Impulsivity and Suicide Risk: Review and Clinical Implications

by E. David Klonsky, PhD and Alexis M. May, MA

Impulsivity, a frequently misunderstood aspect of suicide risk, has long been considered important to the etiology of suicide. However, impulsivity is highlighted for its role in facilitating suicidal actions among those with suicidal ideation. Mann and colleagues developed a clinical model of suicidal behavior which suggests that impulsivity makes individuals more likely to act on suicidal feelings. Similarly, Bryan and Rudd state that impulsivity “may actually be a more significant indicator of suicide attempt than the presence of a specific suicide plan.”

Impulsivity has been adopted as a risk factor or warning sign for suicide. The American Association of Suicidology includes impulsivity as both a chronic and an acute suicide risk factor. Impulsivity is also highlighted by the American Foundation for Suicide Prevention and the Substance Abuse and Mental Health Services Administration. However, as discussed below, these widely held perceptions about impulsivity do not appear to be supported by research.

Correcting misperceptions

The claim that impulsivity facilitates transition from suicidal thoughts to suicide attempts suggests a clear and testable prediction: trait impulsivity should be higher among those who attempt suicide than among those who only consider suicide. However, to the surprise of many, research on the role of impulsivity has routinely failed to support this claim. For example, a 2007 study of young adults by Brezo and colleagues found that attempters scored no higher on the Barratt Impulsiveness Scale than patients with suicidal ideation who had never attempted suicide.

More recently, my colleagues and I examined a military population and found that while both suicide attempters and patients with suicidal ideation scored higher on a measure of impulsivity than those who had never been suicidal, impulsivity scores were equivalent between attempters and patients with suicidal ideation who had never attempted suicide. In other words, impulsivity was moderately elevated in anyone with a history of suicidality (thoughts or behavior), but the study failed to show any further elevation among those who acted on their ideation and progressed to suicide attempts.

On the basis of these surprising findings, we conducted a subsequent analysis using the UPPS Impulsive Behavior Scale. The UPPS develops, Whiteside and Lynam, suggest that impulsivity is a heterogeneous construct. They used a series of factor and psychometric analyses to identify 4 distinct impulsivity-related traits: Urgency (responding rashly to negative emotions), poor Premeditation (difficulties in foreseeing consequences of actions), poor Perseverance (tendency to give up easily), and Sensation seeking (preference for excitement and stimulation).

Using a brief version of the UPPS in a large sample of adolescents and young adults, we found that attempters and individuals with suicidal ideation exhibited equivalent scores on 3 of the dimensions (Urgency, Perseverance, and Sensation seeking) and that attempters scored only very slightly higher on the fourth (Premeditation). Taken together, the findings suggest that suicide attempters and individuals with suicidal ideation exhibit similar levels of trait impulsivity, a pattern that is contrary to clinical beliefs and guidelines.

The studies described above examined impulsivity as a personality trait that could occur at higher or lower levels within an individual. However, there is a second body of research that is also relevant to the role of impulsivity in suicide.

This research examines the impulsive nature of the suicide attempt itself. Many different definitions of attempt impulsivity have been used, including degree of forethought, amount of time spent making the attempt before making the attempt, presence of a suicide plan, and amount of time spent making a plan, among many others. Given the many ways to define or identify an impulsive attempt, it is not surprising that studies on attempt impulsivity produce widely divergent results.
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For example, the percentage of suicide attempts estimated to be impulsive has ranged from a low of 20% to a high of 85%.1,24

Perhaps one of the most surprising findings is that trait impulsivity and attempt impulsivity appear to be unrelated. In other words, among individuals who have made suicide attempts, those who score higher on personality measures of impulsivity are not the ones making the more impulsive suicide attempts. This pattern has been found and reported by 2 separate research teams and thus appears to be true despite its counterintuitive nature.10,11

Taken together, studies indicate 2 critical limitations of current knowledge. First, when it comes to characterizing impulsivity in suicide, there is a disconnect between clinical guidelines and research. The way impulsivity is described in lists of suicide risk factors and warning signs is not supported—and is in some cases disputed—by empirical research. Second, the field continues to struggle to understand the role impulsivity plays in influencing the likelihood and nature of suicide attempts, as well as how to best understand suicide and suicide risk.

Understanding impulsivity

Looking at data from 70 studies, Anestis and colleagues12 examined the association between measures of impulsivity and measures of suicidal behavior (eg, non-lethal attempts,
death by suicide). Across all studies, the relationship between impulsivity and suicidal behavior was modest (Hedges’ g = 0.37). However, most of these studies were cross-sectional; current impulsivity was used to predict a history of suicidal behavior. In the studies that explored impulsivity as a predictor of future suicidal behavior, the association was even smaller, barely above 0 (Hedges’ g = 0.09). The researchers conclude that the role of impulsivity in suicide is likely to be small and indirect rather than central or causal.

Anestis and colleagues next proposed a model specifying the role of impulsivity in suicide. In particular, the authors noted work by Joiner11 that suggested that to make a potentially lethal suicide attempt, one must have the capability to make an attempt. Pain and fear of death serve as barriers to making a suicide attempt, and certain kinds of experiences can allow people to habituate to pain and fear of death and overcome these barriers. These experiences are referred to by Joiner as painful and provocative events and can include a variety of experiences and events, such as exposure to violence, nonsuicidal self-injury, and substance use.

Anestis and colleagues’ theory that rather than a direct relationship, impulsivity has a distal relationship to suicidal behavior by virtue of increasing one’s exposure to painful and provocative events. Indeed, they noted that initial studies have found that painful and provoca-

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If, contrary to commonly held clinical beliefs, impulsivity is not a strong or central predictor of suicide or suicide risk, what may be a more accurate, more useful alternative model?

The ideation-to-action framework

The often cited risk factors for suicide, such as impulsivity as well as depression, hopelessness, and most mental disorders, are indeed greater among suicidal populations, but they distinguish poorly between those who attempt suicide and those who consider but never attempt suicide. In other words, once an individual is known to have suicidal ideation, assessing his or her depression, hopelessness, psychiatric diagnosis, and impulsivity offers little to no information about the risk of acting on that ideation and making a suicide attempt. This distinction is critical because most individuals with suicidal ideation do not go on to attempt suicide.

This pattern of findings, replicated in numerous studies by numerous investigators, led to the ideation-to-action framework. From this perspective, predictors and explanations of prospective, predictors and explanations of a suicide attempt are more likely to facilitate a lifestyle in which painful and provocative events are more likely to be experienced.

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important safety information (continued)

Metabolic Changes (Continued)

- Dyslipidemia: Undesirable alterations in lipids have been observed in patients treated with atypical antipsychotics.

- Weight Gain: Increases in weight have been observed with SAPHRIS. Monitor weight regularly in patients on SAPHRIS.

Hypersensitivity Reactions: Hypersensitivity reactions, including anaphylaxis, angioedema, hypotension, tachycardia, swollen tongue, dyspnea, wheezing, and rash, have been observed in patients treated with SAPHRIS. In several cases, these reactions occurred after the first dose.

Orthostatic Hypotension, Syncope, and Other Hemodynamic Effects: SAPHRIS may induce orthostatic hypotension and syncope. Use SAPHRIS with caution in patients with cardiovascular/cerebrovascular diseases, conditions which predispose to hypotension, and in the elderly. Use SAPHRIS cautiously with other drugs that can induce hypotension, bradycardia, or respiratory or central nervous system depression. Monitor orthostatic vital signs, and consider a dose reduction if hypotension occurs.

Leukopenia, Neutropenia, and Agranulocytosis: Leukopenia/neutropenia have been reported with antipsychotics, including SAPHRIS. Agranulocytosis (including fatal cases) has been reported with other antipsychotics. Monitor complete blood count in patients with pre-existing low white blood cell count (WBC)/absolute neutrophil count or history of drug-induced leukopenia/neutropenia. Discontinue SAPHRIS at the first sign of a clinically significant decline in WBC and in severely neutropenic patients.

QT Prolongation: In an adult QT study, SAPHRIS was associated with increases in the QTc interval from 2 to 5 msec vs placebo. No SAPHRIS patients had QTc increases of ≥60 msec or a QTc of ≥500 msec. Avoid SAPHRIS in combination with other drugs known to prolong QTc interval, in patients with congenital prolongation of QT interval or a history of cardiac arrhythmias, and in circumstances that may increase occurrence of torsades de pointes and/or sudden death in association with the use of drugs that prolong QTc interval.

Hyperprolactinemia: Like other drugs that antagonize dopamine D2 receptors, SAPHRIS can elevate prolactin levels and the elevation can persist during chronic administration. Galactorrhea, amenorrhea, gynecomastia, and impotence have been reported in patients receiving prolactin-elevating compounds.

Seizures: Use SAPHRIS with caution in patients with history of seizures or with conditions that lower the seizure threshold.

Potential for Cognitive and Motor Impairment: Somnolence was reported with SAPHRIS. Caution patients about performing activities requiring mental alertness (eg, operating hazardous machinery or a motor vehicle).

Body Temperature Regulation: Appropriate care is advised when using SAPHRIS in patients who will experience conditions that increase body temperature, eg, exercising strenuously, extreme heat, concomitant anticholinergics, or dehydration.

Suicide: The possibility of suicide attempt is inherent in psychotic illnesses and bipolar disorder. Close supervision of high-risk patients should accompany drug therapy. Prescriptions should be written for the smallest quantity of tablets to reduce risk of overdose.

Dysphagia: Esophageal dysmotility and aspiration have been associated with antipsychotics. Aspiration pneumonia is a common cause of morbidity/mortality in elderly patients, in particular those with advanced Alzheimer’s dementia. SAPHRIS should not be used in patients at risk for aspiration pneumonia.

Drug Interactions: Monitor blood pressure and adjust antihypertensive drugs when taken with SAPHRIS. Based on clinical response, SAPHRIS dose reduction may be necessary when used with strong CYP3A4 inhibitors (fluvoxamine). Reduce paroxetine (CYP2D6 substrate and inhibitor) dose by half when taken with SAPHRIS.

Pregnancy: Advise patients to notify their healthcare provider of a known or suspected pregnancy. SAPHRIS may cause extrapyramidal and/or withdrawal symptoms in neonates with third trimester exposure. Based on animal data, SAPHRIS may cause fetal harm. The National Pregnancy Registry for Atypical Antipsychotics monitors pregnancy outcomes in women exposed to antipsychotics, including SAPHRIS, during pregnancy. For information, contact 1-866-961-2388 or http://womensmentalhealth.org/clinical-and-research-programs/pregnancyregistry/.

Adverse Reactions: In adult clinical trials with SAPHRIS (5 and 10 mg BID) vs placebo, commonly observed adverse reactions (≥5% and at least twice the rate of placebo) were:

- Bipolar I (monotherapy): somnolence (24% vs 6%), dizziness (11% vs 3%), extrapyramidal symptoms other than akathisia (7% vs 2%), and increased weight (5% vs <1%)
- Bipolar I (adjunctive): somnolence (22% vs 10%) and oral hypoglycemia (5% vs 0%)

Postmarketing Experience: Application site reactions, primarily sublingual, have been reported (eg, oral ulcers, blisters, peeling/slaughtering, and inflammation). Choking has been reported, sometimes associated with oropharyngeal muscular dysfunction or hypotesthesia.
for suicide should be classified as to how they approach (a) the risk of suicide ideation, (b) the risk of suicidal attempts among those with suicidal ideation, or (c) both. For example, depression, hopelessness, impulsivity, and most psychiatric disorders appear to be best characterized as predictors of suicidal ideation.15,17

In contrast, fearlessness and reduced pain sensitivity appear to specifically characterize suicide attempters, but not patients with suicidal ideation.14 Other risk factors, such as nonsuicidal self-injury, appear to confer risk of both suicidal ideation and attempts.14 Thus, constructs and clinical models guided by an ideation-to-action framework can greatly improve models of suicide risk as well as efforts to understand and prevent suicide.

A new model of suicide and suicidal risk
A new theory of suicide positioned within the ideation-to-action framework is the 3-step theory (3ST). The 3ST makes 3 central claims, all of which are consistent with existing evidence that is supported by recent findings.20

First, the combination of pain and hopelessness is what brings about suicidal ideation. The nature of pain is intentionally not specified. Any type of pain that makes daily life aversive, regardless of its source, can be implicated in suicidal ideation.

When efforts to engage with life are paired with emotional, psychological, or physical pain, the individual is behaviorally conditioned to want...
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to avoid engaging with life. It is the consequence of pain and hopelessness that leads to suicidal thoughts.

Indeed, pain and hopelessness are the two primary motivations for suicide.

3. More to the point, the recent research suggests that it is the combination of pain and hopelessness that matters. Specifically, suicidal ideation was negligible in those low on both pain and hopelessness and was negligible in those either high on pain or hopelessness; in contrast, suicidal ideation was elevated only in the subgroup high on both pain and hopelessness.

Second, the research suggests prevention suicide risk from escalating to fatal situations in those (i.e., those experiencing both pain and hopelessness). In other words, if connectedness to life—to loved ones, to a role, to any sense of meaning or purpose—exceeds the pain, suicidal ideation will remain at modest levels. However, if pain exceeds the connectedness to or investment in suicidal ideation becomes strong and active. Recent findings support this notion: connectedness was found to be a significant buffer against suicidal ideation only in those with pain and helplessness.
of these 3 variables predicts suicide attempt history, even when controlling for past and current suicidal ideation.62

Dispositional variables are driven largely by genetics, such as pain sensitivity. For example, someone born with low pain sensitivity will have a higher capacity to carry out a suicide attempt. Indeed, more recent work from Smith and colleagues12 indicates that capacity for suicide behavior will generally be acquired. Acquired variables are experiences associated with pain, injury, fear, and death, and they can lead over time to a higher capacity for suicide attempt behavior. Practical variables are concrete factors that make a suicide attempt easier. These factors include access to knowledge of, and comfort with, and lethal means. There are countless ways for someone to increase practical capacity. Each of these 3 factors—dispositional, acquired, and practical—contribute to the capacity for attempted suicide, and an individual with strong suicidal ideation will only make a suicide attempt if and when he has the capacity to do so. (Please see Suicide Risk, page 25)
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One reason the 3ST is useful is that it suggests clear clinical implications for how a specific set of interventions with a particular patient can target and impact suicide risk.

Conclusion

Contrary to commonly held beliefs, a large body of research suggests that impulsivity is not a strong predictor or cause of suicidal behavior. Instead, trait impulsivity appears to be a modest and distal predictor of suicide; however, large, prospective studies that could best address this issue are still needed.

Future studies guided by the ideation-action framework may be able to specifically test the degree to which the impulsivity-suicidality relationship is explained by impulsivity’s impact on the pain and hopelessness that cause ideation and/or on suicide capacity. Instead of focusing on impulsivity, it may be fruitful to focus on domains that have been consistently shown to predict and motivate suicidal ideation and suicide attempts, including pain (especially physical pain associated with illness or injury), depression, and hopelessness. Interventions for suicide risk may be most effective when one or more of these domains is modifiable.
Dr Kbonsky is Associate Professor and Ms May is a Doctoral Candidate in the department of psychology at the University of British Columbia in Vancouver. The authors report no conflicts of interest concerning the subject matter of this article.

References

by Sean Z. Kalinski, MB, BCh, PhD

A t some point in their life, most people are likely to have acted on impulses or reacted to provocations. This is accepted normal human behavior. However, in some cases it is pathological and some individuals behave this way habitually—as part of a pattern of behaviors that may have begun sometime in their youth. Although there are protein manifestations of these behaviors, ranging from suicidal gestures, substance abuse, risk taking, and antisocial behaviors, a subset of individuals are also aggressively and violent.

The terms “impul­ sivity” and “disorders of impulse control” have customarily been used interchangeably. Yet there have been contrasting definitions in the literature. “Impulsivity” has been defined as a decreased sensitivity to negative conse­quences, rapid unplanned reactions to stimuli (without adequate process­ing of information), and lack of regard for long-term consequences. “Disorders of impulse control” have been characterized as repeated fail­ures to resist an impulse or perform an action that is harmful, with a preceding subjective sense of increasing tension (or arousal) and an experience of pleasure or gratification, i.e., catharsis, while committing the act. In both cases, the consequences of the acting out are usually deteri­ orious, with subsequent feelings of re­ gret or guilt.

No studies have directly com­ pared individuals whose impulsivity only takes the form of acting precipi­ tously to stimuli with those who act solely because of impelling urges. In practice, there may not be differences concerning both aspects, such as those with borderline personality disorder who repeatedly act out their urges and can also respond explosively to stimuli. In DSM-5, a new category for borderline per­ sonality disorder is impulsivity, which also encompasses risk-taking activities that are exemplars of poor impulse control, such as excessive spending, promiscuity, and reckless driving. Individuals with intermittent explosive disorder, a “pure” impulse control disorder, exhibit “impulsive (or anger-based) aggressive outbursts” in response to minor provoca­ tions or stressors. Whatever the distinction, individuals with these disorders all have in common a defici­ cy in inhibiting damaging behavior.

In clinical practice, it may actually be difficult to differentiate between compulsions, addictions, and irresistible times meticulously, often report that they had to surrender to overwhelming urges.

Neurobiology and experience
Over 30 years ago, Linnola and colleagues found that impulsive violent offenders had significantly lower cer­ ebrospinal fluid (CSF) concentra­ tions of the major metabolite of sero­ tonin, 5-hydroxyindoleacetic acid (5-HIAA). Their findings have been convincingly validated.1,2 Serotonin is an important inhibi­ tor neurotransmitter, especially in the amygdala, anterior cingulated cortex, and dorsal-lateral prefrontal and orbitofrontal cortices. Reduced or dysregulated serotonin activity is associated with impulsivity and aggression. The possible mechanism may be the disruption of circuits be­ tween the amygdala and the medial prefrontal cortex, which results in amygdala hyperactivity and reduced prefrontal inhibition.3 Impulsive ag­ gression presumably occurs con­ sequent to ongoing arousal (from the amygdala) that primes negative ur­ gency—the tendency to respond impul­ sively and aggressively to provo­ cations or perceptions of threat.

Individuals who have the X-linked allele that codes for low func­ tioning monoamine oxidase A (MAOA-L), the most important en­ zyme for the metabolism of central serotonin, tend to display enhanced activation in subcortical limbicareas (especially the amygdala) and re­ duced prefrontal inhibition. This al­ lele has now acquired the moniker “warrior gene” because of its consis­ tent association with impulsive ag­ gression behavior.

Individuals who have the s allele for the serotonin transporter promot­ er gene also tend to exhibit patterns of impulsiver behavior, probably be­ cause of the reduced presynaptic re­ uptake of serotonin. It may seem paradoxical that low-functioning ver­sions of MAOA and serotonin prom­oter genes are associated with impul­ sive aggression because these genes lead to increased levels of se­ rotonin. The most likely mechanism is that increased levels of serotonin occupy serotonin 1A and serotonin 1B autoreceptors that “switch” the presynaptic neuron off and function­ ally cause a serotonin deficiency.4

(See Violence, page 22)