

The prevalence of environmental exposure to perceived asthma triggers in children with mild-to-moderate asthma: Data from the Childhood Asthma Management Program (CAMP)

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Background: The Childhood Asthma Management Program, a 5-year randomized clinical trial of treatments for childhood asthma, has enrolled and characterized a cohort of 1041 children with mild-to-moderate asthma.

Objective: We sought to describe self-reported sensitivities and environmental exposures and investigate the relationships between self-report of these exposures as asthma triggers and their prevalence in the home.

Methods: Self-reports of sensitivities and home exposures were obtained by interview with the child or parent. Sensitivities were further assessed by using allergy skin testing (prick or puncture) against a core battery of allergens. Home exposures were further assessed by using analysis of a home dust sample.

Results: Environmental exposures were surprisingly common despite self-reported sensitivities to environmental factors. Of patients reporting that cigarette smoking frequently causes asthma symptoms, 26% reported having at least one parent who smokes cigarettes. Thirty-nine percent of patients reporting that exposure to animals frequently causes asthma symptoms live with a furry pet in their home. We found a smaller proportion of homes with a high level of cat allergen ($P < .001$) among the children who reported that animals frequently or always trigger asthma symptoms compared with those who reported that animals never or occasionally trigger asthma symptoms, suggesting modification of the home environment. No such results were seen for dog exposure. However, clinical symptoms did not reduce exposure to parental cigarette smoking ($P = .15$), house dust ($P = .31$), or damp and musty areas ($P = .51$).

Conclusion: These data suggest that children with mild-to-moderate asthma are frequently symptomatic and exposed to a wide variety of environmental exposures that are perceived to trigger symptoms by means of self-report. Although environmental modification of asthmatic homes may occur, many children remain exposed to agents that are known to trigger their asthma. (*J Allergy Clin Immunol* 2001;107:634-40.)

Key words: Allergen exposure, asthma symptoms, childhood asthma, Childhood Asthma Management Program, environmental allergen sensitization, skin prick tests

The Childhood Asthma Management Program (CAMP) has been launched as a multicenter long-term trial aimed at assessing long-term issues in childhood asthma and its management. One thousand forty-one children aged 5 to 12 years at enrollment were recruited from November 1993 to September 1995 at 8 participating clinical centers after satisfying entry criteria that supported the diagnosis of chronic mild-to-moderate asthma and the likelihood of continuing with the trial for 5 years. Children have been randomized to one of 3 treatment groups for the study duration: inhaled corticosteroid (budesonide, 200 mg twice daily), inhaled nonsteroidal anti-inflammatory agent (nedocromil, 16 μ g twice daily), or placebo. β -Agonist (albuterol administered by means of metered-dose inhaler) is provided as a rescue bronchodilator for all children. Procedures for adding or tapering medical intervention for asthma while maintaining the integrity of the study protocol have been standardized. A short course of prednisone is used for severe asthma exacerbation. Although the primary outcome measure of CAMP is lung growth determined by postbronchodilator FEV₁, secondary outcome measures include instruments to assess asthma severity, methacholine airway responsiveness, somatic growth, bone density, neurocognitive development, and individual and family functioning.

The clinical characteristics of mild-to-moderate asthma have not been well described.¹⁻⁹ CAMP is unique in that the trial used criteria based on symptoms and medication use to define a group of children with mild-to-moderate disease. A 28-day screening period during which the child received only β -agonists as needed to relieve asthma symptoms was used to confirm this diagnosis before randomization. Demographic and general characteristics of the CAMP population have been

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Supported by contracts NO1-HR-16044, 16045, 16046, 16047, 16048, 16049, 16050, 16051, and 16052 with the National Heart, Lung, and Blood Institute and General Clinical Research Center grants M01RR00051, M01RR0099718-24, M01RR02719-14, and RR00036 from the National Center for Research Resources. The Harvard Pilgrim Health Care Foundation provides additional support for the CAMP center at Brigham & Women's Hospital.

*See appendix for listing of members of the CAMP Research Group.

Received for publication September 6, 2000; revised December 19, 2000; accepted December 22, 2000.

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0091-6749/2001 \$35.00 + 0 1/81/113869

doi:10.1067/mai.2001.113869

Abbreviation used

CAMP: Childhood Asthma Management Program

reported in detail elsewhere.^{10,11} This article investigates the relationships between self-reported sensitivities and environmental exposures.

METHODS

The details of the CAMP study design, rationale, and methods have been published.¹² Children aged 5 to 12 years with chronic asthma were recruited over a 23-month period from clinic rosters; emergency department records; primary care practices; radio, television, and newspaper advertisements; and other sources. Entry criteria included asthma symptoms, medication use, or both for 6 months or more in the previous year. Patients were excluded if they had needed prednisone bursts more than 5 times or had more than one hospitalization for asthma in the year before their initial interview.

All candidates were evaluated over a period of 5 weeks to 4 months (5 visits) to confirm the presence of mild-to-moderate asthma and for the gathering of baseline information. From the second screening visit on, participants kept asthma symptom and medication use diaries, as well as twice daily peak flow diaries to confirm the presence of ongoing asthma. For at least a 28-day period, children received only as-needed albuterol for asthma therapy. Symptoms and medication use were monitored during this 28-day period. Asthma was considered to be too mild if there were fewer than 8 days with asthma symptoms or with morning or evening peak flow less than 80% of personal best values. Asthma was considered too severe if, in this 28-day period, the child used more than 8 puffs of albuterol for each of 3 consecutive days, had night awakenings caused by asthma averaging more than 1.5 times per week, had mean daily diary card symptom scores of greater than 2 (scale, 0-3), or had used medication other than albuterol to control asthma.

Data were collected by interview between the research coordinator and parents, child, or both (depending on child's age) regarding demographics, history of asthma symptoms and severity, treatment for asthma, asthma triggers, allergy history, features of the home environment, and relevant family history.

Allergy skin testing for pertinent inhalant allergens was carried out by a CAMP-certified technician using the prick or puncture method. Children were tested for sensitivity to a core battery of allergens (Der p 1, Der f 1, cat [standardized], dog [mixed breed], American cockroach, German cockroach, *Penicillium* species mix, *Aspergillus* species mix, Timothy grass, and short ragweed [standardized]), as well as 4 to 6 site-selected allergens. Seven clinical centers tested children for sensitivity to *Alternaria* species.

A home dust sample was obtained after randomization (mean duration, 6 months) with a vacuum cleaner (Douglas Redi-vac Model #6735; Scott-Fetzer, Walnut Ridge, Ariz) fitted with a disposable filter. Dust samples were analyzed at a central laboratory for their content of dust mite, cat, dog, and cockroach allergens. An agar plate was streaked for enumeration of fungal colonies, which were not further identified.¹²

Randomization depended on successful completion of the baseline evaluation and signed consent of the parent or guardian to the child's enrollment. Signed assent of the child participant was also required at some clinics. The institutional review board of each center approved the trial.

The CAMP database involves a distributed data management system. All data are keyed twice in immediate succession by the

same person. Extensive checks and audits are run on a routine basis by the data coordinating center. All analyses were performed with SAS version 6.12 software.¹³ The Pearson χ^2 statistic was used to test the association of a self-report of exposure triggering asthma symptoms and the prevalence of the exposure in the children's homes or the sensitivity to the exposure, as determined by means of skin prick testing.¹⁴

RESULTS

Demographics

Characteristics of the CAMP population are summarized in Table I, and more detailed descriptions can be found elsewhere.^{10,11} The mean age of the children at study entry was 8.4 years, and 60% were boys. Thirty-two percent of the children represented various ethnic minority groups. The educational background of the parent or legal guardian escorting the child to the initial screening visit suggests that most of the children came from well-educated families; 82% had some college or postsecondary education or training or had finished college. Seventy-four percent of the US respondents reported combined family yearly earnings equaling or exceeding \$30,000.

Asthma history and medications

Thirty-six percent of children had their first asthma episode before the age of 2 years, and 90% had their first asthma episode before the age of 7 years. Duration of asthma since onset of symptoms ranged from 6 months to 12 years (median, 5.6 years). A majority of children (62%) had their first identified asthma episode during a respiratory infection (Table I). For 19% of the children, their first asthma episode was associated with allergic exposure. Although most study participants did not require hospitalization in the year before study entry, 35% had required oral corticosteroids in the previous 6 months. Eighty-seven percent of children had asthma symptoms 2 or more times a week for 6 or more months in the year before interview, and 75% required bronchodilators 2 or more times a week for 6 or more months during the year before study entry.

Self-reported asthma triggers for the study group are summarized in Fig 1. Factors perceived by the respondent as causing asthma symptoms at study entry always or frequently included respiratory infections (77%), spring and fall pollens (53%), changes in the weather (48%), exercise (42%), and tobacco smoke (40%). Other factors were less frequently cited as asthma triggers.

Allergy skin testing

Eighty-eight percent of the children had at least one positive skin test response (Table I), and 70% had 3 or more positive test responses. Sensitivities to Der p 1, Der f 1, cat, and Timothy grass were the most common overall and were seen in 43%, 42%, 49%, and 47% of study children, respectively. Thirty-seven percent of the 915 children tested for sensitivity to *Alternaria* species had a positive test response.

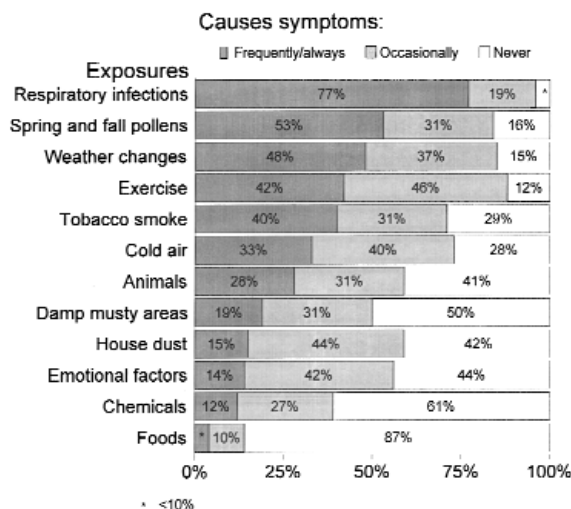


FIG 1. Exposures causing asthma symptoms.

TABLE I. Characteristics of study participants and their families

Characteristic	Combined study group*
Age at randomization, mean $\pm$ SD (range)	8.4 $\pm$ 2.1 y (5-13)
Minority ethnicity-race	32%
Male sex	60%
Parent-guardian attended or completed college or postsecondary education	82%
Annual household income $\geq$ \$30,000	74% [†]
Conditions associated with first asthma episode	
Infection	62%
Exercise	7%
Allergic exposure	19%
Asthma symptoms and medication use	
Symptoms $\geq$ 2 times per wk for $\geq$ 6 mo	87%
Bronchodilator use $\geq$ 2 times per wk for $\geq$ 6 mo	75%
Asthma medication most days for $\geq$ 6 mo	59%
Oral steroids in last 6 mo	35%
At least 1 positive allergen skin test response	88%
Family history of asthma or atopic disorder	
At least 1 parent with asthma	41%
At least 1 parent with hay fever, eczema, or other atopic disorder	64%
Sibling with asthma	37% [‡]
Sibling with hay fever, eczema, or other atopic disorder	38% [§]

*Because of missing data, numbers of patients range from 1021 to 1041, except as noted.

[†]Denominator for percentage is defined as US participants who responded (n = 879).

[‡]Denominator for percentage is defined as CAMP children with siblings for whom asthma status was known (n = 905).

[§]Denominator for percentage is defined as CAMP children with siblings for whom atopic status was known (n = 902).

Family history, smoking history, and the home environment

Many of the children with asthma recruited for this trial came from asthmatic and atopic families. Of the children entering the trial, 41% had at least one parent

TABLE II. Characteristics of the home environment

Characteristic	Combined study group
Mother's previous smoking habits	
During pregnancy	14%
Before 2 y of life	20%
Between 2 y and first grade	21%
Father's previous smoking habit	
Before 2 y of life	32%
Between 2 y and first grade	30%
Current smokers in household	
Mother currently smokes	16%
Father currently smokes	20%
No parent smokes	72%
One parent smokes	20%
Both parents smoke	8%
Child smokes	0%
Pets in the home (self-report)	
Furry pet	53%
Cat	23%
Dog	34%
Rodent	12%
Bird	9%
Heating and cooking devices	
Gas stove	40%
Fireplace	20%
Wood stove	9%
Allergen level (house dust sample)	
Der p 1 + Der f 1 >2000 ng/g (n = 969)	42%
Fel d 1 >8000 ng/g (n = 969)	29%
Can f 1 >8000 ng/g (n = 888)	44%
Mold >25,000 cfu/g (n = 958)	20%
Month of dust collection (n = 968)	
Dec, Jan, Feb	20%
March, April, May	29%
June, July, Aug	32%
Sep, Oct, Dec	19%

Because of missing data, numbers of patients range from 1005 to 1041, except as noted.

with asthma. In addition, 37% of the study children with siblings had siblings with asthma. Sixty-four percent of the children had at least one parent with hay fever, eczema, or another atopic disorder. Thirty-eight percent of the CAMP children with siblings had siblings with hay fever, eczema, or another atopic disorder.

Many of the children with asthma recruited for this study were exposed to tobacco smoke (Table II). Forty-one percent were exposed either during pregnancy or in the first 2 years of life. Surprisingly, many of these children with asthma continued to be exposed to tobacco smoke on a regular basis at study entry, with 28% living in a home with at least one parent smoking cigarettes. None of the children smoked at study entry.

Despite a high prevalence of allergic disease in study families and of skin test reactivity in the children, 53% reported having a furry pet in the home as of study entry (Table II). Other potential home irritant exposures for children with asthma recruited into this study included gas stoves (40% of children), fireplaces (20% of children), and wood-burning stoves (9% of children).

TABLE III. Exposure to triggers in the home or skin test outcome versus self-report of trigger causing asthma symptoms

Trigger/exposure in home or sensitivity to trigger	Trigger frequently/always causes asthma (%)	Trigger occasionally/never causes asthma (%)	P value*
Animals			
Furry pet in home	39%	59%	<.001
Fel d 1 >8000 ng/g	18%	34%	<.001
Can f 1 >8000 ng/g	42%	44%	.63
Skin test response positive to cat	75%	39%	<.001
Skin test response positive to dog	35%	18%	<.001
No. of children reporting effect of trigger	293 [†]	737 [‡]	
Cigarette smoke			
Both parents smoke	6%	9%	.13
At least 1 parent smokes	26%	30%	.15
No. of children reporting effect of trigger	397	606	
House dust			
<i>Dermatophagoides pteronyssinus</i> + <i>Dermatophagoides farinae</i> >2000 ng/g	38%	42%	.31
Skin test response positive to <i>D pteronyssinus</i>	58%	41%	<.001
Skin test response positive to <i>D farinae</i>	50%	40%	.03
Skin test response positive for <i>D pteronyssinus</i> or <i>D farinae</i>	63%	46%	<.001
No. of children reporting effect of trigger	150 [§]	880	
Damp or musty areas			
Mold >25,000 cfu/g	19%	21%	.51
Skin test response positive to <i>Penicillium</i> species	29%	24%	.11
Skin test response positive to <i>Aspergillus</i> species	29%	21%	.02
Skin test response positive to <i>Penicillium</i> or <i>Aspergillus</i> species	39%	30%	.01
No. of children reporting effect of trigger	190 [¶]	823 [#]	

*P values were determined with the Pearson χ^2 statistic (1 df).[†]Number of patients varies from 248 to 293 because of missing data.[‡]Number of patients varies from 630 to 737 because of missing data.[§]Number of patients varies from 133 to 150 because of missing data.^{||}Number of patients varies from 826 to 880 because of missing data.[¶]Number of patients varies from 169 to 190 because of missing data.[#]Number of patients varies from 763 to 823 because of missing data.

Analysis of home dust samples for allergens indicated that many children were living in homes with allergen levels above the threshold for high risk for symptoms (Table II). Dust mite allergen levels above 2000 ng/g, generally regarded as a risk factor for sensitization,¹⁵ were noted in 42% of home dust samples; cat allergen levels above 8000 ng/g¹⁵ were noted in 29% of samples, and mold colony counts above 25,000 colony-forming units (cfu)/g were noted in 20% of homes.

We examined the relationship of a self-report of exposures triggering asthma symptoms and the prevalence of those exposures in the children's homes (Table III). We found a lower proportion of homes with a high level of cat allergen ($P < .001$) among the children who reported that animals frequently or always triggered asthma symptoms compared with those who reported that animals never or occasionally triggered asthma symptoms. No such relationship existed for dogs (Table III). However, clinical symptoms did not reduce exposure to parental cigarette smoking ($P = .15$), house dust ($P = .31$), or damp or musty areas ($P = .51$), and the prevalence of exposures continued to be high, even among children

who reported adverse personal effects of these exposures (parental cigarette smoke, 26%; furry pet, 39%; and house dust, 38%). The self-reports of animals, dust, and mold causing asthma correlated with the skin test results for these triggers (Table III). Sensitization to cats or dogs was significantly more prevalent in subjects reporting that animals frequently cause asthma symptoms relative to those subjects reporting that animals cause asthma symptoms occasionally or never (Table III). However, there were large numbers of children who reported that animals frequently or always cause asthma symptoms who did not have positive test responses to cat allergen (25% of those reporting animals frequently cause symptoms) and who did not have positive test responses to dog allergen (65%).

For all allergens examined (cat, dog, dust mite, *Penicillium* species, and *Aspergillus* species), higher proportions of the children reporting that allergen exposure frequently or almost always causes asthma had positive skin test responses to the exposure compared with those reporting that exposures never or occasionally cause asthma (Table III).

DISCUSSION

The characteristics of the CAMP participants approximate the goals of the trial reasonably well, which were to enroll children with mild-to-moderate asthma that might potentially benefit from long-term anti-inflammatory therapy and hence influence growth of lung function. The descriptive characteristics of the CAMP participants demonstrate a consistency with known truisms about prepubertal childhood asthma.^{1-9,16} In general, this disease has a male predominance, as is found in the CAMP participants, and usually asthma onset occurs before the age of 6 years.¹⁷ In the CAMP trial 84% of our patients had disease onset before the age of 6 years. Information on oral steroid use and asthma symptoms demonstrates that the CAMP enrollees had significant disease, at least symptomatically. Almost 90% of patients had symptoms more than 2 times a week for at least 6 months during the year before enrollment, and 75% used a bronchodilator at least twice a week for at least 6 months during the year before enrollment.

Our study documents for the first time in a large sample the environmental burden faced by children with mild-to-moderate asthma with regard to the home environment. High levels of parental cigarette smoking (28% with one or both parents as smokers), furry pets in the home (present in over 50% of CAMP patients' homes), house dust mite allergen (>2000 ng/g in 42% of homes), and mold counts (>25,000 cfu/g in 20% of homes) signify the importance of environmental exposure for this disease.^{15,18-21} This is underscored by the prevalence of skin test reactivity of 88% in the CAMP cohort (≥ 1 positive prick skin test response). Clearly, the bulk of childhood asthmatic subjects are atopic, and this has been shown in multiple investigations.^{22,23} It remains to be seen whether atopic or nonatopic asthmatic subjects have a more benign clinical course.^{16,24}

Our study design does not allow us to determine whether CAMP parents were counseled about environmental exposure before collection of these baseline data. Because all trial participant families were repeatedly counseled about these exposures after study entry, our longitudinal data should allow us to assess the effectiveness of this intervention.

Analysis of exposure to triggers versus self-report of triggers causing asthma attacks (Table III) indicates less exposure in the more sensitive children with respect to cigarette smoke, cat allergen in the home, damp and musty areas, and house dust. However, only analysis related to cat allergen yielded statistically significant differences ($P < .001$). Although the lesser amount of exposure reported for the more sensitive children suggests that families of more sensitive children may be trying to reduce environmental exposures, the nontrivial proportion of children reportedly having high sensitivity and high exposure demonstrates the challenge for the clinician in attempting to keep patients with mild-to-moderate asthma symptom free and functioning at their highest level. The correlation between sensitivity to the allergen

and self-report of the allergen causing asthma is supportive of the accuracy of the patients' self-reports.

It will be important to determine in the CAMP trial the extent to which sensitization and allergen level interact with treatment in determining growth of lung function in the CAMP children. It is important to recognize the relatively preserved pulmonary function in the face of moderate symptoms seen in the CAMP patients. This scenario of relatively frequent symptoms with preserved lung function is typical of childhood asthma and has often caused clinicians to consider the disease benign and not worthy of aggressive treatment. Although in the short term, such as in the 28-day screening period before randomization, the morbidity associated with minimal medication use is relatively small, the obvious concern and rationale for the CAMP trial is that continued symptoms and inflammation can ultimately result in deterioration of lung function and less than optimal lung growth. The central premise behind the trial is that control of the environment, avoidance of triggers, and continuous anti-inflammatory treatment should improve and maximize growth of lung function, thus working to prevent the development of disease progression and, ultimately, to prevent the development of fixed airflow obstruction. Only the completion of this trial and the analysis of its longitudinal data will allow us to determine the answer to this question.

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University of New Mexico, Albuquerque, NM: H. William Kelly, PharmD (director); Robert Annett, PhD (coinvestigator); Naim Bashir, MD (coinvestigator); Michael Clayton, MD (coinvestigator); Angel Colon-Semidey, MD (coinvestigator); Mary Spicher, RN (coordinator); Marisa Braun; Shannon Bush; David Hunt, RRT; Elisha Montoya; Margaret Moreshead; Barbara Ortega, RRT; Hengameh H. Raissey; Roni Grad, MD (coinvestigator, 1993-1995); Bennie McWilliams, MD (coinvestigator, director, 1992-1998); Shirley Murphy, MD (coinvestigator, 1992-1994); Sandra McClelland, RN (coordinator, 1993-1995); Teresa Archibeque (1994-1999); H. Selda Bereket (1995-1998); Sara Devault (1993-1997); Jeanne Larsson, RN (1995-1996); David Weers (1997-1998); Jose Zayas (1995-1996).

Washington University, St Louis, Mo: Robert C. Strunk, MD (director); Leonard Bacharier, MD (coinvestigator); Gordon R. Bloomberg, MD (coinvestigator); James M. Corry, MD (coinvestigator); Ellen Albers, RN, CNS-P, MSN (coordinator); W. Patrick Buchanan; Greg Belle, MA; Marisa Dolinsky, MA; Edwin B. Fisher, PhD; Stephen J. Gaioni, PhD; Emily Glynn, RN, MSN, CSPNP; Bernadette D. Heckman, MA; Cathy Hermann; Debra Kemp, RN, BSN; Claire Lawhon, BS; Cynthia Moseid; Tina Oliver-Welker, CRTT; Denise Rodgers, RPFT; Sharon Sagel, MD; Deborah K. White, RPFT, RRT; Mary Caesar, MHS (coordinator, 1993-1996); Diana S. Richardson (1994-1997); Elizabeth Ryan, PhD (1994-1996); Thomas F. Smith, MD (coinvestigator, 1994-1998); Susan C. Sylvia, PhD (1994-1996); Carl Turner (1995-1997).

Resource centers

Bone Age Reading Center, Washington University, St Louis, Mo: William McAlister, MD (director); Keith Kronemer, MD (coinvestigator); Patty Suntrup.

Chair's Office, National Jewish Medical and Research Center, Denver, Colo: Reuben Cherniak, MD (study chair).

Coordinating Center, The Johns Hopkins University, Baltimore, Md: James Tonascia, PhD (director); Curtis Meinert, PhD (codirector); Debra Amend-Libercci; Marc Bacsafra; Patricia Belt;

Karen Collins; Betty Collison; Christopher Dawson; Dawn Dawson; John Dodge; Michele Donithan, MHS; Vera Edmonds; Cathleen Ewing; Judith Harle; Robert Huffman; Rosetta Jackson; Kung-Yee Liang, PhD; Jill Meinert; Jennifer Meyers; Deborah Nowakowski; Laura Oelberg; Bonnie Piantadosi, MSW, MPH; Michael Smith; Alice Sternberg, ScM; Mark Van Natta, MHS; Robert Wise, MD.

Dermatology, Allergy and Clinical Immunology (DACI) Reference Laboratory, Johns Hopkins University School of Medicine, Asthma & Allergy Center, Baltimore, Md: Robert G. Hamilton, PhD, DABMLI (director); Carol Schatz (business office manager); Jack Wisenauer, MT (laboratory supervisor).

Drug Distribution Center, McKesson BioServices Corp, Rockville, Md: Robert Rice, PhD, DVM (Director of Pharmaceutical Services Division Operations); Bob Hughes (Director of Pharmaceutical Repository); Tom Lynch (repository technician); Ken Farris; Jun Lee, RPh.

Fundus Photography Reading Center, University of Wisconsin, Madison, Wis: Barbara Klein, MD, MPH (director); Larry Hubbard; Michael Neider; Kurt Osterby; Nancy Robinson; Hugh Wabers.

Immunology and Complement Laboratory, National Jewish Medical and Research Center, Denver, Colo: Ronald J. Harbeck, PhD, DABMLI (director); Rhonda Emerick, MT (ASCP) SM; Brian Watson, MLT (ASCP).

Patient Education Center, National Jewish Medical and Research Center, Denver, Colo: Stanley Szeffler, MD (director); Bruce Bender, PhD (coordinator); Harold Nelson, MD; Cindi Culkin, MEd (coordinator, 1996-1997); Jeryl Feeley, MA (coordinator, 1992-1995); Sarah Oliver, MPH (co-coordinator, 1992-1996); Colleen Lum Lung, RN (1992-1994); Ann Mullen, RN (1994-1996); Christine Szeffler (1992-1994); Anne Walker, MPH (1998-1999).

PDS Instrumentation: Arlin Lehman, RCPT (president).

Project Office, National Heart, Lung, and Blood Institute, Bethesda, Md: Virginia Taggart, MPH (project officer); Pamela Randall (contracting officer); James Kiley, PhD; Margaret Wu,

PhD; Paul Albert, PhD (1991-1999); Suzanne Hurd, PhD (1991-1999); Sydney Parker, PhD (1991-1994).

Serum Repository, DACI Reference Laboratory, Johns Hopkins Asthma & Allergy Center, Baltimore, Md: Robert Hamilton, PhD, DABMLI (director); N. Franklin Adkinson, MD (codirector).

The University of Iowa, College of Pharmacy, Division of Pharmaceutical Services, Iowa City, Iowa: Rolland Proust, PhD (director); David Herold, RPh; Dennis Elbert, RPh.

Pharmaceutical services

ASTRA USA, Inc, Westborough, Mass

Glaxo, Inc, Research Institute, Research Triangle Park, NC

Rhone-Poulenc Rorer, Collegeville, Pa

Schering-Plough, Kenilworth, NJ

Committees

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