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From Social Isolation to Social Justice: The Neuroscience of Solitary Confinement

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In 2010, a sixteen-year-old boy named Kalief Browder was arrested in New York City for allegedly stealing a backpack. Unable to afford bail, he was sent to Rikers Island where he spent almost two years in solitary confinement. During that time, he became floridly psychotic. Browder maintained his innocence throughout his incarceration, and in 2013 the charges against him were dropped. But the damage was done. Though he completed his GED and started college, he continued to struggle with paranoia and depression, requiring multiple hospitalizations. In June 2015, he died by suicide (1).

Unfortunately, stories like Browder's are appallingly common. Almost 2 million people are behind bars in the United States – of those, more than 20% have not been convicted of any crime (2). Approximately 1 in 5 inmates has spent time in solitary confinement within the past year, including a disproportionately high percentage of Black individuals (above and beyond the already disproportionately high rate of detainment (2,3)). Symptoms of "serious psychological distress" occur in approximately 25% of individuals (a term used to connote severe psychiatric illness) (3). The practice is rampant: how can we understand its biological impact??

200 years ago, Charles Bonnet, a naturalist and philosopher, was studying entomology and botany when something personal caught his attention. Bonnet's grandfather was losing his vision due to bilateral cataracts and began to experience hallucinations of people, animals, and even buildings. Bonnet became the first to describe this phenomenon and, in a stroke of insight, postulated that the hallucinations originated from the visual cortex rather than the eyes themselves (4).

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Bonnet's work echoed earlier discoveries. Ambroise Pare, a "barber" in the 1500s, described the peculiar experience of a man complaining of leg pain – peculiar because the leg had been amputated months prior (a phenomenon later termed "phantom limb pain"(5,6)). Multiple physicians, including the famed neurologist, Oliver Sacks, described musical hallucinations emerging in elderly adults experiencing hearing loss (7). Common to these stories was the idea that losing sensory input could cause perceptual disturbances within the same domain.

Of course, establishing causation requires more than retrospective accounts. Enter Donald Hebb, already famous for his foundational work on synaptic plasticity. Hebb was studying the impact of boredom (!) when he designed what seemed like a simple enough experiment. He recruited college students and placed them into small cubicles. He blocked their vision with goggles, played a low frequency hum to suppress outside sound, and prevented tactile input by placing their arms and legs within tubes. Hebb expected that subjects would remain in this space for weeks. Not a single subject lasted more than seven days. Some experienced intense delusions, others cognitive impairment, some frank hallucinations (8). The effect was so profound that it became part of the CIA's MK Ultra Project and was ultimately incorporated into "enhanced interrogation" practices (9). It was clear that depriving people of sensory and social input could induce psychotic symptoms. But it wasn't clear *how*.

One early insight came from a model known as signal detection theory. The basic idea is that the brain is never silent – even at rest, neurons fire intermittently. A task of the cortex, at any moment, is to determine whether a signal from the periphery reflects a novel stimulus or is just random firing. Critically, in the absence of external input, the brain may lower its threshold for what it interprets as a stimulus and be more likely to misinterpret background noise as "real" (10).

More recently, the *predictive coding* model builds on this using a Bayesian approach: the task of the cortex is not just to receive sensory data, but to interpret them based on prior probabilities. Thus, even if an incoming signal is low (or absent), a person may still have a perceptual experience – a hallucination – if they have strong prior expectations. For example, if subjects are repeatedly presented with a paired light and soft sound, they will be more likely to report hearing the sound when they see a light. This phenomenon has been reliably shown in numerous experiments across sensory modalities (11).

Signal detection and Bayesian theories offer a compelling model for how sensory deprivation could lead to hallucinatory experiences. But what about more complex neurobiological systems, like those responsible for social perception and behavior?

Harry Harlow was studying rhesus macaques at his lab in Madison, Wisconsin when an outbreak of tuberculosis precipitated an unexpected discovery. To prevent the spread of disease, Harlow was forced to separate infant monkeys from their mothers. In the aftermath, he observed a curious behavior: when someone tried to take away an infant's dirty diaper, they would become upset and cling to it. Harlow took this to mean that they craved physical contact. The observation prompted what became one of the most famous experiments in the history of psychology, in which he showed that infant monkeys preferred an inanimate monkey covered in soft cloth to a wire monkey with a bottle of milk (12). The need for

physical contact seemed even greater than the need for food. But this was only a small part of what happened.

Rarely discussed was that *all* of the infant monkeys showed significant long-term harm. They lacked basic social skills, sexual behavior with peers was absent, and when the females did get pregnant, they abused their young (12). While they preferred a soft monkey to a wire monkey, what they *needed* was an actual mother.

But Harlow wasn't done. He became fixated on the impact of early social relationships. In the wire monkey study, the animals had contact with the experimenters and indirect contact with other animals. To more rigorously test his ideas, he created what he called *total social isolation* (13). He raised infant monkeys in a cage surrounded by steel walls, with no outside contact. Then, after up to a year, he released them back into a space with other animals. The results were devastating. The monkeys emerged in a "state of emotional shock." Some refused to eat and died from starvation. The autopsy identified the cause of death as "emotional anorexia" (13). Harlow's work, along with notable others', formed the foundation of what we now call *attachment* (14).

Decades later, another researcher, Ralph Hoffman, became fascinated by peoples' social relationships and their connection to psychosis. Clinical dogma at the time was that social deficits were a *consequence* of schizophrenia. But Hoffman was struck by data that social isolation frequently *preceded* the first experience of a hallucination. In the same way that sensory deprivation could cause auditory and visual disturbances, Hoffman wondered whether the absence of social input could cause hallucinations or delusions with "compelling, aberrant social meaning." He spent years studying this and ultimately brought these ideas together into the Social Deafferentation Hypothesis (15).

Tragically, Hoffman died before he could test the model. Nevertheless, the relationship between loneliness and the incidence of hallucinations has been demonstrated multiple times since, including one recent study that showed a specific increase in hallucinations with social meaning (16). Others have documented additional pathways through which social isolation can cause profound neurobiological harm (17).

This begs the question: if the absence of social stimulation can lead to psychosis, can enhancing social connectivity help to treat psychosis?

Early data are promising. A 2012 meta-analysis of 19 studies found evidence of benefit for social cognitive training in people with schizophrenia (18). A more recent study found improved global function over antipsychotic medication alone when individuals with schizophrenia had their social support bolstered through participation in a community club (19).

Yet somehow, social interventions do not resonate with clinicians or the public as being "real" in the same way as clozapine or ECT. This is despite the fact that psychosocial interventions like family psychoeducation, supportive employment, and supportive housing often show larger effect sizes than medications (20).

These findings force us to reconsider the false dichotomy of biologic vs psychosocial factors. Social isolation *is* a biological insult. And social treatments *are* biological treatments.

It is hard to confront the truth of Kalief Browder's story: that an innocent child was imprisoned, subjected to what many would consider torture, and died soon after. The work of Hebb, Harlow, and Hoffman offer a stark perspective: whether we attribute Browder's death to "emotional anorexia" or "psychosis" or "depression" the cause is the same. We now know that social isolation can cause irreparable biological harm.

And still: hundreds of thousands of Americans are subjected to this each year. Many of these are individuals who have never been convicted of a crime; many are adolescents, in the age of maximum vulnerability to developing psychosis; many are people with severe mental illness who, due to the history of transinstitutionalization, have *de facto* had their care relegated to the carceral system.

Researchers often focus on the importance of translating their work from basic science into clinical settings. Sometimes, the need is more acute: to reach beyond the bedside, to engage in broader public dialogue, and to advocate for social justice.

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