

Correspondence

Reply

To the Editor:

We thank Lukkarinen and Jartti¹ for their interest in our study. We completely agree with them that early respiratory infections preceding asthma are probably markers of the underlying susceptibility to asthma. Indeed, it is our main hypothesis that the association between early respiratory infections and later asthma is mainly due to an underlying host factor. Children who are susceptible to asthma have a tendency to early respiratory episodes, and virus infections will therefore often be documented, particularly for the most common viruses, such as rhinoviruses (RVs) and respiratory syncytial virus (RSV).

Lukkarinen and Jartti may be missing this important key point of our study.² They focus entirely on RV infections, but our main point is that the association between early respiratory infections and later asthma is not restricted to this specific virus type but rather is a common phenomenon for all respiratory viruses. We did find an association between RV infections in the first 3 years of life and later asthma (odds ratio, 2.58; 95% CI, 1.36–4.89), but this association was similar for all other viruses. Moreover, in analyses adjusted for the total number of episodes, there was no species-specific effect, in particular no effect from RV. This strongly suggests that the respiratory episode, that is, the host factor rather than the specific trigger, is the important marker of asthma susceptibility. Most previous studies focused only on a specific virus (mainly RSV or RV) and did not adjust for other infections. In the Childhood Origins of ASThma (COAST) study, it was found that RV infection was a stronger risk factor than RSV infections,³ but both RV and RSV infections were associated with asthma and the limited sample size opens the possibility that the very high risk estimate for RV was a spurious finding as reflected in the very wide CIs. The other cohort study⁴ referenced by Lukkarinen and Jartti also found that RSV infections were associated with later asthma but did not adjust for other viruses either.

Studies focusing on specific viral triggers obviously make sense in order to prevent and treat specific infections, which is a completely different question not covered by our study. Vaccination can prevent specific infections, and it is also possible that specific genetic variants affect susceptibility to specific infections. For example, it has been suggested that genetic variation in the 17q21 locus might be associated with susceptibility to RV infections,⁵ and a recently discovered susceptibility gene, *CDHR3*,⁶ might function as a receptor for RV species C⁷ and thereby specifically confer susceptibility to RV species C infections.

In conclusion, our study suggests that no specific viral infection is more “dangerous” than others and that studies of the relationship between early infections and asthma, such as those suggested by Lukkarinen and Jartti, should not be restricted to RV infections but include all respiratory virus as well as bacteria.

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