis

All variables were tested for normality. We compared the distribution of HFE genotypes in both athlete and control groups with the Fisher exact test. Ferritin data for athletes were log transformed to normalise the distribution before analysis. Differences between genotypes in the athlete group were identified by one way analysis of variance. In this group, the paired t test was used to compare the variables according to the presence or lack of mutations. Correlation between paired quantitative data was assessed by the Spearman test. All statistical analyses were performed using SPSS 9.0 software for Windows. The level of significance was set at 0.05.

RESULTS

Table 1 shows the prevalence of the different HFE gene mutations in the two groups. The proportion of subjects without a HFE gene mutation was significantly higher in the control group than in the athlete group (66.5% vs 50.8%; p = 0.03). H63D heterozygosity occurred in 41.5% of the athletes and 24.6% of the controls (p = 0.01). No other significant difference was found between groups. No homozygote for the C282Y mutation was detected in athletes or controls.

Table 2 shows biometric variables, maximum aerobic capacity, and blood iron markers in subgroups of athletes established according to the type of HFE mutation carried. No significant differences in any of the variables (p>0.05) were detected among subgroups.

Table 1: Percentage distribution of HFE genotypes in elite endurance athletes and sedentary controls

<table>
<thead>
<tr>
<th>Genotype</th>
<th>Athletes (n = 65)</th>
<th>Controls (n = 134)</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>wt/wt</td>
<td>50.8</td>
<td>66.5</td>
<td>0.03</td>
</tr>
<tr>
<td>C282Y/wt</td>
<td>3.1</td>
<td>4.5</td>
<td>NS</td>
</tr>
<tr>
<td>H63D/wt</td>
<td>41.5</td>
<td>24.6</td>
<td>0.01</td>
</tr>
<tr>
<td>C282Y/H63D</td>
<td>1.5</td>
<td>0.7</td>
<td>NS</td>
</tr>
<tr>
<td>H63D/H63D</td>
<td>3.1</td>
<td>3.7</td>
<td>NS</td>
</tr>
</tbody>
</table>

wt/wt, Wild-types; C282Y/wt, heterozygotes for the C282Y mutation; H63D/wt, heterozygotes for the H63D mutation; C282Y/H63D, compound heterozygotes; H63D/H63D, homozygotes for the H63D mutation; NS, not significant.
ELECTRONIC PAGES

BJSM Online case reports: http://bjsm.bmjournals.com/

The following electronic only articles are published in conjunction with this issue of BJSM.

Upper airway obstruction masquerading as exercise induced bronchospasm in an elite road cyclist
K E Fallon

This case concerns an elite road cyclist who complained of occasional dyspnoea and inspiratory difficulty during intense exercise. Clinical examination was normal and the final diagnosis was vocal cord dysfunction, a paradoxical closure of the vocal cords during inspiration which is highly associated with inspiratory stridor at high rates of ventilation. Awareness by the sports physician of this not uncommon condition is important to avoid misdiagnosis.

( Br J Sports Med 2004;38:e9) http://bjsm.bmjournals.com/cgi/content/full/38/4/e9

Cardiovascular stress on an elite basketball referee during neonatal competition
A S Leicht

This case report examined the cardiovascular stress imposed on an experienced elite basketball referee during national competition. The average heart rate was similar for all matches, approximated 73% of maximum heart rate, and was experienced for most (>63%) of the match. Similar relative exercise intensity was demonstrated regardless of match play (men’s v women’s) and officiating type (two v three-referee). Further study is needed to document the physiological characteristics of elite basketball referees for greater performance.

( Br J Sports Med 2004;38:e10) http://bjsm.bmjournals.com/cgi/content/full/38/4/e10

An unusual presentation of immersion foot
D M Macgregor

We report a case of “green foot” in a child with a plaster cast applied for a fractured metatarsal who subsequently represented with circulatory compromise. The foot was green and smelly and profuse Pseudomonas aeruginosa was cultured. The infection cleared with simple exposure to air. Perhaps this diagnosis should be considered in patients presenting with circulatory compromise in a cast as severe infection can result in amputation.

( Br J Sports Med 2004;38:e11) http://bjsm.bmjournals.com/cgi/content/full/38/4/e11
Mutations in the hereditary haemochromatosis gene HFE in professional endurance athletes

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doi: 10.1136/bjsm.2002.003921

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/content/38/6/793.1.full.pdf

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Sodium ingestion and the prevention of hyponatraemia during exercise

The study of Twerenbold et al. is important for a number of reasons, not all of which may have been emphasised sufficiently by the authors.

Firstly, it confirms that a rate of fluid intake of 1000 ml/h is too high for a group of female runners running at ~ 10 km/h and who would therefore complete a 42 km marathon in about 4.25 hours. As the athletes drank 4 litres and gained 2 kg during the trial, their average rate of weight loss (as opposed to sweat rate) was about 500 ml/h. As not all of the weight lost during exercise is sweat and as much as 1–3 kg of this weight loss may result from fuel and water losses that do not contribute to dehydration, the absolute maximum rate at which these athletes should have ingested fluid during exercise was probably even less than 500 ml/h. This is substantially less than the drinking guidelines of the American College of Sports Medicine and the Gatorade Sports Science Institute which recommended rates of fluid ingestion of up to 1200–1800 ml/h. As there is no evidence that gaining weight during exercise improves performance there is good evidence that athletes who either lose no weight or who gain weight during exercise are increasingly likely to (a) have an impaired performance, (b) develop troubling gastrointestinal symptoms, or (c) finish the race with serum sodium concentration below the currently recommended 137 mmol/l.

As the currently established best practice is not to ingest fluid at such high rates that weight is gained during exercise, because this practice can produce a fatal outcome, so this study design should, in retrospect, not have been sanctioned. Rather, the control group in the study should have ingested fluid according to guidelines based on the strongest body of current information. It is, for obvious reasons, my biased opinion that the guidelines that come closest to a defendable best practice are those that have been recently accepted by the United States Track and Field and the International Marathon Medical Interest Group which specifically advised not to overdrink during exercise. This recommendation is currently established “best practice”.

Fortunately the data of Twerenbold et al. do allow some calculations to estimate the likely value of the extra sodium that was ingested by two of their groups. Thus, the athletes in their study could have consumed 54 mmol/l of ingested hyponatraemic encephalopathy, it is not immediately clear why the authors chose such high rates of fluid intake in these athletes. Except, perhaps, if they wished to prove the value of sodium ingestion during exercise. I note, for example, that the study was funded by a commercial company that, to my knowledge, markets a sports drink containing sodium chloride.

For it seems highly probable that if athletes overdrink so that they retain fluid and gain weight, then the extent to which their serum sodium concentration falls will be influenced, albeit to a quite limited extent, by the sodium content of the ingested fluids. This indeed was shown by the results of this study. But whether that finding has relevance to the sodium requirements of athletes who are specifically advised not to overdrink during exercise to ensure that they do not develop hyponatraemic encephalopathy is an entirely different question, which cannot be answered with the study design chosen by these authors.

For example, the presence of a control group who drank according to the dictates of thirst (“ad libitum”) and not according to the guidelines of influential sports medical and commercial organisations, so that they may be less prone to overdrink and so to gain weight during exercise, would have established that athletes who lose more than 1–3 kg during exercise do not develop symptomatic hyponatraemic encephalopathy even though they are both dehydrated and sodium deficient. Rather, they are more likely to finish such races with raised serum sodium concentrations.

I would rather argue that a fundamental feature of all prospective trials that aim to evaluate a novel intervention such as the role of sodium ingestion in the prevention of hyponatraemia during exercise should be to compare the new intervention with the currently established best practice.

As the currently established best practice is not to ingest fluid at such high rates that weight is gained during exercise, because this practice can produce a fatal outcome, so this study design should, in retrospect, not have been sanctioned. Rather, the control group in the study should have ingested fluid according to guidelines based on the strongest body of current information. It is, for obvious reasons, my biased opinion that the guidelines that come closest to a defendable best practice are those that have been recently accepted by the United States Track and Field and the International Marathon Medical Interest Group which specifically advised not to overdrink during exercise. This recommendation is currently established “best practice”.

For example, if each group did indeed lose 84 mmol Na+ as did group H (table 1), a value that seems eminently reasonable as it equates to a quite reasonable sweat Na+ of ~40 mmol/l, then the true ECF volume in the W group after the race would have been 14.5 litres—that is, it is unchanged from the starting value. The value (expressed in litres) is calculated as: (pre-race ECF Na+ content – 84) in mmol divided by post-race serum Na+ in mmol/l.

Indeed, if subjects in the W group did lose 84 mmol Na+ during the race but also had a post-race ECF volume expanded to 14.95 litres, then their post-race [Na+] would have been even lower (128 mmol/l) than that actually measured after the race (132 mmol/l; table 1). It is therefore almost certain that the authors should have ingested fluid during exercise. This calculation elegantly shows why small changes in ECF volume determine whether or not hyponatraemic
encephalopathy will develop in those who overdrink, regardless of whether or not they also incur a Na⁺ deficit either during exercise or at rest. A recent paper confirms these predictions by showing that mathematical modelling supports the argument that changes in TBW exert a much greater effect on serum [Na⁺] than does whole body Na⁺ content in those who overdrink and hence gain weight during exercise. Perhaps the point of these calculations is to show that it is not possible to calculate the state of Na⁺ balance in athletes during exercise and so to determine whether or not athletes have developed a Na⁺ deficit, simply by measuring serum [Na⁺]. This is because the ECF volume will not be the same before, during, and after exercise and will change depending on the nature of the fluid ingested and the extent of any fluid deficit or excess that develops during exercise. But more importantly, these calculations clearly show why the regulation of the TBW and the ECF volume will have a much greater influence on serum [Na⁺] than will either the expected Na⁺ losses in sweat or the amount of Na⁺ ingested from sodium-containing sports drinks.

For example a 1 litre (7%) reduction in the ECF volume would “release” 140 mmol Na⁺ into the contracted ECF volume. This means that it is possible to lose 140 mmol Na⁺ in sweat and urine without any change in serum [Na⁺] provided that the ECF volume were to contract by only 7%. If sweat [Na⁺] is about 40 mmol/l, as appears to have been the case in this study of Twerrenbold et al. (table 1), then this 140 mmol is the equivalent of the Na⁺ content of about 3.5 litres of sweat.

As athletes in this study sweated at a maximum rate of only 500 ml/h when running at 10 km/h, this means that simply by losing 140 mmol Na⁺ in sweat and urine without any change in serum [Na⁺], it is possible to lose 140 mmol Na⁺ even in the face of quite large and unrequited Na⁺ loss in sweat, and (b) why the ingestion of sodium-containing sports drinks in the vain hope of matching the rates of Na⁺ loss in sweat can only lead to fluid retention and progressive hyponatraemic encephalopathy, as elegantly shown by this study. Indeed if this inappropriate behaviour is adopted, the result will be death from hyponatraemic encephalopathy, which, as these calculations and this study again show, cannot occur without the presence of distinct fluid overload.

Finally, it is important to note that, even though Na⁺ ingestion marginally increased serum [Na⁺] in the group that ingested the most concentrated Na⁺ drink, this practice was without benefit as running performances were unaltered by Na⁺ ingestion, and the incidence of symptoms was no different between the groups as no athletes reportedly developed symptoms. However, the symptoms of mild hyponatraemic encephalopathy are mild and may not have been sought with sufficient diligence. For example, all subjects, myself included, in our study in which mild hyponatraemia was induced by fluid overload at rest, developed quite disabling symptoms at serum [Na⁺] of ~136 mmol/l or lower. Indeed it would have been most interesting to determine whether the presence of subtle mental symptoms was different in the three groups in this study, as all had similar degrees of fluid overload despite different serum [Na⁺]. If the symptoms in this condition are due purely to fluid overload, then the incidence of symptoms should have been the same in all groups despite different serum [Na⁺]. Alternatively, if the symptoms are related to the degree of hyponatraemia, then they should have been most obvious in the W group, who finished with the lowest post-race serum [Na⁺]. My bias would be to expect that the extent of any symptoms are more likely related to the degree of fluid overload, and hence the increase in the ICF, than to the level to which the serum [Na⁺] has been reduced.

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doi: 10.1136/bjsm.2004.014191
Conflict of interest: none declared

References


Table 1 Sodium balance calculations for three groups of runners running at ~10 km/h for four hours while ingesting solutions with different [Na⁺]

<table>
<thead>
<tr>
<th>Pre-race</th>
<th>Pre-race ECF volume (litres)</th>
<th>Pre-race Na⁺ content (mmol)</th>
<th>Post-race</th>
<th>Post-race ECF volume (litres)</th>
<th>Post-race Na⁺ content (mmol)</th>
<th>Post-race Na⁺ balance (mmol)</th>
<th>Amount of Na⁺ ingested (mmol)</th>
<th>Apparent amount (mmol)/rate of Na⁺ loss during exercise (mmol/h)</th>
</tr>
</thead>
<tbody>
<tr>
<td>H</td>
<td>137.3</td>
<td>14.50*</td>
<td>1991</td>
<td>134.8</td>
<td>15.02</td>
<td>2025</td>
<td>-34</td>
<td>71</td>
</tr>
<tr>
<td>L</td>
<td>137.2</td>
<td>14.50</td>
<td>1989</td>
<td>132.8</td>
<td>14.95</td>
<td>1985</td>
<td>-4</td>
<td>7</td>
</tr>
<tr>
<td>W</td>
<td>137.5</td>
<td>14.50</td>
<td>1993</td>
<td>131.8</td>
<td>14.95</td>
<td>1970</td>
<td>-23</td>
<td>0</td>
</tr>
</tbody>
</table>

H, High sodium intake; L, low sodium intake; W, water during exercise.

*Based on 25% of mean body weight of 57.7 kg for the total group of runners. Weights for different groups were not reported.

†From table 2 of Twerrenbold et al.: to convert mg sodium (table 2) into mmol sodium, divide by the molecular weight of sodium (22.99).

BOOK REVIEWS

**Tennis**

**Rating**

- **Presentation**: 16/20
- **Comprehensiveness**: 15/20
- **Readability**: 15/20
- **Relevance**: 16/20
- **Evidence basis**: 13/20
- **Total**: 75/100

**T Wood**

**Dying to win**


Dying to win gives an accurate account of the problem of doping in sport and the difficulties and complexities in finding solutions to the problems. It makes interesting and provocative reading for anyone interested in sport, from the athlete and coach to the sport administrator, the medical profession, and governments.
The UK Radiological Congress (UKRC) provides a forum in which to bring together clinicians, scientists, radiographers, technicians, and other professionals to present and discuss the latest developments and challenges in diagnostic imaging, radiology, and allied radiological sciences.

The UKRC provides a forum in which to bring together clinicians, scientists, radiographers, technicians, and other professionals to present and discuss the latest developments and challenges in diagnostic imaging, radiology, and allied radiological sciences.

Key subjects to be covered include: diagnostic radiology; ultrasound; nuclear medicine; interventional radiology; veterinary radiology; emerging technologies; image analysis; computer applications; PACS; radiobiology; radiological physics; management & audit; computed tomography; magnetic resonance; equipment development. Expected attendance (conference and exhibition): 4000

Further details: UKRC 2005 Organisers, PO Box 2895, London W1A 5RS, UK; Website: www.ukrc.org.uk; Fax: +44 (0)20 7307 1410; Email: conference@ukrc.org.uk; Exhibition tel: +44 (0)20 7307 1410, Email: exhibition@ukrc.org.uk

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BASEM Conference 2006
5 – 7 October 2006, Oxford, UK
Further details: Email: basemoffice@compuserve.com

CORRECTIONS
doi: 10.1136/bjsm.2004.009211corr1

doi: 10.1136/bjsm.2004.009244corr1
Dadebo B, White J, George K P. A survey of flexibility training protocols and hamstring strains in professional football clubs in England (Br J Sports Med 2004;38:388–94). The multiple regression equation within the Abstract section of this paper was published incorrectly. The correct equation is:

\[ \text{HSR} = 37.79 - (0.33 \text{SHT} + 10.05 \text{SSP} + 2.24 \text{STE}) \]

We apologise for this error.

doi: 10.1136/bjsm.2004.010876corr1

The volume number for reference 23 (Sran et al) was incorrectly published as 24; the correct volume number is 29.

In Table 2 the results for Giles and Muller should read: Greater short term benefit for back pain with manipulation, but not for neck pain. Acupuncture more effective for neck pain.

In the section “Definitions and search strategy” the first line of paragraph 2 should read: I searched Medline, Cinahl, and Embase databases for randomised clinical trials comparing manual therapy, including spinal joint mobilisation (with or without manipulation) or manipulation only with other conservative treatments for back or neck pain.

We apologise for these errors.