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REVIEW

# Iron deficiency and susceptibility to infections: evaluation of the clinical evidence

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**Abstract** Iron is a fundamental nutrient for human and microbial life. We sought to examine the association of iron deficiency versus normal iron status with the susceptibility to infections. A systematic search in the PubMed and Scopus databases was performed to identify relevant clinical studies. Six studies (including a total of 1,422 participants) met the inclusion criteria: four prospective cohort (859 participants), one retrospective case–control (115 participants), and one retrospective cohort study (448 participants). Intensive care unit (ICU)-acquired and postoperative infections were more common in patients with iron deficiency than among those with normal iron status in two studies, while no difference was reported in another study. In one study examining pregnant women with normal mean iron values, higher soluble transferrin receptor values independently predicted vaginosis-like microflora. Iron deficiency anemia was an independent predictor of respiratory tract infections in one study, and postoperative urinary tract infections were more common in patients with iron deficiency anemia in another. The limited available evidence suggests that individuals with iron deficiency and those with iron deficiency anemia may be more susceptible to infections than patients with normal iron status. Future studies should elucidate further these findings.

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## Introduction

Iron deficiency is the most common hematological disorder, especially among women of childbearing age [1]. Controversial hypotheses have been formulated arguing either for higher susceptibility of the iron-deficient individuals to infections, due to impaired cell-mediated or humoral immunity [2–6], or for protection of the iron-deficient individuals from infections, associated with restriction of the host iron for use by the pathogens [7]. Controversy also characterizes the issue of the safety of iron supplementation, with studies suggesting that patients who receive iron are more susceptible to infections than those who do not [8, 9], others supporting that iron supplementation has no effect on the infection incidence [10, 11], and others suggesting that iron administration may reduce the susceptibility to infections [12].

In this context, we aimed to systematically review the available evidence to examine whether iron deficiency, compared with normal iron status, is associated with susceptibility to infections.

## Methods

### Literature search

A search in the PubMed and Scopus databases was performed in March 2012. The following search term was applied: ("iron deficiency") AND (respiratory OR urinary OR bacteremia OR septic OR infection). Abstracts presented after 2000 at ICAAC (Interscience Conference on Antimicrobial Agents and Chemotherapy) and ECCMID (European Congress of Clinical Microbiology and Infectious Diseases) conferences were also searched. Articles published in a language other than English, French, German, Spanish, Italian, or Greek were not evaluated.

Study selection

We included only studies measuring markers of iron status or hemoglobin in patients before an infection occurred (as opposed to during or after an infection). Iron status markers considered in this regard included serum iron, ferritin, transferrin, red blood cell indices, serum transferrin receptors, red cell zinc protoporphyrin levels, and bone marrow iron stores. Studies comparing iron-deficient patients with patients with iron surplus were excluded. In addition, studies reporting on malaria were not considered to be eligible for our review, because it is well-known that iron deficiency protects children from malaria in endemic areas due to pathogen-specific characteristics [12, 13].

Data extraction

Data were extracted from each of the included studies regarding the study characteristics (design, time period, country) and the characteristics of the included subjects (number, patient population, groups that were compared). The data on susceptibility to infections among the compared groups of participants in the univariate or multivariate analyses were also extracted.

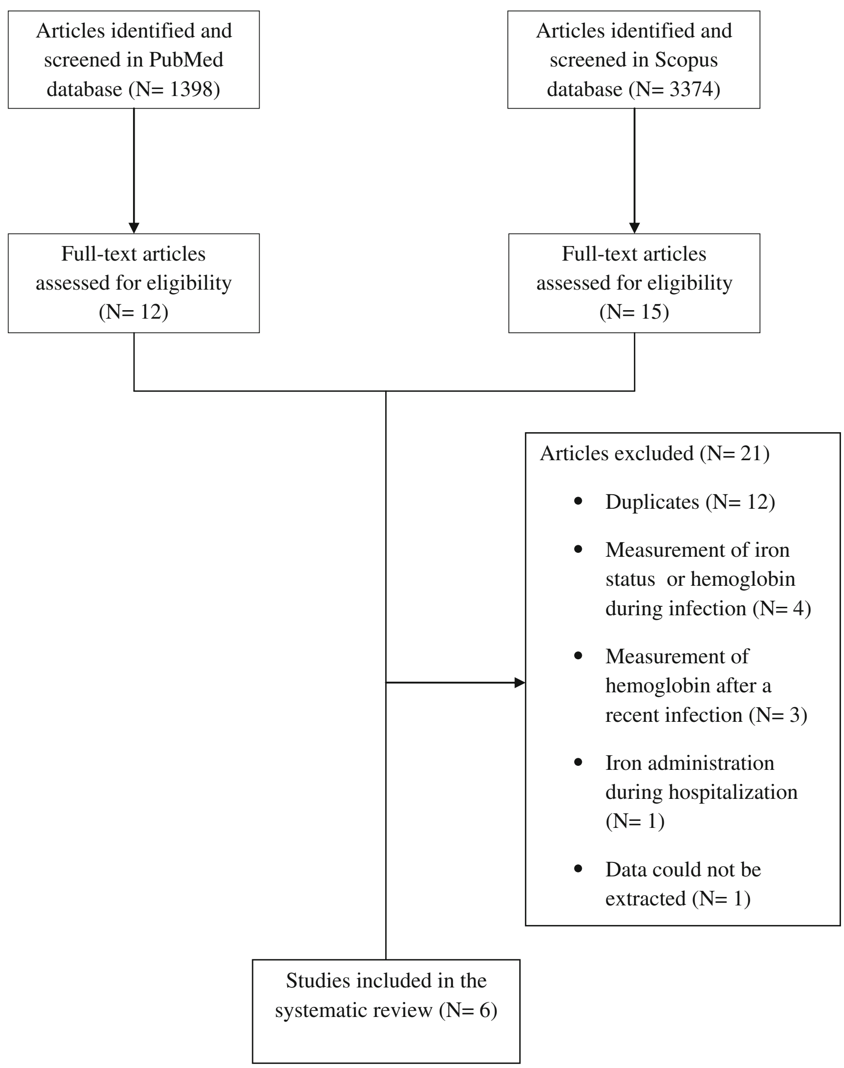
Definitions and outcomes

Iron deficiency or iron deficiency anemia were defined according to the definitions used by the investigators of each study.

## Results

A total of 4,772 articles (1,398 in PubMed, 3,374 in Scopus) was retrieved during our bibliographic search, of which six were finally considered to be eligible for inclusion in this review [14–19]. The study selection process is depicted in Fig. 1. More specifically, of the studies that were selected for further evaluation, seven were excluded because they

**Fig. 1** Flow diagram of the systematic search and study selection process



measured the markers of iron status or hemoglobin during [7, 20–22] or after [23–25] the infection. One study was also excluded because some of the included patients had infections at baseline [26] and another because patients received iron supplementation during their hospitalization [27].

The characteristics of the included studies are presented in Table 1. Four studies were prospective cohort studies [14, 16–18], one was a retrospective case–control study [19], and the remaining study was a retrospective cohort study [15]. Four studies (676 participants) [14, 15, 18, 19] and two studies (746 participants) [16, 17] of the included investigations reported on the impact of iron status and iron deficiency anemia, respectively, on the susceptibility to infections. Regarding the characteristics of the study population, two studies evaluated surgical patients [15, 17], two studies evaluated intensive care unit (ICU) patients [14, 18], another study evaluated pregnant women [19], and the final study evaluated healthy infants and toddlers [16].

## Iron deficiency and infection

In two studies, ICU-acquired infections and postoperative infections following abdominal surgery, respectively, were more common in patients with iron deficiency compared to those with normal iron status, 30.4 % vs. 10.3 % ($p$=0.04) [14] and 10.1 % vs. 2.3 % ($p$<0.001) [15], respectively. However, in another study, no difference was reported between ICU-hospitalized patients with iron deficiency and those with normal iron status regarding the incidence of infections [18].

Another study, with a case–control design, evaluated the iron status in pregnant women on their first antenatal visit [19]. Higher soluble transferrin receptor (sTfR) values were independently associated with the presence of a vaginosis-like microflora, a condition that is thought to precede the development of bacterial vaginosis [odds ratio=4.5, 95 % confidence interval (1.4–14.2)] [19].

**Table 1** Characteristics and outcomes of the included studies

| Author, year | Study design; period, country | Number of patients; characteristics | Compared groups | Susceptibility to infections (uni-/multivariate analysis) |
|---|---|---|---|---|
| Studies reporting on iron deficiency and infections | | | | |
| Fernandez et al., 2010 [14] | Prospective cohort; 2007, Spain | 62; ICU patients | Low[a] CHr vs. normal CHr | ICU-acquired infection: more common in patients with low CHr, 30.4 % vs. 10.3 %, $p$=0.04 (univariate analysis) |
| Harju, 1988 [15] | Retrospective cohort; NR, Finland | 448; abdominal surgery patients | Empty iron stores (low ferritin)[a] vs. normal iron stores (normal ferritin) | Postoperative infections: more common in patients with empty iron stores, 10.1 % vs. 2.3 %, $p$<0.001 (univariate analysis) |
| Patteril et al., 2001 [18] | Prospective cohort; NR, United Kingdom | 51; ICU patients | FID (hypochromasia)[a] vs. normal iron status | No difference in infections: 50 % vs. 45.5 %, $p$=0.85 (univariate analysis) |
| Verstraelen et al., 2005 [19] | Retrospective case–control; 2003, Belgium | 115; pregnant women on the first antenatal visit | Women with vaginosis-like microflora vs. those with healthy microflora | Higher[a] sTfR: an independent predictor of vaginosis-like microflora: [OR=4.5, 95 % CI (1.4–14.2)] (multivariate analysis) |
| Studies reporting on iron deficiency anemia and infections | | | | |
| Levy et al., 2005 [16] | Prospective cohort; 1989–1992 and 1994–1997, Israel | 521; healthy Bedouin infants aged up to 18 months | Anemic due to ID[a] vs. non-anemic | Anemia at 6 months: an independent risk factor for ≥5 respiratory[c] and otitis media episodes from 7 to 18 months of age: [RR=1.99, 95 % CI (1.07–3.71), $p$=0.03] and [RR=2.23, 95 % CI (1.19–4.19), $p$=0.01], respectively (multivariate analysis)Anemia at 6 months increased the risk for respiratory disease after that age by 2-fold [95 % CI (1.1–3.7), $p$=0.03] (multivariate analysis) |
| Myers et al., 2004 [17] | Prospective cohort; 1999–2000, Ireland | 225; elderly with THR | Anemic[a,b] vs. non-anemic | Postoperative UTI: more common in anemic, 28 % vs. 14 %, $p$=0.04 (univariate analysis)Postoperative RTI: 14 % vs. 12 %, $p$=0.55 (univariate analysis) |

*CHr* reticulocyte hemoglobin content (used as the iron deficiency index), *ICU* intensive care unit, *FID* functional iron deficiency, *sTfR* soluble transferrin receptor (used as the iron deficiency index), *ID* iron deficiency, *UTI* urinary tract infection, *THR* total hip replacement, *RTI* respiratory tract infection

[a] As defined by each study's investigators

[b] 21 out of 35 (60 %) patients with preclinical anemia had iron deficiency anemia

[c] Respiratory episodes were cold, tonsillitis, bronchitis, pneumonia, and asthma

Iron deficiency anemia and infection

In one study, iron deficiency anemia at the age of 6 months was an independent risk factor for the occurrence of at least five respiratory disease or otitis media episodes between 7 and 18 months of age, while the risk for respiratory disease (cold, tonsillitis, bronchitis, pneumonia, asthma) increased 2-fold after that age [95 % confidence interval (1.1–3.7), $p$=0.03] [16]. In another study, postoperative urinary tract infections were more common in anemic patients after total hip replacement than in non-anemic patients, 28 % vs. 14 % ($p$=0.04). However, the incidence of postoperative respiratory tract infections did not differ between the two patient groups [17]. In this study, anemia was due to iron deficiency in 60 % of patients.

## Discussion

Our systematic review shows that there is a scarcity of published clinical evidence regarding the association between iron deficiency and the susceptibility to any kind of infection. However, five of the six relevant studies that we identified found that iron deficiency or iron deficiency anemia was associated with a higher occurrence of different types of infections in the different patient populations evaluated in each of the studies.

Iron constitutes an essential nutrient for most of the human pathogens [28–31]. Accordingly, there are host defense mechanisms that tend to restrict the available iron from the invading pathogens [7]. On the other hand, iron appears to be a valuable contributor for normal human immunity. First of all, iron is required for the normal proliferation of tissues with a rapid turnover such as the intestinal epithelium, which constitutes a physical barrier to infection [32]. It is also a component of the biological systems that mediate the biosynthesis of peroxide-generating enzymes and nitrous oxide-generating enzymes or the regulation of cytokine production [33]. Many studies have suggested that both cell-mediated and humoral immunity can be impaired in iron deficiency anemia [2, 3, 5, 6]. For example, the phagocytic activity of the monocytes has been found to be decreased in patients with iron deficiency anemia and no other underlying disease [2]. Enzymes that mediate bacterial killing, such as hydroxyl radicals, are not adequately produced by the macrophages in iron-deficient patients [34]. Premenopausal women with iron deficiency anemia as a solitary pathology have been shown to have fewer T-lymphocytes compared to those without anemia [35]. Lymphocyte responses, particularly interleukin-2 production, has also been shown to be impaired in iron-deficient children [36].

One study showed that iron deficiency anemia in infants was an independent risk factor for the occurrence of respiratory disease or otitis media later in infancy [16]. This may likely be related to the nasal obstruction caused by acute adenoiditis, which is quite common among children of this age [37, 38]. Accordingly, iron deficiency in infancy might cause vulnerability to acute adenoiditis. The higher susceptibility to infections in iron deficiency patients compared to those with normal iron status shown in the majority of the studies included in this review could indicate, if confirmed, that iron deficiency may be more important for human immunity than for pathogen proliferation. However, this balance could be pathogen-specific [39, 40]. Examples of pathogens whose virulence is thought to particularly depend on the uptake of iron from the environment include *Yersinia* spp., *Vibrio vulnificus*, *Listeria monocytogenes*, the agents of mucormycosis, mycobacteria, and several other intracellular pathogens [39, 41–44]. It should, by all means, not be disregarded that excess iron can adversely influence various functions of the immune system. Specifically, excess iron can impair the phagocytic and chemotactic properties of macrophages and neutrophils, and affect T-cell responses as well [32, 45, 46]. Therefore, the association between iron status and the immune system is probably that of a “double-edged sword” [46].

The implications of a potential association between iron deficiency and an increased risk for the development of infections in everyday clinical practice cannot be well established. Many other factors have already been found as contributors to the emergence of infections, both in outpatients and in hospitalized patients. Diabetes mellitus, renal failure, or obesity are such examples [39, 40, 47–49]. Whether careful iron supplementation to correct the iron deficit could reverse the association between iron deficiency and the susceptibility to infections is also highly uncertain [10, 50]. There are small studies showing that iron supplementation has been beneficial for chronic infectious syndromes, such as mucocutaneous candidiasis and recurrent furunculosis [51, 52]. In other studies, judicious iron replacement did not lead to a higher infection risk, even in critically ill patients [53]. Attention, however, should be paid specifically in regions where malaria is endemic (where children are commonly iron-deficient), because iron supplementation can confer an increased risk for the development of malaria [6, 54], if proper public health surveillance is not undertaken.

Our review should be interpreted in view of certain limitations. First, adjustment for the possible confounders of the association between iron status and infection was not performed in most of the included studies [14, 15, 17, 18]. Therefore, factors other than iron deficiency, which were not evaluated, such as underlying diseases associated with blood loss, could be responsible for the higher incidence of infections in the iron-deficient patients in those studies [55]. This particularly applies to patient groups other than children and premenopausal women, because, in these groups, iron deficiency typically relates to high growth requirements and blood loss due to menstruation, respectively. It should be mentioned that anemia was due to iron deficiency in only 60 % of the studied patients in one of the included studies [17]. The effect to which anemia *per se*, rather than iron deficiency, contributes to the infection risk cannot be accurately established on the basis of the reviewed data. Overall, few studies reporting on iron deficiency or iron deficiency anemia exclusively met the inclusion criteria of this review in order to allow for safe conclusions to be drawn.

In conclusion, the limited available evidence suggests that iron-deficient individuals might be more susceptible to the acquisition of different types of infections compared with those with normal iron status. However, further well-designed studies with adjustment for potential confounders are needed so as to verify these findings.

**Conflict of interest** The authors declare that they have no conflict of interest.

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