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 Lucia

**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 SnaBl and BclI were used to detect the mutations 845G→A (C282Y) and 187C→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.

**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 (VO2MAX) 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...
(determined 12 and 6 months before the study) were recorded.

Serum iron concentrations were measured using a standard colorimetric method (Roche/Hitachi 714; Roche Diagnostics Corporation, Indianapolis, Indiana, USA). Transferrin concentration was determined by rate immunoturbidimetry (OSAX anti-serum for the Behring nephelometer; Dade Behring Marburg GmbH, Marburg, Germany). Serum transferrin saturation was calculated from these data as follows:

\[ \text{Transferrin saturation} \% = \frac{\text{serum iron concentration} \, (\text{mol/l})}{(2 \times \text{transferrin concentration} \, (\text{mol/l}))} \times 100 \]

Serum ferritin concentrations were measured by chemiluminescence immunoassay (N-latex ferritin kit; Dade Behring Marburg GmbH). The coefficients of interassay and intraassay 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 µmol/l for iron, 24–336 g/l for transferrin, 24–45% for transferrin saturation, and 20–300 µg/l for ferritin.19

**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. DNA amplification was performed using polymerase chain reaction (PCR) with specific primers for the two HFE gene mutations as described previously.20 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).

**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.

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 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 was significantly higher (p = 0.01) than in controls (41.5% vs 24.6%). Our results are in agreement with those of previous research showing that in the general population of Spain the prevalence of the H63D mutation ranges from 16% to 30.4%,21 and C282Y mutations occur in 2% to 4.4% depending in both cases on the geographical region.22

Thus, the prevalence observed here (especially for H63D) is much higher than that previously reported in non-athletic subjects, yet is similar to rates recently observed by Deugnier et al.,23 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.

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

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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.
The prevalence of mutations in the HFE gene (responsible for hereditary haemochromatosis, a disease in which the body’s iron stores are increased) seems to be high among elite endurance athletes (about 49%). As most elite endurance athletes take iron supplements, regular assessment of their iron reserves is recommended to prevent iatrogenic iron overload.

In conclusion, the prevalence of HFE gene mutations is high among elite endurance athletes (runners and professional cyclists). Regular determination of their iron stores is thus recommended.

ACKNOWLEDGEMENTS
We thank Ana Burton for translation of the manuscript.

References


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ELECTRONIC PAGES

BJSM Online case reports: http://bjsm.bmj.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:e10) http://bjsm.bmj.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.bmj.com/cgi/content/full/38/4/e11
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Sodium ingestion and the prevention of hyponatraemia during exercise

The study of Twerenbold et al 11 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, 7 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 8 and the Gatorade Sports Science Institute 9 and the guidelines of the American College of Sports Medicine 8 and the Gatorade Sports Science Institute 9 and the Guidelines of the American College of Sports Medicine 8 and the Gatorade Sports Science Institute 9 .

Fortunately the data of Twerenbold et al 11 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 who drank 14.95 litres during the race (W) ingested 58 kg of fluid containing 34 mmol/l NaCl (table 1). It is probable that, of this ingested fluid, 34 mmol/l NaCl is less than one third of that in the group who ingested the most NaCl (H) during the race.

For example, if each group did indeed lose 84 mmol NaCl as did group H (table 1), a value that seems eminently reasonable as it equates to a quite reasonable sweat [NaCl] 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. This value (equivalent to the ECF volume) is calculated as: (pre-race ECF Na content − 84) in mmol divided by post-race serum [NaCl] in mmol/l.

Indeed, if subjects in the W group did lose 84 mmol NaCl during the race but also had a post-race ECF volume expanded to 14.95 litres, then their post-race [NaCl] would have been even lower (128 mmol/l) than that actually measured after the race (132 mmol/l; table 1). It is therefore plausible that the sweat content of the ingested fluid ([NaCl]) in the three groups of runners was ~137 mmol/l—this is probably close to a defendable value base are those that have been recently accepted by the United States Track and Field and the International Marathon Medical Interest. 10 This value (equivalent to the ECF volume) is calculated as: (pre-race ECF Na content − 84) in mmol divided by post-race serum [NaCl] in mmol/l. For example, if each group did indeed lose 84 mmol NaCl as did group H (table 1), a value that seems eminently reasonable as it equates to a quite reasonable sweat [NaCl] 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. This value (equivalent to the ECF volume) is calculated as: (pre-race ECF Na content − 84) in mmol divided by post-race serum [NaCl] in mmol/l.

Unfortunately, the vital importance of these small changes in ECF volume in determining whether hyponatraemic encephalopathy will develop is well impressed by those who overdrink during exercise: 11–15 (table 3 of their article), the average total ECF Na content of the three experimental groups was 1189–1993 mmol at the start of the race. As weights increased by 1.8–2.1 kg in the three groups during exercise (table 3 of their article), the increases in ECF volume would have been 450–525 ml in the respective groups, assuming that the ECF increased in proportion to the increase in total body water (TBW). Multiplying this new ECF volume by the serum [NaCl] after the race gives the new total ECF Na content after the race. As shown in table 1, the total ECF Na content increased by 34 mmol/l in the group that ingested the highest salt drink (H) during the race, but fell by 23 mmol/l in the group drinking water (W). As all groups ran for about four hours, according to these calculations and based on these assumptions, the hourly rates of NaCl loss would have varied from 6 to 21 mmol/l, giving a sweat [NaCl] of 12–42 mmol/l in the W and H groups respectively (as their total sweat losses were ~2 litres in each group).

The clear paradox identified by the calculations in table 1 is that (a) the total NaCl loss apparently increases with increased NaCl intake and (b) the estimated NaCl loss in the group who ingested only water during the race (W) is less than one third of that in the group who ingested the most NaCl (H) during the race.

As these calculations are based on two real measurements (body weight changes and changes in plasma [NaCl]), this paper apparently ludicrous conclusion can only be explained if (a) NaCl ingestion during exercise increases whole body NaCl losses in sweat and urine or (b) the estimated ECF volume in the W group after exercise is less than the value calculated. That is, specifically in the W group, the ECF volume contracted despite an increase in TBW of 1.9 litres. Indeed, this response is to be expected. There is consistent evidence that the response of the ECF and the intracellulare fluid (ICF) volumes to fluid ingestion during prolonged exercise are influenced by the NaCl content of the ingested fluid 22,23 so that the ICF volume is likely to be expanded if the ingested fluid is ingested, 22 to fall less if either water 22 or a dilute NaCl drink is ingested, 22,23 or to expand if a concentrated (50–100 mmol/l) NaCl drink is ingested at the same rate that body weight is lost during exercise. 22 In the latter case, any reduction in the TBW appears to come from a reduction in the ICF. 22
encephalopathy will develop in those who overdrink, regardless of whether or not they also incur a Na⁺ deficit either during exercise \(^1\text{11,15,17}\) or at rest. \(^1\text{17}\) A recent paper\(^\dagger\) 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’’\(^\dagger\) 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.\(^\dagger\)\(^\dagger\) 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.\(^\dagger\)

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 Twerenbold et al\(\dagger\) (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 reducing their ECF volume by 1 litre, those athletes could have maintained their pre-race serum [Na⁺] while running for seven hours and drinking just sufficient water to allow for a 1 litre reduction in ECF volume and without requiring any Na⁺ replacement whatsoever. This simple calculation explains why those endurance athletes who, before about 1969, were advised either not to drink at all, or only sparingly during exercise,\(^\dagger\) always finished these races with raised serum [Na⁺] despite having incurred what might have been quite sizeable Na⁺ deficits.\(^\dagger\)

In contrast, athletes in this study who believed the incorrect advice that ingesting Na⁺ at high rates is essential to maintain a normal serum [Na⁺] during exercise,\(^\dagger\)\(^\dagger\)\(^\dagger\)\(^\dagger\)\(^\dagger\) so they overdrank sufficiently to increase their ECF volume by 1 litre, would need to ingest and retain at least an additional 140 mmol Na⁺ in addition to the ~80 mmol lost in sweat (table 1). This is equivalent to the Na⁺ content of 1.2 litres of the low and 7.5 litres of the high sodium drinks respectively in this trial. To maintain fluid balance in this four hour trial when drinking at those high rates and sweating at about 500 ml/h, they would then need to urinate at rates of 1375–2600 ml/h. Both of these rates exceed the maximum at which human kidneys are able to produce urine at rest,\(^\dagger\) let alone during and after prolonged exercise.\(^\dagger\) Drinking at such rates would therefore only lead to progressive fluid accumulation and ultimately death from hyponatraemic encephalopathy.\(^\dagger\)

In summary, these calculations explain (a) why contraction of the ECF in athletes who lose body weight during exercise will maintain the serum [Na⁺] even in the face of quite large and unreplace 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 hyponatraemia, as elegantly shown by this study.\(^\dagger\)

Indeed if this inappropriate behaviour is approached with sufficient vigour, ultimately the result will be death from hyponatraemic encephalopathy,\(^\dagger\) which, as these calculations and this study again show, cannot occur without the presence of distinct fluid overload.\(^\dagger\)

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,\(^\dagger\) 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 sublethal 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.

T Noakes

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Conflict of interest: none declared

References


<p>| Table 1 Sodium balance calculations for three groups of runners running at ~10 km/h for four hours while ingesting solutions with different [Na⁺] |
|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|
| Pre-race ECF volume (litres) | Pre-race Na⁺ content (mmol/l) | Post-race ECF Na⁺ content (mmol/l) | Post-race ECF Na⁺ balance (mmol) | Amount Na⁺ ingested | Apparent amount Na⁺ (mmol/l) |</p>
<table>
<thead>
<tr>
<th>A</th>
<th>B</th>
<th>C</th>
<th>D</th>
<th>E</th>
<th>(C − A)</th>
<th>(D − B)</th>
<th>(E − C)</th>
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<tr>
<td>H</td>
<td>137.3</td>
<td>4.50</td>
<td>1991</td>
<td>134.8</td>
<td>15.02</td>
<td>2025</td>
<td>-34</td>
</tr>
<tr>
<td>L</td>
<td>137.2</td>
<td>4.50</td>
<td>1989</td>
<td>132.8</td>
<td>14.95</td>
<td>1985</td>
<td>-4</td>
</tr>
<tr>
<td>W</td>
<td>137.5</td>
<td>4.50</td>
<td>1993</td>
<td>131.8</td>
<td>14.95</td>
<td>1970</td>
<td>-23</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 Twerenbold et al\(\dagger\) to convert mg sodium (table 2) into mmol sodium, divide by the molecular weight of sodium (22.99).

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Tennis


It is widely recognised that each sport has its own unique demands and injuries. Therefore the IOC, ITF, ATP, WTA, and Society for Tennis Medicine and Science should be congratulated on producing, in this publication, a comprehensive overview of tennis sports medicine. Together they have assembled an impressive array of experts in this field to write succinct and relevant chapters.

Every aspect of tennis is covered to cater for a broad range of readers, including players themselves. Some areas are covered in a high level of technical detail to please the biomechanists, in particular. However, some of the sports medicine is basic in concept and lacking significant evidence based validity.

Nevertheless, I would highly recommend this book to any health professional who treats a large number of tennis players. Most chapters provide a link between common sports medicine problems and their occurrence in tennis, including conditions that are unique to this sport. At times, some authors are somewhat optimistic with their view of recovery time from surgery—for example, three weeks for arthroscopic debri- dement of the infrapatellar fat pad.

Overall it is well presented with relevant and useful photographs and diagrams to aid the reader, and each chapter gives a list of further recommended reading. Unfortunately the book does not provide an answer to where 14 million tennis balls go, imported each year into Australia, as discussed by the editor recently!

BOOK REVIEWS

Tennis

Reviewed by T Wood

Dying to win


Dying to win gives an eye opening account of the extent to which drugs play a major role in sport. Doping is not new and has been used in sport since ancient Olympic times; it is just that drug use in modern times is at such a level of sophistication, it is now an industry in its own right. The book describes the privileged position sport holds in society, having appeal for both the participant and the spectator. This has led to the massive media interest, commercialism, professional- ism, and governmental regulation and manipulation. Economic pressure in the industrialised world and governmental propaganda in the former East Germany, and more recently China, paved the way for increasing pharmaceutical intervention in sport.

With the fall of the GDR, the world saw for the first time what it had long suspected, the extent of systematic doping on a State run basis, and the most interesting fact is that the East Germans kept their records! Further, the book takes a look at the next big issue surrounding drugs in sport—genetic engineering.

Dying to win does not just describe the evolution of doping. It explains the complex relation between anti-doping policy, implementa- tion of those policies, and the role of governments, the IOC, and international and national sporting organisations. With the ever increasing involvement of the legal profession, a vicious circle occurs: it becomes too costly for sporting organisations to fight court battles, with their reliance on Government funding depending on results and punishments set in accordance with what will stand up in courts. This all leads to the relative inertia of the governing bodies to be pro-active in the anti-doping campaign.

The inception of the World Anti-Doping Agency (WADA) after the 1998 Winter Olympics was a way forward to standardise and implement anti-doping policy across the world by an independent body.

Problems and solutions to anti-doping policy are addressed. The major problem is inadequate definition of doping—to quote Arthur Gold “The definition lies not in words but in integrity of character.” It is interesting to note that those behind the athlete, namely coach, administrators, medical profession, and scientists, all seem to lose perspective in the concept and lacking significant evidence based validity.

Every aspect of tennis is covered to cater for a broad range of readers, including players themselves. Some areas are covered in a high level of technical detail to please the biomechanists, in particular. However, some of the sports medicine is basic in concept and lacking significant evidence based validity.

Nevertheless, I would highly recommend this book to any health professional who treats a large number of tennis players. Most chapters provide a link between common sports medicine problems and their occurrence in tennis, including conditions that are unique to this sport. At times, some authors are somewhat optimistic with their view of recovery time from surgery—for example, three weeks for arthroscopic debri- dement of the infrapatellar fat pad.

Overall it is well presented with relevant and useful photographs and diagrams to aid the reader, and each chapter gives a list of further recommended reading. Unfortunately the book does not provide an answer to where 14 million tennis balls go, imported each year into Australia, as discussed by the editor recently!
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CORRECTIONS

doi: 10.1136/bjsm.2004.039211corr1

doi: 10.1136/bjsm.2004.000044corr1
Sran M M. To treat or not to treat: new evidence for the effectiveness of manual therapy (Br J Sports Med 2004;38:521–5). 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.