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Size matters! Peripheral blood leukocyte telomere length and survival after critical illness

Florian B. Mayr [1,2] and Sachin Yende[1,2]

Affiliations: [1]VA Pittsburgh Healthcare System, Pittsburgh, PA, USA. [2]CRISMA Center, Dept of Critical Care Medicine, University of Pittsburgh School of Medicine, Pittsburgh, PA, USA.

Correspondence: Florian B. Mayr, VA Pittsburgh Healthcare System, University Drive C, Building 1, Rm 5A112, Pittsburgh, PA 15240, USA. E-mail: florian@pitt.edu

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The association of shorter leukocyte telomere length and reduced survival in critical illness may prove crucial in our quest to improve outcomes http://bit.ly/2qDAZSq

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In 2009, Elizabeth Blackburn, Jack Szostak and Carol Greider were awarded the Nobel Prize in Physiology or Medicine for their pioneering work that led to the discovery of telomeres and the enzyme complex telomerase responsible for maintaining its structure [1]. Over the past four decades, the classic view of telomeres protecting the natural ends of linear chromosomes and telomerase as telomere-terminal transferase necessary for the replication of chromosome ends has significantly evolved. Many diverse fields have matured, including the discovery of key molecular components of telomerase, implications for limits to cellular replication, and identification and characterisation of human genetic disorders that result in premature telomere shortening [2]. For example, the short telomere syndromes are a group of genetic disorders that are caused by mutations in components of the telomerase enzyme and other telomere maintenance genes [3]. The most common associated pathologies involve respiratory disorders such as idiopathic pulmonary fibrosis (IPF) and related interstitial lung disorders and severe emphysema, alone or combined with fibrosis [4–6]. Mutations in the telomerase enzyme genes are the most common cause of IPF, and the frequency of these mutations in severe, early-onset emphysema rivals the prevalence of α_1-antitrypsin deficiency [4], making short telomere syndromes the most-common premature ageing disorders, with ∼10 000 affected individuals in the USA alone [3].