

Distribution of *Dermatophagoides* spp., *D. farinae* and *D. pteronyssinus*, Antigen in Homes of Patients with Asthma in Eastern Massachusetts

Paul J. Hannaway, M.D., and Carlene Roundy, M.L.T.

ABSTRACT

Regional analysis of dust mite species distribution is pertinent to clinical practice as atopic patients should be tested and, when indicated, receive appropriate immunotherapy with house dust mite antigens indigenous to their geographic locale. Surveys in selected areas of the United States have found *D. farinae* and *D. pteronyssinus* to be the two most prevalent mite species. None of these surveys encompassed the populous Northeastern corridor of the United States. This present study describes the distribution of *Dermatophagoides* spp. antigens within the homes of asthma sufferers living in suburban Massachusetts. A total of 60 dust samples were collected from carpets in the homes of 46 patients with chronic asthma and documented house dust mite sensitivity. Dust samples were obtained from bedroom carpets in 46 homes and both bedroom and family room carpets in 14 homes. *Dermatophagoides* spp. bedroom carpet levels ranged from 2.3 ug/g to 138.5 ug/g; (mean level 36.1 ug/g). *D. farinae* was the dominant species in the majority (78%) of sampled homes. *D. pteronyssinus* predominated in only three (7%) homes. Nearly 80% of surveyed homes had carpet dust mite antigen levels exceeding 10 ug/g. Mean bedroom carpet levels were more than two-fold higher than family room levels, 47.7 ug/g versus 19.5 ug/g. Distribution pat-

terns of *D. farinae* and *D. pteronyssinus* in bedroom and living room carpets was similar in 13 of 14 surveyed homes. Although *D. farinae* is clearly the dominant allergen in the surveyed homes, this study suggests that both *D. farinae* and *D. pteronyssinus* antigens should be used for diagnostic testing and, when indicated, immunotherapy in the northeastern United States. (Allergy and Asthma Proc 18:177-180, 1997)

Five house dust mite species belonging to the family Pyroglyphidae, *D. farinae*, *D. pteronyssinus*, *D. microceras*, *Euroglyphus maynei*, and *Blomia tropicalis*, are known sensitizers for asthma and allergy sufferers.¹ As the prevalence of house dust mites varies between continents, climactic zones, and indoor microclimates, analysis of the distribution of specific house dust mite species is relevant to clinical practice, as atopic patients should be skin tested and, when indicated, receive immunotherapy to house dust mite antigens indigenous to their geographic locale.

Preliminary surveys in selected areas of the United States have found *D. farinae* and *D. pteronyssinus* to be the two most prevailing mite species.²⁻⁶ A more comprehensive analysis of 252 homes in eight more humid southern and westerly regions of the United States found that the majority (91%) of sampled homes were populated by both *D. farinae* and *D. pteronyssinus*.⁷ All homes in all locations contained *Dermatophagoides* species, and few homes were infested by either *D. farinae* or *D. pteronyssinus* alone.

Presently there are little data describing the distribution of *Dermatophagoides* species in the northeastern United

From Tufts University School of Medicine, Salem, Massachusetts
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Address correspondence and reprint requests to Dr. Paul J. Hannaway, 114 Highland Avenue, Salem, MA 01970

States. This most densely populated region of the United States is an area with colder, drier winters where one might expect *D. farinae* to be more prevalent. The present study details the distribution of *D. farinae* and *D. pteronyssinus* antigens within the homes of asthma sufferers residing in eastern Massachusetts.

METHODS

A total of 60 dust samples were collected from the homes of 46 patients residing at or near sea level within a 10 mile radius of Salem, Massachusetts, a coastal suburb at latitude 42°, located 20 miles north of Boston. All patients had chronic asthma (mean duration 12 years), documented house dust mite skin sensitivity, (>5 mm wheal on prick testing to *D. farinae* or *D. pteronyssinus* antigen) and a positive Acarex (Center Laboratories, Port Washington, NY) test on vacuum bag dust samples collected by patients. Dust samples were obtained from bedroom carpets only ($n = 46$) and both bedroom and family room carpets ($n = 14$) from January to October. The majority of dust samples 48/60 (80%) were collected in October and November. As part of a larger environmental control study 29 homes were subjected to a site visit during which the following home parameters were investigated: age and type of dwelling, in-home (bedroom) temperature and relative humidity, carpet thickness, and type of heating and air conditioning system.

DUST COLLECTION AND ANALYSIS

In 17 homes, fine dust was extracted from vacuum bag samples where large dust particles were removed, and in 29 homes dust samples were collected by attaching the ALK Indoor Allergen Analysis Kit to the home vacuum and sampling a 3 mm³ area of carpet for 2 minutes. Patients were instructed to vacuum the bedroom carpet near their dressing area and family room carpet near chairs or sofas where human skin scales might be expected to be in greater abundance. All dust samples were then analyzed by specific immunoassay techniques.⁸ As 5/46 (11%) of dust samples that tested positive by the screening Acarex test had undetectable or clinically insignificant levels, <2 ug/g of *Dermatophagoides* spp., on immunoassay analysis, this report analyzes the distribution of *Dermatophagoides* spp. antigen in 41 homes.

RESULTS

Dermatophagoides antigen levels in bedroom carpets ranged from 2.3 ug/g to 138.5 ug/g with a mean level of 36.1 ug/g of house dust. *D. farinae* was the dominant species in 78% (32/41) of sampled homes, 15% (6/41) of homes were inhabited by both *D. farinae* and *D. pteronyssinus*, and *D. pteronyssinus* was dominant in only three (7%) of sampled homes (see Fig. 1). The nine homes with significant levels of *D. pteronyssinus* antigen had higher mean dust mite levels (53.6 ug/g) than those infested with

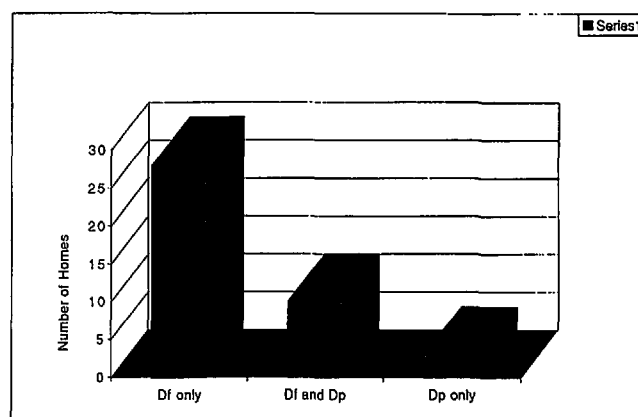


Figure 1. *Dermatophagoides* species distribution in bedroom carpets of 41 homes.

D. farinae alone where the mean level was 23.6 ug/g. Twenty-two percent (9/41) of homes had bedroom carpet dust mite levels ranging from 2 to 10 ug/g per gram of house dust, 56% (23/41) had levels between 10–50 ug/g, and in 22% (9/41) of homes dust mite levels exceeded 50 ug/g (see Fig. 2).

In the 14 homes where both the bedroom and family room carpets were sampled, bedroom carpet levels were significantly higher; range 3.1–138.9 ug/g; mean 47.7 ug/g, versus family room carpets; range 3.3–46.4 ug/g, mean 19.5 ug/g (see Fig. 3). Only one home (No. 10) with bedroom carpet dust mite infestation had nondetectable dust mite levels in the family room carpet. Two of 14 homes (Nos. 11, 14) had higher antigen levels in family room carpets. The pattern of *D. farinae* and *D. pteronyssinus* antigen distribution in bedroom and living room carpets was similar in 13 of 14 surveyed homes.

Twenty-seven homes subjected to an on-site visit were analyzed in detail. The majority of these homes were older, wooden, suburban, single family dwellings ranging in age from 2 to 102 years (mean 41 years). Temperature and humidity levels were recorded in mid to late October. In-home bedroom temperatures ranged from 68–74 °F (mean 72 °) and relative humidity ranged from 39%–56% (mean 45%). There was no correlation between levels of house dust mite infestation and indoor temperature or relative humidity, nor was there any relationship between dust mite levels and the age of the home, type of heating system (hot water versus hot air), air conditioning system (central air versus window units), or carpet thickness.

Vacuum dust samples from all homes in this survey were initially screened with the semiquantitative Acarex test. There were 5/23 (23%) false positive Acarex tests (nondetectable immunoassay or <2 ug/g levels) when the Acarex test was read as slightly positive. Immunoassay levels of dust samples with a moderate Acarex reading ranged from 15.8 ug/g to 95.1 ug/g (mean 38 ug/g) and samples with a highly positive reading ranged from 7.7 ug/g to 138.9 ug/g (mean 50 ug/g). There were no false positive Acarex tests when the Acarex reading was moderate or highly positive.

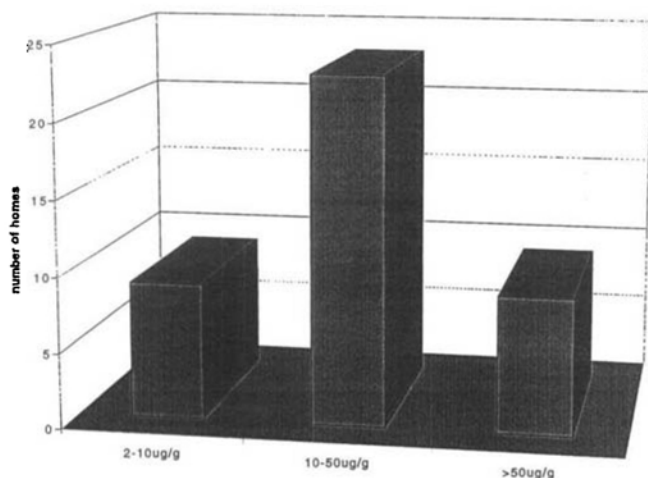


Figure 2. House dust mite antigen levels in 41 homes.

DISCUSSION

The five species of house dust mites that have identified worldwide as risk factors for allergic rhinitis and asthma include *D. farinae*, *D. pteronyssinus*, *D. microceras*, *Euroglyphus maynei*, and *Blomia Tropicalis*. The most prevalent species in England and Europe are *D. pteronyssinus* and *Euroglyphus maynei*, whereas *D. pteronyssinus* and *D. farinae* are widespread in North America.^{2,9} *E. maynei* and *Blomia Tropicalis* have been found in lesser numbers in warmer, more humid areas of the United States and South America.¹⁰

United States surveys in southern California,² Ohio,³ and southeastern Wisconsin⁴ revealed widespread distribution of *D. pteronyssinus* and *D. farinae*, with varied species prevalence and distribution between homes within geographic areas. *D. farinae* was found to be the dominant species in Kansas,¹¹ Texas,¹² and Ohio³ homes. Both *D. farinae* and *D. pteronyssinus* were prevalent in 20 homes studied in Atlanta, Georgia.¹³ An analysis of 40 homes in the Tampa Bay area found *D. farinae* and *D. pteronyssinus* to be the dominant species and *B. tropicalis* was also present in 30% of sampled Florida homes.¹⁰

A more comprehensive survey assayed mite density and species prevalence in 252 homes in eight geographic areas of the United States including Cincinnati, Ohio; Memphis, Tennessee; Greenville, North Carolina; Delray Beach, Florida; New Orleans, Louisiana; Galveston, Texas; and San Diego and Los Angeles, California.⁷ The most common species were *D. farinae*, *D. pteronyssinus*, *Euroglyphus maynei*, and *Blomia Tropicalis*. All homes in all locations contained *Dermatophagoides* spp. mites, and few homes were populated by *D. farinae* or *D. pteronyssinus* alone. *E. maynei* occurred in significant numbers (36%) of homes in New Orleans, Memphis, Galveston, Delray Beach, and San Diego. *B. tropicalis* was found in these same cities, but in smaller numbers.

As a result of this survey and previous studies, it was recommended that both *D. farinae* and *D. pteronyssinus*

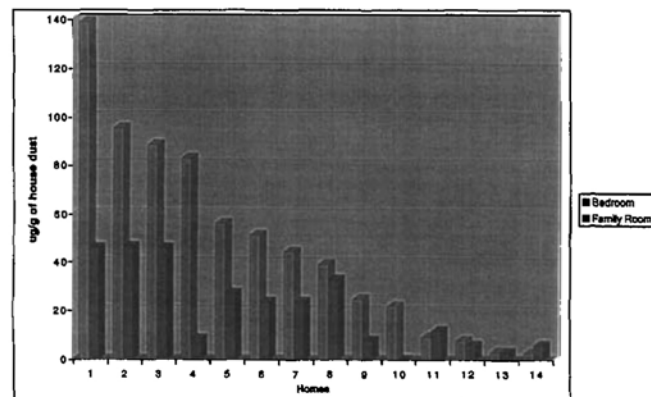


Figure 3. Dermatophagoides species distribution, bedroom versus family room.

should be used for diagnostic testing and immunotherapy. Additional studies are needed to determine the clinical significance of *B. tropicalis* and *E. maynei* species, neither of which is presently available for skin testing or immunotherapy.

Presently there is little or no data depicting the prevalence or distribution of *Dermatophagoides* spp. in the northeastern United States, an area where drier colder winters should favor *D. farinae* survival. The single New England study, which surveyed 15 homes in the rural Upper Connecticut River Valley, collected samples in June and September.¹⁴ Surprisingly *D. pteronyssinus* was the major species in 10 of these 15 homes and clearly dominated in six. Many of the homes in this study were described as being damp and heavily shaded in the summer months.

Our survey of 41 homes in a suburb of eastern Massachusetts reveals *D. farinae* to be the dominant antigen in the majority of sampled homes. While *D. pteronyssinus* was found in 20% of surveyed homes, it dominated in only 7% or 3 of 41 homes sampled. This pattern of *Dermatophagoides* spp. distribution in eastern New England is probably a reflection of colder, drier winters, creating a more suitable habitat for *D. farinae* survival.

The high level of carpet mite infestation in these 41 homes was somewhat surprising considering that the majority of dust samples were collected in October and November when mites are entering a more dormant stage of their life cycle. Nearly 80% (32/41) of sampled homes had levels >10 ug/g of house dust and in 22% (9/41) of homes house dust mite levels exceeded 50 ug/g of house dust. These high levels of mite infestation may be a reflection of infrequent use of air conditioning in the more humid summer months in coastal New England, an active period of mite proliferation. Our higher levels may also be due to sampling carpet areas where a greater abundance of skin scales might be expected, i.e., bedroom dressing areas and family room carpets adjacent to chairs or sofas.

The ratio of house dust mite antigen levels in bedroom carpeting versus family room carpets was 2.5 to 1. The higher house dust mite levels in bedroom carpets (47.7 ug/g)

versus family room carpets (19.5 ug/g) may be due to a greater abundance of food supply (skin scales) in the bedroom. Species distribution of *D. farinae* and *D. pteronyssinus* in bedroom and family room carpets was similar in 13 of 14 homes, and only 1 of 14 homes with significant dust mite levels in bedroom carpeting had no detectable levels in the family room carpet. Thus it appears that antigen analysis of bedroom carpeting reflects mite species distribution within other carpeted areas of the home (see Fig. 3).

Previous surveys in the United Kingdom and Denmark have shown a direct correlation between age of home and mite allergen levels.^{9,15} In this survey, the age of the home was not a significant factor in the level of mite infestation. The Acarex test, which unfortunately is no longer available, proved to be a useful and inexpensive screening test for finding homes with significant levels of house dust mite allergen. Whereas 5/23 (22%) of vacuum dust samples with a "slight" Acarex reading were falsely positive, all 23 homes with a "moderate to high" Acarex reading had significant levels of house dust mite allergen on specific immunoassay analysis; range 7.7 ug/g–138.9 ug/g; mean 42.2 ug/g.

In summary, analysis of the distribution of *Dermatophagoides* antigen in 41 homes in eastern Massachusetts reveals that *D. farinae* to be the dominant species in 80% of surveyed homes. Enough sampled homes (20%) contained *D. pteronyssinus* antigen to suggest that both *D. farinae* and *D. pteronyssinus* be used for diagnostic testing and immunotherapy in the northeastern United States.

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In the treatment of seasonal allergic rhinitis

CLARITIN® Offers Patients a Unique Combination of Benefits



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In studies with CLARITIN® at doses 2 to 4 times higher than the recommended dose of 10 mg, a dose-related increase in the incidence of somnolence was observed.

[†]In seasonal allergic rhinitis patients, relief began in 13% of treated patients vs 4% of placebo-treated patients within 30 minutes (P=.04). At 2 hours, 48% of patients receiving placebo experienced relief. Distribution of onset times was significantly earlier for CLARITIN® vs placebo (P=.03).

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2. These findings were based on a sales comparison of loratadine (U.S. dollars) with other leading antihistamines. *IMS International Prescription Audit.* September 1994 to present (January 1997).

Once-a-day

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10 mg (loratadine)
TABLETS

Please see next page for brief summary of Prescribing Information.

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CLARITIN® brand of loratadine TABLETS, SYRUP, and RAPIDLY-DISINTEGRATING TABLETS

BRIEF SUMMARY (For full Prescribing Information, see package insert.)

INDICATIONS AND USAGE: CLARITIN is indicated for the relief of nasal and non-nasal symptoms of seasonal allergic rhinitis and for the treatment of chronic idiopathic urticaria in patients 6 years of age or older.

CONTRAINDICATIONS: CLARITIN is contraindicated in patients who are hypersensitive to this medication or to any of its ingredients.

PRECAUTIONS: General: Patients with liver impairment or renal insufficiency (GFR < 30 mL/min) should be given a lower initial dose (10 mg every other day). (See **CLINICAL PHARMACOLOGY: Special Populations.**)

Drug Interactions: Loratadine (10 mg once daily) has been coadministered with therapeutic doses of erythromycin, cimetidine, and ketoconazole in controlled clinical pharmacology studies in adult volunteers. Although increased plasma concentrations (AUC 0-24 hrs) of loratadine and/or descarboethoxyloratadine were observed following coadministration of loratadine with each of these drugs in normal volunteers (n = 24 in each study), there were no clinically relevant changes in the safety profile of loratadine, as assessed by electrocardiographic parameters, clinical laboratory tests, vital signs, and adverse events. There were no significant effects on QT intervals, and no reports of sedation or syncope. No effects on plasma concentrations of cimetidine or ketoconazole were observed. Plasma concentrations (AUC 0-24 hrs) of erythromycin decreased 15% with coadministration of loratadine relative to that observed with erythromycin alone. The clinical relevance of this difference is unknown. These above findings are summarized in the following table:

Effects on Plasma Concentrations (AUC 0-24 hrs) of Loratadine and Descarboethoxyloratadine After 10 Days of Coadministration (Loratadine 10 mg) in Normal Volunteers

	Loratadine	Descarboethoxyloratadine
Erythromycin (500 mg Q8h)	+ 40%	+46%
Cimetidine (300 mg QID)	+103%	+ 6%
Ketoconazole (200 mg Q12h)	+307%	+73%

There does not appear to be an increase in adverse events in subjects who received oral contraceptives and loratadine.

Carcinogenesis, Mutagenesis, and Impairment of Fertility: In an 18-month carcinogenicity study in mice and a 2-year study in rats, loratadine was administered in the diet at doses up to 40 mg/kg (mice) and 25 mg/kg (rats). In the carcinogenicity studies, pharmacokinetic assessments were carried out to determine animal exposure to the drug. AUC data demonstrated that the exposure of mice given 40 mg/kg of loratadine was 3.6 (loratadine) and 18 (descarboethoxyloratadine) times higher than in humans given the maximum recommended daily oral dose. Exposure of rats given 25 mg/kg of loratadine was 28 (loratadine) and 67 (descarboethoxyloratadine) times higher than in humans given the maximum recommended daily oral dose. Male mice given 40 mg/kg had a significantly higher incidence of hepatocellular tumors (combined adenomas and carcinomas) than concurrent controls. In rats, a significantly higher incidence of hepatocellular tumors (combined adenomas and carcinomas) was observed in males given 10 mg/kg and males and females given 25 mg/kg. The clinical significance of these findings during long-term use of CLARITIN is not known.

In mutagenicity studies, there was no evidence of mutagenic potential in reverse (Ames) or forward point mutation (CHO-HGPRT) assays, or in the assay for DNA damage (rat primary hepatocyte unscheduled DNA assay) or in two assays for chromosomal aberrations (human peripheral blood lymphocyte clastogenesis assay and the mouse bone marrow erythrocyte micronucleus assay). In the mouse lymphoma assay, a positive finding occurred in the nonactivated but not the activated phase of the study.

Decreased fertility in male rats, shown by lower female conception rates, occurred at an oral dose of 64 mg/kg (approximately 50 times the maximum recommended human daily oral dose on a mg/m² basis) and was reversible with cessation of dosing. Loratadine had no effect on male or female fertility or reproduction in the rat at an oral dose of approximately 24 mg/kg (approximately 20 times the maximum recommended human daily oral dose on a mg/m² basis).

Pregnancy Category B: There was no evidence of animal teratogenicity in studies performed in rats and rabbits at oral doses up to 96 mg/kg (approximately 75 times and 150 times, respectively, the maximum recommended human daily oral dose on a mg/m² basis). There are, however, no adequate and well-controlled studies in pregnant women. Because animal reproduction studies are not always predictive of human response, CLARITIN should be used during pregnancy only if clearly needed.

Nursing Mothers: Loratadine and its metabolite, descarboethoxyloratadine, pass easily into breast milk and achieve concentrations that are equivalent to plasma levels with an AUC_{milk}/AUC_{plasma} ratio of 1.17 and 0.85 for loratadine and descarboethoxyloratadine, respectively. Following a single oral dose of 40 mg, a small amount of loratadine and descarboethoxyloratadine was excreted into the breast milk (approximately 0.03% of 40 mg over 48 hours). A decision should be made whether to discontinue nursing or to discontinue the drug, taking into account the importance of the drug to the mother. Caution should be exercised when CLARITIN is administered to a nursing woman.

Pediatric Use: The safety of CLARITIN Syrup at a daily dose of 10 mg has been demonstrated in 188 pediatric patients 6-12 years of age in placebo-controlled 2-week trials. The effectiveness of CLARITIN for the treatment of seasonal allergic rhinitis and chronic idiopathic urticaria in this pediatric age group is based on an extrapolation of the demonstrated efficacy of CLARITIN in adults in these conditions and the likelihood that the disease course, pathophysiology, and the drug's effect are substantially similar to that of the adults. The recommended dose for the pediatric population is based on cross-study comparison of the pharmacokinetics of CLARITIN in adults and pediatric subjects and on the safety profile of loratadine in both adults and pediatric patients at doses equal to or higher than the recommended doses. The safety and effectiveness of CLARITIN in pediatric patients under 6 years of age have not been established.

ADVERSE REACTIONS: CLARITIN Tablets: Approximately 90,000 patients, aged 12 and older, received CLARITIN Tablets 10 mg once daily in controlled and uncontrolled studies. Placebo-controlled clinical trials at the recommended dose of 10 mg once a day varied from 2 weeks' to 6 months' duration. The rate of premature withdrawal from these trials was approximately 2% in both the treated and placebo groups.

REPORTED ADVERSE EVENTS WITH AN INCIDENCE OF MORE THAN 2% IN PLACEBO-CONTROLLED ALLERGIC RHINITIS CLINICAL TRIALS IN PATIENTS 12 YEARS OF AGE AND OLDER

	LORATADINE 10 mg QD n = 1926	PLACEBO n = 2545	CLEMASTINE 1 mg BID n = 536	TERFENADINE 60 mg BID n = 684
Headache	12	11	8	8
Somnolence	8	6	22	9
Fatigue	4	3	10	2
Dry Mouth	3	2	4	3

Adverse events reported in placebo-controlled chronic idiopathic urticaria trials were similar to those reported in allergic rhinitis studies.

Adverse event rates did not appear to differ significantly based on age, sex, or race, although the number of nonwhite subjects was relatively small.

CLARITIN REDITABS (loratadine rapidly-disintegrating tablets): Approximately 500 patients received CLARITIN REDITABS (loratadine rapidly-disintegrating tablets) in controlled clinical trials of 2 weeks' duration. In these studies, adverse events were similar in type and frequency to those seen with CLARITIN Tablets and placebo.

Administration of CLARITIN REDITABS (loratadine rapidly-disintegrating tablets) did not result in an increased reporting frequency of mouth or tongue irritation.

CLARITIN Syrup: Approximately 300 pediatric patients 6 to 12 years of age received 10 mg loratadine once daily in controlled clinical trials for a period of 8-15 days. Among these, 188 children were treated with 10 mg loratadine syrup once daily in placebo-controlled trials. Adverse events in these pediatric patients were observed to occur with type and frequency similar to those seen in the adult population. The rate of premature discontinuation due to adverse events among pediatric patients receiving loratadine 10 mg daily was less than 1%.

ADVERSE EVENTS OCCURRING WITH A FREQUENCY OF ≥ 2% IN LORATADINE SYRUP-TREATED PATIENTS (6-12 YEARS OLD) IN PLACEBO-CONTROLLED TRIALS, AND MORE FREQUENTLY THAN IN THE PLACEBO GROUP

	LORATADINE 10 mg QD n = 188	PLACEBO n = 262	CHLORPHENIRAMINE 2-4 mg BID/TID n = 170
Nervousness	4	2	2
Wheezing	4	2	5
Fatigue	3	2	5
Hyperkinesia	3	1	1
Abdominal Pain	2	0	0
Conjunctivitis	2	<1	1
Dysphonia	2	<1	0
Malaise	2	0	1
Upper Respiratory Tract Infection	2	<1	0

In addition to those adverse events reported above (≥ 2%), the following adverse events have been reported in at least one patient in CLARITIN clinical trials in adult and pediatric patients:

Autonomic Nervous System: Altered lacrimation, altered salivation, flushing, hyposthesia, impotence, increased sweating, thirst.

Body As A Whole: Angioneurotic edema, asthenia, back pain, blurred vision, chest pain, earache, eye pain, fever, leg cramps, malaise, rigors, tinnitus, viral infection, weight gain.

Cardiovascular System: Hypertension, hypotension, palpitations, supraventricular tachyarrhythmias, syncope, tachycardia.

Central and Peripheral Nervous System: Blepharospasm, dizziness, dysphonia, hyper-tonia, migraine, paresthesia, tremor, vertigo.

Gastrointestinal System: Altered taste, anorexia, constipation, diarrhea, dyspepsia, flatulence, gastritis, hiccup, increased appetite, nausea, stomatitis, toothache, vomiting.

Musculoskeletal System: Arthralgia, myalgia.

Psychiatric: Agitation, amnesia, anxiety, confusion, decreased libido, depression, impaired concentration, insomnia, irritability, paranoia.

Reproductive System: Breast pain, dysmenorrhea, menorrhagia, vaginitis.

Respiratory System: Bronchitis, bronchospasm, coughing, dyspnea, epistaxis, hemoptysis, laryngitis, nasal dryness, pharyngitis, sinusitis, sneezing.

Skin and Appendages: Dermatitis, dry hair, dry skin, photosensitivity reaction, pruritus, purpura, rash, urticaria.

Urinary System: Altered micturition, urinary discoloration, urinary incontinence, urinary retention.

In addition, the following spontaneous adverse events have been reported rarely during the marketing of loratadine: abnormal hepatic function, including jaundice, hepatitis, and hepatic necrosis; alopecia; anaphylaxis; breast enlargement; erythema multiforme; peripheral edema; and seizures.

OVERDOSAGE: In adults, somnolence, tachycardia, and headache have been reported with overdoses greater than 10 mg with the Tablet formulation (40 to 180 mg). Extrapyramidal signs and palpitations have been reported in children with overdoses of greater than 10 mg of CLARITIN Syrup. In the event of overdosage, general symptomatic and supportive measures should be instituted promptly and maintained for as long as necessary.

Treatment of overdosage would reasonably consist of emesis (ipecac syrup), except in patients with impaired consciousness, followed by the administration of activated charcoal to absorb any remaining drug. If vomiting is unsuccessful, or contraindicated, gastric lavage should be performed with normal saline. Saline cathartics may also be of value for rapid dilution of bowel contents. Loratadine is not eliminated by hemodialysis. It is not known if loratadine is eliminated by peritoneal dialysis.

No deaths occurred at oral doses up to 5000 mg/kg in rats and mice (greater than 2400 and 1200 times, respectively, the maximum recommended human daily oral dose on a mg/m² basis). Single oral doses of loratadine showed no effects in rats, mice, and monkeys at doses as high as 10 times the maximum recommended human daily oral dose on a mg/m² basis.

Schering

Schering Corporation
Kenilworth, NJ 07033 USA

CLARITIN REDITABS (loratadine rapidly-disintegrating tablets) are manufactured for Schering Corporation by Scher DDS, England.

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