

ACCELERATED BIOLOGICAL AGEING AMPLIFIES THE ASSOCIATION BETWEEN DIETARY ADVANCED GLYCATION END-PRODUCTS AND HEMATOLOGIC MALIGNANCY PREVELANCE: A US POPULATION BASED STUDY

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BACKGROUND

- Advanced Glycation end-products (AGEs) are pro oxidative compounds implicated in chronic inflammation, immune dysfunction, and cellular senescence.
- Biological ageing metrics such as Levine Phenotypic Age capture cumulative physiologic deterioration beyond chronological ageing and may better reflect cancer susceptibility.
- However, the interplay between dietary AGE exposure and biological age acceleration in hematologic malignancies has not been characterised.
- We evaluated whether dietary AGEs and accelerated phenotypic ageing independently and synergistically associate with hematologic malignancy prevalence in U.S. adults.

CONTACT

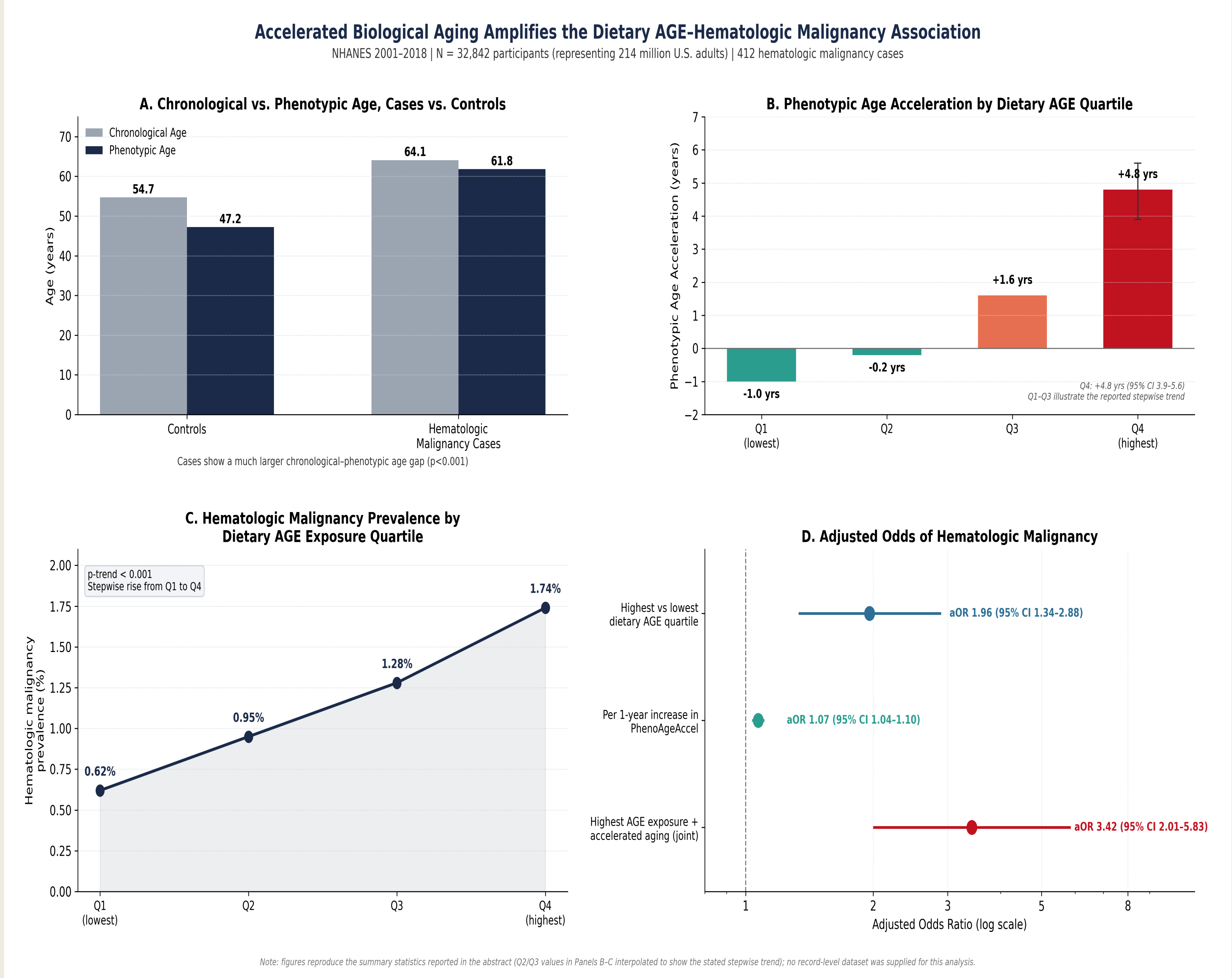
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METHODS

- We performed a cross-sectional analysis of adults aged ≥20 yrs from NHANES 2001–2018.
- Dietary advanced glycation end-product exposure was quantified by mapping NHANES dietary recall data to experimentally validated food AGE databases
- Biological ageing assessed using Levine Phenotypic Age and phenotypic ageing acceleration (PhenoAgeAccel).
- Hematologic malignancy prevalence included self-reported leukaemia, lymphoma, or multiple myeloma.
- Survey-weighted logistic regression models adjusted for demographic, socioeconomic, metabolic, and lifestyle variables were used to evaluate associations.

RESULTS

- The study included 32,842 participants representing 214 million U.S. adults, among whom 412 reported hematologic malignancies.
- Participants with hematologic malignancies demonstrated substantially older biological age profiles than controls (mean Phenotypic Age 61.8 vs 47.2 years; $p<0.001$), whereas chronological age differences were proportionally smaller (64.1 vs 54.7 years).
- Mean phenotypic age acceleration increased progressively across dietary AGE quartiles, reaching +4.8 years (95% CI 3.9–5.6) in the highest exposure group.
- Hematologic malignancy prevalence rose stepwise from 0.62% in the lowest dietary AGE quartile to 1.74% in the highest ($p\text{-trend}<0.001$).
- After full adjustment, individuals in the highest dietary AGE quartile exhibited nearly twofold higher odds of hematologic malignancy prevalence compared with the lowest quartile (adjusted OR 1.96, 95% CI 1.34–2.88).
- Each additional year of phenotypic age acceleration was independently associated with a 7% increase in hematologic malignancy odds (aOR 1.07, 95% CI 1.04–1.10).
- Participants exhibiting both elevated dietary AGE exposure and accelerated biological ageing had the highest observed prevalence burden (aOR 3.42, 95% CI 2.01-5.83).



CONCLUSION

Dietary glycotoxicity and accelerated biological ageing demonstrate convergent associations with hematologic malignancy prevalence at the population level. These findings support a potential mechanistic link between metabolic ageing, chronic inflammatory stress, and hematologic carcinogenesis, while highlighting biological ageing markers as emerging tools for precision risk stratification in hematologic oncology