# Cockroach allergen abatement: The good, the bad, and the ugly

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Allergists are better trained in environmental avoidance as a treatment for asthma than any other specialists. We understand the immunology of IgE-mediated sensitization and the exquisite sensitivity to immediate and chronic airway disease from environmental exposures that sensitization confers. However, we are just beginning to understand the important details of indoor environmental exposures and their relationship to chronic asthma, helped along by the available assays for mite, pets, and cockroach allergens. The article by Arbes et al[1] in this issue adds significantly to our understanding of the specifics of abatement procedures for cockroach allergen, now recognized as an important indoor allergen in increasing morbidity from asthma.

First, the study tells us that it is possible to eliminate the allergen source, even in multifamily dwellings. Pest control experts[2] think of controlling pest populations rather than exterminating them because they believe that it is not realistic to eliminate all cockroaches. The currently accepted approach is called integrated pest management and combines pesticide application with family education and structural elimination of hiding places ("harborages"). Recently, toxicologists have raised issues about the safety of the application of pesticides in homes, especially at the high doses required to reduce cockroach populations. The pesticide used by Arbes et al,[1] hydromethylnon, is a newer pesticide that is both quite effective and safe. It is available as a gel bait, licensed to pest control companies, and available in lower concentrations in bait traps available to the consumer in grocery and hardware stores. The gel baits described in the article are odorless and colorless and are placed by the pest control technician in blobs smaller than a dime in multiple areas of a room. Because they are unobtrusive and because they harden quickly, these agents are not generally attractive to pets and children. Other newer pesticides, including fipronyl, sulfluramide, and abamectin, are also available in gel baits and are more effective or safer than older agents, such as organophosphates and boric acid. Boric acid is quite safe but is much less effective than any of the newer agents. In heavily infested homes, such as the ones included in this study, a second or third application is required because the roaches consume them. Family expectations need to be addressed because these new agents are somewhat slow to act, taking several weeks to have maximum effect.

Of the older agents, the class that raised most safety concerns was the organophosphates, which were effective but had acute neurologic toxicity in large doses and were thought to have long-term neurologic and developmental toxicity in human subjects as well.[3,4] In 2000, the Environmental Protection Agency removed its certification for indoor use of chlorpyrifos, one of the organophosphates, because of this issue.[5] Another older group of pesticides, pyrethrin and the synthetic pyrethrins deltamethrine and fenvalerate, are also effective. Because they give cockroach treatment a characteristic pungent odor that is less acceptable to consumers and because there are emerging questions concerning chronic toxicity,[6] they are less used than previously. Abamectin is one of the safest new pesticides. It has a substantial history of oral use in animals and human use as an antihelminthic[7] and as an oral treatment for scabies.[8]

Using integrated pest management, Arbes et al[1] were able to markedly reduce cockroach populations in heavily infested apartments in low-income apartment buildings. The homes chosen for this study were an exterminator's nightmare. Every home had dozens of cockroaches collected in traps, and every bedroom had high levels of Bla g 1 contamination; these would be in the top 5% of real-life infested homes. Such homes could be identified in a clinical practice by asking, "When do you see roaches: daytime, nighttime, or both?" Roaches usually hide in cracks and crevices and then forage at night; therefore if roaches are seen in the daytime, it is a sign of heavily infested homes, such as the ones included in the trial. By successfully dealing with infestation such as this and reducing populations to the point that 6 of the 16 treated bedrooms had no roaches in the traps within a month of beginning treatment and that 10 to 11 of the treated homes had no roaches for the last 3 months of the study, Arbes et al[1] demonstrated unequivocally that huge cockroach populations can be controlled in multifamily dwellings. This is the answer to skeptics who say that cockroaches cannot be treated successfully and that reinfestation will always occur. Our own studies[9,10] were just as successful as this one, but we studied row homes rather than multifamily apartment buildings. It would appear that by using effective pesticides and helping families to seal all sources of food for the roaches, it is possible to create conditions in which the insects use their heads (or instincts) and stay in adjoining apartments, where living is easier.

Because this is the most aggressive intervention effort yet published, the limits found on allergen reduction are an important biologic lesson. The authors were only able

From Johns Hopkins University, Baltimore, Md.

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Reprint requests: Peyton A. Eggleston, MD, CMSC 1102, Johns Hopkins Hospital, 600 North Wolfe St, Baltimore, MD 21287.

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to reduce settled dust Bla g 1 levels to less than 8 U/g in 9 of 16 bedrooms and less than 2 U/g in 3 of 16 bedrooms in the treatment group. Using even more aggressive cleaning than we used in our own studies,[9,10] they were not able to eliminate residual allergen within 6 months after eliminating the source. Thus residual cockroach allergen appears to be even more difficult to remove from homes than animal dander. Cat allergen takes 6 months of household cleaning to remove from a home once the cat is removed.[11] The most likely explanation for this difficulty is that the particles carrying cat allergen are adherent and contaminate so much of the home that a home allergen reservoir exists that is very large and complex and requires months of cleaning to remove. We would agree with Arbes et al[1] that the ecology of the cockroach also contributes to this difficulty. Roaches seek hiding places where both their backs and bellies are touching a hard surface, and therefore they tend to cluster in narrow crevices, where allergen-laden carcasses are difficult to remove. In addition, roaches regurgitate digestive fluids while feeding, leaving a residue that might be visible as a brownish stain resembling cooking grease. For all these reasons, it is not surprising that it might take more than 6 months of cleaning to remove the last traces of allergen contamination.

Another important finding in this study was that bedding was heavily contaminated. In the intervention group Bla g 1 levels decreased in bedding from 6.1 U/g at baseline to 1.0 U/g, and this decrease occurred somewhat more slowly than other household levels. The authors proposed that bedding had become contaminated passively, with allergen carried into the bed on feet and clothing. This was our own experience in a trial of reducing allergen in bedding by treating the home and providing clean bedding every 2 weeks.[12] We found allergen in the occupant's clothing and found that bedding became contaminated within days of placement. Passive distribution also seemed to be the best explanation for our finding that middle-class homes in which infestation had never occurred contained low levels of allergen in kitchens, presumably tracked in on shoes and paper products.[13] Bedding contamination is very important because cockroach allergen is carried on large particles that do not become airborne under usual circumstances.[14] Thus exposure to cockroach allergen is likely to be through intimate contact with bedding while sleeping, just as in the case of house dust mite allergen.

The authors question whether the reductions that they were able to achieve would have an effect on symptoms and morbidity in allergic asthmatic residents. Although there are no trials of cockroach allergen avoidance yet reported with health outcomes, it is likely that significant health improvement would be seen. House dust mite allergen exposure is a good example of an allergen in which exposure occurs primarily in bedding and in which successful bedding treatment leads to long-term improvement in allergic asthma. In reported house dust mite intervention trials, those that focused on bedding and were able to achieve a 50% reduction in bedding allergen levels generally showed significant reduction in morbidity (symptoms, peak flow rates, and medication use) in asthmatic patients allergic to mites.[14] There does not seem to be a safe threshold, and as long as this proportional decrease is achieved, significant improvement in allergic asthma is seen, regardless of the starting level of allergen. The National Cooperative Inner City Asthma Study intervention trial mentioned in the article was a global intervention, with only modest attention to a cockroach allergen intervention.[15] The global intervention was effective and reduced asthma morbidity, but cockroach allergen did not decrease, and therefore cockroach allergen abatement could not be said to have contributed.[16] There are now at least 3 clinical trials ongoing that are testing the clinical effects of cockroach allergen reduction on asthma severity. The Inner City Asthma Study is a National Institute of Allergy and Infectious Disease–sponsored multicenter trial that has enrolled over 300 asthmatic children. In preliminary results presented at the American Academy of Allergy, Asthma and Immunology annual meeting, these researchers reported significant clinical results with a lesser reduction in cockroach allergen. Other clinical trials are ongoing, and results are not available, but these early results provide hope that cockroach allergen abatement will have a significant health effect on asthmatic patients living in US inner cities.

The article by Arbes et al[1] in this issue of the *Journal* is a model of the sort of difficult, well-planned, and well-executed clinical research that will move the field of environmental avoidance forward. It represents what is technically called an efficacy trial in that the investigators chose to apply the treatment with as little reliance on the adherence of the families participating as possible. This is as opposed to an effectiveness trial that would apply the treatment as it might be applied in clinical practice or by a public health department, usually relying on participant adherence. Hopefully, it will be followed not only by effectiveness trials to see whether this success can be accomplished on a larger scale by using less intensive efforts but also by clinical trials to document the clinical effect of cockroach allergen abatement. We should be encouraged by these successful early efforts to develop an asthma intervention that is appropriate for public health agencies and does not rely on chronic drug treatment.

*Peyton A. Eggleston, MD*
*Baltimore, Md*

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## Corrections

The following corrections pertain to the February 2003 *Primer on Allergic and Immunologic Diseases* (2003;111:S441-S778).

*Chapter 4 (Prussin C, Metcalfe DD. IgE, mast cells, basophils, and eosinophils. S487-S494):* In Fig 1 (p S487), illustrating molecular interactions between $T_H2$ cells and B cells required for IgE synthesis, the accessory binding molecules CD40 and CD40L (CD154) on the T cell and the antigen-presenting cell, respectively, were inadvertently reversed. The correct designation should be that the CD40L (CD154) is on the T-cell surface and the CD40 molecule is on the antigen-presenting cell.

*Chapter 5 (Steinke JW, Borish L, Rosenwasser LJ. Genetics of hypersensitivity. S495-S501):* In the section entitled "Candidate gene studies" (p S497), it is stated in the fifth sentence of the third paragraph that ADAM33 (protease) is expressed in epithelium, smooth muscle, and inflammatory cells. In fact, ADAM33 is expressed in lung fibroblasts, myofibroblasts, and smooth muscle cells, but there is little or no expression in epithelial cells or leucocytes.

*Chapter 12 (Bonilla FA, Geha RS. Primary immunodeficiency diseases. S571-S581):* In Table III (p S574), which presents lymphocyte phenotypes characteristically associated with particular forms of severe combined immunodeficiency (SCID), it is indicated that IL-7 receptor–deficient SCID is similar to other forms of $T^-B^+$ SCID in having reduced natural killer cells. This is incorrect. In IL-7 receptor deficiency, natural killer cell numbers are normal.