Status report

Patterns of age of puberty among children in the Mid-Ohio Valley in relation to Perfluorooctanoic Acid (PFOA) and Perfluorooctane Sulfonate (PFOS)

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This status report summarizes the findings of a statistical analysis of the relationship between levels of perfluorooctanoic acid (PFOA, also called C8), and perfluorooctane sulfonate (PFOS) measured in the blood serum of the children who participated in the C8 Health Project, and puberty. A full report of these findings will be submitted to a peer-reviewed scientific journal.

Introduction.

It has been suggested that some polyfluoroalkyl compounds (PFCs) may alter animal sexual maturation. The aim of this study was to examine the relationship between levels of two PFCs – PFOA (or C8) and PFOS – with puberty based on sex hormone levels and self-reported onset of menstruation. We used data from the C8 Health Project supplemented with detailed date of birth available from those who consented to be in the Science Panel studies.

Methods.

Among the young participants aged 8-18 at the time of the C8 Health Project survey (2005-2006), we examined data for 3076 boys and 2931 girls, all residents for at least a year in the six water districts which had been contaminated with PFOA. They were classified as having reached puberty at the time of interview based on either sex hormone blood levels (testosterone >50 ng/dL or free testosterone >5 pg/mL for boys, and estradiol >20 pg/mL for girls), or having reported that they had started menarche (periods). Statistical models estimated the chance of reaching puberty in relation to PFOA, and PFOS levels, while controlling for other potential explanatory factors. From these models, we could also estimate the average age of reaching puberty for children with different exposure levels, and present the difference (earlier or later) in days of reaching puberty between different exposure to PFOS and PFOA.

Results.

The mid-point of PFOA and PFOS serum levels were 26 and 20 ng/mL in boys, and 20 and 18 ng/mL in girls. For boys, there was a clear relationship of reduced odds of having reached puberty with increasing PFOS (delay of 190 days between the highest and lowest quartile), but not PFOA. For girls, higher exposure to either PFOA or PFOS was associated with reduced odds of having reached puberty. The highest PFOA group had an average age of puberty 130 days later than the lowest exposure group, and for PFOS, the delay was estimated as 138 days comparing the highest and lowest exposure group. These results are consistent in direction and magnitude with one published study which suggested delayed puberty (also measured as self-reported menarche) in relation to PFOS exposure in girls, and in contrast to another study which reported younger puberty (measured as breast maturation) in girls in relation to PFOA exposure.

Conclusions.

Delays of puberty have been observed in this population correlated with PFOS in boys and PFOA and PFOS exposure in girls. Caution is needed in interpreting these results, due to the fact that blood PFC levels and puberty status based on sex hormone levels were determined at the same time, and menarche was self-reported. For example, it may be that growth changes associated with puberty lead to changes in PFOA and PFOS blood levels, rather than these compounds having any effect on age at puberty. Further work is planned to investigate patterns of puberty by age in relation to exposure prior to puberty.