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What suicide interventions should target

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Recent reviews and national statistics indicate that, so far, our field has made limited progress on fulfilling its central mission of preventing future suicidal thoughts and behaviors (STBs). We posit that a fundamental reason for our lack of progress is the way in which our field tends to think about and select STB intervention targets. Specifically, the vast majority of our intervention targets are derived from untested theoretical assertions, moderate correlates of STBs, or weak risk factors for STBs. None of these forms of evidence permits causal inferences, which is problematic because successful STB interventions must target the causes of STBs. To develop effective interventions, we must employ experimental designs to identify targets that are causal, necessary, and viable.

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How can we keep suicidal thoughts and behaviors (STBs) from happening? This is perhaps the most central and difficult question in suicide research. After several decades of work, we have made little progress in answering this question. Recent reviews indicate that few interventions consistently reduce STBs in comparison to an active control group [1,2,3••,4] and that some of our most promising interventions — cognitive-behavioral approaches — are much less promising when publication bias is considered [5]. Our lack of success is reflected in continued high STB rates, especially in the United States. The United States suicide rate in the year 1900 was approximately 10 per 100 000 people, which was also the rate in the year 2000 [6,7]. Since then, rates have steadily climbed to over 13 suicide deaths per 100 000 people [6]. The rates of suicidal thoughts, plans, and nonfatal attempts have followed a similar pattern [8]. Over the past several decades, research and prevention efforts have increased dramatically in an effort to address this international public health problem [1,2,3••]. So why have we made so little progress on figuring out how to keep suicidal behaviors from happening?

There are many reasons for this, including the limited scalability, affordability, and accessibility of most traditional interventions [9,10]. But we propose that an even more fundamental issue has been central to obstructing progress in STB interventions: how we think about and select STB intervention targets. Contained within the Discussion Sections of a large proportion of research articles in our literature is some variant on the conclusion that '*these findings suggest that future interventions should target X*.' But do a large proportion of our studies actually allow for this conclusion? Exactly what kind of evidence would we need to make this kind of conclusion? In other words, what should suicide interventions target and what is the best way to identify those targets?

As a field, we have rarely thought through these kinds of questions, and this has led to a haphazard and largely ineffective approach to identifying suicide intervention targets. This is true of traditional interventions and of the most recent and novel STB interventions, including our own [11]. Such novel interventions may show some promise and may improve on traditional scalability limitations, but they still suffer from a core deficit: a target that has an unclear, little, or no causal role in STBs. Even in the best-case scenario of an initially promising intervention, this deficit can prevent progress for decades by making it nearly impossible to effectively refine the target or the way in which the intervention attacks the target. As a result, we expend tremendous resources grasping in the dark for ways to improve an intervention and often see effect sizes decline over time. In other words, it is nearly impossible to optimize *how* to target a given factor in the absence of proper evidence about *which* factors should be targeted. Across the many decades of STB intervention research, we are aware of no intervention that has improved since its inception. This problem is not unique to suicide intervention research. Within the much larger research field aimed at improving cognitive behavioral therapy for depression, meta-analytic evidence indicates that its efficacy has substantially diminished (rather than improved) over the decades [12•].

In the hopes of establishing a more strategic pathway for identifying effective STB intervention targets, we will

briefly outline the limitations of traditional approaches and then describe what suicide interventions should target and how to identify those targets.

Traditional intervention target identification approaches

A common but generally inadvisable approach to identifying intervention targets is to identify STB correlates. Especially when consistent with a popular theory, this rationale can seem convincing. However, even two highly correlated phenomena may have little or nothing to do with one another. For example, between 1999 and 2009, the correlation between United States suicides by suffocation and the United States budget allocation for science and technology was nearly perfect ($r = .99$) [6,13]. Few people would take this evidence to mean that we should reduce spending on science and technology to reduce suicide. Yet, if the same correlation existed between suicide and hopelessness, many may consider this sufficient rationale to target hopelessness within suicide interventions. Regardless of their strength, consistency, and relevance to a given theory, correlates provide little useful information about intervention targets.

Risk factors (i.e. longitudinal predictors) are more compelling than correlates, but they still fall far short of sufficient evidence for designating a given factor as an intervention target. Risk factors provide some evidence for directionality within an association, but it is important not to conflate longitudinal prediction with cause [14]. A given risk factor may simply be correlated with a STB cause; as a result, targeting a risk factor may have no effect on STBs. For example, a history of psychiatric hospitalization is one of the single strongest risk factors for suicide death [15•], but this does not mean that psychiatric hospitalization causes suicide and that ending psychiatric hospitalizations would prevent suicide. Instead, it may be that psychiatric hospitalization is correlated with several potential causes of suicide. As with correlates, risk factors provide little useful information about intervention targets. This is especially true within STB research as recent meta-analyses have shown that no known risk factor, out of the hundreds tested, accurately predicts STBs [15•].

Unlike correlates and risk factors, certain causal factors may represent potentially promising intervention targets. Unfortunately, suicide research has produced little information about causal factors because experimental designs are necessary to make causal inferences. Although the term 'experiment' has been used to refer to a range of designs in our field, causal inferences can only be drawn from designs where the effects of deliberately introducing a manipulation in one group are compared to the effects of not introducing that manipulation in another group (i.e. the counterfactual dependence test of causation) [16]. Randomized controlled trials (RCTs) are experiments, and hundreds of STB RCTs have been conducted; however, none of these have identified intervention targets because: first, they presuppose a target and do not directly test STB causes (i.e. they test the causal effect of an intervention); second, any conclusions from RCTs about STB causes would be based on problematic *ex juvantibus reasoning* [17], which involves reasoning backwards from an effective treatment (e.g. if aspirin relieves a headache, concluding that the headache was caused by insufficient aspirin in the brain); and third, RCTs indicate that existing interventions (and by extension, their targets) are largely ineffective [3••]. Few experiments have been aimed at directly testing potential causes of suicide ideation [18–21] and we are aware of none that have directly tested potential causes of suicidal behavior.

By this metric, most STB theories — which are primarily theories about STB causes — have not been directly tested. Thousands of studies have concluded that they have found support for various STB theories with correlational or longitudinal evidence. But this kind of evidence is very limited because it leaves open so many alternative explanations for construct–suicide associations that it is impossible to justify even the most tenuous of causal inferences. It follows that most of our theories, and the intervention targets deduced from those theories, have little empirical verification or refutation because they have not been directly tested within experimental designs. This helps to explain how so many different STB theories currently co-exist [15•] — without direct tests of causal hypotheses, falsification evidence cannot be used to winnow the theories. Likewise, this helps to explain the limited efficacy of existing interventions: these interventions primarily target moderate correlates and weak risk factors, none of which have been shown to cause STBs.

Complications to identifying intervention targets

We believe that future work should focus heavily on experimental designs to elucidate STB causes; however, three issues should be kept in mind when conducting and interpreting this work. First, experimental designs cannot prove that a particular factor causes STBs [22], they can only help to rule out potential alternative explanations for an association (e.g. that a given factor is a concomitant). More stringent experimental designs (e.g. randomized groups, powerful control manipulations), larger sample sizes, and consistent findings across studies can rule out more alternative explanations and correspondingly can justify stronger causal inferences.

Second, there are obvious ethical limitations to conducting experiments aimed at increasing STBs, but such studies are still possible. Through careful procedures, multiple labs have conducted experiments aimed at testing the potential causes implicit and explicit suicidal thoughts [20,21]. Although experimental studies of