Peanut in house dust linked to peanut allergy in children with skin gene mutation

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A new study led by researchers at King's College London in collaboration with the University of Manchester and the University of Dundee has found a strong link between exposure to peanut protein in household dust during infancy and the development of peanut allergy in children genetically predisposed to a skin barrier defect.

Around 2% of school children in the UK and the US are allergic to peanuts. Severe eczema in early infancy has been linked to food allergies, particularly peanut allergy. A major break-through in the understanding of eczema developed with the discovery of the FLG gene which codes for the skin barrier protein filaggrin. Mutations in the FLG gene result in an impaired skin barrier which is thought to allow allergens to penetrate the skin and predispose the body towards an allergic response.

The study, published this month in the *Journal of Allergy and Clinical Immunology*, looked at the amount of peanut protein children were exposed to in household dust in their first year of life by vacuuming dust from the living room sofa and measuring peanut in the dust. A group of 577 children were assessed at 8 and 11 years of age for peanut allergy and their DNA was checked for FLG mutations. The study was conducted in children recruited to the Manchester Asthma and Allergy Study.

A strong link was found between early-life exposure to peanut protein in household dust and peanut allergy in children with FLG mutations. A three-fold increase in house dust peanut exposure during infancy was associated with a three-fold increase in risk of school-age peanut allergy. One in five children with peanut allergy had an FLG mutation. There was no significant effect of environmental peanut exposure in children without FLG mutations.

Dr Helen A Brough, first author from the Department of Paediatric Allergy, King's College London, said: "Our findings provide evidence that peanut allergy may develop via the skin in children with mutations in the gene that codes for filaggrin which damage the function of this important skin protein. These findings are also an example of how an individual's response to their environment can be modified by their genes. Our study raises the possibility of being able to identify a group of children with FLG mutations through genetic testing in the future, and altering their environmental exposure to peanut early in life to reduce the risk of developing peanut allergy."

Professor Gideon Lack, senior author from the Department of Paediatric Allergy, King's College London, said: "This is further evidence for the dual-allergen-exposure theory which suggests food allergies develop through exposure to allergens via the skin, likely through a disrupted skin barrier, whilst consumption of these food proteins early in life builds up tolerance in the body. Previous guidelines recommending that mothers should avoid peanuts during pregnancy and breastfeeding have now been withdrawn. Ongoing studies at King's aim to find if exposure to solids in early infancy might actually help to prevent allergies. It may be that the timing and balance of skin and oral exposure to a particular food early in life determines whether a child develops an allergy or tolerance to that food."

Over the past two years, the King's College London

team has been investigating the level of peanut protein in household dust, how this correlates with household peanut consumption, and how peanut protein in dust can trigger an immune response in the cells of children who are allergic to peanut. Peanut protein is present on hands and in saliva for up to three hours after peanuts or peanut-based food has been eaten, and can persist on table surfaces and sofa or pillow dust even after routine cleaning.

More information: 'Peanut allergy: Effect of environmental peanut exposure in children with filaggrin loss-of-function mutations' by Brough et al is published in the *Journal of Allergy and Clinical Immunology*: dx.doi.org/10.1016/j.jaci.2014.08.011

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