Drug use, drug abuse and heterogeneity

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ABSTRACT

Concepts and criteria for drug abuse have significant impacts on research and the development of interventions. The empirically supported differentiation of drug use and drug abuse has led to important gains in understanding the aetiology (etiology) of drug abuse. The need for further distinctions that reflect the heterogeneity of drug abuse is explored. The need for more sophisticated models of drug abuse is discussed.

Keywords: drug use (drug abuse); drug dependence; epidemiology; etiology; heterogeneity; developmental psychopathology.

Drug use and drug abuse

At one point, every drug abuser was a non-user, an abstainer who initiated drug use, later became an escalating user and eventually became an abuser. Every drug abuser follows this progression. While there is considerable variation in drug abuse (the use of different drugs, the quantity and frequency of use etc.), there are also critical commonalities. Drug abuse intrinsically involves the compulsive use of illicit psychoactive substances despite significant potential or already manifest harm.

This commonality suggests to some that drug abuse and drug abusers are fundamentally homogeneous in terms of the primary inherent determining factors. While acknowledging the variable patterns of drug abuse behaviour, this perspective assumes that drug abuse by different individuals is essentially alike in terms of the critical characteristics. These might be any of a number of possible factors, for example, a common underlying neurobiological process, comparable environmental conditions or equivalent behavioural conditioning. Taking this a step further, some believe that there is one basic pathway or trajectory leading to drug abuse and one set of predisposing or risk factors for vulnerability to drug use as well as abuse.

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However, drug abuse is not just a lot of drug use. Obviously, drug use and drug abuse are not independent discrete phenomena. Nevertheless, despite the unquestionable relation of use to abuse, research shows that vulnerability to use seems to be largely distinct from vulnerability to abuse. Further, the quantitative and qualitative differences between drug use and drug abuse are so critical that for heuristic and research purposes, they are in many ways fundamentally distinct phenomena despite their quantitative and behavioural association. This might be thought of as the contrast between a problem behaviour and a psychopathological condition.*

During the late 1960s and 1970s, there was a dramatic growth of illegal drug use in the United States of America and in many other countries. In response, there was a major increase in research attempting to describe and explain both drug abuse in general and the new “drug culture” phenomenon, which involved the use of drugs by population subgroups not previously associated with illegal drugs. Whereas the majority of early investigations had typically studied addicts in treatment and other heavily drug-dependent individuals, the subjects of the new explosion of research were often students in their second year of college (aged 18-19). A common research design of the time tested single factor models of drug abuse by attempting to find a correlation between assessments of a trait or characteristic and a minimal measure of drug involvement, both assessed at a single point in time. Most of the subjects in those studies had relatively limited experience with illegal drug use, which resulted in relatively little variability on the measure of drug experience. Perhaps as a means of achieving variability, the drug abuse measures usually categorized the subjects either as abstainers and experimenters who had used an illegal drug only a few times or as abusers, a category including most other degrees and forms of drug involvement. The published investigations often reported that the researchers’ theory of drug abuse had been supported by research because a correlation had been found between the trait being studied and the drug abuse that it “caused”. Not surprisingly, the studies contributed little to the understanding of drug abuse. Fortunately, some researchers used more sophisticated longitudinal research designs and took a more sophisticated approach to measuring drug involvement.

In the early 1990s, under the co-sponsorship of the National Institute on Drug Abuse (National Institutes of Health) and the American Psychological

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*Use of an illegal drug automatically qualifies as "abuse" in some nomenclatures because it involves criminal activity. This is in contrast to categorizations of alcohol use. Alcohol is legally available although there are restrictions such as age limitations or prohibitions on use while driving. Alcohol "abuse" usually refers to pathological and/or problematic use of alcohol. For the purposes of this article, the term "drug use" is used analogously to the term "alcohol use". "Drug use" refers to experimentation or low-frequency, typically irregular use of illicit drugs that has not (or at least not yet) resulted in significant negative consequences. "Drug abuse" refers to regular and/or compulsive use of illicit drugs. While such a distinction may seem artificial to some, it serves to distinguish people on the basis of whether their illicit drug use has or has not become a significant feature of their lifestyle and whether it is likely to have a psychopathological character. This is not intended to discount the significance of engaging in an illegal behaviour as opposed to one that is legal nor is it intended to minimize the potential danger of even a single use of illicit drugs, particularly under certain circumstances. The intention is to focus on the characteristics and differences underlying the behaviours of use and abuse and to facilitate the discussion of ideas.
Association, drug abuse researchers conducting major longitudinal studies of adolescents were asked to conduct special analyses to explore vulnerability to drug abuse, focusing specifically on those factors and patterns associated with the transition from drug use to abuse. More sophisticated differentiations of use and abuse were encouraged. Despite the variations in the studies, their designs and their subjects, the research projects consensually found that the predisposing or risk factors for drug use were different than those for drug abuse. In addition, the sets of risk factors identified by the diverse studies followed a single basic pattern [1]. Research in the past decade has further corroborated and extended those findings.

The consensus that emerges from the research is that drug use (including initiation and lower levels of involvement) appears to be more a result of social, peer and environmental factors, while drug abuse (including higher levels of drug involvement, drug dependence and drug use disorders) appears to be more a function of biological psychological and psychiatric factors. The risk factors for drug use do not necessarily predict the transition to drug abuse, addiction or disorder. This categorical differentiation of drug use from drug abuse is not just semantic; it highlights different paths and different phenomena. Risk factors for drug use and abuse include the following:

**Risk factors for initiation/low involvement**

- Some “problem behaviours”, “bad” conduct
- Friends with problem behaviour
- Friends involved with drugs, particularly friends with “problem behaviours”; peer influences encouraging and facilitating drug involvement
- Drug availability
- Unconventionality; rebelliousness
- Low involvement with traditional value-oriented institutions (i.e. family, religious institutions, school)
- Poor academic achievement
- Poor quality relations with (and attachment to) parents and/or having parents with problems; poor parenting

**Risk factors for abuse/addiction**

- Multiple risk factors
- Multiple and/or severe problem behaviours
- Early age of onset or initiation of drug use
- High frequency use of drugs
- Parental substance abuse and/or antisocial behaviour, sibling drug abuse
- Family history of psychopathology
Severe family disruption and/or dysfunction, including significantly problematic divorce

Neurobiologic dysfunction related factors

Some psychopathologies: antisocial personality disorder, conduct disorder and criminal behaviour; affective disorders, including depression, bipolar disorder, anxiety disorders (in particular, post-traumatic stress disorder)

Severe childhood conduct/behaviour problems involving aggressivity, acting out and a high childhood activity level

Multiple psychopathologies, that is, more than one childhood psychiatric disorder, in particular a combination of an internalizing and externalizing disorder

Emotional/behavioural arousal self-regulation difficulties (possibly including sensation seeking), impulsivity and attention deficit disorder (called attention deficit/hyperactivity disorder in the United States) if coupled with aggressivity/conduct disorder; deficits in executive cognitive function and affect regulation may also be predisposing conditions

Traumatic experiences, including childhood physical and sexual abuse, particularly if the experience results in post-traumatic stress disorder

Other factors: generally poor function and difficulties in coping, social isolation, interpersonal difficulties

This means that only some individuals who use drugs will be at high risk for making the transition to drug abuse or addiction. In fact, data from general population surveys reveal that only a minority of illicit drug users meet the criteria for dependence. The National Comorbidity Survey conducted in the period 1990-1992 indicated that in the United States 51.0 per cent of the population 15-54 years of age reported illicit drug use at some time in their lives but only 7.5 per cent of the population had a history of drug dependence based on meeting diagnostic criteria at some time in their lives [2].

Not only has research consistently shown that the large majority of those who use drugs are not drug-dependent abusers, but it is also clear that in the large majority of cases, such drug use does not escalate to drug abuse. Given the difference in rates of dependence among users of different substances, it is not surprising that the latency of onset of dependence varies by substance. In a recent re-analysis of National Comorbidity Survey data [3], survival analysis was used to track the development of dependence on cannabis, cocaine and alcohol. Focusing on the transition from use to dependence, cocaine dependence (reported for 16.7 per cent of users) exhibited rapid development, 5-6 per cent of users having met the criteria within one year after initial use and approximately 11 per cent (the majority of eventual cases) within three years. Within 10 years, approximately 15 per cent of cocaine users had developed dependence. Cannabis dependence (reported for 9.1 per cent of users) developed less rapidly, less than 2 per cent of users having met criteria within one year and approximately 5 per cent in three years; rates were close to asymptotic within 10 years.
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Rates of lifetime dependence among lifetime users 15-44 years of age


For alcohol (reported for 15.4 per cent of all users), dependence developed at rates similar to those for cannabis in the first four years (reaching 5-6 per cent). Unlike cannabis dependence, alcohol dependence continued to increase at steady rates up to around seven years, when it reached around 11 per cent, and to continue to increase, though at somewhat diminishing rates, 15-20 years after initial use and beyond [3].

A relatively large number of people engage in some drug use, including drug use that involves experience with substances that have a high abuse liability (i.e. substances shown to be the most addictive). In the United States, for example, 39 per cent of the population 12 years of age and older (some 87 million persons) have tried illicit drugs at some time in their lives, 67 per cent have used cigarettes and 81 per cent have used alcohol (2000 estimates from the National Household Survey on Drug Abuse) [4]. Among the illicit drugs, lifetime exposure to cannabis is reported by 34 per cent, 11 per cent have used cocaine and 1.2 per cent have used heroin. Initiation of cigarette, alcohol and illicit drug use frequently occurs at an early age. The 2001 Monitoring the Future Study revealed that, in the United States, 53 per cent of students in grade 10 (aged 15-16) had used cigarettes in their lifetime, 70 per cent had consumed alcohol, 48 per cent had “been drunk” on alcohol, 46 per cent had used an illicit drug, 40 per cent had used cannabis, 6 per cent had been exposed to cocaine and 1.7 per cent had used heroin [5].

The prevalence rates vary considerably across countries. Paralleling the estimates for students 15-16 years old in the United States, the 1999 European School Survey Project on Alcohol and Other Drugs [6] reported that, among the 30 participating countries, the percentage of students reporting any cigarette smoking in their lifetime varied from 50 per cent in Cyprus to 86 per cent in Greenland.
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Obviously, it is common for adolescents and young adults to have experience with drug and alcohol use; one important question is why more drug use does not escalate to drug abuse and dependence. There appear to be two probably complementary reasons why most drug users do not become drug abusers. The influence of protective factors may be the first reason. It is very likely that no single factor "causes" drug abuse. Evidence indicates that drug abuse develops from the interaction of multiple influences, including biological, psychological and social/environmental factors. In addition, models that consider only risk factors ignore an important set of influences in the system of factors determining risk for drug abuse. Protective factors also play a critical role and they appear to work in opposition to risk factors. If the right protective factors are present at the right time and if they are sufficient in influence, they may nullify the predispositional influence of even potent risk factors. Research on protective factors provides support for interventions that attempt to increase these factors as an approach to prevention.

Secondly, examination of the risk factors associated with drug abuse and addiction shows that for the most part, these factors are powerful but, fortunately, relatively uncommon, at least at higher levels. This suggests that there are significant individual differences between those who make the transition from drug use to abuse and those who do not. Those at higher risk for drug abuse are likely to be influenced by both drug use and drug abuse predisposing factors. That provides encouragement for developing targeted interventions for children and adolescents showing evidence of drug abuse risk factors to supplement universal prevention programmes. It also suggests an explanation for those studies reporting findings that universal prevention interventions benefit even individuals at high risk.

Many prevention programmes target drug use initiation as a prevention strategy. The logic that someone who never uses drugs can never become a drug abuser seems irrefutable. This may be sufficient for those who are primarily at risk only for drug use. Even if they do have some drug involvement, there is a good chance it will be mitigated by the preventive intervention. Alternatively, children...
and adolescents who are subject to abuse risk factors may benefit—but benefit much less—from the same prevention programmes because a substantial part of the influence towards drug involvement is not ameliorated. Even before any drug experience, those who are more vulnerable to future drug abuse are likely to already be on different paths because they are also influenced by the drug abuse risk factors.

Further, prevention programmes that target drug use initiation usually do not include an intervention for those who are already past the point of initiation and already have some drug involvement and/or some experience with illegal drugs or with other (usually illicit) psychoactive substances such as alcohol and/or nicotine (whose purchase and/or use is prohibited for most adolescents). As discussed above, this includes a large percentage of adolescents and young adults and is likely to include a high percentage of those at higher risk for drug abuse. Incorporating escalation intervention components into prevention programmes may significantly increase their ultimate effectiveness.

It is also important to remember that drug use has consequences that in turn become predispositional influences on subsequent behaviour. It is likely, for example, that drug use impairs an adolescent’s social and psychological development, making them more vulnerable to maladaptive behaviour and other problems, including more drug use [7]. Curtailing or even stopping drug abuse will not restore a person to his or her pre-abuse state. Drug abuse is not just the use of drugs that got out of control and that, if brought back under control, will leave no lasting consequences. A person who stops drug use will not simply become the same person with the same development and coping resources that they would have had if they had not used drugs. Heavier drug users whose use has impaired their maturation will not, for example, suddenly catch up on the development of problem-solving, coping and other skills [8]. For those who are vulnerable to abuse or who are already beginning to escalate their use, this is even more the case. Successful drug abuse prevention programmes for vulnerable adolescents may also need to help those individuals mitigate the impaired development and functional maturation resulting from their drug involvement.

The dynamic interaction between risk and protective factors occurs in the context of the individual’s development. In other words, etiologic factors are not merely antecedents exerting influence in a simple linear cause-effect fashion. Individual and environmental influences interact at any given developmental stage of the child or adolescent to lead them to certain behaviours. Those behaviours have consequences and the consequences then become influences on subsequent interactions and behaviours. For example, a young child with a difficult temperament being raised by parents who are not able to help the child to develop self-regulation may then have a more difficult time making friends and doing well in school. In turn, this may predispose the child to associate with peers who engage in delinquent behaviour, creating an opportunity for ready availability of cigarettes and alcohol and encouragement for their use [9, 10]. A more extensive discussion of a developmental conceptualization of drug abuse aetiology can be found in Blackson [11] and Glantz [12].
Many models of drug abuse aetiology assume that there is a single primary pathway or trajectory leading to drug abuse. Many versions of the single trajectory perspective tacitly assume that the aetiology of drug abuse is a linear continuum, a single trajectory that could potentially end in drug abuse for most people who start down the path, that is, individuals who are drug users. Individual differences in vulnerability are assumed to be minimal; that is, given sufficient drug use experience, most people will become drug abusers. Single trajectory concepts typically minimize the role of pre-drug-use differences as determinants of later drug use/abuse.

Conceptualizations of drug abuse aetiology that are based primarily on a single risk factor or even a set of risk factors, even conceptualizations that include the role of protective factors, usually assume that the primary etiological factors are relatively unchanging over the course of the aetiology of drug abuse and that most of the determination of the individual’s course of drug behaviour is exerted at the beginning of the chain of behaviour. They are “ballistic” models that suggest that the aetiology of drug abuse is much like the firing of a cannonball. Once fired, the trajectory of the cannonball is largely predetermined and changes in course are possible only with extreme external influences. The best hope for stopping the cannonball from striking the point at the end of its trajectory is to prevent it from being fired in the first place. While this is the case for cannonballs, it is not true for a complex course of human behaviour.

Aetiology is not a straight line course but more like a spiralling line with a feedback loop that keeps reorienting before it moves again, possibly in a new direction [13]. Every consequence becomes a “cause” influencing the next step, and all of those influences are complex interactions taking place in the context of development. In more severe instances, the course of normal development may be impaired or redirected. Furthermore, changes in trajectory for an individual can occur at any point because of the influence of strong positive or negative factors in the environment. Risk factor models conceptualizing drug abuse aetiology as a ballistic event ignore both the developmental nature of the aetiology and the developmental and environmental contexts in which it usually occurs.

The best theoretical formulation to account for the development of drug abuse appears to be an interactive risk and protective factor model that takes into account individuals’ developmental stage and processes. That type of aetiology model is most useful when viewed with the perspective of patterns of factors and the results of their interaction [14]. It places more emphasis on the outcome of the interactions of risk and resilience and individual and environmental factors rather than on the specific original factors themselves. Adding in a developmental framework that views the course of the patterns over time supports a pattern or path model. Some patterns will be more common and, as the patterns involve interactions of factors over time, the metaphor of “paths” is particularly helpful.

Kazdin [15, p. 180] has provided a brief definition of developmental psychopathology as “the study of clinical dysfunction in the context of maturational and developmental processes”. (For more comprehensive expositions of developmental psychopathology, see Cicchetti [16], Cicchetti and Rogosch [17] and Luthar
This perspective is particularly helpful in understanding drug abuse etiology, especially because most of the formative steps in the etiology of drug abuse typically occur during the periods of child and adolescent development [19].

Such a developmental psychopathology model of interacting factors implies that more than one pattern may lead to drug abuse. Evidence indicates that there is not a single path or combination of factors that leads to drug abuse; instead, there are numerous possible etiological patterns. Some paths, however, are more common than others. Evidence indicates that one particularly common pathway to drug abuse may be through a path involving conduct problems while another may be through a childhood psychopathology pathway. It is probably the case that a small number of paths or patterns will account for the drug abuse etiology of the majority of individuals [20, 21].

Differentiating drug use from abuse is only a part of a larger issue of the heterogeneity of drug abuse. Just as it is important to recognize the qualitative differences of use compared with abuse, other distinctions may also be important. There is no question that there are variations in a number of dimensions of drug use/abuse. There is diversity in the drugs and combinations of drugs used, the routes of administration, the quantity and frequency patterns of use, the “career” stage of abuse, the age when use and abuse began, whether there is a comorbid psychiatric condition, what the consequences are, whether there is a physical dependence etc. Clearly, drug use/abuse is highly heterogeneous.

Concepts, models and research on drug abuse, however, do not always reflect this heterogeneity. For example, research measurements often view drug abuse as a dichotomous variable with few or no variations and as a final outcome, a static endpoint. Individual differences in drug abuse and related factors are often minimized in data analysis and are often considered to be “noise”, measurement error to be controlled for. While some variability probably reflects differences in less significant factors, some of the variability in drug abuse is likely to be related to crucial distinctions (see Zucker, Fitzgerald and Moses [22] for a discussion of this and associated issues of heterogeneity related to alcoholism). Even if there are critical factors common to all cases of drug abuse and related disorders (for example, the involvement of particular neurological processes), the variations may denote significantly different subtypes or patterns. To use an analogy, the general category of “cancers” includes a large number of relatively distinct subtypes. While there are some significant core commonalities among the different types of cancer, there are also critical differences that distinguish the subtypes. Failure to consider the different clinical entities represented by the nosology of cancers would result in a limited understanding and ability to treat the different conditions. The same principle applies to drug abuse and the variable characteristics associated with it.

The question is, which if any of the characteristics relate to critical differences in drug involvement and which are relatively non-critical variations? There is no consensus among clinicians or researchers. The research data point to the probability that some variations may relate to critical heterogeneities. Often, however,
findings indicate some variations are more associated with degrees of severity without providing information about whether there is a qualitative difference involved. Identifying a common characteristic is helpful but it falls far short of identifying the set of fundamentally distinguishing characteristics of a phenotype. A big part of the problem is that it is not possible to determine whether a particular variation relates to a critical defining characteristic of drug abuse when there is no agreed upon set of critical defining characteristics of drug abuse. This issue is often referred to as the phenotype question.

The phenotype question

The concept of phenotype is prominent in genetics research, where the goal is often to relate a particular “genotype”, a particular genetic constellation, to a particular “phenotype”, the observable traits or characteristics of an organism. A phenotype is not, however, a direct manifestation of the genotype. The phenotype results from the genotype as it manifests and develops in a particular environment. Differences in environmental influences and experiences are the reason that, for example, even though identical (monozygotic) twins have identical deoxyribonucleic acid sequences (DNA), they have similar but not identical fingerprints and people who know them well can usually tell them apart even as infants. The relationship of the genotype to the phenotype varies with different traits. Eye colour, for example, is highly related to specific gene characteristics, while other phenotypes, particularly those that are more behavioural in nature, may be less directly related. For some traits and behaviours, there may not be a specific genotype. In other cases, a particular genotype may be less directly related to a phenotype but is more strongly associated with an intermediary, an “endophenotype”, which is an underlying biological characteristic or mechanism.

Obviously, determining a particular genotype-phenotype relationship requires a clearly distinct and preferably consensually accepted definition of both the genotype and the phenotype. In some cases, it can be difficult to formulate a consensus of a reliably identifiable, appropriately inclusive and concretely defined phenotype. The greater the heterogeneity of the phenotypic characteristic, the more difficult the problem. Not surprisingly, this has been an issue for genetic research related to drug abuse.

These are not esoteric theoretical questions. Defining a phenotype for drug abuse is an operational necessity for investigations of the relationship of genetic characteristics to drug abuse, and there is no simply defined drug abuse phenotype. Many researchers have used an individual’s meeting the diagnostic criteria of the International Classification of Disease (ICD) or the Diagnostic and Statistical Manual (DSM) for a drug abuse disorder as the phenotype while others have relied on other criteria. Even when these types of standardized assessments and categorizations are used, questions remain. For example:

(a) Should the criteria for the phenotype be drug-specific?
(b) Is there a predispositional endophenotypic intermediary that must be identified in order to determine the genetic contribution to drug abuse and perhaps to understand vulnerability to drug abuse?

(c) Is the phenotype of drug abuse too narrow and should it be broadened to include other forms of substance abuse, other antisocial behaviours or other psychopathological characteristics?

(d) As individuals go through different stages of drug involvement, are different characterizations necessary to correctly identify drug abuse? To what extent is it necessary to modify the characterizations necessary to correctly identify drug abuse in order to take into account the age, developmental level and life circumstances of the individual?

(e) Is the behavioural drug involvement pattern (defined by such factors as age of onset, rate of escalation, relapse history, experience of and tolerance for negative consequences and quantity and frequency of use) a more useful phenotype?

(f) Does using a phenotype defined by an individual’s meeting the criteria for a DSM drug abuse disorder exclude significant alternative drug abuse manifestations?

(g) Is a more useful phenotype of drug abuse defined by an individual’s reaching some ultimate common condition or process such as a particular neurochemical or neuroanatomical equifinal state?

(h) Is a currently abstinent drug abuser still appropriately classified as a drug abuser; is this classification appropriate even if they never relapse?

(i) Should drug abuse be thought of as having multiple phenotypes?

(j) Should the basic model of a drug abuse phenotype be based on the characteristics of a disease? A psychopathology? An antisocial behaviour?

There has been general recognition of the importance and lack of resolution of the phenotype issue in drug abuse genetics research. There has been less attention and concern in many other areas of drug abuse research. Some researchers have considered the issue to be little more than a semantic disagreement, but the way that drug abuse is defined has significant pragmatic and heuristic consequences: for example, using “drug use” as the phenotype for drug abuse obscured critical differences. By differentiating drug use and abuse, researchers were able to determine that different factors, in fact different paths, led to one versus the other. The importance of formulating an empirically based phenotype for application in other areas of drug abuse research besides genetic research is at least as great.

Asking the question “What is the phenotype of drug abuse?” is asking “What are the critical defining characteristics of drug abuse?”. Each different phenotype conceptualization focuses on different aspects and models of drug abuse and sets different criteria for the determination of drug abuse. An important issue is whether it is appropriate to use different defining criteria depending on the
question being asked. In some cases this appears to be necessary. Some research questions seem to call for different phenotype definitions because they focus on different aspects or stages of drug abuse: for example, comparisons of drug abuse among adolescents versus adults. Other circumstances seem to require still other phenotype definitions: for example, identifications of marijuana abusers versus cocaine abusers.

The general principle of using different phenotypes in relation to different questions seems reasonable to some, in that it responds to the variations in population and behaviour. This leaves the question of whether a phenotype must have some core characteristics in order to be a drug abuse phenotype. Consider the phenotype of skin cancer. Different diagnostic criteria are applied for different stages and variations of the illness and different populations may have different risks and manifestations. Certain environmental factors and behaviours may change the likelihood and course of the disorder and therefore different prevention and treatment strategies may be most effective depending on a variety of factors. Nevertheless, there is a core concept of defining criteria that denote skin cancer and those criteria are involved in all concepts, forms and interventions for skin cancer. Whether there is more heuristic benefit in formulating one phenotype for drug abuse varying by stage and variation or in developing multiple phenotypes is unknown. Ultimately, the resolution must be driven by empirical findings.

The encouragement for the recognition and exploration of the heterogeneity of drug abuse is not intended to minimize the commonalities inherent in drug abuse. It is obviously the case that each instance of drug abuse is not unique. The issue is that identifying critical characteristics common to all instances of drug abuse has not been accomplished. Even for a single drug-abusing individual, the defining characteristics of their involvement with drugs changes over time. If the cardinal characteristics of a phenomenon cannot be specified, it is difficult to understand and develop the most effective interventions for that phenomenon. It is essential to determine which characteristics of drug abuse are universal, which are common, which are particular to specific subgroups and which are idiosyncratic. However, even seemingly simple classifications can present considerable challenges.

To use a mundane example, it would be difficult to research the nature of desserts without first determining their cardinal characteristics. While everyone knows what “dessert” is, developing a classification that would facilitate research and understanding requires some organized conceptualization. To illustrate, it is not possible to formulate a single recipe for all types of desserts, for example, none of the ingredients in a fruit salad are found in a chocolate cake. A particular response to the food would help to discriminate desserts from other foods. Most (and for the purposes of the discussion, all) desserts are sweet. This could be agreed to be a universal characteristic of desserts even though the experience of sweetness is not achieved by the same ingredient (although the sweetening agent is usually sugar in some form or a related substance). Interestingly, this does not resolve the question of whether it is the taste of sweetness or the particular component (sugar) that is the defining characteristic of desserts. This is similar to the
controversy over whether the characteristics of drugs interacting with biological systems or some more behavioural or subjective phenomenon “cause” or at least determine drug abuse. Further, not all sweet foods and not all foods containing sugar (sweet and sour chicken, chutney, barbecued pork, etc.) are desserts. Nevertheless, identifying an almost universal characteristic (sweetness) and an almost universal component creating that characteristic (sugar or a sugar substitute) would make research on desserts practical and more productive. It would not, however, identify a dessert phenotype.

Breaking the general category of desserts into distinctive major subtypes—creating a nosology of desserts—would be very helpful, especially if distinguishing critical characteristics of each subtype could be identified. Public consensus alone would probably not be adequate. Research would have to be done to identify some underlying principles or critical characteristics, in order to differentiate the subtypes into relatively exclusive categories. Classification would not be easy. For example, establishing the categories “pastry”, “ice cream”, “pudding”, “candy” and “fruit desserts” would lead to such questions as whether fruit pies are a separate category, a form of pastry, or a fruit dessert. However, if a categorization were developed that described the “real” subtypes, a prototype recipe for each subtype might be possible and that would greatly facilitate research on desserts. Without this classification step, the heuristic potential of dessert research would be limited. This is approximately the current status of drug abuse research.

The discussion of formulating a phenotype and related nosology for desserts is facetious and intended only as a non-controversial example. Unfortunately, in the area of behavioural research, not having a phenotype and classification of subtypes for many behaviours has been a serious obstacle. For example, the field of resilience research has not successfully developed either a phenotype or a classification for resilience; even the identification of a universal characteristic has proven to be elusive. For this reason, at least in part, the extensive research done on resilience has not been nearly as productive as once expected. (See Glantz and Sloboda [23] and Kaplan [24] for in-depth discussions of resilience.)

While a considerable understanding of compulsive psychoactive drug use, despite negative consequences (the universal characteristic of drug abuse), has been achieved and a great deal of knowledge about drugs of abuse (the universal component) has been ascertained, new quantum steps forward in progress probably require identifying the subtypes or patterns of drug abuse. This is the next frontier for drug abuse research. In all likelihood, it is a pivotal accomplishment that will be the foundation of future critical progress.

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