Risk Factors for Suicidal Thoughts and Behaviors: A Meta-Analysis of 50 Years of Research

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Suicidal thoughts and behaviors (STBs) are major public health problems that have not declined appreciably in several decades. One of the first steps to improving the prevention and treatment of STBs is to establish risk factors (i.e., longitudinal predictors). To provide a summary of current knowledge about risk factors, we conducted a meta-analysis of studies that have attempted to longitudinally predict a specific STB-related outcome. This included 365 studies (3,428 total risk factor effect sizes) from the past 50 years. The present random-effects meta-analysis produced several unexpected findings: across odds ratio, hazard ratio, and diagnostic accuracy analyses, prediction was only slightly better than chance for all outcomes; no broad category or subcategory accurately predicted far above chance levels; predictive ability has not improved across 50 years of research; studies rarely examined the combined effect of multiple risk factors; risk factors have been homogenous over time, with 5 broad categories accounting for nearly 80% of all risk factor tests; and the average study was nearly 10 years long, but longer studies did not produce better prediction. The homogeneity of existing research means that the present meta-analysis could only speak to STB risk factor associations within very narrow methodological limits—limits that have not allowed for tests that approximate most STB theories. The present meta-analysis accordingly highlights several fundamental changes needed in future studies. In particular, these findings suggest the need for a shift in focus from risk *factors* to machine learning-based risk *algorithms*.

Keywords: meta-analysis, prediction, risk factors, suicidal behavior, suicide

Suicidal thoughts and behaviors (STBs) are major public health problems that have devastating impacts on individuals, families, and communities. Suicide is among the leading causes of death worldwide, accounting for more than 40,000 annual deaths in America (Centers for Disease Control and Prevention [CDC], 2014) and an estimated one million annual deaths across the globe (World Health Organization [WHO], 2012). To put this in perspective, suicide accounts for more annual deaths than homicide, AIDS, car accidents, and war (CDC, 2014; WHO, 2012). These suicide deaths are in addition to an estimated 25 million annual suicide attempts (Crosby,

Gfroerer, Han, Ortega, & Parks, 2011) and 140 million annual suicide ideators worldwide (Borges, Angst, Nock, Ruscio, & Kessler, 2008). Unfortunately, the STB problem does not seem to be improving. Suicide rates have not declined appreciably in many decades (CDC, 2014; McKeown, Cuffe, & Schulz, 2006; Nock et al., 2008) and large national surveys indicate a similar pattern for suicide ideation, plans, and attempts (Kessler, Berglund, Borges, Nock, & Wang, 2005). The scope, severity, and consistency of this problem has prompted decades of research devoted to understanding how STBs work, how to accurately predict STBs, and how to best treat and prevent STBs.

Risk factors are a critical component of each of these branches of research. As noted by the World Health Organization (2012),

> The identification of risk and protective factors is a key component of a national suicide prevention strategy, and can help determine the nature of type of interventions required. Risk factors, in this context, are indicative of whether an individual, a community or a population is particularly vulnerable to suicide. (p. 13)

The present meta-analysis of risk factors for STBs may accordingly inform suicide theory, prediction, and treatment.

Risk Factors and Theories

Many different theories of suicide have been proposed over the last century. These include biological (e.g., Oquendo et al., 2014) and sociological approaches (e.g., Durkheim, 1897), and psychological theories that conceptualize suicide as a phenomenon related to the following: psychache (Shneidman, 1993); escape from aversive self-awareness (Baumeister, 1990); hopelessness (e.g., Beck, Steer, Kovacs, & Garrison, 1985); emotion dysregulation (Linehan, 1993); perceived burdensomeness, thwarted belongingness, and capability for suicide (Joiner, 2005; Van Orden et al., 2010); defeat, entrapment, and low social support (Williams, 2001); various diathesis-stress models (e.g., Mann, Waternaux, Haas, & Malone, 1999; O'Connor, 2011; Wenzel & Beck, 2008); and "ideation to action" frameworks (see Nock, Kessler, & Franklin, 2016 for a discussion), among several others. Each of these approaches is actively researched, with several relevant publications each year.

Such diversity is healthy for a young field, but may not be a good sign for the suicide research field, which has been around since at least Durkheim (1897). In many other fields, a broad set of early theories gives way to a dominant paradigm (or a small set of paradigms) that eventually shifts to new paradigm to account for anomalous findings (see Kuhn, 1962/2012). The current theoretical diversity of the suicide research field means that it is still in a preparadigmatic phase. Each STB theory specifies a unique set of risk factors (or specifies a unique relation among a set of risk factors) that drive STBs; each of these theories (and by extension, each set of risk factors) cannot completely explain STBs. It is therefore likely that some of these theories are largely inaccurate, others are partially accurate, and still others may only apply to specific populations or situations. For the field to progress to a paradigmatic phase, empirical data must be employed to winnow the accurate theories or accurate theory elements from the less accurate theories. The present meta-analysis can facilitate this winnowing process by: (a) determining whether the risk factor data necessary for theory evaluation exists; and (b) if so, testing the existing risk factor literature to ascertain which theories or theory elements are most promising.

Risk Factors and Prediction

As described in more detail in the next section, STB risk factors are essentially longitudinal predictors of STBs. Each day, thousands of health care professionals are tasked with predicting whether their patients will engage in STBs in the future (especially the near future). In addition, many nonprofessionals may encounter friends or family members who may seem to be at elevated risk for STBs. The primary aim of most studies in the STB risk factor literature is to improve the prediction of STBs by identifying a predictor (or set of predictors) that professionals and nonprofessionals can use to detect STB risk. Many studies conclude that they have identified strong STB risk factors that should inform STB prediction and treatment.

For example, Beck, Brown, and Steer (1989) concluded that,

> The results of the present study . . . extend the evidence for the importance of clinical ratings of hopelessness for the prediction of eventual suicide. Furthermore, because hopelessness can be reduced fairly rapidly by specific therapeutic interventions, the assessment of hopelessness can potentially improve the prevention as well as the prediction of suicide. (p. 310)

Likewise, Coryell and Schlesser (2001) suggested that, "In efforts to predict and prevent suicidal behavior in patients with major depressive disorder, HPA-axis hyperactivity, as reflected in DST results, may provide a tool that is considerably more powerful than the clinical predictors currently in use" (p. 748). Similarly, Chronis-Tuscano et al. (2010) noted,

> All subtypes of ADHD in young children robustly predict adolescent depression and/or suicide attempt 5 to 13 years later. . . . Identifying high-risk young children with ADHD sets the stage for early prevention trials to reduce risk for later depression and suicidal behavior. (p. 1044)

To aid both professionals and nonprofessionals in accurately identifying STB risk, several organizations have summarized such information from the STB risk factor literature to produce risk factor guidelines (see Table 1). These guidelines typically include lists of both risk factors and "warning signs" for STBs, and usually conceptualize risk factors as more distal risk indicators and warning signs as more proximal risk indicators (cf. Rudd et al., 2006). However, there is a substantial overlap between these categories, with some organizations listing "multiple warning signs" as a risk factor (see Table 1). Consistent with more general taxonomies of risk (Kraemer et al., 1997; see next section), the present meta-analysis conceptualizes both "risk factors" and "warning signs" as risk factors.

Although these guidelines are likely helpful, there is much room for improvement. Most of these guidelines are long lists of relatively nonspecific factors (see Table 1). Taken together, these guidelines indicate that any individual with nearly any type of mental illness (i.e., internalizing, externalizing, psychotic, or personality disorder symptoms), serious or chronic physical illness, life stress (e.g., social, occupational, or legal problem), special population status (e.g., migrant, prisoner, nonheterosexual), or access to lethal means (e.g., firearms, drugs, high places) may be at risk for STBs. A large proportion of the population possesses at least one of these factors at any given time, with many people possessing multiple factors. This lack of specificity makes it dif-

Table 1
Lists of Suicide Risk Factors and Warning Signs From Several Health- and Suicide-Focused Organizations

Organization	Risk factors	Warning signs
American Association of Suicidology (Adults) (AAS, 2015)	More than one warning sign; anticipated or actual losses or life stresses (e.g., romantic break-ups, legal problems, academic failures); prior suicide attempts	Increased substance use; no reason or purpose for living; anxiety, agitation, or sleep problems; feeling trapped; hopelessness; social withdrawal; rage and anger; reckless or risky behaviors; dramatic mood changes
American Association of Suicidology (Youths) (AAS, 2015)	More than one warning sign; new or increased warning signs; actual or anticipated losses or life stresses	Talking about or planning suicide; hopelessness; severe or overwhelming emotional pain/distress; worrisome behaviors or marked changes in behaviors (e.g., social withdrawal; changes in sleep; anger or hostility; increased agitation or irritability)
American Foundation for Suicide Prevention (AFSP, 2015)	Depression; bipolar disorder; schizophrenia; borderline or antisocial personality disorders; conduct disorder; psychotic disorders or symptoms; substance abuse disorders; serious or chronic physical health problems; exposure to others' suicide; access to lethal means; stressful life events; family history of suicide or mental health problems; childhood abuse; prior suicide attempt	Talking about suicide, having no reason to live, being a burden, feeling trapped, or unbearable pain; increased substance use; planning suicide; acting recklessly; social withdrawal; sleep problems; saying goodbye to people; giving away prized possessions; aggression; depression; loss of interest; rage and irritability; anxiety; humiliation
Centers for Disease Control (CDC, 2015)	Family history of suicide or childhood maltreatment; prior suicide attempt; mental disorders (especially depression); substance abuse; hopelessness; impulsive or aggressive tendencies; cultural or religious beliefs that suicide is noble; local suicide epidemics; social isolation; barriers to mental health treatment; loss; physical illness; access to lethal means; unwillingness to seek help due to stigma concerns	Talking about hurting oneself; increased substance use; changes in mood, diet, or sleeping patterns
National Institute of Mental Health (NIMH, 2015)	Mental disorders (especially depression and substance abuse); prior suicide attempt; family history of mental disorder, substance abuse, suicide, violence, or physical/sexual abuse; guns or firearms in the home; incarceration; being exposed to others' suicidal behaviors	Threatening or talking about killing oneself; planning suicide; hopelessness; rage or anger; reckless behaviors; feeling trapped; increased substance abuse; social withdrawal; anxiety, agitation, or sleep problems; dramatic mood changes; lack of purpose in life
World Health Organization (WHO, 2015)	Mental disorders (especially depression and substance abuse); moments of crisis; chronic pain and illness; conflict, disaster, violence, abuse, and loss; being a refugee, migrant, indigenous person, prisoner, or non-heterosexual person; prior suicide attempt	None noted

Note. Warning signs are usually conceptualized as proximal indicators of suicide risk; risk factors are sometimes conceptualized as more distal indicators, but may also be proximal. Consistent with more general taxonomies of risk (e.g., Kraemer et al., 1997), the present meta-analysis conceptualizes both "risk factors" and "warning signs" as risk factors.

ficult for professionals and nonprofessionals to accurately predict who will engage in future STBs. Moreover, there is some inconsistency across guidelines from different organizations, making it unclear which guidelines should be followed. For example, some organizations emphasize the importance of both depression and substance abuse, others only emphasize depression, and still others do not mention depression. Similarly, the World Health Organization (2015) advises that, "by far the strongest risk factor for suicide is a previous suicide attempt," but no other organizations make a similar claim.

All of these guidelines were developed primarily from expert recommendations based on qualitative reviews of the STB risk factor literature. Because of this literature's size and many conflicting findings, there are likely to be many inconsistencies across qualitative expert reviews. The present meta-analysis may be able to provide a quantitative basis for STB guidelines. This could establish a standard set of guidelines, inform whether there should be separate guidelines for different populations (e.g., adults vs.

youths), and provide information about how to best distinguish between distal and proximal predictors (i.e., "risk factors" vs. "warning signs").

Risk Factors and Treatment

A large number of treatment approaches have been applied to STBs; unfortunately, few of these have been shown to consistently reduce STBs relative to a control group (for recent reviews, see: Brown & Jager-Hyman, 2014; Glenn, Franklin, & Nock, 2015; Ward-Ciesielski & Linehan, 2014). As with many mental illness treatments, STB treatments have not grown out of a large corpus of empirical data on the causal risk factors that drive these thoughts and behaviors. Rather, these treatments coalesced from clinical experiences, originated from a particular STB theory, or were developed for a separate phenomenon (e.g., depression) but applied to STBs. By determining which risk factors are most promising, the present meta-analysis may help to provide an em-

pirical foundation for STB treatments. In other words, extant STB treatment targets were derived from a top-down approach; the present meta-analysis may provide the basis for a bottom-up approach to identifying STB treatment targets.

After 50 years of STB risk factor research and little progress toward the field's most important goal—the reduction of STBs on a large scale—a comprehensive review of this literature would be helpful. Such a review would serve to summarize how this field has progressed, identify crucial gaps in knowledge, and establish the best way forward for future research. To address this need, the primary purpose of the present paper was to metaanalyze existing STB risk factor studies. To ensure clarity, we will start by defining what we mean by the term "risk factor."

What Are Risk Factors?

As in many other fields (see Kraemer et al., 1997), the STB field has often been inconsistent and imprecise with how it has used terms such as correlate, risk, and risk factor. Confusion about these terms can lead to miscommunication between researchers, unfounded assumptions about how suicidal behavior works and how to best predict it, and misguided research, treatment, and policy decisions about suicide. To avoid such confusion in the present meta-analysis, we first note how we defined these terms.

We subscribe to the risk factor typology described by Kraemer et al. (1997). According to this typology, a *correlate* is a factor that is associated with another factor. Correlates can have a range of associations with a factor, and specific types of research designs are necessary to determine the specific nature of the association. For example, if a study found that people who attempted suicide tended to display depression symptoms, this could indicate that depression symptoms are a correlate of suicide attempts; however, it would be unclear how or why depression symptoms and suicide attempts were correlated.

A *risk factor* is a special type of correlate that precedes the outcome of interest and can be used to divide the population into high- and low-risk groups. Continuing the example, if people with depression symptoms (at Time 1) were more likely than others to attempt suicide (at Time 2), depression symptoms would be a risk factor for suicide attempts. Cross-sectional studies are sufficient to establish correlates or concomitants, but longitudinal studies are necessary to identify risk factors. Within our example, if thousands of cross-sectional studies showed that depression was a strong correlate of suicide attempts, it would be tempting to consider depression a powerful risk factor and to make this factor a centerpiece of theories, risk assessments, and treatments. But without evidence from longitudinal studies, there would be no empirical justification for considering depression a risk factor or for integrating this factor into theory and practice. As such, the present meta-analysis included only longitudinal studies.

A *causal risk factor* is a special type of risk factor that is identified when the manipulation of a risk factor systematically changes the probability of the outcome of interest. Further continuing our example, if experimentally decreasing (or increasing) depression symptoms at Time 1 altered the probability of suicide attempt at Time 2, depression symptoms could be considered a causal risk factor. By this definition, there is very little existing research on the causal risk factors for STBs. As a result, the present meta-analysis will be most helpful for distinguishing STB risk factors from well-established STB

concomitants. Promising risk factors may then be tested as possible causal risk factors in future studies. The distinction between these three terms is crucial because causal risk factors are predictors and valuable treatment targets; noncausal risk factors are predictors, but less effective treatment targets; and correlates/concomitants may be poor predictors and ineffective treatment targets. Mistaking one type for another can have major consequences for STB theory, research, and practice.

Kraemer et al. (1997) also argued that it is important to evaluate risk factors based on clinical significance rather than statistical significance. Many studies in the STB risk factor literature include thousands or even millions of participants; in these instances, it is difficult *not* to detect a statistically significant effect. There are no widely accepted objective criteria for assessing the clinical significance of a risk factor, but one potentially helpful guideline is to estimate how the magnitude of a given risk factor might translate into population-level risk. For example, approximately 20 of every 100 people in the United States are annually infected with influenza (CDC, 2015). If a particular risk factor (e.g., age, location) tripled the risk of infection, many people may consider it clinically significant because it would signify that an individual with this characteristic would be more likely than not to develop the infection.

Carrying this idea forward to suicide risk, approximately 0.013 of every 100 people in the United States died by suicide in 2013 (CDC, 2014). In contrast to the influenza example, many people may not consider a factor that tripled suicide risk to be clinically significant because it would only increase this rate to 0.039 per 100 people. In other words, even if someone possessed a characteristic associated with a 300% increase in the 1-year likelihood of suicide death, in absolute terms they would still have a near-zero risk of dying by suicide that year. Given the higher base rates for suicide attempts (approximately 0.33 per 100 people) and suicide ideation (approximately 2 per 100 people), a threefold risk would be more meaningful—but still weak—for these less severe outcomes.

Any attempts to establish specific criteria for a clinical significance of STB risk factors would be highly subjective. In the present paper, we will estimate how the present findings might translate into 1-year population-level risk for STBs and leave it to the individual reader to determine subjective clinical significance. As very rough guidelines based on epidemiological data and large national studies (Borges et al., 2008; CDC, 2014; Crosby et al., 2011; Kessler et al., 2005), we estimate here that to increase the 1-year risk of a given outcome to 10%, a factor would need to approximate an odds ratio of 750 for suicide death, 30 for suicide attempt, and 5 for suicide ideation. Although it is important for clinicians to be aware of a ~10% risk of suicidal behavior over a long period of time (e.g., a year, decade, or lifetime), clinicians are often asked to determine whether a given individual has a greater than 50% chance of suicide death or attempt over the course of a day, week, or month. To meet such criteria, odds ratios in the present meta-analysis would need to be in the hundreds or thousands.

The Present Meta-Analysis

A meta-analysis of the STB literature is necessary because it is difficult for a single study to provide an accurate estimate of the true magnitude of a risk factor. There is often a wide variation in effect magnitude across studies. For instance, many studies have found that depression is a relatively strong STB risk factor (odds

ratios > 10.0; e.g., Kishi & Robinson, 1996; Wolk, Weissman, & Puig, 1996) but several others have found that depression is not a risk factor (odds ratios < 1.0; e.g., Beck, Steer, & Trexler, 1989; May, Klonsky, & Klein, 2012). A meta-analysis can reconcile such divergent findings. Similarly, within a narrative review it is difficult to make sense of findings from studies with a wide range of methods and sample types. A meta-analysis can quantify the effects of these variations on risk factor magnitude. Accordingly, we conducted the present meta-analysis to address several broad questions about STB risk factors.

First, what are the basic characteristics of the STB risk factor literature? We examined a range of characteristics, including the following: (a) the number of papers and risk factor tests across the decades of suicide research; (b) characteristics of study samples such as psychopathology severity, age, and number of STB participants; (c) the frequency of various broad risk factor categories; (d) the frequency of outcome types (i.e., ideation, plans, attempts, deaths); (e) variation in follow-up length across studies; (f) the overall predictive ability of the literature (measured in terms of odds ratios, hazard ratios, and diagnostic accuracy statistics); (g) potential publication bias; and (h) study quality based on factors like retention rates. This information may reveal patterns and gaps in the literature that help to establish important future directions for the field.

Second, has predictive ability (i.e., risk factor magnitude and accuracy) improved across the decades of suicide research—why or why not? We hypothesized that suicide risk factor research has been progressive—later studies have built on earlier studies, knowledge has accrued over time, and the ability to predict STBs has steadily improved. If this hypothesis is supported, we will examine which factors appear to be most promising and attempt to gain insight into how the field might accelerate this rate of improvement. But if predictive ability has not improved, we will attempt to determine why this has been the case and what might be done to place the field on a more progressive path.

Third, does predictive ability vary by outcome type? One possibility is that higher base rates translate into better prediction, with risk factor magnitude and accuracy improving from suicide death to suicide attempts to suicide ideation. Yet it is also possible that more severe outcomes (i.e., suicide death) are associated with more distinctive risk factors, making it easier to predict more severe outcomes. Related to this question, we were curious whether outcome characteristics such as definition and assessment strategy affected predictive ability. It is possible that more stringent definitions (e.g., explicit inclusion of "suicide intent" for suicide attempts) and more thorough assessments improve predictive ability. It could also be that such outcome characteristics improve the reliability and validity of results but have little bearing on the strength of prediction.

Fourth, are longer follow-up intervals associated with improved predictive ability? Follow-up intervals in this literature range from several days (e.g., one week in Britton, Ilgen, Rudd, & Conner, 2012) to several decades (e.g., 76 years in Lahti et al., 2015). Given the low base rates of STBs, it is possible that longer follow-up intervals provide stronger prediction because they allow for more events to accumulate. But it is also possible that many factors only confer increased risk for STBs for a short period of time, such that prediction is more accurate over shorter intervals.

For example, losing one's job may sharply increase risk of STBs for a few days or weeks, but not for years or decades. Whatever the pattern of results, these findings may help to establish empirical guidelines for the follow-up intervals of future studies.

Fifth, does predictive ability vary by sample characteristics? In the present meta-analysis, we analyzed sample types in three general ways: sample severity (general vs. clinical vs. prior STB sample reference groups); sample age (adolescent vs. mixed vs. adult samples); and sample size (number of STB participants). In terms of sample severity, we hypothesized that predictive ability would improve as the sample reference group became less severe. This is because differences between participants with STB outcomes and participants without those outcomes (i.e., reference participants) increase as samples become less severe. In effect, general community sample studies represent "extreme groups" designs that may generate larger effect sizes. We further examined the potential impact of clinical sample type (e.g., depression, psychosis), STB sample type (e.g., ideation, attempt), and clinical/STB sample origin (e.g., inpatient, community) on predictive ability. Regarding sample age, it was unclear whether predictive ability varies across adult, mixed, and adolescent samples. Within sample size analyses, we hypothesized that a larger number of participants with a history of STBs would allow for a larger number of observed STBs and would produce more reliable prediction. But it was unclear whether this more reliable prediction would translate into stronger or weaker prediction.

Sixth, we had several questions about broad STB risk factors categories (see Table 2 for a list of these categories). Some questions centered on the composition of these risk factors—has the literature tested a diverse array of risk factors or settled on a narrow set of factors? Have these risk factors become more heterogeneous or homogeneous over time, or have these factors clearly shifted along with the introduction of new theories? Other questions concerned the strength of risk factors—do any risk factors stand out as particularly strong or weak? Are the strongest risk factors the ones that have received the most attention from researchers, or has there been a discordance between risk factor strength and popularity? A final set of questions were related to the possibility that each STB outcome may be associated with a unique set of risk factors. Researchers have long been interested in such questions, particularly the possibility that certain factors may predict the *transition* from suicide ideation to suicide attempt or death (e.g., Kessler et al., 1999). Because of the methodological limitations of most existing studies, the present meta-analysis was unable to directly address questions about such transitions; however, we examined whether predictive ability differed by outcome and whether any broad categories or subcategories of risk factors were uniquely associated with an outcome.

Seventh, is there any evidence for protective factors within this literature (i.e., broadly, factors that are associated with fewer STBs)? Many fewer studies have focused on this important complement to risk factors. The present meta-analysis examined the basic descriptive features of protective factors within this literature and estimated the magnitude of protective factor effects.

Through addressing these questions, the present meta-analysis has the potential to provide new insight into STBs and to map out important future directions for research.

Table 2

Broad Risk and Protective Factor Categories and Examples

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1. Biology
Examples: CSF metabolites; dexamethasone suppression test; genes; hormones; peripheral physiology
2. Screeners
Examples: specific STB screening instruments; clinician prediction; patient prediction
3. Cognitive problems (and cognitive abilities)
Examples: cognitive difficulties; intelligence; mental state; problem-solving ability; school performance
4. Demographics
Examples: age; education; employment; ethnicity; gender; marital status; religion; socioeconomic status
5. Externalizing psychopathology
Examples: aggressive behaviors; impulsivity; incarceration history; antisocial behaviors; substance abuse
6. Family history of psychopathology or self-injurious behaviors
Examples: maternal depression; paternal alcoholism; first-degree relative suicide attempt
7. General psychopathology
Examples: presence or number of psychiatric symptoms; presence or number of psychiatric diagnoses
8. Implicit/explicit processes
Examples: implicit/explicit attentional bias; implicit/explicit identification
9. Internalizing Psychopathology
Examples: anxiety disorders; mood disorders; hopelessness; emotion dysregulation; sleep disturbances
10. Normative personality traits
Examples: type A personality; extraversion; openness; assertiveness; masculinity; agreeableness
11. Physical illness (and physical health/characteristics)
Examples: height; weight; asthma; heart disease; cancer; allergies; migraines; physical disability
12. Psychosis
Examples: schizophrenia; positive, negative, or disorganized psychotic symptoms
13. Prior self-injurious thoughts or behaviors
Examples: prior deliberate self-harm, nonsuicidal self-injury, suicide attempt, suicide ideation
14. Exposure to self-injurious thoughts and behavior
Examples: suicide attempt or completion by a friend, schoolmate, or acquaintance
15. Social factors
Examples: abuse history; family problems; isolation; peer problems; stressful life events
16. Treatment history
Examples: prior psychiatric assessment, hospitalization, or treatment

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Method

Inclusion and Exclusion Criteria

The aim of the present meta-analysis was to provide a broad overview of risk factors for suicidal thoughts and behaviors. All potentially eligible studies were examined for risk (or protective) factor tests (hereafter, "effect sizes"). We define an effect size as any instance where a particular factor was used to longitudinally predict a relevant outcome; as described below, most studies included multiple effect sizes (i.e., examined several risk or protective factors within the same study). The following criteria were established to select relevant effect sizes.

Language.   Only English language articles were included.

Outcome specificity.   Eligible effect sizes had to predict a specific form of suicidal thought or behavior; namely, suicide ideation, suicide plan, suicide attempt, or suicide death. This criterion was necessary because we aimed to provide a meta-analysis of all suicide-related outcomes (vs. potentially nonsuicidal) and because there are important ontological and empirical distinctions among specific STBs (see Nock, 2010). Effect sizes predicting outcomes such as nonsuicidal self-injury, deliberate self-harm, and mixed outcomes (e.g., accidental and suicide death) were excluded. However, these latter factors were eligible to be included as predictors.

Outcome specificity was complicated by the tendency for different researchers use different criteria to determine what did and did not qualify as a specific type of suicidal thought or behavior. These variations reflect the fact that, as with most other psychological constructs, there are no universally accepted definitions of the specific forms of suicidal thoughts and behaviors. To account for variations in definitions across studies, we coded several features of each outcome for each effect size (see below) and tested whether these variations impacted results. We excluded studies that clearly were not consistent with general definitions of suicidal thoughts and behaviors, which we operationalized in accordance with definitions of Nock et al. (2010, p. 342). Specifically, suicide ideation was defined as "thoughts of killing oneself"; suicide plan as "consideration of a specific method through which a person intends to kill oneself"; suicide attempt as "engagement in potentially self-injurious behavior in which there is some intent to die from the behavior"; and suicide death as "death that results from intentionally self-injurious behavior."

Slight variations on these definitions were allowed (but coded and tested for their impact on results); however, many definitions clearly assessed qualitatively different phenomena and were excluded. For example, several studies were excluded because they included deaths classified as "undetermined" as suicide deaths.

Longitudinal prediction.   Because risk factors must precede outcomes (Kraemer et al., 1997), only effect sizes that longitudinally predicted a relevant outcome were included. Most qualifying studies included longitudinal designs (i.e., measuring the risk factor before the outcome), but a few studies were able to establish longitudinal associations from cross-sectional designs (e.g., associations between serotonin transporter polymorphisms and suicide attempts; Bellivier et al., 2000).

Nontreatment studies.   Because we did not aim to provide a meta-analysis of STB treatments and because treatment effects may influence risk factor effects, we excluded effect sizes that occurred within studies where treatment was the primary aim. Predictors relevant to prior treatment history in a general sense (rather than within a specific study that tested a particular intervention) were included.

Effect size independence.   The present meta-analytic strategy assumes that all effect sizes are independent of one another. However, because many studies produced multiple effect sizes and some of the predictor variables for these effect sizes may be correlated (e.g., measures of depression and anxiety), it is likely that there is moderate dependence among some effect sizes in this meta-analysis. Simulation studies indicate that not accounting for dependence when it exists has a minimal impact on pooled effect estimates, but leads to slight underestimations of variance and confidence intervals, thereby increasing Type I error (e.g., Thompson & Becker, 2014). The best way to model dependence is to account for the covariance structure in each study; unfortunately,

this level of information is rarely presented in research articles. Fortunately, several other strategies for handling this issue exist. In the present meta-analysis we took the following steps to reduce dependence and to model the potential effects of dependence.

First, to reduce dependence and avoid redundancy, we excluded any effect sizes that were published two or more times. This occurred when the same effect sizes from the same dataset were reanalyzed across multiple publications. Such instances were easy to detect when data sets were from large studies such as the National Longitudinal Study of Adolescent Health ($n = 12$ included studies) and the Baltimore Epidemiologic Catchment Area study ($n = 7$ included studies). Repeated dataset use was more difficult to detect in smaller studies because these papers often did not note when prior papers had been published on the same or partially overlapping dataset. Notably, this exclusion criterion only applied to *effect sizes* reported multiple times across multiple publications. This means that the same dataset could produce multiple publications that met inclusion criteria for the present meta-analysis if each publication included unique effect sizes. Second, several studies also reported the same effect sizes across multiple time points. In these instances, we only included the effect sizes from the farthest time point because it was the most inclusive. We note here that patterns and statistical significance were the same when other time points or all time points were included in analyses.

Third, to model the potential effects of dependence in this meta-analysis, we conducted analyses that assumed and accounted for complete dependence among effect sizes within each study. Specifically, we averaged all effect sizes within each study (see Scammacca, Roberts, & Stuebing, 2014). Although these analyses assume far greater dependence than likely exists in this meta-analysis, they are useful in that they provides an extreme upper-bound of the potential effects of dependence on our results. Given space limitations and the minimal differences between estimates produced by this method and our primary method, we only present results for overall pooled estimates for each outcome.

Main effects tests. We had hoped to include information about interactions among risk factors, but—precluding reliable meta-analyses—these tests were both rare and idiosyncratic (i.e., the same interaction was seldom examined in more than one study). As a result, effect sizes that directly tested interaction effects were not included in the present meta-analysis. We note here that these interaction effects typically produced magnitudes that were similar to main effects. We had also hoped to examine differences between static risk factors (i.e., factors assessed at one moment in time) and time-varying risk factors (i.e., factors that change during a study). However, only two qualifying studies included time-varying risk factors ($n = 4$ risk factor effect sizes; Keilp et al., 2010; Morrison & O'Connor, 2008), both of which examined change on a measure before and after an experimental manipulation. This low number of time-varying effect sizes is attributable in part to the fact that time-varying factors are usually most appropriately modeled with advanced statistics (e.g., latent growth curve modeling), especially across longer time periods. Unfortunately, as described below, these statistics could not be converted into one of the common metrics used in the present meta-analysis. This meant the exclusion of a few studies that examined time-varying risk factors. However, the major reason that the present meta-analysis does not include a large number of

time-varying effect sizes, effect sizes from combinations of risk factor, and effect sizes from interactions is that the vast majority of existing STB risk studies have tested static factors in isolation.

Necessary statistical and design information. The primary metrics for the present meta-analysis were odds ratios (i.e., relative odds of an event) and hazard ratios (i.e., relative frequency of an event over a certain time interval). Because most statistics can be converted into an odds ratio (see Meta-Analytic Methods section below), we were able to include effect sizes from a wide range of statistical tests. However, inclusion was not possible when an index of variance was not provided (e.g., beta weights with no additional information), certain statistical tests were used (e.g., standardized mortality ratio; latent growth curve modeling), or with certain design features (e.g., unclear or nonexistent comparison group or condition; qualitative outcome descriptions; unclear if STB outcome was assessed as a lifetime variable or *since baseline* variable). Effect sizes with these features were excluded. Another exception to odds ratio conversion was hazard ratios, but because 20.45% of effect sizes were reported as hazard ratios, we elected to include hazard ratio analyses in addition to odds ratio analyses.

Published in print or online by January 1, 2015. We were primarily interested in the available scientific information on STB risk factors that researchers and clinicians use to inform theory and practice. As such, we only included published studies through January 1, 2015 (the earliest qualifying article was published in December 1965). We calculated several publication bias estimates to quantify and correct for this bias. As indicated by publication bias correction statistics (see Results below), the true risk factor effects were likely weaker than the published literature indicates. In other words, the present results should be regarded as slightly optimistic estimates of risk factor magnitude and accuracy.

Literature Search, Papers, and Prediction Effect Sizes

We conducted literature searches across PubMed, PsycInfo, Google Scholar, and the reference sections of all papers included in the present meta-analysis. Searches included permutations of several terms related to the words "longitudinal" and "suicidal thoughts and behavior." These terms included: "longitudinal," "longitudinally," "predicts," "prediction," "prospective," "prospectively," "future," "later," "follow-up," "followed," "risk factor," "risk," "protective factor," "protective," "protect," "genes," "genetic," "self-injury," "suicidality," "self-harm," "suicide," "suicidal behavior," "suicide attempt," "suicide death," "suicide plan," "suicidal thoughts," "suicide ideation," "suicide gesture," "suicide threat," "SITB," "parasuicide," "self-mutilation," "self-cutting," "cutting," "self-burning," "self-poisoning," "deliberate self-harm" (DSH), "DSH," "nonsuicidal self-injury" (NSSI), and "NSSI." As shown in Figure 1, these searchers ultimately yielded 4,084 total effect sizes (after removal of outliers and redundant effect sizes) from 365 studies that were included in the present meta-analysis.

Data Extraction and Coding

Data extraction. Each paper was examined for relevant effect sizes. Each effect size that met inclusion/exclusion criteria was coded on all dimensions noted below. Codes were developed by the lead author in consultation with coauthors. Codes were com-
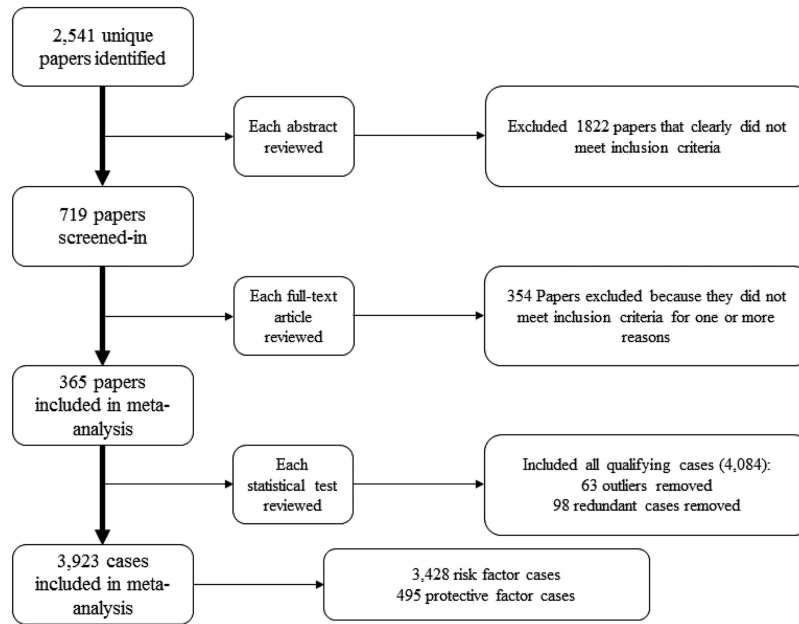
Figure 1. PRISMA diagram for the selection of studies and effect sizes for the present meta-analysis.

pleted via an iterative process. The initial version of each code was completed by one of the authors with an advanced degree in psychology (i.e., masters- or doctoral-level). Each code was then independently checked for accuracy by two additional authors with advanced degrees in psychology. Any discrepancies were discussed and resolved such that all three authors agreed on the coding decision. All authors then reviewed each code; any discrepancies were discussed and resolved, with all authors agreeing on the final versions of each code for each effect size.

Author, year, and era codes. The author and year of publication were recorded for each effect size. Era of research was coded in three separate ways: (a) actual year of publication; (b) 5-year intervals (starting with 2010–2014 and working backward to 2005–2009, 2000–2004, etc. until pre-1980 effect sizes, which were all included as a single interval due to few effect sizes in this era); and (c) 10-year intervals (starting with 2005–2014, and working backward to 1995–2004, 1985–1994, and pre-1985 effect sizes, which were coded as a single interval due the scarcity of effect sizes during this era). We ran analyses across all three coding schemes, but we elected to present only the 10-year interval findings because they provided the clearest depiction of results. We note here that patterns and statistical significance were the same across all three codes.

Follow-up length codes. Follow-up length was coded in terms of the months of follow-up. When studies listed variable follow-ups, we included the longest-noted follow-up interval. Due to the skew of follow-up lengths, we divided these into six class intervals designed to categorize follow-up lengths into very short, short, medium, long, very long, and extremely long intervals. For ease of interpretation, these intervals are described in more detail in the Results section below. We conducted analyses across both raw and class interval coding schemes, but only present class interval code results. As with era of research analyses, there were no differences in terms of patterns or statistical significance across

the two codes, and the class interval code permitted clearer presentation of the data.

Sample severity code. Sample severity was coded based on the nature of the reference group for individuals with STB-related outcomes for a given effect size. If members of a reference group were selected on the basis of a prior self-injurious thought or behavior history, the sample was coded as "STB." When at least some of the members of a reference group were selected on the basis of psychopathology (and the "STB" sample criterion was not met), the sample was coded as "clinical." For instances where neither of these criteria was met, the sample was coded as "general."

Clinical sample type code. It is possible that the type of clinical sample influences risk factor magnitude. We accordingly coded each sample based on the primary type of diagnosis or symptoms for which participants were selected. Each sample with any participants selected for psychopathology were coded as one the following: (a) general psychiatric sample (i.e., multiple primary diagnoses or symptoms); (b) mood and anxiety disorders; (c) substance use disorders; (d) psychotic disorders; (e) eating disorders; (f) attention-deficit/hyperactivity disorder; (g) borderline personality disorder; or (h) other personality disorders.

STB sample type code. It is also possible that the type of prior self-injurious thought and behavior influences risk factor magnitude. To test this possibility, we coded all samples with any participants selected for STBs as one of the following: (a) general suicidality and self-injury (e.g., parasuicide, deliberate self-harm); (b) prior suicide ideation; (c) prior suicide attempt; or (d) suicide death (for studies that selected suicide decedents for study post hoc).

Clinical and STB sample origin. These two sample types were coded based on the locations from which participants were recruited. Specifically, samples were coded as inpatient, outpatient, mixed inpatient and outpatient, or community.

Sample age code. Sample age was coded based on the age range of the sample at the baseline time point of the effect size. When all participants were below age 18, the sample was coded as "adolescent," and when all participants were 18 and above, the sample was coded as "adult." In cases where participants were both above and below 18, the sample was coded as "mixed."

STB sample size code. Sample size is a potentially important variable because larger studies typically record more STBs and accordingly provide more precise and reliable results. However, there were several population level studies (with millions of participants) that recorded a very low proportion of STB-related events and several smaller studies that recorded an extremely high proportion of STB-related events. In other words, the number of participants with STB-related events at any point during the study was the most relevant aspect of sample size for the present meta-analysis. We present analyses based on this STB-related sample size, but note here that analyses based on overall sample size provided very similar results.

General outcome code. Outcomes were coded as one of the following categories: suicide ideation, suicide plan, suicide attempt, or suicide death. As described above, effect sizes that could not be classified cleanly into one of these categories were excluded. In addition to this code, we additionally included codes that assessed outcome characteristics including the nature of the outcome definition, outcome assessment type, and whether suicide attempt outcomes were initial or repeated attempts (see below).

Outcome definition codes. The goal of these codes was to account for variations in acceptable outcome definitions. We employed a slightly different code for each outcome.

For suicide ideation outcomes, we were particularly concerned about differentiating between passive and active ideation. Moreover, many common suicide ideation outcome measures include a mix of passive and active suicide ideation items. We coded suicide ideation outcomes as (a) definition not provided, (b) passive, (c) mixed passive and active, or (d) active. For suicide attempt outcomes, we were primarily interested in assessing suicide intent. As noted above, studies were excluded if their attempt definitions clearly indicated that intent was not necessary. For the included studies, suicide attempt outcomes were coded as (a) definition not provided, (b) intent implied but not explicitly stated, or (c) intent explicitly stated. For suicide death outcomes, we coded whether studies only included suicide deaths within their suicide death outcomes. As noted above, several studies (primarily large European studies) were excluded because they explicitly mixed suicide deaths and undetermined deaths (i.e., explicitly noted that outcomes were ICD codes for suicide death and death from accidental/undetermined causes). Within included studies, we coded suicide death outcomes as (a) definition not provided; (b) did not explicitly include or exclude ambiguous deaths; and (c) explicitly excluded ambiguous deaths.

Outcome assessment codes. There are many different ways to assess STBs, and it is currently unclear if the type of assessment impacts risk factor magnitude. For suicide ideation and attempt outcomes, assessments were coded as (a) assessment type not provided, (b) single-item assessment, or (c) multi-item questionnaire/interview. For suicide death outcomes, assessments were coded as (a) assessment type not provided, (b) family report, or (c) official legal/medical report (e.g., death certificate).

Suicide attempt repetition outcome code. It is possible that risk factors for an initial suicide attempt differ from risk factors for subsequent attempts. We accordingly coded suicide attempt outcomes as (a) unclear if attempts were initial or repeated; (b) repeated attempts; (c) mix of initial and repeated attempts; and (d) initial attempts.

Risk and protective factor codes. The majority of predictors could be reasonably hypothesized to increase risk for future suicidal thoughts or behaviors (i.e., risk factors), but there were several exceptions and complications to this general rule that required attention. A smaller proportion of predictors met our broad definition of a protective factor. A few predictors met classic definitions of protection or resilience (e.g., high omega-3 intake), but many others simply indicated the absence of risk (e.g., "no psychopathology"), a relatively low degree of risk (e.g., rarely drinks alcohol vs. drinks alcohol daily), or potentially lower risk in certain circumstances (e.g., male gender). We included all of these types of effect sizes within a broadly defined category of protective factors.

Categorization was complex for effect sizes that were associated with risk in certain circumstances and (broadly defined) protection in others. For example, suicide attempts are not associated with age in a clear linear pattern—attempts peak in late adolescence and early adulthood (Nock et al., 2008). Complicating matters, there was high heterogeneity among age grouping across studies, with some studies using age as a continuous variable and many others comparing idiosyncratic age group intervals (e.g., 18–24) to other idiosyncratic age group intervals (e.g., 55–64). In all such instances (not just with age), we drew on the best available information to determine whether the reference group would be expected to possess greater or lesser risk for a specific suicide-related outcome (e.g., CDC, 2014; Kessler et al., 2005; Nock et al., 2008). If the reference group was expected to have lesser risk, the effect size was coded as a risk factor; if the reference group was expected to display greater risk, the effect size was coded as a protective factor. For example, if an 18–24 age group was the reference for a 55–64 age group predictor, the effect size would be coded as a protective factor if suicide attempt was the outcome (fewer attempts would be expected in the 55–64 age group) and a risk factor if suicide death was the outcome (more deaths would be expected in the 55–64 age group).

Broad risk/protective factor category codes. Across the 4,084 total effect sizes, there were thousands of predictor types—too many for the present paper to examine individually or even as moderately specific category types. To permit intelligible analyses, we classified each individual predictor into one of 16 broad categories. Table 2 lists these categories and provides general examples of the types of predictors included within each category. Effect sizes were also classified into much smaller subcategories, but space limitations precluded the full presentation of these analyses. Moreover, in terms of overall risk factor magnitude, these subcategory analyses did not provide much additional information. As described in the Results section below, no subcategory was substantially stronger than any broad risk factor category.

Adjusted and unadjusted estimate code. We attempted to include zero-order effects (i.e., unadjusted effects) for each effect size to provide the purest estimation of effects. This was possible for most effect sizes, but not all. Effect sizes were coded based on whether their effects were adjusted or unadjusted, and analyses

were conducted to examine the effects of statistical adjustment (see below).

Predictor scale code. Odds and hazard ratios determine the change in odds/hazard for a given outcome *per each unit of change in the predictor variable.* For example, if the predictor variable is dichotomous (depression diagnosis vs. no depression diagnosis), there would be one unit of change. If the predictor variable was continuous (e.g., scores on the Beck Depression Inventory), there would be many units of change. These scale differences do not impact the statistical significance of results (because standard errors shift with this scaling), but they do affect the effect size magnitudes. Odds/hazard ratios tend to become smaller as the number of units of change increases because the differences between units becomes less extreme (e.g., the difference between Beck Depression Inventory scores of 16 and 17 vs. the difference between having/not having a depression diagnosis). To account for these statistical artifacts, we coded the scale of each predictor variable as (a) dichotomous or (b) continuous.

Outlier code. Outliers were coded as any values above three standard deviations beyond the unweighted mean of the overall odds ratio magnitude ($uwOR = 2.53$, $SD = 5.01$, cutoff = 17.62; $n = 43$ outliers) or overall hazard ratio magnitude ($uwHR = 2.26$, $SD = 2.78$, cutoff = 10.60; $n = 20$ outliers). These 63 outliers accounted for just 1.54% of all effect sizes. We note here that inclusion of these effect sizes did not affect the general patterns or statistical significance of the reported results.

Study quality. Meta-analyses often code for study quality when they include a high degree of methodological variability (e.g., wide range of study designs and outcomes) that may impact conclusions. Such codes are especially common among meta-analyses of treatments because factors like study design (e.g., single-group, case-control, double-blind), sample type (e.g., severity, representativeness), treatment provision (e.g., fidelity, compliance), and outcome type (e.g., anxiety symptoms, alcohol use) may powerfully influence results. Relative to many other meta-analyses, however, the present meta-analysis included a highly uniform set of studies. All were required to share a common core design (i.e., longitudinal) and all were required to include a specific STB outcome. Of course, smaller methodological differences existed among the present set of studies (e.g., follow-up length, sample severity, sample age, sample size), but there are no objective criteria for assessing how variations among these factors are related to study quality.

Although there are no objective criteria for study quality in this literature, we took four steps to gauge the impact of how various methodological features may impact risk factor magnitude. First, we conducted moderation analyses with all codes noted above to examine how variations in these factors may affect effect sizes. Second, we additionally coded each study for the following: (a) whether the study reported a recruitment rate; (b) whether the study reported information necessary to calculate a retention rate; and (c) the calculated retention rate of the study. Third, we similarly tested the effect of the following codes on risk factor magnitude in a simultaneous multiple meta-regression analysis: (a) study recruitment rate reporting; (b) study retention rate reporting; (c) outcome assessment code (described above); and (d) outcome definition code (described above). Fourth, within a separate meta-regression analyses we tested the effect of the calculated retention rate on effect magnitude. This variable was included in a separate meta-regression because it only included the subset of effect sizes that reported information sufficient to calculate a retention rate.

Meta-Analytic Methods

General strategy. All analyses were conducted in accordance with our research questions. Most of these questions pertained to the general effect of a particular variable (e.g., study length, sample severity, sample age, outcome definition, etc.) on overall risk factor strength. Relevant analyses accordingly tested the effect of a particular moderator (e.g., study length) on the overall pooled estimate across risk factor types. Other questions concerned the general effect of a group of variables related to a particular construct (e.g., study quality) on overall risk factor strength. Analyses for these questions examined the combined effect of multiple variables on the overall pooled estimate across risk factor types within simultaneous meta-regressions. Still other questions focused on variations in risk factor magnitude across broad risk factor categories (e.g., internalizing psychopathology) or specific risk factor types (e.g., depression). We examined these questions by calculating and comparing risk factor magnitudes across broad risk factor categories and specific risk factor types.

Programs. Diagnostic accuracy analyses were conducted with Meta-Disc 1.4 (Zamora, Muriel, & Abraira, 2014), a program designed specifically for meta-analysis of diagnostic accuracy statistics. All other analyses were conducted with Comprehensive Meta-Analysis 3.0 (Borenstein, Hedges, Higgins, & Rothstein, 2014).

Random effects models. The present meta-analysis only used random effects models (see Borenstein, Hedges, Higgin, & Rothstein, 2009). Fixed effects models assume that there is one true effect and that all effect sizes of a given association approximate this effect. This assumption is rarely valid in meta-analyses, leading to inaccurate effect size estimates because of a high degree of heterogeneity between effect sizes due to methodological variations. This heterogeneity is quantified with a statistic called I^2. Guidelines suggest that I^2 values of 0% to 30% indicate low heterogeneity, 31–60% indicate medium heterogeneity, and 61–100% indicate high heterogeneity (Higgins, Thompson, Deeks, & Altman, 2003). To justify the use of random effects models in the present meta-analysis, we provided I^2 for major analyses.

Random effects models estimate a distribution of effects rather than a single true effect, and accomplish this by estimating both between- and within-effect size variance (vs. only within-effect size variance with fixed effects models). As a result, potential between-effect sizes heterogeneity (e.g., population, methods, etc.) is factored into the calculation and weighting of each effect size. Random effects models are recommended over fixed effects models for virtually all types of meta-analyses. Given the high degree of heterogeneity in the present meta-analysis (see Results section), random effects analyses were especially necessary.

Odds and hazard ratios. Odds and hazard ratios were two of the three primary metrics in the present analysis (diagnostic accuracy statistics were the other, see below). These results are presented in terms of weighted odds ratios (*wORs*) or weighted hazard ratios (*wHRs*). Statistical significance occurs for both of these metrics when an accompanying 95% confidence interval does not include 1.0. The meta-analytic programs automatically calculated within- and between-effect size variance to weight each effect size

(see Borenstein et al., 2009). In general, the lower the variance for a particular effect size (i.e., larger studies, studies with more precise or consistent results), the greater the weight it received.

Odds ratios are a ratio of the odds of an event in one group compared with another. For example, the odds of a suicide attempt in a group of females compared with a group of males. Most effect sizes were either reported in terms of odds ratios or converted into odds ratios by the Comprehensive Meta-Analysis program (see Borenstein et al., 2009). Specifically, the following data structures were able to be converted into odds ratios: Pearson product-moment correlations; *t* tests and their variants; Cohen's *d* and its variants; means and standard deviations; chi-squared analyses; and 2 × 2 tables with rates or raw information. Hazard ratios are similar to odds ratios except that hazard ratio calculations integrate information from time intervals within the study. Practically, hazard ratios and odds ratios generally produce similar numbers and interpretations; nevertheless, given the special time-based features of hazard ratios, they cannot be converted into odds ratios.

Diagnostic accuracy analyses. Although the odds and hazard ratio analyses are necessarily relevant to prediction, diagnostic accuracy provides additional dimensions for assessing predictive accuracy. Compared with odds and hazard ratio analyses, these statistics are less vulnerable to misinterpretations due to low base rates. These statistics are most often used in medical science to estimate the accuracy of diagnosing a specific condition (e.g., lung cancer). In the present meta-analysis, diagnostic accuracy refers to the correct 'diagnoses' of STB-related outcomes rather than diagnoses of medical conditions or psychiatric disorders.

Accuracy analyses can illuminate specific strengths or weaknesses in prediction (e.g., low false positive rate, high false negative rate), providing greater clarity for clinical decisions and future research directions. Whenever possible, we obtained the raw data necessary to calculate diagnostic accuracy statistics. A total of 926 effect sizes produced sufficient information for these analyses. Each of these effect sizes was also calculated in terms of odds ratios, so we emphasize that these analyses produced a different dimension of information rather than completely new information. A meta-analysis of this information was conducted with Meta-Disc 1.4, which uses random effects models and weighting procedures similar to Comprehensive Meta-Analysis, but is specialized for diagnostic accuracy statistics.

All diagnostic accuracy analyses are constructed from a 2 × 2 table where the four cells represent true positive, false positive, true negative, and false negative events. From these cells, sensitivity (true positive rate) and specificity (true negative rate) were calculated (Šimundić, 2008; Zamora et al., 2014). From these statistics, area under the curve (AUC) was calculated by plotting each effect size along dimensions of sensitivity and 1 minus specificity. An AUC of .5 indicates chance prediction and an AUC of 1.0 indicates perfect prediction. Guidelines suggest that AUCs above .90 indicate excellent prediction, .80 to .89 good prediction, .70 to .79 fair prediction, .60 to. 69 poor prediction, and .50 to .59 extremely poor prediction (Šimundić, 2008). Weighted AUC (*wAUC*) was the primary metric of diagnostic accuracy evaluation in the present meta-analysis, and weighted sensitivity and specificity were provided for major analyses.

Publication bias. Publication bias estimates were included for overall analyses. Several statistics were calculated to estimate different aspects of bias (see Borenstein et al., 2009 for more information on each of these indices).

Two types of fail-safe *N*s were calculated; these statistics estimate the robustness of an observed effect. First, classic fail-safe *N* analyses determine how many studies with a null effect size (i.e., odds and hazard ratios of 1.0) would be needed to make the meta-analytic effect nonsignificant. However, some have pointed out that this analysis is limited because it relies on statistical significance (which is often a function of power) and null effects (unpublished studies may have odds or hazard ratios lower than 1.0). Second, Orwin's fail-safe *N* addresses these issues by allowing the researcher to set the level of a 'trivial effect' (rather than statistical significance) and the effect size of missing studies (rather than 1.0). There is no objective way to set these levels, but in the present meta-analysis we wanted to ascertain the robustness of results if the true effect had been 1.0 and unpublished findings were the inverse magnitude of published findings. Accordingly, we set the trivial effect size to odds and hazard ratios of 1.0 and the effect size of missing studies to 1.0 minus the effect size above 1.0 of the observed fixed effect (e.g., if the observed fixed effect was 1.05, the missing study effect size was set to .95).

We also examined bias in relation to funnel plots, which chart standardized effect size against variance around the observed meta-analytic mean. Compared with large studies, small studies are more likely to obtain extremely positive and extremely negative results. Because of publication bias toward publishing positive findings—especially extreme positive findings—publication bias tends to produce larger effects from smaller studies. This can be visually examined within funnel plots. In the absence of publication bias, the funnel plot is symmetrical, with studies equally likely to fall above and below the mean regardless of study size. Publication bias produces asymmetry in this plot, as small studies with positive findings (especially large positive findings) are more likely to be published than small studies with negative findings.

Because interpretations of funnel plots can be subjective, we calculated three indices of this asymmetry. First, Begg and Mazumdar's rank order correlation investigates the association between standardized effect sizes (log odds or hazard ratios) and the meta-analytic weighting factors (i.e., primarily sample size). A significant negative correlation indicates that smaller studies are associated with larger effects, which suggests publication bias. Second, Egger's test of the intercept regresses standardized effect sizes onto study precision (i.e., inverse of standard error). A significant positive intercept indicates that less precise studies are associated with larger effects, again suggesting publication bias. Third, Duval and Tweedie's trim and fill analysis determines how many studies would need to be included below the meta-analytic mean to make the funnel plot symmetrical. A higher number of studies denotes greater publication bias. This analysis also imputes these missing studies and calculates a meta-analytic effect size that adjusts for publication bias.

Meta-regression. Meta-regression is similar to traditional regression, except that meta-regression includes standardized effect sizes as the dependent variable and weights each independent variable effect size differently (as in a meta-analysis). In the present meta-analysis, we used meta-regression to analyze associ-

ations between independent variables (i.e., moderators) and standardized odds or hazard ratios. For clarity of interpretation, we present these results as *wORs*, *wHRs*, or *wAUCs* and 95% confidence intervals at each level of the independent variable. For example, rather than a single linear coefficient for the effect of sample severity type on effect sizes, we present a weighted mean and confidence intervals for each sample type—general, clinical, and STB. The two methods generate identical results, but the latter provides much more detailed information about specific data patterns.

Analytic Strategy

Overview of analytic strategy. The analytic strategy included three different phases. First, we provided a descriptive account of the literature. Second, we analyzed the overall ability of all available effect sizes to predict a given outcome. For example, we combined all predictors from all effect sizes with a suicide attempt outcome to produce a single aggregated estimate of how well all extant predictors—as a whole—predict suicide attempts. Third, we examined several potential moderators of the association between these predictors and a given outcome. As described above, the aim of moderation analyses to address specific questions (e.g., the effect of sample age on effect sizes) rather than to account for all variation among effect sizes. To maximize the presentation of data patterns, we provided weighted means and 95% confidence intervals at each level of each moderator.

Overall description of the literature. We first calculated the overall descriptive statistics for the literature as a whole, including the number of studies and effect sizes across the eras of STB risk factor research. For clarity, all other descriptive information is included within the appropriate subsections (e.g., descriptive information about follow-up intervals in the follow-up length moderator subsection).

Overall effect size for each outcome. To provide a general estimate of risk factor magnitude and accuracy, we conducted nonmoderated analyses for each outcome (all moderation analyses were also organized by outcome). As noted above, odds ratio, hazard ratio, and diagnostic accuracy analyses were conducted separately. For odds and hazard ratio analyses, we calculated overall estimates, confidence intervals, and I^2. For diagnostic accuracy analyses, we calculated estimates and standard errors for sensitivity, specificity, and *wAUCs*.

Overall publication bias for each outcome. For each overall outcome analysis, we calculated publication bias statistics. These included classic and Orwin's fail-safe Ns, Begg and Mazumdar's rank-order correlation test, Egger's test of the intercept, and Duval and Tweedie's trim and fill analysis. This latter analysis provided estimates of overall effects corrected for publication bias. This correction was not available for diagnostic accuracy estimates.

Overall dependence effects for each outcome. Within models that assumed and accounted for complete dependence among effect sizes (i.e., averaged effect sizes within each study to produce one effect size per study), we recalculated the overall pooled estimates and primary publication bias estimates for each outcome.

Simulation of prediction with base rates. Although overall analyses provide an index of how well risk factors predict STB outcomes beyond chance prediction (i.e., odds/hazard ratios of 1.0, diagnostic accuracy statistics of 0.50), many clinicians may consider the low STB base rates when estimating STB risk. To provide an index of how well risk factors predict STB outcomes compared with base rate guessing, we conducted simulation tests. For suicide death, we created a simulation where there were 13 instances of suicide death across 100,000 person-years (i.e., the United States suicide death rate in 2013; CDC, 2014). We then randomly selected 13 of these 100,000 person-years to see whether any of them were one of the 13 predetermined simulation suicide death instances. In other words, each simulation used base rate guessing to predict suicide death. We then repeated this simulation 10,000 times. We conducted similar simulations for suicide attempt (assuming a prevalence of 325 per 100,000) and suicide ideation (assuming a prevalence of 2,000 per 100,000). From these simulations, we calculated sensitivity and specificity statistics and compared them to prediction by risk factors.

Moderation by outcome characteristics. The effects of outcome definition and assessment strategy were calculated for each outcome; suicide attempt repetition status was additionally examined for suicide attempt outcomes effect sizes.

Moderation by adjusted versus unadjusted estimates. Adjusted and unadjusted estimates were compared for each outcome.

Moderation by predictor scale. Dichotomous predictors were compared with continuous predictors for each outcome.

Moderation by era of research. Dividing the eras of suicide risk factor research into 10 year intervals, we calculated effect estimates for each era for each outcome.

Moderation by follow-up length. We calculated effect estimates for each follow-up length class interval for each outcome.

Moderation by sample characteristics. The effects of sample severity, clinical sample type, STB inclusion type, clinical/STB sample origin, age, and STB sample size on effect estimates were calculated for each outcome.

Moderation by study quality. Separate meta-regressions tested the effect of (a) recruitment rate reporting, retention rate reporting, outcome definition type, and outcome assessment type, and (b) calculated retention rates on effect estimates for each outcome.

Moderation by broad risk factor categories. We calculated effect estimates for each broad risk factor category across each outcome. To demonstrate that no moderately specific predictor category substantially deviated from overall estimates, we also calculated the top five predictors (in terms of *wORs*) for subcategories with effect sizes from at least five studies.

Protective factor analyses. Similar to risk factor analyses, for protective factor analyses we calculated descriptive information, overall protective factor magnitude and accuracy estimation across each outcome, and moderation by broad risk factor categories.

Results

Overall Description of the Literature

Number of papers across time. A total of 365 papers met inclusion criteria. The earliest paper was Motto (1965), but less than one qualifying paper per year was published over the ensuing 20 years. Only 3.29% of papers meeting criteria were published before 1985. The number of papers meeting criteria nearly doubled during each 10-year period after 1984 (see Figure 2). In fact,
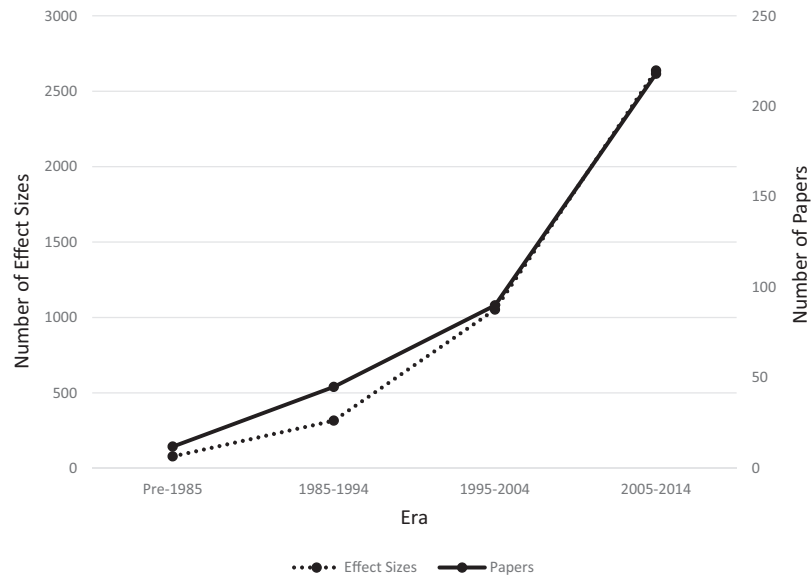
Figure 2. Number of papers and effect sizes across each era of STB research.

60.73% of qualifying papers were published in the last decade and more than half were published after 2006.

Number of effect sizes across time. These 365 papers produced a total of 4,084 effect sizes. Of these, 495 effect sizes (12.78% of all effect sizes) were categorized as pertaining to protective factors (broadly defined) rather than risk factors. Protective factor, outlier, and redundant effect sizes were not included in risk factor analyses (see below for a specific section on protective factors); the total number of risk factor effect sizes analyzed was 3,428. Following the paper-based pattern described above, only 1.93% of these effect sizes were published before 1985, 64.57% were published after 2004, and more than 50% were published after 2007 (see Figure 2). Overall, there was an average of 11.22 effect sizes per paper, with effect sizes per paper doubling in the mid-1990s and holding nearly constant since. In short, most of the empirical information on risk and protective factors for STBs was published in the last few years.

Overall Analytic and Publication Bias Information for Risk Factors

Overall suicide ideation prediction and publication bias. For odds ratio analyses, there were 572 total suicide ideation effect sizes. There was extremely high heterogeneity among these effect sizes ($I^2 = 90.52\%$), and these effect sizes generated an overall weighted mean odds ratio of 1.50 (CI: 1.47 to 1.54). Although fail-safe N analyses indicated that this was a robust nonzero effect, a Begg and Mazumdar rank correlation test and Egger's test of the intercept suggested significant publication bias (see Table 3). These tests show that smaller and less precise studies were more likely to produce larger odds ratios than larger and more precise studies. Similarly, the funnel plot was highly asymmetrical, with Duval and Tweedie's trim and fill analysis determining that 106 effect sizes below the mean were missing. If these effect sizes had been published and factored into analyses, it is estimated that the weighted mean

odds ratio would have dropped to 1.30 (CI: 1.27, 1.34). Unfortunately, there were not enough effect sizes to conduct reliable hazard ratio or diagnostic accuracy meta-analyses for suicide ideation.

Within models that assumed and accounted for complete dependence among effect sizes (i.e., models that averaged all effect sizes within each study, producing one effect size per study), the overall weighted mean odds ratio was 1.51 (CI: 1.45, 1.57). Duval and Tweedie's trim and fill analyses indicated that 22 studies below the mean were missing and, if included, would have lowered the overall weighted mean odds ratio to 1.30 (CI: 1.25, 1.35).

Overall suicide attempt prediction and publication bias. There were 1,281 odds ratio effect sizes with suicide attempt as an outcome. As with suicide ideation, there was very high heterogeneity among these effect sizes ($I^2 = 86.09\%$). These effect sizes produced a weighted mean odds ratio of 1.51 (CI: 1.49, 1.54). Fail-safe N analyses suggested that this was a robust nonzero effect, but a Begg and Mazumdar rank correlation test and an Egger's test of the intercept both indicated substantial publication bias toward publishing large positive findings from small and imprecise studies (see Table 3). Consistent with these tests, the funnel plot was highly asymmetrical, with Duval and Tweedie's trim and fill test finding that 344 effect sizes below the mean were missing. If these omitted findings had been published and included in analyses, it is estimated that the overall weighted mean odds ratio would have fallen to 1.26 (CI: 1.24, 1.29).

Odds ratio models that assumed and accounted for complete dependence among effect sizes produced a weighted mean odds ratio of 1.49 (CI: 1.44, 1.53). According to Duval and Tweedie's trim and fill analysis, 50 studies below the mean were missing. Inclusion of these studies would have produced a lowered estimate of 1.30 (CI: 1.26, 1.33).

A total of 192 hazard ratio effect sizes included suicide attempt as an outcome. These effect sizes were highly heterogeneous ($I^2 = 89.16\%$) and produced an overall weighted mean hazard ratio of

Table 3
Publication Bias Statistics by Outcome Across Odds Ratio and Hazard Ratio Effect Sizes

Statistic	Suicide ideation	Suicide attempt	Suicide death
	Odds ratio analyses		
Classic Fail-Safe N	306,405 effect sizes	1,254,253 effect sizes	1,105,490 effect sizes
Orwin's Fail-Safe N	385 effect sizes	1,117 effect sizes	767 effect sizes
Begg & Mazumdar's Rank Correlation Test	$B = -.16, p < .001$	$B = -.12, p < .001$	$B = -.12, p < .001$
Egger's Test of the Intercept	$B(0) = 1.87, p < .001$	$B(0) = 1.62, p < .001$	$B(0) = 1.63, p < .001$
Duval & Tweedie's Trim and Fill Method	106 effect sizes missing	344 effect sizes missing	0 effect sizes missing
Overall Degree of Publication Bias	High	High	Moderate
	Hazard ratio analyses		
Classic Fail-Safe N	n/a	40,313 effect sizes	119,637 effect sizes
Orwin's Fail-Safe N	n/a	127 effect sizes	322 effect sizes
Begg & Mazumdar's Rank Correlation Test	n/a	$B = -.03, p = .28$	$B = -.16, p < .001$
Egger's Test of the Intercept	n/a	$B(0) = 2.21, p < .001$	$B(0) = 1.73, p < .001$
Duval & Tweedie's Trim and Fill Method	n/a	70 effect sizes missing	0 effect sizes missing
Overall Degree of Publication Bias	n/a	Moderate	Moderate

Note. Fail-Safe N Tests assess the robustness of findings whereas Begg & Mazumdar's, Egger's, and Duval and Tweedie's tests each directly assess publication bias. If all three of these latter tests indicated bias, we categorized bias as high; if two indicated bias, we categorized bias as moderate; and if one indicated bias, we categorized bias as low. It should also be noted that Fail-Safe Ns are distinct from overall Ns (see Publication Bias subsection of the Method section).

1.23 (CI: 1.20, 1.26). Fail-safe N analyses found that this was a robust nonzero effect, and a Begg and Mazumdar rank correlation test did not detect significant publication bias (see Table 3). However, an Egger's test of the intercept did detect significant publication bias (see Table 3), and the funnel plot was asymmetrical. A Duval and Tweedie trim and fill analysis indicated 70 effect sizes below the mean were missing, and that including these effect sizes would have reduced the overall weighted mean hazard ratio to 1.13 (CI: 1.11, 1.16).

A total of 367 suicide attempt prediction effect sizes included information sufficient for diagnostic accuracy analyses. Overall accuracy for suicide attempt prediction was poor—with accuracy being only slightly better than chance ($wAUC = 0.58$, $SE = 0.01$). This appeared to be attributable to very poor sensitivity (i.e., true positive rate; *weighted sensitivity* = 0.26; CI: 0.25, 0.27), meaning that risk factors rarely correctly identified individuals who actually went on to make a suicide attempt. Specificity (i.e., true negative rate; *weighted specificity* = 0.75; CI: 0.74, 0.76) was much higher, but this may be a methodological artifact of crossing a low base rate phenomenon (suicide attempt) with low base rate risk predictors (e.g., prior suicide attempts).

Overall suicide death prediction and publication bias. There were 912 odds ratio effect sizes that included suicide death as an outcome; as with suicide ideation and attempts, there was extremely high heterogeneity among these effect sizes ($I^2 = 98.45\%$). These effect sizes generated an overall weighted mean odds ratio of 1.50 (CI: 1.46, 1.56). Fail-safe N analyses showed that this was a robust nonzero effect, but a Begg and Mazumdar rank correlation test and an Egger's test of the intercept indicated significant publication bias (see Table 3). However, the funnel plot appeared to be symmetrical, with Duval and Tweedie's trim and fill analysis finding no evidence of missing effect sizes.

Assuming and accounting for complete dependence among effect sizes, the weighted mean odds ratio would have been slightly higher ($wOR = 1.64$; CI: 1.53, 1.76); however, this model also indicated greater publication bias. Duval and Tweedie's trim and fill analysis identified 25 missing studies below the mean. If these had been included, the weighted mean odds ratio would have been reduced to 1.45 (CI: 1.36, 1.55).

A total of 346 hazard ratio effect sizes included suicide death as an outcome. These effect sizes were very heterogeneous ($I^2 = 90.01\%$) and produced an overall weighted mean hazard ratio of 1.66 (CI: 1.59, 1.74). Fail-safe N analyses once again indicated that this was a robust nonzero effect, but a Begg and Mazumdar rank correlation test and an Egger's test of the intercept both detected significant publication bias (see Table 3). As with odds ratio analyses, however, the funnel plot appeared symmetrical as a Duval and Tweedie's trim and fill analysis did not detect any evidence of missing effect sizes.

There were 358 suicide death effect sizes that included information sufficient for diagnostic accuracy analyses. As with suicide attempts, diagnostic accuracy for suicide death prediction was poor ($wAUC = 0.57$, $SE = 0.01$). Echoing suicide attempt analyses, suicide death prediction followed a pattern of moderately strong specificity (*weighted specificity* = 0.81; CI: 0.80, 0.82) but very poor sensitivity (*weighted sensitivity* = 0.09; CI: 0.08, 0.10). In sum, current risk factors rarely correctly identify individuals who actually go on to die by suicide, and have a high true negative rate that may be a methodological artifact of a low base rate outcome crossed with low base rate predictors.

Suicide plans analyses. There were too few effect sizes for suicide plans ($n = 88$ effect sizes; 2.57% of all risk effect sizes) to conduct reliable meta-analyses. There has been a recent increase in longitudinal studies of these behaviors, with 74% of suicide plan effect sizes coming from the last decade. Nevertheless, many more studies are needed to provide reliable information about risk factors for suicide plans.

Simulation of base rate prediction accuracy. To compare the accuracy of STB prediction with risk factors (i.e., the preceding diagnostic accuracy analyses) to prediction with base rate guessing, we conducted simulation analyses. Across 10,000 simulations of 100,000 person-years, base rate guessing correctly identified

401,419 of 20,000,000 suicide ideators and 960,401,419 of 980,000,000 nonideators. These simulations produced a mean sensitivity of 0.02 (SD = .003) and a mean specificity of 0.98 (SD < .001). Simulations for suicide attempt showed that base rate guessing correctly identified 10,361 of 3,250,000 suicide attempters and 993,510,361 of 996,750,000 nonattempters. Results indicated a mean sensitivity of 0.003 (SD = 0.003) and a mean specificity of 0.99 (SD < .001). For suicide death analyses, simulations for base rate guessing correctly identified 18 of 130,000 suicide deaths and 999,740,018 of 999,870,000 nondeaths. These simulations generated a mean sensitivity of 0.0001 (SD = 0.003) and a mean specificity of 0.99 (SD < .001). As with predictive accuracy based on risk factors (see above), base rate guessing rarely correctly identified instances of STBs (i.e., low sensitivity/true positive rate) but accurately identified non-STB instances (i.e., high specificity/true negative rate). Compared with prediction with risk factors, base rate guessing produced moderate declines in sensitivity and large increases in specificity.

Brief summary. These broad analyses show that overall prediction of STBs is poor in terms of odds ratios, hazard ratios, and diagnostic accuracy statistics. Specifically, odds and hazard ratio analyses indicate that existing factors do not substantially increase the risk of STBs and diagnostic accuracy analyses suggest that existing factors rarely correctly identify people who go on to engage in suicidal behavior. Models that assumed and accounted for complete dependence among effect sizes produced virtually identical results.

Outcome Characteristics

The preceding analyses assumed that all outcomes for a given STB were assessed and defined the same way. However, as in most other areas of psychology, many suicide researchers disagree about the best way to assess and define each STB. It is possible that heterogeneity in these outcome characteristics across effect sizes impacted results. To test this possibility, we examined the effect of various outcome characteristics on the prediction of each type of STB.

Suicide ideation assessment type. Weighted mean odds ratio analyses showed a small but statistically significant reduction in magnitude for single-item assessments (n = 353; wOR = 1.43; CI: 1.39, 1.47) compared with questionnaire/interview-based assessments (n = 219; wOR = 1.68; CI: 1.60, 1.77). Only one effect size included an unclear/unstated suicide ideation assessment strategy.

Suicide ideation definition type. Ideation definitions that were clearly passive produced significantly smaller weighted mean odds ratios (n = 65; wOR = 1.19; CI: 1.13, 1.26) compared with definitions that were unclear/unstated (n = 33; wOR = 1.56; CI: 1.30, 1.87), clearly active (n = 239; wOR = 1.52; CI: 1.46, 1.58), or clearly a mix of active and passive ideation (n = 236; wOR = 1.67; CI: 1.58, 1.76).

Suicide attempt assessment type. Questionnaire/interview assessments (n = 401; wOR = 1.45; CI: 1.41, 1.50) and single-item assessments (n = 551; wOR = 1.53; CI: 1.49, 1.57) produced similar weighted mean odds ratios. Interestingly, effect sizes that included an unclear/unstated suicide attempt assessment strategy generated a significantly higher weighted mean odds ratio (n = 329; wOR = 1.72; CI: 1.63, 1.81). Hazard ratio analyses indicated a slightly different pattern, with unclear/unstated (n = 71; wHR = 1.23;

CI: 1.19, 1.28), single-item (n = 39; wHR = 1.28; CI: 1.20, 1.37), and questionnaire/interview assessments (n = 82; wHR = 1.25; CI: 1.20, 1.31) producing nearly identical results. Diagnostic accuracy was uniformly poor across single-item (n = 103; $wAUC$ = 0.58; SE = 0.01), questionnaire/interview (n = 132; $wAUC$ = 0.60; SE = 0.01), and unclear/unstated assessment strategies (n = 132; $wAUC$ = 0.58; SE = 0.01).

Suicide attempt definition type. Suicide attempt definitions that explicitly included suicide intent produced significantly smaller weighted mean odds ratios (n = 486; wOR = 1.43; CI: 1.39, 1.47) compared with definitions where suicide intent could only be inferred (n = 510; wOR = 1.60; CI: 1.55, 1.65) and unclear/unstated definitions (n = 285; wOR = 1.59; CI: 1.53, 1.66). Hazard ratio analyses once again produced a slightly different pattern, with suicide definitions that were unclear/unstated (n = 13; wHR = 1.15; CI: 1.09, 1.21) or explicitly included suicide intent (n = 142; wHR = 1.21; CI: 1.18, 1.24) generating significantly smaller magnitudes than definitions where suicide intent could only be inferred (n = 37; wHR = 1.83; CI: 1.59, 2.12).

There were no significant differences in diagnostic accuracy across suicide attempt definitions that were unclear/unstated (n = 111; $wAUC$ = 0.59; SE = 0.02), explicitly included suicide intent (n = 173; $wAUC$ = 0.57; SE = 0.01), or where suicide intent could only be inferred (n = 83; $wAUC$ = 0.59; SE = 0.02).

Initial or repeated suicide attempt. Weighted mean odds ratio analyses did not reveal any significant differences among attempt frequency types. Only two effect sizes included only first-time attempters (wOR = 1.25; CI: 0.53, 2.95) and these effect sizes were not significantly different from effect sizes that included only repeat attempters (n = 151; wOR = 1.67; CI: 1.51, 1.85), a mix of first-time and repeat attempters (n = 1,046; wOR = 1.51; CI: 1.48, 1.53), or participants with an unclear attempt status (n = 82; wOR = 1.56; CI: 1.41, 1.73). With hazard ratio analyses, there was only one effect size that included only first-time attempters, precluding a meta-analysis. Effect sizes that included only repeat attempters (n = 9; wHR = 1.93; CI: 1.37, 2.71) generated significantly higher magnitudes than effect sizes that included a mix of first-time and repeat attempters (n = 132; wHR = 1.19; CI: 1.16, 1.23) and participants with an unclear attempt status (n = 50; wHR = 1.29; CI: 1.23, 1.35); however, the repeat attempter figure may not be reliable because it is only based on nine effect sizes. Diagnostic accuracy was similarly poor across effect sizes that included only repeat attempters (n = 83; $wAUC$ = 0.60; SE = 0.02), a mix of first-time and repeat attempters (n = 266; $wAUC$ = 0.57; SE = 0.01), and participants with an unclear attempt status (n = 16; $wAUC$ = 0.65; SE = 0.04). There were too few effect sizes that included only first-time attempters to permit a diagnostic accuracy meta-analysis (n = 2).

Suicide death assessment type. There were no significant differences among suicide death assessment types, with similar weighted mean odds ratios regardless of whether suicide death was assessed with legal/medical documentation (n = 723; wOR = 1.53; CI: 1.46, 1.59), family reports (n = 13; wOR = 0.77; CI: 0.38, 1.58), or unclear/unstated assessment strategies (n = 178; wOR = 1.42; CI: 1.27, 1.57). All hazard ratio effect sizes included legal/medical documentation, precluding moderation analyses. As with odds ratio analyses, diagnostic accuracy analyses did not detect any significant differences across legal/medical documentation (n = 276; $wAUC$ = 0.55; SE = 0.01), family report (n = 3;

$wAUC = 0.31$; $SE = 0.30$), and unclear assessment strategies ($n = 79$; $wAUC = 0.62$; $SE = 0.02$).

Suicide death definition type. Weighted mean odds ratio analyses showed that all definition types generated similar magnitudes, with similar results for suicide death definitions that explicitly excluded ambiguous deaths ($n = 332$; $wOR = 1.56$; CI: 1.47, 1.66), did not explicitly include or exclude ambiguous deaths ($n = 416$; $wOR = 1.48$; CI: 1.39, 1.57), and unclear/unstated definitions ($n = 164$; $wOR = 1.38$; CI: 1.24, 1.53). As noted above, effect sizes with suicide death definitions that explicitly included ambiguous deaths were excluded; however, we note here that the weighted mean odds ratio was comparable for these excluded effect sizes ($n = 206$; $wOR = 1.38$; CI: 1.31, 1.47). Echoing these findings, hazard ratio analyses revealed nearly identical magnitudes for definitions that did not explicitly exclude or include ambiguous deaths ($n = 84$; $wHR = 1.61$; CI: 1.51, 1.72) and definitions that explicitly excluded ambiguous deaths ($n = 262$; $wHR = 1.60$; CI: 1.49, 1.72). Excluded effect sizes that explicitly included ambiguous deaths produced very similar effect estimates ($n = 78$; $wHR = 1.67$; CI: 1.45, 1.93).

Diagnostic accuracy analyses revealed poor prediction across definitions that were unclear/unstated ($n = 71$; $wAUC = 0.62$; $SE = 0.02$), did not explicitly include or exclude ambiguous deaths ($n = 175$; $wAUC = 0.56$; $SE = 0.02$), and explicitly excluded ambiguous deaths ($n = 112$; $wAUC = 0.55$; $SE = 0.02$).

Brief summary. Viewed as a whole, these outcome characteristic analyses show that factors such as assessment type, outcome definition, and attempt status had little effect on predictive ability within the present meta-analysis. In short, predictive ability was poor regardless of outcome characteristics.

Adjusted Versus Nonadjusted Risk Factors

Descriptive characteristics. Every effort was made to obtain unadjusted estimates for risk factor magnitudes to provide the cleanest possible estimates. This was possible for most effect sizes (82.06%), with most adjusted analyses controlling for basic demographic factors such as age and gender.

Adjustment and suicide ideation prediction. The weighted mean odds ratio for adjusted suicide ideation analyses ($n = 103$; $wOR = 1.28$; CI: 1.23, 1.34) was significantly smaller than the weighted mean odds ratio for unadjusted analyses ($n = 469$; $wOR = 1.59$; CI: 1.54, 1.63).

Adjustment and suicide attempt prediction. As with ideation, suicide attempt analyses indicated that weighted mean odds ratios were significantly smaller for adjusted ($n = 155$; $wOR = 1.38$; CI: 1.33, 1.44) compared with nonadjusted effect sizes ($n = 1126$; $wOR = 1.55$; CI: 1.52, 1.58). Hazard ratio analyses revealed that weighted means were statistically similar for adjusted ($n = 15$; $wHR = 1.19$; CI: 1.13, 1.26) compared with nonadjusted effect sizes ($n = 177$; $wHR = 1.26$; CI: 1.22, 1.29).

Adjustment and suicide death prediction. Weighted mean odds ratio analyses showed that adjusted ($n = 162$; $wOR = 1.71$; CI: 1.51, 1.94) and nonadjusted effect sizes ($n = 750$; $wOR = 1.47$; CI: 1.42, 1.52) produced statistically similar magnitudes. Likewise, hazard ratio analyses indicated a statistically significant difference between adjusted ($n = 148$; $wHR = 1.48$; CI: 1.33, 1.65) and nonadjusted effect sizes ($n = 198$; $wHR = 1.76$; CI: 1.67, 1.85).

Brief summary. These analyses indicate that adjusting for other potential risk factors (again, adjustments typically included one or two demographic factors) may slightly reduce effect size magnitude, but that magnitude is small regardless of adjustment.

Predictor Scale

Descriptive characteristics. The majority of risk factor effect sizes included a dichotomous predictor scale ($n = 2,504$; 73.05% of all effect sizes) rather than continuous predictor scales ($n = 924$; 26.95% of all effect sizes). This was expected as most demographic variables and psychiatric diagnoses (i.e., the majority of risk factors) are in a dichotomous format.

Predictor scale and suicide ideation prediction. Odds ratio analyses revealed a small but statistically significant drop in risk factor magnitude from dichotomous ($n = 414$; $wOR = 1.58$; CI: 1.50, 1.66) to continuous predictor scales ($n = 158$; $wOR = 1.32$; CI: 1.29, 1.36).

Predictor scale and suicide attempt prediction. Suicide attempt prediction analyses showed a similar drop in weighted mean odds ratios from dichotomous ($n = 846$; $wOR = 1.80$; CI: 1.72, 1.87) to continuous predictor scales ($n = 435$; $wOR = 1.24$; CI: 1.22, 1.27). Hazard ratio analyses also revealed a pattern of reduced magnitude from dichotomous ($n = 115$; $wHR = 1.84$; CI: 1.67, 2.02) to continuous predictor scales ($n = 77$; $wHR = 1.11$; CI: 1.09, 1.13).

Predictor scale and suicide death prediction. Echoing findings for other outcomes, suicide death prediction analyses indicated that weighted mean odds ratios diminished from dichotomous ($n = 694$; $wOR = 1.57$; CI: 1.49, 1.65) to continuous predictor scales ($n = 218$; $wOR = 1.22$; CI: 1.17, 1.27). Despite including many fewer effect sizes, hazard ratio analyses showed a similar pattern, with magnitudes diminishing from dichotomous ($n = 328$; $wHR = 1.71$; CI: 1.60, 1.83) to continuous predictor scales ($n = 18$; $wHR = 1.07$; CI: 1.03, 1.10).

Brief summary. These highly consistent findings show that risk factor magnitudes tend to be slightly larger when risk factors are in a dichotomous format. It is important to note that this does not mean that dichotomous risk factors are "better" predictors. This effect is explained by a mathematical feature of odds and hazard ratios: they describe the change in odds/hazard *per unit of change* in the predictor variable. Because dichotomous predictors only have one unit of change (i.e., from Group A to Group B), they tend to produce larger odds and hazard ratio magnitudes; however, the tradeoff is that estimates of dichotomous risk factor magnitudes are less accurate. The present analyses reflected this tradeoff: a greater number of effect sizes should produce tighter confidence intervals, but dichotomous effect sizes produced wider confidence intervals despite including a much larger number of effect sizes.

Risk Factor Prediction Trends Across Eras of Research

Trends for prediction of suicide ideation. The earliest qualifying suicide ideation effect size was from Petrie and Chamberlain (1985), but suicide ideation outcome studies did not become common until the late 1990s, with qualifying effect sizes increasing steadily from 1985 to 1994 ($n = 16$; 5.69% of all effect sizes in this era), to 1995 to 2005 ($n = 167$; 18.43% of effect sizes), to

2005 to 2014 ($n = 413$; 19.87% of effect sizes). Although the number of effect sizes increased steadily over time, weighted mean odds ratios were very similar across eras of research (see Figure 3).

Trends for prediction of suicide attempt. The earliest qualifying suicide attempt effect sizes were from Greer and Bagley (1971), with suicide attempt effect sizes increasing in near-exponential fashion across later eras, especially over the last decade. A total of seven suicide attempt outcome cases qualified during the pre-1985 era (9.45% of all effect sizes in this era), 67 during 1985 to 1994 (23.84% of effect sizes), 342 during 1995 to 2004 (37.75% of effect sizes), and 1,057 during 2005 to 2014 (50.84% of effect sizes). Despite this marked increase in research, weighted mean odds ratios have not improved over time (see Figure 3). Similar analyses for hazard ratio effect sizes are limited as there were relatively few overall effect sizes and only one qualifying study appeared before 2004. Magnitudes were nearly identical from 1995 to 2004 ($n = 30$; $wHR = 1.23$; CI: 1.17, 1.31) to 2005 to 2014 ($n = 162$; $wHR = 1.24$; CI: 1.21, 1.28). Echoing these patterns, diagnostic accuracy analyses showed no significant change in weighted AUCs for suicide attempt prediction across eras of research (see Figure 4).

Trends for prediction of suicide death. The earliest qualifying suicide death effect sizes were reported in Motto (1965), with effect sizes increasing gradually from the pre-1985 era ($n = 67$; 90.54% of all effect sizes in this era), to 1985 to 1994 ($n = 198$; 70.46% of effect sizes), to 1995 to 2004 ($n = 397$; 43.81% of effect sizes), to 2005 to 2014 ($n = 609$; 29.29% of effect sizes). Weighted mean odds ratios were small in magnitude across each decade (see Figure 3). Time-based hazard ratio analyses are limited because of the low overall number of effect sizes and the fact that the first qualifying effect sizes were reported relatively recently (i.e., Brown, Beck, Steer, & Grisham, 2000). There was a small but statistically significant increase in effect magnitude from 2000 to 2004 ($n = 35$;

$wHR = 1.25$; CI: 1.18, 1.34) to 2005 to 2014 ($n = 311$; $wHR = 1.64$; CI: 1.54, 1.75). Consistent with the preceding analyses, diagnostic accuracy analyses indicated poor accuracy across each era of suicide research (see Figure 4).

Brief summary. Although the number of studies and effect sizes for all outcomes increased markedly across time, there was little change in predictive ability across time. This trend is shown clearly in Figure 5. This figure also shows that there is a highly restricted range of effect sizes in the existing literature, meaning that the poor overall predictive ability described above is unlikely to be meaningfully moderated by any methodological or psychological factor. In other words, because a cluster of abnormally large effect sizes does not exist in this literature, we are unlikely to find a moderator (e.g., outcome characteristics, time, and other moderators tested below) that separates abnormally large effect sizes from other effect sizes in this literature.

Risk and Follow-Up Length

Descriptive statistics. The overall mean follow-up length was 108.82 months ($SD = 108.58$ months), with a median of 72 months and a range of .50 to 912 months. To facilitate analyses, we grouped these heavily skewed follow-up lengths into six class intervals: 0 to 6 months ($n = 149$; 4.37% of cases); 7–12 months ($n = 410$; 12.02% of cases); 13–24 months ($n = 382$; 11.20% of cases); 25 to 60 months ($n = 687$; 20.13% of cases); 61 to 120 months ($n = 785$; 23.01% of cases); and 121+ months ($n = 999$; 29.28% of cases). Notably, 0.10% of effect sizes had follow-up lengths that were less than one month, and 0.90% of effect sizes had follow-up lengths that were one month or less; in stark contrast, nearly one third of effect sizes had follow-up lengths that were 10 years or longer.

Follow-up lengths have shortened slightly across the decades of suicide research, with lengths being longest before 1985 ($M =$
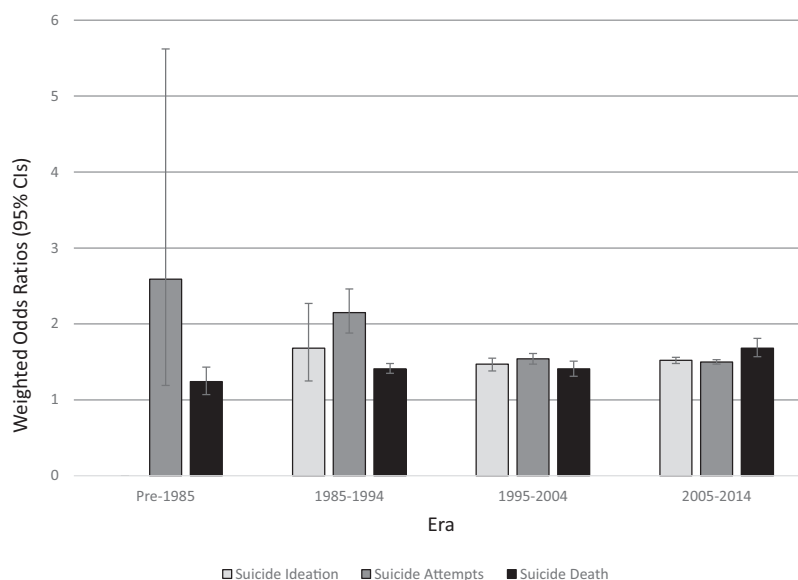
Figure 3. Weighted odds ratios by outcome type across each era of STB research. Error bars represent 95% confidence intervals. An odds ratio of 1.0 indicates chance-level prediction; if error bars include 1.0, it indicates that the effect was not significant. There were no eligible suicide ideation effect sizes before 1985.
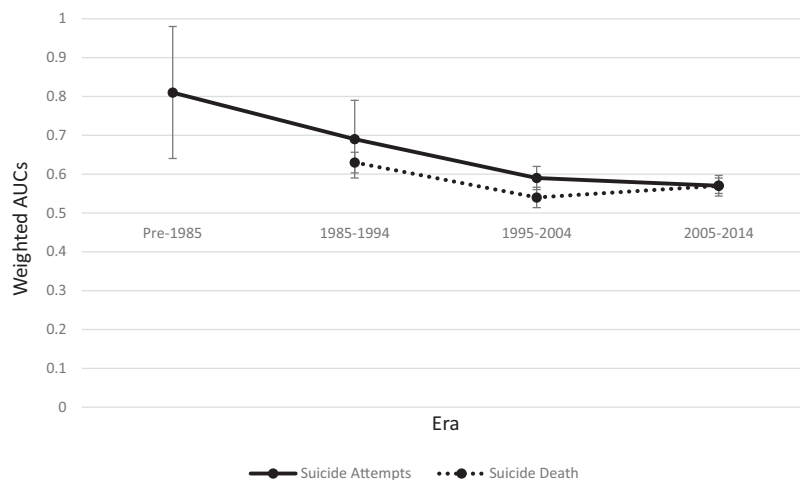
Figure 4. Weighted AUCs for suicide attempt and death prediction across each era of STB research. Errors bars = 95% confidence intervals. AUCs of .50 represent chance-level prediction of suicide attempt or death (i.e., random guessing), and an AUC of 1 would indicate perfectly accurate prediction (i.e., no false negatives or false positives).

234.19 months; SD = 153.95 months), dropping during the 1985 to 1994 era (M = 127.80 months; SD = 103.67 months), remaining similar during the 1995 to 2004 era (M = 127.79 months; SD = 94.87 months), and decreasing slightly during the 2005 to

2014 era (M = 94.24 months; SD = 108.51 months). Follow-up lengths for suicide death outcome effect sizes (M = 170.30 months; SD = 123.77 months) tended to be longer than lengths for suicide ideation (M = 80.72 months; SD = 88.39 months) and
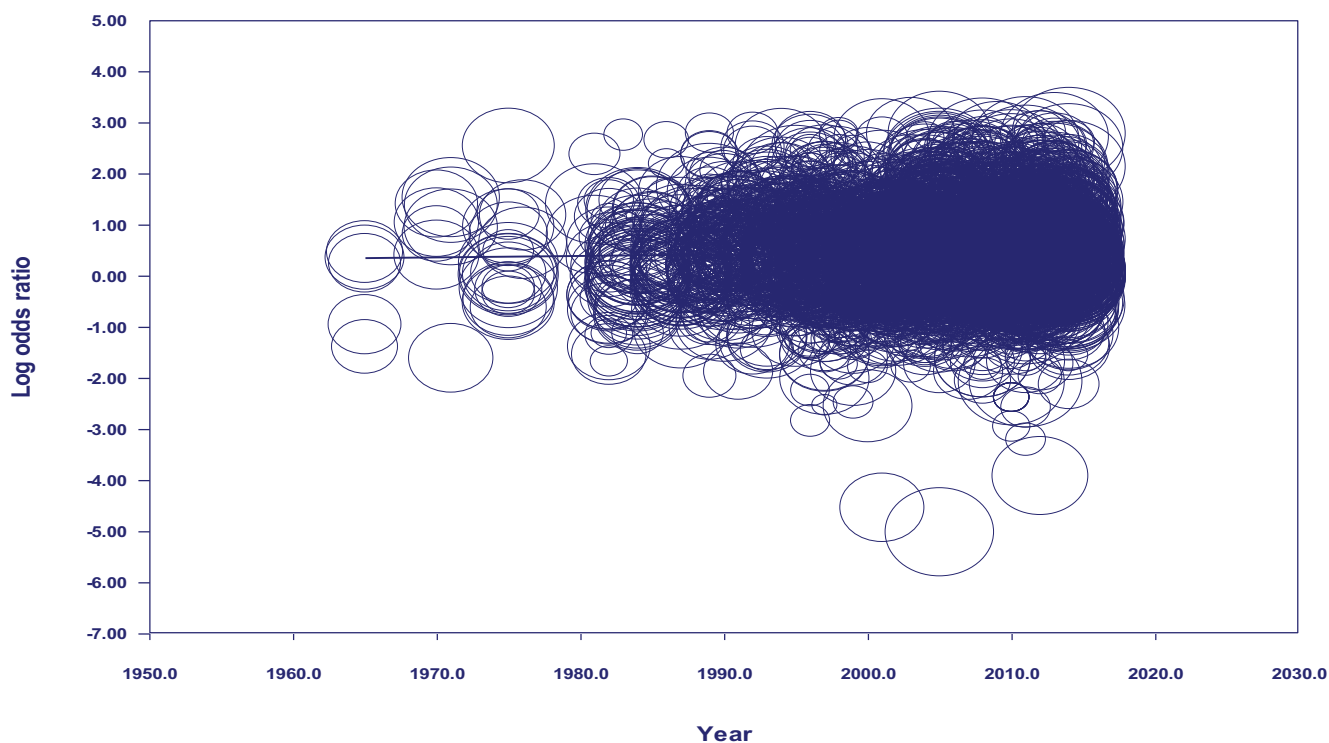

Figure 5. Meta-regression plot of log odds ratios for all STB outcomes across time. Meta-regression was restricted maximum likelihood; the center line in the figure is the regression line; each circle represents an effect size and the size of the circle is proportional to the effect size weight in the random-effects meta-analysis. The plot shows that all effects have always occurred within a tight range and have not improved over time. See the online article for the color version of this figure.

suicide attempt outcome effect sizes ($M = 68.25$ months; $SD = 75.21$ months).

Follow-up length and suicide ideation prediction. Weighted mean odds ratios across follow-up class intervals indicated that odds ratio magnitude decreased slightly from 0–6 months to all other intervals (see Figure 6).

Follow-up length and suicide attempt prediction. Across follow-up class intervals, there was an inconsistent pattern of weighted mean odds ratios (see Figure 6). Hazard ratio analyses showed a similar trend, with no valid effect sizes for the 0 to 6 month interval and an inconsistent pattern of effect sizes across intervals of 7 to 12 months ($n = 10$; $wHR = 1.06$; CI: 1.02, 1.10), 13 to 24 months ($n = 73$; $wHR = 1.12$; CI: 1.08, 1.15), 25 to 60 months ($n = 26$; $wHR = 2.36$; CI: 1.91, 2.92), 61 to 120 months ($n = 59$; $wHR = 1.31$; CI: 1.25, 1.37), and 121+ months ($n = 24$; $wHR = 1.97$; CI: 1.58, 2.46). Likewise, diagnostic accuracy analyses revealed an inconsistent pattern: 0 to 6 months ($n = 8$; $wAUC = .78$; $SE = .08$); 7 to 12 months ($n = 61$; $wAUC = .57$; $SE = .02$); 13 to 24 months ($n = 76$; $wAUC = .57$; $SE = .02$); 25 to 60 months ($n = 104$; $wAUC = .58$; $SE = .01$); 61 to 120 months ($n = 90$; $wAUC = .59$; $SE = .02$); and 121+ months ($n = 19$; $wAUC = .65$; $SE = .04$).

Follow-up length and suicide death prediction. Weighted mean odds ratios across follow-up class intervals suggested a slight decline between intervals covering 0 to 12 months and all other intervals (see Figure 6). The only eligible effect sizes for hazard ratio analyses included long follow-up intervals: 25 to 60 months ($n = 42$; $wHR = 1.87$; CI: 1.55, 2.25); 61 to 120 months ($n = 110$; $wHR = 1.86$; CI: 1.66, 2.09); and 121+ months ($n = 194$; $wHR = 1.43$; CI: 1.36, 1.50). Diagnostic accuracy analyses also followed this general pattern, with accuracy diminishing from the 7 to 12 month interval ($n = 13$; $wAUC = .68$; $SE = .05$) to intervals of 25 to 60 months ($n = 72$; $wAUC = .53$; $SE = .04$), 61 to 120 months ($n = 57$; $wAUC = .56$; $SE = .03$), and 121+ months ($n = 212$; $wAUC = .58$; $SE = .02$).

Brief summary. Predictive ability was poor across all follow-up intervals. Although a few statistically significant differences were detected, no consistent patterns emerged.

Risk and Sample Severity

Descriptive characteristics. Most effect sizes were tested within general samples ($n = 1,400$ effect sizes; 40.84% of all effect sizes) or clinical samples ($n = 1,339$ effect sizes; 39.06% of all effect sizes). Fewer were tested within samples where all participants had a history of some form of self-injurious thought or behavior (i.e., STB samples; $n = 689$ effect sizes; 20.10% of all effect sizes). General sample effect sizes accounted for a disproportionate amount of suicide ideation (72.48%) and death outcome effect sizes (40.44%), and clinical sample effect sizes were responsible for the majority of suicide attempt outcome effect sizes (52.82%). General sample effect sizes ($M = 125.32$ months; $SD = 119.91$ months) were longer than clinical sample effect sizes ($M = 102.29$ months; $SD = 108.99$ months), which in turn were longer than STB sample effect sizes ($M = 88.14$ months; $SD = 73.05$).

Sample severity and suicide ideation prediction. Weighted mean odds ratios for suicide ideation dropped significantly from general to clinical to STB sample effect sizes (see Figure 7). However, it should be noted that effect sizes were small across all sample types and there were relatively few effect sizes from clinical ($n = 133$) and STB samples ($n = 31$).

Sample severity and suicide attempt prediction. Weighted mean odds ratios for suicide attempt prediction were similar across all sample severity types, with slightly but significantly reduced magnitudes in clinical samples compared with general and STB samples (see Figure 7). Hazard ratio analyses showed a different pattern, with weighted means decreasing significantly from general ($n = 13$; $wHR = 2.13$; CI: 1.60, 2.85) to clinical ($n = 142$; $wHR = 1.33$; CI: 1.28, 1.37) and STB samples ($n = 37$; $wHR = 1.05$; CI: 1.03, 1.08). Similarly, diagnostic accuracy analyses indicated that AUCs were significantly larger in general samples
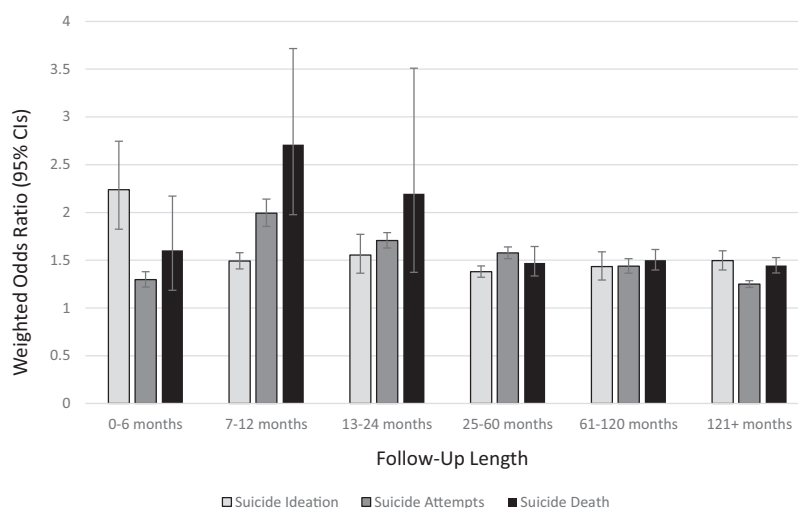
Figure 6. Weighted odds ratios by outcome type across follow-up length classes. Error bars represent 95% confidence intervals. An odds ratio of 1.0 indicates chance-level prediction; if error bars include 1.0, it indicates that the effect was not significant.

Figure 7. Weighted odds ratios by outcome type across each sample severity type. Error bars represent 95% confidence intervals. An odds ratio of 1.0 indicates chance-level prediction; if error bars include 1.0, it indicates that the effect was not significant.
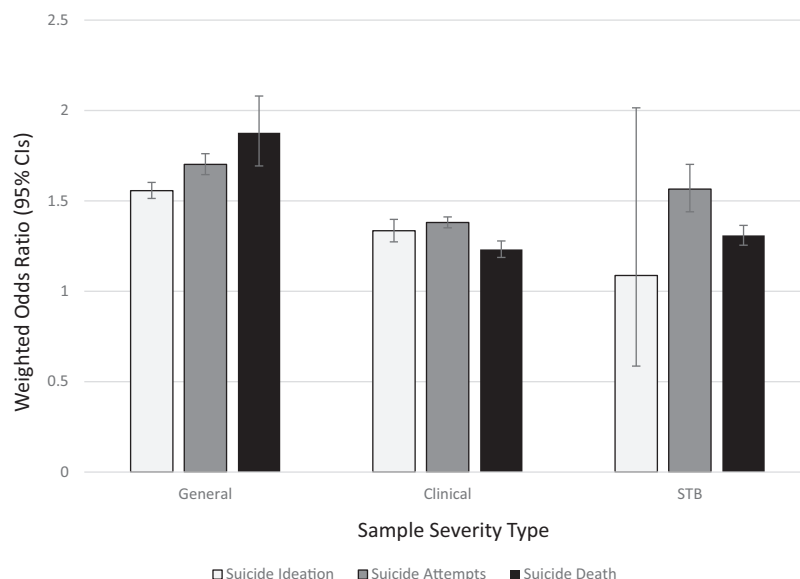
($n = 40$; $wAUC = 0.60$; $SE = 0.02$) compared with clinical ($n = 234$; $wAUC = 0.58$; $SE = 0.01$) and STB samples ($n = 93$; $wAUC = 0.62$; $SE = 0.02$), though accuracy was poor for all sample types.

Sample severity and suicide death prediction. Similar to ideation and attempts, analyses showed that weighted odds ratios for suicide death declined from general samples to clinical and STB samples (see Figure 7). The pattern for hazard ratio analyses was slightly different, with weighted means being similar across general ($n = 214$; $wHR = 1.61$; CI: 1.48, 1.74) and clinical samples ($n = 79$; $wHR = 1.44$; CI: 1.35, 1.53), and slightly higher in STB samples ($n = 53$; $wHR = 1.91$; CI: 1.67, 2.19). Diagnostic accuracy analyses showed that accuracy was similarly poor for general ($n = 70$; $wAUC = 0.58$, $SE = 0.02$), clinical ($n = 181$; $wAUC = 0.59$, $SE = 0.02$), and STB samples ($n = 107$; $wAUC = 0.51$, $SE = .02$).

Brief summary. Although there were a few exceptions, as a whole analyses indicated that general samples tended to produce slightly greater predictive accuracy than clinical or STB samples. As discussed below, this is consistent with the idea that clinical and STB samples represent more stringent reference groups.

Risk and Subtypes of Clinical and Self-Injurious Samples

Although the preceding sample severity analyses did not produce strong effects, it is possible that more specific aspects of clinical and self-injurious samples may influence STB prediction. Below, we test this possibility by examining the potential impacts of psychiatric issue type, clinical sample origin, and STB sample type. Because there were too few hazard ratio and diagnostic accuracy effect sizes for these subsample analyses, only weighted mean odds ratio results are reported below.

Type of psychiatric issue. Odds ratio analyses showed that the type of psychiatric issue reported for a given sample had little effect on suicide ideation prediction, with only one significant difference across effect sizes from samples with general psychopathology ($n = 54$; $wOR = 1.71$; CI: 1.41, 2.07), mood or anxiety disorders ($n = 55$; $wOR = 1.23$; CI: 1.16, 1.30), substance use disorders ($n = 7$; $wOR = 1.44$; CI: 1.19, 1.74), and psychotic disorders ($n = 11$; $wOR = 1.13$; CI: 0.63, 2.05).

There were likewise few significant differences in weighted mean odds ratio magnitude for the prediction of suicide attempts, with similar magnitudes for effect sizes from samples with general psychopathology ($n = 112$; $wOR = 1.76$; CI: 1.60, 1.94), mood and anxiety disorders ($n = 298$; $wOR = 1.56$; CI: 1.48, 1.64), substance use disorders ($n = 89$; $wOR = 1.63$; CI: 1.49, 1.78), psychotic disorders ($n = 56$; $wOR = 1.16$; CI: 1.08, 1.24), eating disorders ($n = 25$; $wOR = 1.99$; CI: 1.56, 2.54), attention-deficit/hyperactivity disorder ($n = 3$; $wOR = 3.56$; CI: 1.76, 7.21), borderline personality disorder ($n = 29$; $wOR = 1.22$; CI: 1.17, 1.28), and other personality disorders ($n = 23$; $wOR = 1.95$; CI: 1.50, 2.53). Although samples with psychotic disorders and borderline personality disorders produced significantly smaller weighted mean odds ratios than other sample types, these magnitude differences were very small in an absolute sense.

Similar to ideation and attempt findings, the type of psychiatric issue within a sample had little effect on suicide death prediction. There were no significant differences in weighted mean odds ratios across samples with general psychopathology ($n = 120$; $wOR = 1.56$; CI: 1.33, 1.82), mood and anxiety disorders ($n = 150$; $wOR = 1.29$; CI: 1.23, 1.34), substance use disorders ($n = 36$; $wOR = 1.19$; CI: 1.02, 1.39), or psychotic disorders ($n = 38$; $wOR = 1.15$; CI: 0.75, 1.77).

Type of STB sample. Suicide ideation prediction was similar within self-injurious samples selected for prior suicide ideation ($n = 16$; $wOR = 1.96$; CI: 1.24, 3.10), prior suicide attempt ($n = 10$; $wOR = 1.58$; CI: 0.42, 5.92), and general self-injurious

thoughts or behaviors ($n = 17$; $wOR = 2.30$; CI: 1.45, 3.63). A similar pattern emerged for suicide attempt prediction, with no significant differences across samples selected for prior ideation ($n = 69$; $wOR = 1.33$; CI: 1.26, 1.58), prior suicide attempt ($n = 192$; $wOR = 1.73$; CI: 1.57, 1.89), and general self-injurious thoughts or behaviors ($n = 42$; $wOR = 1.76$; CI: 1.46, 2.13). Echoing these findings, there were no significant differences in suicide death prediction across samples selected for prior suicide ideation ($n = 5$; $wOR = 1.91$; CI: 1.22, 3.00), prior suicide attempt ($n = 211$; $wOR = 1.35$; CI: 1.25, 1.46), general self-injurious thoughts or behaviors ($n = 71$; $wOR = 1.31$; CI: 1.20, 1.44), and suicide death ($n = 139$; $wOR = 1.68$; CI: 1.46, 1.59).

Clinical and STB sample origin. Suicide ideation prediction was similar across community ($n = 36$; $wOR = 1.47$; CI: 1.28, 1.69), inpatient ($n = 48$; $wOR = 1.38$; CI: 0.94, 2.02), outpatient ($n = 27$; $wOR = 1.14$; CI: 1.04, 1.24), and mixed inpatient/outpatient origins ($n = 31$; $wOR = 1.35$; CI: 1.24, 1.46). As with most other statistically significant differences in the present results, the significant differences between community and outpatient sample origins is small in an absolute terms. Analyses showed that suicide attempt prediction was slightly but significantly stronger in inpatient ($n = 344$; CI: 1.43, 1.52) and mixed inpatient/outpatient samples ($n = 362$; $wOR = 1.48$; CI: 1.43, 1.54) compared with community ($n = 55$; $wOR = 1.27$; CI: 1.18, 1.37) and outpatient samples ($n = 125$; $wOR = 1.32$; CI: 1.21, 1.44). Suicide death analyses revealed the opposite pattern, with significantly stronger prediction in outpatient samples ($n = 28$; $wOR = 2.60$; CI: 1.91, 3.55) compared with inpatient ($n = 485$; $wOR = 1.26$; CI: 1.21, 1.31) and mixed inpatient/outpatient samples ($n = 94$; $wOR = 1.21$; CI: 1.16, 1.27). There were too few community sample effect sizes with a suicide death outcome ($n = 1$) to permit meta-analysis.

Brief summary. As might be expected from the overall restricted range of effect sizes (see Figure 5) and the small effects within sample severity analyses, variations in clinical and STB sample types had little effect on results. A few significant effects were detected, but these effects were small and did not coalesce into a consistent pattern.

Risk and Sample Age

Descriptive statistics. The majority of effect sizes were from adult samples ($n = 2{,}114$ effect sizes; 61.67% of total effect sizes), with many fewer effect sizes from mixed ($n = 539$ effect sizes; 15.72% of total effect sizes) and adolescent samples ($n = 775$ effect sizes; 22.61% of total effect sizes). These proportions have held nearly constant since 1985, with adult effect sizes always accounting for nearly 60% of effect sizes and mixed and adolescent effect sizes each always accounting for nearly 20% of effect sizes. Follow-up lengths for mixed samples ($M = 115.36$ months; $SD = 83.34$ months) and adult samples ($M = 112.19$ months; $SD = 118.69$ months) tended to be longer than those of adolescent sample studies ($M = 95.18$ months; $SD = 93.49$ months). Compared with adult and mixed samples, adolescent samples included a high proportion of suicide ideation and suicide attempt outcomes; however, very few adolescent samples included suicide death as an outcome (see Figure 8). Adolescent effect sizes were primarily from general sample studies (70.71% of effect sizes), adult effect sizes included a high proportion of clinical sample studies (46.59% of effect sizes), and mixed samples were predominantly from STB sample studies (52.32% of effect sizes).

Sample age and suicide ideation prediction. Analyses revealed that weighted mean odds ratios for suicide ideation were similar across all sample age types (see Figure 9).

Sample age and suicide attempt prediction. Weighted mean odds ratios for suicide attempt were also very similar across all sample age types for suicide attempt outcomes (see Figure 9). Hazard ratio analyses detected a slightly different pattern, with a smaller effect size in adult samples ($n = 141$; $wHR = 1.17$; CI: 1.14, 1.19) compared with mixed ($n = 26$; $wHR = 1.98$; CI: 1.72, 2.27) and adolescent samples ($n = 25$; $wHR = 1.66$; CI: 1.39, 1.97). Diagnostic accuracy was poor for adult ($n = 261$; $wAUC = 0.58$, $SE = 0.01$), mixed ($n = 24$; $wAUC = 0.59$, $SE = 0.01$), and adolescent samples ($n = 82$; $wAUC = 0.59$, $SE = 0.02$).

Sample age and suicide death prediction. As with other outcomes, analyses did not detect any significant differences
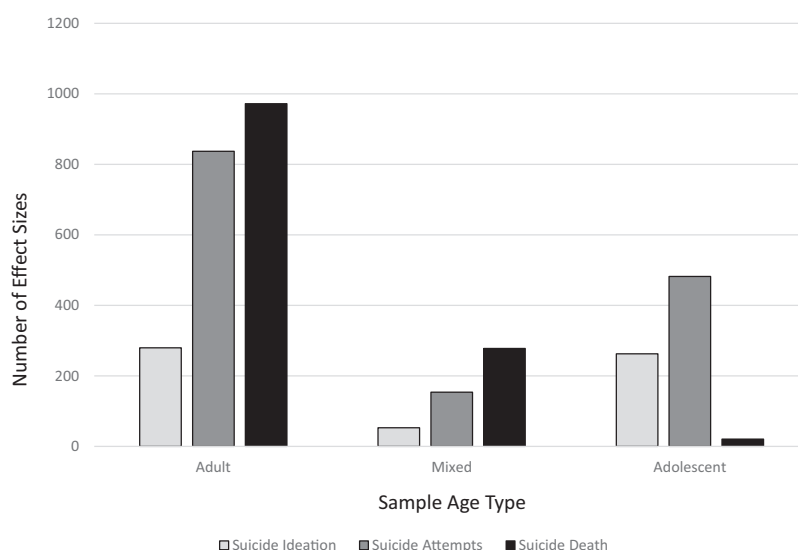

Figure 8. Number of effect sizes by outcome type across each sample age type.
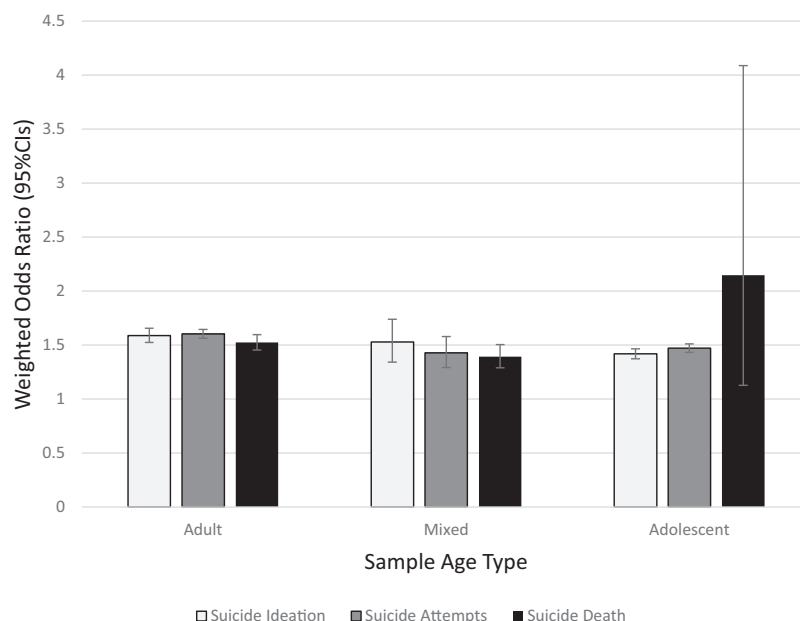
Figure 9. Weighted odds ratios by outcome type across each sample age type. Errors bars = 95% confidence intervals. An odds ratios of 1.0 indicates chance-level prediction; if error bars include 1.0, it indicates that the effect was not significant.

among weighted mean odds ratios across sample age types for suicide death outcomes (see Figure 9). Hazard ratio analyses detected statistically smaller magnitudes in adult samples ($n = 220$; $wHR = 1.50$; CI: 1.43, 1.58) compared with mixed ($n = 112$; $wHR = 1.79$; CI: 1.61, 2.00) and adolescent samples ($n = 14$; $wHR = 2.16$; CI: 1.69, 2.76). As with attempts, diagnostic accuracy analyses showed that accuracy was poor across adult ($n = 280$; $wAUC = 0.57$, $SE = 0.01$), mixed ($n = 74$; $wAUC = 0.55$, $SE = 0.03$), and adolescent samples ($n = 4$; $wAUC = 0.50$, $SE = 0.08$).

Brief summary. Sample age had little effect on predictive accuracy. Notably, however, results revealed a scarcity of risk factor studies with adolescent suicide death as an outcome.

Risk and Number of STB Participants

Descriptive statistics. Across all effect sizes, the mean number of STB participants was 166.53 ($SD = 696.68$; $Mdn = 57$; $range = 27,095$). To facilitate analyses, the number of STB participants for each effect size was categorized into one of four STB size classes: (a) 1 to 25 participants (i.e., very small; $n = 891$ effect sizes; 25.99% of all effect sizes); (b) 26 to 100 participants (i.e., moderately small; $n = 1375$ effect sizes; 40.11% of all effect sizes); (c) 101 to 500 participants (i.e., moderately large; $n = 782$ effect sizes; 22.81% of all effect sizes); and (d) 501 + participants (i.e., very large; $n = 300$ effect sizes; 8.75% of all effect sizes). Outcomes were mostly proportionately distributed across STB size classes, except that very large samples primarily included suicide death outcomes (67.00%). Follow-up intervals were not systematically related to the number of STB participants, with shorter follow-ups in very small ($M = 100.89$ months; $SD = 89.24$ months) and moderately large samples ($M = 92.67$ months; $SD =$

92.48 months) compared with moderately small ($M = 124.50$ months; $SD = 129.85$ months) and very large samples ($M = 119.37$ months; $SD = 86.90$ months). The number of SITB participants was proportionately distributed across sample age types, except that adult samples accounted for a disproportionate share of very small samples (70.03%).

Number of STB participants and suicide ideation prediction. Weighted mean odds ratio analyses for suicide ideation showed that magnitudes were similar across all STB sample size classes (see Figure 10).

Number of STB participants and suicide attempt prediction. Similar to ideation analyses, weighted mean odds ratios for the prediction of suicide attempts was similar across all classes (see Figure 10). Deviating from this pattern slightly, hazard ratio analyses found that magnitudes were significantly smaller for very small ($n = 25$; $wHR = 1.12$; CI: 1.06, 1.18) than for moderately small ($n = 109$; $wHR = 1.24$; CI: 1.20, 1.29) and moderately large samples ($n = 58$; $wHR = 1.31$; CI: 1.25, 1.37). There were no eligible effect sizes for the very large sample size class. Diagnostic accuracy analyses found that accuracy was poor across all intervals, with low mean weighted AUCs for effect sizes across very small ($n = 110$; $wAUC = 0.61$, $SE = 0.02$), moderately small ($n = 198$; $wAUC = 0.58$, $SE = 0.01$), and moderately large classes ($n = 59$; $wAUC = 0.57$, $SE = 0.01$). There were no valid effect sizes for the very large class interval.

Number of STB participants and suicide death prediction. Analyses showed that weighted mean odds ratios for suicide death were small in magnitude across all intervals (see Figure 10). Weighted mean hazard ratios produced a similar pattern of small magnitude effects for all class intervals: very small ($n = 21$; $wHR = 1.43$; CI: 1.10, 1.85), moderately small ($n = 138$; $wHR = 1.44$; CI:
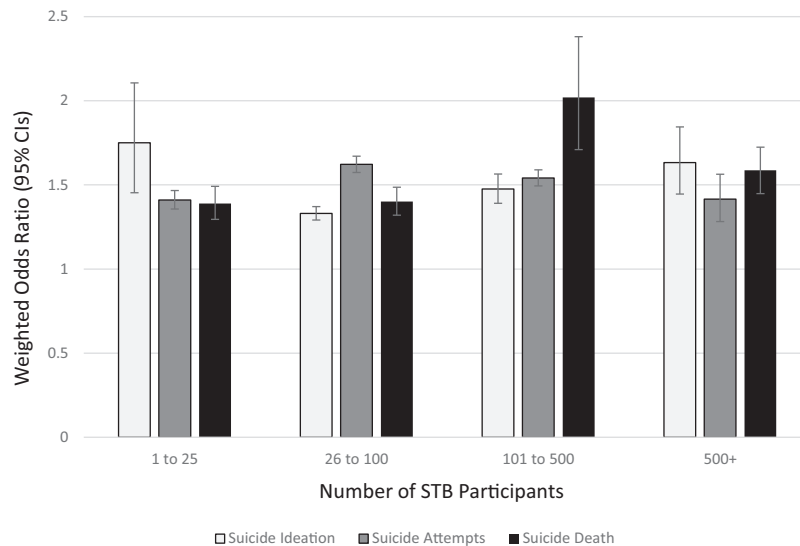
Figure 10. Weighted odds ratios across each STB sample size class. Error bars represent 95% confidence intervals. An odds ratio of 1.0 indicates chance-level prediction; if error bars include 1.0, it indicates that the effect was not significant.

1.36, 1.52), moderately large ($n = 146$; $wHR = 1.74$; CI: 1.56, 1.94), and very large classes ($n = 40$; $wHR = 1.64$; CI: 1.39, 1.91). Echoing these findings, diagnostic accuracy analyses revealed little variation in weighted mean AUCs, with similar findings across very small ($n = 141$; $wAUC = 0.61$, $SE = 0.02$), moderately small ($n = 175$; $wAUC = 0.56$, $SE = 0.01$), moderately large ($n = 27$; $wAUC = 0.50$, $SE = 0.05$), and very large classes ($n = 15$; $wAUC = 0.60$, $SE = 0.07$).

Brief summary. As with other potential moderators described above, the number of STB participants had little effect on predictive accuracy. Although there were a few significant effects across class intervals, no consistent patterns emerged—very small samples appeared to produce effects that were as strong as those from very large samples.

Risk and Study Quality

Descriptive characteristics. To examine the potential impact of study quality on STB prediction, we conducted a series of meta-regression analyses. As noted above, the relative homogeneity of the present set of studies (e.g., longitudinal designs) made it difficult to parse study quality. However, we employed two general types of variables to estimate study quality within the present meta-analysis: (a) variables related to assessment quality (described in Outcome Characteristic analyses above); (b) variables related to study recruitment/retention. Most studies did not report any information about recruitment rates (32.06% of all effect sizes, precluding reliable outcome-specific analyses on actual calculated recruitment rates), but most studies included information sufficient to calculate a retention rate (74.18% of all effect sizes). Across all studies, the average retention rate was moderate ($M = 80.76\%$; $Mdn = 86.53\%$; $SD = 21.31\%$). Retention varied significantly across outcome types, $F(2, 2,236) = 339.66$, $p < .001$, increasing from suicide ideation ($M = 69.94\%$; $SD = 24.77\%$) to suicide attempt ($M = 76.45\%$; $SD = 19.99\%$) to suicide death outcomes

($M = 95.32\%$; $SD = 9.06\%$). Retention may be extremely high for suicide death outcomes because researchers typically relied on national death registries for follow-up information rather than continued direct contact with participants. Because only a subset of studies provided retention rates, we tested the effect of calculated retention rates on STB prediction in separate meta-regressions for each outcome. All results reported below are odds ratio analyses; there were too few hazard ratio effect sizes to permit reliable analyses.

Study quality and suicide ideation prediction. In addition to codes for whether or not recruitment and retention rates were reported, study quality was assessed with suicide ideation assessment type (i.e., single-item vs. questionnaire/interview) and definition (i.e., unclear vs. passive, mixed passive/active, and active). Although no specific variable in the meta-regression analysis reached significance, the overall meta-regression model was significant ($Q[6] = 25.31$, $p < .001$). However, the model only accounted for a small proportion of the variance in weighted mean odds ratios for suicide ideation prediction ($R^2 = 0.04$). In a separate meta-regression on the subset of effect sizes with retention rate data ($n = 390$), higher retention rates were not significantly associated with suicide ideation prediction ($Q[1] = 0.45$, $p = .50$, $R^2 < .01$).

Study quality and suicide attempt prediction. Study quality was assessed with codes for recruitment and retention rate reporting as well as the suicide attempt assessment (unclear vs. single-item and questionnaire/interview) and definition codes (unclear vs. intent inferred and intent explicitly stated) described above. The meta-regression analysis showed that no specific variable reached significance and that the overall effect of the model was not significant ($Q[6] = 9.23$, $p = .16$, $R^2 < .01$). A separate meta-regression on the subset of effect sizes with retention rate data ($n = 1,045$) showed that higher retention rates were significantly associated with stronger suicide attempt prediction

($Q[1] = 5.93$, $p = .01$); however, this was a very small effect ($R^2 = 0.01$).

Study quality and suicide death prediction. Similar to the preceding analyses, study quality was assessed with a combination of codes for recruitment and retention reporting and the suicide death assessment (unclear vs. legal/medical documentation and family report) and definition codes (unclear vs. ambiguous deaths likely excluded and explicitly excluded) described above. Analyses indicated that no specific variable within the meta-regression reached significance and that the overall model was not significant ($Q[6] = 9.28$, $p = .16$, $R^2 < .01$). Within a separate meta-regression on the subset of effect sizes with retention rate data ($n = 510$), higher retention rates were not significantly associated with suicide death prediction ($Q[1] = 0.16$, $p = .69$, $R^2 < .01$).

Brief summary. Taken together, these findings indicate that study quality variables generally had little to no impact on STB prediction strength in the present meta-analysis. As discussed above, this may be due to a combination of the homogeneity of research designs and the restricted range of effect sizes in the present meta-analysis. Additionally, it may be that "high quality" studies obtain more *reliable* and *valid* results, but this does not necessarily mean that these studies will obtain *stronger* results.

Broad Risk Factor Categories and Prediction

Risk factor categories. Space limitations preclude a full examination of the thousands of specific constructs that have been tested as risk factors. To provide a general index of the magnitude of various types of risk factors, however, we distilled all effect sizes into 16 broad risk factor categories (see Table 2). We note here that no risk factor subcategory (e.g., depression symptoms) substantially deviated from the risk magnitudes of these 16 broad risk factor categories (e.g., internalizing psychopathology). This is illustrated by Table 4, which shows the top five subcategory predictors for each outcome. None of these subcategory predictors stood out as particularly strong, especially in terms of absolute odds, and most were not significantly different from one another (see Table 4).

Category popularity across time. The most popular broad risk factor categories were internalizing psychopathology, demographics, externalizing psychopathology, prior STBs, and social factors (see Table 5). These five categories accounted for 77.45% of all effect sizes, with internalizing psychopathology and demographics together accounting for 41.42% of effect sizes. Moreover, these five categories have been the top five most popular categories since the inception of longitudinal STB research, accounting for an increasing share of effect sizes each research era (see Table 5). This suggests that the field has primarily focused on the same risk factors for the past 50 years, with risk factors becoming increasingly homogenous over past five decades.

Risk factor categories and suicide ideation. Weighted mean odds ratio analyses for suicide ideation revealed that prior STBs was the strongest broad risk factor category, though risk factor magnitude for even this category was weak in an absolute sense (see Figure 11). No other category exceeded a weighted mean odds ratio of 2.0. In terms of subcategories, prior suicide ideation was by far the strongest predictor, and hopelessness was the only other predictor to exceed a weighted odds ratio of 3.0 (see Table 4).

Table 4

Top Five Subcategory Predictors (in Terms of Weighted Odds Ratio Magnitude) Across Each STB Outcome

Rank	Subcategory	wOR	(CIs)	Number of effect sizes
	Top 5 suicide ideation subcategories			
1	Prior suicide ideation	3.55	(2.64, 4.78)	22
2	Hopelessness	3.28	(1.49, 7.22)	6
3	Depression (diagnosis)	2.45	(1.39, 4.34)	11
4	Abuse history (any kind)	1.93	(1.59, 2.33)	16
5	Anxiety (diagnosis)	1.79	(1.34, 2.40)	25
	Overall wOR (all effect sizes)	*1.50*	*(1.47, 1.54)*	*572*
	Top 5 suicide attempt subcategories			
1	Prior NSSI	4.15	(2.89, 6.92)	8
2	Prior suicide attempt	3.41	(2.71, 4.30)	42
3	Screening instrument	2.51	(1.82, 4.36)	10
4	Axis II diagnosis (any kind)	2.35	(1.88, 2.93)	40
5	Prior psychiatric hospitalization	2.32	(1.58, 3.39)	14
	Overall wOR (all effect sizes)	*1.51*	*(1.49, 1.54)*	*1281*
	Top 5 suicide death subcategories			
1	Prior psychiatric hospitalization	3.57	(2.81, 4.53)	31
2	Prior suicide attempt	2.24	(1.69, 2.97)	19
3	Prior suicide ideation	2.22	(1.45, 3.41)	10
4	Socioeconomic status (lower)	2.20	(1.32, 3.67)	10
5	Stressful life events	2.18	(1.63, 2.93)	23
	Overall wOR (all effect sizes)	*1.50*	*(1.46, 1.56)*	*912*

Note. CI = Confidence interval; STB = Suicidal thoughts and behaviors; NSSI = nonsuicidal self-injury; wOR = weighted odds ratio. To be eligible for inclusion on these lists, a subcategory need to be statistically significant and to include effect sizes from at least five different studies. For reference, *wOR*s calculated from all eligible effect sizes for each outcome are included. Although these risk factors are strong relative to other risk factors, they are weak in an absolute sense and should not be regarded as a list of "strong risk factors."

Risk factor categories and suicide attempt. Suicide attempt analyses revealed that weighted mean odds ratios for all categories were weak in an absolute sense, with no broad risk factor category exceeding a weighted mean odds ratio of 2.37 (prior SITBs; Figure 12). A few categories clustered around a weighted mean odds ratio of 2.0 and several others clustered around 1.5 (see Figure 12). Despite being by far the most popular category of risk factors, internalizing psychopathology was a relatively weak category ($wOR = 1.69$). As shown in Table 4, subcategory analyses revealed that prior nonsuicidal self-injury was the strongest predictor, outpacing prior suicide attempts and screening instruments (which mostly consisted of questions about prior STBs); however, none of these top five subcategories were significantly different from one another.

Hazard ratio analyses produced similar results, with most categories clustering around a weighted mean hazard ratio of 1.50 and

Table 5

Top Five Broad Risk Factor Categories in Terms of Popularity (i.e., Proportion of Total Effect Sizes) Across Each Era of STB Research

	Pre-1985		1985–1994		1995–2004		2005–2014	
Rank	Category	% Effect sizes	Category	% Effect sizes	Category	% Effect sizes	Category	% Effect sizes
1	Demographics	29.73	Internalizing	29.89	Internalizing	28.26	Internalizing	22.81
2	Internalizing	14.86	Prior STBs	13.88	Externalizing	14.67	Demographics	19.14
3	Prior STBs	10.81	Demographics	11.03	Prior STBs	11.85	Externalizing	16.02
4	Externalizing	9.46	Externalizing	10.68	Demographics	11.85	Prior STBs	11.52
5	Social Factors	5.41	Social Factors	9.25	Social Factors	8.37	Social Factors	9.61
Total		70.27		74.73		75.00		79.10

Note. STBs = Suicidal thoughts and behaviors.

only two categories exceeding 2.0 (see Table 6). The strongest overall category was prior psychiatric treatment, though it should be noted that this estimate may be unreliable as this category only included nine effect sizes for these analyses. Diagnostic accuracy analyses continued this pattern, with no category exceeding a weighted AUC of .61 (see Table 6). In short, no category improved predictive accuracy much beyond random guessing.

Risk factor categories and suicide death. Weighted mean odds ratio analyses for suicide death found that only one category generated a magnitude above 2.0—prior psychiatric treatment history (see Figure 13). All other categories clustered around a weighted mean odds ratio of 1.50, including popular categories such as demographics, internalizing psychopathology, and prior STBs. Subcategory analyses showed that prior psychiatric hospitalization was the strongest overall predictor of suicide death, exceeding the magnitudes for prior suicide attempts and ideation

(see Table 4); however, none of these top five subcategory predictors were significantly different from one another.

Analyses for hazard ratios generated similar results, with most categories clustering around a weighted hazard ratio of 2.0 (see Table 6). Diagnostic accuracy analyses once again revealed poor prediction, with few categories reaching significance and only two statistically significant categories exceeding weighted AUCs of .60—social factors and prior psychiatric treatment history (see Table 6). No category exceeded a weighted AUC of .67 (see Table 6).

Protective Factor Analyses

Descriptive statistics. After removing outliers and redundant effect sizes, 495 effect sizes were categorized as protective factors. These effect sizes were accordingly analyzed separately from risk

Figure 11. Weighted odds ratios by broad risk factor categories for suicide ideation outcome effect sizes. Error bars represent 95% confidence intervals. Broad risk factor categories are presented only if there were at least five eligible effect sizes in the category. Prior STBs = prior self-injurious thoughts and behaviors; Family History = family history of psychopathology or STBs; Internalizing = internalizing psychopathology; externalizing = externalizing psychopathology.
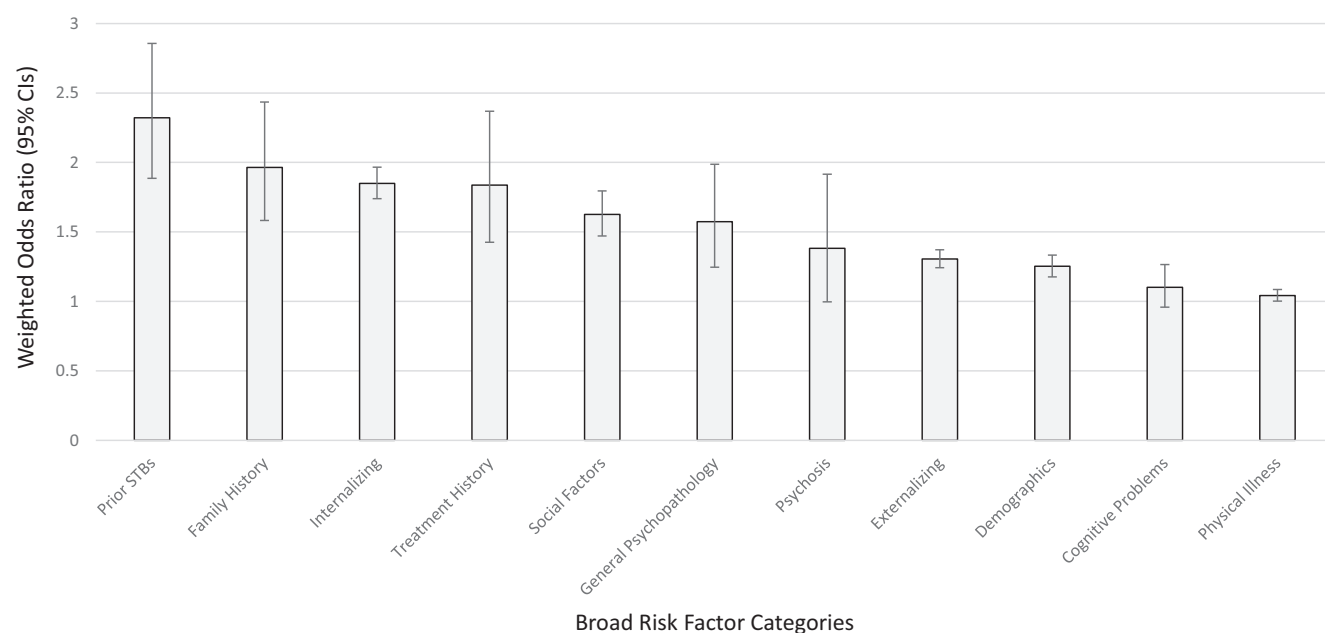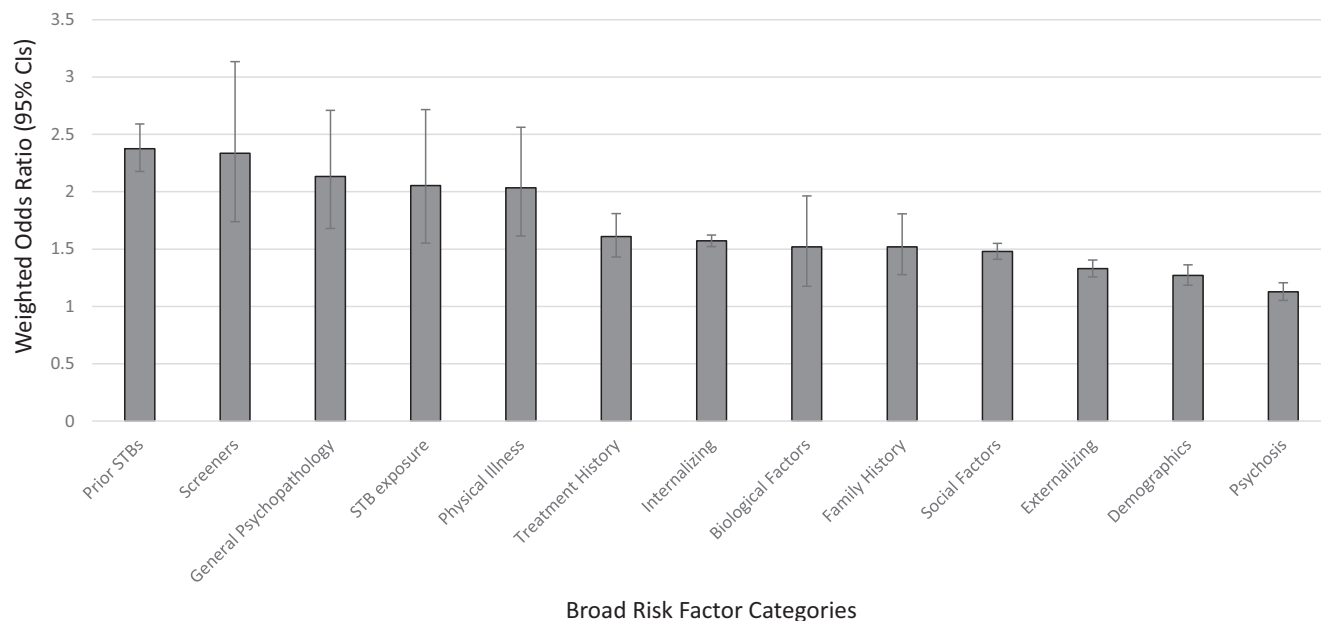
Figure 12. Weighted odds ratios by broad risk factor categories for suicide attempt outcome effect sizes. Error bars represent 95% confidence intervals. Broad risk factor categories are presented only if there were at least five eligible effect sizes in the category. Prior STBs = prior self-injurious thoughts and behaviors; Screeners = STB-related screening instruments; STB exposure = exposure to self-injurious thoughts or behaviors; Internalizing = internalizing psychopathology; Family History = family history of psychopathology or STBs; Externalizing = externalizing psychopathology.

factor effect sizes. These protective factor effect sizes generally followed the same descriptive pattern as risk factor effect sizes, with comparable follow-up lengths (*M* = 112.82 months; *SD* = 121.96 months; *Mdn* = 72 months) and numbers of self-injury participants (*M* = 231.78 participants; *SD* = 903.13 participants;

Mdn = 67 participants). Few protective factor effect sizes were published before 1985 (*n* = 3; 0.61% of effect sizes), with effect sizes increasing sharply between eras of 1985 to 1994 (*n* = 32; 6.46% of effect sizes), 1995 to 2004 (*n* = 109; 22.02% of effect sizes), and 2005 to 2014 (*n* = 351; 70.91% of effect sizes).

Table 6

Weighted Hazard Ratio and Diagnostic Accuracy Results Across Suicide Attempt and Death Outcomes by Each Broad Risk Factor Category

	Suicide attempt				Suicide death			
Category	*n*	*wHR* (95% CI)	*n*	*wAUC* (SE)	*n*	*wHR* (95% CI)	*n*	*wAUC* (SE)
Biology	4	—	14	.61* (.03)	9	1.30 (.99, 1.69)	16	.58 (.05)
Screeners	1	—	1	—	—	—	3	—
Cognitive problems	—	—	—	—	—	—	1	—
Demographics	19	1.52 (1.26, 1.82)	34	.55* (.02)	126	1.33 (1.23, 1.44)	66	.55* (.02)
Externalizing	37	1.37 (1.24, 1.42)	44	.57* (.02)	49	1.57 (1.32, 1.87)	33	.46 (.07)
Family history	2	—	17	.57* (.02)	2	—	14	.53 (.04)
General Psychopathology	5	1.02 (.90, 1.15)	20	.60* (.03)	10	2.51 (1.49, 4.24)	12	.64 (.07)
Implicit/explicit	—	—	1	—	3	—	—	—
Internalizing	50	1.17 (1.12, 1.22)	106	.59* (.02)	38	1.71 (1.56, 1.88)	94	.55* (.02)
Normal personality	—	—	—	—	—	—	1	—
Physical illness	2	—	4	—	35	1.78 (1.49, 2.12)	12	.61 (.07)
Psychosis	4	—	23	.49 (.05)	2	—	22	.61 (.07)
Prior SITBs	26	1.25 (1.17, 1.34)	52	.61* (.02)	35	2.82 (2.22, 3.60)	42	.59* (.03)
SITB exposure	—	—	1	—	—	—	—	—
Social factors	33	2.10 (1.73, 2.55)	40	.61* (.02)	29	1.17 (.99, 1.38)	25	.66* (.03)
Treatment history	9	2.74 (1.65, 4.55)	15	.51 (.05)	8	2.70 (1.79, 4.08)	17	.67* (.06)

Note. *wHR* = weighted hazard ratio; *wAUC* = weighted area under the curve. Confidence intervals for *wHR*s that did not include 1.0 were statistically significant. As with odds ratio analyses, only analyses that included at least five effect sizes are presented.
* A statistically significant weighted AUC.
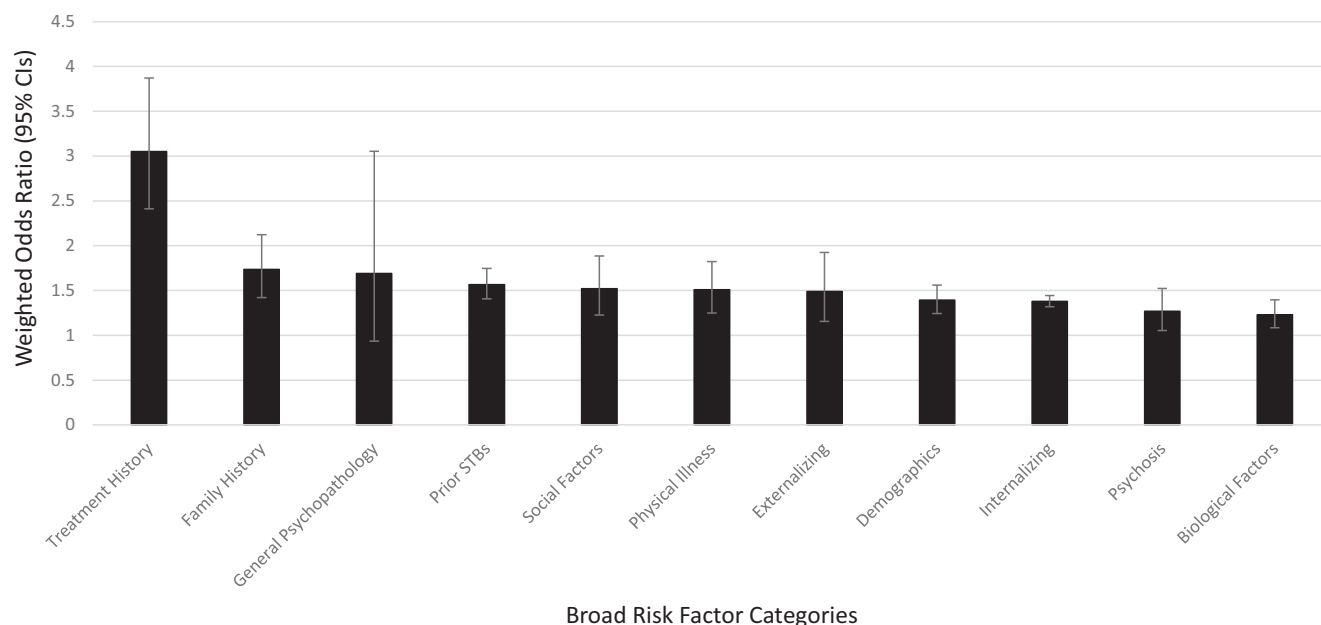
Figure 13. Weighted odds ratios by broad risk factor categories for suicide death outcome effect sizes. Error bars represent 95% confidence intervals. Broad risk factor categories are presented only if there were at least five eligible effect sizes in the category. STB exposure = exposure to self-injurious thoughts or behaviors; Family History = family history of psychopathology or STBs; Prior STBs = prior self-injurious thoughts and behaviors; Externalizing = externalizing psychopathology; Internalizing = internalizing psychopathology.

Protective factor effect sizes included a high proportion of suicide attempt (42.63% of outcomes) compared with suicide death outcomes (27.47% of effect sizes) and suicide ideation outcomes (26.46% of effect sizes); very few effect sizes included suicide plan as an outcome (3.43% of effect sizes). Most protective factor effect sizes were examined in general (52.86% of effect sizes) or clinical samples (35.70% of effect sizes), with many fewer in STB samples (11.44% of effect sizes). Similarly, the majority of protective factor effect sizes were examined in adult samples (54.75% of effect sizes), with many fewer effect sizes from adolescent (10.51% of effect sizes) or mixed age samples (34.75% of effect sizes). In terms of broad categories, 55.56% of effect sizes were classified as demographic variables and 14.14% were classified as social factors; no other categories exceeded 4.84%. Because there were so few category-specific effect sizes across data types (i.e., odds/hazard ratios) and outcomes, reliable protective factor category analyses could not be performed.

Protective factors and suicide ideation prediction. Odds ratio analyses for suicide ideation revealed a small but significant protective effect ($n = 126$; $wOR = 0.93$; CI: 0.90, 0.96; note: converse to risk factor effects, an OR/HR significantly below 1.0 indicates a significant protective factor effect). There were too few effect sizes to conduct hazard ratio or diagnostic accuracy analyses.

Protective factors and suicide attempt prediction. Protective factor effect sizes generated a weighted mean odds ratio for suicide attempt that was slightly but significantly below 1.0 ($n = 184$; $wOR = 0.93$; CI: 0.91, 0.95). The weighted mean hazard ratio was similar in magnitude ($n = 27$; $wHR = 0.93$; CI: 0.89, 0.96). Diagnostic analyses also indicated a slight protective effect, but this did not reach significance ($n = 36$; $wAUC = 0.48$, $SE = 0.03$).

Protective factors and suicide death prediction. Analyses revealed that protective factors did not produce a weighted mean odds ratio that was significantly below 1.0 ($n = 76$; $wOR = 0.95$; CI: 0.81, 1.11). In contrast, hazard ratio analyses did indicate a slight protective effect ($n = 60$; $wHR = 0.94$; CI: 0.91, 0.98). Echoing odds ratio analysis findings, protective factors did not produce a significant $wAUC$ ($n = 29$; 0.45, $SE = 0.03$).

Discussion

Suicidal thoughts and behaviors are among the most common, deadly, and potentially preventable public health problems. Despite major advances in medical and psychological science, the devastating impact of this problem has remained constant for at least several decades (see CDC, 2014; Kessler et al., 2005; McKeown et al., 2006; Nock et al., 2008). Knowledge about STB risk factors is essential for crafting scientific theories, accurate risk assessments, and effective treatments. Each day, thousands of clinicians rely on a half century of risk factor research to inform critical decisions about suicide risk and treatment. The primary purpose of the present meta-analysis was to estimate the power and accuracy of these risk factors.

These analyses produced several unexpected findings that may be surprising to many researchers and clinicians. Chief among them was the finding that, *at least within the narrow methodological limits of the existing literature,* existing risk factors are weak and inaccurate predictors of STBs. Analyses also revealed the following: predictive ability has not improved over the past 50 years; most studies included very long follow-up intervals (~5–10 years), but longer intervals were not associated with improved predictive ability; predictive ability was slightly better when a general

sample reference group was used; risk factor categories have been homogenous and have become increasingly homogenous over time; no risk factor category or subcategory is substantially stronger than any other; there is no compelling evidence that any specific STB outcome is associated with a unique set of risk factors; and protective factors are rarely studied and are generally weak.

These findings unfortunately have much greater implications for future research than for current suicide theory, prediction, and treatment. This meta-analysis was unable to establish the most promising future theoretical directions because, despite hundreds of studies across several decades, the available data were not sufficient to evaluate most hypotheses and theories about suicide. When relevant data were available for a particular approach (e.g., biological risk factors, hopelessness), study methods were usually too constrained to provide a helpful evaluation of a theory or hypothesis. Likewise, this meta-analysis was unable to provide clarity about risk factor and warning sign guidelines. As noted above, no broad or specific risk factors stood out as particularly strong and all risk factors have been evaluated within narrow methodological limits.

It follows that this meta-analysis also cannot directly inform STB treatment and prevention efforts. Because the meta-analytic data could not winnow theories or risk factors, it cannot provide much useful information about treatment and prevention targets. The limited ability of this meta-analysis to inform suicide theory, prediction, and treatment emanates from one major source: the methodological limitations of the existing literature. This meta-analysis accordingly represents a clarion call to researchers to modify their methods in a way that allows STB risk factor research to have greater implications for suicide theory, prediction, and treatment. Below, we discuss specific meta-analytic findings in greater detail and provide recommendations for future research.

Overall Risk Factor Effects

Given the degree of difficulty involved in conducting a longitudinal STB study, this literature produced a surprisingly high number of qualifying studies ($n = 365$), which in turn produced 3,428 risk factor effect sizes. Unfortunately, this large amount of research did not produce strong overall risk factor effects. Weighted mean odds and hazard ratios were around 1.50 for all outcomes and dropped significantly when publication bias was taken into account. These small effects were not meaningfully moderated by outcome definition (e.g., type of ideation), outcome assessment strategy (e.g., single-item vs. questionnaire/interview), or study quality (e.g., retention rate). As discussed in the next section, these overall effects also were not meaningfully moderated by broad risk factor categories (e.g., internalizing psychopathology) or specific risk factors (e.g., depression). In terms of clinical significance, assuming that these weighted odds ratio figures would apply on a population level, these combined risk factor effects would increase the 1-year odds of suicide death from 0.013 to 0.019 per 100 people; suicide attempt from 0.33 to 0.49 per 100 people; and suicide ideation from 2 to 3 per 100 people. These may not represent clinically significant effects, especially when considering clinicians often are tasked with determining STB risk over the course of hours, days, or weeks rather than over an entire year.

Diagnostic accuracy analyses were consistent with these findings. There were too few eligible effect sizes for suicide ideation analyses, but diagnostic accuracy was only slightly above chance for both suicide attempt prediction ($wAUC = 0.58$) and suicide death prediction ($wAUC = 0.57$). Sensitivity (i.e., true positive rate) analyses showed that risk factors rarely correctly identified instances of suicide attempt (26% correct) or suicide death (9% correct). At first glance, specificity (i.e., true negative rate) was much better, with acceptable levels for both suicide attempt (75% correct) and suicide death (81% correct). However, these rates appear to be inflated by a methodological artifact of crossing a low base rate predictor (e.g., prior suicide attempt) with a low base rate outcome (e.g., suicide death). In such scenarios, most participants will test negative for the predictor (e.g., will not have a suicide attempt history) and most also will test negative for the outcome (e.g., will not die by suicide). As a result, even if the predictor is not strongly related to the outcome, there will be a high true negative rate in these scenarios. It is notable that simulations showed that predicting solely according to base rates may be comparable to prediction with current risk factors. Compared with prediction with risk factors, prediction with base rates produced poorer sensitivity rates (~ 0–2% correct) and better specificity rates (~ 98–99% correct). In any case, both strategies produce poor STB prediction.

These small effects across odds ratio, hazard ratio, and diagnostic accuracy analyses may be slightly optimistic estimates of effect size two reasons. First, we used unadjusted effect sizes whenever possible ($\sim 82\%$ of effect sizes). Adjusted effect sizes, which control for various factors and more effectively isolate the contribution of a specific risk factor, were significantly smaller than unadjusted effect sizes. Second, we only included published studies and only calculated publication bias adjustments for overall odds and hazard ratio analyses (including these adjustments for all analyses would have been unwieldy and largely redundant with overall publication bias analyses). These publication bias adjustments substantially reduced the effect sizes of risk factors for suicide ideation ($\sim 40\%$ reduction) and suicide attempt ($\sim 50\%$ reduction).

Specific Risk Factor Effects

Composition of risk factors. Across our 16 broad risk factor categories, nearly 80% of risk factors fit into one of the top five most popular categories and nearly 95% of risk factors fit into one of the top nine most popular categories. Risk factors related to internalizing psychopathology accounted for nearly 25% of all effect sizes and risk factors related to demographics accounted for an additional 17%. This means that almost half of the 3,428 risk factor effect sizes fit into one of two categories. In short, STB risk factors have been very homogenous.

This homogeneity has been present across all eras of STB risk factor research. The top five most popular broad risk factor categories were the same in each era (though in slightly different orders). These five categories have always accounted for at least 70% of all risk factors, with this proportion approaching 80% in the last decade. These patterns suggest that the STB risk factor literature has not become increasingly diverse, followed novel theoretical directions, or systematically built on or innovated beyond earlier work. Rather, the set of risk factors from pre-1985

studies is virtually indistinguishable from the set of risk factors from 2014 studies, indicating that the field developed a narrow set of potential risk factors early on and stuck to them. There were, of course, some exceptions to this rule, but these have represented a small minority of effect sizes. By and large, the STB risk factor field appears to have conducted essentially the same studies over and over again throughout the last 50 years. In light of this pattern, it is not surprising that predictive ability has remained nearly constant over the last 50 years. Similarly, in light of these two patterns, it should not be surprising that STB rates have also remained nearly constant over the last 50 years.

Strength of particular risk factors. Although overall risk factor magnitude and accuracy were weak (see above), we hypothesized that certain broad risk factor categories would stand out as much stronger than others. Results did not support this hypothesis. Even the strongest categories did not exceed weighted mean odds ratios of 2.5 for any outcome, and all categories clustered within a tight range of 1.0 to 2.5. Interestingly, the most popular risk factor categories tended to be among the weakest. Internalizing psychopathology, demographics, externalizing psychopathology, and social factors together accounted for 66% of all risk factors; however, they tended to be among the weakest half of categories for each outcome. The reason for the popularity-strength discordance is unclear. One possibility is that these categories remain popular because of tradition. Another possibility is that, because these categories are strong correlates of STBs, researchers have assumed that they are also strong risk factors. Regardless of the reason for this discordance, the present findings show a need for novel risk factors.

After obtaining these category-level results, we speculated that these broad categories may have concealed a few powerful subcategories of risk factors. For example, although internalizing psychopathology as a whole is not a strong STB risk factor, it may be that depression, posttraumatic stress disorder, or similar subcategories stand out as especially powerful risk factors. Results did not support this hypothesis. Even the top five most powerful risk factor subcategories were weak in absolute sense (see Table 4). Across all outcomes, only four of these subcategories exceeded a weighted mean odds ratio of 3.0 (three were prior STBs, one was prior psychiatric hospitalization) and few were significantly different the rest of the top five. Taken together, these findings indicate that, *at least within the narrow methodological limits of the existing literature*, there is no evidence that any known risk factors—broad or specific—approach what many might define as clinical significance.

Outcome-risk factor specificity. As noted above, researchers have long been interested in the possibility that each STB outcome may be associated with at least a partially unique set of risk factors. Although we tested this intriguing hypothesis in multiple ways, the present meta-analysis found no evidence for any such distinctions within the existing literature. First, the most general test of this hypothesis found that combined risk factor magnitudes and accuracies were almost identical across suicide ideation, attempt, and death outcomes. As risk factor types were similar across all outcomes, this indicated that, at least in a highly general sense, there were likely to be few risk factor differences across outcomes.

Second, a more specific test of this hypothesis showed that broad risk factor categories tended to display similar magnitudes and accuracy across outcomes. In general, magnitudes rarely var-

ied more than 0.20 and confidence intervals almost always overlapped. For example, the demographics category displayed similar magnitudes across suicide ideation ($wOR = 1.25$; CI: 1.18, 1.33), attempt ($wOR = 1.27$; CI: 1.19, 1.36), and death outcomes ($wOR = 1.39$; CI: 1.24, 1.57). Third, an even more specific test of this hypothesis revealed that risk factor subcategories were also consistent across outcomes. Prior STBs tended to be the strongest subcategories, followed by factors that signified elevated levels of general psychopathology, prior STBs, and stress (e.g., prior psychiatric hospitalization, screening instruments, stressful life events; see Table 4).

Considering all of these findings together, there appears to be no evidence of outcome-risk factor specificity. It may be tempting to interpret some of the small differences across outcomes as having meaningful implications. As emphasized above, however, we note here that all risk factors were weak in magnitude and that any differences across outcomes (or even categories) are not likely to be highly meaningful. Also as emphasized above, however, we do not take these results as proof that there are no meaningful differences between outcomes or categories. Rather, we strongly believe that there are many extremely important differences but that these differences have been obscured by the significant methodological limitations of the existing literature. Correspondingly, we stress that the present null outcome-risk factor specificity findings only apply to these associations *within the narrow methodological limits of the existing literature*.

Moderator Effects

We investigated several methodological features as potential moderators of general risk factor magnitude and accuracy. Although overall predictive ability and predictive ability across specific risk factors was weak, it was possible that certain methodological features were associated with improved prediction. Given the restricted range of effect sizes in the existing literature (see Figure 5), however, any moderation effects were likely to be modest.

Risk factor effects across time. Since 1985, the number of qualifying papers and effect sizes nearly doubled every 10 years. More than half of all papers and effect sizes were published after 2007 and, if these growth trends hold, the 2015 to 2024 decade will produce more STB risk research than all pre-2015 years combined. The sharp increase in research was not accompanied by a sharp increase in predictive ability. Weighted odds ratios, hazard ratios, and diagnostic accuracy statistics were highly consistent across eras and, if anything, predictive ability diminished slightly over time. These findings suggest that, as a whole, STB risk factor research has not been progressive—later research has not built on earlier research, there has been little innovation, and knowledge has not steadily accrued. Only 3.29% of eligible papers and only 1.93% of eligible effect sizes were published before 1985; yet, if the present meta-analysis had been conducted in 1985, it would have yielded nearly identical findings. This fact signals the need for major changes in STB risk factor research.

Follow-up length. Studies were very long on average. The mean follow-up length was nearly 10 years and the median follow-up length was five years. A much higher proportion of effect sizes had follow-up lengths of longer than 10 years (29%) than lengths of six month or shorter (5%). Less than 1% of effect

sizes had follow-up lengths of one month or shorter. These findings show that there are very few studies on short-term or acute STB risk, which in turn means that there are very few longitudinal studies of "warning signs" for suicidal behavior. This is concerning given that clinicians are often asked to make decisions about acute risk rather than decisions about risk over months, years, or decades.

Presumably, most studies have had long follow-up lengths because researchers aimed to capture as many STB events as possible and because they reasoned that longer lengths may translate into better predictive ability. Results did not support this possibility as weighted odds ratios, hazard ratios, and diagnostic accuracy statistics were consistent across all follow-up length intervals. These findings suggest that, at least in terms of predictive ability, there is no compelling reason to a conduct STB study with a long follow-up interval. On the other hand, new studies with very short follow-up lengths may yield tremendous theoretical and empirical advances. In particular, the present findings show that the existing literature can only speak to risk factor magnitude and accuracy *over long intervals*. It is possible that risk factors, especially time-varying risk factors (vs. stable or trait-like risk factors), are exponentially stronger over very short intervals. For example, an intense relationship breakup may confer high STB risk for minutes, hours, or days. If a study examines this factor over the course of months, years, or decades, this initial high-risk "signal" may get lost in an ever-increasing cacophony of "noise."

Sample characteristics. There were large variations in the severity, age ranges, and sizes of samples across STB risk factor studies. General and clinical sample studies were much more common than STB sample studies, and this gap has grown over the last 20 years. Prediction tended to be stronger in general samples compared with clinical and STB samples, but these differences were small in an absolute sense. Likewise, there was no meaningful moderation of these effects based on type of clinical sample (e.g., depression, psychosis), type of STB sample (e.g., ideators, attempters), or clinical/STB sample origin (e.g., inpatient, community). Stronger predictive ability in general samples may have occurred because risk factors related to psychopathology, prior STBs, and social factors tend to be correlated. Reference groups in clinical and STB samples likely acted as controls for these correlations, which slightly reduced effect sizes. It follows that, although general samples tended to produce better predictive ability, they also produced more ambiguity about the specificity of risk factor effects. For example, if depression was found to be a significant risk factor in a general community sample, it may be unclear whether this effect was due to depression or psychopathology more generally. If this same effect were found in a severe clinical sample, there could be greater confidence this effect was due to depression.

Adolescent sample studies have increased in recent decades, but adult sample studies have increased more sharply and have always accounted for the majority of prediction effect sizes. There was a notable scarcity of adolescent sample effect sizes with suicide death as an outcome. Predictive ability was comparable across adult, mixed, and adolescent samples for all outcomes. This indicates that there are few meaningful age-related differences in risk factors from the existing literature. However, we again caution that this conclusion only apples to STB risk factors *within the narrow methodological limits of the existing literature*. Future studies that examine more innovative risk factors with improved methods (see below) may detect important risk factor differences among these age groups.

The size of studies in terms of STB participants increased sharply in the mid-1990s and then leveled off. Over time, very small (1 to 25 participants) and very large (500+ participants) studies have become less common, and moderately sized studies (26 to 500 participants) have become more common. The median number of STB participants, 57, has remained relatively small across the eras of suicide risk factor research. Analyses revealed that risk factor magnitude and accuracy were comparable across all sample sizes for all outcomes. These findings suggest that extremely large samples (e.g., population-level samples or national survey samples) carry no benefit in terms of prediction strength. But because larger samples more accurately estimate true effects (even if those effects are small), future studies would benefit from including at least moderately large samples (e.g., 100 to 500 participants), especially for suicide attempt and death outcome studies.

Protective Factors

There were many fewer protective factor effect sizes than risk factor effect sizes, and studies rarely set out a priori to investigate protective factors. The majority of these effect sizes were demographic factors that we coded as protective factors based on their expected associations with each outcome according to epidemiological statistics on STBs (see Method section). Protective factor effect sizes generally mirrored risk factor effect sizes, nearly doubling every decade and producing weak overall effects. These findings make clear the need for studies specifically designed to evaluate protective factors, particularly studies with factors that are not simply the inverse of risk factors (e.g., "no psychopathology," "no alcohol use vs. alcohol abuse," and "male gender predicting suicide attempt").

Limitations

This meta-analysis should be interpreted in light of its limitations. First, we were unable to accommodate the inclusion of studies that employed advanced statistical techniques, some of which included time-varying risk factors. Although these would have represented a small proportion of all studies, it is possible that their inclusion may have generated larger effect estimates. Second, many of the reported estimates may overestimate risk factor effects because we only included published studies and did not estimate publication bias within subanalyses (because this would have been unwieldy and largely redundant with overall analyses). Based on overall analyses, we estimate that effects for subanalyses are 40% to 50% weaker than reported for suicide ideation and attempt outcomes. Third, although we took multiple steps to reduce dependence among effect sizes (see Methods section), our primary analytic strategy treated each effect size within a study as if it were independent from all other effect sizes within that study (i.e., zero correlation among various types of risk factors in the same study). This analytic strategy slightly decreased confidence intervals and Type I error, increasing the likelihood of significant effects. Importantly, however, an analytic strategy that assumed and accounted for complete dependence among effect sizes within a

given study produced nearly identical results. Given that data in the present study are likely much closer to complete independence than complete dependence, we estimate that our primary analyses produced very slight increases in Type I error (i.e., slightly more significant effects than truly exist).

Fourth and most importantly, the overwhelming limitation of this meta-analysis reflects the overwhelming limitation of the existing STB risk factor literature: the methods of most existing studies have been extremely narrow and homogenous, and have not allowed for tests that approximate how STB risk may work in nature. This major limitation means that all of the present results should be interpreted with the caveat that these findings only apply to STB risk factors *within the narrow methodological limits within which STB risk factors have been studied for the past 50 years*. As a result, the present meta-analysis cannot support conclusions like "internalizing psychopathology is a weak STB risk factor." Instead, it can only support conclusions like "internalizing psychopathology is a weak STB risk factor *within particular methodological constraints, but it remains unknown if internalizing psychopathology is a strong or weak risk factor outside of these constraints*." Specifically, conclusions about the existing literature are constrained by at least three major methodological limits: (a) long follow-up intervals; (b) risk factors measured in isolation rather than in combination; and (c) risk factors measured in a static or trait-like fashion. That is, most existing studies have tested whether a single isolated factor measured at one moment in time predicts STBs over the course of years or even decades.

The poor predictive ability produced by this modal research design is consistent with evidence that the vast majority of people who possess a specific risk factor never engage in suicidal behavior. For example, mood disorders and prior psychiatric treatment (especially inpatient) are widely considered to be major risk factors for suicide death. However, the lifetime probability of suicide death is low (in an absolute sense) among mood disorder outpatients (2%) and mood disorder inpatients (4%; Bostwick & Pankratz, 2000); for reference, approximately 1.6% of the general population dies by suicide (CDC, 2014). Because 96% to 98% of mood disorder patients will *not* die by suicide, a mood disorder diagnosis in isolation is inherently limited in its ability to accurately predict future suicide death: this prediction will be wrong 96% to 98% of the time. The present meta-analysis highlights the need for studies that overcome the limitations inherent in using a single, inaccurate factor to predict STBs.

It is notable that the modal research design in the STB risk factor literature does not approximate most hypotheses or theories about STB risk. Although the specific content of these hypotheses and theories varies widely, most subscribe to a basic process whereby a subset of trait-like distal factors and highly volatile proximal factors combine in a complex way to raise STB risk for a few minutes, hours, or days. For example, few would expect hopelessness measured as an isolated trait-like factor to accurately predict suicide death over the course of a decade. But many might expect that, among older males who own a gun and have a prior history of self-injury and very little social support, a rapid elevation in hopelessness after the unexpected death of a spouse would greatly increase suicide death risk for a few hours or days. Yet, most of the existing literature has tested the former hypothesis rather than the latter. Viewed in this light, the present results may

not be surprising: most hypotheses and theories are actually consistent with these weak meta-analytic findings.

Summary

This meta-analysis found that, based on the existing literature, all STB risk (and protective) factors are weak and inaccurate. This general pattern has not changed over the past 50 years and was not meaningfully moderated by study characteristics (e.g., length, sample severity) or type of risk factor (e.g., internalizing psychopathology, prior STBs). These results may be surprising and disappointing to many researchers and clinicians, and may be easily misinterpreted. To help facilitate the interpretation of the present findings, below we articulate what these findings *do* and *do not* mean for research and practice. Most of these points stem from the importance of distinguishing between (a) the actual nature of STB risk and (b) the narrow band of this nature examined by the existing literature.

What the present findings *do not* mean. First, the present results do not mean that widely used STB risk guidelines (see Table 1) are invalid or useless. As emphasized above, the existing STB risk factor literature can only speak to STB risk within narrow methodological constraints. These constraints have greatly limited the ability of the existing literature to speak to the validity or utility of these guidelines. For example, suicide "warning signs" are widely used in clinical settings to gauge suicide risk. These warning signs are thought to indicate imminent suicide risk and often involve changes in factors (e.g., dramatic changes in mood, increased alcohol use, social withdrawal; see Rudd et al., 2006). Unfortunately, the existing literature has been unable to evaluate these warning signs because very few studies examined risk over a short time period (i.e., days or weeks) or examined changes in risk factors over time.

Second and similarly, the present findings do not mean that traditional risk factors and theories based on these risk factors have little relevance to STBs. As noted above, the existing literature has been unable to test most STB hypotheses and theories about risk factors.

Third, the present results do not mean that the field should lessen its focus on STB risk factors or prediction. Only a narrow band of the nature of STB risk has been examined. Failing to investigate the rest of this nature would be detrimental to the understanding, prediction, and prevent of STBs.

What the present findings *do* mean. First, the present results mean that existing STB risk guidelines are rationally derived and have not been appropriately evaluated by the existing literature. As most of these guidelines were produced by expert consensus, there is reason to believe that they may be useful and effective. Accordingly, we recommend that these guidelines remain in use, but emphasize that there is an urgent need to empirically evaluate these guidelines within longitudinal studies.

Second, traditional risk factors are poor predictors of STBs *within the narrow methodological constraints of the existing literature*, but it is unknown how these risk factors perform outside of these narrow methodological limits. Whatever the role of these risk factors, it is likely to be complex. The present results show that single risk factors (e.g., a depression diagnosis considered in isolation) are inherently limited in their ability to accurately predict future STBs. We speculate that the additive or interactive

effects of a small number of risk factors would also produce inaccurate prediction. Consistent with recent research (Kessler et al., 2015) and many hypotheses and theories about suicide, accurate STB prediction will likely require a complex combination of a large number of factors (i.e., >50), many of which are time-varying. Moreover, it is likely that there are many different paths to STBs. This equifinality means that a single one-size-fits-all algorithm for STB prediction is unlikely; researchers will likely need to develop separate algorithms different populations (e.g., adolescents; the elderly; inpatients; soldiers/veterans; prisoners; mood disorder patients; psychotic disorder patients; etc.).

Third, the present findings indicate a need for *more* STB risk factor studies, but clarify that these studies must overcome the methodological limitations of the existing literature. It may be helpful for future STB risk factors studies to be evaluated in part based on how they overcome these limitations. Similarly, it may be helpful for these future studies to be evaluated based on their clinical significance in addition to their statistical significance. In most fields, a factor that increases risk by 10% may be extremely important. But the low bases rates of STBs mean that even a statistically significant factor that increases suicide risk by 1,000% may not be clinically significant. In short, the present meta-analysis suggests that, to advance knowledge about STB risk, we must change our approach to conducting and evaluating studies.

Future Directions

The present findings show a clear need for studies that are capable of testing complex hypotheses about STB risk. We propose four suggestions for future directions that may facilitate such studies. First, studies should include short follow-up intervals—on the order of minutes, hours, or days. Second, studies would benefit from repeatedly or continuously measuring constructs. For example, it would be helpful to measure rapid increases or decreases in hopelessness rather than trait hopelessness. Third, there is a need for novel risk factors. Several such factors have recently been proposed, but they have rarely (e.g., implicit identification with death/suicide; Nock et al., 2010) or never been directly tested as risk factors (e.g., suicidal capability; Joiner, 2005). Fourth and finally, risk factors should be combined in a complex but replicable manner. In other words, we recommend that the field shift from a focus on risk *factors* to a focus on risk *algorithms*.

Machine learning approaches are ideal for this latter recommendation (see Ribeiro et al., 2016). Traditional statistical techniques (e.g., regression) are helpful for testing relatively simple hypotheses about STBs (e.g., Diathesis × Stress models), and machine learning approaches confer little advantage for smaller data sets that include few predictors. As indicated by the present meta-analysis, however, accurate STB prediction may require models that consider complex relationships among hundreds of predictors. Within sufficiently large data sets, distinct advantages of the machine learning approach emerge, including the following: (a) the machine determines the optimal algorithm for prediction (vs. a human deciding which variables should be included and what the relationship among the variables should be); (b) many techniques can model highly complex relationships among predictors, going far beyond traditional additive, interactive, and linear models; and (c) these techniques are often applied with generalizability in mind, with most studies including a range of strategies to prevent

overfitting (e.g., cross-validation, bootstrap aggregating). In short, these techniques have the potential to produce models that accurately reflect the complex nature of STB risk. Just as Internet search algorithms require the consideration of hundreds of variables to accurately answer a search query, complex machine learning algorithms are likely necessary for accurate STB prediction. Pioneering retrospective work showed the promise of this approach (e.g., Delgado-Gomez et al., 2012; Lopez-Castroman et al., 2011; Mann et al., 2008), with later prospective work confirming the utility of this approach for suicide prediction (Kessler et al., 2015).

The four suggestions outlined above were nearly impossible to heed until recent advances in technology. These new technologies make it possible to repeatedly assess risk factors and STBs on the scale of minutes or hours rather than years or decades. Recent advances also make it easy to create mobile app versions of novel cognitive and affective tasks traditionally confined to laboratory studies (cf. Franklin et al., 2016). Similarly, it is now possible for most researchers—even those with no computer science knowledge—to employ machine learning algorithms to form complex (but robust and replicable) combinations of a large number of potential risk factors within large data sets. Perhaps most critically, the proliferation of powerful mobile technologies now makes it possible to quickly and economically recruit thousands of individuals from around the world who may be at elevated risk for STBs. This represents a democratization of STB risk factor research whereby most researchers can now conduct studies with the suggested methods without need of large grant funding, several research assistants, or connection to a large psychiatric hospital.

This meta-analysis brought to light many concerning patterns. In doing so, however, it also provided a clear path toward progress. Studies that leverage recent technological advances and more closely approximate theories about STBs have the potential to produce rapid advances in knowledge. Such knowledge would bring about much needed advances in understanding, predicting, and preventing STBs.

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