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# THE HEALTH EFFECTS OF ECONOMIC DECLINE

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## Abstract

The recent recession and lingering high unemployment will likely lead to a burst of research studying the health effects of economic decline. We aim to inform that work by summarizing empirical research concerned with those effects. We separate the studies into groups defined by questions asked, mechanisms invoked, and outcomes studied. We conclude that although much research shows that undesirable job and financial experiences increase the risk of psychological and behavioral disorder, many other suspected associations remain poorly studied or unsupported. The intuition that mortality increases when the economy declines, for example, appears wrong. We note that the research informs public health programming by identifying risk factors, such as job loss, made more frequent by economic decline. The promise that the research would identify health costs and benefits of economic policy choices, however, remains unfulfilled and will likely remain so without stronger theory and greater methodological agreement.

### Keywords

Unemployment; recession; illness; disorder; public health

## INTRODUCTION

Research into the health effects of economic decline appears “countercyclical” - good times yield fewer publications while bad times produce more (47). We, therefore, expect that the

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worst recession since the Great Depression will generate a burst of research. We hope to contribute to this effort by summarizing research testing hypothesized associations between economic decline and incident illness. More specifically, we assign selected publications to the cells of tables defined by three dimensions (mechanism, line of inquiry, and outcome), described below. We also offer our assessment of whether findings in populated cells warrant inferring an association.

We used several online search engines to identify publications tagged with "unemployment," "economy," "recession," or "job loss" in addition to the psychological, behavioral, and somatic outcomes shown in tables 1 and 2. We also traced references in the literature to find additional publications not identified in the online searches. We excluded research concerned with the effect of macroeconomic conditions on medical care. We note, however, that much research suggests that contracting economies affect decisions to seek medical care and the willingness to diagnose illness, independent of the biology or behavior of patients (24). We also excluded work that, in our judgment, failed to meet minimum standards of internal and external validity. This meant excluding reports based on cross-sectional associations or on longitudinal associations not adjusted for autocorrelation. We also excluded work that inferred individual associations from ecological correlations.

We focus on work published in the last two decades because reviews published in the early 1990s cover much of the earlier work (49). We, however, cite selected earlier work when needed to set the context for more recent publications.

## DEFINING THE CONCEPTUAL SPACE

### Mechanisms

The research we identified explicitly or implicitly speculates that declining economies affect somatic as well as psychological and behavioral morbidity primarily through three broadly defined mechanisms. The literature suggests that these mechanisms can increase (i.e., "countercyclical" effect) or decrease (i.e., "procyclical" effect) the incidence of illness in populations embedded in declining economies. We describe the counter- and procyclical variant of each mechanism below.

The stress mechanism dominates the literature. The countercyclical literature reports that declining economies increase the incidence of stressful job and financial events and that these events increase the likelihood of experiencing other stressors not intuitively associated with the economy (such as marital difficulties) (28, 144). The literature also includes reports of economic decline increasing the anticipation of stressful experiences, including job loss and difficulty meeting financial obligations (104).

Procyclical invocation of the stress mechanism assumes that much of the stress of everyday life comes from working (84). A reduction in time at work, therefore, may reduce the prevalence of stress-induced illness. The procyclical literature also notes that the capacity to cope with workplace safety hazards increases when the pace of work slows (10, 22, 33, 44, 92).

A second mechanism, frustration-aggression, stems from the much-tested argument that individuals denied an expected reward may experience psychosomatic antecedents of aggression (7). Some will exhibit antisocial behaviors while others cope by using alcohol or drugs. The countercyclical camp argues that contracting economies increase the perception of "unfair" loss of earned rewards, and thereby increase the incidence of intra-family and workplace violence as well as of substance abuse (39).

The procyclical literature asserts a special case of the frustration-aggression mechanism, referred to as the "inhibition effect" (32). This argument asserts that workers who fear job loss will do whatever they can to avoid it. This may include reducing the use of alcohol and drugs and avoiding the antisocial behavior that make employees more likely to be laid off when the demand for labor wanes.

A third mechanism subsumes a broad group of possible connections we collectively label "effect budgeting." Both the pro- and countercyclical versions of this mechanism assume that we have limited time, energy, and money to manage our environments and experiences. We budget these resources in ways that reflect expected costs and benefits. "Investments" that improve or protect our health or that of others in our social ecology may, depending on our explicit or implicit cost/benefit analyses, rank high or low.

The countercyclical literature asserts that a declining economy not only reduces the money available to many households, but also forces them to invest time and effort in managing the sequelae of lost jobs or lost income. These new allocations require taking time, effort, and money from existing investments. An individual who ranks salutary uses of time, energy, or money (e.g., exercise, child care, socially supportive behavior, medication, good nutrition, surveillance of one's own or others' biology or behavior) relatively low in the hierarchy will stop investing in them and thereby increase his or her risk of illness (17).

Unlike the countercyclical variant of the effect budget mechanism, the procyclical version assumes that many individuals and households have risky goods and services at the bottom of their lists. Such households or individuals who lose income will, for example, consume less alcohol or illicit drugs, thereby avoiding the somatic toxicity of these substances as well as the risky behaviors associated with them (e.g., drunk driving) (55).

A second procyclical form of effect budgeting assumes that a contracting economy will reduce time at work, making more available for salutary behaviors (e.g., exercise or monitoring the health of chronically ill relatives) that previously fell below the investment line (128).

## Line Of Inquiry

Researchers in this field intend, explicitly or implicitly, to further either of two lines of inquiry. The first asks whether exposure to contracting economies changes the somatic, psychological, or behavioral health of individuals. These "risk factor studies" typically compare the health of individuals exposed or unexposed to contracting labor markets, or to undesirable job or financial experiences, such as involuntary job loss.

A second line of inquiry yields research using population aggregates as the unit of analysis (e.g., national or state populations) and measures the association over time between economic indicators (e.g., the unemployment rate) and incidence of pathology. These "net effect studies" try to estimate the sum of pro- and countercyclical effects on population health (39). Estimating net effects can inform efforts to anticipate changes in aggregate demand for, and cost of, public health services. We do not subscribe to the criticism that net effect studies axiomatically assume that an association between two characteristics of a population generalize to individuals in that population (i.e., the "ecological fallacy") (33). The net effects literature we review does not make such assumptions.

## Outcomes

We organize the research by the outcomes most frequently addressed by the literature. These include psychological and behavioral morbidity (including substance abuse), nonspecific morbidity and mortality, cardiovascular morbidity and mortality, and gestational morbidity

and infant mortality. Some of these outcomes have relatively few studies while others have many. We review what we consider the best work, based on internal and external validity, for each outcome.

The dimensions described above define tables 1 and 2. Table 1 includes psychological and behavioral morbidity because the rubric subsumes several relatively well-studied outcomes that deserve separate consideration. Table 2 includes somatic outcomes.

## PSYCHOLOGICAL AND BEHAVIORAL MORBIDITY

The largest subgroup of literature concerned with the health effects of economic decline focuses on psychological and behavioral morbidity. We subdivide this literature into that dealing with depression and anxiety, substance use and abuse, violent behavior, and suicide.

### Depression and Anxiety

**Risk factor studies—**Most of the individual-level research concerned with symptoms of depression and anxiety focuses on job loss as the risk factor. In the work that best controls for the possibility of confounding by reverse causation and other relevant factors, odds ratios for increased risk of symptoms associated with job loss average around 2.0, and range from 1.15 to 5.67 (14, 25, 65, 87, 142). Linear regression results from well-designed studies suggest a 15% to 30% increase in reported symptoms among job losers compared to those who remain stably employed (e.g., (21)). A recent meta-analysis suggests that the work concerned with job loss converges on effect sizes in these ranges (117).

Unlike the relatively large body of work concerned with symptoms of depression and anxiety, we found no strong studies focused on clinically significant or diagnosable anxiety and only three that measured clinically significant depression as an outcome. Of these three, one, which examined job loss in a Mexican-American sample (25), reported a significant association. Two studies found more clinical depression among job losers than other workers but the difference did not reach conventional levels of statistical significance (14, 48).

Although involuntary job loss remains the most commonly studied risk factor associated with economic decline, researchers have increasingly become interested in other forms of economic adversity. Transitions to inadequate employment (52), insecure or temporary employment (59, 142), and income loss (121), all reportedly increase symptoms of depression and anxiety. Perceived job insecurity also increases risk of depression symptoms (60), as well as incident use of antidepressants (124). The effect sizes in these studies generally fall below those reported for involuntary job loss.

Employment among previously unemployed persons significantly reduces depression symptoms (e.g., (88, 120). A series of studies using a randomized controlled jobs training intervention program also reports reductions in symptoms or depression (122, 145, 146).

Much research has focused on age and gender as moderators of the job loss-psychological distress relationship. The majority of longitudinal studies that assessed effect modification by gender reported no significant interaction effect (e.g., (8, 52, 75, 114, 121, 124, 142)). Longitudinal studies of older adults have consistently reported that losing a job increases psychological distress (e.g., (11, 65, 103)), but findings in young adults include positive associations (75, 111, 112) as well as no effect (14, 130).

Very little risk factor research discusses mechanisms, and even less attempts to empirically discriminate between mechanisms. Nearly all of the work that discusses any mechanism

explicitly or implicitly invokes some variant of the stress mechanism. Financial strain has been reported as mediating the association between unemployment and minor psychiatric morbidity in three studies (59, 87, 144). In contrast, neither marital strain nor social interaction nor non-financial stressful events appear to mediate the job loss to morbidity association (52, 87).

**Net effect studies—**The literature includes virtually no longitudinal studies of the net effect of potential pro- and countercyclical mechanisms on psychological distress. The sole study we identified used sequential population surveys to measure prevalence of major depression in Hong Kong before and after the 2008 financial crisis (98). The authors attributed a significant increase in depression rates to unemployment and losses in investment income. This study invoked the stress mechanism, although tested against the null rather than other mechanisms.

**Conclusion—**In sum, the risk factor and net effects research suggests that involuntary job loss increases risk or severity of symptoms of depression and anxiety. We know nothing of the association, if any, of job loss or other forms of economic adversity with clinically diagnosable anxiety, and very little of the association with clinically important depression. The research available on the latter suggests a modest association. The work rarely invokes theory and virtually never attempts to empirically pit mechanisms against each other.

## Substance Use and Abuse

**Risk Factor studies—**A number of well-executed studies suggest that unemployment increases alcohol (80), cannabis (74, 76), and other drug use (110). Unemployment (>20 weeks) has been found to be associated with double the alcohol intake overall, and with four times the rate of heavy drinking among males, in a Swedish population (80). In the National Longitudinal Survey of Youth (NLSY), long term unemployment (≥3 years) increased the risk of heavy drinking (≥ 5 drinks on one occasion) by approximately 50% (113). Among older workers, Gallo et al. (64) found that involuntary job loss increased the onset of drinking, but had no effect on total alcohol consumption. Dooley and colleagues, using NLSY data, noted that people with a history of substance use may be at higher risk: unfavorable employment changes were found to predict increased heavy drinking among previous heavy drinkers (50). Also using NLSY data, Arkes reported that high state unemployment rates led to increased marijuana and cocaine use among teenagers, adjusting for individual risk factors (3).

Countercyclical results also emerge from work focused on clinically significant alcohol abuse rather than utilization. The Christchurch Health and Development Study (CDHS) reported that the variation in unemployment among young people accounted for 8–17% of substance abuse risk, and that those who had been unemployed for >6 months had increased rates of disorder (57, 58). Using panel data from the Epidemiologic Catchment Area Study (ECA), Catalano et al. (29) found that involuntary job loss increased risk of meeting standards of “caseness” by sixfold.

The above studies reporting countercyclical results allocate little discussion to theory or mechanisms. The work, however, typically alludes to unemployment as a stressor. We, therefore, have included the work among that assuming the stress mechanism.

Support for procyclical associations come from the ECA study, alluded to above, and from analyses of data from the National Health Interview Survey (NHIS). Among ECA respondents, Catalano and colleagues found that those who remain employed amid high unemployment showed reduced risk of alcohol abuse. The authors attributed these results to the inhibition mechanism (29). Using NHIS data, Ettner et al. (55) found evidence that new

unemployment reduces alcohol consumption dependence symptoms, and that this effect is likely driven by reduced income rather than psychological distress. Ettner, however, also reported that involuntary unemployment increased alcohol consumption, providing some evidence for a countercyclical relationship.

The Winnipeg Health and Drinking Survey likewise yielded both pro- and countercyclical results. Although clinical alcohol dependence and alcohol problems did not correlate with employment status (90), short-term unemployment was associated with reduced alcohol use, which the authors attributed to both inhibition and income effects. We, therefore, include these findings in both the frustration-aggression and effect budget groups. In contrast, the authors also reported that long-term unemployment predicts increased alcohol use and attributed the association to the stress mechanism.

**Net effect studies—**Ecological evidence for a procyclical association between macroeconomic conditions and substance use in populations comes mostly from two studies. Using 1975–1988 state-level data, Ruhm (125) found that reductions in personal income predicted decreased alcohol consumption. Freeman (61) replicated and expanded these results, suggesting a robust procyclical relationship between state unemployment rates and consumption. Dee (45) also reported that increases in state-level unemployment predicted decreases in overall consumption. While this work does not offer theory or discussion of mechanisms, the text implies an income effect and we therefore assign the articles to the effect budget group.

Other net effects studies support the countercyclical hypotheses. Dee (45) reported that increases in state-level unemployment predicted increases in binge drinking, and speculated that “recession-induced increases in binge drinking” could be explained by stress in the labor force. A study using WHO and International Labour Organization (ILO) data showed that increases in unemployment contributed to excess alcohol-related deaths among those <65 years (134). This paper also indicated that dips in employment may significantly increase alcohol-related deaths among working age people (134). High regional unemployment rates also have been associated with high rates of opiate exposure (67), whereas a similar study found no consistent, significant relationship between aggregate unemployment and substance use (131).

**Conclusion—**Risk factor research supports a countercyclical association in that individuals appear to consume more alcohol after losing jobs or suffering long bouts of unemployment. The net effects research, on the other hand, appears mixed and defies clear inference. The relative rarity of studies focused on substances other than alcohol makes conclusions regarding cyclical and procyclical effects for these substances more tenuous.

## Violent Behavior

**Risk factor studies—**While highly visible violent acts committed by laid off workers have led to popular reports of an association between job loss and violence (1), relatively little research met our standard for longitudinal analysis. Researchers have invoked frustration-aggression theory and stress to hypothesize that the incidence of intra-family abuse responds to economic conditions. A case-cohort study found that children whose mothers were unemployed for at least 21 weeks in the previous year experienced increased odds of being hospitalized for abuse or neglect (OR=1.3) (42).

Also citing frustration-aggression theory and using the ECA panel data alluded to above, Catalano and colleagues (27) reported a countercyclical effect in that layoff increased the likelihood of violence nearly six times among people not violent at baseline. Retaining a job amid high rates of layoffs, however, reduced violent behavior, thereby suggesting a

procyclical inhibition effect. The authors also found support for selection in that a history of violence increased the likelihood of job loss nearly 16-fold.

**Net effect studies—**Three ecological studies have used time-series methods to examine the relationship between weekly unemployment insurance claims and civil commitment for danger to others. The work invokes both the pro- and countercyclical variants of frustration aggression theory by arguing that violence should increase with job loss in a community, but that very high rates of unemployment should inhibit violence among those who remain employed. Results supported this argument for men in San Francisco and Pittsburgh; the effect also appeared for women in San Francisco (31, 32) and for men in Florida (86). The incidence of civil commitment for violence against others increased as layoffs began to rise but then declined at high levels of job loss.

The literature argues that the countercyclical effect may arise not only from increased incidence of psychogenic violence, but also from reduced community tolerance for the chronically mentally ill (36). This argument invokes frustration-aggression theory by asserting that frustrated persons will use socially and legally acceptable forms of aggression such as calling the police to report persons they find most noisome.

Returning to the hypothesis that economic contraction induces intra-family violence, Paxson found that a decrease in state welfare benefit levels was associated with an increased rate of foster home placements; the study invokes both effect budgeting and stress but lacks the data to directly test either mechanism (119). Replicating Steinberg et al.'s earlier work (133) and citing the stress mechanism, a study in Scotland (70) found positive correlations between male unemployment and physical abuse of children at the local level. Lastly, Catalano and colleagues identified both pro- and countercyclical effects in the relationship between foster home placements in California and the state unemployment rate (39). These results imply that rising job loss in the community increases the incidence of foster home placements, but the incidence peaks and declines as job loss continues to rise.

**Conclusions—**Risk factor and net effects tests of the association between economic decline and violence reconcile pro- and countercyclical arguments. The reconciliation implies that frustration-aggression mechanisms induce increased violence among those who lose jobs or income. As layoffs in a community mount, however, fear of such losses increases in the population remaining employed. This fear causes individuals to reduce their chances of layoff by altering their behavior, including reducing abusive behavior. The net effect of economic contraction on antisocial behavior potentially reduces incidence as job losses reach levels that transmit fear through media attention and social networks.

## Suicide

**Risk factor studies—**Data on the role of individual-level economic adversity in predicting suicide or parasuicide (serious suicide attempts) emerge from case-control and longitudinal cohort studies conducted in a variety of countries. Seven risk factor studies (9, 15, 56, 89, 93, 99, 123) – all of which explicitly or implicitly invoke the stress mechanism – provided support for a modest but positive longitudinal association between unemployment and suicide when controlling for confounding factors such as socioeconomic status, marital status, and age (RRs: 1.8–3.8).

Attempts to "sort out" the associations among unemployment, prior mental illness, and suicide have not produced clarity. Whether such associations reflect causation or the increased propensity of psychologically vulnerable individuals to both undesirable economic experiences and suicide remains controversial. Some work reports that unemployment predicts suicide after adjusting for confounding by mental illness (9, 15, 123), while other

studies report that the association disappears after control for prior (56) or current (6, 81) psychiatric disorder and/or familial and interpersonal problems. Because current psychological disorder may fall on the causal pathway between new unemployment and suicide, these last two studies may have over-controlled for the association.

Among the risk factor studies, only Jones et al. (81) attempted to examine intervening mechanisms. While unemployment significantly increased parasuicide in univariate analyses, the inclusion of post-job loss psychological and relationship problems as covariates removed the association, suggesting that these variables may act as mediators.

**Net effect studies—**Suicide rates vary positively with the unemployment rate in time-series analyses of the United Kingdom (73), the U.S. (148), Spain (138), Russia (68, 108), Japan (91), and several other Asian countries (41). Several studies using fixed-effects models in a variety of countries have also found countercyclical effects for suicide (126, 134, 137, 139, 140). Increases in suicide rates have been particularly marked in the aftermath of severe economic crises – nearly 45% in some Asian countries after the 1998 financial crisis (41) – and appear to be explained primarily by the concomitant rises in unemployment (41, 108). Coefficients for the effect size across normal economic cycles tend to be smaller, but remain significant (73, 91, 138, 148). Much of the net effects work also finds that the strength of the relationship differs by gender or age, although the risk factor literature remains less consistent (71).

Only four studies we identified found either procyclical (5, 115) or null (2, 79) associations between suicide and economic decline. Although reasons for this heterogeneity remain unclear, possibilities include substantial differences in method and relatively low power for detecting associations.

As with the risk factor literature, net effects studies often invoke the stress mechanism to explain the association between unemployment and suicide. Net effects research also occasionally invokes the frustration aggression mechanism, noting that increases in alcohol consumption during economic decline may increase suicide rates.

**Conclusion—**The majority of risk factor and net effects research concerning suicide converges, suggesting that economic decline increases the incidence of self-destructive behavior. The diversity of the populations and time periods studied strengthens this inference. Nevertheless, the lack of clarity regarding mechanisms operating in the pathway between economic adversity and suicide suggests an important area for future research.

## CARDIOVASCULAR DISEASE

### Risk factor studies

Emotional states associated with job loss, including anxiety and depression, may influence cardiovascular health through excessive activation of stress response pathways including the sympathetic nervous system and hypothalamic-pituitary-adrenal axis (95). However, explicit tests of stressful experiences as a mechanism linking unemployment and heart disease remain rare.

Individual-level, prospective studies of this association vary in design and analytic approach, but seem to converge on the finding that job loss and job insecurity later in life increase risk of cardiovascular outcomes in the United States. Using data from the U.S. Health and Retirement Survey (HRS), Gallo demonstrated that involuntary job loss later in life predicts significantly increased hazards of both heart attack and stroke over the subsequent ten years (63, 66). However, the small number of stroke and cardiovascular events in this sample limit

these findings. Data from the much larger Nurses Health Study, however, support the HRS findings in that self-reported job insecurity in women (ages 46–71) predicted almost twice the risk of nonfatal myocardial infarction in the subsequent two years (97).

Studies of Northern European cohorts do not find similar associations. In a large randomly sampled Finnish cohort (ages 36–64), workplace downsizing was not associated with coronary heart disease (CHD) mortality (106). Among Danish men (aged 40–50 years old) job loss was not associated with heart disease events (94). While age-stratified analyses in a Swedish cohort suggested increased risk of hospitalization due to stroke for men aged 35–49 following job loss, no relationship with hospitalization due to myocardial infarction was detected in either the whole cohort or age-stratified analyses (53). Disparate findings from the U.S. and Northern Europe may be explained by differences in the social and financial services available following job loss (54) as well as by the relative younger ages of the cohorts studied in Northern Europe.

**Net effect studies—**In early work citing the role of psychosocial stressors, Bunn (20) and Brenner (13) showed that increasing unemployment predicted increased heart disease mortality in Australia and in nine developed countries, respectively. However, the methods used in these investigations have since been widely criticized (72).

More recently, several studies have suggested procyclical effects of economic insecurity on risk of death from heart disease (69, 128). These studies, based on longitudinal data from 23 OECD countries (1960–1997) and the United States (1979–1988), found that decreasing unemployment resulted in increased deaths from CVD mortality. While the authors have proposed multiple mechanisms to explain these findings, they broadly fall into the category of effect budgeting, such that a decline in working hours may provide time for health-promoting behaviors and reduce consumption of cigarettes and obesogenic foods (127). More time and effort spent at work might also reduce early detection of CVD symptoms among workers' older relatives, who would otherwise have benefitted from life-saving procedures used early in episodes of acute CVD (128).

These conflicting findings have provoked several investigations to examine whether these relationships hold in other settings. Using panel data of eight Asia-Pacific countries (1976–2003), Lin (101) found that decreases in unemployment predicted an increase in CVD mortality. However, studies in Scandinavia do not yield the same results; instead they suggest countercyclical effects. Svensson found that decreases in unemployment predicted decreases in mortality from heart disease using Swedish panel data from 1976–2005, with evidence that these effects may be most pronounced for those in prime working age (20–49) (136).

**Conclusion—**The CVD research poses an inferential puzzle. The U.S. risk factor research reports countercyclical associations while the Scandinavian findings suggest no connection. The U.S. net effects research, conversely, suggests a procyclical association while the Scandinavian works suggests a countercyclical pattern. Differences among net effects studies may be due to differences in method, while those among risk factor findings could arise from differences in study population demographics or social welfare systems.

## GESTATIONAL MORBIDITY AND INFANT MORTALITY

### Birth Weight

**Risk Factor Studies—**One individual-level study (51) based on NLSY data reports countercyclical reductions in birth weight among infants whose mothers shifted from employment to unemployment or to involuntary part-time employment during pregnancy.

The authors attempted to discriminate between the stress mechanism, operationalized by gestational age and maternal weight gain, and effect budgeting, measured by utilization of prenatal care. Only gestational age was found to meet the authors' criteria for mediation, implying a stress mechanism. Whether gestational age gauges stress, however, remains controversial.

**Net effect studies—**Findings from net effects studies differ by population and methodology. Of the studies with strong internal validity, Catalano and colleagues – using data from populations ranging from California counties to Scandinavia – reported countercyclical increased rates of low birth weight (LBW) and very low birth weight (VLBW) during months with higher-than-expected unemployment rates and lower-than-expected employment rates (30, 35). These authors also tested the stress mechanism by demonstrating increased rates of LBW immediately following a period of threatened, but unrealized, job loss (35).

Dehejia and Lleras-Muney (46), using U.S. data from all 50 states, found procyclical reduced rates of VLBW and LBW associated with increasing annual unemployment rates, especially among black mothers. They argued that the association most likely arises from an effect budget mechanism in which couples least likely to have low weight babies choose not to start pregnancies during economic contractions. Two studies by Joyce and colleagues, however, found no association between unemployment rates and LBW in New York City (82) and in Tennessee (83).

**Conclusion—**Although one risk factor and two net effect studies reported countercyclical associations between economic contraction and birth weight, two studies found null and one found procyclical associations, limiting inference from this literature. Net effects studies also differed by methodology and population.

### Fetal Death

**Net effect studies—**The stress mechanism purportedly connects economic contraction to fetal death. Natural selection conserved mechanisms by which women give birth to fetuses most likely to survive environments prevailing at birth. While stressful environments reduce the survival chances of all infants, small males suffer the largest decline (147). Conserved mechanisms, therefore, supposedly allow women to spontaneously abort small males during stressful times (132). This "culling" of small male fetuses supposedly lowers the ratio of male to female births (i.e., the secondary sex ratio) (143).

Although no risk factor studies have assessed fetal death or sex ratios as a function of undesirable job or financial events, four net effects studies have tested the association between economic contraction and the secondary sex ratio. The first found that the fetal death (≥20 weeks) sex ratio increased significantly when monthly unemployment rates rose above their expected value in California (26). The second (37) reported that the secondary sex ratio in East Germany during the economic collapse of 1991 was lower than its expected value. The third found a decrease in the secondary sex ratio in years characterized by a reduction in household consumption of goods and services in Sweden between 1862 and 1991 (38). Helle et al. (78), however, found that secondary sex ratios were not associated with the annual percentage change in gross domestic product (GDP) in Finland between 1865 and 2003. This inconsistency may arise due to a difference in methodology.

**Conclusion—**No risk factor tests of an association between undesirable job and financial events and fetal death appear in the literature. Three net effect studies, all from the same

research group, support the countercyclical hypothesis, while one supported neither hypothesis. We conclude that no inference can be drawn from the existing literature.

### Infant mortality

**Risk factor studies—**No risk factor research addresses whether undesirable job or financial events affect infant mortality. Bruckner (17), however, finds that parental report of high-risk infant sleeping position (associated with sudden infant death syndrome (SIDS)) increases when the economy contracts.

**Net effect studies—**A small literature examines the net effects of economic contractions on the infant mortality rate (IMR) in developing nations, and reports predominantly countercyclical associations (4, 43, 62, 118). The variable quality of IMR data from these countries may, however, limit the validity of these studies.

Studies examining associations between unemployment rates and the IMR in developed nations also support a countercyclical association. Lin (100), Menifield (109), and Chan (40) reported associations between increasing unemployment rates and increasing infant mortality rates. Bruckner (16, 19) reported evidence of increased rates of infant mortality due to unintentional injury and SIDS following economic contractions in multiple populations, explicitly testing the effect budget notion that parents have less time and effort to invest in monitoring children when the economy contracts. One study reports associations in the opposite direction (46) and one finds no significant association (115).

Several net effect studies have revisited Brenner's seminal 1973 report of a net countercyclical association between economic adversity and neonatal mortality. Catalano and Serxner (34) reexamined this question by applying time-series methods data from Los Angeles and Orange counties, California, and found that neonatal mortality among black males rose when employment was unexpectedly low. Three studies have used fixed-effect methods to examine the association between rates of unemployment and neonatal mortality. Two of these studies found no association between unemployment rates and neonatal mortality in the U.S. (46) and Germany (115), while one found a countercyclical increase in the neonatal mortality rate associated with unemployment rates in Taiwan (100).

The net effects studies primarily invoke effect budgeting but do not empirically test for mechanism. A few also discuss stress mechanisms or effect budgeting (34, 46, 100, 115), or the inhibition variant of the frustration-aggression theory (46, 115).

**Conclusion—**The majority of net effects studies examining infant mortality support the countercyclical hypothesis. The work connecting SIDS to the economy deserves further attention, as one individual-level study has reported that parents are less likely follow "back to sleep" guidelines when the economy contracts. We draw no inferences from the work examining neonatal mortality, however, because two of the four studies found no associations. The different time and geographic units as well as statistical methods of these works, moreover, make comparing and drawing conclusions from them difficult at best.

## GENERAL MORBIDITY AND MORTALITY

### General Morbidity

**Risk factor studies—**All of the studies that met our inclusion criteria for the effect of the economy on general morbidity focus on individual experiences of job loss due to business closure. Using data from the entire Swedish population, Eliason & Storrie (54) found a countercyclical effect of job loss due to plant closure on hospitalization during the following

12 years for alcohol-related conditions in men and women, and traffic accidents and self-harm among men (hazard ratios ranging from 1.2–1.4). They found no increase in severe cardiovascular disease.

Kuhn et al. (96) suggested both stress and effect-budgeting mechanisms, but did not explicitly test either theory in their study of job loss due to plant closure in Austria. They found that job loss predicted an increase in prescriptions and hospitalizations due to mental health problems in men only. Payments of sickness benefits strongly increased after job loss, due to benefit policies rather than a real change in health.

Salm et al. (129) found no effect of job loss due to business closure on self-reported short-term physical or mental health using data from the Health and Retirement Study. These authors attempted to test for reverse causation but did not invoke a mechanism.

**Conclusions—**Risk factor studies have reported small but significant countercyclical associations between job loss and hospitalization, used as a surrogate for general morbidity. Research has not attempted to address the net effects of economic contraction on general morbidity.

### General Mortality

**Risk factor studies—**Several studies have invoked the stress mechanism to explain a countercyclical relationship between economic contraction and mortality. Job loss due to plant closure (53) and unemployment (102) have been associated with increased risk of mortality among men in Sweden. Likewise, both the neighborhood unemployment rate and individual unemployment have been associated with increased risk of mortality for men and women in Denmark (116), even after adjusting for individual-level risk factors. The effects on mortality appear strongest for alcohol-related causes and within the first four years after job loss.

Three studies invoke both stress and effect-budgeting theories to explain the countercyclical effect of unemployment or job loss on mortality. Two studies from Finland reported that the effect of unemployment on mortality varied inversely with the national unemployment rate, suggesting a selection effect (105, 107). A U.S. study found a 50–100% increase in the risk of death in the first year after job loss for high-seniority male workers, which decreased substantially to a 10–15% additional risk twenty years later (135).

**Net effect studies—**Several studies have found evidence that economic growth reduces mortality in England and Wales (12) as well as Denmark and the U.S. (23). An analysis using fixed-effects models for 26 European Union countries found that suicide, death from alcohol abuse, and homicides rose during economic contraction, whereas traffic deaths decreased (134). These studies suggested stress (12), effect budgeting (23), or implicitly invoke both (134) as the mechanisms.

However, a series of studies have identified procyclical effects of the economy on all-cause mortality. Tapia Granados has reported reduced mortality during economic contraction in the U.S. during the Great Depression (140) and throughout the 20$^{th}$ century (137), in Spain (138), and in postwar Japan (139). Countercyclical effects, however, appear for suicide in all cases, as well as for diabetes and hypertension in Japan. Tapia Granados also suggested that the relationship in Sweden reversed from countercyclical in the 19$^{th}$ century to procyclical in the 20$^{th}$ century (141). These studies invoke, but do not directly test, stress and effect-budgeting mechanisms.

Two additional studies, which invoke the theory of effect budgeting, found that mortality increased as the economy grew across 23 Organization for Economic Cooperation and Development (OECD) countries (69) and in the U.S. (126), with suicide as an exception in the US.

**Conclusion—**Risk factor findings suggest that job loss increases the risk of premature mortality across the developed world. The net effects work in this literature offers less convergence. Suicide, however, appears to be an exception, with countercyclical effects predominating.

## CONCLUSIONS

Our review of this wide-ranging literature leads us to several overarching conclusions. The risk factor research converges on the finding that undesirable job and financial experiences increase the risk for psychological and behavioral morbidity. The net effects research focusing on alcohol consumption remains mixed, and that concerned with violence suggests countercyclical associations during shallow economic declines but procyclical effects with steeper declines.

Findings from the work focusing on somatic outcomes do not converge. Risk factor research suggests that undesirable job and financial experiences will increase the risk of acute cardiovascular disease, non-specific morbidity, and mortality. Risk factor research on gestational outcomes remains generally too sparse to infer effects. Results from net effects research on somatic outcomes continue to diverge, thereby defying strong inference. Whether this failure to converge arises from differences in method (e.g., time-series vs. fixed-effect approaches), variability in the aggregation of time (e.g., months vs. years) or populations (labor markets vs. states or nations), or from real differences in associations across time and place, remains unclear.

Third, the field lacks empirical testing of mechanisms. We assigned research to the mechanisms shown in tables 1 and 2, but that assignment reflects invocation, rather than testing, of theory. The work predominantly tests against the null hypothesis rather than pitting conflicting theories against each other. Indeed, it remains unclear if this work, in whole or in part, contributes to the resolution of any basic controversies concerning biology or behavior. This strikes us as unfortunate since economic decline has been, and will likely remain, among the most feared of human scourges. Our adaptations to it must surely tell us something about our nature.

Tables 1 and 2 provide support for several inferences, including that contributors to the research most frequently invoke the stress mechanism. Perhaps reflecting the greater involvement of economists, the net effects work more frequently invokes effect budgeting than does the risk factor research. The involvement of psychologists in the research concerned with behavioral morbidity may likewise explain the appearance of frustration-aggression theory in that work.

We suggest that all the work in this field essentially invokes an “adaptation” model in which economic forces create circumstances different from those for which individuals, households, institutions, and communities had prepared. This difference requires individuals to adapt somatically and behaviorally in ways that allow them to meet social and economic obligations. In the case of economic decline, some of these adaptations increase the risk of illness while others reduce it. Such a perspective connects work in this field not only to human ecology (77) and ecological psychology (85), but also to that on tolerance among humans for biological and behavioral deviance (36).

The literature described in this review contributes to the field of public health by examining the health implications of some of our most important collective choices – those concerning the regulation of our economy. Findings from this type of research could, and should, contribute to cost-benefit analyses that presumably precede the formation of economic policy. The failure to test mechanisms and lack of consensus on methodology, however, keep most of the current literature from making a meaningful contribution to the debate over policy making. We suggest that, through more careful consideration and empirical testing of theory and mechanisms, future examinations into the health effects of contracting economies could not only inform economic policy, but could also identify vulnerable populations (e.g., job losers) and targets for intervention (e.g., reducing financial insecurity) during economic downturns.

## LIST OF KEY TERMS

| | |
|---|---|
| **Procyclical** | An adjective used to describe an association in which pathology declines when the economy declines |
| **Countercyclical** | An adjective used to describe an association in which pathology increases when the economy declines |
| **Time-series** | An adjective used to describe tests of association over time in which the researcher derives the counterfactual from historical values in the test population when unexposed |
| **Fixed-effect** | An adjective used to describe tests of association over time in which the researcher derives the counterfactual from values in unexposed populations at times when the test population has been exposed |
| **Autocorrelation** | Patterns that, by extrapolation, allow an observer to predict the next value of a time series more accurately than simply predicting the mean of all previous values. Autocorrelation must be controlled in tests of association because they assume that the mean of a series best predicts its next value |

## LIST OF IMPORTANT ACRONYMS

| | |
|---|---|
| **ECA** | Epidemiologic Catchment Area Study – a study supported by the National Institute of Mental Health intended to determine the true prevalence of psychological and behavioral disorder in the United States in the early 1980s |
| **NLSY** | National Longitudinal Survey of Youth – a longitudinal panel study of the experiences and well-being of young Americans |
| **HRS** | Health and Retirement Study – a longitudinal panel study of the experiences and health of persons nearing and following retirement in the United States |
| **CVD** | Cardiovascular disease |
| **NHIS** | National Health Interview Survey – a regularly conducted survey of American households intended to estimate health and health-related phenomena |
| **OECD** | Organization for Economic Cooperation and Development – An association of national governments, mostly those of the largest market economies, that studies and describes international economic policy |

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## SUMMARY POINTS

1. Research shows that undesirable job and financial experiences increase the risk of psychological and behavioral disorder, including violence and suicide.
2. The intuition that mortality increases when the economy declines appears wrong.
3. Tests of many hypothesized associations, including those between economic contraction and the aggregate consumption of alcohol, do not converge, leaving inferences unclear.
4. The existing research informs public health programming by identifying risk factors, such as job loss, made more frequent by economic decline.
5. The promise that the research would identify health costs and benefits of economic policy choices remains unfulfilled, and will likely remain so without stronger theory and greater methodological agreement.

## FUTURE RESEARCH

1. Research needs to better identify and test mechanisms that may connect declining economies to behavioral and somatic health. Ideally the field would attract more basic science researchers so that connections to well-understood processes that affect health and well-being can be identified and tested.
2. Research into the effects of economic contraction as an environmental stressor needs to adopt methodological conventions so that differences in results can be better understood.
3. We need more research into the effects of job or income loss in families on gestation and infant health.
4. We need more research into the effects of job or income loss in families on anxiety.

**TABLE 1**

Summary of Cited Literature Concerned with Psychological and Behavioral Morbidity

| Outcome | Association | Risk Factor Studies | | | Net Effect Studies | | |
|---|---|---|---|---|---|---|---|
| | | Mechanism | | | Mechanism | | |
| | | Stress | Effect Budgeting | Frustration-Aggression | Stress | Effect Budgeting | Frustration-Aggression |
| Depression/Anxiety | Procyclical | — | — | — | — | — | — |
| | Counter-cyclical | 8, 14, 20, 24, 47, 51, 58, 59, 64, 74, 86, 87, 102, 110, 111, 113, 120, 121, 123, 141, 144, 145 | — | — | 97 | — | — |
| Suicide | Procyclical | — | — | — | 5, 114 | 5, 114 | — |
| | Counter-cyclical | 6, 9, 15, 55, 80, 88, 92, 98, 122 | — | — | 67, 72, 90, 107, 125, 133, 136, 137–140, 147 | — | 90, 107, 125, 133, 137–140 |
| Violent Behavior | Procyclical | — | — | 26 | — | — | 30, 31, 38, 85 |
| | Counter-cyclical | 41 | — | 26 | 69, 118 | 118 | 30, 31, 35, 38, 85 |
| Substance Abuse | Procyclical | — | 54, 89 | 28, 89 | — | 44, 60, 124 | — |
| | Counter-cyclical | 3, 28, 49, 56, 57, 63, 73, 75, 79, 89, 109, 112 | — | — | 44, 66, 133 | — | — |

**TABLE 2**

Summary of Cited Literature Concerned with Mortality and Somatic Morbidity

| Outcome | Association | Risk Factor Studies | | | Net Effect Studies | | |
|---|---|---|---|---|---|---|---|
| | | Mechanism | | | Mechanism | | |
| | | Stress | Effect Budgeting | Frustration-Aggression | Stress | Effect Budgeting | Frustration-Aggression |
| Cardiovascular disease | Procyclical | — | — | — | — | 68, 100, 127 | — |
| | Countercyclical | 62, 65, 96 | — | — | 13, 19, 135 | 135 | 135 |
| Birth weight | Procyclical | — | — | — | — | 45 | — |
| | Countercyclical | 50 | 50 | — | 29, 34 | — | — |
| Fetal death | Procyclical | — | — | — | 25, 36, 37 | — | — |
| | Countercyclical | — | — | — | — | — | — |
| Infant mortality | Procyclical | — | — | — | — | — | — |
| | Countercyclical | — | 17 | — | 33 | 4, 16, 18, 33, 39, 42, 61, 99, 117 | — |
| General Morbidity | Procyclical | — | — | — | — | — | — |
| | Countercyclical | 53, 95 | 95 | — | — | — | — |
| General Mortality | Procyclical | — | — | — | 136–140 | 68, 125, 136–140 | — |
| | Countercyclical | 52, 101, 104, 106, 115, 134 | 104, 106, 134 | — | 12, 140 | 22 | — |