Original Article

White opioids: Pharmaceutical race and the war on drugs that wasn't

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Abstract The US 'War on Drugs' has had a profound role in reinforcing racial hierarchies. Although Black Americans are no more likely than Whites to use illicit drugs, they are 6–10 times more likely to be incarcerated for drug offenses. Meanwhile, a very different system for responding to the drug use of Whites has emerged. This article uses the recent history of White opioids – the synthetic opiates such as OxyContin® that gained notoriety starting in the 1990s in connection with epidemic prescription medication abuse among White, suburban and rural Americans and Suboxone® that came on the market as an addiction treatment in the 2000s – to show how American drug policy is racialized, using the lesser known lens of decriminalized White drugs. Examining four 'technologies of whiteness' (neuroscience, pharmaceutical technology, legislative innovation and marketing), we trace a separate system for categorizing and disciplining drug use among Whites. This less examined 'White drug war' has carved out a less punitive, clinical realm for Whites where their drug use is decriminalized, treated primarily as a biomedical disease, and where their whiteness is preserved, leaving intact more punitive systems that govern the drug use of people of color.
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Introduction

The US 'War on Drugs' has had a profound role in reinforcing racial hierarchies. Drug offenses accounted for two-thirds of the rise in the federal inmate population and more than half of the rise in state prisoners between 1985 and 2000, with more than half of young Black men in large cities in the United States currently under the control of the criminal justice system (Alexander, 2010), and middle aged Black men more likely to have been in prison than in college or the military (Rich *et al*, 2011). Although Black Americans are no more likely than Whites to use illicit drugs, they are 6–10 times more likely to be incarcerated for drug offenses (Bigg, 2007; Goode, 2013). Alexander (2010), Wacquant (2009), Hart (2013) and others make the case that the criminal justice system is, in effect, a new state-sponsored racial caste system.

Meanwhile, a very different system for responding to the drug use of Whites has emerged. Beginning in the 1990s, rates of prescription opioid misuse – primarily OxyContin® – began to rise dramatically, particularly among Whites. The press reported a suburban and rural White prescription opioid epidemic (Tough, 2001; Ung, 2001a, b) as prescription drug overdose deaths rose 117 per cent between 1999 and 2012 (CDC, 2014). By 2010, the National Institute on Drug Abuse (NIDA) noted increasing numbers of prescription opioid dependent people turning to heroin as prescription opioids became harder to misuse (Volkow, 2014) because of new prescription monitoring programs and tamper resistant opioid formulations. Thousands of White addicted people found themselves the center of media and political debates about how the country should respond to the latest drug crisis.

The public response to White opioids looked markedly different from the response to illicit drug use in inner city Black and Brown neighborhoods, with policy differentials analogous to the gap between legal penalties for crack as opposed to powder cocaine. This less examined 'White drug war' has carved out a less punitive, clinical realm for Whites where their drug use is decriminalized, treated primarily as a biomedical disease, and where White social privilege is preserved. This 'White drug war' has historical precedents in which predominantly White populations have used social privilege to invoke 'medical need' to secure or maintain access to powerful sedatives or stimulants in the mid to late twentieth century (see Herzberg, 2013). But in the case opioids, addiction treatment itself is being selectively pharmaceuticalized in ways that preserve a protected space for White opioid users, while leaving intact a punitive, carceral system as the appropriate response for Black and Brown drug use.

Whiteness is a sociocultural achievement: it is actively maintained through the shoring up of social boundaries distinguishing White and from not White (Frankenberg, 1993; Allen, 1994; Fine *et al*, 1996; Daniels, 1997; Wray, 2006; Roediger, 2007; Twine and Gallagher, 2008; Hughey, 2010). The seeming inevitability and naturalness of whiteness allows those within the category 'White' to be unmarked, to think of themselves as simply human and without race, while those who fall outside the bounds of whiteness are the racialized Other (Dyer, 1988). Scholars have explored the ways in which whiteness shapes housing (Low, 2009), education (Leonardo, 2009), politics (Feagin, 2012), law (Lopez, 2006), research methods (Zuberi and Bonilla-Silva, 2008) and our understanding of society (Lipsitz, 2006 [1998]; Feagin, 2010). Yet, despite almost two decades of research in the field of whiteness studies, there remains relatively little literature that explores the myriad connections between whiteness and health in the US context (Katz Rothman, 2001; Daniels and Schulz, 2006; Jones, *et al*, 2008; Daniels, 2012; Daniels, 2013) and only a few studies exploring the issue of whiteness and psychoactive substances (Steiner and Argothy, 2001; Murakawa, 2011; Daniels, 2012; Linnemann and Wall, 2013).

In one of the only studies to explicitly look at how drug policy is used to carve out White spaces exempt from punitive more approaches, Lassiter (2015) takes a historical look at the roots of the White opioid crisis of today. Looking back to marijuana policies of the 1970s, he states: "exemptions created for white middle-class participants in the underground marketplace were not merely epiphenomenal but rather constitutive of the expansion of the carceral state (p. 127)". The drug war operates because of a reciprocal relationship between the criminalization of blackness and the decriminalization of whiteness. Lassiter notes that, when the Rockefeller Drug Laws instituted harsh mandatory minimums, White suburban youth found themselves facing significant jail time for low-level marijuana possession. Parents of

White suburban youth banded together to create policy changes that exempted marijuana from the Rockefeller Drug Laws, essentially decriminalizing low-level possession in some jurisdictions. This was possible, in part, because of the racial dynamics and the portrayal of White youth as sympathetic victims of the organized narcotics trade. The relative paucity of studies, such as Lassiter's examining the constitutive role of whiteness in the drug war, is remarkable given the wealth of sociocultural research on pharmaceuticals, addiction and race, which has illuminated myriad ways that the biochemical stratification of people of African and Latin American origin has been represented and materially shaped in sites ranging from genetics labs, medical clinics and neighborhoods, to popular media, courtrooms and policy debates (c.f. Campbell, 2000; Courtwright, 2001; Fullwiley, 2007; Singer, 2008; Bourgois and Schonberg, 2009; Roberts, 2011; Tiger, 2012).

While not focused on addiction, some scholars have traced the ways that medicine has always been racialized and used to justify less punitive responses for Whites than Blacks. For example, Solinger (2013) traces how a racial divide was produced and maintained by the discourses surrounding "illegitimate" childbirth and the medicalization of abortion. While abortion became available and somewhat acceptable for Whites, the welfare system was being used to publicly shame and punish women of color who had children out of wedlock. Other studies trace how race became embedded in medical technologies and rhetoric in ways that become taken for granted and that reinforce notions of racial difference. Pollock (2012), in a study of the racialization of heart disease, notes that whiteness is reinforced by its using the famous Framingham study as the norm. Braun (2014) traces the ways that the spirometer, originally developed in the antebellum South to demonstrate the physiological inferiority of Black slaves, continues to naturalize racial difference in contemporary usage through "race correction" of "normal" reference ranges. These works remind us that racial projects are inherently implicated in medicine and that race is reified in discourses of legitimacy, normativity and technological precision.

White race is encoded into biomedical technologies and practice in particularly covert, implicit rather than explicit ways and in ways that may not be intentional on the part of key actors. Our argument here focuses on racializing consequences of pharmaceutical reason that stem from pre-existing structures of exclusion, inclusion, political pragmatism and industry that over-determine the strategies of scientists, treatment advocates, policymakers and pharmaceutical executives. Opioids in the United States are subjected to a racial biopolitics that constrains available strategies, even to those who oppose social hierarchies and strive to de-racialize drug policy. Efforts to de-racialize any arena of life in the United States, which do not directly address racism, risk reproducing racial hierarchy, because White privilege is typically reproduced by eliminating racial references from seemingly universal policies and practice; in the latter part of the twentieth century the social policy reforms that did the most to intensified racial inequalities in income, employment and incarceration were undertaken under the guise of "color blind ideology" (Alexander, 2010).

This article uses the recent history of White opioids – the synthetic opiates OxyContin® and Suboxone® that gained notoriety starting in the 1990s in connection with epidemic prescription medication abuse among White, suburban and rural Americans – to show how American drug policy is racialized, using the lesser known lens of decriminalized White drugs. Examining four 'technologies of whiteness' (neuroscience, pharmaceutical technology, legislative innovation and marketing), we trace a separate system that categorizes and

disciplines drug use among Whites, while not naming White race explicitly, given that White is the unmarked, assumed norm. Maintaining the boundaries of whiteness under racially segregated drug policy requires constant vigilance and ongoing political work. Our thesis is that these technologies of whiteness are fueled by two biopolitical currents that trade on hidden or misrecognized racial coding: on one hand, a partnership of the pharmaceutical industry with lawmakers who are conscious of the racial symbolism of addiction risk and legitimate use of opioids, and on the other, an unconscious collusion of liberal neuroscientists seeking to decriminalize opioid misuse and democratize treatment using a discourse of addiction as a brain disease, while failing to recognize the (White) racial coding in the universal brain disease framework and its disparate consequences.

Addiction Neuroscience as a Technology of Whiteness

The mass incarceration of people of color for drug offenses is, in part, legitimated by the belief that drug use results from a failure of will or morality. People are responsible for their use and, therefore, must be held accountable or punished. However, at the same time that more punitive War on Drug policies were enforced in Black and Latino city neighborhoods, President Bush I ushered in the Decade of the Brain at the National Institute on Drug Abuse. The richly funded neuroscience program at NIDA in the 1990's provided a scientific rationale for addiction as a clinical disease, focusing on altered brain chemistry as the source of addiction and on neuroactive pharmaceuticals and clinical (rather than law enforcement) interventions as the appropriate response. Simply put, neuroscience provided a scientific rationale for treating addictions – at least some addictions – as disease needing medical intervention rather than as crime requiring punishment. While the race of the addict was excluded from this universalizing biological discourse, the very absence of a language of race indexed White subjects. The scientists involved in this project probably did not intend to exacerbate racial inequalities with their work; on the contrary, many saw their efforts to establish the neurochemical basis for addiction as a way to counteract the social injustice of addiction stigma, and, by extension, racial injustice. Yet, the implicit racial logic of universalist clinical research, which has long held the 70 kg White male as its standard subject (Epstein, 2007), channeled the efforts of even egalitarian neuroscientists toward stratified results.

In the United States, NIDA spent millions of dollars promoting one simple message: 'addiction is a brain disease'. In 1997, Alan Leshner, then Director of NIDA, published a landmark article entitled, "Addiction is a Brain Disease, and It Matters". In the article, Leshner argues that addiction is as much a medical as a social problem and that the field and the public has focused too much attention on the latter:

> That addiction is tied to changes in brain structure and function is what makes it, fundamentally, a brain disease. … Understanding that addiction is, at its core, a consequence of fundamental changes in brain function means that a major goal of treatment must be to either reverse or compensate for those brain changes.
>
> (1997, p. 46)

It is this perspective that has guided NIDA since. It has also permeated the field of addictions research through the 1990s, as demonstrated by a landmark article in the *Journal of the*

American Medical Association, co-authored by four prominent addictions researchers, and entitled "Drug Dependence, A Chronic Medical Illness". The article argued that drug dependence was comparable to asthma, diabetes and hypertension in terms of heritability, causation, pathophysiology and adherence to treatment, and that it should be treated as a chronic medical illness for purposes of treatment approaches, insurance coverage and outcome measures (McLellan *et al*, 2000). In 2003, Nora Volkow, a prominent neuroscientist who pioneered the use of PET scans in addiction research, became the Director of NIDA. Since her appointment, she has vociferously championed the brain disease model in both NIDAs scientific and public education arms. For example, in 2005 NIDA underwrote a widely-cited issue of Nature devoted to addiction neurobiology. NIDA has also produced a series of curricula for elementary and high school students to explain addiction. These include: "Brain Power", "Mind Over Matters", "Heads Up" and "The Brain". NIDAs educational materials closely track the scientific literature. NIDAs power in the field of addiction neuroscience has led one scholar to describe them as largely responsible for making "the neuroscientists' laboratory … an obligatory passage point for the production of truths about addiction" (Vrecko, 2010, p. 58).

Addiction neuroscience is connected to whiteness and addiction is three key ways. First, whiteness and health are implicated in the use of brain imagery. Images, such as PET scans, of the 'addicted brain' are increasingly popular in addiction studies and in reporting on addiction. While images of drug-related brain damage or craving-induced brain activity are compelling and are liberally used in NIDAs educational materials, several scholars have noted that imaging technologies, far from being objective or neutral, are shaped by their social and economic context and involve a series of subjective decisions by researchers (see especially, Beaulieu, 2001; Dumit, 2004; Joyce, 2005). 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). 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. 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.

Neuroscience and whiteness are further connected through the 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 (for example, studies demonstrating how brain 'lights up' when a drug user is shown a picture of heroin or cocaine). Even when environmental forces are acknowledged, 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.
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> (2005, p. 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 their stressors. They offer an individually focused clinical and physiological, rather than a public health, model of addiction.

The third connection between addiction neuroscience and whiteness is what neuroscience frames as the appropriate response to addiction. When addiction is conceptualized as a 'brain disease' involving genetically and physiologically determined neuroreceptors, what is called for is a medication that specifically targets faulty neuroreceptors. This stands in stark contrast to the prevalent response to addiction in the United States as part of the War on Drugs in which incarceration is the ready response. When addiction is framed as a brain disorder, rather than a crime, its cultural work to racially recode prescription opioid addiction as White is unrecognized. This ultimately leads to a bifurcated discourse of White addicts as having a 'brain disease' and needing 'treatment', and of non-White addicts as 'criminals' that require incarceration to protect the public.

It seems, however, that some neuroscientists are unaware of the role their work is playing in reinscribing racialized notions about drug users. Indeed, some addiction neuroscience researchers have said that their political project was to delink addiction from crime. They see themselves as reframing addiction as a brain disease for the precise purpose of destigmatizing and decriminalizing drug use and bringing it under the purview of medicine, rather than the criminal justice system. Dackis and O'Brien, for instance, claim that neuroimaging will:

> substantiate the biological basis of addiction and […] ultimately erode entrenched societal attitudes that prevent addiction from being evaluated, treated, and insured as a medical disorder.
>
> (2005, p. 1431)

Such researchers believe that promoting a neurological basis for addiction will erode the persistent idea that addiction is "a character flaw rather than a bona fide brain disease" (Chou and Narasimhan, 2005, p. 1427), leading to the end of stigma and criminalization. Importantly, both models – the moral/criminal and the medical – root the etiology of addiction at the level of the individual, absenting from consideration structural and environmental factors.

These discussions do not explicitly reference race, but it is notable that the neurobiological, universal, by extension 'White' and therefore less stigmatizing and criminalizing concept of addiction, was aggressively developed during a decade (the 1990s) when middle class, White

heroin use was growing. This change in the demographics of heroin consumption was because of an influx of heroin from newly planted poppy fields in Colombia, whose cartels were competing with established Middle Eastern and Asian heroin distributors by dramatically lowering the price and increasing the purity of the heroin they sold in the United States cities. This attracted middle class users who found they could snort instead of injecting the purer heroin (Hamid *et al*, 1997). In addition, with the 1996 approval of OxyContin® as a "minimally addictive pain reliever" marketed to generalist physicians in rural and suburban areas for their use in moderate pain (Van Zee, 2009), by the late 1990s the specter of the coming White prescription opioid addiction epidemic had been raised.

By focusing on brain neurochemistry, the neuroscientific model of addiction, referred to by many neurophysiology researchers as 'chronic relapsing brain disease', erases and obscures the role of race and other social differences in ways that privilege whiteness. The brain disease model of addiction reduces any discussion about poverty, exposure to drugs, racism and other environmental factors to problems of the brain and its response to 'stress'. 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, p. 101).

As we discuss further below, this neuroscientific racial recoding of prescription opioid addiction was one basis for legislative innovations that created a new, separate track for treating the addiction of White opioid users.

Pharmaceutical Technologies of Whiteness

Opioids have long blurred the line between legitimate medications and drugs of abuse. Morphine went into widespread use during the Civil War in the treatment of injured soldiers, and among middle class White Victorian housewives for a range of problems including menstrual pain. Morphine 'habits' only began to be seen as problematic at the end of the nineteenth century; in 1898 Bayer Corporation introduced heroin as a non-addictive cure for morphine addiction as well as a cough syrup for children. However, changes in medical practice and international drug policy led the media and policymakers to associate opiates with poverty and ethnic minorities, and prohibitionists succeeded in banning opiate maintenance from clinical practice with passage of the 1914 Harrison Act (Musto, 1999; Courtwright, 2001). Methadone was introduced as a synthetic opioid pain reliever in Germany during World War II when morphine supplies from Asia ran short, was re-introduced in the 1960s as a maintenance medication for heroin addiction following successful clinical trials among African Americans in Harlem (Dole and Nyswander, 1966), and was named a primary weapon in President Nixon's War on Drugs in 1971 (White, 1998). Methadone quickly become the focus of tight surveillance and regulation by the DEA, however, based on reports of methadone abuse and diversion, and on methadone's symbolic association with Black and Brown inner city drug use (Hansen and Roberts, 2012). New opioids have therefore undergone a predictable cycle of optimism touted as the holy grail of a non-addictive pain reliever, followed by challenges to their legitimacy as

medications, their association with poverty and ethnic minorities, and stigmatization as addictive drugs.

One strategy for reassuring the government and the public that these medications are used only 'as prescribed' and only get into the hands of 'real' patients is to develop new drug delivery technologies. For example, to justify its widespread use by generalist physicians for the treatment of chronic pain, the makers of OxyContin® claimed that its sustained release formulation would prevent its diversion and abuse. When it became clear that, in fact, OxyContin®, was easy to abuse by crushing and snorting or injecting it, **drug developers at Purdue Pharmaceuticals** began experimenting with new 'tamper-resistant' formulations. Binding oxycodone to polymers, they made OxyContin® more difficult to cut, break, chew, crush, or dissolve and thus the time release mechanism harder to circumvent. Despite research suggesting that this led some users switch from OxyContin® to heroin (Cicero and Surratt, 2012), tamper resistant formulations are clearly intended to promote the perception that OxyContin® is used by 'legitimate' pain patients – those with personal doctors, not those associated with poverty and street trade.

Suboxone®, currently advocated by Federal agencies as the first-line treatment for prescription opioid addiction, is itself a formulation that combines an opioid (buprenorphine) with the opiate antagonist naloxone, in order to deter division and abuse (SAMHSA, 2015). If Suboxone® is crushed and injected, the naloxone effect can precipitate opioid withdrawal, including symptoms such as nausea, sweating, cramps and agitation. If Suboxone® is taken as prescribed (that is, dissolved under the tongue), the naloxone has no effect. This formulation was publicized as making a Suboxone® a relatively safe medication that would not be abused and, as we discuss further below, helped justify its marketing to generalist physicians for the treatment of addiction. Unlike methadone, which had come to be seen as a very abusable drug requiring tight regulation and government oversight, not to mention closely associated with poverty and Black or Latino ethnicity in the American imagination, Suboxone® was seen as a technologically tailored 'smart drug' that could and should be moved into the medical mainstream. In a race- and class-stratified health-care system, such as that in the United States, where doctors are privatized and access to them largely limited to those who can pay, technologies of pharmaceutical delivery in themselves encoded whiteness by assuring the identity and legitimacy of medication users as patients within a medical context.

In fact, science had few interventions to offer for the "bona fide brain disease" of opioid addiction until Suboxone® was made available through a remarkable series of legislative changes. Suboxone® is an opioid partial agonist that works by blocking the effects of other opioids. Buprenorphine, the generic version of Suboxone®, was not a new scientific discovery that transformed understandings of addiction neuroscience. Rather, it was developed in 1966, failed to sell as an opioid analgesic, and was brought back to market 30 years later at a time when White opioid use was on the rise, a medical solution to addiction was being fervently sought, and neuroscientific understandings of addiction were gaining momentum in the scientific and popular press. Suboxone®, as one of the only medications available to treat opioid addiction besides methadone, played a central role in bolstering the claims of addiction neuroscience, and it became the center of discursive and political arguments over the nature of addiction. Campbell and Lovell (2012) trace the political and symbolic importance of Suboxone® to NIDA, which had been fervently searching for a compound that had the properties of an opioid but with technological safeguards against abuse and overdose, to

bolster the brain disease model of addiction and to justify continued government investment in research on pharmacological addiction treatment. While race was not an explicit part of the scientific-political discourse of Suboxone®, Campbell and Lovell note the lengths to which Suboxone's® manufacturer went to distinguish it from methadone and to shed the stigma associated with addiction treatment, as it lobbied for the lower abuse potential scheduling of Suboxone®, and for its positioning in the "treatment space" of generalist doctors' offices, based on its claimed molecular superiority.

Like Suboxone®, OxyContin® fit neatly into the neuroscientific paradigm, laying the groundwork for framing addiction to OxyContin® as a medical disorder appropriately treated with a medical intervention (that is, Suboxone®). As OxyContin® was being introduced to the market, neuroscience was increasingly used to explain how drugs activated the brain's reward centers leading to experiences of craving and withdrawal. These same neuroscientific concepts, played a central role in explaining how OxyContin® – originally purported to have a low potential for addiction – came to be the source of addiction for thousands of Americans.

Oxycodone, which had been used as a pain medication for decades and is the active ingredient in OxyContin®, was known to have a large abuse potential. Oxycodone's addictiveness was understood as resulting from the formulation of the product in ways that made it particularly rewarding from a neurochemical standpoint – it gave users an immediate sense of relief and/or euphoria that was seen as especially reinforcing. According to Purdue, OxyContin® addressed this problem through its sustained released formulation of oxycodone. Consistent with neuroscientific understandings of the brain's reward centers, the claim was the slower absorption of oxycodone would be less rewarding and therefore less addicting. As Seppala explains:

> FDA officials state that when OxyContin was approved, the agency believed that the controlled-release formulation would result in less abuse potential, because when taken properly, the drug would be absorbed slowly, without providing the rapid, intense drug effect sought be abusers.
>
> (2010, p. 180)

The data used to bolster the claim of lower abuse potential have since been criticized for selection bias, since they were derived from cancer patients being treated in tertiary care centers, who can hardly be seen a representative of the chronic pain patients that were the primary market for OxyContin®. These claims were further discredited in a series of lawsuits against Purdue for misrepresentation the addictiveness of OxyContin®.

As it turned out, the sustained release formulation of OxyContin® likely enhanced, rather than diminished, its abuse potential. Davis *et al* (2003) note that because it was a sustained release formulation, OxyContin® contained higher doses of oxycodone than previous preparations, and when crushed and injected, it was extremely rewarding and addictive. In the General Accounting Office Report about OxyContin® abuse released in 2003, investigators concluded:

> While OxyContin's potency and controlled release feature may have made the drug beneficial for relief of moderate-to-severe pain over an extended period of time, the DEA has stated that those attributes of its formulation have also made it an attractive target

> for abuse and diversion. According to recent studies, oxycodone, the active ingredient in OxyContin, is twice as potent as morphine. In addition, OxyContin's controlled-release feature allows a tablet to contain more active ingredient.
>
> (2003, p. 29)

Despite clear evidence of illegal abuse and diversion, the problem is not framed as one of criminal addicts who cannot control their behavior; rather the "attributes of its [OxyContin] formulation" that are at fault. Neurochemistry, not a failure of will or character is the root of the problem, and a neurochemical fix, not prison or a punitive behavioral intervention, is the solution.

In the technological parlance of both OxyContin® and Suboxone®, the problem is identified as not the user but the medication and its impact on brain chemistry. The solution is not to penalize those who misused them, but rather, to develop new technologies and reformulate the medications seal them off from the realm of illicit drugs that are bought and sold on the street and/or injected by 'addicts'. Inherent in the effort to distinguish licit from illicit drugs is an often unspoken racial symbolism of White biology and Back crime, least visible at the molecular level of pharmaceutical development, but progressively more apparent in the State apparatus of drug control and in the corporate apparatus of marketing in popular media.

Legislating and Regulating Whiteness

The focus on pharmaceutical and clinical, rather than punitive, interventions for White prescription opioid addiction necessitated a third technology of whiteness. This was a novel set of legislative and regulatory guidelines: both OxyContin® and Suboxone® have unprecedented monitoring and certification requirements for prescribers. These legislative innovations are notable, not only because they create access to opioids and opioid dependence treatment for Whites that is largely unavailable for Black and Brown opioid users, but also because they shift the focus of law enforcement from drug users to surveillance of physicians and pharmacies.

OxyContin®, the primary driver of the dramatic rise in prescription drug abuse through the 2000s, stimulated many states to pass unique prescription drug monitoring laws, which have been adopted by 49 states and which mandate prescriber participation in 22 states (Haffajee *et al*, 2015), although data sharing across state lines is limited by lack of data sharing agreements and technology (ONDCP, 2013). In 2005, congress passed the National All Schedules Prescription Electronic Reporting (NASPER) Act and authorized US $60 million in spending through grants at the US Department of Health and Human Services to establish state-run prescription drug monitoring programs, although no funding was committed in 2006–2008, limiting its implementation. States with rigorous prescription monitoring have shown a decrease in the number of opioid prescriptions filled, but neighboring states without such laws have shown an increase in filled opioid prescriptions (Manchikanti, 2007).

The emphasis of state and federal agencies, including the DEA, on prescription monitoring has made pharmacies and doctors, rather than patients/prescription abusers, the primary target of law enforcement. The wide variation in regulation by state and the resulting inter-state pattern of traffic of prescription opioids, primarily OxyContin®, has led to state-specific interventions against "rogue pain clinics", their prescribing doctors who often purchase

opioids in bulk and dispense them at a profit, as well as Internet pharmacies operating without proper physician verification. Florida, considered "the nation's epicenter for the illegal distribution of prescription drugs" (Fletcher, 2011), was one of the last states to implement a prescription drug monitoring program. The majority of Florida's pain clinic clients came from out of state or purchase supplies over the Internet, and the number of pain clinics in Florida increased exponentially in the 2000s; in Broward County, for example, the number of pain clinics grew from 4 in 2007 to 142 in 2010. As of 2009, Florida was home to 97 of the top 100 practitioners purchasing bulk Oxycodone in the country; these Florida physicians bought 96.5 per cent of the total units purchased by US practitioners.

In June 2010, the DEA took action against four wholesale distributors who supplied these 'pill mills', leading to a drop in number of oxycodone sales in the Florida from over 8 million units in May 2010 to under 500 000 by October 2010 (under the national average of less than one million units). The state of Florida followed with legislation banning pain clinics from advertising that they sell narcotics and from dispensing more than a 72 hour supply of narcotics at a time, as well as requiring physicians in the clinics to have formal training in pain management and to be free of a history of conviction or revoked registration (Santos, 2012). OxyContin's® manufacturer was implicated in Florida's drug monitoring plans as well: Purdue Pharmaceuticals pledged $2 million in funds to support the development of a prescription drug monitoring program in Florida in 2007, but the state never claimed the funds because of its failure to pass the appropriate legislation (Manchikanti, 2007).

The other innovation to curb the abuse of prescription opioids has been DEAs National Prescription Drug Take-Back programs, which allow individuals to dispose of unused prescription medications. Partnering with local law enforcement agencies, the DEA program collected 2 million pounds (1018 tons) of prescription medications in 4 years (DEA, undated). In 2010, Congress passed the Secure and Responsible Drug Disposal Act of 2010, amending the Controlled Substances Act to allow DEA to develop permanent, ongoing and responsible methods for medication disposal. Before the passage of the Act, there were no legal means for transferring possession of controlled substance medications from prescription holders to other individuals for disposal. The Act specifically cites the rising number of deaths, violent crime and property crime from the abuse of prescription medications as the reason for the new piece of legislation (Secure and Responsible Drug Disposal Act, 2010). Like drug-monitoring programs, this strategy is notable for its non-punitive nature and its focus on upstream causes of use, rather than on penalizing individual users, even though the Act explicitly acknowledges the death and crime associated with the misuse of prescription medications. It strains the imagination to picture a similar program aimed at reducing the death and crime associated with heroin use.

In fact, although by 2004 prescription opioids overtook heroin as the primary opiate of abuse in the United States, arrests of prescription opioid abusers and street-level dealers were far outnumbered by arrests of users and dealers of illicit narcotics. For example, in 2009, the arrest rate per 100 000 was 7.5 for the sale and 15.6 for the possession of manufactured or synthetic drugs, compared with 29.5 for the sale and 72.8 for the possession of heroin or cocaine (U.S. Census Bureau, 2009), even though the prevalence of prescription drug misuse far exceeds that of heroin. Not coincidentally, the "non-medical use" of pain relievers is

almost twice as high among Whites as Blacks (SAMHSA, 2010), while rates of heroin use among Blacks, Latinos and Whites are almost identical (Office of National Drug Control Policy (ONDCP), 2011). Because of the highly racialized war on drugs, stereotypes of heroin users tend to cast them as Black urban dwellers (see Scotti and Kronenberg, 2001; Steiner and Argothy, 2001) and as the appropriate targets for law enforcement.

The language of control used by the DEA and other regulators paints prescription opioid abusers as patients who ultimately deserve protection from unscrupulous prescribers and suppliers. The White market of OxyContin® and related prescription opioids, it was thought, could be controlled through medicalized, sanitized interventions focused on surveying and credentialing pharmacies, clinics and professionals, while leaving the more punitive world of incarceration intact to respond to use of illicit drugs in predominantly African American and Latino communities.

Inconveniently, a series of Federal and state-level crackdowns on physician and pharmacy-run prescription opioid rings were followed by news reports that prescription opioid users were turning to heroin as the street prices of prescription opioids rose. Many of these reports painted a picture of an emerging 'suburban' heroin problem among former OxyContin® abusers: one such story on NBC news, entitled "Painkiller Use Breeds New Face of Heroin Addiction", described a White Chicago police captain who moved to the suburbs in search of a safe environment for his son. His son started using OxyContin® at the age of 14, was pushed to heroin by rising illicit market pill prices, and died of heroin overdose at 19. The reporter sums up the problem with a rare explicit reference to race: "Close to 90 percent of teen heroin addicts are White, data show" (Schwartz, 2012).

The same story then moves to another case, that of a White college student from a wealthy Boston suburb who graduated from pills to heroin. It states: "For a while, O'Connor was able to hide the fact that he was driving to the city on a regular basis to score heroin from dealers on the street" (Schwartz, 2012). This story foreshadows the racial threat of this migration to heroin: young suburbanites who are drawn to the city streets where they mingle with Black and Brown dealers. Such a mingling could preclude therapeutic, rather than punitive, responses for White youth, a threat that the reporter forestalls with a hopeful reference to treatment in his closing line: "I had so many psychologists and therapists. The best ones weren't the smartest ones, they were the ones that cared the most" (Schwartz, 2012).

One under-examined question is the degree to which the new prescription opioid drug monitoring programs not only encouraged White suburban heroin use, but also discouraged overburdened public sector physicians from legitimately prescribing opioid pain relievers to non-White and low income people, possibly reinforcing what has been described as systematic under-treatment of pain among ethnic minority patients (Hausmann *et al*, 2013).

While attempts to control the supply of prescription opioids, like OxyContin®, focused largely on pharmacies and doctors, the response of the thousands of (mostly White) prescription opioid addicts was to seek Suboxone®, a medication that marked a radical policy turn away from both imprisonment and methadone – the primary responses to heroin use. Between 1914 and 1970s, when methadone was first introduced, doctors were not allowed to prescribe any opioids as a treatment for addiction. While methadone marked an important turning point in drug policy as the first opioid-based addiction treatment since the 1914 Harrison Act, it never entered the medical mainstream. To the present day,

methadone is restricted to specialized clinics with strict DEA oversight, directly observed dosing and frequent urine toxicology screens. Methadone clinics are typically concentrated in African American and Latino communities (Hansen and Roberts, 2012; Hansen *et al*, 2013). In 2000, the Drug Addiction Treatment Act (DATA) was passed and allowed any certified physician to prescribe an opioid – Suboxone® – in the privacy of their own offices to treat addiction. This remarkable legislative change marked a clear shift away from the 'war on drugs' policy and rhetoric that had dominated US drug policy for decades. The policy responses seen as appropriate for Black and Brown addicts – methadone and prison – were not seen as a viable option for White addicts. New alternatives were needed, and DATA 2000 provided them.

An analysis of the congressional records surrounding the passage of DATA 2000 shows Congress turning to a medicalized response for addiction, especially for certain kinds of addicts they characterized as young and suburban. The congressional record seldom mentions race explicitly. However, code words for race, such as "urban", "suburban", "new user" and "hard core user" abound. As Davis notes, "when used indexically, code words or phrases are deployed to create racial meaning that generates a sort of pathological profiling of groups without direct reference to race" (2007, p. 251). Within discussions of DATA 2000, there is a clear emphasis on a "new kind of drug user" one that is young, suburban and "not hardcore" and, implicitly, White. For example, we learn from the congressional record that: "Narcotic addiction is spreading from urban to suburban areas. The current system, which tends to concentrate in urban areas, is a poor fit for the suburban spread of narcotic addiction" (Congressional Record, 1999a, p. S1092). Although no scientific evidence supported the claims that Suboxone® was more effective among new opioid users than for 'hardcore' users, this assumption was taken for granted in the congressional record.

A corollary to the notion that young, new opioid users would do better on Suboxone® was the idea that these same addicts should not be exposed to the stigmatized world of methadone – which remained the appropriate form of treatment for 'urban, hard core users'. The congressional committee report on DATA 2000 specifies that a certain 'hard core' addict is appropriate for methadone:

> Methadone treatment is largely reserved for those who have been addicted to relatively high levels of opioids (generally heroin) for a relatively long period of time.
>
> (Congressional Record, 1999, p. 13)

In contrast, Alan Leshner of NIDA testifies at length about how buprenorphine is uniquely appropriate for a new kind of opioid user:

> Narcotic addiction is spreading from urban to suburban areas. The current system, which tends to concentrated in urban areas, is a poor fit for the suburban spread of narcotic addiction … [There is] an increase in the number of younger Americans experimenting with and becoming addicted to heroin. … Treatment for adolescents should be accessible, and graduated to the level of dependence exhibited in the patient. Buprenorphine products will likely be the initial medications for most opioid-dependent adolescents.
>
> (Congressional Record, 1999a, p. S1092)

Then Health & Human Services Director Secretary Shalala notes that the alternative to methadone would serve a new kind of addict:

> It would be available not just to heroin addicts, but to anyone with an opiate problem, including citizens who would not normally be associated with the term addiction.
>
> (Congressional Record, 2000, p. S9113)

This new kind of addict necessitated a new kind if policy response, while those 'not normally associated with the term addiction' could remain within the existing system of publicly funded methadone or prison. The only opposition to DATA 2000 in the entire congressional record was from a group of legislators who noted that this kind of reasoning – Suboxone® as a treatment best suited to new users and available to those with access to a physician – would consign 'hard core' users to the existing and widely recognized as failed system. They summarized their opposition this way:

> The bill may help some … These will be mild to moderately addicted persons with the financial resources to obtain access to a physician or other healthcare provider who will either dispense or prescribe the medication. The bill does not address the need of most heroin addicts; namely, those who are severely addicted or who lack the financial resources to see a doctor.
>
> (Congressional Record, 1999, p. 29)

Presumably, DATA 2000 could have, and perhaps should have, changed the landscape for all opioid users, not just White ones. However, as presaged by the legislators above, DATA 2000 had the effect of creating two tiers of treatment. DATA 2000 kept the methadone system intact and did nothing to alter the draconian drug laws that mandate thousands of people of color to prison, but it did create a new treatment track for those had the resources to take advantage of it. Nor did DATA 2000 move beyond a medical model to envision a response to drug use rooted in public health – one that accounts for the social determinants of health, such as geography, race and class. By retaining focus on the individual level, systemic issues, such as racism, poverty and inequality are essentially erased, while, as pointed out by Smith (2005), racial segregation in American health care persists into the present due to systems of finance and regulation that address health care as a business, rather than social investments.

DATA 2000 had several provisions that restricted the widespread adoption of Suboxone® but did make it available to those with access to health care and resources. Suboxone® manufacturers cooperated with the DEA to require an 8 hour training course and registration number to certify prescribers, essentially limiting the availability of Suboxone® providers to those with a high income clientele who could pay fees that would motivate private practitioners to become certified. In addition to the 8-hour training, physicians had also to apply for a special waiver from the DEA to prescribe Suboxone® – which no other medication requires. DATA 2000 restricted the number of patients for whom a physician could prescribe Suboxone® (originally 30, and in 2007 amended to100), further restricting the number and type of physicians prescribing Suboxone®. In order to access Suboxone® as a patient, one had to know about the medication, have access to a regular health care, and be able to locate a prescribing physician. At $5.30–9.60 per tablet, Suboxone® is considerably more expensive than methadone: the drug cost of methadone averages $8 per month (Silverman, 2012; Toombs and Kral, 2005).

Finally, buprenorphine is only covered by State funded Medicaid in some states (Clark *et al*, 2011). Pharmaceutical regulations and reimbursements, therefore, direct buprenorphine to uniquely affluent, White markets building in an invisible but enduring racial bias (see also, Braun, 2014).

Crafting White Markets

The fourth technology that created White opioids was marketing. Pharmaceutical manufacturers shape markets by selectively targeting consumer groups, cultivating the public image of their drug and its benefits. In the case of both OxyContin® and Suboxone®, the crafting of their markets began well before their release to the public. In fact, the markets to which manufacturers attached each drug were critical to their gaining FDA approval.

OxyContin®'s legendary commercial success as a pain reliever hinged on its designation as a "minimally addictive" opioid analgesic. When OxyContin® was under review at the FDA, Purdue estimated the addictive potential of OxyContin® to be "less than 1 per cent" based on its testing among terminally ill cancer patients for a 3-month period. This low level of reported dependence led the FDA to approve the drug for "moderate" and chronic pain; which opened the door for Purdue to pursue new markets for OxyContin®. Rather than focus on acutely ill hospitalized or debilitated patients – those who were traditionally offered opioids for pain – Purdue used this designation to market OxyContin® among primary care providers with patients who had lower back and other chronic pain. To create this new, larger and therefore lucrative market of patients with moderate, chronic pain, Purdue hired a cadre of 671 drug representatives who canvassed a call list of up to 94 000 physicians, with a focus on primary care physicians, leading to a 10-fold increase in prescription of OxyContin® for moderate, chronic pain (Van Zee, 2009). The disproportionate uptake of OxyContin® by rural and suburban prescribers in majority White states (Maine, Kentucky, West Virginia) is notable in light of the historical hostility of regulatory agencies such as the DEA to the expansion of opioid use. Urban markets would have brought with them race and class imagery of illicit drug use that may have made expanded prescription of OxyContin® for moderate pain a hard sell to regulators.

Purdue's segmented marketing of OxyContin® certainly had an impact on the geography of its use. The earliest reported cases of OxyContin® abuse were in Kentucky, West Virginia, rural Maine, Western Pennsylvania and Eastern Ohio (Tough, 2001). OxyContin® gained the nickname "hillbilly heroin" based on its predominance in these rural White states, but prescription opioids were equally linked to affluent Whites in the suburbs and with celebrities such as conservative talk show host Rush Limbaugh, who went public with his OxyContin® dependence in 2003 (CNN, 2003), and Cynthia McCain, wife of presidential candidate John McCain, whose prescription opioid dependence made the press nine years before (Silverman and Voas, 1994).

As Federal and state regulators tightened prescription monitoring and quotas on OxyContin® production, reporters quoted physicians who pleaded for their chronic pain patients. For example, an article entitled "For those who need painkiller, stigma hurts" stated "Increased vigilance in administering OxyContin may be causing people to suffer needlessly (Ung, 2001a)". As with Limbaugh and McCain, these 'legitimate' pain patients who became addicted to OxyContin® are portrayed sympathetically in the media as innocent victims of

iatrogenic medicine or as people struggling with real or existential pain – terms rarely associated with the drug use of Black or Brown individuals. For example, in an article titled "In the Grip of a Deeper Pain", Newsweek notes: "most people acquire the drugs innocently enough by prescription … The problem with painkillers is they also work on existential pain" (Adler, 2003). In another story titled "In Neighborhoods, Mourning the Lives Lost to a legal drug", both the police and the users note the racial dividing line between heroin and OxyContin®. For example, a teenage OxyContin® user from a predominantly White neighborhood in Philadelphia says, "It's weird, because the kids in my neighborhood think if you are on heroin, you're a junkie. You're no good. You're the filth of the earth. If you do Oxys, it's not that bad" (Ung, 2001b).

Despite these sympathetic portrayals of OxyContin® abusers, public pressure was mounting for a technological intervention. In August of 2010, Purdue Pharmaceuticals introduced its tamper-resistant time-release formulation, imbedding oxycodone into polymers that convert tablets into 'gummies' should users attempt to crush or dissolve them (Cicero and Surratt, 2012). On the heels of patent expiration for OxyContin®, Purdue lobbied the FDA to refuse approval of generic OxyContin® that did not have Purdue's newly patented tamper-resistant technology. Their marketing efforts moved from their original extended release tablets – no longer on patent – to new tamper-resistant, patented formulations that had the potential to shut incoming generics out of the market. By keeping prices high, and preserving the image of OxyContin® as technologically sealed off from illegitimate use, the manufacturer strove to keep OxyContin® symbolically a step ahead of darkening street markets.

Suboxone®, for its part, was developed and congressionally approved as a destigmatized, fully medicalized opioid maintenance treatment for those addicted to White prescription opioids. To cultivate that market, the manufacturer selected its medium carefully with target audiences in mind: the Internet, rather than broadcast television or radio, was their primary marketing venue. Web-based public service announcements by the National Alliance of Advocates of Buprenorphine Treatment, sponsored by the manufacturer, featured White professionals and business owners on Suboxone® maintenance. Mike, for example, a diner owner in Ohio, sits in front of an American flag as he tells us that he coaches his son's baseball team and sings in his church choir, and was addicted to prescription opioids following a work related injury:

> I used to look at people who used and abused drugs as people who were making a choice … this disease devours your total being; I had to get better … being able to visit the doctor like an ordinary patient has made all the difference in the world to my recovery.
>
> (naabt.org, 2014)

This same Website features a link to a Suboxone® prescriber locator service created by the federal Substance Abuse and Mental Health Services Administration; browsers simply enter their zip code to generate a list of physicians with Suboxone® prescribing certification, most of them in private practice. More recently, Reckitt Benckiser, the manufacturer of Suboxone®, selected Mike "the situation" Sorrentino – a White reality t.v. star from Jersey Shore, who became addicted to Oxycontin® and other opioid pain killers following an injury – to act as its spokesperson. These strategies created a selective yet lucrative segment of the market for Suboxone®, making it a blockbuster drug at $1.4 billion in sales in the United States in 2012 alone (CESAR FAX, 2012), among consumers that, according to the last nationally

representative study, were 91 per cent White, over half with at least some college education and 75 per cent of whom were prescription opioid dependent (Stanton *et al*, 2006), with recent studies showing Suboxone® patients in New York City to be concentrated in majority White, upper income neighborhoods (Hansen *et al*, 2013). OxyContin®, for its part, earned its manufacturer over $3 billion in 2009 alone (Eban, 2011).

Discussion

The recent history of White opioids shows that whiteness is a moving target. The whiteness of a given product, and thus, of its consumer base, is continuously under threat of invasion and miscegenation. As OxyContin® began to cross over into inner city drug markets, its manufacturer attempted to preserve its whiteness by introducing tamper resistant/abuse resistant technologies such as resins that prevent injection and that preserve its time-release mechanism. A more remote history indicates that, while many opiates have been introduced to the market as non-addictive pain relievers or even cures for opiate addiction, including heroin in 1898, no opiate has managed to stay a medication and concomitantly no opiate has managed to stay White. What the stories of OxyContin® and Suboxone® reveal is how much political work is required to keep White opiates out of the War on Drugs and maintain them in a White medicalized space. The constant threat of miscegenation and invasion requires marketers, legislators and manufacturers to stay one step ahead of the darkening of the drug; they ultimately fail and have to (re)invent new White opioids.

Another aspect of the racialization of drugs revealed by White opioids is the way that racial ideology works as a crucial element of post-industrial narco-capitalism. The very racial segmentation of markets into licit and illicit, White and Black, clinical and recreational as dictated by the War on Drugs and by the profit imperative of opioid manufacturers helps to drive cycles of demand and sustains a moving target of time-bound patents on new technologies of bioactive molecules and delivery devices. Pharmaceutical manufacturers exploit this temporal cycle in their claim to bring the latest technology to bear to limit the consumption of narcotics by the wrong people for the wrong reasons. The trope of racial invasion and miscegenation upon the province of a White narcotic also builds public political support for segmented marketing and regulation of reformulated drugs and creates pharmaceutical demand among Whites for whom the trope augments the appeal of reformulations as legitimate products for White consumption, while eventually heightening demand among non-Whites for whom such new reformulations are aspirational products, such as among Blacks and Latinos among whom a discourse of unequal treatment and need for access to opioid analgesics for undertreated pain and to buprenorphine for opioid dependence is building.

An international comparison reveals how profoundly the racial political agendas underlying drug control shape population patterns of drug consumption. In France, where buprenorphine was adopted for generalist physician treatment of opiate dependence in 1996, well before the United States, buprenorphine was billed as a public health intervention to stem HIV transmission and opiate overdose deaths among low income, largely immigrant heroin injectors. As a result, buprenorphine was widely adopted among primary care doctors serving

poor communities, who encountered no certification requirements or prescribing restrictions (Lovell, 2006), and the opioid overdose rate in France dropped 80 per cent in the 7 years after buprenorphine's approval (Emmanuelli and Desenclos, 2005).

What this suggests for scholars of race, US drug policy and its 'War on Drugs', is that the fields of medicine, pharmaceuticals and biotechnology must be carefully studied for their contribution to the racialization of drugs through alternative ideologies, technologies, regulation and marketing. Public health may be a third alternate ideology to a medical or punitive frame – one that may be able to encompass important structural issues, such as race, geography and class. However, to date, the discourse surrounding addiction and drug policy remain largely driven by NIDAs commitment to the brain disease model; public health frameworks have not been a large part of the public debate surrounding drug policy. Rather, whether operating within the criminal justice or medical frame, addiction remains a highly individualized problem, making it easy to erase systemic factors and obscure built in biases (cf, Pollock, 2012; Braun, 2014). Since whiteness is a designation of exclusion – signaled by the absence of mention of race itself – these arenas will not lend themselves to an easy reading of their racial intents and effects. However, a racially segregating drug policy can only be sustained if there is a separate route to categorizing and disciplining drug use for Whites, and that route must appear, at least on its face, to be race neutral. The ambiguity of the Greek word pharmakon – it's dual identity as medicine and poison – is exploited in this little known White 'war on drugs' in which unprecedented profits are made in the liminal space between licit and illicit sales; a space that is protected by its symbolic whiteness.

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