measure aortic PWV occurred in a large proportion of patients at baseline (n=51, 24%), which is not our or other groups' usual experience and may therefore have inadvertently generated bias.

There is a great deal of heterogeneity between the studies published to date in terms of type and duration of exercise intervention, the magnitude of improvements in the functional outcome measures, inclusion of those with or without established cardiovascular disease, and the arterial bed assessed (carotid–femoral *versus* carotid–brachial), all of which undoubtedly will have contributed to the conflicting results. In the past, a similar controversy regarding the benefits of exercise intervention after myocardial infarction was resolved by the use of well-designed randomised controlled trials, which should be considered before discarding the likely cardioprotective role of pulmonary rehabilitation and the strong public health message of exercise improving cardiorespiratory health.

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RCTs of #pulmrehab or #exercise in patients with #COPD are required to establish potential cardioprotective role http://ow.ly/tROVc

Charlotte E. Bolton[1], Nichola S. Gale[2] and John R. Cockcroft[2]
[1]Nottingham Respiratory Research Unit, School of Medicine, University of Nottingham, Nottingham, and [2]Wales Heart Research Institute, Cardiff University, Cardiff, UK.

Correspondence: Charlotte E. Bolton, Nottingham Respiratory Research Unit, The University of Nottingham, School of Medicine, Clinical Sciences Building, City Hospital NUH Trust Campus, Hucknall Road, Nottingham NG5 1PB, UK. E-mail: charlotte.bolton@nottingham.ac.uk

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From the authors:

We thank C.E. Bolton and colleagues for their interest in our report about arterial stiffness in patients with chronic obstructive pulmonary disease (COPD) [1]. Our paper has two major findings: 1) aortic pulse wave velocity (PWV) in COPD is not related to systemic inflammation; and 2) aortic PWV does not respond to pulmonary rehabilitation [1]. According to C.E. Bolton and colleagues, these findings are unexpected as two previous papers did report small, but significant reductions in aortic PWV with exercise and/or pulmonary rehabilitation [2, 3]. However, VIVODTZEV *et al.* [2] used carotid-brachial pulse wave velocity as an outcome of arterial stiffness, which is known to be more susceptible to modification by exercise training than central (aortic) elastic arteries [4]. GALE *et al.* [3] did use aortic PWV as an outcome of arterial stiffness and used similar methodology to our study. While GALE *et al.* [3] suggested that a reduction in mean arterial pressure accounted for the reduction in aortic PWV following rehabilitation in their sample of 22 patients with

pre- and post-rehabilitation data, our study in a much larger COPD population did not confirm either of these findings.

C.E. Bolton and colleagues suggest the lack of change in aortic PWV in our study can be attributed to the ability of the participants to maintain sufficient exercise intensity, or confounders such as medications and timing of assessments, or failure to measure aortic PWV. Even though we very much appreciate this correspondence, we disagree with the speculative arguments of C.E. Bolton and colleagues. Unfortunately, the individual training schedules of our study sample cannot be provided. Nevertheless, the pulmonary rehabilitation programme was organised and executed according to international recommendations [5]. Our programme consisted of 40 sessions, twice as many as the number of sessions in the studies by GALE *et al.* [3] and VIVODTZEV *et al.* [2]. Mean changes in 6-min walking distance (+31 m), constant work-rate cycle test (+198 s) and disease-specific health status (St George's Respiratory Questionnaire total score: -4 points) were very acceptable when compared with existing literature [6]. Therefore, we believe that training intensity was sufficient. All aortic PWV assessments were carried out on sober patients early in the morning after 15 min of rest in a supine position. Stratification for the use of lipid-lowering or blood pressure-lowering drugs did not affect aortic PWV results (see table 5 of the original paper [1]). In the study by GALE *et al.* [3], patients with COPD were excluded from the study if they had known ischaemic heart disease, cardiac failure, diabetes mellitus, malignancy, or any other inflammatory or metabolic condition, or were receiving oral corticosteroids, disease modifying or weight loss drugs. By contrast, our study sample had multiple objectified comorbidities, which may explain the failed aortic PWV measurements at baseline. Indeed, patients with unsuccessful aortic PWV measurements had a significantly higher body mass index, and levels of triglycerides and glucose [1].

Interestingly, we found a rather high variability in the (nonsignificant) change in aortic PWV following pulmonary rehabilitation, which is very similar to data presented in figure 1 of the article by GALE *et al.* [3]. So, while some patients with COPD may have a reduced aortic PWV following pulmonary rehabilitation this is not the case for all patients.

We agree with C.E. Bolton and colleagues that aortic PWV is a robust predictor of cardiovascular risk. Therefore, it seems of clinical relevance to better understand the underlying mechanisms, as well as the pharmacological and non-pharmacological treatment of increased aortic PWV in patients with COPD. Large-scaled randomised controlled trials are necessary to take possible confounders into consideration.

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Aortic PWV is a predictor of cardiovascular risk: understanding underlying mechanisms in COPD is clinically relevant http://ow.ly/u3dXe

Lowie E.G.W. Vanfleteren, Martijn A. Spruit, Emiel F.M. Wouters and Frits M.E. Franssen
Program development centre, CIRO+, Centre of Expertise for Chronic Organ Failure, Horn, The Netherlands.

Correspondence: Lowie E.G.W. Vanfleteren, Program development centre, CIRO+, Centre of Expertise for Chronic Organ Failure, Hornerheide 1, Horn, 6085NM, The Netherlands. E-mail: lowievanfleteren@ciro-horn.nl

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