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Invitation to review a manuscript for Clinical Epigenetics - CLEP-D-15-00189

From: **Clinical Epigenetics Editorial Office** (em@editorialmanager.com)
Sent: Thu 11/19/15 3:02 AM
To: Shicheng Guo (shicheng.guo@hotmail.com)

CLEP-D-15-00189
Methylation, telomeres, and frailty: an epidemiological study on the interplay of epigenetic, genomic and clinical correlates of age
Lutz P Breitling; Kai-Uwe Saum; Laura Perna; Ben Schöttker; Bernd Holleczek; Hermann Brenner
Clinical Epigenetics

Dear Dr. Guo,

I would like to invite you to review the manuscript above which has been submitted to Clinical Epigenetics. Further details including the full abstract can be found at the end of this email.

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Thank you for your time, and I look forward to hearing from you.

Best wishes,

Ahihiro Umezawa
Clinical Epigenetics
http://www.clinicalepigeneticsjournal.com/

CLEP-D-15-00189
Research
Methylation, telomeres, and frailty: an epidemiological study on the interplay of epigenetic, genomic and clinical correlates of age
Clinical Epigenetics

Abstract: Background. The epigenetic clock, in particular epigenetic pre-aging quantified by the so-called DNA methylation age acceleration, has recently been suggested to closely correlate with a variety of disease phenotypes. There remains a dearth of data, however, on its association with telomere length and frailty, which can be considered major correlates of age on the genomic and clinical level, respectively.
Results. In this cross-sectional observational study on altogether 1820 subjects from the elderly general population in Germany, no correlation of epigenetic age acceleration with telomere length was found. However, there was an association of DNA methylation age acceleration with a comprehensive frailty measure, such that the accumulated deficits significantly increased with increasing age acceleration. This association was independent from a variety of confounding variables considered.
Conclusions. The results of the present study suggest that epigenetic age acceleration is correlated with clinically relevant aging-related phenotypes through pathways unrelated to genomic age as assessed by telomere length. Innovative approaches like Mendelian randomization will be needed to elucidate whether epigenetic age acceleration indeed plays a causal role for the development of clinical phenotypes.