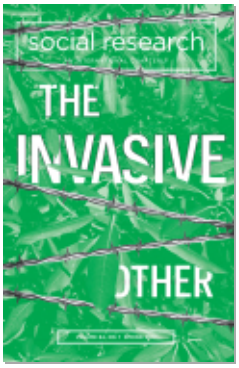
Epidemics and Xenophobia, or, Why Xenophilia Matters

A. David Napier



Social Research: An International Quarterly, Volume 84, Number 1, Spring 2017, pp. 59-81 (Article)

Published by Johns Hopkins University Press

A. David Napier

Epidemics and Xenophobia, or, Why Xenophilia Matters

SELF AND OTHER

Research on the relationship between self and other is today mediated by new knowledge that forces us to ask questions regarding connections between the biological and social sciences. Such research also demands a reconsideration of the possible effects of answering those questions on age-old forms of discrimination and xenophobia. Indeed, today we stand at a crossroad that demands our rethinking the ancient idea that foreigners (refugees, immigrants) carry diseases and should be feared—that foreigners are, primarily, *invasive*.

If science is to be taken seriously, not only is this picture incorrect, but also the metaphors that sustain this view may be equally wrong. The old moral divide between those who dislike outsiders and those who behave charitably toward them needs retooling, given what new science suggests about the relevance of others to individual health. The new field of epigenetics has amply shown how social encounters can influence genetic makeup over generations—that is, we do not always revert to the same inherited genetic blank slate with each coming generation. In addition, other, new forms of scientific knowledge have shown how our ability to thrive is as much a function of the way we adjust symbiotically to environmental and social stimuli as it is of evolutionary adaptations that affect our fitness.

Though it is hard to say what future effect the rethinking of biodeterminism will have on our views of self and other (Napier 1996; 2017), it is also clear that old assumptions die hard. Right-wing nationalism has re-emerged, as have xenophobia and antimigration rhetoric, alongside counterepidemic hysteria that attributes new diseases and their mutations to the behaviors of foreigners and alien cultural practices. If anything, they have grown exponentially as global migration increases alongside a sharp rise in numbers of stateless peoples worldwide.

It is for these reasons that there is today an especially urgent need to rethink the relationship between epidemics and xenophobia, and to ask how new knowledge might dampen, if not completely reverse, the human tendency to take bad meaning over no meaning, as Nietzsche so aptly put it, reverting to scapegoat narratives that should have no place or register in the multicultural settings that world populations increasingly inhabit (https://www.change.org/p/united-nations-general-assembly-epidemics-and-xenophobia).

Contemporary Anti-immigration Poster
"Creating safety"
SVP (Swiss People's Party)

Indeed, the urgent need to rethink basic ideas about self and other is not only reflected in the unacceptable rise in prejudice about purity and pollution that expresses itself through overt biological racism; it is also reflected positively in changes in science that demand a reconsideration of basic notions of self and other. Several areas of science have emerged to examine and rethink the symbiotic relationship between human identity and its boundaries. New research on the microbiome has focused, for example, on the vast diversity of commensal organisms that occupy our bodies and influence and adjust for our interactions with our local environments. These, we now know, are not only critical for organic health, but have significant effects on our susceptibility to allergies, our chances of contracting noncommunicable diseases (such as diabetes), and our ability to adjust to irritants and related toxic stimulants. Moreover, there are many examples of plant, animal, and cross-species dependency that are literally life-giving and without which entire species would disappear (e.g., Margulis 1999).

But there are three areas of contemporary scientific research that are not only symbiotic in focus, but also especially and explicitly *xenophilic*—in other words, that express a critical need for attraction to or engagement with difference as a means not only of facilitating survival and reproduction but also of reshaping the very concept of selfhood as an Enlightenment construct (Napier 2012b). These areas are the new science of epigenetics, that of stem cells and regenerative medicine, and that of theoretical immunology—an older field now rapidly transforming its foundational assumptions into a wholly new domain of epistemological inquiry.

EPIGENETICS

Epigenetics is, quite simply, the science that studies "the set of modifications to our genetic material that change the ways genes are switched on and off" (Carey 2011, 7). Epigenetics focuses not merely on gene expression, but also on how variations in our natural and social environments affect what genes are expressed and the ways in which they are expressed.

There are many famous examples of epigenetic phenomena that show the profound effect of environmental factors on gene expression. Back in the 1950s, for example, a Soviet geneticist named Dmitry Belyaev began to consider whether tame foxes could be bred for the fur industry, for foxes are notoriously unpleasant in captivity. Denied their natural habitats and confined to cages, they are understandably hostile and often overtly aggressive creatures. So Belyaev began by selecting the more tolerant foxes, combining over generations both breeding selection and the active socializing of selected foxes to the presence of humans. His experiments, in other words, were not just about breeding; they were also overtly "polluted" by social interference, selecting breeding foxes depending on their responses to human keepers and their potential for domesticated relations with humans.

What began to emerge, astonishingly within a decade, was not only a breed of domesticated foxes, but actually a rather different sort of animal, with drooping ears, a change in fur color, and a pronounced increase in barking and in responding when called by name—an animal no longer shy, but indeed one even capable of being playful with humans.

For some, these experiments stand as proof of the power of evolutionary selection—that selection makes all the difference. But for others, what they show is the power of social, cross-species interactions on selection, and the extreme impacts of domestication on biology—of the effects of social encounters and controlled living conditions on the biology of animals that, in mere years rather than thousands of years, changed from being difficult and aggressive in captivity to being compatible, attentive, and affectionate. Indeed, within 40 years of selecting for sociability, the experiment had produced an "elite" group of foxes; these were not only domesticated but also now involved in novel biological behaviors, such as breeding out of season (Trut 1999, 168–9).

That such changes could happen so quickly would surely have alarmed Darwin, who was convinced that nature makes no leap ("*Na-*

tura non facit saltum"). But perhaps what was even more astonishing than the rapidity of the physical and behavioral changes in the foxes were the complex knock-on effects of socialization on a much wider range of gene expression than anyone might possibly have imagined. After all, archaeologists have attributed processes of animal domestication to thousands and even tens of thousands of years of social evolution. Now here, in a matter of years, domestication (through the control of selection, but also of active human-animal socialization) had not only made possible significant behavioral changes, but also caused an unexpected cascade of unanticipated, related biological change.

Importantly, what we see here is not biology or sociology only. What is demonstrated in these experiments is much more complex and profound; for not only had changes occurred more rapidly than ever expected, but those changes in socialization, eating habits, and reproductive control also had significant and unanticipated additional impacts—a fact that can perhaps make us hopeful about altering, for example, the course of socially fuelled illnesses such as obesity and diabetes, but that also sets off all sorts of alarms when we think of how quickly conditioning can influence biology.

For a long time, some geneticists thought that Belyaev was surely tweaking his data. After all, each generation in spite of social conditions should go back to the same genetic blank slate; for how could such profound biological changes take place in such a short time—over a few generations and with such overt social consequences for biology? But the legitimacy of the actual genetic truth of what Belyaev accomplished has more recently been demonstrated by a number of experiments, two sets of which make the point adequately.

The first came in 2006 when Andy Fire and Craig Mello were awarded the Nobel Prize for their 1998 work on genetic interference, or what is now commonly referred to as "gene silencing." Genetic interference is a process by which RNA molecules create a biochemical block in cells that transmit genetic codes. Though genes are of course transmitted over generations, interference causes such a deg-

radation of those molecules that they can even disappear over time. While silencing had been observed previously, what Fire and Mello realized was that the effects of such silencing could be permanent (Moulin 2012, 154). Though some argue that the effects of silencing are normally insignificant, the fact is that many—actually countless—viruses possess the very kind of double-stranded RNA that can cause a permanent silencing of gene expression. All these double-stranded molecules need is an environmental opportunity. And this acknowledgment—that inheritance is, to the lament of many more traditional geneticists, deeply a function of how an organism relates to its environment—in itself makes a second line of research really rather astonishing.

Recent work at Washington State University by Michael Skinner and his colleagues has demonstrated that epigenetic changes in mice can be witnessed across several generations (Skinner 2014, 49):

> Despite the mounting evidence, many biologists still recoil from the idea that environmentally induced epimutations can settle into the germ line. The hypothesis seems to contradict a long-established belief that nearly all epigenetic marks are erased from the DNA and then rewritten during the reproductive process—not just once but twice. These processes, the reasoning goes, should wipe clean any acquired epimutations before they can cause trouble in the next generation. (51)

Though there is so much more to be said about the potential impact of new epigenetic research on our understanding of biology, these two examples are sufficient for making an important point: the less aware we are of the effects of our social and natural environments on our genetic makeup—that is, what stands at the edges of our personal world waiting to have an impact on us—the less likely we are to adjust creatively and constructively.

Belyaev's fox breeders, for example, could easily have gone on building stronger cages to distance themselves from the aggression

of the foxes they sought to confine and exploit; but engagement with those very animals allowed for profound adjustments. Not only did Belyaev's foxes become less aggressive, but their domestication also made much harder their culling by breeders. Just ask dog lovers about how they feel when they hear of societies that consume dogs for food and you will see the potential implications of epigenetics for well-being and world peace.

STEM CELLS AND REGENERATIVE MEDICINE

Regenerative medicine is a wholly new field of medical research devoted to the restoration of human health through the remaking of tissue, organs, and cells. Today, popular Internet search engines tell us that regenerative medicine began in 1992 in an article about hospital administration, of all things (https://en.wikipedia.org/wiki/Regenerative_medicine). Though scholarly papers do not, as a practice, cite Wikipedia as a key source, I raise the point here to demonstrate a social fact: once an idea takes hold, we not only explore its new potential but also seek to identify where it came from. We look back, in other words, to go forward, even if looking back is actually a new recursive activity that itself both creates and reshapes history (Napier 2003a).

Though most doctors, for instance, cannot really tell you much about regenerative medicine or which particular stem-cell therapies are likely to yield broad medical advances, the idea that regenerative medicine is the future of healthcare is already being socially driven to describe the relationship between a "self" (your body) and its behavioral environment (the tolls of living that produce disease or health within your body). This reshaping is what historians mean by *historiography* (the actual study of how history gets written). It is a kind of critical practice in its own right, and one that scholars know they need to be careful about. So we should thank them for their caution and be careful too about science's desire to find deep pedigrees of knowledge in areas not yet broadly understood.

Reading, for instance, books on the history of the Vietnam War often creates the impression that the public became uniformly suspi-

cious of what the US government was up to in Vietnam during the Tet Offensive in 1968. But I graduated from high school that year, was president of my class at one point (meaning I knew most of my peers), and can assure you that we knew very little (next to nothing) about what our government was up to. In fact, the Vietnam War is mentioned absolutely nowhere in our yearbook! So we need to be careful when we try to establish a pedigree for a new idea when most don't yet even know much about that idea, or its range of applications, or its possible impact.

Today many physicians and even researchers are unclear about what is meant by regenerative medicine, and some research companies (in an era of personalized medicine, where trials themselves become individual therapies) have allowed those who pay for participating in a personalized trial—even at some of our most famous and especially our financially driven universities—to think of regeneration in its very broadest terms; for deception can be lucrative.

Though there are huge profits to be made by offering personalized therapies that describe regeneration as all but a fountain of youth, there is in fact a massive lack of clarity that only now we are seeking to address (Cossu et al. forthcoming). For example, in his foreword to the Nuffield Council on Bioethics Report, *Emerging Biotechnologies: Technology, Choice, and the Public Good*, Michael Moran (2012), who chaired Nuffield's Working Party on Emerging Biotechnologies, addresses the problem of this emerging field within the context of the public good:

> When we began to look at the field of emerging biotechnologies … their sheer breadth became apparent and their differences perhaps more important than their similarities. The only cross-cutting issue common to all emerging biotechnologies is indeed that they are "emerging". Therefore we have focused precisely on this process of emergence, and on the conditions that shape it. We are concerned, above all, with how reflection on decisions con-

cerning biotechnology innovation can produce outcomes better aligned with the public good.

The point here is that uncertainty creates opportunities for real innovation, as much as it creates opportunities for opportunists. The problem is, to be blunt, that medicine has been both lazy (because it knows people will always become sick) and disinterested (because for a very long time it has not thought of health as having to do with more than illness).

But what these realities also mean is that few of us have actually taken seriously the implications of personalizing medicine on the ethical allocation of limited resources. Should significant public funds be invested in therapies that only work when they are targeted at individuals, especially when scarce resources already cannot meet public health needs? And what of public health itself if medicine now becomes wholly personal? What role will healthcare play when a system designed not for health but for illness is required to think about how causes of ill health can be reversed?

One point is clear: part of the conundrum we face has to do with medicine's own unfamiliarity with the idea that healthcare may become much more than the science of sickness. Indeed, those who have taken the knock-on effects of personalized medicine seriously are led to calling for major paradigm changes in how we think of illness and, in practical terms, how we treat it. And it is the very seriousness of this challenge that pushes us not only into new and hopeful ideas, but also, when we are timid, into premature closure on those real possibilities that we have not allowed ourselves to consider.

After all, the entire process of regeneration is based on a relatively simple if medically and clinically unfamiliar concept. Pathologists, for example, try to determine the origins of illnesses, and often they do so by examining how cells evolve and how evolving is influenced by the kinds of epigenetic, environmental, and social processes we have just briefly described. In this line of thought, regeneration is not about extending life or growing new hair. It is about cell and

tissue cultures and how they hinder or help to regenerate organic health.

However, for many, including researchers and clinicians, stem-cell therapies are also considered regenerative because they involve healthy cell and tissue regrowth, and a process of regrowth that is fundamentally recursive. They are, that is, based on the idea that an unhealthy organism can be healed not, say, by the surgical removal of unhealthy tissue—by directly "fighting" illness (killing unhealthy tissue)—but by the replacement of unhealthy cells with either regenerated healthy ones or new, immunologically compatible ones that are modified (as in stem-cell therapies) to perform different functions. This is in theory all well and good, but it is not ideologically something anyone working as a clinician today has much training in or can tell you much about.

So, let's set aside medicine's current identity crisis and consider the basic theoretical (i.e., practical) problem: when an embryo divides, a single cell splits and "commits" to a particular way of growing. Every human being begins as a single fertilized egg. To make cells later in life perform different functions without a body's trying to reject them, it is necessary either to suppress immune responses to what a body perceives as foreign or to rebuild cells that are compatible for carrying out other tasks. The potential of such retraining is a key reason the new field of regenerative medicine has emerged as science's most promising way of developing cures for cell- and tissue-related diseases and why stem cell research works similarly to take your own naïve (uncommitted ["stem"]) cells and reprogram them to assist in healing—that is, taking cells in their uncommitted state and restructuring them to perform other functions, often by using viruses as "vectors" to convey information (Napier 2015).

But terms and definitions are still being negotiated, and that leaves quite open the space in which ancient prejudice can emerge and haunt us—a point to which we will return shortly. First, however, we need to understand why it is so critical for these fields to define the scope of what those who work in them actually do.

By and large, researchers now agree that regenerative medicine should focus primarily on innovations in tissue engineering, and on cell and gene clinical therapies and their related molecules. To the degree that pluripotent (multiply applicable) and embryonic stem cells are designed to affect, replace, or otherwise augment damaged, pathogenic, or unhealthy cells, stem cells can also be considered "regenerative." After all, regenerative medicine also has great potential for transplantation medicine, and indeed for any condition where biological information can be conveyed across cell plasma barriers through so-called "vectors" (e.g., viral vectors, liposomes, and nanoparticles). Moreover, because of this ability to transfer information, regenerative medicine remains intimately tied both to immunology and to virology, fields where acceptance and rejection are key to health and morbidity.

Indeed, in spite of immunology's emergence in the post-war era out of microbiology—where alien organisms actually do invade us—today the idea of information conveyance is at least as important, if not more important, than simple bodily defense (Anderson and Mackay 2014; Napier 1992, 2003a, 2012b, 2015; Zinkernagel 1996). In other words, regenerative medicine is also demanding a serious rethinking of immunity, which is to say that our general understanding that viruses principally invade us is one that needs to be transcended quickly if we are to avoid broad misconceptions about what regenerative medicine and stem-cell research can and cannot do.

We will get to this transformation in thinking in a moment. But to understand how these processes relate conceptually to a fear of (or attraction to) things foreign, we first need to sense the major change in point of view represented by such new therapies. For remember: stem cell research has advanced our understanding of how we can reverse the effects of some diseases, not only by killing cells carrying an illness—the model whereby deadly microbes are selectively eliminated by antibiotics, for example. Locating biologically compatible cells from a given organism and retraining them from their naïve state can also alter the course of diseases, including those

caused by the interaction of our genetic makeup with the social and ecological environments we inhabit.

In other words, the simplest way of understanding regeneration is to think of it as an attempt to "try again" to shape a body's relationship with whatever in its social and natural environment made it sick in the first place. And what this in turn means is that we now have another chance at rethinking our health, what we have come to be, and what we might become. So why not embrace the opportunity? Well, that would mean being creative, and creativity is, as we know, both a rare commodity and hard work. It is also what attracts us to difference.

However, part of the problem with being creative is that biological regeneration is itself a new and unfamiliar idea for medicine, and one that now makes it necessary not only to "try again," as it were, but also to do so with an understanding of what stimuli made an organ grow symbiotically in a particular way. And that in itself is not conceptually easy; for these new fields of research and practice are built not on the eradication of the foreign, but on an understanding of how that which is foreign influences our living out of the genetic data we have inherited. And that kind of major conceptual shift asks a lot of us.

Given what we have already described for epigenetics, these processes, then, require a much more complete understanding of the foreign, rather than an ignorance or outright rejection of it. Part of making cells grow involves not only knowing how to start growth caused by human intervention, environmental conditions, or social practices but how to stop it. Otherwise, regeneration can itself run amok, producing new aberrations, such as novel cancers. And that is where immunology and the science of compatibility and incompatibility come into play.

IMMUNOLOGY AND "NONSELF HELP"

On a hot Virginian day in the summer of 1974, I accidentally drove the tip of a scythe I was using to clear a farm pasture into a nest of

aggressive hornets. By the time I realized what was happening, I had already been stung more than 30 times by the swarm. As I ran in absolute panic while they continued to sting me, my body did exactly the wrong thing that all our bodies are primordially trained to do—shut down to preserve blood flow to the brain. I knew enough about shock to know that it could also be psychogenic. On the race to the hospital some 20 miles away, I had the odd sensation of having to be mindful of trying to regulate my fluttering heartbeat. I felt also the strong sense of doom that is common to those experiencing anaphylactic shock, and needed to be clear and determined about not giving in to my anxieties. I was, in short, in the midst of a bio-psycho-social traumatic experience.

Walking into the hospital, I was seized by a nurse and told to lie down. After a shot of epinephrine I was told to relax. In about an hour I began to feel better except for the fact that—this being a teaching hospital—virtually every medical student the head resident could grab was brought to visit me. After all, as future doctors they needed to know anaphylactic shock when they saw it.

As each ad hoc group of young students peered through the curtain of the emergency room, the resident in charge took the stage and ceremonially explained the details of what had happened. I did not mind. After all, my life, I was told, had been saved. I could easily have died. So, instead, I too listened to the lectures. As the child of a family with both a medically trained grandfather and a sister then in medical school—and as one who had been destined at one point for the same profession—I found myself oddly distanced from the fact that I was the very specimen being examined.

In each mini-lecture the head resident explained that a massive dose of venom had caused a systemic reaction mediated by my immune system, in which my body had overreacted to an antigenic stimulant by releasing basophils, white blood cells that become active in inflammatory responses. What I was experiencing was a cell-mediated immune response in which my body, as it were, was attacking itself. For this reason, the lecturer went on, anaphylaxis is at the

foundation of our understanding of autoimmune processes in which immune cells (which normally only recognize and eliminate nonself invaders) actually "recognize" the self and go after it.

This then-emerging notion of the immune system was based upon a number of postwar discoveries, but perhaps most importantly those that led to the 1960 Nobel Prize for Medicine that went to Peter Medawar and F. McFarland Burnett for work that helped us to conceptualize the systemic nature of immune responses. For Medawar, it was his work on acquired immunologic tolerance that won him his prize. He showed that mice could tolerate difference until they acquired immunologic intolerance. In other words, identity would be acquired as the body learned how to defend itself from outside invasion. Burnett, in turn, showed that the body produced antibodies ("anti-foreign-bodies") that would recognize and eliminate antigens ("anti-body-generators"), and that immunity, hence, was a function of recognizing the foreign but not recognizing (and attacking autoimmunologically) the self that immunity served to protect. Together, these and related discoveries lead to the emergence of the concept of the immune system in the late 1960s (Moulin 1989, 1991; Napier 2003a, 2012a, 2012b).

But there was another, rather separate line of research that also played a major role in this conceptual evolution, and that was the field of vaccines and vaccinology. As a child of the 1950s, raised in Pittsburgh, Pennsylvania, I have distinct memories of those men in white lab coats—Jonas Salk's team of inoculators from the University of Pittsburgh—who would come to our nursery school to administer polio vaccines from unpleasantly large glass syringes. My first cousin had contracted polio, and our parents and their generation were terrified of it.

At the time, scientists argued vigorously about whether viruses were or were not living (indeed, they still do), but that did not stop anyone from conceptualizing the diseases they produced—from smallpox to AIDS more recently—as infectious diseases (Lwoff 1957). Moreover, Salk's miraculous ability to halt polio in its track was

enough for us. We did not need conceptually to solve the existential character of viruses in order to think of their invasiveness in the same broad terms as we would any other kind of bodily invasion. In fact, the idea that things foreign could be secretly undermining our health through invasive activity had its perfect social and political parallel in the Cold War fears of that era that most everyone shared. The idea of nefarious invaders, that is, was easily conceptualized more broadly (Napier 1992; 2003a; 2003b; 2012b). And because of these circumstances, it is surely no accident that the immune system emerges as an acknowledged fact only in the scientific literature of the late 1960s (Moulin 1991).

But to study immunology in those days required, because of this easy slippage between microbe and virus, a firm grasp of microbiology, and most leading immunologists came to immunology having studied parasites, true living invaders. By the late 1960s, this easy merging of alien forms meant that much of the necessary scientific, public health, and political pieces had come together for immunologists to take the stage as the medical heroes of the future—vanguard researchers whose many deserved awards and prizes would help firmly cement the idea that immunity was about protections, and in particular about recognizing and eliminating "nonself." Indeed, as immunologists have reiterated to me for decades, without "recognition and elimination" we have no immunology, leaving the body a parasitized "toxic dumpsite," as Melvin Cohen, head of the Salk Institute's theoretical immunology group, once described a hypothetically immune-free body to me.

But while this idea of immunity has prevailed and continues to be promoted in research—as well as in clinical practices, medical training programs, and most school campaigns that urge children to wash their hands regularly—certain nagging conceptual problems had to be overlooked or at least glossed over. First, as already mentioned, was the existential oddity of positing a protective mechanism that only worked by not recognizing the organism it was designed to protect. Second was the fact that the body's major generators of

immunity (the thymus and bone marrow, our creators of "T" and "B" cells) seemed to be engaged in a constant creation of mutants, making them nothing short of random mutation/transformation factories.

This second problem so vexed biologists that it became known as the *evolutionary paradox*, for our bodies create a huge number of randomly diverse cells—somewhere between ten to the fifth power and ten to the sixteenth power in number. In fact, the quantity of random mutations each of our bodies produces is so large as to become irrelevant, especially when we realize that most antigens that we describe as attacking us have many markers on them that tell us they are "outsiders." These are the "epitopes," or amino acids and sugar residues that function as outside determinants and that need to be recognized by our response cells (lymphocytes) for us to react immunologically. Compounding the configuration of epitopes that our bodies must respond to actually means that the numbers of lymphocytes are much larger than the numbers of antibodies in our repertoire (Napier 2012b and citations).

So, for all intents and purposes, the scope of this mutational diversity is simply staggering—a theoretical conjecture. The paradox, however, is not; for why, it posits, should we continue to produce randomly? After all, evolution inveighs profoundly against randomization without targeted adaptation. We don't produce cells for big feet just in case the earth turns into a mud bath, or wings because global warming might flood the planet. But in evolutionary terms our immune systems do. They are, in fact, constantly creating freaks of nature—some have no target whatsoever but are necessary, immunologists think, because the system is there to create whatever diversity it can in order to respond to invasion, and to replicate quickly the customized cells we need to defend ourselves.

But how, as Arthur Silverstein wrote decades ago in his *History of Immunology* (1989, 147),

> could the gene pool be maintained when any given organism was likely to employ such a small proportion of its

> specificity repertoire [its unique number of specific antibodies] during its lifetime and when so many of the specificities that it did employ were against antigens that posed little threat to survival? In the absence of positive selective pressure, it would not take long for such unused or "unimportant" genes to lose their identity.

Now this is a real problem—and not only a problem for short-term cost savers flummoxed by apparent waste. According to natural selection, these numbers should be limited because mutant, maverick cells are and ought to remain superfluous, especially when they have no potential application beyond the prospect of their interacting with some yet-nonexistent stimulus.

However, even were we to argue counterintuitively that the immune system protects what it cannot recognize by producing random, untargeted, spontaneous mutants, this configuration is further compounded by a third problem: the number of immune cells your body creates is less important at the level of contracting disease than is the relation of that number to the number of outsiders who can influence you. In other words, what exists outside is really as important to your survival as is your ability to keep unfamiliar things out. And that's because survival is not only about getting through Darwin's jungle intact; it's also about making a future you can live with. Survival, that is to say, is not just the colonial experience of getting home relatively unharmed on a leaky buoy called *The Beagle*. Survival is about making sure that the world out there is also a part of your future.

In fact, in spite of the extraordinarily high number of mutants produced by your thymus and bone marrow (and, by the way, humans do not produce the highest numbers of antibodies), some organisms do quite well with very limited repertoires, whereas others produce antibodies in seemingly promiscuous numbers and nonetheless do not meet their targets with great accuracy. After all, so goes the extinction of many species in the course of a single year.

Having more antibodies, in other words, is only a part of the picture, because the scope of the immunological repertoire is only relevant to the environmental information that can affect an organism. What is more, that number is in itself no guarantor of protection unless the right antibody or dendritic ("presentation") cell is available and able to produce immune responses rapidly when a novel stimulus appears.

What these facts tell us is that our ability to respond is as much a function of what the environment offers us as it is a function of the immune system itself. Some organisms do very well with a quite limited repertoire. Indeed, no thymus would be necessary were it not for environmental conditions that provoke responses. And this is where we need to confront yet a fourth and final paradox.

Though immunology, as stated earlier, grew in the postwar era out of microbiology (where autonomous living microbes often actually invade us and make us unwell), viruses are entirely different. They have no mobility and cannot create. In fact, they are just information that our cells assimilate and bring to life. Viruses can remain frozen for tens of thousands of years in the polar icecaps. Many we respond to are altogether harmless; and successful vaccines are completely dependent on the idea that limited doses of some are positively conditioning us for better health, as Salk and his researchers realized and made the best of when they used my nursery school as a laboratory. Yes, cells with certain kinds of viral information can replicate rapidly and be transmitted from person to person—influenza, HIV, Ebola—but this viral information in fact cannot on its own invade cells. Cell, much to our surprise, bring life to viral information, even dangerous information.

Now, for those who consider the immune system as only a defense mechanism, and for those who think of defense as an evolutionary fitness exercise, this is a hard pill to swallow; for even if our dendritic cells display information that the body tries to respond to defensively, that information cannot do anything without our cells bringing it to life.

The truth, we must accept, is that there is no invasion by viruses, even if humans can be invaded by viral information transmitted by other humans. And the reason for this is the fact that, while your immune system may indeed function to defend you when your body successfully assimilates information from its environment, what that system actually is doing—for better or worse—is seeking out information.

In other words, your immune system is as much a *search engine* of difference as it is a *defense mechanism* (Anderson and Mackay 2014; Napier 2003a, 2012b, 2013). It is there to bring new information to you so that you can adapt, survive, and not die—as did so many isolated Amazonian natives—from truly alien forms of influenza that sometimes created in invading conquistadores the symptoms of a common cold.

XENOPHILIA, OR WHY INFORMATION MATTERS

Many of us are familiar with the concept of the avant-garde. For most, it has to do with radical artistic activity and unusual and unconventional behaviors—some potentially interesting; some novel and informative; some dangerous; and some just silly. But, in fact, much of what we call avant-garde activity is not at all unlike what your immune system is constantly engaged in. And that's because the term itself is really about information gathering. The term avant-garde was not originally applied to groups of artists: it was a French military term (literally the "advanced guard"). It referred to soldiers in war who climbed out of the trenches in days when people fought that way, trying to figure out what those foreigners over there were up to.

The avant-garde, then, is about intelligence gathering: if you don't take risks, if you don't go far enough out into zones of danger, you never learn enough to make informed decisions. If you go out like a berserk Rambo, you will probably die, and nobody will learn one thing from your recklessness.

So, that in a nutshell is what your immune system is up to: it looks for information and tries—for better, but sometimes for

worse—to assimilate what it gathers in the world of living microbes and the eternal libraries of viral information. When it assimilates information successfully, we consider its activities defensive. When it fails to assimilate successfully, we later claim it unfit, coming back at its funeral wake to lament its having succumbed to one or another evolutionary pressure. And while both of these outcomes in the end give us nice stories that make heroes of those who succeed and failures of those who don't, in fact they really are just nice stories; for nature itself functions much more symbiotically than we might ever have imagined—and, in the end, is not necessarily as impressed as we are by Darwin.

But there is one more part of this particular story that needs telling. To say that your immune system is as much a *search engine* as it is a *system of defense* is in fact not to tell the whole story; for immunity is not just about the intelligence industry and learning about what someone else knows that you don't. It is also about interacting with the unknown, and that's what is lacking in the *search engine* idea; for a *search engine* has a particular meaning in information theory—one that applies, for example, to the Google search engine I used to find quickly some of the citations I only partially remembered when I wrote this paper. A search engine identifies what doctors and others call an *evidence base*—a foundation of what is known that presumably can better inform decision-making.

However, your immune system does much more than that. Because it is the key source of diversity in your body, it is also central to making novel baseline data. It is as much creating new cells and a future evidence base as it is mining what is already known or what can be known. In fact, it is one of our major sources of creation, and as such very unlike a computer putting together novel molecules with data it is fed. The random patterns of your immune system are more than the product of the algorithms that some technicians at Google put together to figure out who you are, what you are likely to buy, or what you might be persuaded to like.

Rather, your immune system constantly changes algorithms based on environmental stimuli. Its "evidence base" is always transforming because it is not only merging and recombining what is known; it is also encountering and interacting with the unknown to create something new out of that incompleteness we call living. And this is how it differs from any search engine your computer taps into. Your immune system is actually beautifully designed for the recursive engagement with its environment and your memory of it (Napier 2003a; 2012b; 2013). As such, it not only searches for existing information but also brings that information back, creating a new evidence base that it will continually modify in order to go forward creatively.

Here, yet another French word aptly comes to mind: *reconnaissance*—the seeking out and assimilating of information that is not only defensive but that also forms the recursive basis of creation itself. For immunity is about energy flow and vitality: it can kill or cure; it can create or destroy. In fact, it is amoral in its meaning and function—not immorally harmful; nor a thing to comfort us with epic stories of war and conquest; nor the Darwinian allegory of fitness we apply once we witness its outcomes—just an open acknowledgment of the xenophilic need inside each of us to be curious about the foreign, even if we risk becoming its victim.

There is more information to be gathered in the world around us than any immune repertoire could ever suppose. Like the very migrants the xenophobic seek to keep out, the world coming your way has more on you—knows more about you—than you have on it, or probably know about it. Yes, we can fence ourselves in when we feel a deep and abiding fear about our own future welfare. We can claim that the house is full instead of admitting that its cupboards need replenishing from the fields beyond the fortress. But closing off the outside is only a short-term answer that can bring no new life. Left wholly to ourselves we become highly susceptible to disease, just like the isolated Amazonians—perhaps romantically alone, yet also equally so very vulnerable.

REFERENCES

Anderson, Warwick and Ian R. Mackay. 2014. *Intolerant Bodies: A Short History of Autoimmunity*. Baltimore: Johns Hopkins University Press.

Carey, Nessa. 2011. *The Epigenetic Revolution: How Modern Biology Is Rewriting Our Understanding of Genetics, Disease, and Inheritance*. London: Icon Books.

Cossu, Giulio et al. 2017. Forthcoming. "Stem Cells and Regenerative Medicine." *The Lancet Commissions*.

Lwoff, A. 1957. "The Concept of Virus." *Journal of General Microbiology* 17: 239–53.

Margulis, Lynn. 1999. *Symbiotic Planet: A New Look At Evolution*. New York: Basic Books.

Moran, Michael. 2012. "Foreword." *Emerging Biotechnologies: Technology, Choice, and the Public Good.* London: Nuffield Council on Bioethics Report.

Moulin, Anne-Marie. 1989. "The Immune System: A Key Concept for the History of Immunology." *History and Philosophy of the Life Sciences* 11: 221–36.

———. 1991. *Le Dernier Language de la Médecine: Histoire de l'Immunologie de Pasteur au Sida.* Paris: Presses Universitaires de France.

———. 2012. "Immunology, a Dubious Ally of Anthropology?" *Cultural Anthropology* 27 (1): 153–61.

Napier, A. David. 1992. *Foreign Bodies: Essays in Performance, Art, and Symbolic Anthropology.* Berkeley and Los Angeles: University of California Press.

———. 1996. "Unnatural Selection: Social Models of the Microbial World. In *Vaccinia, Vaccination, and Vaccinology: Jenner, Pasteur and Their Successors*. Edited by S. Plotkin and B. Fantini. Proceedings, International Meeting on the History of Vaccinology, Marnes-la-Coquette, France. New York: Elsevier.

———. 2003a. *The Age of Immunology: Conceiving a Future in an Alienating World.* Chicago: University of Chicago Press.

———. 2003b. "Stressful Encounters of an Immunological Kind: The Social Dimensions of Psychoneuroimmunology." In *Social and*

Cultural Lives of Immune Systems. Edited by J. Wilce, 269–81. New York: Routledge.

———. 2012a. "Introduction" (Anthropology, Immunology, and Neoliberalism Special Edition) *Cultural Anthropology* 27 (1) 2012: 118–21.

———. 2012b. "Nonself Help: How Immunology Might Reframe the Enlightenment." *Cultural Anthropology* 27 (1): 122–37 (with response papers by Paul Clough, Michael Fischer, George Marcus, Anne Marie Moulin, Nancy Scheper-Hughes, and Paul Stoller).

———. 2013. "A New Sociobiology: Immunity, Alterity, and the Social Repertoire." *Cambridge Anthropology* 31 (2): 20–43.

———. 2015. "Immunity as Information, or Why the Foreign Matters." *Somatosphere*, March 2. http://somatosphere.net/?s=Napier.

———. 2017. Forthcoming. "Anthropology's End to Biodeterminism: A New Sociobiology." *The End of Biodeterminism*. Edited by Jens Seeburg, Andreas Roepstorff, and Lotte Meinert. Durham: Duke University Press.

Silverstein, Arthur M. 1989. *A History of Immunology*. San Diego: Academic Press.

Skinner, Michael. 2014. "The Case for Inheritance of Epigenetic Changes in Chromosomes." *Scientific American* 311 (2), July 15.

Trut, Lyudmila. 1999. "Early Canid Domestication: The Farm-Fox Experiment." *American Scientist* 87 (2): 160–69.

Zinkernagel, Rolf M. 1996. "Immunology Taught by Viruses." *Science* 271, January 12: 173–78.