LETTERS

Quantifying physical activity in COPD: different measures for different purposes

We have read with great interest the recent article by Walker *et al*[1] and the accompanying editorial by Morgan[2] on the measurement of physical activity in chronic obstructive pulmonary disease (COPD). Walker *et al* compared an activity monitor (DynaPort) with a leg-mounted uniaxial accelerometer (Actiwatch). They concluded that "lower limb activity is the major determinant of whole body activity". In the accompanying editorial Morgan states that it therefore "does not seem necessary to use overly complicated devices" any more to measure physical activity. The latter statement is, in our opinion, an oversimplification.

First, the validity of the Actiwatch to assess leg activity in these patients seems insufficient. Walker *et al* reported an inverse and poor relation ($r = -0.42$) between "leg activity" from the Actiwatch and walking time from the DynaPort. Since the DynaPort showed excellent agreement with video recordings,[3] this inverse relation suggests that the Actiwatch does not accurately measure patients' walking at low walking speed.

Second, movement intensity and overall movement time (including minor movements such as fidgeting) were chosen as the main outcomes from the DynaPort to represent "whole body activity". These outcomes do not reflect the full scope of information that activity monitors can provide. Activity monitors differentiate between postures (ie, standing, sitting and lying) and movements (ie, walking and cycling) and classify intensities of movements. By being able to measure these outcomes, activity monitors provide information that is easily interpretable both for healthcare providers and patients. In contrast, uniaxial accelerometers register "activity counts" as an abstract overall measure of daily activity that combines intensity and time spent in physical activity.[4]

In general, both accelerometers and activity monitors can provide useful information depending on their purpose of use. Validated accelerometers are useful as an overall measure of physical activity, discriminating physically active from physically inactive populations. Most accelerometers and pedometers seem, however, not to be sensitive enough to pick up changes in physical activity in slowly moving patients.[5 6] Whenever one wishes to quantify daily time spent in different leg activities (ie, walking or cycling) and postures one will have to rely on activity monitors. Facilitating interpretability of results in this way is of special interest when one aims at increasing patients' awareness of their activity levels.

In summary, the study by Walker *et al* does not provide enough evidence to allow the conclusion that the Actiwatch accurately measures "leg activity" in patients with COPD. This uniaxial leg accelerometer should therefore not be regarded as a surrogate measure for an activity monitor in this population. Efforts should be undertaken to make activity monitors as user-friendly as possible. These should lead to the next generation of physical activity monitors with larger memory and smaller size that are affordable for use in both research and clinical practice.

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Authors' reply

We would like to thank Dr Langer and colleagues for the interest shown in our paper.[1] The authors appear to have interpreted our article and the accompanying editorial as suggesting that accelerometers measure exactly the same outcomes as activity monitors. This is not the case, as evidenced by the fact that we used an activity monitor (Dynaport) to validate the recordings made with an accelerometer (Actiwatch). However, the data we presented show a close correlation between measurements of overall activity recorded by the two devices (overall activity score: $r = 0.92$, intensity of activity score: $r = 0.83$). This supports our assertion that leg activity measured by the Actiwatch is the major determinant of whole body activity measured by the Dynaport.

Accelerometers do not try or claim to specifically measure time spent walking, which is not the sole contributor to overall whole body activity. In our COPD population, time spent cycling is rarely of relevance and no patient spent any time cycling during our Dynaport recordings. We do not dispute that time spent walking is a useful measure and an easy concept for an individual to understand, but we disagree that level of physical activity is conceptually difficult for a patient to comprehend. In fact, the UK government has tried specifically to address the issue producing guidance on how to increase physical activity in the overall population.[2] We believe that improving level of physical activity after an intervention is an outcome with which patients can identify. Despite the concerns raised, the Actiwatch was able to detect change in activity in slow moving patients after a standard exercise programme, even with a similar level of disease severity and improvement in walking distance compared with previously published results.[3]

Clearly the information obtained from precisely measuring time spent walking and cycling has to be balanced against the lower cost and ease of use of accelerometers. In effect, purchase of current activity monitors is impossible for almost all rehabilitation programmes so, although this outcome is an important one, it will not be measured. In other studies we have found that a significant number of patients considered the activity monitor cumbersome and difficult to use and, as a consequence, failed to complete adequate recording time.[4] We agree that, in time, activity monitors will advance technologically and current problems will be overcome but, at present, they are likely to remain a research tool because of the additional information they supply. For these reasons we feel that accelerometers are a more appropriate device for clinical practice because they accurately measure activity, are affordable and easier to use.

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Non-atopic persistent asthma in children

Henderson and colleagues[1] interestingly describe six different wheezing phenotypes among which persistent wheeze is, they say, less associated with atopy than intermediate or late-onset wheeze (but with similar lung function deficits, suggesting a mixture of structural airway abnormalities and atopic wheeze).

We would like to emphasise the fact that children with persistent asthma without allergic sensitisation (ie, non-atopic persistent asthma (NAPA)) constitute a phenotype of its own that should be accounted for separately, since its clinical features differ noticeably from atopic persistent asthma (APA).

At our reference centre for paediatric asthma in a north-eastern region of Italy, there were 14 patients with NAPA out of 1280 seen in the last 5 years (1%). In this series, 12/14 patients with NAPA (84.7%) had clinical features of moderate and severe persistent asthma vs 304/1266 patients with APA (24%, $p<0.001$); 8/14 patients with NAPA (57%) required hospital admission compared with 130 patients with APA (10%, $p<0.001$). The transition from first wheezing (usually viral) and persistent asthma symptoms was much faster in patients with NAPA than in those with APA (mean (SD) 0.5 (0.8) years vs 3.6 (2.4) years; $p = 0.001$). Moreover, only one of the patients with NAPA had a clinical history of atopic dermatitis compared with 785 (63%) of those with APA.

Just as children with APA and adults with "intrinsic" asthma, children with NAPA do have intense eosinophilic inflammation of the respiratory airways.[1] In agreement with this finding, inhaled steroids were an effective treatment in our patients.

Despite its low incidence, this infrequent—although not very rare—paediatric asthma phenotype should not be missed in large epidemiological cohort studies. Nothing is known about which treatment is best for NAPA and—even more importantly—nothing is known about the natural history of this severe disease.

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Competing interests: None.

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Authors' reply

We thank Dr Ventura and colleagues for their interest in our paper and for drawing attention to the phenomenon of non-atopic persistent asthma (NAPA). We agree that non-atopic airway inflammation is a potential mechanism for modification of early life influences on airway development. In our paper we highlighted the strong positive association of persistent wheeze with atopy[1] but, as shown in table 2, the prevalence of atopy in this group was 42%. As persistent wheeze comprised 7% of the total population, non-atopic persistent wheeze would be expected to occur in around 4% of this unselected population-based sample. This contrasts with the prevalence of NAPA of 1% reported by Ventura and colleagues from their specialist clinic population in Italy and raises important questions about comparing results from different population samples. It is not clear from their letter how Ventura and colleagues defined persistent asthma but, without frequent early life measures, it would be difficult to disentangle intermediate onset (62% atopic) from persistent wheeze in our model. As both these phenotypes were very strongly associated with persistent wheezing and with physician-diagnosed asthma in later childhood, such misclassification could alter the inferences of their respective associations with objective markers of atopy and airway function. Ventura *et al* make some interesting observations about the early life course of NAPA which are highly relevant to our attempts to disentangle the course of early childhood wheezing trajectories. Clearly, there are more complexities to emerge from approaches to defining the various subphenotypes of asthma, and detection of rare phenotypes—even in relatively large population samples—remains a challenge. The trade-off between population size and the intensity of detailed phenotyping with relevant biomarkers that is feasible in very large epidemiological surveys exemplifies some of these difficulties, although the application of non-invasive markers of inflammation in this setting shows some promise.[2] There is a need to understand the variation of phenotypes within asthma and how these relate to clinical and pathological end points. A greater understanding of genetic determinants of components of the asthma phenotype,[3] harmonisation of outcomes between studies and exploration of environmental interactions with genetic variants will hopefully reveal modifiable targets for disease treatment and prevention.

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CORRECTION

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T Sandström, F J Kelly. Traffic-related air pollution, genetics and asthma development in children. *Thorax* 2009;**64**:98–99. Reference 6 in this editorial is incorrect, it should be Salam MT, Lin P-L, Avol EL, *et al*. Microsomal epoxide hydrolase, glutathione S-transferase P1, traffic and childhood asthma. *Thorax* 2007;**62**:1050–7.