

Review

Food-Specific IgG Guided Elimination Diet; A Strategy for Weight Loss?

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Being obese or overweight arises from excess energy consumption, relative to expenditure, over long periods. Although eating patterns are influenced by many factors, such as cultural, social and commercial factors [1], the regulation of food intake and uptake in the body is a very complex process, which involves biochemical signals and hormones from many sources including the braingastrointestinal axis, fat stores and the pancreas. Even the fat cells, adipocytes, themselves are highly specialised to play important roles in energy storage, fatty acid metabolism and glucose regulation. Obesity is also correlated with inflammation in the body [2] and, once overweight or obese, shifting the balance to lose weight may not be as simple as just reducing energy consumption and increasing energy output.

Many reports collectively implicate the inter play between the intestinal microbiota (the microbiological environment in the gut or gut flora), intestinal permeability ("leaky gut"), and the immune system as one mechanism linking diet, obesity, and associated diseases. Cox et al (2015) [3] propose that reducing intestinal permeability through interventions that improve the intestinal microbiota, and reducing the activation of the immune system and associated inflammatory responses, could be a key strategy to address obesity and obesity-related disease. It is well known that dietary changes produce changes in gut flora, but the composition of the gut microbiota varies substantially amongst individuals [4]. Dietary changes to lose weight may not just be about reducing calories, more about shift changes that are specific to each individual.

A strategy to address obesity based on reducing intestinal permeability, improving the gut flora, and reducing activation of the immune system is significant when considering the part that an elimination diet based on food-specific IgG antibodies might play. "Leaky gut" increases the probability that larger food particles can enter the blood stream and this creates the potential for those food particles to trigger a food-specific IgG immune response. It is well known that elimination diets based on food-specific IgG measurement have been shown to be beneficial for conditions such as migraine [5] and digestive disorders [6,7] but, what evidence links raised food-specific IgG antibody levels with obesity and

inflammation?

One key study took two groups of children, the first group were obese and the second were in the normal weight range. Blood was tested for food-specific IgG antibodies and the inflammatory marker C Reactive Protein (CRP). The obese group had two and a half times the IgG antibodies against certain foods than the children in the normal weight range. They also had three times the levels of CRP than the normal weight group [8]. Food-specific IgG antibodies are tightly associated with low grade systemic inflammation and the authors at the time, 2008, postulated that anti-food IgG is involved in the development of obesity. This finding is supported by another more recent study that analysed levels of yeast-specific IgG antibody levels, comparing obese and normal weight individuals. The presence of this antibody was positively associated with weight, BMI, and waist circumference [9].

It is interesting, too, to consider the impact that other biochemical messengers or hormones might play. The IgG antibodies that are present in obese people have been shown to bind the "hunger hormone" ghrelin much more that than the IgG antibodies from non-obese people. It appears that increased appetite and cravings may be mediated, at least partly, by circulating IgG enhancing the orexigenic (appetite stimulating) effect of the ghrelin [10]. In addition, chronic inflammation has been shown to impair the brain's ability to receive the hormone leptin's appetite suppressing messages, called leptin resistance, and so you keep eating [11]. It has

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been demonstrated that dietary fat excess can have inflammatory effects on the hypothalamus [12] which, in turn has a well-known role in appetite and thermogenesis [13]. Indeed, obesity in humans is associated with hypothalamic inflammation and disturbances in the gut-brain axis which are known to be influenced by dietary, environmental and genetic factors [14]. These findings may, in part, explain the "addiction" to food and cravings often reported by those trying to lose weight. What strategy then to break this cycle of inflammation and addiction?

In 2012, Lewis et al [15] published a study that looked at the effect of eliminating immunologically reactive foods from the diet of overweight individuals. They not only measured changes in weight, but also body composition, blood pressure, heart rate and physical and mental measures of quality of life; scales of vitality, bodily pain and general health. 120 overweight subjects were involved and were prescribed an elimination diet based on food-specific IgG antibody results. The subjects had to eliminate all their reactive foods for 90 days. Participants lost, on average, 5kg in weight and 8cm in waist circumference over the 90 day period. In addition to the positive changes associated with body composition there was a significant drop in diastolic blood pressure and substantial improvements in both physical and mental quality of life.

A recent survey [16] analysed results from 38 individuals who had taken a food-specific IgG test programme, including the option of Nutritional Therapist support, and who had reported weight loss after making dietary changes according to the test results. For the majority (87%), the main reason for using the programme was not related to a desire to lose weight; primary reasons included digestive symptoms such as IBS and bloating, skin symptoms such as eczema and rashes, migraines and fatigue. In terms of weight loss, 52% started to notice a reduction in weight within the first 2 weeks, with 31% starting to lose weight between 2 and 4 weeks.48% lost up to 4kg, 43% lost between 5-9kg and the remainder (9%) lost over 9kg in weight; all over a greater than 3 month period. Only 55% said that, looking back, they had been concerned about their weight before they took the test, but yet 92% said that the weight loss that they achieved was desirable. Sometimes positive outcomes owe more to serendipity than careful design, think of Viagra, and the fact that weight loss was not the original intention of the majority of participants adds weight to this survey.

So, can a personalised IgG-guided elimination diet help shift change the gut microbiota, reduce the activation of the immune system and associated inflammatory responses, and aid weight loss? It is clear that IgG testing for food is not considered diagnostic for "food intolerance" per se because a direct cause-effect relationship has not been firmly established. However, an increasing number of studies are emerging that show a correlation between food-specificIgG guided elimination diet and improvement in a variety of conditions. There are countless dietary programmes on the market, but none of them recognises the possibility that certain foods, even healthy ones like tomatoes or carrots, could be problematic if they trigger an immune system response in overweight individuals. The point being here that each dietary intervention, on this basis, is personalised; dependent on specific tailored food-IgG test results; providing a unique targeted approach. Eliminating foods that are IgG-reactive, while replacing them with similar, non-reactive foods to ensure that nutrient deficiencies do not occur, is a relative new concept for a diet; showing promise as a highly effective strategy for addressing obesity.

References

- Lean M, Malkova D (2016) Altered gut and adipose tissue hormones in overweight and obese individuals: cause or consequence? Int J Obes 40(4): 622-632.
- Ellulu M, Khaza'ai H, Rahmat A, Patimah I, Abed Y, et al. (2016) Obesity can predict and promote systemic inflammation in healthy adults. Int J Cardiol 215: 318-324.
- 3. Cox A, West NP, Cripps AW (2015) Obesity, inflammation, and the gut microbiota. Lancet Diabetes Endocrino I3(3): 207-215.
- 4. Carding S, Verbeke K, Vipond DT, Corfe BM, Owen LJ (2015) Dysbiosis of the gut microbiota in disease. Microbial Ecology 26: 26191.
- Alpay K, Ertas M, Orhan EK, Ustay DK, Lieners C, et al. (2010) Diet restriction in migraine, based on IgG against foods: a clinical doubleblind, randomised, cross-over trial. Cephalagia 30: 829-837.
- Atkinson W, Sheldon TA, Shaath N, Whorwell PJ (2004) Food elimination based on IgG antibodies in irritable bowel syndrome: a randomised controlled trial. Gut 53(10): 1459-1464.
- Bentz S, Hausmann M, Piberger H, Kellermeier S, Paul S, et al. (2010) Clinical relevance of IgG antibodies against food antigens in Crohn's disease: a double-blind cross-over diet intervention study. Digestion 81(4): 252-264.
- Wilders-Truschnig M, Mangge H, Lieners C, Gruber H, Mayer C, et al. (2008) IgG antibodies against food antigens are correlated with inflammation and intima media thickness in obese juveniles. ExpClinEndocrinol Diabetes 116 (4): 241-245.
- 9. Salamati S, Martins C, Kulseng B (2015) Baker's yeast (Saccharomyces cerevisiae) antigen in obese and normal weight subjects. Clin Obes 5(1): 42-47.
- Takagi K, Legrand R, Asakawa A, Amitani H, François M, et al (2013) Anti-ghrelin immunoglobulins modulate ghrelin stability and its orexigenic effect. Nat Commun 4:2685.
- 11. Zhou Y, Rui L (2013) Leptin signalling and leptin resistance. 7(2): 207-222.
- Viggiano E, Mollica MP, Lionetti L, Cavaliere G, Trinchese G, et al. (2016) Effects of a high-fat diet enriched in lard or in fish oil on the hypothalamic amp-activated protein kinase and inflammatory mediators. Front Cell Neurosci 10:150.

- Monda M, Viggiano AN, Viggiano A, Viggiano E, Lanza A, et al. (2005) Hyperthermic reactions induced by orexin A: role of the ventromedial hypothalamus. European Journal of Neuroscience 22(5): 1169-1175.
- 14. Kreutzer C, Peters S, Schulte DM, Fangmann D, Turk K, et al. (2017) Hypothalamic inflammation in human obesity is mediated by environmental and genetic factors. Diabetes May db170067.
- Lewis J, Woolger JM, Melillo A, Alonso Y, Rafatjah S, et al (2012) Eliminating immunologically-reactive foods from the diet and its effect on body composition and quality of life in overweight persons'. J Obes Weig los Ther 2:1.
- 16. Survey by YorkTest Laboratories (2016)