Wendy Klag Center for Autism
& Developmental Disabilities

at the Johns Hopkins Bloomberg School of Public Health

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A Tale of Traffic and Smog: How the Air We Breathe Can Affect the Developing Brain

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Johns Hopkins University

A.J. Drexel Autism Institute

Fourth Annual Autism Public Health Lecture

March 29, 2016

Autism Spectrum Disorder

SOCIAL SKILLS
nonverbal interactions
friendship
joint attention
reciprocity

COMMUNICATION
language
conversation
play

UNUSUAL BEHAVIORS
obsessive interests
rigid rituals
preoccupation with
parts of objects

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ASD Prevalence in the United States

Statistics from the Autism and Developmental Disabilities Monitoring Network (ADDM)				
Surveillance Year	Birth Year	Number Locations	Prevalence per 1,000 Children	~ 1 in X children…
2000	1992	6	6.7	1 / 150
2002	1994	14	6.6	1 / 150
2004	1996	8	8.0	1 / 125
2006	1998	11	9.0	1 / 110
2008	2000	14	11.3	1 / 88
2010	2002	11	14.7	1 / 68

Family History

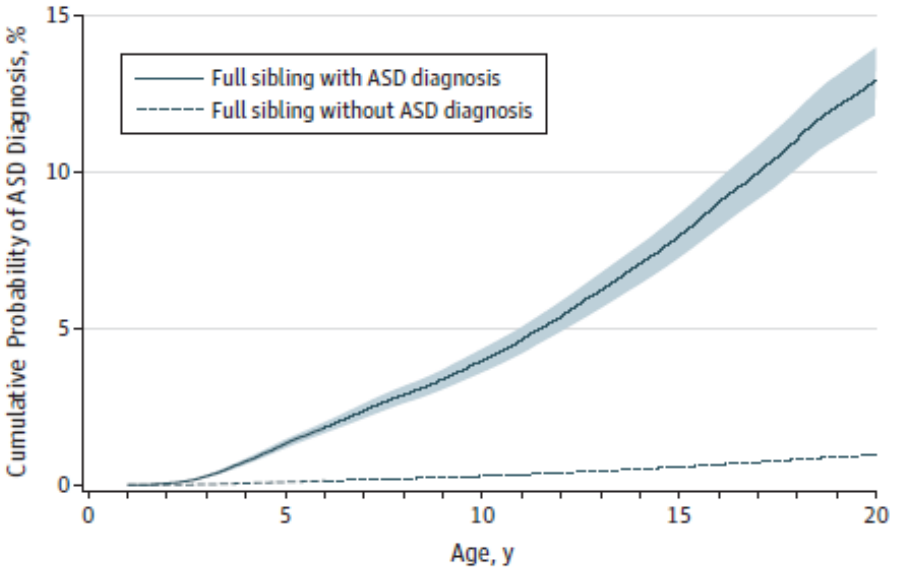
Original Investigation

The Familial Risk of Autism

Sven Sandin, MSc; Paul Lichtenstein, PhD; Ralf Kuja-Halkola, MSc; Henrik Larsson, PhD; Christina M. Hultman, PhD; Abraham Reichenberg, PhD

More likely to have ASD diagnosis if have a sibling with an ASD diagnosis

Figure 1. Age-Cumulative Probabilities for ASD Diagnosis in Siblings With a Full Sibling With ASD and in Siblings With a Full Sibling Without an ASD Diagnosis



ASD indicates autism spectrum disorder. Shaded areas represent 95% 2-sided point-wise confidence interval bands. The siblings who had a full-sibling with ASD were followed for 76 481 person-years resulting in 634 ASD events. The siblings who had a full sibling without ASD were followed for 35 486 922 person-years resulting in 17 327 ASD events.

PEDIATRICS®
OFFICIAL JOURNAL OF THE AMERICAN ACADEMY OF PEDIATRICS

Recurrence Risk for Autism Spectrum Disorders: A Baby Siblings Research Consortium Study

Sally Ozonoff, Gregory S. Young, Alice Carter, Daniel Messinger, Nurit Yirmiya, Lonnie Zwaigenbaum, Susan Bryson, Leslie J. Carver, John N. Constantino, Karen Dobkins, Ted Hutman, Jana M. Iverson, Rebecca Landa, Sally J. Rogers, Marian Sigman and Wendy L. Stone

Pediatrics 2011;128;e488; originally published online August 15, 2011;
DOI: 10.1542/peds.2010-2825

WHAT'S KNOWN ON THIS SUBJECT: The sibling recurrence risk of autism has been estimated to be between 3% and 10%. Previous research was affected by small samples and selection, stoppage, and reporting limitations. Updated estimates of recurrence risk are needed.

WHAT THIS STUDY ADDS: Studying a large sample and using a prospective longitudinal design, this study demonstrated that the sibling recurrence risk of autism spectrum disorder is substantially higher than previous estimates. This elevated risk has important implications for infant screening and genetic counseling.

abstract

FREE NIH

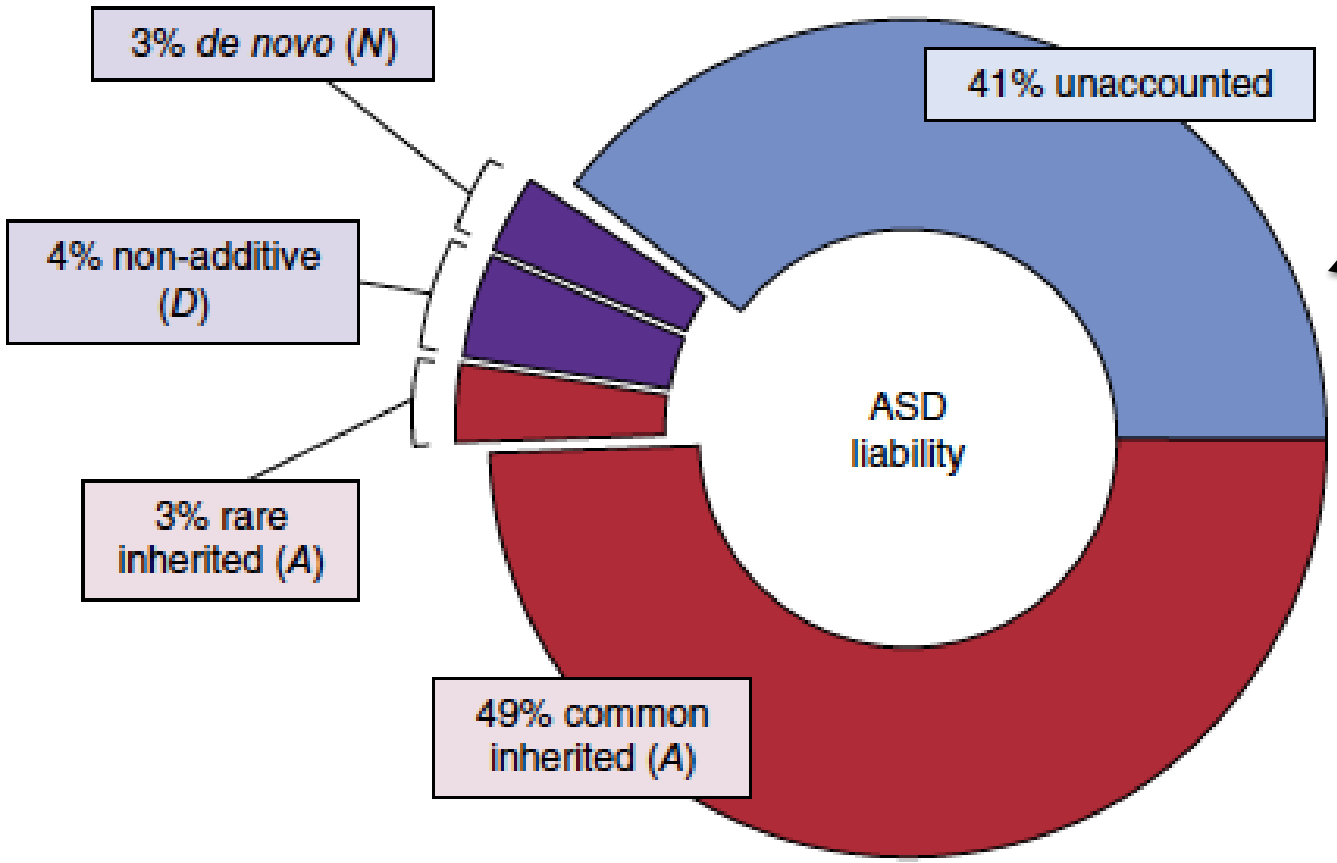
OBJECTIVE: The recurrence risk of autism spectrum disorders (ASD) is estimated to be between 3% and 10%, but previous research was limited by small sample sizes and biases related to ascertainment, reporting, and stoppage factors. This study used prospective methods to obtain an updated estimate of sibling recurrence risk for ASD.

METHODS: A prospective longitudinal study of infants at risk for ASD was conducted by a multisite international network, the Baby Siblings Research Consortium. Infants ($n = 664$) with an older biological sibling with ASD were followed from early in life to 36 months, when they were classified as having or not having ASD. An ASD classification required surpassing the cutoff of the Autism Diagnostic Observation Schedule and receiving a clinical diagnosis from an expert clinician.

RESULTS: A total of 18.7% of the infants developed ASD. Infant gender and the presence of >1 older affected sibling were significant predictors of ASD outcome, and there was an almost threefold increase in risk for male subjects and an additional twofold increase in risk if there was >1 older affected sibling. The age of the infant at study enrollment, the gender and functioning level of the infant's older sibling, and other demographic factors did not predict ASD outcome.

CONCLUSIONS: The sibling recurrence rate of ASD is higher than suggested by previous estimates. The size of the current sample and prospective nature of data collection minimized many limitations of previous studies of sibling recurrence. Clinical implications, including genetic counseling, are discussed. *Pediatrics* 2011;128:e488–e495

Genetic Influences on ASD

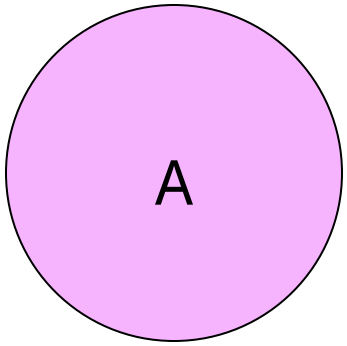


Environmental Effects and GxE Interactions

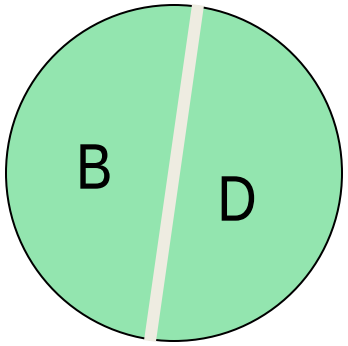
Adapted from Gaugler et al., 2014

Autism Causation is Multifactorial

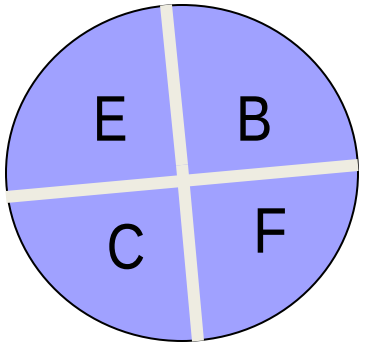
Likely to be many causes across the population *and* within an individual…



More Rare:
1 Cause



Possible:
2 Causes



More Common:
3 or More Causes

…from both genes and / or the environment(s)

Recognition

Psychogenic Risk Factors

Genetic Risk Factors

Genes & Environment Today?

1940 1950 1960 1970 1980 1990 2000

Kanner / Asperger describe Autism - Kanner hints at in born biologic and psychogenic factors

Rimland publishes book that includes cogent nine-point argument favoring biologic as opposed to psychogenic causation of autism

Bettleheim publishes "The Empty Fortress" which asserts that "refrigerator mothers" are a primary cause of autism

Folstein and Rutter publish first autism twin study indicating substantive heritable component to autism

Courtesy of Dr. Craig Newschaffer

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Early Life Exposure Matters

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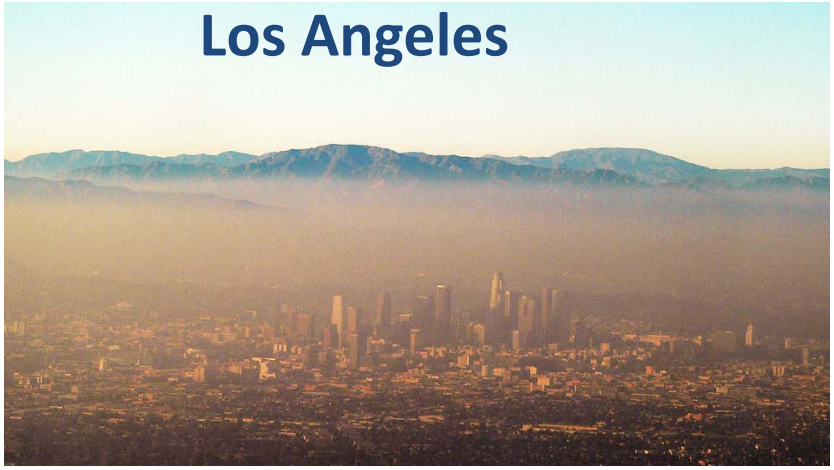
A CENTURY OF SAVING LIVES
100
MILLIONS AT A TIME
1916–2016



We are ALL exposed….

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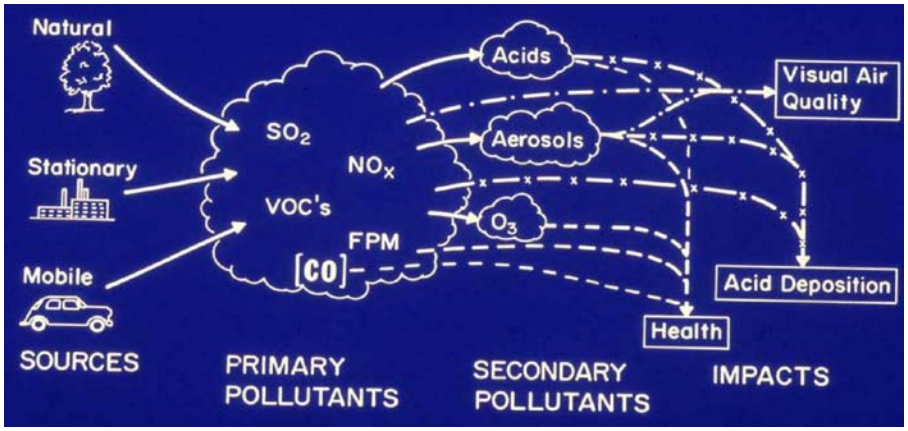
A CENTURY OF SAVING LIVES
100
MILLIONS AT A TIME
1916–2016

…air pollution involves more than just cars…



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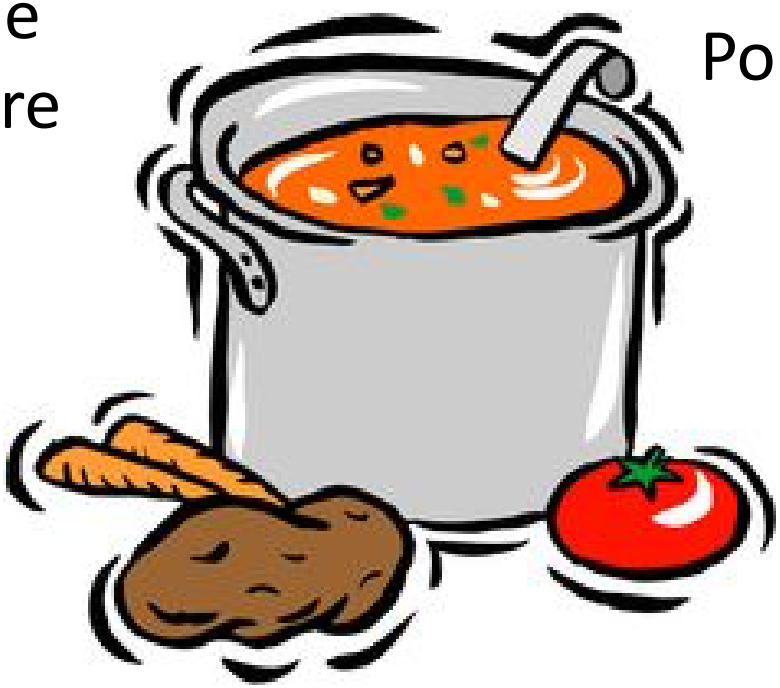
Air Pollution Is A Mixture

Gases

Nitrogen Dioxide
Ozone
& More

Volatile Agents

Polycyclic Aromatic
Hydrocarbons
& More



Particles

Coarse
Fine
Ultrafine
Metals

What Do We Mean When We Talk About Particles?

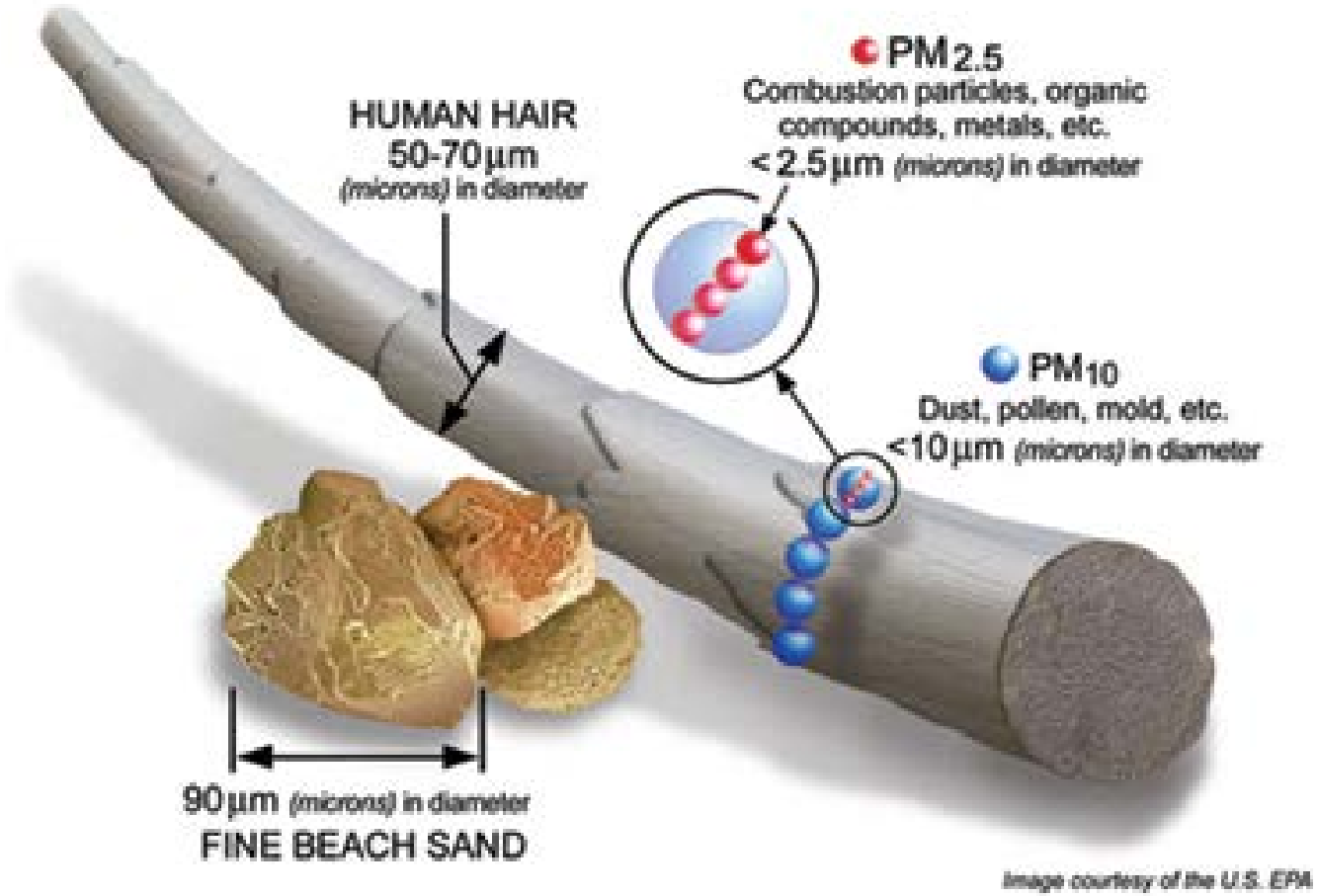


Image courtesy of the U.S. EPA

Health Effects of Air Pollution

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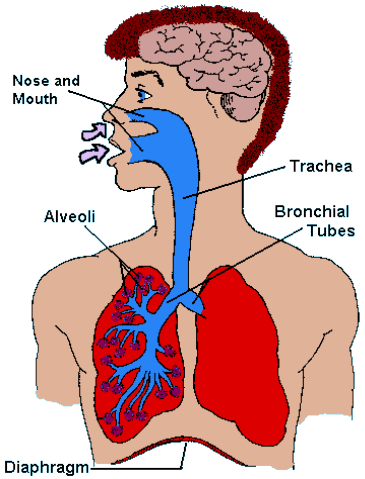
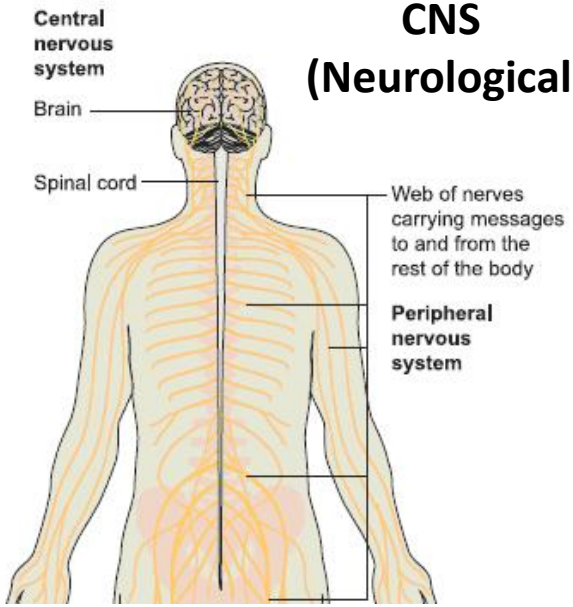
A CENTURY OF SAVING LIVES
100
MILLIONS AT A TIME
1916–2016

Cardiovascular Morbidity & Mortality

Respiratory Morbidity & Mortality



CNS (Neurological)

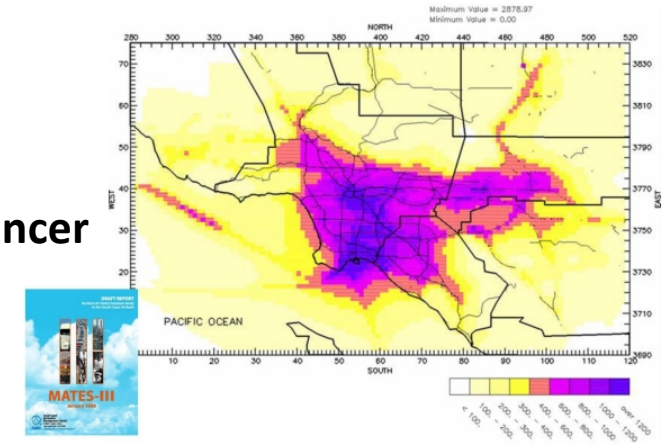


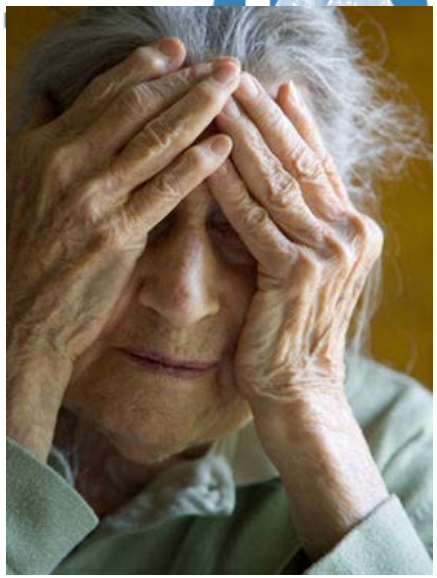
Perinatal Development

Assorted Birth Outcomes

Cancer

MATES-III Modeled Cancer Risk






F
SEE ME AFTER CLASS!
BOO
's good. I liked it a lo
have any pictures
derstand


a u t i s m

What Can Animal Studies Tell Us?

Direct Effects of Exposure

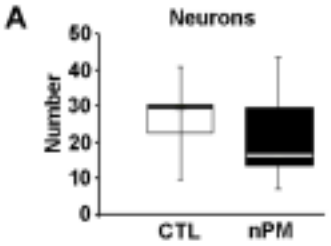
- Re-aerosolized UFPM from LA roadways
  - Increased inflammation in brain
  - Inflammatory cytokine activation
  - Changes in neuronal growth and differentiation
  - Depression symptoms

Trans-placental Effects

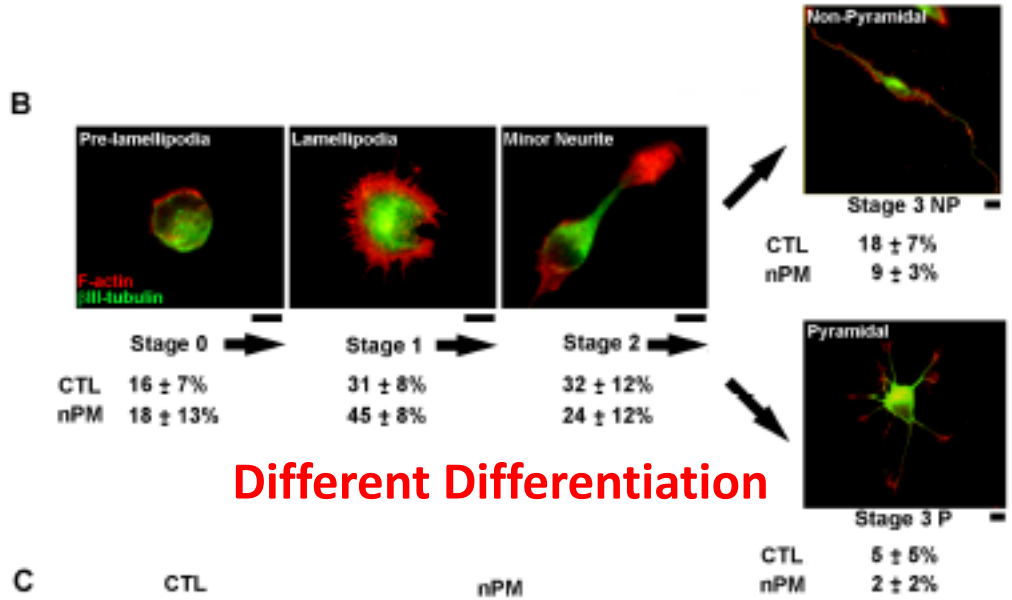
- *In utero* DEP exposure
  - Inflammation in brains of exposed pups
  - Male-specific effects

Kleinman et al., 2008
Morgan et al., 2011
Davis et al., 2013
Bolton et al., 2013

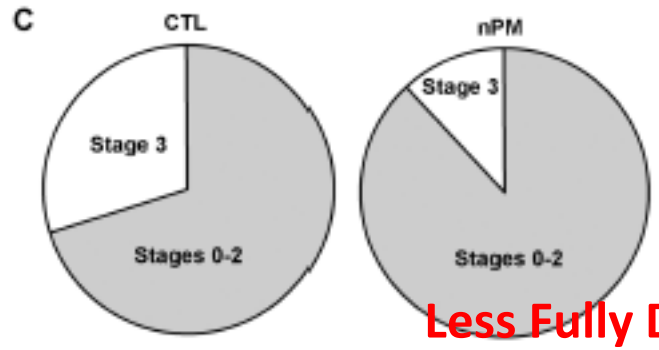
So What ... Brain?



Same Number



Different Differentiation



Less Fully Developed and Differentiated

OPEN ACCESS Freely availa

PLOS ONE

Prenatal Exp ... Causes Alter ... Like Respon ... es in Mice ... Depression-

David A. Davis[1☯], Mar ... o Iwata[1], Payam Pakbin[4], Jean ... utas[4], Caleb E. Finch[1,5*☯], To ...

1 Davis School of Gerontology, USC ... California, United States of America, 3 Dornsife College of Letters, Arts an ... USC, Los Angeles, California, United States of America, 5 Dept. of Neuro ... ool of Medicine, USC, Los Angeles, California, United States of America, ... Kansas, United States of America

Figure 1. Prenatal exposure to maternally inhaled nPM impairs postnatal differentiation and neurite initiation in cultured neurons. Cerebral cortex neurons from day 1 pups were grown 24 hours in primary culture. a) Neuronal numbers (median±IQR) did not differ by prenatal nPM inhalation exposure (nPM) vs. filtered room air (CTL). b) Stages of neuron differentiation, green: βIII-tubulin and red: F-actin; scale bar, 10 μm. Prenatal exposure to nPM reduced the proportion of Stage 3 neurons with definitive neurite extension (Stage 3 non-pyramidal & pyramidal, with elongated neurites ≥15 μm) (median±IQR). c) Summary of prenatal nPM effect on neurite elongation: CTL Air, 30% of neurons had neurites ≥1.5 μm; nPM, 12% of neurons in nPM Air cultures ($P<0.0001$). N = 5 neuronal cultures/group.

doi:10.1371/journal.pone.0064128.g001

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So What Does Air Pollution Do To the Brain?

Oppenheim *et al. Particle and Fibre Toxicology* 2013, **10**:62
http://www.particleandfibretoxicology.com/content/10/1/62

PARTICLE AND FIBRE TOXICOLOGY

RESEARCH Open Access

Exposure to vehicle emissions results in altered blood brain barrier permeability and expression of matrix metalloproteinases and tight junction proteins in mice

Hannah A Oppenheim[1], JoAnn Lucero[1,4], Anne-Cécile Guyot[2], Lindsay M Herbert[3], Jacob D McDonald[1], Aloïse Mabondzo[2] and Amie K Lund[1,4*]

Leaky Blood Brain Barrier

Conclusions: These data indicate that inhalation exposure to traffic-generated air pollutants promotes increased MMP activity and degradation of TJ proteins in the cerebral vasculature, resulting in altered BBB permeability and expression of neuroinflammatory markers.

So What Does Air Pollution Do To the Brain? Can It Even GET There?

TOXICOLOGICAL SCIENCES 140(1), 160–178 2014
doi: 10.1093/toxsci/kfu059
Advance Access publication April 1, 2014

Developmental Exposure to Concentrated Ambient Ultrafine Particulate Matter Air Pollution in Mice Results in Persistent and Sex-Dependent Behavioral Neurotoxicity and Glial Activation

ity. Although mechanisms of these effects remain to be fully elucidated, findings suggest that neurodevelopment and/or adulthood air pollution exposure may represent a significant underexplored risk factor for central nervous system diseases/disorders and thus a significant public health threat even beyond current appreciation.

How Do We Measure This In Humans?

Biologic Measures of Exposure

Personal Monitoring

Geographic Linkages To Monitoring Networks

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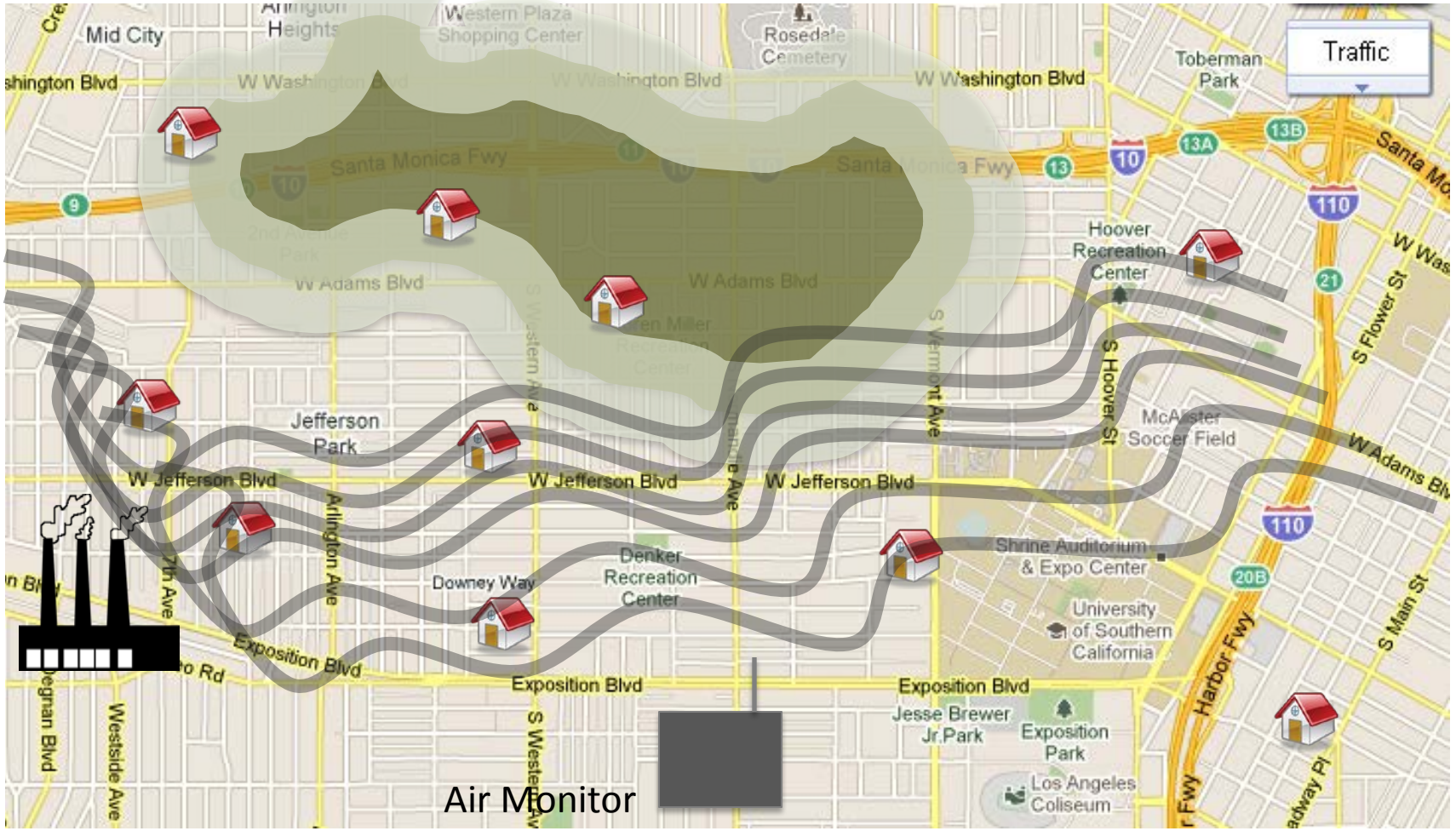
Measures of Air Pollution



Air Pollution Exposure and Early Life Neurodevelopment

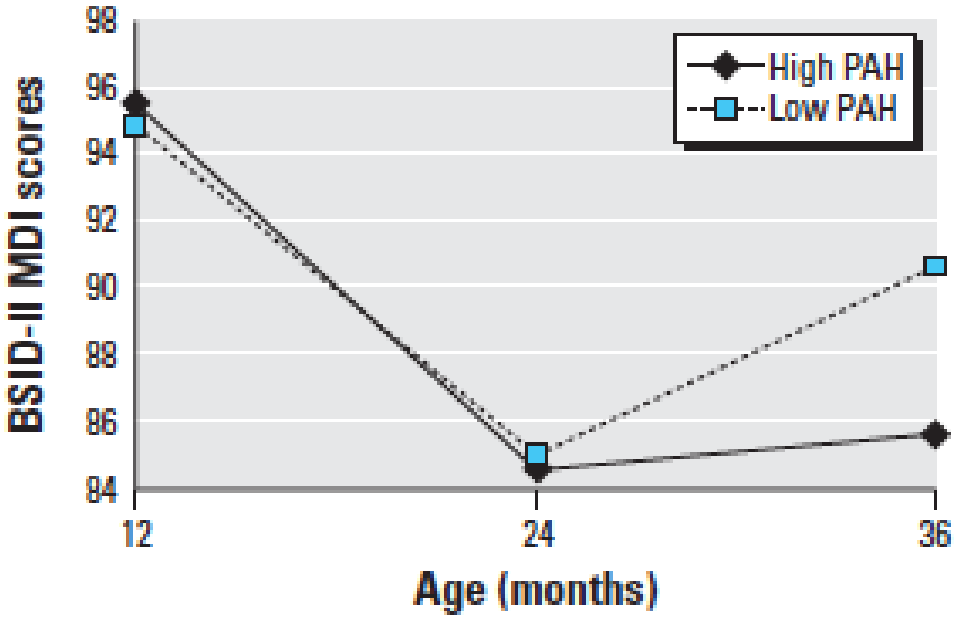


Figure 1. Estimated effects of prenatal PAH exposure on cognitive development in children 12 months through 36 months of age by GEE. The model was adjusted for the child's exact age at test administration, child's sex, ethnicity, gestational age at birth, quality of the (caretaking) home environment, and prenatal exposure to ETS and CPF.

Decreased cognitive development score with increasing exposure

Perera et al., 2006

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Roadway Proximity and Cognitive Development at Age 8

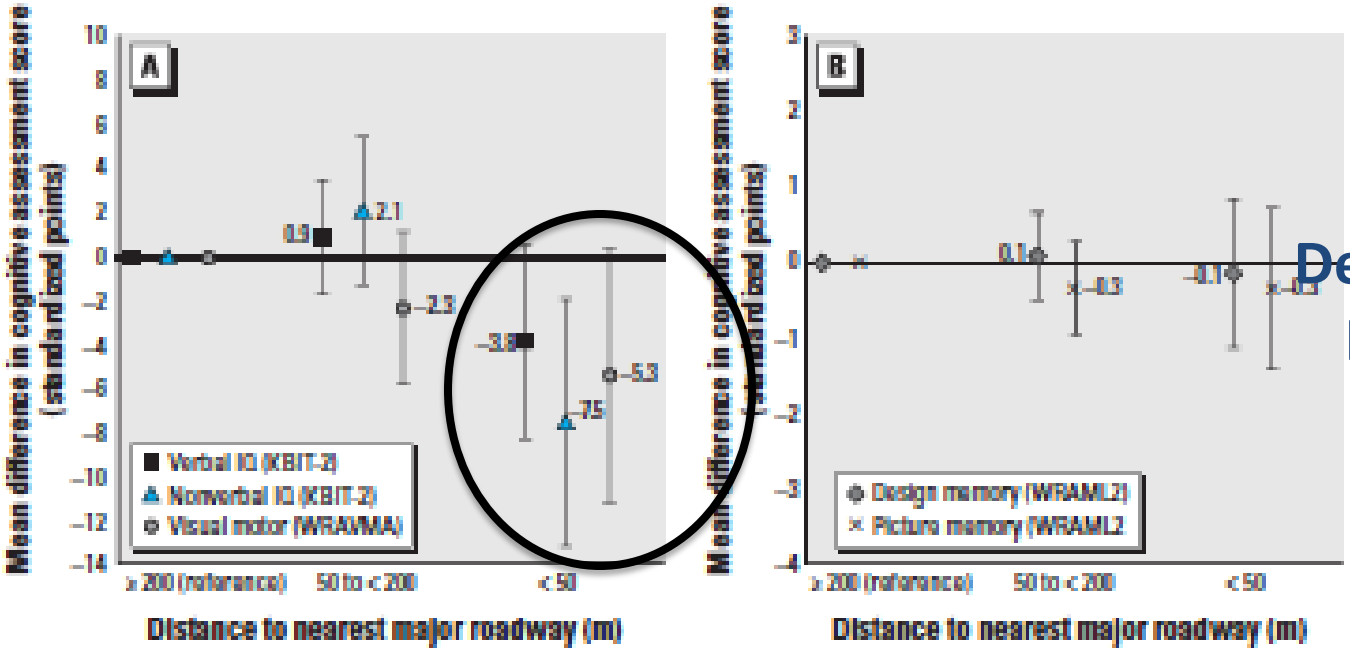

Figure 1. Mean differences (95% CIs) in cognitive assessment scores associated with residential proximity to major roadway at birth. (*A*) Results for standardized cognitive assessment scores scaled to mean ± SD = 100 ± 15 (KBIT-2 verbal and nonverbal IQ; WRAVMA visual motor). (*B*) Results for standardized cognitive assessment scores scaled to mean ± SD = 10 ± 3 (WRAML2 design memory and picture memory). All models were adjusted for characteristics of child (age, sex, breastfeeding duration, early childhood blood lead), mother (age, parity, race/ethnicity, education, IQ, marital/cohabitation status, and blood lead, smoking, secondhand smoke exposure, and alcohol in pregnancy), father (education), household (income, home caretaking environment, gas stove), and neighborhood (census tract median income).

Decreased non-verbal IQ with increasing proximity

Harris et al., 2015

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Air Pollutants and ASD

Hazardous Air Pollutants

- Mercury, cadmium, nickel, vinyl chloride, DEP
  - California
  - Windham et al., 2006
- Methylene chloride, styrene, quinoline
  - North Carolina
  - Kalkbrenner et al., 2010
- Styrene, chromium, PAH, methylene chloride
  - Pennsylvania
  - Talbott et al., 2015
- Metals (lead, manganese, mercury), methylene chloride, DEP
  - Across USA
  - Roberts et al., 2013
- Traffic and industry emissions
  - LA County
  - Von Ehrenstein et al., 2014

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Particles and Metals

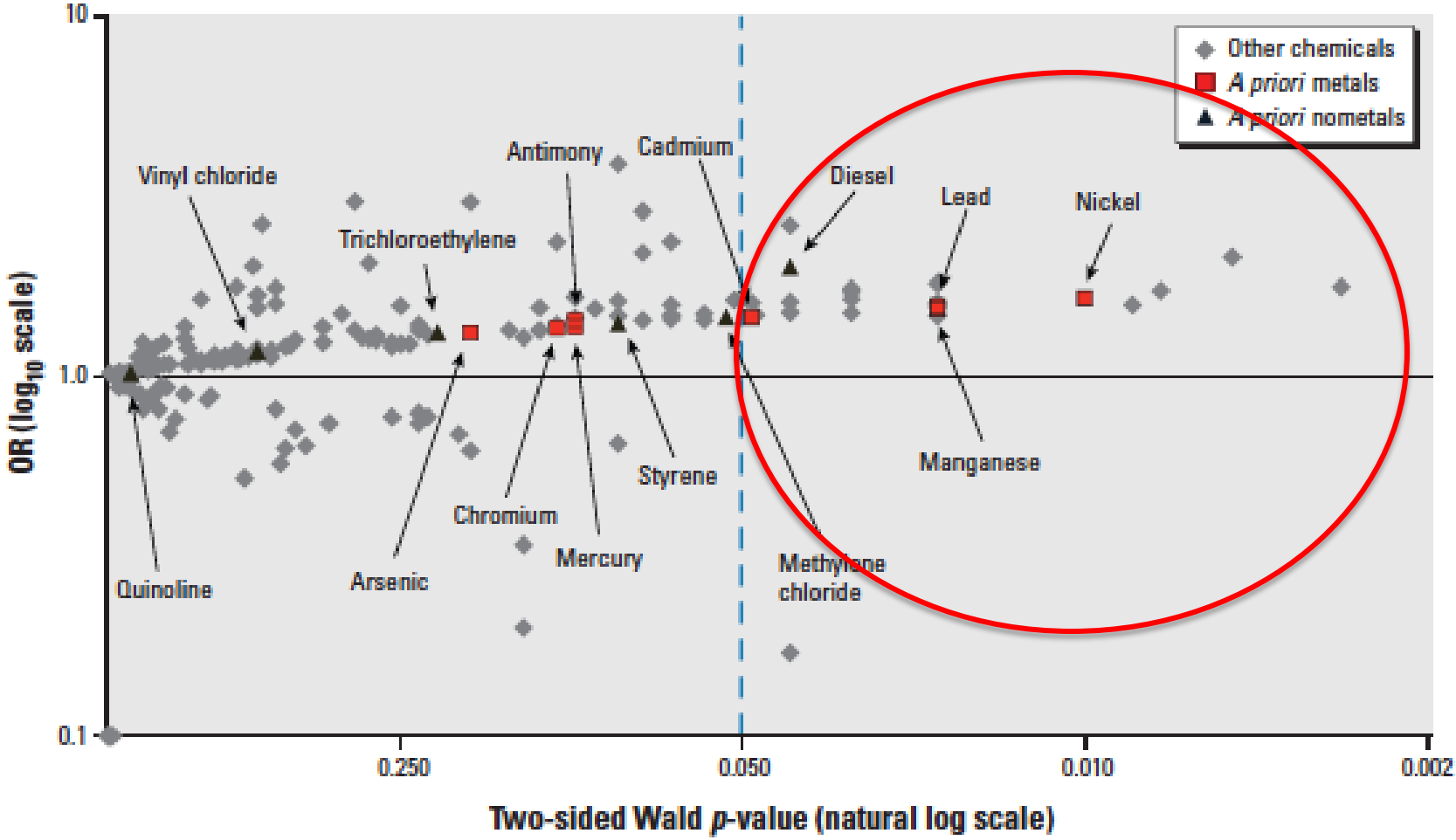

Figure 2. Association of ASD with air pollutant concentration, highest quintile versus lowest quintile ORs by Wald two-sided *p*-value, children of the Nurses' Health Study II ($n = 22,101$ controls, $n = 325$ cases).

Roberts et al., 2013

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Air Pollutants and ASD

Near Roadway Air Pollution and NO_2 in the US

- California
  - Volk et al., 2011, 2013
- LA County
  - Becerra et al., 2011

Studies from Abroad

- No association with NO_2 in 4 European birth cohorts
  - Guxens et al., 2016
- No association with NO_2 in Sweden
  - Gong et al., 2014
- NO_2, Ozone, SO_2, CO in Taiwan
  - Jung et al., 2013

Particulate Matter ($PM_{2.5}$ and PM_{10})

- California
  - Volk et al., 2013
- Across USA
  - Raz et al., 2015
- California and North Carolina
  - Kalkbrenner et al., 2014
- Pennsylvania
  - Talbott et al., 2015
- LA County
  - Becerra et al., 2013

Study Design

- Childhood Autism Risks From Genetics and the Environment (CHARGE) Study
  - Born 1996-2007
- Autism Cases
  - First diagnosed by Regional Center (Department of Developmental Services (DDS))or clinical referral
  - Positive for autism on gold standard assessments
- Controls
  - Typically developing children from birth records

NIEHS R01 ES015359 , PI: Hertz-Picciotto

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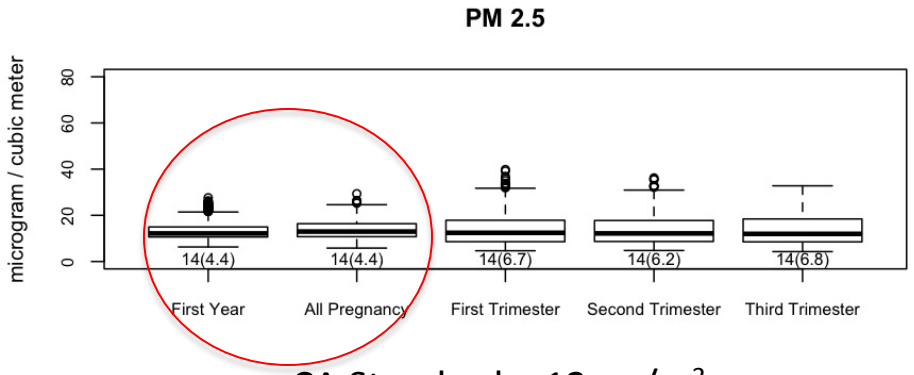
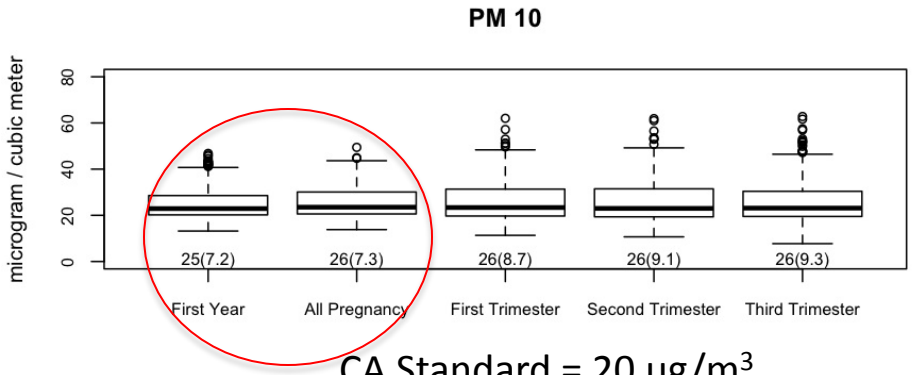
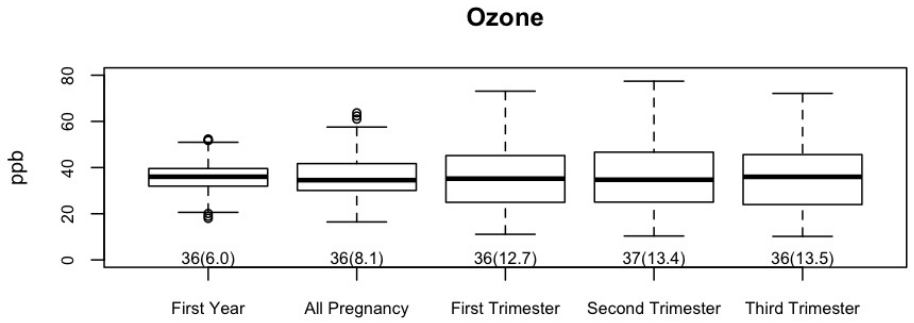
Air Pollution In CHARGE

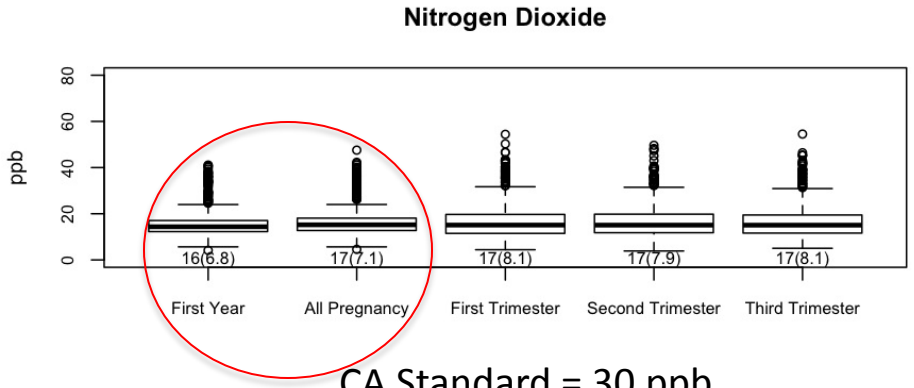


CA Standard = 12 ug/m^3



CA Standard = 20 ug/m^3





CA Standard = 30 ppb

Distance From Freeway at Birth
(304 Cases and 259 Controls)

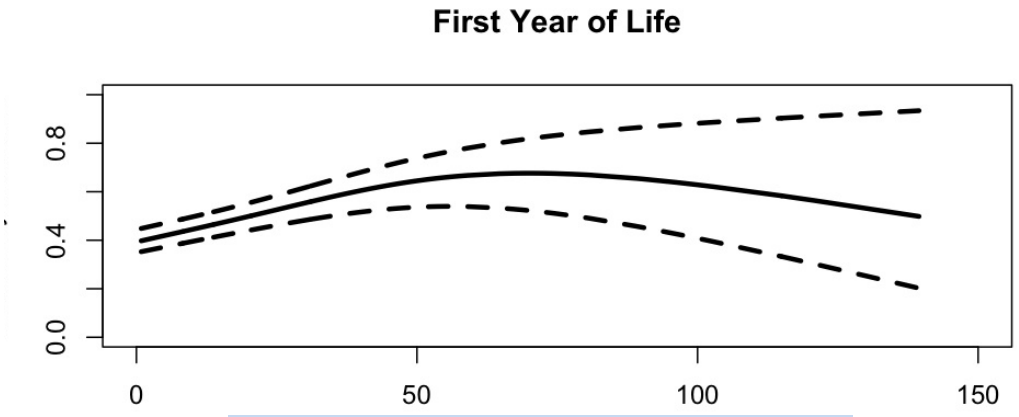
	Crude OR	Adjusted OR*
Birth Address <309m	1.86 (1.04-3.45)	1.86 (1.03-3.45)
Birth Address 309-647m	0.98 (0.60-1.59)	0.96 (0.58-1.56)
Birth Address 647-1419m	1.14 (0.76-1.71)	1.11 (0.73-1.67)
Birth Address >1419	Reference	Reference

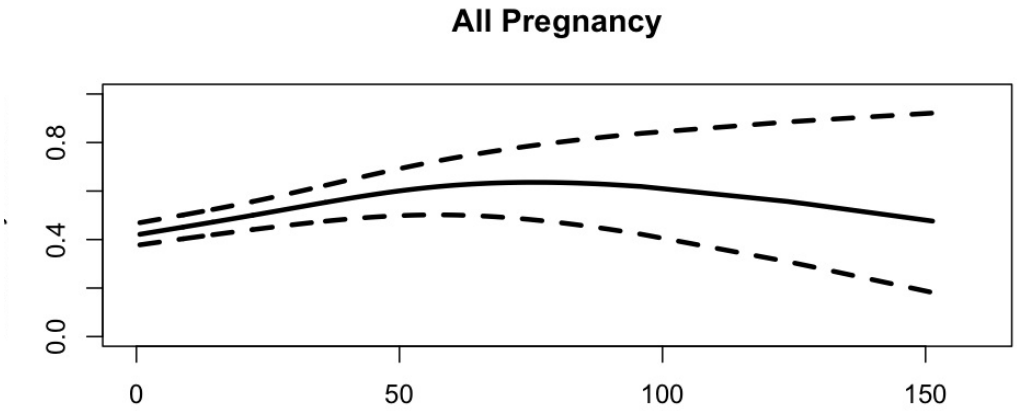
*Model adjusted for child male gender, child ethnicity (Hispanic vs. White, Black/Asian/Other vs. White), maximum education in home (college degree or more), maternal age > 35 years, and prenatal smoking

SCEHSC Pilot Project, PI: Volk

Volk, et al., 2011

Near Roadway Air Pollution (279 Cases and 245 Controls)





Probability of ASD Increases as NRAP Increases

Volk, et al., 2013

Regional Pollutant Exposure
(279 Cases and 245 Controls)

	First Year of Life OR*	All Pregnancy OR*
$PM_{2.5}$	2.12 (1.45-3.10)	2.08 (1.93-2.25)
PM_{10}	2.14 (1.46-3.12)	2.17 (1.49-3.16)
Nitrogen Dioxide	2.06 (1.37-3.09)	1.81 (1.23-2.65)
Ozone	1.15 (0.72-1.86)	1.09 (0.76-1.55)

***Regional pollution effects reflect risk of autism based on 2 standard deviations from the mean value, specifically per increase of 8.8 mg/m3 $PM_{2.5}$, 12.4 mg/m3 PM_{10}, 9.0 ppb NO_2, and 13.6 ppb ozone. The top TRP quartile refers to estimated exposure levels of 30.4ppb or greater.**

Models adjusted for child male gender, child ethnicity (Hispanic vs. White, Black/Asian/Other vs. White), maximum education of parents (parent with highest of four levels: college degree or higher vs. some high school, high school degree, or some college education), maternal age (>35 years vs. 35years), prenatal smoking, population density.

NIEHS R21 ES19002, PI: McConnell, Co-I: Volk

Volk, et al., 2013

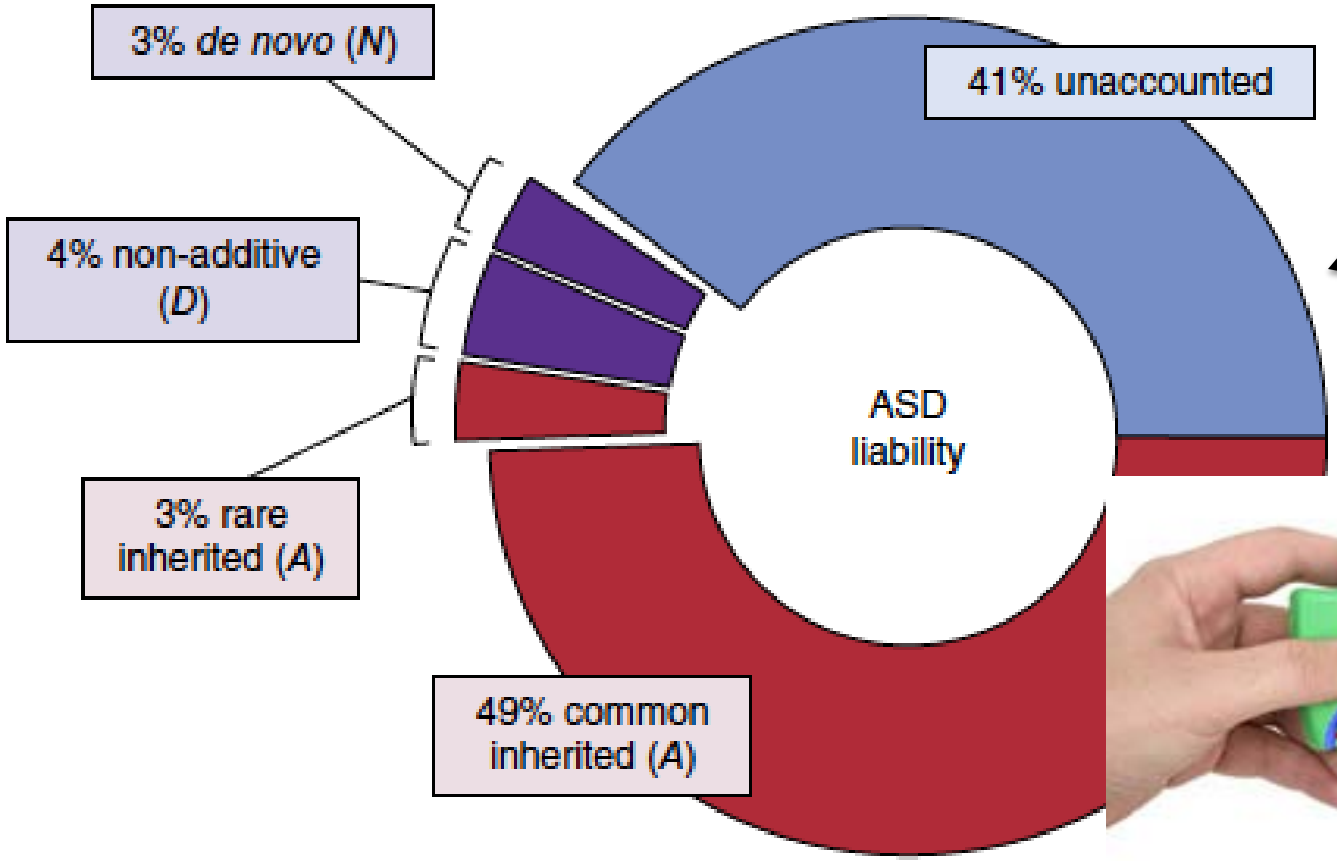
What Do We Know So Far?

- Air Pollution Associated with ASD
- Air Pollution Associated with Worse Functioning in ASD

What Do We Want To Know Next?

- So How Might This Work?
- What's the Responsible Ingredient?

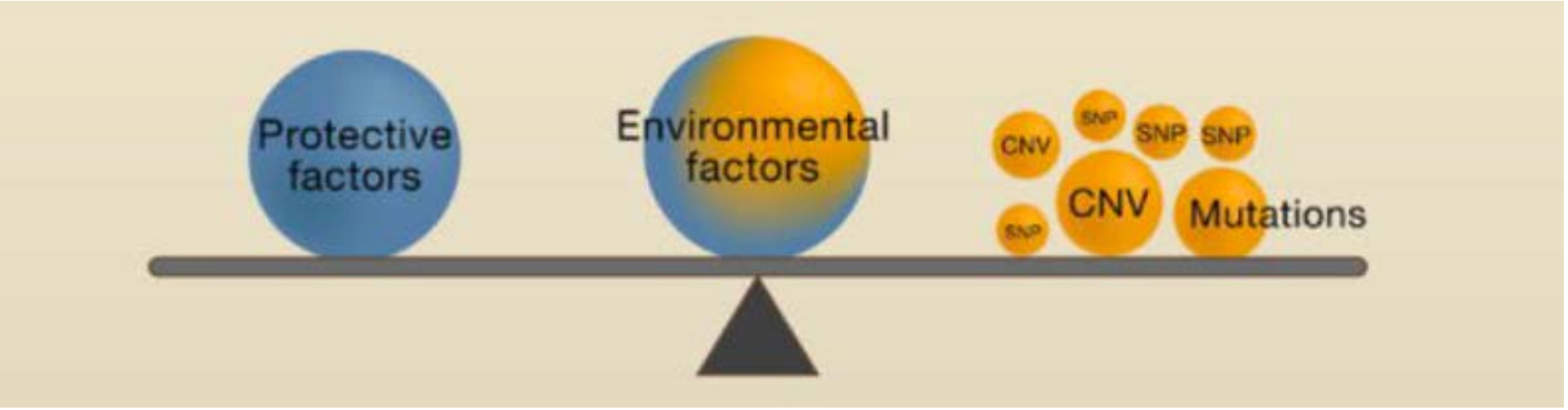
Where Do Genes Fit In?



Environmental Effects and GxE Interactions

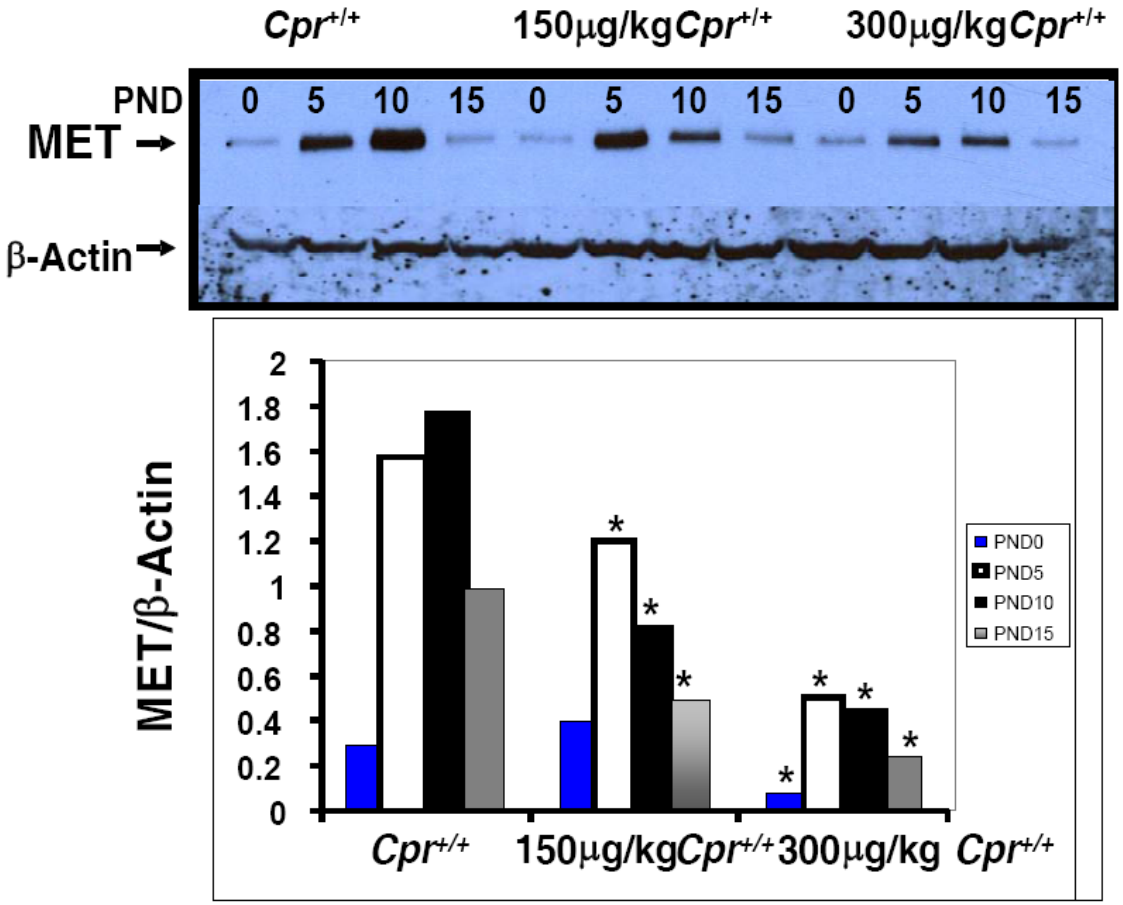
Adapted from Gaugler et al., 2014

Simple Model for Gene and Environment Effects



Adapted from Geschwind et al., 2008

Prenatal PAH Exposure Reduces MET Protein Expression in Mouse Cortex (Benzo(a)Pyrene)



Sheng et al., 2010

Replications of *MET* rs1858830 Variant Association with Autism

	Reference	Study Design	Sample Source
1	Campbell et al. 2006. *PNAS*.	Family-based and case-control	Italy (Rome 1)
2	Campbell et al. 2006. *PNAS*.	Family-based and case-control	NIMH
3	Campbell et al. 2008. *Autism Res*.	Family-based	Vanderbilt
4	Sousa et al. 2009. *Eur J Hum Genet.*	Family-based and case-control	Europe (UK)
5	Sousa et al. 2009. *Eur J Hum Genet*.	Case-control	Italy (Bologna)
6	Jackson et al. 2009. *Autism Res*.	Case-control	South Carolina
7	Jackson et al. 2009. *Autism Res*.	Case-control	Italy (Rome 2)
8	Thanseem et al. 2010. *Neurosci Res*.	Family-based	Japan
9	Thanseem et al. 2010. *Neurosci Res.*	Family-based	AGRE
10	Rajamma et al. 2011. SfN poster	Family-based	India

Joint Effect of *MET* rs1858830 and Air Pollution

Near Roadway Air Pollution		
	MET rs1858830 Genotype	
	C/C	C/G or G/G
Exposed	2.9 (1.0-10.4)	1.3 (0.73-2.2)
Unexposed	0.80 (0.47-1.4)	reference
Regional Nitrogen Dioxide		
	MET rs1858830 Genotype	
	C/C	C/G or G/G
Exposed	3.6 (1.3-12.7)	1.2 (0.71-2.1)
Unexposed	0.72 (0.41-1.3)	reference

Volk et al, 2014

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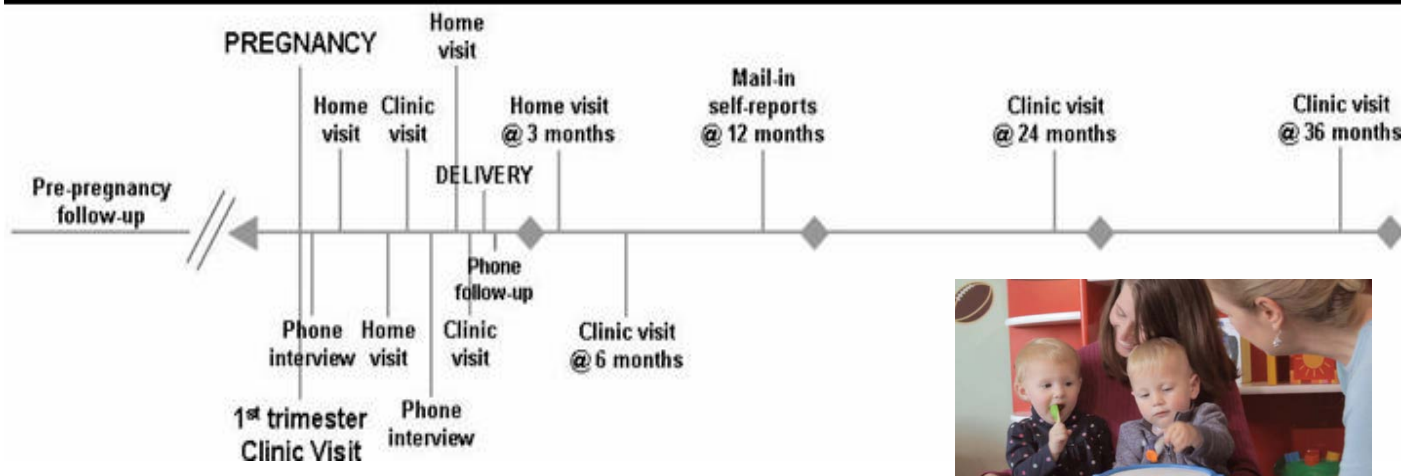
What Are The Next Steps?

MARBLES
Markers of Autism Risk in Babies - Learning Early Signs

IBIS Network
Infant Brain Imaging Study
Study Sites

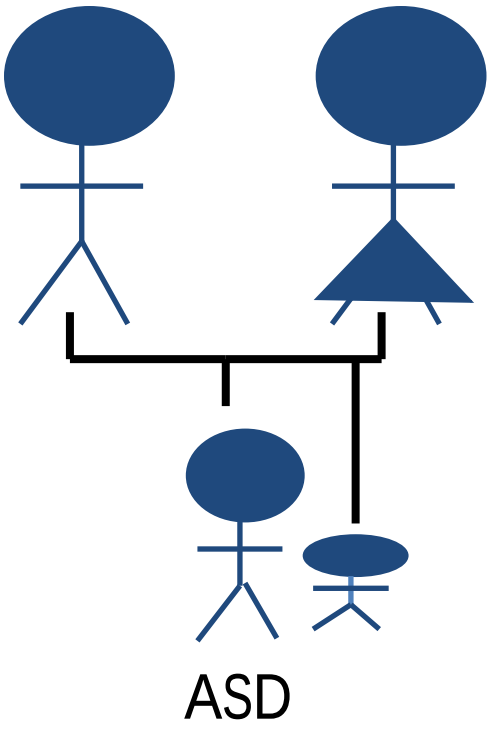
EARLI
Early Autism Risk Longitudinal Investigation

Figure 6. EARLI Network core protocol data collection schedule



Prospective Evaluation of Air Pollution, Cognition, and Autistic Traits from Birth Onward (PEACABO)

- Collaboration with At-risk Infant Sibling Study Designs
  - Early Autism Risk Longitudinal Investigation (EARLI)
    - R01 ES016443 (Newschaffer)
  - Markers of Autism Risk in Babies-Learning Early Signs (MARBLES)
    - R01 ES020392 (Hertz-Picciotto)



NIEHS R01 ES023780, PI: Volk

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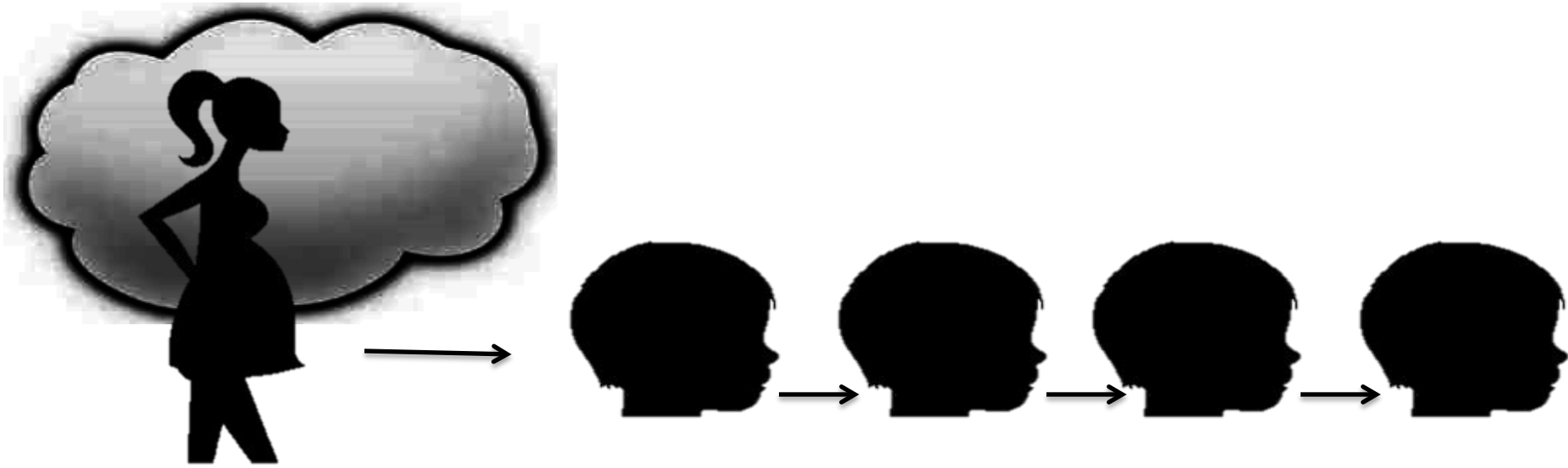
Air Pollution on Development and ASD Over Time



Prenatal Exposure

Modeled Residential Exposure

Polycyclic Aromatic Hydrocarbon (PAH) Biomarkers

6, 12, 24, and 36 months

Cognitive Development
Adaptive Function

ASD and Related Traits

NIEHS R01 ES023780, PI: Volk

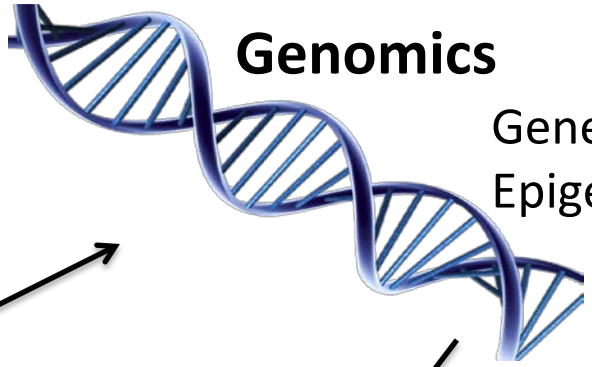
Virtual Consortium for Transdisciplinary Environmental Research (ViCTER)

- Understand the MECHANISM of the effect of air pollution on ASD
- Combine Both Human and Animal Models

RNA seq

Behavior

Step 1



Genomics

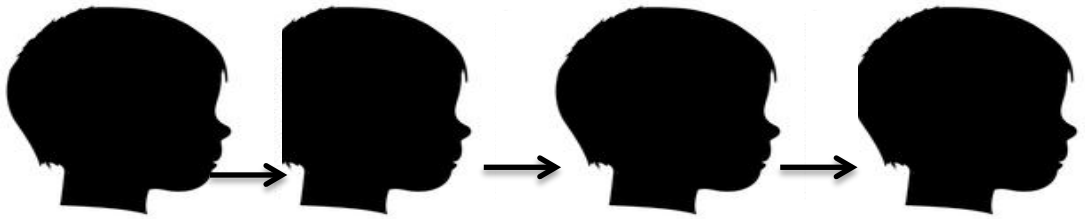
Gene Expression

Epigenetic Effects

Step 2

Step 3

Genetic Associations



Prenatal Exposure

Traffic and $PM_{2.5}$

12, 24, and 36 months

Child Cognitive Development

ASD and Related Traits

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Air Pollution Exposure Effects on Brain Structure

Effects of Prenatal Exposure to Air Pollutants (Polycyclic Aromatic Hydrocarbons) on the Development of Brain White Matter, Cognition, and Behavior in Later Childhood

Bradley S. Peterson, MD; Virginia A. Rauh, ScD; Ravi Bansal, PhD; Xuejun Hao, PhD; Zachary Toth, BA; Giancarlo Nati, BA; Kirwan Walsh, BA; Rachel L. Miller, MD; Franchesca Arias, MS; David Semanek, BA; Frederica Perera, DrPH, PhD

CONCLUSIONS AND RELEVANCE Our findings suggest that prenatal exposure to PAH air pollutants contributes to slower processing speed, attention-deficit/hyperactivity disorder symptoms, and externalizing problems in urban youth by disrupting the development of left hemisphere white matter, whereas postnatal PAH exposure contributes to additional disturbances in the development of white matter in dorsal prefrontal regions.

Peterson et al., 2015

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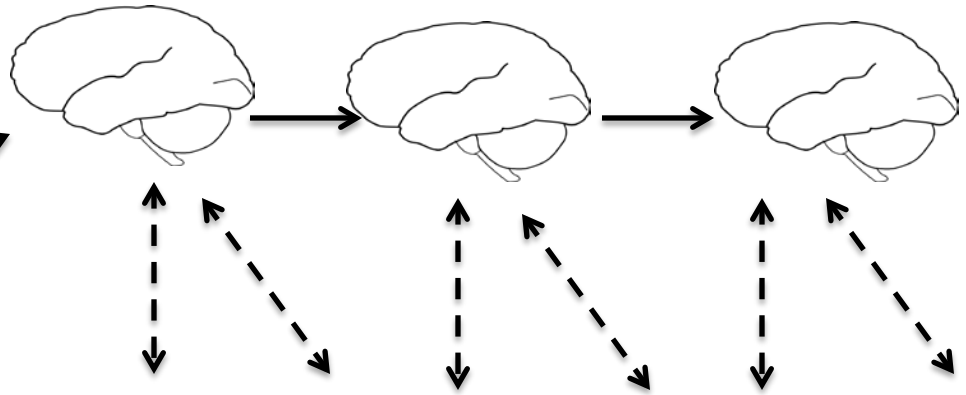
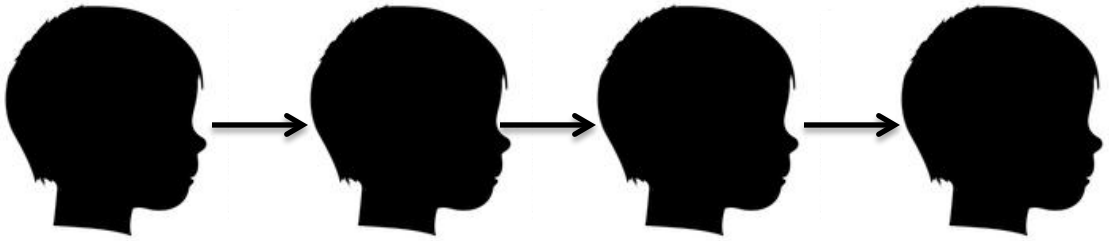
IBIS Collaboration

Prenatal Exposure
Modeled Residential Exposure

Enrollment, 12, and 24 months
Brain Volume, Surface Area, EAF Volume,
CC Morphology, WM Fiber Tract Structure





6, 12, 24 and 36 months
MSEL and VABS
ASD and Related Traits

So Is It All True?

Curr Envir Health Rpt (2015) 2:430–439
DOI 10.1007/s40572-015-0073-9

AIR POLLUTION AND HEALTH (JD KAUFMAN AND SD ADAR, SECTION EDITORS)

Air Pollution and Autism Spectrum Disorders: Causal or Confounded?

Marc G. Weisskopf[1,2] · Marianthi-Anna Kioumourtzoglou[1] · Andrea L. Roberts[3]

Given the general consistency of findings across studies and the exposure-window-specific associations recently reported, the overall evidence for a causal association between air pollution and ASD is increasingly compelling.

What Might Be the Impact?

Population Attributable Risk Percent

=

The percent of cases that would NOT occur if that risk factor were eliminated

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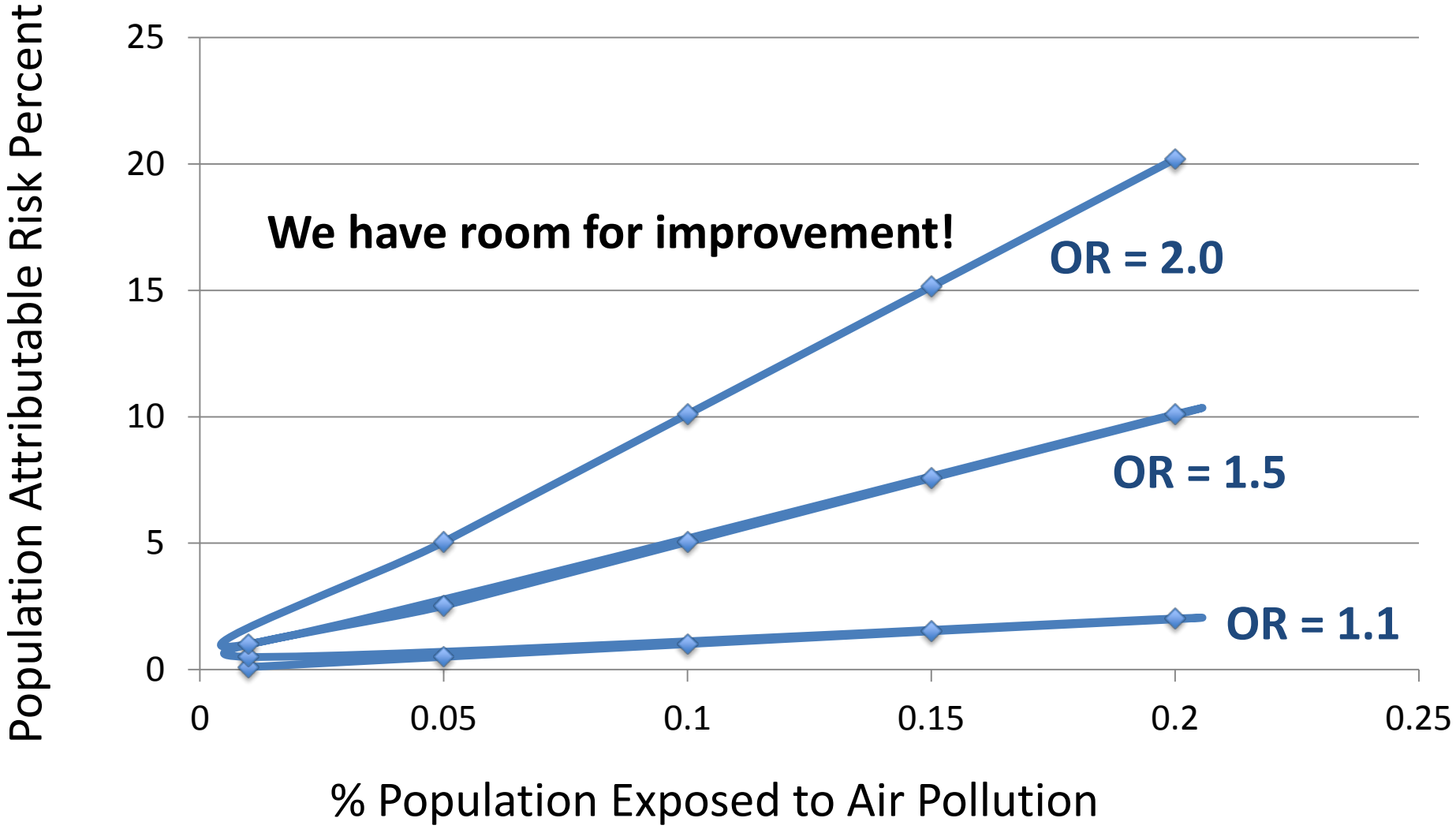
What Might Be The Impact?



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