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RESEARCH ARTICLE

FOOD ALLERGIES: CAUSE AND PREVENTION

***Dr. Gerry A Smith**

Biochemistry Dept. Cambridge, retired, 14, Lantree Cres, Trumpington, Cambridge UK, CB2 9NJ

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INTRODUCTION

It is often reported that a food sensitivity arises without previous contact with the food e.g. peanuts, soy, other nuts and dairy. There is one enlightening publication by Gideon Lack[1] taken from earlierwork[2]who has demonstrated that emollient treatment leads to peanut allergy. Also reported elsewhere[3]. This is also given prominence in the press, https://www.sciencenews.org/blog/food-thought/unexpected-sources-peanut-allergy o rhttps://www.telegraph.co.uk /news/world news/ north america/usa/1424292/Peanut-allergy-is -linked-to-creams-for-skin-rashes.html.(The authors name was a typo in the telegraph, given as Lock).With this coverage and what I now present the observation, amazingly, was then forgotten.

Causes of Food Allergies: The problem is with the industrial processing of triglycerides which come from sources that food allergies have arisen to. This simple prevention should become industry wide, not just peanut. It is my belief that virtually all peanut allergies are attributable to the following as peanuts and peanut oil are not specifically allergenic. It also applies to soy and other sources like butter.

The underlying chemistry: The startpoint is as any chemist will agree under basic conditions N-acylation is much faster than O-acylation. In production of mono- and di- glycerides from triglyceride they use base catalysed fatty-acyl transfer to added glycerol. Hence any peptide present, either dissolved or in suspension, will have a fatty-acyl group put on the N-terminal amino-acid and elsewhere. Dissolution of any suspended peptides will be aided by the added glycerol. These fatty-acyl peptides will contaminate the mono- and di-glycerides produced, and would need anion-exchange chromatography to remove.

***Corresponding author: Dr. Gerry A Smith,**
Biochemistry Dept. Cambridge, retired, 14, Lantree Cres, Trumpington, Cambridge UK, CB2 9NJ.

On a quick look on the web I cannot see any fatty-acyl peptide antigen use in immunisation, they all concentrate on the use of adjuvants as required for simple acyl peptides as antigens.

Administration to humans: Mono- and di-glycerides are widely used in two ways, either in emollients, see above press clipping, and also added to industrialised foodstuffs to disperse the fats. Hence when applied to broken skin the emollients get absorbed into the bloodstream along with the fatty-acyl peptides. When ingested orally as food the fatty-acyl peptides will be protonated in the gut and hence will not carry a charge, so they will be insoluble and associated with fat particles as they go through to the lower intestines. In the lower intestines they will get absorbed with the fats present. This amounts to mass immunisation of the public with the fatty acyl peptides. The glycerides get absorbed by the body and metabolised as normal.

The immune-response, why IgEis produced giving anaphylactic responses: When in the bloodstream the fatty-acyl peptides will be bound to cell surfaces by the fatty-acyl moiety and thus presented as membrane-boundantigens to the immune system. It is my belief that antigens presented thus are powerful and will not need adjuvants. Furthermore, as the antigen is on a cell surface, to the immune system it will appear as an invasive species. I seem to recall that IgE is produced in response to an invasive species, e.g. trypanosomes etc.

Hence the anaphylactic responses with these allergies, viz. IgE activated release of histamine. Someone needs do an animal immunisation with a fatty-acyl peptide to get IgE. This alone could be somebody's research project, they may find it interesting per se. A review published 2017 may prove useful[4]. I now make a conjecture. With the occurrence of fatty-acyl antigen on the same cell surface as native proteins and hence possible interaction between them there is the probability of triggering an auto-immune response. Is there any spatial or temporal occurrence correlation of food allergy with auto-immune disease?

The ultimate prevention of peanut and other related food allergies: Inexpensive total removal of the peptides in triglyceride is priority, this is down to the industry, they may have better ideas. It may be sufficient to simply dilute aqueous mineralacid wash the triglyceride before use, maybe in easily removed light petroleum spirit with fine filter to remove interfacial particulates, to facilitate careful layer separation and discarding of the aqueous wash. For use a bicarbonate wash may be needed prior to evaporation of the petroleum ether. Fat soluble peptides will be short, ion pairedcyclic, so appear uncharged and somewhat hydrophobic, hence acid wash should do the trick. Alternatively pass through/over an absorbent such as activated silica, ion exchange resin etc. to remove all peptides, whatever the source of triglyceride. Clearly removal of peptides before triglyceride processing is chemically easier, the glyceride emulsifiers are difficult to manipulate.

Next to do: I think with Lack's work[1] as proof of the thesis this could have been just patented as is and let the chemists in the industries follow up. It is so important as it means the end of peanut and other related allergy epidemics and anaphylactic deaths. To isolate any suspected fatty acyl peptide one should take the emollient or mono-/di-glyceride and pass through a diethylamino based anion exchange polysaccharide column in aqueous methanol, wash off the glycerides with aqueous methanol then extract any fatty-acyl peptide from the anion exchanger with suitable ionic solution, e.g. acetic acid in aqueous methanol.

One can identify the fatty-acyl peptide and its amino-acid sequence by chromatography and/or mass spec etc. Frying the peptides in the oil containing them may also contribute, but I doubt this as the allergy epidemics would have appeared much earlier especially with the onset of arachis oil in kitchens.

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