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Autoimmunity and Affect: Tolerance and Time in Three Narratives

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Abstract

This essay, co-written by three art and technology humanities scholars from standpoints of autoimmune experience, takes up the longstanding critique of historical self/non-self models of the immune system to introduce a discussion about biopsychosocial and political affect in autoimmune experience of the 2020s. They argue that an ethos of tolerance and intolerance, long operative in biopolitical strategies of state power (Brown 2009), continues to factor into newer 21st century models of autoimmunity as biopsychosocial, ecological, distributed, and networked response. Drawing provisionally on the danger model (Matzinger 1994), which posits that immune responses are triggered by the perception of threat and not by the presence of "foreign" entities, the authors highlight the kernel of negative political affect in the signaling of danger. Drawing on the work of Silvan S. Tomkins, they describe, theorize, and interpret the role of sustained negative affect signaling, specifically (d)anger, distress, and disgust, in their own respective autoimmune experiences, which they describe as unfolding in achronic time (Freeman 2010, 126). It is proposed that closer scrutiny is needed for the negative affects that shape the infrastructure of (in)tolerance and care that gives life to autoimmune symptoms, diagnosis, and treatment experience in the 2020s, an era in which post-COVID autoimmune conditions unfold achronically in ongoing COVID-19 pandemic time.

Keywords

autoimmunity, biopolitics, affect, Lyme, Lupus

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Introduction

The immune system, perhaps more than any other bodily system, has long been studied as a domain around which the concept of the self is both integral and highly contested. The modeling of autoimmunity on the binary of self/other and the paradigm of (auto)immune responses as conditional (in)tolerance has been subject to criticism and rebuttal for some time.[2] Immunologists and philosophers of immunology have introduced and debated an array of alternative concepts, such as the network, cognitivist, ecological, and danger models. Common to these approaches is some notion of cellular agency: the ecological and network models see cells as socially interconnected and engaged; the cognitivist sees them as entities with perceptual abilities; and in the danger model (Matzinger 1994), affective signaling is regarded as a tissue-system response to damage. One could say that models are just metaphors, but they structure research strategies and the culture of treatment.

Tolerance and intolerance, long operative in biopolitical strategies of state power (Brown 2009), factor into these immune models, including those that assume a decentered network model (after Jerne 1974). (In)tolerance is present in contemporary immune discourse, where we propose it to be a normalizing anatomopolitics, a biopolitics enacted at the scale of the individual biological body.[3] Tony Sandset (2021), among others, has introduced the concept of necropolitics to the discussion of COVID-19. He proposes that "conditions of slow death and necropolitical outcomes are themselves not only the outcomes of a form of 'state of exception' but rather [occur] through what we can call a 'state of acceptance'.... Necropolitics is often 'chronic' and slow, yet its influence on people's lives often comes to the fore when an abrupt crisis emerges such as COVID-19" (1411). He makes a case for the importance of understanding tolerance as it is enacted through the slow death of acceptance.[4] The writer Sarah Manguso, describing her experience with Guillain-Barré syndrome, said, "All autoimmune diseases invoke the metaphor of suicide. The body destroys itself from the inside" (quoted in Anderson and Mackay 2014, 5). Her remark gives voice to the affective dimensions of the immune system in the lingering fatalist "mystical trappings" of a self/non-self (Silverstein and Rose 1997, 197), a biometaphysical conception that inexplicably persists (Pradeu 2012; Pradeu and Carosella 2006) through network and open models that figure (auto)immunity as smart and fluidly heterogeneous (Jerne 1974; Shi 2020; Varela and Coutinho 1991;), or which characterize immune systems as "selfless selves" (Varela 1991). These and other models from newer fields such as systems biology have replaced the old self/non-self binary, but retain a core aspect of it—what we will call a *tolerance binary*.[5] Newer models such as the socially engaged, situated cell, or the shift of focus to danger and the tissue system are no less imbricated in the (in)tolerance affective schema than the binary (non)self model. As Thomas Pradeu and Edwin Cooper write, "advocating that immune responses are multifactorial, complex, and contextual" is all too easy (2012). We are especially interested in the danger theory of autoimmunity

proposed by Polly Matzinger (1994), because it offers a means through which to understand the affect system's role. Matzinger (2002) has proposed that what triggers an immune response is not "foreignness," but the release of "alarm signals" by damaged tissues. We propose that we grasp the affective dimensions of autoimmunity's "signals" over time. This model is explicit in its attribution of negative affect to cells: damage triggers danger signals, or a danger signal response mechanism is somehow tripped in the absence of damage. This is theorized as a biopsychosocial communication systems problem. It has generated research into danger and its relationship to brain function (Cserr, Harling-Berg, and Knopf 1992; Louveau et al. 2015; Matzinger and Kamala 2011). Immunologist Tirumalai Kamala (n.d. [2015]) states, "What held up the discovery of brain lymphatics is the thrall of immune privilege," the idea that certain sites of the mammalian body, such as the eyes, the central nervous system, and the testicles, will tolerate antigens without launching an immune response. Previously it was believed the brain was among the privileged "tolerant" systems.

We perceive the systems problem of immune systems communication of "danger," a highly psychobiosocial concept, to be in the domain of affect study. Following Silvan S. Tomkins's general understanding of affect as a response, feelings as a response with insight, and emotion as a response with insight and historical reflection (Tomkins Institute, n.d.), we try, in this paper, to capture affect and/as (in)tolerance in self-experience of responses to autoimmune experience in diagnosis and treatment. We want to better understand the relationship between the biopolitical, anatomopolitical, and necropolitical dimensions of (dis)regulation and normalization of living in a state of self-destruction as an autologous telos, enacted through successors to the self/non-self model. We propose that most contemporary autoimmunity models, while dispensing with the older paradigm of self/non-self, retain the older models' ethos of affective (in)tolerance as normative.[6] Colin Koopman (2018, 108) reminds us that Foucault distinguished between the casting out of lepers at the end of the Middle Ages and the inclusion of plague victims in early disciplinary modernization. The latter "is not exclusion but quarantine"—not a matter of driving individuals out but of "establishing and fixing them, of giving them their own place, of assigning places and of defining presences and subdivided presences" (Foucault 2003, 46). We see this occurring in the contemporary COVID-19 moment of the 2020s at the anatomopolitical level of the understanding of the (auto)immune system, especially in illness experience and in treatment encounters, as they play out in their affective "post-COVID" dimensions.

The immune system has been variously characterized as an intersectional system or network since Niels K. Jerne (1974). It is presently described in terms such as *distributed*, *flexible*, *adaptive*, *dynamic*, *chimeric*, *intersectional*, and *open*, with attention to the role of the tissue system that makes up organs (Matzinger and

Kamala 2011). But these models retain the consternation of "horror autotoxicus," a doctrine introduced by Paul Ehrlich (1900), who proposed that a normal (anti)body has powers of recognition, contrivances that allow the immune system to be smart, to know its enemies—and to have the sense *not* to turn against its own kind. In this model of (auto)immune action, the state and its citizens are the model for the body and its immune system, which has the political insight to recognize external agents as threat (Martin 1994). Late in his life, Jacques Derrida (2003) reversed this formula, interpreting 9/11 as the rogue action of a state system that produced its own death by training foreign agents who then performed suicidal acts of terror inside its borders. His account modeled the relay between the necropolitical and the anatomopolitical levels in a modern, twenty-first-century autoimmune state. Below we track the reverse flow, narrating how treatment responses produce immune system experiences of relayed biopolitical affect (Protevi 2009, 115–40) in which (in)tolerance figures centrally in an ethos of inclusion and care.

We emphasize that immune modeling is not a mere problem of clinical metaphors (Melander 1993; Tauber 1994, 2018). Models project the shape of practice to come through treatment and disease experience. Expression is complexly biopsychosocial, making language and comportment inextricable from disease etiology and experience. More work is needed about the production of traffic between immune system biopolitics, autoimmune necropolitics, and anatomopolitics in the embodied and clinical experience of diagnoses, from the situated standpoint of patient experience. We therefore track flows of affect over time in the situated contexts of tolerating autoimmune diagnosis experience, regarding autoimmunity as a queer art of failure (Halberstam 2011). Following from the work of Mel Y. Chen (2007, 2011) and others who have insisted on theorizing the place of affect, diagnosis, institutional classification, and the state in health and ability experience, we pursue auto-biopsychosocial interpretations that aim to grasp the depth of distress, disgust, and anger in the production of immune system pathology, and in our socialization into a culture of tolerance—in which we learn to put up with our autoimmune experience in a networked, heterogeneous, transient state of biopsychosocial being. We seek to understand how the immune system is produced in affective relays among the biopolitical, the necropolitical, and the anatomopolitical scales of experience in and around tolerance, a primary aspect of the ethos of care and inclusion that drives the contemporary moment.

At the center of our concern is the temporality of the production of contemporary biopsychosocial (in)tolerance of danger, a term that has anger at its core and is closely associated with another characteristic of our time: chronic distress. We ask how and where exactly the relays between self and other and tolerance and intolerance are produced in the body and its biopsychosocial mangle of autoimmunity over time, in acute, chronic, and post-illness states of autoimmune

disease. How necropolitical regulatory (in)tolerance of disease and disability connects to the normalization of autoimmune individuals through toxic affective exchanges, and how theories of autoimmunity update in order to stay the same (Chun 2016), are questions we share. Addressing these questions has required, for us, detailed tracking of timelines. How, we wondered, did Manguso come to feel her condition to be awash in suicidal feelings, and why did this make us angry? By this, we do not mean to ask how she, or we, came to hold socially imposed bad feelings, but how did our conditions come to be a mangle of experience that makes banal the model of biological, psychological, and social self-(in)tolerance as a deeply felt condition understood to be classic, timeless (chronic), and definitive of autoimmunity. Addressing our timelines in some detail below, we try to better understand how the autoimmune life we put up with is modeled and produced through a structure that sustains negative affective registers, and to discern how this experience is normalized through a treatment framework that operates at different scales, from bio- to anatomo- to necropolitical.

Wendy Brown (2008), in *Regulating Aversion*, reminds us that to tolerate is not to affirm but to conditionally allow and make room for what is unwanted or deviant. Tolerance is not an affect. Nor it is just a strategic, provisional comportment. It is a boundary condition in which the self is produced at a border that may be the elastic tissue of the skin (Anzieu 1989) or the shifting territories modeled on the redrawn face of a map or a diagnostic category (Bowker and Star 1999). Brown traces mobilizations of tolerance that circulate and perpetuate rather than alleviate racism, homophobia, and ethnic hatred in domestic practices of sexual and racial regulation and in international strategies of imperialism. Tolerance, as a strategy of governmentality, requires the production of normalization at the scale of the individual subject. To tolerate is to suffer. It is a sustained negative affective state, with its own infrastructure. In this framework, tolerance is acquired in the response to an entity recognized as the other. That recognition of an entity as other is a precondition of tolerance. Intolerance may be exerted against the invading other but also against the self , as in the psychological processes differently theorized as "internalization" by psychiatrists Frantz Fanon (1952) and Alvin Poussaint (1966). The internalization of necropolitical (in)tolerance as a slow state of discernment of other and negotiating acceptance in relationship to an internal entity that has triggered danger "rightly" or not ensures its status as a chronic oscillating condition of judgment, which requires a subject and object. Amelia Jones quotes Antonio Damasio, who states that our experiences "tend to have a consistent perspective, as if there were indeed an owner and knower for most, though not all, [psychic] contents." He continues: "I imagine this perspective to be rooted in a relatively stable, endlessly repeated biological state. The source of the stability is the predominantly invariant structure and operation of the organism, and the slowly evolving elements of autobiographical data" (Jones 2009, 59, quoting Damasio (1995, 238).

But what happens to the bio-logic of the "autobiographical data" when it is self-eliminating? Following the self/non-self model, one would say there is a breakdown of the autologic, and someone must step in with care. The self as always already constituted through misrecognition, fracture, and interdependence has been a cornerstone of analytic thought for decades, exposing the fantasy of autonomy that links immune system theory to the other AI (artificial intelligence) with which it rose to prominence.[7] Polly Matzinger, chief of the US National Institute of Allergy and Infectious Disease's T-Cell Tolerance and Memory Section, has insisted that not only is "foreignness" *not* the important feature that triggers an immune response in the individual (2002, 302); the immune system *does not care* about the self. Its drive is simply (or, more complexly) to protect against danger (Matzinger 1994). Proclaiming abandonment of the self model, Matzinger has continued to argue that the field should move off its perpetual question, "what causes a break in self-tolerance?" and focus instead on how—in what moments, in what systems in the body, in what agents, in what ways—the immune system identifies danger. Matzinger (2022) notes the co-agential function of bodily tissues in (auto)immune activity (Matzinger and Kamala 2011).[8] Her proposal aligns with the STS dictum that agents, including technology and like language systems, do not have intentionality, except when they become durable and compelling tools in specific actions (Suchman 1987). Intentionality is the cognitive map of plans, not the affective, messy process of situated actions. It also reinforces a view of the (immune) system as open, its elements non-autonomous. "Auto" immune responses are in fact complexly intersectional, involving multiple actors inside and outside the body. Pleasure and danger (Vance 1984) can be mitigated, like when the toy licked by the child in Chen's (2011) account of lead toxicity is removed from the mouth of the child and sucked into a biopsychosocial public health treatment plan, recalled as a danger in an environment where anything can be found to have always already been toxic. Immune logic projects the affordances and risks of the shifting signifier onto all things. Medical normalization is pinged back to the biosocial model of environmental regulation and tolerance, with the toy subject to regulation, and tolerance identified as a boundary, a threshold, in the child.

The self model and the tolerance paradigm add up to a suicide plan inflicted on subjects held in contempt for insisting on feeling, and on describing the open ambiguity of their experience, in ways that are inadequately modeled by research and treatment protocols that insist on the co-production of body and environment (yet another binary). Kimberle Crenshaw's (1991) concept of intersectionality has been widely used to account for the multiplicity of forms and registers of discrimination that coincide in lived experience, describing "where power interlocks and intersects" (Crenshaw 2017) in order to understand the multifaceted experiences of marginalized subjects, in particular Black women. In his doctrine of "horror autotoxicus," Ehrlich (1900) famously described antibodies

and antigens as existing in a bespoke "lock-and-key" relationship that he modeled in a sketch (Figure 1). It is important to note that the antibody ("antitoxin") is not the key but the lock. The lock is drawn as a black fish with an open mouth that seeks out specific toxins, the keys (protruding from the cell) for which its orifice is uniquely molded. Like Chen's child, the antitoxin is driven by a desire to incorporate a danger into itself.

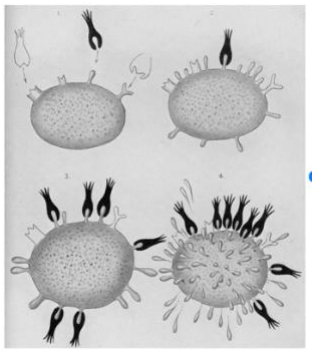

Figure 1. Paul Ehrlich, the lock-and-key principle behind immunoreaction. Drawing reproduced as Plate 7 between pages 437 and 438 of Ehrlich, 1900. Source: Wellcome Collection, https://wellcomecollection.org/works/eprmw6yc/items?canvas=30.
Alt text: A black and white print of a drawing of fours oval shaped blobs with spikes. They are surrounded by floating black tweezer-like pincers aiming for the spikes. On the past blob, seven pincers have attached themselves to the spikes.

This sketch is followed by a second drawing that demonstrates a more ambiguous interaction. There are a few interlocking pairs of antitoxins and antigens. Many antigens float free, and numerous antitoxins find no match.

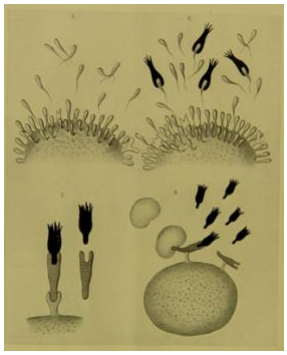

Figure 2. Drawing reproduced as Plate 8 between pages 437 and 438 of Ehrlich, 1900.
Alt text: A black and white print of a drawing of the tops of two oval cell-blobs, one with pincers (antitoxins) aiming for the spikes (antigens) that surround it on the cell, some finding and some missing a lock-and-key fit.

Ehrlich notes that in the blood of animals, there is the presence of excess, "normal" bodies analogous to the antibody. "The formation of these antitoxins lacks all of the characteristics of purposeful, intelligently directed, remarkable

process" (Ehrlich 1900, 440). So where is the intelligence in the system? Matching is characterized as an innate contrivance, a feeling—perhaps something like desire. Immunity, he speculates, may be part of a more open system, influenced by diet and responsive to tissue changes. He speculates on these factors' contribution to variations in reaction to infection intragroup, among members of "the same race and species" (441).

"The problem with identity politics," Crenshaw wrote in 1991, "is not that it fails to transcend difference, as some critics charge, but rather the opposite—that it frequently conflates or ignores *intragroup* difference" (Crenshaw 1991, 1242, emphasis added). Variation and ambiguity in the transition of antigens from healthy to pathological and the sheer range and volume of antigens and antibodies in the immune system undo both the foreign-self dichotomy and the tolerance paradigm. In Ehrlich's brief speculation about diet and tissue (which we regard as the interiorized skin of affect transmission), we see a glimmer of recognition of intragroup variation in the possible situational action of multiple systems and diet on immune response, casting it as open and socially produced. But racialized and sexualized mechanisms of (in)tolerance through a self-other schema remain firmly in place: while there is internal diversity, there are unique sets of oppositions that ensure discrimination within that system.

Later, immunology will shift from type to process: What makes a good cell go bad? The self/other model remains tacitly active in attitude and regard of patients even among some researchers and physicians who have moved on to other more dynamic and open, contemporary models of immunity. Multiple factors propel the self/tolerance paradigm, including the requirement that a diagnosis be fixed to qualify for treatment. Matzinger (2022) asks for more adherence to the danger model, which we interpret here through autobiographies of distress, disgust, and anger (double d-anger), moving off the analysis of what specifically signals "danger," and onto the specifics of the affective spectrum triggered. Affects are, of course, experienced intersubjectively, relayed between self and caregivers, friends, family members, and colleagues entangled in the chronicity of ugly feelings (Ngai 2009). How ugly feelings and ugly symptoms are mutually constitutive over time, especially when they surprise and alarm us in their presentation as ambiguous and chimeric, is of great interest to us. It is the convoluted narrative force of relay and repetition between affect and symptom on face, skin, and tissue that we seek to understand in our interpretation of this danger model through experience over time.

How do we tolerate the affects associated with autoimmune experience? A 1985 study of affect's social and biological dimensions defines "affect tolerance" as "the ability to respond to a stimulus which would ordinarily be expected to evoke affects by the subjective experiencing of feelings, rather than by an apparent non-reaction response or a discharge pattern of response such as impulsive behavior,

somatic dysfunction, or personality disorganization" (Sashin 1985, 175). In this account three variables influence the ability of a subject to tolerate affect: "the capacity to fantasize, the state of the inner container, and the capacity to verbalize affect" (175). Here we shift our attention to time and tissues in self-narratives of experience with autoimmune conditions and diagnoses, offering to readers what we hope to be useful verbalizations of relays of affect (in)tolerance informed by the insight of feeling and the retrospective historicity of emotion. We each have responded to our respective experiences with explanatory tactics that aim not to preclude or pathologize impulsive behavior, somatic dysfunction, and personality disorganization—ours or our caregivers. These are reasonable responses to the biopsychosocial mangle of autoimmunity. Elizabeth Freeman's work on chronicity (2010) and bodily endurance over time (2019) helps us to describe the experience of the specific course of (not) tolerating a distributed affective state, distress, in a case eventually diagnosed as lupus, followed by accounts of anger-rage in the experience of Lyme disease and its diagnosis, and a the volley of affect between caregiver and self in a misdiagnosis of anti-DPPX syndrome around a more likely case of parasitic bacterial infection with *Bartonella* or *Borrelia burgdorferi* bacteria. These conditions are understood as adjacent to the range of autoimmune and immune deficiency conditions now grouped under post-COVID-19 autoimmune conditions, aka long COVID.[9] We do not discuss COVID or post-COVID conditions specifically. Rather, we describe self-experiences of and with autoimmune conditions that inform the context in which long COVID is emerging.

The 1989 redefinition of AIDS as a chronic illness, anthropologist Jean Scandlyn (2000) reminds us, was contingent on access to antiretroviral therapy, which has been vastly uneven, with Western countries experiencing the highest degree of stability in treatment access. Lucas Pereira de Melo et al. (2020) note that in Brazil, the state is the primary agent in guaranteeing drug access, and thus in securing the experience of HIV and/or AIDS as chronic rather than acute conditions by people who are HIV positive. They use the phrase "fragile chronicity" to address the experience of having HIV/AIDS over time marked by state-controlled access and withdrawal of access to treatment drugs. Among members of an online group they observed, the chronic illness state was experienced as a normalizing state that was in fact fragile, interrupted by periods of acute disease when the state medical apparatus experienced periods of upheaval and treatment drugs were not available for a time. To become a chronic sufferer again was to reassume a normal state. In addition to experiencing a new, intermittent "order of life" on treatment drugs, members of the group experienced the "suffering and fear of HIV/AIDS as emotions from a distant history" during those gaps when "there was no political, social and biotechnological support" (Pereira de Melo et al. 2020).

In our account, we approach time over which we received unstable, misapplied, or reluctant diagnosis and treatment, with medical consternation and antagonism among the responses to our symptoms and shared distress, disgust, and anger a feature of our care. We elaborate these experiences to reflexively trace the chronicity of distress, (d)anger-rage, and disgust, horror, and humiliation. As we passed through acute affect tempered by the insight of chronic feeling, we struggled to find ways to be tolerated and not shunned by our caregivers, friends, and family, and we fought to tolerate our own disintegrating self-control to achieve a survivable state of normalcy. These are, we acknowledge, accounts of white middle class fragility—illness experienced with the affordances of income, health insurance, and white skin, all of which we did not lose despite age, sexuality, and ability destabilizing our attempts to live a normal life. Destabilization occurred in part through challenges to our mental health status and our capacity for self-determination, couched in the normalizing language of care.[10] Scandlyn states that whereas "the battle against acute illness is dramatic and heroic, the management of chronic illness, despite its complexity, is banal" (2000). We are reminded of Lauren Berlant's concept of "crisis ordinariness" (2011, 10). What makes something negative banal is tolerance. Any condition can be normalized. But the ordinariness of being chronically sick with indeterminate cause and no endgame requires an especially complicated and exhausting degree of adaptation, accommodation, and masquerade to survive the sustained state of acute panic, the intense multidirectional volleying of disgust (received, self-directed, mirrored), and to forestall open intolerance and rejection. (D)anger must be normalized. This requires self-control of affect—immunity to the triggers of distress, disgust, and rage. There are multiple affects for stigma, a form that exists in a lock-and-key relationship with tolerance of danger, which we regard as a condensation of difference and anger. Chronicity driven by panic about self-contamination demands a performance of self-containment that we hope to chronicle here in its achronicity, a concept we explain below.

We each have heard something like this: "We don't know much about this condition and how or when it began for you. We are not even sure if you have it. There are no definitive markers to confirm its existence before, or now. But you may experience its symptoms all your life, intermittently." The face that offers these words is sometimes performing the composure of care, sometimes struggling to hide bemusement or vague disgust, indicated by a tug of the mouth toward a grimace Silvan S. Tomkins (2008b, 22) in a reaction has called dissmell, a drive-like response that protects the infant from (d)angerous substances. Often the face mouthing dissmell is skeptical, looking at a computer screen as it reacts to us. Sometimes the words come through an online message, and there is no showing of face, but you can hear the dissmell reaction that keeps the caregiver safe from the danger we pose. Along the way, in these accounts, we touch on visibility, masking, and the process of maintenance of appearance and behaviors of care in managing suspected or diagnosed chronic autoimmune disease. We

discuss some of our tactics for avoiding internalization of the stigma associated with the appearance of these signs and symptoms in our caregivers.

Our use of the term *achronical* relates to this unevenness, and also to the ways in which this autoimmune experience of dissmell toward us is produced as banal, normal, not worthy of notice. *Acronycal* is a term derived from *ákronux* (nightfall) to describe the contrapuntal temporality of a star that rises while the sun sets. It is the star that is "acronycal" and not the sun, whose time is the standard against which the star's weak power is observed. A distinctive characteristic of acronycity is that the star is always present, but noticed only when the power of the sun has weakened enough for the weak light of the star to become visible. The star is the sun's other. The certainty of the sun's relative strength and position in the next cycle makes it remain the dominant measure against which acronycal time is registered. We propose autoimmunity may be usefully understood as achronical (Freeman 2010), meaning not outside of time or timeless but remotely sensible, as a past and future condition. A primary disease, its history, and its sequelae in compromised immunity or autoimmune disease are interconstitutive and co-defining.

Chronicity demands a performance of self-containment that we hope to chronicle here in its unevenness, its achronicity. Our accounts focus on affective experiences of symptoms associated with lupus, Lyme disease, and anti-DPPX syndrome. We propose, overall, a critical and reflexive engagement with autoimmune and deficiency syndromes' achronicity across subject positions. The model of consecutive time in which acute disease is followed by chronic post-disease syndrome does not capture this uneven experience adequately.

The affect we begin with is distress. The verb *distress* descends from *dis (apart)* and *strengere* (to draw tight). It is an archaism that describes the act of stretching apart. In law, the noun describes a claim made over the goods of another to satisfy a debt. Use of *distress* to describe anguish, grief, and pain, dating back to the 1300s, begs the question of who or what performs the stretching and seizing, and how the term came to privilege not the act of taking away, but the emotional state of the subject on whom tearing and loss is inflicted. Health, for example, is the transacted object taken away from the body, but we do not use the noun form of "distress" to name that process of separation. Rather, "distress" becomes the sequela of this loss—it describes the subsequent state of the loser. To put this another way, when the body is in ill health, control over the body is understood to have been taken away from the subject and put in the hands of the illness state, which assumes power over the afflicted person. Distress is the feeling they are left with. The distress-anguish spectrum is among the affective processes described by Tomkins (2008a), and he characterizes it as tending to exist in a prolonged, high-steady state (289–323), as compared to, say, anger, which is more short-fused (323). Distress, at the low end of the distress-anguish spectrum, tends to be

maintained and tolerated by the subject for a very long time. The *APA Dictionary of Psychology* (2023) defines distress as a type of stress that results from being overwhelmed by negative life conditions, resulting in maladaptive behaviors.

In the following sections, we oscillate between "I" and "we" as we shift to our personal narratives, approaching them individually and together.

Narrative #1: The Unhealing Wound: Distress and Eustress

I start with a narrative about a wound. The art historian Amelia Jones (2009), in an extensive treatment of wounds in performance-based art practice, points out that wounds are powerful signifiers that are both agential and enacted. They announce injury and, when self-inflicted by the artist, as they are in the works by Ron Athey and Catherine Opie discussed by Jones, they serve a discursive purpose. In the cases she discusses, one purpose is to insist that the person in pain maintains control over enunciation of experience—that pain is not debilitating but, rather, productive of a register and politics of pleasure. In this context, the adage "time heals all wounds" takes on a different meaning. Standardly, this phrase assigns agency and intentionality to time in the process of resolving the emotional distress of a loss, typically of another person from one's life. Progressive, linear time is understood to have a curative, healing power. In this adage, a physical wound is a metaphor for an emotional injury, for hurt feelings. The traumatic cause (a breakup, a death) becomes a memory, like a scar, that times blunts and erases. But in performance-based self-inflicted pain, control over the chronicity of distress is in the hands of the subject who experiences the injury and bears the wound. The performance makes clear that a physical wound and wounded feelings are not simply analogous but are intersectional, co-productive. A physical wound is a trigger of feelings of distress. Emotional distress produces somatic outcomes: a rash, the marks of self-cutting. The case of masochistic performance art demonstrates the problematic of agency and control in the experience of distress, and underscores the reality of the wound as not simply an outcome of injury but a process in the negotiation of relational agency and power. The distressed subject is always negotiating with time and in this sense has the ability to bring out its achronicity.

Freeman (2016) proposes that chronic illness belies narrative, with chronicity assuming a kind of shapelessness in comparison to linear time. In this first account, we together unpack the chronicity of distress associated with a physical event that was an outcome of chronic illness that failed to resolve, becoming unending—or, rather, becoming repetitive, with multiple beginnings and endings. The narrative arc of hemostasis, inflammation, proliferation, and remodeling/repair unfolded only to begin again before repair is complete. An acute wound that refuses to stay healed is not exactly constant, not exactly

chronic. A chronic wound, one learns by experience, perseverates—has its own cyclical rhythms that are acronycal, irrupting like a rising star just when you thought the infection cycle was resolving.

Throughout the first decade of the 2000s, I began developing lesions without having been noticeably injured. These wounds appeared with increasing frequency and severity. Each refused to fully heal. One wound of about three inches in diameter that had formed on the back of my left thigh around 2009 remained unhealed for two years. "Healing" is a process referred to often in health discourse. *Unhele* is an obsolete term meaning to uncover or reveal that I revive here to describe the process of a pathology bent on revealing itself achronically. It is not such a simple matter as "non-healing," for the wound did heal itself—only to unheal again and again. In this section below, I establish the different patterns of chronicity that became interlaced as the wound took on a rhythm of its own, as it drained and re-established itself, and as medical visits became a routine recurrence, punctuating the routines of work life and adding stress and anxiety as dressing the wound—washing and bandaging it and taking different courses of antibiotics in hopes of making it heal—became a regular part of dressing for work. The life of the wound was accommodated in patterns of comportment and appearance to mitigate the stress of achronical temporality: having to speak in public with an open wound, while in pain.

I ignored the wound for about six months, watching it grow with mounting anxiety, as I attended to the more urgent crisis of my pet's fatal illness. When I finally sought treatment for myself, I was seen by a nurse practitioner in my primary care practice. They drained the lesion in-office, treating it as a cyst or boil. However, they raised questions about the nature of the wound, speculating about its behavior and appearance. It did not act like a cyst or a boil, they said. It lacked the characteristic sac or wall of a cyst; the sinus tract was too deep for a boil. Although the draining brought temporary relief, the wound did not permanently heal, but instead became more inflamed. The area became larger and more swollen, abscessing and bursting only to then re-establish itself as an open sore rather than closing up into a scar.

I began to measure time by the wound. Sometimes the cycle took days. Other times it completed itself over the course of hours. I went into this process with a tacit understanding that wounds heal over time. The life of a lesion follows a pattern that ends in closure of the tear. Even an infected wound will open and drain, close, and become a scab. Perhaps it will produce a scar that gradually fades: time heals all wounds, after all. What I was not prepared for, however, beyond the delay of healing, was the erratic recurrence of the lesion's life and death, as it stretched apart and drained only to re-form in a more volatile state, with days or hours of acute fulminant activity. There was no finality, no resolution of the acute state, only its inevitable yet unpredictable repetition. Wound time

was achronical—not exactly shapeless, but apparent only when compared to the regulating time of the imagined normal wound.

This account may summon feelings of disgust. But what was most prominent for me was my own sense of distress faced with a painful bodily state sure to be received with disgust if revealed in public. Our experience has been that physicians who are not specialists in feelings don't know where to look when faced with feelings of distress they find ugly rather than those that engender tender feelings of compassion and care. Many look away, a response that may be an indication of disgust, dissmell, a mirroring of our presumed shame, or they simply express mild annoyance in the form of a distancing brusque professionalism. Feelings interfere with the path to treatment of the body. They need to be put to the side. We each have been referred to psychological or psychiatric resources to help with our intrusive affective states, imagining this referral is a caring act that will address a perceived need for supplying us with strategies of self-management geared toward normalization.

Distress mounted as the experience of repeating an acute process over and over, at different unpredictable rates, became a chronic state, or rather a state of achronicity. I came to recognize draining and diminishment as stages of a deception, after which, at some point—unpredictably but within a short period of time—the lesion would not heal but instead "unheled" (uncovered itself), over and over.

Chronic suggests constancy, perpetuity, continuity, unending sameness. Words like *eruptive*, *disruptive*, *respite*, and *remission* hint at the ways in which repeated bouts of acute symptoms, remission, and recurrence are the fat of chronic disease, morphing its basic process, and triggering anticipation and dread in the subject who tries to maintain control over its erratic course. Distress becomes normalized. It becomes productive, takes action. One of the actions I took was to reassign agency over the process from myself to the lesion. Whereas the nurse practitioner had drained the wound at my request, now it drained itself, according to its own will and clock. I was beginning to realize it was a chronic thing that might never succumb to the normative linear time of health, healing, and cure. I started to worry about the consequences of living with this thing that would always be infected, that might become septic, that might never go away. And so my own sense of agency in the process diminished. I was at the mercy of an unhealed lesion. It was a danger to me. Areas of tissue and skin cells had begun to go rogue.

I had no idea what chronic condition I "had" that was causing this perpetual wound-state. I continued to seek help from my medical caregivers, hoping for treatment and answers. My primary care doctor recommended waiting, giving the lesion more time to heal. However, my dermatologist began to associate a

different narrative with my symptoms: he diagnosed me with the inflammatory skin disease hidradenitis suppurativa (HS). In the following months he put me on repeated courses of various oral antibiotics, a standard (and generally ineffective) treatment for HS. None of these helped. My primary care doctor did not agree with the HS diagnosis and persisted with the narrative of an eventual fading of symptoms. And so I walked around for two years with a wound that was perpetually open, in various stages of healing and unhealing.

I became very inventive and resourceful in devising ways to bandage and medicate the wound, accommodating it into everyday life by improving methods to cover the thing that insisted on uncovering itself. My dermatologist eventually suggested surgical removal, and although my primary doctor insisted on waiting, I finally elected for this surgery in 2011. A critical aspect of successful elimination of an HS lesion is the removal of the sinus tract feeding the lesion. The surgeon reported removing a tract as long as my finger, and I thought the saga was finally over. But within two weeks the abscess returned. This is a common outcome of hidradenitis suppurativa surgeries, since the subcutaneous sinus tracts that feed their chronicity can be insidious—difficult to trace, hard to remove in total, and extensive, sometimes even opening into body cavities and internal organs.

This one long-term lesion finally did resolve itself after some novel treatment by an HS specialist. But its capricious pathology disclosed yet another unheled condition associated with supreme achronicity and ever-shifting, ever-unpredictable symptomology and acute phases. The HS diagnosis implicated more sinister factors entangled with the chronic skin disorder, namely systemic lupus erythematosus (SLE). Cause was nested within cause. In medical parlance *co-occurrence* has replaced *comorbidity*. In the nineteenth century, the use of *morbid* to describe disease states was transferred onto negative affect (morbid feelings). With *co-occurrence*, diseases are characterized as companions that co-exist (a)chronically, like the bright sun and the weak star. HS often co-occurs with such chronic and incurable autoimmune diseases as lupus and rheumatoid arthritis.

Lupus has ever-changing manifestations that can impact any part of the body. Being female, my profile is consistent with lupus's characteristic gender associations: women constitute roughly 90 percent of the people diagnosed with systemic lupus erythematosus and lupus nephritis (LN), a condition associated with renal disease. My diagnosis in my early fifties was unusual because the disease typically emerges in adolescence or early adulthood. I am also an outlier because of my race. I am white. Systemic lupus erythematosus prevalence for African American women is double that for white women, and both forms of the disease occur with far greater frequency among Native American and Alaska Native, Asian/Pacific Islander, and Latinx women than among white women. Candace Feldman et al. describe the various intersectional influences suspected of

contributing to the onset and development of systemic lupus erythematosus and lupus nephritis via the "complex interplay of genetic, hormonal, and environmental factors" that likely contribute to their "variations by gender, race/ethnicity, and income level" (2013, 760). Among these are workplace and environmental exposures to toxins, smoking, and psychosocial stress. I suspect certain of these, particularly past domestic violence, workplace stress, and PTSD, as factors in my disease, and those of many women across these demographic sectors.

The living pattern of achronicity embedded in such intersecting risk factors amplifies the experience of intolerance and discrimination faced by women whose also experience poverty or low income, racial and ethnic marginalization, precarious housing and work conditions, and domestic or sexual violence and abuse. The psychosocial and embodied distress associated with erratic, unpredictable temporality of autoimmune disease compounds the achronicity of precarious housing, employment, financial and personal resources, and healthcare access.

Caregiver disgust reduces the human to an imagined being without volition. Disease terminology for "lupus" was taken from the Latin word for wolf by Rogerius Frugardi (1180), a surgeon who thought his patient's facial rash resembled an animal's bite. This displacement of the auto-agency of an autoimmune attack onto an undomesticated predator is rooted in the ascription of immune agency to a foreign body. It makes us rethink the legal "debt" distress scenario in terms of what is taken away: in this case, the patient's agency over their body's own actions against itself. Predation by the body's own immune system is a terrifying thing to experience, impeding the exercise of a person's will against the body's own internal defense mechanisms. Enter masochistic performance art, the action of self-injury made plain under circumstances that insist on being on top in the narration of pleasure and (d)anger in chronic time.

Again the news of my condition was broken with the tragic statement (and affective comportment) conveying that time and symptomatology exceed my control: like HS, lupus has no cure. The doctor expressed uncertainty about time—my lupus might have been in preliminary stages, or maybe its onset was already underway. This uncertainty impacted my predictive thinking and planning about the future, deeply altering my self-perception and sense of my life's past, and of its overall temporality. Receiving the diagnosis that one has a chronic, incurable disease immediately launches an acute emotional crisis of remembering in which one's sense of time is reconfigured, becoming operant on a lifelong scale that slides in all directions at once. One begins to edit and re-edit one's history with each new causal thesis in mind. Looking backward, I remembered joint pain in my hips in my late twenties. I wondered whether it was related to lupus. I thought about my childhood. I do not recall having the classic red-cheek butterfly rash

associated with lupus, but I nonetheless searched my memory for predictive signs. My frequent bouts of respiratory infections and the fact that when I got sick I fell ill very rapidly and severely all took on new meaning.

The present, past, and future are all implicated in this tectonic shift as diagnosis with a chronic illness is triggers the affect of surprise, which startles memory into the emotional process of re-examining past and present experiences and future expectations. An uncertain new landscape in which this condition will be lived achronically in the months, years, and decades ahead. Lupus frequently attacks and destroys the kidneys; would I end up in dialysis, and if so, when? Was I in danger of needing a kidney transplant at some future date? Anticipation and dread amplify distress, prompting responses (such as the pressure to recall past symptoms) that, rather than alleviating stress, lead one to dwell in the achronic, relative temporality of reflective emotion in which autoimmune conditions make themselves visible in the temporality of an entire planet, a past and future life visible at a distance, in a curious weak light. The lupus diagnosis required me to maintain a schedule of dermatology and rheumatology checks—visual scans of my skin and joints, X-rays, blood, and urine testing—that I would describe as chronic, in that its pattern was clear as day—it would be a lifelong, predictable schedule driven by hopes for improvement and cycles of remission and recurrence, not cure.

Rewriting the narrative of one's life story in ways that make sense and provide a clear path forward driven by one's memories and desires becomes a way of reasserting control over time. Chronicity, as opposed to achronicity, is found in the desire for causality and early signs when in fact there may be no such factors to unearth, nothing to see that could have made life turn out otherwise. Achronicity describes the speculative and intercutting nature of self-historicizing experienced as an art of fragmentation, disjuncture, and bricolage—the activated distress of seizing the story and tearing it apart yourself. This approach is triggered when labs and medical professionals deliver a life sentence with the cool, clinical detachment that often characterizes doctor-patient communications. My account is not a disavowal or refusal of either temporality, but an insistence on the importance of tolerating (a)chronicity—or, rather, recognizing (a)chronicity as a mechanism of (in)tolerance.

Recognizing the highly varied assortment of rheumatoid skin, joint, and connective tissue diseases described as co-occurring—intersectional if not interconstitutive—strengthens the case for using *achronicity* as the relevant term for the real time of autoimmune diseases. One has such conditions perpetually. As the acute crises resulting in the primary diagnosis and those that follow gives way to the more general task of integrating disease awareness and accommodation into one's tactics of self-care and time, mental activities like those I experienced started to focus on piecing together a new, broken narrative that follows an

acronycal arc, finding meaning in those things that appear in a weak light at the endpoints of knowledge.

The construction of autoimmunity experience and life stories as a diagnosis response frequently incorporates scars of past experiences largely attributed not to somatic conditions but to emotional and psychological trauma, and states of mental suffering such as depression, PTSD, and "stress." Stress, and specifically the stress of needing to go on, to speak in public—the event associated with the "positive" stress engendered by "welcome" life challenges—is another factor here. To work, for all three of us, is to act and to speak in public, to teach, which is also to survive (it is how we make our income and receive health insurance). The thought of speaking in public while in distress, and while having evidence of a condition that might be seen as disgusting was, of course, profoundly distressing.

Hans Selye, a near contemporary of Tomkins, rendered stress binary, coupling distress with "eustress," a neologism that neutralized stress, rendering it quite simply a "nonspecific response of a body to a demand" (1976, 137). This concept reflects the affordances of banal anatomopolitics for capitalism. Eustress, associated in its medical literature with better survival, is classified as a positive response type generated by "positive" life stressors. The stresses of life in industrial capitalism become, quite simply, motivational forces. In the Tomkins affect system, there is no such positive valence for stress, which is always prefixed by *dis* (apart). The felt quality of the negative affect that is distress is first described by Tomkins through anecdotes about the performed behavior in which the feeling makes itself seen and heard: crying, with and without tears, is presented through examples that are, by and large, in response to loss (2008, 313–30). But for Selye, stress has its positive potential—indeed, he warned, "complete freedom from stress is death!" (Selye 1976, 137).

We return to our question about agency and causality in autoimmune syndromes, and to the act of stretching apart, understanding it as not so easily attached to the primary disease of which the autoimmune syndrome is understood to be a sequel. We might consider appropriating *eustress* ironically, as a hallmark of the neoliberal normalization of crisis in chronic pandemic time. Eustress is a useful concept for characterizing accommodation, by which we mean not simply learning to live with our autoimmune syndromes, but finding ways to perform (to act out, to speak in public), in order to provoke accommodation and normalization of our immune-system divergence in the social world of people with AI syndromes, which, with the COVID-era recognition of post-COVID conditions, will now be a vast medical demographic about which little is known.

Eustress is a normalizing concept that suggests there is value in performing positive thinking while in distress: we may respond behaviorally to distress, interpreting it as a motivational force. Trying to avoid income and advancement

lost while on disability by characterizing your condition as a prompt to work harder is a foundation of the supercrip phenomenon (Garland-Thomson 2002; Schalk 2016) in which you strive harder and do better because of your condition. The moment of diagnosis and the naming of the disease or condition of disablement are often held up as significant breakthroughs in this behavioral reversal of outlook from negative to positive. A diagnosis startles you, makes you react. You charge ahead toward a cure or accommodations: positive steps, one might say.

But with autoimmune conditions, there is always the awareness that the diagnosis does not provide a clear treatment pathway or a predictable future. This opens the door to a web of potentialities that are both certain and random: co-occurrences will crop up, making themselves known through the stepped-up schedule of testing or through compounding symptoms; self-care habits similarly are added and become part of everyday life, and the reality that they will not end with any diagnosis sets in. Layered upon this are the existing chronic patterns and the baffling complexity of acute recurrences that eventually become both predictable and erratic in their path and duration. Characterizing distress as "eustress" is classic cruel optimism (Berlant 2011) that is not so much felt or internalized but performed strategically. You learn to say "I'm better now" when people ask. It is an avoidance mechanism that allows you to bypass the humiliation of having your condition tolerated (Tomkins 2008a, 435). You learn to accept that some friends and colleagues will start to view you as the person who has become a flake, a no-show, or an obsessive. Their skeptical but accommodating expressions are another facet of an immune response, at once a symptom and a trigger. Horror autotoxicus rears its ugly head not in your body, but in the social space where they perform their affect in suppressed form, as in the uneasy expression on the face that masks the automatic physical disgust reaction that is like vomiting a toxin (what Tomkins, in 2008b, 22, calls dissmell). A classic immune response on the part of an interlocutor is to feel uneasy, annoyed, or, as we have described, mildly disgusted—an expression that may not be shown to you directly, but may be relayed through friends and colleagues, after your condition is discussed with care and concern, and their kindly words are conveyed back to you, perhaps in an HR meeting about work performance. They want to help but they are tired of your story and want you to tell them an ending you do not have to give—and they want to help you find it if you are unable to do so yourself, perhaps through a special office set up to perform this care. Because surely if you don't have a cure or a treatment regimen that keeps you stable *after all this time*, then you didn't help yourself, or perhaps you have simply become obsessed with your symptoms, attached to repeating them—this must be psychopathology, and for this there are also treatment providers at hand to help you, even in the workplace.

To put this in Tomkins's terms, avoiding your expression of affect can serve to deny the press of aggression in your interlocutor's response, be it concern or consternation. We note that Tomkins spent some years of his career as an industrial psychologist, evaluating the psychological states of both managers and workers on the factory floor, specifically focusing on weeding out those who posed a danger to other workers or to themselves in the workplace. Like some of the men Tomkins described in his account of humiliation, you might learn that it is safer to put your head down and quietly do your job (Tomkins 2008a, 555–61). It is not a coincidence that his passage on humiliation draws from the organizational psychology of Bertram P. Karon (1964), whose highly problematic study of Black factory workers in the US south relied on a projective test devised by Tomkins, and whose ideas were an important source material for Tomkins's own ideas about Black affect (Tomkins 2008a, 561). "If you cannot control your anger in a world where you are constantly being provoked, and where the direct expression of anger brings inevitable disasters," wrote Karon, then "you are lost" (1964, 166).

Normalizing distress as eustress as a behavioral tactic has the ironic affordance of making you stop saying there is something wrong (which is not to stop thinking and feeling it). The social benefit of performing a positive affect toward your own immune response, tactically, is to receive a positive social immune response in return. You are less likely to provoke pity, consternation, and anger. Doing this becomes easier when you can normalize asking for accommodations in your workplace based on a written diagnosis, rather than having to show and tell how you feel. The diagnosis is an account of yourself from a doctor, someone who is only passingly familiar with your life history, and whose response to the affective crisis the diagnosis provokes can seem indurate, even dismissive. But using that physician's immune response of writing a note as an institutional act of regulatory compliance that makes you one of a biosocial class of people who are disabled can earn you a pass at qualifying for individualized accommodation. We characterize these approaches as facets of tolerance, the product of a "healthy" immune system exchange, according to the model of the immune-system-as-networked-self, which is compelled to graciously accept institutional tolerance and care to survive.

We underscore the irony of this approach, and highlight its basis in a 1960s psychology of liberalism. The immune system is profoundly a site of alienation in this scenario. It is here that we get angry at health discourse for taking the modern tenet of the immune-system self to the extreme of attributing intentionality, motivation, and the innate ability to identify (d)anger to a system they admit they do not even understand, and which is rarely understood to incorporate affect in its social context—except as a tolerance or helping mechanism in which accommodation is reduced to regulatory instrumental (in)tolerance strategies in the service of the heterogenous system. We move on to

our second account, in which Tomkins's affective spectrum of anger-rage features prominently as something that must be thusly contained.

Narrative #2: Lyme Time: Anger-Rage

Lyme disease made me angry. Really angry, with senseless bouts of rage that recurred for months on end. Or, to put it more accurately, what made me angry was not Lyme disease, but *Bartonella*, an intracellular parasite that possibly entered my bloodstream through a bite that I never felt or saw, from a tick that I never felt or saw. The facticity of my *Bartonella* infection and its role in my experience of symptoms associated with Lyme disease and post-Lyme autoimmune syndrome is something that my doctors confirm well into this experience. Lyme disease screening tests are in fact blood tests that look for and measure antibodies against bacteria. Like lupus, Lyme has no definitive test.

Bartonella and *Borrelia* are genera of bacteria carried by any number of flies, ticks, mites, fleas, and small animals that serve as hosts to these insects. They are associated with various diseases: Bartonellosis, cat scratch disease, and Lyme. While not classified as an autoimmune condition in itself, *Bartonella* is understood to cause an infection that is part of the immunological spectrum of post-Lyme syndrome.[11] In my case, a *Bartonella* infection produced a state of mind that I didn't imagine possible, with episodes of pure rage towards anything and everything, with no cause at all that I can identify, beyond this biochemical factor of an infection discovered well into my year of rage.

In the months before the COVID pandemic, I began to lose weight. My body ached, and I was spiking fevers and contracting various influenzas in the middle of summer, out of sync with their peak seasons. My joints were inflamed, the swelling and pain of the inflammation impairing my mobility. I could no longer lift my t-shirt above my shoulders. I experienced migraines, nausea, and the sick spell of an insomnia that keeps you at two hours of sleep maximum in any given twenty-four-hour period. There was a tightness in my throat, a response that much later would be diagnosed as mast cell activation syndrome, a condition in which white blood cells in connective tissue are overactive in their release of inflammatory mediators, causing severe allergy symptoms. Foods and beverages I normally tolerated caused my throat to constrict. This, along with the nausea, made eating a major challenge and a very real risk.

I also experienced bouts of anger. It would seem logical to say that my anger was provoked by my physical state—that I was angry about the unfairness of becoming so chronically impaired. But it was not, and I was not. In any case, there was no space to think about the cause. The anger was there, along with these other symptoms, pure in its abstract form. In writing about anger, Tomkins (2008b) speaks of "reasons" (a word he puts in quotes) as paradoxical, coincidental causes of anger. If circumstances were related to my anger, they

were experienced as coincidental to it, simply co-occurring with it. They were no more a cause of my anger than they were a product of it. The reader may rightfully ask, how do you know this? My response is that, at this point in time, the evidence points to *Bartonella* as the common causal factor, and to anger as a part of the syndrome that comprises the Lyme to post- or chronic Lyme spectrum. This is the conclusion I came to over the years that followed acute infection. Cognitive conditions are now established as possible sequelae of Lyme disease, with the term *Lyme neuroborreliosis* entering the medical literature with increasing regularity with respect to acute disease (regarded as treatable in early stages), but approached more generally with skepticism with respect to "nonspecific" and post-acute symptoms such as chronic pain, fatigue, difficulty concentrating, and emotional volatility (Rauer et al. 2018).[12]

My experience of anger in the acute time of this experience was as a pure affect state in which I did not experience insight (feeling) or reflection over past or future times of similar experiences (emotion). It was indiscriminate—against everyone and everything in its path. It had no intentionality, only coincidental targets. Tomkins (1981, 448) has written that cognition without affect is weak but affect without cognition is blind. His comment is striking because it associates affect with power, and cognition with ethical deliberation and feeling. My account demonstrates the continuity of chronic as chronic, and as an indiscriminate immune action. *Response* would be the wrong word, as there was no apparent trigger. This was a cognitive state that was not particularly psychosocial. The brain is somatic, and chemical impacts, and their temporality, are poorly recognized and understood. And yet the disgust, horror, fear, and shame with which I and those around me responded to my anger was very much psychosocial, evoking the obsolete model of horror autotoxicus (Ehrlich 1900). Anger is an unexamined component of the longstanding immune system self/other model. The self is understood not simply to feel disgust at itself, but to target aspects of interiority and attack them indiscriminately. That is anger.

Tomkins wrote, "Distress is the affect of suffering, making of the world a vale of tears…But anger is problematic above all other negative affects for its social consequences…Of all the negative affects it is the least likely to remain under the skin" (2008b, 687). An entire volume of *Affect, Imagery, Consciousness* is devoted to anger and fear.[13] In my episodes of anger, I was ready to strike at those closest to me, loved ones and friends and family—people with whom I felt safe. There was no clear trigger or limit that I could feel at the onset of each episode. Anger would emerge, full-force and ready to pounce on whoever was near. And here I reference Tomkins on joy to say that I think part of me came to enjoy the swell of energy that accompanied this anger. These outbursts offered a brief suspension of an otherwise rotting feeling, providing a social bond, albeit one that was not so joyful for my patient interlocutors.

I didn't notice my behavioral changes at first. After demanding a Lyme test and receiving a diagnosis via a positive blood test, an infectious disease specialist at a well-regarded New York hospital began a first try at treating me with doxycycline. The two-tier Lyme blood tests check for the presence of antibodies that respond to, among other things, the bacteria that cause Lyme disease. Availability of the test is mixed, depending on the willingness of the healthcare provider to entertain the possibility of Lyme disease in the absence of physical signs, such as a bite (recall the imagined animal bite sign on the face of the lupus sufferer). Often tests will not be administered if there is no telltale tick bite or characteristic rash. On the matter of tests, Marvin Fritzler et al. (2021) explain that some autoimmune disease tests are "offered as 'Research Use Only (RUO),' some as 'Laboratory Developed Tests (LDT),' some enter Health Technology Assessment (HTA) pathways, while others are relegated to a 'death valley' of autoantibody discovery and become 'orphan' autoantibodies. Those that achieve regulatory approval are further threatened by the business world's 'Darwinian Sea of Survival.'" Lyme tests belong to the latter category, with the Center for Disease Control's 2021 guidelines on testing and diagnosis making clear the pervasive skepticism of test results as definitive indications of disease compounding the general reticence of medical care providers to agree to administer the test without physical evidence of a bite.

I took the potent antibiotic offered to me for twenty-one days. In retrospect I know this to have been a woefully inadequate and misguided treatment for my infection, particularly late-stage or symptom-onset, as Lyme literate medical practitioners have been arguing for years (see, e.g., Embers et al. 2017). A year later, after symptoms had not abated and under the care of a naturopath, I then began a series of treatments with Chinese skullcap, Japanese knotweed, cryptolepis, homeopathic antibiotics that would begin my real attempt at recovery. Lyme disease is transmitted by spirochetal bacteria, and spirochetes are very hard to kill. Other spirochetal diseases include syphilis, called the great imitator due to the wide array of ailments that present in each patient. That nickname now belongs to Lyme.

A reaction in the body is produced when antibiotic treatment is potent enough to induce the die-off of these corkscrew-shaped bacteria, generating a storm of endotoxin-like products into the bloodstream. Named after Karl Herxheimer, one of the dermatologists who observed this reaction in patients who were given mercury to treat syphilis, herx reactions are a massive rejection response that can last hours or days, though my experience suggests their symptoms can have a much longer duration. The drug triggers the body into a disgust-performance of expulsion. Herx reactions are a performance of the immune self expelling the enemy.

This performance is heightened by cognitive changes. Occasionally, spirochetes make their way across the blood-brain barrier. This has been shown to be a possible cause of severe insomnia and mood disturbances, as has the *Bartonella* bacteria itself. Thus, the person under treatment for Lyme is at double risk of a period of sepsis and related abstract, non-provoked episodes of emotional instability. Mine leaned toward rage.

One night in particular made this instability and its abstract form quite clear. On this occasion I experienced what I would describe as a manic episode out of the blue. It started with a pay-per-view boxing match between a YouTube star and a former world champ. I felt anger mounting as I watched that match. That anger, directed both at the sport of boxing and its reduction of the fight to mere social media spectacle, was overpowering and truly bizarre. I had been neither a fan nor a critic of boxing before. I am not really sure why I tuned in. But there I was, consuming the match and finding my anger ascending, singularly settling on the match as my coincidental "cause" simply because it was there. This anger came to visit most evenings for the next year, when it would be directed at whatever and whoever happened to be co-present with me. Just as, in Tomkins's narrative of distress, the infant does not know "why" they are crying, or that it might be stopped, or how to stop it, so my anger unfolded as an abstract condition of my disease. Confronted with a deep and enduring challenge that could not be mitigated, I had no immunity to anguish. I developed a new personality component, and violent action ensued.

During the course of Lyme treatment, I moved from an apartment on the east coast to my childhood home, where I lived with my parents. This was not an unusual transition for my demographic: young adults pursuing higher education. The slightest disagreements in view or perceived slights would become the coincident cause of my outburst of rage. Thus I behaved, mostly in the evenings, with my parents, family, and friends—those closest to me who met me with unconditional love. In the throes of anger, I felt as if I was kicking down the gates of hell when in reality I'd done absolutely foolish things—stomped my foot through a styrofoam board on the floor, for example, gaining a severely sprained ankle that lasted six weeks. The anger had me red hot and looking for an opening out of my infected body, which I experienced psychologically as decaying, rotting—and which I struggled to stomp to life through whatever target for displaying my anger was closest at hand. This was exhausting, and it simply wasn't "me." But it was a primary survival mode through months of reactions I associated, in retrospect, with prolonged herxing and the cognitive impact of the *Bartonella* infection.

I understand my anger and my bacterial load to have worked in concert, though perhaps inversely. While herxing, I felt every nerve ending in my body at once. When the herxing was at its peak, I would become so exhausted from the

unending inflammation and internal fire control as to have no energy left for feeling. Then, as the inflammation died down, it was rage that came back, in full force, replacing a wave of fear that preceded it. The relationship of fear to anger in immune system rage should not be discounted. The feelings during these episodes were of constant alarm, and everyone became an enemy I needed to fight. It made me feel alive, following days with a dull and monotonous routine of meds, rest, and ice, to fight. Anger fleshes out the inadequacies of the spirit and the perceived world. When herxing was linked to fear and disgust, leaving me immobilized on the couch, tightly wound into a fetal position as I shed toxins and drowned in nausea, anger swooped in with my body's high-frequency trading mechanisms, capitalizing on my arbitrage in mood to supercharge my endorphins. What a corrupt economy of the body, I think now. Both states were affectively impoverished, offering too much or too little motivation to feel the affect. The co-occurrence of herxing and anger deserves consideration beyond the idea that herxing is causal and anger reactive. Herxing in/as anger is a concept worth examining beyond the frameworks of analogy and co-occurrence.

After some time spent ricocheting between these states, from lead-bound to hot air, my faculties for feeling, the state after pure affect, began to step up. First came the activation of childish pride, in the form of megalomaniac self-proclamations: a last ditch effort at narcissistic vanity from my exhausted mind. I felt righteous about my anger. I had just gone through all of this suffering from illness, day after day submerged in miasmas of medical literature, litanies of medical procedures that I had to show up for and monitor and track, to show my progress. The rage began to have intentionality. It was now directed at doctors, healthcare systems, and insurance companies that made every act an effort in overtime. My body's efforts had resulted in outcomes. Lyme disease is an invisible illness. Rage made it visible. What symptoms are rendered manifest and believable before the medical establishment is a constant negotiation. They are conditions for a communal rage that falls and is received unevenly across racialized, gendered, and socioeconomic lines. One discriminatory theory that skews diagnosis is that affluent households are regarded as being at greater exposure risk for Lyme due to their location in suburbs or periphery areas closer to forested lands and the ticks that inhabit them, tilting research sample populations and credibility towards those that are white and wealthy. Doctors are often not trained to distinguish the telltale erythema migrans "bull's-eye rash," an early-stage indicator of a Lyme infection, on patients with darker skin tones, leading to disproportionate late-stage progression and complications of the disease with its myriad of affects in people with darker skin.

After the period of herxing, I was glad to have killed off the *Bartonella*, and to be able to focus on rest and recovery rather than managing active disease. I can't remember any specific instance where the tipping point of a relatively normal day suddenly switched into rage mode for the first time, and just the same I don't

remember when anger's unyielding grip let go. What does remain is the memory of a searing vision of having something, anything, in your crosshairs, a recall of the unrelenting agitation that caused me to chase people from rooms, and a desire to not be that person again. This comes very close to Tomkins's description of the person who experiences loss having had no prior script for successfully negotiating a severe stress through the iceberg that is their mass structure, their memory bank of distress (2008a, 330–31).

To accept that my Lyme disease had given way to a "post" or "chronic" syndrome—that certain symptoms may very well be with me for the rest of my life, and to have reached a point of accomplishment by having put the most acute phase of recovery behind me, opened a deep speculative hole of "what ifs." Reflection on experience (emotion) was a stress trigger to be avoided. As rage gave way to shame, I repeatedly realized that the only support I had had come from my family and my community, many of them people with autoimmune illnesses with whom I'd shared experimental treatment anecdotes and medical timelines online and while hooked up to IV units together in the second waiting room, that room beyond the front desk, where we, with our weakened immune systems, were kept well apart from other people waiting for COVID testing and treatment. If distress is a process of tearing apart, then we can apply this term to the separation of people with long COVID from those of us who carry other designated or adjacent, overlapping autoimmune and impaired immunity conditions for some time, and who have come to terms with achronicity and its affective cycles. At the very least, the cognitive data on post-COVID conditions suggests that learning from Lyme holds value for people with long COVID. The terrible irony is that whereas long COVID is widely accepted as a diagnostic term, even as it is poorly understood, long Lyme remains subject to skepticism, medical disparagement, and humiliation.

Narrative #3: Anti-DPPX: Disgust and Humiliation

My house was a sanctuary during the first pandemic spring. House wrens took up residence outside the window in a nook where I had set up my Zoom teaching office. I worked in view of their nest as eggs hatched and fledglings were fed. When the birds left the nest, I opened the window wide to let in the warm spring air. That was not all I let in. Tiny insects were now crawling on the papers on my work table. Occasionally I swiped one off my leg or keyboard as I ignored the problem for a day or two, trying to meet a deadline. But then one of my dogs, who slept by my feet in the nook, began to cry. Elderly and having lost the motor capacity to scratch, he struggled to relieve what appeared to be an agonizing itch. I spent time on the floor with him, trying to ease his discomfort. Within days, I too experienced a terrible crawling sensation and awoke one day to find my body covered in bites. Bed bugs, disgusting, I thought as I made an appointment with my doctor and proceeded to have my house treated with pesticides, wondering

how this could have happened despite having no guests or travel for three months.

I was terribly concerned about my pets and my own health, but I also felt no small degree of disgust and embarrassment about my body and my home. An inspector from a fumigation company asked me to raise my shirt and show him my bites before agreeing to treat my environment. But even after I had followed all the treatment protocols to for my pets, my home, and my body, the dog continued to scratch and cry and my bites and the rash escalated, covering my legs, arms, and torso, and reaching even my face and hands. I simultaneously developed a mysterious fine motor tremor and a gastrointestinal infection that left me dehydrated and weak. The early lesions appeared not to be healing. With the continuing bites, which occurred even in the absence of visible evidence of source, I experienced a distressing sensation on my skin that reached epic proportions at night, leaving me in anguish that was amplified by the heightened stress induced by the steroids prescribed to bring down my immune response.

After initial treatments with permethrin, a common treatment for the bird mites my doctor had assumed were the culprits, had failed, I was referred me to a dermatologist, a new doctor who saw me in office during pandemic months when most patients were seen online. In the first visit, the dermatologist also assessed my rash as an immunological reaction to bird mite bites and prescribed a new dose of permethrin and various ointments to quell the itch. When I returned uncured in a follow-up, the dermatologist had me seen first by their resident, who gave me a cursory examination and left the room to deliver a verbal report to the dermatologist. The dermatologist then entered the exam room, announcing as they walked in that they were surprised to see me back. I should have been cured by the treatment I'd been prescribed. The student's assessment, they explained, followed a pattern they often saw: a case of actual infestation and immune response to bites is followed by delusional parasitosis, a psychological disorder in which people who undergo a parasitic infestation believe they continue to be attacked after the crisis is over. They immediately referred me to a neurologist. This startled me. Whereas in the previous visit they had examined me, provided a diagnosis of insect bites and prescribed treatment, now they appeared annoyed and disgusted. They spoke in a manner that was strained, and which I perceived to be both patronizing and imperious as they struggled to explain the psychological pattern of behavior they "often saw in other patients." Standing as far from me as was possible in the small exam room, their face assumed a comportment of mild contempt, an affect that Tomkins describes, in his third volume, as a combination of dissmell, the protective comportment of spitting out something toxic, and anger (Tomkins 2008b, 22). Whereas before the dermatologist was interested and caring, invested in providing a cure, now, when the cure had not worked, they acted annoyed and dismissive, and spat out words invalidating my symptoms,

including the physical ones. They insisted the many bites, including those in the middle of my back where I could not reach, must in fact be self-inflicted wounds.

Adam Frank and Elizabeth Wilson (2021, chapter 6) remind us that for Tomkins, whereas shame is the affect of sociality, contempt is the affect of hierarchization. They quote Tomkins: "contempt strengthens the boundaries and barriers between individuals and groups and is the instrument par excellence for the preservation of hierarchical, caste and class relationships" (Tomkins 2008b, 216). I recall feeling a vague sense of fear in response to the doctor's obvious annoyance verging on anger and disgust at my ongoing physical state. If they could not help but spit out my experience, how would I get treatment? Their response was, in turn, a danger to me. Now it was my turn to feel defensively angry. When I objected that they had not even so much as examined me, and asked how they could levy a psychiatric diagnosis without an examination of the skin evidence I was there to show them, a dermatologist, to assess, they raised their chin and left their post near the still open door, approached my body where I was sitting on the examining table, and told me to open my examination gown. As I held open the garment, they tilted their head back in the classic posture of dissmell and, without touching me, wrinkled their nose, pointed, and gestured to the resident to take a few biopsies at this and that spot. Explaining to me that they were conceding to a biopsy and testing to humor me (they expected no evidence of arthropod toxins would be detected by the lab, they restated), they then left the room without further word. Clearly they were sick of me.

What was abundantly clear was that the dermatologist was disgusted, or more accurately dissmelled, by my unhealed flesh. My heightened state of anxiety, my ragged sleepless face, and my newly gaunt body seemed to trigger in them a kind of expulsive anger that I had brought a danger upon myself through delusional thinking and self-attack. My anxiety and my disgusting body, I insisted, were not the product of psychopathology, I did not have the mechanical means to produce these lesions—but they did not agree. I was confounded by their danger response.

Writing about the biosocial, Frank and Wilson (2021) note that displaced anger (which we read as danger, difference plus anger) confounds all parties. *Confound* means both surprised and mixed up. My anxiety was experienced as displaced by a doctor who was certain I was in fact cured of a heightened immune response to insect bites and was now self-inflicting wounds due to elevated stress. Startle is a reset button, electrical—it makes things happen. Like laughter and rage, it is contagious. But what happens when the startle button is held down for too long? While I waited for the outcome of the punch biopsy, the circle of confounded feelings grew. I saw the neurologist to whom the dermatologist had referred me. As incensed as I was that a dermatologist would deign to offer a psychological diagnosis, based on a verbal report of the resident and without examination no less, I figured no harm would come from following through. A psychological

disorder would have been welcome, in fact. Imagining this problem would have been far better than the reality of having to address a disgusting and recalcitrant physical cause.

The neurologist was, like the dermatologist, new to the clinic. Their affect was that of interest, which I appreciated greatly, responding in kind to their ideas and answering all of their questions. After some motor tests and a short interview, they offered, with great excitement, that they believed I may have a rare autoimmune condition, anti-DPPX encephalitis. When I met their statement with a blank stare, they offered a list of criteria, most of which I met (tremor, GI disturbance)—except for one hallmark sign: mental disturbance. They then offered, with mounting excitement, a description of a 2016 film based on a novel about this rare neurological syndrome. I would need a spinal tap to confirm, of course. But the decisive factor to justify that test, they said, was proving that my bites were self-inflicted, the result of a delusional state of mind.

At about this time I lifted my shirt and held out my arms, not for the first time, and asked the neurologist to simply take a look at the many little wounds. How could anyone produce this configuration of inflamed bites by self-infliction? Surely they did not think I was subjecting my body to hundreds of tiny puncture wounds, each forming with the same general shape and development of inflammation and (by this time) scarring pattern. They avoided the question, commenting with an expression of care that people did strange things in states of mental instability. If it turned out that I did have anti-DPPX syndrome, this would be an important case for their practice; the condition was still regarded as rare.

I was okay with this pursuit. The neurologist was clearly sharp. If I had this rare condition, I wanted to know. But in the week that followed, they hounded me with notes on my university medical center's app, probing for evidence of my (in)sanity. Their excitement was acute. They asked for contact with my friends and family members to confirm their hypothesis. I mentioned that I was in pandemic isolation and therefore not in contact with many people. I suggested we wait for the pending pathology report on the punch biopsies as a source that would provide them with the needed proof that my wounds were no self-inflicted, but they brushed it off, saying the dermatologist was certain the pathology report would be negative. They became quite insistent, adding to the sense of distress and anxiety I was already experiencing from the bites, exacerbated no doubt by the prednisone.

At the end of this grueling week, I received a phone call from the dermatologist. I'm very sorry, they said. The diagnosis of delusional parasitosis was wrong. Your pathology reports are consistent with arthropod bites. At this point I found this result to be great news, as I was no longer feeling sanguine about the spinal tap the neurologist had scheduled for me, based on them now clearly mistaken

diagnosis of a rare autoimmune encephalitis. I asked how to proceed. "Get an exterminator and clean your house," they advised before terminating the call.

Tests that followed with a GI specialist confirmed an *E. coli* and, later, a small intestinal bacterial overgrowth (SIBO) infection. Denied further diagnosis (such as a Lyme test, which could have revealed a possible *Bartonella* or *Borrelia* infection), I embarked on a regimen of environmental, dietary, and pharmaceutical treatments, following the advice of members of a group I'd met online composed of people undergoing similar circumstances. The range of issues and outlooks in this group was broad, and I followed what I would describe as the most skeptical and conservative path, resolving the array of problems, including the infection and the immune system reaction, over many months. My assumption, not proven because my doctors refused to order the necessary blood tests, is that I contracted a bacterial infection from bites from the mites that entered my home through the opened window after the departure of the house wrens from the nest outside. Having regularly handled and removed abandoned nests before with no ill effects, I surmised that the lowered immunity of my sick and elderly dog, and my own immune system being compromised by the dual infections, left us vulnerable to an insect that is, after all, everywhere outdoors.

The experience triggered distress, disgust, shame, humiliation, fear, anxiety, anger, dissmell, and—here is the organizing principle—interest. What got me through this experience and allowed me to come through on the other side without offing myself, a feeling shared by my colleagues here, by Manguso, and perhaps by others reading this who are dealing with something that causes immunological torment and upheaval with no end in sight, is interest in the unfolding of medical and self-treatment as I joined a support group and addressed the conditions, environmental and bodily, through the guidance of others who have been through the same ordeal. Without this shared experience and the ability to co-produce and co-perform affective transformation with the community of achronic "sufferers" who shared treatment strategies, stories of misfortune and loss, macabre jokes, and tactics for respite and relief, I would not have been able to tolerate the distress caused by the bioaffective muddle of feeling and fascination to which I was subject in the medical care of the dermatologist and the neurologist.

Anger carries difference. Tomkins characterizes it as toxic and to be avoided. Anger is distressing in part because it is forbidden in a culture of tolerant inclusion. My bites were angry, and therefore not tolerated. Distress about *other* affects, Tomkins tells us, is an archaic infantile taboo ignored by psychoanalysis. The infant is restrained from doing things that might be self-injurious. The child who pets a dangerous animal is punished and warned. The childlike adult who imagines themselves to live in harmony with insects and animals, who refuses pesticides and harsh cleaning products, is subject to ridicule and skepticism, or to

approval for their tolerance. Moral responses to lifestyle choices amplify the stress of zoonotic conditions, the reality of which remains subject to skepticism and disbelief even among veterinarians, who are amongst their most frequent human sufferers. Tomkins writes of the benefits of going "no affect" when one's distress or anger are likely to generate further distress, in the form of humiliation, punishment, or censure by "the parent." When the neurologist pressed me for family contacts, they were intensely excited, seeking to find a rare condition, to the point that they were willing to walk the line between ethical and unethical intrusion. Their interest and excitement (positive affects) led them to subject me to shame. My response to these excited demands was intensification of distress and, by the end of the week, consternation bordering on anger. I spit them out, shutting down communication with them, realizing that this was the only way to end this chain reaction of affects that I was no longer able to tolerate. With apologies, the providers voided the reports in my chart. My danger immune response was successful.

I was able to get past the press of their sense of (d)anger and its provocation of my own feelings of self-disgust, self-contempt (shame), and humiliation, as well as the disbelief and disgust I similarly faced from others, only because I gradually became engaged in affective and practical exploration of the mysteries of this (d)anger elsewhere, with interest and action sustained around a community that acknowledged the reality of zoonotic transmission with humor and which offered self-treatment and self-management guidance without the moral (in)tolerance paradigm, and with plans and situated actions organized around performative encounters with the reality of a world in which parasites have become a routine occurrence we must adjust to living with. The queer art of living with immune system failure requires shared experience-based knowledge, humor, and tactical science, not (in)tolerance. The cruel optimism of the institutional cure economy demands tolerance of a comportment of care in the form of treatment protocols that stubbornly cling to the stigma of foreign danger and the horror of dirty infectious things. To date, only eighty autoimmune diseases are formally classified and many care providers remain skeptical about their causal agents, their chronicity, and their recurrence, as well as their etiology and "nonspecific," non-acute cognitive and physical components. Contempt is rampant and skepticism is both legion and contagious. While the source of the intensity of my immune system's response to insect bites remains inconclusive (my doctors refused to administer a Lyme test despite my dog having tested positive and been treated for tick disease), I found myself able to tolerate the condition of living in the achronical biosocial state of crowd-sourced autoimmune experience through group self-care, and to find routes to cure in this context in which (d)anger was destigmatized, recognized as the pervasive and banal thing that it is. The sequelae of shame around institutional stigma, however, is something we all share as we endure our respective experiences of post-traumatic stress in the achronic time of medical (in)tolerance.

Conclusion

Our intention has been to bring forward a range of concerns about autoimmune affect and (in)tolerance. One of these concerns is the biopsychosocial mangle in which emotions emerge acutely and are later described ambiguously, in diagnosis, as a bio-psychological response to a pathogen (or illness associated with it), and as a co-occurring, self-intolerant condition—a delinked, irrational *emotional* response. As well, we described the temporal experience of intermittence and recurrence as *achronical* characteristics of chronic autoimmune disorders and feelings, folded into patterns of symptomatology. In many disease narratives, diagnosis is described as a goal or a climax after which feelings and their motivating sources change. It feels better to know, one would think. Anxiety is replaced by relief and, perhaps, determination to follow a new path to cure. In our narratives, we perseverate on the experience of living with and through speculative and retroactive diagnoses that forced reverse speculation, the rereading of one's past life, and which, rather than affirming a future in which a known condition and its way of life will always be present, generates skepticism about the thingness, the autonomy, of what we "have." We insist on the value at this moment in time of living with ambiguity in the affective mangle of autoimmunity rather than living with the flawed model of autoimmunity as a navigable system in which an immune self is replaced by a paranoid system of conquering unknown dangers.

Immunodeficiency and AI syndromes, understood as "post" acute conditions, make plain the cruelty of optimism that accompanies narratives of recovery and cure. These conditions are produced through an organizational arrangement of individuals, data, and time. That the present pandemic times in which we live will become achronically endemic, and that our bodies will harbor the viral matter and impacts they gleaned along the way, is a reality long understood and experienced by people with HIV (an immune deficiency syndrome) and autoimmune syndromes ranging from the widely recognized (such as lupus) to those cruelly subject to skepticism (such as post-Lyme), as well as those widely believed to be going undiagnosed, or those having been missed or mistaken as rare (such as anti-DPPX). Why medicine wants to seek out rare danger in something as dramatic as anti-DPPX encephalitis while refusing a test for the equally devastating but less novel syndrome of post-Lyme is a matter for another time, as we chose instead to take up our experience narratives, providing autodata that we believe will, in time and across other disjointed stories about affective experience in the time of autoimmunity, provide the empirical base for eroding endemic (in)tolerance on the matter of feelings as interconstituents of autoimmunity as a social condition.

Frank and Wilson explain that Tomkins offers a "disarmingly concise characterization of the negative affects as *inherently unacceptable*" (2021, 61). Experienced as punitive, their toxicity requires control. "All the negative affects

trouble human beings deeply. Indeed, they have evolved just to amplify and deepen suffering and to add insult to the injuries of the human condition" (Tomkins 2008b, 111). When your body is angry at itself and you can't fix it, you have to find means to produce self-tolerance, and affect is key. Affect is the antigen in this model. We need to remember the arbitrary nature of the antigen. A good antigen can go rogue. A bad antigen can be harnessed to cure. Perhaps it is time to let go of the coding of affect as negative and positive, so we can grasp the arbitrary nature of the toxic danger that is tolerance.

Notes

[1] We are three historians of art, science, and media, white and middle class. Two of us are tenured arts and humanities professors, cis female and over sixty, one straight and one queer. One of us is an arts and humanities graduate student, a unionized teacher who is cis, male, and straight. We identify as people with disabilities living in some relationship to autoimmunity. The link between autoimmunity and artificial intelligence implied in our acronym will be examined in another essay. Contact us at aihumanitiesresearch@gmail.com

[2] For an overview of models and discussion of the persistence of the self/non-self model, see Matzinger 1994; Pradeu 2012, 2020; Pradeu and Cooper 2012; Swiatczak and Tauber 2020; Tauber 1994, 2000. For critical theory, STS, history, disability studies, and medical science studies discussions of these models, see Anderson and Mackay 2014; Chen 2007, 2011; Derrida 2003; Ferri 2018; Haraway 1989; A. Martin 2010; E. Martin 1994; Margulis, Asikainen, and Krumbein 2011; Sengupta 2014; Tauber 1994; Titchkosky 2015; Tsai 2011; Wald 2008; and Weasel 2001. A sample of the biomedical literature: Ahsan 2023; Burnet, Fenner, and Clark 1949; Dumes 2020; Fritzler et al. 2021; Jerne 1966, 1974; Lynch and Platt 2009; Matzinger 1994, 2002, 2022.

[3] On anatomopolitcs, see the review of Foucault's use of the term in Koopman 2018, esp. 107–8. Also see Foucault 1977, 177–83; 2009, 243; and Adams 2017. On the relay between biopolitics and anatomopolitics, see Protevi 2009. On the difference between biopolitical regulation and anatomopolitical normalization, see Adams 2017 and Tremain 2018.

[4] On necropolitics, see Mbembé 2003. On necropolitics in "compassionate" care, see López 2020.

[5] See, for example, Matzinger (1994, 2000, and 2022), especially on the matter of danger, and Carter (2000) on the immune system as a model for designing a pattern recognition engine, expanding immune computation as a field that understands the immune system as paradigmatically open to learning, a trait viewed as a design asset to be emulated. We are exploring the autoimmune

system's historical relationship to artificial intelligence in another paper in development.

[6] In addition to the works by Tomkins cited, we acknowledge Frank and Wilson (2021) and Sedgwick and Frank (1995) as canonical sources for understanding the affect theory of Tomkins.

[7] On artificial intelligence and affect, see Wilson 2010.

[8] For some of the key feminist STS literature on tissue cultures, see Cooper and Waldby 2014; Landecker 2007; and Waldby and Mitchell 2006. We do not have space here to develop our argument, building on Cooper and Waldby (2014) and Wilson (2010), that tissue is an affective interface meriting analysis along the lines of Tomkins's understanding of the skin and the face.

[9] We acknowledge the difference between immunodeficiency and autoimmune conditions. Whereas the former describes lacking or compromised immune power, the latter describes a misdirected active response. Previously understood as being exclusive, the two sets of conditions are increasingly recognized as inter-causal, co-productive, and not simply co-occurring. As one rheumatology researcher puts it, they are two sides of the same coin (see Schmidt, Grimbacher, and Witte 2018).

[10] There is no space to discuss concerns with work on care. We point to Andrea M López (2020) on care/brutality as a cautionary read in the context of the politics of interdependence (Care Collective 2020), María Puig de la Bellacasa (2011), and the special issues of *Social Studies of Science* (Martin, Myers, and Viseu 2015) devoted to this concept. We share the sentiment of the edited volume *Against Health* (Metzl and Kirkland 2010) in addressing the affective and physical dimensions of care as a strategy in biomedical normalization.

[11] The causality of persisting symptoms in Lyme patients is a highly contentious topic in the medical field, and is explored in greater detail in *Divided Bodies*, in which Abigail Dumes (2020) contends with the split-self concept of autoimmunity.

[12] Here we note that post and chronic are not the same thing. Their corollaries are *after* and *always*, which may co-occur, or not.

[13] Anger has yet to be widely discussed in the feminist STS and critical theory literature. For an analysis of anger in feminist film aesthetics, see McHugh 2021.

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professors, cis female and over sixty, one straight and one queer. One is a technology and arts and humanities graduate student, a unionized teacher who is cis, male, and straight. We identify as people with disabilities living in some relationship to autoimmunity. The link between autoimmunity and artificial intelligence implied in our acronym will be examined in another essay. Contact us at aihumanitiesresearch@gmail.com.