Response to correspondence ENVINT_2020_552 “Can habitual exercise really increase serum concentrations of persistent organic pollutants?”

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Response to correspondence ENVINT_2020_552 “Can habitual exercise really increase serum concentrations of persistent organic pollutants?”

Handling Editor: Lesa Aylward

We appreciate the interest by Lee & Lee in our recent study that reported high circulating plasma polychlorinated biphenyls (PCBs) in fit and lean children (Domazet et al., 2020). Below we provide responses to the points raised by Lee & Lee.

The first comment raised questioned the validity of investigating the relationship of exercise with lipophilic persistent organic pollutants (POPs) in a child cohort, since weight loss or weight gain are very important determinants of serum POPs. This is an important point, however, we disagree. Firstly, BMI status and obesity have been found to track strongly throughout childhood and adolescence (Freedman et al., 2018), wherefore we assume our study population to be relatively stable in body composition. The growth rate typically reaches its lowest point just before the initiation of the adolescent growth spurt, which is estimated around 10 years for girls and 12 years for boys (Tanner et al., 1966) and therefore at an older age than our study population. Secondly, the peak velocity during the adolescent growth spurt correlates strongly with the initiation of puberty (Tanner et al., 1976), which was evaluated in our study; only 16.8% had started puberty (girls only). Our study population consisted of a random sample of exclusively third grade students from public elementary schools with a mean age of 9.7 years (SD 0.4 years). This narrow age-ranged population-based cohort ensures a homogeneous sample with respect to growth and maturation. Yet, we still carefully normalized cardiorespiratory fitness for body mass (watt/kg) in all analyses, statistically evaluated the effect of conditioning on body composition (subcutaneous fat mass) and performed a separate analysis stratified by pubertal status in girls. In summary, our results clearly showed (1) a positive association between fitness and PCB independent of adiposity level and (2) an inverse association between fatness and PCB independent of fitness. We cannot see any argument that a study in an adult cohort such as the National Health and Nutrition Examination Surveys (NHANES) would be more suited for the purpose. On the contrary, such adult cohort is much more heterogeneous and weight cycling are likely more prevalent, which requires even more careful control of body size and composition when investigating the association of PCB with fitness or habitual physical activity.

We agree with Lee & Lee that overweight/obese individuals may sequester more PCB in adipose tissue relative to plasma due to a larger adipose tissue reservoir. Therefore, we have described this issue as a limitation in the discussion of our paper “our findings could be biased due to overweight children having lower plasma PCB concentrations not as a result of lower lipolytic activity but as a result of sequestering PCBs to adipose tissue”. However, our results do not suggest that this explains the ENTIRE association between fitness and PCB, since the relationship was highly significant even after adjustment for subcutaneous fat mass, which has been found to correlate highly with DEXA-measured total body fat percentage (Gutin et al., 1996).

We agree with Lee & Lee that it is plausible that POPs may be excreted at a faster rate in fit individuals through increased circulation and biotransformation through liver enzyme activity. Nevertheless, it cannot explain the associations that we observe. If the fit and lean children excreted higher amounts of PCB, we would not see an almost three-fold higher plasma PCB concentration among these children. Quite the reverse, we would expect a much weaker or an opposite direction of the association presented by either a smaller mean difference in plasma PCB concentrations between fit/lean versus unfit/overweight children or a higher PCB level among unfit and overweight/obese children. We are not able to comment on Lee and colleagues’ paper from 2020 as the paper is in press (Lee et al., 2020 in press) however, the other paper that Lee & Lee refer to showing lower levels of organochlorines among athletes could have major flaws (Pelletier et al., 2002). The study, which we also cite in our paper, is highly prone to bias caused by selection into the study as their study population comprised of three very small groups that was selected completely independent of each other and at different points in time and as a consequence, clearly does not stem from the same source population. Thus, selection bias and residual- and unknown confounding could be major sources of bias unaccounted for in the study by Pelletier et al.

The other major issue raised by Lee & Lee relates to the study design. They argue that a cross-sectional study must be the most appropriate to examine our research question.

When we want to estimate the causal effect of regular engagement in physical activity on circulating levels of PCB we want either to conduct a true experiment (i.e. a randomized controlled trial with random allocation to groups discordant in assigned habitual physical activity level over a period of time (i.e. an exercise program)) or propose to ask the similar causal question in an observational study. In a prospective observational study, we could emulate such a study by investigating changes in PCB against changes in habitual physical activity level over a period of time if repeated participant information on physical activity engagement, body composition (including change in

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adiposity level), and PCB were available. This would allow us to control for the influence from i.e. weight loss or gain or model the synergy of changes in circulating PCB or other lipophilic POPs. A cross-sectional study is unable to account for this in the same detail and clearly less suitable to answer the causal question. Also, it is well known that it is rather difficult to attain weight loss through exercise alone due to i.e. a relatively small calorie-burning effect of a normal exercise regime and increased exercise-induced calorie consumption (Thomas et al., 2012), which limits the issue raised by Lee & Lee that exercise-related weight loss can distort such results.

Lee & Lee also suggest that hypertrophic adipocytes are able to increase the circulating levels of POPs due to uncontrolled lipolysis among unhealthy obese individuals. That might be so, but not in our case. Our study population comprised of ~5% obese children, who were definitely not clinically obese (class II-III according to CDC). Nor were they otherwise unhealthy. Also, our results showed the opposite of a high plasma PCB concentration among overweight and obese children.

We acknowledge that the internal pathways should be explored to decrease POP-related health risk. However, we do not see this as the primary aim, and it should not overshadow our pursuit to eliminate POP exposure in the environment. We do not believe that controlling the toxicokinetic of POPs through calorie restriction, intermittent fasting, increased intake of phytochemicals, moderate fat intake, and increased physical activity is a practical way to mitigate the impact of unavoidable exposures to low-dose chemical mixtures such as POPs. On the contrary, exposure to harmful environmental toxins should not be up to the individual in the first place. Instead, exposure should be regulated through political and structural initiatives to target the entire population. Our study asked a basic science question that we propose should be further investigated in more rigorous designed studies considering the size of the associations we observe. Regardless of all the positive effects of exercise on health, the possible effects of regular exercise on PCB or other lipophilic POPs stored in human body tissues is a topic of major relevance to examine in future studies.

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Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

References


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