Body iron excretion by healthy men and women

Janet R Hunt, Carol Ann Zito, and LuAnn K Johnson

ABSTRACT

Background: Iron excretion measured by isotope dilution has been a primary basis for the factorial derivation of recommendations for iron intake, but the results have been available for men only.

Objective: The objective of this study was to confirm iron excretion measurements in healthy men and extend them to women.

Design: The turnover rate of $^{55}$Fe that had been administered $\geq$ 1 y earlier was determined from blood sampled semiannually for up to 3 y from 53 subjects in the community. Body iron was determined from hemoglobin, serum ferritin, and transferrin receptor. Complete menstrual collections were obtained from 13 women.

Results: The total median (range) iron excretion was 1.18 (0.11–2.07) mg/d for 29 men, 1.58 (0.65–4.88) mg/d for 19 menstruating women, and 0.99 (0.86–1.57) mg/d for 5 postmenopausal women. When hormonal contraceptive users were omitted, the median for 15 menstruating women increased to 1.66 mg/d. The distribution of iron excretion was normal for the men and postmenopausal women and was strongly skewed for the menstruating women; menstrual iron accounted for 90% of the variation. Iron excretion was not strongly related to body weight. Body iron in menstruating women decreased somewhat (by 4.6%) in the men and tended to increase (by 1.5%) during the study.

Conclusions: The results extend direct iron excretion measurements in men to include similar measurements in women. The results emphasize the wide range of iron excretion in humans, which results in a 40-fold range of requirements for absorbed iron. This trial was registered at clinicaltrials.gov as NCT00755105.


INTRODUCTION

The body's iron content of $\approx$ 1.5–6 g is mainly controlled at the site of intestinal absorption, with an absorptive efficiency that can be up-regulated $\geq$ 15-fold in inverse proportion to body iron stores (1). Excluding iron losses from menstruation, other bleeding, or pregnancy, body iron is highly conserved. Basal iron excretion, principally from cellular exfoliation, is limited, and is the primary factor determining men's nutritional requirements for absorbed iron. Using isotope dilution methods, Green et al (2) measured basal iron losses of 0.95 ± 0.30 mg/d for white men in the United States, 0.90 ± 0.31 mg/d for Mestizo men in Venezuela, and 1.02 ± 0.22 mg/d for Indian men in South Africa. Higher and more variable losses of 2.42 ± 1.09 and 2.01 ± 0.94 mg/d for Bantu men in Johannesburg and Durban, South Africa, respectively, were attributed to greater than normal body iron stores in the Bantu population. Since publication in 1968, the iron excretion measurements made by Green et al in the non-Bantu men have been used as the principal basis for factorial estimations of dietary iron requirements and recommendations for US men. They are further used to estimate basal iron losses of women and children, for whom direct measurements of basal iron excretion have been unavailable (3).

The objective of the present research was to strengthen the basis for such factorial-derived recommendations by directly measuring total iron excretion in women as well as in men. Using these measurements we evaluated the relation between iron excretion and body iron stores (as measured by current methods using serum ferritin and transferrin receptor; 4). Within the constraints of a relatively small sample size, we also evaluated assumptions used in setting the Dietary Reference Intakes for iron that 1) iron excretion correlates with and has a variation similar as body weight, and 2) iron excretion follows a normal distribution, with the exception of menstrual iron losses, which have a highly skewed population distribution (5, 6). Finally, new iron excretion measurements in men allowed an evaluation of a possible over-estimation of basal iron excretion by Green et al (2) when they eliminated subjects whose multyear decline in blood $^{55}$Fe concentrations was not statistically significant ($P < 0.05$).

SUBJECTS AND METHODS

Subjects

The study was approved by the University of North Dakota's Institutional Review Board. The study was explained verbally and in writing by the investigators, and written informed consent was given by each subject.

The subjects were recruited from among men and women who had participated for $\geq$ 1 y earlier in studies of healthy subjects who were administered $^{55}$Fe. All subjects meeting this criterion were enrolled in a 3-y study that involved semiannual blood
sampling and questionnaire completion if they agreed not to take dietary supplements containing iron (unless medically prescribed), not to donate blood during the study, and not to provide blood samples for optional medical screening. If blood was removed for medical reasons, the subject worked with their healthcare professional to report the volume.

At the beginning and end of the study, the subjects completed questionnaires on general health and factors that might affect the care professional to report the volume.

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Women were also asked about pregnancy history, menstrual patterns, chemical forms of birth control, and hormone replacement therapy. Every 6 mo during the study, the subjects completed a questionnaire to confirm that they were not taking nonprescribed iron supplements, having optional blood removed for donation or screening, and to update information about health, iron supplement use, or blood losses due to medical conditions or care, pregnancy, use of chemical forms of birth control or hormone replacements, and dates of menstruation.

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Complete data were obtained from 53 subjects who provided semiannual blood samples for ≥1 y, did not use iron supplements, did not become pregnant, did not experience menopause, did not undergo surgery, or did not donate blood. The final data set included 19 menstruating women (4 of whom used a hormonal contraceptive), 5 postmenopausal women, and 29 men, as further described in the top portion of Table 1.

Iron excretion measurements

The $^{55}$Fe radioisotope emits weak X-rays with a range of 1–2 mm in tissues and decays with a physical half-life of 2.7 y. This isotope had been administered to all subjects in prior research. Eight years before the beginning of the present 3-y study, 11 subjects had received a single intravenous dose of 5 μCi $^{55}$Fe mixed with each subject's own plasma (7). One to two years before the present study, the remaining 42 subjects had received 2 oral doses separated by several weeks, with a total dose of 1–2 μCi $^{55}$Fe as hemoglobin iron (8–13).

The biological half-life of $^{55}$Fe was determined for each subject from blood samples collected semiannually for 1–3 y. To allow for tracer equilibration in the body, blood sampling began no sooner than 1 y after $^{55}$Fe administration. The average sampling duration for the subgroups was 32–36 mo (Table 1). Each subject’s frozen blood samples were held for analysis of $^{55}$Fe in a single analytic batch. Sample weights were monitored to rule-out any freezer evaporation of sample. A single batch analysis reduced analytic variability and eliminated the need to correct for isotopic decay. Sufficient time had lapsed to enable the physical decay of another isotope, $^{55}$Fe (44.5 d half-life), from the previous studies to levels that were no longer detectable. For each blood sample, duplicate 10-μL aliquots were prepared by acid digestion and by conversion of the iron to an insoluble white ferriphosphate complex (14, 15). This was dissolved in 1 mL 6 N HCl and mixed with 19 mL of a scintillation fluid recommended for strong mineral acids (Ultima Gold AB; Perkin-Elmer, Shelton, CT). Dark-acclimated samples were counted for 1 h/sample by using a matrix-matched quench correction curve, a high sensitivity mode, and a 1–5-KeV window on the Packard Tri Carb 1600 scintillation counter (Meriden, CT). All sample counts were >3 times background.

Body iron excretion was calculated as described by Green et al (2), with modification to estimate body iron stores using serum transferrin receptor and ferritin concentrations. For each individual, the decrease in blood activity of $^{55}$Fe, reflecting biological

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Iron excretion by men and women</th>
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<tbody>
<tr>
<td></td>
<td>Men (n = 29)</td>
</tr>
<tr>
<td></td>
<td>(n = 19)</td>
</tr>
<tr>
<td>Initial age (y)</td>
<td>43 ± 8 (30-58)</td>
</tr>
<tr>
<td>Body weight (kg)</td>
<td>92 ± 15 (62-131)</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>28.8 ± 3.7 (21.8-35.3)</td>
</tr>
<tr>
<td>Hemoglobin (g/L)</td>
<td>15.5 ± 0.6 (14.0-16.5)</td>
</tr>
<tr>
<td>Serum ferritin (μg/L)</td>
<td>142 [82, 248] (51-357)</td>
</tr>
<tr>
<td>Sampling duration (mo)</td>
<td>33 ± 5 (23-37)</td>
</tr>
<tr>
<td>Biological half-life of iron (y)</td>
<td>7.8 [4.8, 12.7] (4.3-31.6)</td>
</tr>
<tr>
<td>Turnover rate (%/y)</td>
<td>9.7 ± 3.6 (2.2-16.1)</td>
</tr>
<tr>
<td>Total body iron (g)</td>
<td>4.4 ± 0.7 (3.0-6.5)</td>
</tr>
<tr>
<td>Change in total body iron (g/study)²</td>
<td>0.07 ± 0.19 (0.23-0.62)</td>
</tr>
<tr>
<td>Change in total body iron (%/study)²</td>
<td>1.5 ± 4.4 (4.4-15.7)</td>
</tr>
<tr>
<td>Change in total body iron (%/yp²</td>
<td>0.6 ± 1.8 (1.9-6.3)</td>
</tr>
<tr>
<td>Iron excretion (mg/d)</td>
<td>1.07 ± 0.47 (0.11-2.07)</td>
</tr>
<tr>
<td>Iron excretion (μg · kg⁻¹ · d⁻¹)</td>
<td>12 ± 5 (1-21)</td>
</tr>
</tbody>
</table>

¹ All values are arithmetic means ± SDs for normally distributed data or geometric means (with SD and 95% confidence intervals) for skewed distributions; ranges in parentheses. HC, hormonal contraceptive.

² The men experienced a nonsignificant tendency for total body iron to increase (0.05 < P < 0.10).

³ Significantly different from zero, P < 0.05 (Student’s t test).
retention, follows an exponential curve that is linear when expressed as a semilogarithmic retention plot (the natural logarithm of \(^{55}\text{Fe}\) activity versus time; see examples in Figure 1). The biological half-life \((t_{1/2})\) of body iron (in mg) was calculated from the slope of each of these retention plots, as follows:

\[
t_{1/2} = -\ln(2)/\text{slope} \quad (1)
\]

This relation was used to determine the turnover rate of the tracer, which was assumed to be fully equilibrated in the body 1 y after isotope administration. Expressed as the percentage of the labeled body iron pool lost per year (since the beginning of the present protocol), the turnover rate was calculated as:

\[
\text{Turnover rate} = \ln(2) \times 100/t_{1/2} \quad (2)
\]

Body iron was determined as the sum of circulating hemoglobin iron plus body iron stores, based on measurements from samples collected on 2 separate days at the beginning and again at the end of each subject’s participation. Blood iron concentration was calculated from the blood hemoglobin concentration by using the conversion factor 3.39 mg Fe/g hemoglobin, as used by the Institute of Medicine to derive dietary iron recommendations (16). Total blood volume was estimated from sex-specific formulas based on height and weight (17, 18). Body iron stores were calculated from serum concentrations of transferrin receptor and ferritin, as described by Cook et al (4).

Body iron loss was calculated from body iron and the turnover rate (Equation 2) by using the following formula:

\[
\text{Body iron loss (mg/d)} = \text{body iron (mg)} \times \text{[turnover rate(\%)/100\%]} / 365 \text{~d} \quad (3)
\]

Physiologic iron excretion was calculated by subtracting from this body iron loss the iron removed as part of the present investigation or medical care. This was determined from the volume of blood removed by phlebotomy, which averaged 0.2 (range: 0.1–0.3) mL/d by using the most recent hemoglobin concentration and the conversion factor 3.39 mg Fe/g hemoglobin (16).

**Menstrual iron excretion**

Thirteen of the menstruating women provided complete menses collections for 1 to 3 menstrual cycles. Commercial sanitary products, selected for low iron content (by analysis), were provided. Menstrual samples were extracted from sanitary products with 0.12 N HCl, followed by digestion with nitric and perchloric acids, and iron was analyzed by inductively coupled plasma optical emission spectroscopy.

**Other clinical chemistry**

Hemoglobin and hematocrit were measured by using a Celldyn 3500 (Abbott Laboratories, Abbott Park, IL). Serum iron was determined colorimetrically with a commercial chromagen (Ferene; Raichem Division of Hemagen Diagnostics, San Diego, CA). Serum ferritin was measured by immunometric assay (Abbott Laboratories, Abbott Park, IL or Immunite Ferritin, Diagnostic Products Corp, Los Angeles, CA), as was serum transferrin receptor (R & D Systems Inc, Minneapolis, MN). Because commercial serum transferrin receptor measurements have not been standardized, the strong correlation \((R^2 = 0.86)\) (19) between the R & D Systems method and the Ramco (Houston, TX) method was applied to convert to the Ramco transferrin receptor units used by Cook et al (4) for calculating body iron.

**Statistics**

The Shapiro-Wilk statistic was used to test whether the data were normally distributed. Descriptions of the data used geometric means and SDs when data were skewed. Data were evaluated by regression analysis with SAS software (version 9.1.3; SAS Institute Inc, Cary, NC). Student’s t test was used to test whether the change in total body iron was significantly different from 0. Using 2-tailed probabilities, \(P \leq 0.05\) was considered significant.

**RESULTS**

Blood isotope activity declined with time for all subjects, and this negative relation was statistically significant for 49 of the 53 subjects’ exponential retention plots. The other 4 subjects were all men, with negative retention slopes that were among the least steep but which had a goodness-of-fit (based on the root mean square error of the regression) similar to the others (see Figure 1 for examples). Therefore, these men were deemed to have some of the lowest relative turnover rates of body iron, and their data were retained so as not to overestimate mean body iron excretion by eliminating the men with the lowest excretion rates.

Iron excretion was based on the average total body iron of each subject, disregarding any slight differences between the initial and final measurements of total body iron (considered further below). Mean (± SD) iron excretion by the 29 men was 1.07 ± 0.47 (range: 0.11–2.07 mg/d) (Table 1); the data are described as an arithmetic mean ± SD because they followed a normal distribution, as indicated by the Shapiro-Wilk test \((W = 0.97, P < 0.67)\) (Figure 2). In contrast, iron excretion by the menstruating women was highly skewed, with
values. Each point represents one subject; the horizontal lines indicate median contraceptive (HC) users (n = 19), menstruating women with omission of hormonal excretion. Because the differences in total body iron were small and may reflect differences in absorption rather than differences in menstruating women still decreased by 2.3 ± 4.8% for these 2 women were eliminated, the total body iron of the was not otherwise explained by the data collected. When the data for these 2 women were eliminated, the total body iron of the menstruating women still decreased by 2.3 ± 4.8% (P = 0.06) during the study, or 1.0 ± 1.8%/y (P = 0.05). Total body iron in the men tended to increase by 1.5 ± 4.4% during the study (P = 0.07), or 0.6 ± 1.8%/y (P < 0.06) and did not relate to the amount of iron excreted. Because the differences in total body iron were small and may reflect differences in absorption rather than differences in excretion, they were not factored into the calculations of body iron excretion, which otherwise would not have changed by >0.04 mg/d. We conclude that, whereas the menstruating women’s total body iron decreased significantly and the men’s body iron tended to increase with time, these slight changes did not contribute substantial error to the calculations of physiologic iron excretion.

Iron excretion per menstrual period varied considerably between the subjects (SD: 14 mg), but varied less within subjects (SD: 9 mg). Menstrual iron ranged from 0.5 to 56 mg iron/menstrual period, or, adjusted for the reported number of menstrual periods per year, 0.015–1.86 mg Fe/d (n = 13). The menstrual iron losses determined by elemental analysis accounted for 90% of the variation in total iron excretion that was independently determined by isotope dilution (Figure 3). Menstrual iron losses fully explained the skewed distribution of iron excretion for this subset of 13 menstruating women. Calculated on a daily basis, these women excreted (geometric means, ± 1 SD) 0.28 (0.08, 1.05) mg/d menstruated iron and 1.53 (0.94, 2.50) mg/d total iron (n = 13).

To investigate whether body iron stores influenced body iron excretion, we examined the relation between serum ferritin and body iron excretion. Because these 2 variables were not independently derived (both use serum ferritin measurements), we also examined the relation between total body iron and turnover rate. For the men, neither of these relations approached statistical significance (see serum ferritin comparison in Figure 4). In contrast, for the menstruating women, iron excretion was inversely related to serum ferritin ($R^2 = 0.41, P < 0.01, n = 19$) following a power curve that becomes linear after logarithmic transformation of both variables; Figure 4), and the turnover rate decreased linearly with total body iron ($R^2 = 0.40, P < 0.01, n = 19$). These relations support the conclusions that high iron excretion by menstruating women results in low iron stores, that nonmenstrual iron excretion is not related to serum ferritin concentrations between 50 and 350 µg/L, and that iron excretion is not regulated to maintain body iron homeostasis.

For the men, iron excretion was directly related to body weight ($R^2 = 0.16, P < 0.05, n = 29$), consistent with greater cellular exfoliation with a larger body size. A similar, but nonsignificant relation was observed for the 13 women for whom nonmenstrual iron losses could be calculated (by subtraction of menstrual losses) ($R^2 = 0.20, P = 0.12, n = 13$). When these 2 groups plus the postmenopausal women were combined, there was a weak but marginally significant direct relation between nonmenstrual iron excretion and body weight ($R^2 = 0.08, P = 0.06, n = 47$). However, the turnover rate was not related to body weight, and

![Figure 2](image1.png)

**FIGURE 2.** Distributions of iron excretion by men (n = 29), menstruating women (n = 19), menstruating women with omission of hormonal contraceptive (HC) users (n = 15), and postmenopausal women (n = 5). Each point represents one subject; the horizontal lines indicate median values.

![Figure 3](image2.png)

**FIGURE 3.** Menstrual iron excretion accounted for 90% ($R^2$) of the variation in total iron excretion for the subset of women who provided complete menstrual collections (n = 13) and accounted for the skewed distribution of iron excretion in these women. Each dot represents one woman; the line is the best fit from linear regression analysis.
expression of iron excretion per kilogram body weight did not improve the relation of iron excretion with serum ferritin for either sex (data not shown). Thus, the relation of iron excretion with body weight appears weak, if it occurs at all.

**DISCUSSION**

Iron excretion measurements, as reported herein, involve relatively sensitive determinations of iron turnover rates based on isotope dilution, combined with estimates of the size of the exchangeable body iron pool. Previously reported turnover rates are comparable but tend to be slightly greater than the present measurements for men and slightly less than the present measurements for menstruating women. For men (excluding the Bantu, who commonly have unusually high iron stores), Green et al (2) reported turnover rates of 10.8 ± 4.1%, 12.4 ± 4.8%, and 14.1 ± 2.5% and Heinrich (20) reported rates of 11.7 ± 12.7% and 11.7 ± 8.8% compared with 9.7 ± 3.6% per year in the present study (Table 1). If the present study had excluded the 4 men with nonreporter exchangeable iron pool estimates of 3.3 ± 0.3, 2.7 ± 0.4, and 2.7 ± 0.5 g for men (excluding the Bantu). These estimates are less than the estimate for men of 4.4 ± 0.7 g in the present study, which assumes 100% exchangeability of the body iron with the isotope after ≥1 y after isotope administration. Our larger estimate of the exchangeable iron pool is consistent with data available for a subset of 40 of the subjects in the present study for whom we also had measurements of erythrocyte incorporation of total absorbed isotope, based on blood and whole body measurements of $^{59}\text{Fe}$ 2 wk after oral isotope administration of both $^{56}\text{Fe}$ and $^{59}\text{Fe}$ (8-13) (see method described in reference 8). The average erythrocyte incorporation of iron isotope (n = 2 measurements per subject) was 64 ± 13% (35-88%) for 23 men, 79 ± 11% (57-100%) for 14 menstruating women, and 84 ± 15% (66-93%) for 3 postmenopausal women. These erythrocyte incorporation measurements provide a second check of our estimation of exchangeable body iron. For the subset of 23 men for whom erythrocyte incorporation measurements were available, exchangeable iron calculated as blood iron divided by fractional erythrocyte incorporation (4.3 ± 1.1 g, 3.0-6.5 g) was not significantly different (P = 0.33) from total body iron estimated with our present method (4.5 ± 0.8 g, 3.1-6.5 g), based on blood iron plus iron stores derived from ferritin and transferrin receptor concentrations. This supports the possibility that Green et al (2) may have underestimated the exchangeable iron pool size of the men in their study.

Mainly because of these differences in exchangeable body iron, the present iron excretion results for men (1.07 mg/d; range: 0.11-2.07 mg/d) are somewhat greater than those reported by Green et al (2): 0.95, 0.90, and 1.02 (range: 0.49-1.63) mg/d for 3 groups (excluding the Bantu). The range of iron excretion results in the present study would be further narrowed to 0.49-2.07 mg/d if we, like Green et al, had eliminated the non-significant long-term retention plots (n = 4) that, in our retention plots, had flat slopes but an acceptable goodness of linear fit. Overall, these comparisons generally validate the present data, which extend human iron excretion results to include women.

The women’s iron excretion results were separately validated by the independent measurements of menstrual iron excretion. The menstrual iron losses accounted for most of the variation in excretion and for the skewed distribution of iron excretion in these women.

Current US dietary recommendations for iron use the body weights and the daily iron losses of the subjects of Green et al (2) to derive an average estimated iron loss of $\sim 14 \mu\text{g} \cdot \text{kg}^{-1} \cdot \text{d}^{-1}$ (16). To estimate a Recommended Dietary Allowance (RDA) that meets or exceeds the requirements of 97.5% of the population, the distribution of iron losses is assumed to be similar to the distribution of body weight. The median iron losses used to derive the estimated average requirements (EAR) for men, menstruating women, and postmenopausal women are 1.08, 1.41, and 0.896 mg/d, respectively (16), compared with medians of 1.18, 1.58, and 0.99 mg/d in the present study. However, the distribution of iron losses in the present study is generally wider (in both directions) than the distribution based on body weight that was used to determine the RDAs for iron. As an example, 3 of 19 (16%) of the menstruating women in the present study exceeded the estimated iron loss of 3.15 mg/d at the 97.5th percentile, which is the basis for the RDA (16). The present data indicate only a weak relation between body weight and iron excretion and do not support the use of body weight data for estimating the population distribution of iron excretion.
The present data are consistent with the early observations of McCance and Widdowson (21, 22) that body iron excretion does not contribute to the regulation of iron homeostasis. This emphasizes the importance of iron absorption in controlling iron balance, adjusting for a range of ≥40-fold in an individual’s iron excretion: in the present study, male subjects needed to replace as little as 0.11 mg/d, and female subjects needed to replace as much as 4.88 mg/d. On the basis of turnover rates, these subjects needed to replace as little as 2% and as much as 95% of their body iron annually!

Despite this substantial range in iron excretion, homeostatic control mechanisms were effective at maintaining body iron homeostasis for most subjects, with substantially impaired iron-status indexes in only one menstruating woman (as delineated above). These considerable differences in iron excretion and resulting requirements can generally be appropriately met by physiologic control of iron absorption, provided that dietary iron is accessible and reasonably bioavailable. In previous investigations, after 10 wk of equilibration to controlled diets containing 15 mg Fe per 2300 kcal, total (heme plus nonheme) iron absorption varied inversely with serum ferritin from 0.46 to 5.26 mg/d (3–41% of dietary iron) for men and women consuming a high-bioavailability diet and from 0.05 to 0.68 mg/d (0.4–5% of dietary iron) for those consuming a low-bioavailability diet (8, 11; individual results previously reported as group summaries). These absorption results suggest that the women and most of the men in the present study would be able to absorb sufficient iron from a high-bioavailability diet to replace their excreted iron, but that some of the men in the present study may benefit from a somewhat lower iron bioavailability diet, so as not to absorb more iron than they excrete (Table 1). This is consistent with the finding that, after 10 wk, men significantly reduced their total iron absorption from the high-bioavailability diet from 0.96 to 0.69 mg/d (geometric means) (8). This reduction, together with the tendency in the present study to (nonsignificantly) increase their body iron in 3 y may mean that dietary recommendations for men in Western countries may best focus on preventing body iron accumulation rather than iron deficiency.

Current iron recommendations for women are intended to maintain sufficient body iron for functional needs, with minimal iron stores, as indicated by a serum ferritin concentration of 15 μg/L (16). Subjects with this minimal level of body iron are estimated to absorb 18% of the iron from a high-bioavailability diet (16), an efficiency confirmed in our previous study of women’s iron absorption from high- and low-bioavailability diets (11). However, in that same study (11), one-third (week 0) to one-half (week 10) of the women, generally with serum ferritin values <15 μg/L, absorbed >18% of the iron from the high-bioavailability diet (11; individual results previously reported as group summaries). In the present study, 4 of the 19 menstruating women had average serum ferritin concentrations <15 μg/L, but only one developed iron deficiency anemia. Together, these data suggest that the upper ends of the distributions of both iron excretion and iron absorption by menstruating women are greater than have been used in deriving current recommendations.

In conclusion, this study extends isotope dilution measurements of men’s body iron excretion to include women. The median results are generally consistent with, although slightly greater than, previous results, and body iron excretion measurements had broad ranges, with a normal distribution in men and a skewed distribution in women. The latter was determined by a skewed distribution of menstrual iron losses. The distribution of body iron excretion was not strongly related to body weight.

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