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Exposure to Environmental Tobacco Smoke and Cognitive Abilities Among U.S. Children and Adolescents

In the article "Exposure to Environmental Tobacco Smoke and Cognitive Abilities Among U.S. Children and Adolescents," Yolton et al. (2005) stated that the data "indicate an inverse association between ETS exposure and cognitive deficits among children …." They do not. They indicate an inverse association between ETS exposure and scores, but a direct association between ETS exposure and cognitive deficits.

The author declares he has no competing financial interests.

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Reference

Yolton K, Dietrich K, Auinger P, Lanphear BP, Hornung R. 2005. Exposure to environmental tobacco smoke and cognitive abilities among U.S. children and adolescents. Environ Health Perspect 113:113:98–103.

Editor's note: In accordance with journal policy, Yolton et al. were asked whether they wanted to respond to this letter, but they chose not to do so.

Maternal and Paternal Risk Factors for Hypospadias

Hypospadias is a common congenital anomaly, characterized by incomplete fusion of the urethral folds during fetal development, which results in the urethra opening on the ventral surface of the penis or on the scrotum. In their article, Pierik et al. (2004) proposed multiple possible maternal and paternal risk factors related to the development of isolated hypospadias, including genetic, endocrine, and environmental factors.

Concerns and alarms have been raised about the potential effects of endocrine disruptors, which include derivatives of polyaromatic hydrocarbons and pesticides, on the developing male reproductive tract. We have recently published two case–control studies on risk factors and hypospadias, one on a possible association with maternal age (Bianca et al. 2005) and the other on the role of endocrine disruptors (Bianca et al. 2003).

In the first study (Bianca et al. 2005), we evaluated 415 newborns with isolated hypospadias and 812 controls. Our results suggest that an increased risk for hypospadias exists in women at the extremes of the age distribution (< 20 years and > 40 years; p = 0.000 and 0.026, respectively) relative to women in the middle of the distribution, with a mechanism probably related to hormonal disruption. It has been postulated that changes in concentrations of sex hormones during the fetal critical period of genital development (weeks 8–14), caused by endogenous or exogenous factors, may play a role in the development of hypospadias and that hypospadias could be associated with early malfunction of the placenta, resulting in decreased secretion of placental and fetal hormones that could in turn disturb fetal development (Akre et al. 1999). Mothers at the extremes of the age distribution may be more susceptible to this hormonal disruption. The association between hypospadias and maternal age, both for younger and older women, might be explained as a "defect in nature's quality control" caused by a reduction of defensive maternal mechanisms that would be less efficient in the elimination of malformed fetuses in the mothers of hypospadias cases. This hypothesis may also apply to other birth defects where a relationship with maternal age has been demonstrated.

In the second study of 68 cases and 211 controls (Bianca et al. 2003), we identified a high incidence of hypospadias in two towns in southeastern Sicily, which have intense industrial (Augusta) and agricultural (Vittoria) activities. Our results showed an incidence that was 3.8 [95% confidence interval (CI), 2.16–6.14] and 2.3 times (95% CI, 1.48–3.43) higher, respectively, than expected (3.2 per 1,000 male live births in southeastern Sicily). The odds ratios for fathers' job exposure alone were 5.5 (95% CI, 1.22–24.7) for working in an oil refinery in Augusta and 2.9 (95% CI, 1.01–8.55) for working in hothouses in Vittoria.

Pollutants in both areas include compounds with proven estrogenic activity and other chemicals: pesticides and herbicides and their metabolites; dieldrin, chlordane, and endosulfan; polychlorinated biphenyls and dioxins; bisphenols used in epoxy resins; and some phthalates used as plasticizers in a wide variety of applications, including polyvinyl compounds and alkyphenol ethoxylates. The direct cause–effect relationship between environmental pollutants, which act as endocrine disruptors, and the increased incidence of hypospadias in these areas is difficult to establish and demonstrate. Our study (Bianca et al. 2003), suggested that exposure to large amounts of industrial and agricultural pollutants is sufficient to increase the risk of hypospadias.

Other risk factors, such as reproductive history, may be involved in the etiology of hypospadias. Disturbances in sexual differentiation occur when endogenous and/or exogenous factors act to disrupt the metabolism of gonadal hormones during development.

Further epidemiologic and biologic studies are needed to explain, in a multifactorial model, which factors (genetic and/or environmental) interact and influence the etiology of this congenital abnormality.

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Editor's note: In accordance with journal policy, Pierik et al. were asked whether they wanted to respond to this letter, but they chose not to do so.

Errata

In Table 1 of "Estimating the Exposure–Response Relationships between Particulate Matter and Mortality within the APHEA Multicity Project" by Samoli et al. [Environ Health Perspect 113:88–95 (2005)], the values for CVD deaths are incorrect. The corrected table is shown below. The authors apologize for the errors.

Table 1. City descriptive data on the study period, population, exposure (PM_{10} and BS), outcome (daily number of deaths), and selected effect modifiers (region, mean temperature, mean NO_2 over 24 hr, and directly standardized mortality rate).

City	Study period (month/year)	Population (× 1,000)	No. of deaths per day			PM_{10} (μg/m^3) percentile		BS (μg/m^3) percentile		Geographic region	Mean temperature	NO_2 (24-hr)	SDR
			Total	CVD	Respiratory	50th	90th	50th	90th				
Athens	1/92–12/96	3,073	73	36	5	40[a]	59	64	122	South	18	74	784
Barcelona	1/91–12/96	1,644	40	16	4	60	95	39	64	South	16	69	740
Basel	1/90–12/95	360	9	4	1	28[a]	55			West	11	38	678
Bilbao	4/92–3/96	667	15	5	1			23	39	South	15	49	711
Birmingham	1/92–12/96	2,300	61	28	9	21	40	11	22	West	10	46	895
Budapest	1/92–12/95	1,931	80	40	3	40[a]	52			East	11	76	1,136
Cracow	1/90–12/96	746	18	10	0	54[a]	86	36	101	East	8	44	1,009
Dublin	1/90–12/96	482	13	6	2			10	26	West	10	—	940
Erfurt	1/91–12/95	216	6	—	—	48	98			West	9	40	972
Geneva	1/90–12/95	317	6	2	0	33[a]	71			West	10	45	608
Helsinki	1/93–12/96	828	18	9	2	23[a]	49			West	6	33	915
Ljubljana	1/92–12/96	322	7	3	0			13	42	East	11	46	823
Lodz	1/90–12/96	828	30	17	1			30	77	East	8	39	1,231
London	1/92–12/96	6,905	169	71	29	25	46	11	22	West	12	61	851
Lyon	1/93–12/97	416	9	3	1	39	63			West	12	63	579
Madrid	1/92–12/95	3,012	61	22	6	33	59			South	15	70	636
Marseille	1/90–12/95	855	22	8	2			34	56	West	16	71	666
Milan	1/90–12/96	1,343	29	11	2	47[a]	88			West	14	94	632
Netherlands	1/90–9/95	15,400	342	140	29	34	67	63	122	West	10	43	757
Paris	1/92–12/96	6,700	124	38	9	22	46	21	45	West	12	53	644
Poznan	1/90–12/96	582	17	9	1			23	76	East	9	47	1,106
Prague	2/92–12/95	1,213	38	22	1	66	124			East	10	58	984
Rome	1/92–12/96	2,775	56	23	3	57[a]	81			South	17	88	585
Stockholm	1/94–12/96	1,126	30	15	3	14	27			West	8	26	666
Tel Aviv	1/93–12/96	1,141	27	12	2	43	75			South	20	70	430
Teplice	1/90–12/97	625	18	10	1	42	83			East	9	32	1,173
Torino	1/90–12/96	926	21	9	1	65[a]	129			West	14	76	724
Valencia	1/94–12/96	753	16	6	2			40	70	South	19	66	820
Wroclaw	1/90–12/96	643	15	9	1			33	97	East	9	27	970
Zurich	1/90–12/95	540	13	6	1	28[a]	54			West	11	40	666

Abbreviations: —, no data; CVD, cardiovascular deaths; SDR, directly standardized mortality rate. Mean temperature in degrees centigrade.
[a]PM_{10} were estimated using a regression model relating collocated PM_{10} measurements to the BS or total suspended particles.

Bonner et al. would like to correct a factual error in "Occupational Exposure to Carbofuran and the Incidence of Cancer in the Agricultural Health Study" [Environ Health Perspect 113:285–289 (2005)]. In the second paragraph of the introduction, the fourth sentence was incorrect. The two studies cited demonstrated that another carbamate pesticide, carbendazim, and not carbofuran, induced lymphoma. The sentence should read: "While two studies demonstrated that the carbamate pesticide carbendazim was able to induce lymphoma in Swiss mice (Borzsonyi and Pinter 1977; Borzsonyi et al. 1976), carcinogenicity of carbofuran was not evident in several 2-year dietary studies conducted on rats (Gupta 1994)."

Despite the error, the authors stand by the validity of the analysis and the interpretation of the results. The authors apologize for the error.

The March Focus article ["Great Lakes: Resource at Risk," Environ Health Perspect 113:A164–A173 (2005)] stated that Dow Chemical released about 400 tons of mercury into Lake Superior from two chloralkali plants. In fact, these two plants were located in Sarnia, Ontario. Thus, the discharges were made into Lake Huron. *EHP* regrets the error.