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# Has 'lifetime prevalence' reached the end of its life? An examination of the concept

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**Abstract**

Many cross-sectional surveys in psychiatric epidemiology report estimates of lifetime prevalence, and the results consistently show a declining trend with age for such disorders as depression and anxiety. In a closed cohort with no mortality, lifetime prevalence should increase or remain constant with age. For mortality to account for declining lifetime prevalence, mortality rates in those with a disorder must exceed those without a disorder by a sufficient extent that more cases would be removed from the prevalence pool than are added by new cases, and this is unlikely to occur across most of the age range. We argue that the decline in lifetime prevalence with age cannot be explained by period or cohort effects or be due to a survivor effect, and are likely due to a variety of other factors, such as study design, forgetting, or reframing. Further, because lifetime prevalence is insensitive to changes in treatment effectiveness or demand for services, it is a parameter that should be dropped from the lexicon of psychiatric epidemiology. *Copyright © 2009 John Wiley & Sons, Ltd.*

## Introduction

Lifetime prevalence is one of the most commonly encountered parameters in psychiatric epidemiology. It is usually estimated from cross-sectional data as the proportion of a sample having had at least one episode of illness in their life up to the time of sampling. The popularity of this parameter reflects a view that most mental disorders are chronic conditions in which symptomatic episodes are interspersed with periods of remission.

A puzzling and paradoxical finding in many psychiatric epidemiologic surveys is that, while the point prevalence of depression may increase with age (e.g. Roberts *et al.*, 1997; Roberts *et al.*, 1991), the lifetime prevalence of disorders such as anxiety and depression has been reported to decrease with age (e.g. Hasin *et al.*, 2005; Kessler *et al.*, 2005; Robins *et al.*, 1984; Somers *et al.*, 2006; Streiner *et al.*, 2006). If this were found only in studies conducted within a narrow time frame, it could be explained as either a period or a cohort effect; that is, due to external events that affected everyone who was alive at the time or those who were in a specific age cohort (Yang, 2007). However, it has been seen in surveys conducted 25 years apart (e.g. Robins and Regier, 1991; Gravel and Béland, 2005); in geographically and culturally different areas of the world, such as New Zealand (Oakley Browne

 

*et al.*, 2006), Mexico (Slone *et al.*, 2006), Germany (Wittchen, 1986), Turkey, Greece, India, Nigeria, and other countries (Simon *et al.*, 1995); and with almost all psychiatric diagnoses (Simon and VonKorff, 1992).

In a closed cohort with no mortality, lifetime prevalence would represent a cumulative number or proportion that would increase or remain constant with age, in that once a person has had an episode, his or her value can only remain the same. As a result, each initial episode of illness should move a person from a lifetime prevalence negative category to a lifetime prevalence positive category, but movement in the reverse direction cannot occur. If this closed cohort with no mortality consisted only of at-risk (no prior lifetime episodes) individuals, then lifetime prevalence at any age would be synonymous with cumulative incidence by that age. In reality, lifetime prevalence is estimated from surveyed populations subject to mortality, so that a valid age-specific lifetime prevalence estimate represents cumulative incidence among survivors to that age.

In order for mortality to account for declining lifetime prevalence, mortality rates in those with a disorder would need to exceed those without a disorder by a sufficient extent that cases were removed from the prevalence pool at a rate exceeding their replenishment through incidence. This is unlikely to occur across most of the age range. As Robins *et al.* (1984) state, 'While the pattern [of lifetime prevalence] should vary across diagnoses, depending on the ages of onset, for *all* diagnoses, the rate in a younger age group should never be *higher* than the rate in the next older age group; it should equal the prevalence in the next older age group if both groups are past the age of risk onset and it should be less than the rate in the next older group if the age of onset includes the current ages of one or both groups' (p. 954; original emphasis).

These considerations help to distinguish various possible interpretations of the decline in lifetime prevalence. If the decline represents a cohort or period effect, then it indicates an actual change in population health status. If the decline is interpreted as an effect of mortality then it provides a valid reflection of population health status, but not a valid reflection of cumulative incidence. Another possibility is that the effect is not valid, but is rather an artifact of defective study design or bias. In this paper, we review the history of the concept of lifetime prevalence in psychiatric epidemiology, discuss the arguments for and against its use, and examine the possible causes for the observed decline in lifetime prevalence by age. We conclude that the parameter has outlived its usefulness.

## Where did the concept of lifetime prevalence come from?

Traditionally, prevalence measures are divided into two types: point prevalence and period prevalence. Point prevalence is the number or proportion of people in a population with a specified condition at a point in time, whereas period prevalence refers to the number or proportion of people with that condition during a period of time. In situations in which health status changes can be considered permanent (such as conversion to seropositivity for infections conveying lifetime immunity) or a chronic disorder from which there is no recovery (e.g. Alzheimer's disease or autism), prevalence can be viewed as a number or proportion within the population who are in or have permanently moved into another health state: lifetime prevalence. The same perspective has been applied in psychiatric epidemiology where, for example, a first episode of mania is interpreted as a permanent entry into the diagnostic category of Bipolar I Disorder. Lifetime prevalence is an unusual form of period prevalence in that the reference period is variable depending on a person's age. A person who has met diagnostic criteria for a disorder at any point in his or her life up to the time of assessment is included in the numerator of the lifetime prevalence proportion. Within psychiatric epidemiology, lifetime prevalence has become one of the most common types of prevalence reported. This phenomenon probably relates to the current limitations of psychiatric diagnoses. Unlike most other categories of disease, there are no adequately validated biological gold standard measures to define who has a disorder. As a result, psychiatric diagnoses depend on signs and symptoms, yet signs and symptoms at a point in time do not in themselves embody enough information to support a diagnosis. For example, a depressive episode may indicate a recurrent depressive disorder (i.e. major depressive disorder) or, if there is a history of past manic, hypomanic, or mixed episodes, may be (by definition) a manifestation of a bipolar disorder. Moreover, signs and symptoms are subjectively experienced and, as a result, may be interpreted differently at different times in a person's life.

There are major conceptual differences between point and period prevalence, on the one hand, and lifetime prevalence on the other hand. The first two terms include a fixed time component in the denominator (e.g. the number of cases within a six-month window) and are therefore justifiably called 'rates' (noting that some epidemiologists restrict the use of the term 'rate' to estimates with person-time denominators); whereas lifetime prevalence has a variable amount of time per person in the

denominator and therefore is not a rate. Consequently, it does not make sense to determine the average 'lifetime prevalence in a population' of a condition based on estimates from different age groups, although this is commonly done in psychiatry.

## Value of lifetime prevalence

The popularity of lifetime prevalence in psychiatric epidemiology cannot be attributed solely to the need to incorporate past history into diagnostic algorithms. Fully structured diagnostic interviews used in psychiatric epidemiology tend to emphasize lifetime prevalence as their target of assessment, e.g. the World Health Organization (WHO) version of the Composite International Diagnostic Interview (CIDI) (WHO, 1990). These interviews could have emphasized current mental state, supplemented by some historical data, as opposed to the reversal of this emphasis. A part of the value of lifetime prevalence may relate to higher prevalence proportions that emerge from this approach, which may have a particular value in advocacy because they emphasizing the high frequency of occurrence of these disorders. The concept of lifetime prevalence also fits with the view that mental disorders are chronic rather than acute conditions.

## Problems with lifetime prevalence as an epidemiologic parameter

In spite of the positive features of lifetime prevalence in terms of advocacy and clinical salience, the parameter is plagued by epidemiological disadvantages. Nearly half a century ago, Gruenberg (1963) said of lifetime prevalence that 'This particular measure is an example of new gimmicks introduced into a field of mensuration which has enough real troubles without being further burdened by unhelpful tricks. Lifetime prevalence measures are of no visible usefulness. They depend not only on the limited reliability of present case finding and identifying techniques, but also on the distant memory of respondents' (p. 92). This was echoed by Kramer *et al.* (1980), who wrote that 'Lifetime prevalence has been an exceptionally popular morbidity measure in epidemiological surveys of mental disorders. It is doubtful that this popularity is deserved' (p. 429).

There are a number of problems that result from using lifetime prevalence to reflect changing patterns in mental health epidemiology. First, a number of authors have interpreted the higher rates of lifetime depression among younger respondents to mean that there is a growing epidemic of mental disorders, and that we are entering an 'age of depression' (Cross-National Collaborative Group, 1992; Klerman and Weissman, 1989; Wickramaratne *et al.*, 1989). Second, it has been hypothesized that the inappropriate averaging of lifetime prevalence rates discussed earlier may result in 'pseudocomorbidity' (Kraemer *et al.*, 2006); that is, the appearance of comorbidity even when disorders are randomly associated.

Finally, although lifetime prevalence may have some salience in relation to long-term treatment needs, it correlates poorly with current treatment needs. A person with a single transient episode occurring many years or decades earlier appears in the numerator of a lifetime prevalence calculation in the same way as does a person having multiple persistent episodes, despite the fact that their treatments needs may differ greatly. For the same reason, the parameter is likely to be a poor metric of the effectiveness of health services. A person with an optimal outcome (rapid help seeking, rapid initiation of effective treatment, and rapid resolution of the episode) appears in a lifetime prevalence calculation in exactly the same way a person with a poor outcome. Jacobi *et al.* (2004), for example, found that disability days among those with a lifetime prevalence of mental disorder were comparable to those with no disorder, and twice as high as for people with a 12-month prevalence. It seems likely that lifetime prevalence can be, at best, a parameter of minor interest to psychiatric epidemiology, perhaps for advocacy purposes. However, drawing even this conclusion requires an understanding of what the parameter means, and whether estimates of it can be considered valid. The most compelling basis for exploring these questions is the unexpected behavior of the parameter in relation to age, as discussed in the following sections.

## Possible reasons for the decline in lifetime prevalence with age

There are (at least) five possible reasons that the prevalence of a lifetime disorder may decline with age: (a) period/cohort effects; (b) the types of studies; (c) a survivor effect; (d) forgetting; or (e) reframing. We will discuss each of these in turn.

### Period/cohort effects

The participants who were 65 and older at the time that many of the currently available surveys were conducted grew up during two very stressful periods: the Great Depression, followed by World War II. It is possible that there may be a cohort effect, in that those who survived these events developed greater resilience and were better able to withstand other vicissitudes that may lead to depression or other disorders (Elder, 1974). Of course, this

explanation is clearly rooted in a psychosocial determinants paradigm, where factors such as stress and personal coping skills and resources are not only viewed as primary risk factors for depression, distress, and anxiety; they are themselves a product of the social environment (Pearlin, 1989). Thus, it is not age *per se*, but rather one specific group of people for whom there is a lower incidence of disorder. These events may have also resulted in a period effect, in that they affected people of all ages, not just those who were born during this period, or an interaction between the two, whereby some age groups (cohorts) are more adversely affected by the event than others (e.g. Elder, 1974).

Age, period, and cohort effects are very difficult to disentangle, because most of the data come from piecing together cross-sectional surveys of people of different ages, or assembling birth cohorts from the same survey, which confounds age (with the concomitant problems of recall, discussed later) with cohort (e.g. Klerman *et al.*, 1985; Lasch *et al.*, 1990). There are few longitudinal surveys that have examined age effects within different birth cohorts and in different time periods. The two longest studies are the Stirling County Study (Murphy *et al.*, 2000) and the Lundby Study (Mattison *et al.*, 2005), neither of which reported lower incidence rates in elderly respondents. However, the Alameda County Study (Roberts *et al.*, 1991; Roberts *et al.*, 1997), which followed three waves of people for 18 years, found both period effects (rates in 1974 higher than either 1965 or 1983) and cohort effects (older participants had a higher two-week prevalence than younger ones). Consequently, evidence supporting or refuting period and cohort effects must come from comparing results across surveys that were done at different times and with different age groups of participants. The monotonic decrease in lifetime prevalence is seen in surveys that span at least a quarter of a century (e.g. Somers *et al.*, 2006) and are from different continents (e.g. Kessler *et al.*, 2005; Oakley Browne *et al.*, 2006). This would tend to rule out period or cohort effects, leading us to look elsewhere for an explanation of the declining lifetime prevalence.

## The types of studies

One difficulty in interpreting the results of lifetime prevalence studies is closely related to age/period/prevalence effects; we are trying to make inferences about longitudinal trends by stitching together the results of cross-sectional surveys (Simon and VonKorff, 1992). It has been noted that this is akin to doing a cross-sectional survey of Miami and concluding that people speak Spanish when they are young, English in their middle ages, and Yiddish when they grow old. Indeed, much of the early work that purportedly showed an age-related decline in all areas of intelligence was based on cross-sectional data, which confounded age with cohort effects (e.g. the more limited educational opportunities of earlier cohorts). Later longitudinal studies showed far less of a decline in verbal ability (e.g. Hertzog and Schaie, 1986; Horn and Donaldson, 1976; Schaie, 1994). In a similar way, most of what we know about the lifetime prevalence of psychiatric disorders is based on cross-sectional surveys, such as the Canadian Community Health Survey, Cycle 1.2 (Gravel and Béland, 2005) or the National Comorbidity Survey (Kessler, 1994). However, given the evidence cited earlier, that this decline has been seen in surveys separated by at least a generation, it is unlikely that period or cohort effects are sufficient to explain the decline.

## Survivor effect

It is possible that lifetime prevalence appears to decrease with age because those who suffer from psychiatric disorders either die at a younger age than non-patients, or are in hospitals or nursing homes, which are usually excluded from the sampling frames of surveys. The result of this would be a declining number of respondents at each age who have had a prior episode of a disorder. There is evidence from a number of directions that would support this hypothesis. First, there is a strong link between depression and suicide; indeed, depression is the major risk factor for attempted and completed suicide (e.g. Préville *et al.*, 2005); and higher suicide rates are seen with other psychiatric disorders, such as schizophrenia (Capasso *et al.*, 2008; Palmer *et al.*, 2005). Moreover, suicide rates increase with age (Conwell and Brent, 1995), although it has been argued that the earlier data may have over-estimated the risk (Bostwick and Pankratz, 2000). Furthermore, those suffering from a wide range of psychiatric disorders appear to be at a higher risk of morbidity and mortality from other causes. The Framingham Offspring Study, for example, found the relative risk (RR) for the 10-year incidence of definite coronary heart disease to be 1.25 among those with 'increased tension,' and the RR for mortality was 1.22 among anxious men and 1.27 in women (Eaker *et al.*, 2005). In one large cohort involving nearly 20,000 people, participants who had had a major depressive disorder were 2.7 times more likely to die from ischemic heart disease than those who did not (Surtees *et al.*, 2008); and a meta-analysis of 37 articles found an all-cause standardized mortality rate of 2.58 among people diagnosed with schizophrenia (Saha *et al.*,

2007). A meta-analysis of studies examining the impact of depressive disorders on all-cause mortality led to an estimated RR of 1.8 (Wulsin *et al.*, 1999). However, this may be an over-estimate, since most of the studies in this review involved clinical samples, which may have more severe depressive disorders than those identified in community samples.

Kramer *et al.* (1980) attempted to take mortality into account regarding lifetime prevalence, by using a life-table approach, and assuming various age-standardized mortality ratios for people with schizophrenia. Their results indicated that not taking mortality into account could lead to underestimates of lifetime prevalence at older ages. However, the decline in lifetime prevalence begins in young adulthood, in the twenties to forties in most published studies (e.g. Oakley Browne *et al.*, 2006; Robins *et al.*, 1984). These are age ranges in which mortality rates are probably too small to have a substantial impact on lifetime prevalence estimates.

The other component of a survivor phenomenon is that people who may have been diagnosed at a younger age were in hospitals or long-term care institutions at the time the surveys were conducted. In other words, various disorders may affect survival not only in the sense of life or death, but also survival in terms of remaining a member of the target population for surveys in which declining lifetime prevalence has been observed. An effect of this nature could also be regarded as a form of selection bias since the sampling frames of many large-scale survey do not include the institutionalized segment of the population. In principle, institutionalization could account for a declining lifetime prevalence of major depression if major depression is a strong determinant of institutionalization and if the frequency of institutionalization is sufficiently high to remove a large proportion of lifetime cases from community populations. For example, McDougall *et al.* (2007) found that the prevalence of depression among those living in institutions was 27.1%, compared to 9.3% for those living at home. However, as noted earlier, the decline in lifetime prevalence observed in epidemiologic studies generally begins in respondents in their twenties to forties (e.g. Oakley Browne *et al.*, 2006; Robins *et al.*, 1984) and continues into older age categories. The trend, therefore, begins in age ranges where institutionalization is too infrequent to account for the decline. Furthermore, when the prevalence data in the Canadian Community Health Survey are stratified by gender, the decline in depression with age is largely seen for women; the curve for men is generally flat. This means that not only would institutionalization have to occur earlier than expected, but that it is selective by gender, which does not conform to the demographics for institutionalization.

However, institutionalization may pose a problem for those suffering from dementing disorders. The prevalence of Alzheimer's disease and vascular dementia, for example, is about 1.4% for those between the ages of 65 and 69, increasing to roughly 10% for those 80 to 84, and about 25% for those 85 and older (Berr *et al.*, 2005; Jorm *et al.*, 1987).

The issue is whether effects of mortality and institutionalization are sufficient to account for the observed decline in lifetime prevalence. A simulation study by Kruijshaar *et al.* (2005) determined that even very high RRs for mortality, in the range of RR = 4.0, would have only a small effect on lifetime prevalence up to age 65, and that more plausible values (RR = 1.8) had almost no effect. Another simulation model compared a RR for mortality of 1.1 to that of 1.8, again finding no substantial effect on lifetime prevalence (Patten, 2007).

## Forgetting

Another possible explanation for the decline in lifetime prevalence is that people simply forget that they had had an episode of a disorder, a form of recall bias. Even with a short time-span, healthy, well-educated men forgot between 34% and 46% of significant life events that had been reported nine months previously (Jenkins *et al.*, 1979). Naturally, the problem is magnified if the episode occurred 10 or 20 years previously. There is ample evidence that recall, even of serious illnesses, is far from perfect. In the now-classic Health Interview Study, Cannell *et al.* (1965) found that 42% of people did not recall that they had been in hospital one year after the event. Under-reporting was negatively correlated with duration of stay, but positively related to the respondent's age. Similarly, Means *et al.* (1989) reported a 69% false-negative rate in the recall of serious medical events after one year. A study of nearly 10,000 relatives of 2000 patients initially found that lifetime prevalence rates of epilepsy decreased with age and appeared to increase in successive generations. However, further analyses of the data showed that these were artifacts due to forgetting; there was in fact no cohort effect, and lifetime prevalence actually increased with age, as would be expected (Ottman *et al.*, 1995).

Needless to say, the same phenomenon exists in the recall of psychiatric disorders. Pulver and Carpenter (1983) found that the Diagnostic Interview Schedule failed to detect psychiatric symptoms, such as hallucinations and delusions, from 12% to 80% of the time, when

compared to ratings that were done 11 years earlier. Andrews *et al.* (1999) followed a cohort of patients after an inpatient admission. Of those who had a major depression at the time of their admission, only about half (14/27) recalled symptoms sufficient to make a CIDI diagnosis of major depression 25 years later. Consequently, the decline in lifetime prevalence can be due simply to the respondents having forgotten previous episodes of anxiety and depression.

### Reframing

A different explanation of the decline is not that respondents have forgotten previous episodes, especially of depression, but rather that they more positively reinterpret prior experiences in light of current circumstances. This could lower the sensitivity of items in structured diagnostic interviews, leading also to recall bias. According to adaptation theory (Helson, 1964), people react acutely to positive and negative events in their lives, but eventually return to a stable level. Albrecht and Devlieger (1999) refer to this phenomenon as the 'disability paradox;' the fact that people with often severe innate and acquired disabilities report their quality of life to be at the same level as that of non-handicapped people. In a longitudinal study of nearly 25 000 residents of Germany, for example, Lucas *et al.* (2003) found that immediately following the death of a spouse, there is a significant drop in satisfaction with life, but that most people return to their pre-widowhood level within eight years. Even in community studies of the oldest-old (ages 80 and over), where the prevalences of physical disability and social isolation are high, depression is not commonly reported (Johnson and Barer, 2003). Thus, episodes that may have been labeled 'sadness' or 'depressive' when they occurred may be seen in retrospect as merely part of the normal ups and downs of everyday life when viewed from the vantage point of 50 years of experience.

## Conclusions and recommendations

As noted earlier, lifetime prevalence may reflect cumulative incidence under certain artificial conditions, but these do not exist in real-world populations. Since mental disorders can contribute to mortality and institutionalization, it is possible in theory to regard lifetime prevalence as a composite measure that reflects both incidence and mortality. However, the effect of mortality is probably dependent on age, with a larger impact occurring in more elderly age groups. Even if valid, this parameter would seem to be of little value for epidemiologic description, at least not in the contemporary context. Furthermore, there are important reasons to believe that available estimates are not valid. A particularly compelling case can be made that recall bias probably distorts all available lifetime prevalence estimates.

Lifetime prevalence served an important historical role in psychiatric epidemiology. It helped to confirm that mental disorders occur commonly and that such disorders were often untreated. Lifetime prevalence estimates are not very helpful for moving beyond such basic questions, yet the predominance of this parameter has continued in psychiatric epidemiology. Psychiatric epidemiologic surveys are extremely expensive and the choice of measurement and estimation strategies is likely to be influenced by perceptions of the credibility of available instruments. The currently favored approaches reflect decades of development and evolution of structured diagnostic interviews. They may continue to be chosen for this reason despite their emphasis on lifetime prevalence.

More detailed measures of current functioning, current symptom levels, and current treatment have often become mere 'add on' elements to survey interviews, unnecessarily disconnected from the prevalence measures. For example, scales that assess symptom levels, functional impairment, and quality of life typically refer to essentially 'current' reference periods such as the last week or past month. While such scales can be included in psychiatric epidemiologic studies, they are disconnected from diagnostic measures in the sense that detailed diagnostic information is most often collected from past episodes (e.g. first episode or worst episode). Another concern is that reliance on lifetime prevalence-based measures may be producing a distorted picture of the burden of mental illness. In particular, the impression that the prevalence of mental disorders declines with age may be an artifact that could nevertheless influence policy.

Given that prevalence data are necessary for both scientific and planning purposes, a question remains regarding the most appropriate time frame. In our opinion, a one-month period is an appropriate compromise between the need for a window of some duration to allow for the fact that most disorders do not have an acute onset, with a measure that is responsive to changes in health care delivery. With respect to a wider window, we do not see any convincing argument that would favor six months over 12 months, or vice versa. A 12-month window allows the detection of brief episodes of a disorder that may be missed using six-month prevalence, but is less reflective of change. However, gathering both one- and 12-month prevalence in a survey may be a good balance between competing requirements.

The continued use of lifetime measures may now be doing more harm than good and it may be impeding the progress of the discipline of psychiatric epidemiology. While it is currently not feasible or desirable to eliminate historical information from psychiatric diagnostic interviews, the time has come to reconsider the relative emphasis on lifetime versus current measurement.

## Declaration of interest statement

No author has any conflict of interest for this paper.

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