Effects of Perinatal Exposure to PCBs and Dioxins on Play Behavior in Dutch Children at School Age

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Polychlorinated biphenyls (PCBs) and dioxins are known as neurotoxic compounds that may modulate sex steroid hormones. Steroid hormones play a mediating role in brain development and may influence behaviors that show sex differences, such as childhood play behavior. In this study we evaluated the effects of perinatal exposure to environmental levels of PCBs and dioxins on childhood play behavior and whether the effects showed sex differences. As part of the follow-up to the Dutch PCB/dioxin study at school age, we used the Pre-School Activity Inventory (PSAI) to assess play behavior in the Rotterdam cohort (n = 207). The PSAI assesses masculine or feminine play behavior scored on three subscales: masculine, feminine, and composite. Prenatal exposure to PCBs was defined as the sum of PCB 118, 138, 153, and 180 in maternal and cord plasma and breast milk. For breast milk we measured additional PCBs as well as 17 x. Respondents returned 160 questionnaires (age 7.5 years ± 0.4). Effects of prenatal exposure to PCBs, measured in maternal and cord plasma, on the masculine and composite scales were different for boys and girls (p < .05). In boys, higher prenatal PCB levels were related with less masculinized play, assessed by the masculine scale (p = .042; p = .001) and composite scale (p = .011); whereas in girls higher PCB levels were associated with more masculinized play, assessed by the composite scale (p = .028). Higher prenatal dioxin levels were associated with more feminized play in boys as well as girls, assessed by the feminine scale (p = .048). These effects suggest prenatal steroid hormone imbalances caused by prenatal exposure to environmental levels of PCBs, dioxins, and other related organochlorine compounds. Key words: dioxins, endocrine disruption, play behavior, polychlorinated biphenyls, prenatal exposure, sex-specific, sex steroids. Environ Health Perspect 110:A593–A598 (2002). [Online 13 September 2002] http://ehpnet1.niehs.nih.gov/docs/2002/110pA593-A598vreugdenhilabstract.html

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design and recruitment process, chemical analysis, and PCB and dioxin concentrations have been described in detail elsewhere (20). Pregnancy and delivery were uncomplicated. Only first- or second-born children, born healthy at term, were included. Half of the group of children was breast-fed (BF) (*n* = 105) for at least 6 weeks; the others were formula-fed (FF) (*n* = 102) during infancy. All FF infants received formula from a single batch (Almiron M2; Nutricia NV, Zoetermeer, The Netherlands) from birth until 7 months of age. In this formula, PCBs and dioxins were not detectable. The medical ethics committee of the University Hospital Rotterdam/Sophia Children’s Hospital approved the study design, and the parents gave informed consent.

**Assessment of exposure variables.** Plasma samples were collected from the mothers during the last month of pregnancy, and cord plasma samples were collected directly after birth. These samples were analyzed for four PCB congeners: International Union for Pure and Applied Chemistry (IUPAC) numbers 118, 138, 153 and 180. Two weeks after delivery, a 24-hr representative breast-milk sample was collected from the mothers who were breast-feeding their children. Breast-milk samples were analyzed for 17 dioxins (PCDDs and PCDFs), 6 dioxin-like PCBs (3 planar PCBs and 3 mono-ortho PCBs), and 20 non-dioxin-like PCBs. Toxic potency of the mixture of dioxins and dioxin-like PCBs was expressed by using the toxic equivalent (TEQ) approach (27).

We estimated prenatal exposure to PCBs in the total study population by using the sum of the four PCB congeners in maternal (ΣPCBmaternal) and in cord plasma (ΣPCBcord). In the BF group additional prenatal exposure measurements were used: the ΣPCBmilk (the sum of PCB118, 138, 153, and 180), the dioxin, planar, mono-ortho, and total TEQ value (the sum of the TEQ values of the 17 dioxins and the 6 dioxin-like PCBs), and the ΣPCB20 nondioxin PCBs (the sum of 20 non-dioxin-like PCBs).

**Postnatal exposure to PCBs and dioxins through lactation was estimated by multiplying the number of weeks of breast-feeding with, respectively, ΣPCBmilk; the dioxin, planar, mono-ortho, and total TEQ; and ΣPCB20 nondioxin PCBs. Concentrations in breast milk.**

**Assessment of play behavior.** Parents were asked to complete the Dutch version of the Pre-School Activities Inventory (PSAI) (22) (Appendix) when the children reached school age. This questionnaire, along with a questionnaire on problem behavior and a health questionnaire, was sent to the parents in two mailings, depending on the age of the child (in 1998 and 1999), near the end of a school year.

The PSAI is designed to discriminate play behavior both within and between the sexes. It consists of 24 questions addressing three aspects of play behavior: type of toys, activities, and child characteristics. Answers are given on a 5-point scale ranging from never to very often. The questions assess either feminine or masculine play behavior from which 3 scales are derived: a composite scale, integrating both masculine and feminine play behavior, and a masculine and a feminine scale. The composite scale is essentially defined as the difference: feminine scale minus masculine scale. A negative score on the composite scale implies masculine play behavior and a positive score feminine play behavior. A higher score on the feminine scale indicates more feminine play behavior, whereas a higher score on the masculine scale indicates more masculine play behavior.

The questionnaire has been validated in a group of preschool English children (*n* = 102); additionally, a test–retest reliability for the scores on the PSAI of 0.62 for boys and 0.66 for girls has been found (22). The PSAI has been assessed in various cohorts for standardization and norming purposes. These cohorts include normal preschool children across several samples in the United Kingdom (pilot study (*n* = 75); validation study (*n* = 102); and a cohort obtained through the magazine Practical Parenting (*n* = 1,643), in the United States (*n* = 203), and also in the Netherlands, using a Dutch translation of the questionnaire (*n* = 341) (22).

**Assessment of other variables.** Variables that may influence child neurodevelopment have been assessed and include birth weight, duration of gestation, fetal exposure to alcohol and cigarette smoking, maternal age at birth of the child, parity, type of feeding during infancy, duration of breast-feeding, sex, and parental education level. The verbal IQ of the parent who spends the most time with the child (usually the mother) was assessed during the follow-up session at 42 months by two subtests, Information and Vocabulary from the Dutch version of the Wechsler Adult Intelligence Scale (WAIS) (23). At 7 years of age, follow-up assessment in this cohort was done at home by a psychologist (H.V.). During this visit, the child’s home environment was assessed by the Home Observation for Measurement of the Environment (HOME) (24).

**Data analysis.** To compare groups for a single variable we used the Student’s *t*-test (for continuous variables), the chi-square test (for categoric variables), or the Mann-Whitney *U* test. Plasma and milk PCB and dioxin values were positively skewed and were therefore normalized by natural logarithmic transformation.

**Table 1. Characteristics of the total study population and breast-fed and formula-fed boys and girls separately.**

| Characteristics | Total (*n* = 158) | Breast-fed boys (*n* = 53) | Breast-fed girls (*n* = 32) | Formula-fed boys (*n* = 35) | Formula-fed girls (*n* = 38) |
|-----------------|------------------|-----------------------------|----------------------------|-----------------------------|----------------------------|
| Breast feeding period (weeks) | 17 (6–72) | 16 (6–72) | 19 (6–54) | 15 (43) | 20 (53) |
| Number of first born (%) | 80 (51) | 28 (53) | 17 (53) | 22 (14) | 15 (45) |
| Parental education (%) | Low 15 (10) | 2 (4) | 4 (13) | 6 (17) | 3 (8) |
| Medium 52 (33) | 17 (32) | 6 (19) | 12 (34) | 17 (49) | 18 (47) |
| High 91 (58) | 34 (64) | 22 (69) | 17 (49) | 17 (49) | 17 (49) |
| Parental verbal IQ | HOME 48.3 (± 3.0) | 48.1 (± 3.1)* | 49.6 (± 2.7)** | 47.8 (± 2.8) | 48.1 (± 3.1)* |
| Age at assessment | 7.5 (± 0.4) | 7.6 (± 0.4) | 7.5 (± 0.3) | 7.5 (± 0.4) | 7.5 (± 0.4) |
| Exposure variables | ΣPCBmaternal (µg/L) | 2.06 (0.73–5.08) | 2.16 (0.73–4.21) | 2.09 (0.87–4.87) | 2.04 (0.88–5.08) |
| ΣPCBcord (µg/L) | 0.42 (0.08–1.99) | 0.44 (0.11–1.72) | 0.40 (0.08–1.99) | 0.38 (0.09–1.21) | 0.40 (0.08–1.99) |
| ΣPCBmilk (µg/kg fat) | 39.3 (10.2–66.6) | 42.7 (20.0–80.5) | 36.0 (174–795) | 36.0 (10.2–66.6) | 36.0 (10.2–66.6) |
| ΣTEQplanarPCB (ng/kg fat) | 15.3 (4.4–45.7) | 14.6 (4.4–45.7) | 16.6 (5.3–30.0) | 16.6 (5.3–30.0) | 16.6 (5.3–30.0) |
| ΣTEQmonoPCB (ng/kg fat) | 13.9 (3.2–25.8) | 14.2 (4.4–25.8) | 12.4 (3.2–24.8) | 12.4 (3.2–24.8) | 12.4 (3.2–24.8) |
| ΣTEQ20 nondioxin (ng/kg fat) | 68.1 (27.7–135.2) | 68.1 (27.7–135.2) | 67.1 (28.1–108.9) | 67.1 (28.1–108.9) | 67.1 (28.1–108.9) |

Values are numbers (percentages), means ± SDs, or medians (ranges).

*p* < 0.05 comparing sexes within feeding groups; *p* < 0.05 comparing feeding groups within sexes.
We studied the effects of PCB and dioxin exposure on the scores for the play behavior scales using multiple linear regression analyses (SPSS, version 9; SPSS, Chicago, IL, USA). Variables that were likely to affect play behavior were included in the regression model as a fixed set of variables. These variables were: sex (0/1 = boy/girl), highest education level of either parent (0/1 = low [primary school, secondary school not finished]/middle [secondary school finished]/high [school finished, professional and university training]), parental verbal IQ, type of feeding during infancy (0/1 = BF or FF), duration of breast-feeding (0 for FF children), HOME score, and assessment age. Additionally, confounding variables, i.e., variables that correlated (r < 0.2), were adjusted for the fixed set of variables, with one of the exposure variables and with scores of one of the play behavior scales included in the final regression model. Candidate confounders were alcohol use (0/1 = no/yes) and smoking (0/1 = no/yes) during pregnancy, duration of gestation, birth weight, maternal age at birth, and parity (0/1 = 1st/2nd born). This procedure resulted in the following regression model: sex, parental education level, parental verbal IQ, feeding type, duration of breast-feeding, HOME score, age at assessment, and parity. We studied sex differences in the effects of exposure to PCBs and dioxins by including an interaction term, the product of sex and exposure (sex*exposure), in the regression model. The effect of exposure on the outcome variables in boys and in girls, and the difference between these effects (girls minus boys) are estimated through the interaction term sex*exposure in essentially the same regression model by reparameterizing the sex effect. Results were considered significant if p ≤ 0.05.

Results

In the follow-up assessment at school age, 189 of the 207 children in the original cohort were re-examined and 160 of these parents returned the PSAl questionnaire (84% were filled out by mothers, 6% by fathers, and 10% by both parents). Two children were excluded from data analyses due to circumstances other than PCB and dioxin exposure that are likely to influence play behavior: a girl with Turner syndrome and a boy with a pervasive developmental disorder. Four questionnaires had missing data and were therefore excluded from data analyses.

Compared to the nonparticipating children (including both children who did not participate in the follow-up at school age and children whose questionnaires were not returned), prenatal PCB and dioxin exposure levels were comparable with the levels in children whose parents returned the questionnaire. Moreover, the distribution of children over the feeding groups in the participating group was not statistically different from that of the nonparticipating group (BF n = 20; FF n = 22). In regard to the other variables used in the regression model, these groups were also generally comparable except for the parental education levels (p = 0.011), parental verbal IQs (p = 0.009), and HOME scores (p = 0.021), which were higher in the participating group.

The mean age of the children at assessment was 7.5 (± 0.4) years old. The descriptive statistics for the total study group, and for BF and FF boys and girls separately, are presented in Table 1. The characteristics of all boys and girls were not significantly different. Comparing characteristics of boys and girls within feeding groups, the HOME score was significantly higher in BF girls than in BF boys. The HOME score and the parental education level were significantly higher in BF girls than in FF girls.

Boys and girls scored significantly differently on the three PSAl scales [mean (SD) composite scale: boys –14.6 (5.8), girls 14.0 (3.3); masculine scale: boys 24.2 (5.3), girls 12.6 (4.5); feminine scale: boys 9.6 (3.3), girls 26.4 (6.2); all p-values < 0.001]. Table 2 presents effects of PCBs and dioxins on the PSAl scales for boys and girls including the sex difference in effect, adjusted for all other variables. Effects of prenatal exposure to PCBs on the scores on the composite scale and masculine scale were significantly different for boys and girls. In boys, higher prenatal PCB exposure was related with higher scores on the composite scale and lower scores on the masculine scale, both indicating less masculine play behavior. In girls, effects of prenatal PCB exposure moved in opposite directions on the composite and masculine scales; however, relations were not significant. We saw no sex-specific effects of prenatal PCB exposure on scores on the feminine scale. As an example of the relation between prenatal PCB exposure and play behavior in both sexes, adjusted for confounding variables, the relation between lnΣPCBmilk and scores on the masculine scale are visualized in a partial regression plot (Figure 1).

In the BF group (Table 3), effects of ΣPCBmilk on the scores on the composite scale were also significantly different for boys and girls (p = 0.020). In girls, higher exposure to these compounds was related to lower scores (p = 0.028), indicating more masculine play behavior, whereas in boys the relation was in the opposite direction, although not significant (p = 0.369). We saw

Table 2. Results of multiple regression analyses in the total population.

| Sex*exposure | Boys | Girls |
|--------------|------|-------|
| β | SE | p-Value | β | SE | p-Value |
| lnΣPCBmilk | Composite scale | –5.93 | 2.69 | 0.029 | 2.73 | 1.78 | 0.127 | –3.20 | 2.13 | 0.137 |
| Masculine scale | 4.50 | 2.04 | 0.029 | –2.77 | 1.35 | 0.042 | 1.73 | 1.62 | 0.286 |
| Feminine scale | –1.20 | 1.95 | 0.590 | –0.04 | 1.30 | 0.975 | –1.24 | 1.55 | 0.423 |
| lnΣPCBmaternal | Composite scale | –5.51 | 2.08 | 0.009 | 4.06 | 1.56 | 0.011 | –1.45 | 1.46 | 0.323 |
| Masculine scale | 5.94 | 1.56 | 0.001 | –3.85 | 1.17 | 0.001 | 2.09 | 1.10 | 0.059 |
| Feminine scale | 0.56 | 1.52 | 0.712 | 0.15 | 1.15 | 0.998 | 0.71 | 1.07 | 0.508 |

Effects of prenatal exposure to PCBs on scores on the PSAl scales (composite, masculine, and feminine), adjusted for type of feeding, duration of breast-feeding, sex, parity, parental education level, parental verbal IQ, HOME score, and age at examination. The effect of exposure on the PSAl scores in boys and in girls and the difference between these effects (girls minus boys) are estimated through the interaction term sex*exposure in essentially the same regression model by reparameterizing the sex effect. *Regression coefficient, SE, and p-value of the interaction variable sex*exposure (lnΣPCBmilk or lnΣPCBmaternal) on outcome variable when in the regression model boy is coded 0 and girl = 1, p < 0.05 indicates a significantly different effect of prenatal exposure on PSAl scores between boys and girls. *Regression coefficient of exposure, SE, and p-value, on PSAl scores in boys. * Regression coefficient of exposure, SE, and p-value, on PSAl scores in girls.

Figure 1. Relation in boys (A) (r partial = –0.29) and in girls (B) (r partial = 0.417) between scores on the masculine scale and levels of lnΣPCBmilk, adjusted for confounding variables; partial regression plot.
no sex-specific effects of \( \Sigma \text{PCB}_{\text{milk}} \) on scores on the masculine and feminine scale. Effects of prenatal levels of dioxin, planar and mono-ortho TEQs, total TEQ, and the sum of the 20 nondioxin-like PCBs on play behavior were not significantly different for boys and girls. Prenatal dioxin TEQ levels were significantly related with higher scores on the feminine scale in the total group of boys and girls (\( p = 0.048 \)), indicating more feminized play behavior in both sexes.

Postnatal exposure, through lactation, to \( \Sigma \text{PCB}_{\text{milk}}, \) dioxin, planar and mono-ortho TEQs, total TEQ, and \( \Sigma \text{PCB}_{\text{20 nondioxin}} \) was not related to play behavior in the total BF group nor in boys and girls separately.

### Discussion

In this study we described sex-specific effects of prenatal exposure to PCBs on play behavior in healthy Dutch children at school age. Higher prenatal exposure to PCBs was associated with less masculinized play behavior in boys and with more masculinized play behavior in girls. Effects of prenatal exposure to dioxins were seen on feminine play behavior. In boys as well as in girls, higher prenatal dioxin levels were associated with more feminized play behavior.

Childhood play behavior shows marked sex differences and is likely to be influenced by the prenatal steroid hormone environment. We therefore suggest that these results may indicate behavioral effects of steroid hormone imbalances early in development related to prenatal exposure to PCBs and dioxins, their metabolites, and/or related compounds.

In the Yu-Cheng cohort, researchers observed sex-specific effects of prenatal exposure to high levels of PCBs and PCDFs on the scores on the Raven’s Colored Progressive Matrices (CPM) and Standardized Progressive Matrices (SPM) (11). These tests are considered to be tests for general cognitive development that appeal more on spatial rather than verbal capabilities. Spatial abilities form another domain of nonreproductive sex-specific behaviors that provide evidence for prenatal steroid hormone involvement. In the Yu-Cheng cohort, prenatally exposed boys were affected in their scores on the CPM and SPM tests, whereas in exposed girls no effect was seen. Because boys typically develop better spatial abilities than girls (25,26), these results were interpreted as demasculinizing or feminizing effects caused by disturbances in steroid hormones by prenatal exposure to PCBs/PCDFs (11). On the basis of results of play behavior studies in several groups of children that were prenatally exposed to abnormal levels of endogenous or exogenous steroid hormones, it has been hypothesized that there is evidence for prenatal androgen influences on sexual differentiation of childhood play (16).

Masculinized or defeminized childhood play behavior was reported in genetic females who were exposed to elevated androgens (27,28), whereas demasculinized or feminized play behavior was associated with prenatal exposure to progestogenic compounds that are assumed to interfere with androgen action in genetic females and, more subtly, in genetic males (29).

In adults prenatally exposed to diethylstilbestrol (DES)—a group that might be seen as a model group in studying potential estrogenic effects of prenatal PCB and dioxin exposure—childhood play behavior has been studied retrospectively. Males prenatally exposed to DES recalled slightly more masculinized play behavior than nonexposed controls, assessed by an interview covering childhood play behavior (30). In DES females no difference in childhood play, retrospectively assessed by questionnaires filled out by the DES subjects and their mothers, has been reported (31,32). The effects of prenatal exposure to PCBs and dioxins on childhood play behavior we reported in this study are opposite to the results of these DES studies. This difference in effect can be related to the retrospective nature of these DES studies and to differences in timing and duration of exposure to these chemicals in these groups. Moreover, differences in behavioral effects can be related to the level of exposure, which is likely to be higher in DES-exposed children. Many studies have reported that effects of exposure to hormones and hormone-mimicking chemicals show nonmonotonic dose–response curves, such as U-shaped or inverted U-shaped (33–37).

The current knowledge on the mechanisms of action of PCBs and dioxins and their metabolites, such as hydroxylated PCBs, on prenatal steroid hormone metabolism is still limited. Complex interactions with various steroid hormone systems are suggested, including estrogen and androgen hormone systems (3). These systems can be affected on various levels and estrogenic (38,39), antiestrogenic (8,40–42), and antiandrogenic (7) effects have been described in in vivo and in vitro studies, possibly depending on congener type or metabolites. In this study we lack information on prenatal steroid hormone levels, and although play behavior studies suggest that childhood play behavior is mediated predominantly by prenatal androgen action, our data are insufficient to show that multiple endocrine effects are not involved in the mechanism of action of prenatal exposure to PCBs and dioxins.

In the environment, PCBs and dioxins are present as complex mixtures of various congeners that may vary in metabolism, toxicity, and endocrine-disrupting properties. In this study we measured PCBs 118, 138, 153, and 180 in maternal and cord plasma samples. The sum of these four most abundant congeners constitutes 46% of the total PCBs (43). In the BF group various PCB and dioxin congeners were measured in breast milk. Prenatal levels of \( \Sigma \text{PCB}_{\text{milk}} \) were associated with masculine play behavior, similar to what was seen using maternal and cord \( \Sigma \text{PCB} \) levels as prenatal exposure levels. Dioxin exposure was related with more feminine play behavior. Nondioxin-like PCB levels and dioxin-like PCB and total TEQ levels were not significantly associated with play behavior. Whether these results reflect effects that are specific to PCB or dioxin congeners or the limited power of analyses in this subgroup of BF children.

### Table 3. Results of multiple regression analyses in the BF group.

| Sex*exposure | Boys | Girls | Total BF group |
|-------------|------|-------|----------------|
| lnPCBmilk | \( \beta \) | SE | \( p \)-value | \( \beta \) | SE | \( p \)-value | \( \beta \) | SE | \( p \)-value |
| Composite scale | –9.53 | 3.98 | 0.020 | 2.15 | 2.37 | 0.369 | –7.39 | 3.29 | 0.028 |
| Masculine scale | 4.94 | 3.14 | 0.121 | –0.07 | 1.87 | 0.970 | 4.86 | 2.59 | 0.085 |
| Feminine scale | –4.40 | 2.71 | 0.094 | 2.08 | 1.62 | 0.203 | –2.3 | 0.24 | 0.263 |
| lnDioxinTEQ | Composite scale | –2.85 | 4.69 | 0.546 | 6.19 | 3.56 | 0.088 | 3.34 | 2.77 | 0.312 | 4.63 | 2.46 | 0.066 |
| Masculine scale | 1.70 | 3.77 | 0.653 | –2.18 | 2.87 | 0.449 | –0.48 | 2.63 | 0.856 | –1.25 | 1.98 | 0.529 |
| Feminine scale | –1.15 | 3.18 | 0.720 | 4.00 | 2.42 | 0.103 | 2.86 | 2.22 | 0.203 | 3.38 | 1.67 | 0.048 |

**Effects of prenatal exposure to PCBs on scores on the PSAI scales (composite, masculine, and feminine), adjusted for type of feeding, duration of breast-feeding, sex, parity, parental education level, parental verbal IQ, HOME score, and age at examination. The effect of exposure on the PSAI scores in boys and in girls and the difference between these effects (girls minus boys) are estimated through the interaction term sex*exposure in essentially the same regression model by reparameterizing the sex effect.**

*Regression coefficient, SE, and \( p \)-value of the interaction variable sex*exposure (lnPCBmilk or lnDioxinTEQ) on outcome variable when in the regression model boy is coded 0, and girl = 1; \( p < 0.05 \) indicates a significantly different effect of prenatal exposure on PSAI scores between boys and girls. **Regression coefficient of exposure, SE, and \( p \)-value on PSAI scores in boys. ***Regression coefficient of exposure, SE, and \( p \)-value on PSAI scores in girls. ****Regression coefficient of exposure, SE, and \( p \)-value on PSAI in the total BF group, not including the sex*exposure interaction term in the regression model.
cannot be concluded from these results. Moreover, total TEQ levels, the sum of the nondioxidin-like PCBs, and the four PCBs in breast milk and maternal and cord plasma correlated highly with each other (20).

Play behavior in our study was not associated with postnatal exposure to PCBs and dioxins through breast-feeding. We therefore suggest that childhood play behavior is sensitive to endocrine-disrupting behavioral effects of exposure to PCBs and dioxins early in development, as is supported in females with congenital adrenal hyperplasia (28) and by studies in other mammals (44,45).

In conclusion, this is the first behavioral study in humans to show effects of prenatal exposure to environmental levels of PCBs and dioxins on behavior that shows marked sex differences. Moreover, sex-specific effects of background prenatal exposure to PCBs have not been reported previously in human PCB studies. The results of this exploratory study give evidence for steroid hormone involvement in the neurotoxic mechanism of action of prenatal exposure to environmental levels of PCBs, dioxins, and other related organochlorine compounds. Evaluation of the relation between prenatal steroid hormone status and PCB and dioxin exposure is needed to further confirm these findings; in addition, follow-up of this cohort will be necessary to assess potential implications of these results on later development.

Appendix

Pre-School Activity Inventory

Copyright Susan Golombok and John Rust (22)

Name:
Age:
Sex: M/F (delete as appropriate)

Instructions
This inventory is about everyday activities of preschool children. It is in three sections: toy preferences, activities, and characteristics. Each question asks how frequently the child plays with particular toys, engages in particular activities or shows particular characteristics. There are five possible answers: (N) Never, (HE) Hardly Ever, (S) Sometimes, (O) Often, or (VO) Very Often. Answer each question by circling the response which best describes the child.

[e.g., N HE S O VO]

Please answer all of the questions. If you are unsure about which response best describes the child for any of the questions then please answer according to the response which seems most appropriate.

[Key: (N) Never, (HE) Hardly Ever, (S) Sometimes, (O) Often, or (VO) Very Often]

Part 1: TOYS: Please answer the questions according to how often the child played with the following toys during the past months.

1. Guns (or used objects as guns) N HE S O VO
2. Jewelry N HE S O VO
3. Tool set N HE S O VO
4. Dolls, doll’s clothes, or doll’s carriages N HE S O VO
5. Trains, cars, or airplanes N HE S O VO
6. Swords (or used objects as swords) N HE S O VO
7. Tea set N HE S O VO

Part 2: ACTIVITIES: Please answer these questions according to how often the child engaged in the following activities during the past month.

1. Playing house (e.g., cleaning, cooking) N HE S O VO
2. Playing with girls N HE S O VO
3. Pretending to be a female character (e.g., princess) N HE S O VO
4. Playing at having a male occupation (e.g., soldier) N HE S O VO
5. Fighting N HE S O VO
6. Pretending to be a family character N HE S O VO
7. Sports and ball games N HE S O VO
8. Climbing (e.g., fences, trees, gym equipment) N HE S O VO
9. Playing at taking care of babies N HE S O VO
10. Showing all interest in real cars, trains, and equipment N HE S O VO
11. Dressing up in girlish clothes N HE S O VO

Part 3: CHARACTERISTICS: Please answer questions according to how often the child shows the following characteristics:

1. Likes to explore new surroundings N HE S O VO
2. Enjoys rough and tumble play N HE S O VO
3. Shows interest in snakes, spiders, or insects N HE S O VO
4. Avoids getting dirty N HE S O VO
5. Likes pretty things N HE S O VO
6. Avoids taking risks N HE S O VO

NOW PLEASE CHECK THAT YOU HAVE ANSWERED ALL THE QUESTIONS.
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Kids, Questions, Clarification?...

No, we don’t have all the answers to pediatric environmental health questions, but we can provide answers to many of the medical, exposure and risk questions.

The Pediatric Environmental Health Specialty Units (PEHSU) offer clinical consultation and education for the health professional needing information about children and families with environmental exposures.

The Association of Occupational and Environmental Clinics (AOEC) sponsored Pediatric Environmental Health Specialty Units provide:

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