P-26 Occupational pesticide exposure in early pregnancy associated with impaired glycaemic control in adolescents

Helle Raun Andersen[1], Jeanette Tinggaard[2], Philippe Grandjean[1], Tina K Jensen[1], Christine Dalgård[1], Katharina M Main[2]

[1]Institute of Public Health, University of Southern Denmark, Odense, [2]University Dept. of Growth and Reproduction, Rigshospitalet, Copenhagen , Denmark.

Background

Occupational pesticide exposure has been associated with increased risk of type 2 and gestational diabetes mellitus but potential effects of gestational exposure on glucose regulation in children have not been investigated. Previously, we found associations between occupational pesticide exposure in pregnancy and higher childhood body fat content, blood pressure, and serum markers of metabolic dysfunction. The associations were affected by sex and PON1 Q192R (rs662) polymorphism influencing the antioxidative capacity of the paraoxonase 1 enzyme. We explored in the same cohort, if maternal pesticide exposure was associated with elevated glycated hemoglobin (HbA1c) in their children.

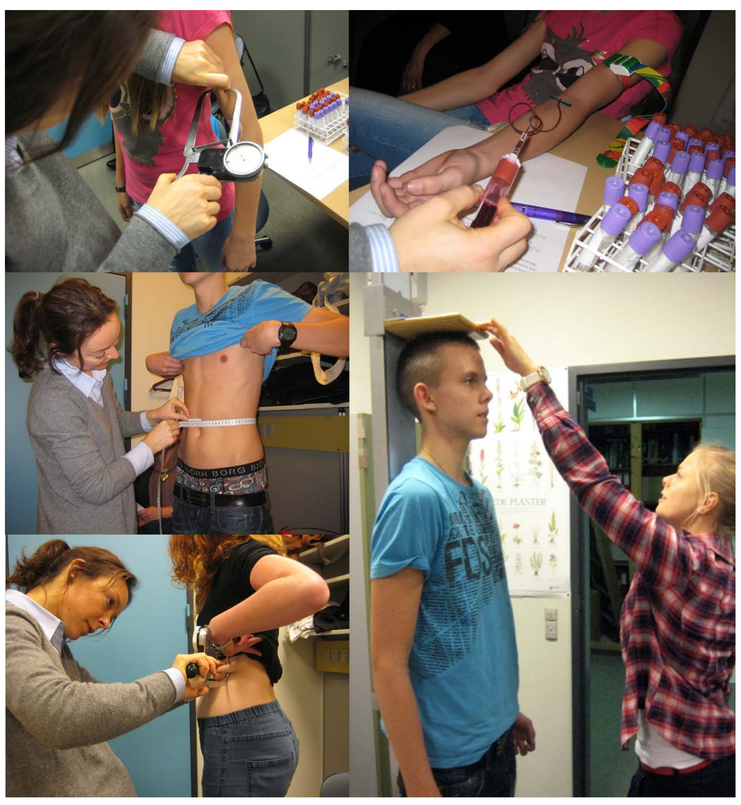
Methods

Pregnant women working in greenhouse horticultures were recruited in the beginning of their pregnancy and categorized as occupationally exposed or not exposed to pesticides. Exposed women were moved to unexposed work functions or went on paid leave. At age 10-to-16 years, the children underwent a clinical examination including anthropometry, pubertal stage assessment (accepted by 141 children) and blood sampling. Serum was analyzed for HbA1c. PON1-genotype was available for 152 children and 112 mothers.

Table1. Characteristics n (%) or mean (95% CI) of 141 children whose mothers were occupationally exposed or unexposed to pesticides during early pregnancy

	Unexposed (N=55)	Exposed (N=88)	P-value
SES	21/23/9 (40/43/17)	17/49/20 (20/57/23)	0.04
Child age	12.8 (12.3; 13.2)	13.2 (12.9; 13.5)	0.08
Puberty, Tanner stage (1/2/3/4/5)	7/10/10/13/13 (13/19/19/24.5/24.5)	4/15/21/24/24 (5/17/24/27/27)	0.45
Waist circumference ,cm	67.0 (64.6; 69.5)	70.0 (68.0; 72.0)	0.06
Body fat % (skinfolds)	19.2 (16.7; 21.8)	23.2 (21.2; 25.2)	0.02
BMI, kg/m^2	19.1 (18.1; 20.2)	20.6 (19.8; 21.4)	0.02

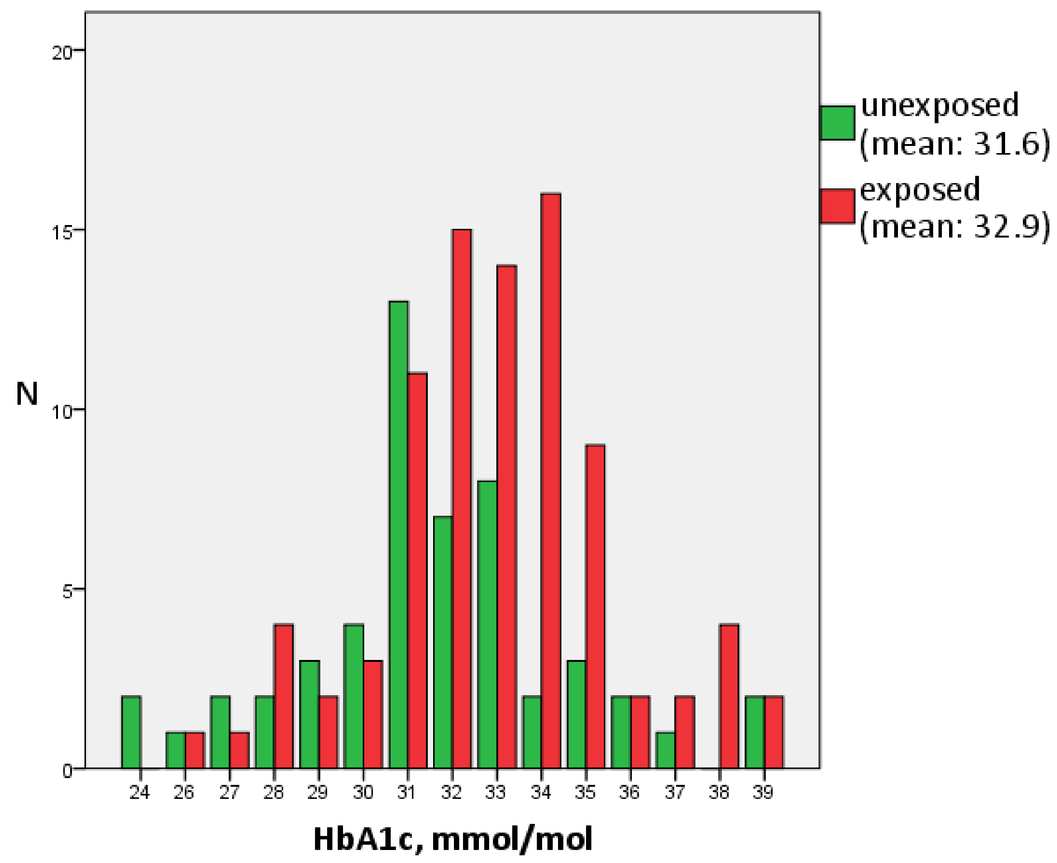
SES: Socioeconomic status, social class 1-3/4/5; Puberty: Highest of Tanner Pubic hair stage, breast stage (girls) and/or genital stage (boys)e



Conclusion

Our results suggest an adverse effect of prenatal pesticide exposure on glucose homeostasis in adolescents and support previous findings from this cohort of an exposure-associated adverse metabolic risk profile along with higher susceptibility related to female sex and genetic predisposition due to the PON1 192R-allele

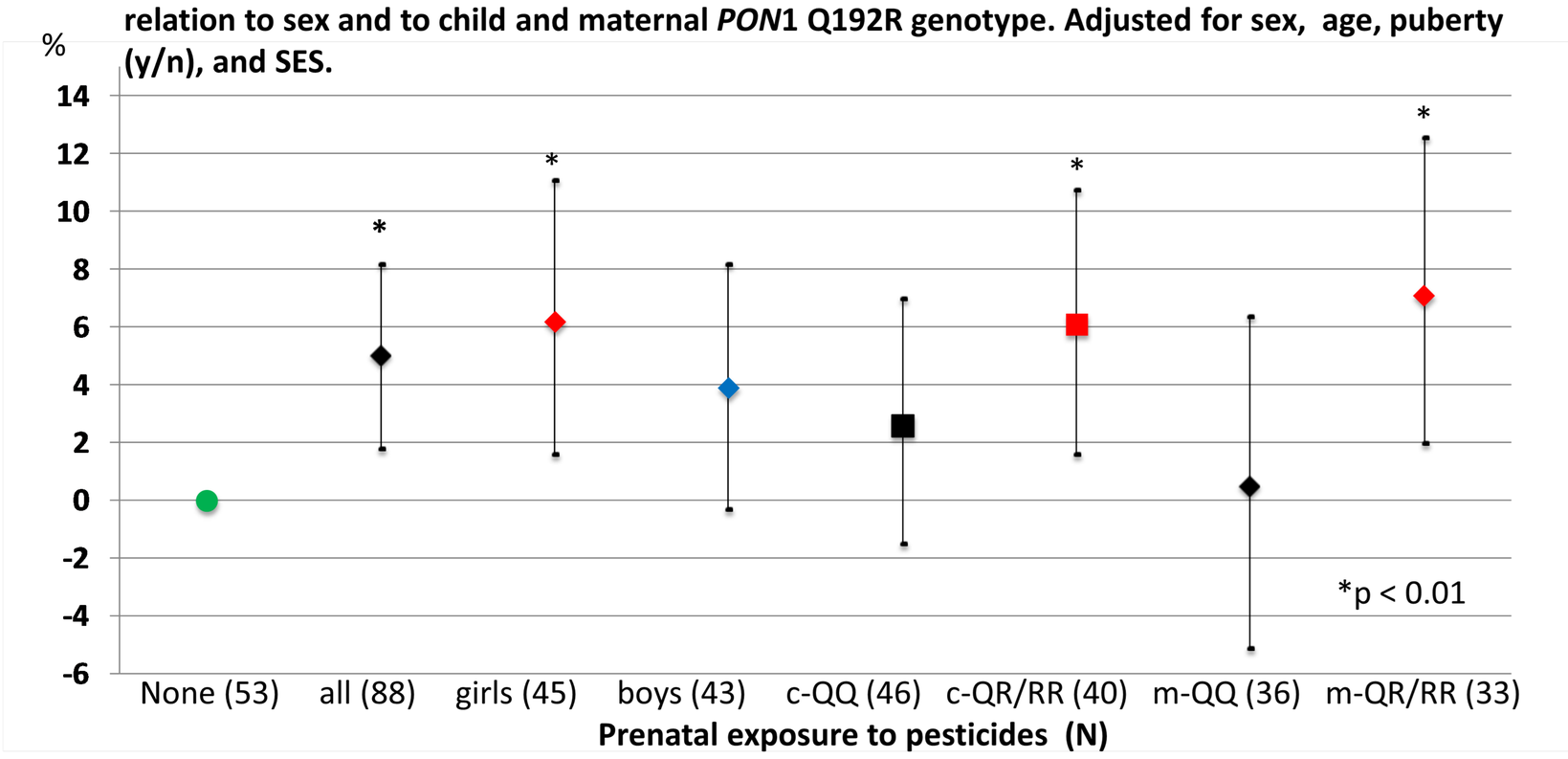
Figure 1 Distribution of prenatally pesticide exposed and unexposed children in relation to HbA1c



Results

Prenatally pesticide exposed children had higher mean HbA1c than unexposed children as reflected by a shift of the distribution curve towards higher levels for the exposed children(Figure 1). After adjustment for age, sex, socio-economic status, and puberty, prenatal exposure was associated with a 5.0% higher HbA1c compared to unexposed children ($p < 0.001$). The association was stronger in girls (6.2%, $p = 0.007$) than boys (3.9%, $p = 0.07$) and if the child or mother had the PON1 192R-allele (6.1% and 7.1%, $p < 0.01$) (Figure 2). Controlling for body fat% did not affect the results.

Figure 2 Relative difference in % (95% CI) of HbA1c between unexposed and exposed children in relation to sex and to child and maternal *PON*1 Q192R genotype. Adjusted for sex, age, puberty (y/n), and SES.



Correspondence:
Helle Raun Andersen, Email: hrandersen@health.sdu.dk