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A Viral Pedagogy: Undoing Things with Long COVID's Autoimmunities

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Abstract

This article focuses on the scientific and political transformations that the COVID-19 pandemic continues to engender through its impact on the epistemological and ontological foundations of autoimmunity. By discussing an online seminar taught during the COVID pandemic winter of 2020–21, the article considers how the viral autoimmunities involved in severe and long-haul COVID pathogenic mechanisms have fostered transformative pedagogical and therapeutic experiences, spaces, and events. As a highly contagious viral disease, COVID highlights disparities in access to healthcare, whereby certain (racialized, disabled, ageing) bodies are unequally burdened with the labor of staying alive. Predicated on a foundational separation between inside and outside, or between the internal, vulnerable, visceral space of the individual(ized) "human" body and an external(ized), hazardous, contaminated, foreign environment, COVID measures for avoiding viral exposure became paramount, reciting a genealogy of distancing, protection, containment, and isolation. By examining how concepts and practices of autoimmunity animate cutting-edge biomedical research, this article asks, How does autoimmunity undo normative models of health predicated on a militarized model of immunity? How might the viral shift our understanding of autoimmunity and what might autoimmune conditions teach us about virality? In short, what can autoimmunity and the viral show us about uncertainty, care, and transformation in therapeutic and pedagogical contexts?

Keywords

autoimmunity, COVID, long COVID, uncertainty, pedagogy, care

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> Stop playing pretend that we know when we don't.
> Stop playing pretend that we're well when we're not.
> Stop playing pretend that the world is certain when it's absolutely the opposite of it.
> —ALOK (@alokvmenon), excerpt from a video posted on Instagram, August 15, 2022[1]

Introduction

In *Virology: Essays for the Living, the Dead, and the Small Things in Between,* Joseph Osmundson observes that "we live on a viral planet. They were here first. We are their guests, not hosts" (2022, 61). As a virologist, queer activist, and self-identified "fag," Osmundson is interested in pinning down the inherent queerness at the core of viral activity—what a virus can do (and not do), and specifically what a virus can do (and not do) to human cells, human bodies, human life. Osmundson theorizes the queer relations that emerge in response to viruses, spanning experiences and networks of care, pain, fear, desire, mourning, and loss. As a microbiologist, he offers readers an informative and poetic remix of viral and immunological expertise coupled with intimate accounts of his own queer encounters with viruses. Osmundson is as invested in communicating what he (or the collective "we" of universalized scientific subjectivity) knows about the viral as in articulating what is not known, either because it cannot be known or because that knowledge is yet to come. Much like in ALOK's remarks above, this investment in the unknown, in what is uncertain or indeterminate, is not simply an intellectual exercise. It is rather an urge to *stop pretending* that "we're well when we're not" in order to accept and articulate how odd, how queer, being alive really is—and to use uncertainty as a therapeutic, pedagogical, transformative political tool, not to recuperate it but to put it to good use, informing queer epistemologies and ethics. It is an urge, shared by me and many others, to do queer things with uncertainty, and specifically, with the uncertainties at the core of viruses and other pathogens, of pandemics and epidemics, of cells that burst open, of bodies that break down, fade away and die.

In this article, I consider the uncertainty at work in viral autoimmunity, particularly in the autoimmune mechanisms involved in severe and long COVID, to show the pedagogical and therapeutic potential of not knowing. In tandem with autoimmunity's riddles, I examine the Staying Alive online seminar I taught at Humboldt University of Berlin during the first winter of the COVID pandemic (from November 2020 to February 2021), when (unvaccinated and under lockdown) we tried to survive another gloomy Berlin winter, this time one with a darker core and a more sinister edge. As daily life seemed to be reduced to prevention measures and the dynamics of infection and treatment, followed by convalescence or death, my biweekly discussions with the students became long

collective meditations on fragility, debility, disability, and socioeconomic privilege as a determining factor in quality of care and COVID outcomes. We talked about who is left to die and who gets to stay alive, but we also discussed the blurry lines between health and illness, the vital and the deadly we carry inside and encounter every day. The lifeworlds we brought with us and shared during those Zoom sessions were filled with uncertainty about what viruses and bodies can do. As we stopped "playing pretend that we're well when we're not" (ALOK 2022), we were able to talk about COVID and AIDS, polio and measles, avian flu and swine flu, about depression and social anxiety, loneliness and immunodeficiency, chronic pain and delicate lung cells. We also talked about the clinical puzzle of autoimmunity and of chronic autoimmune conditions, particularly in relation to artist Carolyn Lazard's writing on chronic illness . As students shared stories about their own experiences of grappling with illness, pain, and disabling conditions, from mental to physical health issues, we became better equipped to digest and respond to the course readings. Learning about (auto)immunity was a way of learning about our bodies (our/selves), but it was also a way of undoing, disassembling the restrictive limitations of the "self," that isolated unit, to revel in the permeable, reactive, disorderly, multispecies entity formerly known as "ourselves." The collectivity summoned by the viral spans its vestigial, nearly imperceptible formations, as well as its massive, humanly incomprehensible scale. Feeling somewhat lost in this immensity, we grappled in the darkness of that long semester with all the unknowns COVID had irrevocably introduced into our lives, as we sought to tolerate far more uncertainty than we could bear. This, I argue, is the challenge that autoimmune phenomena place on our bodies, clinical protocols, and biomedical studies, demanding an unconditional acceptance of uncertainty, ambiguity, unpredictability, even chaos.

In what follows, I deploy what Vicky Kirby (2017) calls "autoimmunity's riddle" as a pedagogical strategy to engage with uncertainty in (post)pandemic times, using the Staying Alive seminar as a case study. I begin by considering some of the political, conceptual, and material entanglements of virality and autoimmunity. I engage with theoretical work on autoimmunity and on the politics of immunity in the context of biopolitics and sovereignty, in particular Kirby's (2017) transdisciplinary critique of autoimmunity and Emily Martin's (1994) anthropological research on immunology and the "flexible" body. I propose the concept of immunoplasticity to account for the ways in which immune systems learn, change, and adapt, modulating their responses according to interactions with external actants, from pathogens to vaccines. In the second section, I offer a critical overview of the Staying Alive seminar, paying particular attention to the pedagogical tools the students and I employed to make it a space of radical hospitality that enabled transformative, therapeutic experiences to take place. In spite of all our limitations and shortcomings, especially during such an uncertain, turbulent time, we managed to collectively engage in daring, candid conversations on topics from ableism in academia to racism in healthcare. In the

third section, I return to an examination of how autoimmunity functions in long COVID clinical scenarios. Finally, I return to how uncertainty can be productive, engendering pedagogical and therapeutic experiences with unexpected outcomes.

Viral Autoimmunity

Authors such as Ed Cohen (2009), Emily Martin (1994), Vicky Kirby (2017), Jacques Derrida (2003, 2005), and Roberto Esposito (2011) have theorized immunity as a biomedical and political concept, considering its intellectual genealogies alongside clinical and cultural practices. In *Intolerant Bodies: A Short History of Autoimmunity,* Warwick Anderson and Ian R. Mackay (2014) frequently mention the impact of contagious diseases as contributing to the onset of autoimmune conditions. The authors approach autoimmunity as a "concept of pathogenesis," one that is supported by the epistemological framework of a highly individualized and specialized entity called the Self: "Notions of *individual* reactivity and sensitivity flourished best where the prevailing thought styles and sensibilities nurtured such ideas" (2014, 5, emphasis added).

Evidence of viral involvement in the pathogenesis of a vast range of autoimmune conditions (including lupus, type 1 diabetes, multiple sclerosis, and celiac disease) is well documented across clinical and biomedical literatures. Causality, however, remains remarkably difficult to establish, and the role of viral infection is usually presented as one factor (among others) involved in autoimmune pathogenesis. To use one specific example from a recent review article on lupus (systemic lupus erythematosus): "A *causal link* between viral infections and autoimmunity has been studied for a long time and the role of some viruses in the induction or exacerbation of systemic lupus erythematosus (SLE) in genetically predisposed patients *has been proved*" (Quaglia et al. 2021, 277, emphasis added). This citation corroborates similar statements from original scientific articles and review articles on autoimmune pathogenic mechanisms across the fields of biomedicine, clinical medicine, and public health, as well as more specialized fields like virology, immunology, and microbiology (Getts et al. 2013; Hejrati et al. 2020). Viruses occupy a liminal position between the living and the nonliving, becoming enlivened through their interaction with living cells. Unlike nonliving matter, viruses have the capacity to replicate exponentially, which gives them an uncanny resemblance to living beings, in particular bacteria and fungi. Although organic and inorganic matter also has the capacity to become enlivened through interaction with other substances, it cannot replicate itself exponentially. This crucial difference is what makes viruses so common and their presence so pervasive. Viruses are, quite simply, everywhere.

Given this well-established link between viral infection and autoimmunity, even if it has not (yet) been proven to be a causal one, why does long COVID seem to pose such a challenge to standard Western clinical practice? And why has it

remained a clinical puzzle in recent biomedical research? Partly, the answer resides in the characteristics of the SARS-CoV-2 virus itself: its molecules have the capacity to affect virtually any cell in the human body, dispersing its effects across organs and introducing a high degree of variability in the range of symptoms. However, there is also, I argue, a paradigmatic clash at the core of COVID models of pathogenesis, one in which germ theories of disease, epigenetic models, and the specificity of (auto)immunity compete and collide. The notion that every (human) body responds to pathogens in the same standardized manner, a central tenet of germ theories of disease, is contradicted by the principles of genetic diversity and variability. Moreover, it is at odds with the highly idiosyncratic, disparate responses of autoimmune mechanisms defined by the "individual reactivity and sensitivity" Anderson and Mackay (2014) highlight above, understood as a series of responses that are both unpredictable and changeable over time.

Although the production of autoantibodies in small amounts is common, it does seem to affect immune function and it can cause tissue damage. In COVID clinical scenarios, it is difficult to assess whether autoantibodies are produced due to more severe COVID infection, resulting in inflammation and cellular damage, or whether it is precisely the presence of autoantibodies that precipitates a severe or acute clinical scenario. In an overactive immune response, cytokines (signaling proteins that are part of the immune system) produced in excessive amounts can cause cellular damage. By contrast, autoantibodies "gone rogue" no longer recognize the tissues and cells that "belong" to the self and are thus unable to distinguish them from pathogens (Khamsi 2021).[2]

I want to propose here that the neologism *immunoplasticity* (which appears across a variety of studies on immunity across fields like internal medicine, neurology, psychology, immunology and cardiology) might capture something of the flexibility (Martin 1994) and plasticity characteristic of this disparate collective of cells and molecules known as the "immune system."[3] While their activity remains to some extent coordinated and recognizable as an epistemological and ontological identity, I contend that the cells and molecules involved in immune activity function less as an organized system than as a heterogeneous network of actants distributed across a given organism. Each participant in immune functioning, from T cells to macrophages, has some degree of ability to act, react, and overreact independently, including in ways that are damaging and even deadly to a living body. Immune activity shifts over time in ways that can be unpredictable, making it difficult to detect and measure variance and variability. These unpredictable shifts can be understood along the lines of plasticity, in the sense of an organism's ability to change and adapt to changes in its environment and more specifically in relation to neuroplasticity, or "the ability of the nervous system to form and reorganize connections and pathways, *as during development and learning or following injury*" (*OED*, n.d., emphasis added). In turn,

immunoplasticity might thus provide a useful model for considering how immunity is also highly responsive, and not always in a beneficial way for the self/body/organism. Ultimately, the immune system, an agglomerate of cells and molecules, does not operate as a singular, coordinated unit, but rather as a heterogeneous collective of partly autonomous entities, acting in an agential manner that is not necessarily serving the perpetuation of any single organism's life. Understood perhaps as the biological materialization of suicidal intent targeting the suffering or dying body, autoimmunity manages to exceed such a reading once we consider the foundational *otherness* of both the innate and the adaptive immune networks circulating within a living (human) body.

Distinguishing between the beneficial and damaging effects of any treatment, encapsulated in the term *pharmakon* as deployed by Derrida (1981) and Elizabeth Wilson (2015), remains difficult when critically engaging with autoimmunity. In politically charged situations, particularly in clinical settings, having to quickly decide what is good or bad, right or wrong, helpful or destructive, is never a straightforward matter. To make empirical decisions informed by reason, it becomes often necessary to reduce the complexity of a problem, aiming to (hopefully) increase predictability and minimize the risk of error. Statistical probability, complemented by pragmatism and medical intuition, might indeed save lives—but whose lives? For those whose bodies fall outside normative parameters, often defined according to racist, ableist, ageist, and heterosexist scientific standards, the simplistic imposition of normative expectations can prove harmful or even fatal.

Elsewhere, I highlight how the biomedical concept of "viral load" might be useful for theorizing the uneven distribution of the pandemic effects so many had to bear (Chen 2021), in addition to the already unbearable burdens of paid and unpaid labor, disabling and chronic conditions, and the bodily impact of dealing with violence, racism, ableism, heterosexism, poverty, and social exclusion on a daily basis (Varino 2017, 2021, forthcoming). In a study by Jesse Fajnzylber et al. (2020), COVID severity and mortality are closely linked to viral load, with samples collected from the respiratory tract, plasma, and urine showing a higher prevalence of detectable SARS-CoV-2 in patients with severe COVID. The study is corroborated by similar scientific findings about the role of immune dysregulation in severe COVID clinical scenarios (Giamarellos-Bourboulis et al. 2020). Several studies also suggest a strong connection between COVID severity and risk of developing long COVID (Del Rio, Collins, and Malani 2020; Subramanian et al. 2022). More recently, strong links between long COVID and autoimmunity have been observed, with research showing increased risk of acquiring an autoimmune or autoinflammatory disease after COVID infection (Hosseini et al. 2022; Kocivnik and Velnar 2022; Mobasheri et al. 2022; Sharma and Bayry 2023), and autoimmunity emerging as a common feature of long COVID clinical scenarios (Mehandru and Merad 2022; Rojas et al. 2022). Taken as a whole, these studies

point towards links between viral infection and autoimmunity that continue to dispute the militarized model of immune responses mounted by a "sovereign self" against "foreign invaders." The collapse between self and nonself is part of the ontological and epistemic crisis that autoimmunity inaugurates. I argue in this article that it is also part of the transformative potential that the viral instigates as the catalyst for chronic autoimmune and immunodeficiency conditions, from chronic and long-term conditions directly caused by a specific virus, like long COVID and AIDS, to autoimmune conditions perhaps more sinuously linked to viral pathogenesis, like multiple sclerosis, lupus, celiac disease, or type 1 diabetes.

It is tempting, of course, to try to turn all the pathos and confusion of a global pandemic, or the derangement of a personal health crisis, into a lesson, a didactic experiment, something "we" can all learn and grow from, a process of healing that returns the fallen body/self to a prior, original state of order, purity, and productivity afforded by good health. I want to avoid that schema while still finding a way to account for the (often convoluted, transversal, tangential, disruptive) learning that did take place during the Staying Alive online learning experiment in the winter of 2020–21. It was transformative, in a distinctly therapeutic and pedagogical manner, to grapple with the troubles and joys of living in a body that breaks down, eluding our control attempts, and to consider how social systems fail specific groups and communities. During the semester, we returned over and over to historian of science Edna Bonhomme's (n.d.) generative question, "what makes people sick?" and sought to answer it in different ways each time. The experiment was not conclusive, though; its results only yielded opaque, partial, highly subjective, and non-replicable data, demanding many more questions and tearing apart what we thought we already knew.

Staying Alive: A Pandemic Seminar

Over the course of the winter semester 2020–21, I taught a graduate seminar called Staying Alive: Queer and Feminist Interventions on Illness and Disability from the AIDS Crisis to the COVID-19 Pandemic at Humboldt University of Berlin.[4] The seminar was aimed at students in the master's program in gender studies, but students from all subjects were allowed to participate, and there were medicine and biology students enrolled alongside psychology, social sciences, and humanities students. For many of them, this was the first semester of their MA and several had moved to Berlin to study. Advanced BA students were also welcome, provided they felt that they could follow the readings and engage in the seminar discussions. My main goal was to offer students (several of whom were doing their exchange year or semester abroad as part of the Erasmus program, a student exchange program established among European universities) the time and space to discuss what was happening in their own lives and in the world as that first pandemic winter unfolded. To accomplish this, I sought to place the pandemic within wider contexts, encompassing continuities and discontinuities with the AIDS crisis and other previous pandemics and epidemics (such as SARS,

the 1918 flu, polio, and measles), but also within wider contexts of health activism, racial and disability justice, climate change, and environmental rights. I chose readings that highlighted disability and illness narratives, which demonstrated the unevenly distributed physical and emotional labor demanded from certain bodies in order to stay alive in a racist, ableist, ageist, classist, and heterosexist world. We read disability scholars such as Mel Chen (2012, 2021), Lisa Diedrich (2016), Margrit Shildrick (2009), and Rosemarie Garland-Thomson (2017) alongside excerpts from *Disability Visibility* (2020), edited by Alice Wong, and from *Care Work* (2018), edited by Leah Lakshmi Piepzna-Samarasinha. The students especially responded to Carolyn Lazard's writing on chronic illness and to critical work by Cindy Patton (1990) and Paula Treichler (1999) on the AIDS crisis, a crisis that some younger European (and particularly German) students, who have free access to widely available PrEP (pre-exposure prophylaxis, a preventive course of medication that reduces the risk of HIV infection by over 90 percent), had simply assumed was over.

We took an intersectional approach to discussing how the COVID pandemic was affecting each one of us in remarkably different ways, from week to week, and to think about how it was affecting the people we knew or heard about, via the media or other sources, in places far away. This was speculative, demanding work that required creativity, empathy, and solidarity—resources we did not necessarily have at the beginning of the semester, but which we gradually developed through systematic, slow, careful engagement with the readings and media materials we shared on a weekly basis. Foregrounding first-person narratives about disability, illness, and mental health was crucial for me as a lecturer to destabilize the notion of normative health as a given for most. Rather, we started from the premise that most people undergo periods of disability during a lifetime, and that many of us deal everyday with disabling and/or chronic conditions (including mental health issues such as depression, anxiety, and post-traumatic stress), either our own or experienced by those around us, including those for whom we provide paid or unpaid care labor. Examining how these conditions are exacerbated by racism, ableism, sexism, and poverty became a core component of the course, especially as we reflected on the rampant ableism still prevalent in academic settings.

As is usually the case with most humanities and social sciences courses at German universities, attendance was not mandatory, and as a lecturer I emphasized that attending the Zoom sessions was completely voluntary and that no one should feel pressured to attend, participate, or even to turn on their cameras. All readings and course materials were available online and all exercises and activities could be completed asynchronously.[5] My aim was to engender a welcoming, radically accessible atmosphere that centered care, support, and mutual responsibility rather than academic achievement. This atmosphere was informed by but departed from the current frameworks of diversity, equity, and inclusion prevalent in North American and in some European universities, which still presume a

universalized set of standards concerning individual class participation and academic output, predicated on principles of equal access to education that can never be actualized under the grueling conditions of late-stage capitalism. Rather, the temporary learning community we formed was supported by the freedom-oriented radical pedagogies of educators such as Paulo Freire, bell hooks, Augusto Boal, and Gloria Anzaldúa, which question the very conditions of possibility for learning and how power structures shape educational institutions.

The student responses, from their contributions to the weekly Zoom sessions to their required course assignments, astounded me. Gathered together, they formed a collection of insightful and sophisticated accounts of how our bodies limit, fail, support, and enable our lives. The transformative work this seminar did was both pedagogical and therapeutic, replacing the ableist normativity that regards good health as a given standard, and disease and disability as abnormal or deviant, with an acknowledgment of how particular bodies and communities bear the brunt of staying alive with minimal support and resources. Instead of assuming states of health and disease as singular experiences, we approached them as interconnected events, socially and materially mediated. The viral, then, emerged as a collective and relational category in this seminar, something that binds human and nonhuman bodies across historical times and geographical regions, rendering what may appear to be discreet units as living assemblies.

Together, we made a concerted effort to think about COVID and other medical conditions beyond the binary of either health or disease, challenging the dualism of "bad" foreign pathogens trying to invade and conquer our "sovereign" bodies while "good" immune systems defend the territory of a familiar self. As one student succinctly put it, "at the end of the day it is our immune systems that make us sick," either by doing too much or not doing enough. As intricately distributed networks, immune systems rely on shared resources, including collective immunity and environmental conditions, to be able to keep bodies alive. As a pedagogical and therapeutic tool, viral autoimmunity encapsulates how a single pathogenic agent can instigate a broad spectrum of immune responses among individuals and across time, some of which are not entirely understood within current scientific and clinical frameworks of causality, predictability, or replicability. In what follows, I continue to examine the confounding phenomena involved in viral autoimmunity, initially introduced in those seminar readings and discussions, and much indebted to the incredible generosity, honesty, and creativity of the students participating in the Staying Alive seminar. The viral is transformative and relational, and engaging with its uncanny, at times volatile and destructive liminality can indeed foster learning and healing—even as it kills, harms, and terrifies us, even as we mourn our losses while striving to stay alive.

Breaking It Apart: (Un)doing Things with (Long) COVID

Dr. Ashish Jha (2022), the White House COVID-19 response coordinator from April 2022 to June 15, 2023, observed during a White House press teleconference in 2022 that "long COVID is almost surely not one clinical condition; it's probably multiple different things—how do we understand that, *break that apart*?" (emphasis added). Clinical medicine's intolerance for the multiplicity of long COVID may lead to a more in-depth understanding of each of its myriad manifestations, which might result in better treatment, care, and disease management in the near future. This is especially the case if medical specialties can study and mitigate some of the more specific symptoms or conditions present in long COVID clinical scenarios. But while it is true that cardiology, gastroenterology, or neurology, for example, might excel at providing treatment for a specific array of symptoms, there is also value in considering how a condition might benefit from a more generalist clinical approach. Immunology, especially in its attention to the idiosyncrasies of autoimmune activity and its historical interest in understanding variability in individual responses, may well have such a role to play in addressing long COVID. During the Staying Alive seminar, students routinely highlighted precisely this variability, commenting on how the disorienting clinical scenarios of COVID spanned a confounding range of physical, psychological, and neurological symptoms, even at what was then still an early stage of the pandemic.

At the time of writing, the unsettling temporalities of COVID continue to make it a major public health concern on a global scale, both in terms of the effects of COVID infection on older, disabled, or immunocompromised individuals and communities, especially with recurring COVID waves caused by the spread of new variants, and in terms of the long-term lingering symptoms of what has become known as long COVID (also known as long-haul COVID, post-COVID, chronic COVID, or post-COVID syndrome, sometimes abbreviated as PCS). Since July 2021, long COVID has been recognized in the United States as a disability under the Americans with Disabilities Act. The World Health Organization (2021) currently defines long COVID as "a condition characterized by symptoms impacting everyday life, such as fatigue, shortness of breath and cognitive dysfunction, which occur after a history of probable or confirmed SARS-CoV-2 infection." The chronic condition, with its confounding, often vague range of symptoms, continues to pose a causality riddle. Why do some patients remain sick for so long, instead of recovering from COVID after infection subsides and the SARS-CoV-2 virus is no longer detectable? As already mentioned above, the probability of developing long COVID appears to be, at least to some extent, correlated with severe COVID symptoms, and severity of symptoms seems in turn to be correlated with both viral load (quantified levels of detectable SARS-CoV-2 measured in samples collected from the respiratory tract, blood plasma, or urine) and with the presence of autoantibodies in blood samples.

Long COVID's disorienting clinical scenario is not by any means unusual. The sequalae left by acute or severe infectious disease often manifest in ambiguous clinical scenarios that can extend over the course of weeks, months, or years. Currently, there is no standardized testing or specific clinical protocol to determine whether someone might indeed have long COVID. Healthcare professionals assess the probability of long COVID by considering clinical history, a standard medical examination, and possibly a range of tests, from blood tests to X-rays. In addition to this, whether long-haul, critical, severe, moderate, or mild, COVID cases have tended to present a vast range of symptoms, with differently affected tissues and organs. Although the condition often produces respiratory symptoms ranging from mild to acute, its gastrointestinal, cardiovascular, neurological, and dermatological symptoms have also been thoroughly documented and studied since the early stages of the pandemic (Ciechanowicz et al. 2020; Jiang et al. 2020; Mehandru and Merad 2022). In addition, the role of inflammation and autoimmunity has also been central in biomedical and clinical studies of COVID pathogenic mechanisms (Ehrenfeld et al. 2020; Khamsi 2021). In severe and critical COVID cases, lowering an excessive immune response with immunosuppressing drugs such as steroids must be carefully balanced with the risk of increasing the degree of infection due to a lowered immune response, thus potentially triggering viral replication resulting in cellular and tissue damage.

Autoimmune processes are in fact an integral aspect of immune function regulation, rather than an exceptional or rare occurrence. For example, while every living body produces autoantibodies, they can either instigate autoimmune pathogenesis or, conversely, play a pivotal role in regulating immune responses such as inflammation (immunosuppressants like steroids, for example, seek to mimic this regulatory effect). Similarly, T cells can contribute to the onset of autoimmune pathogenic mechanisms while also, paradoxically, helping guard against autoimmune pathogenesis and even engendering protective autoimmunity (for example, in the case of central nervous system injuries). As Kirby has observed, "autoimmunity's riddle must accommodate the following paradox: the concept of *autos*, or *ipse* can no longer be some*thing* which is compromised, threatened or even secured, and this is because an identifiable self which appears under attack may never have existed" (2017, 50). It is thus not surprising to see that autoimmunity unsettles a modern biomedical paradigm where "proper" immune bodies are the norm, either through innate immunity or by acquiring it through contact (and possible infection) or through vaccines and other inoculation techniques. In line with Kirby's assertion that an "identifiable self…may never have existed" in the first place, I want to suggest that something far more complex than a scenario of self-destruction is at work in autoimmune conditions. I want to consider how any given immune system does not in fact belong to a specific "self" or even to the organism within which it operates, but is rather a heterogeneous agglomerate participating in a vast network of ecosocial

immune functioning, eventually determining the survival or death of an organism or even of an entire community.

Autoimmunity ostensibly inaugurates the collapse between self and nonself, brought along by a disassembling self that can no longer maintain the only thing that made it recognizable in the first place: its distinguishability from the nonself. Chronically autoimmune bodies may not directly infect others, but neither do they endanger themselves exclusively. They also pose a threat to the stability of normative models of health and disease, dis/order, dys/function, cure and harm, and perhaps even more so, to models of causality and normativity, installing a foundational ambiguity (Kirby's "riddle," mentioned above) that can never be finally resolved. The paradoxical ambiguity at the core of autoimmunity clashes with modern scientific paradigms founded on causality and replicability. In turn, clinical protocols rely on some degree of predictability, so that error can be minimized during diagnostic procedures and ensuing treatment recommendations. The "auto" introduced by autoimmunity refers to a modality of reflexive immunity that is oriented towards the (ostensibly) singular, unified, autonomous, individuated organism. In the standard, dominant biomedical narrative, autoimmune pathogenic mechanisms can then be reduced to a matter of error or failure, whereby a body attacks its own cells and tissues *by mistake*, losing the ability to recognize its own visceral materiality and to distinguish friend from foe, self from nonself. Caught up in the circuitry of pathogenic autoimmunity, this dysfunctional, disorderly body harms "itself" by mounting what is deemed as an unnecessary immune response that causes inflammation and possibly cellular and/or tissue damage. By contrast, autoimmune mechanisms that have a beneficial effect on an individual organism (like the autoantibodies involved in immune self-regulation or the T cells providing protective immunity mentioned above) are relegated to the entirely different category of immune functioning, foundationally distinct from autoimmunity in the clinical sense of pathogenic mechanisms involved in the onset of allergic reactions and of chronic autoimmune conditions. Even though the effects are diametrically opposed, the autoimmune mechanisms at work appear to be essentially the same. This duality at the core of autoimmunity, this ability in some cases to protect and regulate immune functioning, and in others to have harmful, incapacitating, or even deadly effects, further complicates "autoimmunity's riddle" (Kirby 2017).

Ambiguity and unpredictability are not well tolerated by current scientific and clinical paradigms seeking to prevent, control, and manage disease transmission by eradicating doubt and minimizing error. The ambiguity at the core of autoimmunity poses numerous epistemological and ontological challenges for current immunological knowledge. Similarly, a lingering, unresolved case of prolonged COVID, presenting with an array of diverse symptoms ranging from mild to debilitating, troubles assumptions about normative immunity. The current clinical expectation is that an appropriate, standard immune response should

return the body to its previous (pre-COVID) healthy state. Instead, bodies exhibiting COVID-related symptoms long after the standard (a statistical average based on what is more frequently observed) four- to six-week period of convalesce are seen as unruly, deviant, damaged, and damaging bodies whose "rogue" antibodies and autoantibodies cause unnecessary trouble to the sovereign (yet subservient) citizen-self, one who had ample opportunity to return to its proper, (re)productive, healthy condition prior to COVID infection. This refusal to return to a pure, original, pre-COVID state undermines the sovereignty of the self just as much as it undermines the economic sovereignty of the nation-state, with wealthy countries such as Germany and the US now grappling with the long-term effects and added costs of a reduced and/or less productive workforce due to long COVID. Autoimmunity is transformative, I argue, for the uncertainty it entails and the ambiguity it installs within an immune enterprise predicated on defending a sovereign self that may well not exist—its ontological and epistemological contours too blurry to maintain any recognizable stability. Autoimmunity, as a clinical and social phenomenon, demands massive adjustments on a visceral level, by cells and tissues, and large-scale accommodations on an epistemic, ethical, political, and socioeconomic level. Autoimmunity arrives at the scene of biopolitics to disassemble the self, that kernel whose freedom, integrity, and autonomy still animates neoliberal late capitalist paradigms, although probably not for much longer.

In fact, I would offer that long COVID's clinical puzzle might seem far less puzzling after a paradigmatic shift to autoimmunity as a core immune mechanism that is not necessarily pathogenic nor foundationally opposed to immune response, but might be rather contiguous with it. Far from rare or exceptional, autoimmune responses occur alongside immune ones, mounted by an immune system that is not the property nor the extension of a sovereign self, but simply an agglomerate of actants that may or may not behave in a way that is (seemingly) beneficial for a specific body, community, or population. Since it is remarkably difficult to distinguish between right or wrong, good or bad, cure or harm, avoiding nihilism or political apathy may not be simply a matter of taking sides or of developing reasonable arguments based on empirical evidence. As outlined in the previous section, it may instead require an increased capacity for tolerating paradoxical ambiguity, ambivalence, uncertainty, indeterminacy, accompanied by an commitment to engage with the imperceptible (Murphy 2006), vestigial, or negligible—that is, towards not knowing, by making ethical and epistemic space in an argument, clinical protocol, or laboratory study, for what we do not know (yet) about what we know and for what we do not know that we don't know. Throughout the Staying Alive seminar, the students and I experienced again and again how developing a taste for the intolerable and stomaching failure and error are key therapeutic and pedagogical outcomes of a transformational approach to autoimmunity in general, and specifically towards its role in pathogenic mechanisms, including COVID's erratic clinical expression.

Centering the role of autoimmunity in immune functioning enables a renewed understanding of long COVID. Conversely, COVID data, narratives, and practices provide robust evidence of autoimmunity's central role in infectious disease mechanisms, corroborating previous studies on the links between autoimmune and viral pathogenesis. While the immune system remains in many situations a highly adaptive, enclosed, self-regulating network, using its own resources to manage excessive activity, it is also true that the many immune actants at work within a body are not simply aligned with keeping it alive and well. They are also capable of harming and even destroying it, demonstrating how no action or entity can aspire to be entirely pure, benign, or beneficial. The murky ethics and politics at the core of immune functioning should come as no surprise, since an immune system is an agglomerate of interconnected yet independent entities whose agency does not align in any conscious or intentional manner with that of a living body in all its visceral, affective, and cognitive complexity. The immune system behaves less as a seamless, coordinated system than as an eclectic collective of highly specialized living cells and molecules, in close cooperative and at times antagonistic relations with the unicellular life forms (such as bacteria and fungi) that make multicellular life possible. In fact, the (innate and adaptive) immune system active inside each living body, remarkably similar across all vertebrate life forms, may well be as foreign as anything else circulating within a body or interacting with it.

Understanding the autoimmune mechanisms at the core of COVID across its myriad clinical manifestations can be as useful for biomedical research and clinical practice as it is for transformational political projects. The modern Western sovereign self that "we," the individual subject-citizens of overdeveloped western(ized) affluent societies, so dearly cultivate and manage, can barely sustain itself. Its ecologies can be stormy, its immunoplasticity unruly, its multispecies heterogeneity a hybrid gone rogue. If we take seriously the paradox at the core of autoimmune mechanisms, we must conclude that there is very little of the self's sovereignty, autonomy, or even distinguishability left at all. What we might find instead is an intricate community of beings, clashing as well as cooperating, socially and materially bound to one another. This, then, might be what can be called a politics beyond the (human) self.

Accepting and imaginatively engaging with autoimmunity's paradoxical indeterminacy and uncertainty, as ALOK incites us to do in the epigraph, rather than seeking to minimize or eradicate them altogether, is a pedagogical and therapeutic strategy that can also function as a methodology, perhaps even as an ethos, for queering knowledge production. Acknowledging what we do not know (yet), and perhaps cannot know, about a pandemic, about a virus, about autoimmunity, and finding ways to notice and articulate the foundational ambiguity and variability at the core of life, might function simultaneously as a

radical political and scientific gesture. It is also very necessary queer work, feminist work, anti-racist work. In a similar manner, it can be therapeutic and pedagogical to acknowledge and document the many ways in which embodied, sensory, cognitive, and affective experiences undermine ableist models of normative health, often framed as a standard to which all living bodies must aspire.

Conclusion: The Afterlives of COVID (Auto)immunity

To circle back to the Staying Alive seminar, I have come to realize that the pedagogical and therapeutic transformations that made space for the unknown were also ways to welcome, articulate, and even cultivate ambiguity as a method, as a way of being and thinking together, as a praxis for reading, writing, documenting experiences and events that fall outside dominant epistemic regimes. In the seminar, I interacted with and collaborated with the students to foster an atmosphere of collective disorder(ing) where each participant was welcome to share their own experiences and responses, not as unique or individual, but as part of a collective, contributing to an unwieldy bunch of narratives and conversations. There were, I'm sure, plenty of shortcomings, uneasy conversations, perhaps even triggering or at least uncomfortable. The technical possibilities offered by Zoom, from switching off the camera to typing into the chat box, as well as editing one's name, afforded those who needed it an anonymous, confidential setting close to group therapy. Talking about COVID and AIDS, chronic conditions and disabilities, neurodivergence, mental health issues, and the lingering states between health and illness, convalescence, and debility, allowed us to talk about "differential bodily burdens" (Chen 2021, 22) and social justice in terms of racism, ableism, heterosexism, poverty, and classism. We worked together to dislocate health and illness from the privatized realm of the sovereign self and placed it squarely in the politicized arena of public affairs, where of course it always is.

I believe it is urgent to create more spaces of radical inclusion and experimentation across educational settings and institutions, to discuss difficult matters around the politics of access, the uneven demands of (self)care, and the (always tempting) impossibility to performatively (re)produce the illusion of a sovereign, rational, autonomous subjectivity in our daily lives. Talking openly and reading widely about illness and disability, infection and contagion, prevention and containment, loss and mourning, pathogenic mechanisms and autoimmune responses allowed us as a group to do some of this difficult and nourishing work over the course of sixteen weeks. Our seminar was an exercise in collective immunoplasticity and autoimmune functioning, where we learned to be highly responsive, to care for ourselves and each other, to be adaptable, and to increase our tolerance for ambivalence, failure, antagonism, and uncertainty. This demanded creativity and intellectual curiosity, a capacity to stay present against all odds. We came closer, we withdrew, we asked uncomfortable questions and

shared awkward silences, we turned off our cameras and confessed our exhilarating, at times limiting and at others generative, embodied and affective experiences on a weekly basis. In this article, I have aimed to show how critically engaging with COVID, especially in its long-haul autoimmune clinical manifestations, might engender a radical, transformative pedagogical and therapeutic realignment informed by autoimmunity and autoimmune phenomena that centers on a collective, networked, relational subjectivity rather than insisting on the individualized, sovereign immunity of an autonomous self. Immunity is a messy, collective affair—just like the rest of life.

Notes

[1] ALOK, also known as Alok V. Menon, is a non-binary queer activist, author, and performer.

[2] See Khamsi (2021) for an excellent overview of COVID autoimmune mechanisms, aimed at a general audience.

[3] At German universities, the winter semester's lecture period usually runs between October and February, followed by an examination period. The beginning of the winter semester of 2020–21 was postponed to November 2020 due to the COVID pandemic.

[4] Students enrolled in the course got a pass grade if they completed the two required course assignments, thus earning four credit points within the European Credit Transfer System. If they did not complete the two assignments, they did not receive credit points. The course assignments consisted of critical responses to course-related topics and/or readings, in any medium or genre, from traditional academic essays and creative writing to a podcast, comic, or zine.

[5] For examples of how the neologism "immunoplasticity" has been used across a broad range of research studies on immunity see, for example, Moser, Salzer, and Krause (2011) and Pakulak, Stevens, and Neville (2018).

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