Explaining Rare Acts of Violence: The Limits of Evidence From Population Research

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After the tragic mass shooting in Tucson, experts struggled to explain why such horrific events occur, in order to prevent them in the future. The author argues that homicides perpetrated with firearms against strangers by individuals with mental disorders occur far too infrequently in the population to allow explanatory statistical modeling and predictability. However, from a public health perspective that seeks to reduce violence in populations, it is likely that efforts to improve treatment access, continuity, and adherence for people with serious mental illnesses will also prevent some violent episodes, even if it remains impossible to reliably predict which specific individuals would otherwise engage in the most serious acts of violence. (Psychiatric Services 62:1369–1371, 2011)

“Tucson” now settles in the collective memory of senseless rampages by troubled young men. The pundits are moving on, but their questions linger. Does mental illness make people violent? Was this a preventable tragedy? Experts are expected to answer such questions on the basis of solid research evidence. Their answers come up short. This Open Forum discusses some of the reasons why.

The rare-event problem
When researchers talk about violence and mental disorders in general—and about what they know of the prevalence and correlates of violence among people with serious mental illness in the community—they are not talking about mass shootings, which are extremely rare. They are talking largely about behaviors that are common enough to study systematically with representative samples, such as fist fights, pushing and shoving among family members, and sometimes threats made with weapons (1–3). Epidemiological studies cannot tell us what caused Jared Loughner to shoot a U.S. congresswoman and 18 other people. Structured risk assessment instruments could not have predicted it either. We do not possess the data to do this reliably (4).

The population-to-individual problem
The data that we have suggests that mental illness plays only a weak and minor role in explaining violence in populations. Of course, that statement is not necessarily true of any one individual. Some perpetrators of horrendous crimes clearly are driven by psychosis beyond responsible volition, which is the meaning of “not guilty by reason of insanity.” At the same time, there is a good reason that simply having a mental illness is not criminally exculpatory: the vast majority of people with mental illness do not commit violent crimes.

Asking whether mental illness causes violence is a bit like asking whether political dysfunction causes war. The two correct answers are “sometimes” and “it depends.” Research findings on the link between acts of violence and mental illness vary widely because study designs, sampling frames, operational definitions, and comparison groups also vary widely (5,6). Some studies may find high rates of violence in samples of former prisoners and of patients discharged from psychiatric hospitals mainly because violent behavior is the reason many people become prisoners and patients in the first place—and those are the populations to whom such studies generalize. And with respect to any sample, the question, “Are people with mental illness more violent, . . . ?” begs another question: “More violent than whom?” More violent than people without mental illness or substance abuse? More violent than the general population? More violent than residents of the same high-crime neighborhoods? Or more violent than the participants themselves at different points in time, such as before versus after treatment? Different questions, different answers.

Predicting and preventing violence
Nevertheless, when we consider ways to “prevent Tucson,” it is not unreasonable to ask, if science and medicine can explain and reduce other causes of mortality, why not this? If we can generalize to individual cases from average causal effects in clinical trials of, say, beta blockers and myocardial infarction, why can’t we do the same with population-level studies of violence and mental illness? Why can’t we apply general knowledge to save the next Virginia Tech from the next Cho?

Neither is it unreasonable to ask, if we can successfully triangulate what we know of such things as geological fault lines, tectonic plates, and frequent small temblors to learn why rare, massive earthquakes occur, what is so different about violence? Why can’t we take studies of garden-variety psychiatric symptoms, social dis-
advantage, alienation, addiction, trauma, and minor aggressive behavior and put them together to learn just why Jared Loughner shot 19 people in Tucson?

To be clear, we can and should sometimes apply general empirical knowledge to the individual case; this is the heart of evidence-based clinical practice in medicine. Indeed, with respect to the possibility of studying average causal effects and applying knowledge of a class to explain the particular, one need not distinguish in theory between heart attacks and firearm attacks (or nuclear attacks, for that matter). The important difference lies in the nature, quality, and amount of data available to do this reliably for one type of event versus another and in the feasibility of ever obtaining such data.

The population-to-individual problem links to the rare-event problem. In a U.S. city the size of San Jose, California, (population about 1,000,000), about 4,000 people every year will have a heart attack; perhaps one or two will be killed by someone with mental illness wielding a gun. Treatment evidence for preventing death from myocardial infarction has piled up from hundreds of clinical investigations over several decades, involving more than 50,000 patients in randomized trials by the early 1980s (7). When it comes to persons with mental disorders who kill strangers, there is nothing remotely resembling such an empirical evidence base.

Describing the inexpllicable
Nielsenen and colleagues (8) identified seven population studies in six countries (none in the United States) that reported stranger-homicides by people with schizophrenia or other psychotic illness. Seventy-eight of these incidents were counted across all investigations over several decades, involving more than 50,000 patients in randomized trials by the early 1980s (7). When it comes to persons with mental disorders who kill strangers, there is nothing remotely resembling such an empirical evidence base.

Nielsenen and colleagues (8) offer a modest summation of their study’s implications, which surely seems sensible even if the data cannot prove them right: “Measures that ensure earlier treatment of psychosis and continued treatment in the community would be likely to prevent homicides of both strangers and family members. However, the extreme rarity of these events means that identification of individual patients who might kill a stranger is not possible.”

With respect to prevention, the data provide general hints at best. Reports from the National Confidential Inquiry Into Suicide and Homicide by People With Mental Illness (9) in the United Kingdom suggest, for example, that reducing co-occurring substance abuse and ensuring regular outreach to improve treatment adherence among patients with a history of violence might prevent future violence. But we know that already, and it does not prevent a Tucson. It does not stop a mass shooting by a disturbed individual untouched by the mental health service system who can legally buy a handgun with a high-capacity magazine that fires 31 bullets in 15 seconds.

The limits of common evidence for uncommon things
Either of two factors can thwart a public health intervention study: a small effect size or a rare outcome event. Studies of how to prevent gun violence by people with mental illness would face both of these challenges at once.

This brings up the question of triangulating evidence of common things to inform understanding of similar but far less common things. Can studies of minor aggressive acts by psychiatric patients tell us anything useful about the one-in-millions mass homicide perpetrator? Maybe, or maybe not. We don’t know—at least not on the basis of the evidence we have now. By analogy, studies of hypertension treatment alone would never have produced the medical knowledge that beta blockers can lower death risk in heart attack victims. To fully understand and explain such events, we need to study them directly and describe them.

But even then, description is not the same as prediction. Profiles of mass killers suggest that they tend to be troubled young men. This does not tell us which one of the very large population of troubled young men in the world will become the next mass killer; the vast majority of them will not. And even if we could statistically predict such violent events, it would not necessarily mean that we could explain them. History of previous violent behavior is a relatively strong statistical predictor of any future violent behavior. It does not explain violence any more than a history of previous cancer explains why people get cancer.

When asked to explain individual acts of horrific violence committed by people who appear to have been mentally ill, perhaps the best that a researcher can do—at least for now—is to restate what we know about any and all violence risk in populations, and to say it in several ways: absolute risk, relative risk, and attributable risk. The “absolute risk” message is that the vast majority of people with
mental illness in the community are not violent. The “relative risk” message is that people with serious mental illness are, indeed, somewhat more likely to commit violent acts than people who are not mentally ill. And the “attributable risk” message is that violence is a societal problem caused largely by other things besides mental illness (ready availability of guns, for example) (10.)

That said, I am surely not suggesting that we are ultimately powerless to prevent rare and terrible things (such as 9/11) simply because they almost never occur and we cannot explain them by using population databases. Such a position would amount to irresponsible nihilism. From a public health point of view, we face some problems that almost inherently defy explanation and predictability, yet we prevent them anyway. We act on reasonable assumptions despite idiosyncratic and fragmentary evidence, because we have no other choice.

The non-news

Considering violence risk in the aggregate—that is, as a descriptor of population well-being over time—we are slowly moving in the right direction; for example, the U.S. homicide rate has been cut almost in half since 1990. And after all, the human species has somehow managed for 66 years to forestall a repeat of a nuclear war, an almost unimaginable event that occurred only once in history. Such a thing requires study of the particular in its own context—not because it represents some possible universe of similar events from which we will then generalize to the next instance, but precisely because it is unique.

In some respects, horrific acts of violence by people with serious mental illness are all unique. Systematic attempts to characterize, categorize, and compare these rare occurrences to other events and actors are useful as far as they go, but they may never yield a full explanation or satisfying answer to the “Why?” and “What if?” questions that Tucson poses.

In the meantime, we should pause to consider the “non-news.” We seldom read banner headlines announcing terrible events that might have happened but did not. Yet the truth is that an untold number of potential tragedies have been averted by the everyday provision of timely interventions and appropriate treatment (8,11). Many other acts of violence—harmful acts of aggression across the spectrum of severity—could be prevented in the future if mental health services were more consistently available and accessible to all those who need them, and especially to those with known risk factors for harming others or themselves. We may never know who the next mass shooter could have been, but then why would it matter?

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