# Interaction of electromagnetic fields with infectious disease, including AIDS

Based on mechanism « INH » of antigen recognition by lymphocytes, low power effects of exposure to electromagnetic waves on cancer and auto-immune diseases depend on high/low exposure transitions. These effects are non-existent at constant exposure power. They were observed at power levels of about $10^{-6}$ W/m2 (0.02 V/m=20 mV/m).

But theory predicts that mechanism « INH » can affect antigen recognition at extremely weak power, since spectral power needs only be higher than the spectral power of electromagnetic waves which are of thermal origin. As an order of magnitude, for a signal occupying the full frequency band of 0 to 3 GHz, the threshold power is about de $10^{-8}$ W/m2 (2 mV/m). But for a signal which occupies a smaller bandwidth, the threshold can be lower. For example for a digital TV signal on 8 MHz, an effect can conceivably exist down to $10^{-12}$ W/m2 (0,02 mV/m). Theory predicts that mechanism « INH » may cause a pro-pathogen effect for a constant exposure, independent of any signal variation.

Thus theory predicts a pro-pathogen effect at very low power, at least from 2mV/m (authorized exposure limit is 60.000 mV/m and the threshold recommended by associations is 600 V/m). The fundamental question is thus: how does our immune system still resist pathogens ?

In fact, the existence of very low power effects does not imply that our immune system is necessarily overwhelmed. These effects essentially cause protection gaps to appear: certain antigens are no more recognized by either the humoral or the cellular immune system. But most antigens are still recognized in the presence of low power electromagnetic waves. Therefore, most pathogens are not overly advantaged by the presence of electromagnetic waves.

However, some pathogens are advantaged, due to particular mechanisms. HIV is a virus which evolved in the presence of electromagnetic waves towards a mechanism which optimizes use of protection gaps, by mutating so as to occupy protection gaps in each individual infected host. To best understand HIV, it is necessary to also understand the interaction of other pathogens with electromagnetic waves. This interaction will be described as follows:

1-Exposure to electromagnetic waves creates protection gaps.

2-Antigenic variance allows bacterias and protozoas to take limited advantage of protection gaps.

3-The dual cellular and humoral immune system limits the use of protection gaps by viruses.

4-HIV neutralizes the humoral immune system.

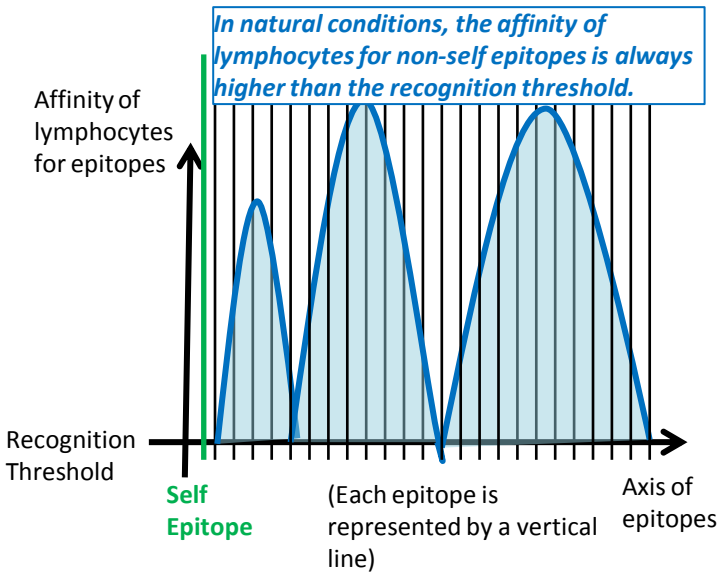
5-In the final phase, HIV escapes immune protection (almost) entirely

**Exposure to electromagnetic waves creates protection gaps.**

Each lymphocyte is capable of recognizing a large number of different epitopes (the epitope is the part of the antigen which is recognized by an individual lymphocyte)

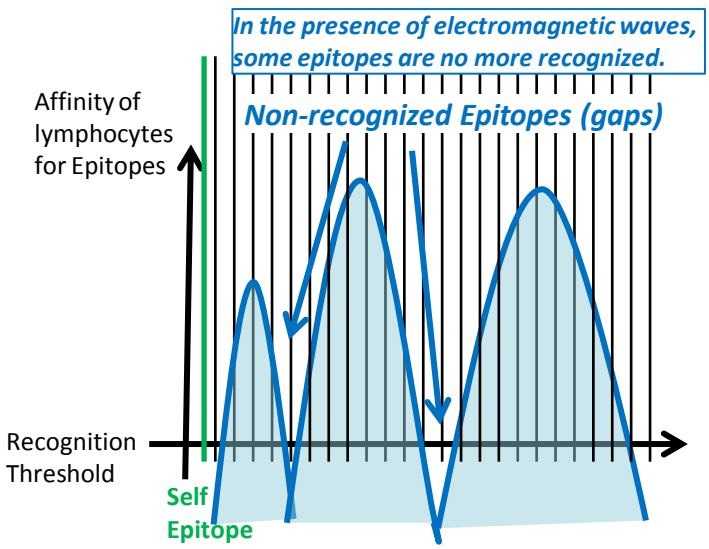
The lymphocyte cover in the absence of electromagnetic waves results from millions of years of evolution and is adapted so that almost any non-self epitope can be recognized.

But this lymphocyte cover represents a cost for the organism and is not over-abundant. The affinity of lymphocytes for some epitopes is positive but very low.




Where electromagnetic waves are present, the affinity of lymphocytes for epitopes diminishes. Some epitopes are no more recognized. Lymphocytes being randomly generated in the bone marrow, non-recognized epitopes depend on the individual host and can differ even amongst twins.

It can occur that all epitopes of a pathogen are non-recognized, in which case the immune system is powerless against the pathogen. But this is unlikely, and even if it occurs the pathogen cannot propagate because if it escapes the immune system of one individual host it will still be caught by immune systems of other hosts.




**In most cases, the protection gaps (non-recognized epitopes) do not strongly affect the propagation and pathogenicity of pathogens.**

**Antigenic variance allows bacterias and protozoas to take limited advantage of protection gaps.**

Some pathogens can within certain limits adapt to protection gaps of different hosts, through an adaptation of surface proteins ( « antigenic variance » ).

For example, certain protozoas (Trypanosoma) or bacterias (Borrelia Borreliosis) can modify their surface proteins. The evolutionary advantage of that capacity was to escape successive responses elaborated by the immune system, by changing the surface protein when the immune pressure was too strong. However in the present situation (with artificial electromagnetic waves) the modifications of surface proteins also allow the pathogen to occupy protection gaps in each host.

This capacity remains limited, due to the limited number of surface proteins that can be generated, and further because other, non-modifiable proteins must also appear at the surface from time to time to ensure interaction with the outer world.

**Protozoas and bacterias that use antigenic variance have an improved capacity to benefit from protection gaps as compared to similar micro-organisms that do not use anigenic variance. This capacity is limited by the number of available surface proteins and by the existence of other, non-variable proteins.**

**The dual cellular and humoral immune system limits the use of protection gaps by viruses.**

Many viruses mutate fast enough to take advantage of protection gaps in each individual host. Mutations affect all viral proteins, and are thus not limited to surface proteins, which gives viruses a particular capability to benefit from protection gaps. Why does it not make viruses highly dangerous in the presence of electromagnetic waves and protection gaps ?

The adaptive immune system comprises two sub-systems, both of which attack viruses (whilst protozoas and most bacterias are attacked only by the humoral immune system).

| **Adaptive cellular immune system:** | **Adaptive humoral immune system:** |
|---|---|
| CD8+ T lymphocytes (effectors) directly eliminate infected cells. Epitopes are produced by restriction (« cutting ») of antigens (viral proteins) by the class I Major Histocompatibility Complex. | CD4+ T lymphocyte are needed to generate antibodies. Epitopes are produced by restriction (« cutting ») of antigens (viral proteins) by the class II Major Histocompatibility Complex. |

An antibody is said to be in a gap of the adaptive (resp.humoral) immune system if no MHC-class I (resp. class II) – restricted epitope of this antibody is recognized by CD8+ (resp. CD4+) T lymphocytes.

But gaps of the cellular adaptive immune system do not coincide with gaps of the humoral adaptive immune system. So generally an antigen cannot be simultaneously in a gap of the adaptive cellular immune system and in a gap of the adaptive humoral immune system. Thus an antigen cannot generally be both in a gap of the adaptive cellular immune system and in a gap of the adaptive humoral immune system. For example dengue or Hepatitis C viruses escape the adaptive humoral immune system by antigenic variation, but they do not simultaneously escape the cellular immune system.

**Viruses have a capacity to use antigenic variation but can only escape one of the two adaptive immune systems (humoral or cellular) which limits their capacity to take advantage of protection gaps.**

**HIV neutralizes the humoral immune system.**

In the adaptive humoral immune system a CD4+ T lymphocyte meets an antigen-presenting cell (APC, for example a dendritic cell or a macrophage) which presents an epitope coming from a pathogen and restricted by the MHC class II. The CD4+ T lymphocyte, if it recognizes the presented epitope, becomes « armed ». If thereafter it meets a B cell capable of producing antibodies against that epitope, it provides the B cells a signal authorizing it to produce antibodies. B cells continuously need signals from CD4+ T lymphocytes so as to manufacture antibodies. When a pathogen is entirely eliminated, CD4+ T lymphocytes do no more meet epitopes from that pathogen and can no more activate B cells producing antibodies against that pathogen, so that production of these now useless antibodies ceases.

An antigen presenting cell (APC) which presents HIV epitopes has been in contact with HIV and is often infected. When the CD4+ T lymphocytes come near the APC to recognize the epitope, they often becomes infected, because CD4+ T lymphocytes are the main target of HIV. If a CD4+ T lymphocyte is not infected in this manner, it can be infected through other pathes. In follicles where B cells produce antibodies, a number of CD4+ T lymphocytes supply B cells with the positive signals which they need to produce antibodies, but these lymphocytes are infected by HIV. When B cells produce anti-HIV antibodies, these antibodies bind armed CD4+ T lymphocytes, which are then destructed by the immune system. B cells then cease antibody production due to the lack of CD4+ armed lymphocytes. The initial production of anti –HIV antibodies ceases.

But HIV can mutate. When CD4+ T lymphocytes become dominantly infected by a new HIV variant, the production of antibodies directed against the ancient variant resumes insofar as there remain enough armed CD4+ T lymphocytes or enough exemplaries of the ancient variant of HIV to generate an immune response. This antibody production is not directed against the new HIV variant but against the old variant, and does not significantly affect the new variant. It does not cause destruction of armed CD4+ T lymphocytes which emit positive signals towards B cells as these lymphocytes are not dominantly infected by the new HIV variant but by the ancient variant.

**HIV neutralizes antibody production directed against the current dominant viral variant. But antibodies directed against ancient variants can be produced.**

This system accelerates the elimination of ancient and non-optimal viral variants and thus the mutation of the virus.

**In the final phase, HIV escapes immune protection (almost) entirely**

HIV neutralizes the adaptive humoral immune system. It is thus only attacked by the adaptive cellular immune system. HIV variants which best survive have antigens which occupy protection gaps of the adaptive cellular immune system, i.e. antigens which MHC class I- restricted epitopes are not recognized by CD8+ effector T lymphocytes.

After a sufficient number of mutations, the dominant HIV variant yields antigens which are non-recognized by the adaptive cellular immune system. The dominant variant of HIV is thus not recognized at all by the adaptive (celluar and humoral) immune system.

Most protection gaps are near self antigens because self antigens are themselves non-recognized (because negative thymus selection eliminates lymphocytes that recognize self antigens) and immune protection is thus generally weaker near self antigens. Thus the dominant HIV variant presents essentially near-self antigens, and it is little attacked by the innate (non-adaptative) immune system, which does not have a sufficient fine recognition capacity to discriminate self antigens from non-self antigens.

**After a sufficient number of mutations, the dominant HIV variant escapes the immune system almost entirely. There is no barrier to the proliferation of the virus, which finally causes death.**