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SES gradient in psychological distress revisited: a dynamic perspective on the mediating effect of financial strain and mastery

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**SES gradient in psychological distress revisited:
A dynamic perspective on the mediating effect of financial strain and mastery**

by

Chih-Yuan Weng

A dissertation submitted to the graduate faculty
in partial fulfillment of the requirements for the degree of
DOCTOR OF PHILOSOPHY

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ABSTRACT

There is a well-established literature that both psychological distress and mental disorders are linked to the gradient of socioeconomic status (SES). According to the stress process model or the life stress paradigm, SES could affect mental health in at least two ways: first, by creating situations where lower SES people tend to experience stressors in greater quantity; second, by enhancing (e.g., due to underexposure of stress for high SES people) or undermining (e.g., due to overexposure of stress for low SES people) coping resources that are beneficial to psychological wellbeing. While the stress process model or the life stress paradigm underscores an intra-personal process where changes in stress, resources, and distress are hypothesized to be inter-correlated within the same individual over time, most previous research on testing relevant hypotheses has been cross-sectional by design, focusing on between-person differences in stress, resources, and distress across the SES spectrum. Even among those exceptions that have collected data at multiple occasions in time, the prevailing analytic approaches have failed to take into account individual variations in the trajectories (either growth or decline) of stress, resources, and distress across time. This study extends previous research by using panel data and latent growth curve (LGC) modeling to examine the extent to which intra-individual changes in depressive symptoms are related to

fluctuations in financial strain and mastery, which in turn, are conditioned by chronic level of income as a relatively stable SES attribute. This study also adds to previous research by investigating the causal sequence between psychological distress as indexed by depressive symptoms and a major form of personal resources as reflected in one's sense of mastery, since they have appeared to be causally reciprocal in their strong inverse correlation with each other as part of the general sense of demoralization.

CHAPTER 1

INTRODUCTION

Disruptions of mental health, either in terms of psychological distress or mental disorders, have been disproportionately present among those located at the bottom of the socioeconomic status (SES) hierarchies (Dohrenwend, Levay, Shrout, Schwartz, Link, Skodol, and Stueve 1992; Holzer, Shea, Swanson, Leaf, Myers, George, Weissman, and Bednarski 1986; Kessler, McGonagle, Zhao, Nelson, Hughes, Eshleman, Wittchen, and Kendler 1994; Link and Dohrenwend 1989; Link, Lennon, and Dohrenwend 1993; Miech, Caspi, Moffitt, Wright and Silve 1999; Williams, Takeuchi, and Adair 1992; Wheaton 1978). While, from a historical point of view, the strength of the relationship between SES and mental disruptions has been heavily contingent on what kind of health outcome was under consideration as well as how such outcome was conceptualized and measured (Dohrenwend and Dohrenwend 1982; Kohn, Dohrenwend, and Mirotznik 1998), nevertheless, depression, be it defined as distress or disorder, has been consistently documented to be inversely associated with SES. That is, individuals with higher SES are less likely to be afflicted by depression. Alternatively, the probability for one to suffer from depression increases with progressively lower SES (Gallo and Matthews 2003).

Other than previous work, this study intends to elucidate the underlying mechanisms of

observed disparities in non-disordered depression (i.e., a distress view of depression) across SES spectrum from a dynamic perspective. Before directly pursuing this substantial goal, it is important to outline some preliminary issues. To date, research on the association between SES and psychological disturbances has stimulated four major points of inquiry. The first one relates to a long-standing debate about whether symptomatology (e.g., depressive symptoms) or diagnosis (e.g., major or clinic depression) is more appropriate to serve the purposes of social epidemiology or sociology of mental health and illness (Horwitz 2002; 2007; Kessler 2002; Mirowsky and Ross 2002). There has been a discipline-oriented preference involved in choosing one over the other: sociologists generally prefer symptomatology, whereas psychiatrists use diagnostic categories more often. In essence, the choice between symptom counts and diagnostic categories largely reflects the history of instrument refinement, on the one hand, and has tremendous implications for the conclusions one may draw about the relationship between SES and mental disruptions, on the other (Kohn et al. 1998).

The second major research inquiry focuses on the causal direction between SES and disruptive mental health. Two common explanations have been suggested (Dohrenwend et al. 1992; Goldman 2001; Hudson, 2005; Johnson, Cohen, Dohrenwend, Link, and Brook 1999; Ritsher Warner, Johnson, and Dohrenwend 2001). The “social causation” perspective argues

that psychological disturbances are overrepresented in the lower social strata to the extent that the disadvantaged social and economic circumstances undermine the mental health of the poor by generating higher levels of stress and adversity. In contrast, the “selection/drift” perspective proposes that psychological disturbances either impair upward mobility (inter-generation process) or contribute to downward mobility (intra-generation process). This debate of causation vs. selection has long lived in the literature due to its profound implications for both theory and policy (Muntaner, Eaton, Miech and O’Campo 2004). Analytically, the choice between causation vs. selection prescribes the treatment of either SES or measures of mental disruptions as the dependent variable; for policy-makers, it determines whether limited budgets should be spent primarily on mental health services and pharmacotherapy (selection/drift perspective) or on other intervention/prevention efforts directed at reducing relative economic disadvantages and socially-arranged risk factors of mental disruptions (social causation perspective).

The third subject of inquiry concerns the relevance and prominence of more proximal psychosocial risks that may link SES to psychological disturbances. Since the middle of last century, Pearlin and colleagues (Pearlin 1989; Pearlin and Schooler 1978; Pearlin, Menaghan, Lieberman, and Mullan 1981; Pearlin, Mullan, Semple, and Skaff 1990) have formulated an

influential “stress process model,” which has become paradigmatic by attributing apparent health disparities to unequal distribution of psychosocial risk factors (including stressors and coping resources) in a convincing manner (Aneshensel 1992; Elliott 2000; House 2002).

With stress process integrated into the proposition of SES gradient in mental health, it was generally found that a substantial portion of the distressed or disordered individuals belonged to groups “most exposed to hardship” but “least equipped to deal with it” (Pearlin and Schooler 1978: 18).

As far as depression is concerned, particularly in terms of non-disordered form of depression (Horwitz 2007), past research has identified a wide range of stressors derived from social arrangements that might have been implicated in the “social patterns of distress” (Mirowsky and Ross 2003:253). In essence, much attention has been paid to financial or economic strain (e.g., Angel, Frisco, Angel, and Chiriboga 2003; Keith 1993; Krause 1997; Pearlin and Schooler 1978; Pearlin et al. 1981; Price, Choi, and Vinokur 2002; Voydanoff 1990). Generally speaking, evidence suggests that financial strain, usually chronic in nature, tends to generate secondary or follow-up stressors in both the original economic domain and other non-economic life domains, thus triggering depressive responses from time to time across life course to the extent that recurrent and diverse stressors could profoundly

undermine one's coping resources. Among others, the sense of mastery, a feeling that everything in one's life is under one's own control (Pearlin 1999a; 1999b), is probably one of the most-investigated personal resources that have been shown to bear an immediate and negative influence on the episode of depression (Hobfoll, Johnson, Ennis, and Jackson 2003; Mirowsky and Ross 2003; Turner and Lloyd 1999; Turner and Roszell. 1994).

However, two questions remain unsettled about financial strain and mastery as the potentially crucial psychosocial factors linking SES and depression. First, given that mastery and depression are both psychological constructs that cannot be directly observed but can only be inferred, more evidence is needed to demonstrate whether they are just causally reciprocal in their inverse correlation with each other as part of the general sense of demoralization, or the causal flow does pass from mastery (personal resources) to depression (affective outcome) as postulated by the stress-distress paradigm. Second, little is known about whether financial strain and mastery vary over time to a significant degree and, if they do, how changes in financial strain and mastery mediate between relatively stable rank in the macro-level stratification system (i.e., SES) and fluctuating individual depressive responses over a specific period of time (George and Lynch 2003; Holahan, Moos, Holahan, and Cronkite 1999; Hobfoll et al. 2003).

With more and more attention paid to time in the stress research, the fourth and last issue centers on the within-person dynamics in the SES-depression relationship (George and Lynch 2003). As noted before, our understanding of the mechanism underlying the SES gradient in depression has been greatly improved by the implementation of stress process model and its variants in the sociology of mental health and illness (e.g., Ensel and Lin 1991; Lin and Ensel 1989). For example, the sense of mastery has been identified as both a direct function of SES and an important dimension of human agency that is able to foster or suppress depression. (Mirowsky and Ross 2003; Pearlin 1999a; 1999b; Thoits 1995; 2006; Turner and Roszell 1994). However, most previous research on the mediating or moderating effect of mastery has largely been cross-sectional by design (e.g., Mirowsky, Ross, and Van Willigen 1996; Turner and Lloyd 1999; Turner Lloyd, and Roszell 1999), such that intra-personal increases or decreases in, say, depressive responses that are attributable to prior change in stress or resources within the same individual (e.g., growth/decline in the sense of mastery followed by decline/growth in depressive responses) can only be inferred from the between-individual differences in corresponding measures (e.g., those with higher/lower sense of mastery reporting less/greater level of depressive responses). This apparent inconsistency between the unit of actual analysis (i.e., individual) and the unit of what is inferred (i.e., time) lends itself

to a danger analogous to “ecological fallacy” (Grzywacz, Almeida, Neupert, and Ettner 2004).

While a few noticeable studies have adopted panel designs, change was predominantly estimated in terms of “residualized scores” with autoregressive models (e.g., Maciejewski, Prigerson, and Mazure 2000; Pearlin et al. 1981; Price et al. 2002; Turner and Noh 1988).

Such an approach to studying change, however, has long been criticized because of its insensitivity to the absolute within-individual change over time and between-individual variation in the trajectories of development on variables of interest. Fortunately, some advanced analytic frameworks for longitudinal data have been developed to deal with the shortcomings of autoregressive models; one of which is so-called latent growth curve (LGC) analysis (Lorenz, Wickrama, and Conger 2004a; Rogosa, Brandt, and Zimowski 1982; Willett and Sayer 1994; Wickrama, Beiser, and Kaspar 2000).

In keeping with previous findings on SES-depression relationship that mostly support the social causation hypothesis (inquiry 2) particularly when depression was defined as distress and measured by symptomatology (inquiry 1), this study addresses the remaining two research inquiries (inquiry 3 and 4) by (1) bringing SES back in as the fundamental cause of mental wellbeing instead of a mere control, (2) including financial strain as the cognitive component of SES and the primary stressor based on social arrangements, and (3) from a

dynamic perspective, examining the mediating effect of financial strain mastery in linking SES and depression by evaluating the extent to which changes in financial strain (stressor), mastery (personal resources), and depressive symptoms (health outcome) are associated with one another within the LGC analytic framework.

Based on a panel of sample that was originally designed to investigate how families in rural Iowa responded to economic stresses brought by the farm crisis of the 1980s (Conger and Elder 1994; Elder and Conger 2000), this study traces the decade-long (1991-2001) trajectories of financial strain, sense of mastery and depressive symptoms of a panel of 403 women and 305 men who were mothers and fathers of adolescent children in 1991, respectively, and investigates how these trajectories are causally inter-correlated under the contextualizing effect of SES as measured by chronic income between 1991 and 1994. As will be discussed later in greater detail, these families were especially well-suited for a study of stress and health because they were included in a project that was launched in the wake of the financial farm crisis of the mid-1980s. It was during this time that some but not all farm families lost their farms, and some but not all families living in rural villages and cities lost their jobs as implement dealers and other agricultural related companies went out of business. In effect, the farm crisis elongated the distributions of income, with some losing money and

incurring debt, financial strain, deprivation of mastery, and psychological distress to a significant extent. There was even a higher incidence of diagnosed mental disorders.

Briefly put, as outlined in Figure 1, it is hypothesized by this study that when considered together, (a) the trajectory of financial strain acts as a complete mediator between chronic income and the trajectory of mastery (i.e., P_2 and P_4 are hypothesized to be significant but P_5 is not in the full model), and (b) the trajectory of mastery acts as a complete mediator between the trajectory of financial strain and the trajectory of depressive symptoms (i.e., P_4 and P_6 are hypothesized to be significant but P_3 is not in the full model). More details about the research hypotheses and the characteristics of the panel sample used by this study are discussed later.

The remainder of this dissertation is arranged as follows. Chapter 2 reviews literature of stress research in greater detail and formulates more specific research hypotheses. Chapter 3 describes the sample, measures, and methods. Result is presented in Chapter 4. Finally, Chapter 5 discusses the potential limitations of current research and suggests what else needs to be done in the future.

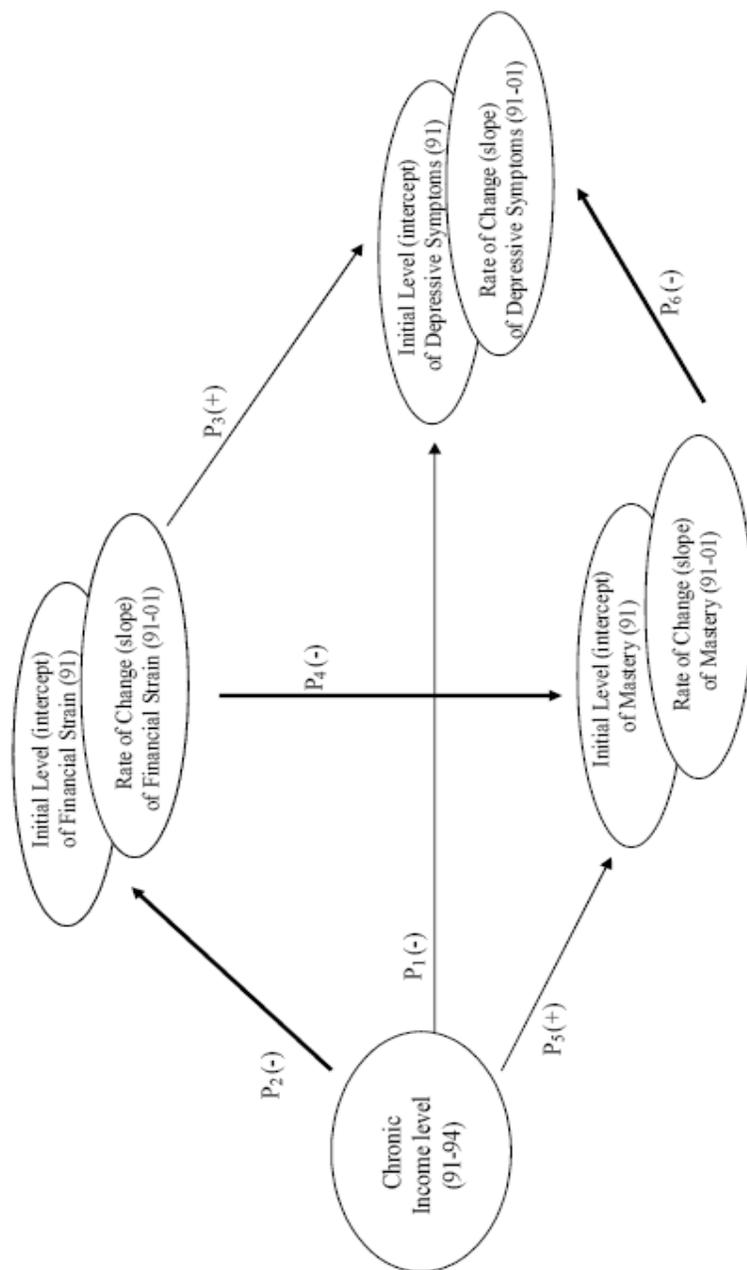


Figure 1 Conceptual model for the dynamic relationship between SES and depressive symptoms mediated by financial strain and mastery

CHAPTER 2

LITERATURE REVIEW AND THEORETICAL FRAMEWORK

2.1 Measurement of Depression: Symptoms or Diagnosis?

2.1.1 Theoretical debate

There has long been a conflation between distress (usually measured by symptomatology) and disorder (usually reflected by a diagnostic category) as the outcome variable in the sociology of stress. As indicated by Horwitz (2007), distress stands for natural rather than pathological responses made by non-disordered people that are proportionate to stressful social conditions. Distress is usually transitory insofar as it tends to abate or disappear as the stressful conditions are removed or as people gradually achieve a positive adjustment. In contrast, disorders are syndromes for internal psychological dysfunction that are better predicted by previous episodes or/and family history of disorders than by stressful social arrangements. Generally speaking, disorders are disproportionate to external conditions: they are more likely to be chronic as well as recurrent but less responsive to changing circumstances. Since the sociology of stress is more interested in the impact of stressful social arrangements on health and wellbeing, Horwitz (2007) further argues that it is consequential to distinguish distress from disorders and to realize that what we usually study in sociology of mental health and illness is normal responses to stressful situations other than

mental disorders that are characteristic of pathological syndromes.

This study embraces the distress view of depression for two reasons: first, as already noted, there has been a discipline-oriented focus on the stressful aspects of social conditions and reluctance to medicalize largely natural responses to external stressful situations; second, from a measurement point of view, a distress index is more compatible than a diagnostic category of disorder with the purpose of sociology of stress, in general, and of this current study, in particular.

Specifically, depression has been conceptualized and measured by two major traditions. While some view depression as a manifestation of a specific dimension of psychological distress, others define depression as a medical condition or psychiatric disorder one either has or does not have.

The emphasis on dichotomies, not surprisingly, results from a medical way of thinking about the disruptions to mental health (Luhmann 2000). In principle, the clinical perspective of major depression distinguishes “cases” (i.e., those who are diagnosed as clinically depressed by reporting more symptoms than a somewhat arbitrarily pre-specified threshold) from “non-cases,” as has been the conventional practice of psychiatrists (Pearlin 1999a; Mirowsky and Ross 1989). Within this tradition, more importantly, there has been a belief

that without first obtaining a clinical diagnosis, it is hardly possible to make a treatment decision which is also categorical (i.e., to treat or not to treat), to identify the untreated people who are actually in need of clinical treatment, and to gather lifetime information on episode occurrences, duration of episodes, and interval between episodes of a specific discrete syndrome (Kessler 2002).

It is noteworthy that virtually all diagnostic instruments specify a minimum number of symptoms as a *sine non qua* for ascertaining a positive diagnosis (Aneshensel, Rutter, and Lachenbruch 1991; Mirowsky and Ross 2003). However, by relying on sharp cut points between health and illness, say, 7 symptoms are required to achieve a diagnosis of major depression, those who endorse 5 or 6 symptoms are, paradoxically, placed in the same category as those with no symptoms and together as a whole considered clinically different than those with 7 symptoms (Mirowsky and Ross 2002, 2003).

Finally, the categorical view tends to portray psychiatric disorders as discrete entities that do not overlap with one another. For example, one's affective disturbances, if already diagnosed to be pathological, need to be determined as either depressive disorder or anxiety disorder, but not both. That is, one has to be assigned into one, and only one, disorder at a time. With different disorders portrayed as discrete entities, however, the diagnostic

categories tend to proliferate to accommodate the situations where people endorse symptoms simultaneously present in more than one presumed clusters. “Comorbidity” is therefore created to reflect the presence of more than one discrete disorder. For example, “schizo-affective disorder” refers to those who happen to have symptoms belonging to both schizophrenia and affective disorder (Horwitz 2002; Mirowsky and Ross 2002; 2003).

As far as the distress view is concerned, in contrast, it is neither desirable nor possible to assign individuals to a set of mutually exclusive diagnostic categories. Rather, each particular *type* of psychological problem (e.g., depression or anxiety) can be indexed by a *severity* score which registers each individual’s own level of suffering along a specific dimension of psychological distress, even though different dimensions generally intersect in reality to some extent by sharing common symptoms (i.e., it is legitimate for one to be both depressed and anxious). In essence, the psychological distress view assesses depression with symptoms check-lists of both mood (feelings) and malaise (bodily state) items, hence reflecting the degree to which one is depressed on a continuum ranging from not at all to extremely severe. According to this view of depression, it is not necessary for people to be “cases” to get help. What is of more interest is to know why some suffer more than others, even by only a small margin, so that an intervention can be designed as a result (Mirowsky and Ross 2002, 2003).

Obviously, the choice between diagnoses and symptom counts involves trade-offs, in both theoretical and practical terms. Kessler (2002) argues that the dichotomous measures can be useful even when the existence of true discrete mental disorder lacks reasonable justification. He continues to suggest that it be a desirable goal for sociological studies of mental health and illness to use diagnoses and symptom counts in tandem and explicitly compare findings from different outcome measures whenever is possible.

Despite the fact that it seems optimal to include both categorical assessments and continuous indexes, practical considerations usually dictate an either-or decision. Generally speaking, sociological studies of mental health and illness prefer continuous symptoms to dichotomous diagnoses because the diagnostic categories are less informative to the extent that they toss out substantial variation in the social distribution of psychological disturbances. For example, the stressful consequences of social arrangement can be underestimated if one just disregards the borderline disruptions to mental health (e.g., individuals reporting 5 or 6 depressive symptoms for the 7-symptom diagnostic criteria) and positive extreme of mental well-being (e.g., individuals reporting 0 depressive symptoms) that fail to make the “case” (Horwitz 2002; Mirowsky and Ross 2002, 2003). In addition to the greater capability to preserve the between-individual variation, continuous scales of depression are also more

likely to reveal within-individual changes in one's depressive responses across time, which is exactly the focus of the current study. On the contrary, dichotomous diagnoses are not sensitive to any temporal fluctuations in depressive responses that fail to re-classify one's diagnostic status (i.e., diagnosis of depressive disorder) between two points in time.

In sum, the categorical diagnosis of depressive disorder is not really compatible with the sociological portrayal of depression as normal distressed response. Moreover, the utilization of the diagnostic criteria is apparently at the cost of the rich information about between- and within-individual variation in depressive responses that is otherwise available in terms of symptom counts.

2.1.2 Empirical findings: The three generations of studies

In general, the relationship between SES and diverse mental disruptions appears to be susceptible to how the latter is identified and categorized. In particular, different approaches to measuring depression ever developed have had important historical implications for the strength of the SES-depression relationship. It has been argued that sociological research on psychopathology can be divided into three recognizable generations, depending primarily on how health outcome was measured and what samples of respondents were obtained (Dohrenwend and Dohrenwend 1974; 1982; Horwitz 2002; Kohn et al. 1998). Table 1

summarizes the distinguishing measurement and sample characteristics of each of the three generations of studies.

Table 1 Distinguishing characteristics of three generations of psychopathological studies

<i>Psychopathological Assessment</i>	Clinical Diagnosis	Symptom Counts
<i>Sample Selection</i>		
Clinical or Practitioner Sample	1 st Generation	
Community Sample	3 rd Generation	2 nd Generation

The first generation of studies, mostly conducted before World War II, relied heavily on the records of mental hospitals and other official agencies for measures of outcome. With an exclusive focus on the clinical cases (i.e., those who received treatment) and a premature nomenclature for psychopathology, the population of those with psychiatric disorders could have been seriously underestimated. For example, the median prevalence rate was only 3.6% for all recognizable types of disorders combined in the first generation of studies (Dohrenwend and Dohrenwend 1974, 1982)¹. More importantly, the correlation between SES

¹ According to the epidemiological conventions, incidence, usually better at identifying risk factors, is the rate at which a disorder develops after a follow-up interval among a cohort of individuals who were previously unaffected. More useful for evaluating the need of health services, prevalence is the proportion of population that

and psychopathology could also have been confounded by the availability of medical facilities and public attitude toward the use of treatment (Dohrenwend and Dohrenwend 1974): these two factors might have substantially contributed to a sampling bias insofar as those who were highly mentally-ill did not receive treatment due to the lack of mental hospitals in their geographic proximity or their fear of being alienated by others.

The second generation of studies was largely characterized by expanding the definition of psychiatric disorders, directly interviewing untreated community residents, and summing self-reported symptoms from continuous scales. The median prevalence rate for all types of disorders combined was approximately 20% among these studies after WWII (Dohrenwend and Dohrenwend 1974; 1982). A number of investigators in this generation dispensed with clinical judgments altogether and instead used seemingly arbitrary cutoff scores in screening scales that were composed of symptom items to distinguish likely clinical disorder from less severe distress. Examples include 22-item Langner Scale (Langner 1962), the Center for Epidemiological Studies Depression Survey (CES-D: Radloff 1977), and Symptom Checklist 90 (SCL-90: Derogatis 1977; 1983). During the same period, considerable effort was devoted to elaborating sociological theories of stress (e.g., Pearlin 1989; Pearlin et al. 1981; Lin and

has a positive diagnosis for a disorder within a specific time period, which could be affected by any factors that may influence the course or duration of a disorder after onset (Gallo and Lebowitz 1999).

Ensel 1989; Ensel and Lin 1991; Thoits 1987), whereas less attention was paid to refining the outcome measures (Horwitz 2002). Despite the obviously economical advantages, these screening scales were criticized mainly in two respects: first, earlier global measures usually included confounding questions that were physiological in content and reflected only non-specific distress that might not correspond to diagnosable discrete mental disorders; second, even for more specific measures like CES-D that is used to manifest one's depressive symptomatology, it was still difficult to tell whether higher scores revealed the substantial existence of mental disorders or they just indicated non-disordered responses proportionate to highly stressful situations (Dohrenwend and Dohrenwend 1974; 1982; Horwitz 2007; Kohn et al. 1998).

While providing no direct information on the link of SES to major or clinical depression, notably, the second-generation of studies largely confirmed a linear inverse relationship between SES and depressive symptomatology, with the health outcome generally interpreted as “demoralization” (e.g., Kessler 1982) and measured with objective screening scales, such as CES-D (e.g., Aneshensel, Frerichs, Clark, and Yokopenic 1982), Langner scale (e.g., Cockerham 1990), and Psychiatric Epidemiology Research Instrument Demoralization Scale (e.g., Dohrenwend, Levav, Shrout, Link, Skodol, and Martin 1987).

The third and current generation of studies was initiated by the third edition of Diagnostic and Statistical Manual of Mental Disorders (DSM-III) criteria published by American Psychiatric Association in 1980. Since then researchers have been able to test the association between SES and specific mental disorders among community residents with structured clinical interview schedules and explicit diagnostic instruments. Representative examples of the third-generation instruments are the International Classification of Disease (ICD) criteria (World Health Organization 1978), Diagnostic Interview Schedule (DIS: Robins, Helzer, Croughan, and Ratcliff 1981), and the Composite International Diagnostic Interview (CIDI: Robins, Wing, Wittchen, Helzer, Babor, Burke, Farmer, Jablenski, Pickens, Regier, Sartorius and Towle 1988).

Despite the significant improvement in diagnostic reliability, findings based on the third-generation instruments were less likely to establish the linear link between SES and depressive disorder, indicating that the relationship between the stressfulness derived from social arrangements and major or clinical depression may not be as straightforward as suggested by previous studies with non-specific distress or depressive symptom counts as outcome. Instead, the relationship between SES and major or clinical depression has been found to vary by sex, urbanicity, ethnicity, age, and sometimes the diagnostic instrument

itself, as demonstrated by a series of studies from the Epidemiologic Catchment Area (ECA) program (Kohn et al. 1998).

The ECA was a typical example of the collaborative multi-site studies in the third generation. Panel surveys of population were collected with one-year interval from five metropolitan Community Mental Health Center catchment areas in the United States, including New Haven (CT: Yale), Baltimore (MD: Johns Hopkins), St. Louis (MO: Washington University), Durham (NC: Duke), and Los Angeles (CA: UCLA). The primary aims of ECA were to estimate prevalence and incidence of specific mental disorders as diagnosed by DSM-III, investigate risk factors, and examine the patterns in use of facilities (Eaton, Regier, Locke, and Taube 1981).

The conclusions obtained from various ECA reports have been inconsistent about the relationship between SES and major or clinical depression (Kohn et al. 1998). Weissman and colleagues (1991) found that, even though 1 year prevalence was not significantly associated with education, income, or occupation, subjects receiving public financial assistance had greater risk of major depression. The Eastern Baltimore Mental Health Study (EBMHS), an offshoot from the Baltimore ECA study (Robins, Helzer, and Weissman 1984), however, reported no significant inverse relationship between current episodes of major depression and

SES as measured by education, employment or receipt of public assistance after adjusting for sex, race, age, and marital status. While an inverse association between 6 month prevalence and SES was discovered, the relationship appeared to hold only for women (Holzer et al. 1986) and whites (Williams et al. 1992).

What may have suppressed the link of SES to depressive disorder deserves further systematic investigation. Notably, the DIS has been reported to possibly bias the inverse association between major depression and SES, making the sampled relationship weaker than it really is in the population (Kohn et al. 1998). Part of the reason could be that DIS tended to underdiagnose major depression (Helzer, Robins, McEvoy, Spitznagel, Stoltzman, Farmer, and Brockington 1985). Although it is still unclear whether the same bias applies to other diagnostic instruments that produce dichotomous diagnosis, it does not seem very surprising that the third-generation research on the whole failed to detect the socioeconomic origins of depression, which had been more easily revealed by their predecessors (i.e., studies in the second generation): first, a dichotomized measure usually has lower reliability given the lack of precision in a crude split; second, as already mentioned, a substantial loss of information is always inevitable when using a categorical diagnosis, such that the resulting measure of health outcome has limited variation and its correlation with SES are in all likelihood

disguised (Mirowsky and Ross 2002; 2003).

More recent evidence also confirms that the SES gradient in depression is more readily observed in terms of depressive symptoms than major or clinical depression. For example, Gallo and Matthews (2003) concluded in their extensive review that a majority of the associations they examined suggested a strong inverse relationship that tended to be linear between SES and depressive symptoms (64% of the studies examined), whereas evidence was somewhat weaker for incident depressive disorders (57% of the studies examined) and for prevalent depressive disorders (50% of the studies examined). It is also worth mention that evidence for the linear inverse association seemed to be stronger and more consistent among studies using either income alone or some composite measures of SES than among those approximating SES by education or occupation, respectively (Gallo and Matthews 2003; Lorant, Deliège, Eaton, Robert, Philippot, and Ansseau 2003).

Consistent with the methodological standpoint that symptom scores are more capable of reflecting both between- and within-individual differences in depression than categorical diagnoses, to conclude, accumulating empirical findings have consistently suggested that depressive symptomatology works better than clinical depressive diagnosis for researchers, first, to discern the social origins of stressful consequences (i.e., the SES gradient in

depression), and, second, to look into the psychosocial pathways that may mediate between the depressive responses and the relative position in the overarching hierarchies. As far as the measures of SES are concerned, equally important, more recent review studies have indicated that the linear inverse association between SES and depression is most evident when SES is indexed by either a single measure of income or a composite measure that combines income, education, occupation, and more.

2.2 Causal Order of the SES-Depression Relationship: Social Causation or Social Selection?

Despite the appreciable fluctuation in the effect size due to alternative outcome measures, sample characteristics, and analytic procedures, at present there is little doubt about the existence of the inverse SES-depression association, particularly when SES is denoted by income and the symptom scores of depression are used as the outcome measure. However, the underlying mechanisms and the causal direction implicated in such an association have remained a major issue (see Hudson 2005 for a historical review). For the past decades, two dominant interpretations have arisen with respect to the causality between SES and psychological wellbeing. First, the selection-drift hypothesis predicts that various forms of mental disruptions (inherited or not) may channel individuals into the bottom of society or

prevent them from rising out of the 'residue group' of low SES. According to the social causation model, alternatively, it is the adversities associated with low SES that cause the psychological disturbances of virtually all kinds.

Researchers have come to an agreement that social causation processes are responsible for most of the observed disparities in physical health (Goldman 2001). When attention is directed to the psychological realm, however, evidence has shown that social causation and social selection processes collectively contribute to the SES gradient in mental health, and the relative importance of either direction could in fact depend on the health outcomes of interest (Johnson et al. 1999). For example, most of the support for selection-drift hypothesis came from studies examining schizophrenia and other severe cognitive disorders (Dohrenwend et al. 1992; Eaton 1980; Eaton and Lasry 1978; Eaton and Muntaner 1999; Goldberg and Morrison 1963; Turner and Wagenfeld 1967; West 1991). Conceivably, severe cognitive disorders such as schizophrenia tend to strike in the late adolescence or early adulthood, which is a critical period in one's lifetime to establish (or fail to establish) a career. In contrast, social causation is more likely to be set in motion for affective impairments such as anxiety and depression, especially when they are interpreted as distress or demoralization and indexed by symptomatology (Dohrenwend et al. 1992; Hudson 2005; Link et al. 1993;

Moos, Cronkite, and Moos 1998; Wheaton 1978). As far as depression is concerned, in essence, the causal route from earlier achieved status to subsequent onset prescribed by social causation seems to be particularly evident for adult samples relative to adolescents (Costello, Compton, Keeler, and Angold 2003; Kessler, Davis, and Kendler 1997; Miech et al. 1999; Muntaner, Eaton, Diala, Kessler, and Sorlie 1998), and tends to hold even after controlling for parental general psychopathology (Johnson et al. 1999) and parental depression (Ritsher et al. 2001). In sum, there has been a consensus in social epidemiology or sociology of mental health and illness: it is the SES characteristics that determine the onset of depression rather than the other way around, at least when the outcome is measured by depressive symptomatology instead of major or clinical depression.

Although the causation vs. selection debate is not yet, and apparently will not be, completely settled, from a pragmatic viewpoint, it may prove more valuable to identify the mechanisms that intervene in the association between SES and depression than to repeatedly test the causal order per se. Once the intervening mechanisms are revealed, regardless of the casual direction, some life-improving programs may be designed as a result to either prevent the onset of mental disruptions for people of lower SES (causation hypothesis) or to minimize the adverse consequences of psychological disturbances on ensuing status

attainment process (selection hypothesis) (Eaton and Muntaner 1999).

2.3 Mechanism Linking SES to Depression: The Utility of the Stress Process Model

With respect to physical health outcome such as mortality, more recent research has argued that social conditions, generally, and SES, particularly, are the “fundamental causes” of fatal diseases given the fact that the SES-mortality relationship is so robust across time and places. The SES gradient in mortality, as suggested by the fundamental cause perspective, cannot be fully understood with an exclusive focus on the intervening risk factors (e.g., smoking, exercise, diet, etc.), because the definitions of risk factors are time-varying such that factors deemed risky in a specific historic period may not be considered so in another. However, there is little exception that lower SES people throughout the history are more likely to get exposed to their contemporaneous risk factors for fatal diseases (House, Kessler, and Herzog 1990; House and Mortimer 1990; Link and Phelan 1995; Phelan and Link 2005; Phelan, Link, Diez-Roux, Kawachi, and Levin 2004).

While the idea of “fundamental causes of diseases” points out the importance of “bringing the social back in” for the sociological study of physical health, considerable more and more research with mental impairments as outcome has shown that, compared to relatively objective indicators of SES, some psychosocial risk factors such as financial strain,

sense of mastery, or social support have contributed to the discernible SES gradient in psychological wellbeing in a consistent and substantial way (Pearlin et al. 1981; Turner and Lloyd 1999; Turner and Noh 1983; Turner, Wheaton, and Lloyd 1995; Turner et al. 1999).

Since the beginning of the second generation of sociology of mental health and illness, as described earlier, there has been considerable effort on elaborating the socio-psychological variables that are potentially responsible for the stressful consequence of social arrangements. Formulated by Pearlin and colleagues (Pearlin 1989; Pearlin and Schooler 1978; Pearlin et al. 1981; Pearlin et al. 1990) in the second half of last century, in particular, the “stress process model” incorporates stress (e.g., general vs. specific, acute vs. chronic), coping strategies (e.g., problem- vs. emotion-focused), and other psychosocial resources (e.g., self-efficacy and social support) into a single conceptual framework to account for the social distribution of distress². Building on Pearlin and colleagues’ work, the “life stress paradigm,” as coined by Lin and Ensel (Ensel and Lin 1991; Lin and Ensel 1989), has further laid out a comprehensive set of competing hypothetical interrelationships among stressors, coping

² Evidence is relatively equivocal about how coping strategies work in the stress process (Aneshensel 1999): not only may the activation of a specific coping style (e.g., problem-focused vs. emotion-focused) be contingent on individual dispositions (Endler and Parker 1990) or the nature of specific stressful situations (Lazarus 1981; Lazarus and Folkman 1984), but its effectiveness for adaptation may vary such that what seems to be adaptive in one context may not be the case in another (Pearlin 1989; Pearlin and Schooler 1978).

resources, and health outcomes that can be most appropriately tested using panel data.

Guided by the “stress process model” or the “life stress paradigm,” ensuing sociological studies of mental health and illness have been able to explain substantial variability in individuals’ health outcome as a consequence of their social statuses, either ascribed or achieved. For example, Marmot, Fuhrer, Ettner, Marks, Bumpass, and Ryff (1998) collected cross-sectional data in the U.S. to explore the association among educational attainment, nine types of psychosocial factors (neighborhood poverty, childhood socioeconomic environment, smoking, social relationships, social support, relationship strain, perceived inequalities, psychosocial work environment, and perceived control), and psychological wellbeing comprised by six dimensions of positive psychological functioning (autonomy, environmental mastery, personal growth, positive relationship with others, purpose in life, and self-acceptance). Despite some noticeable overlap between the measurement of psychosocial factors and that of psychological wellbeing, they found controlling for all psychosocial factors at the same time accounted for substantial effect of education attainment on psychological wellbeing. Specifically, with all explanatory variables taken into account, the odds ratio for fair/poor psychological wellbeing in the lowest educational group (i.e., less than high school graduate) dropped from 4.83 to 3.81 for men and from 5.92 to 3.07 for

women, as compared to those in the highest educational group (i.e., BA/graduate degree). Another cross-sectional study using data from Canada (Turner and Lloyd 1999) found that insofar as all six selected components of the stress process (i.e., stress, mastery, self-esteem, emotional reliance, assertion of autonomy, and social support) were held constant, the regression coefficient on SES dropped by more than 91 percent in magnitude for one model with depressive symptoms as dependent variable and by 47 percent for another model predicting depressive disorder. When simultaneously entered into the regression equation, furthermore, all six elements of stress process significantly mediated the SES effects in the model for symptomatology, but only two factors (i.e., stress and mastery) had significant independent effect in the model for major depression. It is particularly noteworthy that mastery was the most important mediating factor across model for depressive symptoms and model for depressive disorder.

Similar evidence is also available from longitudinal research for the great utility of psychosocial risk factors to account for the SES gradient in depression. For example, Eaton, Muntaner, Bovasso, and Smith (2001) demonstrated that by adjusting for the family history of depression, the incidence of depressive syndrome (as defined in a way closely related to the diagnosis of minor depression) was minimally implicated in the process of status

attainment either as cause (selection/drift hypothesis) or consequence (social causation hypothesis), whereas the psychological demand associated with the pace of work was significantly related to higher odds of the outcome variable (i.e., depressive syndrome). One interpretation for this finding offered by Eaton and colleagues (2001) is that high job demand, particularly when combined with perceivable financial dependence on some form of welfare, lends itself to the adverse psychological feeling comparable to powerlessness (Seeman 1959; Seeman, Seeman, and Budros 1988), helplessness (Seligman 1975), hopelessness (Abramson, Metalsky, and Alloy 1989), or being trapped in a punishing situation that never ends (Brown, Harris, and Hepworth 1995), which in turn have a demoralizing effect to provoke non-specific distress, in general, or depression, in particular (Mirowsky and Ross 2003). As a conclusion, they argued that variables drawing on more social-psychologically oriented theories, such as psychosocial risk factors associated with work environment and poverty status, might be more relevant and prominent etiologic sources of depression than the macro-level socioeconomic rank.

A more recent study in the United Kingdom also showed that even with adjustment for initial psychiatric morbidity and SES, reporting of financial difficulties at baseline was still significantly associated with an increased risk for major depression at follow-up, suggesting

that apart from the objective indicators of SES, more subjective measures of financial hardship might be equally, if not more, important for the subsequent occurrence or persistence of clinical depression (Skapinakis, Weich, Lewis, Singleton, and Araya 2006).

Finally, Lorant, Croux, Weich, Deliège, Mackenbach, and Ansseau (2007) revealed that, during a seven-year study period (1992-9) in Belgium, increases in both depressive symptoms and incidents of major depression across annual waves were associated with soaring deprivation index and subjective financial strain. While becoming poor (defined as living in household with an income less than half of the population median income) was linked to increases in depressive symptoms but not in cases of major depression, in contrast, neither changes in income nor changes in employment status were related to changes in depressive symptoms or changes in cases of major depression.

From a theoretical point of view, the psychosocial approach to the study of mental health and illness has in principle emphasized two key elements (Aneshensel 1999). First, the concept of *stress* refers to the demands imposed by the circumstances. Second, *resources* are anything that can be used by individuals to offset demands from outside, be they material (e.g., financial assets), social (e.g., ties to or support from others), or personal (e.g., a sense of self-efficacy or mastery). This psychosocial orientation is crucial to understanding the social

origins of distress because it not only places individuals within a social context but explicitly pinpoints the interaction between the individual and the environment. Stress (or social stress) can thus be defined in a broader sense by the mismatch between individual and environment to the extent that demands imposed by the environment either exceed individuals' ordinary adaptive capacity or ability to make sought-after ends meet (Aneshensel 1992; 1999; Pearlin 1983). In addition to the interactional nature, there is also an important cognitive component involved in the concept of stress. According to the paradigm of stress research, individual responses that may have health implications can only be triggered when the given social conditions and associated environmental stressors come to be appraised as threatening. Moreover, appraisal of and response to given potential environmental stressors depends on social, psychological, and physical characteristics of individuals and of their situations (House 1974; 1981; 2002). It is therefore hypothesized by this study that SES affects depression mainly through the appraisal or recognition of objective economic adversity as reflected by financial strain, which in turn undermines the sense of mastery, one of the most-investigated personal resources that have been shown to bear an immediate and negative influence on the episode of depression (Hobfoll et al. 2003; Mirowsky and Ross 2003; Turner and Lloyd 1999; Turner and Roszell 1994).

2.3.1 Appraisal of economic adversity: Financial strain

Wheaton (1994; 1999) has argued that stress is such a complicated concept that cannot be adequately operationalized by the mere use of a checklist of recent life events. In the theoretical “stress universe” are there at least two major dimensions, along which different types of stressors can be arrayed. Along the first dimension, or what Wheaton refers to as “stress continuum,” stressors differ in terms of their discreteness (or acuteness) vs. continuity (or chronicity) as a problem, from sudden traumas at the most discrete end, through life-change events, daily hassles, nonevents (i.e., when something did not happen as anticipated, such as graduating from school) to chronic stressors at the most continuous end. Along the other dimension, stressors differ in terms of the level of social reality at which they occur and can be distinguished into micro events that occur at the junctures of daily life (e.g., daily hassles) and macro events that occur at the level of larger social system above the individual (e.g., economic recessions). Wheaton further suggests that, essentially, different types of stressors have distinct and cumulative effects on mental health, and one cannot fully understand the role of stress without incorporating a variety of acute and chronic stressors having ever occurred over the life course.

Several empirical prospective studies have provided evidence that later increases in

psychological distress over time could follow a prior exposure to daily hassles (Grzywacz et al. 2004), major life events (Aneshensel and Frerichs 1982; Ensel and Lin 1991; Maciejewski et al. 2000), chronic life strains (Aneshensel 1985; Pearlin and Lieberman 1979), or the interaction of the last two (Pearlin et al. 1981). Though not adopting a longitudinal design, Turner and colleagues (Turner et al. 1995; 1999) have further demonstrated that an extensive measure of stress made up of acute life events, chronic life strains, and major life-time traumas has a greater role to account for between-individual variability in both depressive symptoms and major depressive disorders than suggested by previous studies.

Not surprisingly, it is usually difficult to obtain a comprehensive as well as precise stress measure, particularly within a panel research design. On the one hand, it is costly for both researchers and subjects to extract multiple sources of stress across time. On the other, the information about early traumas is all but retrospective such that it is inevitably biased to some degree, depending on individuals' various capacities to recall things. As a result, it is more typical for stress research to focus on a limited number of stressors from the "stress universe," instead of including all conceivable categories of them.

Nonetheless, stress studies that only measure the simple "counts" of stressors of specific and limited kinds could be quite misleading for the purpose of understanding the stressful

consequences of SES or other social conditions. The first reason for this concern is that, even though it is very likely for lower SES people to experience a greater amount of “overall” stress than higher SES people, it may not be the case if the measure of stress is non-inclusive. That is, there is always a danger that selected stressors are not representative of the true “overall” stress.

By employing a wide range of measures of stress (i.e., recent life events, chronic stressors, lifetime major events, daily discrimination, and total combined stress), for example, Turner and Avison (2003) found that a significant monotonic SES-stress relationship (i.e., the lower the SES, the higher the stress) was most evident in terms of the total combined stress. Similar SES gradient in stress was also observed in each separate dimension of stress except for the category of recent life events. Therefore, previous research that relied exclusively on a life events checklist might have seriously underestimated the SES variation in “overall” stress exposure. As compared to recent life events that mediated little of the SES effect on depressive symptomatology, more importantly, the total combined measure of stress accounted for almost half of the observed SES differences in the same health outcome.

Second, as much as one is lucky enough to measure the “fundamental” stressors that are proportional in quantity to the more comprehensive version, exposures to a comparable

amount of selected stressors may still in fact have qualitatively different implications, depending on distinct stressor contents and individual perceptions.

For example, Grzywacz et al. (2004) found that, contrary to previous studies, college-educated individuals, despite revealing fewer physical symptoms and less psychological distress, reported more daily stressors than those with high school and less education. Their interpretation was that better-educated individuals were less vulnerable to daily stressors to the extent that those with less education reported higher levels of psychological distress and physical symptoms than their better-educated counterparts on days when individuals experienced stressors, and there were no education differences in psychological distress and physical symptoms on days when individual reported no stressors. A follow-up study (Almeida, Neupert, Banks, and Serido 2005) further indicated that the differential vulnerability to daily stressors could be accounted for by stressor *severity* (i.e., less-educated individuals experienced objectively more severe stressors) and stressor *appraisal* (i.e., less-educated individuals reported that daily stressors were more likely to pose risks to their financial situations and self-concept), suggesting that in order to better understand the effects of SES on psychological wellbeing through differential exposure to environmental demands, a proper stress measure should be able to reflect (1) the objective

magnitude of given or potential economic difficulties, and (2) individual's recognition of such given or potential economic difficulties.

Although there might be no "fundamental stress," as suggested by Wheaton (1999), this study chooses to approximate the latent "overall" stressfulness of social arrangements with financial strain based on two reasons: presumably as a direct function of SES, financial strain is not only one of the most influential primary stressors that can trigger or exacerbate other secondary stressors in both the economic and non-economic life domains (Pearlin 1989; Pearlin et al. 1981; Pearlin, Schieman, Fazio, and Meersman 2005), but it also has a cognitive component that corresponds to the emphasis of stress research paradigm on the appraisal of the social conditions and environmental demands as a prerequisite for subsequent health consequences (House 1974; 1981; 2002).

Originally elaborated by Lazarus and Folkman (Folkman 1984; 1992, Folkman and Lazarrus 1980; Lazarrus 1981; Lazarus and Folkman 1984), the appraisal-based stress model views coping as a dynamic process where recognitions of potential threat on the cognitive level mediate between life stressors and subsequent adaptive responses. With the distinction between sources of danger (primary appraisal) and the availability of coping resources and options to deal with the danger (secondary appraisal), appraisal is understood to be the

cognitive process through which individuals define a specific person-environment relationship in one of the three ways: already-occurred damage (harm/loss), yet-to-occur damage (threat), and opportunity for mastery or gain (challenge). According to this contextual approach to the stress process, coping is not a stable personal trait but varies over time in response to changing appraisals of the demands imposed by the situation.

In essence, this study draws on the idea of appraisal from the stress research paradigm and proposes that “feeling poor,” as reflected by the elevated level of financial strain, is an important cognitive pathway linking “being poor” in terms of low levels of chronic income and the occurrence/persistence of psychological distress as indexed by depressive symptoms (Blacksher 2002). By asking whether one has enough income to meet daily expenses or basic needs, specifically, the scale of financial strain simply measures the level of “income adequacy” or perceived economic deprivation that can substantially complement what might have been missing in the more objective measures of SES. As being indicative of the lack of “adequate” income, financial strain is not only a primary stressor itself in both objective and subjective respects but a major source of other derivative chronic or acute stressful experiences (Angel et al. 2003).

There has been considerable evidence that financial strain, particularly that attributable to

job loss or the aging process, come to be part of a general sense of demoralization and therefore one of the risk factors for lower self-esteem, decreased self-mastery, and heightened distress (Angel et al. 2003; Armstrong and Schulman 1990; Blacksher 2002; Chou and Chi 2001; de Leon, Rapp, and Kasl 1994; Keith 1993; Kessler, Turner, and House 1987; 1988; Krause and Baker. 1992; Pearlin et al. 1981; Price, van Ryn, Vinokur 1992; Vinokur, Price, and Caplan 1996). For example, Kessler and colleagues (1987) found that compared to other hypothesized mediators, including marital conflict and loss of work relationship, financial strain alone accounted for 90% of the variance in mental health problems that had been explained by unemployment status. For unemployed samples in panel studies, financial strain was also found to significantly mediate between job loss at previous point in time and manifestation of depressive symptoms at later point in time (Price et al. 2002; Vinokur and Schul 1997). In another longitudinal study, Weich and Lewis (1998) further revealed that financial strain at baseline was strongly associated with both onset and maintenance of common mental disorders at follow-up , and the relationship tended to hold even net of more objective indices of standard of living, such as poverty and unemployment status.

At last, previous work has suggested that either there is an immediate association between financial strain and psychological distress, or financial strain is linked to

psychological distress by interfering with one's daily work or routines (Mills, Grasmick, Morgan, and Wenk 1992; Voydanoff 1990), by undermining the ability to fulfill specific role obligations (Ross and Huber 1985), by reducing supportive behaviors and eliciting undermining behaviors in couple relationships (Vinokur et al. 1996), and by lowering the sense of control over one's life (Keith 1993; Chou and Chi 2001; Krause and Baker 1992; Lincoln, Chatters, and Taylor 2003; Pearlin et al. 1981). Among the previously-investigated mediating mechanisms between financial strain and psychological distress, self-control or the sense of mastery has been studied most and has received substantial supporting evidence.

2.3.2 Psychological coping resources: The sense of mastery

SES not only shapes the experience of financial strain that can trigger or exacerbate a cascade of other secondary stressors, but it regulates access to various resources (e.g., material assets, coping styles, perceived control, and interpersonal support) that can be used to avoid or minimize the risk for disease and distress. Indeed, previous research has discovered that people of lower SES commonly have low self-esteem, self-mastery, and self-efficacy (Blacksher 2002; Lantz, House, Lepkowski, Williams, Mero, and Chen. 1998; Lynch, Kaplan, and Salonen 1997), all of which tend to be related to increasing distress (Mirowsky and Ross 1984; Thoits 1985; Wheaton 1980; 1983). Therefore, the objective

economic hardship of low SES is expected to have its greatest impact on distress only when the worse-off also subjectively perceive their impoverishment, which in turn deteriorate the preservation of psychosocial resources that are beneficial to mental health (Ensel and Lin 1991; Wheaton 1985). Compared to “being poor,” which is more relevant to worsening physical health (e.g., Lynch, Davey Smith, Kaplan, and House 2000), “feeling poor” lends itself more to the detriment of psychosocial self (e.g., self-esteem, self-efficacy, and self-mastery) and moral agency (i.e., the capacity for self-determination and crafting a life of one’s own), which is usually ominous for ensuing serious mental disruptions (Blacksher 2002).

The belief in being able to take control of one’s life has been expressed in a number of closely related terms, including locus of control (Lefcourt 1981; Rotter 1966; 1975), mastery (Pearlin and Schooler 1978), personal control (Gurin, Gurin, and Morrison 1978; Mirowsky and Ross 2003; Ross, Mirowsky, and Cockerham 1983), and instrumentalism (Mirowsky et al. 1996; Wheaton 1980; 1983). Aspects of the same belief have also been presented in negative labels, such as powerlessness (Seeman 1959), helplessness (Seligman 1975), hopelessness (Abramson et al. 1989), and fatalism (Ross et al. 1983; Wheaton 1980; 1983).

However put in words, these related constructs seem to agree in the following three ways: (1)

they imply the importance of “*perceived causal relevance* ... as a contingency in human development and functioning” (Turner and Roszell 1994: 181); (2) they deal only with the sense of one’s own *personal control*, thus having little to do with the belief about how much influence others have over their life, be it referred to as *universal control* (Mirowsky and Ross 2003) or *control ideology* (Gurin et al. 1978); (3) they represent a *global* judgment of personal control over one’s own life in virtually all respects rather than *specific* to action in certain realms (Mirowsky and Ross 2003). As suggested by Mirowsky and Ross (2003), consequently, all the constructs mentioned above can be regarded as alternative labels for the same personal resources in terms of human agency, except for Rotter’s (1966; 1975) locus-of-control, the scale of which includes statements referring to people in general besides oneself and to action in specific realms such as politics and school (e.g., “Most students don’t realize the extent to which their grades are determined by accidental happenings,” and “The average citizen can have an influence in government decisions”).

Among all the similar constructs described above, the sense of mastery has probably drawn the most attention and has remained in frequent use by many sociologists (Hobfoll et al. 2003; Mirowsky and Ross 2003; Turner and Lloyd 1999). Building on this tradition, this study adopts the classic mastery scale developed by Pearlin and Schooler (1978) and their

classic definition of mastery as “the extent to which one regards one’s life-chances as being under one’s own control in contrast to being fatalistically ruled (p5).”

There has been a rich body of evidence with respect to the mediating role of psychosocial resources as a linkage of stress to distress. Specifically, the sense of mastery is frequently hypothesized to change as a function of various stressors and to bear a direct impact on health outcomes in the more integrative stress process (Ensel and Lin 1991; Pearlin 1999b; Wheaton 1985). Evidence from past research suggests that the sense of mastery could intervene between psychological distress and different types of control-deterrents, including SES (Mirowsky and Ross 1984; 1989; Mirowsky et al. 1996; Turner and Lloyd 1999; Turner et al. 1999), life events (Maciejewski et al. 2000; Pearlin et al. 1981), chronic strain (Pearlin et al. 1981), minority status (Mirowsky and Ross 1984; Mirowsky et al. 1996), material loss (Hobfoll et al. 2003), demanding work condition (Link et al. 1993), negative aspects of interaction (Lincoln et al. 2003; Vinokur et al. 1996), and distress history (e.g., earlier depression) (Maciejewski et al. 2000; Turner and Noh 1988).

However, there has been a serious limitation involved in previous findings on the mediating role of mastery. Past studies were mostly cross-sectional by design and just assumed that, as prescribed by the stress process model or life stress paradigm, depression

was more reactive to mastery, rather than the other way around. Given that mastery and depression are both psychological constructs that can only be inferred without being directly observed, more evidence is needed to support one of the three competing hypotheses about the causal relationship between mastery and depression: (1) they are pretty much inversely-correlated as part of the general sense of demoralization and their relationship with each other is therefore causally reciprocal, (2) the causal flow does pass from mastery (personal resources) to depression (affective outcome), as suggested by the stress-distress paradigm, and (3) contrary to the dominant theory, mastery is in fact more reactive to depression than depression is reactive to mastery.

2.4 Dynamics in SES-Depression Relationship over Time

Little work has been done using longitudinal or panel design to investigate the extent to which the major components of the stress process change within an individual over time and how the changes in different components are interlocking with one another. While both the stress process model (Pearlin 1989; Pearlin and Schooler 1978; Pearlin et al. 1981; Pearlin et al. 1990) and life stress paradigm (Ensel and Lin 1991; Lin and Ensel 1989) have implied an intra-personal process where changes in stress, resources, and distress are hypothesized to be inter-correlated within the same individual, most previous research on testing relevant

hypotheses has been cross-sectional in nature, focusing instead on the between-person differences in stress, resources, and distress across the SES spectrum (e.g., Mirowsky et al. 1996; Turner and Lloyd 1999; Turner Lloyd, and Roszell 1999). Based on the cross-sectional design, intra-personal increases or decreases in the level of distress that are attributable to prior change in stress or resources within the same individual (e.g., increase/decrease in the sense of mastery followed by decrease/increase in the level of distress) can only be inferred from the between-individual differences in corresponding constructs (e.g., those with higher/lower sense of mastery reporting less/greater level of distress). As a result, stress research adopting cross-sectional design has long suffered from an apparent inconsistency between the *unit of actual analysis* (i.e., individual) and the *unit of what is inferred* (i.e., time) (Grzywacz et al. 2004).

Why is it so important to take time into account for stress research, then? Empirical evidence has shown that the levels or statuses of major components in the stress process are by no means static from one point in time to another. Considered separately, even the onset of depression as measured by diagnostic criteria is not so much constant as episodic (Kessler 2002). When defined along a continuum, as revealed by Lorenz and colleagues (1997; 2000; 2004b; 2006), depressive symptoms fluctuate over time in a more appreciable manner.

Financial strain, likewise, is seldom steadfast insofar as it can intensify or diminish, for example, as a direct function of employment status (Price et al. 2002; Vinokur and Schul 1997). However, one question that is often raised and remains unsettled concerns the extent to which personal resources, such as mastery, are trait-like and therefore temporally invariant.

Whether personality (in a broader sense) changes in adulthood has been hotly debated in development psychology (see Aldwin and Levenson 1994; Costa and McCrae 1997 for reviews). In their review of previous longitudinal studies, Aldwin and Levenson (1994) indicated that, first, personality measures are heterogeneous in terms of their volatility across time; second, while the average stability coefficient for the measure of *general personality* was only moderate (no greater than .6), the same coefficient for what they called *personality processes* (e.g., mastery and self-esteem) was even lower than that for temperament or trait measures such as the Big-Five *personality dimensions* (see John and Srivastava 1999 for the Big-Five taxonomy). Thus, one can at least expect that lower-order personality traits such as mastery are readily subject to change over time, even during one's adulthood (Aldwin, Sutton, and Lachman 1996; Gecas 1989). Evidence has shown that, for example, not only may the sense of mastery vacillate as people age (Gurin and Brim 1984; Mirowsky 1995; Ross and Mirowsky 2002; Woodward and Wallston 1987), unscheduled or off-time life events can also

lead to a significant decrement in mastery (Gurin and Brim 1984), one example of that being too early first birth (McLaughlin and Micklin 1983).

Even though it is now commonly believed that mastery, be it defined as a lower-order personality trait or personal coping resources, does change over time to an appreciable degree, a further question pertinent to stress research is yet to be answered: what are the major antecedent stressors and potential health consequences for the temporal variation in mastery? As suggested by Holahan and associates (1999: 620), “despite the central role of resources in contemporary theories of the stress and coping resources, researchers know little about the nature, consequences or determinants of resource change.”

Unfortunately, most previous stress studies were either cross-sectional by design or less concerned with changes in psychosocial resources as a central mechanism in their overall model (Hobfoll et al. 2003). While some noticeable exceptions had tried to estimate change in the sense of mastery based on a panel design (e.g., Maciejewski et al. 2000; Pearlin et al. 1981; Price et al. 2002; Turner and Noh 1988), they largely employed “autoregressive models” that assess change in terms of the “residualized scores” from a prediction equation whereby mastery measured at follow-up was regressed on its earlier counterpart measured at baseline (Bollen and Curran 2004; Kessler and Greenberg 1981). However, there has been a

great deal of criticism about the conceptual and statistical flaws with respect to the idea of change implied by autoregressive models (For reviews, see Lorenz et al. 2004a; Rogosa et al. 1982; Wickrama et al. 2000; Willett and Sayer 1994). In contrast, the latent growth curve (LGC) analysis is more appropriate for the purpose of measuring change in that, first, it takes into account the individual variation in absolute within-person change over time, rather than reflects only relative change and implies that the change pattern is common for everyone in the sample; second, LGC is also convenient in revealing how changes in one variable is related to changes in another insofar as it is possible to separate the chronic level (intercept) from the rate of change (slope) for any variable repeatedly measured at multiple points in time (Lorenz et al. 2004a; 2006; Wickrama et al. 2000). Chapter 3 provides more detailed comparisons between autoregressive model and LGC as well as more systematic expositions of the LGC framework.

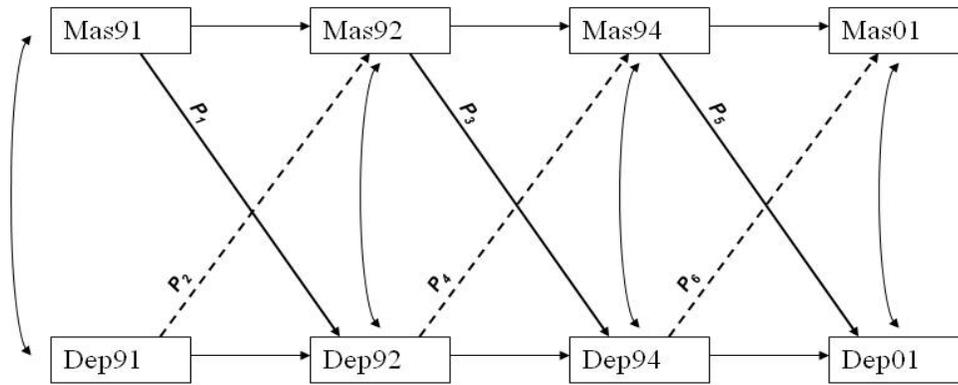
To the extent that substantial knowledge about resource change in the stress process can be greatly improved by sustained methodological advancement, analyzing panel data in the LGC framework allows us to take a more nuanced look at the process whereby relatively stable SES attributes lead to changes in distress through changes in financial strain and personal resources such as mastery. With SES as an ultimate origin of psychological

wellbeing, to be more specific, a distinction between chronic dimension (level) and acute dimension (rate of change) for the major components in the stress process may more clearly reveal the extent to which (1) chronic levels and acute changes associated with financial strain are conditioned by one's relatively durable socioeconomic rank in the macro society as reflected by the measure of chronic income; (2) chronic levels and acute changes associated with financial strain, mastery, and depressive symptoms reverberate with one another in terms of interlocking trajectories.

2.5 Research Hypotheses

The first set of analyses, as shown in Figure 2, is to fit a cross-lagged model with mastery and depressive symptoms each repeatedly measured at four points in time. The hypothesis tested by the cross-lagged model is that the causal flow does pass from mastery (personal resources) to depressive symptoms (affective outcome), as suggested by the stress process model or life stress paradigm. In other words, when estimated simultaneously, the cross-lagged paths from mastery to depressive symptoms (i.e., P_1 , P_3 , and P_5) are hypothesized to be significant, whereas the cross-lagged paths from depressive symptoms to mastery (i.e., P_2 , P_4 , and P_6) are not.

Referring back to Figure 1, the second set of analyses begin with a baseline model that



Note:

1. Mas91, Mas92, Mas94, and Mas01 are mastery scores measured in 1991, 1992, 1994, and 2001.
2. Dep91, Dep92, Dep94, and Dep01 are depressive symptoms measured in 1991, 1992, 1994, and 2001.
3. Solid lines are hypothesized to be significant paths, whereas dotted lines are hypothesized to be not significant

**Figure 2 Hypothesized 4-wave cross-lagged model
between mastery and depressive symptoms**

includes only chronic income and depressive symptoms in order to test whether and to what extent SES reflected by chronic income affects both initial level and subsequent rate of change associated with depressive symptoms. As indicated in Figure 1 by path P_1 , the direct effect of SES (chronic income) on two trajectory parameters (i.e., initial level and rate of change) of depressive symptoms is hypothesized to be significant in this baseline model.

Next, both initial level and rate of change associated with financial strain are included to evaluate the extent to which the contextualizing effect of SES (chronic income) on initial level and rate of change associated with depressive symptoms is mediated by its relatively

alterable cognitive component. It is hypothesized that the trajectory of financial strain is a complete mediator between SES (chronic income) and the trajectory of depressive symptoms such that when estimated simultaneously, P_2 (negative) and P_3 (positive) are significant whereas P_1 (originally negative) ceases to be.

When mastery is finally added in, the full model includes (1) the trajectory of financial strain as a potential complete mediator between SES (chronic income) and the trajectory of mastery (i.e., when estimated simultaneously, P_2 and P_4 are hypothesized to be significant and negative but P_5 is not), and in turn, (2) the trajectory of mastery as a potential complete mediator between the trajectory of financial strain and the trajectory of depressive symptoms (i.e., when estimated simultaneously, P_4 and P_6 are hypothesized to be significant and negative but P_3 is not).

For each of four models (i.e., one cross-lagged model and three LGC models) described above, a set of competing socio-demographic variables (i.e., age, education, and marital status) as measured in the beginning of the decade (i.e., 1991) are also included as time-invariant controls. It is noteworthy that marital status is controlled only for women but not for men because all male samples were married when first included in this study (see chapter 3 for more detailed description of the sample).

CHAPTER 3

METHODOLOGY

3.1 Sample

The sample used to test the research hypotheses includes both fathers and mothers from the Iowa Midlife Transitions Project (MTP: Lorenz et al. 2006), a decade-long panel study of 484 rural families with various socioeconomic backgrounds. Being drawn from a cluster of eight counties in north central Iowa that closely mimicked the economic diversity of the rural Midwest, these families were originally part of the Iowa Youth and Families Project (IYFP: Conger and Elder 1994) from 1989 to 1992 and the Iowan Single Parent Project (ISPP: Simons & Associates 1996) between 1991 and 1993. Beginning in 1989, the IYFP study recruited 451 families with the purpose of understanding the relationship between rural economic stress and well-being. With a particular interest in child development, the IYFP families included two biological parents of a target child who was in seventh grade in 1989 and had a sibling within 4 years of the target child's age. Two years later in the same study site, the ISPP study was initiated by recruiting 205 recently divorced mothers with two children. Within each ISPP household was there a mother who had permanently separated from her husband within last two years and was living with at least two of her biological children, one of whom was a ninth-grader in 1991 and the other a close-aged sibling. By

using this selection criterion, ISPP households included adolescents of comparable age in 1991 to those in IYFP households. Among the eligible families that were identified through contacts with public and private schools and then randomly selected into the IYFP, seventy-eight percent of the married couples agreed to participate. In contrast to IYFP, ninety-nine percent of the eligible single mothers agreed to participate in ISPP (Conger and Elder 1994; Simons & Associates 1996). As a combined sample, 415 IYFP families and 105 ISPP families remained in 1994 interview and were invited to continue participating in the MTP, for which only adult participants were interviewed in the early month of 2001. As a result, a four-wave (1991, 1992, 1994, and 2001) panel study was formed with 416 families (86% of the 484) that participated in the MTP across all four waves of data collection during the decade (Lorenz et al. 2006; Wickrama, Lorenz, Conger, Elder, Abraham, and Fang 2006). From this panel, 381 fathers and 468 mothers provided at least one wave of data during the study period from 1991 to 2001.

A notable feature of MTP is that the information was gathered from multiple informants. During each visit to participating families in their homes, trained field interviews asked each family member to independently complete a detailed questionnaire and kept confidential their respective responses to a series of questions about family life, work, finances, friends,

mental and psychological health status, and health behaviors. In addition to the self-report of each family member, other information were also collected from (1) trained observers who rated videotapes of family discussions in their homes, (2) teachers of the target children, and (3) school records of academic performance and achievement. This multi-informant measurement strategy not only helped reduce potential biases inherent in single-informant approaches but also provided an opportunity to assess the discrepancies in perspectives among family members (Conger and Conger 2002; Lorenz and Melby 1994).

The MTP sample was exclusively composed of the residents in rural Iowa, which is distinctive from most previous similar research that used either community samples located in urban areas, e.g., Epidemiologic Catchment Area (for review, see Robins and Regier 1991), or national-representative samples that contained a relatively small portion of rural residents, e.g., National Comorbidity Survey (for review, see Kessler, Zhao, Blazer, and Swartz 1997).

Among a limited number of studies to which this panel design can be compared, two complementary traditions have emerged in the epidemiological investigations of the rural-urban distinctiveness in mental health (Lorenz et al. 2004b). In the first tradition, random rural samples are deliberately drawn to estimate the independent contribution of place to the aggregate incidents and prevalence of mental disruptions, net of individual-level

attributes such as age, sex, race, and poverty. Overall, the evidence suggests that except for higher suicide rates among rural men, there is remarkable similarity between rural and non-rural areas in terms of types, levels, and rates of broadly-defined psychological distress and specific mental disorders. The second tradition has focused more narrowly on rural community samples. Generally guided by the “life stress paradigm” mentioned above, the second tradition explores the mental health consequences of socioeconomic status through mediating pathways of stressors and resources. It is of particular interest for this tradition to investigate the extent to which specific intervening mechanisms have been substantially altered in a health-devastating sense by historical contingencies such as plant closings in rural communities and the “farm crisis” of the late 1980s – i.e., the impact of “being in the wrong place during the wrong time (Lorenz et al. 2004b:79).”

As part of the efforts in the second tradition, the combined MTP families were sampled from a relatively homogeneous subpopulation of rural America in the wake of the financial “farm crisis” of the 1980s. Although many families were untouched by the farm crisis, some others experienced substantive loss of farm and agriculture-related jobs (Conger and Elder 1994; Lorenz et al. 2006). Designed as a rural epidemiological community, families participating in the MTP were monitored to understand whether and how differential

exposure to economic hardship had made the developmental trajectories divergent for each of the family members. Findings from the analyses of the MTP data have resulted in a large number of published articles (e.g., Lorenz et al. 2000; 2006; Wickrama et al. 2006).

While some doubts may be raised about the generalizability of the findings from the current study to metropolitan areas or the nation as a whole (a theme to which the discussion chapter will return), there is an obvious advantage of focusing on this particular sample of rural Iowa families with children, which had been differentially exposed to the adversities brought by the “farm crisis” during the decade of 1980s. According to Conger and Elder (1994), the “farm crisis” that can be regarded a valuable natural experiment had set in motion diversified financial trajectories that might reflect at least four economic scenarios throughout the 1980s: (1) those who had not yet recovered from a hard hit by the “farm crisis” during the early and mid-1980s; (2) those who had regained viable economic options after a period of financial difficulties; (3) those who had been able to maintain their financial wellbeing to a satisfactory level over the decade; (4) those who were already economically disadvantaged in the beginning of the decade and either maintained or improved from their initial status. The important point here is that, this panel of rural Iowa families with children provides an observation of variable financial trajectories and their psychosocial consequences

following a protracted length of economic crisis in the rural heartland.

Moreover, given that current sample was highly homogeneous in terms of other primary socio-demographic characteristics such as age, education attainment, and marital status (as shown in next section), it is also a great opportunity to test the robustness of inverse SES-depression association by examining to what extent the chronic level and acute rate of change associated with depressive symptoms are shaped by the fine-grained stratification of economic status through mediating pathways of change in financial strain and mastery, even with other individual attributes being roughly comparable. Recall that Whitehall Study of civil servants in England (Marmot, Shipley, and Rose 1984; Marmot, Smith, Stansfeld, Patel, North, Head, White, Brunner, and Feeney 1991) became classic for it provided impressive evidence that even in the population comprised by one-ethnic adults who were residing in close proximity to one other, stably employed in white-collar jobs, and limitedly exposed to industrial hazards, each higher level of employment grade was consistently associated with better health status (i.e., lower rates of disease and death).

3.2 Measures of Concepts

Table 2 presents the summary statistics for the major variables outlined in the conceptual framework (Figure 1). With list-wise deletion, 305 fathers out of 381 (80%) and 403 mothers

Table 2 Descriptive statistics for variables of interest by sex

		Fathers (N = 305)				
		Range	Min.	Max.	Mean	Std. Dev.
Gross per capita family income (\$1,000)						
	91	81.05	-39.25	41.80	9.09	7.40
	92	135.75	-77.75	58.00	9.16	10.37
	94	167.76	-36.01	131.75	11.73	10.95
	01	513.59	-17.14	496.45	34.47	39.80
Financial Strain						
	91	4.00	1.00	5.00	2.44	0.76
	92	4.00	1.00	5.00	2.56	0.80
	94	4.00	1.00	5.00	2.41	0.76
	01	3.71	1.00	4.71	2.26	0.64
Mastery						
	91	3.43	1.57	5.00	3.82	0.50
	92	3.00	2.00	5.00	3.77	0.53
	94	2.71	2.29	5.00	3.76	0.52
	01	3.43	1.57	5.00	3.81	0.53
Depression						
	91	3.77	1.00	4.77	1.30	0.43
	92	2.92	1.00	3.92	1.29	0.42
	94	2.62	1.00	3.62	1.36	0.44
	01	2.69	1.00	3.69	1.41	0.43
Education (91)		13.00	7.00	20.00	13.72	2.19
Age (91)		25.00	34.00	59.00	41.93	4.61

Table 2 (Continued)

		Mothers (N = 403)				
		Range	Min.	Max.	Mean	Std. Dev.
Gross per capita						
family income (\$1,000)						
	91	76.50	-21.00	55.50	8.48	6.96
	92	110.49	-52.49	58.00	8.63	8.60
	94	167.76	-36.01	131.75	11.41	10.06
	01	513.59	-17.14	496.45	31.63	35.49
Financial						
Strain						
	91	4.00	1.00	5.00	2.72	0.89
	92	4.00	1.00	5.00	2.71	0.89
	94	4.00	1.00	5.00	2.58	0.88
	01	4.00	1.00	5.00	2.29	0.76
Mastery						
	91	3.57	1.43	5.00	3.71	0.62
	92	3.71	1.29	5.00	3.76	0.62
	94	4.00	1.00	5.00	3.67	0.61
	01	3.29	1.71	5.00	3.81	0.60
Depression						
	91	3.38	1.00	4.38	1.55	0.59
	92	3.54	1.00	4.54	1.50	0.55
	94	3.15	1.00	4.15	1.59	0.57
	01	3.00	1.00	4.00	1.54	0.54
	Education (91)	10.00	9.00	19.00	13.56	1.70
	Age (91)	24.00	31.00	55.00	39.94	3.95
	Marital Status (91)	N	%			
	<i>married</i>	320	79.4			
	<i>divorced or separated</i>	83	20.6			

out of 468 (86%) remained for subsequent analyses without having missing values on each variable across the study period (i.e., 1991-2001).

For *financial strain*, respondents were asked to indicate, without reference to a specific time frame, the extent to which they approved each of 7 items on a scale from 1 (strongly agree) to 5 (strongly disagree). The items were, “my family has enough money to afford the kind of home we should have,” “we have enough money to afford the kind of clothing we should have,” “we have enough money to afford the kind of furniture or household equipment we should have,” “we have enough money to afford the kind of car we need,” “we have enough money to afford the kind of food we should have,” “we have enough money to afford the kind of medical care we should have,” and “my family has enough money to afford the kind of leisure and recreational activities we want to participate in.” A mean score was computed and assigned to those who responded to at least three items, even though nearly all respondents answered all of the items. As a consequence, the financial strain index had a range from 1 to 5 such that higher mean scores reflected a higher level of financial strain. As Table 2 indicates, the average level of financial strain reported by both fathers and mothers generally decreased across time, with a particularly noticeable drop between 1994 and 2001. This declining trend in average level of actual as well as perceived economic hardship could

have been attributed to a typical transition from young adulthood to mid-life (e.g., Schieman, Van Gundy, and Taylor 2001), on the one hand; it could have reflected a gradual recovery from the “farm crisis” back in 80’s for this particular sample, on the other. For the index of financial strain, additionally, fathers had the internal consistency estimates of reliability of .91 in 1991 (n = 327) and 2000 (n = 379), and .92 in 1992 (n = 318) and 1994 (n = 338). Mothers, on the other hand, had the alphas of .92 in 1991 (n = 424), 1992 (n = 424), 1994 (n = 431), and 2001 (n = 465).

Mastery was obtained from a Pearlin’s scale (Pearlin et al. 1981), which was designed to measure respondents’ sense of control. Specifically, each respondent was asked to indicate to what extent on a scale from 1 (strongly agree) to 5 (strongly disagree) he or she identifies with each of the seven Likert items as follows: (1) I have little control over the things that happen to me; (2) there is really no way I can solve some of the problems I have; (3) there is little I can do to change many of the important things in my life; (4) I often feel helpless in dealing with the problems of life; (5) sometimes I feel that I’m being pushed around in life; (6) what happens to me in the future mostly depends on me; (7) I can do just about anything I really set my mind to. To maintain as large effective sample size as possible, a mean score was computed and assigned to those who responded to at least three items so that the mastery

index presumably ranged from 1 to 5. Essentially, higher mean scores reflect a higher sense of control or mastery after responses to positively worded items were reverse-coded. As indicated in Table 2, the mean mastery scores reported by fathers by and large decreased across time, while the reverse is true of mothers. The index of mastery had internal consistency estimates of reliability of .79 in 1991 (n = 327), .81 in 1992 (n = 318) and 1994 (n = 337), and .77 in 2001 (n = 380) for fathers. For mothers, the alphas are .83 in 1991 (n = 424) and 1994 (n = 431), .85 in 1992 (n = 424), and .79 in 2001 (n = 468).

Depression was measured in terms of depressive symptoms using 12 items from the SCL-90-R (Derogatis 1983). After the preamble “During the past week, how much were you distressed or bothered by ...,” respondents were asked how much, along a five-point scale from “not at all” to “extremely,” they were bothered by symptoms of depression such as crying easily, feeling low in energy or slowed down, thoughts of ending your life, feeling of being trapped or caught, blaming themselves for things, feeling lonely, feeling blue, and feeling worthless, with scores on the 12 items ranging from 12 to 58. A mean score was computed and assigned to those who responded to at least six items, again, to increase the effective sample size. In so doing, the depression index presumably ranged from 1 to 5 with higher mean scores reflecting a severer depressive symptomatology. It can be observed from

Table 2, importantly, that fathers' average depressive symptoms steadily increased across time, achieving its peak in 2001. In contrast, mothers' pattern was less obvious with a generally increasing trend for the first three waves followed by significant drop in the last wave. In terms of the absolute level of depression, however, fathers reported significantly less average depressive symptoms than mothers in each of the four occasions. Finally, the depression index for fathers had the internal consistency estimates of reliability of .92 in 1991 (n = 327), .90 in 1992 (n = 318), and .91 in 1994 (n = 338), and .90 in 2000 (n = 379). Mothers, for the same scale, had the alphas of .91 in 1991 (n = 422), 1992 (n = 424), and .90 in 1994 (n = 428) and 2001 (n = 465).

Gross per capita family income was measured across 4 waves and used to reflect one's socioeconomic status at different time points. A significantly increasing trend in average gross per capita family income over the decade was easily noticed in Table 2 for both fathers and mothers. The chronicity of relative income level instead of its rate of change is of particular interest to this study because a significant variation is only observed in the initial level but not in the rate of change with respect to income for this particular sample, as will be shown in later analyses on the univariate growth curve of income. It is therefore hypothesized by this study that SES, represented by the chronic level of income, serves as the

ultimate exogenous variable that exerts a contextualizing effect on (1) the recognition or appraisal of both absolute and relative economic adversity, (2) the development of psychological resources, and (3) the occurrence or persistence of psychological distress. In order to remain the temporal precedence, the chronic level of income was measured by only the first three waves of data (i.e., 1991-1994).

Other important social demographic attributes served as *control variables* include age, education, and marital status. Table 2 shows that when the study began in 1991, fathers were all married, averaged 41.93 years of age, and had completed an average of 13.72 years of schooling. A closer examination indicates that in 1991, (1) roughly two-thirds of the fathers (66.9%) were between 40 to 50 years of age, (2) more than one-third of fathers (39.0%) had a high school diploma (exactly 12 years of schooling) as their highest degree, (3) almost half of the fathers (48.2) had highest degree of some college or a B.A. / B.S. diploma (13 to 16 years of schooling), and (4) about one-tenth (9.8%) of the fathers had more than 16 years of schooling (i.e., above Bachelor). On the other hand, 79.4 percent of mothers were married in 1991, with the remaining either divorced or separated. Averaging 39.94 years of age and 13.56 years of schooling in 1991, mothers on the whole were slightly younger and less educated than fathers in the beginning of the study. Further analyses show that, also in 1991,

(1) more than two-thirds of the mothers (68.5%) were between 39 to 49 years of age; (2) 34.5 percent of the mothers had the highest degree of high school graduate, (2) 60.8 percent of mothers had some college or a B.A. / B.S. degree, and (4) less than 4 percent (3.7%) was above Bachelor's degree. Overall, both fathers and mothers in this particular sample were highly homogeneous in terms of the primary socio-demographic variables considered here.

3.3 Analytic Strategy: Latent Growth Curve (LGC) Modeling

Panel studies make it possible for researchers not only to explore the causal structure between variables of interest but to examine how change in one variable is related to other time-invariant or time-varying variables. Generally speaking, most previous longitudinal stress research has employed “autoregressive models,” in which case structure equations were used to estimate the net effects of predictor variables on the final measure of the outcome after adjusting for the immediately prior level of the same outcome, and change was therefore defined by the “residualized scores” from the regression of the follow-up outcome on its baseline value. Specifically, autoregressive models can be expressed in terms of multiple regression as follows,

$$y_{it} = \beta_0 + \beta_1 x_i + \beta_2 y_{t-1,i} + \varepsilon_i \quad \varepsilon_i \sim \text{NID}(0, \sigma^2)$$

β_1 is the effect of predictor x on change in y , and β_2 is the *stability coefficient* or

autoregressive effect, which provides useful information about the relative predictive strength of the relation between outcomes over time (i.e., y_{t-1} and y_t). To include both *autoregressive effect (stability coefficient)* and *cross-lagged coefficient*, the multiple regression equations can be extended in the following way:

$$y_{it} = \beta_{0y} + \beta_{y_t x_{t-1}} x_{t-1,i} + \beta_{y_t y_{t-1}} y_{t-1,i} + \varepsilon_{iy} \quad \varepsilon_{iy} \sim \text{NID}(0, \sigma_y^2)$$

$$x_{it} = \beta_{0x} + \beta_{x_t x_{t-1}} x_{t-1,i} + \beta_{x_t y_{t-1}} y_{t-1,i} + \varepsilon_{ix} \quad \varepsilon_{ix} \sim \text{NID}(0, \sigma_x^2)$$

Again, $\beta_{x_t x_{t-1}}$ and $\beta_{y_t y_{t-1}}$ are the *autoregressive (or stability) parameters* for predictor x and outcome y , respectively. $\beta_{y_t x_{t-1}}$, on the other hand, is the *cross-lagged coefficient*, representing the longitudinal prediction of y_t from x_{t-1} above and beyond the autoregressive prediction from y_{t-1} . $\beta_{x_t y_{t-1}}$ can be interpreted in a similar way when the prediction of x_t is also of interest (Bollen and Curran 2004; Kessler and Greenberg 1981; Lorenz et al. 2004a; Wickrama et al. 2000).

Despite the noteworthy contribution to the broader stress-distress tradition in medical sociology or sociology of mental health and illness (e.g., Aneshensel, Frerichs, and Huba 1984; Ensel and Lin 1991; Lin and Ensel 1989), the autoregressive model has been criticized for its misrepresentation of change (Lorenz et al. 2004a; Wickrama et al. 2000). Conceptually, outcome variables in the autoregressive model are treated as a discrete “status” that can be

“caused” by an earlier status instead of a continuous “process” that may unfold over time. Moreover, change is regarded as common to all individuals in the sample to the extent that the autoregressive effects (or stability coefficients) and cross-lagged effects are shared by everyone. Determined by the same stability coefficients, the residualized score is therefore not unique to the individual in an explicit sense. Statistically, it is not likely for the autoregressive model to reveal the intricacies of non-linear trajectory. More importantly, the residualized scores can only reflect relative change, but they in fact say nothing about absolute change in the outcome variable. For example, if the rank order of the values on the outcome variable remains over time (i.e., each individual has occupied the same relative position in the distribution of the outcome variable across two points in time), then the stability arises even though there is a significant shift (either increase or decrease) in the mean of the outcome variable, which also makes it difficult for other predictors to have significant effects on the final measure of the outcome variable (Lorenz et al. 2004a; Wickrama et al. 2000).

By estimating the underlying time-related factors of growth and decline with the information on the means of observed indicators, latent growth curve (LGC) modeling has then been advocated as a better alternative to measure change due to (1) its greater capability

of portraying “individual time path,” i.e., individual growth or decline over time (Lorenz et al. 1997; 2000; 2004a; Rogosa 1980; Rogosa et al. 1982; Rogosa and Willett 1985; Willett and Sayer 1994), and (2) its readiness to be estimated with structural equation framework (Bollen 1989; Bollen and Curran 2004; Singer and Willett 2003; Wickrama, Lorenz, and Conger 1997).

The estimation of latent growth curve includes two steps. Initially, a regression equation (or a line segment) linking the outcome variable to a corresponding point in time is fit for each individual in the sample, with an intercept (π_{0i}) representing the initial level, a slope (π_{1i}) representing the rate of change over a fixed period of time (e.g., 1 year), and the error term representing within-subject error (ε_{it}). Thus,

$$y_{it} = \pi_{0i} + \pi_{1i} t + \varepsilon_{it} \quad \varepsilon_{it} \sim \text{NID}(0, \sigma_{it}^2)$$

represents that outcome variable y for the i^{th} individual either grows or declines linearly over time (t). Within the framework of structural equation modeling (George and Lynch 2003; Jöreskog and Sörbom 1996; Lorenz et al. 2004a; Wickrama et al. 1997; 2000; Willet and Sayer 1994), the initial level and rate of change are defined as two latent constructs, which are measured by the manifest variable of interest observed repeatedly at multiple time points. In the current case, the observed measurements of a variable at four points in time ($t_1, t_2, t_3,$

and t_4) for each person ($i = 1, 2, \dots, n$) can be represented by a vector (i.e., $[y_{i,t1}, y_{i,t2}, y_{i,t3}, y_{i,t4}]^T$), which can in turn be decomposed into a linear combination of two parameters of initial level and rate of change (i.e., $[\pi_{0i}, \pi_{1i}]^T$) and a vector of error terms for the four measurements (i.e., $[\varepsilon_{i,t1}, \varepsilon_{i,t2}, \varepsilon_{i,t3}, \varepsilon_{i,t4}]^T$):

$$\begin{bmatrix} y_{i,t1} \\ y_{i,t2} \\ y_{i,t3} \\ y_{i,t4} \end{bmatrix} = \begin{bmatrix} \lambda_{11} & \lambda_{12} \\ \lambda_{21} & \lambda_{22} \\ \lambda_{31} & \lambda_{32} \\ \lambda_{41} & \lambda_{42} \end{bmatrix} \begin{bmatrix} \pi_{0i} \\ \pi_{1i} \end{bmatrix} + \begin{bmatrix} \varepsilon_{i,t1} \\ \varepsilon_{i,t2} \\ \varepsilon_{i,t3} \\ \varepsilon_{i,t4} \end{bmatrix}$$

The common practice is to fix the factor loadings of the initial level on all four indicators (i.e., $\lambda_{11}, \lambda_{21}, \lambda_{31},$ and λ_{41}) to 1. Depending on the form of change to be tested (e.g., linear, quadratic, or other higher-order trajectories), the construct of rate of change is given appropriate loadings to the same set of indicators (i.e., $\lambda_{12}, \lambda_{22}, \lambda_{32},$ and λ_{42}) to reflect the distance from the baseline in terms of time unit (e.g., “year” in the current case). For the simplest case of a linear model, 0(1991), 1(1992), 3(1994), and 10(2001) are substituted into $\lambda_{12}, \lambda_{22}, \lambda_{32},$ and $\lambda_{42},$ respectively. When the time scores are specified in such a way that the slope factor loads nothing (i.e., 0) on the first-wave indicator of a specific variable, π_{0i} in fact reflects the earliest measurement or the initial level of such variable. Essentially, the time-specific errors (ε_{it}) are assumed to be normally distributed across n individuals with mean vector of 0 and a diagonal covariance matrix σ_ε (i.e., $\varepsilon_p \perp \varepsilon_q, \forall p \neq q$). The covariance

matrix σ_e could also incorporate correlated errors off the diagonal to reflect otherwise undetected correlations between measures at two points in time. However, adding such off-diagonal correlations should be cautioned against an apparent lack of theoretical and methodological justifications to do so.

In so far as the individual trajectories indexed by two growth parameters (i.e., initial level and rate of change) differ from one another in a sample of n individuals, there are potentially n different intercepts and slopes. In essence, π_{0i} and π_{1i} are assumed to have a multi-normal distribution with a mean vector of $[\mu_{\pi_0}, \mu_{\pi_1}]^T$ and a covariance matrix Σ , which summarizes the variability around the average trajectory characterized by the mean vector across the n individual trajectories. One can then aggregate the n line segments across the whole sample to obtain an average intercept (mean of π_{0i}), an average slope (mean of π_{1i}), and estimated variances for both the intercept (σ_{π_0}) and the slope (σ_{π_1}). Specifically, the mean of the intercept and that of the slope describe, respectively, the overall average of the individual-specific levels and changes of an attribute over time; the intercept variance and slope variance reflect the dispersion from the overall average in the corresponding growth parameter across individuals.

Significant variances in the growth parameters are required, both statistically and

conceptually, for the LGC to further proceed to predict the heterogeneous growth trajectories in the population. If the individual-specific intercepts and slopes are so alike in the population, conceivably, one needs nothing but the overall average to make a good enough prediction. In the second step of the LGC, one can seek to explain the significant variation in the initial level and rate of change by regressing each individual's intercept (π_{0i}) and slope (π_{1i}) against theoretically important covariates, either time-invariant (x_{1i}) or time-varying (x_{2it}) as follows:

$$\begin{aligned}\pi_{0i} &= \gamma_0 + \gamma_{01} x_{1i} + \gamma_{02} x_{2it} + \delta_{\pi_{0i}} & \delta_{\pi_{0i}} &\sim \text{NID}(0, \sigma_{\pi_0}^2) \\ \pi_{1i} &= \gamma_1 + \gamma_{11} x_{1i} + \gamma_{12} x_{2it} + \delta_{\pi_{1i}} & \delta_{\pi_{1i}} &\sim \text{NID}(0, \sigma_{\pi_1}^2)\end{aligned}$$

where γ_0 and γ_1 are adjusted means or the intercepts of the prediction equations for initial level and rate of change, respectively. While γ_{01} and γ_{11} are path coefficients linking growth parameters to time-invariant predictor variables (x_{1i}), γ_{02} and γ_{12} are those linking growth parameters to time-varying predictor variables (x_{2it}). Finally, still assumed to have a mean vector of 0 and a covariance matrix Ψ , the error vector $[\delta_{\pi_{0i}}, \delta_{\pi_{1i}}]^T$ represents disturbances or unexplained variances between individuals. For time-varying predictor, notably, the SEM framework makes it possible to use growth parameters of the predictor variable to predict those of the outcome variable. The paths or correlations between growth

parameters associated with two different variables are usually referred to as “inter-locking” trajectory. Technically, it is also possible to use the growth parameters of time-varying predictor to predict a time-invariant outcome in the same framework. However, a sheer statistical consideration (e.g., improving model fit) apparently does not warrant such a practice, unless it is backed up by a well-founded hypothesis.

CHAPTER 4

RESULTS

The analyses begin from estimating two cross-lagged models, as specified by Figure 2, one for fathers and the other for mothers. The purpose of fitting the cross-lagged mode is to reveal, after controlling for socio-economic and social-demographic characteristics, the causal structure between personal resources as reflected in the sense of mastery and distress as represented by depressive symptoms. The second step is to estimate the univariate growth curves that separate absolute levels (intercept) from rates of change (slope) with respect to *gross per capita family income (PCI)*, *financial strain (FS)*, *sense of mastery (MAS)*, and *depressive symptoms (DEP)*, respectively, as illustrated by the overlapping ellipses in Figure 1. Finally, the conceptual model in Figure 1 is tested in a progressive manner. That is, a series of models are estimated to examine to what extent the dynamic relationship between PCI and DEP was mediated by FS and MAS in terms of the “interlocking trajectories” (i.e., significant correlations between growth parameters associated with relevant variables) for this particular sample from rural Iowa.

In view of previous findings on sex differences in depressive responses to stress (Kessler 2003; Kessler and McLeod 1984; Mirowsky and Ross 1995; Nolen-Hoeksema, Larson, and Grayson 1999; Rieker and Bird 2000; Rosenfield 1989, 1992; Turner and Avison 1989; Wu

and DeMaris 1996), all analyses are conducted for fathers and mothers separately to examine whether and how the hypothesized mediation mechanisms might be different depending on the social roles assumed by adults of different sexes in a family with adolescent children. It is noteworthy that a multiple-group approach to testing the equivalence of a measurement, path, or full latent-variable models across subjects categorized on a specific ground (Jöreskog and Sörbom 1996) may not be appropriate for the current study because IYFP fathers and IYFP mothers were dependent to the extent that each couple came from the same family.

All model parameters are estimated by the matrices of covariance and the method of maximum likelihood using LISREL (Jöreskog and Sörbom 1996). Along with parameter estimates, commonly-used goodness-of-fit indices such as chi-square with degrees of freedom, Root Mean Square Error of Approximation (RMSEA), Standardized Root Mean Square Residual (SRMR), CFI (Comparative Fit Index), Goodness of Fit Index (GFI), and Adjusted Goodness of Fit Index (AGFI) are also presented, since it usually takes the combination of at least two of them to achieve a reasonable as well as adequate model evaluation. For example, Hu and Bentler (1999) have recommended a number of “combinational rules” for determining whether a model fit is acceptable, one of which is to use a cutoff value close to .95 for CFI coupled with a cutoff value close to .09 for SRMR.

4.1 Attrition Analyses

Due to concerns that sample attrition may introduce biases to the result, a comparison was conducted between those who provided complete data across 4 waves throughout the decade ($n = 305$ for fathers and $n = 403$ for mothers) and those who were excluded from the analyses because they dropped out of the panel at certain point in time during the study period ($n = 76$ for fathers and $n = 65$ for mothers). With respect to fathers, overall, those who were excluded from further analyses were somewhat older (43.06 vs. 41.93) and less well educated (12.89 vs. 13.72 years of schooling) in 1991, but the differences failed to reach statistical significance. However, fathers who dropped out of the panel did report significantly lower PCI in 1991 (6.09 vs. 9.09, $p = .003$, unit: thousand dollars) and significantly higher DEP in 2001 (1.56 vs. 1.41, $p = .01$). For mothers, on average, those who did not provide complete data across four occasions were slightly younger (38.40 vs. 39.94, $p = .095$), less well educated (12.90 vs. 13.56 years of schooling, $p = .094$), and more likely to be divorced or separated in 1991 (82% vs. 21%). Besides, the excluded mothers had significantly lower PCI in 1991 (3.79 vs. 8.48, $p < .001$, unit: thousand dollars) and 1992 (4.53 vs. 8.63, $p < .01$, unit: thousand dollars), and they also reported slightly higher FS in 2001 (2.48 vs. 2.29, $p = .071$). For both fathers and mothers alike, in general, differences in

FS, MAS, and DEP throughout the decade were negligible between the analyzed sample and the excluded sample, whereas the average PCI for the latter was lower to a noticeable degree at the beginning of the decade.

4.2 Causal Sequence between Mastery and Depressive Symptoms

A pair of four-wave, two-variable cross-lagged autoregressive modes was first fit to the data for mothers (Figure 3) and fathers (Figure 4), respectively, where both MAS and DEP were measured at four points in time. Both models were also adjusted for chronic PCI (1991-1994) and baseline (i.e., in 1991) age, education, and marital status (for mothers only).

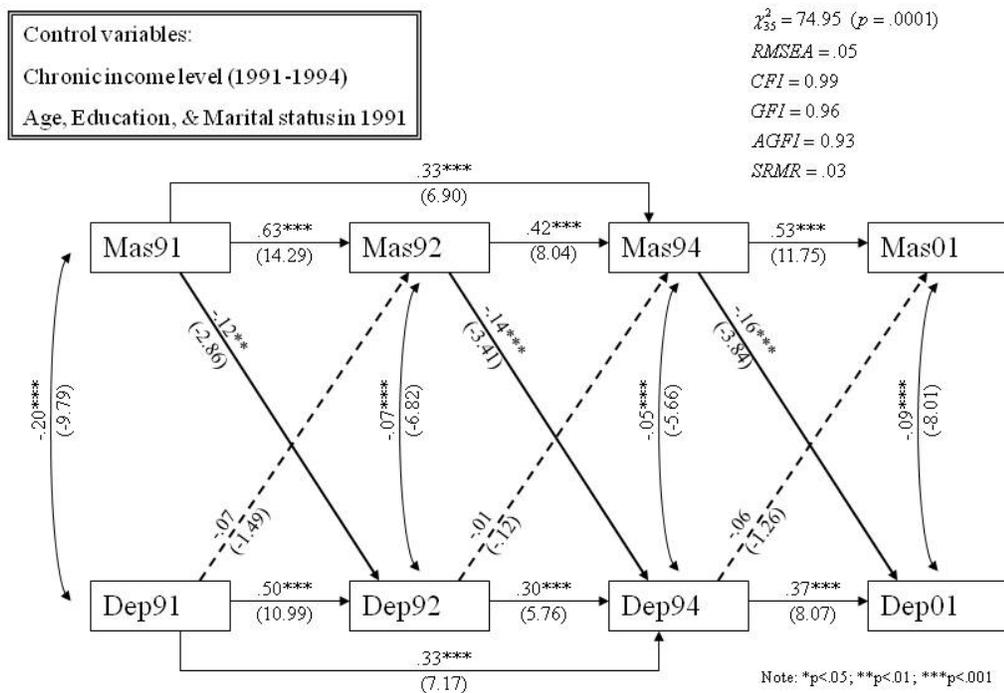


Figure 3 Four-wave cross-lagged model between mastery and depression for mothers (unstandardized coefficients with t-values in the parentheses)

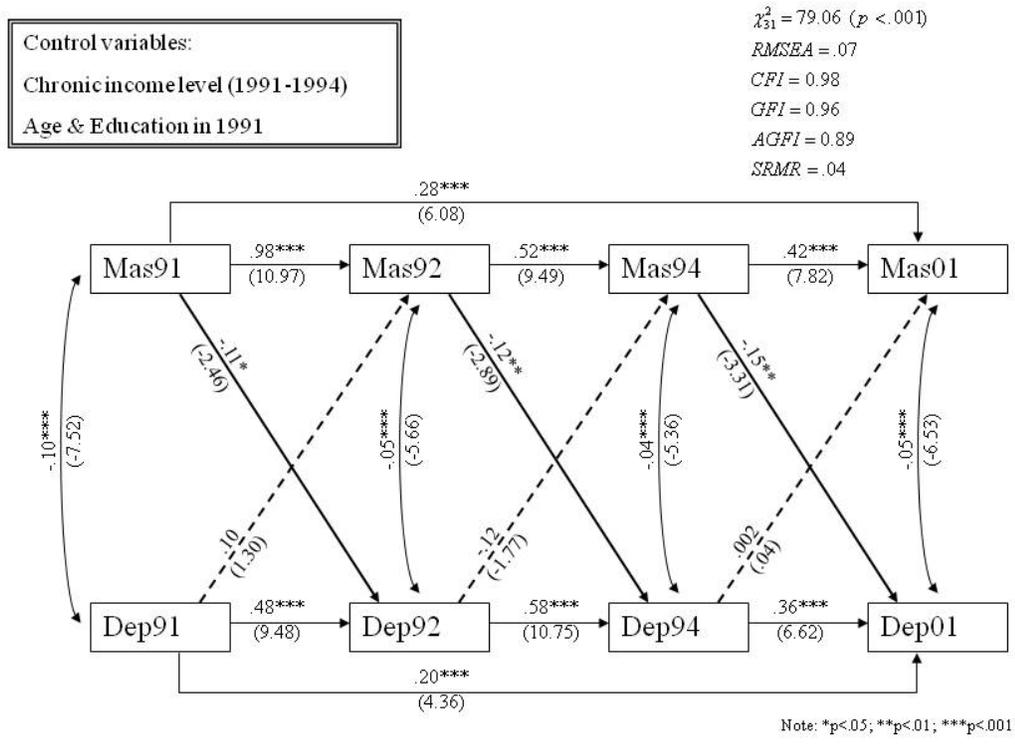


Figure 4 Four-wave cross-lagged model between mastery and depression for fathers (unstandardized coefficients with t-values in the parentheses)

Essentially, the coefficients linking a specific variable at one point in time to itself at the next point in time denote the stability effect. Within each sex group, all of the stability coefficients for both MAS and DEP were positive and large in magnitude with high statistical significance, meaning that in the population, rank order (i.e., relative levels) of both MAS and DEP largely remained over the decade. In other words, people with relatively high levels of MAS/DEP at one point in time tended to occupy roughly the same relative position in the distributions of MAS/DEP at next point in time; alternatively, people with relatively low levels of MAS/DEP at baseline were more likely to stay low in the follow-up distributions of

MAS/DEP. Still across sex groups, stability effects estimated from each of the three first-order intervals (i.e., 91-92, 92-94, and 94-01) were generally higher for MAS than those for DEP, according to the standardized coefficients (not shown in the figures: .64 vs. .53 between 91 and 92, .42 vs. .29 between 92 and 94, .54 vs. .39 between 94 and 01 for mothers; .64 vs. .53 between 91 and 92, .43 vs. .30 between 92 and 94, .84 vs. .39 between 94 and 01 for fathers).

With the stability effect being simultaneously estimated, a path from one variable, say x , at one point in time (e.g., t_1) to the other, say y , at the next point in time (e.g., t_2) denotes the effect of x at t_1 on subsequent change in y at t_2 insofar as the prior level of y (i.e., y at t_1) has been accounted for (Kessler and Greenberg 1981). In regard to mothers, the result shows that over the decade, there was a significant and negative association between MAS at one point in time and DEP at next point in time (unstandardized coefficient: -.12, $t = -2.86$ between 91 and 92; -.14, $t = -3.41$ between 92 and 94; -.16, $t = -3.84$ between 94 and 01), but DEP at one point in time was not related to MAS at next point in time in a significant manner (unstandardized coefficient: -.07, $t = -1.49$ between 91 and 92; -.01, $t = -.12$ between 92 and 94; -.06, $t = -1.26$ between 94 and 01). For fathers, the story was basically the same: on the one hand, earlier MAS was negatively and significantly linked to later DEP in each of the

three first-order intervals; on the other, the associations between earlier DEP and later MAS from three first-order intervals were inconsistent in their signs and all failed to achieve statistical significance (unstandardized coefficients: $-.11, t = -2.46$ vs. $.10, t = 1.30$ between 91 and 92; $-.12, t = -2.89$ vs. $-.12, t = -1.77$ between 92 and 94; $-.15, t = -3.31$ vs. $.002, t = .04$ between 94 and 01).

As hypothesized by Figure 2, to summarize, earlier levels of MAS were significantly predictive of later change in DEP (i.e., $P_1, P_3,$ and P_5 in Figure 2), but there was little evidence for the causal sequence that proceeds from DEP to MAS (i.e., $P_2, P_4,$ and P_6 in Figure 2). Moreover, the cross-lagged effects of MAS on DEP remained significant even after controlling for important socio-economic and socio-demographic variables. Finally, the causal primacy of MAS over DEP held for both mothers and fathers, in spite of the well-documented sex differences in depressive responses to stressors related to societal arrangements.

According to the LISREL automatic modification indices (MIs) (or otherwise called Lagrange multiplier statistics), there would be a substantial improvement in model fit with some additional parameters to be estimated for each sex group. For mothers, on the one hand, MIs suggest that initial levels (in 1991) of MAS and DEP remain to have significantly

positive effects on their respective counterparts measured three years later (in 1994), as shown in Figure 3. For fathers, on the other, MIs suggest that initial levels (in 1991) of MAS and DEP exert even longer-lasting impacts on their respective counterparts measured at the end of the decade (in 2001), as shown in Figure 4. Although there has been a persistent caution against post-hoc modification without a good starting model and rigorous theoretical grounds (e.g., MacCallum 1986, 1995; MacCallum, Roznowski, and Necowitz 1992), it seems legitimate, or at least innocuous, in the current case to estimate additional parameters as described above, even though they were not specified a priori. In essence, doing so not only enhances our understanding of the changing nature of relevant variables, but, more importantly, it makes little difference in concluding the ascendance of MSA over DEP in terms of causal order.

As mentioned before, both cross-lagged models have been adjusted for important socioeconomic and socio-demographic variables, including chronic PCI, marital status (only for mothers), age, and education. Although they were included only as exogenous controls, their correlations with one another and their independent effects on each wave of MAS as well as DEP could also add to our understanding of potential correlates of causal agency (i.e., sense of control or mastery) and psychological distress (i.e., depressive symptoms).

Table 3 shows that, for mothers, chronic PCI was correlated positively with age and education but negatively with marital status (0 = married; 1 = divorced/separated). In other words, mothers who were older, better educated, and married in 1991 tended to have perennially higher income. Chronic PCI also had a significantly positive association with, and only with, initial levels of MAS in 1991, as indicated in Table 4, whereas age and education were not associated with either MAS or DEP at each of the four waves to a discernible extent. Among the few significant correlations between controls and the two psychological measures (i.e., MAS and DEP) across the four waves, it is in particular noteworthy that marital status (0 = married; 1 = divorced/separated) in 1991 was positively linked to 1st-wave DEP and 2nd-wave MAS. On the one hand, it has been well-documented that depression in the sense of psychological distress is volatile to the extent that increases in depressive symptoms due to the termination of marriage are usually followed by rapid declines immediately after divorce or separation (e.g., Lorenz et al. 1997; 2006). On the other, usually described as “resource mobilization” in previous stress literature (e.g., Ensel and Lin 1991; Wheaton 1985), the phenomenon that women’s causal agency (i.e., sense of mastery or control) or other relevant self-concepts improve rather than deteriorate in the wake of divorce or separation has also received empirical support to a certain degree (for review, see Baum,

Rahav, and Sharon 2005).

Table 3 Completely standardized maximum-likelihood estimates (with t-ratios) of covariances between control variables for mothers

	Chronic income	Marital status	Age	Education
Chronic income	-			
Marital status	-.26*** (-4.31)	-		
Age	.25*** (4.23)	-.11* (-2.16)	-	
Education	.30*** (4.96)	-.09 (-1.70)	.34*** (6.47)	-

Note: *p<.05; **p<.01; ***p<.001

Table 4 Unstandardized maximum-likelihood estimates (with t-ratios) of the path coefficients linking control variables to 4 waves of mastery and depressive symptoms for mothers

	Chronic income	Marital status	Age	Education
Mas91	.02* (2.42)	-.15 (-1.84)	-.02* (-2.30)	.003 (.13)
Mas92	.01 (1.47)	.21*** (3.63)	-.01 (-1.31)	.01 (.41)
Mas94	-.003 (-.52)	.03 (.59)	-.003 (-.45)	.01 (.40)
Mas01	.01 (1.94)	.06 (1.04)	-.01 (-1.06)	.01 (.56)
Dep91	-.01 (-1.80)	.26*** (3.50)	-.003 (-.41)	.002 (.12)
Dep92	-.01 (-1.16)	.01 (.19)	-.003 (-.53)	.01 (.97)
Dep94	-.004 (-.76)	-.05 (-.87)	-.004 (-.70)	.03* (2.10)
Dep01	-.01 (-1.94)	.03 (.59)	-.01 (-1.63)	-.005 (-.32)

Note: *p<.05; **p<.01; ***p<.001

As far as fathers are concerned, Table 5 shows that chronic PCI was positively related to education in a significant manner. However, correlations of age with chronic PCI as well as

with education were not significant for these mid-aged rural fathers. Finally, there were few noticeable additive effects of chronic PCI, age, and education on any wave of MAS and DEP over the decade, as indicated in Table 6.

Table 5 Completely standardized maximum-likelihood estimates (with t-ratios) of covariances between control variables for fathers

	Chronic income	Age	Education
Chronic income	-		
Age	.06 (.99)	-	
Education	.27*** (4.07)	.11 (1.93)	-

Note: *p<.05; **p<.01; ***p<.001

Table 6 Unstandardized maximum-likelihood estimates (with t-ratios) of the path coefficients linking control variables to 4 waves of mastery and depressive symptoms for fathers

	Chronic income	Age	Education
Mas91	.01 (1.90)	.001 (.17)	.006 (.46)
Mas92	.01* (2.24)	-.01* (-2.25)	.01 (1.13)
Mas94	.005 (1.03)	.000 (.11)	.01 (1.18)
Mas01	.002 (.49)	-.01 (-1.36)	.02* (2.04)
Dep91	-.01* (-2.12)	.002 (.33)	.02 (1.51)
Dep92	-.002 (-.63)	.008 (1.91)	.008 (.87)
Dep94	-.002 (-.42)	.000 (.05)	-.01 (-1.43)
Dep01	-.000 (-.01)	.003 (.64)	-.004 (-.42)

Note: *p<.05; **p<.01; ***p<.001

Overall, the negligible relationships between baseline controls and the two psychological constructs (i.e., MAS and DEP) measured across the four waves suggest two important points that have been argued throughout this study from both methodological and theoretical perspectives. First, the idea of change implied by autoregressive models is flawed insofar as stability coefficients tend to increase in magnitude when rank order remains over time, which makes it difficult for other predictors to have independent effects on the follow-up measure of the outcome variable with its prior value adjusted for (Lorenz et al. 2004a; Wickrama et al. 2000). Second, if there is any substantial effect of objective socioeconomic indicators and socio-demographic attributes on psychological distress, it is most likely indirect and can be better understood by including relevant psychosocial risk factors or/and coping resources as potential mediators (Eaton et al. 2001).

4.3 Univariate Growth Curves of Major Variables

Results from fitting linear growth curves to the four waves (1991, 1992, 1994, and 2001) of (1) PCI, (2) FS, (3) MAS, and (4) DEP are summarized in Table 7 and 8 for mothers and fathers, respectively, and the unstandardized coefficients are estimated by LISREL, using matrices of covariances and methods of maximum likelihood.

As shown in Table 7, PCI for mothers was estimated to be a linear function of time ($t = 0$,

1, 3, and 10) based on the following fitted equation with t-values in parentheses and

chi-square test statistic to the right:

$$PCI_t = 8.30 + .99t, \chi^2_{(4)} = 7.58$$

$$(24.48) \quad (6.64)$$

Table 7 Univariate growth curves of gross per capita income (\$1,000), financial strain, mastery, and depressive symptoms for mothers (N = 403)

Gross per Capita Income	$PCI_t = 8.30 + .99t$ (t = 24.48) (t = 6.64)	t = 0, 1, 3, 10 $\chi^2_{(4)} = 7.58$ (p = .11)	$\hat{\sigma}_L^2 = 31.43$ (t = 7.79) $\hat{\sigma}_S^2 = 1.05$ (t = 1.61)
Financial Strain	$FS_t = 2.73 - .04t$ (t = 63.54) (t = -12.37)	t = 0, 1, 3, 10 $\chi^2_{(4)} = 3.83$ (p = .43)	$\hat{\sigma}_L^2 = .69$ (t = 12.98) $\hat{\sigma}_S^2 = .003$ (t = 3.28)
Mastery	$MAS_t = 3.73 + .008t$ (t = 128.88) (t = 2.93)	t = 0, 1, 3, 10 $\chi^2_{(4)} = 3.58$ (p = .47)	$\hat{\sigma}_L^2 = .27$ (t = 11.54) $\hat{\sigma}_S^2 = .001$ (t = 2.89)
Depression	$DEP_t = 1.57 - .003t$ (t = 56.83) (t = -.86)	t = 0, 1, 3, 10 $\chi^2_{(4)} = 7.47$ (p = .11)	$\hat{\sigma}_L^2 = .22$ (t = 10.98) $\hat{\sigma}_S^2 = .002$ (t = 3.25)

Note: t-ratios in parentheses; $\hat{\sigma}_L^2$: estimated level variance; $\hat{\sigma}_S^2$: estimated slope variance.

*p<.05; **p<.01; ***p<.001

The intercept was arbitrarily set to reflect the “initial level,” i.e., the overall mean of PCI at first wave (1991). With different set-up for the loadings of the indicators measured at four points in time on the slope factor, to be sure, the intercept could be otherwise set to reflect the “2nd-wave level,” “3rd-wave level,” or the “final level” (see previous chapter for details). In the current case, the intercept of 8.30 for mothers in the equation is the estimated average

PCI reported by all mothers in 1991 (i.e., mean of π_{0i}), which is close to the observed average of 8.48 in Table 2. Not surprisingly, the average initial level of PCI was significantly different from 0 ($t = 24.48$). It is even more informative that the individual initial level also varied significantly from one mother to another, which is revealed by the significant variance of π_{0i} ($\hat{\sigma}_L^2 = 31.43$, $t = 7.79$), indicating that mothers entered the decade with substantial differences in their starting points of PCI. This finding demonstrates that even for this highly homogeneous sample of rural mothers that had relatively comparable demographic profile, there could still exist clear-cut social hierarchies among them defined by objective measures of income.

The average slope of 0.99 (mean of π_{1i} , $t = 6.64$) attests that PCI increased significantly between 1991 and 2001 for mothers as a whole (i.e., the shift in the overall mean or the amount of absolute increase was significantly different from 0). However, the estimated variance in rates of change was not significantly different from 0 (i.e., variance of π_{1i} , $\hat{\sigma}_S^2 = 1.05$, $t = 1.61$), meaning that almost every mother experienced increases of similar rate in PCI between 1991 and 2001. Taken together, the absolute differences in PCI for mothers revealed at first wave (1991) remained largely intact (i.e., the gaps neither converged nor diverged) over the study period.

In general, the overall 10-year PCI trajectory of fathers greatly resembles that of mothers (see Table 8), except that fathers had somewhat higher average initial level (9.04 vs. 8.30) but slightly lower average rate of increase (.91 vs. .99)³. In view of the lack of significant between-individual variation in the rates of change associated with PCI for both mothers ($\hat{\sigma}_S^2 = 1.05$, $t = 1.61$) and fathers ($\hat{\sigma}_S^2 = 1.64$, $t = 1.87$), only the chronic level of PCI across the first three waves (1991-1994) would be estimated in subsequent analyses.

Table 8 Univariate growth curves of gross per capita income (\$1,000), financial strain, mastery, and depressive symptoms for fathers (N = 305)

Gross per Capita Income	PCI _t = 9.04 + .91 _t (t = 21.33) (t = 5.38)	t = 0, 1, 3, 10 $\chi^2_{(4)} = 7.26$ (p = .12)	$\hat{\sigma}_L^2 = 49.03$ (t = 8.15) $\hat{\sigma}_S^2 = 1.64$ (t = 1.87)
Financial Strain	FS _t = 2.59 - .03 _t (t = 53.32) (t = -7.45)	t = 0, 1, 3, 10 $\chi^2_{(3)} = 1.43$ (p = .70)	$\hat{\sigma}_L^2 = .55$ (t = 11.01) $\hat{\sigma}_S^2 = .003$ (t = 3.70)
Mastery	MAS _t = 3.79 + .001 _t (t = 144.27) (t = .40)	t = 0, 1, 3, 10 $\chi^2_{(4)} = 9.46$ (p = .05)	$\hat{\sigma}_L^2 = .17$ (t = 10.01) $\hat{\sigma}_S^2 = .003$ (t = 3.92)
Depression	DEP _t = 1.30 + .01 _t (t = 59.94) (t = 4.58)	t = 0, 1, 3, 10 $\chi^2_{(4)} = 8.06$ (p = .09)	$\hat{\sigma}_L^2 = .10$ (t = 8.47) $\hat{\sigma}_S^2 = .001$ (t = 3.13)

Note: t-ratios in parentheses; $\hat{\sigma}_L^2$: estimated level variance; $\hat{\sigma}_S^2$: estimated slope variance.

*p<.05; **p<.01; ***p<.001

³ Since IYFP couples shared exactly the same amount of gross per capita family income, the estimated difference in the univariate growth curve of PCI between fathers and mothers was due to measurement error, some missing data (e.g., a husband of a participating women did not participate), and for the most part the mixture of married and single mothers in the sample of mothers.

Last but not least, a model-building process is not complete until the model fit to the data is explicitly evaluated. Specifically, the linear univariate growth models of PCI for both mothers and fathers fit the data adequately, as indicated by the small chi-square statistics relative to their respective degrees of freedom (for mothers: $\chi^2_{(4)} = 7.58$; for fathers: $\chi^2_{(4)} = 7.26$), and no significant improvement is observed by fitting other higher-order functional forms such as the quadratic or cubic (result not shown).

The results of other univariate growth curves with respect to FS, MAS, and DEP shown in Table 7 and 8 can be interpreted in the same fashion as demonstrated above. By and large, the linear growth curves specifying monotonic change with constant rate over time perform well in depicting the trajectories of these three social-psychological variables across sex groups and therefore would be employed as the base for further analyses. Notably, other than PCI for which there was no significant between-individual variation in the rates of change, in light of Table 7 and 8, the estimated variances of the slope factors for FS, MAS, and DEP, respectively, were all significant for both mothers and fathers ($ps \leq 0.1$). Specifically, for mothers, the estimated slope variance was .003 ($t = 3.28$) for FS, .001 ($t = 2.89$) for MAS, and .002 ($t = 3.25$) for DEP; for fathers, the estimated slope variance was .003 ($t = 3.70$) for FS, .003 ($t = 3.92$) for MAS, and .001 ($t = 3.13$) for DEP. Of particular importance, a

significant variance in the rates of change shows that individual growth trajectories over time were substantially heterogeneous in at least two ways. First, some individuals experienced increases in FS, MAS, and DEP, whereas others experienced declines in the corresponding measures. For those who experienced change in the same direction, moreover, some experienced more dramatic change than others (e.g., moderate increase vs. drastic increase within the increase group, or moderate decrease vs. drastic decrease within the decrease group). In other words, for the sample as a whole, some were above and others were below the average rate of change to a significantly varying degree. Given the significant variations in rates of change, the slope factors associated with FS, MAS and DEP would remain to be estimated in later analyses as either latent predictor variables or latent response variables to be predicted.

To conclude this section, some notable sex differences are summarized in terms of the linear univariate growth curves for the four variables of major interest. First, while fathers had higher average initial levels of PCI (9.04 vs. 8.30) and MAS (3.79 vs. 3.73) as compared to mothers, the mean levels of FS and DEP at baseline were higher for mothers than for fathers (2.73 vs. 2.59; 1.57 vs. 1.30). Taken together, this corresponds to previous cross-sectional findings that females are more financially disadvantaged and

depression-prone (Nolen-Hoeksema 1990, 2001; Nolen-Hoeksema et al. 1999; Rieker and Bird 2000; Rosenfield 1989, 1992; Wu and DeMaris 1996). Second, mothers as a whole experienced significant increases in PCI (.99, $t = 6.64$) as well as MAS (.008, $t = 2.93$).

While their average slopes associated with FS as well as DEP were negative, only the former was not significantly different from zero in the population ($-.04$, $t = -12.37$; $-.003$, $t = -.86$).

Despite resembling mothers in terms of significantly positive average slope associated with PCI (.91, $t = 5.38$) and significantly negative average slope associated with FS ($-.03$, $t = -7.45$), fathers in general experienced significant increases in DEP (.01, $t = 4.58$) and, to a minimal extent, in MAS (.001, $t = .40$).

4.4 Direct Effect of SES on Trajectories of Depressive Symptoms

Figure 5 and 6 were two models that included chronic PCI between 1991 and 1994 as a predictor of both growth parameters (i.e., initial level and rate of change) of DEP from 1991 to 2001, for mothers and fathers, respectively. In subsequent analyses, again, all models of mothers were adjusted for marital status, age, and education, while those of fathers included only age and education as controls

With reference to Figure 1, the baseline model that includes only SES as indexed by chronic PCI and health outcome as represented by DEP tests the hypothesis that there is a

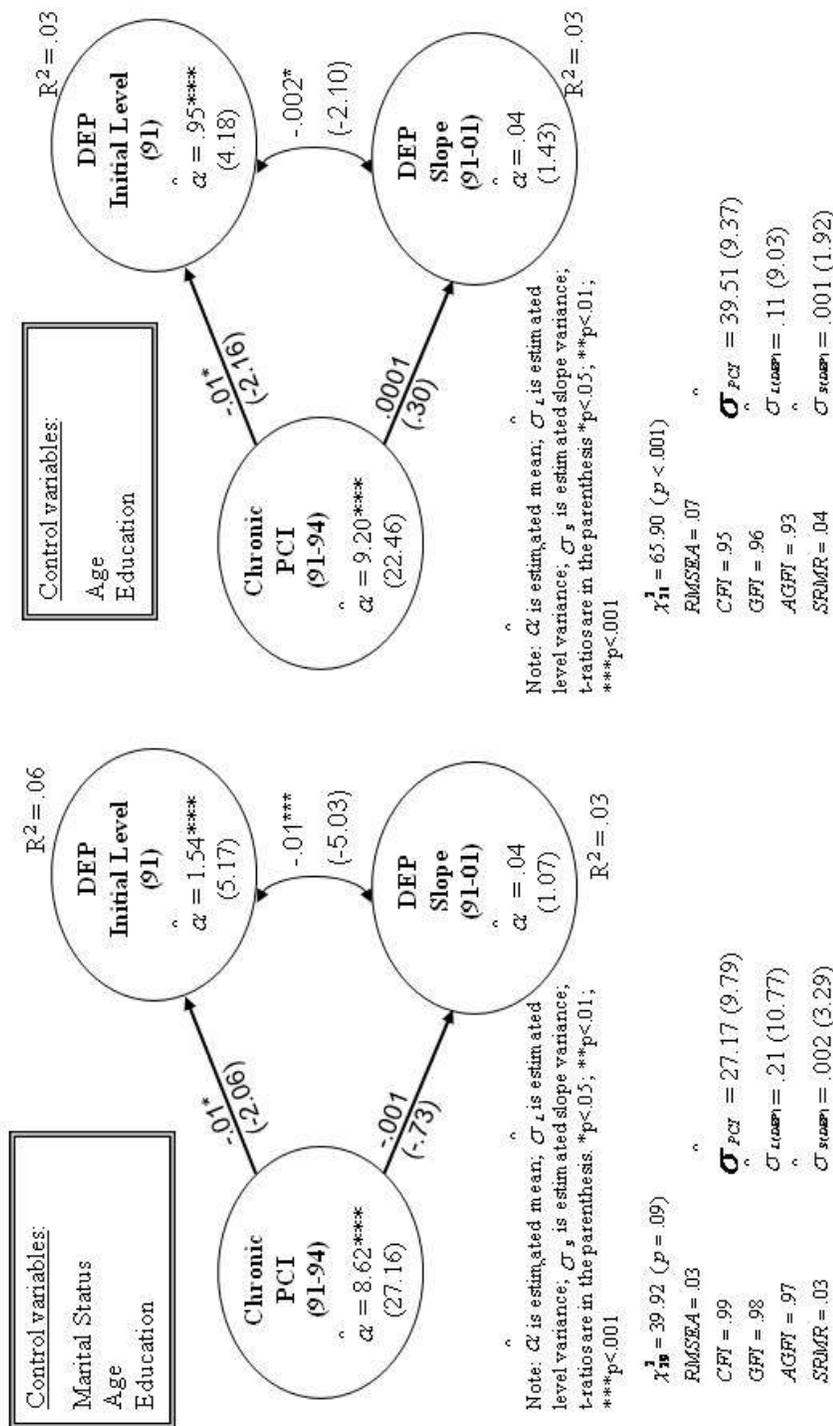


Figure 5 Direct effect of SES (chronic per capita income) on change in depressive symptoms for mothers

Figure 6 Direct effect of SES (chronic per capita income) on change in depressive symptoms for fathers

substantial SES gradient in the trajectory of DEP (i.e., p_1 is hypothesized to be significant without taking into account other relevant psychosocial variables). As indicated by Figure 5 and 6, on the whole, chronic PCI (1991-1994) was negatively related to initial levels of DEP in 1991, for mother and fathers alike ($-.01$, $t = -2.06$ for mothers; $-.01$, $t = -2.16$ for fathers). Compared to the modest but still significant associations between chronic PCI and initial levels of DEP, chronic PCI had only negligible impact on the rates of change for DEP from 1991 to 2001 for both mothers and fathers ($-.001$, $t = -.73$ for mothers; $.0001$, $t = .30$ for fathers), suggesting that chronic PCI per se did not interact with time when influencing DEP. Taken together, again for mothers and fathers alike, chronic PCI set the stage for cross-sectional differentiation in DEP at baseline (in 1991), and the SES disparities in DEP remained about the same from the beginning to the end of the decade. In addition, the unexplained negative covariation between two growth parameters (i.e., the level factor and the slope factor) of DEP is a commonly recognized phenomenon called “regression to the mean” ($-.01$, $t = -5.03$ for mothers; $-.002$, $t = -2.10$ for fathers), a tendency for high (or low) initial levels of a specific individual attribute to be followed by successive decreases (or increases) in levels toward the population average.

For a better illustration of models in Figure 5 and 6, Figure 7 plots the mean DEP across

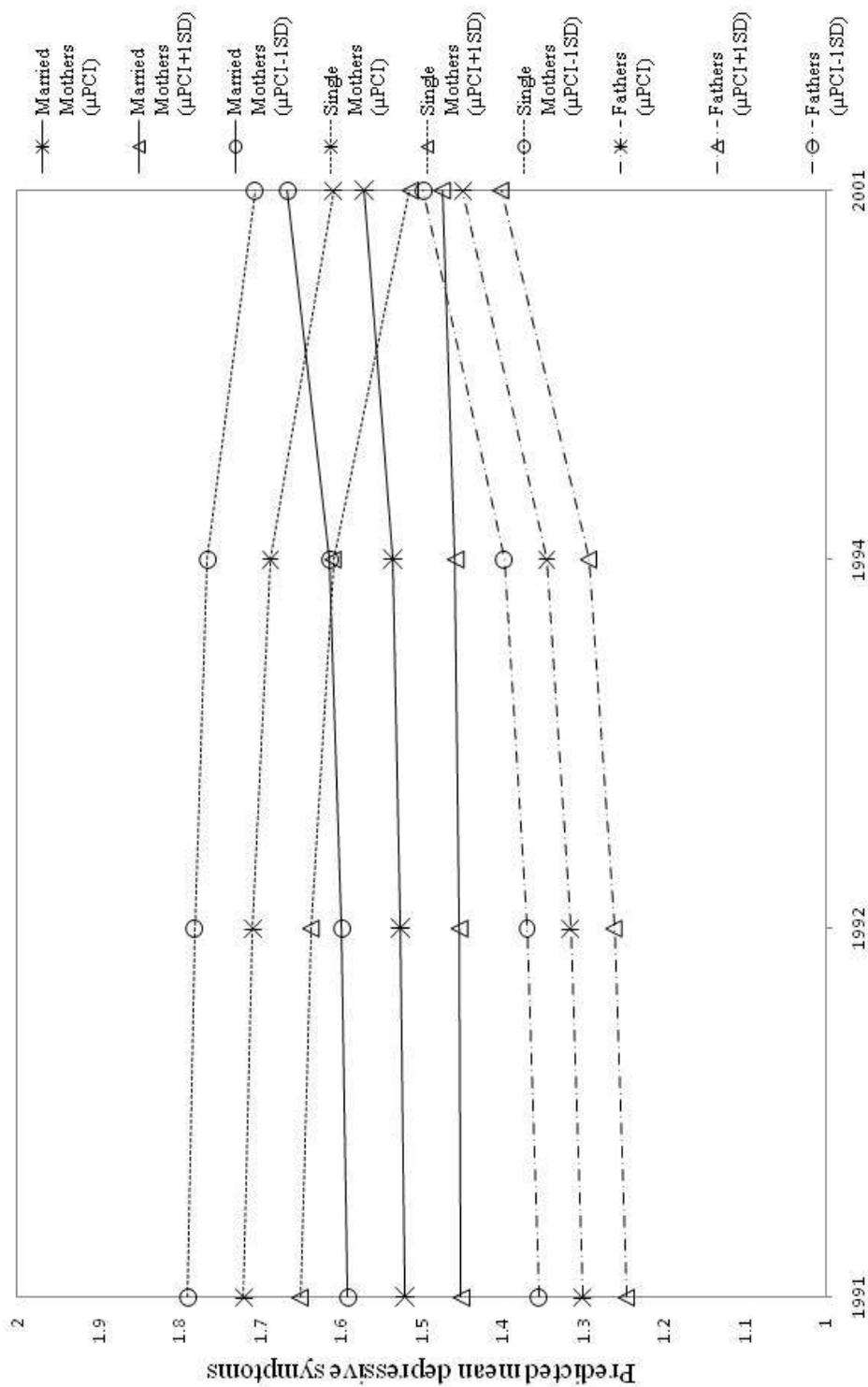


Figure 7 Mean trajectories of depressive symptoms from 1991 to 2001 predicted by chronic income (1991-1994), gender, and marital status

four waves predicted by three levels of PCI within each of the two sex groups (and within each of the two categories of marital status for mothers). Across sex groups and marital statuses, as indicated by Figure 7, the overall 10-year trajectories of DEP with respect to average PCI (μ_{PCI}), one standard deviation above average PCI ($\mu_{\text{PCI}} + 1\text{SD}$), and one standard deviation below average PCI ($\mu_{\text{PCI}} - 1\text{SD}$), respectively, are in essence parallel, denoting no “SES-by-time” interaction effect on change in DEP. Thus, people with higher chronic PCI tended to report persistently lower levels of DEP. In contrast, people characteristic of lower chronic PCI were more likely to experience higher levels of DEP in a perpetual manner.

Across SES groups, however, there is some evidence that sex and marital status each interacted with time when influencing DEP. First, although fathers as a whole reported chronically less DEP than mothers as a whole, the former also experienced progressively more DEP over the decade, which corresponds to what has been shown by their univariate growth curve of DEP in previous section. Despite the fact that mothers on average reported more DEP than fathers throughout the decade, there was no such an increasing trend in DEP for mothers as seen in the case of fathers. Among mothers, second, the interaction effect between marital status and time on change in DEP is even more impressive. Compared to single mothers, married mothers at large had chronically lower DEP within each of the three

PCI levels, and their DEP across all three PCI levels tended to remain about the same over the decade, except for a noticeable jump experienced only by those with below-average PCI at the end of the decade. As far as single mothers are concerned, in general, they not only had much higher baseline DEP (in 1991) but reported more DEP with persistency than married mothers of comparable PCI levels throughout the decade. However, a decreasing trend in DEP was evident for single mothers as a whole, in particular for those with above-average PCI. For all their markedly high mean levels of DEP at baseline (1991), in essence, single mothers as a whole reported progressively less DEP over the decade, such that within each of the three PCI levels, their final average levels of DEP (in 2001) converged to those of married mothers. Once again, this significant and rapid recovery for single mothers from extreme feelings of depression immediately after the divorce or separation has been observed elsewhere (e.g., Lorenz et al. 1997; 2006; Turner and Avison 1992).

According to the two baseline models estimated in Figure 5 and 6, to recap, cross-sectional differences in DEP among mothers and fathers earlier in the decade (1991) could be significantly explained by their relatively persistent SES characteristics indexed by chronic PCI, even though the effect size was modest. More importantly, the SES disparities in DEP revealed in 1991 largely remained the same throughout the decade. Notwithstanding

the homogeneous basic demographic profile among the current sample of rural mothers and fathers, as a result, there is substantial evidence that chronic levels of DEP were in general linked to the gradient of SES, of which income is an important, if not foremost, dimension (Adler, Boyce, Chesney, Cohen, Folkman, Kahn, and Syme 1994; Gallo and Matthews 2003; House et al. 1994; Lorant et al. 2003).

Some sex differences found in these two baseline models, though not dramatic, deserve a close scrutiny. First of all, mothers as a whole seemed to be more readily responsive to their enduring SES in terms of the initial levels of DEP, given their higher percentage of variance of initial DEP levels explained by chronic PCI than that of fathers (6% vs. 3%). Second, consistent with the result from univariate growth curves, there was more individual variation in rates of change (i.e., slopes) with regard to DEP for mothers than for fathers (.002, $t = 3.29$ vs. .001, $t = 1.92$). Third, the “regression to the mean” phenomenon associated with DEP seemed to be more evident for mothers than for fathers as a whole: not only did mothers have greater variation in initial DEP levels (.21, $t = 10.77$ vs. .11, $t = 9.03$), but the unexplained negative covariation between DEP level and DEP slope was stronger in magnitude for mothers than for fathers (-.01, $t = -5.03$ vs. -.002, $t = -2.10$), which is apparently, at least in part, due to the initially elevated levels of DEP followed by a rapid relief from the

remarkably negative affect for single mothers.

The fact that the association between chronic PCI and initial levels of DEP was modest (though significant) and chronic PCI did not interact with time when influencing DEP suggests that the effect of relatively stable SES characteristics on change in DEP, if there is any, might not be in a direct manner but mediated by some social-psychological variables indicative of either risk factors or coping resources for depressive responses. Including relevant psychosocial measures should increase the explained variance (i.e., R^2) in the trajectories of DEP and help illuminate why the SES gradient in DEP remained about the same over the decade for both mothers and fathers, despite the general trend of “regression to the mean.” As already discussed in previous chapter, this study mainly hypothesizes that SES represented by chronic PCI stipulates more closely the trajectories of appraisal or recognition of objective economic adversity as reflected by FS, which in turn has a more immediate consequence for change in MAS and DEP. Therefore, models in Figure 8 and 9 were estimated as variants of Figure 5 and 6, for mothers and fathers, respectively, by including chronic PCI between 1991 and 1994 as a predictor of both growth parameters (i.e., initial level and rate of change) of FS from 1991 to 2001.

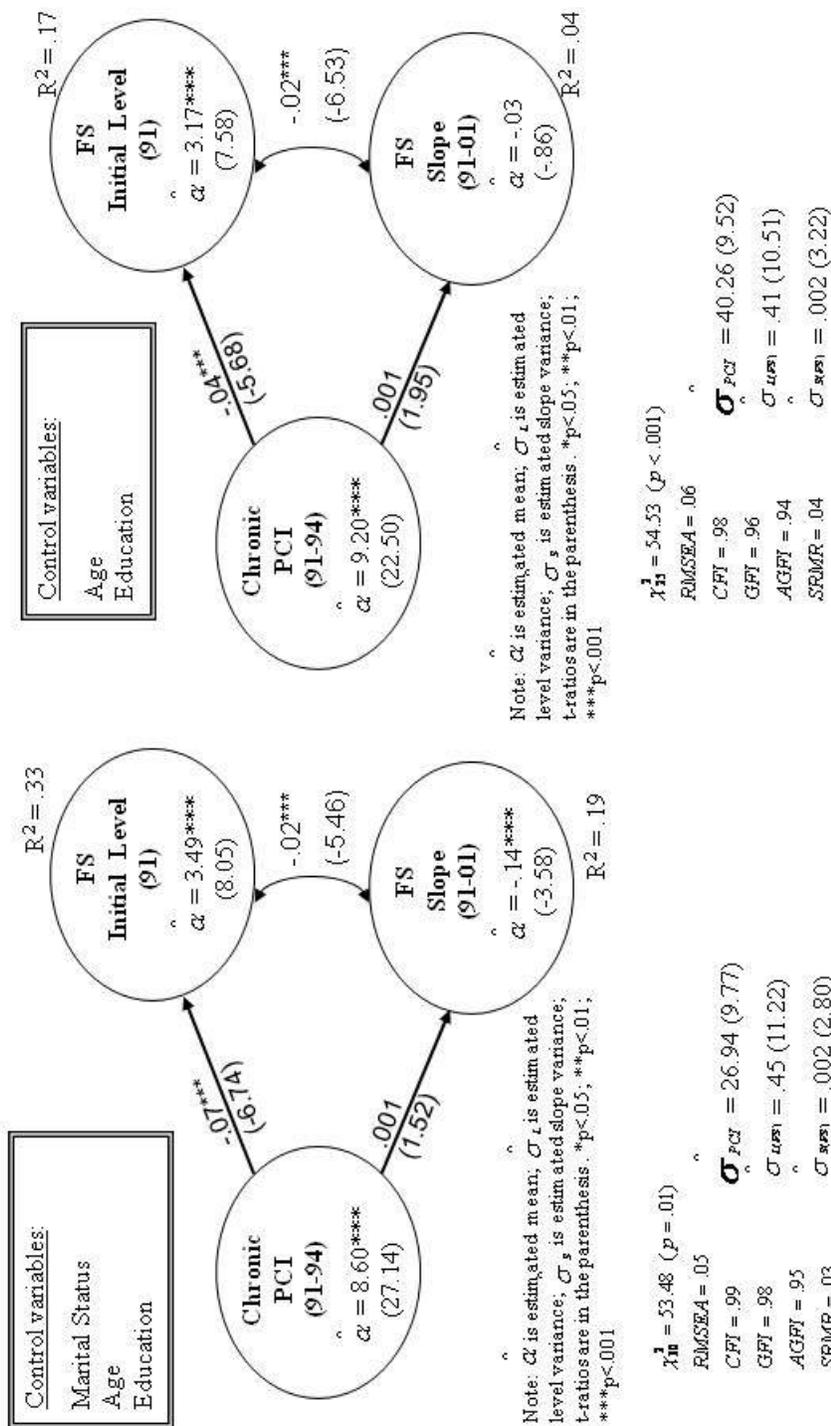


Figure 9 Direct effect of SES (chronic per capita income) on change in financial strain for fathers

Figure 8 Direct effect of SES (chronic per capita income) on change in financial strain for mothers

As shown in Figure 8 and 9, chronic PCI (1991-1994), for mother and fathers alike, was negatively related to the initial levels of FS in 1991 ($-.07$, $t = -6.74$ for mothers; $-.04$, $t = -5.68$ for fathers). It is evident that, across sex groups, the negative associations between chronic PCI and initial FS levels were stronger in magnitude and greater in significance than those between chronic PCI and initial DEP levels, according to the corresponding unstandardized estimates. To a less significant extent, in addition, there was a notable positive effect of chronic PCI on the rates of change for FS between 1991 and 2001 for both mothers and fathers ($.001$, $t = 1.52$ for mothers; $.0001$, $t = 1.95$ for fathers), suggesting that chronic PCI marginally interacted with time when influencing FS. Taken together, across sex groups, chronic PCI not only contributed to substantive cross-sectional differentiation in FS levels at the beginning of the decade, but also shaped the rates of change for FS throughout the decade with borderline significance. The positive association between chronic PCI and FS slopes, nonetheless, does not mean that individuals with higher chronic PCI tended to experience a progressive escalation in FS over time. With in mind that the average slope of FS was negative for both mothers and fathers ($-.14$, $t = -3.58$ for mothers; $-.03$, $t = -.86$ for fathers), a more appropriate interpretation is that, on average, those with higher chronic PCI would have experienced a slower rate of decrease in FS, since their initial levels of FS were

presumably low enough such that it was unlikely for their FS levels to precipitate at the same rate as experienced by those with lower chronic PCI. Figure 10 provides a graphic illustration of models in Figure 8 and 9 by plotting the predicted mean FS levels across four waves given three benchmarks of PCI within each of the two sex groups (and within each of the two categories of marital status for mothers).

Across sex groups and marital statuses, as indicated by Figure 10, people with higher chronic PCI tended to experience enduring lower FS levels. In contrast, people characteristic of lower chronic PCI were more likely to report higher levels of FS in a perennial manner.

Across SES groups, there was an overall decreasing trend in FS levels throughout the decade.

However, compared to those with average chronic PCI, those with below-average chronic PCI in general had a faster rate of decrease in FS levels, while the overall mean slope of FS was much flatter for those with above-average chronic PCI. Within each of the three chronic PCI levels, single mothers as a whole had the highest chronic FS levels, followed by married mothers and then fathers⁴. On the other hand, the average rate of decrease in FS levels was faster for single mothers than for married mothers across three chronic PCI levels, but it was

⁴ Given that IYFP couples shared exactly the same amount of gross per capita family income, again, the estimated difference in the univariate growth curve of FS between married fathers and married mothers was small and basically due to measurement error and maybe some missing data (e.g., a husband of a participating women did not participate). The more important difference was then between married and divorced mothers.

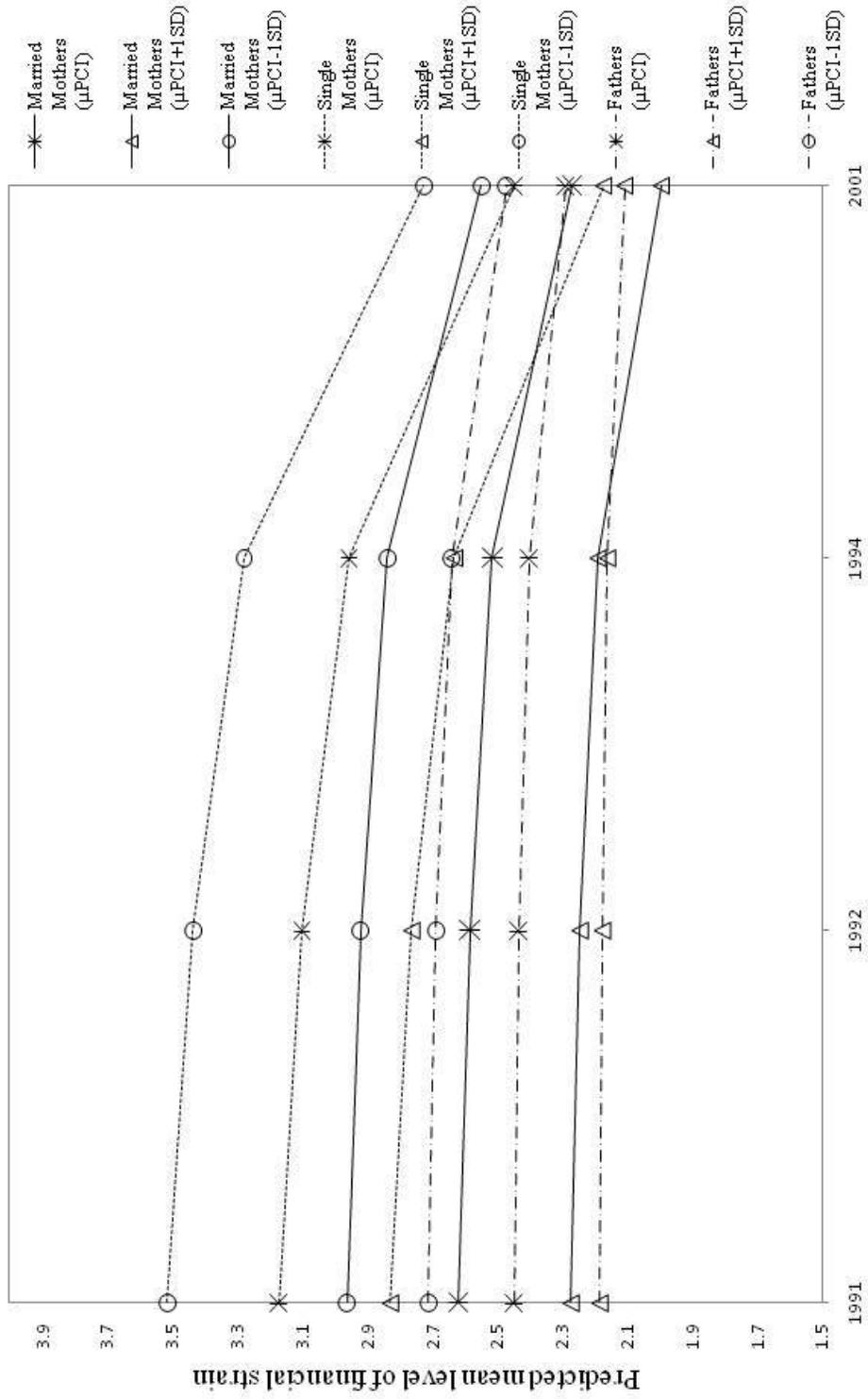


Figure 10 Mean trajectories of financial strain from 1991 to 2001 predicted by chronic income (1991-1994), gender, and marital status

not obvious for fathers as a whole to have a decreasing trend in FS levels throughout the decade.

4.5 Mediation of Financial Strain Trajectories between SES (Chronic Income) and Change in Depressive Symptoms

Combining models in Figure 5 and 8, the model in Figure 11 was estimated to examine to what extent the trajectories of FS mediated between chronic PCI and the trajectories of DEP for mothers. Within the context of Figure 1, this model tests the hypothesis that originally significant direct effect of SES (chronic PCI) on the trajectory of DEP is totally mediated by the trajectory of FS, i.e., when estimated simultaneously, P_2 (negative) and P_3 (positive) are significant whereas P_1 (originally negative) ceases to be.

Essentially, Figure 11 shows that for mothers as a whole, chronic PCI had a significant and negative impact on initial levels of FS ($-.07$, $t = -6.75$) in 1991. There were also a significant and positive contemporaneous association between initial levels of FS and those of DEP ($.24$, $t = 5.97$). It is manifest that the effect of chronic PCI on initial levels of DEP found significant in previous section was completely mediated by initial levels of FS to the extent that the direct association between chronic PCI and initial levels of DEP dropped out ($.002$, $t = .30$) with initial levels of FS accounted for. In addition, the indirect effect of

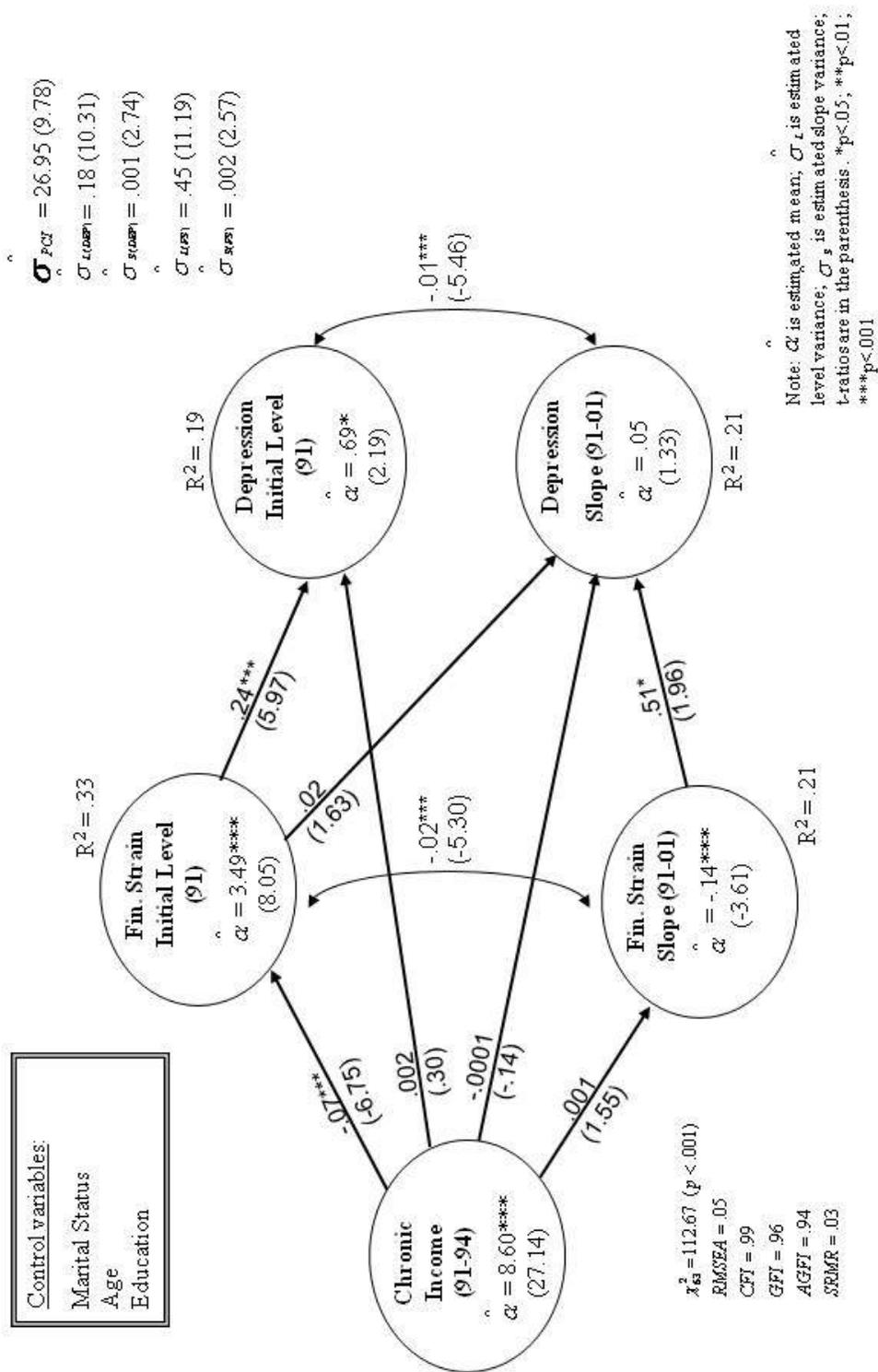


Figure 11 Mediation of financial strain trajectory between SES (chronic per capita income) and change in depressive symptoms for mothers

chronic PCI on initial levels of DEP estimated by LISREL with Sobel test was significant and negative ($-.02$, $t = 4.44$). Taken together, for mothers as a whole, the total effect of chronic PCI on initial levels of DEP was primarily indirect and completely mediated by initial levels of FS.

Chronic PCI, on the one hand, was positively related to the rates of change for FS, though only to a marginal degree ($.001$, $t = 1.55$). Noting that the average slope of FS for mothers was negative ($-.14$, $t = -3.61$), again, this indicates that, in general, mothers who had higher chronic PCI and presumably lower initial levels of FS would have experienced a slower rate of decrease in FS throughout the decade than those who had lower chronic PCI and most likely higher initial levels of FS. On the other hand, the association between FS slope and DEP slope was significant and positive ($.51$, $t = 1.96$), suggesting that any acute increases or decreases in the levels of FS over time was largely accompanied by simultaneous increases or decreases in the levels of DEP. Of greater interest, initial levels of FS marginally interacted with time when influencing DEP ($.02$, $t = 1.63$). That is to say, higher initial levels of FS tended to accelerate subsequent increases in DEP over time; stated otherwise, lower levels of FS at baseline were generally predictive of slower rate of increase in DEP afterwards. Finally, neither the direct effect ($-.0001$, $t = -.14$) nor the indirect effect ($-.0004$, t

= -.64) of chronic PCI on DEP slope was significant, as estimated by LISREL with Sobel test.

Findings from mothers can thus be summarized as follows: (1) the overall effect of chronic PCI on initial levels of DEP was essentially indirect and completely mediated by initial levels of FS; (2) chronic PCI had neither direct nor indirect effect on DEP slope; (3) there were positive level-to-level (i.e., chronic) and slope-to-slope (i.e., acute) associations between FS and DEP; (4) even with only marginal significance, a positive and modest level-to-slope association between FS and DEP indicates that initial levels of FS might influence the rates of change in DEP over time.

Figure 12 tells a pretty similar story for fathers. Generally speaking, most of the findings revealed from mothers not only hold but turn out to be more statistically significant for fathers. First of all, the direct association between chronic PCI and initial levels of FS was negative and significant ($-.04$, $t = -5.66$) for father as a whole. That is, for fathers, on average, the higher the chronic PCI, the lower the initial levels of FS. Next, chronic PCI significantly interacted with time when influencing FS ($.001$, $t = 1.98$), suggesting that in general, given the negative average slope of FS ($-.04$, $t = -.95$), fathers with higher chronic PCI would have experienced a slower rate of decrease in FS, since their initial levels of FS might be too low

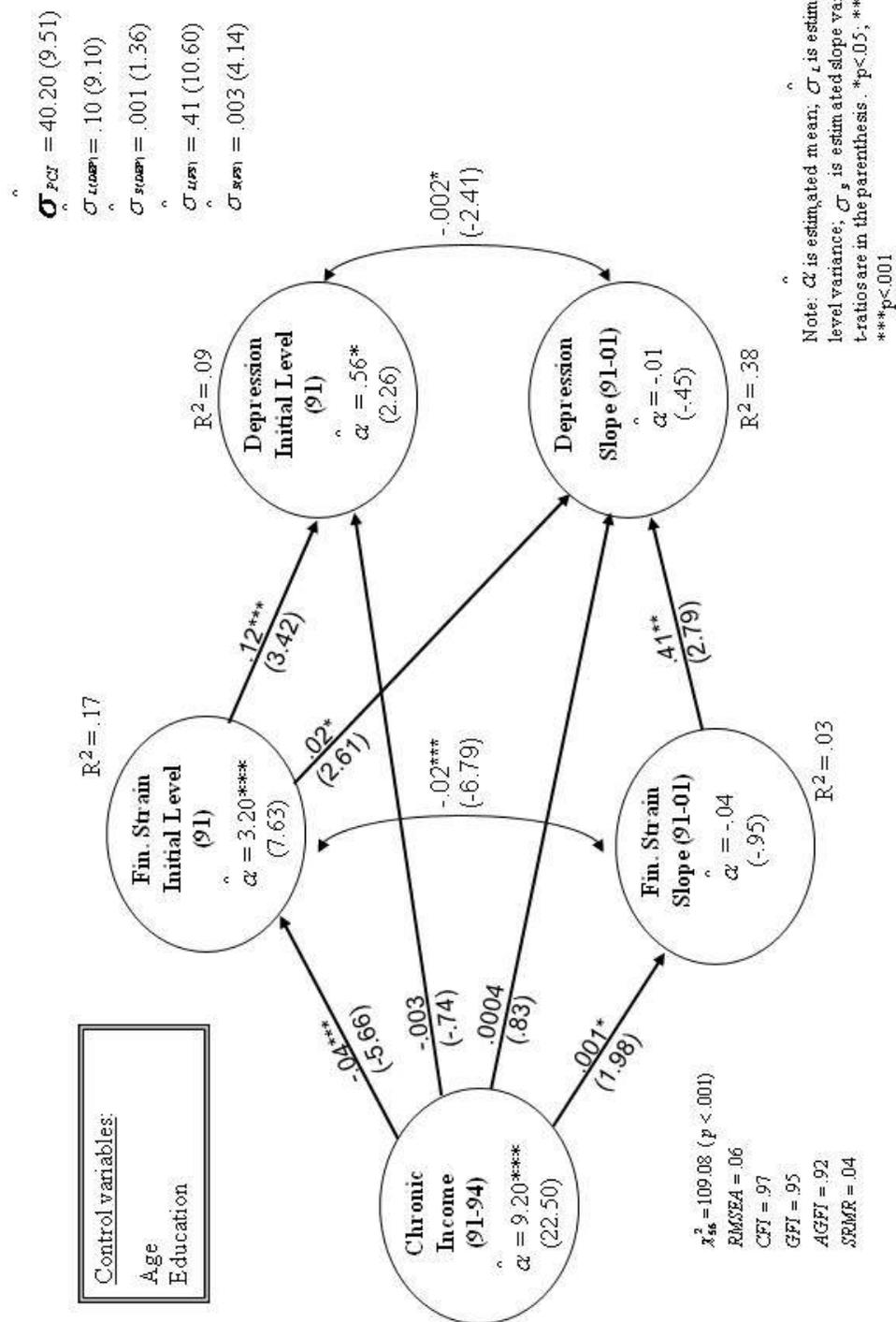


Figure 12 Mediation of financial strain trajectory between SES (chronic per capita income) and change in depressive symptoms for fathers

to further descend at the same rate as experienced by those with lower chronic PCI and supposedly higher initial levels of FS.

As in the case of mothers, there were also two systematic associations between FS and DEP for fathers in terms of interlocking levels as well as interlocking slopes. In essence, both the level-to-level (.12, $t = 3.42$) and the slope-to-slope (.41, $t = 2.79$) associations were positive and significant. Thus, from a cross-sectional point of view, fathers with higher (or lower) initial levels of FS tended to report higher (or lower) initial levels of DEP. In terms of a longitudinal perspective, furthermore, fathers going through acute increases (or decreases) in levels of FS over time were more likely to experience simultaneous increases (or decreases) in levels of DEP.

The fact that the direct association between chronic PCI and initial levels of DEP was not significant ($-.003$, $t = -.74$) with initial levels of FS adjusted for signifies that on average, relationship between chronic PCI and initial levels of DEP was completely mediated by initial levels of FS, which can be further confirmed by a significant and negative indirect effect of chronic PCI on initial levels of DEP ($-.01$, $t = -2.95$) as estimated by LISREL with Sobel test. Therefore, just like their female counterparts, chronic PCI influenced initial levels of DEP altogether by way of initial levels of FS for father as a whole. The direct association

between chronic PCI and DEP slope was not significant (.0004, $t = .83$) when controlling for FS slope, likewise, implying a potential mediation of FS slope between chronic PCI and DEP slope. However, the indirect effect of chronic PCI on DEP slope estimated by LISREL with Sobel test was not significant (-.0003, $t = -1.08$).

Finally, unlike mothers, there is stronger statistical evidence for fathers as a whole that initial levels of FS interacted with time when influencing DEP, as indicated by the significant and positive effect of FS level on DEP slope (.02, $t = 2.61$). In view of the comparable unstandardized coefficient of .02 on the level-to-slope path across sex groups, there seems to be little doubt that the overall trend for initial levels of FS to influence the rates of change in DEP later in time was equivalently substantial for both fathers and mothers.

The whole picture about the dynamics implicated in the SES gradient of depression has been so far looming out of the enormous similarities between the separate findings from mothers and fathers. Above all, the effect of chronic PCI on chronic levels of DEP was indirect and completely mediated by chronic levels of FS. In addition, FS trajectories and DEP trajectories were virtually interlocking insofar as there were three specific types of positive associations observed (1) between FS level and DEP level, (2) between FS slope and DEP slope, and (3) between FS level and DEP slope.

Despite the positive slope-to-slope correlation between FS and DEP, notably, there was also an overall trend that increasing chronic PCI was linked to slower rates of decrease in FS for the current sample of rural mothers and fathers. However, both of these two direct associations were modest and barely significant. In particular, there was no significantly negative indirect effect of chronic PCI on DEP slope through FS slope. Thus, it should be cautioned to make any inferential statement about the “slope-to-slope” part of interlocking trajectories. For example, it may be true that inadequate chronic PCI could lend itself to decreases in FS at a faster rate, in turn leading to a precipitous drop in DEP simultaneously in the short run. This, however, does not necessarily mean that the financially disadvantaged as a whole could eventually experience as low chronic levels of DEP as reported by the well-to-do in the long run, for which there are at least two reasons.

First, the direct level-to-level correlations between chronic PCI and FS as well as between FS and DEP were highly significant and substantial, and so was the negative indirect effect of chronic PCI through FS level on DEP level. Therefore, from the cross-sectional point of view, the gradient of chronic PCI was readily translated into chronic levels of FS and DEP. More importantly, there was also a positive, though modest, level-to-slope association between FS and DEP, whereby initial levels of FS interacted with time when influencing DEP.

Hence, the higher the levels of FS were at baseline, the more rapidly the levels of DEP increased afterwards; alternatively, lower initial levels of FS tended to be associated with subsequent increases in DEP at a much slower rate over time. Taken together, one can try to picture the dynamic relationship between SES and trajectories of DEP in the following ways: (1) the SES gradient as reflected by distinct levels of chronic PCI could be largely projected onto initial levels of DEP by differentiating initial levels of FS first; (2) to the extent that heightened initial levels of FS might accelerate the rates of increase in DEP afterwards, the SES differentials in initial levels of DEP could remain intact or become even divergent over time, despite the general “regression to the mean” phenomenon.

4.6 Mediation of Mastery Trajectories between Change in Financial Strain and Change in Depressive Symptoms

As previously indicated, the last puzzle piece for understanding the dynamics implicated in the SES gradient of depression would be the potential mediation of mastery trajectories. To recap, the full model in Figure 1 hypothesizes that the trajectory of financial strain as a potential complete mediator between SES (chronic income) and the trajectory of mastery (i.e., when estimated simultaneously, P_2 and P_4 are hypothesized to be significant and negative but P_5 is not), and in turn, (2) the trajectory of mastery as a potential complete mediator between

the trajectory of financial strain and the trajectory of depressive symptoms (i.e., when estimated simultaneously, P_4 and P_6 are hypothesized to be significant and negative but P_3 is not). Models in Figure 13 and 14 were thus estimated to examine the extent to which MAS trajectories mediated between FS trajectories and DEP trajectories for mothers and fathers, respectively.

It is not difficult to tell that, again, initial levels of MAS completely mediated between initial levels of FS and initial levels of DEP for both mothers and fathers. First of all, the level-to-level correlations were significant between FS and MAS (for mothers: $-.34$, $t = -9.18$; for fathers: $-.28$, $t = -8.04$) as well as between MAS and DEP (for mothers: $-.56$, $t = -10.96$; for fathers: $-.55$, $t = -10.07$), but it was not the case for the correlation between FS and DEP (for mothers: $.05$, $t = 1.46$; for fathers: $-.03$, $t = -1.05$). In addition, the level-to-level indirect effect estimated by LISREL with Sobel test was significant between chronic PCI and MAS (for mothers: $.02$, $t = 5.48$; for fathers: $.01$, $t = 4.75$), between chronic PCI and DEP (for mothers: $-.02$, $t = -4.88$; for fathers: $-.01$, $t = -3.31$), and between FS and DEP (for mothers: $.19$, $t = 7.06$; for fathers: $.16$, $t = 6.21$).

Taken together, the SES gradient as indexed by chronic PCI was primarily responsible for the analogous differentiation in initial levels of FS and MAS, which in turn literally

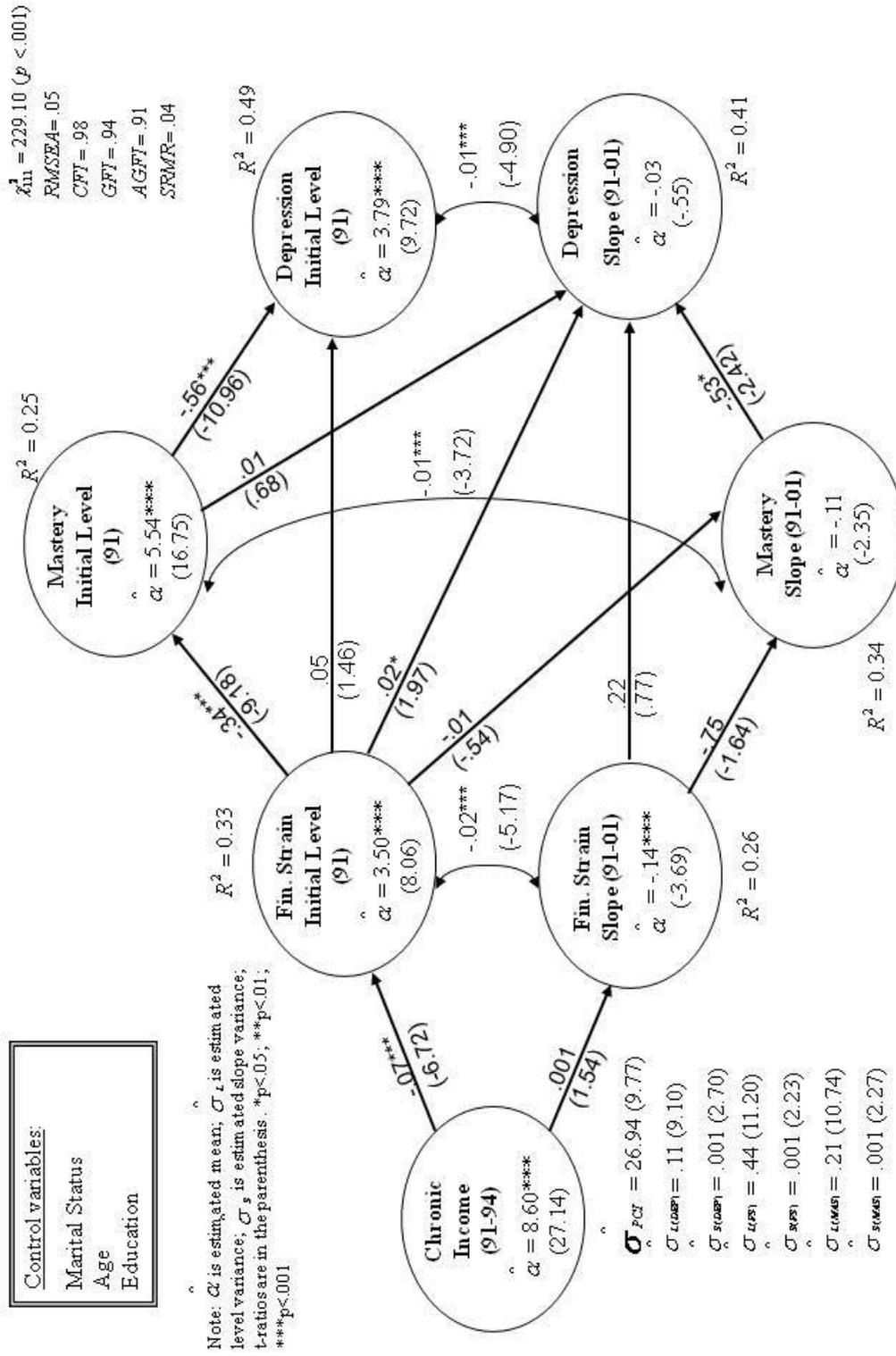


Figure 13 Mediation of mastery trajectory between change in financial strain and change in depressive symptoms for mothers

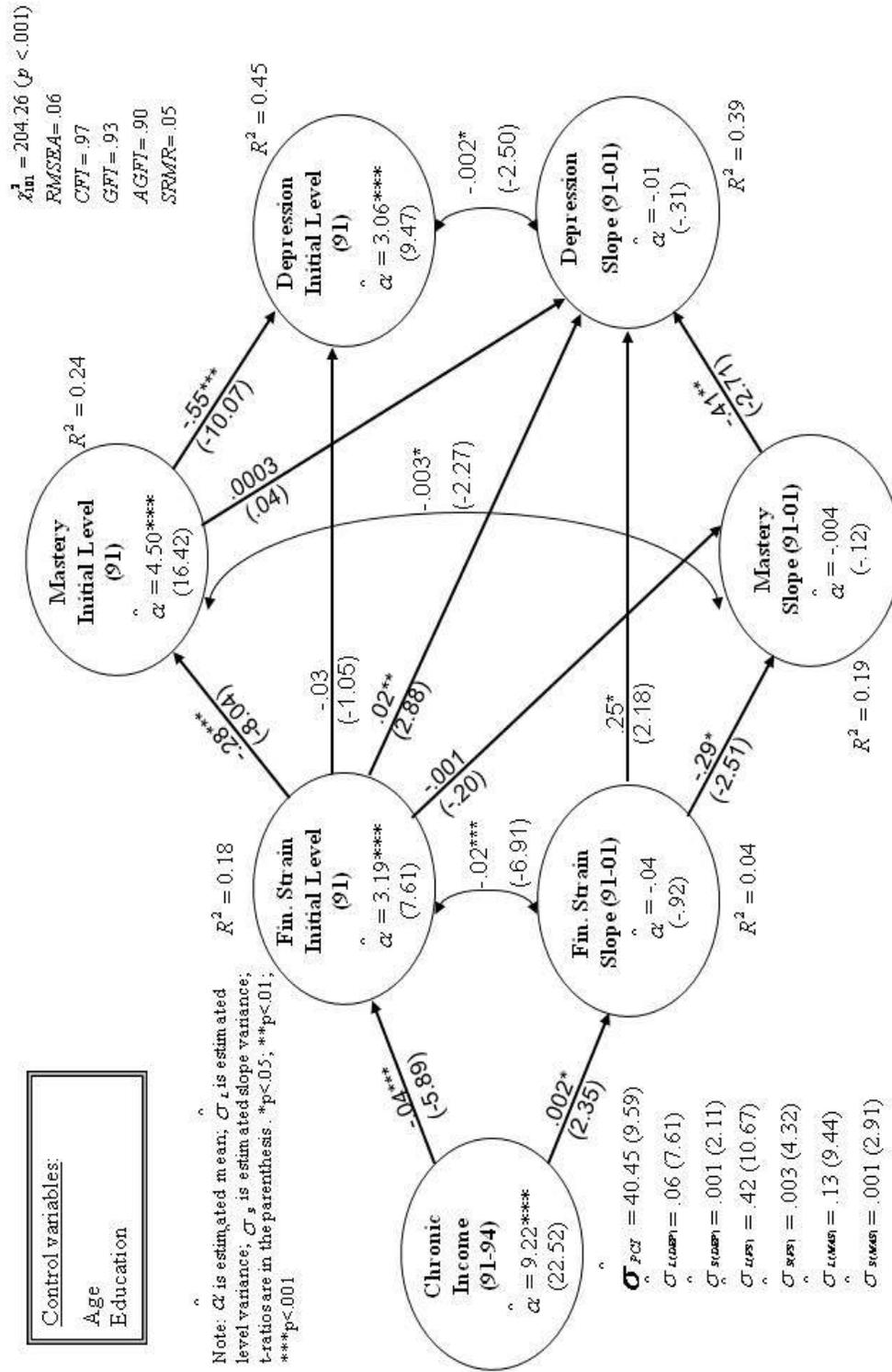


Figure 14 Mediation of mastery trajectory between change in financial strain and change in depressive symptoms for fathers

translated into corresponding hierarchies in initial levels of DEP.

For mothers, the slope-to-slope association was only significant between MAS and DEP (-.53, $t = -2.42$), but it was not the case between FS and MAS (-.75, $t = -1.64$) as well as between FS and DEP (.22, $t = .77$).

For fathers, the slope-to-slope correlations between FS and MAS (-.29, $t = -2.51$), between FS and DEP (.25, $t = 2.18$), and between MAS and DEP (-.41, $t = -2.71$) were all significant, even though all three of them were modest in magnitude.

Regardless of statistical significance, however, the overall trend revealed by the slope-to-slope correlations is consistent across sex groups: acute increases in FS levels over time was largely related to simultaneous reduction in MAS levels and concurrent growth in DEP levels; alternatively, FS levels on the decrease were in general accompanied by enhancement of MAS levels and diminution of DEP levels at the same time. Thus, as suggested by previous literature, “resources deterioration” seems to have more empirical grounds than “resources mobilization” (Ensel and Lin 1991; Wheaton 1985).

Although the positive association between chronic PCI and FS slope was still present here (for mothers: .001, $t = 1.54$; for fathers: .002, $t = 2.35$), it was apparently subject to the “regress to the mean” phenomenon. Once again, it deserves great caution against inferring

too much about the interlocking slopes, given that most of the slope-to-slope correlations were barely significant and extremely modest in magnitude.

Last, and most importantly, a significant and positive level-to-slope association between FS and DEP held for both mothers (.02, $t = 1.97$) and fathers (.02, $t = 2.88$). This again indicates a “level by time” interaction, whereby initial levels of FS interacted with time when influencing DEP. To wit, higher initial levels of FS tended to pave the way for more rapid increases in DEP levels later on. Stated otherwise, lower initial levels of FS were generally predictive of subsequent increases in DEP levels at a slower rate.

Taken together, again, there seems to be a “double jeopardy” for those in relatively persistent poverty as a whole in terms of mental health: (1) chronically adverse economic situation was in general related to higher cross-sectional levels of DEP, first by enhancing contemporaneous levels of FS, and next by suppressing contemporaneous levels of MAS; (2) despite the general trend of “regression to the mean,” the SES differentials projected onto initial levels of DEP through corresponding hierarchies in initial levels of FS and MAS could remain intact or even become divergent over time insofar as the initial levels of FS might affect the way DEP levels changed subsequently by either accelerating the rates of increase or slowing down the rates of decrease in levels of DEP for the financially disadvantaged.

4.7 Change in Marital Status

As shown in Table 9 and 10, change in marital status did occur sometime between 1991 and 2001 for some cases among the sample. Of all 305 fathers who were from IYFP and therefore married in 1991, 17 (5.6%) got divorced sometime after 1991. For IYFP mothers who were married in 1991, there was no change in marital status for 305 out of 320 (95%) between 1991 and 2001, while the remainder 15 (5%) changed to being divorced during the same period of time. Finally, the ISPP mothers who were divorced in 1991 can be subdivided into those who remained divorced (37 out of 83) till the end of the observation and those who recoupled (46 out of 83, either by remarrying or cohabiting) at some point in time over the decade.

Table 9 Change in marital status during 1991-2001 for fathers

<i>Marital Status after 1991</i>	Married	Divorced
<i>Sample Source (1991)</i>		
IYFP (married)	288	17

Even though it was a relatively small fraction of the sample that had experienced a transition in the state of marriage, models in Figure 13 and 14 were re-estimated with additional control variables to capture the possible confounding effects of change in marital

Table 10 Change in marital status during 1991-2001 for mothers

<i>Marital Status after 1991</i>	Married	Divorced
<i>Sample Source (1991)</i>		
IYFP (married)	305	15
ISPP (divorced)	37	46

status after 1991. Specifically, a dummy variable was created for fathers, distinguishing between those who remained married throughout the decade (no change in status: coded 0) and those who did not (changed to being divorced: coded 1).

For mothers, given the four categorizations of change in marital status (i.e., IYFP-remained married; IYFP-changed to divorced; ISPP-remained divorced; ISPP-recoupled), three contrast variables were created: the first tested the overall difference between IYFP mothers (coded 1) and ISPP mothers (coded -1); the second examined the specific divergence between the two groups within IYFP mothers (-1= “IYFP-married” vs. 1 = “IYFP-divorced”); the third drew an explicit comparison between ISPP mothers with change in marital status and those without (-1= “ISPP-divorced” vs. 1 = “ISPP-recoupled”).

In general, the re-estimated models with additional control variables to account for possible confounding effects of change in marital status (result not shown) did not differ to

any appreciable degree from what has been shown in Figure 13 and 14, and the result presented above thus received further support.

CHAPTER 5

DISCUSSION

5.1 Findings

Result from the current study adds to previous similar research in at least two ways. First, it sheds light on the causal sequence between personal resources as reflected in the sense of mastery and psychological distress as measured by continuous scales of depressive symptoms. Although it has been argued that mastery is neither intrinsic to the very nature of depression nor an inherent as well as inseparable symptom of depression (Pearlin et al. 1981), little is known about which takes the causal precedence in their highly negative association with each other. The cross-lagged autoregressive models demonstrate that (1) mastery was less volatile than depressive symptoms to the extent that the stability effect was greater for mastery than for depressive symptoms, (2) depressive symptoms at one point in time were to a lesser extent conditioned by its own earlier levels and therefore more reactive to mastery at previous point in time, and (3) the causal primacy of mastery over depressive symptoms held for both sexes even when controlling for other important socioeconomic and socio-demographic variables.

Second, by implementing the LGC analytic framework that separates the absolute levels of variables from their rates of change, this study also illuminates the dynamics involved in

the seemingly stable SES gradient of depression by including psychosocial constructs as intervening processes, in particular by taking into account financial strain as a measure of stress and mastery as an index for personal resources. This dynamic process can be briefly summarized as follows: (1) the SES gradient as reflected by distinct levels of chronic per capita family income among individuals could at large translate into corresponding hierarchies in initial levels of depressive symptoms by differentiating initial levels of financial strain and mastery; (2) in the short run, acute changes in financial strain, mastery, and depressive symptoms were modestly inter-correlated, and these interlocking slopes, in turn, were modestly conditioned by chronic per capita family income; (3) in the long run, as indicated by the significant positive “level-to-slope” correlation between financial strain and depressive symptoms, the SES disparities reflected in cross-sectional levels of depressive symptoms at baseline could remain intact or even get divergent over time to the extent that those families with high initial levels of financial strain also experienced accelerated rates of increase in subsequent levels of depressive symptoms, thus counteracting the general trend of “regression to the mean.”

The trajectories were interlocking in an immediate or acute sense between financial strain and mastery as well as between mastery and depressive symptoms: as reflected by significant

level-to-level and slope-to-slope associations between financial strain and mastery as well as between mastery and depressive symptoms, mastery was only reactive to contemporaneous levels of and proximate fluctuations in financial strain, and it was the same case for the dynamic relationship between depressive symptoms and mastery. In contrast, the significant and positive level-to-slope correlation between financial strain and depressive symptoms implies a longer-lasting sense of devastation for psychological wellbeing attributable to a distal perception of economic adversity. To wit, those who once recognized that they were economically deprived to a significant degree were very likely to experience chronically higher levels of depressive symptoms in that the heightened levels of financial strain (presumably accompanied by heightened levels of depressive symptoms) at certain point in time could either prevent subsequent levels of depressive symptoms from moving toward the mean (by slowing down the rates of decrease in subsequent levels of depressive symptoms), or, even worse, could drive subsequent levels of depressive symptoms to move further away from the mean (by accelerating the rates of increase in subsequent levels of depressive symptoms).

5.2 Implications

The findings have both theoretical and policy implications. In terms of the stress research,

first, it confirms previous observation that despite the clear conceptual distinctions, financial strain seems to be highly intertwined with negative cognitions and emotions that are characteristic of demoralization (Angel et al. 2003; Dohrenwend, Shrout, Egri, and Mendelsohn. 1980). Second, it reveals that levels of financial strain that are once intensified, even though followed by a gradual relief from the hardship, may still provoke a internal transition from “being poor” to “feeling poor” (Blacksher 2002), and exert a protracting effect to escalate depressive symptoms in the long run.

What may account for this “level by time” interaction between financial strain and depressive symptoms deserve further investigation. One possible mechanism pertains to the notion of “stress proliferation” that serious stressors experienced early in time (i.e., primary stressors) tend to give rise to following additional stressors (i.e., secondary stressors) that (1) may mediate between initial (primary) stressors and later measures of psychological distress or (2) may themselves have independent and additive effect (beyond and above initial stressors) on the subsequent health outcome (Kahn and Pearlin 2006; Pearlin 1989; Pearlin et al. 2005; Pearlin, Aneshensel, and LeBlanc 1997). Another possible scenario is analogous to what has been termed “kindling/sensitization” process (see Hammen 2005 for a review), which suggests that the association between stressors and depression may be progressively

diminishing to the extent that repeated stressors and/or recurrent depressive episodes could lead to some neurobiological changes that further render individuals sensitized or kindled to have *spontaneous* depressive responses afterwards. This “wear and tear” effect of persistent or chronic stressors has been referred to as “allostatic load,” whereby organism becomes vulnerable by degree to health-damaging agents and less able to fully recover from the resulting physical as well as mental impairment (Kahn and Pearlin 2006; McEwen and Seeman 1999; Singer and Ryff 1999). Apparently, both “kindling/sensitization” process and “allostatic load” posit an increasing susceptibility to current stress as a result of over- or repeated exposure to previous stress. Future research should extend this study by including a comprehensive set of stress measures other than financial strain and separate their chronic levels from rates of acute change, as has been demonstrated here, to determine whether stress proliferation or kindling/sensitization/allostatic load better accounts for the “level by time” interaction between financial strain and depressive symptoms.

For policy makers, one message from this study seems clear. The rural American families that make up the IYFP and ISPP samples constitute a “distressed region” (Elder and Conger 2000: 12). The farm crisis in 1980s impacted farm families and rural communities alike through the misfortunes of the farm families that lost their farms and the non-farm families

that lost their jobs in the economic downturn. The events of the late 1980s defined these rural communities as a high-risk environment by launching a trend toward increasing and continuing economic disadvantages (i.e., chronic condition of poverty) for many families in this study. The interpretation of and response to socioeconomic change of these families may to some degree be unique to rural families and may reflect a distinctive “rural character.” However, this study may generalize beyond time and place regarding the role of economic hardship in family life. In essence, the experiences of these families not only substantially replicate what urban families had faced during the Great Depression (Conger and Elder 1994; Elder and Conger 2000), they may well predict the fate of those who are facing the current housing “credit crunch” or anticipating other macro financial crises to come. After entering the 21st century, in particular, we need to become more capable of successfully adapting to economic uncertainty and adversity due to the dramatic structural changes resulting from the irreversible and ongoing trend of globalization.

5.3 Limitations

This study has two major limitations. First, although financial strain and depressive symptoms were selected as principal stressor and health outcomes, respectively, to demonstrate the dynamics implicated in the SES gradient of psychological distress, these two

alone do not capture the full universe of stressors or the full spectrum of mental disruptions that are associated with SES over the life course (Aneshensel 1992; 1999; 2005; Wheaton 1994; 1999). Future research on the dynamic process of stressful consequences due to SES should include a more comprehensive array of stressors and mental health measures across the life span, such as negative life events, daily hassles, and life-time traumas for the former, and various types of symptoms (e.g., anxiety, anger, hostility), behavioral problems (e.g., alcohol and substance use, violent and antisocial behavior), and maybe clinical mental disorders (e.g., major depression, anxiety disorder) for the latter.

The second major limitation concerns the potential bias and generalizability of the result presented here. As indicated by the attrition analyses, those who dropped out of the panel tended to be among the financially worst-off. Their absence from the analyses might have introduced bias into the sample. More importantly, one may doubt how representative this panel of Caucasian mid-aged parents in rural Iowa is when generalizing the findings here to people of different demographic characteristics and in other ecological or geographical settings. The answers to previous questions are speculative but auspicious. First of all, had the drop-out cases been included to increase the variation of the financial trajectories in the sample, the associations found here would likely have been much stronger. Next, not only did

the experiences of these rural Iowa families with children going through the “farm crisis” extend previous research findings derived from urban families during the Great Depression (Conger and Elder 1994), there is also accumulating evidence that relationships found in this panel have been replicated in minorities, urban contexts, and even foreign countries (see Lorenz et al. (2006) for a partial list of replicated sites). Finally and most importantly, this particular sample is unique and, in some sense, desirable for stress studies to the extent that the “farm crisis” served as a natural experiment: these families became more diversified during the farm crisis. The financial trajectories of these rural residents, which were highly homogeneous at the beginning, became more differentiated as the farm crisis unfolded, with some but not all losing their farms and/or their jobs. This situation, though unfortunate, is perfect for testing the robustness of dynamic relationship between income-oriented SES and psychological distress. However, as noted before, it would still be desirable for findings presented here to be replicated with other families in other places and at different points in time based on a similar longitudinal analytic framework and including a more comprehensive set of stress and health outcome measures.

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