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Children's Environmental Health in Agricultural Settings

Catherine Karr, MD, PhD [Associate Professor]

Department of Pediatrics and Department of Environmental and Occupational Health Sciences, NW Pediatric Environmental Health Specialty Unit, University of Washington, Seattle, Seattle, Washington, USA

Abstract

Children residing in rural settings may encounter environmental hazards derived from agricultural production activities. Health consequences of organic dusts, farm chemicals including pesticides, machinery noise, excess sun exposure, and zoonotic infectious agents have been clearly described among farm-working adults. The author reviews the related evidence base on child health with a life-stage perspective on their differential exposure and vulnerabilities. Methemoglobinemia among infants consuming nitrate-contaminated well water, neurodevelopmental health impacts associated with early life exposure to organophosphate pesticides, and diarrheal disease due to zoonotic infectious agents are well-described pediatric concerns. There is suggestive but more limited evidence for respiratory health consequences from air contaminants associated with confined animal feeding operations and hearing deficits for children exposed to machinery-related noise. Many contaminants of concern for children in these environments remain largely understudied—diesel exhaust, biomass burning, solvents, veterinary antibiotics, and silica-containing particulate matter. Overall, the state of knowledge and programmatic activities on agriculturally derived environmental contaminants and child health is immature and much less complete than for working adults. This overview provides a context for research, policy, and programmatic needs.

Keywords

Agricultural health; children's environmental health; rural health

INTRODUCTION

The influential role of environmental factors on children's health has been increasingly appreciated in the last two decades. The extent and type of physical, chemical, and biological hazards encountered by children varies considerably across households and communities. The agricultural setting poses some unique exposures related to agricultural production. Physical factors and mechanical exposures resulting in acute traumatic injuries and musculoskeletal disorders are among the most well-described agricultural hazards for both children and adults. This review focuses on data relevant to the role of agricultural

Address correspondence to: Catherine Karr, MD, PhD, Department of Environmental and Occupational Health Sciences, University of Washington, Seattle, Box 354695, 4225 Roosevelt Way NE, Seattle, WA 98105, USA (ckarr@u.washington.edu).

production activities on illness and disease among children who reside in agricultural environments.

The well-established relationships of pesticides, organic dusts, toxic gases, and other farm chemicals in acute and chronic respiratory and neurologic health conditions among occupationally exposed adults raise suspicion for similar or unique adverse impacts on children.^{1,2} Childhood cancer and developmental delays are unique outcomes of concern for chemical toxicant exposures in the pediatric population. In addition, infectious agents associated with farm animals, sun exposure from outdoor work, and high noise levels associated with farm tools and machinery are well-characterized workplace hazards in adult agricultural workers.^{3,4} These nonchemical exposures may present health hazards for children as well.

UNDERSTANDING RISKS TO CHILDREN—A LIFE STAGE PERSPECTIVE ON EXPOSURE AND TOXICITY

Crop and animal production activities may produce contaminants in multiple media in agricultural communities. Soil where children play, the ambient air, dust in homes, drinking water supplies, and food crops represent the range of sources that must be considered. Children are not small adults and life stage from birth to adulthood influences both exposure variability and toxicological sensitivity to contaminants in these media (Figure 1).⁵

Children are anabolic with rapid growth phases. Compared to adults, children consume more calories, more water, and breathe more air per unit of body weight.⁶ Compared to adults, toxicants in food are delivered at 2 to 3 times higher rate, and in water 5 to 7 times higher rate.⁷ Age-related differences in skin absorption are not prominent,⁸ but specific dietary constituents vary considerably through life stages. For example, apple product consumption is an order of magnitude higher in early life.⁵ Normal exploratory behavior in young children is consistent with data that demonstrate that the average toddler ingests twice as much soil as an adult, and the child in the 95th percentile can ingest 8 times more.⁹ Studies of children's activity patterns demonstrate that dermal contact with environmental contaminants on surfaces and objects is a concern throughout childhood, whereas activities such as hand-to-mouth and object-to-mouth behaviors contributing to nondietary ingestion may decline with age.¹⁰

Based on these factors, children in agricultural communities with contamination of soil, water, foods, or air are at risk of receiving higher doses than adult residents. For developmental toxicants, the same dose may have no consequences in an adult yet portend devastating consequences on a fetus or child if exposure occurs in a critical developmental window. For example, the physiologic vulnerability of young infants to nitrate contamination of drinking water is discussed below. Paraoxonase I detoxification enzymes which interact with certain organophosphates (OPs) occur at lower levels and activity in young children compared to adults.¹¹ Among pesticides or other farm chemicals that are mutagenic carcinogens, life stage-based risk assessment acknowledges that risk is higher for exposures that occur in early life with a longer opportunity for development of latent disease.¹²

In this broad overview, brief episodic illnesses and chronic developmental toxicity are considered. Of particular interest are the major chronic morbidities that affect US children today, such as attention-deficit/hyperactivity disorder (ADHD), adverse birth outcomes, autism, asthma, and cancer. Reliance on previous reviews as available and primary citations was utilized and synthesized. Discrepancies and limitations in the literature are noted. This can help frame the research needs and future directions to understand the role of the agricultural environment on child health.

PRIMARY ENVIRONMENTAL HAZARDS OF CONCERN FOR CHILDREN IN AGRICULTURAL COMMUNITIES

Pesticides

Pesticides are among the increasingly well-documented hazardous exposures among children in agricultural settings. Unintentional ingestion from improperly stored acutely toxic pesticide products, as well as inhalation and skin contact from drift or spills, has the potential for immediate, devastating, and sometime lethal consequences for farm children.^{13–16} Fortunately, such severe acute toxicity is rare in US farm children. Not rare in agricultural settings is the proximity of housing, schools, and play areas to agricultural fields or livestock production where regular pesticide application occurs.

The opportunity for “take home” exposure, where parents serve as “vectors” for residues transferred from occupational activities via their skin, clothing, and footwear into family vehicles and residences is now well established.^{17–20} Pesticides may contaminate rural drinking water supplies. Wells that rely on shallow groundwater sources are at highest risk; approximately 61% of shallow groundwater samples from agricultural areas contain at least one detectable pesticide according to the National Water Quality Assessment Program.²¹ Very few (1.2%) of the pesticides detected exceed their individual benchmark levels that are intended to be protective of the general population. However, detection of more than one pesticide contaminant is common and health-based benchmarks are not available for combined exposures. The use of biological markers of pesticide exposure such as urinary metabolites have clearly demonstrated uptake in farm children and pregnant women that exceeds levels observed in reference populations.^{17,22,23}

Characterizing the scope of the problem is impossible due to lacking data systems for comprehensive tracking and surveillance. Although 12 states maintain a National Institute for Occupational Safety and Health (NIOSH)-sponsored Sentinel Event Notification System for Occupational Risks (SENSOR) that collects medical provider reports of pesticide-related illness or injury, these are focused primarily on occupationally related exposures. In rare instances, these have included some pediatric relevant outcomes such as a SENSOR-prompted birth defect cluster analysis among migrant workers²⁴ and the use of SENSOR data to review poisoning events associated with schools, which included spray drift onto school grounds from neighboring farmland.¹⁵ Although the Poison Control Centers Toxic Exposure Surveillance System (TESS) publishes annual summaries of *self-reported calls* to the centers from the public and medical community and include pesticide incidents, these

represent acute exposure concerns, are subject to reporting bias and selection bias, and do not provide information specific to the agricultural setting.¹⁴

Underreporting in existing systems is highly problematic given the poor recognition of pediatric pesticide exposure among health care providers.^{25,26} This reflects the limited attention in current medical education to topics in children's environmental health.²⁷⁻³⁰ Also, clinical presentations in children may be more subtle and less specific than in adults.^{26,31} This is aggravated by the lack of diagnostic tools to confirm that a pesticide overexposure occurred.^{32,33} Furthermore, long waits at some rural health clinics or limited access to health care may preclude farmworkers and their children from presenting to medical care for suspected pesticide illness.³⁴

Agricultural pesticide chemicals include neurotoxicants, mutagens, carcinogens, immunotoxicants, and endocrine disruptors based on experimental toxicology data. These provide biological plausibility for an expanding and relatively consistent epidemiological evidence base that supports a link between pesticide exposure in early life with some of the most prevalent and severe chronic health conditions in US children.

Pesticide exposures in fetal life and early childhood are associated with risk of behaviors seen in ADHD, autism, as well as cognitive effects, adverse birth outcomes including low birth weight and birth defects, and pediatric cancer. Fewer epidemiological studies designed to address immune dysfunction, asthma, or reproductive system development and health have been conducted, although several studies in adult workers have linked pesticides to respiratory health outcomes including wheeze.^{35,36} Toxicological mechanisms of pesticide active ingredients support the need for better characterization of these health endpoints.

Research emphasis in the last decade on the widely used organophosphate class of insecticides has revealed much about the unique vulnerability of the developing central nervous system, including elucidation of toxicological mechanisms, genetic vulnerabilities, and risk of clinically relevant outcomes in observational epidemiological studies. Similar to observations of chronic sequelae of OP poisoning in adult workers, children who experienced an early life acute OP poisoning (age <3 years) demonstrate subtle but identifiable ongoing neurobehavioral deficits at school age.³⁷

Of even greater public health concern are the findings from three recent and ongoing well-designed prospective birth cohort studies in US children, including one in a Mexican American agricultural farmworker community (the Center for the Health Assessment of Mothers and Children of Salinas or CHAMACOS). These studies demonstrate relative consistency of poorer cognitive and behavioral outcomes based on validated assessment tools in infants, toddlers, preschoolers, and early-school-aged children with increased prenatal exposure to organophosphate pesticides.³⁸⁻⁴⁴ In addition, several studies have implicated early life (prenatal and/or postnatal) OP exposure with symptoms and/or diagnosis of ADHD or symptoms of pervasive developmental delay (autism).^{40,41,45,46} Whereas the birth cohort studies have identified cognitive impacts associated with prenatal exposure but not postnatal exposure, others have reported associations of measures of

postnatal OP exposures with adverse effects on measures of neurobehavioral function, including short-term memory, executive function, reaction time, and motor skills.^{26,47,48}

There has also been high attention to pediatric cancer. Several review articles describe multiple ecologic and case-control studies exploring parental exposures or pesticide use in the home with childhood brain tumors, leukemias and lymphomas, and a number of other tumor types.^{49–52} Reliance on retrospective, nonspecific exposure assessment as well as limited sample size are common limitations in this literature. These reviews, along with a recent meta-analysis of two cohorts and 38 case-control studies, demonstrate a body of evidence with strongest links to leukemia and brain cancer.⁵³ The associations for leukemia appear greatest for maternal exposure through household use or occupational exposures preconceptionally and prenatally. Incidence of brain cancer appears to be influenced more by paternal exposure and has been linked to occupational exposure before conception through birth.

The chronic exposure patterns and health implications of non-organophosphate agricultural pesticides or the influence of mixtures have received much less focused study. The use of pyrethroid insecticides has been increasing in agriculture and toxicological data for active ingredients in this group raise concern for neurodevelopmental toxicity and carcinogenicity.⁵⁴ Preliminary recent findings in an urban setting are suggestive that pyrethroid-containing products may have adverse neurodevelopmental effects, but confirmatory studies from well-designed epidemiological studies in agricultural settings are lacking.⁵⁵

Recognizing the concern for children's routine exposure to pesticides from multiple sources, the American Academy of Pediatrics Council on Environmental Health is developing a new technical report and policy statement that reviews the evidence for pediatric harm, the data gaps, and policy recommendations (personal communication, American Academy of Pediatric Executive Council on Environmental Health).

Organic methods of agricultural production represent an approach to growing crops and raising livestock that avoids synthetic chemicals such as pesticides as well as hormones, antibiotics, genetic engineering, and irradiation. Organic produce contains lower levels of pesticide residue and experimental studies of children demonstrate that a diet of organic produce can reduce exposure levels in children.^{56,57} There are no studies that directly examine exposure to pesticides from conventionally grown foods and the development of adverse health outcomes. However, the exposure to organophosphate measured in the cohort studies described above have found adverse neurodevelopmental effects in populations exposed in ranges comparable to those observed among children consuming conventional diets.⁵⁷

Organic agriculture may also benefit the growing problem of antimicrobial-resistant organisms. Studies demonstrate that antimicrobials applications in food production promotes the development and subsequent dissemination to humans of resistant organisms.⁵⁸ Although the proportion of the problem attributed to nontherapeutic livestock use is not well understood, the largest use of antimicrobial agents outside human medicine is in food animals.⁵⁹

Air Contaminants Derived From Animal Production

Animal feed handling, movement of animals on manure, and the storage and removal of their manure produce a complex mixture of air contaminants. The type and extent released reflect animal type, management practices, and facility type and size. Known respiratory irritants and proinflammatory components, including ammonia, hydrogen sulfide, volatile organic compounds, and particulate matter and bioaerosols (glucans, endotoxin), are of primary concern.⁶⁰ Perception of odors has been linked to respiratory complaints and reduced measures of quality of life. An immunosuppressive effect of odor on mucosal immunity has also been hypothesized.⁶¹

Livestock production in the United States has sharply transformed in recent decades, from small family operations to an industry dominated by large and concentrated production processes.⁶² The impact of these facilities on community air quality and child health is of high concern to communities but available data are largely limited to occupational exposures and investigation of adult health outcomes.^{63,64}

Respiratory health consequences among workers in high-density animal production are well documented.^{65,66} Effects include mucous membrane irritation, bronchitis, asthma, chronic obstructive pulmonary disease, and interstitial lung disease. Health impacts on adults from environmental exposures assessed as proximity to animal operations have also been demonstrated, including deficits in lung function (forced expiratory volume in 1 second [FEV₁]) and asthma symptoms.⁶⁷ A systematic review of research on the association between animal feeding operations (AFOs) and the health of individuals living nearby notes the strong limitations of the evidence base. Among nine relevant studies examined, the authors conclude that existing data provide inconsistent evidence of a weak association between self-reported adverse health outcomes, particularly among individuals with allergies or family history of allergic disease.⁶⁸

Data on child exposures are particularly scarce. Limited surveys suggest exposure to large-scale animal production is associated with increases in asthma prevalence and asthma symptoms among US children.⁶⁹⁻⁷¹ This area of research is critical, given that asthma is the most prevalent chronic disease among US children, affecting roughly 10% and rates have been increasing over the last two decades.⁷²

Exposure characterization for child residents in areas with large animal feeding operations is needed. Longitudinal studies that can decipher impacts of these exposures across life stages of children on mechanistically and clinically relevant endpoints associated with allergic disease, immune function, lung function, and lung function growth will be most informative.

This is underscored by ongoing investigations of a role for early life exposure to endotoxin or other microbiological factors associated with animals or farm life (e.g., raw milk consumption) decreasing risk of the development of asthma or other allergic disease.⁷³ These observations are derived from studies largely conducted in nonindustrial farm settings in Europe. This effect has been less evident in the US context.^{70,74}

Nitrate

Nitrate is a common contaminant in rural well water. Important sources are nitrogen-containing fertilizer use and/or high-volume manure waste. The most recently available survey data from the United States Geological Survey indicate that 20% of US agricultural area wells exceed the nitrate maximum contaminant level (MCL) set by the United States Environmental Protection Agency (US EPA).⁷⁵ The MCL is set to protect infants from the development of methemoglobinemia or “blue baby” syndrome. This severe syndrome of inadequate oxygenation of tissues can be fatal. Among the reported cases of methemoglobinemia in US infants, most have been attributed to the use of contaminated well water for preparation of infant formula.⁷⁶

Infants are physiologically vulnerable to the development of methemoglobinemia due to several factors. Their higher gastric pH favors nitrate-reducing bacteria that convert ingested nitrate into methemoglobin-producing nitrite. In addition, fetal hemoglobin, the predominant form in infants up to 3 months of age, is oxidized more readily to methemoglobin by nitrite than is adult hemoglobin. Lastly, the activity of the red blood cell enzyme systems that reduce methemoglobin back to normal hemoglobin is reduced by about half in infants compared with adults. Prompt recognition is vital to ensuring delivery of potentially lifesaving treatment.

Less well-established child health consequences are under investigation. Findings linking maternal exposure to nitrate in pregnancy with subsequent birth defects are suggestive but equivocal.^{77–80} Studies of nitrate in drinking water and development of type I diabetes support a hypothesized linkage but are ecological in design. Results from case-control studies, which have all been done outside the United States, are inconsistent.⁸¹

Whereas large suppliers of public water sources are required to monitor nitrate concentrations regularly, private wells are not. Few rural wells are routinely tested for nitrates.⁸² Well water recommendations targeted to pediatric care providers have recently become available.⁸³ Effective dissemination of these recommendations, improved surveillance of agricultural drinking water, and well-designed epidemiological investigations of developmental and chronic health outcomes in higher risk communities are needed.

Noise

Noise exposure related to agricultural equipment is a well-recognized hazard with high exposure levels experienced by farmworkers and families.⁸⁴ Impacts of early life exposure in the farm environment on the function of the developing ear have not been studied, although it has been hypothesized that the developing child’s vulnerability to acoustic trauma and strain on the middle and inner ear is qualitatively different.⁸⁵ Existing studies suggest that hearing impairment among agricultural workers develops early in life.⁸⁶ The limited studies assessing hearing loss in farm youth have documented increased risk compared to national samples or nonrural counterparts and increased risk associated with active involvement in farm work.^{87,88} Longitudinal studies in children that address noise as well as exposures to potentially synergistic ototoxic chemical exposures on the farm, including solvents and pesticides, are needed.⁸⁵

Sun Exposure

Sun exposure is the major environmental risk factor associated with nonmelanomatous skin cancer (basal cell carcinoma, squamous cell carcinoma).⁸⁹ Review of epidemiologic studies is also suggestive that sunlight exposure and sunburn during childhood and adolescence increases the risk of melanoma.⁸⁹ In addition, reviews and meta-analyses of cancer epidemiological studies are suggestive of increased risk of skin cancers for adult agricultural workers that may reflect higher sun exposure as well as other environmental factors such as pesticides.⁹⁰ In the large cancer incidence study of pesticide applicators enrolled in the Agricultural Health Study, spouses of applicators were observed to have a significant excess of melanoma (standardized incidence ratio [SIR] 1.64, 95% confidence interval [CI] 1.27–2.09), although the effect was not observed among the applicators themselves.⁹¹ In a study of risk factors for squamous cell carcinoma in Saskatchewan, a largely rural agricultural Canadian province, agricultural occupation was a significant factor 1.49 (95 % CI 1.22–1.82).⁹²

Skin cancer is rare in children, although the risk of melanoma increases considerably in the adolescent years. Few studies address risk in this age group. One study done in a high-exposure setting (Australia) found that among the more well-established risk factors (propensity to develop nevi and freckles, red hair, blue eyes, inability to tan, and a family history of the disease), adolescents who had lived or currently lived on a farm had a significantly increased risk (odds ratio [OR] = 1.9, 95% CI = 1.1–3.3).⁹³ Reported exposures to sunlight, pesticides, fertilizers, or aerial spraying were not significant predictors in this study.

These observations raise concern given that rural children may spend more time outdoors compared to their urban counterparts.⁹⁴ A survey of 11- to 18-year-old US youth demonstrates that among factors influencing risk of sunburn is the number of hours spent outside.⁹⁵

Zoonotic Organisms

Several case and outbreak reports of zoonotic disease transmission to children who work or live on or near farm animal operations have been described. Transmission routes include oral, respiratory, direct, and indirect contacts with infected animals or contaminated soil and water.^{4,96,97} Outbreaks in communities downwind of infected animals have been reported.⁹⁸ The zoonotic diseases found in agricultural settings for which young children or the developing fetus may be at increased risk include campylobacteriosis, coxiellosis, cryptosporidiosis, enterohemorrhagic *Escherichia coli* (EHEC), listeriosis, salmonellosis, toxoplasmosis, and yersiniosis.⁴

The developing immune system of very young children, particularly infants, places them at greater risk for acquiring serious infections and developing infections of antibiotic-resistant organisms connected to the agricultural use of antimicrobials.⁹⁹ For example, whereas most strains of *E. coli* are harmless and commensal in healthy mammals, *E. coli* 0157:H7 produces a powerful toxin and can cause severe hemorrhagic enterocolitis. Children under the age of 5 years are at risk of hemolytic uremia syndrome as a complication.¹⁰⁰

As with pesticide illness or nitrate excesses in private wells, there are no systematic surveillance data on transmission of infections related to farm animals. Although health care providers are trained in the diagnosis and prevention of zoonoses, the nonspecific clinical presentation in children (e.g., diarrheal disease) requires an index of suspicion for animal reservoirs and farm-related transmission. The true prevalence of zoonoses among agricultural community residents is unknown. Seroprevalence studies may help identify high-exposure subgroups as well as specific animal and child behavior risk factors that influence exposure.¹⁰¹ Effective surveillance efforts will require coordination and education of the veterinary, medical, and public health community.⁴

OTHER EXPOSURES OF CONCERN FOR CHILDREN IN THE AGRICULTURAL SETTING

The exposures summarized above have focused on the more well-characterized environmental contaminants associated with agricultural production that may affect child health. There are many other contaminants of concern based on demonstrated toxicity among children in nonagricultural settings or plausibility for harmful exposure from agricultural production. In addition to those discussed above, exposure characterization and child health impacts research in agricultural settings is needed for diesel exhaust, biomass burning, solvents, and silica-containing particulate matter.

MAKING SENSE OF MIXED EXPOSURES AND COMPLEX, MULTIFACTORIAL DISEASES IN THE RURAL CONTEXT AND FOR VULNERABLE SUBGROUPS

Most research on agriculturally related environmental hazards for children focus on a single contaminant or source. However, the major morbidities of concern for children in both agricultural and nonagricultural settings are complex and multifactorial diseases (e.g., asthma, ADHD, autism, diabetes, cancer, obesity). Numerous environmental, genetic, and nonenvironmental factors interact to influence the development and severity of these outcomes. Agricultural health research approaches that are health outcome focused and can encompass multiple factors within a rural health context should be promoted. The landmark National Children's Study conceptual design serves as an example of an approach to investigate major child health and development concerns in relation to the complexity of the social, nutritional, biological, chemical, and genetic environment from preconception into adulthood.¹⁰²

Addressing cultural factors, legal status, and/or socioeconomic-driven barriers that may be prominent in rural settings is critical. These factors influence access to education and prevention programs as well as access to quality health care services and contribute to health outcome disparities. Addressing these disparities is discussed in detail elsewhere in this issue (J. McLaurin, "Unique Agricultural Safety and Health Issues of Migrant and Immigrant Children"; D. Kraybill et al., "Culturally Competent Safety Interventions for Children in Old Order Anabaptist Communities"; and D. Helitzer, "Children's Safety on Native American Farms: Information and Recommendations"). Identification of uniquely

vulnerable subpopulations is important for research design, educational messages, and policy decision-making.

CONCLUSIONS AND RECOMMENDATIONS

Overall, the state of knowledge on environmental contaminants from agricultural activities and their health consequences for children is quite limited and much less complete than the effects of these exposures on working adults. Furthermore, ongoing children's environmental health research and programs in rural settings are far fewer than efforts in urban areas. A synthesis of the body of evidence suggests that children in agricultural settings may be at high risk of exposures that disrupt normal development and health. The existing data considered here provide a framework for research, program, and policy needs in children's environmental health in agricultural settings

Program Recommendations

Provide opportunities for training and improved capacity of clinicians and public health professionals who serve agricultural families on identification and reduction of environmental health risks of children.

Such programs could encompass development of data-gathering approaches in communities to define factors that influence exposure, prevalence of health conditions associated with agricultural hazards, and improvements associated with intervention strategies. Programs that foster collaborations of medical, public health, agricultural science, and veterinary health sectors should be prioritized. This should be done at the local, state, and federal level.

Develop programs for education of agricultural communities on reduction of environmental health risks for children in their homes, schools, and public areas. Engage local youth in environmental health programs and provide exposure to environmental health careers.

Research Recommendations

Provide empirical data on exposure levels children experience in agricultural settings to key known environmental hazards—noise, animal operation-related air contaminants, agricultural chemicals including pesticides, drinking water contaminants particularly nitrates, and zoonotic infectious agents. Examine the variability in these exposures in relationship to lifestyle and agricultural production factors. Consider all pathways and combined exposures in air, water, dietary, soil, and house dust.

Support epidemiological research that evaluates the impact of nonacute agricultural chemical exposures and important chronic pediatric morbidities or suspected toxicities such as autism, ADHD, asthma, allergic rhinitis, skin disease, diabetes, low birth weight, premature birth, birth defects, obesity, cancer, and hearing loss. Promote approaches that evaluate multiagent effects and timing of exposures.

Policy Recommendations

Establish a comprehensive national surveillance program for identification and tracking of important environmental hazards in agricultural communities, including pesticide-related

incidents, drinking water contaminants, and zoonoses. Data system applications that can integrate and incorporate existing relevant data such as outcomes in electronic medical records and exposures collected from regulatory authorities (e.g., pesticide applications) should be advanced. Surveillance activities should establish strong linkages among the medical, veterinary, and public health communities that serve agricultural communities.

Developmental toxicity endpoints including but not limited to neurodevelopment, reproductive health, and endocrine disruption should be a priority requirement when evaluating the health impacts of agricultural contaminants, including new agricultural chemicals for licensing, regulatory decisions, or re-registration of existing products. If evidence supports reasonable concern for developmental health consequences rather than demonstrated negative health effects, regulatory authorities including US EPA and USDA should adapt policy approaches to limit childhood and pregnancy exposure.

Include overall hazardous exposure reduction for children as a goal in agricultural chemical use and agricultural production policies and require assessment of alternatives where child health concern exists. For example, enhance promotion of integrated pest management and organic methods of agriculture, require assessment of alternatives, increase well water monitoring, and promote effective farm waste management strategies and zoning to reduce exposures to children their homes, daycares, and schools from contaminants.

References

1. Kirkhorn SR, Garry VF. Agricultural lung diseases. *Environ Health Perspect.* 2000; 108(Suppl 4): 705–712. [PubMed: 10931789]
2. Alavanja MC, Hoppin JA, Kamel F. Health effects of chronic pesticide exposure: cancer and neurotoxicity. *Annu Rev Public Health.* 2004; 25:155–197. [PubMed: 15015917]
3. Frank AL, McKnight R, Kirkhorn SR, Gunderson P. Issues of agricultural safety and health. *Annu Rev Public Health.* 2004; 25:225–245. [PubMed: 15015919]
4. LeJeune J, Kersting A. Zoonoses: an occupational hazard for livestock workers and a public health concern for rural communities. *J Agric Saf Health.* 2010; 16:161–179. [PubMed: 20836437]
5. Firestone M. Protecting children from environmental risks throughout each stage of their childhood. *J Expo Sci Environ Epidemiol.* 2010; 20:227–228. [PubMed: 20407448]
6. US EPA (Environmental Protection Agency). Exposure Factors Handbook. 2011. Washington, DC: National Center for Environmental Assessment; 2011. EPA/600/R-09/052F. Available from the National Technical Information Service, Springfield, VA. Available at: <http://www.epa.gov/ncea/efh> [Accessed March 9, 2012]
7. Miller MD, Marty MA, Arcus A, Brown J, Morry D, Sandy M. Differences between children and adults: implications for risk assessment at California EPA. *Int J Toxicol.* 2002; 21:403–418. [PubMed: 12396687]
8. Bearer CF. How are children different from adults? *Environ Health Perspect.* 1995; 103(Suppl 6):7–12.
9. US EPA. (Environmental Protection Agency). Child-Specific Exposure Factors Handbook (Final Report). Washington, DC: National Center for Environmental Assessment; 2008. EPA/600/R-06/096F
10. Freeman NC, Jimenez M, Reed KJ, et al. Quantitative analysis of children's microactivity patterns: The Minnesota Children's Pesticide Exposure Study. *J Expo Anal Environ Epidemiol.* 2001; 11:501–509. [PubMed: 11791166]

11. Huen K, Harley K, Brooks J, et al. Developmental changes in PON1 enzyme activity in young children and effects of PON1 polymorphisms. *Environ Health Perspect.* 2009; 117:1632–1638. [PubMed: 20019917]
12. US EPA. (Environmental Protection Agency). Risk Assessment Forum, Supplemental Guidance for Assessing Susceptibility from Early-Life Exposure to Carcinogens. Washington, DC: National Center for Environmental Assessment; 2005. EPA/630/R-03/003F
13. Washington State Department of Health Pesticide Program. Illness Monitoring and Prevention. [Accessed May 18, 2011] Hazards of home foggers and aerosol insecticides: observations and recommendations prepared by Barbara Morrissey, Toxicologist. Jul 18, 2008. Available at: <http://www.doh.wa.gov/ehp/pest/fog-epa.pdf>
14. Bronstein AC, Spyker DA, Cantilena LR Jr, Green JL, Rumack BH, Giffin SL. 2009 Annual Report of the American Association of Poison Control Centers' National Poison Data System (NPDS): 27th Annual Report. *Clin Toxicol (Phila).* 2010; 48:979–1178. [PubMed: 21192756]
15. Alarcon WA, Calvert GM, Blondell JM, et al. Acute illnesses associated with pesticide exposure at schools. *JAMA.* 2005; 294:455–465. [PubMed: 16046652]
16. [Accessed May 18, 2011] Washington State Department of Health Pesticide Incident Reporting and Tracking (PIRT) Review Panel. 2002 Annual Report. Dec, 2002. Available at: <http://www.doh.wa.gov/ehp/PIRT/PIRT2002AR-2000data.pdf>
17. Lu C, Fenske RA, Simcox NJ, Kalman D. Pesticide exposure of children in an agricultural community: evidence of household proximity to farmland and take home exposure pathways. *Environ Res.* 2000; 84:290–302. [PubMed: 11097803]
18. Curl CL, Fenske RA, Kissel JC, et al. Evaluation of take-home organophosphorus pesticide exposure among agricultural workers and their children. *Environ Health Perspect.* 2002; 110:A787–A792. [PubMed: 12460819]
19. Quirós-Alcalá L, Bradman A, Nishioka M, et al. Pesticides in house dust from urban and farmworker households in California: an observational measurement study. *Environ Health.* 2011; 10:1–15. [PubMed: 21205326]
20. Fenske RA, Lu C, Barr D, Needham L. Children's exposure to chlorpyrifos and parathion in an agricultural community in central Washington State. *Environ Health Perspect.* 2002; 110:549–553. [PubMed: 12003762]
21. US Geological Survey. Pesticides in the Nation's Streams and Ground Water, 1992–2001—A Summary. Washington, DC: US Department of the Interior; 2006. Available at: <http://pubs.usgs.gov/fs/2006/3028/pdf/fs2006-3028.pdf> [Accessed May 28, 2011]
22. Arcury TA, Grzywacz JG, Barr DB, Tapia J, Chen H, Quandt SA. Pesticide urinary metabolite levels of children in eastern North Carolina farmworker households. *Environ Health Perspect.* 2007; 115:1254–1260. [PubMed: 17687456]
23. Bradman A, Eskenazi B, Barr DB, et al. Organophosphate urinary metabolite levels during pregnancy and after delivery in women living in an agricultural community. *Environ Health Perspect.* 2005; 113:1802–1807. [PubMed: 16330368]
24. Calvert GM, Alarcon WA, Chelminski A, et al. Case report: three farmworkers who gave birth to infants with birth defects closely grouped in time and place—Florida and North Carolina, 2004–2005. *Environ Health Perspect.* 2007; 115:787–791. [PubMed: 17520069]
25. Zwiener RJ, Ginsburg CM. Organophosphate and carbamate poisoning in infants and children. *Pediatrics.* 1988; 81:121–126. [PubMed: 3336578]
26. Ruckart PZ, Kakolewski K, Bove FJ, Kaye WE. Long-term neurobehavioral health effects of methyl parathion exposure in children in Mississippi and Ohio. *Environ Health Perspect.* 2004; 112:46–51. [PubMed: 14698930]
27. Karr C, Murphy H, Glew G, Keifer M, Fenske M. Pacific Northwest health professionals survey on pesticides and children. *J Agromedicine.* 2006; 11:113–120. [PubMed: 19274903]
28. Schenk M, Popp SM, Neale AV, Demers RY. Environmental medicine content in medical school curricula. *Acad Med.* 1996; 71:499–501. [PubMed: 9114870]
29. Roberts JR, Gitterman BA. Pediatric environmental health education: a survey of US pediatric residency programs. *Ambul Pediatr.* 2003; 3:57–59. [PubMed: 12540256]

30. Hiott AE, Quandt SA, Early J, Jackson DS, Arcury TA. Review of pesticide education materials for health care providers providing care to agricultural workers. *J Rural Health*. 2006; 22:17–25. [PubMed: 16441332]
31. Reigart JR, Roberts JR. Pesticides in children. *Pediatr Clin North Am*. 2001; 48:1185–1198. ix. [PubMed: 11579668]
32. Keifer M, Gasperini F, Robson M. Pesticides and other chemicals: minimizing worker exposures. *J Agromedicine*. 2010; 15:264–274. [PubMed: 20665311]
33. American Public Health Association. [Accessed March 9, 2012] APHA Policy Number: 20108: Requiring clinical diagnostic tools and biomonitoring of exposures to pesticides. Policy Date: November 9, 2010. Available at: <http://www.apha.org/advocacy/policy/policysearch/default.htm?id=1400>
34. O'Malley M, Barry T, Ibarra M, Verder-Carlos M, Mehler L. Illnesses related to shank application of metamsodium, Arvin, California, July 2002. *J Agromedicine*. 2005; 10:27–42.
35. Hoppin JA, Umbach DM, London SJ, Lynch CF, Alavanja MC, Sandler DP. Pesticides and adult respiratory outcomes in the agricultural health study. *Ann N Y Acad Sci*. 2006; 1076:343–354. [PubMed: 17119214]
36. Hoppin JA, Umbach DM, London SJ, Lynch CF, Alavanja MC, Sandler DP. Pesticides associated with wheeze among commercial pesticide applicators in the Agricultural Health Study. *Am J Epidemiol*. 2006; 163:1129–1137. [PubMed: 16611668]
37. Kofman O, Berger A, Massarwa A, Friedman A, Jaffar AA. Motor inhibition and learning impairments in school-aged children following exposure to organophosphate pesticides in infancy. *Pediatr Res*. 2006; 60:88–92. [PubMed: 16788088]
38. Engel SM, Berkowitz GS, Barr DB, et al. Prenatal organophosphate metabolite and organochlorine levels and performance on the Brazelton Neonatal Behavioral Assessment Scale in a multiethnic pregnancy cohort. *Am J Epidemiol*. 2007; 165:1397–1404. [PubMed: 17406008]
39. Young JG, Eskenazi B, Gladstone EA, et al. Association between in utero organophosphate pesticide exposure and abnormal reflexes in neonates. *Neurotoxicology*. 2005; 26:199–209. [PubMed: 15713341]
40. Eskenazi B, Marks AR, Bradman A, et al. Organophosphate pesticide exposure and neurodevelopment in young Mexican-American children. *Environ Health Perspect*. 2007; 115:792–798. [PubMed: 17520070]
41. Rauh VA, Garfinkel R, Perera FP, et al. Impact of prenatal chlorpyrifos exposure on neurodevelopment in the first 3 years of life among inner-city children. *Pediatrics*. 2006; 118:e1845–e1859. [PubMed: 17116700]
42. Rauh V, Arunajadai S, Horton M, et al. Seven-year neurodevelopmental scores and prenatal exposure to chlorpyrifos, a common agricultural pesticide. *Environ Health Perspect*. 2011; 119:1196–1201. [PubMed: 21507777]
43. Bouchard MF, Chevrier J, Harley KG, et al. Prenatal exposure to organophosphate pesticides and IQ in 7-year old children. *Environ Health Perspect*. 2011; 119:1189–1195. [PubMed: 21507776]
44. Engel SM, Wetmur J, Chen J, et al. Prenatal exposure to organophosphates, paraoxonase 1, and cognitive development in childhood. *Environ Health Perspect*. 2011; 119:1182–1188. [PubMed: 21507778]
45. Bouchard MF, Bellinger DC, Wright RO, Weisskopf MG. Attention-deficit/hyperactivity disorder and urinary metabolites of organophosphate pesticides. *Pediatrics*. 2010; 125:e1270–e1277. [PubMed: 20478945]
46. Marks AR, Harley K, Bradman A, et al. Organophosphate pesticide exposure and attention in young Mexican-American children: the CHAMACOS study. *Environ Health Perspect*. 2010; 118:1768–1774. [PubMed: 21126939]
47. Grandjean P, Harari R, Barr DB, Debes F. Pesticide exposure and stunting as independent predictors of neurobehavioral deficits in Ecuadorian school children. *Pediatrics*. 2006; 117:e546–e556. [PubMed: 16510633]
48. Rohlman DS, Arcury TA, Quandt SA, et al. Neurobehavioral performance in preschool children from agricultural and non-agricultural communities in Oregon and North Carolina. *Neurotoxicology*. 2005; 26:589–598. [PubMed: 16112324]

49. Wigle DT, Turner MC, Krewski D. A systematic review and meta-analysis of childhood leukemia and parental occupational pesticide exposure. *Environ Health Perspect.* 2009; 117:1505–1513. [PubMed: 20019898]
50. Flower KB, Hoppin JA, Lynch CF, et al. Cancer risk and parental pesticide application in children of agricultural health study participants. *Environ Health Perspect.* 2004; 112:631–635. [PubMed: 15064173]
51. Infante-Rivard C, Weichenthal S. Pesticides and childhood cancer: an update of Zahm and Ward's 1998 review. *J Toxicol Environ Health B Crit Rev.* 2007; 10:81–99. [PubMed: 18074305]
52. Zahm SH, Ward MH. Pesticides and childhood cancer. *Environ Health Perspect.* 1998; 106:893–908. [PubMed: 9646054]
53. Vinson F, Merhi M, Baldi I, Raynal H, Gamet-Payrastré L. Exposure to pesticides and risk of childhood cancer: a meta-analysis of recent epidemiological studies. *Occup Environ Med.* 2011; 68:694–702. [PubMed: 21606468]
54. Centers for Disease Control and Prevention. Fourth National Report on Human Exposure to Environmental Chemicals. Pyrethroids. Atlanta: Centers for Disease Control and Prevention; Dec, 2009. Available at: <http://www.cdc.gov/exposurereport/> [Accessed March 9, 2012]
55. Horton MK, Rundle A, Camann DE, Boyd Barr D, Rauh VA, Whyatt RM. Impact of prenatal exposure to piperonyl butoxide and permethrin on 36-month neurodevelopment. *Pediatrics.* 2011; 127:e699–e706. [PubMed: 21300677]
56. Baker BP, Benbrook CM, Groth E 3rd, Lutz Benbrook K. Pesticide residues in conventional, integrated pest management (IPM)-grown and organic foods: insights from three US data sets. *Food Addit Contam.* 2002; 19:427–446. [PubMed: 12028642]
57. Lu C, Toepel K, Irish R, Fenske RA, Barr DB, Bravo R. Organic diets significantly lower children's exposure to organophosphorus pesticides. *Environ Health Perspect.* 2006; 114:260–263. [PubMed: 16451864]
58. Shea KM. Antibiotic resistance: what is the impact of agricultural uses of antibiotics on children's health? *Pediatrics.* 2003; 112:253–258. [PubMed: 12837918]
59. Swartz, MN. Human Health Risks With the Subtherapeutic Use of Penicillin or Tetracyclines in Animal Feed. Washington, DC: National Academies Press; 1989. Committee on Human Risk Assessment of Using Subtherapeutic Antibiotics in Animal Feeds, Institute of Medicine, Division of Health Promotion and Disease Prevention.
60. National Research Council. Air Emissions from Animal Feeding Operations: Current Knowledge, Future Needs. Washington, DC: National Academies Press; 2003.
61. Avery RC, Wing S, Marshall SW, Schiffman SS. Odor from industrial hog farming operations and mucosal immune function in neighbors. *Arch Environ Health.* 2004; 59:101–108. [PubMed: 16075904]
62. MacDonald, JM., McBride, WD. The Transformation of U.S. Livestock Agriculture: Scale, Efficiency, and Risks. Washington, DC: United States Department of Agriculture, Economic Research Service; Jan, 2009. Economic Information Bulletin Number 43. Available at: <http://www.ers.usda.gov/Publications/EIB43/EIB43.pdf> [Accessed May 19, 2011]
63. Mitloehner FM, Schenker MB. Environmental exposure and health effects from concentrated animal feeding operations. *Epidemiology.* 2007; 18:309–311. [PubMed: 17435438]
64. Heederik D, Sigsgaard T, Thorne PS, et al. Health effects of airborne exposures from concentrated animal feeding operations. *Environ Health Perspect.* 2007; 115:298–302. [PubMed: 17384782]
65. Respiratory health hazards in agriculture. *Am J Respir Crit Care Med.* 1998; 158:S1–S76. [PubMed: 9817727]
66. Omland Ø. Exposure and respiratory health in farming in temperate zones—a review of the literature. *Ann Agric Environ Med.* 2002; 9:119–136. [PubMed: 12498578]
67. Radon K, Schulze A, Ehrenstein V, van Strien RT, Praml G, Nowak D. Environmental exposure to confined animal feeding operations and respiratory health of neighboring residents. *Epidemiology.* 2007; 18:300–308. [PubMed: 17435437]
68. O'Connor AM, Auvermann B, Bickett-Weddle D, et al. The association between proximity to animal feeding operations and community health: a systematic review. *PLoS ONE.* 2010; 5:e9530. [PubMed: 20224825]

69. Sigurdarson ST, Kline JN. School proximity to concentrated animal feeding operations and prevalence of asthma in students. *Chest*. 2006; 129:1486–1491. [PubMed: 16778265]
70. Merchant JA, Naleway AL, Svendsen ER, et al. Asthma and farm exposures in a cohort of rural Iowa children. *Environ Health Perspect*. 2005; 113:350–356. [PubMed: 15743727]
71. Mirabelli MC, Wing S, Marshall SW, Wilcosky TC. Asthma symptoms among adolescents who attend public schools that are located near confined swine feeding operations. *Pediatrics*. 2006; 118:e66–e75. [PubMed: 16818539]
72. Centers for Disease Control and Prevention. [Accessed May 28, 2011] Asthma in the US: growing every year. *Vital Signs*. May, 2011. Available at: <http://www.cdc.gov/VitalSigns/Asthma/index.html>
73. von Mutius E. Environmental factors influencing the development and progression of pediatric asthma. *J Allergy Clin Immunol*. 2002; 109:S525–S532. [PubMed: 12063508]
74. Thorne PS, Kulhánková K, Yin M, Cohn R, Arbes SJ Jr, Zeldin DC. Endotoxin exposure is a risk factor for asthma; the national survey of endotoxin in United States housing. *Am J Respir Crit Care Med*. 2005; 172:1371–1377. [PubMed: 16141442]
75. Burow KR, Nolan BT, Rupert MG, Dubrovsky NM. Nitrate in groundwater of the United States, 1991–2003. *Environ Sci Technol*. 2010; 44:4988–4997. [PubMed: 20540531]
76. Greer FR, Shannon M, American Academy of Pediatrics Committee on Nutrition; American Academy of Pediatrics Council on Environmental Health. Infant methemoglobinemia: the role of dietary nitrate in food and water. *Pediatrics*. 2005; 116:784–786. [PubMed: 16140723]
77. Arbuckle TE, Sherman GJ, Corey PN, Walters D, Lo B. Water nitrates and CNS birth defects: a population-based case-control study. *Arch Environ Health*. 1988; 43:162–167. [PubMed: 3377550]
78. Cedergren MI, Selbing AJ, Löfman O, Källén BA. Chlorination byproducts and nitrate in drinking water and risk for congenital cardiac defects. *Environ Res*. 2002; 89:124–130. [PubMed: 12123645]
79. Croen LA, Todoroff K, Shaw GM. Maternal exposure to nitrate from drinking water and diet and risk for neural tube defects. *Am J Epidemiol*. 2001; 153:325–331. [PubMed: 11207149]
80. Fan AM, Steinberg VE. Health implications of nitrate and nitrite in drinking water: an update on methemoglobinemia occurrence and reproductive and developmental toxicity. *Regul Toxicol Pharmacol*. 1996; 23:35–43. [PubMed: 8628918]
81. Longnecker MP, Daniels JL. Environmental contaminants as etiologic factors for diabetes. *Environ Health Perspect*. 2001; 109:871–876.
82. US Environmental Protection Agency. National Pesticide Survey: Project Summary. Washington, DC: US Environmental Protection Agency; 1990. EPA Publication No. 570990NPS6
83. Rogan WJ, Brady MT. Committee on Environmental Health; Committee on Infectious Diseases. Drinking water from private wells and risk to children. *Pediatrics*. 2009; 123:1599–1605. [PubMed: 19482772]
84. Milz SA, Wilkins JR 3rd, Ames AL, Witherspoon MK. Occupational noise exposures among three farm families in northwest Ohio. *J Agromedicine*. 2008; 13:165–174. [PubMed: 19064421]
85. Perry MJ, May JJ. Noise and chemical induced hearing loss: special considerations for farm youth. *J Agromedicine*. 2005; 10:49–55.
86. Marvel ME, Pratt DS, Marvel LH, Regan M, May JJ. Occupational hearing loss in New York dairy farmers. *Am J Ind Med*. 1991; 20:517–531. [PubMed: 1785614]
87. Broste SK, Hansen DA, Strand RL, Stueland DT. Hearing loss among high school farm students. *Am J Public Health*. 1989; 79:619–622. [PubMed: 2784948]
88. Renick KM, Crawford JM, Wilkins JR 3rd. Hearing loss among Ohio farm youth: a comparison to a national sample. *Am J Ind Med*. 2009; 52:233–239. [PubMed: 19089836]
89. Balk SJ. Council on Environmental Health. Section on Dermatology. Ultraviolet radiation: a hazard to children and adolescents. *Pediatrics*. 2011; 127:588–597. [PubMed: 21357336]
90. Blair A, Freeman LB. Epidemiologic studies in agricultural populations: observations and future directions. *J Agromedicine*. 2009; 14:125–131. [PubMed: 19437268]
91. Alavanja MC, Sandler DP, Lynch CF, et al. Cancer incidence in the agricultural health study. *Scand J Work Environ Health*. 2005; 31(Suppl 1):39–45. [PubMed: 16190148]

92. Hogan DJ, Lane PR, Gran L, Wong D. Risk factors for squamous cell carcinoma of the skin in Saskatchewan, Canada. *J Dermatol Sci*. 1990; 1:97–101. [PubMed: 2100549]
93. Youl P, Aitken J, Hayward N, et al. Melanoma in adolescents: a case-control study of risk factors in Queensland, Australia. *Int J Cancer*. 2002; 98:92–98. [PubMed: 11857391]
94. Hansen AM, Raaschou-Nielsen O, Knudsen LE. Urinary 1-hydroxypyrene in children living in city and rural residences in Denmark. *Sci Total Environ*. 2006; 363:70–77. [PubMed: 16832893]
95. Davis KJ, Cokkinides VE, Weinstock MA, O’Connell MC, Wingo PA. Summer sunburn and sun exposure among US youths ages 11 to 18: national prevalence and associated factors. *Pediatrics*. 2002; 110:27–35. [PubMed: 12093943]
96. Miron D, Kenes J, Dagan R. Calves as a source of an outbreak of cryptosporidiosis among young children in an agricultural closed community. *Pediatr Infect Dis J*. 1991; 10:438–441. [PubMed: 1852540]
97. Studahl A, Andersson Y. Risk factors for indigenous *Campylobacter* infection: a Swedish case-control study. *Epidemiol Infect*. 2000; 125:269–275. [PubMed: 11117949]
98. Hawker JI, Ayres JG, Blair I, et al. A large outbreak of Q fever in the West Midlands: windborne spread into a metropolitan area? *Community Dis Public Health*. 1998; 1:180–187.
99. Donham, KJ., Thelin, A. *Agricultural Medicine: Occupational and Environmental Health for the Health Professional*. Ames, Iowa: Wiley-Blackwell; 2006. Special risk populations in agricultural communities; p. 29-63.
100. Bell BP, Griffin PM, Lozano P, Christie DL, Kobayashi JM, Tarr PI. Predictors of hemolytic uremic syndrome in children during a large outbreak of *Escherichia coli* O157:H7 infections. *Pediatrics*. 1997; 100:E12. [PubMed: 9200386]
101. Belongia EA, Chyou PH, Greenlee RT, Perez-Perez G, Bibb WF, DeVries EO. Diarrhea incidence and farm-related risk factors for *Escherichia coli* O157:H7 and *Campylobacter jejuni* antibodies among rural children. *J Infect Dis*. 2003; 187:1460–1468. [PubMed: 12717628]
102. Landrigan PJ, Trasande L, Thorpe LE, et al. The National Children’s Study: a 21-year prospective study of 100,000 American children. *Pediatrics*. 2006; 118:2173–2186. [PubMed: 17079592]

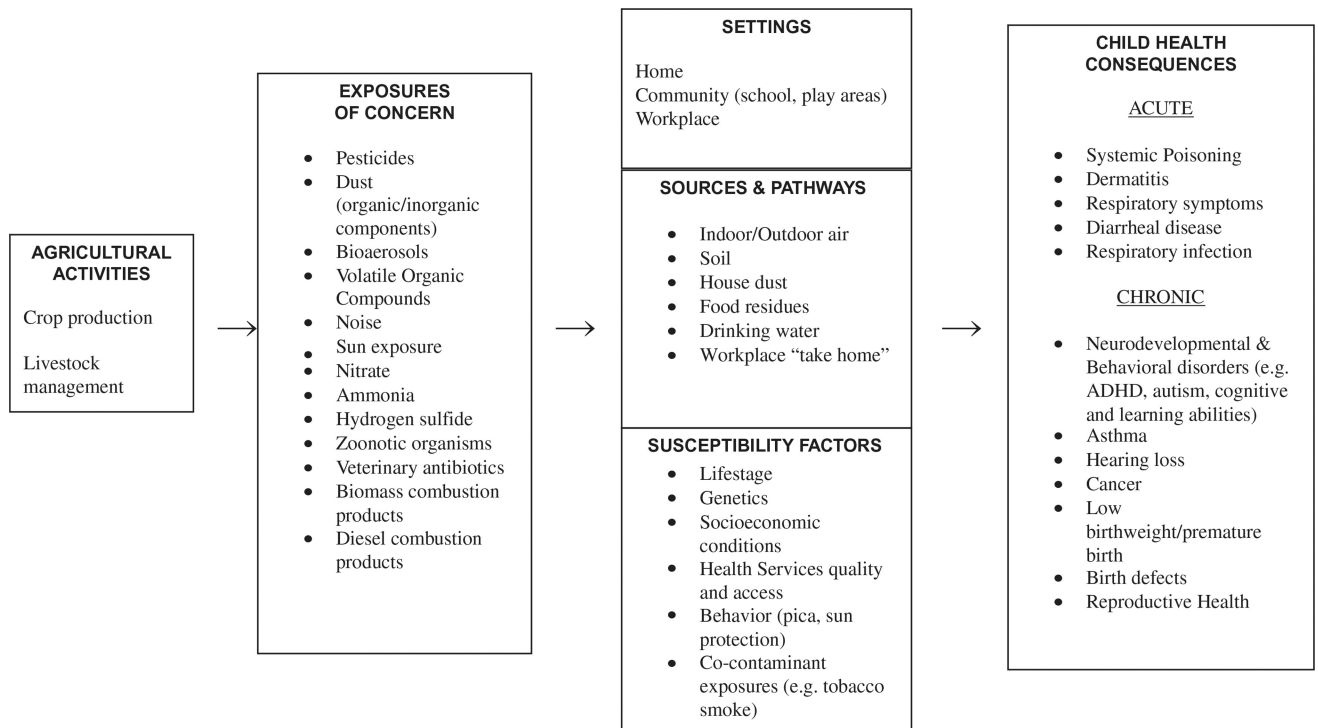


FIGURE 1. Factors influencing environmental health consequences of agricultural production on children.