

Vaccinations and Smallpox: What you need to know  
by Philip Incao, M.D. *Tuesday December 10, 2002 at 05:10 PM*

## Reflections On Immunity, Vaccinations And Smallpox

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Part One: The Phenomenon of Immunity

Illness is a process that everyone experiences repeatedly in one's lifetime. Until our modern era, illnesses were classified according to their recognizable signs and symptoms. Today, in addition to the outward appearance of an illness, we also classify it according to its unique features detectable with the microscope and with biochemical tests. Thus many illnesses of similar or identical appearance which were lumped together in the past can now be distinguished from one another based on their microscopic or biochemical features. For example, what for hundreds of years was called influenza is now described as a group of "influenza-like illnesses," each one associated with a different virus.

On the other hand, many diseases known for centuries and recognizable by their typical signs and symptoms have been confirmed by modern science to be distinct entities, i.e. to be associated each with its own particular virus or bacterium and with no other. Measles, chicken pox and scarlet fever are examples of these.

It has long been known that in some illnesses such as these, one experience of the illness usually confers lifelong immunity. A second experience with measles or scarlet fever is extremely rare.

These observations by physicians and patients throughout history, as well as careful observations of the stages in a patient's recovery >from an acute inflammatory illness like measles or scarlet fever, have led to certain basic concepts in medicine.

One of these concepts was formulated as "Hering's Law" in the 19th century, although it was well-recognized and mentioned by the ancient Greek physician Hippocrates. This law states that as an illness resolves, its manifest signs and symptoms travel from the inner vital organs and blood circulation to the outer surface of the body, often visible as a rash or as a discharge of blood, mucus or pus. In this way we "throw off" an illness.

Another basic concept arising from the phenomenology of illness, i.e. >from observations of the directly perceptible behavior of human illness, is the concept of immunity to or protection from an illness that one has had before.

This immunity to second episodes of certain illnesses like measles or scarlet fever reveals a knowing function of the human being in relation to illness. This inner knowing allows us, without any conscious knowledge or effort, to recognize an illness we've had before and to thereby resist it or quickly repulse it.

Hering's law on the other hand is evidence of an innate doing function of the human being in healing, i.e. we actively clear the illness from our body, we get it out of our system as we heal. These inner activities of doing and knowing work more strongly during illness than in the healthy state, and they were clearly recognized by the ancient physicians. Hippocrates said illness consisted of the active element *pónos* (labor) as well as the passive element *pathos* (suffering). Illness is intense inner work. Hippocrates perceived this labor as a cooking and digesting (*pepsis*) of our inner poisons during an inflammatory illness. Today we regard our inner work as a battle against a hostile virus or bacterium. The all-too-often overlooked point however, is that it is we ourselves who inwardly, unconsciously determine whether or not to engage in the battle. The great medical pioneer Hans Selye, M.D., who introduced and elucidated the role of stress in health and illness explained, "Disease is not mere surrender but also fight for health; unless there is fight there is no disease (emphasis mine)." 1

The symptoms of an acute inflammatory-infectious illness begin not when we are infected by a virus or bacterium, but when we respond. The magnitude of our response is influenced not only by the magnitude of the infection, but also by the inherent strength of what is responding in us. For the ancient physicians the responder in us was an aspect of our human spirit and our inner vitality; our inner healing force. Today the physical basis of our inner responder is what we call our immune system. The phenomenon of immunity hasn't changed, but our thinking about it has.

The severity of the early symptoms of a particular illness is directly proportional to the vigor of our immune response and indirectly to the burden and noxiousness of the infection to which we are responding. The surprising fact is that most of the symptoms of an infectious disease are caused not by the germs themselves but by our own activity of the immune system in fighting the germs. The germ "invasion" of our body is often silent, and can take place gradually over a long period of time without disturbing us. It is only when our immune system decides to do battle with the encroaching germs that we start to feel sick.

The metaphor of battle is a convenient, but not fully accurate description of the relationship between our immune system and the proliferating viruses or bacteria during an acute inflammatory/infectious illness. Pasteur's germ theory assumes that disease germs have a predatory nature: that they prey on our flesh for their own survival, while contributing nothing to us in return. The germ theory further assumes that the harmful or lethal effects of infectious/inflammatory diseases are a direct result of this predation of the human body by germs.

In early microscopic studies of host tissues in acute inflammatory/infectious diseases, Pasteur, Koch and their colleagues repeatedly observed that germs were proliferating while many host cells were dying. They made the critical assumption, upon which all further thinking has been based, that the germs attack and destroy otherwise healthy cells, thus causing direct harm to the human body.

It would have been equally justified by the observable facts to assume that the cells were dying for inapparent biochemical reasons and that the proliferating germs were attracted to the site of increased cell death and decay just as flies, crows and vultures are attracted to death in outer nature. A choice was available early on between regarding germs as predators and regarding them as scavengers. The nineteenth-century thinking of the time was captivated by the Darwinian images of "Nature red in tooth and claw" and the relentless struggle for survival. The decision to see germs as predators was perhaps inevitable, and that has made all the difference in our current thinking about illness and health. That early decision by Pasteur and his followers led to medicine's present nearly-exclusive focus on combating germs, while neglecting all the subtle but far-reaching ways to strengthen the host against lasting harm from inflammatory/infectious illness.

Just as flies, crows and vultures were regarded by the Native Americans as playing a necessary and helpful role in the great chain of Being, so too with germs which scavenge death and decay within our bodies. The true causes of inflammatory/infectious illnesses will ultimately be found to reside not in the germs, but in the various human frailties which allow the forces of death and decay to predominate in us. The scavenging germs are the markers of our waxing and waning states of physiologic imbalance when cell death and decay temporarily exceed their normal limits.

The metaphor of battle between immune system and germs is justified provided we remember that our real enemies are the forces of death and decay. The germs themselves become sacrificial victims marked for destruction by our immune system because their role is to absorb the products of death and decay. Germs become poisonous to us through embodying the poisons we create. In "battling" germs, the real battle is to overcome ourselves and to refine our nature. This concept is implicit in the following discussion of how our immune system does battle with germs.

Using battle as our metaphor, we can imagine three possible scenarios. In the first, the attacking army is not strong, but the defenders are, and the attackers are routed from the field in a bloody but one-sided and brief battle in which the defenders suffer no casualties. This describes a typical case of a benign but acute inflammatory-infectious illness like roseola which usually expresses itself in a very high fever of 105° or 106°F and an extensive rash despite being no threat whatsoever to the host.

A second scenario would be when the opposing armies are evenly matched and there is a fierce battle with many casualties on both sides. This could describe an acute life-threatening inflammatory illness like septicemia or an overwhelming pneumonia, in which recovery or death is equally likely.

In the third scenario, the war reporter arrives late at the battlefield and finds no carnage, in fact little or no evidence of any previous battle. The defending army is quiet and no attackers can be seen. The reporter at first concludes that it was a very quick and easy victory for the defenders and the attackers have fled. On closer investigation, however, he finds that no battle took place

because the defenders were unable or unwilling to fight. What our reporter at first thought was the defending army in reality consists of non-combatant defenders who have been quietly and massively infiltrated by the attackers. The attackers blend in, occupying the defenders' homeland, and any defenders who would fight them have gone underground where they intermittently harass and provoke the occupying enemy.

The point of this elaborate metaphor is to demonstrate by analogy that the absence of fevers and other symptoms and signs of inflammatory illness (the absence of a battle) does not always mean that our immune system (the defending army) has been victorious!

Today it is more often the case that when we don't fight our battles vigorously and often enough, i.e. when our fevers and discharging inflammations are very seldom and mild, then we are liable to be infiltrated by the enemy in disguise and suffer from chronic allergic or autoimmune disorders. This concept today is called the hygiene hypothesis. In the 1920's Rudolf Steiner expounded essentially the same concept as a mutual interplay between opposing forces of inflammation and of sclerosis, in which the healthy state is a dynamic balance between the two.

Returning to our third scenario, there are of course times when the absence of a battle, i.e. absence of obvious disease symptoms, indeed does mean that the defending army has easily routed the enemy and is truly immune from further attack. Thus we see that two entirely opposite outcomes, 1. immunity from attack and 2. quiet infiltration by the attackers into the defenders' homeland (the host body) can have the exact same appearance superficially. This analogy applies precisely to another pair of similar-appearing but inwardly opposite states, i.e. the true immunity conferred by overcoming illness as opposed to the apparent immunity conferred by vaccination. In both cases the host appears to be healthy due to the absence of illness, but true health is much more than the absence of overt illness. We will illustrate this point further when we discuss smallpox in part 3.

To complete our phenomenological description of immunity, we must note that in addition to the functions of clearing illnesses from the body and of recognizing the illnesses it has previously encountered, the immune system has another cognitive or knowing capacity. This is the discrimination of self from non-self and the ability to "tolerate," i.e. to not treat as foreign and to not react to, any elements of self. This remarkable knowing of the immune system also extends to its ability to tolerate, in pregnancy, a massive foreign presence in the body, the fetus, without reacting to it at all.

Thus we see the incredible skill and apparent purposefulness of doing and the discriminating capacity of knowing possessed by the immune system. Although modern science rarely uses the words "knowing" and "doing" in its descriptions of the immune system, nevertheless distinct knowing and doing functions are very clearly and unavoidably implied in all scientific writing on immunology. Science prefers to focus on the molecular level, hoping to find in molecular events the elusive key to understanding, if not why, at least how the immune system does what it does.

Today the immune system is most often described in articles and textbooks as comprising those bodily organs, cells and functions which discriminate between self and non-self. The molecules of self or non-self which the immune system can recognize are called antigens. One branch of the immune system, called the humoral immune system, consists primarily of antibodies which are protein molecules made by the body to specifically interact with foreign antigens. Antibodies attach themselves to any foreign antigens like bacteria or parasites which may exist in blood or body fluids outside of the body's cells. Antibodies are attracted to such extracellular antigens and usually coat these antigens as one step in the complex process of the destruction, digestion and elimination of foreign matter in us by our immune system.

We come now to a beginner's question, one seldom or never asked in the science of immunology. It is, why does our immune system work in such an inconsistent way, providing for permanent immunity from recurrence only after certain illnesses and not after others? A "why" question such as this is usually considered irrelevant in modern science, while the equivalent "how" question is actively pursued. In the case of immunity to illness, it is the "how" questions that have led science to the idea and the practice of vaccination.

For science the pertinent question is, how can we imitate nature and bring about lifelong immunity to an infectious-inflammatory illness, but without having to experience the illness first? The first task would be to learn exactly how nature itself manages to maintain permanent immunity in us after a first experience of illness. What is this process of lifelong maintenance of resistance to a particular illness? Can science duplicate it?

## Part Two: How Do Vaccinations Work?

It is an interesting fact that sometimes a practical scientific breakthrough happens out of an intuition, a hunch, long before the discoverer or anyone else is able to explain just how and why this particular breakthrough works. This is true of the work of Jenner and Pasteur, the great initiators of the practice of vaccination. Astoundingly, in our modern era when vaccinations are so widely acclaimed and practiced, science still cannot explain how they work.

In the New Scientist magazine of May 27, 2000, an article on AIDS vaccine research quotes the following from two scientists: "I'm amazed by the amount of basic science we don't know," and "the assumption that successful vaccines work by simply producing antibodies is almost certainly wrong." The article then describes how one vaccine researcher found that in a certain viral disease of horses, vaccination was successful in inducing antibodies against the virus, nevertheless the vaccinated horses died faster than the unvaccinated ones! Referring to our present ignorance as to just why these vaccinated horses would succumb, he stated, "It's an issue people haven't wanted to think about, but we might have to."

Vaccine science and practice have always been based on certain assumptions, which we are only now beginning to examine. One of these is that antibodies in the blood (humoral immunity) confer protection against an illness, and that the level of antibodies correlates with

the degree of protection. This relationship between measurable antibodies in the blood and apparent protection >from illness has been observed for decades in many types of infectious diseases. It is not known however whether the antibodies persisting in the blood for months or years after an infectious disease are themselves responsible for protecting us from recurrences of that disease or whether they are merely markers of a protection that is accomplished by another part of the immune system. It is also not known whether the apparent protection associated with vaccination-induced antibodies is a benefit pure and simple or whether a hidden cost to the immune system is involved. The idea of a hidden cost is considered unthinkable by vaccine researchers for obvious practical reasons, yet it continues to be a nagging doubt among an ever-widening circle of parents, consumer advocates, chiropractors, holistic physicians and other discerning people.

The AIDS research quoted at the beginning of this article suggests that it's not the antibodies which protect us, but rather it's the cellular immune system. Also called the cell-mediated immune system, it comprises the white blood cells, all the lymph nodes and lymphatic tissue throughout the body and is concentrated in the thymus, tonsils, adenoids, spleen and bone marrow. It is generally agreed that the primary function of the cellular immune system is to destroy foreign intracellular antigens like viruses and some bacteria as well as the cells that harbor them. This is accomplished by the various white blood cells which are able to move inside, outside and through the walls of our blood vessels and to access every part of the body.

In the past I have been tempted to assign the immune system's doing function to the cell-mediated branch and its knowing function to the humoral antibody-mediated branch. This neat division of function is not borne out by the facts. Research shows us that each branch participates in functions of both knowing and doing, although most of the immune system's muscle to destroy, digest and drive out intruders is flexed by its cell-mediated branch. Thus, while immune system functions of knowing and doing may be conceptually distinct, in the physical reality they are overlapping in an exceedingly complex orchestration of organs, cells, molecules, hormones and chemical messengers.

There are also other aspects of the immune system which are beyond the scope of this article. Reading a modern textbook of immunology can be frustrating as one finds a bewildering array of cellular, molecular and antibody-mediated processes which science has discovered without knowing how they all fit together and manage to cooperate in health and in illness in the human being. It's something like hoping to find an understanding of how an automobile performs by studying its disassembled parts in an auto parts shop.

At the present time, it is thought that the encounter between self and non-self, that is, between the immune system and a foreign "invader" like a virus or bacterium begins in the domain of the cellular immune system with a cell called the antigen-presenting cell. If the foreign guests are not great in number or in their noxiousness, the cellular immune system is able to dispatch them, digest them and clear them from the body without ever calling into action its coworker the humoral or antibody-mediated immune system. This explains the very important fact that without our awareness we are continually infected with many small numbers of different germs

in our body, some of them nasty, and the cells of our immune system continually shepherd them and keep them in check without the assistance of antibodies.

Like dust and other unseen debris, these microorganisms enter our bodies as we breathe, eat and drink. Only when the number or rate of growth of germs exceeds a certain threshold are they then recognized by the humoral immune system, resulting in the formation of antibodies specific to the particular provocative bug. At this stage we may have only mild fleeting symptoms or none whatsoever. This explains how we may be found to have antibodies against illnesses we don't remember ever having had! This is called "subclinical infection," i.e. infection without symptoms, and it happens commonly.

Thus science has discerned three levels of infection. The lowest level is our steady-state equilibrium of everyday life in which we peacefully co-exist with our inner menagerie of germs without needing to form detectable antibodies against them. At this lowest level our cellular immune system is quietly busy keeping our bugs in line and when necessary pruning the flock. Thus, although small numbers of disease agents are within us, our cellular immune system sees to it that we remain well and free of disease symptoms, and that our germs are under control.

At the second level of infection, we temporarily relax our vigilance and allow a certain group of germs to begin rapidly multiplying to the point where the humoral immune system is alerted and begins to produce antibodies against the offending bugs. This sets off a cascade of immune system functions which succeed in fairly quickly quelling our rebelling germs, so quickly that the person hosting all these inner happenings is unaware of having just gone through a subclinical illness. The identity of the wayward germ can afterwards be diagnosed by the presence in the blood serum of the specific antibodies produced against it by the humoral immune system.

At the third level of infection things get seriously out of control and all our inner alarm bells go off as a tribe of germs proliferates wildly and provokes the full defensive reaction of our immune system. This is called the "acute inflammatory response," which usually includes fever, release of stress hormones by the adrenal glands, increased flow of blood, lymph, mucus, and a streaming of white blood cells to the inflamed area. The human host of these wisdom-filled events now feels sick and may experience pain, nausea, vomiting, diarrhea, weakness, chills and fever. We have now emerged from the realm of the subclinical to a full-blown clinical illness, with all of its intense and often frightening symptoms. It is critical to a healthy understanding of these things to realize that we never merely suffer through an illness in a passive, one-dimensional way. In an acute illness, parts of us that in health are most active, like our mind and our muscles, are subdued, while other parts like our blood, glands and immune system are much more active than normal. Thus every illness rouses us to become more inwardly active than usual, and this inner activity of ours is the cooking through, the sweating out and the throwing off of the illness. This is hard work, and every illness calls upon and exercises capacities in us which otherwise would have remained dormant. Adults often notice these new capacities as a change in attitude or outlook after an illness. Children often manifest

positive changes in their behavior or development after overcoming an acute inflammation or fever.

Having successfully passed the challenge of a particular illness, we may not need to experience it again. Something about the illness and our response to it has made us immune to its recurrence. If we knew what that something was, perhaps we could learn how to use it to create health and prevent illness. Of course, this is the basic concept of vaccination, but the all-important question is, does vaccination accomplish what we think it does?

We've already suggested that it's probably the cellular immune system, and not antibodies, which protect us against illness. Surely antibodies can have no role in either preventing or overcoming first bouts of infectious-inflammatory illness, because they are formed only after the illness has peaked. It must be the cellular immune system which confers the resistance to, as well as the capacity to overcome, both first episodes and subsequent episodes of infectious disease. To understand how this might happen, it is helpful to examine more closely the very illness and its vaccination which started the whole debate: smallpox.

### Part Three: Smallpox And Its Vaccination

That vaccines can confer a degree of protection from certain infectious-inflammatory illnesses is clear. What is not clear, as mentioned earlier, is exactly what vaccinations do to the immune system to bring about their protective effect. Researchers generally agree that vaccines do not prevent the particular virus or bacterium from entering the body nor from beginning to multiply within it. It is thought instead that the vaccines stimulate or "prime" the immune system to quickly eradicate the offending germ soon after it begins to infect the host.

Let us consider how this process might work in the case of smallpox. Our knowledge about smallpox and its vaccination is based on over 200 years of study of this dramatic and much-feared illness by physicians in many countries.

The natural course of the illness begins when one "catches" smallpox from someone with a smallpox rash or from the mucus or pus of smallpox on a patient's bedclothes or dressings. For the next twelve days there are no signs or symptoms at all and the new patient is not contagious even though the smallpox virus is multiplying within the body. On or about the twelfth day large numbers of smallpox virus enter the blood (viremia) and the "toxemic" phase of the illness begins, meaning a poisoning or contamination of the blood circulation. This blood poisoning of smallpox is the beginning of the overt illness, with symptoms of fever, prostration, severe headache, backache, limb pains and sometimes vomiting. After three or four days of these symptoms the typical smallpox rash begins to erupt and in the next one to two days the fever falls to almost normal and the patient feels much better.

The skin eruption begins as red spots which over the next few days evolve into raised pimples, which then change to blisters which then become pus-filled (pustules). On the 11th to 13th day of the illness the pustules begin to dry up and form crusts or scabs which then fall off by the end

of the third week of the illness. The fever usually returns, less severely, after the pustules appear and then becomes normal as the crusts and scabs form. If one dies >from smallpox, it may be in the first week of the illness if the toxemia is very severe, but most smallpox deaths have occurred toward the end of the second week after the pustules appear.

The majority of smallpox patients survive, and the falling away of the dried-up scabs from the skin signifies the final stage of healing, approximately 33 days after catching the infection. The dramatic course of smallpox illustrates very well some of the concepts discussed earlier in this article. The twelve-day incubation period during which the smallpox virus actively multiplies in the body without provoking the slightest symptom confirms the point that it is our response to infection, not the infection itself, which causes the typical disease symptoms of fever, aches and pains and extreme weakness.

The fact that the fever drops and the patient feels much better after the rash breaks out illustrates Hering's Law. The poisons circulating in the blood during the toxemic phase cause the most severe symptoms of smallpox. These symptoms improve considerably once the blood clears out its poisons by discharging them through the skin, producing the typical pus-filled blisters of smallpox. The chief danger of smallpox consists in the degree of blood poisoning and in the huge and exhausting effort required for the immune system to push the poisons out of the blood and through the skin. When the toxemia, the poisons, are overwhelming and the patient lacks the strength to discharge them out of the body, then the patient may die in the effort, either before the eruption ever appears or else, utterly spent, afterwards.

The patients who survive smallpox will have lifelong neutralizing antibodies to smallpox virus in their blood and permanent immunity to a second episode of the illness. What does this mean?

Using the battle metaphor from part one, we could say that the victorious defending army has acquired much valuable skill, know-how, and confidence through its combat experience as well as certain medals awarded to acknowledge their participation in combat. The first three attributes are comparable to the inner strengthening of the cellular immune system which is attained through overcoming an illness like smallpox. The medals as visible tokens of achievement are roughly comparable to the antibodies visible on simple blood tests indicating that the host has already won that battle and is likely to be immune to future attacks of the same illness.

If a foolish general were under the illusion that merely wearing a combat medal actually conferred the know-how, skill and confidence gained in battle, then he might propose pinning medals on soldiers with no combat experience to make them immune to dangerous future battles. That would bestow the same outward appearance to the seasoned and unseasoned soldiers alike, belying their experience.

In the same way, science bestows antibodies through vaccination and mistakenly assumes that it is bestowing the immune strength that can only be developed through the experience of illness.

In equating the significance of vaccine-induced antibodies with that of illness-induced antibodies, science confuses the outer sign of the battle experience with the experience itself. Antibodies arising through illness are markers of immunity and (unlike the medals in our battle metaphor) also contribute to immunity, but antibodies alone are not sufficient to confer lasting immunity to a particular illness. There are several diseases which may recur repeatedly, such as herpes outbreaks, despite high antibody levels. The evidence suggests that it is our cellular immune system which confers lasting immunity, with antibodies playing a secondary role in the process.

Immunity is really the result of our experience, of having gone through, along with our cellular immune system, an active process (the combat in the metaphor) of learning and strengthening. The immune system is a limb of us, and it learns from experience just as we do. Antibodies signify that we've had experience of illness, often repeatedly, but not necessarily that we've gained anything from the experience. When on some level we respond with greater initiative to our experience of illness, actively processing, digesting and ultimately learning from such experience, then we are usually immune from having to repeat it. In such cases our cellular immune system has strengthened itself through its active encounter with, and overcoming of, the illness. In this view, immunity is the result of having successfully met the challenge of a particular illness and having gained mastery over it. It is like learning a particular skill, such as riding a horse, which is then usually retained for life. On the physiologic level, the skill and mastery we gain in overcoming illness accrue to our cellular immune system.

This active process of acquiring mastery cannot be replaced by a vaccination unless the host's immune response to the vaccination is essentially identical to its response to the illness itself, even though reduced in intensity. This would mean that in order to produce genuine cellular immunity, a vaccination would have to reproduce the experience of the illness, causing some of the same signs and symptoms, though milder, that are caused by the illness. To see if this is true, let us look at smallpox vaccination.

The vaccination consists of introducing live cowpox (vaccinia) virus into the skin by multiple superficial punctures in a small area about 1/8 inch diameter on the upper arm. The vaccination site is then inspected twice after 3 and 9 days to determine if the vaccination "takes" or not. A primary reaction or "take" evolves as follows: for three days after the vaccination there is no reaction whatsoever. On the fourth day a small red pimple appears which gradually grows into a blister which becomes pus-filled, surrounded by a zone of redness and often with tender, swollen glands in the armpit and mild fever. This reaction peaks on the 8th to 10th day, after which the pustule gradually dries up and forms a scab which eventually falls off leaving a scar.

Clearly the primary "take" reproduces the experience of smallpox itself described earlier, but of course in a very limited way so as to generate only one pock rather than many dozens of them. The cellular immunity produced by smallpox vaccination is also limited, lasting from six months to three years. This immunity probably coincides with the length of time that the exercised "muscle" of the cellular immune system remains strengthened from its labor of discharging the single cow pock resulting from the vaccination. The antibodies appearing in the

blood after primary smallpox vaccination may remain for over ten years, but these antibodies cannot be taken as a trustworthy sign of immunity. The official description of the currently available smallpox vaccine in the U.S., which was manufactured by Wyeth Laboratories, states vaguely "the level of antibody that protects against smallpox infection is unknown"<sup>2</sup> If we can state blandly that the protective level of antibody is still unknown after having assumed for several decades that protection is directly correlated with antibody level, then surely it is time to rethink that assumption.

In practice antibody levels were seldom used in the smallpox era as a measure of immunity. Anyone not vaccinated in the previous three years was considered to be susceptible to smallpox, regardless of their antibody level.

The all-important question is how to interpret the meaning of reactions to smallpox vaccination which are milder and briefer than the primary "take" which peaks in ten days, and which does result in a genuine though short-lived immunity of the cell-mediated system.

Since the early 1970's only two types of reactions to smallpox vaccination have been officially recognized, as recommended by the World Health Organization (WHO). For purposes of greater clarity, in this discussion I will be referring to the older classification which recognized three types of normal reactions to smallpox vaccination.

The second type of normal skin reaction to smallpox vaccination was called the accelerated or vaccinoid reaction, usually in people who had some immunity to smallpox at the time of vaccination, either from a previous experience of the disease or from a previous smallpox vaccination. In the accelerated reaction, the skin blister which forms is smaller and reaches its maximum size and intensity between the 3rd and 7th day after the vaccination. This reaction works in exactly the same way as the primary reaction but to a lesser degree, boosting the cell-mediated immunity that is already present, but waning, from the previous vaccination.

It is the third type of reaction to smallpox vaccination that in my opinion has created all the problems, that has been at the root of a 200 year old controversy over the usefulness of smallpox vaccination. This stems from the fact that this reaction for years was interpreted as indicating immunity to smallpox, when it often meant exactly the opposite. In many cases the bearers of this reaction may have had a suppressed cellular immunity, making them on repeated revaccination more susceptible to smallpox than an unvaccinated person!

This third type of reaction to smallpox vaccination was originally called an immune reaction, then later renamed an early or immediate reaction. A small pimple forms at the vaccination site which may evolve into a tiny blister, peaking on the second or third day and diminishing thereafter. An earlier textbook of viral diseases >from the smallpox era states the following: "The early or immediate reaction is an indication of sensitivity to the virus and may be given by persons who are either susceptible or immune to smallpox.[It] cannot be regarded as a successful result and cannot be guaranteed to induce or increase the person's resistance to smallpox."<sup>3</sup> This is a typical scientific understatement that glosses over years of devastating

results of smallpox vaccination in which thousands of vaccinated people who were thought to be immune based on their so-called "immune reaction" to vaccination later caught smallpox and died.

Ian Sinclair, writing on the history of smallpox, states:

"After an intensive four-year effort to vaccinate the entire population between the ages of 2 and 50, the Chief Medical Officer of England announced in May 1871 that 97.5% had been vaccinated. In the following year, 1872, England experienced its worst ever smallpox epidemic which claimed 44,840 lives. In the Philippines, prior to U.S. takeover in 1905, case mortality [death rate] from smallpox was about 10%. In 1918-1919, with over 95% of the population vaccinated, the worst epidemic in the Philippines' history occurred resulting in a case mortality of 65%. The 1920 Report of the Philippines Health Service [stated] 'hundreds of thousands of people were yearly vaccinated with the most unfortunate result that the 1918 epidemic looks prima facie as a flagrant failure of the classic immunization toward future epidemics.'"<sup>4</sup>

How can this be? How can these historical facts be reconciled with my earlier statement that a primary take in response to a first smallpox vaccination results in genuine cellular immunity for up to three years? The usual explanation offered is that the vaccine used was inactive due to loss of potency in storage, but this clearly cannot be the whole answer to the many documented instances of failure of smallpox vaccination to protect from smallpox.

The answer is an open secret which has been very well known for years, but never fully understood: that many first recipients of smallpox vaccine fail to produce a take (primary reaction) and continue to fail to do so even when revaccinated many times. The textbook states,

"Easton (1945) records of one man who died of confluent smallpox that vaccination had been attempted at birth, again in 1941 and ten times in 1943 without a take, thus emphasizing the danger of accepting even repeated unsuccessful vaccination as evidence of insusceptibility to smallpox.." <sup>5</sup>

This is an excellent example of a vitally important observation leading to an irrelevant, though not incorrect, conclusion. This example begs the question: how many repeated failures to react does it take to justify the concern that continuing to revaccinate may be doing more harm than good?

The relevant conclusion, in my opinion, is that due to differences in immune response capability among individual human beings at the time of first vaccination, in some individuals the cellular immune system lacks the muscle to push out the single pock eruption that is the primary take. The scratching of the virus into the skin of the arm is a strong challenge to the immune system. A successful take depends on the ability of the cellular immune system to respond to that challenge in an equally vigorous way, to push the intruding virus right back out of the body. It is a simple matter of action and reaction, of challenge and response. If Charles Atlas challenges a 97-pound weakling to arm wrestling and his opponent's arm immediately collapses, we would

not think that the challenge ought to be repeated indefinitely if the weak condition of the responder had no means of improving! Yet in thousands of individuals in the last 200 years who may have been weakened through stress, poor nutrition and poverty, whose cellular immune systems were not vigorous enough to respond to smallpox vaccination with a take, the effect of repeated revaccination, which was commonly practiced, was to weaken these individuals' immune systems still further, making them no doubt more vulnerable to smallpox than they had been before vaccination! This would explain the disastrous results of the above-mentioned smallpox vaccination campaigns in England, the Philippines and in many other countries as well.

The ambivalent nature of the early reaction to smallpox vaccination is analogous to the third battle scenario mentioned in part one of this article. When little or no signs of battle (reaction) are visible, it may mean that the defenders were easily victorious (the host is immune) or contrariwise it may mean that the defenders lacked the strength to fight and their homeland was subsequently quietly infiltrated by the attackers. When a smallpox vaccine recipient lacks the immune muscle to respond to the viral intrusion of his or her body with a vigorous pock-forming discharge, then we might expect that most of the intruding virus has remained in the body. With each revaccination the burden of vaccinia virus in the body increases, and the suppressive effect of this viral burden on the cellular immune system also increases, eventually resulting in a dangerous state of immunosuppression. This may also explain the occasional catastrophic effects that were observed resulting from a brief medical fad in the 1970's: treating recurrent herpes infections with repeated smallpox vaccinations.

The disease smallpox and its vaccination are fruitful subjects to study in order to understand how the immune system works, because we can observe what happens on the skin as vital clues to what might be happening inside the body. The main lesson from this study is the exceedingly important fact that a lack of a vaccine reaction, and by extension a lack of illness symptoms, can by no means be taken as a sign of immunity or of health.

The other critical fact confirmed by our historical experience with smallpox vaccination is that individual differences in response to vaccination are extremely important. One size most definitely does not fit all. It is clear that although the smallpox vaccine was effective in conferring a temporary immunity in some individuals, an unknown number of other individuals were probably harmed by the vaccine. With the smallpox vaccination the adverse effects were fairly obvious, they often appeared on the skin. With other vaccines in use today the adverse effects may not be so obvious. We've seen with smallpox that the same vaccination procedure which temporarily strengthened the cellular immune system in some individuals probably weakened it in others, especially upon repeated revaccination.

The possibility, that the up to 39 doses of 12 different vaccines which children today receive by school entry may be impacting the cellular immune systems of many individual children in a negative way, suggests itself to the open mind. Science has most of the knowledge and the tools it needs to investigate and to find answers to these unanswered questions. All it needs now is the will. May it come soon, for our children's sake.

1 Selye, Hans. The Stress of Life. New York: McGraw-Hill, 1978, p.12

2 <http://www.cdc.gov/mmwr/preview/mmwrhtml/rr5010a1.htm>

3 Rivers, T.M., and Horsfall, F.L., Jr. Viral and Rickettsial Infections of Man. Philadelphia: Lippincott, 1959, p.686.

4 <http://www.whale.to/cvaccines/sinclair.html>

5 Rivers, T.M., and Horsfall, F.L., Jr. Viral and Rickettsial Infections of Man. Philadelphia: Lippincott, 1959, p.687.

[http://www.vaccineinfo.net/issues/how\\_vaccines\\_work.htm](http://www.vaccineinfo.net/issues/how_vaccines_work.htm)

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