Association of ferritin and transferrin saturation with all-cause mortality, and the effect of concurrent inflammation: a danish cohort study

Nikki H. Mitchell, Henrik L. Jørgensen, Fie J. Vojdeman, Henriette P. Sennels, Christen L. Andersen, Margit Kriegbaum, Mia K. Grand, Christine W. Bang and Bent S. Lind

ABSTRACT

The association between ferritin and transferrin saturation (TS), respectively, and all-cause mortality is unclear. Furthermore, the influence of concurrent inflammation has not been sufficiently elucidated. We investigated these associations and the effect of concurrently elevated C-reactive protein (CRP), and accordingly report the levels associated with lowest all-cause mortality for females and males with and without inflammation.

Blood test results from 161,921 individuals were included. Statistical analyses were performed in sex-stratified subpopulations, with ferritin or TS level as continuous exposure variables, and were adjusted for age, co-morbidity and inflammation status using CRP. An interaction was used to investigate whether the effect of ferritin or TS on all-cause mortality was modified by inflammation status (CRP ≥ 10 mg/L or CRP < 10 mg/L). Low and high ferritin and TS levels were respectively associated with increased all-cause mortality in females and in males. These associations persisted with concurrent CRP ≥ 10 mg/L. The ferritin level associated with lowest mortality was 60 µg/L for females and 125 µg/L for males with CRP < 10 mg/L. It was 52 µg/L for females and 118 µg/L for males with CRP ≥ 10 mg/L. The TS level associated with lowest mortality was 33.9% for females and 32.3% for males with CRP < 10 mg/L. It was 28.7% for females and 30.6% for males with CRP ≥ 10 mg/L.

Our findings can nuance clinical interpretation and further aid in defining recommended ranges for ferritin and TS.

Introduction

Ferritin and transferrin saturation (TS) levels in blood can be used to assess the iron metabolic state. This is useful as both iron deficiency and iron overload can cause disease [1–7]. Ferritin and TS levels are affected during an acute-phase response [8]. In relation to an increase in C-reactive protein (CRP), ferritin levels have been shown to increase and TS levels to decrease [9]. Consequently, concurrent inflammation can affect the clinical interpretation of these parameters.

Ferritin and TS levels have been investigated for their respective associations with mortality in several population studies. These studies have found either a positive association between all-cause mortality and high ferritin [10], low ferritin [11], both high and low ferritin [12] or no association at all [13,14]. Similarly for TS, studies have found either an association between all-cause mortality and high TS [15,16], a U-shaped association [17], no association [13] or an inverse association [14]. Thus, findings have been inconsistent, and only a subset of the studies adjusted for CRP [12,13] to account for the known acute-phase reactivity of ferritin and TS [9].

We examined the associations of ferritin and TS measurements as continuous variables with all-cause mortality in a Danish primary care population. We furthermore determined how inflammation affected these associations, using concurrent CRP measurements. Finally, we report the level of ferritin and TS respectively associated with the lowest mortality in females and males, with and without concurrently elevated CRP.

Materials and methods

Study population

This study used data from primary care patients in the greater Copenhagen area of Denmark. Danish citizens can consult their GP free of charge, and the primary health care system is thus equally accessible for every Danish citizen regardless of socioeconomic status. The GPs in the
Copenhagen area were served by the Copenhagen General Practitioners’ Laboratory (CGPL). All results of the analyses performed at the CGPL between July 2000 and December 2015 have been saved in the Copenhagen Primary Care Laboratory (CopLab) database.

According to Statistics Denmark in 2008 (midway through our study period), the inhabitants of the Copenhagen area were mainly of Danish descent (83%) and other European descent (9%) [18].

The initially included population comprised all individuals with at least one measurement of either ferritin, iron or transferrin between 2000 and 2015. Only the first appearing requisition was included for each individual. We then excluded: all individuals with measurements from the year 2000 to ensure that we only included incident measurements; pregnant individuals from 12 months before the beginning of their pregnancy until 12 months after giving birth, with pregnancy defined as described in The Copenhagen Primary Care Laboratory Pregnancy (CopPreg) database [19]; individuals under the age of 18 years; non-GP blood sampling requisitions and requisitions with missing and/or invalid data (Figure 1).

This resulted in a population of 161,921 unique individuals. Of these 147,142 individuals were included in the 'ferritin population' by having a ferritin measurement and a concurrent measurement of CRP, and 43,963 individuals were included in the 'TS population' by having concurrent measurements of transferrin, iron and CRP on the same requisition. 29,184 individuals were in both the ferritin and in the TS population.

The ferritin and TS populations were stratified by sex, giving a total of four sub-populations. The sex distribution was 91,106 females and 56,036 males in the ferritin population, and 27,026 females and 16,937 males in the TS population (Figure 1).

In each of these sub-population, the CRP-level on the given requisition was used to define whether inflammation was present (CRP \( \geq 10 \text{ mg/L} \)) or absent (CRP \( < 10 \text{ mg/L} \)) at the time of measurement.

**Endpoint and covariates**

All subjects were followed from the date of blood measurement until the date of death. Alternatively, they were censored at the end of the registries on 31 December 2017, or at date of first emigration. The personal identification number (CPR number) is unique to every citizen in Denmark and enables matching of individuals across registries. To adjust for possible confounding by comorbidity, we computed Charlson Comorbidity Index (CCI) scores [20] which are based on chronic diseases, each weighted according to its potential to influence mortality. CCI was computed on the basis of all available diagnoses for each patient, stemming from the Danish National Patient Register going back to 1977 [21].

**Biochemical analyses**

The blood levels of ferritin, transferrin, iron, and CRP were measured as described in Supplementary Material 1.

**Statistical analyses**

We used Cox proportional hazard models for analyses of all-cause mortality. The analyses were performed in the sex-stratified subpopulations. In addition to the exposure variable, which was either ferritin or TS level, the models were adjusted for age, CCI and inflammation status, defined using CRP levels as either present (CRP \( \geq 10 \text{ mg/L} \)) or absent (CRP \(< 10 \text{ mg/L} \)). The effects of the exposures and age were modelled using penalized splines. The best fit for the splines was determined using 10-fold cross-validation. The baseline hazard was stratified by inflammation status. We further included an interaction between exposure and inflammation status, to be able to investigate whether the effect of the exposure (ferritin or TS) on the outcome (all-cause mortality) was modified by inflammation status. The model assumptions were checked by inspection of diagnostic plots. All statistical analyses were performed in SAS (9.4) and R (4.0.3).

**Ethical approval**

According to Danish legislation, no ethical approval or patient consent was required because the patients were not approached at any time during the conduct of the study.

The study was granted approval by the Danish Data Protection Agency to The Faculty of Health and Medical Sciences at the University of Copenhagen. (j.no. 2015-57-0121).

**Results**

The median follow-up time in the ferritin population was 6.5 years for females and 5.9 years for males. In the TS population, it was 8.8 years for females and 7.6 years for males. During the follow-up, 13,767 females and 11,275 males died in the ferritin population; 5285 females and 4184 males died in the TS population. Baseline characteristics are presented in Table 1.

The distributions of ferritin, TS and CRP can be seen in Supplementary Material 2 to Supplementary Material 5.

**Ferritin and all-cause mortality, with and without concurrent inflammation**

Regardless of sex and of the presence of concurrent inflammation, both low and high ferritin levels were associated with an increased relative hazard of death from all causes (Figure 2).

In females, the most pronounced effect was seen for high ferritin levels. In males, high and low values were associated with equally increased all-cause mortality.
In females without present inflammation, the ferritin level associated with lowest all-cause mortality was 60 µg/L. This was considered as a relative hazard of 1.00. The values of ferritin where the relative hazard was different from 1.00 at a 95% confidence level were 37 µg/L and 101 µg/L, respectively.

Corresponding values were: 52 µg/L, 16 µg/L and 68 µg/L in females with present inflammation; 125 µg/L, 79 µg/L and
193 μg/L in males without inflammation; and 118 μg/L, 90 μg/L and 220 μg/L in males with present inflammation. In a subanalysis, we defined four age-groups: (18-35 years), (36-50 years), (51-65 years) and (≥66 years), and performed an analysis for men and women, separately. The impact of the age groups was similar for men and women. However, there was a significant interaction by age group in both sexes.

The absolute survival probabilities for different ferritin levels are illustrated in Supplementary Material 6.

The absolute survival probabilities for different TS levels are illustrated in Supplementary Material 7. The association of transferrin and iron, respectively, and all-cause mortality with and without concurrent inflammation is presented in Supplementary Materials 8 and 9.

**Discussion**

In this study comprising 161,921 individuals from Danish primary care, we showed that both low and high ferritin and TS were associated with increased all-cause mortality in both females and males. These associations persisted with concurrent inflammation.

The ferritin level associated with the lowest all-cause mortality was 60 μg/L for females and 125 μg/L for males when CRP < 10 mg/L. These values were respectively considered as representing a relative hazard of 1.00. The values of TS where the relative hazard was different from 1.00 at a 95% confidence level were 27.9% or 44.1%, respectively.

Corresponding values were: 28.7%, 24.4% and 54.7% in females with present inflammation; 32.3%, 26.7% and 42.3% in males without inflammation; and 30.6%, 25.5% and 46.7% in males with present inflammation.

The ferritin level associated with the lowest all-cause mortality was 60 μg/L for females and 125 μg/L for males when inflammation was absent, and 52 μg/L for females and 118 μg/L for males when inflammation was present.

The TS level associated with the lowest all-cause mortality was 33.9% for females and 32.3% for males when inflammation was absent, and 28.7% for females and 30.6% for males when inflammation was present.

Regardless of sex and the presence of concurrent inflammation, both low and high TS levels were associated with an increased relative hazard of death from all causes.

In females, high and low TS were equally associated with all-cause mortality regardless of concurrent inflammation. Similarly, in males, the associations were U-shaped with and without inflammation (Figure 3).

In females without present inflammation, the TS level associated with lowest all-cause mortality was 33.9%. This was considered as a relative hazard of 1.00. The values of TS where the relative hazard was different from 1.00 at a 95% confidence level were 27.9% or 44.1%, respectively.

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The TS level associated with the lowest all-cause mortality was 33.9% for females and 32.3% for males when inflammation was absent, and 28.7% for females and 30.6% for males when inflammation was present.

**Ferritin and all-cause mortality**

In a clinical setting, ferritin levels of 20–300 μg/L for males and postmenopausal females and 15 to 200 μg/L for premenopausal females have been proposed as normal values [22].

Our analyses of ferritin showed that levels of 60 μg/L for females and 125 μg/L for males were associated with the lowest all-cause mortality when CRP < 10 mg/L. These values were respectively considered as representing a relative
hazard of 1.00. The values with a relative hazard different from 1.00 at a 95% confidence level were: 37 and 101 μg/L for females, and 79 and 193 μg/L for males. Thus, recommended ferritin ranges include levels that we showed to be associated with increased all-cause mortality. Although all-cause mortality and iron deficiency and overload are different outcomes, it is still reasonable to take the shown association to mortality into account when interpreting ferritin levels in a clinical setting, as well as when defining optimal ranges for ferritin in populations.

When CRP levels were ≥ 10 mg/L, 52 μg/L for females and 118 μg/L for males were associated with the lowest all-cause mortality. These values represented a relative hazard of 1.00. Ferritin levels below 16 μg/L and above 68 μg/L in females, and below 90 and above 220 μg/L in males had a relative hazard greater than 1.00 at a 95% confidence level.

Ferritin cut-off values for iron deficiency are <15 to 30 μg/L in healthy subjects, and <70 to 100 μg/L in individuals with inflammation [23]. Our results indicate that the established ferritin cut-off for iron deficiency in a setting with inflammation could be too high in females, bearing in mind that our outcome was mortality rather than iron deficiency.

Ferritin cut-offs of >150 μg/L in apparently healthy menstruating females and >200 μg/L in apparently healthy males and non-menstruating females, along with a cut-off of >500 μg/L in adult non-healthy individuals, have been recommended to evaluate possible iron overload [24]. The latter cut-off value was based on very limited evidence, and our results indicate that this level could be too high.

Our findings are in overall agreement with two earlier studies: A Danish study reported that increased ferritin (≥ 200 μg/L) was associated with increased all-cause mortality in a population-based follow-up study [10]. Another recent study found that absolute iron deficiency (ferritin ≤ 30 μg/L) was associated with all-cause mortality [11]. We add to their results by showing that the associations of high as well as low ferritin with increased all-cause mortality seem to be persistent regardless of elevated CRP.

Another study, which did include CRP as a control variable, investigated the association of ferritin as quartiles with all-cause mortality, and found that the lowest ferritin quartile was associated with increased all-cause mortality for females, whereas the highest ferritin quartile was associated with increased all-cause mortality for males with no major chronic diseases [12]. In our analyses, we found both high and low ferritin to be associated with increased mortality for females and males alike, and further that high ferritin levels seemed more deleterious than low levels in females. This discrepancy might be due to their use of ferritin levels.

Figure 2. Relative hazards for the associations between ferritin level and death for females and males, given absent (CRP < 10 mg/L) or present (CRP ≥ 10 mg/L) inflammation. The dashed lines represent 95% confidence intervals. CRP: C-reactive protein.
quartiles [12] as opposed to our continuously expression of ferritin.

Contrary to us, two earlier studies found no associations of ferritin quartiles [13] or quintiles [14] with all-cause mortality, both using the Third National Health and Nutrition Examination Survey population from the US. Only the former study included CRP as a control parameter [13]. These results conflict with ours. This could be due to our Danish primary care population differing from the US population, and/or because we expressed ferritin as continuous variables rather than using percentiles.

**TS and all-cause mortality**

TS levels of 25 to 45% have been proposed as normal values for both sexes in a clinical context [22]. Likewise, 1/3 saturation of transferrin with iron has been described as normal [25].

We showed that TS levels of around 33% for both females and males were associated with the lowest all-cause mortality when inflammation was absent. The values of TS with a relative hazard different from 1.00 at a 95% confidence level, which corresponded to the respective lowest relative hazards, were: 27.9 and 44.1% for females, and 26.7 and 42.3% for males.

In the presence of elevated CRP, a TS of 28.7% for females and 30.6% for males were associated with the lowest all-cause mortality, corresponding to a relative hazard of 1.00. TS levels below 24.4% and above 54.7% in females, and below 25.5% and above 46.7% in males had a relative hazard different than 1.00 at a 95% confidence level.

Thus, a TS level of around 29 to 34% was associated with the lowest all-cause mortality, irrespective of sex and the presence of inflammation. This is line with already proposed normal ranges [22,25].

A population-based study combining two Danish Cohorts reported that a stepwise increase in TS was associated with an increased risk of mortality from values \(\geq 40\%\) (with TS <20% as reference) [15]. A US population-based study showed that individuals with a TS \(\geq 55\%\) compared with \(\leq 55\%\) had increased all-cause mortality [16]. Finally, another US study showed, that compared with the third TS quartile (23.7 to 31.3%), being in the first, second or fourth quartile increased HR for all-cause mortality [17]. These three studies all reported findings generally in line with ours, albeit none of them had CRP as a control variable. Our study therefore adds this explanation to the possible

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**Figure 3.** Relative hazards for the associations between TS level and death for females and males, given absent (CRP < 10 mg/L) or present (CRP \(\geq 10\) mg/L) inflammation. The dashed lines represent 95% confidence intervals. TS: transferrin saturation; CRP: C-reactive protein.
underlying mechanisms of the associations with all-cause mortality.

Two studies using The Third National Health and Nutrition Examination Survey investigated the associations of TS quartiles [13] and quintiles [14], and their reported findings opposes ours. One study found that TS was not associated with altered risk of mortality [13], one found an inverse association between higher TS quintile and all-cause mortality [14]. These different findings could be explained by differences in the population, and by TS percentiles being used.

The median level of TS in our population was 22.2% for females and 26.4% for males, which is below the optimal level in relation to mortality that we found in this study. It is further below what by some are considered normal [22,25]. Moreover, it is close to the cut-off of 20% set by a recent study for non-anemic iron deficiency (with or without inflammation) [23].

This indicates that the majority of the TS population in our study would benefit from an increase in their TS in relation to their survival, and it also indicates that some degree of iron deficiency is quite common – although causality cannot be established on the current basis. Although we do not know the indications for the blood tests in our primary care population, the TS levels in our population are nevertheless similar to what is seen among healthy adults [26].

The role of concurrent inflammation

The finding that the associations of ferritin and TS with all-cause mortality persisted in the presence of inflammation indicates that the acute phase properties of these parameters are not a main factor in driving the associations shown by us and others as well [10–12,15–17].

This could mean that the iron load level itself is a main driving force regarding the altered mortality.

Strengths and limitations

The main strength is the size of our included population, and that we had concurrent CRP-values. Furthermore, our results stem from a consecutive series of patients who did not have to opt-in as is often the premise of general population studies.

Access to data regarding co-morbidities gave us the opportunity to control for possible confounders in our data analyses. It would however have been beneficial to have access to even more co-variates for our analyses.

The biochemical analyses were all performed at the same accredited laboratory and all the assays underwent quality assurance (Supplementary Material 1). Our data stem from GPs from the same well-defined geographical area. The inhabitants were of predominantly Danish or other European descent during the study period [18], which is important as there are known ethnic differences in levels of ferritin and TS [27]. All blood samples were collected during daytime, minimizing the diurnal fluctuations of the investigated parameters, which have been demonstrated in an earlier study [28].

The lack of indications for the blood tests makes it uncertain if these results can be generalized to the entire population. However, it seems reasonable that our findings can be generalized to new individuals who are tested by their GP, given that the incentive for investigating ferritin and TS stays the same.

Conclusions

In a Danish primary care population, both low and high levels of ferritin and TS were associated with increased all-cause mortality in both females and males.

The ferritin levels associated with the lowest all-cause mortality were 60 μg/L for females and 125 μg/L for males without inflammation. With concurrent inflammation, it was 52 μg/L for females and 118 μg/L for males. For TS, a level of 29 to 34% was associated with the lowest all-cause mortality in both sexes, irrespective of the presences of inflammation.

Our findings indicate that inflammation does not substantially modulate the association between ferritin, TS and all-cause mortality. Our findings further raise the question, if current recommended ranges and cut-offs for ferritin and TS are optimal, especially in the presence of inflammation.

Confirmation of our results, and clarification of whether these associations are related to the iron balance itself and to what extent other underlying morbidities contribute to these associations are warranted. This would aid interpretation in a clinical setting. It would further provide a stronger basis for general recommendations regarding optimal levels of ferritin and TS.

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Author contributions

NHM, BSL, HLJ, CLA, MKG and MK were involved in designing the study. MK, MKG and CWB were responsible for the data requisition and analyses. BSL contributed with information on CopLab methods and materials. All authors contributed to the interpretation of the data. NHM drafted the manuscript and all additional authors were involved in the revision.

All authors approved the final version of the manuscript.

NHM is the corresponding author. The corresponding author attests that all listed authors meet authorship criteria and that no others meeting the criteria have been omitted.

Disclosure statement

No potential conflict of interest was reported by the author(s).
The data providing the grounds of this study is located at server housed by Statistics Denmark. Access to the data is restricted, and data is thus not available to the public.

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