We thank Drs. Aligne (1) and Grant (2) for their stimulating comments concerning causal inference in the association between asthma and caries risk reported in our systematic review and meta-analysis (3).

Dr. Aligne suggests that secondhand exposure to tobacco smoke could represent a common environmental determinant predisposing people to both asthma and caries (1), following our original idea that one potential pathway is a common genetic or environmental determinant for both of these conditions (3). There is substantial evidence that the risk of asthma is causally related to secondhand smoke (SHS) exposure in both children (4) and adults (5). Dr. Aligne points out that there is also some evidence from a cross-sectional study (6) of an association between SHS and caries, which is consistent with the hypothesis that SHS causes caries. This finding as such is not sufficient to confirm a causal link, but it raises an important hypothesis. If we accept this hypothesis,
SHS could be a confounder that could explain at least part of the observed association between asthma and caries.

Figure 1 shows 3 alternative causal models for elaborating a role of SHS in causal inference concerning asthma and caries. Given the existing epidemiologic evidence and biologic plausibility (7), it is reasonable to assume that SHS truly causes asthma. In model A, SHS causes asthma—that is, SHS increases the risk of asthma and asthma increases the risk of caries independently of SHS. This would result in an association between SHS and caries even if SHS were not a causal determinant of caries. In model B, SHS causes dental caries in addition to asthma and could therefore confound the relation by either enhancing or masking the relation between asthma and caries. In this situation, adjustment for SHS would correct the association between asthma and caries. In model C, SHS causes asthma and asthma increases the risk of caries, but in addition there is another factor X which is both empirically associated with SHS and a causal determinant of caries (for example, some dietary factors). In this situation, adjustment for SHS could bias the relation between asthma and caries, resulting in either underestimation or overestimation of the true relation.

Dr. Grant presents evidence that vitamin D deficiency increases the risks of both asthma and dental caries and could therefore explain the observed association between asthma and dental caries (2), which again would be compatible with our suggestion of a common determinant predisposing people to both conditions (3). This is a plausible explanation in settings where vitamin D deficiency is common, and it would correspond with causal model B, where SHS is replaced with vitamin D. However, our meta-analyses included studies conducted worldwide, including geographic areas with an abundance of ultraviolet B irradiance and a low probability of vitamin D deficiency, thus excluding vitamin D as the only explanation for the observed relation between asthma and caries. Vitamin D could serve both as a confounder and an effect modifier of this relation. To elaborate on the role of vitamin D, we would like to either 1) study the association between asthma and caries in environmental and dietary conditions where vitamin D deficiency is rare or nonexistent or 2) measure vitamin D levels and adjust for them in the analyses or study them as potential modifiers of the relation between asthma and caries.

Our meta-analysis indicated that persons with asthma are at a higher risk of caries whatever the causal pathways underlying the observed associations. From a public health perspective, our recommendations are valid: Dentists should pay extra attention to the oral status of patients with asthma, since they are more susceptible to caries and its progression. The suggestions of links with SHS exposure and vitamin D deficiency provide interesting hypotheses for future studies that may lead to additional tools for treating caries in persons with asthma.

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REFERENCES

Salla Alavaikko1,3, Maritta S. Jaakkola1,2, Leo Tjäderhane3, and Jouni J. K. Jaakkola1 (e-mail: jouni.jaakkola@oulu.fi)
1 Center for Environmental and Respiratory Health Research, Institute of Health Sciences, University of Oulu, FI-90014 Oulu, Finland
2 Center for Environmental and Respiratory Health Research, Respiratory Medicine Unit, Institute of Clinical Medicine, University of Oulu, FI-90014 Oulu, Finland
3 Institute of Dentistry, University of Oulu, Oulu University Hospital, FI-90220 Oulu, Finland

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