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Letter to the Editor

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Dear Editor,

In a recent article by Ospina et al., the authors explore interesting associations between adverse childhood experiences (ACE) and development of asthma reported in adulthood.¹ The study replicates findings from a vast evidence base: an authoritative meta-analysis that included data from more than 250,000 individuals established that ACE were associated with detrimental outcomes of somatic illness across virtually all organ-systems, including the respiratory system.² The strong association between ACE and respiratory disease is consistent between the current study and the meta-analysis; however, the etiology behind the association is multifaceted and largely unknown. The authors suggest several biological explanations for this association, including stress-mediated imbalances in the hypothalamic–pituitary–adrenal axis, increased levels of inflammatory markers associated with asthma as well as immune dysregulation. However, the risk of misclassification of asthma is imminent when using a self-reported measure as the primary outcome, since objective measures are central to the diagnosis.³ Hence, other important possible explanatory mechanisms behind the association should be considered.

In this context, dysfunctional breathing (DB), also commonly referred to as hyperventilation syndrome, is a relevant topic for discussion. DB is defined as changes in breathing patterns without an underlying physiological demand and can cause shortness of breath and dizziness, regardless of the presence of respiratory or cardiac diseases.⁴ DB is considered to be a respiratory disorder that can have both physical and mental etiology. To date, no golden standard exist in the diagnosis of DB and the most common method to diagnose DB is the Nijmegen Questionnaire.⁵ The prevalence of DB is 6%–10% in the general population,⁶ whereas the prevalence of DB is significantly higher among patients with asthma in primary care settings (29%).⁷ DB is associated with panic and anxiety-related disorders.⁸ Anxiety and depression often co-occur with asthma and may influence the level of asthma control.⁹ Thus, evidence exists to suggest a complex interplay between anxiety disorders and respiratory problems. The risk of misclassification and misinterpretation arises when the symptoms of DB are attributed to asthma with no objective confirmation. This is of particular importance when investigating effects of ACE, as the findings may largely reflect underlying functional somatic illness or anxiety.

In summary, the study by Ospina et al. has several shortcomings which they themselves acknowledge: a) the exposure variable was based on retrospective reports, b) the outcome variable of asthma was self-reported, and c) no information about a wide array of possible confounding factors are available. Consequently, can this study possibly provide any new answers to an already well-established association?² Although the authors applied latent class analyses to identify patterns of ACE,¹ applying a relatively complex statistical model does not solve the problem that the data behind the current study are sparse and inadequate.¹⁰ Instead, in order to progress the field and get any closer to solving this problem, it is crucial that resources are directed toward the strenuous process of gathering comprehensive data and not to conduct futile secondary analyses of poor-quality data.

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