

BIOLOGICAL AGEING ACCELERATION, GENETIC SUSCEPTIBILITY AND RISK OF CHRONIC LIVER DISEASES: TWO OBSERVATIONAL COHORT STUDIES

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Poster

Hepatobiliary

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Accelerated biological ageing, measured by deviation between phenotypic age and chronological age, was independently associated with increased risk of alcoholic liver disease, metabolic dysfunction-associated steatotic liver disease, and liver fibrosis/cirrhosis in two large cohort studies.

KEY POINTS

- In the UK Biobank cohort, accelerated PhenoAge was associated with more than doubled risk of alcoholic liver disease (HR 2.29, 95% CI 2.03-2.58) and liver fibrosis/cirrhosis (HR 2.22, 95% CI 2.01-2.46), and modestly increased risk of metabolic dysfunction-associated steatotic liver disease (HR 1.10, 95% CI 1.04-1.16).
- Similar associations were observed in the All of Us cohort, with accelerated PhenoAge linked to alcoholic liver disease (HR 1.97, 95% CI 1.00-3.88), metabolic dysfunction-associated steatotic liver disease (HR 1.35, 95% CI 1.15-1.58), and liver fibrosis/cirrhosis (HR 2.19, 95% CI 1.48-3.22).
- The association between accelerated biological ageing and chronic liver diseases remained significant across polygenic risk score levels and was independent of genetic risk.
- Participants with both high genetic risk and accelerated biological ageing exhibited the highest risk of developing chronic liver diseases compared to those with biologically younger ages and low genetic risk.
- This research may be most relevant to clinicians and researchers interested in risk stratification for chronic liver disease using integrated biological ageing metrics alongside genetic susceptibility markers.

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