Life Expectancy

1-Associations between biological ageing and the risk of, genetic susceptibility to, and life expectancy associated with rheumatoid arthritis: a secondary analysis of two observational studies

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Abstract

Background Rheumatoid arthritis is a chronic autoimmune disorder that affects life expectancy. Accelerated biological ageing is thought to be a major risk factor for age-related diseases, but its role in rheumatoid arthritis remains uncertain. We aimed to assess the associations between biological ageing and risk of rheumatoid arthritis and genetic susceptibility to the disease. We also aimed to assess the effect of biological ageing on the life expectancy of people with rheumatoid arthritis. Methods We calculated the chronological age-adjusted biological age-by both the Klemera-Doubal method (KDMAge) and phenotypic age (PhenoAge)-as a surrogate measure for biological ageing in participants from the US National Health and Nutrition Examination Survey (NHANES) and UK Biobank study. KDMAge or PhenoAge acceleration was defined as the residual of the regression of KDMAge or PhenoAge based on chronological age.

Participants with accelerated biological ageing had KDMAge or PhenoAge acceleration values greater than 0, whereas those without accelerated ageing had values less than or equal to 0. We did cross-sectional analyses to assess the association between biological ageing and prevalent rheumatoid arthritis in both cohorts and prospective analyses to assess the association between biological ageing and incident rheumatoid arthritis in the UK Biobank. Logistic regression and Cox proportional hazards models were used to analyse these associations. Polygenic risk scores were used to establish genetic susceptibility to rheumatoid arthritis and to analyse the interaction between biological ageing and genetic risk. We also assessed the association between life expectancy and biological ageing status in people with rheumatoid arthritis. Findings In the cross-sectional analyses, each 1-year increase in age-adjusted biological age was associated with an increase in the risk of rheumatoid arthritis of between 1% and 10%.

In the NHANES, individuals with accelerated ageing had a higher risk of rheumatoid arthritis than non-accelerated ageing individuals, with odds ratios of 1 center dot 21 (95% CI 1 center dot 03-1 center dot 42; p=0 center dot 018) for KDMAge acceleration and 1 center dot 46 (1 center dot 26-1 center dot 69; p<0 center dot 0001) for PhenoAge acceleration. Similarly, in the UK Biobank, the risk of rheumatoid arthritis was increased in individuals with accelerated ageing compared with individuals with no accelerated ageing (KDMAge odds ratio 1 center dot 96 [95% CI 1 center dot 71-2 center dot 24]; PhenoAge 2 center dot 71 [2 center dot 51-2 center dot 92]). In the prospective analyses of the UK Biobank population, accelerated biological ageing was associated with an increased risk of incident rheumatoid arthritis as measured by both KDMAge (hazard ratio 1 center dot 27 [95% CI 1 center dot 03-1 center dot 55]) and PhenoAge (1 center dot 70 [1 center dot 52-1 center dot 92]).

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Among participants with high genetic predisposition to rheumatoid arthritis, accelerated biological ageing was associated with an increased risk of incident disease, and we noted significant additive interactions between accelerated biological ageing and genetic risk. At age 45 years, people with rheumatoid arthritis had reduced life expectancy compared with those without rheumatoid arthritis. Among people with rheumatoid arthritis, accelerated biological ageing was associated with reduced life expectancy compared with not having accelerated biological ageing. Interpretation Accelerated biological ageing could increase the risk of rheumatoid arthritis, especially among people with high genetic risk, and could reduce the life expectancy of people with rheumatoid arthritis. The identification of populations with accelerated biological ageing has important implications for reducing the risk of rheumatoid arthritis and of lowered life expectancy. Copyright (c) 2023 The Author(s). Published by Elsevier Ltd. This is an Open Access article under the CC BY-NC-ND 4.0 license.

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