Selected Recent References:
Some ambient pollutants and human health effects

AIR PARTICLES AND INCREASED RISK OF SUDDEN DEATH

Result <1>
Unique Identifier
18192596
Status
MEDLINE
Authors
Ballester F. Medina S. Boldo E. Goodman P. Neuberger M. Iniguez C. Kunzli N. Apheis network.
Authors Full Name
Ballester, Ferran. Medina, Sylvia. Boldo, Elena. Goodman, Pat. Neuberger, Manfred. Iniguez, Carmen. Kunzli, Nino. Apheis network.
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Title
Reducing ambient levels of fine particulates could substantially improve health: a mortality impact assessment for 26 European cities.
Source
Journal of Epidemiology & Community Health. 62(2):98-105, 2008 Feb.
Abstract
Recently new European policies on ambient air quality--namely, the adoption of new standards for fine particulate matter (PM(2.5)), have generated a broad debate about choosing the air quality standards that can best protect public health. The Apheis network estimated the number of potential premature deaths from all causes that could be prevented by reducing PM(2.5) annual levels to 25 microg/m3, 20 microg/m3, 15 microg/m3 and 10 microg/m3 in 26 European cities. The various PM(2.5) concentrations were chosen as different reductions based on the limit values proposed by the new European Directive, the European Parliament, the US Environmental Protection Agency and the World Health Organization, respectively. The Apheis network provided the health and exposure data used in this study. The concentration-response function (CRF) was derived from the paper by Pope et al (2002). If no direct PM(2.5) measurements were available, then the PM(10) measurements were converted to PM(2.5) using a local or an assumed European conversion factor. We performed a sensitivity analysis using assumptions for two key factors--namely, CRF and the conversion factor for PM(2.5). Specifically, using the “at least” approach, in the 26 Apheis cities with more than 40 million inhabitants, reducing annual mean levels of PM(2.5) to 15 microg/m3 could lead to a reduction in the total burden of mortality among people aged 30 years and over that would be four times greater than the reduction in mortality that could be achieved by reducing PM(2.5) levels to 25 microg/m3 (1.6% vs 0.4% reduction) and two times greater than a reduction to 20 microg/m3. The percentage reduction could grow by more than seven times if PM(2.5) levels were reduced to 10 microg/m3 (3.0% vs

0.4%). This study shows that more stringent standards need to be adopted in Europe to protect public health, as proposed by the scientific community and the World Health Organization.
Publication Type
Journal Article. Multicenter Study. Research Support, Non-U.S. Gov't.

Result <2>
Unique Identifier
18308966
Status
MEDLINE
Authors
Fullerton DG. Semple S.
Authors Full Name
Fullerton, D G. Semple, S.
Title
Air pollution and health: indoor air pollution in the developing world is the real key to reducing the burden of ill health.[comment].
Comments
Comment on: Thorax. 2007 Sep;62(9):748-9; PMID: 17726169
Source
Thorax. 63(3):288; author reply 288, 2008 Mar.
Publication Type
Comment. Letter.

Result <3>
Unique Identifier
18197308
Status
MEDLINE
Authors
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Title
Air pollution and postneonatal infant mortality in the United States, 1999-2002.
Source
Environmental Health Perspectives. 116(1):110-5, 2008 Jan.
Abstract
OBJECTIVE: Our goal was to evaluate the relationship between cause-specific postneonatal infant mortality and chronic early-life exposure to particulate matter and gaseous air pollutants

across the United States. METHODS: We linked county-specific monitoring data for particles with aerodiameter of < or = 2.5 microm (PM2.5) and < or = 10 microm (PM10), ozone, sulfur dioxide, and carbon monoxide to birth and death records for infants born from 1999 to 2002 in U.S. counties with > 250,000 residents. For each infant, we calculated the average concentration of each pollutant over the first 2 months of life. We used logistic generalized estimating equations to estimate odds ratios of postneonatal mortality for all causes, respiratory causes, sudden infant death syndrome (SIDS), and all other causes for each pollutant, controlling for individual maternal factors (race, marital status, education, age, and primiparity), percentage of county population below poverty, region, birth month, birth year, and other pollutants. This analysis includes about 3.5 million births, with 6,639 postneonatal infant deaths. RESULTS: After adjustment for demographic and other factors and for other pollutants, we found adjusted odds ratios of 1.16 [95% confidence interval (CI), 1.06-1.27] for a 10-mug/m3 increase in PM10 for respiratory causes and 1.20 (95% CI, 1.09-1.32) for a 10-ppb increase in ozone and deaths from SIDS. We did not find relationships with other pollutants and for other causes of death (control category). CONCLUSIONS: This study supports particulate matter air pollution being a risk factor for respiratory-related postneonatal mortality and suggests that ozone may be associated with SIDS in the United States.
Publication Type Journal Article.

Result <4>
Unique Identifier
18285643
Status
MEDLINE
Authors
Chen G. Song G. Jiang L. Zhang Y. Zhao N. Chen B. Kan H.
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Chen, Guohai. Song, Guixiang. Jiang, Lili. Zhang, Yunhui. Zhao, Naiqing. Chen, Bingheng. Kan, Haidong.
Institution
Shanghai Environmental Monitoring Center, School of Public Health, Fudan University, China.
Title
Short-term effects of ambient gaseous pollutants and particulate matter on daily mortality in Shanghai, China.
Source
Journal of Occupational Health. 50(1):41-7, 2008 Jan.
Abstract
Identification of the specific pollutants contributing most to the health hazard of the air pollution mixture may have important implications for environmental and social policies. In the current study, we conducted a time-series analysis to examine the specific effects of major air pollutants [particulate matter less than 10 microns in diameter (PM(10)), sulfur dioxide (SO(2)), and nitrogen dioxides (NO(2))] on daily mortality in Shanghai, China, using both single-pollutant and multiple-pollutant models. In the single-pollutant models, PM(10), SO(2), and NO

(2) were found to be associated with mortality from both all non-accidental causes and from cardiopulmonary diseases. Unlike some prior studies in North America, we found a significant effect of gaseous pollutants (SO(2) and NO(2)) on daily mortality even after adjustment for PM (10) in the multiple-pollutant models. Our findings, combined with previous Chinese studies showing a consistent, significant effect of gaseous !
pollutants on mortality, suggest that the role of outdoor exposure to SO(2) and NO(2) should be investigated further in China.
Publication Type
Journal Article. Research Support, Non-U.S. Gov't.

Result <5>
Unique Identifier
17700248
Status
MEDLINE
Authors
Wong CM. Ou CQ. Lee NW. Chan KP. Thach TQ. Chau YK. Ho SY. Hedley AJ. Lam TH.
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Wong, Chit-Ming. Ou, Chun-Quan. Lee, Nga-Wing. Chan, King-Pan. Thach, Thuan-Quoc. Chau, Yuen-Kwan. Ho, Sai-Yin. Hedley, Anthony Johnson. Lam, Tai-Hing.
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Title
Short-term effects of particulate air pollution on male smokers and never-smokers.
Source
Epidemiology. 18(5):593-8, 2007 Sep.
Abstract
BACKGROUND: Numerous studies have shown that ambient air pollution and smoking are both associated with increased mortality, but until now there has been little evidence as to whether the effects of these 2 factors combined are greater than the sum of their individual effects. We assessed whether smokers are subject to additional mortality risk from air pollution relative to never-smokers. METHODS: This study included 10,833 Chinese men in Hong Kong who died at the age of 30 or above during the period 1 January to 31 December 1998. Relatives who registered for deceased persons were interviewed about the deceased's smoking history and other personal lifestyle factors about 10 years before death. Poisson regression for daily number of deaths was fitted to estimate excess risks per 10 microg/m increase in particulate matter with aerodynamic diameter <10 microm (PM10) in male smokers and never-smokers in stratified data, and additional excess risk for smokers relative to never-smokers in combined data. RESULTS: In smokers there was a significant excess risk associated with PM10 for all natural causes and cardio-respiratory diseases for men age 30 years or older and men 65 or older. For all natural causes, greater excess risk associated with PM10 was observed for smokers relative to never-smokers: 1.9% (95% confidence interval = 0.3% to 3.6%) in men age 30 and older and 2.3% (0.4% to 4.3%) in those age 65 and older. CONCLUSIONS: Ambient particulate air

pollution is associated with greater excess mortality in male smokers compared with never-smokers.
Publication Type
Comparative Study. Journal Article. Research Support, Non-U.S. Gov't.

Result <6>
Unique Identifier
17425638
Status
MEDLINE
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Title
Time-varying coefficient models for the analysis of air pollution and health outcome data.
Source
Biometrics. 63(4):1253-61, 2007 Dec.
Abstract
In this article a time-varying coefficient model is developed to examine the relationship between adverse health and short-term (acute) exposure to air pollution. This model allows the relative risk to evolve over time, which may be due to an interaction with temperature, or from a change in the composition of pollutants, such as particulate matter, over time. The model produces a smooth estimate of these time-varying effects, which are not constrained to follow a fixed parametric form set by the investigator. Instead, the shape is estimated from the data using penalized natural cubic splines. Poisson regression models, using both quasi-likelihood and Bayesian techniques, are developed, with estimation performed using an iteratively re-weighted least squares procedure and Markov chain Monte Carlo simulation, respectively. The efficacy of the methods to estimate different types of time-varying effects are assessed via a simulation study, and the models are then applied to data from four cities that were part of the National Morbidity, Mortality, and Air Pollution Study.
Publication Type
Journal Article.

Result <7>
Unique Identifier
17766181
Status
MEDLINE
Authors

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Title
Fine particles, a major threat to children. [Review] [66 refs]
Source
International Journal of Hygiene & Environmental Health. 210(5):617-22, 2007 Oct.
Abstract
BACKGROUND: There is a growing body of evidence for serious health consequences of exposure to ambient air pollution. The general question of who is susceptible is one of the most important gaps in current knowledge regarding particulate matter (PM)-related health effects. Who is susceptible depends on the specific health endpoint being evaluated and the level and length of exposure. Here, we restrict the review on the impact of fine particle exposure on children's health to the following outcomes: infant death, lung function, respiratory symptoms and reproductive outcomes. METHODS: This is a strategic review of children's susceptibility to ambient fine particles and characteristics of infant and children which underlie their increased susceptibility to PM. RESULTS: Ambient fine PM is associated with intra-uterine growth retardation, infant mortality; it is associated with impaired lung function and increased respiratory symptoms, particularly in asthmatics. Concerning infant mortality, exposure to PM is strongly and consistently associated with postneonatal respiratory mortality and less consistently with sudden infant death syndrome. Although most of the studies reported adverse effects for this health outcome, the evidence is weaker than for infant death. Exposure to fine PM has been associated with impaired lung function and lung function growth. Most of the studies reported increased prevalence of symptom with increased exposure to fine PM. CONCLUSION: Fine PM is a major threat to children, because of their higher exposure to PM compared to adults, the immature state of the lung in childhood and also of the immune function at birth. The first months of life might be a period of particular sensitivity. Although the mechanisms of air pollution effects have not yet been completely understood, pregnant women, infants and children need specific protection against exposure to fine particles. [References: 66]
Publication Type
Journal Article. Review.

Result <8>
Unique Identifier
18080899
Status
MEDLINE
Authors
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Title
Evaluation and identification of priority air pollutants for environmental management on the basis of risk analysis in Russia.
Source
Journal of Toxicology & Environmental Health Part A. 71(1):86-91, 2008 Jan.
Abstract
Since 1997, more than 30 health-risk analyses were conducted using Russian data sets. These studies demonstrated that air pollution is the most important environmental contributor toward morbidity and mortality risk in Russia, with 90% of the total human health risk coming from the criteria pollutants total suspended particulate (TSP), SO2, and NO(x). This article contributes to the ongoing discussion of the magnitude of this health issue in Russia by providing an estimate of both the mortality rate attributed to airborne pollutants and the associated economic damages. The 90% confidence interval of mortality is 46,000-132,000, and the associated economic damages are between 2.6 and 6.5% of gross domestic product (GDP). The largest source of uncertainty in mortality is the concentration-response parameter, accounting for 50-60% of the total variability in the estimate. The point estimate of 87,000 implies that mortality due to airborne pollutants is threefold higher than reported due to tuberculosis, twofold due to transportation accidents, and about the same as that from suicide and homicide combined. By 2002 there was enough evidence regarding potential health hazard and air pollution exposure in Russia to start environmental management reform. In 2004 Russia officially adopted guidelines for health risk analysis associated with air pollution. The next step is to use this health-risk assessment approach as a lead for sensible reforms of the emissions-permit system and environmental finance.
Publication Type
Journal Article.

Result <9>
Unique Identifier
17998361
Status
MEDLINE
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Title

Indoor air pollution: a poverty-related cause of mortality among the children of the world. [Review] [50 refs]
Source
Chest. 132(5):1615-23, 2007 Nov.
Abstract
This article reviews the research on the relation between indoor air pollution exposure and acute respiratory infection (ARI) in children in developing countries. ARI is a cause of death globally, causing approximately 19% of all deaths before the age of 5 years, according to a World Health Organization estimate. Indoor air pollution from biomass fuels, which is strongly poverty related, has long been regarded as an important risk factor for ARI morbidity and mortality. The empirical base for this view is comparatively narrow, with few empirical studies in relation to the magnitude of the global public health importance of the problem. Most existing reports consistently indicate that indoor air pollution is indeed a risk factor for ARI, but studies are generally small and use indirect indicators of pollution, such as use of biomass fuel or type of stove. Exposure assessment for indoor air pollution in developing countries is recognized as a major obstacle because of high cost and infrastructural limitations to chemical pollution sampling. Use of proxy indicators without measurement support may increase the risk of both misclassification of exposure and of confounding by other poverty-related factors. The issue of sufficient sample size further underlines the need for decisions to invest in this research field. Areas where further research is needed also include exploring qualitatively options for interventions that are culturally and economically acceptable to local communities. [References: 50]
Publication Type
Journal Article. Review.

Result <10>
Unique Identifier
17886048
Status
MEDLINE
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Title
Mortality effects of longer term exposures to fine particulate air pollution: review of recent epidemiological evidence. [Review] [43 refs]
Source
Inhalation Toxicology. 19 Suppl 1:33-8, 2007.
Abstract

This article evaluates the dynamic exposure-response relationship between particulate matter air pollution (PM) and mortality risk by integrating epidemiological evidence from studies that use different time scales of exposure. The evidence suggests that short-term exposure studies are observing more than just harvesting or mortality displacement. There is little evidence of short-term compensatory reduction in deaths, and estimated PM effects are generally larger for intermediate and longer term time scales of exposure. Although proximity in time matters, with most recent exposure having the largest health impact, there is evidence that the short-term exposure studies capture only a small amount of the overall health effects of long-term repeated exposure to PM. The overall epidemiological evidence suggests that adverse health effects are dependent on both exposure concentrations and length of exposure, and that long-term exposures have larger, more persistent cumulative effects than short-term exposures. [References: 43]
Publication Type
Journal Article. Research Support, Non-U.S. Gov't. Review.

Result <11>
Unique Identifier
17761602
Status
MEDLINE
Authors
Kabir Z.
Authors Full Name
Kabir, Zubair.
Title
Decrease in U.S. deaths from coronary disease.[comment].
Comments
Comment on: N Engl J Med. 2007 Jun 7;356(23):2388-98; PMID: 17554120
Source
New England Journal of Medicine. 357(9):941; author reply 941, 2007 Aug 30.
Publication Type
Comment. Letter.

Result <12>
Unique Identifier
17630372
Status
MEDLINE
Authors
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Institution

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Title
Outdoor air pollution and infant mortality: analysis of daily time-series data in 10 English cities.
Source
Journal of Epidemiology & Community Health. 61(8):719-22, 2007 Aug.
Abstract
BACKGROUND: There is growing concern that moderate levels of outdoor air pollution may be associated with infant mortality, representing substantial loss of life-years. To date, there has been no investigation of the effects of outdoor pollution on infant mortality in the UK. METHODS: Daily time-series data of air pollution and all infant deaths between 1990 and 2000 in 10 major cities of England: Birmingham, Bristol, Leeds, Liverpool, London, Manchester, Middlesbrough, Newcastle, Nottingham and Sheffield, were analysed. City-specific estimates were pooled across cities in a fixed-effects meta-regression to provide a mean estimate. RESULTS: Few associations were observed between infant deaths and most pollutants studied. The exception was sulphur dioxide (SO2), of which a 10 mug/m(3) increase was associated with a RR of 1.02 (95% CI 1.01 to 1.04) in all infant deaths. The effect was present in both neonatal and postneonatal deaths. CONCLUSIONS: Continuing reductions in SO2 levels in the UK may yield additional health benefits for infants.
Publication Type Journal Article.

Result <13>
Unique Identifier
17366813
Status
MEDLINE
Authors
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Title
The effects of components of fine particulate air pollution on mortality in california: results from CALFINE.
Source
Environmental Health Perspectives. 115(1):13-9, 2007 Jan.
Abstract
OBJECTIVE: Several epidemiologic studies provide evidence of an association between daily mortality and particulate matter < 2.5 pm in diameter (PM2.5). Little is known, however, about the relative effects of PM2.5 constituents. We examined associations between 19 PM2.5

components and daily mortality in six California counties. DESIGN: We obtained daily data from 2000 to 2003 on mortality and PM2.5 mass and components, including elemental and organic carbon (EC and OC), nitrates, sulfates, and various metals. We examined associations of PM2.5 and its constituents with daily counts of several mortality categories: all-cause, cardiovascular, respiratory, and mortality age > 65 years. Poisson regressions incorporating natural splines were used to control for time-varying covariates. Effect estimates were determined for each component in each county and then combined using a random-effects model. RESULTS: PM2.5 mass and several constituents were associated with multiple mortality categories, especially cardiovascular deaths. For example, for a 3-day lag, the latter increased by 1.6, 2.1, 1.6, and 1.5% for PM2.5, EC, OC, and nitrates based on interquartile ranges of 14.6, 0.8, 4.6, and 5.5 pg/m(3), respectively. Stronger associations were observed between mortality and additional pollutants, including sulfates and several metals, during the cool season. CONCLUSION: This multicounty analysis adds to the growing body of evidence linking PM2.5 with mortality and indicates that excess risks may vary among specific PM2.5 components. Therefore, the use of regression coefficients based on PM2.5 mass may underestimate associations with some PM2.5 components. Also, our findings support the hypothesis that combustion-associated pollutants are particularly important in California.
Publication Type Journal Article.

Result <14>
Unique Identifier
17343725
Status
MEDLINE
Authors
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Title
Does respiratory health contribute to the effects of long-term air pollution exposure on cardiovascular mortality?.
Source
Respiratory Research. 8:20, 2007.
Abstract
BACKGROUND: There is growing epidemiological evidence that short-term and long-term exposure to high levels of air pollution may increase cardiovascular morbidity and mortality. In addition, epidemiological studies have shown an association between air pollution exposure and respiratory health. To what extent the association between cardiovascular mortality and air pollution is driven by the impact of air pollution on respiratory health is unknown. The aim of

this study was to investigate whether respiratory health at baseline contributes to the effects of long-term exposure to high levels of air pollution on cardiovascular mortality in a cohort of elderly women. METHOD: We analyzed data from 4750 women, aged 55 at the baseline investigation in the years 1985-1994. 2593 of these women had their lung function tested by spirometry. Respiratory diseases and symptoms were asked by questionnaire. Ambient air pollution exposure was assessed by the concentrations of NO2 and total suspended particles at fixed monitoring sites and by the distance of residency to a major road. A mortality follow-up of these women was conducted between 2001 and 2003. For the statistical analysis, Cox' regression was used. RESULTS: Women with impaired lung function or pre-existing respiratory diseases had a higher risk of dying from cardiovascular causes. The impact of impaired lung function declined over time. The risk ratio (RR) of women with forced expiratory volume in one second (FEV1) of less than 80% predicted to die from cardiovascular causes was RR = 3.79 (95%CI: 1.64-8.74) at 5 years survival time and RR = 1.35 (95%CI: 0.66-2.77) at 12 years. The association between air pollution levels and cardiovascular death rate was strong and statistically significant. However, this association did only change marginally when including indicators of respiratory health into the regression analysis. Furthermore, no interaction between air pollution and respiratory health on cardiovascular mortality indicating a higher risk of those with impair ed respiratory health could be detected. CONCLUSION: Respiratory health is a predictor for cardiovascular mortality. In women followed about 15 years after the baseline investigation at age 55 years long-term air pollution exposure and impaired respiratory health were independently associated with increased cardiovascular mortality.
Publication Type Comparative Study. Journal Article. Research Support, Non-U.S. Gov't.

Result <15>
Unique Identifier
17360888
Status
MEDLINE
Authors
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Title
Use of biomass fuel is associated with infant mortality and child health in trend analysis.
Source
American Journal of Tropical Medicine & Hygiene. 76(3):585-91, 2007 Mar.
Abstract
Biomass fuel used for cooking results in widespread exposure to indoor air pollution (IAP), affecting nearly 3 billion people throughout the world. Few studies, however, have tested for an exposure-response relationship between biomass fuel and health outcomes. The aim of this study

was to explore the relationship between biomass fuel, infant mortality, and children's respiratory symptoms. Eighty households in a rural community in Ecuador were selected based on their use of biomass fuel and questioned regarding a history of infant mortality and children's respiratory symptoms. Carbon monoxide (CO) and particulate matter (PM) were measured in a subset of these homes to confirm the relationship between biomass fuel use and IAP. Results showed a significant trend for higher infant mortality among households that cooked with a greater proportion of biomass fuel (P=0.008). Similar trends were noted for history of cough (P=0.02) and earache (P<0.001) among children living in these households.
Publication Type
Journal Article.

Result <16>
Unique Identifier
16847936
Status
MEDLINE
Authors
Forastiere F. Stafoggia M. Tasco C. Picciotto S. Agabiti N. Cesaroni G. Perucci CA.
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Title
Socioeconomic status, particulate air pollution, and daily mortality: differential exposure or differential susceptibility.
Source
American Journal of Industrial Medicine. 50(3):208-16, 2007 Mar.
Abstract
BACKGROUND: Short-term increases in particulate air pollution are linked with increased daily mortality and morbidity. Socioeconomic status (SES) is a determinant of overall health. We investigated whether social class is an effect modifier of the PM(10) (particulate matter with diameter <10 micron)-daily mortality association, and possible mechanisms for this effect modification. METHODS: Area-based traffic emissions, income, and SES were available for each resident in Rome. All natural deaths (83,253 subjects) occurring in Rome among city residents (aged 35+ years) during the period 1998-2001 were identified. For each deceased individual, all the previous hospitalizations within 2 years before death were available via a record linkage procedure. PM(10) daily data were available from two urban monitoring sites. A case-crossover analysis was utilized in which control days were selected according to the time stratified approach (same day of the week during the same month). Conditional logistic regression was used. RESULTS: Due to the social class distribution in the city, exposure to traffic emissions was higher among those with higher area-based income and SES. Meanwhile, people of lower social class had suffered to a larger extent from chronic diseases before death

than more affluent residents, especially diabetes mellitus, hypertension, heart failure, and chronic obstructive pulmonary diseases. Overall, PM(10) (lag 0-1) was strongly associated with mortality (1.1% increase, 95%CI = 0.7-1.6%, per 10 microg/m(3)). The effect was more pronounced among persons with lower income and SES (1.9% and 1.4% per 10 microg/m(3), respectively) compared to those in the upper income and SES levels (0.0% and 0.1%, respectively). CONCLUSIONS: The results confirm previous suggestions of a stronger effect of particulate air pollution among people in low social class. Given the uneven geographical distributions of social deprivation and traffic emissions in Rome, the most likely explanation is a differential burden of chronic health conditions conferring a greater susceptibility to less advantaged people. (c) 2007 Wiley-Liss, Inc.
Publication Type Journal Article.

Result <17>
Unique Identifier
17301770
Status
MEDLINE
Authors
Moorhouse S.
Authors Full Name
Moorhouse, Steve.
Title
US air pollution is harmful and fine particles can kill.[comment].
Comments
Comment on: Nature. 2006 Nov 16;444(7117):248-9; PMID: 17108921, Comment on: Nature. 2007 Jan 4;445(7123):21; PMID: 17203038
Source
Nature. 445(7129):709, 2007 Feb 15.
Publication Type
Comment. Letter.

Result <18>
Unique Identifier
17135427
Status
MEDLINE
Authors
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Title
Relation between concentration of air pollution and cause-specific mortality: four-year exposures to nitrogen dioxide and particulate matter pollutants in 470 neighborhoods in Oslo, Norway.
Source
American Journal of Epidemiology. 165(4):435-43, 2007 Feb 15.
Abstract
This study investigated the concentration-response relation between air pollution (nitrogen dioxide and particulate matter pollutants PM(10) and PM(2.5)) and cause-specific mortality. The population included all inhabitants of Oslo, Norway, aged 51-90 years on January 1, 1992 (n = 143,842) with follow-up of deaths from 1992 to 1998. An air dispersion model (AirQUIS; Norwegian Institute for Air Research (NILU), Oslo, Norway) was used to estimate levels of exposure in 1992-1995 in all 470 administrative neighborhoods. These data were linked to census, education, and death registries. A consistent effect on all causes of death was found for both sexes and age groups by all indicators of air pollution. The effects appeared to increase at nitrogen dioxide levels higher than 40 micro g/m(3) in the youngest age group and with a linear effect in the interval 20-60 micro g/m(3) for the oldest. An effect of all indicators on cardiovascular causes, lung cancer, and chronic obstructive pulmonary disease was also found in both age groups and sexes. The effects were particularly strong for chronic obstructive pulmonary disease, which appeared to have linear effects, whereas cardiovascular causes and lung cancer seemed to have threshold effects. Results show that vulnerable persons with chronic obstructive pulmonary disease and the elderly seem to be susceptible to air pollution at lower levels than the general population.
Publication Type
Journal Article. Multicenter Study.

Result <19>
Unique Identifier
17187238
Status
MEDLINE
Authors
Moreno T. Querol X. Alastuey A. Ballester F. Gibbons W.
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Title
Airborne particulate matter and premature deaths in urban Europe: the new WHO guidelines and the challenge ahead as illustrated by Spain.
Source
European Journal of Epidemiology. 22(1):1-5, 2007.

Abstract
Twenty first century epidemiological publications on urban air pollution are confirming that inhalation of fine, airborne particulate matter (PM) has serious chronic human health effects and is a major cause of premature death worldwide. Recently updated recommendations by WHO identify three “Interim Targets” for the stepped reduction in PM levels within world cities in the quest to achieve an annual mean Air Quality Guideline (AQG) concentration of 20 mug/m(3) for particles less than 10 microns in size (PM(10)). In this paper we offer a perspective from Spain, a country with the longest record of reporting pollution data from large numbers of urban traffic sites to a central European database (AIRBASE). We can demonstrate that average annual PM concentrations at urban traffic monitoring stations in many European cities continue to be 50-100% above the WHO AQG, a situation exacerbated by high urban PM(2.5/10) ratios which indicate a dominance of finer, more deeply inhalable particles potentially more detrimental to health. Given that WHO has estimated in 2000 there were well over 250,000 premature deaths in Europe attributable to PM inhalation, such continuing high urban pollution levels are placing a huge burden on European medical resources.
Publication Type
Journal Article. Research Support, Non-U.S. Gov’t.

Result <20>
Unique Identifier
16959922
Status
MEDLINE
Authors
Gamble JF. Nicolich MJ.
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Gamble, John F. Nicolich, Mark J.
Title
Comments on the updated Harvard Six Cities study.[comment].
Comments
Comment on: Am J Respir Crit Care Med. 2006 Mar 15;173(6):667-72; PMID: 16424447
Source
American Journal of Respiratory & Critical Care Medicine. 174(6):722; author reply 722-4, 2006 Sep 15.
Publication Type
Comment. Comparative Study. Letter. Multicenter Study.

Result <21>
Unique Identifier
16882800
Status
MEDLINE
Authors

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Title
Air pollution and infant death in southern California, 1989-2000.
Source
Pediatrics. 118(2):493-502, 2006 Aug.
Abstract
OBJECTIVE: We evaluated the influence of outdoor air pollution on infant death in the South Coast Air Basin of California, an area characterized by some of the worst air quality in the United States. METHODS: Linking birth and death certificates for infants who died between 1989 and 2000, we identified all infant deaths, matched 10 living control subjects to each case subject, and assigned the nearest air monitoring station to each birth address. For all subjects, we calculated average carbon monoxide, nitrogen dioxide, ozone, and particulate matter < 10 microm in aerodynamic diameter exposures experienced during the 2-week, 1-month, 2-month, and 6-month periods before a case subject's death. RESULTS: The risk of respiratory death increased from 20% to 36% per 1-ppm increase in average carbon monoxide levels 2 weeks before death in early infancy (age: 28 days to 3 months). We also estimated 7% to 12% risk increases for respiratory deaths per 10-microg/m3 increase in particulate matter < 10 microm in aerodynamic diameter exposure experienced 2 weeks before death for infants 4 to 12 months of age. Risk of respiratory death more than doubled for infants 7 to 12 months of age who were exposed to high average levels of particulates in the previous 6 months. Furthermore, the risk of dying as a result of sudden infant death syndrome increased 15% to 19% per 1-part per hundred million increase in average nitrogen dioxide levels 2 months before death. Low birth weight and preterm infants seemed to be more susceptible to air pollution-related death resulting from these causes; however, we lacked statistical power to confirm this heterogeneity with formal testing. CONCLUSIONS: Our results add to the growing body of literature implicating air pollution in infant death from respiratory causes and sudden infant death syndrome and provide additional information for future risk assessment.
Publication Type
Journal Article. Research Support, N.I.H., Extramural.

Result <22>
Unique Identifier
16393654
Status
MEDLINE
Authors
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Title
Fine particulate air pollution and mortality in nine California counties: results from CALFINE.
Source
Environmental Health Perspectives. 114(1):29-33, 2006 Jan.
Abstract
Many epidemiologic studies provide evidence of an association between daily counts of mortality and ambient particulate matter<10 microm in diameter (PM10). Relatively few studies, however, have investigated the relationship of mortality with fine particles [PM<2.5 microm in diameter (PM2.5)], especially in a multicity setting. We examined associations between PM2.5 and daily mortality in nine heavily populated California counties using data from 1999 through 2002. We considered daily counts of all-cause mortality and several cause-specific subcategories (respiratory, cardiovascular, ischemic heart disease, and diabetes). We also examined these associations among several subpopulations, including the elderly (>65 years of age), males, females, non-high school graduates, whites, and Hispanics. We used Poisson multiple regression models incorporating natural or penalized splines to control for covariates that could affect daily counts of mortality, including time, seasonality, temperature, humidity, and day of the week. We used meta-analyses using random-effects models to pool the observations in all nine counties. The analysis revealed associations of PM2.5 levels with several mortality categories. Specifically, a 10-microg/m3 change in 2-day average PM2.5 concentration corresponded to a 0.6% (95% confidence interval, 0.2-1.0%) increase in all-cause mortality, with similar or greater effect estimates for several other subpopulations and mortality subcategories, including respiratory disease, cardiovascular disease, diabetes, age>65 years, females, deaths out of the hospital, and non-high school graduates. Results were generally insensitive to model specification and the type of spline model used. This analysis adds to the growing body of evidence linking PM2.5 with daily mortality.
Publication Type
Journal Article.

Result <23>
Unique Identifier
17214288
Status
MEDLINE
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Institution

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Title
Health impact assessment of particulate matter in Tokyo, Japan.
Source
Archives of Environmental & Occupational Health. 60(4):179-85, 2005 Jul-Aug.
Abstract
Among industrialized countries, Japan still maintains an old set of guidelines for particulate matter (PM); therefore, we assessed the public health impacts of PM exposure in various situations using data from the Tokyo metropolitan area. Subjects were 7.8 million people aged older than 30 years. Based on a linear relationship between exposure and health effects, we estimated attributable cases of mortality caused by PM. Even at the recent exposure level, the number of deaths will occur after both short- and long-term exposure. When the guideline for PM2.5 (particles < 2.5 microm in diameter) long-term exposure was set at 12 microg/m3, we could prevent 8% of all causes mortality or 6,700 deaths in the Tokyo metropolitan area per year. This assessment shows that guidelines for long-term exposure, especially for PM2.5, should be recommended in Japan.
Publication Type
Journal Article.

ENVIRONMENTAL ESTROGENS

Result <1>
Unique Identifier
18410776
Status
MEDLINE
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Title
High growth rate of girls with precocious puberty exposed to estrogenic mycotoxins.
Source
Journal of Pediatrics. 152(5):690-5, 695.e1, 2008 May.
Abstract
OBJECTIVE: To test the hypothesis that human puberty timing can be advanced by environmental estrogen exposure. STUDY DESIGN: We analyzed serum mycoestrogen contamination via high-performance liquid chromatography (HPLC) in 32 girls affected by

central precocious puberty (CPP) and in 31 healthy female control subjects. All 32 patients received triptorelin (TR) for more than 12 months after diagnosis. RESULTS: Increased serum levels of zearalenone (ZEA; 933.7 +/- 200.3 pg/mL; 95% CI, 723.5-1143.9) and of its congener alpha-zearalenol (106.5 +/- 1.9 pg/mL; 95% CI, 104.5-108.5) contaminated 6 girls with CPP, who were from a bounded Tuscany area. At diagnosis, ZEA levels correlated with patient height ($r = 0.906$, $P < .05$) and weight ($r = 0.887$, $P < .05$), but not with bone age. In patients who were mycotoxin-positive, height ($F = 4.192$; $P < .01$), weight ($F = 3.915$; $P < .01$), and height velocity ($F = 2.777$, $P < .05$) were higher than patients who were mycotoxin-negative during 12-months TR treatment. Height correlated with weight both in patients who were mycotoxin-positive ($r = 0.986$, $P < .001$) and in patients who were mycotoxin-negative ($r = 0.994$, $P < .001$). Body mass index, bone age, and gonadal secretion was not different in patient groups before and during TR treatment ($P > .05$). CONCLUSIONS: Mycoestrogenic zearalenone is suspected to be a triggering factor for CPP development in girls. Because of its chemical resemblance to some anabolic agents used in animal breeding, ZEA may also represent a growth promoter in exposed patients.
Publication Type
Journal Article.

Result <2>
Unique Identifier
16832329
Status
MEDLINE
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Title
How do environmental estrogen disruptors induce precocious puberty?. [Review] [94 refs]
Source
Minerva Pediatrica. 58(3):247-54, 2006 Jun.
Abstract
Puberty is regulated by the endocrine system. Disruption of that system by exposure to environmental hormone-mimicking substances (i.e. endocrine disruptors) may, therefore, affect this development profoundly. There has been a great secular trend in the earlier timing of puberty such as both puberty onset and menarche age. This is apparently caused by environmental factors such as improved socioeconomic status, better healthcare and improved nutrition. However, part of the phenomenon could be associated with exposure to endocrine disruptors that have intrinsic estrogen activity or increase endogenous sex hormone levels. These estrogen pollutants tend to degrade slowly in the environment, to bioaccumulate in the food chain and to have long half-lives in humans. Because most of environmental chemicals, called estrogen disruptors or

xenoestrogens, are toxic and estrogen/antiandrogen active, they can disregulate hypothalamic-pituitary-gonadal axis potentially inducing reproductive disorders. There are several case reports of accidental exposure to estrogenic compounds in cosmetic products, food and pharmaceuticals. The outbreak of epidemics of premature thelarche in some geographical areas has also been suggested to be linked to exposure to estrogen disrupters such as dioxins, furans and organohalogens. We review data on adverse health and reproductive outcomes have been attributed to estrogen disruptors in laboratory animals and in wildlife as well as in humans, specially focusing on the puberty timing. [References: 94]
Publication Type
Journal Article. Review.

Result <3>
Unique Identifier
16690809
Status
MEDLINE
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Title
Adverse effects of the model environmental estrogen diethylstilbestrol are transmitted to subsequent generations. [Review] [54 refs]
Source
Endocrinology. 147(6 Suppl):S11-7, 2006 Jun.
Abstract
The synthetic estrogen diethylstilbestrol (DES) is a potent perinatal endocrine disruptor. In humans and experimental animals, exposure to DES during critical periods of reproductive tract differentiation permanently alters estrogen target tissues and results in long-term abnormalities such as uterine neoplasia that are not manifested until later in life. Using the developmentally exposed DES mouse, multiple mechanisms have been identified that play a role in its carcinogenic and toxic effects. Analysis of the DES murine uterus reveals altered gene expression pathways that include an estrogen-regulated component. Thus, perinatal DES exposure, especially at low doses, offers the opportunity to study effects caused by weaker environmental estrogens and provides an example of the emerging scientific field termed the developmental origin of adult disease. As a model endocrine disruptor, it is of particular interest that even low doses of DES increase uterine tumor incidence. Additional studies have verified that DES is not unique; when other environmental estrogens are tested at equal estrogenic doses, developmental exposure results in increased incidence of uterine neoplasia similar to that caused

by DES. Interestingly, our data suggest that this increased susceptibility for tumors is passed on from the maternal lineage to subsequent generations of male and female descendants; the mechanisms involved in these transgenerational events include genetic and epigenetic events. Together, our data point out the unique sensitivity of the developing organism to endocrine-disrupting chemicals, the occurrence of long-term effects after developmental exposure, and the possibility for adverse effects to be transmitted to subsequent generations. [References: 54]
Publication Type
Journal Article. Research Support, N.I.H., Intramural. Review.

Result <4>
Unique Identifier
16393666
Status
MEDLINE
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Title
The estrogenic effect of bisphenol A disrupts pancreatic beta-cell function in vivo and induces insulin resistance.
Source
Environmental Health Perspectives. 114(1):106-12, 2006 Jan.
Abstract
The function of the pancreatic beta-cell is the storage and release of insulin, the main hormone involved in blood glucose homeostasis. The results in this article show that the widespread environmental contaminant bisphenol-A (BPA) imitates 17beta-estradiol (E2) effects in vivo on blood glucose homeostasis through genomic and nongenomic pathways. The exposure of adult mice to a single low dose (10 microg/kg) of either E2 or BPA induces a rapid decrease in glycemia that correlates with a rise of plasma insulin. Longer exposures to E2 and BPA induce an increase in pancreatic beta-cell insulin content in an estrogen-receptor-dependent manner. This effect is visible after 2 days of treatment and starting at doses as low as 10 microg/kg/day. After 4 days of treatment with either E2 or BPA, these mice developed chronic hyperinsulinemia, and their glucose and insulin tolerance tests were altered. These experiments unveil the link between environmental estrogens and insulin resistance. Therefore, either abnormal levels of endogenous estrogens or environmental estrogen exposure enhances the risk of developing type 2 diabetes mellitus, hypertension, and dyslipidemia.
Publication Type
Journal Article. Research Support, Non-U.S. Gov't.

Result <5>
Unique Identifier
15605068
Status
MEDLINE
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Title
Human breast milk and xenoestrogen exposure: a possible impact on human health.[see comment]. [Review] [33 refs]
Comments
Comment in: J Perinatol. 2005 Aug;25(8):558-9; PMID: 16047036
Source
Journal of Perinatology. 25(4):282-8, 2005 Apr.
Abstract
Human milk is the best natural and optimal food for neonates with several immunologic, developmental and practical advantages throughout childhood. Although the World Health Organization strongly supports breastfeeding, it recognizes the potential health risks posed by the presence of environmental toxicants in breast milk. Contamination of human milk is widespread and due to decades of inadequately controlled pollution by toxicants, persistent pesticides or chemical solvents. These chemicals tend to degrade slowly in the environment, to bioaccumulate in the food chain and to have long half-lives in humans. Many of these environmental pollutants have estrogen-like activities and, thus they are called environmental estrogen disruptors or xenoestrogens. Certain adverse health and reproductive outcomes are attributed to these chemicals in laboratory animals and in wildlife as well as in humans. Here, we review available data from breast milk monitoring studies suggesting the environmental chemicals that may affect child health through breastfeeding. [References: 33]
Publication Type
Journal Article. Review.

Result <6>
Unique Identifier
11712665
Status
MEDLINE
Authors
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Title
Evaluation of developmental toxicity in rats exposed to the environmental estrogen bisphenol A during pregnancy.
Source
Life Sciences. 69(22):2611-25, 2001 Oct 19.
Abstract
Bisphenol A (BPA) is an essential component of epoxy resins used in the lacquer lining of metal food cans, as a component of polycarbonates, and in dental sealants. The present study was conducted in an attempt to evaluate the adverse effects of the environmental estrogen BPA on initiation and maintenance of pregnancy and embryofetal development after maternal exposure during the entire period of pregnancy in Sprague-Dawley rats. The test chemical was administered by gavage to mated females from days 1 to 20 of gestation (sperm in varginal lavage = day 0) at dose levels of 0, 100, 300, and 1000 mg/kg. All females were subjected to caesarean section on day 21 of gestation and their fetuses were examined for external, visceral and skeletal abnormalities. In the 1000 mg/kg group, significant toxic effects including abnormal clinical signs, decreased maternal body weight and body weight gain, and reduced food consumption were observed in pregnant rats. An increase in pregnancy failure was also found in the successfully mated females. In addition, increased number of embryonal deaths, increased postimplantation loss, reduced litter size and fetal body weight, and decreased number of fetal ossification centers of several skeletal districts were seen. On the contrary, no significant changes induced by BPA were detected in the number of corpora lutea and implantation sites and by fetal morphological examinations. In the 300 mg/kg group, suppressed maternal body weight and body weight gain, decreased food intake and reduced body weight of male fetuses were seen. There were no adverse signs of either maternal toxicity or developmental toxicity in the 100 mg/kg group. It was concluded that BPA administration during the entire period of pregnancy in rats produced pregnancy failure, pre- and postimplantation loss, fetal developmental delay and severe maternal toxicity, but no embryo-fetal dysmorphogenesis at an oral exposure level of 1000 mg/kg.
Publication Type
Journal Article.

Result <7>
Unique Identifier
11535251
Status
MEDLINE
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Title
Genetic susceptibility and environmental estrogen-like compounds. [Review] [35 refs]
Source
Mutation Research. 482(1-2):77-82, 2001 Oct 1.
Abstract
Environmental chemicals with estrogenic activities have been suggested to be able to interact with the endocrine system. Endogenous estrogen is synthesized in the ovarian theca cells of premenopausal women or in the stromal adipose cells of the breast of postmenopausal women and minor quantities in peripheral tissue. These cells, as well as breast tissue, express all the necessary enzymes for this synthesis, CYP17, CYP11a, CYP19, 17-beta-hydroxysteroid hydrogenase, steroid sulfatase as well as enzymes further hydroxylating estradiol, such as CYP1A1, CYP3A4, CYP1B1, catechol-o-methyltransferase (COMT). Polymorphisms in these enzymes may have a possible role in the link between environmental estrogens and hormone-like substances and the interindividual risk of breast cancer. [References: 35]
Publication Type
Journal Article. Research Support, Non-U.S. Gov't. Review.

Result <8>
Unique Identifier
12084301
Status
MEDLINE
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Title
The possible effects of environmental estrogen disrupters on reproductive health. [Review] [61 refs]
Source
Current Urology Reports. 1(4):253-61, 2000 Dec.
Abstract
The term environmental estrogen refers to chemical substances that exhibit some degree of estrogen-like activity. The primary emphasis for potential adverse effects resulting from exposure to environmental estrogens is on in utero exposure because such exposures can occur during critical periods of organogenesis. Assessment of biological plausibility can be based, in part, on the extensive data on the effects of diethylstilbestrol (DES). The available evidence is too limited

to judge with any confidence whether sperm counts have declined during the past 50 years. Based on both animal and human data with DES, it is biologically plausible that in utero exposures to exogenous estrogenic compounds are capable of reducing sperm production in adult men. However, the apparent existence of a maternal dose threshold for DES-induced effects on sperm count undermines the likelihood that environmental estrogens, which are substantially less potent, are capable of causing similar effects. [References: 61]
Publication Type
Journal Article. Review.

Result <9>
Unique Identifier
9699867
Status
MEDLINE
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Title
An updated review of environmental estrogen and androgen mimics and antagonists. [Review] [74 refs]
Source
Journal of Steroid Biochemistry & Molecular Biology. 65(1-6):143-50, 1998 Apr.
Abstract
For the last 40 y, substantial evidence has surfaced on the hormone-like effects of environmental chemicals such as pesticides and industrial chemicals in wildlife and humans. The endocrine and reproductive effects of these chemicals are believed to be due to their ability to: (1) mimic the effect of endogenous hormones, (2) antagonize the effect of endogenous hormones, (3) disrupt the synthesis and metabolism of endogenous hormones, and (4) disrupt the synthesis and metabolism of hormone receptors. The discovery of hormone-like activity of these chemicals occurred long after they were released into the environment. Aviation crop dusters handling DDT were found to have reduced sperm counts, and workers at a plant producing the insecticide kepone were reported to have lost their libido, became impotent and had low sperm counts. Subsequently, experiments conducted in lab animals demonstrated unambiguously the estrogenic activity of these pesticides. Man-made compounds used in the manufacture of plastics were accidentally found to be estrogenic because they fouled experiments conducted in laboratories studying natural estrogens. For example, polystyrene tubes released nonylphenol, and polycarbonate flasks released bisphenol-A. Alkylphenols are used in the synthesis of detergents (alkylphenol polyethoxylates) and as antioxidants. These detergents are not estrogenic; however, upon degradation during sewage treatment they may release estrogenic

alkylphenols. The surfactant nonoxynol is used as intravaginal spermicide and condom lubricant. When administered to lab animals it is metabolized to free nonylphenol. Bisphenol-A was found to contaminate the contents of canned foods; these tin cans are lined with lacquers such as polycarbonate. Bisphenol-A is also used in dental sealants and composites. We found that this estrogen leaches from the treated teeth into saliva; up to 950 microg of bisphenol-A were retrieved from saliva collected during the first hour after polymerization. Other xenoestrogens recently identified among chemicals used in large volumes are the plastizicers benzylbutylphthalate, dibutylphthalate, the antioxidant butylhydroxyanisole, the rubber additive p-phenylphenol and the disinfectant o-phenylphenol. These compounds act cumulatively. In fact, feminized male fish were found near sewage outlets in several rivers in the U.K.; a mixture of chemicals including alkyl phenols resulting from degradation of detergents during sewage treatment seemed to be the causal agent. Estrogen mimics are just a class of endocrine disruptors. Recent studies identified antiandrogenic activity in environmental chemicals such as vinclozolin, a fungicide, and DDE, and insecticide. Moreover, a single chemical may produce neurotoxic, estrogenic and antiandrogenic effects. It has been hypothesized that endocrine disruptors may play a role in the decrease in the quantity and quality of human semen during the last 50 y, as well as in the increased incidence of testicular cancer and cryptorchidism in males and breast cancer incidence in both females and males in the industrialized word. To explore this hypothesis it is necessary to identify putative causal agents by the systematic screening of environmental chemicals and chemicals present in human foods to assess their ability to disrupt the endocrine system. In addition, it will be necessary to develop methods to measure cumulative exposure to (a) estrogen mimics, (b) antiandrogens, and (c) other disruptors. [References: 74]
Publication Type
Journal Article. Research Support, Non-U.S. Gov't. Research Support, U.S. Gov't, Non-P.H.S.. Research Support, U.S. Gov't, P.H.S.. Review.

Result <10>
Unique Identifier
9182870
Status
MEDLINE
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Title
Profound effects of the weak environmental estrogen-like chemical bisphenol A on the growth of the mammary gland of Noble rats.
Source
Journal of Steroid Biochemistry & Molecular Biology. 60(1-2):153-60, 1997 Jan.

Abstract
In the present study we have examined the effects of the environmental estrogenic chemical bisphenol A on proliferative activity, cell cycle kinetics and differentiation of the mammary gland of female Noble rats. Differentiation measured by the degree of lobular maturation revealed that the conversion of immature structures to mature structures was significantly increased in response to exposure to both low (0.1 mg/kg/day) and high (54 mg/kg/day) doses of bisphenol A compared to controls. The proliferative activity of epithelial cells was increased by 143% over controls by the exposure of animals to the low dose of bisphenol A, whereas a 220% increase over controls was observed for the high dose of bisphenol A. The labelling index and growth fraction were 19% and 27%, respectively, for a low dose of bisphenol A; and 27% and 45%, respectively, for a high dose of bisphenol A, compared to 18% and 31%, respectively, in controls. A significant increase in the conversion of mamm!
ary epithelial cells from G0, to G1, and S-phase cells by 1.8 and 4.5-fold, respectively, was observed in animals exposed to the high dose of bisphenol A compared to that of controls. Based on the previously reported estrogenic activity of an equivalent dose of bisphenol A to that of diethylstilbestrol (DES) (0.1 mg/kg/day), a calculated theoretical dose of the order of 10(6)-fold higher of bisphenol A will be required to produce the same biological effects as DES. A comparison of the proliferative activity of DES and that of an equivalent dose of bisphenol A observed in this study, however, revealed that its influence on proliferative activity in the epithelial cells of the mammary gland was profound. The weak estrogenic activity of bisphenol A does not explain its profound effect on cell proliferation observed in this study. Perturbation of the cell cycle is considered a risk factor for the development of cancer. Bisphenol-mediated perturbation of the cell cycle in epithelial cells may produce adverse effects in the mammary glands of Noble
rats.
Publication Type
Journal Article.

Result <11>
Unique Identifier
9175721
Status
MEDLINE
Authors
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Title
Transcriptional activation of the human estrogen receptor by DDT isomers and metabolites in yeast and MCF-7 cells.

Source
Biochemical Pharmacology. 53(8):1161-72, 1997 Apr 25.
Abstract
In this study, we determined whether the DDT isomers p,p'-DDT [1,1,1,-trichloro-2,2-bis(p-chlorophenyl)ethane], o,p'-DDT [1,1,1-trichloro-2(p-chlorophenyl)-2-(o-chlorophenyl)ethane], and their metabolites p,p'-DDD [1,1-dichloro-2,2-bis(p-chlorophenyl)ethane], o,p'-DDD [1,1-dichloro-2-(p-chlorophenyl)-2-(o-chlorophenyl)ethane], p,p'-DDE [1,1,-dichloro-2,2-bis(p-chlorophenyl)ethylene], o,p'-DDE [1,1-dichloro-2-(p-chlorophenyl)-2-(o-chlorophenyl) ethylene], and p,p'-DDA [2,2-bis(p-chlorophenyl)acetic acid], could bind to and transcriptionally activate the human estrogen receptor (hER). Novel results from competitive binding assays showed that o,p'-DDD, o,p'-DDE, and p,p'-DDT, as well as the established environmental estrogen o,p'-DDT, were able to bind specifically to the hER with approximately 1000-fold weaker affinities for the hER than that of estradiol. In contrast, only o,p'-DDT, but not p,p'-DDT, bound to the rat estrogen receptor. Moreover, two yeast expression-reporter!
systems, constructed to test if the DDT isomers and metabolites could transcriptionally activate the hER, demonstrated that an o,p'-DDT metabolite could transactivate the hER or LexA-hER fusion protein with just a 140- to 300-fold weaker potency than that of estradiol. The DDT isomers and metabolites that bound the hER in vitro triggered estrogen receptor-mediated transcription of the lacZ reporter gene in the yeast systems. Furthermore, the DDT isomers and metabolites that transactivated the hER elicited an additive response when given together or with estradiol. The DDT isomers and metabolites that triggered transcription of the yeast expression-reporter systems also stimulated two estrogenic endpoints in estrogen-responsive MCF-7 cells: the induction of the progesterone receptor and the down-regulation of the hER. Thus, in MCF-7 cells and in yeast expression-reporter systems, certain DDT isomers and metabolites act directly as agonists and transactivate the hER at concentrations found in human tissues.
Publication Type
Journal Article. Research Support, U.S. Gov't, P.H.S..

Result <12>
Unique Identifier
9168009
Status
MEDLINE
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Title
Developing a marker of exposure to xenoestrogen mixtures in human serum.
Source

Environmental Health Perspectives. 105 Suppl 3:647-54, 1997 Apr.

Abstract

It has been hypothesized that environmental estrogens may play a role in the increasing incidence of breast cancer, testicular cancer, and other problems of the reproductive system. While a single causal agent can be identified in cases in which humans have had occupational exposures, wildlife showing signs of reproductive damage have usually been exposed to a combination of endocrine disruptors that may act cumulatively. The development of appropriate biomarkers of cumulative exposure, and their measurement at developmental points where exposure is critical, are required to test the environmental estrogen hypothesis. Measuring levels of each of the xenoestrogens in blood is a better approximation of real exposure at the target organ level than inferring cumulative exposure by estimating from mass balance of dietary levels. However, the cumulative estrogenicity of mixtures cannot be directly concluded from individual xenoestrogen plasma levels. Two approaches may be used to assess total load: a) the development of methods to study mixtures of these xenoestrogens, to quantify their cumulative effects, and to begin to understand their interactions (i.e., additivity, synergy, antagonism, or independent action), so that plasma concentrations may be translated into units of activity such as “estradiol equivalents”; and b) the development of methods to separate xenoestrogens from ovarian estrogens in blood and to directly measure the estrogenic activity of the xenoestrogen extract using a bioassay. The cumulative activity may be used as a marker of exposure to xenoestrogens. This article reports the development of a method to extract and separate xenoestrogens from ovarian estrogens using human serum as a source, followed by using a bioassay for determination of the cumulative xenoestrogen load as “estradiol equivalents.”

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