From genes to latrines—Vikings and their worms provide clues to emphysema

February 4 2016

In a paper published today in *Scientific Reports* a group of researchers led by LSTM have found that the key to an inherited deficiency, predisposing people to emphysema and other lung conditions, could lie in their Viking roots.

Archaeological excavations of Viking latrine pits in Denmark have revealed that these populations suffered massive worm infestations sciencenordic.com/dna-study-vi … intestinal-parasites. The way that their genes developed to protect their vital organs from disease caused by worms has become the inherited trait which can now lead to lung disease in smokers.

Chronic obstructive pulmonary disease (COPD) and emphysema affect over 300 million people, or nearly 5% of the global population. The only inherited risk factor is alpha-1-antitrypsin (A1AT) deficiency, and this risk is compounded if individuals smoke tobacco.

A1AT protects the lungs and liver from enzymes called proteases that are produced by cells of the immune system, but also by parasitic worms. In the absence of A1AT these proteases can break down lung tissue leading to COPD and emphysema.

Deficiency of A1AT is genetically determined and is due to deviants of A1AT that are surprisingly common, particularly in Scandinavia, where they evolved in Viking populations more than two thousand years ago. Why these disease-causing deviants of A1AT are so common in human

populations today has long been a mystery.

LSTM's Professor Richard Pleass is senior author on the paper. He said: "Vikings would have eaten contaminated food and parasites would have migrated to various organs, including lungs and liver, where the proteases they released would cause disease."

In this latest paper the authors show that these deviant forms of A1AT bind an antibody called immunoglobulin E (IgE) that evolved to protect people from worms. The binding of A1AT to IgE prevents the antibody molecule from being broken down by such proteases.

"Thus these deviant forms of A1AT would have protected Viking populations, who neither smoked tobacco nor lived long lives, from worms." Continued Professor Pleass, "it is only in the last century that modern medicine has allowed human populations to be treated for disease causing worms. Consequently these deviant forms of A1AT, that once protected people from parasites, are now at liberty to cause emphysema and COPD."

More information: Phyllis M. Quinn et al. IgE-tailpiece associates with α-1-antitrypsin (A1AT) to protect IgE from proteolysis without compromising its ability to interact with FcεRI, *Scientific Reports* (2016). DOI: 10.1038/srep20509

Provided by Liverpool School of Tropical Medicine