LETTERS TO THE EDITOR

doi:10.1093/occmed/kqs039

Re: Self-reporting height and weight

Dear Sir,

In their recent short report [1], Lois et al. make the point that self-reported height is often unreliable, particularly in men. The context of their report is the use of self-reported data in a health-promotion programme targeted at obesity. However, there are lessons here for occupational health practice during measurement of lung function using forced expiratory volume (FEV1) and forced vital capacity (FVC), for which height is a principal reference variable. Lois et al. compared the self-reported height with that obtained by a standard method; this was referenced but not detailed in their study. Some may conclude that details do not need repeating because they are a part of the lifetime experience of most children. However, the measurement is still sometimes made using unsuitable equipment, for example, a vertical rod with a sliding horizontal strut that can stick or wobble. In addition, the strut can be applied with variable pressure, or without considering the posture of the subject. In any of these circumstances, an error of 2.5 cm (1 inch) in the measurement of height is not uncommon. In their study, Lois et al. reported differences ranging from −16 to +12 cm between the estimated and the measured heights in men. Such inaccuracies can lead to material errors in estimating the reference values for lung function on a single occasion and to an unacceptably large error for follow-up assessments, unless the same measurement fault is present throughout. As an example, in Caucasian men, the regression coefficient of the FVC with reference to height is −5 l/m [2]; so, an error of 2.5 cm equates to an inaccuracy of 125 ml in the reference value, which is equivalent to 20–25% of the equation’s residual standard deviation (RSD) and hence is of clinical importance. For FEV1, the equivalent error is ~95 ml, or ~20% of the equation’s RSD. Such inaccuracies can lead to serious errors in the interpretation of results of the lung function tests and lead to misdiagnosis.

In the case of serial measurements, the error in interpretation can be compounded if concomitant longitudinal changes in body fat and muscle are not taken into account [3]. Interpretation should be based on separate measurements of these two components of body composition because, for most indices of lung function, their effects are opposite.

We hope that in any study of lung function, the operators would measure the subject’s height directly (and accurately) and not rely on a self-report. However, this may not always be the case in a busy clinic. In occupational health practice, we devote considerable effort to training staff to measure lung function accurately. Sloppy measurements of height or reliance on self-report can devalue these efforts and invalidate the interpretation of the results. Lois et al. have reinforced the lesson that self-reports of anthropometry can be unreliable. This provides an opportunity to re-emphasize the general message that accuracy counts.

John E. Cotes and David J. Chinn
e-mail: coterie@globalnet.co.uk

References


doi:10.1093/occmed/kqs040

Long-term pesticide exposure and the risk of testicular cancer

Dear Sir,

In their recent article, Frost et al. [1] found significantly elevated incidence of testicular cancer (TC) in British users of pesticides, with a non-significant excess in mortality. This is not surprising as many occupational pesticide studies are designed to examine mortality rather than the incidence of TC and find conflicting results. Considering the recent improvements in therapy, mortality rates due to TC have declined sharply in developed countries, while its incidence has more than doubled over the past four decades [2]. Therefore, the use of mortality rates should be considered suboptimal for the study of TC. The Pesticide Users Health Study [1] could represent a significant contribution for ascertaining the possible causal role of pesticides in TC and could be placed in the group of studies concerning the potential effects of long-term exposure to pesticides. Several studies on its incidence revealed that long periods of exposure to pesticides significantly increased the risk...
of TC. Guo et al. [3] found positive exposure–response relationships between TC and occupational exposure to pesticides, particularly for insecticides (≥0.002 mg/m²-years; relative risk (RR) = 3.26, 95% confidence interval (CI) = 1.20–8.83), with significantly elevated standardized incidence ratios (SIRs) after 10- and 20-year lag periods of exposure. The incidence of TC was also significantly elevated among licensed pesticide applicators in Florida during follow-up in the period 1975–93 (SIR = 2.48, 95% CI = 1.57–3.72) [4]. The SIR for TC was significantly increased with 'time since license' >10 years in pesticide applicators in Swedish agriculture (SIR = 2.54, 95% CI = 1.1–5.00) [5]. The Agricultural Health Study showed a slight increase in the risk of TC (without statistical significance) among commercial applicators; but only 11% of this group had applied pesticides for periods >20 years [6]. Finally, Frost et al. have now found significantly elevated incidence of TC in British users of pesticides followed up between 1987 and 2004 (SIR = 1.26, 95% CI = 1.04–1.53) [1].

The possible causal relationship of TC with long-term pesticide exposure is also corroborated by the accumulation of these chemicals over time in the serum of subjects with TC. To date, six case–control studies have examined the serologic measures of pesticides and TC [2,7]. A recent review noted that increased serum levels of organochlorine pesticides were consistent with a positive association with TC, with the two studies that included prediagnostic serum samples providing the strongest evidence [2]. We also recently reported some further evidence of this association, finding that TC was significantly associated with higher serum concentrations of organochlorine pesticides in diagnosed cases, compared with controls (OR_adjusted = 3.34, 95% CI = 1.09–10.17) [7].

Some pesticides have been classified as endocrine-disrupting chemicals because they may mimic the actions of estrogens or have anti-androgenic effects. Exposure to these compounds has recently been hypothesized to increase the risk of TC by interfering with the regular hormonal balance of the subject, but more research is needed to confirm this hypothesis [2]. The current findings available from the literature suggest that the increase in TC incidence in recent decades could, at least in part, be related to the accumulation of some pesticides in the environment.

Fabrizio Giannandrea
e-mail: fabrizio.giannandrea@uniroma1.it

References