

Asthma caused by live fish bait

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Abstract

TOP

Background: Larvae of insects and worms are commonly used as live fish bait (LFB) by anglers. Asthma, rhinoconjunctivitis, and urticaria related to various kinds of LFB have been reported.

Methods: We studied 14 subjects with respiratory symptoms associated with exposure to LFB. Skin prick tests and RASTs with *Lucilia caesar*, *Galleria mellonella*, and *Tenebrio molitor* extracts were carried out in all subjects. Monitoring of peak expiratory flow rate and evaluation of bronchial responsiveness to methacholine before and after exposure to LFB were performed in seven subjects.

Results: Thirteen subjects had asthma, all 14 had rhinoconjunctivitis, and three had contact urticaria. Eleven subjects had respiratory symptoms after fishing with LFB, and the other three subjects had symptoms during their work in a commercial fish bait farm. Positive skin prick test results or significant RAST binding to *L. caesar* extract were observed in 13 subjects, to *G. mellonella* extract in four subjects, and to *T. molitor* extract in three subjects. After exposure to LFB, two subjects had an early asthmatic response, three had a late asthmatic response, and two had no asthmatic response. Late asthmatic response was associated with a long-lasting increase in bronchial responsiveness.

Conclusions: This study demonstrates that emanations from LFB are sensitizers, which have the potential to elicit IgE-mediated asthma. Exposure to LFB is common and LFB

should be considered as a possible sensitizing agent for asthma. (J ALLERGY CLIN IMMUNOL 1994;93:424-30.)

EAR	Early asthmatic response
FEV₁	Forced expiratory volume in 1 second
GM	<i>Galleria Mellonella</i>
LAR	Late asthmatic response
LC	<i>Lucilia caesar</i>
LFB	Live fish bait
PD₂₀M	Methacholine provocative dose needed to decrease FEV ₁ by 20%
PEFR	Peak expiratory flow rate
SPT	Skin prick test
TM	<i>Tenebrio molitor</i>

Key words: [Live fish bait](#), [late asthmatic reaction](#), [occupational asthma](#)

The larvae of flies and moths, as well as worms, are commonly used as bait for angling. There have been several reports of asthma, rhinoconjunctivitis, and urticaria related to fish bait in anglers¹⁻⁶ or in occupationally exposed workers.⁷⁻¹⁰ Asthma and rhinitis are associated with exposure to larvae such as bee moth (*Galleria mellonella* [GM])⁷⁻⁹ mealworm (*Tenebrio molitor* [TM])^{8, 10} housefly (*Musca domestica*),² bluebottle (*Calliphora vomitoria*),³ greenbottle (*Lucilia caesar* [LC]),⁴ *Calliphora erythrocephala*,⁶ or worms such as *Marphysa sanguinea*¹ and *Eisenia foetida*.⁵ We have previously described five cases of asthma and rhinoconjunctivitis caused by LC larvae.¹¹ In this article we have extended the clinical and immunologic study to 14 cases and have evaluated two other cases of allergy related to exposure to larvae of bee moth and mealworm.

METHODS

Subjects

We studied 14 subjects with respiratory symptoms associated with exposure to live fish bait (LFB). Three were employed in commercial fish bait establishments and had work-related symptoms, and 11 were anglers and had symptoms when fishing with LFB (■Table I). Three were current smokers and five were former smokers. The three occupationally exposed workers were older, had lower forced expiratory volume in 1 second (FEV₁) and higher hyperresponsiveness to methacholine than the anglers. Duration of exposure to

LFB was 15.4 years in fish hobbyists and 9.3 years in those who were occupationally exposed. Exposure to *LC*, *GM*, and *TM* was estimated according to a 4-point scale, as follows: 0 = no exposure; 1 = infrequent exposure (less than 1 time per month); 2 = moderate exposure (1 to 4 times per month); 3 = frequent exposure (2 to 5 times per week) (■Table II). Duration of LFB-induced symptoms was 6.3 years in anglers and 2.3 years in occupationally exposed workers. LFB-induced symptoms resulted in asthma in all but one subject, rhinoconjunctivitis in all subjects, and contact urticaria in three.

TABLE I. Characteristics of the subjects studied

Subject No.	Age (yr)	Duration of exposure LFB (yr)	Duration of to LFB-related symptoms (yr)	FEV ₁ (% pred)	PD ₂₀ M* (µg)	LFB-induced symptoms
Anglers						
1	27	20	19	112.8	3500	Asthma, RC
2	27	10	9	121.8	ND	Asthma, RC, urticaria
3	35	10	6	100.2	1394	Asthma, RC
4	18	4	2	82.5	127	Asthma, RC
5	29	19	3	87.3	3500	Asthma, RC
6	17	6	2	120.9	3500	RC
7	36	25	4	113.3	1438	Asthma, RC, urticaria
8	33	26	1	95.0	3500	Asthma, RC
9	30	15	1	134.9	3500	Asthma, RC
10	30	15	4	115.6	250	Asthma, RC, pruritus
11	35	19	18	114.0	3500	Asthma, RC, urticaria
Mean (SEM)	28.8 (1.9)	15.4 (2.2)	6.3 (2.0)	108.9 (4.8)	1610 (1)	
Occupationally exposed						
12	24	4	4	116.0	81	Asthma, RC
13	50	14	2	77.2	52	Asthma, RC
14	43	10	1	69.8	46	Asthma, RC
Mean (SEM)	39.0 (7.8)	9.3 (2.9)	2.3 (0.9)	87.7 (14.3)	58 (1)	

RC, Rhinoconjunctivitis; ND, not done.

*Geometric mean (geometric SEM)

TABLE II. Immunologic data

<u>Intensity of exposure to LFB*</u>	<u>Skin test result to LFB</u>	<u>RAST to LFB (% binding)</u>
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Subject No.	Family history of atopy	Personal history of atopy	Skin test results to common allergens	Other									
				LC	GM	TM	LFB	LC	GM	TM	LC	GM	TM
Anglers													
1	Yes	No	Neg	2	1	0	2	.02	Neg	Neg	37.2	3.2	2.3
2	Yes	No	Grass + +	2	0	2	2	ND	ND	ND	11.1	1.1	ND
3	Doubtful	No	Neg	3	0	2	2	0.02	Neg	Neg	20.3	3.3	1.2
4	Yes	Yes	Grass + +	3	2	2	2	0.05	Neg	0.1	41.5	4.7	21.3
5	No	No	Neg	2	2	2	2	Neg	Neg	0.01	4.5	3.6	17.1
6	Yes	No	Grass + + +, cypress + + +	2	1	1	1	0.01	Neg	Neg	43.8	12.1	4.0
7	Doubtful	Yes	Grass + + +	3	3	3	1	0.02	Neg	Neg	17.0	1.0	1.3
8	Doubtful	No	Neg	3	1	1	0	0.01	Neg	Neg	27.5	1.3	1.1
9	No	No	Neg	2	0	0	0	0.02	Neg	Neg	27.7	1.0	1.0
10	No	Yes	<i>Parietaria</i> + + +	2	0	0	0	0.01	0.01	Neg	10.0	9.3	1.5
11	Yes	No	Cat + + +, Dog + + +	2	0	0	0	0.01	Neg	Neg	ND	ND	ND
Mean (SEM)											24.1 (4.4)	4.1 (1.2)	5.6 (2.6)
Occupationally exposed													
12	No	Yes	Grass + + House dust mite + + +	3	3	3	1	0.01	Neg	Neg	40.0	4.0	1.3
13	No	No	Cockroach + +	3	3	3	3	0.01	0.02	0.01	41.3	7.8	37.8
14	No	Yes	Storage mites +	3	3	3	3	0.01	Neg	Neg	23.1	11.1	4.1
Mean (SEM)											34.8 (5.9)	7.6 (2.1)	14.4 (11.7)

Neg, Negative; ND, not done.

*Exposure to LFB was estimated as follows: 0 = no exposure; 1 = infrequent exposure (< 1 time/month); 2 = moderate exposure (1 to 4 times/month); 3 = frequent exposure (2 to 5 times/week)

LFB extracts

One hundred grams of LC, GM and TM were frozen at -20° C for 48 hours. LFB bodies were washed with phosphate-buffered saline at a concentration of 1:10 wt/vol. The washing fluid of LC extract was collected, filtered (Millex Millipore filters, 0.22 µm; Millipore Corp., Bedford, Mass.) and dispensed in vials of 3 ml. The washed bodies of LC, GM, and TM were homogenized with three-blade Politron (Kinematica GmbH, Kriens, Switzerland), then extracted in phosphate-buffered saline for 6 hours at room temperature. The extracts obtained were filtered in series with 0.8, 0.45, and 0.22 µm filters and dispensed in vials. About half of the obtained extract was dialyzed in Pierce tubes (Kinematica GmbH) versus

phosphate-buffered saline for 48 hours at room temperature. The total protein content of the extracts was 4.00 mg/ml for *LC*, 9.00 mg/ml for *GM*, and 3.85 mg/ml for *TM*.

Immunologic studies

Skin prick tests (SPTs) with common allergens were carried out. LFB extracts had been used both for SPTs and RASTs. Serial dilutions of 1:10, 1:20, 1:50, and 1:100 were prepared for SPTs. Results of SPTs were read after 20 minutes.

Specific IgE to *LC*, *GM*, and *TM* extracts were measured by the direct RAST with methylcellulose discs previously activated by cyanogen bromide coupled with the same extracts (*LC*, *GM*, and *TM*) used for SPTs.¹² Results were expressed as a percentage of binding, calculated by comparing the counts per minute of the sample with the total added iodine 125-labeled anti-IgE. *LC* extract binding specificity was evaluated from a serum pool with known IgE activity to house dust mites, *Parietaria* species, and milk. The threshold of 5% binding was considered the positive lower limit.

The RAST inhibition assay was performed with the three extracts in a serum pool from subjects with positive RAST results to *LC*, *GM*, or *TM* extracts. This was accomplished by preparing a series of dilutions of *LC*, *GM*, and *TM* allergen extracts. The dilutions of each extract were tested against the other two allergens, bound to a cellulose disc. In this way a competitive system was created between two different allergen extracts towards the serum antibody of the serum. The proportion of IgE linked to the allergen extract on the disc, measured by a system ¹²⁵I-anti-IgE gamma counter, provides the required indication regarding the ability of the second allergen extract (soluble) to inhibit the linkage IgE-antigen on the disc. We repeated this procedure for the three extracts. Inhibition percentage was calculated as follows:

$$\% \text{Inhibition} = 1 - \frac{(\text{cpm bound in the presence of inhibitor} - \text{cpm bound nonspecifically})}{(\text{cpm bound in the absence of inhibitor} - \text{cpm bound nonspecifically})} \times 100$$

SPT and RAST results to *LC*, *GM*, and *TM* extracts were negative in a group of nine healthy control subjects.

Monitoring of peak expiratory flow rate

Seven subjects agreed to take part in monitoring of peak expiratory flow rate (PEFR) before and after exposure to LFB. PEFR was recorded every hour from 6:00 AM to 10:00 PM the day before (control day) and the day of exposure to LFB. Spirometry and methacholine challenge tests were done before and 24 hours after first exposure to LFB. A definite asthmatic response was considered to be a fall in PEFR of at least 20% of the subject's preexposure baseline value on the exposure day. An early asthmatic response (EAR) was defined as a fall in PEFR that developed within 1 hour after first exposure to LFB, and a late asthmatic response (LAR) was defined as a fall in PEFR that started 2 hours or more after first exposure.

Methacholine challenge test

A breath-activated dosimeter (ME.FAR, Brescia, Italy) was used. Double cumulative doses of methacholine were administered, beginning with a dose of 25 µg (25, 50, 100, 200, 400, 800, 1600, and 3200 µg).¹³ We considered as baseline FEV₁ that recorded after phosphate buffer (control reagent) inhalation. FEV₁ was recorded 1 minute later and was followed by the next dose, respectively. The challenge sequence terminated when FEV₁ had decreased by 20% or more or when the maximum cumulative dose (3.2 mg) had been given. The methacholine provocation dose necessary to decrease FEV₁ by 20% (PD₂₀M), was obtained from the dose-response curve plotted on a semilogarithmic scale; the abscissa represents logarithmic methacholine doses, and the ordinate represents the percentage decrease in FEV₁. The challenge was performed with buffered lyophilized methacholine (phosphate buffer) (Lofarma, Milano, Italy); 0.2% and 2% methacholine solutions were prepared by diluting methacholine in distilled water. The dosimeter delivered methacholine from an MB2 ampule (ME.FAR) by means of an air compressor (driving pressure = 1.5 kg/cm²).¹³ Every inhalation delivered 25 to 250 µg methacholine, from 0.2% and 2% concentrations, respectively.

RESULTS

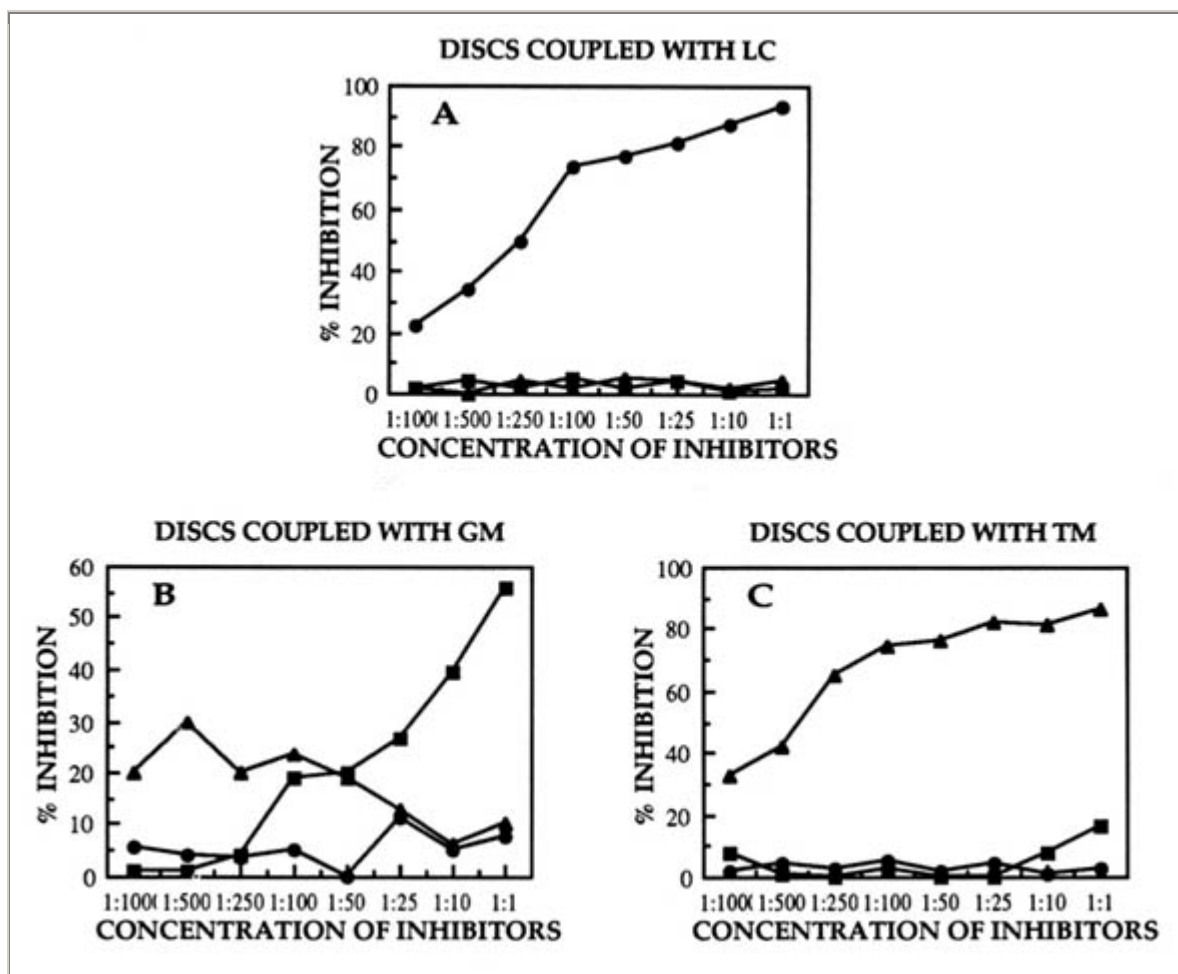
Five subjects had a positive family history of atopy, five had a personal history of atopy, and eight had positive SPT results to common allergens (■Table II). Subjects 1 and 11 are brothers.

In all subjects, exposure to *LC* was moderate to frequent. Five subjects had no exposure to *GM*, and four subjects were not exposed to *TM* or other LFB. Twelve of 13 subjects exhibited positive SPT results to *LC* extracts. In subject 2, SPT was not performed. SPT results to *LC*, *GM*, and *TM* extracts were negative in nine normal control subjects. Two subjects had positive SPT results to *GM* and three had positive results to *TM*.

Significant RAST binding to *LC*, ranging from 10.0% to 43.8%, was demonstrated in 12 of 13 subjects. In subject 11 RAST was not performed. Six subjects also had significant RAST binding, three to *GM* and one to *TM*.

The inhibitory capacities of different concentrations of *LC*, *GM*, and *TM* extracts on solid-phase *LC*, *GM*, and *TM* extracts are illustrated in ■Fig. 1. When *LC* and *TM* were coupled to the discs, almost complete inhibition was obtained with the homologous allergen at the highest concentration used (1:1 dilution) (■Fig. 1, A and C). When *GM* was used in the solid phase, only partial inhibition (56.1%) was obtained with the homologous allergen at the highest concentration used (■Fig. 1, B). There was neither inhibition from *GM* and *TM* on *LC* nor from *LC* and *TM* on *GM* nor from *LC* and *GM* on *TM*.

FIG. 1. RAST inhibition. The results are illustrated in the three panels with the three extracts as the solid-phase antigen. **A**, *LC*; **B**, *GM*; **C**, *TM*. Inhibitors: (solid circles) *LC*; (solid squares) *GM*; (solid triangles) *TM*. Protein concentration of 1:1 wt/vol: *LC*, 4.00 mg/ml; *GM*, 9.00 mg/ml; *TM*, 3.85 mg/ml.



Binding specificity was also evaluated by testing a serum pool of subjects with positive RAST results to *Dermatophagoides* and *Parietaria* species and milk with LC-coated methylcellulose disks. There was no significant inhibition of ^{125}I -labeled anti-IgE binding to LC-coated methylcellulose discs by the above-mentioned serum pool.

Monitoring of PEFR was carried out in seven subjects, and FEV₁ measurements were performed before and 24 hours after LBF exposure (Table III). After exposure to LFB, two subjects (subjects 13 and 14) had an EAR, three (subjects 1, 3, and 5) had an LAR, and two (subjects 10 and 12) had no asthmatic response. PEFR was used to document EAR and LAR. Of the two subjects with EAR, both of whom were occupationally exposed, one (subject 14) had a short-lasting reaction (less than 1 hour) and the other had a long-lasting reaction (several hours). In subject 1, LAR started 7 hours after exposure to LC and lasted 4 hours. In subject 3, LAR started 12 hours after exposure to LC, lasted 12 hours, and was associated with recurrent nocturnal asthma (for 3 nights) and an increase in bronchial responsiveness, which lasted for more than 2 months (Fig. 2). In subject 5, LAR started 16 hours after exposure to GM and TM and was associated with recurrent nocturnal asthma (for 3 nights) and an increase in bronchial responsiveness, which lasted almost 1 month. Subject 10 had an equivocal LAR (FEV₁ decreased 13.4%) associated with an increase in bronchial responsiveness (PD₂₀M was 250 µg before exposure and 36 µg 24 hours after exposure to LC).

TABLE III. Effect of exposure to LFB on PEFR and bronchial reactivity to inhaled methacholine

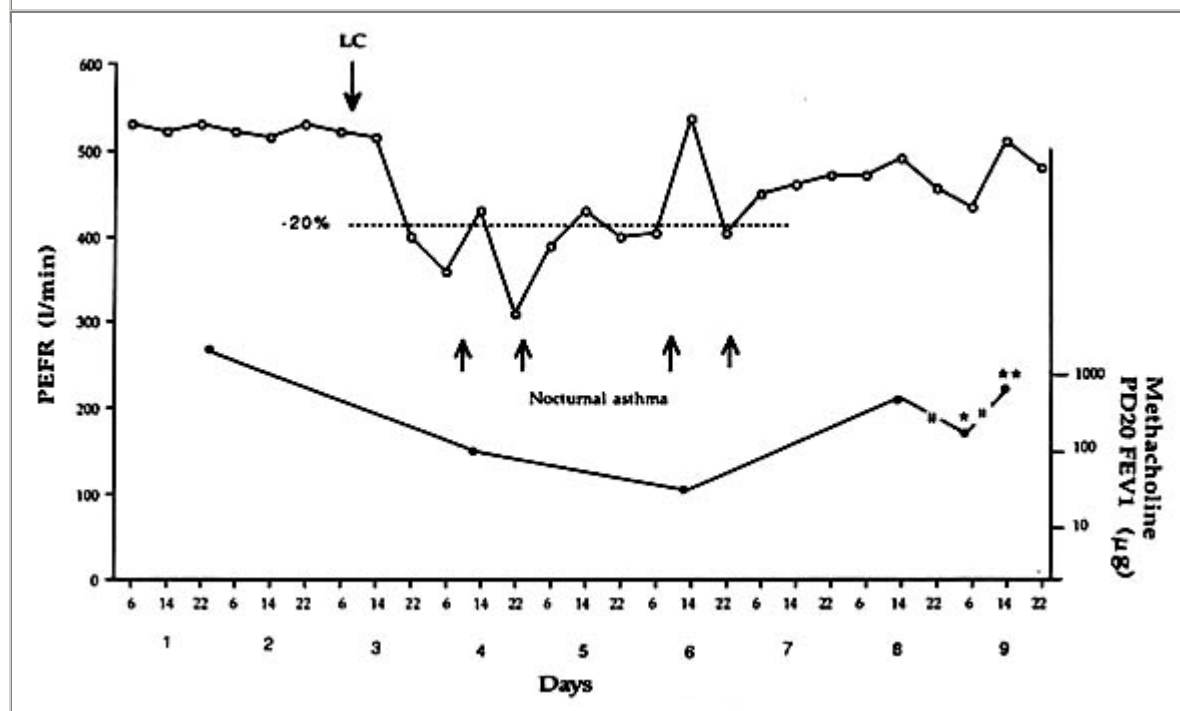
Subject No.	Type of exposure	Duration of exposure (hr)f	Baseline PEFR* (L/min)	<u>Maximum fall in PEFR (%)</u>		<u>FEV₁ (% pred)</u>		<u>PD₂₀M (mg)</u>	
				EAR	LAR	Before†	After†	Before	After
Anglers									
1	LC	5.0	515	-1.0	-24.3	106.4	ND	>3.200	ND
3	LC	2.2	520	-5.8	-34.6	100.2	77.1	1.394	0.090
5	GM, TM	3.2	425	0	-22.4	87.3	80.7	>3.200	0.036
10	LC	7.0	635	0	-13.4	112.2	105.7	0.250	0.036
Occupationally exposed									
12	LC	8.0	505	-1.0	-6.9	99.8	102.0	1.296	2.956
13	LC	1.0	410	-26.8	-41.5	109.8	83.2	0.052	0.016
14	LC	1.0	480	-21.9	+1.0	69.8	94.6	0.046	0.825

ND, Not done.

*Preexposure PEFR value on the exposure day.

†Before and 24 hours after exposure to LFB

FIG. 2. Late asthmatic reaction after exposure to larvae of greenbottle (LC) associated with recurrent nocturnal asthma and increase in bronchial responsiveness. (open circles) PEFR; (solid circles) PD₂₀FEV₁. *PD₂₀FEV₁ 25 days after exposure. **PD₂₀FEV₁ 67 days after exposure.



DISCUSSION

Three centuries ago Bernardino Ramazzini¹⁴ described respiratory symptoms caused by exposure to insects. He wrote that "silkworm as many other animals of the same order such as caterpillars have some injurious material and a corrosive matter, in particular for lungs." More recent studies have identified subjects with asthma, rhinoconjunctivitis, and urticaria related to larvae or worms used as LFB. Exposure may occur during angling¹⁻⁶ or in handling of LFB.⁷⁻¹⁰

In the present study *LC* was probably responsible for symptoms in 13 of 14 patients. In one subject (patient 5), *TM* seemed more significant than *LC* in causing asthma and rhinoconjunctivitis. According to ■Table II, *LC* seems to be more commonly used among Italian anglers than *GM* and *TM*, which tend to be used more often in the United States.⁷⁻¹⁰ The total protein content is not very different among the three extracts, but *LC* may be a more potent sensitizer than *GM* and *TM*.

Asthmatic reactions to LFB may be early,^{4, 8} late,^{1, 3, 6} or dual.⁷ We have presented three cases of late asthmatic reaction after exposure to *LC* emanations during angling. In LAR, described by both Stockley et al.³ and by ourselves, a short immediate reaction might escape hourly monitoring of PEFR. In fact, isolated LARs are very common in occupational asthma because of low molecular weight compounds¹⁵ but are rarely described in allergic asthma.^{16, 17}

In our study a positive family history of atopy in five subjects, a personal history of atopy in five subjects, and positive skin test results to common allergens in eight subjects highlight the important role of atopy for the development of LFB hypersensitivity. In one subject (patient 2) seasonal rhinoconjunctivitis caused by grass developed 8 years after the onset of *LC*-induced asthma. This is in agreement with the results of other studies.^{2, 3, 8}

Gold et al.⁹ described a subject who had independent sensitizations to two insects, caused by occupational exposures at different times. We also found multiple independent sensitization to LFB in five subjects, two of whom had been occupationally exposed (■Table II).

In subjects with respiratory symptoms caused by exposure to insect larvae, IgE-mediated hypersensitivity to unknown antigens of larvae have been demonstrated in many studies.^{1-10, 18} We have also confirmed, by means of SPTs and RASTs, that symptoms caused by LFB exposure were associated with specific IgE to *LC*, *GM*, and *TM* extracts in serum and skin. There was a good correspondence between SPT and RAST responses to *LC*, *GM*, and *TM* extracts (■Table II). RAST inhibition demonstrated specificity of IgE binding to *LC*, *GM*, and *TM* antigens and no cross-reactivity with *Dermatophagoides* and *Parietaria* species and milk antigens or within the three larvae extracts (*LC*, *GM*, and *TM*). These observations are in agreement with those of other studies, which have also demonstrated a lack of cross-reactivity between orders of insects.⁸

In conclusion, our results suggest that asthma caused by LFB is common both in anglers and occupationally exposed workers. *LC* extract seems to include a particularly potent and specific antigen that elicits IgE-mediated asthma.

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Allergic rhinoconjunctivitis caused by *Harmonia axyridis* (Asian lady beetle, Japanese lady beetle, or lady bug)

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