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NONSELF HELP: How Immunology Might Reframe the Enlightenment

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Tat tvam asi

[That art Thou]

—Chandogya Upanishad 6.8.7

I. ILLOGICAL “SELF”

In 1960, Peter Medawar and F. MacFarlane Burnet were awarded the Nobel Prize for Medicine for their groundbreaking work on immunity and the role of “self” and “nonself” in maintaining and destroying organic integrity. Medawar’s ideas on acquired immunologic tolerance developed from his demonstration that mice “learned” to accept foreign tissue if injected with allogenic bone marrow at or before birth—that is, that one could tolerate difference until defensive intolerance was acquired.

Burnet’s contribution was that the body produced the protective antibody (“defender” of “Self”—that is *anti*-foreign *body*) for recognizing a foreign invading antigen (“Nonself”—that is, *antibody generator*); and that in so doing it failed to recognize “self.” To put it simply, one’s defense mechanism worked by not recognizing the self it protected.

By the early-1970s the idea that there was something called an “immune system” was just beginning to take root in general practice, having only first appeared in the scientific literature in the mid-1960s (Moulin 1989:221–222; 1991), which

1
2 is why, I would argue, its recent history is so important. This change was subtle but
3 significant; for the science of immunology moved away from an earlier, reactive
4 definition of itself that went far back into the 19th century to one now characterized
5 by protection through expulsion—an earlier belief that dramatic, hypersensitive
6 reactions (such as in anaphylactic shock) were “the unavoidable preliminary step in
7 the production of immunity” (Moulin 1989:232), now gave way to the idea that
8 immunity once acquired stood principally as a mechanism of defense and boundary
9 maintenance.

10 Immunology courses at medical schools have, since then, borne such titles as
11 “Identity: Microbes and Defense” As one leading introductory textbook puts it:

12
13 While normally acquired immunity is carefully regulated so that it is not
14 induced against components of “self,” for various reasons, when this regulation
15 is defective, an immune response against “self” is mounted. This type of
16 immune response is termed *autoimmunity*. . . .

17 In many cases, exposure to foreign substances results from clinical situations
18 in which tissue is transplanted or blood is transfused from one person to
19 another. . . . Rejection of the transplant or transfusion is not a manifestation of
20 some force of nature designed to frustrate the physician and the patient. Rather,
21 such rejection occurs because of the central tenet of acquired immunity—
22 recognition and elimination of “not-self.” [Benjamini and Leskowitz 1988:10]

23 For several decades, the work of the early pioneers of this model defined
24 the rapidly growing field of immunology, while inspiring our present understand-
25 ing of transplantation biology. In the absence of a fully articulated and well-
26 understood immune system (which, to our surprise when we now look back,
27 first appeared in the literature only in the 1960s) no one could foresee that the
28 human “self” was now defended by a mechanism that could only function by not
29 “knowing,” or otherwise “recognizing,” the very “self” it was designed to look
30 after. Indeed, this may be immunology’s first and ultimate paradox of identity—
31 namely, how can a mechanism defend by not identifying the thing, the person,
32 defended?

33 In successive decades (following Medawar and Burnet), several complex theo-
34 retical models emerged: the systemic network theory of Niels Jerne, the associative
35 recognition theory of Melvin Cohn, and the diversified cell theories of Talmage
36 and Burnet, to name some important examples. Through these and others, the
37 *systemic* nature of immune function became solidified, explaining the idea of im-
38 munologic “memory” and the logarithmic rate of antibody formation by showing

2 how molecules could quickly replicate on binding to an injected antigen. Through
3 evidencing such an orchestrated defense against what was viewed as a pathogenic
4 assault, the concept of the “immune system” became an acknowledged fact, even
5 though yet today its very existence remains based, to quote Melvin Cohn, the Salk
6 Institute’s dean of theoretical immunology and head of its Conceptual Immunology
7 Group, “on experimental systems of such great complexity that many interpreta-
8 tions are possible and reproducibility becomes a luxury” (Cohn et al. 1980; see also
9 Cohn 1992, 1997a, 1997b, 1998a, 1998b, 1998c).

10 Almost overnight the killing and consuming concepts of pathogenic microbial
11 invasion took root in immunology even though at the time the nature of viruses
12 (which I will get to in a moment) was yet very poorly understood.

13 Indeed, despite early optimism about the explanatory usefulness of an im-
14 mune system structured to defend against invaders, the fundamental paradigm
15 of immunology—the recognition and elimination of “nonself”—has yet to re-
16 solve the field’s major concerns of autoimmunity, transplantation, and tumor
17 immunology (Carosella and Pradeu 2006, 2010; Carosella et al. 2006; Pradeu
18 2009; Pradeu and Carosella 2006). Indeed, “the self–nonself” model has created so
19 many intractable problems (e.g., Tauber 1991, 1994, 1999, 2000, 2003; Tauber
20 and Chernyak 1991) that theorists and bench researchers alike frequently suggest
21 that it ought to be put to rest (Tauber 1994), or replaced by a model in which
22 antigen-presenting cells (macrophages and dendritic cells) allow the body to be-
23 come “aware” of viral “danger.” This was, and yet remains, the contested view
24 of Polly Matzinger (e.g., Matzinger and Fuchs 1996; Schaffner 1997), one of the
25 most popularly acknowledged (e.g., Pennisi 1996) and most controversial of U.S.
26 immunologists.

27 There is still a fascinating book to be written specifically about how these
28 models were rapidly transformed between the 1960s and the new millennium (see,
29 e.g., Eichmann 2008)—both about how notions of what viruses were evolved out
30 of the study of microbes, and about how immune systems models related in the
31 1950s and 1960s to the social information feedback studies of the Macy Foundation
32 being carried out by the father of cybernetics, Norbert Wiener, along with Harvard
33 sociologist, Talcott Parsons, and anthropologist, Gregory Bateson.

34 However, what is so often overlooked throughout these debates over models of
35 immunity is the degree to which immunological identity hinges on culture-bound
36 notions of a wholly autonomous “self”—a concept made manifest in two basic
37 assumptions: (1) that organic integrity depends on the recognition and elimination
38 of biological difference (there would be no immunology without recognition and

1
2 elimination); and (2) that the notion of a prior and persistent “self” is not contestable.
3 Overall, these premises converge on a singular assumption: that a “self” must
4 preserve its integrity through a protective mechanism. Were a “self” not salient,
5 persistent, and protectionist, that “self” would soon become, as Cohn himself once
6 reminded me, a toxic dumpsite.

7 Whilst the “recognition-and-elimination” model worked well for autonomous
8 biological pathogens (such as those bacteria and parasites which really do attack
9 us), we now know that defending the body against a viral “attack” is nothing like
10 defending it from invading organisms. Viruses need cells to achieve vitality, and
11 cannot attack without the life that autogenous, “self-made” cells (ones made by our
12 own bodies) bring to each and every viral encounter.

13 Furthermore, because immunity demands the ongoing and ever-changing ac-
14 quisition of tolerance to one’s own proteins, it is often argued by contemporary
15 immunologists who are dissatisfied with the self–nonself model that if, as we
16 now know, the self “is constantly being defined anew” through defensive anti-
17 body production and related acquired immunity, isn’t that another way of saying
18 “that it doesn’t really exist at all”? (Richardson 1996:5). In other words, if the
19 self is a prior and persistent Cartesian entity, then what we now know about
20 immunity makes its focus on self both misconstrued and inappropriate. Either
21 immunology is not about the self, or, if it is about selfhood, then that self is not
22 Cartesian.
23

24 II. A BASIC CONUNDRUM

25 If so much in immunology depends on what a “self” is, can what we have
26 learned from immunology, to reverse our terms, make possible a rethinking of our
27 time-honored concept of selfhood? Does immunology, to reverse or inquiry, tell
28 us something new regarding our cultural and historical assumptions about identity
29 and what it is that makes for a “self”?

30 Although good bench science depends on building on existing assumptions,
31 anthropologists can look toward other cultures for models of self and other; for
32 there are many examples (Hinduism being one famous one) in which body image
33 boundary is defined by a careful familiarity with, and a regulation of, new stimuli
34 that may in turn be helpful or harming.

35 Without providing the extensive inventory of possible models that a full
36 historical and anthropological assessment of these questions would require—a
37 thing, by the way, very much in need of being done—we may and should ask what
38 might be gained by such an inquiry.

2 Are other notions of “self” applicable in any way to theoretical immunology?
3 To answer this we need briefly to revisit the famous question of whether viruses
4 are living things; for we will see that the virus stands somewhere at the borders
5 of “self” and “nonself,” and is thus as much a conditioner and definer of a body’s
6 boundaries as it is a “single-minded attacker.”

7 A comparison of an excellent contemporary introduction to cellular biology
8 (Becker, Reece, and Poenie) and any of the early popular attempts at explaining
9 whether viruses are living things, reveals that our assumptions about the vitality of
10 the virus have changed little over the past 40 years; for despite the fact that viruses
11 have no mobility, we continue to describe them as if they do. Indeed, today’s texts
12 put the question aside as intractable:

13 “The question is sometimes asked whether or not viruses are living. The
14 answer depends crucially on what we mean by “living,” and it is probably
15 worth pondering only to the extent that it helps us more fully understand what
16 viruses are—and what they are not. The most fundamental properties of living
17 things are *mobility*, *irritability* (perception of, and response to, environmental
18 stimuli), and the *ability to reproduce*. Viruses clearly do not satisfy the first two
19 criteria. Outside their host cells, viruses are inert and inactive. They can,
20 in fact, be isolated and crystallized almost like a chemical compound. It is
21 only in an appropriate host cell that a virus becomes functional, undergoing a
22 cycle of synthesis and assembly that gives rise to more viruses.” [Becker et al.
23 1996:105]

24
25 And because it is intractable, we continue to use volitional, intentional,
26 metaphors that encourage us to think of the virus as a living thing that can *commandeer*
27 a healthy cell, *reduce* it to a nursery, and *reproduce* itself at breakneck speed. But
28 how can a virus do such things *if it is inert and without locomotion*? How can viruses
29 *recognize*, *scout*, *trick*, *discover*, *alert*, *evade*, *sense*, *recruit*, *mobilize*, *prod*, *mask*, *defend*,
30 *scavenge*, *attack*, *invade*, *adapt*, *appropriate*, *sacrifice*, and *kill* (Napier 2003b:60) if they
31 *lack mobility* and *do not respond to environmental stimuli*? If these are mere linguistic
32 conventions, why should we continue to employ them when they are so inaccurate?

33 Here is a common example of the consequences of such conceptualizing made
34 famous by Susan Sontag in *AIDS and its Metaphors* (1990).¹ It will resonate easily
35 with what most of us have been told:

36 The invader is tiny, about one sixteen-thousandth the size of the head of a
37 pin. . . . Scouts of the body’s immune system, large cells called macrophages,
38 sense the presence of the diminutive foreigner and promptly alert the immune

1

2 system. It begins to mobilize an array of cells that, among other things, produce
3 antibodies to deal with the threat. Single-mindedly, the AIDS virus ignores
4 many of the blood cells in its path, evades the rapidly advancing defenders
5 and homes in on the master coordinator of the immune system, a helper
6 T cell. . . .

7 On the surface of that cell, it finds a receptor into which one of its envelope
8 proteins fits perfectly, like a key in a lock. Docking with the cell, the virus
9 penetrates the cell membrane and is stripped of its protective shell in the
10 process. . . .

11 The naked AIDS virus converts its RNA into DNA, the master molecule of
12 life. The molecule then penetrates the cell nucleus, inserting itself into a
13 chromosome and takes over part of the cellular machinery, directing it to
14 produce more AIDS viruses. Eventually, overcome by its alien product, the
15 cell swells and dies, releasing a flood of new viruses to attack other cells.
16 [1990:105–107]

17 Here is another example:
18

19 When faced with a *foreign invader*, the immune system *mounts* either of two
20 *defenses*. One, humoral immunity, involves primarily B cells. These white cells
21 *recognize* a particular antigen, then make antibodies that bind to that molecule.
22 The other depends heavily on T cells . . . that can *destroy* tumors and cells
23 infected with viruses and bacteria. These *assassins*, including *natural killer* cells,
24 become part of the cell-mediated immune response.

25 T helper cells are the sergeants that roust T or B cells into action. As *helpers*
26 form in the thymus, each becomes *sensitive* to just one antigen *trigger*. They
27 *drift* in the bloodstream or *hang out* in lymph nodes in a “naïve” state until they
28 *meet* the antigen they were primed to *recognize*. At that moment, a *helper cell’s*
29 *fate* is sealed as either a TH1 or a TH2, or so some researchers think. If it
30 becomes a TH1, the cell then *readies* cytolytic T cells to do *battle*, generating
31 the TH1 response. As a TH2 cell, it *initiates* humoral immunity. . . .

32 These *beneficial assassins* can *destroy* a cell that has been *tricked* into *harboring*
33 *pathogens* where antibodies and TH2 components *can’t get at them*. [Pennisi
34 1994, emphasis added]

35 But if, on its own, a virus remains inert and without locomotion, why should
36 we privilege it with agency? Why should we in fact refer to viruses as foreign *agents*,
37 if a virus is lifeless outside a living cell? And if the floating virus is not an active
38 “other” to be defeated, what generates a so-called “viral attack”? Why, furthermore,

1 CULTURAL ANTHROPOLOGY 27:1

2 should the body's bringing it to life be understood as primarily a defensive activity,
3 if a virus on its own is inanimate? Finally, if the so-called defensive antibodies we
4 create (and those proteins essential for identify foreign bacteria and viruses) are
5 de facto not coded for marking "self," how can we see them at all as "self"—or a
6 defensive mechanism of "self"?

7 Might it be that our persistent characterizations of viruses as active agents,
8 arises partly from the cultural belief that harboring otherness within us is principally
9 dangerous, a belief whereby a persistent "self" must in turn always be protected
10 against things "foreign"? Although we all have cancer cells within our bodies, for
11 example, we never say we have cancer until those cells become problematic. In
12 fact, this ambivalence about, and concern over, things foreign within us is why,
13 until the arrival of stem cell research, studying the maternal–fetal was so often
14 unsatisfying for immunologists (Charlesworth et al. 1989).

15 To put things in perspective in an era of research now dominated by attention
16 to "uncommitted" embryonic stem cells, it is worth listening to a well-known
17 Australian immunologist's view of the maternal–fetal only a few decades ago.
18 In 1975–76, the Nuffield Foundation had supported the distinguished Australian
19 immunologist, Graham Mitchell, in what was hoped to be new, groundbreaking
20 work in this area. But this kind of immunology was unsatisfying for most immu-
21 nologists whose training emerged from the understanding of the invasive agents of
22 bacteriology and parasitology.

23 As Mitchell put it, commenting on this period of his research in the 1970s:

24 The maternal/foetal was a good idea, but we just didn't have the techniques,
25 just didn't have the way of approaching the question. It's something which
26 a lot of people have actually got into for a short time and then got out
27 of—the immunological aspects of nature's most successful foreign graft [i.e.,
28 pregnancy]—how the foetus actually survives inside the immunologically
29 hostile mother. We know she is responding to antigens from the foetus. So
30 we did a little bit on that and then got a couple of publications. But that was
31 a bad year. We had the idea and we thought, wouldn't it be nice to have got
32 some money for it, and then we were under pressure to deliver on the money
33 we got from the Nuffield Foundation. . . . We weren't into the field enough
34 and I must say we really didn't get into the field because I got absolutely
35 seduced by host/parasite relationships. [Charlesworth et al.:214]
36

37 As a group of social scientists then studying Mitchell's work pointed out, it is
38 clear that

1

2 Mitchell's own background, experience and contacts fitted him better for
3 research into the immunology of host/parasite relationships. He had no special
4 background in reproductive physiology [most immunologists didn't prior to
5 the era of stem cell research] and was unfamiliar with key techniques needed in
6 the foetal/maternal studies—as he [Mitchell] put it, “we were not particularly
7 good at tissue culture.” But he was *au fait* with the basics of parasitology: “I
8 didn't [Mitchell claimed] have the hangup, which a lot of students do have,
9 of learning the list of fifty parasites and remembering the life cycles and
10 remembering what host they parasitize and so on. . . . That was behind me as
11 an undergraduate. A little bit of boning up and that's all I needed for that.”
12 [Charlesworth et al.:214–215]

13

14 But Mitchell was far from alone at that time in being unsettled by the areas of
15 embryonic cell research that now dominate the science news media; embryology
16 had since World War II faded in importance as microbiology grew dramatically,
17 leaving immunology and virology to evolve alongside and out of the studies of in-
18vasive agents and infectious diseases. Why *do* autoimmune symptoms often subside
19 in pregnancy? Why do woman have much higher rates of autoimmune infection (as
20 much a seven or eight times) than men? Might even childbirth itself, as immunol-
21ogists often think, be understood in terms of immunological rejection? These are
22 today's question more than yesterday's.

23

24 But given them (perhaps precisely because of them), we should, I would argue,
25 persist in revisiting our earlier question: how can viral antigens be considered *foreign*
26 invaders if *our own cells animate viruses*? In Darwinian evolution there is no reason
27 for a healthy organism to seek out something that might harm itself, except when
28 in retrospect we call that encounter conditioning. And, because organisms do not
29 spontaneously endanger themselves in a “naturally selective” world, viruses must
30 at some level, it is assumed, be aggressive killers, even if they are actually inert
31 until brought to life by a cell.

32

33 There is, however, another possible view—namely, that antibodies are a key
34 to exploring the dividing line between self and other, a mechanism by which one's
35 identity, as a living thing, becomes contested and eventually defined. Identity,
36 in this sense remains—to the considerable relief of immunologists—a prior and
37 persistent condition, but one that is now defined at the peripheries of selfhood
38 where it is contested and challenged. If we can accept such a view of identity
(one widely evidenced in the anthropological literature), there is only one logically
consistent and satisfying conclusion to be drawn: namely, if viruses need cells to

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2 come alive, the so-called immune “self” (our antibodies) must attract, or otherwise
3 absorb those potentially pathological viral antigens. We know, that is, who we are
4 by the risky, dynamic, and sometimes painful process of discovering what we can
5 and cannot accommodate. “If you can’t stand the heat,” as Richard Nixon was so
6 fond of repeating ad nauseam, “get out of the kitchen”—survival, in this view, being
7 predicated on stepping forward, “taking the heat,” and hopefully doing something
8 with it besides caving in.

9 Although a completely different paradigm, the notion that antibodies absorb
10 potentially harmful bits of inert matter to transform (for better or worse) their
11 packaging of information actually makes good sense and withstands all logical trials
12 we might set against immunology’s major conundrums; for an antibody is the tool
13 that enables a living thing to explore the boundaries of life—to engage that danger
14 that is the precondition of real change—whether that change be life giving or
15 life taking. Although many may find the concept of inviting danger intolerable, in
16 some non-Western notions of selfhood it is considered both logical and coherent.
17 There are multiple anthropological examples of how self is made up and defined
18 by potentially dangerous encounters at one’s boundaries.²

20 III. NONSELF HELP: COMPATIBILITY RECONSIDERED

21 I have suggested how an alternative view of viral activation—one arising from
22 within cotemporary immunology—can reframe immunology’s former “paradox of
23 identity”; but what of its other more vexing paradoxes? Can this assimilative view
24 of viral life also reframe them? Let us briefly apply such an interpretation to two
25 other outstanding and famous paradoxes of theoretical immunology.

26 First, the *Evolutionary Paradox* (Silverstein 1989): *How can the human body*
27 *continue to create a broad specificity repertoire—a wide range of “B”- and “T”-cells—when*
28 *natural selection would otherwise demand that apparently superfluous “deformations” be*
29 *eliminated?*

30 The answer is that, in an anthropologically and historically informed model,
31 natural selection is no longer a problem because the body is creating possible
32 versions of “otherness” rather than mutations of “self”—that is, the bone marrow
33 (“B”) and thymus (“T”) cells explore “otherness” rather than defend “self”; for
34 the human body appears to produce an extraordinary number of antigen-inducing
35 antibodies (some threatening, some harmless). Indeed, estimates of the human
36 antibody repertoire run from 10 to the 5th to 10 to the 16th. And if these figures
37 weren’t already mind-boggling, “because most antigens have many epitopes [i.e.,
38 amino acids or sugar residues that are antigenic—‘outside’—determinants] and a

1
2 given epitope can be recognized by more than one lymphocyte [response cells in
3 the lymph nodes], the number of lymphocytes that can respond to a given antigen is
4 much larger than the number of cells possessing a certain antigen receptor” (Becker
5 et al. 1996:788).

6 According to natural selection, these numbers should be naturally limited.
7 Superfluous creations should be eliminated. Bodies don’t, for instance, sponta-
8 neously generate organic variations in anticipation of unanticipated events. In evolu-
9 tion the body responds and adjust to stimuli; it does not create spontaneous mutants,
10 or at least does not do so as a function of health-enhancing activity. Saltation—
11 sudden and unexpected change—troubled Darwin; it was thought unnatural.

12 As, however, cellular diversity by definition proliferates in immunology, this
13 so-called “immunological repertoire” appears “paradoxical” to immunologists when
14 it creates mutants that have no apparent target. A body should not produce spon-
15 taneous, experimental deformities in anticipation of a single cellular mutation out
16 of millions on millions that might be useful for some future, unknown threat.
17 Furthermore, for binding to take place, an antibody must be created with a lock-
18 ing mechanism that works for an unknown antigen. This makes no sense either
19 because variation, by definition, is infinite—unless immunologists, as have some
20 evolutionary biologists, come to accept the possibility that antigens develop from
21 the nucleic acids of normal cell genes (i.e., out of the attenuated nucleic acids of
22 [as it were] “former selves,” or at least something very much like them).

23 If this connection between pathology and prior normality is posited, what of
24 antibodies—those mutations we generate in anticipation? Might they not be seen,
25 then, as *creative attempts to engage risk at the borders of self*? In numerous non-Western
26 models identity depends on just such a dynamic engagement. However, I refrain
27 from making the complex digressions that such an explication would necessitate,
28 but refer to the many examples of spiritual engagement, ritual warfare, and marital
29 customs described in the anthropological literature wherein the assimilation—the
30 vaccination—of “otherness” proliferates. Although elsewhere I discuss alternate
31 concepts of self at some length (e.g., Napier 1986, 1992:190–99; 2003b), suffice
32 it here to say that the anthropological literature is replete with examples not only
33 of how body-image boundaries may become transformed or otherwise malleable
34 but also where they position themselves to assimilate other selves or, as it were,
35 evolutionary “former selves,” as a means of establishing and negotiating identity
36 and body boundaries.

37 If natural selection is the parent of traditional immunological identity, cell
38 biology shows that there is nothing natural about selection, at least as it relates

2 to the benefits and drawback of encountering danger; for a self understood as a
3 “prior and persistent identity” can never ascertain in advance whether a selective
4 pressure is ultimately destructive or constructive. (Which is why, incidentally,
5 natural selection is so frequently accused of being teleological.) Therefore such a
6 “self” can only constantly attempt, as it were “selfishly,” to eliminate difference. The
7 result of this elimination may reflect the immunology we have inherited, but it will
8 not answer the contradictions of the field now being taken on by contemporary
9 immunology—contradictions whose resolutions shake the very foundations of
10 Cartesian thinking about what makes a “self” prior, persistent, and autonomous.

11 So much for the evolutionary paradox and immunology. But there is another,
12 equally famous, outstanding paradox that an assimilative view of viral encounters
13 resolves. This is what is known as the *Repertoire Paradox* (Silverstein 1989). And it
14 goes like this: *How can we argue that the proliferation of B-cells and T-cells is adaptive,*
15 *when neither the size of the immunological repertoire, nor even the presence or absence of the*
16 *thymus (our major generator of immunity), can be construed as an indicator of resistance?*
17 Some organisms do very well indeed with a quite limited repertoire; and it is
18 widely accepted that repertoire scope is only relevant to the number of potentially
19 pathogenic viruses that can influence an organism: one may not need a thymus at
20 all if there is no danger—no alienated variation of identity—no “similar,” “other-,”
21 or “former” self, as it were—being expressed.

22 Furthermore, “foreign” viruses and prokaryotes (those organisms whose DNA
23 is not contained inside a cellular nucleus), *are not always harmful*: some are innocuous,
24 others are real mutation—transformation factories without a clearly defined “target,”
25 undermining the very notion that the body seeks always to rid itself of difference.

26 But identifying “nonself” need not be defensive in this new, immunologically,
27 and anthropologically informed model of “self”; for immunity can now be under-
28 stood more *as a creative attempt to engage difference*, than solely as a battle to eliminate
29 it. Here, immunity is less a system of protection and defense than a system of
30 *information assessment, even of creation*—for we survive through risking engagement
31 with difference—as so many new students of mirror neurons, for instance, are
32 eager to tell us.

33 Today, then, immunology sees itself quite differently, and I would argue
34 is well-positioned—perhaps better positioned than any other domain of modern
35 science—to help us rethink notions of the self that have dominated Western
36 philosophy at least since the Enlightenment; for it is with the Enlightenment that
37 we get the birth of scientific agnosticism and the first consistent argument for a
38 prior and persistent person that is not predicated on divine intervention.³

1

2 Compare the “recognition and elimination” model of immunity with, for
3 instance, a recent Salk Institute discovery that certain skin cells with a hereditary
4 blood disease called *Fanconi anaemia* could be reengineered to revert to stem cells,
5 which could then be recommitted by use of a virus. What this means is that the
6 repaired cells—constructed by reverse engineering to a naive stem cell and then
7 as it were, reinfected it—can now be used to replace the defective bone marrow
8 cells characteristic of a disease that also results in very low blood cell counts. A cure
9 becomes possible because, rather than suppressing immune responses, we reshape
10 them by encouraging and feeding novel viral information—information of a new
11 type that one day may well lead to therapies for what were once incurable genetic
12 disorders.

13 What made this possible, I would argue, is an unrecognized but giant leap that
14 has almost subconsciously become a core immunological precept—namely that
15 *viruses do not invade us, but that we, for better or worse, bring life to the sometimes dangerous*
16 *encounters that define the limits of who we are, that limit what we can be, and that*
17 *(hopefully) do so without taking the very life that those viruses, once embodied,*
18 *now inform—or, as we used to say, infect.*

19 Burnet’s antibodies, which recognize “foreignness” (“nonself”) but fail to rec-
20 ognize “self,” are now replaced by antibodies that function as “*self*” *search engines*—
21 *search engines for the information (harmful or helpful) that sits latently in viruses* like books
22 in a library.

23 If, as evolutionary biologists now widely suppose, pathogens evolve from
24 normal cell genes or entities much like genes (for how else can they bind with cell
25 surface receptors?), what our antibodies must be recognizing are as much aspects
26 of *selfhood* as aspects of *foreignness*. The difference made possible by this view is
27 neither subtle nor semantic; for it shows us that cellular antibodies risk bringing
28 life to alien viruses to revitalize these biologically binding—and therefore related,
29 *even if dangerous*—forms of “self” so as to adjust and respond to them. In this view
30 the “self” stands not only as something prior and persistent but also as a living thing
31 capable of constantly recreating and reclaiming its identity through engaging viral
32 information.

33 “Know your friends well, you enemies better.” Thus understood, the body
34 interacts with its environment in an attempt to create a new future. Burnet, who
35 said “it could be no other way” than for antibodies to fail in recognizing “self,” did
36 not suspect that there was another way of resolving immunology’s paradoxes—a
37 way that might change how we view the self and its environment. Part of this not
38 knowing was because of immunology’s evolving out of cell biology—where real

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2 organisms do attack one another. Part of this was because we needed molecular
3 genetics and stem cell research to demonstrate the real advantages of modifying
4 and loading cells over killing them off.

5 The alternative view outlined here, although not the last word on the immuno-
6 logical self, does provide an explanation by which immunology's paradoxes can be
7 reconciled with what is known. If proven correct, it may or may not help reshape
8 scientific research, as it, however, seems now at the threshold of so doing; but it
9 will certainly force us to reconsider immunology's contribution to the metaphysics
10 of human identity.

11 12 **ABSTRACT**

13 *The classical immunological paradigm is predicated on the body's ability to recognize*
14 *and eliminate "nonself." However, the "self–nonself" model has yet to facilitate any*
15 *resolution of the field's major concerns, and may thus prove to be of limited use.*
16 *Merely discarding it is no solution, as the juxtaposition of "self" and "nonself" persists*
17 *in research, in clinical settings, and in everyday practice despite the best efforts of*
18 *theoretical immunologists. Instead, the very conception of "selfhood" may prove to*
19 *be key. Replacing immunology's prior and persistent "self" with less static concepts*
20 *derived from non-Western contexts not only resolves immunology's famous paradoxes*
21 *but also offers a new and more accurate model that allows immunology to reframe what*
22 *may become an outmoded Enlightenment construct of "self." In such a new paradigm,*
23 *immunology's well-known system of protection and defense is replaced with a view in*
24 *which nonself becomes not only the body's enemy but also its primary mechanism for the*
25 *creative assimilation of difference. This incorporative model—in which the "immune*
26 *system" functions more as a search engine than as an expeller of difference—both resolves*
27 *outstanding paradoxes, and complies more accurately with contemporary knowledge and*
28 *research practice. [medical anthropology, immunology, identity]*

29 **NOTES**

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1. For further discussion see *The Age of Immunology: Conceiving a Future in an Alienating World* (Napier 2003b). See also Cohen 2004, 2009; Martin 1990, 1994; Moulin 1989, 1990; and Napier 1996a, 1996b.

2. See several examples in Napier 2003a, 2003b. On the idea that identity is defined at its peripheries, see Fredrik Barth's landmark *Ethnic Groups and Boundaries: The Social Organization of Cultural Difference* (1969).
3. More specifically, I say "Enlightenment" because Descartes begins his own *Meditations* with the claim that he is about to embark on his famous exploration of (self-) consciousness, and that (like a modern-day "infectious" virus) he comes forth masked.

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