ADHS Forum

Mr. ATOD’s Wild Ride: What Do Alcohol, Tobacco, and Other Drugs Have in Common?

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Abstract. All researchers agree that individuals can become intoxicated by and dependent on alcohol, tobacco, and other psychoactive drugs. But they have disagreed over whether, and to what extent, drug pathologies comprise a unitary medical problem. Most critically, does addiction have a biological common denominator? Consensus on this question has shifted back and forth. In the late nineteenth and early twentieth centuries, physicians often studied and treated various drug addictions together, working under the “inebriety” paradigm. By the mid-twentieth century the inebriety paradigm had collapsed. Tobacco and alcohol had split off, both in the medical research community and in western popular culture. This article argues that neuroscientific, genetic, epidemiological, and historical evidence helped to reunify the addiction field in the late twentieth century. A new unifying paradigm emerged, variously called chemical dependency, substance abuse, or simply ATOD—alcohol, tobacco, and other drugs.

When you write global drug history, you quickly discover the limits of your knowledge, and you rely on the work of others. Many of the scholars whose research has guided and inspired me are present this evening. I first want to express my debt to you and my gratitude for the opportunity to address you. I am truly honored.

My title, “Mr. ATOD’s Wild Ride,” is an anagrammatic play on a celebrated episode from Kenneth Grahame’s The Wind in the

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Willows. Mr. Toad, you will recall, has a thing for fast machines. He is, in fact, unable to control himself around them. They “intoxicate . . . his weak brain.” Disguised as a washerwoman, Toad commandeers a motor car and goes hurtling down the road. When its owner and chauffeur imprudently try to stop him, he sends the car veering across the road, smashes into a hedge, and goes flying through the air.

Mr. Toad’s wild ride has inspired a Monty Python movie and an amusement-park ride in my home state of Florida. Toad’s ride has also inspired me by suggesting a metaphor for shifting western and, more particularly, Anglo-American, attitudes toward ATOD: alcohol, tobacco, and other drugs. In the last century those attitudes have, like Toad’s car, veered sharply. They have gone from a tendency to see ATOD and their pathologies as linked phenomena, then to a tendency to see them as separate phenomena, then, in the last thirty years, to see them again as linked. I’ve sketched these shifting attitudes toward ATOD as a pendular timeline (Figure 1). It’s not an exact timeline, for most of the events depicted occurred over many years, rather than during a single year. But it makes the point. Consensus on this question has lurched from one side of history’s road to the other and then back again.

I use the words “tendency” and “consensus.” I don’t think that all experts shared these attitudes at any given point in time, or that the swings of opinion occurred without dissent. What I propose is a sketch of “governing ideas.” Governing ideas are “cognitive bundles” of related concepts that have had “considerable intellectual appeal and at some point succeeded in capturing the attention, imagination, and actions of the broad population” (Gerstein and Harwood 1990, 42). Specialists framed problems in certain ways, and their views percolated into popular consciousness and language. In the 1960s continental drift and plate tectonics triumphed as governing ideas in geology, and before long school children were getting watered-down versions in their earth science lessons. New ideas about the nature and management of compulsive alcohol and other drug use spread in comparable fashion—with, as Sarah
Tracy points out, input from patients and their families along the way (Tracy 2005).

So what were the governing ideas about psychoactive substances? In the late nineteenth century, the key medical concept, at least in Britain and most of North America, was inebriety. Many physi-
cians who treated alcoholics and narcotic addicts held that these conditions were manifestations of a more general mental disorder called “inebriety.” The Keeley Institute, a large treatment franchise, advertised that the “person addicted to liquor or drugs is a victim of a ravishing disease,” which may nevertheless be successfully treated “by the famous Keeley Treatment” (Keeley Institute 1921, emphasis added). The treatment regimen for residential Keeley patients—who might be dependent on alcohol, or narcotics, or both—required abstention from tobacco and even caffeinated beverages, understood as insalubrious habits likely to trigger relapse (White 1998, 55).

The inebriety theory drew on three other popular nineteenth-century medical ideas. These were neurasthenia, or nervous weakness; diathesis, or constitutional predisposition to chronic disease; and degeneration, or transmission of morbid deviations across generations. Thomas Crothers, a representative figure, taught that alcohol and other drugs had pathological effects on the nervous system. The effects varied. Those suffering from neurasthenia, such as exhausted “brain workers,” were particularly susceptible. Also susceptible were those born with a nervous diathesis. Whether the vulnerability was environmental or constitutional—in our terms, whether it was due to stress, genes, or both—alcohol and other drugs left a permanent pathological impression on inebriates’ nerves. The repeated administration of a drug intensified this impression into a “morbid craving,” the heedless, self-destructive pursuit of the substance that seemed the common denominator of all inebriate behavior (Crothers 1902).

Once acquired, the trait of inebriety could be passed on as degenerate constitutional tendencies. The process was often one of sinister transmogrification. The drunkard’s child might be an opium addict, his grandchild an epileptic, his great grandchild a congenital idiot. Though inebriate degeneration might assume many forms, its final end was always ruin. It was crucial, therefore, to keep the young from acquiring inebriate habits, for their sake and for posterity’s. Drugs were literally “germ poisons” that threatened the “race” by
corrupting sperm and ova. Caleb Saleeby, a prominent English eugenicist and opponent of both tobacco and alcohol, went so far as to argue that alcoholics should be prevented from procreating (Armstrong 2003, 27-42; Woiak 1998).

Other temperance advocates concentrated on preventing drug use from spreading *within*, rather than across, generations. They made tobacco a particular target, for tobacco often led to drink and other drugs. The most trenchant of the early “gateway” theorists was Charles Towns, a lay addiction specialist. Except for a few women, Towns wrote, *every* alcoholic and addict he treated had a history of excessive tobacco use. Smoking magnified any personal predisposition toward inebriety, “because the action of tobacco makes it normal . . . to feel the need of stimulation.” Tobacco’s irritating effects could be blunted by alcohol, on which the smoker in turn became dependent. Then came narcotics to allay hangover and other effects of drinking. “Cigarettes, drink, opium is the logical and regular series” (Towns 1915, 153).

Towns set forth his views in *Habits That Handicap* (1915), a slender little book that was perhaps the most prescient work about ATOD linkages to appear in the early twentieth century. Towns was an intuitive sociologist who saw that smoking was socially conducive to other forms of addiction. Boys sought out the back rooms of pool halls and saloons to smoke in secrecy, and there they also learned to gamble and drink. Better-educated men who refrained until they entered college found themselves “out of it” if they didn’t light up. Sociability was tobacco’s most seductive attraction, and its social utility made it that much harder to quit. Worse, tobacco’s use scandalized others and tempted them to follow the same path to intoxication. “The very openness and permissibility of the vice,” Towns decided, made tobacco the worst of the drug habits (Towns 1915, 172).

When Towns wrote those words the first ATOD paradigm was near—actually, just past—its zenith. As John Burnham and Caroline Acker have shown, beliefs about the pathological links among drugs and other vices had yielded practical political conse-
quences in the broad-gauge progressive campaign against the urban “vice constellation.” The same reformers who went after narcotics and alcohol often tried to suppress cigarettes, prostitution, pornography, and gambling. Essentially, they understood vice as a package deal (Burnham 1993; Acker 2002).

Even in the 1910s, however, forces were emerging that would spell the end of the first inebriety movement. In fact, by the late 1930s, the idea of biosocial linkages among alcohol, tobacco, and other drugs had all but disappeared. True, it persisted in otherwise disparate groups that shared an unremitting ideological hostility to all psychoactive drugs. Nazis, Mormons, and Evangelical Protestants come to mind. Total sobriety makes for strange bedfellows. But in both western medicine and in western popular culture, alcohol and tobacco effectively split off from other drugs, to the point that the ordinary understanding of the word “drugs” came to exclude, rather than include, these substances. Anyone who asked North American high school students in the 1940s what “drugs” meant would have been told they had something to do with hospitals or dope fiends. Why did this happen?

The collapse of the unifying inebriety theory is a good place to begin. Briefly, its proponents never delivered the scientific goods. They never showed, at the level of cellular pathology, the common mechanism by which drugs caused lasting neurological damage in susceptible persons, or caused such symptoms as withdrawal and craving. The few specific hypotheses, such as Ernest Bishop’s antitoxin analogy, were falsified. No one ever discovered the mechanism by which alcohol and other drugs damaged germ plasm. The Lysenko episode aside, belief in Lamarckian ideas collapsed. Western researchers abandoned the assumption that acquired traits could be passed on to offspring. The Nazi horrors and abuses such as involuntary sterilization gave eugenics a bad name. Researchers quit generalizing about alcohol as a “race poison,” or generalizing much about races at all.

What midcentury researchers did do, with the support of both Alcoholics Anonymous and the alcoholic beverage industry, was to
focus on alcoholism. Howard Haggard and E. M. Jellinek’s widely read 1942 book, Alcohol Explored, exemplified the new approach. Alcoholism was still a disease, to be sure, but a specific disease manifest in a minority of users who could not control their drinking. Alcohol itself was not inherently toxic. The authors found no evidence that alcohol damaged offspring, either through heredity or teratogenesis. Indeed, for the majority of drinkers, alcohol was merely a harmless “social condiment.” The old inebriety view of alcohol—in Norman Kerr’s words, that it was a “poisonous narcotic beverage”—now seemed passé (Roizen 2004; Haggard and Jellinek 1942, 12, 208; Kerr 1887, 2).

The widespread social use of alcohol by physicians and their spouses could only have made these new views more congenial. The same may be said of tobacco. The now-infamous Camel claim, that doctors smoked Camels more than any other brand, was false. But the deeper premise—that doctors smoked a lot of cigarettes, at least before the first big cancer scare in the 1950s—was true. Personal use tended to blind physicians and researchers to the harms of tobacco and to its links to other patterns of drug use.

They weren’t alone. As millions of ordinary men and women took up cigarettes, the idea that smoking necessarily led to intoxicating drugs faded. Many of the new smokers were patriotic veterans of the Great War, and they had received their cigarettes from reputable organizations like the Red Cross (Tate 1999). Airplane pilots even dropped cigarettes to doughboys in advanced posts. A few years ago, working in the Library of Congress, I found a gouache drawing with the unlikely title “Colonel Sir Valtravers Plantagenet gladly accepts a light, during a slight lull in the barrage, from a private in the Benin Rifles” (Figure 2). I proposed the illustration for Forces of Habit, only to have one of my editors veto it. She said it played on racial stereotypes. It certainly did, but that’s why I thought it worked. It made my point that cigarettes had transcended the conventional social boundaries of race and class. Only the barrier of gender remained, and that gave way in the 1920s and 1930s, thanks to clever advertising and the influence of the movies.
In the late 1930s, 1940s, and 1950s, cigarette smoking among adults became so widespread as to be unremarkable, unexception-

Figure 2. Gouache drawing in the Library of Congress: “Colonel Sir Valtravers Plantagenet gladly accepts a light, during a slight lull in the barrage, from a private in the Benin Rifles.”
able, socially invisible. Smoking paraphernalia spread everywhere. Ash trays sprouted on bistro tables and soda-fountain counter tops, cigarette lighters appeared on car dash boards, and, if you wanted to make a statement, you could even buy your own ring-cigarette holder. Matchbooks, which doubled as a convenient form of advertising, were the most common sort of paraphernalia. Their covers show how culturally Janus-faced smoking had become. Some of them already nod back in the direction of the underworld, the adult world of risqué jokes and taverns and extra-marital sex. But there’s also the shining upperworld of modernity: nationalism, fast transportation, electrified communication, and consumer conveniences. Smoking is up-to-date and linked to two other kinds of licit drugs, alcoholic and caffeinated beverages. Matchcovers even pitched products designed to ameliorate the consequences of smoking, such as cough drops—an aspect of a broad phenomenon that economists call “externalities” and I call “problem profits.”

Both the masses and elites smoked. Elite smoking habits were so well known that cartoonists could use them as shorthand, identifying Winston Churchill by his long cigar, FDR by his cigarette holder, and Douglas MacArthur by his corncob pipe. Such personal use of tobacco had nothing to do with “real” drugs. Indeed, pamphlets at the Lexington Narcotic Hospital told new patients where to buy their smokes. Clouds of smoke hung over AA meetings. By contrast, the Keeley Institute had refused to accept cigarette smokers. The late-nineteenth-century view was that they had slipped into the use of alcohol and narcotics easily, and that, while they smoked cigarettes, they could not abstain from other drugs.

The decoupling of alcohol and tobacco from other drugs was no accident. It was at least partly the result of a deliberate campaign by powerful and fiscally important industries, which spent millions on advertising and public relations to promote and defend their products. These industries had a huge advantage. Ethical pharmaceutical manufacturers couldn’t advertise drugs like narcotics or stimulants, except within relatively narrow professional medical circles. Folk or bohemian drugs like marijuana spread by word of
mouth. Virtually the only advertising they generated was official warnings aimed at discouraging their use. Makers of alcohol and tobacco, however, were free to present their products as innocent and salubrious, and to do so to a mass audience. Guinness famously reduced the message to a slogan: “Guinness is good for you.”

Abetted by the movies, alcohol and tobacco ads played up themes of youth, glamor, athleticism, and conviviality. True, the manufacturers could never fully escape or suppress the evidence of their products’ toxicity and addictive potential. But they could hire public relations experts to counter unwelcome news. Hill and Knowlton, America’s leading public relations firm, worked for both the Licensed Beverages Industries, Inc., and the cigarette manufacturers. Hill and Knowlton managed the Tobacco Industry Research Committee, a public relations operation intended to assuage public anxieties by raising doubts about the cancer link and fostering the impression that the industry was actively investigating any potential health problems. The British industry created a comparable organization. It challenged purely statistical studies and called attention to the industry’s funding of lung-cancer research. The implicit message: if there’s something bad in cigarettes, the scientists in the white coats will find it and take it out, so relax and carry on smoking (Courtwright 2005).

Of course, there were plenty of honest researchers in the mid-twentieth-century, researchers who operated independently of the tobacco and alcoholic beverage industries. But they all faced the problem of information glut. Another, quotidian reason that the first ATOD paradigm unraveled was specialization. To cope with the literature explosion, researchers had to concentrate on one drug, or one aspect of one drug. I suspect everyone here has come up against this problem. When I began my dissertation on opiate addiction, I found an international bibliography, The Pharmacology of the Opium Alkaloids (Krueger, Eddy, and Sumwalt, 1943), that was full of clinical, epidemiological, and pharmacological material. Too full: there were nearly 10,000 entries. I remember thinking,
“They’ve got to be kidding.” Any notion of studying more than one type of drug in one country quickly departed.

I sank my particular posthole in the mid-1970s and dug in it contentedly for some time. Looking back over thirty years, I’d say the biggest surprise has been the rebirth of inebriety. It doesn’t go by that name, or any single name. It’s been called chemical dependency, substance abuse, and ATOD abuse, among other terms. But there’s no mistaking its presence. Bill White, a clinician and historian, said it best in *Slaying the Dragon*. “For most of the 20th century,” White wrote, “no conceptual umbrella existed to help the field understand and intervene in the destructive use of all psychoactive drugs. The most seminal event in the modern era of addiction treatment was the reformulation of terms analogous to the earlier concept of inebriety.” Researchers and clinicians rethought the addiction experience, emphasizing its commonalities and looking for integrated theories and treatment approaches. The same conceptual shift, White argued, was linked to the “culture’s more temperate relationship with alcohol and its growing rejection of tobacco” (White 1998, 287-88).

So attitudes toward ATOD have swerved backed to the other side of the road. The big increases in youthful and polydrug abuse undoubtedly had much to do with this switch. So did the growing western cultural awareness of, and aversion to, health risks of any variety. But I want to highlight three “internalist” factors: discoveries in neuroscience, genetics, and new epidemiological findings.

Since the 1960s, scientists have worked out, in increasingly refined detail, how psychoactive drugs affected the mesolimbic dopamine system. This is the neural reward pathway that originates in the ventral tegmental area at the base of the brain. From this region the neurotransmitter dopamine carries messages forward to the nucleus accumbens, a key pleasure center beneath the frontal cortex. Experiments on animals that have been taught to self-administer drugs have shown a sure-fire method for eliminating addiction: just destroy this system. Of course, there are minor drawbacks, like loss of interest in food. That’s why drug addiction is so stubborn and
insidious. It represents, at least on one level, the chemical hijacking of the brain’s most basic and ancient system of reward and motivation.

There’s more to the story than a single neural pathway. Drug administration also sends messages to the amygdala and hippocampus, which are important for forming memories and cues associated with craving and relapse. It’s also true that drugs work in different ways. Opiates and alcohol stimulate dopamine production directly, while cocaine blocks its reuptake. But the bottom line, to quote a recent literature review, is “different drugs, same ultimate effect” (Nestler and Malenka 2004, 84). That goes for licit and illicit drugs, and sometimes for licit and illicit drug treatment. Naltrexone, an antagonist that blocks opioid receptors and which has been used to treat narcotic addicts, has shown promising results in preventing craving and relapse among alcoholics (Rawson et al. 2000). Though the degree of Naltrexone’s therapeutic benefit is still under investigation, and though it may not help the severest alcoholics (e.g., Krystal et al. 2001), the findings do suggest that at least some of the opioid receptors are involved in both types of addiction. Also intriguing is the fact that PET brain scans of compulsive gamblers shown slot machines look very similar to those of cocaine addicts shown white lines on a mirror. Compulsive gambling and drug addiction have many symptomatic parallels, and there is a good deal of comorbidity: heavy gamblers are more likely to be ATOD users, and vice versa (Petry 2002).

That’s not to say that all forms of addiction can be reduced to a single neural pathway, or that drug use can be understood apart from its social and cultural context. When critics (e.g., Satel 2002) accuse the National Institute on Drug Abuse of being obsessed with the “chronic relapsing brain disease model,” to the exclusion of other approaches, they make a fair point. Even Alan Leshner, the former head of NIDA who did so much to popularize the neurological model, wrote that “addiction is not just a brain disease. It is a brain disease for which the social contexts in which it has both developed and is expressed are critically important” (Leshner 1997,
46). Still, one does not have to be a biological reductionist to grasp the historical significance of this research trend or to understand how it reinforces the idea that psychoactive drugs have more in common than was once thought. The progressives had a point. Vices are linked.

The predisposition toward vice may also have a genetic basis, if not necessarily a Lamarckian one based on our ancestors’ poisonously altered germ plasm or otherwise wickedly acquired characteristics. “Several twin studies,” to quote a recent JAMA review, have shown “significantly higher rates of dependence among twins than among nontwin siblings and higher rates among monozygotic [identical] than dizygotic [nonidentical] twins.” Heritability measures have been estimated at 0.34 for men dependent on heroin, 0.55 for men dependent on alcohol, 0.52 for women dependent on marijuana, and 0.61 for men and women who are regular cigarette smokers (McLellan et al. 2001). The inebriety theorists’ hunch, that individual reactions to alcohol, tobacco, and other drugs vary, depending on innate neurological susceptibility and family history, have apparently been borne out for several different substances. Some people are disposed to find certain kinds of drug use more rewarding, or less toxic, or both. Addictive predispositions may overlap. Studies have shown that those with a genetic predisposition for alcohol dependence are also more likely to have problems with pathological gambling (Petry 2002, 185).

It is possible that the correlations are due, not so much to neurological susceptibility per se, but to personality traits that are also partially inherited. Children, especially male children, who lack self-control and future orientation, who are impulsive, insensitive to others, and less intelligent, are more likely to experiment with drugs. In fact, this same constellation of traits predicts a wide range of illegal behaviors (Gottfredson and Hirschi 1990). Loutish thrill-seekers will end up with more ATOD problems for the same reason they end up with more jail time for smash-and-grab thefts and joyriding in stolen cars. Personality traits increase the odds of forbidden experimentation, and experimentation increases the
odds of (indeed, is a prerequisite for) long-term conditions like addiction.

Having said this, I should add that I’ve long had reservations about the concept of an “addictive personality,” whether defined in terms of neurological susceptibility or broader antisocial behavior. If personality is so important, why were most American opiate users middle-aged, middle-class females in the nineteenth century and younger, lower class-men in the twentieth? Or why were they once mostly white and Asian and, then, within the space of two or three decades in the mid-twentieth century, mostly black and Hispanic? Were the addictive personality traits “jumping genes” that hopped from one group to another? (Courtwright 2001a, 6, 152-55). Obviously, specific social circumstances—what doctors prescribed, which immigrants were moving into drug-infested slums—mattered a great deal. One might even say that it is the historian’s indispensable task to point out what those specific circumstances were, and how they changed over time. That said, the question of inherited vulnerability has clearly reemerged in the addiction research field; it has important implications for etiology, prevention, destigmatization, and treatment; and it has reinforced the idea that a range of compulsive behaviors may have common biological elements.

The third field that has contributed to the rebirth of the ATOD paradigm has been epidemiology. Even allowing for some beneficial cardiovascular effects from moderate wine drinking, statisticians documented, with increasing precision, the elevated risks of accidents when drinking, and the risks of chronic disease for heavy drinkers. The more one drank, the greater the risks—a dose-response finding central to the emergence of the alcohol public-health paradigm in the mid-1970s (Roizen 2004; Room 2004). The smoking data were even more damning. The relentless accumulation of health evidence, and the related economic evidence of heavier health-care costs, undercut the efforts of alcohol and tobacco companies to portray their products as benign non-drugs. It also explains why these same companies have increasingly
sought to exploit markets in developing countries, where government regulations are fewer and consumers tend to be less wary.

In the mid-1970s, Denise Kandel and others put the gateway theory on a much firmer epidemiological basis. They emphasized the risk of advancing from licit to illicit drugs. Not all smokers or drinkers did so, but their odds of progression were far higher than abstainers’—a finding that held up in France, Israel, and Japan as well. As a recent RAND study shows, adolescent smokers were at especially high risk (Kandel 2002; Ellickson 2002). The same study showed early smoking to be an excellent predictor of subsequent social problems. When the politician and novelist Jeffrey Archer was sent to Belmarsh prison following his 2001 perjury conviction, practically the first thing he noticed was the ubiquity of smoking. It took only a few strip searches before it dawned on him that the inmates, especially the repeat offenders, were also heavily involved in the illicit drug culture (Archer 2003).

The process by which young smokers moved on to other drugs involved three types of causes—social, learning, and neurochemical. None was necessary or sufficient, but all increased the likelihood of other drug use. Socially, “enabling factors” played a key role. These were the influences Charles Towns had in mind when he described boys lighting up in the pool hall. Adolescent smokers were more likely to be part of peer groups in which alcohol and other drugs were available. They were more likely to become curious about them, to observe how they were used, and to receive praise if they tried them for themselves. Ditto other problem behavior, such as petty theft or skipping school.

Learning also played a part. Smoking is an acquired skill, something neither intuitive nor pleasant on first trial. By learning to inhale tobacco smoke, individuals acquired the behavior necessary for consuming marijuana and other smokable drugs. Because underage smoking was illegal, adolescent tobacco users also learned to develop masking behaviors that served to hide forbidden drug use.
Then there is the effect of tobacco itself. Smokers have lower levels of monoamine oxidase-B, the enzyme responsible for breaking down dopamine in the brain. They can sustain higher levels of dopamine for longer periods of time, particularly if they continue smoking. More dopamine means more pleasure: tobacco works synergistically with alcohol, cannabis, cocaine, and narcotics to provide a sustained high. Hence alcohol and other drug users tend to smoke more heavily. The reverse is also true. Alcohol, a depressant, mitigates some of the adverse effects of smoking, such as increased heart rate. And alcohol activates nicotine-metabolizing enzymes, which makes it necessary to consume more tobacco to achieve the accustomed effect (Courtwright and Courtwright, forthcoming).

In brief, the neuroscientific and epidemiological studies suggest that drug consumption is synergistic and crosses licit-illicit lines. I found that out for myself when I began, in the opening chapters of *Forces of Habit*, to describe when various drugs had become important commodities. We have all been taught that description is easier than analysis. I found the opposite to be true. The analytic chapters—how medical drugs caught on in popular culture, why drugs were ideal products, how drugs financed empires and exploited colonial labor, why elites finally imposed restrictions—turned out to be much easier to write. The biggest challenge was describing the globalization of drugs, a process I called the “confluence of the world’s psychoactive resources” (Courtwright 2001b).

I thought I’d tackle one drug at a time. I’d describe the spread of alcoholic beverages, then tobacco, then caffeinated beverages—“the big three”—then move on to opiates, cannabis, coca products, and synthetics. Try as I might to keep my drug histories compartmentalized, they kept sloshing over onto one another. “Confluence” really is *le mot juste*. The Chinese, for example, learned to smoke tobacco, then tobacco mixed with opium, then refined opium. When early modern European sailors went on liberty, pipe in one hand and drink in the other, they instructed natives in the art of smoking *and* drinking. They taught that these two novel practices went together. (As, neurochemically, they in fact do.) I discovered that early
modern European coffee houses were seldom simply coffee houses. Patrons could purchase all sorts of psychoactive products, from chocolates to exotic liqueurs. They could also smoke, which was good for business, because, we now know, smokers metabolized caffeine much faster than nonsmokers. I also found that complex, overlapping patterns of consumption had developed in Europe’s many colonies. In 1945, when Ho Chi Minh declared Vietnamese independence, he condemned the French for foisting opium and alcohol on his people. I suppose he didn’t add tobacco to the list because he himself was an inveterate smoker.

At the deepest level of historical connection, one might say at the point where economic substructure meets the reptile brain, the common denominator of globalized nonmedical drug use was the rise of limbic capitalism. By that I mean the reorientation of capitalist enterprise from basic services and durable goods to the more profitable business of providing transient but habitual pleasures, whether drugs or pornography or gambling or even sweet and fatty foods. In global terms limbic capitalism first became important in the age of transoceanic empire building. Were it not for the trade in sugar, tobacco, tea, and opium, there never would have been a British Empire, with all that entailed for modern history. Limbic capitalism has become even more conspicuous and lucrative in our own advanced (if that is the word) consumer societies. Entrepreneurs exploit evolved drives and then provide the goods and services to cope with the damage. Alcohol sells aspirin, cigarettes sell nicotine patches, sugar sells insulin, heroin sells Naltrexone, which costs addicted patients between $100 and $150 a month (Rawson et al. 2000). A recent book, Investing in Vice, concludes that the best, most recession-proof financial strategy is to buy a portfolio of “sin stocks,” taking advantage of their relatively inflexible demand curves (Ahrens 2004). I would “double down” the bet by adding stocks of the companies that clean up after the sin companies. Logically, the demand curves for the two sorts of products are correlated.
I can’t explore all the ramifications of limbic capitalism. But I have tried to show how biological, genetic, epidemiological, and historical evidence has given momentum and credibility to the revitalized ATOD paradigm. That doesn’t guarantee its permanent or universal triumph. In particular, alcohol may resist the trend. The alcoholic beverage industry has money, influence, and an argument—moderate consumption is healthful—that may continue to win privileges for its product. Time will tell. Invite me back thirty years hence, and I’ll tell you whether Mr. ATOD’s car has swerved back to the other side of the road.

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Notes

1. For reasons of time, I omitted the section on genetics during the original talk.

2. The relationship between nicotine and caffeine is complex, but researchers have shown that rats chronically exposed to caffeine self-administer nicotine at higher-than-control levels. Compared to nonsmokers, heavy smokers prefer more heavily caffeinated beverages, e.g., coffee rather than tea. Other drugs shown to increase nicotine consumption in animals include pentobarbital, amphetamine, methadone, and heroin (Courtwright and Courtwright, forthcoming).

References


Some two hundred years ago—with the help from Benjamin Rush, Thomas Trotter, Christoph W. Hufeland and above all from the Moscow physician v. Brühl-Cramer—a strange disease came into being: people who drank too much suffered from drinking too much. The majority of the colleagues shook theirs heads, and, as everybody knows, to this day the concept remains contested—we cannot even reach an agreement on whether it was discovered or invented. Be that as it may, Brühl-Cramer had labelled it Trunksucht, literally: “addiction to drink,” a term he had modelled from the rampant Lesesucht, the “addiction to read” (especially novels like Goethe’s Werther which caused spectacular cases of suicide). Thus, the Trunksucht was framed by a broader idea of pathological excessive behaviour, be it reading, coffee drinking or craving for certain food. For psychological reasons some people became addicted; admittedly, these reasons were mysterious. Trotter and the famous Hufeland, on the other hand, simply regarded alcohol the cause of the craving for alcohol. The latter spoke of an “infection” with hard spirits which “inevitably makes it necessary to drink ever more.” Admittedly, it was a mystery why only a minority of the consumers became addicted. Hufeland promoted Brühl-Cramer’s trailblazing work enthusiastically—not realising the difference between the two approaches. In other words: right from its origins the concept of addiction bore a crucial etiological vagueness, if not a void. The starting point of this disease could be seen either in malfunctions
in the body which make the individual behave, especially consume pathologically, or in certain substances seeping from outside into the body and then causing malfunctions which lead to pathological consumption. In the first case it would appear that there is only one addiction showing itself in countless forms, including behavioural patterns; in the second case it would appear either that there are different addictions caused by different substances, or—obviously more elegantly—that there is a class of substances causing similar sorts of pathological appetites.

In his stimulating essay, David Courtwright shows how in the last century the attitudes towards alcohol, tobacco, and other drugs shifted from a notion of a single unity of psychoactive—and addictive—substances to separate ones, and then back to a single one (now including excessive behaviours, too). Aware of the traps of such a condensed outline, Courtwright underlines that he talks of the “governing ideas” only. Indeed, busy historians could easily find counter-examples—no wonder, considering the etiological vagueness of the addiction concept! Nonetheless, doubtless there were significant shifts in the prevailing notions. Thus, the following two points intend not to reject but to strengthen Courtwright’s argument: the relationship of internal and external factors, and the periodization of the shifts.

To start with a brief remark on the “governing ideas.” Courtwright regards them—like Kuhn’s “paradigms”—as originating in the scientific community. From the experts they spread to the public and become popular knowledge. However, in order to explain the shifting attitudes, external factors—such as the changing prestige of the cigarette or the global economy—also have to be considered. Admittedly, I do not possess the philosopher’s stone to settle the everlasting struggle between externalists and internalists in the history of science. I only suggest making that point clearer. Apparently there were phases in which weltanschauungen and other external factors had a strong impact on research and vice versa—both spheres being intertwined in many different ways.
For the sake of periodization we better take a look at the world outside America.³ That there is a class of substances which alter the state of mind is a wisdom dating back at least to the eighteenth century.⁴ Hufeland coined the term “narcotic poisons” which later became a “governing idea.”⁵ But then, as Courtwright points out, in particular in the 1930s-50s, the notion of “inebriety” (or with fewer moral undertones “narcomania”) was overthrown by a division into licit, rather harmless substances, on the one hand, and illicit “drugs” on the other. Indeed, since the 1912 Hague Convention the latter had been increasingly put under control. At the same time, however, it was precisely the model of the “addictive personality” that gained ground, at least in Germany and other European countries. This model—following the path of Brühl-Cramer—stated that certain individuals suffer from “addictiveness” [Süchtigkeit].⁶ Be it acquired or inherited, this hidden abnormality manifested itself in numerous forms, ranging from “nicotinism” to addictive gambling. Consequently—now following the path of Hufeland—ATOD were placed under one heading: narcotics.⁷ A further consequence was that alcohol prohibition made no sense; it would only increase the demand for more destructive drugs.⁸ Instead, those whose “addictiveness” was acquired were to be cured and those who suffered from the inherited variation were to be prevented from parenting “degenerates” by sterilisation. After World War II the eugenic argument lost ground, and whiskey and cigarettes symbolized the modern lifestyle. But the notion of an entity comprising all “narcotics” survived, until it became the “governing idea” again, among experts and laypersons alike. So for the purpose of the periodization of the attitudes towards psychoactive substances and excessive behaviours I suggest to add to the criterion “are ATOD seen as a unity or not?” the criterion “where is the source of addiction located?” This would allow for a more complex model which reflects the fundamental ambiguities of the mystery of addiction.

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2. In his pioneering “Discovery of Addiction” (JSA 39/1978), H.G. Levine assumed that the AA and the Yale Center were the first to shift the source of addiction from the substance to the body. This is misleading in three respects: firstly, Jellinek’s model should better be seen as a mediatory one: a specific substance comes across a specific body; secondly, this idea of “intolerance” towards alcohol was in Europe already discussed around 1900; finally, and most important, both sources of addiction—body and drug—(135,530),(914,879) have always been taken into account at the same time.

3. The attitudes towards ATOD on both sides of the Atlantic differ(ed) considerably, the distinctive clue being ‘sin.’ The gradual disappearance of Europe from USA’s intellectual screen is an alarming and somewhat irritating tendency (cf. my remarks on Tate in SHAR 16/2002).

4. They formed a subgroup of the stimulants; see Genussmittel: Eine Kulturgeschichte, ed. T. Hengartner and C.M. Merki (Frankfurt 2001).

5. See esp. N. Kerr: Inebriety or Narcomania: Its Etiology, Pathology, Treatment, and Jurisprudence (London 1894). Occasionally excessive behaviours were also counted among the addictions, whereas the addictive qualities of tobacco seem to have been more of a popular wisdom.

6. See esp. E. Gabriel and E. Kratzmann, Die Süchtigkeit (Hamburg 1936; last ed. 1974!).

7. In Germany, e.g., in 1934 the relevant organisations were gathered in the “National Narcotic Control Board” (Reichsarbeitsgemeinschaft für Rauschgiftbekämpfung).

8. The Nazi regime never promoted “total sobriety” (as Courtwright assumes). Both the “classical” disease model and the model of “addictiveness” were in line with the failure of Prohibition: they shared the notion that alcohol was addictive in only few individuals (whereas “drugs” were much more likely to trigger an acquired addiction).
David Courtwright has given us an exceptionally lucid essay explaining how and why the paradigms governing the study of alcohol and other drugs have changed over time. His essay provides compelling evidence and argument as to why historians ought to consider alcohol together with other drugs in their research. He has conveyed to a wider audience of specialists in the social sciences and humanities fields the findings of neuroscience—and linked the medical evidence to the paradigms of drug use in suggestive ways.

As an historian who has attempted to study alcohol control issues and tobacco, both separately and together, I found a great deal of interest in this paper. For instance, when anti-tobacco reformers referred to tobacco users as “addicts,” “slaves,” “fiends,” etc., it turns out that they were reflecting intuitive knowledge that has since been verified by medical science.

Courtwright’s argument prompts me to raise speculation about the way this study—and the work reported in fields other than history—might influence further research on the social history of alcohol and other drugs. Given the evidence on reward paths in the brains of humans as of other animals, the net could be cast more widely still. How ought we to consider such supposedly “innocent” pleasures as coffee, chocolate or sugar? It seems to me that these also follow similar reward paths and have psychoactive effects. The emphasis is upon those drugs which have been shown to have or argued to have had serious social or medical consequences. The debate has thus been determined by the social, the moral and indeed
the economic rather than the neurological. Where is the line on drugs to be redrawn? Beyond alcohol, beyond sugar and chocolate, beyond gambling (which is mentioned by Courtwright as another addictive set of behaviours) and into the area of foodways? One might well ask how determinative the neurological is. Courtwright certainly recognises the importance of social associations and learned behaviour, and argues that these factors are mutually reinforcing with or connected to the neurological. But with the area of impact and comparison so broad, what is the relative importance of the neurological and the social in some of these outlying cases?

One concern that I have as a student of American history but one who has worked outside the United States for his entire career is the representativeness of the paradigms discussed. Indeed, is there a global model for the consideration of these topics, and when did global convergence of research come about and why? Scholars more familiar with European history may have something more to say about this. In my case, drawing on the Australian evidence of tobacco use and anti-tobacco reform, there is a great deal of murkiness and cultural lag. The predominantly American paradigm described here does not fit the discourse on tobacco closely until the 1950s, when the famous British and American studies on tobacco and cancer began to appear. When does the paradigm about drugs become so synchronised internationally? The spread of modern media may explain convergence, or perhaps the global development of modern consumer capitalism and its effects. But for earlier periods, I did not find the inebriety paradigm to be particularly important in Australian discourse. The late nineteenth century did not see such a clear conceptual apparatus applied across tobacco and alcohol studies. And when the paradigm of convergence on drug use was important (though not hegemonic), after 1900, it was not discarded in the 1920s and 1930s. The old “inebriety view of alcohol” that it was a “poisonous narcotic beverage” was still discussed fairly widely in Australia in the 1930s. Anti-tobacco reformers referred to tobacco as a narcotic until the 1950s. So it seems that there may be useful research done in tracing the spread of (and contestation of) the
presented paradigm internationally, even in societies with a good deal of American and British influence, as in the case of Australia.

Another concern is the way that the use of tobacco is treated by many researchers predominantly in terms of cigarettes. Although cigarette smoking grew to be so ubiquitous in popular culture in the twentieth century, so too was tobacco use of other sorts equally ubiquitous in the nineteenth. Research on the effects of tobacco would need to take into account the different ways in which tobacco has been used—as pipe smoking, chewing and snuff, as well as cigar and cigarette smoking. Perhaps a similar diversity is also true of other drugs.

Though Courtwright quotes Nestler and Malenka (2004) that “different drugs, same ultimate effect,” the historical record of comparing tobacco and alcohol’s treatment in Australia suggests that social observers in the nineteenth century were more impressed by the different effects of different drugs. Some drugs calmed and others excited. Tobacco was considered in the nineteenth century to calm, and for most people, including many doctors, was not seen as part of an inebriety paradigm. Alcohol on the other hand excited and was outside of respectable society. Though the Keeley Institute had refused to accept cigarette smokers, not all temperance people took this stance in nineteenth century. Keeley devotees did not equal the temperance movement people, at least in Australia.

Another concern is over the conceptualisation of paradigmatic changes and their implications as socially determined phenomena. Courtwright argues that the picture of drug consumption and its social and neuro-physiological patterns may change in the future. This leaves us in a possibly relativist state, with a pressing need to continue to do empirical research on the social causes and effects of drug use. Though Courtwright highlights “three ‘internalist’ factors, discoveries in neuroscience, genetics, and new epidemiological findings,” in the shift to the new paradigm, one should not neglect what he calls the “growing western culture awareness of, and aversion to, health risks of any variety.” Courtwright’s point does raise the question of the relationship between the paradigm and the
culture—since the research on tobacco is driven increasingly by government and private health objectives—of saving health dollars in an aging population more exposed to cancer and other risks that can be minimised by reducing tobacco use. Thus economics and the welfare state must come into the picture as well as medical and other scientific and indeed social scientific factors. Of particular importance is the role of “healthism” as a part of modern consumer culture and environmentalism among the middle class in producing opposition to tobacco. This aspect should not be neglected in future research. Indeed, interesting parallels could be drawn between environmentalist discourse and anti-tobacco reform.

I agree emphatically with Courtwright on the role of capitalism in the wider spread of drugs and their social connections, through marketing and consumer patterns. Indeed, “the common denominator of globalized nonmedical drug use was the rise of limbic capitalism.” However one would also need to consider anthropological evidence of the different ways in which non-western and pre-modern peoples used psychoactive substances, a point that hardly undermines Courtwright’s insight, but which needs to be noted in comparative perspective for the widest possible study of drug use.

I would agree, therefore, in concluding that it remains the “historians’ indispensable task to point out” the “specific circumstances” concerning who used drugs, and where and how the use of different drugs changed over time. This admirable paper points us in fruitful directions.

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Morality, Society and the Science of Intoxication: A response to David Courtwright’s “Mr. ATOD’s Wild Ride: What Do Alcohol, Tobacco, and Other Drugs Have in Common?”

James Mills

David Courtwright’s “Mr. ATOD’s Wild Ride: What Do Alcohol, Tobacco, and Other Drugs Have in Common?” is the customary mix of wide understanding and grasp of detail of an author who has contributed so much to the field of the social history of alcohol and drugs. It makes a stimulating attempt to grasp broad patterns over two centuries. Courtwright’s account focuses on the scientists and doctors who were prominent in the various stances on intoxicants that developed in the nineteenth and early twentieth centuries and how their thoughts and theories became the “governing ideas” that “succeeded in capturing the attention, imagination, and actions of the broad population.” As with any such attempt to grasp broad patterns, it cannot be comprehensive and omissions might be expected. The account would have been made more complete if it had considered the role of those who generated the “governing ideas” of his conceptual framework who were not “scientists.” By ignoring them he risks privileging “scientists” as the “specialists [who] framed problems in certain ways, and their views percolated into popular consciousness and language.”

I would argue that it was discussions of “addicts” in Asia and Africa that played a large part in developing popular theories or “governing ideas” about “inebriation” in the early nineteenth and twentieth centuries, and that it was rarely doctors or scientists that took a lead role in these discussions. The most obvious demonstra-
tion of this lies in the campaigning of missionaries, moralists and anti-imperialists throughout much of the nineteenth century on the issue of opium trading and consumption in Asia. Many of the elements of the “inebration” argument outlined by Courtwright were presented to the congregations and voters in order to scandalize them into support for the campaigners. Missionaries regularly dealt in images of opium use that evoked “the haunts of vice, and . . . the wretchedness, poverty, disease, and death which follow the indulgence,” where “the smokers are broken down, their power for procreation is also gone” and in which “children are born with the opium ‘yin’ and die often unless their mothers or friends blow the opium smoke down their throats.”

These images of opium addiction were accompanied by similar ideas about Africans and western spirits or, as the Aborigines Protection Society stated in the 1890s in West Africa “poisonous alcohol injurious to health and [encouraging] widespread drunkenness and laziness.” Drink was represented by such groups as a product sold wantonly in Africa by opportunistic western traders encouraged by colonial governments eager to cream off excise revenues. Such visions of opium and alcohol users were not simply peddled in Church and through temperance societies. They became the stock in trade of those in the alliance between moralists and anti-imperialists who generated the political campaigns of the nineteenth and twentieth centuries on alcohol and opium. These focused on the issue of imperialism and intoxicants and they enjoyed repeated success over decades which included forcing the “Restrictive Measures Concerning the Traffic in Spirituous Liquor” contained in Brussels General Act of 1889-90 governing trade with Africa, the Royal Commission on Opium (and the Indian Hemp Drugs Commission) ordered in the House of Commons in the 1890s and the Geneva Opium Conventions of the 1920s that were founded on the pre-WWI agreements in Shanghai and the Hague. Government, politicians and populations were forced to engage with images of addicts supposedly wrecked by the consumption of intoxicants, not by doctors and scientists working in the United
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Kingdom and the United States, but by moralists and campaigners who drew on their experiences of Asia and Africa and who were fired by the politics of anti-imperialism and evangelism. Their ideas seem to have emerged early in the nineteenth century and to have gathered pace as time went on; a clearer understanding of the relationship between these groups and the doctors and scientists of the nineteenth century is necessary, as the ideas of the moralists often seem to coincide with, and sometimes to antedate, those emerging from the clinics and the university medical departments.

If the most significant omission from the account is the role played by the moralists in the story told, the most obvious inconsistency can be found in the approach adopted to the “science” of intoxication of recent years. The strength of the first half of the paper lies in identifying many of the social and political forces which shaped the development of the science of intoxicants. Courtwright argues, for example, that the first inebriety movement was undermined by the increasing unpopularity of eugenics and racial theories of medicine caused by the horrors of Nazi science which drew heavily on both approaches. He then goes on to suggest that the forgiving attitude towards alcohol and tobacco use in the mid-twentieth century was framed by the popularity of products that transcended national, class and gender boundaries and which were also widely used by doctors and scientists themselves.

The piece then outlines “the discoveries in neuroscience, genetics, and new epidemiological findings” that have governed the “rebirth of the ATOD paradigm.” However, when it moves on to these recent epistemological shifts it seems less concerned to trace the social factors that have driven them. Instead, the tone of the argument seems to implicitly accept that this knowledge is “true” and it fails to explore the forces which have once again driven science in this direction. Many factors suggest themselves, among which are the financial commitment to the “war on drugs” of the United States, the consolidation and expansion of pharmaceutical corporations eager to open new markets in “limbic capitalism,” or the morality of the post-AIDS generation of the 1980s which was
forced to reassess its pleasures within the calculus of cost to health. It may well be that rather than being “true,” the discoveries in neuroscience, genetics and epidemiology are in fact as partial a set of ideas as those that the author so ably outlines as its predecessors in the scientific world.

David Courtwright’s intention in writing the think-piece was to stimulate reflection about developments in “governing ideas” about intoxicants over the last two centuries. It has certainly done that for this author for two related reasons. It has been a reminder not to talk up or over-emphasise the role of scientists when writing the history of knowledge about drugs; they were certainly involved in the emergence of paradigms but they were never sole authors of these, and in the story of opium and alcohol in the nineteenth and early twentieth centuries they may even have been junior partners in the creative process. The piece has also been a reminder that the science of drugs and alcohol should never be unyoked from the society around it. Indeed, among the many involved in the development of contemporary drug and alcohol discourses perhaps the historian is unique. It is often she or he alone who can raise a quizzical eyebrow to current “neuroscience, genetics and epidemiology” in the knowledge that so much of what has gone before as authoritative data has, in fact, simply been morality or prejudice dressed up with the ribbons of science.

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Notes
Addiction Science, History, and the ATOD Paradigm: A Reply to Hasso Spode, Ian Tyrrell, and James Mills

David T. Courtwright

Hasso Spode and Ian Tyrrell are right to point out that, in a global perspective, the debate over addiction began earlier and assumed more complex forms than indicated in my sketch. It is true too that ideas about addiction were “culturally lagged”—a useful phrase—and that the question of ATOD unity begged another issue, namely, where addiction was existentially located. So, yes, the situation a century ago was messier than the inebriety paradigm alone would indicate. Hence my circumspection in limiting the discussion to Britain and North America, places where the unified inebriety concept temporarily dominated medical opinion.

“Medical opinion” was not, as James Mills reminds us, solely the domain of physicians and scientists. My own lay example, Charles Towns, was a salesman who overcame initial skepticism from the medical establishment and who became, for a time, a respected figure in the treatment community. In Britain and North America, however, physicians like Norman Kerr or Thomas Crothers did most of the technical, etiological writing on drugs and the nervous system. Ultimately, it was their failure to create a testable empirical basis for the unified inebriety theory, to advance beyond the vague nineteenth-century concepts of neurasthenia, diathesis, and degeneration, that made the concept vulnerable to scientific and corporate counterattack in the mid-twentieth century.

Today the revitalized ATOD paradigm extends well beyond the
Anglo-American world. It has gone global, as has the scientific enterprise itself. On-line journals carry the latest discoveries about neural pathways, receptors, genetic predispositions, drug synergisms, and gateway epidemiology to researchers and public health officials around the world. Tyrrell captured the spirit of my essay when he suggested that historians can apply these same medical-scientific insights to past patterns of drug use, while avoiding biological reductionism by showing the play of cultural, geographic, and social forces.

All three commentators note that I emphasized medical research discoveries in explaining the rise of the new ATOD paradigm. One reason for doing so is that the lack of biological evidence was a key reason why the old inebriety paradigm collapsed. Nineteenth- and early twentieth-century observers saw that addictive behaviors were like one another in important ways, such as compulsive use, withdrawal symptoms, craving, and relapse. Until the 1970s, however, no one could make neurochemical sense of why different substances produced similar intoxicating and addictive effects. That was the essential breakthrough, though external factors like growing concern over health risks or corporate malfeasance prepared the cultural soil onto which the seed of scientific insight fell.

When I wrote the lecture I knew that drug historians preferred the cultural soil to the scientific seed. Another reason for highlighting scientific advances was to bring them to the attention of an influential scholarly audience that seemed to be getting too comfortable in its social-constructionist ways. James Mills counters that “so much of what has gone before as authoritative data has, in fact, simply been morality or prejudice dressed up with the ribbons of science.” Fair enough. But it is also fair to say that we now know more, and in more fine detail, about how various drugs actually affect the brain than we did in the Victorian era. There is progress in science, and that progress can be liberating. For too long drug historians have been like transportation historians writing separate monographs on cars, trucks, and motorcycles without knowing how internal combustion engines worked, or why they burned hydrocarbon
fuels and generated pollution. Suspicion of scientific arrogance and imperialism ought not to prevent anyone from the selective appropriation of research insights, especially those that illuminate the common or synergistic features of drug action. My subversive advice: break history’s last taboo. Take a hit of neuroscience. Just don’t get addicted.

Tyrrell asks where the line on drugs should be drawn. My rule of thumb is to include any psychoactive substance that stimulates the mesolimbic dopamine system, the primitive neural substrate of pleasure and motivation. Thus cocaine and caffeine are both drugs, though one is good deal stronger than the other. Sugar is a special case. I don’t consider sugar a drug per se, but it has often been made into a drug (alcohol) or used to sweeten drug preparations such as chewing tobacco or tea. A spoonful of sugar makes the alkaloid go down.

If drug history evolves toward the broader study of compulsive behaviors, such as gambling or even certain types of eating, I would be neither surprised nor perturbed. Cheese may not be “morphine on a cracker,” as one activist has claimed, but we should remain open-minded about where research on the brain’s pleasure centers will lead. Social history is about making connections. Medical science keeps suggesting new connections. Is it, for example, just a cultural coincidence that games of chance have long flourished in taverns, or that Las Vegas runs on booze, or that the Koran prohibits both alcohol and gambling? I don’t know the answer. I do know that posing such questions will enrich our field, and align us with researchers who are expanding the boundaries of other disciplines.

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