

ACQUIRED AUTOIMMUNITY AFTER VIRAL VACCINATION IS CAUSED BY MOLECULAR MIMICRY AND ANTIGEN COMPLIMENTARITY IN THE PRESENCE OF AN IMMUNOLOGIC ADJUVANT AND SPECIFIC HLA PATTERNS

Burton A. Waisbren Sr.*

Waisbren Clinic, 2315 N. Lake Drive, Suite 815, Milwaukee, WI 53211, United States
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SUMMARY Acquired autoimmunity syndromes occur after viral vaccinations.

Molecular mimicry is involved in these phenomena as is the necessity for the presence of two chemically complimentary antigens and an immunologic adjuvant. The HLA pattern of the host is also an important factor.

The example used to explain these phenomena is demyelization disease that follow hepatitis B vaccination. The somatic antigen of the hepatitis B virus in the vaccine has chemical complimentarity with the Epstein-Barr virus antigen in the vaccine recipient. The Epstein-Barr virus shows molecular mimicry with human myelin. The immunologic adjuvant is either present in the vaccine or muramyl peptides in the individual who is vaccinated.

Why more than one type of autoimmune disease occurs is explained by the fact that specific autoimmune T-cells have been shown to develop clones that attack multiple human tissues.

HYPTHESES

A given for this hypothesis is that acquired autoimmune diseases have followed viral vaccination. This hypothesis is based on the published publications of Root-Bernstein, Westall, Fijinami, Oldstone, Wucherfennig, Strominger and others.

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Fujinami, Oldstone, Wucherfennig, and Strominger. They all championed the idea that molecular mimicry was an important factor in the development of acquired autoimmunity. Molecular mimicry may be described as an autoimmune reaction caused by a host receiving an antigen that has amino acids homology with amino acid chains in organs of the host's body. This results in the host's immune system attacking these organs.

Westall and Root-Bernstein hypothesized that a double antigen in the presence of an immunologic adjuvant was fundamental in the development of acquired autoimmunity. Their other requirements for acquired autoimmunity to occur were that one of the antigens must show molecular mimicry with human tissue and that the two antigens must show chemical complementarity 1. In addition, an immunologic adjuvant must be present these four requirements the multiple antigenic mediated autoimmunity (MAMA) syndrome.

Heroelen, de Keyser and Ebinger, and Kaplanski et al. suggested that those who suffered an autoimmune syndrome (MS) after a viral vaccine had certain human leukocyte antigens (HLA) patterns. Their finding makes it reasonable to include in my hypothesis a requirement for certain HLA patterns to be present for acquired autoimmunity to occur after hepatitis B vaccination.

Therefore, my hypothesis includes the following five requirements:

1. A double antigen is formed between two viral antigens that exhibit chemical complementarity to each other. The vaccine contributes one of the antigens and the host supplies the other antigen.
2. One of these antigens must exhibit molecular mimicry with a polypeptide present in the host's tissues.
3. The vaccine antigen and the antigen that is contributed by the host must be chemically complementary to each other.
4. An immunologically active adjuvant must be present. This can be supplied by the adjuvant present in the vaccine (mercury) or muramyl peptides contributed

by the monocytes of the vaccine recipient.

5. The vaccine recipient must have a certain genetic make-up (HLA pattern).

DISCUSSION

In the case of MS following hepatitis B vaccination, the vaccine recipient will have a HLA haplotype of DR2 or B7 or another HLA pattern that is associated with MS.

The antigen that most likely would fit the bill for a molecular mimicry characteristic of one of the antigens would be the ubiquitous Epstein-Barr virus antigen that has been shown to exhibit molecular mimicry with myelin.

The loose end of this hypothesis is that, at least in the case of hepatitis B vaccine, a multitude of acquired autoimmune reactions seem to occur. The recent studies of Lehman et al. and McClain et al. may tie up this loose end. They found that the autoimmune T-cells that emerge in the syndrome lupus erythematosus, lose their autoimmune specificity and have off shoots of T-cell clones that attack multiple tissues. These clones run amok and can be called “rogue” T-cell clones.

Finally, the studies of Gran and Martin have added to the concept of autoimmunity being caused by molecular mimicry by showing that less sequence homology than hereto for was thought necessary for this phenomenon, could be its cause. This suggests that it might be worthwhile to monitor anti-myelin T-cell concentrations after vaccine administration to determine if sub-clinical autoimmunity might be caused by vaccinations due to degenerate T-cells responses and or very limited polypeptide homology.

Two experiments that might support this hypothesis are as follows:

1. It could be determined whether the capsular antigen of the hepatitis B vaccine has chemical complementarity with the Epstein-Barr virus, which is known to exhibit molecular mimicry with myelin.
2. Hepatitis B vaccine along with viruses that exhibit molecular mimicry with myelin and that are chemically complementary with the capsular antigen of the hepatitis B virus can be injected into genetically appropriate mice with adjuvant to see if “experimental allergic encephalitis” (EAE) follows. EAE is an accepted animal model for MS.

The fact that the Guillain-Barre syndrome, another acquired autoimmune disease, follows a number of vaccines, might indicate that the phenomenon of acquired autoimmunity after vaccination is more common than heretofore realized. It also suggests that studies regarding molecular mimicry between indigenous virus and vaccine polypeptides might be in order before a vaccine is released for general use.

REFERENCES

Complete references available upon request from Dr. Waisbren.

waisbrenclinic@ameritech.net

Available upon request from the author is a list of 122 articles from 76 journals by 460 authors from 19 countries that report cases of autoimmunity that followed hepatitis B vaccinations.