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Bites and stings

Fatal anaphylaxis following jack jumper ant sting in southern Tasmania

The "jack jumper" ant (*Myrmecia pilosula*) is a major cause of anaphylaxis in Tasmania. We describe four deaths attributed to stings by this ant between 1980 and 1999. All victims were men aged 40 years or over with significant comorbidities; two were taking angiotensin-converting enzyme inhibitors, which may increase risk of severe anaphylaxis. Three victims had known ant-sting allergy, but only one carried adrenaline, which he did not use. Another believed he was protected by previous attempts at hyposensitisation with whole ant-body extract. There is potential to prevent deaths by careful education of people with known allergy, prescribing of adrenaline for auto-injection and development of an effective hyposensitisation therapy.

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MJA 2001; 175: 644-647

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Of 89 described species of bull ants (genus *Myrmecia*), 88 are found only in Australia.¹ Stings are frequent, and allergy is common, especially to *Myrmecia pilosula* in Tasmania,² rural Victoria,³ the Adelaide Hills of South Australia and southern New South Wales. Peptide allergens thought to account for most *M. pilosula* IgE-specific reactions have been identified, cloned and named Myr p I,⁴⁻⁹ II and III.

Stings by *M. pilosula* caused 21%-25% of the 324 cases of anaphylaxis treated with adrenaline in the Royal Hobart Hospital Emergency Department between 1990 and 1998, compared with 13% caused by honeybee stings (Brown, unpublished data). *M. pilosula* is well known to local people, and recognisable by its size and coloration ([Box 1A](#)). It moves in short jerks and jumps, leading to the names "jumper ant", "hopper ant", "jumping jack", and "jack jumper". Nests are defended aggressively and range from a single hole to large mounds a meter in diameter with multiple entrances, typically surrounded by a scattering of fine gravel. Another less common but well-recognised local bull ant is the "inchman" ([Box 1B](#)). Our field trips in Tasmania have revealed this ant to comprise one species, identified as *Myrmecia forficata* by CSIRO (the Commonwealth Scientific and Industrial Research Organisation) Entomology, Canberra. Other species are seldom encountered and unlikely to be a common cause of stings.

We present clinical details of four deaths attributed to *M. pilosula* stings between 1980 and 1999 in southern Tasmania (population, 223 000¹⁰). Cases were identified from a manual search of diagnoses in the hospital's forensic register. Immunological methods are shown in [Box 2](#). The study was approved by the Royal Hobart Hospital Ethics Committee.

Clinical records

Patient 1

In 1989, a 49-year-old man woodcutting in bush told his companions he had been stung by a jack jumper ant. Because of known allergy to these stings, he took two antihistamine tablets. He was left alone for 15-20 minutes and was dead when his companions returned.

Past history included hypertension treated with enalapril. Autopsy revealed cardiomegaly, acute on chronic pulmonary congestion and "unusually fluid blood". The pathologist discounted ant-sting anaphylaxis as "exceedingly rare". Tissue from a suspected forearm bite was negative for snake venoms (Dr Struan Sutherland, Medical Consultant, Commonwealth Serum Laboratories, Melbourne, VIC, personal communication). Tests for serum tryptase and venom-specific IgE were not available in Australia at that time.

Patient 2

In 1995, a 62-year-old man was thought to have been stung by a jack jumper ant while fishing. He subsequently developed tongue and lip swelling and breathlessness. When a paramedic arrived 20-30 minutes later, he was in cardiac arrest. Resuscitation was unsuccessful.

Past history and allergies were not recorded. Autopsy revealed severe oedema of lips and tongue, oedema of the upper airways, extreme lung congestion, diffuse severe atherosclerosis with occlusions of 50%-75%, and marked hypertensive left ventricular hypertrophy. Toxicological screening revealed a blood alcohol level of 0.21 g/100 mL and no other drugs or substances. Serum tryptase level in blood taken three days after death was 2.73 µg/L (reference range [RR], < 2 µg/L). IgE specific to *M. pilosula* venom was detected by IgE antibody-binding studies (uptake of radioactive label, 15.2%). Reactivity to other venoms was not tested.

Patient 3

In 1995, a 40-year-old man reported being stung by a "bull ant" (a term commonly used by local people, including some medical practitioners, when referring to *M. pilosula*) outside his home in a beachside suburb of Hobart. He injected himself with promethazine (50 mg) and continued to drink alcohol. Some time later, he was noted to be slurring his words and to fall over when going to the bedroom. The precise timeframe was difficult to determine from available records. He was found dead in bed in the early hours of the morning.

Past history included obstructive sleep apnoea and progressively worsening systemic allergic reactions to "bull ants", for which he was prescribed intramuscular antihistamine and an adrenaline puffer. The medical records did not mention whether "bull ant" referred to *M. forficata* or *M. pilosula*. He was known to be a heavy alcohol user and was also prescribed diazepam and fluoxetine.

Autopsy results were unremarkable apart from a fatty liver. Toxicological screening revealed a blood alcohol level of 0.22 g/100 mL and non-toxic concentrations of diazepam and fluoxetine. Serum tryptase level in blood taken three days after death was 2.72 µg/L (RR, < 2 µg/L). Tests for venom-specific IgE revealed high radioactive label uptakes for *M. pilosula* (31.7%) and the other *Myrmecia* species tested (*M. tarsata*, 42.1%; *M. simillima*, 39.8%; *M. pyriformis*,

39.6%; *M. gulosa*, 37.0%; and *M. nigrocincta*, 24.5%).

Patient 4

In 1999, a 65-year-old man working in his backyard in Hobart complained that he had just been stung on the knee by a jack jumper ant, feeling immediately unwell, itchy and short of breath. An ambulance was called, and he collapsed.

A paramedic crew arrived five minutes later, when he was found to be deeply cyanosed with no palpable pulses. He had a generalised urticarial rash and was making an occasional respiratory effort without any movement of air. Cardiopulmonary resuscitation was commenced, but bag-valve-mask ventilation was ineffective. Electrocardiogram demonstrated idioventricular rhythm. Paramedics administered a total of 13 mg of adrenaline, but at no stage was a pulse detected. On arrival in hospital 50 minutes after the arrest, laryngoscopy revealed marked laryngeal oedema and a gum-elastic bougie was required to achieve intubation. Resuscitation attempts were ceased shortly thereafter.

Past history included allergy to *M. pilosula*, with unconsciousness on several occasions. Fifteen years earlier, hyposensitisation was attempted with crushed whole-ant preparation. Since then he had been stung once, with what his family recalled to be a less severe reaction, leading him to believe he was protected from further stings. He also had a history of atrioseptal defect repair, chronic atrial fibrillation and impaired left ventricular function. Medications at time of presentation included warfarin, digoxin, bumetanide, carvedilol and foscarnil.

Autopsy revealed considerable oedema of the larynx, aryepiglottic folds and adjacent pharyngeal tissues. The lower airways were clear, indicating that aspiration had not occurred. There was no significant coronary artery disease, but there was marked dilatation of the tricuspid and mitral valves and all cardiac chambers.

Serum tryptase level in blood taken four hours after termination of resuscitation was 51.6 µg/L (RR, < 12 µg/L). IgE specific to the venoms of *M. pilosula* and honeybee was detected (radioactive label uptakes, 10.1% and 2.7%, respectively). IgE uptakes to three synthetic venom peptides, Myr p I-III, were 0.4%, 3.2% and 1.0%, respectively. No significant IgE reactivity with European wasp or other *Myrmecia* venoms was detected. Results of inhibition and immunoblot studies are shown in [Box 3](#).

Discussion It has been argued that deaths caused by sting anaphylaxis are under-reported because of lack of circumstantial evidence and the frequent absence of diagnostic postmortem features.¹⁵⁻¹⁷ This may be compounded by lack of awareness, as suggested by our first case.

IgE specific for *M. pilosula* venom was detected in the three patient sera tested. In Patient 3, binding of IgE to all tested venoms probably represented true immunological cross-reactivity, as the patient was unlikely to have been exposed to all these ant species, which are found in geographically disparate areas. Cross-reactivity between different *Myrmecia* venoms is well recognised⁷ and may lead to anaphylaxis after the sting of a species not previously encountered, which should be made clear to patients. Consequently, the third death may have been caused by the inchman ant, *M. forficata*, rather than the jack jumper, *M. pilosula*.

Postmortem findings supported anaphylaxis as a cause of death in all patients except Patient 3. In about half the deaths caused by anaphylaxis, no cause of death is evident at autopsy.¹⁷ In Patient 3, the apparent long interval between the sting and death, along with the significant comorbidities, raises the possibility that death was caused by a combination of moderately severe anaphylaxis, intoxication (alcohol, benzodiazepine and antihistamine) and obstructive sleep apnoea. Although the median time from sting to cardiac arrest in fatal cases is 15 minutes, significant delays of several hours may occur.^{18,19}

Serum mast-cell tryptase level was markedly raised in Patient 4, but only marginally raised in the other two patients tested. The reference range for the technique used in 1995 was < 2 µg/L,¹¹ and levels do not increase after death.²⁰ Baseline tryptase levels are raised in some people with sting allergy because of underlying mastocytosis.²¹ Using 10 µg/L as the cut-off level, postmortem serum tryptase level has 86% sensitivity and 88% specificity for predicting death caused by anaphylaxis.²² Tryptase level is not raised in many anaphylaxis cases, despite raised histamine levels,²³ and tryptase may not enter the circulation until 30 minutes after exposure, peaking 1-2 hours after exposure if the circulation remains intact.²⁴ Tryptase half-life is two hours with an intact circulation, increasing to four days after death.²⁴

In Patient 4, the clear-cut reactivity of IgE antibodies with whole venom was not replicated with synthetic peptides. While whole venom produced 80%-90% inhibition of IgE binding, synthetic peptides at much higher concentrations produced only 35% inhibition. Immunoblotting results suggested that the peptide

components separated by gel electrophoresis also had low reactivity to IgE in this patient's serum. A review of serum from 273 people allergic to *M. pilosula* venom reveals that, of those with positive reactions to whole venom, 19% show little or no reactivity with Myr p I or Myr p II. Immunoblotting with some of these sera demonstrated four previously unidentified IgE-binding bands with molecular weights 11.7, 16.9, 25 and 43.5 kDa (Wu and Baldo, unpublished data). These findings have significant implications for immunotherapy. In the absence of further data on interactions with T-cell epitopes, it cannot be assumed that these synthetic peptides will be effective substitutes for native venom immunotherapy.

Notably, we did not identify deaths of young healthy individuals. This repeats the pattern observed for bee and wasp sting allergy,^{15,19} and is consistent with the observation that adult males tend to have more severe reactions to bee stings.²⁵ All victims in this series were men aged 40 or over with significant comorbidities.

Two of the patients used angiotensin-converting enzyme (ACE) inhibitors, combined with a β -blocker in one case. These drugs may have contributed to the deaths or may simply have been a marker of underlying conditions that determined outcome. ACE is a kininase; inhibitors of this enzyme can trigger severe anaphylaxis in patients undergoing venom immunotherapy,²⁶ possibly because of decreased breakdown of vasodilator kinins activated during anaphylaxis or compromise of compensatory activation of the renin-angiotensin system.²⁷ In addition, β -blockers may both impair the endogenous adrenergic stress response and counteract and imbalance the effect of exogenous adrenaline. Interestingly, patients with severe venom allergy have reduced activity of the renin-angiotensin system, despite normal kininase activity.²⁸ Drugs that selectively inhibit the angiotensin II-1 (AT₁) receptor, such as losartan, may have less deleterious effects than ACE inhibitors. In patients undergoing haemodialysis, losartan may be associated with a lower incidence of anaphylactoid reactions than kininase inhibitors,²⁹ but no data are available on anaphylactic reactions to external allergens.

Three of the victims had previously sought medical attention for ant-sting allergy. In contrast, larger (and probably more representative) studies of sting-allergy deaths show that a minority of victims have a previous history of systemic sting allergy.^{16,19} Despite the known histories of sting allergy, only one of the victims had been prescribed adrenaline, which was not used. Another patient believed he was protected by previous immunotherapy with crushed whole ant-body extract. This technique has been shown to be no better than placebo in the only rigorously conducted trials available — for bee and wasp sting allergy^{30,31} — and is no

longer available for the treatment of jack jumper venom allergy. The optimal duration of immunotherapy is also unknown — indefinite continuation has been recommended for those with a history of severe reactions, such as Patient 4.³² These cases illustrate that the severity of reactions in allergic individuals can be unpredictable. There is potential to prevent deaths by careful patient counselling, prescribing of adrenaline for auto-injection and development of an effective hyposensitisation therapy. Currently available synthetic allergens may not reproduce enough of the immunological activity of *M. pilosula* venom to produce hyposensitisation. Therefore, we are currently conducting a trial of hyposensitisation therapy using native *M. pilosula* venom.

Acknowledgements

The authors thank Dr Catherine Morgan (Emergency Medicine Department, Royal Hobart Hospital) for her assistance with the manual search of the hospital forensic medical register. This work was supported by a grant from the Royal Hobart Hospital Research Foundation.

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(Received 26 Jun, accepted 15 Oct, 2001)

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1: Common bull ants (genus, *Myrmecia*) of Tasmania

A



B



A: The "jack jumper" ant (*Myrmecia pilosula*) is 10-12 mm long and jet black, except for yellow or orange mandibles and leg tips.

B: The "inchman" ant (*Myrmecia forficata*) is 15-20 mm long with a purple-brown body and black abdomen. Although other *Myrmecia* species appear similar, *M. forficata* appears to be the only such species found in appreciable numbers in southern Tasmania.

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2: Immunological methods

Immunological studies were performed at the Molecular Immunology Unit of the Kolling Institute of Medical Research, Sydney, NSW, during each forensic investigation.

Serum tryptase levels: Tryptase released from mast cells was measured in postmortem serum by radioimmunoassay (reference range, < 2 µg/L in 1995; and < 12 µg/L in 1999, because of reagent changes). [11,12](#)

IgE antibody-binding studies:

Patient IgE specific for venom was measured by incubating patient serum with venom-coated nitrocellulose discs. IgE that bound to the discs was measured using ^{125}I -labelled anti-IgE, and results expressed as percentage uptake of the radioactive label. Uptake > 2% was considered positive.⁷

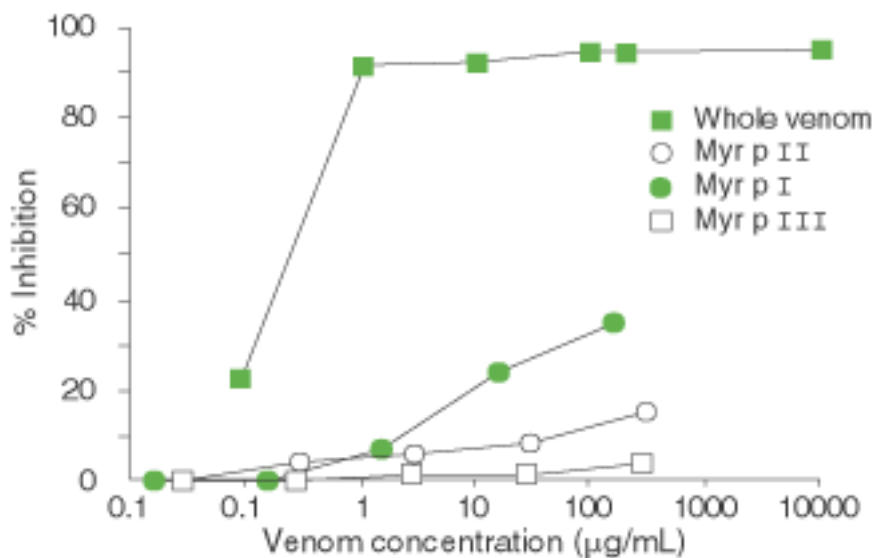
Inhibition studies: The specificity of patient IgE for synthetic venom peptides was investigated as described previously.^{7,13} Patient serum was pre-incubated with whole venom or synthetic venom peptides to neutralise specific IgE. Remaining venom-specific IgE was then measured by uptake to venom-coated discs as described above, and compared with levels in serum that was not pre-incubated. Percentage inhibition of IgE due to the venom or peptide was calculated.

Immunoblot: To investigate specificity of patient IgE further, venom components separated by sodium dodecyl sulfate polyacrylamide gel electrophoresis (SDS-PAGE) were transferred to nitrocellulose and then probed with patient serum. Binding of patient IgE to venom components in the gel was detected using ^{125}I -labelled anti-IgE and autoradiography.^{5,14}

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3: Inhibition and immunoblot studies in Patient 4

Inhibition of IgE binding by *Myrmecia pilosula* whole venom and synthetic venom peptides



Whole *M. pilosula* venom was a potent inhibitor of IgE binding. Of the purified peptides, only Myr p II caused significant inhibition, which was markedly less than that produced by whole venom.

Immunoblot studies (not shown)

No binding of patient IgE to venom components separated by SDS-PAGE and transferred to nitrocellulose was identified initially by autoradiography. However, after 2 weeks of film exposure, bands previously noted to represent Myr p II⁹ became visible. No binding to other bands was apparent.

Interpretation

These findings suggest that the synthetic peptides and separated venom components do not account for the total allergenic activity of *M. pilosula* venom, as represented by IgE binding.

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