In trying to highlight some of the potentially disastrous health problems that may arise from genetic manipulation of our food, it has been necessary to delve quite deeply into the technology. Some of the issues raised by those concerned with genetic manipulation of our food have been treated with disdain and answered with off—handed dismissals on the assumption that those opposed to gene technology do not understand what is going on. It has been suggested that foreign DNA in genetically modified food may cause ‘allergic’ disease and that "allergies occur to the protein produced, not the DNA". This asserts that DNA is not immunogenic. This is not the case!

Immunological reactions in humans can and do occur against DNA fragments as evidenced by the autoimmune disease SLE. In this disease, antibodies are produced against DNA fragments and nucleoprotein released from dying cells. This results in a Type III Immune Mediated Hypersensitivity Reaction. In fact, there are a small number of potentially self-reactive cells with access to their respective autoantigens such as human thyroglobulin, myelin basic protein and DNA normally present in the body. The only thing holding them in check normally is properly functioning homeostatic mechanisms that have obviously gone astray in conditions such as SLE and Rheumatoid arthritis. Immune complex glomerulonephritis is another condition associated with the production of antibodies against DNA and DNA-protein etc. In patients with SLE, immune
complex deposits containing antibodies to single stranded and double stranded DNA have been detected in the kidney tissue.

While these Type III reactions do not cause the classic ‘IgE allergic’ response, they can and do cause insidious disease when these antigen-antibody reactions form insoluble complexes at fixed sites within the body that may give rise to acute inflammatory reactions, eg rheumatoid arthritis. The production and release of inflammatory mediators and proteolytic enzymes etc can damage tissue and further intensify inflammatory responses. Type III complex mediated hypersensitivity reactions can affect the skin producing edema and erythema, or the lungs e.g. farmer’s lung, pigeons fanciers disease & pulmonary aspergillosis.

So what does it matter if we can produce antibodies against DNA and RNA, and what has it to do with any potential DNA fragments in our food supply as a result genetic manipulation? It matters because:

The homeostatic mechanisms that keep Type III autoimmune disorders at bay are poorly defined and understood, and

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There are many examples in which potential autoantigenic determinants are present on an exogenous cross-reacting antigen. These can provoke autoantibody formation.

Examples are

- Post rabies vaccine encephalitis, thought to result from an autoimmune reaction to brain initiated by heterologous brain tissue in the vaccine;
- Some micro-organisms carry determinants which cross react with human antibodies;
- Colon antibodies present in ulcerative colitis have been found to cross react with E Coli 014.
- The anti-RNA antibodies of SLE patients can bind to a t-RNA obtained from E Coli and a bacterial phage.
- The anti-RNA antibodies in SLE sera are heterogeneous, ie able to bind different portions or types of RNA.
- Drugs can result in autoimmunity and production of antinuclear antibodies.
  For example prolonged treatment with Isoniazid may produce arthritis associated with nuclear antibodies, and a high proportion of patients on continued treatment with procainamide develop nuclear antibodies with 40% of these present with clinical signs of SLE.

The pathological effects of autoimmunity vary and may be:

- Innocuous, where immune complexes may be present but do not appear to be causing pathogenic changes, or
- The autoimmunity can be secondary to another disease (surgery for thyroiditis) but once initiated can then be responsible for continuing chronic disease, or
- The autoimmunity can be the casual factor in disease eg autoimmune hemolytic anemia.

Autoimmunity can affect almost every part of the body and can produce either localised (eg joint tissue in Rheumatoid arthritis) or disseminated disease when the responses are directed against widely distributed antigens such as antinuclear antibodies in SLE. Immune responses to persistent and unrecognised extrinsic antigens, as in chronic viral infections can generate chronic allergic disorders that are difficult to distinguish from anti-self immune reactions.

Most immunogenic preparations are a diverse mixture of antigenic molecules, and antisera produced from such preparations will consist of polyclonal antibody populations that are reactive with their own molecule. Even trace levels (1%) of contaminants in a purified protein preparation will elicit detectable amounts of antibody.

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