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Title:

Short-Chain Fatty Acids and Sleep Disturbances in Pediatric ADHD: Microbiome–Brain Axis Links and Dietary Modulation Potential

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Preparation of Your Abstract

1. The title should be as brief as possible but long enough to indicate clearly the nature of the study. Capitalise the first letter of the first word ONLY (place names excluded). No full stop at the end.
2. Abstracts should state briefly and clearly the purpose, methods, results and conclusions of the work.
Introduction: Clearly state the purpose of the abstract
Methods: Describe your selection of observations or experimental subjects clearly
Results: Present your results in a logical sequence
Discussion: Emphasize new and important aspects of the study and conclusions that are drawn from them

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Introduction: Sleep disturbances are common in children with attention-deficit/hyperactivity disorder (ADHD) and may exacerbate symptom severity. Emerging evidence highlights the gut–brain axis as a key pathway linking gut microbiota composition, microbial metabolites, and neurodevelopmental outcomes. Short-chain fatty acids (SCFAs), produced through dietary fibre fermentation, are central to this interaction. However, their relationship with sleep in paediatric ADHD remains poorly defined. This study investigated associations between faecal SCFA concentrations and sleep disturbances in children with ADHD, and explored differences between those treated and untreated with methylphenidate.
Methods: A cross-sectional study was conducted in 25 children with a clinical diagnosis of ADHD. Sleep disturbances were assessed using the Children's Sleep Habits Questionnaire (CSHQ). Faecal SCFA concentrations (acetate, propionate, butyrate, iso-butyrate, valerate, iso-valerate) were quantified using gas chromatography–mass spectrometry. Partial correlation and multiple linear regression analyses were applied, controlling for age and sex.
Results: Clinically significant sleep disturbances (CSHQ > 41) were observed in 92% of participants. Unmedicated children displayed higher bedtime resistance scores and greater concentrations of most SCFAs compared with medicated peers. Acetate was positively correlated with sleep duration problems, while iso-butyrate, iso-valerate, and valerate were negatively correlated with bedtime resistance. Multiple linear regression identified acetate and propionate as significant predictors of sleep disturbances.
Discussion: Distinct SCFA profiles were observed between medicated and unmedicated ADHD children, with acetate and propionate emerging as potential biomarkers for sleep problems. These findings support a mechanistic role of diet-derived SCFAs in modulating sleep via the microbiome–brain axis and suggest that targeted dietary interventions to optimise SCFA production could complement ADHD management. Given that SCFA profiles are strongly influenced by dietary fibre type and food matrix structure, nutritional strategies to modulate gut microbial fermentation may represent a feasible pathway to improve sleep outcomes in paediatric ADHD.