

Euro Dementia 2018: Immunoreactivity of Anti-A β -42 Specific Antibody with Toxic Chemicals and Food Antigens- Aristo Vojdani- Immunosciences Lab Inc.

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Abstract

Objective: The aim of our study was to examine immunoreactivity between A β -42, toxic chemicals, and food proteins that would be involved in AD. Methods: We applied monoclonal anti-A β -42 to a range of chemicals bound to human albumin (HSA) and 208 different food extracts.

Keywords: A β -42; Amyloidogenesis; Alzheimer's Disease; Toxins; Dietary Proteins; Immunoreactivity; Neurodegeneration

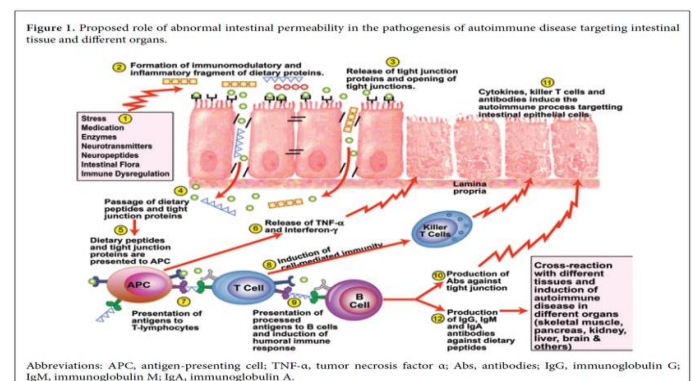
Immunoreactivity to Common Foods: Now that we have some understanding of Immunology under our belts, we will get to the meat of what my clients tend to care about – immunoreactivities to common foods and how this may be a trigger for autoimmunity or one's own immune system seeing self-tissue as a pathogen. This is a very big deal as it is estimated that over 50 million Americans suffer from some kind autoimmunity (comparatively, heart disease “only” affects upwards of 22 million Americans) and the occurrence of autoimmunity is growing exponentially. It is now believed that around 30% of autoimmunity is due to genetic predisposition. But, this implies that 70% of autoimmune cases are potentially caused by environmental and lifestyle triggers – the main culprits being intestinal permeability, infections, and chemical exposures.

“Bacterial toxins, chemicals, foods, and undigested proteins and peptides can induce systemic food immune reactivity by causing failure of immune tolerance. Immune tolerance is the immune system's ability to recognize what is harmful and what is not. If immune tolerance is lost, then inflammation ensues and autoimmunity can occur.”

The digestive system sees over 1 ton of food every year and contains north of 70% of our immune cells. Your gastrointestinal tract also has more contact with the surface world than your skin and this mucosal lining is constantly being exposed to potential antigens. The reason our body doesn't wreck shop on all this food stuff all the time is due to

oral tolerance which occurs through the deletion or immunosuppression of reactive immune cells.

Reacting to common food items isn't normal and may be due to impaired immunological development (which is outside the scope of this post and revolves around the hygiene hypothesis, maternal diet, how one was born, breast feeding, and also the infant gut microbiota) or an immune system that's out of balance (cough low vitamin D) and over stimulated (SAD).



The figure above is from Dr. Aristo Vojdani, if not the leader, in food immunoreactivity. There is a lot going on above, but we can use our newfound understanding of immunology and begin to grasp how this all works.

Materials and Methods: Monoclonal antibodies

Commercially available antibodies were purchased from different companies. Rabbit monoclonal anti-amyloid- β -42 (fibril sequence DAEFRHDSGYEVHHQKLVFFAEDVGSNKGAIIGLMVGGVVIA) produced by Abcam's RabMab® technology was purchased from Abcam. This antibody reacts strongly to human A β 42 monomers, oligomers and fibrils, but not with human muscle fibrils. Additional information about the specificity of this antibody is provided in the Abcam package

insert (ab201061) and in an article by Hatami et al. Affinity-purified mouse anti-amyloid- β 1-42 was purchased from BioLegend, San Diego, CA USA. This antibody reacted strongly with formalin-fixed, paraffin- embedded diseased human brain tissue.

Binding of phthalate to HSA: Preparation of diethyl phthalate was done according to the method described by Zhou et al. briefly, 2 mg of diethyl phthalate was dissolved in methanol and added drop wise to 25 mg of HSA dissolved in 5 mL of 0.14 Tris-HCl buffer.

Preparation of dietary antigens: Food antigens were prepared from products purchased from the supermarket in both raw and cooked forms. For that preparation, 10 g of nutrient was put in a food processor using 0.1 M of phosphate buffer saline (PBS) at pH 7.4. The mixer was turned on and off for 1 h and then kept on the stirrer overnight at 4°C. After centrifugation Citation: Vojdani A, Vojdani E (2018) Immunoreactivity of Anti-A β P-42 Specific Antibody with Toxic Chemicals and Food Antigens. J Alzheimers Dis Parkinsonism 8: 441. doi: 10.4172/2161-0460.1000441 Page 3 of 11 Volume 8 Issue 3 • 1000441 J Alzheimers Dis Parkinsonism, an open access journal ISSN:2161-0460 at 20,000 g for 15 min, the top layer, which contained oil bodies, was discarded The liquid phase was removed and dialyzed against 0.01 M of PBS using dialysis bags, with a cutoff of 6,000 kDa. Dialysis was repeated three times to ensure all small molecules were removed. After dialysis, all samples were filtered through a 0.2 micron filter to remove any debris. Protein concentrations were measured using a kit provided by Bio-Rad (Hercules, CA, USA). Different peptides were purchased from Bio-Synthesis (Lewisville, TX, USA). Lectin and agglutinins including pea lectin and lentil lectin were purchased from SigmaAldrich (St. Louis, MO, USA).

Conclusion: Depend on these results, we hypothesized that reaction between A β P-42 antibody with chemically bound to HSA and numerous food antigens might play a lead role in Alzheimer's disease (AD). These anti-A β P antibodies might be derived from protein mis folding like to β -amyloid, or from antibodies to various food antigens that cross-react with A β P-42. Removal of toxic chemicals and food items that share a homology with β -amyloid is also recommended at least for patients within the early stages of AD. Therefore, the role of A β P-42 cross-reactive foods and chemicals bound to HSA in neurodegeneration should be investigated further.

Results: We have found that anti-A β P-42 reacts from moderately to strongly with mercury-HSA, dinitrophenyl-HSA (DNP-HSA), phthalate-HSA, and aluminum-HSA, but not to many other tested chemicals bound to HSA nor to HSA alone. This antibody also reacted with 19 out of the 208 food antigens utilized in the assay. One example of a food that reacted strongly with anti-A β P-42 in our study was canned tuna, although raw tuna reacted only moderately.

Note: This work is partly presented at 11th International Conference on Alzheimers Disease & Dementia, May 24-25, 2018 at Vienna, Austria