The Epidemiology of ADHD:

Prevalence, Natural History & Clues to Etiology

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Outline

- Prevalence of ADHD and how it varies
- Natural History of ADHD and its impact
- Etiology of ADHD
  - Can exposures to environmental toxicants cause ADHD?
  - How likely is a gene-environment interaction as an explanation?

Estimates of Prevalence of ADHD

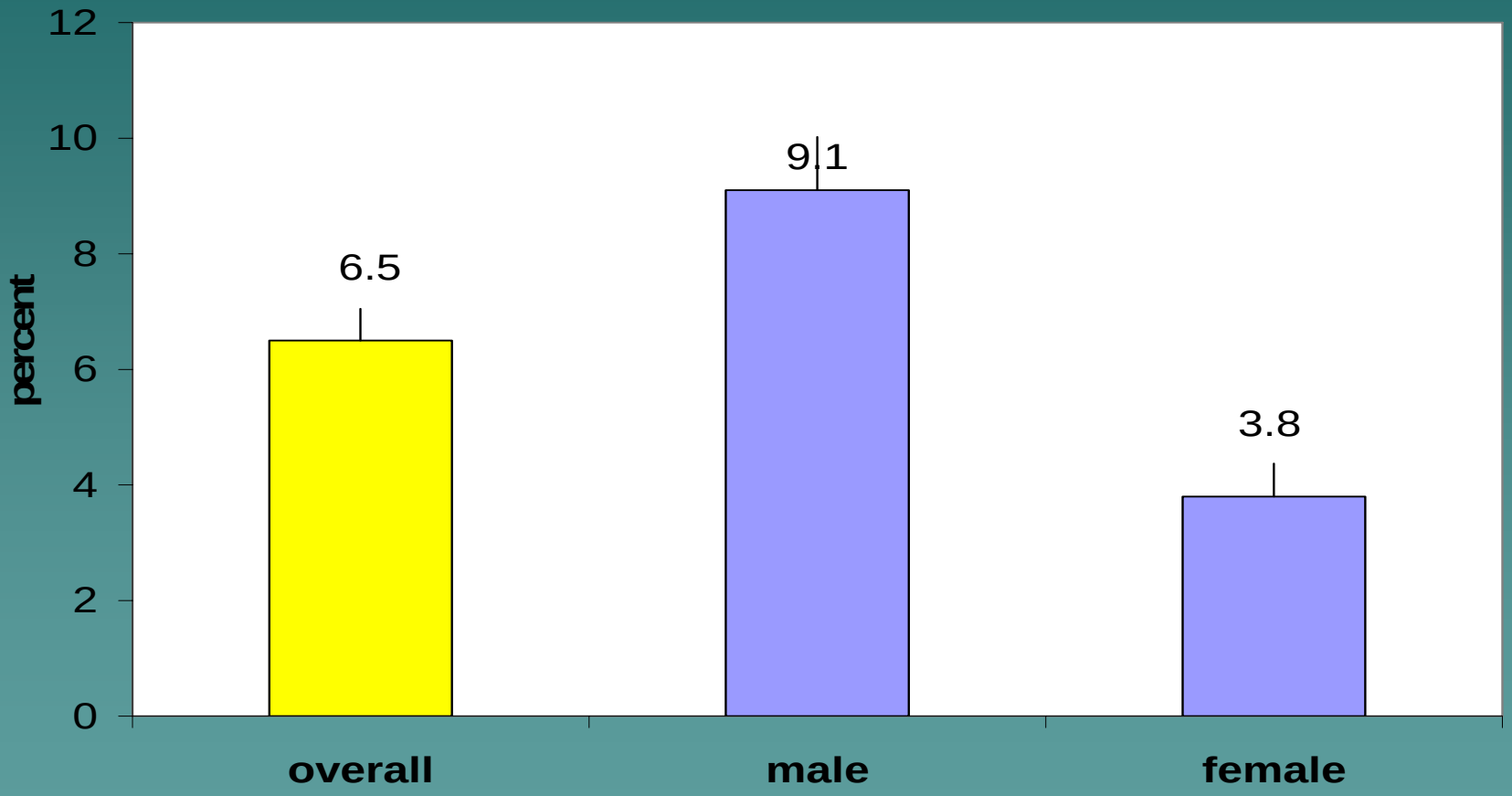
- Prevalence stated in DSM-IV, TR 3-7 %
- Many problems with many current estimates: (Skounti et al 2007)
  - Different definitions of ADHD
  - Clinic samples
  - Use of only 1 informant
  - Children taking medication
  - Symptoms caused by other disorders

Epidemiologic Compass

Prevalence varies by :

- Gender
- Age
- Race/Ethnicity
- SES
- Over time
- Geographically

Age-adjusted Estimates of Parent-reported ADHD National Health Interview Survey, 2005

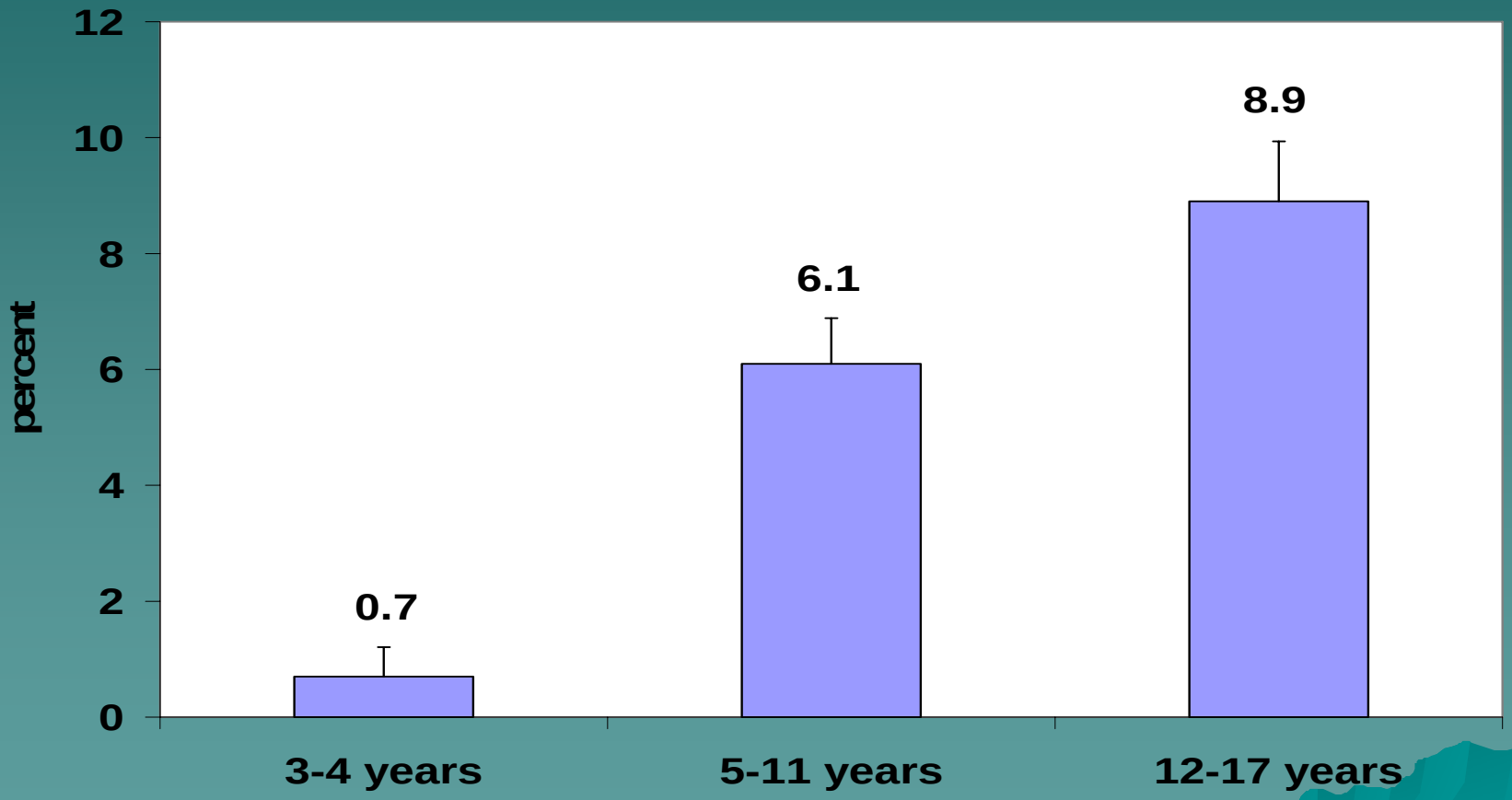


Source: NHIS, Series 10, 231, 2005

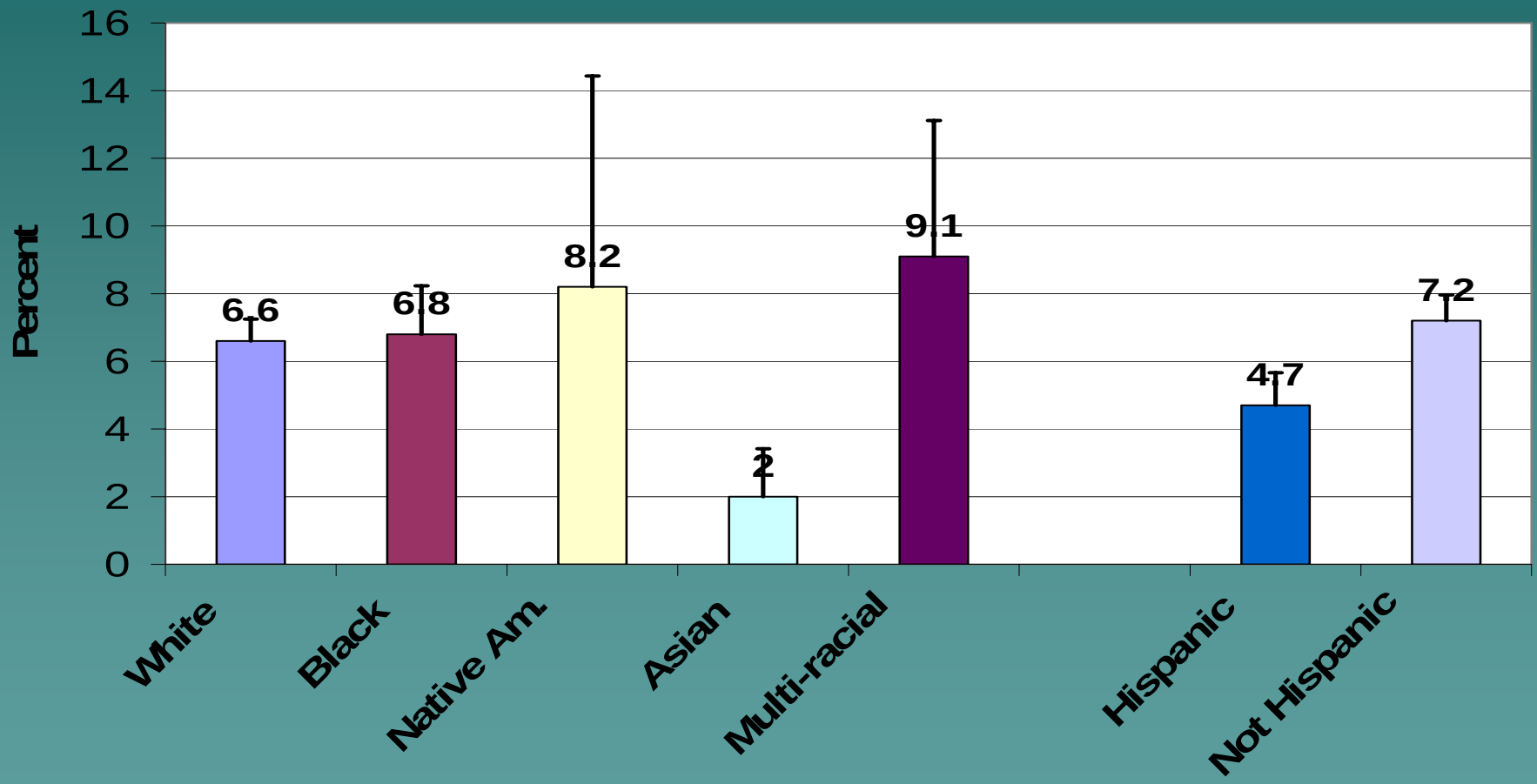
ADHD as a Developmental Disorder

- Sex ratio for ADHD is about 3:1
- Male predominance true for many developmental disabilities
- Male vulnerability through birth, infancy, childhood

Relation between Age and Parent-reported ADHD, NHIS, 2005

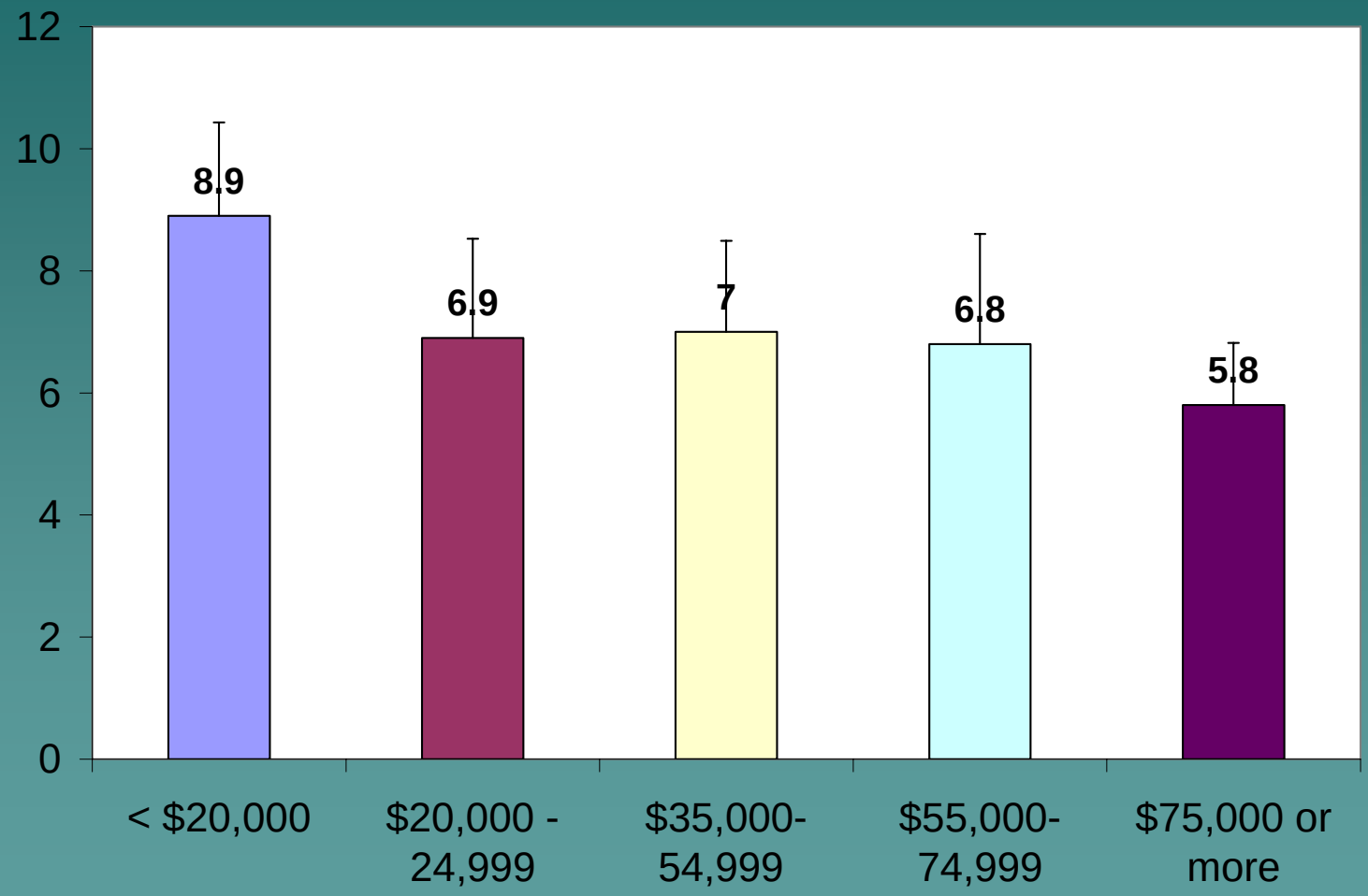


Age-adjusted Prevalence of Parent-Reported ADHD By Race/Ethnicity



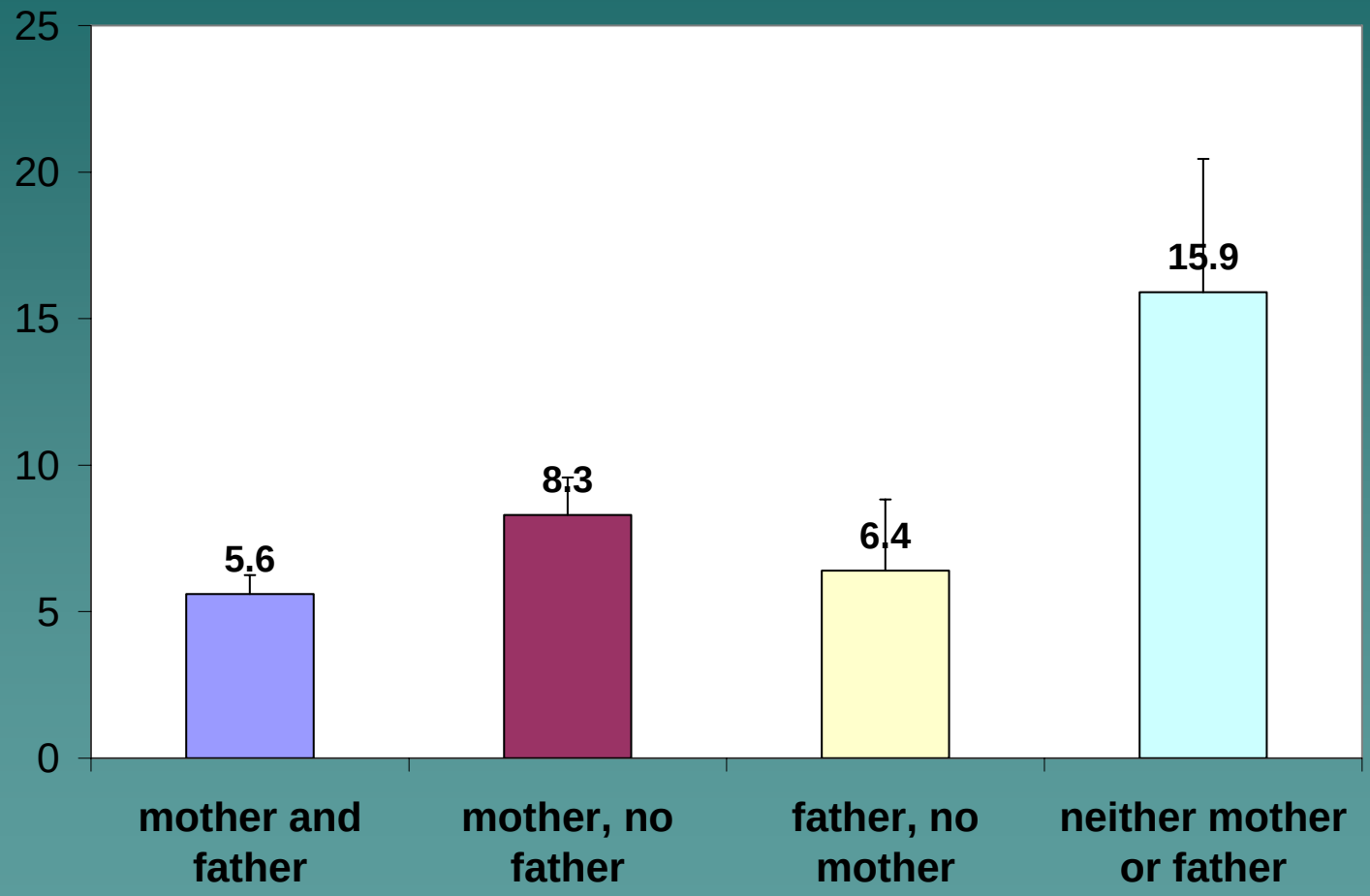
Source: NHIS, 2005

Age-adjusted Prevalence of Parent-reported ADHD by Annual Family Income



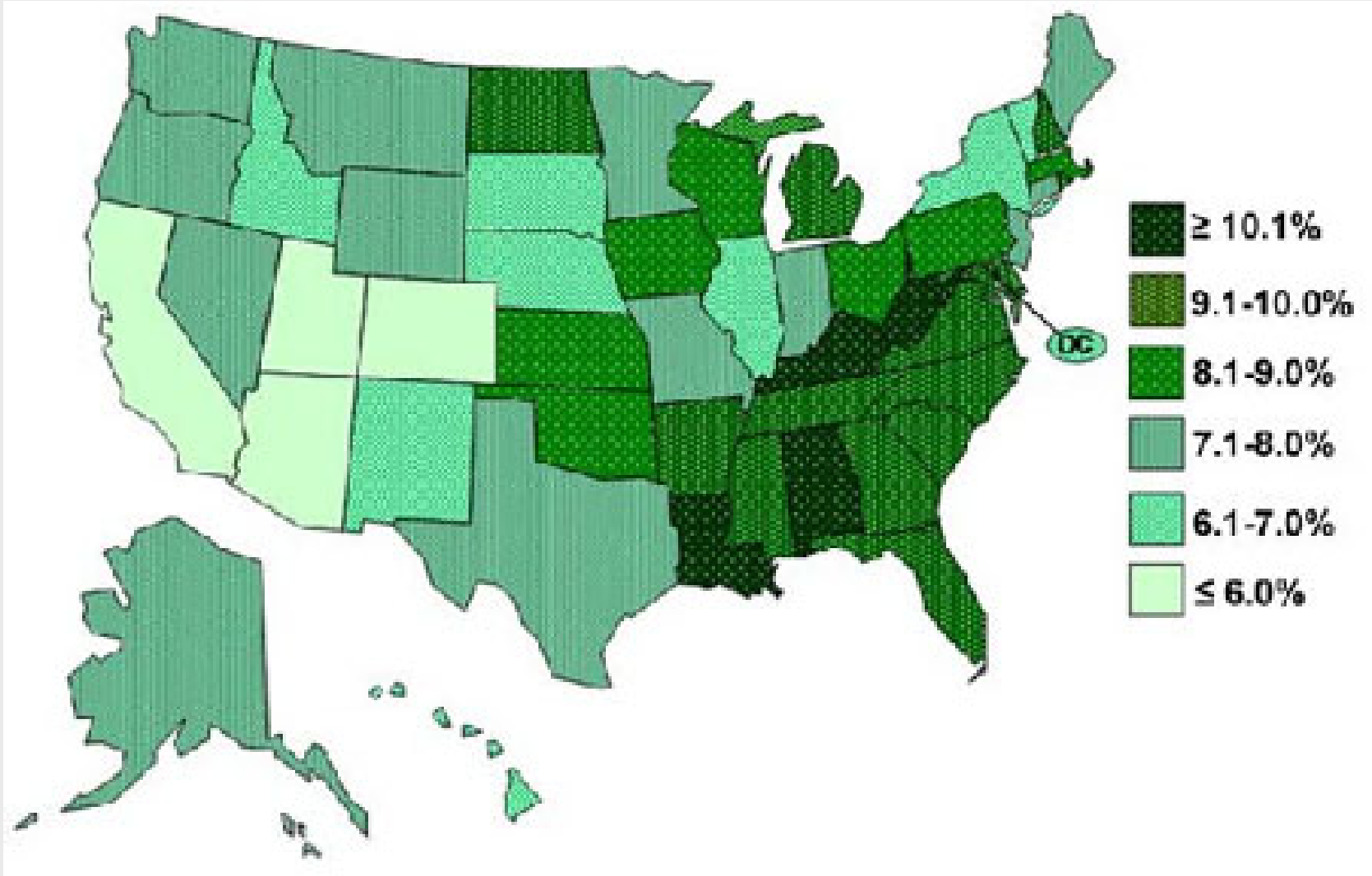
Source: NHIS, 2005

Age-adjusted Prevalence of Parent-reported ADHD By Family Structure

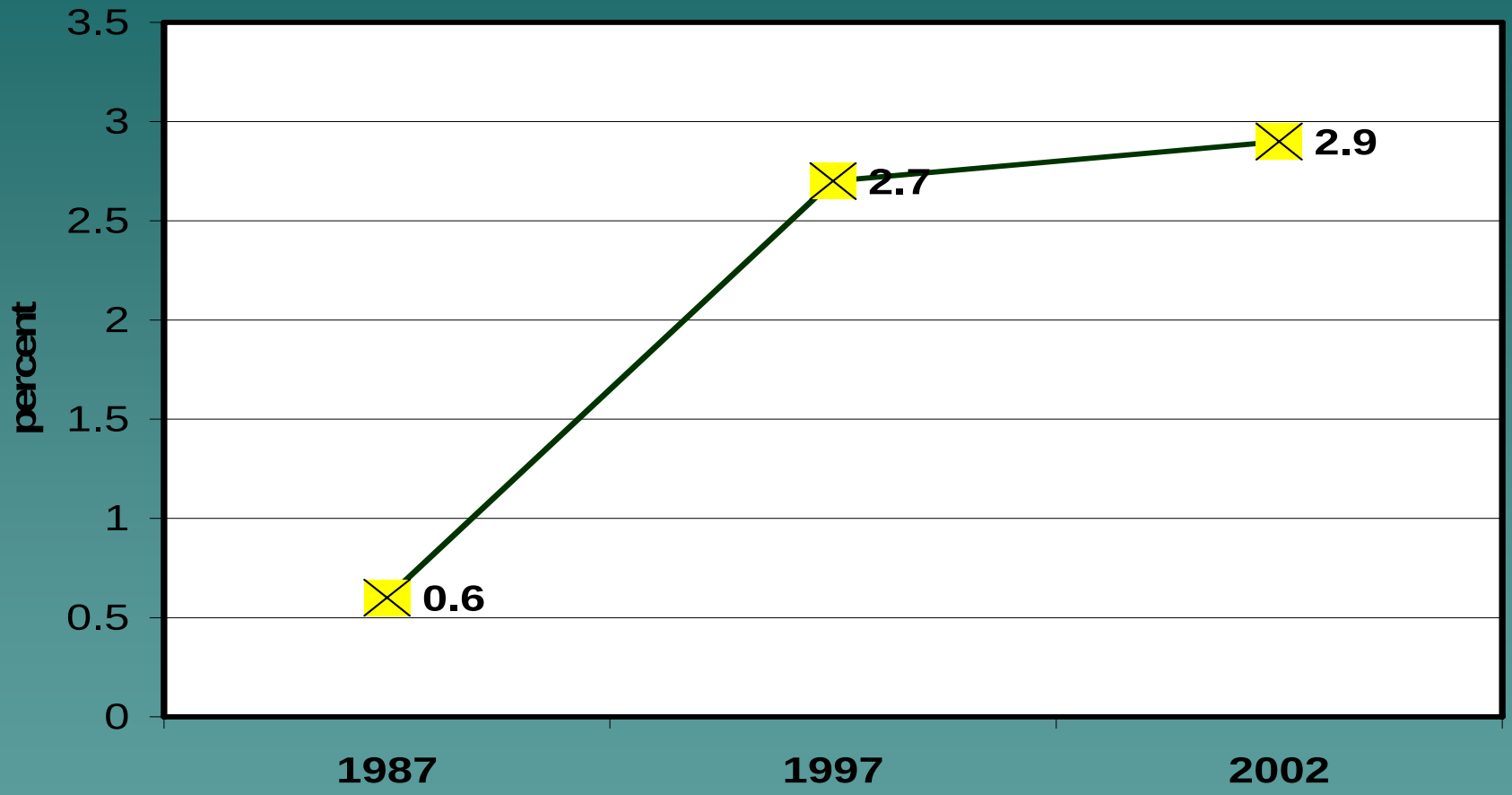


Source: NHIS, 2005

Prevalence of Parent-Reported ADHD by State, National Survey of Children's Health, 2003



Prevalence of Stimulant Use
U.S. Population - Age 18 and Under



Source: Medical Expenditure Database, AHRQ

Impact of Natural History

Percent of ADHD Children with Comordid Conditions Ontario Child Health Study

	% ADHD + Comorbid Conditions
Males 4-11	53.0
Females 4-11	42.1
Males 12-16	36.9
Females 12-16	67.0

Source: Szatmari et al. 1989

Natural History of ADHD

Follow-up studies suggest:

- **30-45% will meet criteria for ADHD at age 20**
- **Risks persist**
- **Risk of substance abuse and conduct disorder**

Accidents and Health Risk behaviors

- **Long term costs** (Discala et al. 1998)
  - **Accidents**
  - **Health risk behaviors**
  - **Arrests**
- **Youth with ADHD + Conduct Disorder at particularly high risk**

Etiology

Genetic Risk of ADHD
Farone et al. 2005

- Familial risk
- Heritability estimates
- Many polymorphisms, weak relationships
- May suggest gene-environment interaction

Pregnancy Complications: Collaborative Perinatal Project 1959-1965

Risk of hyperkinetic-impulsive Behavior

- prenatal smoking
- hospitalized during pregnancy
- convulsions during pregnancy
- breech delivery

Toxicant Exposure and ADHD

- Prenatal exposure to smoking
- Environmental tobacco smoke
- Prenatal exposure to alcohol
- Prenatal stress
- Lead
- Pesticides

Prenatal Smoking and ETS

- Prenatal smoking
  - 2003 review (Linnet et al 2003)
  - evidence mixed, but overall positive
- Environmental tobacco smoke
  - evidence mixed

Prenatal Alcohol / Prenatal Stress

- Nine studies reviewed (Linnet et al, 2003)

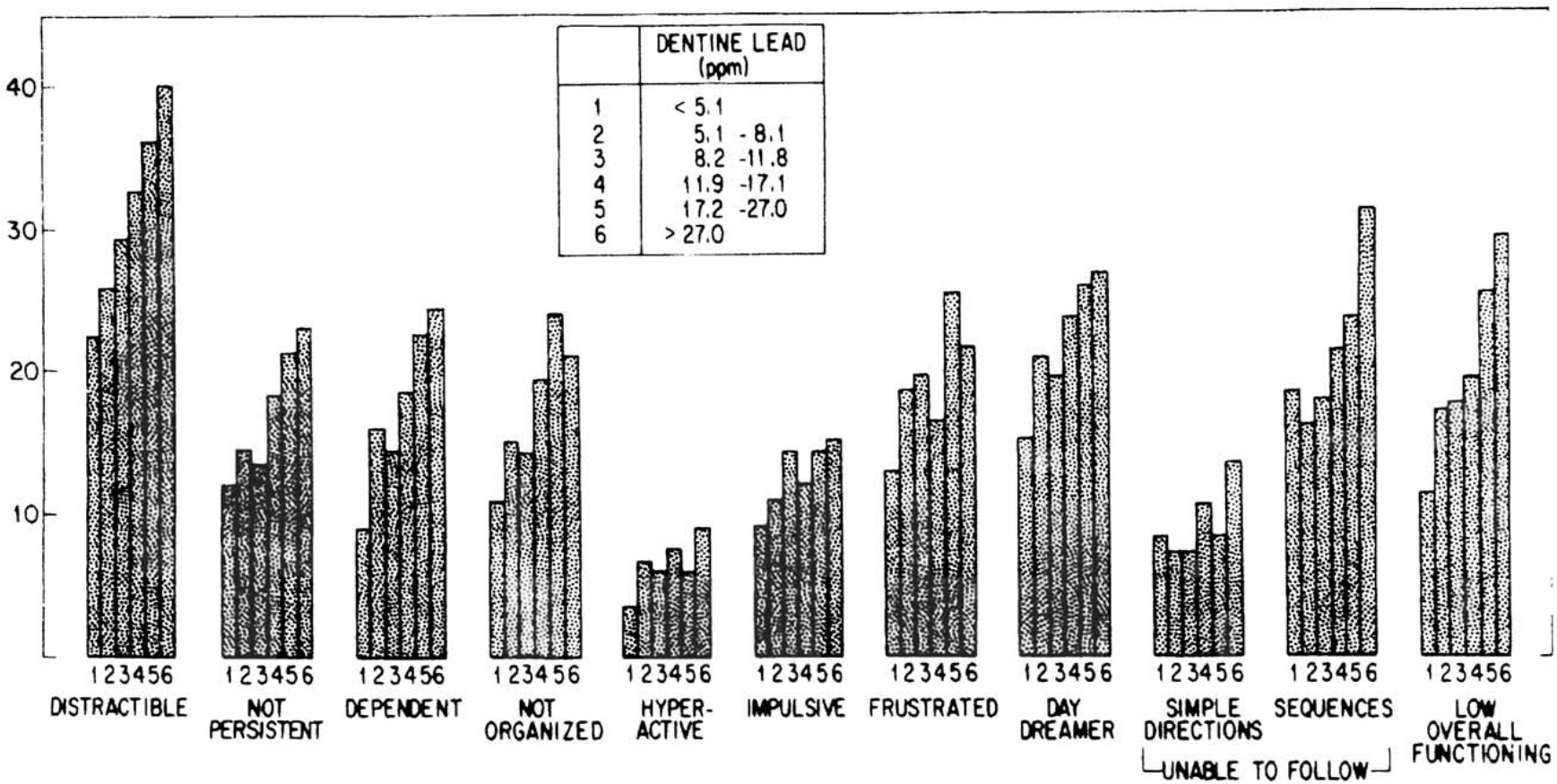
  - Evidence mixed

  - Critique comparing ADHD and FAS/FAE (Coles, 2001)

- Implications for environmental studies

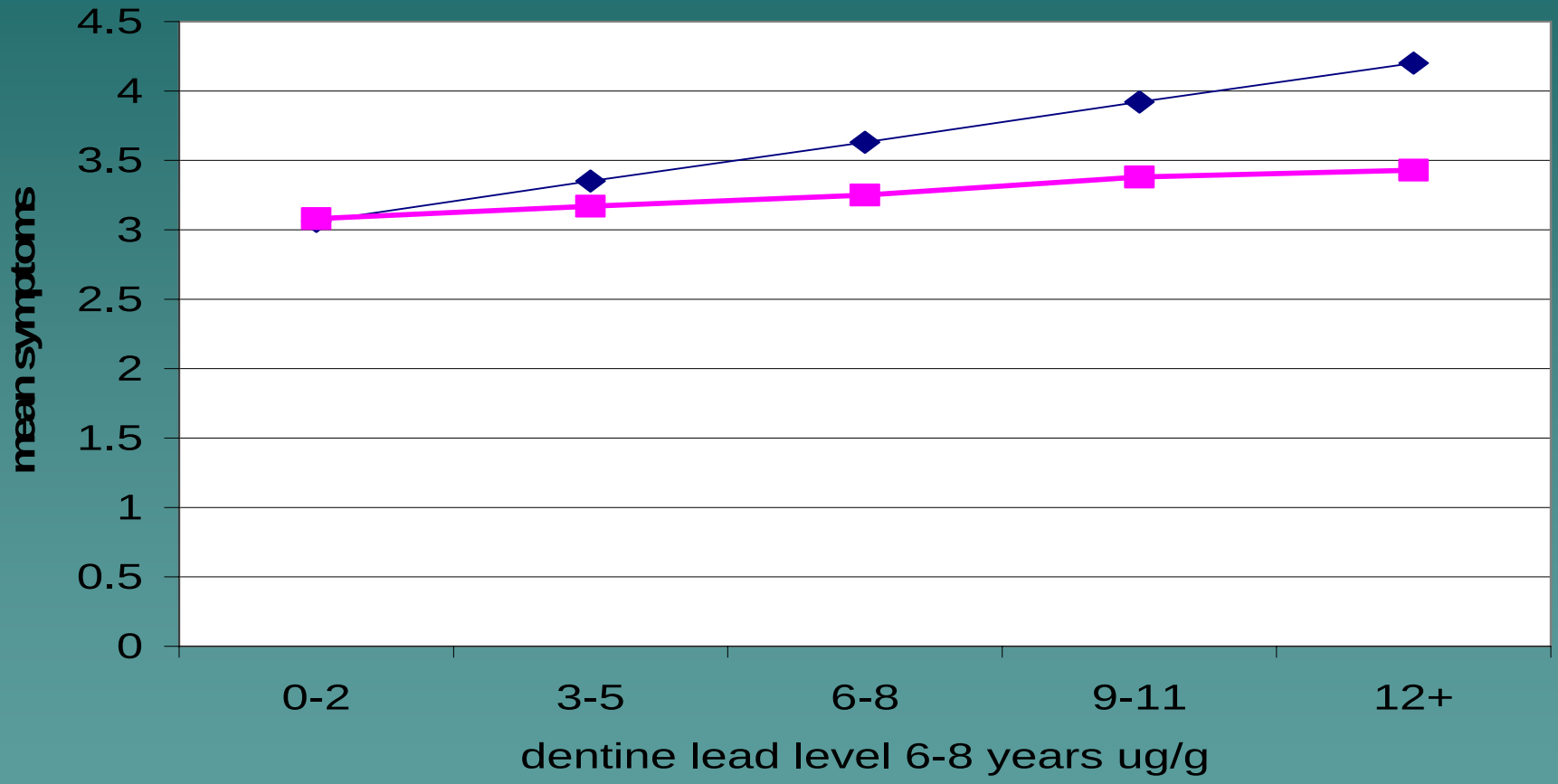
- Prenatal stress and ADHD (O'Connor et al 2002, Rodriguez 2005)

Dentine Lead and Teacher-Reported School Problems



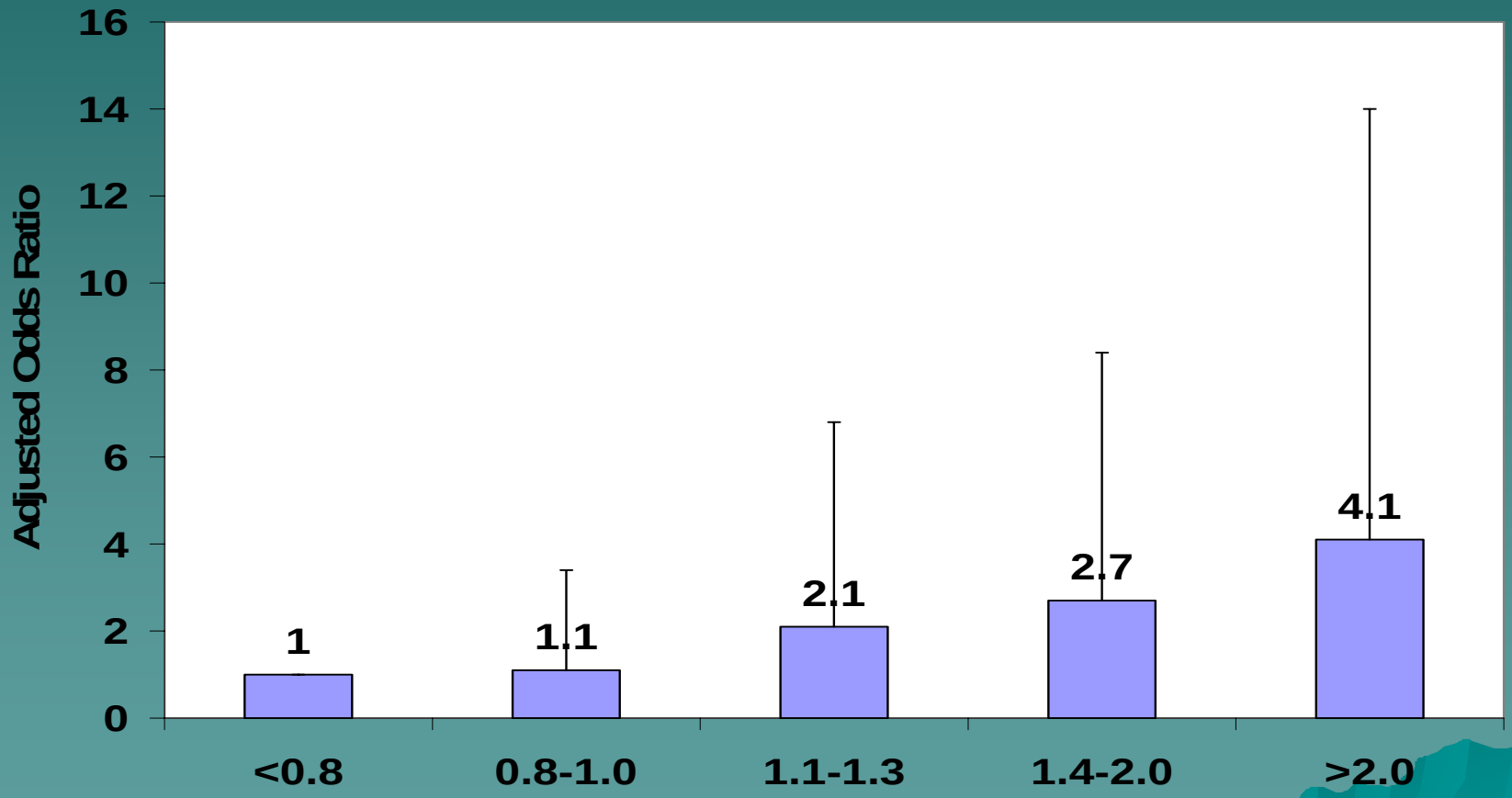
Source: Needleman et al. 1979. NEJM

Relation between Dentine Lead Levels at Age 6-8 and Adjusted Symptoms of Inattention/restlessness at Age 12-13



Source: Fergusson et al. 1993

Relation between Blood Lead and Odds Ratio for ADHD, NHANES 1999-2002



Source: Braun et al. 2006

Adjusted Odds Ratios of Attention Problems at 36 months

Prenatal Exposure	Attention Problems	ADHD Problems
Environmental Tobacco Smoke	2.8 (0.4-17.8)	8.1(1.2-54.7)
Chlorpyrifos	11.3 (1.8-71.0)	6.5 (1.1-38.7)

Source: Rauh et al. 2006

Points to Consider

- Does the endpoint matter?
  - Tests of attention, ADHD symptoms, or ADHD
  - Multi-method approaches
  - Standardization of the case definition

- How can we incorporate social factors into our studies of environmental and genetic risk factors?

- We need to develop more effective ways to control for SES and poverty in our models.

Points to Consider Continued

- Need for more complex models that account for adverse life events and timing of exposures during different stages of child development.
- Epidemiologic tools don’t work very well at low exposures. We need to make good use of the tools we do have.

Collaborators

- **North Carolina**

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