Infant Sleeping Position and Atopic Sensitisation: Evidence from a Birth Cohort Study Commenced before the Back To Sleep Campaign in the UK

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Rationale: Infants who habitually sleep on their front may be exposed to greater concentrations of inhalant allergens than back or side sleepers, thus increasing their risk of of allergic sensitisation to house dust mite allergen *(Der p1)*.

Methods: Data was obtained from the Avon Longitudinal Study of Parents and Children (ALSPAC), a birth cohort study with recruitment during pregnancy (1.4.91 - 31.12.92) and with 13,971 surviving infants at one year after birth. Information about sleeping position was obtained from self -report questionnaires at 6 months after delivery. 7175 children attended a clinic at 7.5 years for physical examination, including skin prick testing to common allergens (*Der p1*, cat and mixed grasses). Atopy was defined as weal diameter >=2mm to any allergen, and house dust mite sensitisation as >=2mm weal diameter to *Der p1*.

Results: Proportions of subjects with atopy and house dust mite sensitisation for each sleeping position are shown in the table.

Sleeping position	Atopy		*Der p1*	
	% (N)*	OR [95% CI]	% (N) **	OR [95% CI]
Back	23.4 (278)	1.0	13.3 (157)	1.0
Side	19.2 (854)	0.8 [0.7,0.9]	11.7 (520)	0.9 [0.7,1.0]
Front	22.9 (57)	1.0 [0.7,1.3]	14.5 (36)	1.1 [0.7,1.6]
Varies	22.0 (124)	0.9 [0.7,1.2]	13.7 (76)	1.0 [0.8,1.4]

*χ^2,p=0.005;**χ^2,p=0.2

Conclusion: We did not find evidence to support the hypothesis that prone sleeping position in infancy is associated with an increased risk of house dust mite sensitisation at age 7.5 years.

This Abstract is Funded by: None

Relationship of Cytokine Production in Infancy and Wheeze in the First Year of Life

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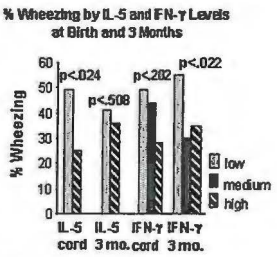
Rationale: There has been considerable recent interest in the relation between cytokine production in infancy and subsequent development of asthma and wheeze. In the ongoing Tucson Infant Immune Study, we looked at Th1 and Th2-like cytokines produced by peripheral blood mononuclear cells at birth and 3 months (mo.) of age and wheeze in the first year of life. **Methods:** Data on wheeze in the first year were determined for 244 children from parental questionnaires. Blood was obtained from children at birth (n=127) and 3 mo. of age (n=190). Cytokine production was assessed by ELISA in supernatants following PBMC stimulation with ConA/PMA. Depending on their distribution, cytokine values were log-transformed, dichotomized or divided into tertiles. **Results:** 40% of children were reported to have wheezed in the first year. Wheezing was associated with low IL-5 production at birth ([Chi]^2, p<.024) and low IFN-γ ([Chi]^2, p<.022) at 3 mo. There was no association between wheezing and either IL-4 or IL-13 production at birth or 3 mo. **Conclusions:** There was no evidence that wheeze in the first year is associated with a Th2- skewed cytokine profile in infancy. Decreased production of IL-5 (a Th2-like cytokine) at birth and IFN-γ (a Th1-like cytokine) at 3 mo. were found to be predictive of wheeze in the first year.



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Sleeping in Lower Bunk Beds Does Not Cause an Increase in Sensitization to House Dust Mite

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Rationale: Children who sleep in the bottom bunk of a pair of bunk beds may have greater overnight exposure to house dust mite, particularly where the upper bunk bed is occupied by another child. This increased exposure could raise the risk of subsequent asthma.

Methods: A parent-completed questionnaire was administered at the age of 8 years to families under follow-up within the Avon Longitudinal Study of Parents and Children (ALSPAC) cohort. Data were collected that detailed children's sleeping habits, in particular whether they routinely slept in bunk beds and whether the other of the pair was simulataneously occupied. The association of bunk bed occupation (in particular lower bunk with occupation of upper bunk) with sensitivity to dust mite allergens (Der p1 and Der f1) was analysed.

Results: 4221 families returned questionnaires. 851 children slept in lower bunk beds with the top bunk usually occupied. 1544 children had never slept in bunk beds. There was no increase in HDM sensitivity in children sleeping in lower bunk beds.

	Case (n=851)	Control n=(1544)
Reaction to Der p1		
Yes (n=299)	11.4% (97)	13.1% (202)
No n=(2096)	88.6% (754)	86.9% (1342)
p=0.233		

Conclusion: Sleeping in bunk beds does not appear to be associated with increased sensitization to house dust mite.

This Abstract is Funded by: Medical Research Council, UK

Parental Asthma as an Independent Risk Factor for the Development of Skin Test Sensitization in Children

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[1]RATIONALE: Little is known about the role of a familial predisposition for asthma in the development of atopy in children. METHODS: Subjects in this analysis were participants in the Tucson Children Respiratory Study. Skin tests to common allergens were performed in parents and in children at age 6, 11 and 16. Skin test positivity was defined as a wheal at least 3 mm greater than control. Parents were considered asthmatic if they reported MD confirmed asthma at enrollment. Parents were divided into 4 mutually exclusive phenotypes based on skin sensitization and asthma status: Skt-/As-, Skt-/As+, Skt+/As-, and Skt+/As+. Proportions of atopic children were compared among phenotypes. RESULTS: There was a differential distribution of skin positive children among parental phenotypes at all ages (p<.0001) (table). Children in the Skt+/As- and Skt+/As+ groups were significantly more likely to be atopic than children in the Skt-/As- group at all ages. Among children with atopic parents, the proportion of positive skin tests was higher in the asthma positive (Skt+/As+) than in the asthma negative phenotype (Skt+/As-) at both yr 6 and yr 11 (p< 0.005), but not at yr 16. These findings held true after stratification and multivariate analyses were performed in order to adjust for potential confounders. CONCLUSION: Our findings suggest that a familial predisposition for asthma may independently contribute to atopic sensitization in childhood.

Atopy in children by parental phenotype

Parental phenotype	% Skin test + yr 6 (n)	% Skin test + yr 11 (n)	% Skin test + yr 16 (n)
Skt-/As-	24.4 (131)	38.2 (123)	48.8 (82)
Skt-/As+	33.3 (21)	42.9 (14)	75.0 (4)
Skt+/As-	37.4 (446)	55.6 (417)	75.1 (307)

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The Influence of Early Respiratory Infections on the Development of Atopy

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RATIONALE: The escalating prevalence of atopic disease has been shown to be predominantly in 'westernised' countries, where major environmental changes have occurred, resulting in reduced exposure to microbial stimulation. Advances in immunology have highlighted the environmental factors implicated in the polarisation of an individual's immune profile towards an atopic state. OBJECTIVE: To investigate the role of acute respiratory illnesses (ARI) on the development of atopy in a 'high risk' cohort of children. METHODS: 250 'high risk' children were recruited prenatally in 1996 and followed up for 5 years. Information on all ARI was collected. The children were checked regularly for atopic dermatitis (AD).Skin prick tests (SPT) were performed at 6 months, 2 and 5 years of age. RESULTS: Children with AD in infancy, and those with persistent disease were more likely to be atopic. In non-atopic children, ARI increased the risk of AD. Upper respiratory illnesses (URI) in infancy was associated with a four-fold increase in persistent AD, whilst infants who had non-wheezy LRI had a two-fold increased risk for AD at 1 year. Children who had URI in their second year of life had a lower risk of positive SPT to ingested allergens at 2 years of age. Conversely, children who experienced non wheezy LRI in the second year were more than twice as likely to have a positive SPT to ingested allergens at 2 years. CONCLUSION: Respiratory illnesses in early childhood alter the susceptibility of 'high risk' children to atopy and atopic dermatitis.

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Parental Atopy and Asthma in Rural Children: Is There a Link?

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Recent literature has reported low asthma and/or atopy prevalence in rural/farm populations. We found relatively high rates of childhood asthma (16%) and atopy (36%) in a completely rural cohort. The highest prevalence of asthma was in farm children, atopy in rural town children. We tested the hypothesis that parental farm allergies were associated with these outcomes in a cross-sectional study of data from Round I of the Keokuk County Rural Health Study. Parental interview, spirometry, methylcholine inhalation challenge, skin-prick testing, and serum specific IgE (sub-sample only) data were included. This study was limited to 487 children ages 3-17 years. Several specific parental allergy and sensitization variables were associated with asthma or atopy, including farm allergies to hogs (31.0% prev., asthma OR=1.98, 95%CI 1.21-3.24), soybeans (16.6% prev., asthma OR= 2.48, 95%CI 1.42-4.34), and chickens (8.2% prev., atopy OR= 1.90, 95%CI 0.99-3.63). After adjustment for other covariates, multiple comparisons within homes, and other environmental exposures; parental allergy to soybeans was associated with increased asthma prevalence in children (OR= 1.12, 95%CI 1.02-1.23). Similarly, parental allergy to chicken was weakly associated with atopy (OR= 1.15, 95%CI 0.99-1.35). Our study suggests that atopy prevalence on farms was low relative to town and rural residents. Parental atopy to farm antigens was a weak contributor to a childs risk of developing atopic illnesses. This abstract does not necessarily represent EPA policy.

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GMISP125        THE CDC GRANTS MANAGEMENT INFORMATION SYSTEM          07/17/03
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AH07AH00                      ( SCREEN NO.1 )

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 CENTERS FOR AGRICULTURAL RESEARCH, EDUCATION, & DISEASE & INJURY PREVENTION
         GRANTEE NAME...: THE UNIVERSITY OF IOWA
         BUSINESS OFFICE: SPONSORED PROGRAMS
         STREET.........: 100 GILMORE HALL
         CITY...........: IOWA CITY
         STATE: IA   ZIP CODE: 52242-1320     PHONE:( 319 ) 335-4189-

         PROJ DIRECTOR..: STEPHEN REYNOLDS, PH.D.
         DEPARTMENT.....: THE UNIVERSITY OF IOWA
         STREET.........: 100 OAKDALE CAMPUS - 140 IREH
         CITY...........: IOWA CITY
         STATE: IA   ZIP CODE: 52242-5000     PHONE:( 319 ) 335-4212-
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          INDIRECT COSTS (FA)............:       178,624
          SBIR FEE.......................:

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