Initiative). This longitudinal prospective study assessed familial atopic predisposition and severity of viral respiratory infections in infants with SHS exposure. Term infants with a birth weight $\geq 2275$ g were enrolled from September to May of 2004 to 2008 when they presented with lower respiratory tract infection for an acute care visit to a single academic medical center.

METHODS. A questionnaire was used for assessment of patient demographic characteristics, familial atopy predisposition, and environmental exposures. In this case, familial atopy predisposition was determined on the basis of 2 factors: (1) a mother with self-reported atopy or allergen sensitization; or (2) atopy in a first-degree relative of the infant. Medical records were reviewed. Hospital length of stay (LOS) and bronchiolitis severity scores (BSS) were used to determine severity of lower respiratory tract infection. Multivariable regression models were then used to investigate the relationship of SHS exposure, bronchiolitis severity, and family atopy.

RESULTS. Of 451 infants with lower respiratory tract infection, more than one-half had SHS exposure, more than one-third had mothers with atopic disease, and more than two-thirds had familial atopy. Univariate analysis showed that SHS resulted in higher BSS. However, when considering maternal atopic disease and allergic sensitization as variables, this study did not find a statistically significant difference in BSS or LOS with SHS exposure. Furthermore, utilizing familial atopy as a predictor of BSS in the SHS exposure groups showed no statistically significant difference versus those with no familial atopy. However, SHS exposure did increase the LOS of those infants with familial atopy. This finding is in contrast to patients with no familial atopy, in whom SHS did not affect LOS.

CONCLUSIONS. SHS was linked to longer LOS, but not BSS, in infants with familial atopy.

REVIEWER COMMENTS. As physicians, we are often faced with the daunting task of smoke-cessation counseling, particularly for the benefit of children exposed to SHS. Studies such as these are often referred to by physicians in daily practice when counseling families regarding the need for parental smoking cessation. Unfortunately, this study is limited by parental reports of SHS that are likely to be underreported. Because this study describes increased LOS for infants with SHS and lower respiratory tract infection in those with familial atopy, it lays the groundwork for further studies that may explore the consequences of these findings on the long-term pulmonary health of children.

URL: www.pediatrics.org/cgi/doi/10.1542/peds.2014-1817Y

Grace T. Padron, MD
Vivian Hernandez-Trujillo, MD
Miami, FL

Passive Smoking Impairs Histone Deacetylase-2 in Children With Severe Asthma

PURPOSE OF THE STUDY. The oxidative stress from tobacco smoke impairs histone deacetylase–2 (HDAC2) via phosphorylation of phosphoinositide-3-kinase (PI3K)/Akt activation, leading to corticosteroid insensitivity. This study tested the hypothesis that passive exposure to tobacco smoke is associated with reduced HDAC2 in alveolar macrophages in children with severe, refractory asthma.

STUDY POPULATION. The study population included children aged 8.5 to 13.5 years with severe asthma, already receiving inhaled corticosteroids plus a long-acting $\beta$-agonist.

METHODS. Bronchoalveolar lavage fluid was obtained from all children during bronchoscopy. Subjects were divided into those exposed to passive tobacco smoke (PS) and those not exposed to PS. Exposure was assessed by using parent surveys and was supported by measurement of cotinine levels in the saliva and urine. Fractional exhaled nitric oxide levels were also measured. HDAC2 expression and activity, Akt/HDAC2 phosphorylation levels, and corticosteroid responsiveness in alveolar macrophages were assessed.

RESULTS. Parental reports of smoking correlated with measurable cotinine levels in the urine and saliva of the children. PS exposure reduced HDAC2 protein expression and activity. PS exposure also reduced the inhibitory effects of dexamethasone of tumor necrosis factor $\alpha$–induced CXCL8 release in the alveolar macrophages. Children exposed to PS had higher neutrophil counts and CXCL8 expression in bronchoalveolar lavage fluid and lower Asthma Control Test scores compared with those not exposed.

CONCLUSIONS. PS exposure in children impairs HDAC2 function via PI3K signaling activation. This finding agrees with previous studies in adults. The high levels of oxidative stress and their end products seem to induce corticosteroid insensitivity with dysfunction of HDAC2. This study supports the conclusion that PS exposure not only worsens asthma symptoms but induces a state of steroid resistance in an already difficult-to-control asthma population.

REVIEWER COMMENTS. It is well known that secondhand tobacco smoke exposure is related to exacerbation of asthma in children and can be a risk factor for persistent asthma in later childhood. This study shows the molecular basis for this finding in children with severe asthma and how PS exposure can induce steroid insensitivity. Patients with severe asthma are already difficult to treat, and steroid resistance makes it even more difficult. This finding highlights the need, even more so, for parental education regarding the effects of tobacco smoke exposure on their children with asthma.
A Majority of Parents of Children With Peanut Allergy Fear Using the Epinephrine Auto-Injector


PURPOSE OF THE STUDY. The purpose of this study was to identify factors that may contribute to parental fear of using an epinephrine autoinjector (EAI).

STUDY POPULATION. The study included 1229 parents of children with peanut allergy, all of whom had been prescribed an EAI. The mothers had a mean age of 37.9 years, and the fathers had a mean age of 40 years.

METHODS. Children with peanut allergy were retrospectively identified from 2000 to 2004 through chart review, and they were prospectively identified between 2004 and 2011 at their visit to Montreal Children’s Hospital. Parents of these children were mailed a questionnaire on whether and why they feared using an EAI, if their child had ever received an EAI, who prescribed the EAI, their level of satisfaction with the EAI training they received, the interval between reaction to prescription of the EAI, the type of EAI they were prescribed, whether they had changed devices, and the number of EAIs purchased. Fear was characterized as “afraid,” “somewhat afraid,” or “not afraid.”

RESULTS. Fifty-six percent of parents reported being afraid or somewhat afraid to use the EAI. The most commonly reported fears were hurting the child, incorrect use of the EAI, or fear of a bad outcome or death. Several predictors of parental fear were identified, including having a younger child and those with a shorter disease duration. In addition, their children were less likely to have experienced a severe reaction or to have required an EAI. With regard to parental characteristics, those who were characterized as having fear were slightly younger, had less satisfaction with EAI training, and were less likely to find the EAI easy to use. Factors associated with less fear included longer disease duration or older age of the mother.

CONCLUSIONS. This study found that a majority of parents have fear regarding use of the EAI. Factors that may predict fear include younger age of children, lack of severe reaction, and dissatisfaction with EAI training.

REVIEWER COMMENTS. This study is the largest to examine parental anxiety and fear regarding EAIs. The presence of parental fear could lead to delayed or lack of use in a severe allergic reaction, which is consistent with previous studies demonstrating low EAI use even in the face of severe reactions. Parents who were dissatisfied with their EAI training were more likely to express fear, highlighting the importance of appropriate EAI training by both prescribing physicians as well as others caring for children who have food allergies. This study was limited with regard to ethnic diversity, and the majority of parents were highly educated and employed.

Anaphylaxis Knowledge and Practice Preference of Pediatric Emergency Medicine Physicians: A National Survey


PURPOSE OF THE STUDY. The goal of this study was to assess pediatric emergency medicine physicians’ knowledge and practice preferences for anaphylaxis.

STUDY POPULATION. A cross-sectional sample was used with participants recruited by using contact information obtained from the American Board of Pediatrics and the American Board of Medical Specialties. Participants were given a 12-item survey.

METHODS. A total of 1124 invitations were sent to the identified participants through SurveyMonkey. The survey included 12 questions covering demographic characteristics, physicians’ practices including medications preference, preferred route of epinephrine administration, duration of patient monitoring, discharge medications, prescription home with epinephrine autoinjectors, referral to specialists, and referral to educational Web sites. Emergency department settings were separated into university hospital, nonuniversity hospital with a residency training program, and community hospital with no residency training program. Data were collected by using the SurveyMonkey software.

RESULTS. Of the 1124 physicians, 56% responded, 3% opted out, and 0.9% no longer practiced medicine. The remaining did not respond. Overall, 93.5% correctly identified epinephrine as the treatment of choice for anaphylaxis but only 66.9% used the intramuscular route, which is the preferred method. Hospitals with residency programs had higher rates of intramuscular epinephrine use and higher volume of anaphylaxis cases, which in turn were associated with a decreased likelihood of admission for patients with anaphylaxis. In addition, 98.7% provided a prescription for an epinephrine autoinjector, 72.4% were referred to an allergy specialist, and 8.7% provided information to an educational Web site.
Passive Smoking Impairs Histone Deacetylase-2 in Children With Severe Asthma
Melinda M. Rathkopf
Pediatrics 2014;134;S147
DOI: 10.1542/peds.2014-1817Z

<table>
<thead>
<tr>
<th>Updated Information &amp; Services</th>
<th>including high resolution figures, can be found at: /content/134/Supplement_3/S147.full.html</th>
</tr>
</thead>
<tbody>
<tr>
<td>Permissions &amp; Licensing</td>
<td>Information about reproducing this article in parts (figures, tables) or in its entirety can be found online at: /site/misc/Permissions.xhtml</td>
</tr>
<tr>
<td>Reprints</td>
<td>Information about ordering reprints can be found online: /site/misc/reprints.xhtml</td>
</tr>
</tbody>
</table>
Passive Smoking Impairs Histone Deacetylase-2 in Children With Severe Asthma

Melinda M. Rathkopf

*Pediatrics* 2014;134;S147

DOI: 10.1542/peds.2014-1817Z

The online version of this article, along with updated information and services, is located on the World Wide Web at:
/content/134/Supplement_3/S147.full.html