House-dust mite and cat allergens in the Antarctic

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Allergens of house-dust mites (HDM) and domestic cats provoke asthma and rhinitis in sensitised individuals.[1] The major allergen of the HDM *Dermatophagoides pteronyssinus*, Der p 1, is found on their relatively large faecal particles, and HDM themselves require an ambient relative humidity (RH) of over 50% to feed and reproduce. Cat allergen, Fel d 1, derives from saliva and other secretions, and is present on very small airborne particles. These different physical characteristics influence the distribution of the allergen within the environment. To examine this distribution further we have measured HDM and cat allergens at Scott Base, the permanent New Zealand Antarctic base on Ross Island on the edge of the Antarctic continent. The dry conditions in the Antarctic, where outdoor RH rarely rises above 20%, is not supportive for HDM proliferation. The Antarctic is also entirely free of cats. Thus any HDM and cat allergens found there would have been introduced passively, most likely from clothing, known to contain HDM and cat allergens.[2,3]

We obtained dust samples by vacuuming three living-room areas and twelve mattresses with established techniques.[1] Additionally, dust samples were obtained from the sweaters of 11 recently arrived Scott Base personnel, seven of whom kept cats in their homes in New Zealand. RH and temperature were continuously recorded in the living-room and bedroom areas, using an Axiom Smart Reader data logger. The dust samples were analysed for Der p 1 and Fel d 1 by double-monoclonal antibody ELISA techniques[4,5] and expressed as μg of allergen per g of dust.[1]

HDM allergen, Der p 1, was undetectable in the three living-room areas, and only one of 12 mattresses sampled contained detectable Der p 1 (0·61 μg/g). Der p 1 was detectable in six of the 11 sweaters, in concentrations ranging from 0·28 to 2·18 μg/g. Cat allergen, Fel d 1, was detectable in one living-room area (0·44 μg/g), and in seven mattresses with Fel d 1 levels ranging from 0·43 μg/g to 1·49 μg/g. Ten of the 11 sweaters had Fel d 1 levels ranging from 0·45 μg/g to 4·18 μg/g. Indoor RH remained constantly below 20% at a temperature averaging 17°C.

This study confirms passive transfer of both HDM and cat allergens, with greater accumulation of cat allergen compared to HDM allergen in the local environment. In the absence of sustainable HDM populations Der p 1 accumulation appears likely to be minimal whereas Fel d 1 appears to accumulate even in remote and cat-free environments. This could result in unexpected symptoms in individuals allergic to cats.

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A Ca^{++} activated serine protease (LOPAP) could be responsible for the haemorrhagic syndrome caused by the caterpillar *Lonomia obliqua*

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Since 1989, a haemorrhagic syndrome produced by contact with the *Lonomia obliqua* caterpillar[1] has become epidemic in southern Brazil. Fatal cases due to renal complications and cerebral haemorrhages have been reported.[2,3] The accidents affect the clotting mechanism, resulting in a drastic decrease of fibrinogen, factors V and XIII. In addition, decreases of plasminogen and α 2-antiplasmin, and of the activity of protein C, the natural inhibitor of clotting are seen. These data indicate a consumption coagulopathy with fibrinolysis.[4]

To assess whether one or more prothrombin activators of the caterpillar toxins could be involved, the soluble proteins of *L obliqua* bristles were purified by gel filtration and reverse phase (HPLC) chromatographies. Prothrombin activator activity was monitored by using prothrombin and a chromogenic substrate specific for thrombin (S2238, Chromogenix). The hydrolysis products of prothrombin were also identified by SDS-PAGE. A single-chain protein of 69 kDa displaying a Ca^{++} activating serine protease activity was shown to directly convert prothrombin into thrombin. It can be included into the group 1 of prothrombin activators. The protein, named *L obliqua* Prothrombin Activator Protease (LOPAP), was purified to homogeneity and aminoacid sequencing indicated no homology with prothrombin activators or with any other known serine proteases. In-vivo experiments in mice showed that intravenous injection of the purified protein, similarly to the crude extract, causes a dose-dependent blood unclottability. The observation was corroborated by observation of cremaster microcirculatory network study after protein injection using intravital microscopy technique. Data obtained showed that the infusion of LOPAP produced intravascular coagulation and thrombosis in post capillary vessels, which often contributes to end-organ damage.[5] This study indicates that LOPAP is probably one of the main factors causing consumption coagulopathy in accidents with *L obliqua—L achlous* may have a different toxin.

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