

Eczema in infancy may be linked to cat ownership in those with a specific gene mutation

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A gene mutation and cat exposure at birth may increase a child's risk of developing eczema during their first year according to a study published in *PLoS Medicine* this week. Researchers led by Professor Hans Bisgaard (University Hospital Gentofte, Copenhagen, Denmark) studied the association between mutations in the filaggrin gene (FLG) and exposure to environmental factors with the development of eczema.

Eczema runs in families and evidence suggests it is caused by genetic and environmental factors. The same researchers recently discovered that two common "loss-of-function" variants in the gene encoding filaggrin (FLG) predispose people to eczema. Filaggrin is a protective protein normally found in skin. It acts as a physical barrier to potentially harmful substances in the environment. The researchers hypothesized that inheriting one or two defective FLG genes might weaken their physical barrier, affecting their response to environmental substances.

The hypothesis was tested by conducting a cohort study in two independent groups of infants – a high-risk group consisting of 379 infants born in Copenhagen, Denmark to mothers with asthma and a group of 503 infants born to women from the general population in Manchester, UK. The researchers determined which FLG variants each child had inherited and classified those with either one or two defective copies of FLG as having an FLG mutation. They determined pet exposure in early life by asking whether a dog or a cat was living in the

parental home when the child was born and then analyzed how these genetic and environmental factors affected the age of onset of eczema. In both groups, children with FLG mutations were twice as likely to develop eczema during the first year of life as children without FLG mutations. For children without FLG mutations, cat ownership at birth had no effect on eczema risk but for children with FLG mutations, cat ownership at birth (but not dog ownership) further increased the risk of developing eczema. However, it is important to note that this last estimate is based on findings in a very small number of children.

The findings provide support for the researchers' suggestion that filaggrin deficiency causes weakening of the skin's protective barrier, increasing a child's susceptibility to factors associated with cat exposure. The limitation of this study is that only a very small number of children in this study both carried FLG mutations and were exposed to cats from birth, so these findings need confirming in independent studies. In addition, it is still not clear how exposure to cats drives the development of eczema. Allergy was not the mechanism as the FLG-deficient children exposed to cats and who developed eczema did not develop cat-specific immunoglobulin E antibodies. Nevertheless, if confirmed, these findings suggest that, to reduce their risk of developing eczema, filaggrin-deficient individuals may need to avoid cats (but not dogs) during the first few months of life.

Source: Public Library of Science

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