# Association between arthritis and cardiovascular risk factors in community-based adults: an opportunity to target cardiovascular risk 

Julia Sewell ${ }^{1 \dagger}$, Sultana Monira Hussain ${ }^{1 \dagger}$, Yuanyuan Wang ${ }^{1}$, Anita E. Wluka ${ }^{1}$, Yuan Z. Lim ${ }^{1}$, Melinda J. Carrington², Katherine Samaras ${ }^{3,4,5}$ and Flavia M. Cicuttini $i^{{ }^{*}}$


#### Abstract

Background: Undertreated risk factors are major contributors to the burden of cardiovascular disease (CVD). Those with arthritis have an increased prevalence of CVD risk factors. CVD risk factors are often asymptomatic, which may be a barrier their treatment. Arthritis causes pain and immobility, and is a common reason for individuals to seek healthcare. Our aims were to (1) examine the relationship between arthritis and CVD risk factors in Australian adults, and (2) calculate the proportion of CVD risk factors that could be reduced if individuals with arthritis were targeted. Methods: This cross-sectional study uses data from the 2017-18 Australian National Health Survey which included 13,776 participants, categorised into young (18-39 years), middle aged ( $40-64$ years) and older ( $\geq 65$ years) adults. Hypertension, height and weight were measured. Arthritis, dyslipidemia and diabetes were self-reported. The associations between arthritis and CVD risk factors were examined using logistic regression, and the population attributable fraction (PAF) of arthritis for each CVD risk factor was calculated. Results: Arthritis was reported by $4.0 \%$ of young adults, $28.8 \%$ of middle-aged adults and $54.5 \%$ of older adults. Those with arthritis were at increased odds of obesity ( 2.07 fold in young, 1.75 fold in middle-aged and 1.89 fold in older adults), increased odds of diabetes ( 5.70 fold in young, 1.64 fold in middle-aged and 1.37 fold in older adults), increased odds of hypertension ( 2.72 fold in young, 1.78 fold in middle-aged and 1.48 fold in older adults) and an increased odds of dyslipidaemia ( 4.64 fold in young, 2.14 fold in middle-aged and 1.22 fold in older adults) compared to those without arthritis. This elevated chance remained significant even after adjusting for obesity, with the exception of diabetes in the older population. This elevated chance remained significant even after adjusting for obesity, with the exception of diabetes in the older population. The PAF of the presence of arthritis for having at least one CVD risk factor was $30.7 \%$ in middle-aged adults and $70.4 \%$ in older adults. Conclusion: Australian adults of all ages with arthritis are at increased odds of having CVD risk factors. For young and middle-aged adults, this increased odds remains significant even when adjusted for obesity. Presentation to


[^0]healthcare practitioners with arthritis is an opportunity to screen for asymptomatic CVD risk factors with the potential of improving outcomes for both diseases. By adopting an approach of managing arthritis and CVD risk factors in parallel, rather than in silos, we could reduce the burden of CVD risk factors by 20-30\%.
Keywords: Arthritis, Osteoarthritis, Inflammatory arthritis, Crystal arthritis, Cardiovascular disease, Cardiovascular risk factors, Obesity, Hypertension, Diabetes, Dyslipidaemia

## Background

Cardiovascular diseases (CVD) remain the leading cause of disease burden in the world [1]. CVD burden has continued to rise for decades in almost all countries, and alarmingly the age-standardized rate of CVD has begun to rise. According to the Global Burden of Disease study, CVD was the underlying cause of 6.2 million deaths occurring between the ages of 30 and 70 years, and onethird of all deaths globally in 2019 [2]. The underlying pathophysiology of CVD is accelerated by obesity, hypertension, dyslipidemia and diabetes [3]. These conditions develop over many years, remaining silent, and thus are usually advanced by the time CVD symptoms occur [4]. Despite traditional risk score screening being recommended in guidelines for identifying individuals at risk of developing CVD [4-7], this remains underutilised [8]. Addressing these risk factors in all affected individuals is challenging, as many people with these CVD risk factors are largely asymptomatic and may not be actively seeking healthcare to specifically address their CVD risk. Hence, using other risk-enhancing factors to identify subsets of people most likely to receive clinical benefit for prevention of CVD offers an alternative approach.

Musculoskeletal conditions such as arthritis, in contrast, cause pain and immobility and are one of the most common reasons for presentation to primary healthcare [9]. People with most forms of arthritis (e.g. inflammatory arthritis, gout and osteoarthritis or OA) have an increased risk of CVD and death from CVD [10-14]. For example, the rate of CVD is increased by $1.5-2.0$ fold in those with rheumatoid arthritis [15], 1.3 fold in systemic connective tissue disorders [16], 1.5 fold in gout [17] and twofold in OA [18]. Shared inflammatory pathways has been suggested as a possible link for any form of these arthritis and CVD. Therefore presentations for arthritis symptoms may be used as a 'teachable moment', or an opportunity for healthcare practitioners to manage arthritis and screen for CVD risk factors in parallel in order to reduce the burden of both conditions. Thus, our aim was to examine the relationship between arthritis and CVD risk factors (obesity, hypertension, dyslipidaemia and diabetes) in young, middle-aged and older Australian adults and to calculate the proportion of individuals with CVD risk factors in the population that could be reduced by targeting arthritis.

## Methods

Study design
This is a cross-sectional study using the National Health Survey (NHS) data conducted by the Australian Bureau of Statistics (ABS) in 2017-18 [19].The NHS was conducted in metropolitan, regional and rural areas of all Australian states and territories. Participants were excluded if they were under the age of 18 since both OA and CVD are less common in this age category [18].

## Demographic, anthropometric and clinical measurement

Trained ABS interviewers conducted interviews between 2 July 2017 and 30 June 2018. Voluntary measures of height and weight were collected from respondents. If respondents elected not to be measured, they were asked to self-report their height and weight. In total $80 \%$ of respondents agreed to be measured and the remainder elected to self-report these data [20]. Body mass index (BMI) was calculated using the formula weight ( kg ) divided by the square of height ( m ), and obesity was defined by BMI $>30 \mathrm{~kg} / \mathrm{m}^{2}$. Voluntary blood pressure measurements were also taken. The second of two readings was counted, unless there was a difference of $>10 \mathrm{mmHg}$ between the two readings in which case a third reading was taken [19]. Hypertension was defined by a systolic blood pressure of $>140 \mathrm{mmHg}$ or a diastolic blood pressure of $>90 \mathrm{mmHg}$ [21].
Respondents were asked whether they had arthritis, including osteoarthritis, rheumatoid arthritis, rheumatism, gout or other types of arthritis [19]. The presence of arthritis was defined if the condition had been diagnosed by a doctor or a nurse, or if it was a current or long-term condition [19]. Respondents were also asked if they had been diagnosed with any type of diabetes or high blood sugar by a doctor or a nurse and were included as having diabetes if they responded yes, except if they had gestational diabetes [19]. Similarly, respondents were asked whether they had ever been told by a doctor or nurse if they had high cholesterol [19].

## Statistical analysis

Study participants were categorised into three age groups: young (18-39 years old), middle-aged (4064 years old), and older (65 years old and above) adults. Descriptive statistics were used to describe the
population characteristics and distribution of arthritis and CVD risk factors. Logistic regression models were used to estimate the odds ratio (OR) with $95 \%$ confidence intervals (CI) for CVD risk factors in relation to the presence of arthritis. Population attributable fraction (PAF) was calculated to determine the proportion of CVD risk factors in the population that could be attributable to having arthritis using the "punafcc" command in Stata, which implements the method recommended by Greenland and Drescher [22]. $P$ values less than 0.05 were considered statistically significant. Analyses were adjusted for obesity $>30 \mathrm{~kg} / \mathrm{m}^{2}$. All analyses were performed using STATA 15.0 SE (StataCorp LP., College Station, TX, USA).

## Results

Our study included 13,776 participants. Arthritis was reported by $4.0 \%$ of young adults, $28.8 \%$ of middle-aged adults and $54.5 \%$ of older adults. Those with arthritis were at increased odds of obesity ( 2.07 fold in young, 1.75 fold in middle-aged and 1.89 fold in older adults), increased odds of diabetes ( 5.70 fold in young, 1.64 fold in middle-aged and 1.37 fold in older adults), increased odds of hypertension ( 2.72 fold in young, 1.78 fold in middle-aged and 1.48 fold in older adults) and an increased odds of dyslipidaemia (4.64 fold in young, 2.14 fold in middle-aged and 1.22 fold in older adults) compared to those without arthritis. This elevated odds remained significant even after adjusting for obesity, with the exception of diabetes in the older population.

## Young adults (18-39 years)

The prevalence, associations and PAF of CVD risk factors and arthritis in young adults are presented in Table 1. Of this population, $29.1 \%$ of those with arthritis were obese, compared to $16.5 \%$ of those without arthritis, indicating
a 2.07 fold higher prevalence ( $95 \%$ CI 1.36-3.16) of obesity in young adults with arthritis. The PAF, or the proportion of the young adult population with obesity that could be attributable to arthritis, was $15.0 \%$ ( $95 \%$ CI $4.1-24.7 \%)$. The prevalence of diabetes was $2.5 \%$ in young adult with arthritis; a 5.70 fold higher prevalence ( $95 \%$ CI 1.74-15.37) compared to those without arthritis, which remained significant after adjusting for obesity. The prevalence of hypertension and dyslipidaemia in young adult with arthritis was $8.9 \%$ for each, a 2.72 (95\% CI 1.534.84 ) and 4.64 fold ( $95 \%$ CI $2.56-8.39$ ) higher prevalence compared to those without arthritis, respectively. These results remained significant after adjusting for obesity. The PAF for hypertension and dyslipidemia in relation to arthritis were $5.6 \%$ ( $95 \%$ CI $0.9-10.2 \%$ ), and $7.0 \%$ ( $95 \%$ CI $2.3-11.5 \%$ ), respectively. In young adult with arthritis, $13.4 \%$ had at least one of hypertension, dyslipidaemia or diabetes compared to $5.2 \%$ of those without arthritis, a 2.82 fold ( $95 \%$ CI 1.74-4.56) higher prevalence and a PAF of $8.6 \%$ ( $95 \%$ CI $2.7-14.1 \%$ ). If obesity was included, $36.4 \%$ of this population with arthritis had at least one CVD risk factor (hypertension, dyslipidaemia, diabetes or obesity), compared to $20.6 \%$ of those without arthritis, a 2.28 fold ( $95 \%$ CI 1.53-3.40) higher prevalence and a PAF of $20.4 \%$ ( $95 \%$ CI 8.2-31.0\%).

## Middle-aged adults (40-64 years)

The prevalence, associations and PAF of CVD risk factors and arthritis in middle-aged adults are presented in Table 2. The prevalence of obesity was $38.3 \%$ in those with arthritis compared to $26.2 \%$ in those without, a 1.75 fold ( $95 \%$ CI 1.54-2.01) higher prevalence. The PAF of arthritis for obesity was $16.5 \%$ ( $95 \%$ CI $12.5-20.3 \%)$. The prevalence of diabetes was $9.2 \%$ in middle-aged adults with arthritis, representing a 1.64 fold (95\% CI 1.33-2.03) higher prevalence compared

Table 1 Cardiovascular risk factors in adults with/without arthritis aged 18-39 years in NHS 2017-18

|  | Total number |  | OR (95\% CI) |  | Population attributable fraction, \% (95\% CI) |
| :---: | :---: | :---: | :---: | :---: | :---: |
|  | No arthritis ( $\mathrm{n}=3773$ ) | Arthritis ( $\mathrm{n}=157$ ) | OR (95\% CI) | OR adjusted for obesity ( $95 \%$ CI) |  |
| Obesity ${ }^{\text {a }}$ | 473 (16.5\%) | 32 (29.1\%) | 2.07 (1.36-3.16) | - | 15.0 (4.1-24.7) |
| Diabetes | 19 (0.5\%) | 4 (2.5\%) | 5.70 (1.74-15.37) | 4.87 (1.34-17.69) | 2.1 (0.00-4.50) |
| Hypertension | 131 (3.5\%) | 14 (8.9\%) | 2.72 (1.53-4.84) | 2.35 (1.17-4.70) | 5.6 (0.9-10.2) |
| Dyslipidaemia | 78 (2.1\%) | 14 (8.9\%) | 4.64 (2.56-8.39) | 4.62 (2.34-9.14) | 7.0 (2.3-11.5) |
| 1 or more of hypertension/dyslipidaemia/ diabetes | 196 (5.2\%) | 21 (13.4\%) | 2.82 (1.74-4.56) | 2.78 (1.59-4.88) | 8.6 (2.7-14.1) |
| 1 or more of hypertension/dyslipidaemia/ diabetes/obesity ${ }^{\text {a }}$ | 613 (20.6\%) | 40 (36.4\%) | 2.28 (1.53-3.40) | - | 20.4 (8.2-31.0) |

Table 2 Cardiovascular risk factors in adults with/without arthritis aged 40-64 years in NHS 2017-18

|  | Total number |  | OR (95\% CI) |  | Population attributable fraction, \% (95\% CI) |
| :---: | :---: | :---: | :---: | :---: | :---: |
|  | No arthritis ( $\mathrm{n}=4055$ ) | Arthritis ( $\mathrm{n}=1638$ ) | OR (95\% CI) | OR adjusted for obesity ( $95 \% \mathrm{Cl}$ ) |  |
| Obesity ${ }^{\text {a }}$ | 868 (26.2\%) | 527 (38.3\%) | 1.75 (1.54-2.01) | - | 16.5 (12.5-20.3) |
| Diabetes | 236 (5.8\%) | 151 (9.2\%) | 1.64 (1.33-2.03) | 1.37 (1.08-1.73) | 3.6 (1.9-5.3) |
| Hypertension | 745 (18.4\%) | 496 (28.6\%) | 1.78 (1.60-2.04) | 1.59 (1.37-1.84) | 5.6 (0.8-10.2) |
| Dyslipidaemia | 469 (11.6\%) | 358 (21.9\%) | 2.14 (1.84-2.49) | 2.04 (1.73-2.41) | 11.6 (9.1-14.0) |
| 1 or more of hypertension/dyslipidaemia/ diabetes | 1101 (27.2\%) | 680 (41.5\%) | 1.90 (1.69-2.15) | 1.73 (1.51-1.98) | 19.7 (16.0-23.2) |
| 1 or more of hypertension/dyslipidaemia/ diabetes/obesity ${ }^{\text {a }}$ | 1421 (42.8\%) | 830 (60.4\%) | 2.03 (1.79-2.31) | - | 30.7 (25.5-35.5) |

to those without arthritis, remaining significant after adjusting for obesity. The PAF of arthritis for diabetes was $3.6 \%$ ( $95 \%$ CI 1.9-5.3\%). The prevalence of hypertension and dyslipidaemia in this population with arthritis was $28.6 \%$ and $21.9 \%$ respectively, with a 1.78 fold ( $95 \%$ CI $1.60-2.04$ ) and 2.14 fold ( $95 \%$ CI 1.84-2.49) higher prevalence compared to those without arthritis, remaining significant after adjusting for obesity. The PAF of arthritis was $5.6 \%$ (95\% CI $0.8-$ $10.2 \%$ ) for hypertension and $11.6 \%$ (95\% CI 9.1-14.0\%) for dyslipidaemia. Among middle-aged adults, 41.5\% of those with arthritis had at least one of hypertension, dyslipidaemia or diabetes, a 1.90 fold ( $95 \%$ CI 1.69-2.15) higher prevalence and a PAF of $19.7 \%$ ( $95 \%$ CI 16.0-23.2\%). If obesity was included, $60.4 \%$ of this population with arthritis had at least one of hypertension, diabetes, dyslipidaemia or obesity, a 2.03 fold (95\% CI 1.79-2.31) higher prevalence and a PAF of 30.7\% (95\% CI 25.5-35.5\%).

## Older adults (>65 years)

The prevalence, associations and PAF of CVD risk factors and arthritis in older adults are presented in Table 3. The prevalence of obesity was $32.9 \%$ among those with arthritis, a 1.89 fold ( $95 \%$ CI 1.62-2.21) higher prevalence compared to those without arthritis. The PAF was $15.5 \%$ ( $95 \%$ CI 12.0-18.9\%). The prevalence of diabetes in those with arthritis was $17.6 \%$, which represents a 1.37 fold ( $95 \%$ CI 1.15-1.62) increased prevalence compared to those without arthritis. The PAF of arthritis for diabetes was $4.7 \%$ ( $95 \%$ CI $2.2-7.2 \%$ ). The prevalence of hypertension and dyslipidaemia in this population with arthritis were $48.2 \%$ and $28.7 \%$ respectively, a 1.48 fold ( $95 \%$ CI 1.31-1.68) and 1.22 fold ( $95 \%$ CI $1.07-1.41$ ) increased prevalence respectively compared to those without arthritis, which remained significant when adjusted for obesity. The PAF was $15.7 \%$ ( $95 \%$ CI 11.0-20.1\%) for hypertension and $5.2 \%$ ( $95 \%$ CI 1.7-8.7\%) for dylipidemia. $61.7 \%$ of older adults with arthritis had at least one of hypertension, dyslipidaemia or diabetes, representing a 1.49 fold (95\% CI

Table 3 Cardiovascular risk factors in adults with/without arthritis aged $\geq 65$ years in NHS 2017-18

|  | Total number |  | OR (95\% CI) |  | Population attributable fraction,\% (95\% CI) |
| :---: | :---: | :---: | :---: | :---: | :---: |
|  | No arthritis ( $\mathrm{n}=1891$ ) | Arthritis ( $\mathrm{n}=2262$ ) | OR (95\% CI) | OR adjusted for obesity ( $95 \%$ CI) |  |
| Obesity ${ }^{\text {a }}$ | 326 (20.6\%) | 610 (32.9\%) | 1.89 (1.62-2.21) | - | 15.5 (12.0-18.9) |
| Diabetes | 256 (13.5\%) | 399 (17.6\%) | 1.37 (1.15-1.62) | 1.15 (0.95-1.39) | 4.7 (2.2-7.2) |
| Hypertension | 730 (38.6\%) | 1091 (48.2\%) | 1.48 (1.31-1.68) | 1.35 (1.18-1.55) | 15.7 (11.0-20.1) |
| Dyslipidaemia | 468 (24.8\%) | 649 (28.7\%) | 1.22 (1.07-1.41) | 1.19 (1.02-1.38) | 5.2 (1.7-8.7) |
| 1 or more of hypertension/dyslipidaemia/ diabetes | 984 (52.0\%) | 1396 (61.7\%) | 1.49 (1.31-1.68) | 1.37 (1.19-1.57) | 20.2 (14.4-25.6) |
| 1 or more of hypertension/dyslipidaemia/ diabetes/obesity ${ }^{\text {a }}$ | 948 (60.0\%) | 1303 (70.4\%) | 1.58 (1.38-1.83) | - | 26.0 (18.8-32.5) |

1.31-1.68) higher prevalence compared to those without arthritis and a PAF of 20.2\% (95\% CI 14.4-25.6\%). Of older adults with arthritis, $70.4 \%$ had at least one of hypertension, dyslipidaemia, diabetes or obesity, which equates to a 1.58 fold ( $95 \%$ CI 1.38-1.83) higher prevalence compared to those without arthritis and a PAF of 26.0\% (95\% CI 18.8-32.5\%).

## Discussion

This study showed high prevalences of treatable CVD risk factors in people with arthritis. The proportion of one or more CVD risk factor (hypertension, dyslipidaemia or diabetes) in the population that could be identified by targeting those with arthritis was $8.6 \%$ in young adults, $19.7 \%$ in middle-aged adults and $20.2 \%$ in older adults. These proportions were significantly higher if obesity was included, being $20.4 \%, 30.7 \%$, and $26.0 \%$, respectively.
The prevalences of arthritis across each age group is similar to those reported in other Australian literature [23, 24] and in other Western countries [25, 26]. Although the type of arthritis was not specified in our study, it is likely to vary across each age group. However, there is evidence that all the common forms of arthritis; osteoarthritis [27, 28], inflammatory arthritis [29-31] and crystal arthritis such as gout [32, 33], are associated with an increased risk of CVD, which is significantly attributed to the traditional risk factors such as hypertension and dyslipidaemia.
CVD remains the number one cause of death worldwide, with significant burden of disease due to untreated or undertreated risk factors [34]. In Australia, it was found that $68 \%$ of people with hypertension were either not treated or undertreated [35], contributing to $48 \%$ of CVD burden [36]. Approximately 80\% of Australians with dyslipidaemia are not on treatment [20], contributing to $21 \%$ of CVD burden [36] while obesity is estimated to contribute to approximately $30 \%$ of CVD burden [36]. Only 55\% of Australians with diabetes are well-controlled, attaining target haemoglobin A1c levels of less than $7.0 \%$ [35]. One barrier to management of CVD risk factors is that they are mostly asymptomatic and thus require specific targeting approaches. As seen in our study, it has been noted over a number of years that those with arthritis have an increased prevalence of CVD risk factors [28].
Arthritis, in contrast to CVD risk factors, causes pain and immobility and is a very common reason to seek health care [9]. Given that CVD risk factors are common in those with all forms of arthritis, and that those with arthritis are more likely contact with health professionals, this point of contact has the potential to be used as a teachable moment to signal the assessment of CVD risk. This may also be a strategy to aid in targeting higher
risk individuals who are less likely to seek medical attention for preventative (asymptomatic) care [37-41]. The potential to target the hidden burden of CVD risk would benefit those with arthritis. Further, given the high prevalence of arthritis this has the potential to impact on the overall burden of CVD. We estimate that targeting those with arthritis has the potential to identify $5.6 \%$ of hypertension in young and middle-aged adults and $15.7 \%$ in older adults in these populations. Similarly, by targeting those with arthritis, there is the potential to identify $7.0 \%$ of dyslipidemia in young adults, $11.6 \%$ in middle-aged adults and $5.2 \%$ in older adults in these populations.
The prevalence of arthritis is high in the community, and arthritis symptoms are a common reason to seek healthcare attention [9]. Finding people with arthritis and screening them for CVD presents an opportunity for improving community-based CVD prevention as cardiovascular risk factors are asymptomatic until CVD develops. The current study shows that there is the opportunity to target people for CVD prevention at their first point of contact for joint pain. This has the potential to increase awareness of cardiovascular prevention by targeting a population with a high prevalence of CVD risk factors. This approach in turn also has the potential of reducing joint pain since CVD and OA, the most common cause of joint pain in adults, share common risk factors and we have shown that targeting these through low-intensity lifestyle intervention improves joint pain [42].
The results of this study need to be considered in context of its limitations. While hypertension and obesity were mostly defined by measurements, the prevalence of dyslipidemia and diabetes was self-reported, which may have been subject to bias and resulted in underestimation. As an example, in the NHS 2017-18 it was estimated that nearly three quarters (73.7\%) of all adults with measured high blood pressure did not report having hypertension [43]. Our results may be limited by the use of dichotomous variables (i.e. the presence or absence) of conditions. There were no supporting clinical, biochemical or radiographic data which not only would confirm diagnoses, but could also be used to grade severity. There may be a stronger association between more severe CVD risk factors and arthritis, which would not be elucidated in our study. The type of arthritis was not specified in our data. While it may be possible to infer that in the younger population inflammatory arthritis may predominate, as with OA in the older population, the types of arthritis are separate entities and may cause an increased CVD risk via different pathophysiological processes. Since the association between inflammatory arthritis (eg. rheumatoid arthritis, gout) and CVD are increasingly
recognised, people with these conditions may have been more likely to be screened for CVD risk factors thereby possibly introducing bias. However, the association between OA and CVD risk factors is not well recognised among healthcare professionals and specific CVD screening is not recommended in this population in either CVD screening guidelines or in guidelines for the management of OA. As the prevalence of inflammatory arthritis is far less common than osteoarthritis in middle aged and older populations, it is unlikely that bias due to screening for CVD risk factors is the only explanation for our findings as arthritis was associated with elevated CVD risk irrespective of age category. Another limitation is that we did not have access to data pertaining to physical activity levels, which could act as a potential confounding factor in some of the relationships drawn. Finally, this is a cross-sectional study, thus a temporal relationship between arthritis and risk factors of CVD could not be established. This study can confirm that both arthritis and CVD coexists and if one of these health risk is present, people should be referred to test the other condition.

## Conclusions

Our study demonstrates that Australian adults of all ages with arthritis are at an increased odds of having CVD risk factors independent of obesity. Presentation to healthcare practitioners with symptoms due to arthritis could provide an opportunity to screen for individuals with asymptomatic CVD risk factors. By adopting an approach of managing arthritis and CVD risk factors in parallel, rather than in silos, there is the potential to improve outcomes in both conditions.

## Abbreviations

CVD: Cardiovascular disease; PAF: Population attributable fraction; OA: Osteoarthritis; NHS: National Health Survey; ABS: Australian Bureau of Statistics; BMI: Body mass index; OR: Odds ratio; CI: Confidence interval.

## Acknowledgements

Not applicable.

## Author contributions

SMH and FC were involved in the conception and design of this paper. SMH performed the statistical analysis. JS, SMH and FC interpreted the data. JS, SMH and FC were major contributors in writing the manuscript. YW, AEW, YZL, MJC and KS substantially involved in the revision of this work. All authors read and approved the final manuscript.

## Funding

SMH is the recipient of National Health and Medical Research Council (NHMRC) Early Career Fellowship (\#1142198). YW is the recipient of NHMRC Translating Research into Practice Fellowship (APP1168185). AEW is the recipient of the RACP Fellows Career Development Fellowship. YZL is the recipient of NHMRC Clinical Postgraduate Scholarship (\#1133903) and Royal Australasian College of Physicians Woolcock Scholarship. FMC is the recipient of NHMRC Investigator Grant (APP1 194829). The funding sources had no role
in the design, conduct, or reporting of the study or the decision to submit the manuscript for publication.

## Availability of data and materials

The datasets used and/or analysed during the current study are available from the corresponding author on reasonable request.

## Declarations

Ethics approval and consent to participate
Not applicable.

## Consent for publication

Not applicable.

## Competing interests

The authors declare they have no competing interests.

## Author details

${ }^{1}$ Department of Epidemiology and Preventive Medicine, School of Public Health and Preventive Medicine, Monash University, 553 St Kilda Road, Melbourne, VIC 3004, Australia. ${ }^{2}$ Pre-Clinical Disease and Prevention, Baker Heart and Diabetes Institute, 75 Commercial Rd, Melbourne, VIC 3004, Australia. ${ }^{3}$ Clinical Obesity, Nutrition and Adipose Biology Laboratory, Healthy Ageing, Garvan Institute of Medical Research, 384 Victoria St, Darlinghurst, NSW 2010, Australia. ${ }^{4}$ Department of Endocrinology, St Vincent's Hospital, Victoria St, Darlinghurst, NSW 2010, Australia. ${ }^{5}$ St Vincent's Clinnical School, University of New South Wales Sydney, Victoria St, Darlinghurst, NSW 2010, Australia.

Received: 11 August 2021 Accepted: 13 May 2022
Published online: 19 May 2022

## References

1. World Health Organization: Cardiovascular diseases (CVDs). Geneva; 2021.
2. Roth GA, Mensah GA, Johnson CO, Addolorato G, Ammirati E, Baddour LM, Barengo NC, Beaton AZ, Benjamin EJ, Benziger CP, et al. Global burden of cardiovascular diseases and risk factors, 1990-2019: update from the GBD 2019 study. J Am Coll Cardiol. 2020;76:2982-3021.
3. Petrie JR, Guzik TJ, Touyz RM. Diabetes, hypertension, and cardiovascular disease: clinical insights and vascular mechanisms. Can J Cardiol. 2018;34:575-84.
4. World Health Organization. Prevention of cardiovascular disease: guidelines for assessment and management of total cardiovascular risk. Geneva; 2007.
5. The Royal Australian College of General Practitioners. Guidelines for preventative activities in general practice, 9th edn. East Melbourne, VIC; 2016.
6. Ministry of Health. Cardiovascular disease risk assessment and management for primary care. Wellington, New Zealand; 2018.
7. National Vascular Disease Prevention Alliance. Guidelines for the management of absolute cardiovascular disease risk; 2012.
8. Greaves K, Smith A, Agostino J, Kunarajah K, Stanton T, Korda R. Crosssectional survey describing general practitioners' absolute cardiovascular disease risk assessment practices and their relationship to knowledge, attitudes and beliefs about cardiovascular disease risk in Queensland, Australia. BMJ Open. 2020;10: e033859.
9. St Sauver JL, Warner DO, Yawn BP, Jacobson DJ, McGree ME, Pankratz JJ, Melton LJ 3rd, Roger VL, Ebbert JO, Rocca WA. Why patients visit their doctors: assessing the most prevalent conditions in a defined American population. Mayo Clin Proc. 2013;88:56-67.
10. Fernandes GS, Valdes AM. Cardiovascular disease and osteoarthritis: common pathways and patient outcomes. Eur J Clin Investig. 2015;45:405-14.
11. Singh JA. When gout goes to the heart: does gout equal a cardiovascular disease risk factor? Ann Rheum Dis. 2015;74:631-4.
12. England BR, Thiele GM, Anderson DR, Mikuls TR. Increased cardiovascular risk in rheumatoid arthritis: mechanisms and implications. BMJ. 2018;361: k1036.
13. Turkiewicz A, Kiadaliri AA, Englund M. Cause-specific mortality in osteoarthritis of peripheral joints. Osteoarthr Cartil. 2019;27:848-54.
14. Hansildaar R, Vedder D, Baniaamam M, Tausche A-K, Gerritsen M, Nurmohamed MT. Cardiovascular risk in inflammatory arthritis: rheumatoid arthritis and gout. Lancet. 2021;3:E58-70.
15. Solomon DH, Goodson NJ, Katz JN, Weinblatt ME, Avorn J, Setoguchi S, Canning C, Schneeweiss S. Patterns of cardiovascular risk in rheumatoid arthritis. Ann Rheum Dis. 2006;65:1608-12.
16. Baene-Díez JM, Garcia GM, Comas-Cufí M, Ramos R, Prieto-Alhambra D, Salvator-González B, Elosua R, Dégano IR, Peñafiel J, Grau M. Association between chronic immune-mediated inflammatory diseases and cardiovascular risk. Heart. 2018;104:119-26.
17. Rahimi-Sakak F, Maroofi M, Rahmani J, Bellissimo N, Hekmatdoost A Serum uric acid and risk of cardiovascular mortality: a systematic review and dose-response meta-analysis of cohort studies of over a million participants. BMC Cardiovasc Disord. 2019;19:218.
18. Wang H, Bai J, He B, Hu X, Liu D. Osteoarthritis and the risk of cardiovascular disease: a meta-analysis of observational studies. Sci Rep. 2016;6:39672-39672.
19. Australian Bureau of Statistics. National Health Survey: users' guide, 2017-18. Canberra, ACT; 2019.
20. Australian Bureau of Statistics. National Health Survey: first results meth odology. Canberra, ACT; 2018.
21. Williams B, Mancia G, Spiering W, Agabiti Rosei E, Azizi M, Burnier M, Clement D, Coca A, De Simone G, Dominiczak A, et al. 2018 Practice guidelines for the management of arterial hypertension of the european society of hypertension and the European Society of Cardiology: ESH/ ESC task force for the management of arterial hypertension. J Hypertens. 2018;36:2284-309.
22. Greenland S, Drescher K. Maximum likelihood estimation of the attributable fraction from logistic models. Biometrics. 1993;49:865-72.
23. March LM, Bagga H. Epidemiology of osteoarthritis in Australia. Med J Aust. 2004;180:S6-10
24. Ackerman IN, Pratt C, Gorelik A, Liew D. Projected burden of osteoarthritis and rheumatoid arthritis in Australia: a population-level analysis. Arthritis Care Res (Hoboken). 2018;70:877-83.
25. Hootman JM, Helmick CG. Projections of US prevalence of arthritis and associated activity limitations. Arthritis Rheum. 2006;54:226-9.
26. Barbour KE, Helmick CG, Boring M, Brady TJ. Vital signs: prevalence of doctor-diagnosed arthritis and arthritis-attributable activity limitationUnited States, 2013-2015. MMWR Morb Mortal Wkly Rep. 2017;66:246-53
27. Singh G, Miller JD, Lee FH, Pettitt D, Russell MW. Prevalence of cardiovascular disease risk factors among US adults with self-reported osteoarthritis: data from the Third National Health and Nutrition Examination Survey. Am J Manag Care. 2002;8:S383-391.
28. Puenpatom RA, Victor TW. Increased prevalence of metabolic syndrome in individuals with osteoarthritis: an analysis of NHANES III data. Postgrad Med. 2009;121:9-20.
29. Radner H, Lesperance T, Accortt NA, Solomon DH. Incidence and prevalence of cardiovascular risk factors among patients with rheumatoid arthritis, psoriasis, or psoriatic arthritis. Arthritis Care Res (Hoboken). 2017;69:1510-8.
30. Khraishi M, Aslanov R, Rampakakis E, Pollock C, Sampalis JS. Prevalence of cardiovascular risk factors in patients with psoriatic arthritis. Clin Rheumatol. 2014;33:1495-500.
31. del Rincón ID, Williams K, Stern MP, Freeman GL, Escalante A. High incidence of cardiovascular events in a rheumatoid arthritis cohort not explained by traditional cardiac risk factors. Arthritis Rheum. 2001;44:2737-45.
32. Krishnan E, Baker JF, Furst DE, Schumacher HR. Gout and the risk of acute myocardial infarction. Arthritis Rheum. 2006;54:2688-96.
33. Kuo CF, See LC, Luo SF, Ko YS, Lin YS, Hwang JS, Lin CM, Chen HW, Yu KH. Gout: an independent risk factor for all-cause and cardiovascular mortality. Rheumatology (Oxford). 2010;49:141-6.
34. Heeley EL, Peiris DP, Patel AA, Cass A, Weekes A, Morgan C, Anderson CS, Chalmers JP. Cardiovascular risk perception and evidence—practice gaps in Australian general practice (the AusHEART study). Med J Aust. 2010;192:254-9.
35. Australian Bureau of Statistics. Australian Health Survey: first results, 2011-12. Canberra, ACT; 2012.
36. Global Burden of Disease Data Visualizations. http://www.healthdata.org/ data-visualization/gbd-compare.
37. Gordon J, Valenti L, Bayram C, Miller GC. An analysis of general practice encounters by socioeconomic disadvantage. Aust Fam Physician. 2016;45:702-5.
38. Australian Institute of Health and Welfare. Rural and remote health. Canberra, ACT; 2019.
39. Australian Institute of Health and Welfare. The health of Australia's males. Canberra, ACT; 2019.
40. Schultz WM, Kelli HM, Lisko JC, Varghese T, Shen J, Sandesara P, Quyyumi AA, Taylor HA, Gulati M, Harold JG, et al. Socioeconomic status and cardiovascular outcomes. Circulation. 2018;137:2166-78
41. Alston L, Allender S, Peterson K, Jacobs J, Nichols M. Rural inequalities in the Australian burden of ischaemic heart disease: a systematic review. Heart Lung Circ. 2017;26:122-33.
42. Wang Y, Lombard C, Hussain SM, Harrison C, Kozica S, Brady SRE, Teede H, Cicuttini FM. Effect of a low-intensity, self-management lifestyle intervention on knee pain in community-based young to middle-aged rural women: a cluster randomised controlled trial. Arthritis Res Ther. 2018;20:74.
43. Australian Bureau of Statistics. Hypertension and measured high blood pressure. Canberra, ACT; 2018.

## Publisher's Note

Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.

## Ready to submit your research? Choose BMC and benefit from:

- fast, convenient online submission
- thorough peer review by experienced researchers in your field
- rapid publication on acceptance
- support for research data, including large and complex data types
- gold Open Access which fosters wider collaboration and increased citations
- maximum visibility for your research: over 100M website views per year

At BMC, research is always in progress.

Learn more biomedcentral.com/submissions
BMC


[^0]:    ${ }^{\dagger}$ Julia Sewell and Sultana Monira Hussain are joint first authors.
    *Correspondence: flavia.cicuttini@monash.edu
    ${ }^{1}$ Department of Epidemiology and Preventive Medicine, School of Public Health and Preventive Medicine, Monash University, 553 St Kilda Road, Melbourne, VIC 3004, Australia
    Full list of author information is available at the end of the article

