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Review

Patterns of muscle strength loss with age in the general population and patients with a chronic inflammatory state

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\textbf{ABSTRACT}

\textbf{Background:} There is growing recognition of the serious consequences of sarcopenia on the functionality and autonomy in old age. Recently, the age-related changes in several inflammatory mediators have been implicated in the pathogenesis of sarcopenia. The purposes of this systematic review were two-fold: (1) to describe the patterns of muscle strength loss with age in the general population, and (2) to quantify the loss of muscle strength in rheumatoid arthritis as representative for an underlying inflammatory state. Handgrip strength was used as a proxy for overall muscle strength.

\textbf{Results:} Results from 114 studies (involving 90,520 subjects) and 71 studies (involving 10,529 subjects) were combined in a meta-analysis for the general and rheumatoid arthritis population respectively and standardized at an equal sex distribution. For the general population we showed that between the ages of 25 years and 95 years mean handgrip strength declined from 45.5 kg to 23.2 kg for males and from 27.1 kg to 12.8 kg for females. We noted a steeper handgrip strength decline after 50 years of age (rate of 0.37 kg/year). In the rheumatoid arthritis population handgrip strength was not associated with chronological age between the ages of 35 years and 65 years and was as low as 20.2 kg in male and 15.1 in female. Rheumatoid arthritis disease duration was inversely associated with handgrip strength.

\textbf{Conclusions:} This meta-analysis shows distinct patterns of age-related decrease of handgrip strength in the general population. Handgrip strength is strongly associated with the presence and duration of an inflammatory state as rheumatoid arthritis. The putative link between age-related inflammation and sarcopenia mandates further study as it represents a potential target for intervention to maintain functional independence in old age.

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1. Introduction

Sarcopenia, age-related loss of muscle mass and strength, is highly prevalent, with reported proportions exceeding 50% in those aged 80 years and older (Iannuzzi-Sucich et al., 2002; Baumgartner et al., 1998). This ageing phenomenon is becoming an important public health concern as it inflicts a profound functional burden on our growing elderly population and contributes to increased morbidity (Rantanen et al., 1994; Rantanen et al., 1999; Taekema et al., 2010) and mortality (Rantanen et al., 2000; Metter et al., 2002; Ling et al., 2010). Several studies have shown that loss of muscle mass occurs as early as the fifth decade of life and accelerates in older age (Lexell et al., 1986; Janssen et al., 2000). The etiology and pathogenesis of sarcopenia is complex and probably involves the interplay of a myriad of factors including physical inactivity, hormonal, metabolic and nutritional factors (Doherty, 2003; Morley et al., 2001). Recent research has also implicated age-related changes in several inflammatory mediators in the pathogenesis of sarcopenia (Krabbe et al., 2004).

Similar to the ageing process, inflammatory cytokines have been shown to have a profound role in the pathogenesis of “rheumatoid cachexia”, the loss of muscle mass and strength with concomitant increase in fat mass, which persists after joint inflammation improves in rheumatoid arthritis (RA) patients (Roubenoff, 2009). In patients with chronic inflammatory diseases such as RA, there is an accelerated loss of muscle mass and strength compared to healthy subjects (Roubenoff, 2000; Madhok et al., 1993).
osteoarthritis, diabetes, growth hormone or testosterone deficiency including diseases or conditions that may affect HGS such as wards or rehabilitation clinics were excluded as well as those with RA.

There were no nationality or ethnicity related selection criteria. There were no enrolment criteria for the general population or subjects with RA; (2) HGS was measured by a handheld dynamometer with results reported in kilogram (kg), Newton (N) or pound (lb) or a sphygmomanometer with results reported in millimeter of mercury (mm Hg) or kilo-Pascal and (3) data on sample size, age and gender distribution. Whenever available, anthropometrical data, RA disease duration, pain-scores (as measured on the visual analogue scale (VAS)), general health (as measured on the health assessment questionnaire (HAQ)), inflammatory markers (C-reactive protein (CRP) or erythrocyte sedimentation rate (ESR)) were also extracted. When HGS measurements were reported separately for male and female or for different age categories or other study specific sub-grouping, these measurements were regarded as being distinct study groups. Therefore, multiple study groups were extracted from the original articles. In case the age category was reported as range without the mean age, the midpoint of the age range was used for the analysis. Study groups were excluded when no lower or upper age cut-off was described for the age category. For longitudinal studies, only baseline data were selected and HGS measurements reported on subjects who died during the study follow-up duration were excluded.

If necessary, data were converted into kg equivalent: newtons (N) were divided by 9.81; pounds (lb) by 0.45; units of pressure (mm Hg) or force (kPa) were converted using validated methods (Desrosiers et al., 1995; Agnew and Maas, 1991). In studies where only the mean HGS was reported without the SD, the latter was estimated based on the linear regression of the variance of HGS on the mean HGS using the groups that provided complete data (mean and SD) for the general or RA population.

2.2. Data extraction

The following information was extracted from each article: year of publication, sample size, mean and standard deviation (SD) of HGS, age and gender distribution. Whenever available, anthropometrical data, RA disease duration, pain-scores (as measured on the visual analogue scale (VAS)), general health (as measured on the health assessment questionnaire (HAQ)), inflammatory markers (C-reactive protein (CRP) or erythrocyte sedimentation rate (ESR)) were also extracted. When HGS measurements were reported separately for male and female or for different age categories or other study specific sub-grouping, these measurements were regarded as being distinct study groups. Therefore, multiple study groups were extracted from the original articles. In case the age category was reported as range without the mean age, the midpoint of the age range was used for the analysis. Study groups were excluded when no lower or upper age cut-off was described for the age category. For longitudinal studies, only baseline data were selected and HGS measurements reported on subjects who died during the study follow-up duration were excluded.

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2.3. Statistical analysis

To determine the mean HGS at different calendar ages in the general and RA population, we performed random effects meta-regression analyses adjusted for age and gender (van Houwelingen et al., 2002). These meta-regression models allowed us to make tables and figures for study groups standardized at 50% females or for males and females separately. For the general population, we first ascertained by eyeballing the scatter plot a change point at which the slope of annual decrease in HGS changed most rapidly. This change point parameter was then included in the meta-regression analysis. This change point parameter was then included in the meta-regression model of the general population, this resulted in inclusion of the gender × age interaction term in the analysis. This interaction indicated that the relation between HGS and age was dependent on the gender distribution. We plotted this relationship standardized at an equal sex distribution. For the RA population, none of the squared terms or interaction terms was significant and therefore, no further extension of the model was needed. Similarly, the method of forward selection was used in the analysis of HGS and RA disease duration, adjusted for age and gender. This resulted in inclusion of disease duration × age interaction to the meta-regression model. Again, the meta-regression line of this model was only plotted for the case of an equal sex distribution. All statistical analyses were performed in STATA 10.
71 studies (102 study groups) of the rheumatoid arthritis (RA) population. Descriptive statistics of extracted means from study groups included in the review. Study groups derived from 114 studies (330 study groups) of the general population and

Table 1

| General population | RA population |
|-------------------|--------------|
| N                 | Median (IQR) |
| Total number subjects | 90,520 |
| Number of subjects per study group | 50.5 (19–189) |
| Age (years) | 65.0 (50.5–72.7) |
| Age SD per study group (years) | 5.2 (3.0–7.8) |
| Gender (% females) | 54.6 (0–1) |
| Disease duration (years) | – |
| Anthropomorphic measurements | – |
| Height (cm) | 163.3 (158.0–172.5) |
| Weight (kg) | 66.7 (58.1–76.4) |
| BMI (kg/m²) | 23.0 (23.0–26.6) |
| Clinical measurements | – |
| Pain, VAS 0–100 | 2 (0–11.5) |
| HAQ, 0–3 | 0.05 (0–0.07) |
| Inflammatory markers | – |
| CRP (mg/L) | 2.98 (1.0–6.7) |
| ESR (mm/h) | 8.8 (–) |

Data are presented as medians with 25th and 75th percentiles (i.e., interquartile ranges [IQR]). N: number of study group with available data. Study groups are defined as whole study groups or sub-study groups based on gender, age categories or study specific sub-grouping.

3. Results

Out of 114 studies related to HGS in the general population we extracted 330 study groups involving 90,520 subjects. For RA, 102 study groups were extracted from 71 studies involving 10,529 subjects (Fig. 1). In the general population, HGS was measured using a hand-held dynamometer in 99.7% of the study groups and a sphygmomanometer in 0.3%, compared to 61.7% and 38.3%, respectively of the study groups in the RA population. Standard deviation of the mean HGS was reported in 82.1% and 79.4% of the study groups for the general and RA population, respectively. Table 1 shows the characteristics of the extracted study groups for the two populations apart. The median number of subjects per study group was 50.5 in the general population and 47.0 in the RA population. The age range of subjects within all study groups for the two populations apart. The median number of subjects per study group was 50.5 in the general population and 47.0 in the RA population. The age range of subjects within all study groups for the two populations apart. The median number of subjects per study group was 50.5 in the general population and 47.0 in the RA population.

Table 2 shows the mean HGS values according to age for the general and RA population dependent on age for male and female.

Table 2

| Age (years) | General population | RA population |
|-------------|-------------------|--------------|
|             | Male | Female | Male | Female |
| Mean in kg (95% CI) | Mean in kg (95% CI) | Mean in kg (95% CI) | Mean in kg (95% CI) |
| 25 | 45.5 (43.2; 47.8) | 27.1 (24.4; 29.7) | – | – |
| 35 | 44.3 (42.7; 45.9) | 27.0 (25.2; 28.8) | 20.2 (15.0; 25.3) | 15.0 (11.2; 18.9) |
| 45 | 43.1 (41.7; 44.6) | 27.0 (25.5; 28.5) | 20.2 (16.1; 24.4) | 15.1 (12.7; 17.4) |
| 55 | 40.4 (39.1; 41.8) | 25.4 (24.1; 26.7) | 20.3 (16.4; 24.2) | 15.1 (13.3; 16.9) |
| 65 | 36.1 (35.0; 37.2) | 22.3 (21.2; 23.3) | 20.4 (16.0; 24.7) | 15.2 (12.4; 18.0) |
| 75 | 31.8 (30.5; 33.2) | 19.1 (17.9; 20.3) | – | – |
| 85 | 27.5 (25.6; 29.5) | 16.0 (14.2; 17.7) | – | – |
| 95 | 23.2 (20.6; 25.8) | 12.8 (10.4; 15.2) | – | – |

Grip strength in kg of each age is standardized for male and female.

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a Meta-regression model with change point at age 50 years.
b Linear meta-regression model.

CI: confidence interval.
Table 3
Change in handgrip strength (kg) per calendar year in the general population and rheumatoid arthritis (RA) population.

|                          | General population\(^a\) | RA population\(^b\) |
|--------------------------|--------------------------|---------------------|
| Change of grip strength  |                          |                     |
| (kg/y)                   |                           |                     |
| Age range in years       | Mean (95% CI)             | Mean (95% CI)       |
| 20–50                    | -0.06 (-0.16; 0.04)       | 0.01 (-0.18; 0.19)  |
| 50–100                   | -0.37 (-0.44; -0.31)      |                     |

Standardized at 50% females.

\(^a\) Meta-regression model with change point at age 50 years.

\(^b\) Linear meta-regression model.

CI: confidence interval.

4. Discussion

We performed meta-regression analyses to evaluate the patterns of HGS loss with age in the general population and quantified the magnitude of strength loss when chronic inflammation is present, using RA as a representative condition. HGS was used as a proxy for overall muscular strength as it has been shown to correlate well with whole body muscle strength (Rantanen et al., 1994). It is also an easily accessible, simple and reliable clinical assessment tool (Innes, 1999). We found an inverse relationship between HGS and age, with a gradual decline starting as early as the third decade of life followed by a steeper deterioration after 50 years of age. Subjects with RA had significantly lower handgrip strength compared to the general population of similar age. HGS was also associated with disease duration of RA. To the best of our knowledge, this study provides the first large sample systematic review of the patterns of loss of muscle strength in the general as well as the in the RA population over a wide age range.

The data from our study on the pattern of HGS loss in the general population support the curvilinear relationship between HGS and age as reported in some earlier published studies (Vianna et al., 2007; Rantanen et al., 1998). In women, the rapid decline in HGS after 50 years of age has been linked to sex hormone deficiency occurring at menopause and lifestyle changes during the transition (Samson et al., 2000; Vianna et al., 2007). In men however, the profile of strength loss is less well established (Metter et al., 2002; Samson et al., 2000; Vianna et al., 2007). Nonetheless, the rate of HGS decline in the general population of this study comprising of 50% females was comparable to that reported by Rantanen et al. (1998).

We also found that HGS in middle-aged RA subjects was approximately half that of the general population. This finding supports previous observations on the negative effects of chronic inflammation on muscle function (van Hall et al., 2008; Bodell et al., 2009). Few studies have reported the crucial role of “sarcoactive” cytokines such as TNF-α and IL-6 and CRP in the pathogenesis of RA (Madhok et al., 1993; Choy and Panayi, 2001; Engvall et al., 2008). These pro-inflammatory cytokines have also been
implicated in the pathogenesis of sarcopenia in “normal” ageing (Schaap et al., 2009).

In RA subjects loss of muscle strength was already observed before the age of 50 years. This supports the concept of RA being a disease of accelerated aging. Recently it has been shown that RA patients suffer from excess ageing occurring prior to RA incidence as well as an acceleration of ageing (Crowson et al., 2010). We postulate that the loss of muscle strength early in life of RA subjects is one of the aspects behind this phenomenon.

Our quantitative analysis of the literature has several limitations. First, there is the limitation due to using aggregate data (study groups) instead of data of individuals. However, the variance that is lost by aggregating individuals into study groups in the general population is limited, because the median SD of age for the study groups is calculated to be only 5.2 years, which is small compared to the between studies variance in mean age. In RA the median SD of age was higher (12.0 years) which could have led to an underestimation of the effect of age on HGS. Despite of aggregating, the statistical heterogeneity between study groups was high. This might be caused by differences between studies in factors such as method of HGS measurement, body composition and ethnicity and in the RA population also by RA disease duration and medication use.

We were unable to directly assess the influence of inflammation on HGS in the RA subjects as we did not account for various other factors that could affect HGS measurements such as pain or fear of pain, stiffness, corticosteroid use, disuse atrophy and mechanical disruption. Furthermore, in the RA population, we observed an inverse association between HGS and duration of RA disease but not the influence of chronological age per se. This could in part be explained by selection as RA subjects with profound muscle weakness were less likely to participate in studies and had higher mortality due systemic disease. Still, the amount of inflammatory pressure during the ageing process is low when compared to RA (Madhok et al., 1993). We showed that at the moment of RA diagnosis mean HGS is already as low as 20 kg. This implies that the impact of chronic inflammation on HGS was the highest already during the months from the earliest onset of RA and the moment of diagnosis, which normally goes together with the start of an anti-inflammatory treatment (Morel and Combe, 2005). For the period after diagnosis we showed RA to be associated with remarkable elevated level of inflammatory markers ESR and CRP.

This meta-analysis confirms previously reported patterns of age-related decrease of HGS in the general population. The putative link between age-related inflammation and sarcopenia mandates further exploration. Investigating RA may serve as representative condition when studying inflammatory pathways that lead to sarcopenia, which are relatively subtle in the general population. These pathways, however, represent a potential target for intervention to prevent disability and maintain functional independence in old age.

Conflict of interest

None of the authors reported any conflict of interest.

Contributors

Study concept and design: Beenakker and Maier. Acquisition of data: Beenakker and Maier. Analysis and interpretation of data: Beenakker, Westendorp, and Maier. Drafting of the manuscript: Beenakker, Ling, Meskers, and Maier. Critical revision of the manuscript for important intellectual content: Ling, Meskers, Westendorp, and Maier. Statistical analysis: Beenakker, de Craen, Stijnen, and Maier. Study supervision: Westendorp and Maier.

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