An estimated 149.2 million children under the age of 5 were physically stunted in 2020, defined as falling at least –2 standard deviations below the height-for-age World Health Organization (WHO) Child Growth Standards median. Stunting is a visible indicator of a deficient environment, the consequences of which include child morbidity and mortality, reduced and delayed neurocognitive development, and an increased risk of long-term chronic diseases. The underlying aetiology and pathophysiological mechanisms leading to stunting remain elusive, and therefore few effective treatment and prevention strategies exist. Here, based on available studies, we present potential mechanistic pathways by which parasitic infection of mother and/or infant may lead to childhood stunting.

The most well-recognised pathway to stunting is a 'vicious cycle' between deteriorating nutritional status and infection, which is evolving to encompass dysbiosis of the gut, local and systemic inflammation alongside energetic, hormonal, and metabolic consequences. Anaemia, which is often presented as co-occurring alongside stunting, may in fact be contributing. The bidirectional relationship between intestinal parasites and the microbiota in early life, and their combined effects, may also play a key role in stunting. And finally, epigenetic regulation of gene expression may link parasitic infections and poor gut health in early life to stunting.

We highlight the need for future multidisciplinary longitudinal studies and clinical trials aimed at elucidating the most influential factors, and synergies therein, that can lead to stunting, and ultimately towards finding solutions to successfully mitigate against it.