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Race as a Ghost Variable in (White) Opioid Research

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This paper traces the unspoken, implicit white racial logic of the emerging brain disease model of addiction, which is based on seemingly universal, disembodied brains devoid of social or environmental influences, and which led to "context free" neuroscience that made the social hierarchies of addiction and its consequences invisible to, and thus exacerbated by, national policies on opioids. The brain disease model of addiction was selectively deployed among the white middle class population that had long accessed narcotics and treatment for narcotics dependence from biomedical clinics, as opposed to from illegal sources subject to law enforcement. In turn, new treatments for opioid addiction were racially marketed to the same white clientele to which newly patented opioid analgesics were marketed, tapping into a circumscribed but highly lucrative consumer base that has long benefitted from a legally protected, racially segregated safe space for white narcotics consumption. The connecting thread for the contemporary white opioid "crisis," therefore, is white race as a ghost variable in addiction neuroscience and in its pharmaceutical and biotechnological translation.

Introduction

In her classic work of post-modern sociology, Ghostly Matters, Avery Gordon (2008) writes of the dilemma of how to detect and represent power relations that are not transparent, that have been forcibly erased, that exist only in traces. The book's first chapter, "Her shape and his hand" refers to the account that critical legal theorist of race Patricia Williams gives of her great-great-grandmother, a slave, who was shaped by her sexual and political domination by Williams' great-great-grandfather, a slave owner (and ironically a well known jurist). The shape of her great-great-grandmother, which is present in Williams herself but not biographically recorded, and the hand of her great-great-grandfather, which is biographically recorded in American jural history, but whose hand in raping her great great grandmother is erased, are both ghosts. These shapes, or traces, signal the presence of something repressed, denied, but involuntarily re-enacted, to produce what Freud called the uncanny; unsettling because it is familiar and at the same time strange outside of conscious control.

In this paper we attempt to exorcise a ghost that haunts addiction science: why the life expectancy of almost all non-white racial groups in the U.S is rising while the life expectancy of middle-aged whites is falling (Case and Deaton 2015). The answer is largely due to opioid overdose, but to understand how it is that opioids are the primary driver of falling life expectancy of U.S whites is not straightforward. We have had to read the dominant narratives about opioids against the grain. Over a four-year period we have observed eight addiction clinics, attended dozens of addiction science and policy meetings, and interviewed over 200 pharmaceutical executives, addiction scientists, policy makers, advocates, clinicians and patients. It turns out that drugs can be designed with white racial identities, and they can serve as pharmaceutical prostheses to enhance the whiteness of people whose privilege is challenged by stigmatizing diagnoses like addiction and threatened by criminalized responses to the war on drugs aimed primarily at people of color..

Whiteness works as a ghost variable in pharmaceutical narcotic science in two ways: as the assumed norm, whiteness operates through racial coding in which research on "universal human neurobiology" implies a white subject; at the same time, whiteness serves as an exclusive category whose boundaries are actively maintained through distinctions from non-white others. A complex web of medicoscientific, commercial, social, and legal factors gives particular drugs a racialized identity that produces real world effects, like racially patterned decreases in life expectancy. In the case of prescription opioids, this process is not overt as it was the case of Bi-Dil, the heart failure treatment for Blacks that became the first medication to be explicitly marketed to a specific racial group (Kahn 2013). As an unmarked, assumed norm, whiteness operates differently, and as a result opioids were not discursively racialized or read as white, rather, they become white through complex links among pharmaceutical and brain science, drug regulations and marketing. This is a dual prong and circular process where on the one hand whiteness is the assumed standard of universal human biology, yet, once a drug is racialized as white through its identification with universal biology, it is differentially distributed, producing racially distinct effects that reinforce its whiteness. Owing to the privileged place of whiteness as both a default "universal" subject category and as a driver of privileged access, whites are ushered into one system that is geared

toward biomedical individual consumption, whereas non-whites are ushered into another system of criminalization and control.

In narcotic science and regulation, this has historically been done through racialized distinctions between legal, prescribed narcotics and illegal street narcotics. White use of prescription narcotics has been sanctioned through constructs such as "medical need" (Herzberg 2009), while Black and Latino narcotics use has been met with stigma and criminalization. More recently, as growing white suburban and rural heroin use has led to a whitening of the image of heroin in popular media, whiteness has been secured through geographically distinct responses to heroin, using biotechnology to treat addiction and overdose in white communities, and law enforcement to control and punish heroin use in black and Latino communities.

Racialized capital – in the U.S. economy, which was founded on, and continues to be fueled by, consumption and labor that are stratified by race (Robinson 1983, Melamed 2015) - renders whiteness a ghost in this story. Whites are dying as a result of their so-called "privilege" in the consumer market, meaning: their occupation of a decriminalized, protected zone of opioid use, their access to legitimating doctors and prescriptions, and the higher prescription rate for white patients than black patients for pain. Narcotics find legal markets when whitened (such as newly patented opioid painkillers and the private office-based alternative to methadone treatment, buprenorphine, an opioid otherwise known as Suboxone). When illegal drugs are whitened, as occurred when prescription opioid users turned to heroin as supplies of prescription opioids dried up, we see bi-partisan support for alternatives to the War on Drugs, such as diversion from incarceration to treatment, where treatment coincidentally in this case means long term maintenance on the patented legal opioid buprenorphine (commercially known as Suboxone).

The story of how racialized capital led opioids to become white involves the combined effects of four "technologies of whiteness": addiction neuroscience, new biotechnologies, regulatory structures and marketing (Netherland and Hansen 2016). In this paper, we focus on the universalizing scientific contexts of addiction research and biotechnologies—in order to make race-making visible where it is, by design, invisible. The invisibility of race defends white space by socially decontextualizing it, unmarking it, and categorizing it as merely "human," even as manufacturers and lawmakers play on coded white imagery in order to selectively deregulate and market biotechnologies.

Mechanisms of Neuroscientific Whiteness [in Addiction Neuroscience]

Addiction neuroscience and biotechnologies have provided the conceptual underpinnings needed for whiteness to operate within the latest opioid crisis. Addiction neuroscience is connected to whiteness and addiction in three key ways. First, the reliance on brain imagery has created a racially unmarked, biological, and, hence, medicalized image of the 'addict' as implicitly white. Second, addiction neuroscience has largely erased the role of environmental factors or discussions of structural causes of drug use and addiction. Third, it has helped frame our policy responses in ways that have created a less punitive, medicalized space for white drug users, while leaving intact more punitive systems for Black and Latino drug users, creating hierarchies of blame and innocence.

Brain Scans and Images of Addiction: Burying race in the brain

What is striking about brain images of addiction is that they are unmarked by race: they convey a sense of universality and timelessness that, by omitting racial identity, help to expunge racial identity of the addict leaving a white, because racially unmarked, backdrop. Brain scans here operate as the unmarked white norm similar to the way in which the Framingham study of predominantly white participants became the norm for heart disease (Pollock, 2012) and white lung function became the norm for spirometer measurements (Braun, 2014).

A neuroscientific model of addiction as brain disease, which extracts the brain from the racially marked addicted body, thereby helps to unmark (biological) addiction, defining it as a human universal, and therefore white. Indeed, according to Daniels and Shultz (2006), "a defining feature of whiteness, then, is the absence or unmarked invisibility of 'white' as a racial category" (p.94). The neuroscientific reframing of addiction is, therefore, a technique for the racial recoding of (certain types of) addiction and of (some) addicted people, which extracts addiction from the association with Black and Latino people inherent in a social, moral, or criminal framing of addiction.

Addiction as an unmarked white brain disease not only provides a mechanism for extracting addiction from its association with people of color, it also helps render those who have it as blameless. They are victims of a disease that, by definition, erodes their will and ability to make "healthy" choices. In sharp contrast, the legions of Black and Latino drug users arrested, imprisoned, and otherwise punished for using drugs after the declaration of the War on Drugs has led the US to the highest incarceration rate in the world with Black men six times and Latino men three times as likely to be sentenced as white men on drug charges despite similar rates of drug use (Alexander 2010). Meanwhile, those with the brain disease of addiction (implicitly coded as white opioid users) biologically can't control themselves, and thanks to neuroscientific breakthroughs, treatment, rather than punishment, is the response. This contrast of the meteoric rise in incarceration for Black and Latino non-violent drug users, with the simultaneous decriminalization and biomedicalization of government responses to drug use among whites, demonstrates the racial targeting of the War on Drugs and also of the supposedly "universal" brain disease model promoted by addiction neuroscientists and the pharmaceutical industry.

Erasing Structure and the Environment

Neuroscience became central to the US approach to addiction during President H.W. Bush's Decade of the Brain (1990-2000). This was an era in which the National Institute on Drug Abuse (NIDA) was directed to look for neuromolecular causes of addiction and for pharmaceutical treatments for addiction, in anticipation of breakthroughs from the Human Genome Project.

Alan Leshner, then Director of NIDA, lobbied to rebrand addiction as a "Chronic Relapsing Brain Disease." Leshner's ambition was shared by other leading NIDA researchers who coauthored a widely cited article in JAMA in 2000 with the title "Drug Dependence: A Chronic Medical Illness" (McLellan et al 2000). In it, they argued that narcotics addiction was comparable to diabetes, hypertension and asthma in terms of

heritability, treatment adherence, and relapse rates, and as such should be treated in a similar way. Images of so-called addicted brains, which populated scientific studies and graced journal covers (see, for example, fig.1), literally took the subject and his or her trappings of gender, race and class out of the picture, and took the offending organ (the brain) out of the body altogether, symbolically conveying an unmarked universality of addiction physiology. In neuroscience laboratories, addiction was further reduced to molecular action at neuroreceptors, the ultimate disembodiment of addiction. The apparent "universality" of this molecularized model excluded the social or political from addiction. This narrowed the field of vision to the body and biology alone. At the same time, however, research on "universal" human biology implicitly assumed a standard 70 kg white male subject (Epstein 2007).

The scientists involved in the movement to biologize addiction often had a social justice intent: they wanted to destigmatize addiction by demonstrating it to be a legitimate medical condition (a disease of the brain), thereby erasing the social and racial foundations of drug use in order to counteract punitive War on Drugs policies. Many of the neuroscientists interviewed in our study commented on the potential for the brain disease model to reduce the stigma and punitive response to addiction across racial groups. Ironically, because their new model located the cause of addiction in the body, it unmoored addiction from social factors such as education, poverty, income equality and unemployment that contribute to drug use and that are deeply imbricated with race and with social justice.

According to Ruha Benjamin, "in the postracial era, subjugation is hardly ever the explicit objective of science and technology; instead, noble aims such as 'health' and 'safety' serve as a kind of moral prophylaxis for newfangled forms of classification and control" (2016, p.150). The biologization of addiction was a neuroscientific version of color blind ideology (see Alexander 2010) that unconsciously whitened opioids by molecular means, paradoxically further racializing them. As we will show below, during the white opioid crisis, the ascendency of the chronic relapsing brain disease model of addiction and the growth of a medicalized and hence less punitive space for white drug use and treatment provided the discursive and material "out" whites needed to escape the War on Drugs that has been directed at people of color.

Neuroscience whitens further by the relative silence in neuroscientific literature about the role of environmental factors contributing to addiction. Social determinants of health, such as geography, income, education, and housing are largely omitted from the description of research subjects and from the lists of relevant variables in neuroscientific papers. Environmental factors in addiction neuroscience are generally reduced to cues or triggers (e.g., studies demonstrating how brain 'lights up' when a drug user is shown a picture of heroin or cocaine) and, more recently, in discussions of neuroplasticity.

Even when environmental forces are invoked, they are of interest primarily for the biochemical processes they engender. Volkow and Li explain the "neural consequences of environmental risk":

> Low socioeconomic class and poor parental support are two other factors [along with drug availability] that are consistently associated with a propensity to self-administer drugs, and stress might be a common feature of these environmental factors […T]here is evidence that corticotropin-releasing factor (CRF) might play

> a linking role through its effects on the mesocorticolimbic dopamine system and the hypothalamic pituitary-adrenal axis. […] If we understand the neurobiological consequences underlying the adverse environmental factors that increase the risk for drug use and addiction, we will be able to develop interventions to counteract these changes (2005: 1436).

Here, environmental influences are acknowledged but understood only in the context of how the stress they induce affects the dopamine system. Volkow and Li (2005) go on to suggest that the future addiction interventions may include medications that act synergistically with behavioral therapies to mitigate the impact of stress. Absent from their view of addiction are features of neighborhood environment or social roles that might hint at the context of drug use, and therefore the race of drug users and the stressors they face, such as racism, poverty and state violence.

By focusing instead on brain neurochemistry, the neuroscientific model of addiction erases and obscures the role of race and other social differences in ways that privilege whiteness. Social issues, such as the mass incarceration of African Americans under harsh drug laws or the lack of viable economic opportunities beyond the drug trade in Black and Latino neighborhoods, have no place in neuroscientific discourse. As Nancy Campbell notes, "as an ideological code, CRBD [chronic relapsing brain disease] does not focus attention on social differences, including the differential histories and cultural geographies within which their subjects encounter drugs (2010: 101)."

Despite the reductionist tendencies of neuroscience, the notion of brain plasticity – the brain's responsiveness to the environment – suggests a more nuanced concept of addiction that considers the interaction of social and biological factors. Neuroplasticity refers to the brain's capacity to reorganize itself in response to experience or injury (Kolb and Wishaw, 1998). Indeed, the neuroscientific notion of plasticity "appears to challenge biological reductionism by providing room for the environment in brain development and function (Pitts Taylor, 2010, p. 636)."

Some neuroscientific addiction researchers cite an interplay between environmental, psychological, and biological factors. For example, in a review article on addiction neurobiology, Chou and Narasimhan (2005) claim that addiction is influenced by the drug, the user's personality, peers, and the environment. However, they also assert: "exposure to drugs causes plasticity in the neural circuits related to reward and motivation, supporting the idea that addiction is a biological disease. Plasticity results from drug use and drug abuse" (2005, p.1427). In this view, addiction remains a biological predisposition, and the external factor of interest is the availability of drugs to initiate it. In fact, scholars have noted the failure of addiction neuroscience to explain either social factors (Campbell, 2010) or the variations in the prevalence of drug use between populations (Acker, 2010). In general, neuroscientific literature on addiction seems to construe the role of environmental influences quite narrowly, in that discussions of plasticity focus on the role that drugs, rather than social environmental factors, play in reshaping the brain.

Without a broader understanding of plasticity and the role of environment, addiction neuroscience's explanations of behavior are consistent with our cultural focus on the individual and interiority (Choudhory et al, 2009) -- a focus also consistent with a whiteness that looks to individual, rather than social-structural, explanations for drug use

and our responses to it. As part of the larger individualization of illness, solutions for addiction framed as a brain disease lay in helping individuals get well. As Krupars and Ehlers note: "The neoliberal assertion of race-transcendent agency eclipses the ongoing impacts of structural racism, such as social-economic disinvestment in minority neighborhoods and the political neglect of people of color (2013, p.17)".

By rooting the cause of addiction in the individual brain, absent any social context, there is an unspoken assumption that all brains are equally exposed to addiction and equally situated to overcome it. Interest in understanding how neighborhood, family, poverty, or experiences of discrimination and violence impact addiction and one's ability to overcome it are minimized. Nor is there any impetus for seeking to resolve the structural forces at play. This is true, not just for low-income communities of color, but low-income white communities as well. This leaves addiction researchers little capacity to look at systemic issues that might be driving the opioid epidemic and the unprecedented numbers of opioid overdose deaths in both white and non-white communities.

Two Tiers of Policy and Punishment in the Decade of the Brain

The brain disease model of addiction led to racially selective, rather than global, changes in drug policy. By erasing the social context of drug use, and of societal responses to drug use, it built upon a pre-existing two tiered system for managing narcotic use in the U.S.: a clinical tier of legally protected, medicalized use for middle class whites with access to prescribing doctors, and a criminalized tier for low income non-whites who have long been the target of prohibitionist law enforcement. This two tiered narcotics policy began in the early twentieth century when racialized images of narcotics use were used to build support for prohibition of heroin, cocaine and marijuana (Courtwright 1982, Musto 1999) while private physicians continued to prescribe patented narcotics at increasing rates to patients who could afford them, leading, for example, to an epidemic of overdose by barbiturates and other sedatives in the post World War II era among middle class whites that rivaled or exceeded the contemporary rates of opioid overdose, as well as high rates of dependence on benzodiazepines such as Valium and Xanax, otherwise known as "mother's little helper," among suburban housewives in the 1960's-70's (Herzberg 2009). On the basis of this segregated system, the federal government's efforts to bring addiction neuroscience to bear on the pharmaceutical management of addiction was destined to have a differential impact on the white, middle class population that had disproportionate access to clinical care and already was accessing narcotics through clinicians. This left non-whites who were disproportionately subject to law enforcement, and accessing narcotics through non-clinical means, outside of the realm of biomedicalization in drug policy.

It is difficult to overemphasize the role of NIDA in promoting the brain disease model of addiction. Until recently, NIDA claimed to be the funder of 85% of the world's research on addiction (Vrecko 2010a); it is behind much of the scientific and popular discourse about addiction as a brain disease (Courtwright 2010). NIDA's neuroscientific model of addiction dates back to the early 1970's when Jerome Jaffe became the first director of the Special Action Office for Drug Abuse Prevention created by President Nixon. Jaffe was "committed to the view that addiction was rooted in an individual's

biochemistry" (Vrecko 2010: 58) and was responsible for promoting methadone and more generally trying to shift national drug policy towards a pharmacological approach. Despite the potential for methadone to fully biologize addiction treatment, however, methadone was from the beginning associated with Black and Latino urban heroin use and marginalized from mainstream clinical practice (Hansen and Roberts, 2012).

How did the brain disease model of addiction have such ascendency at a national level? In his July 17, 1990 proclamation declaring the 1990's the Decade of the Brain, George H. Bush directed the National Institutes of Health to start an initiative to "to enhance public awareness of the benefits to be derived from brain research" (Bush 1990) and to direct research grant support to basic neuroscience and neuropharmacological research. The Decade of the Brain resulted in a number of high level conferences and publications, shoring up neuroscience's status as the way to understand all manner of human behavior and illness. Neuroscience, then, as now, carries with it a mystique and the promise of demystifying complex human behaviors. A broad cultural fascination with the brain and the biological basis of behavior moved in phase with a massive infusion of federal and private funds into brain imaging studies, and ushering in an era of "cerebral subjecthood" (Pickersgill,M et al, 2011; Vidal, 2009).

The National Institute of Mental Health (NIMH) and NIDA came under pressure to embrace this biological reductionism given they were in competition with other NIH institutes centered on biological diseases, because "NIMH and NIDA place themselves at a political disadvantage to the extent that they publicize that their primary phenomena are psychological." (Miller 2010)

By the late 1990's, NIDA was actively promoting the brain disease model in a way that was designed to bring addiction into mainstream medicine, which continues to dominate its rhetoric and their funding today. In 1997, Alan Leshner, then Director of NIDA, published his landmark article entitled, "Addiction is a Brain Disease, and It Matters," stating "that addiction is tied to changes in brain structure and function is what makes it, fundamentally, a brain disease" (Leshner 1997: 46), and that treatment must compensate for or reverse changes in the brain. By the time the Obama Administration launched The Brain Research through Advancing Innovative Neurotechnologies® (BRAIN) Initiative in 2013, NIH invested $85 million in projects aimed at understanding and learning to manipulate neural circuits that may be linked to addictive behaviors. Bolstered by the decade of the brain, the BRAIN initiative, the cultural ascendency of neuroscience and NIDA's desire to legitimate itself amongst "hard science" peers, NIDA committed itself to shifting understandings of addiction from a behavioral to a neuroscientific problem.

It is this perspective that has guided NIDA since. In 2003, Nora Volkow, a prominent neuroscientist who pioneered the use of PET scans in addiction research, became the Director of NIDA and has vociferously promoted the CRBD model both through her powers at NIDA and through her prominence as a public figure. In a *Huffington Post* article, Volkow (2015) refined her articulation of the brain disease model by noting that this "brain" disease also undermines the capacity for free will:

> "Because of drug use, a person's brain is no longer able to produce something needed for our functioning and that healthy people take for granted, *free will*. … We can do much to reduce the shame and the stigma of drug addiction, once

> medical professionals, and we as a society, understand that addiction is not just 'a disease of the brain,' but one in which the circuits that enable us to exert free will no longer function as they should."

Volkow's project has been to use the CRBD model in an attempt to destigmatize addiction and render those who suffer from addiction blameless in part because they are unable to exercise their own free will. This diverged from the historical legacy of racially motivated prohibitionism and discriminatory narcotic law enforcement in the U.S., which invoked the moral depravity of non-white drug users. This ranged from turn-of-the-century media portrayals of Chinese opium dens, "cocaine crazed negroes," and Mexican "marijuana madness" that led to the passage of heroin and marijuana control acts of the early twentieth century, to racial profiling by the U.S. Narcotics Bureau through the post-war period under Harry Anslinger (Lassiter 2015), and later the Black racial coding of crack cocaine as something that produced urban "superpredators," inscribed into federal law by the 1986 Anti-Drug Abuse Act which mandated minimum sentencing for 1/100th the weight of crack cocaine in comparison to powder cocaine, powder cocaine being more expensive and symbolically coded as an affluent white drug (Alexander 2012).

In short, NIDA fostered the creation of a separate, neurobiological etiological explanation of white drug use by raising up addiction neuroscience as *the* future of addiction research, making "the neuroscientists' laboratory ... an obligatory passage point for the production of truths about addiction" (Vrecko 2010: 58), but only for those groups whose addiction is already managed by doctors in clinics, rather than in criminal justice settings.

The Translation of Addiction Neuroscience into Racialized Biotechnologies

Theories and discourses about a brain disease model alone did not accomplish the (selectively white) medicalization of addiction, however. A concrete medical intervention was needed (, 2010). The ability of addiction neuroscience to prevent, diagnose, or treat addiction was thus far limited. While brain imaging was being widely used in addiction research, it had not yet been employed in clinical practice (e.g. Koob and Simon 2009). So the next step in propagating the neuroscientific/brain model of addiction was to translate neuroscientific research into new pharmaceuticals and biotechnologies for treatment. In keeping with the implicit racialization of addiction neuroscience, these new treatments were also racially coded and deployed for white middle class consumption.

In terms of treatment, as an editorial in *Nature* put it: "our understanding of the neurobiology of disease has progressed substantially… [but] researchers have been less successful in translating this knowledge into effective therapies" (Kosten, 2005: 1413). In 2015, respected drug researchers Wayne Hall and colleagues took the brain disease model and NIDA to task in an article entitled "The brain disease model of addiction: is it supported by the evidence and has it delivered on its promises?" (Hall, Carter & Forlini, 2015). On this backdrop, NIDA has largely looked to pharmaceuticals that can address the physiological symptoms of addiction that are increasingly understood in neurological terms. A medication to treat addiction would place it squarely within the medical model, and scientists, with support from NIDA, have pursued this goal vigorously.

Nestler (2005) highlights the three pharmaceutical approaches being pursued by addiction researchers: 1) medications that block the effects of drugs; 2) medications that "mimic" drugs; and 3) medications that directly influence the processes of addiction. Methadone maintenance was introduced in the 1970's, but in highly regulated clinics with daily observed dosing as President Nixon's first weapon in the War on Drugs, for a population of Black and Latino heroin dependent people that was symbolically linked to urban crime and race riots, a setting in which control of unruly populations was a primary goal rather than molecular, individually tailored, privately consumed products for a chronic brain disease. There have been few psychopharmaceutical treatments for addiction introduced since. Aside from naltrexone and naloxone (which block and reverse the effects of opiates), buprenorphine, an opioid that works by the same mechanism as methadone, is the only medication for opiate dependence to enter into widespread usage in the past 40 years. The medicalization of addiction through this limited number of pharmaceuticals has been pervasive and far from racially neutral.

New Biotechnologies – Developing and Marketing White Drugs

The disease model of addiction calls forth biomedical techniques, rather than either intervening on the social environment to reduce the appeal of drug use, or enhancing law enforcement and criminal justice responses in order to punish and suppress drug use. The 'brain disease' concept of addiction involving genetically and physiologically determined neuroreceptors calls for molecular safeguards and treatments, opening racially segmented marketing opportunities for new pharmaceuticals.

The implicit whiteness of neuroscientific framings of addiction itself played a significant role in the creation of the current white "opioid crisis," starting with newly patented opioid pain relievers in the 1990's. While neuroscientists engaged in the avowed color-blind (but implicitly white) neuro-ideology described above believed themselves to be developing universal biological models of addiction, their work unwittingly supported the more deliberately racial strategies of the pharmaceutical industry. Building on a neuroscientific ideology of technological solutions to previously sociopolitical problems, in 1996, Purdue pharmaceuticals got FDA approval for OxyContin as a "minimally addictive opioid pain reliever" suitable for chronic management of moderate pain such as in lower back injuries. This was based on its patented sustained release capsule technology, which in theory lowered the reward for drug abusers by preventing an initial rush. Note that the social context of addiction is again erased in this neurotechnological solution to addiction risk, an erasure consistent with the universalizing logic of neuroscience - that addiction is molecular process that is the same across time and place – that left no room for regulators to ask what OxyContin users would do with the sustained release capsule in real world conditions.

The designation of "minimally addictive," based on the fiction that OxyContin use on the open market would mirror its use in the three month randomized controlled trial of terminal cancer patients upon which OxyContin's FDA approval was based, enabled Purdue to aggressively pursue an opioid market that had previously been restricted to those with severe acute pain like post surgical or cancer pain. They defined a new, much larger market of patients with moderate, chronic pain like lower back pain, hiring almost 700 drug representatives who canvassed a call list of nearly 100,000

primary care doctors in primarily white suburban and rural areas, leading to a ten-fold increase in prescription of opioids nationally (Van Zee 2009).

Of course, what its model of addiction-proof biotechnology left out was social innovation in drug use. Oxycontin users interested in a rush quickly learned to crush and snort or inject the oxycodone in each capsule, oxycodone being more potent than morphine. Unprecedented prescription opioid overdose was followed by heroin overdose as crushable pills became harder to find: as regulators clamped down on prescribers and manufacturers, instituting Prescription Drug Monitoring Programs in 49 states requiring physicians and pharmacists to check patient data bases to reduce duplicate prescriptions, and as manufacturers patented tamper-resistant formulations of opioids that turned into polymer "gummies" should users try to dissolve and inject them.

A biotechnology developed specifically in response to the white suburban and rural prescription opioid epidemic was buprenorphine itself, branded Suboxone and actively distinguished from its stigmatized pharmacological cousin, methadone, which has been symbolically linked to Black and Latino urban heroin since 1971, when it became the first weapon in President Nixon's War on Drugs. Methadone clinics are DEA regulated and oriented towards patient control, requiring daily observed dosing and frequent urine checks, followed by lowered or increased doses of methadone if illegal drug use is detected (Bourgois 2000). Methadone cllinicals are almost exclusively located low income neighborhoods, far away from other clinical services. Even accounting for white methadone patients, this geographic segregation of methadone clinics fosters another mechanism of racialization: locating methadone clinics in low-income Black and Latino neighborhoods also cements the public identity and visibility of patients who live in the community, while it protects and anonymizes the patients who travel to it, which by definition includes those who are higher income and (given residential racial segregation) disproportionately. As a result, methadone clinics are "of color" to a greater extent than methadone users are.

Not coincidentally, buprenorphine was approved for private physician office-based use at the same time that suburban communities were dealing with an increase in heroin and the abuse of prescription opioids was just beginning to rise. Buprenorphine was not a new scientific discovery that transformed understandings of addiction neuroscience. Rather, it was an old drug, developed in the late 1960's, that had failed to sell as a "minimally addictive" opioid pain reliever because it was only moderately effective for pain and was found to have significant addictive potential. Bupenorphine was re-introduced in the late 1990's as an evolving opioid crisis among "suburban youth" (coded language for white users) became visible. NIDA subsidized the manufacturer, Reckitt-Benckiser Pharmaceuticals, with $23 million for clinical trials of buprenorphine for opioid dependence (SAMHSA 2000) for this growing cohort of white opioid users that the popular press sympathetically referred to as patients with a treatable disease of "drug dependence" rather than addicts or criminals (Netherland and Hansen DATE). Buprenorphine was re-framed in neuroscientific terms as a targeted "smart drug" that, as a partial opioid agonist, caused less euphoria that other opioids and less risk of overdose because it did not depress respiration to the degree that other opioids did. In addition, to reassure regulators with further technological safeguards against its abuse, Reckitt-Benckiser manufactured buprenorphine in combination with naloxone, an opioid antagonist (reversal agent) that causes withdrawal in people who attempt to inject it but

not in those who take it as prescribed, under the tongue, where the naloxone is not absorbed into the bloodstream. Congressional lobbying by addiction treatment advocates and pharmaceutical industry leaders that highlighted these features of buprenorphine persuaded congress to pass the Drug Abuse Treatment Act of 2000, legalizing office-based treatment of opioid dependence using schedule III opioid medications. This move reversed an 80-year prohibition on general physician treatment of narcotic dependence using narcotics dating to the 1914 Harrison Act. In order to make buprenorphine eligible for office based treatment, advocates also lobbied the DEA to re-classify buprenorphine to the federal Controlled Substance Schedule III (meaning it poses a low to moderate risk of creating dependence, along with codeine cough syrup), thereby downgrading it from its classification on Schedule II (meaning it poses a high risk of creating dependence, along with methadone and OxyContin) (Jaffe & O'Keeffe, 2003). Buprenorphine bolstered the chronic, relapsing brain disease model that was increasingly being used as the unifying conceptual framework for addiction science, the basis for a significant proportion of NIDA's appropriations, and a source of scientific legitimacy for the field (Campbell 2007).

In the late 1990's congressional debates that led to to the legalization of monthly Suboxone prescriptions in private doctors offices, as opposed to daily observed dosing in DEA regulated methadone clinics, there was a clear emphasis on a "new kind of drug user," one who was young, suburban and "not hardcore" and, implicitly, white. Alan Leshner, then director of NIDA, testified that buprenorphine, as opposed to methadone, was uniquely appropriate for this new kind of opioid user. As Leshner said, methadone "tends to [be?] concentrated in urban areas, is a poor fit for the suburban spread of narcotic addiction" (Congressional Record 1999:S1092). In a subsequent congressional hearing, then Health & Human Services Director Secretary Donna Shalala noted that buprenorphine, as an alternative to methadone, would serve a new kind of addict, "including citizens who would not normally be associated with the term addiction" (Congressional Record 2000:S9113). Adding the potent term "citizen" further bolstered the respectability and implicit whiteness of the burgeoning new cohort of white, middle class opioid dependent people.

The DEA was reassured that Suboxone, itself a potentially abusable opioid, would not be diverted to street markets because of the requirement that a physician undergo 8 hours of certification training and register with the DEA as a Suboxone prescriber. Since public sector doctors working in Black and Brown inner cities did not have incentives to undergo this training, certified prescribers were largely private practitioners who, in NYC for example, charge $1000 for an initial Suboxone induction visit, and whose largely white, affluent patients often pay out of pocket to keep addiction out of their medical record (Mendoza, Rivera and Hansen 2016). They find prescribers through online advertising and internet referral services on websites with racial coding such as images of clean cut white patients in ironed button-down shirts (fig. 2).

In a race-and –class stratified healthcare system such as that in the U.S., where access to generalist doctors is often limited to those who can pay, patented technologies designed for private office delivery in themselves encode white race and middle class. Ultimately these whitening strategies created an exclusive yet lucrative segment of the market for Suboxone, by 2013 a blockbuster drug at over $1.5 billion a year in sales in

the U.S. in alone, second only OxyContin®, which had reported sales of $3 billion in the same year (Drugs.com 2014).

Conclusion

The flip side of these spectacular sales is the haunting specter of overdose. The whitening capitalization of opioid science and marketing, a process that, as we have shown, depends on racial and socioeconomic inequalities in access to treatment and an individual model of addiction risk and behavior, has obscured social contextual understandings of the causes of, and potential institutional interventions for, overdose epidemics. The brain disease model of addiction, based on seemingly universal, disembodied brains devoid of social or environmental influences, led to "context free" neuroscience that made the social hierarchies of addiction and its consequences invisible to, and thus exacerbated by, the national policies that translated this neuroscience into intervention. The brain disease model thus reinforced hierarchies of blame and punishment as it was selectively deployed to apply to the white middle class population while excluding from its purview low income non-white populations that have long been relegated to illicit sources of narcotics, and thus to law enforcement and criminal justice-based responses to narcotics use. The white, middle-class population was already disproportionately able to access—indeed targeted for distribtion of—biomedical sources of narcotics and pharmaceutical treatment for narcotics dependence. In the era of recently patented new opioids, these hierarchies of blame and punishment take the form of configuring some (white) addicted people as patients who have lost their free will as a complication of seeking prescribed, legitimate treatments for pain, while figuring other (non-white) addicted people as criminals who are seeking the pleasure of an opiate high. In turn, the treatments developed as interventions for opioid addiction were racially marketed to the same white clientele to which newly patented opioid analgesics were selectively and aggressively marketed, tapping into a circumscribed but highly lucrative consumer base that has long benefitted from a legally protected, racially segregated safe space for white narcotics consumption. Yet, in the end, this protected space has not proven to be safe, even for its intended middle class white clientele. The profit-driven, racialized healthcare system through which new opioids were disseminated has proven harmful to whites and non-whites alike, as the very accessibility to opioid pain relievers that it provided whites led to unprecedented overdose rates. And the profit-driven nature of its racialized capital has created barriers to population wide overdose prevention measures that have been successfully implemented in less capitalized and racialized public health campaigns of other countries.

With its overwhelming focus on individual risk and response, the US has developed little public health mechanism to stem the tide of overdose. This is most visible through comparison with France, a country with universal healthcare and government pricing and purchasing of medications. There, buprenorphine was widely promoted and adopted among primary care doctors in poor immigrant communities for prevention of overdose and HIV starting in 1996. The opioid overdose rate in France dropped 80% in the seven years after buprenorphine's approval (Auriacombe et al 2004). Contrast this with the US, where drug overdose rates have tripled in the first ten years after buprenorphine's approval (Rudd 2016). The public health potential of

buprenorphine is limited by its whiteness in our racially segregated and market driven healthcare system, which orphans patients that have patchy insurance coverage and tenuous access to prescribers.

Racially segregated drug policies and lucrative yet lethal prescription narcotic marketing can only be sustained if there is a separate route to categorize and discipline drug use among whites, and that route must appear, at least on its face, to be race neutral. Consistent with Marx's predictions, the technologies that whiten opioids deny the social relations that underlie commodities, transforming human producers of commodities into ghosts, and in this case, also transforming the consumers into ghosts.

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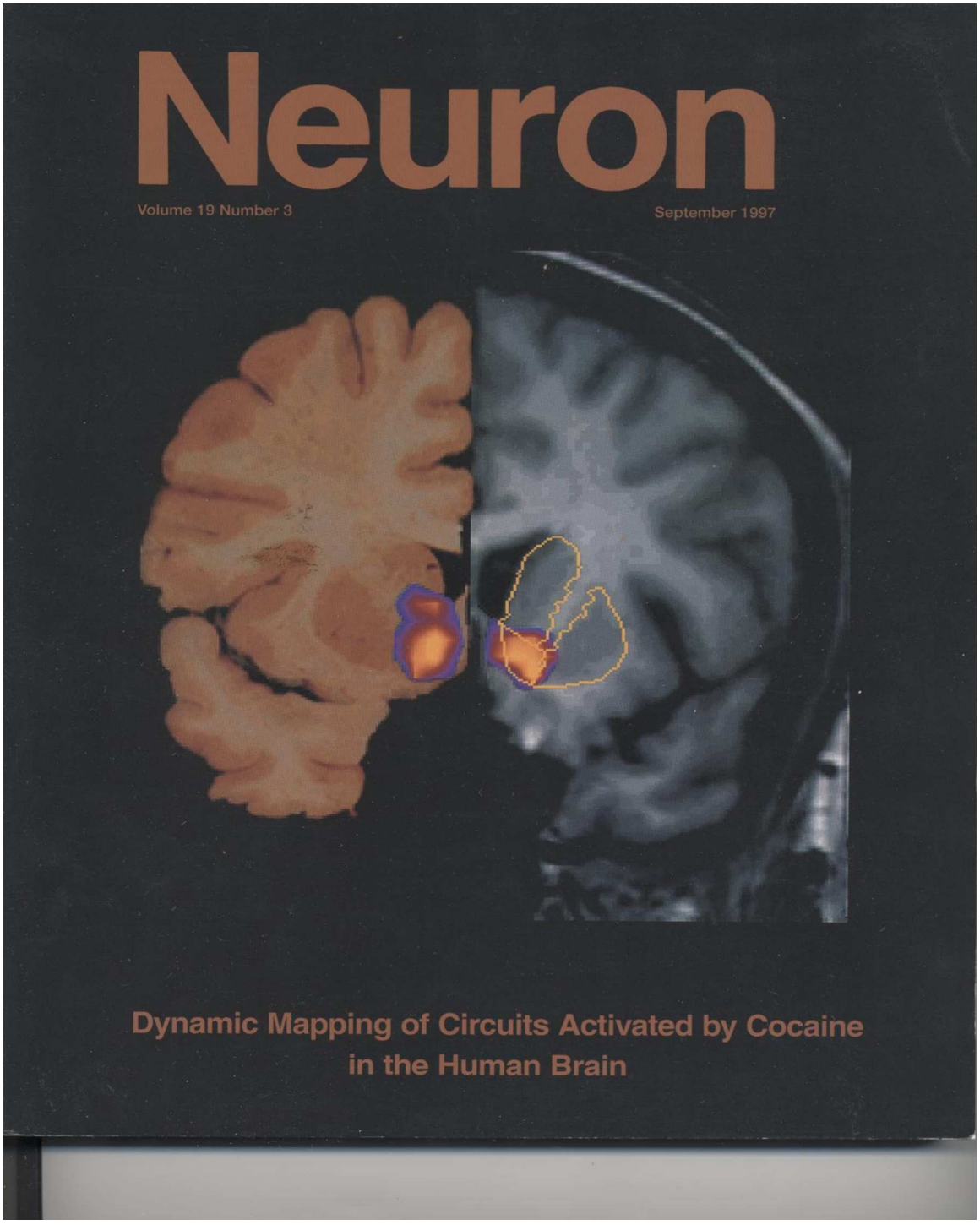
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