Thorax Online First, published on February 7, 2014 as 10.1136/thoraxjnl-2014-205133

CORRESPONDENCE

Authors' response to Young and Hopkins: vitamin D and lung function

We thank Dr RP Young and Ms RJ Hopkins for their interest in our article and their constructive comments.[1]

We agree with Dr RP Young and Ms RJ Hopkins that our analyses may suggest a threshold effect. However, we believe that more studies are needed before we firmly can assume a threshold effect. It is true that that the association between vitamin D level and FEV_1% predicted was mirrored by a corresponding association between vitamin D and FVC% predicted. Therefore in our main analysis, the vitamin D level was not significantly associated with the FEV_1/FVC ratio. This general reduction of lung volumes in individuals with low vitamin D levels could, as suggested by Young and Hopkins, be caused by general frailty or weakness and not by an intrapulmonary process. Yet, as we observed a significant interaction, suggesting a stronger association between vitamin D and FEV_1% pred in individuals with airway obstruction we concluded that low vitamin D levels may play a role with regard to development and in particular progression of COPD.[2] It was not our intent to claim that low vitamin D is a cause of decreased lung function, as observational studies like ours are subject to reverse causation and confounding as rightly pointed out by Young and Hopkins. We rather sought to report the observed association and fully agree that these associations could be confounded by or mediated through, for example, systemic inflammation. Indeed, we ourselves have previously reported that markers of systemic inflammation like elevated fibrinogen and C reactive protein are associated with reduced lung function, increased risk of COPD and prognosis in COPD.[3–5] Therefore, we agree that future studies on the relationship between vitamin D and lung function and COPD should include possible confounding or mediation through systemic inflammation.

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Contributors SA and BGN drafted the response, which was amended and finally approved by PL, SEB and JJF.

Competing interests PL received during the last 5 years pharmaceutical company grants from GSK in 2010 and 2011, Boehringer-Ingelheim and Almirall in 2012 via his home institution and also participated in speaking activities, industry advisory committee or other related activities sponsored by Almirall, Boehringer-Ingelheim, Novartis, GSK, AstraZeneca, Pfizer, Mundipharma and UCB during the period 2008–2012.

Provenance and peer review Not commissioned; internally peer reviewed.

To cite Afzal S, Lange P, Bojesen S E, *et al*. *Thorax* Published Online First: [*please include* Day Month Year] doi:10.1136/thoraxjnl-2014-205133

Received 13 January 2014
Accepted 16 January 2014

Linked

- http://dx.doi.org/10.1136/thoraxjnl-2013-203682
- http://dx.doi.org/10.1136/thoraxjnl-2014-205101

Thorax 2014;**0**:1. doi:10.1136/thoraxjnl-2014-205133

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THORAX

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