Familial Susceptibility to Suicide, Death of Parents in Early Life, and Behavioral Health Disorders in Older Adulthood: F x E Interactions

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ABSTRACT

We examined how familial susceptibility to suicide might interact with early-life parental death to create behavioral health phenotypes in older adulthood. We utilized demographic and pedigree data from the Utah Population Database, linked with Centers for Medicare and Medicaid Services records for the State of Utah between 1992 and 2009, to examine this familiality x environment (F x E) interaction. The final sample included 163,295 individuals. Findings showed that experiencing death of a parent in early life and having a familial predisposition to suicide were each independent risk factors for behavioral health disorders in older adulthood. But, we found no evidence of an interactive effect between the two risk factors. We also found that being male, religiosity, marriage, and higher childhood SES were protective against behavioral health disorders in older adulthood.

INTRODUCTION

Behavioral Health Disorders (BHD's) are very prevalent in older adulthood, representing a significant public health burden (Clark et al. 2009). Suicide and behavioral health disorders are highly positively correlated (Agerbo et al. 2002; McGirr et al. 2009; Schneider 2009), and tend to cluster in families (Brent and Mann 2005; Brent and Melhem 2008; Brent et al 2004). It is further known that the risks for these phenotypes are also affected by environmental exposures, particularly those occurring during the early critical stages of development; experiencing parental death in early life (PD/E) is one such factor (Jakobsen and Christiansen 2011; Niederkrotenthaler et al 2012; Tsuchiya et al 2005). This paper examines whether familial suicide predisposition moderates the effect of early-life parental death (PD/E) on BHD's in older adulthood. In other words: is there a familial x environment (F x E) interaction affecting behavioral health phenotypes in older adulthood?

This research should help (1) facilitate integration of research across traditional disciplines by examining the social within a biological context; (2) clarify whether familial susceptibility and PD/E have synergistic or antagonistic effects; (3) point toward possible demographic patterns of resilience in those who have familial susceptibilities; and (4) provide guidelines for possible personalized medical interventions and targeted public health initiatives.

BACKGROUND

Hypothesized Mechanisms

Biologically, PD/E may directly increase later-life BHD's by directly "scarring" (Preston et al. 1998: 1232) the body. Two such mechanisms predicting increased risks are attachment theory and allostatic load. Attachment theory was introduced by Bowlby (1969), and predicts that the grief experienced by a child following the death of a close parental caregiver may last

throughout life. Allostatic load (McEwen and Stellar 1993) refers to the physiological phenomenon whereby chronic exposure to such stress taxes the hypothalamopituitary-adrenal axis, disrupting regulation of certain chemicals (e.g., cortisol). Research suggests that certain chemical balances in the brain may develop differently for children who are withdrawn from parents at a critical stage of development (Beauchaine et al. 2011; Labonte and Turecki 2010).

Two hypothesized social mechanisms linking early-life stress to later health outcomes are social regulation and socioeconomic status. Rojas and Stenberg (2010) adapted Durkheim's (1951 (1897)) famous concept of social regulation to the individual level. For example, children without a parent may lack social control in the form of boundaries regarding substance abuse (Hill et al. 2000). Socioeconomic status (SES) is another possible mediating social mechanism. It has been shown that early familial disruption may result in downward social mobility for the surviving family (Biblarz and Raftery 1993). Further, such disadvantage may accumulate throughout the life-course, resulting in cumulative disadvantage (Dannefer 2003) and even greater behavioral health risk.

"Correlated environments" (Preston et al. 1998: 1232) may also indirectly link PD/E and later-life phenotypes. It is quite likely that the cause of PD/E will also directly increase the risk for BHD's outside of the aforementioned mediating pathways. For example, chronic alcohol abuse is correlated with premature mortality (Rehm et al. 2007). If both parent and child are part of a religious community that encourages alcohol abstention, this could account for an association between PD/E and BHD. Religious communities also offer network ties which can protect against premature mortality (Hummer et al. 2004) and BHD's (Pescosolido and Sharon 1989).

We hypothesize at least four different mechanisms by which familial suicide susceptibility may moderate the behavioral health risk posed by PD/E. First, familial predisposition to suicide may represent genetic polymorphisms, inherited by descent, that contribute to mental health. These polymorphisms may be necessary for PD/E to initiate a chain of events leading to certain BHD phenotypes. Such gene-environment (G x E) interactions are often referred to as "epigenetic" factors, and are believed to be necessary for a phenotype to manifest (Waddington 1953). Biologically, the stressful life event can alter the expression of DNA, forging a behavioral phenotype (Labonte and Turecki 2010; Waddington 1953). Also, familial susceptibility to suicide may capture some socialized responses to stress (i.e., patterns or traditions of coping). Perhaps families with high susceptibilities suffer a lack of healthy coping patterns in general, including poor coping to PD/E, thereby resulting in increased risks of adverse behavioral outcomes.

Third, perhaps family susceptibility is event- or age-specific. For example, high susceptibility may be due to particular vulnerability to stressful events such as diagnosis with chronic illness or widowhood. As a result, high-risk families may develop efficient kin social support patterns to attempt to cope with the risk. This may have the unintended (though salubrious) effect of facilitating healthier adaptation to PD/E in subsequent generations. Finally, it is possible that there is some "ceiling effect," whereby those with lower familial predispositions may endure the most extreme mental health problems following PD/E, simply because the higher-risk families already have excessive risk.

Review of Literature

We have not yet identified any studies examining how familiality of suicide might interact with early-life conditions to affect BHD's in older adulthood. However, there are similar

studies examining related F x E or G x E relationships. With the population of Sweden from 1952-2003, Tidemalm et al. (2011) analyzed familial clustering to estimate that suicide has both genetic and shared environmental components. With a sample of 1,889 patients for substance dependence, Roy and Janal (2005) found that family history of suicide, early life trauma and female sex were risk factors for suicide attempts, but they did not examine interacting effects among them. In a related study, Brodsky (2008) examined 507 offspring from 271 parents to show that parents experiencing early-life trauma in the form of sexual abuse had offspring more likely to exhibit aggression, a known risk factor for suicide (Gvion and Apter 2011; McGirr et al. 2009). There is also research suggesting an interaction between specific genetic variants and stressful life events may precipitate suicide attempts (Ben-Efraim et al. 2011).

METHOD

For simplicity, the individual serving as the observation in each sample is called the "ego". This is the person at risk for a BHD phenotype. The parents of this person are the "ego's parents". Given the importance of family structure to the study, pedigree data from the Utah Population Database (UPDB) were utilized. From family history records and vital records such as birth and death certificates, this database has linked together a large proportion of the population of Utah over the past few centuries. The unique scope and detail of the database, particularly in terms of enabling family linkages and the construction of familial clustering of phenotypes renders it a unique resource for this study. Additionally, Centers for Medicare and Medicaid Services (CMS) records for the state of Utah were available for the years 1992-2009. Linking these records to the family history and vital records enabled examination of BHD's for much of the Utah population aged 65 and older during these years.

Key Variables

Mean and Maximum Suicide FSIR

Familial Standardized Incidence Ratios (FSIR's), as detailed by Kerber (1995), were used to measure familiality. In this specific implementation of Kerber's method, the suicide FSIR measures the excess relative risk of completed suicide for a pedigree in comparison to controls, while weighting by expected level of genetic relatedness using a kinship coefficient. Death certificates spanning 1904-2010 in UPDB were utilized to identify completed suicides with ICD codes traditionally used in NCHS vital statistics reporting: E963, E970-E979 (ICD6-7), E950-E959 (ICD8-9), and X60-X84, Y87.0, U03 (ICD10). Additionally, a pedigree member was also coded a suicide where a manner of death of suicide was indicated on the certificate. Comparisons of the UPDB suicide counts to published Utah vital statistics since 1910, available upon request, show that the UPDB counts very closely approximate the suicide counts published in vital statistics report by year, suggesting our data can adequately measure suicide (approximately 15,000 cases) for the vast majority of the Utah population over the last century. Note that some individuals had more than one FSIR, due to the fact that the FSIR was assigned from the pedigree, and an individual may have been assigned to more than one pedigree. So, we considered both the mean and the maximum FSIR for each individual.

Early Death of Parent Experienced by Ego (PD/E)

This was a simple binary variable, measured as the recorded death of a parent before the ego reached age 18. Note this was often able to be determined with the historical data in UPDB even in the absence of a death certificate. We note here that preliminary analyses with UPDB data suggested this measure of PD/E is a strong risk factor for suicide, but we have not previously considered PD/E's effects on BHD's.

Behavioral Health Disorders (BHD's)

The term "BHD" has been utilized by previous researchers in the examination of serious behavioral and mental health phenotypes measured by ICD9 diagnosis codes in CMS data (Clark et al. 2009). We had the same ICD9 measures, and several others, available for study. The relevant ICD9 phenotypes available in the data, and for which we have obtained IRB approvals, are presented in Table 1. Codes were taken from the CMS carrier files. In this study we examined the presence or absence of a BHD with a dummy variable, where the record of any BHD claim for that individual constituted testing positive for a BHD.

Control Variables

We controlled for the ego's sex with the inclusion of a *male* dummy variable. *Ego's year of birth* was used to control for cohort effects and, to some extent, age. Ego's religiosity was measured with a record of *baptism* into the Church of Jesus Christ of Latter-day Saints by age 9—which is considered normative for children born into that faith. Controlling for *maternal and paternal age* at ego's birth attempted to approximate a number of potential confounders, including parental health during the ego's childhood, and the unexpectedness of the death based upon the parent's age in cases where PD/E occurred—i.e., deaths to older parents may be more expected, and therefore less stressful, than deaths to younger parents. We controlled for *maternal and paternal suicide*, measured as having a record of that parent committing suicide previous to the ego's death. We also considered early-life SES, measured as the greater of the mother's or father's Nam Powers score (Nam and Powers 1983) obtained from the occupation recorded on the parent's death certificate. This score ranges from 1 to 100, with higher values representing higher SES. We recoded it into six categories: four quartiles; a special category for farmers (Nam Powers score 40), as they represented a large proportion of the sample; and a category for

missing data. We also controlled for whether the *ego ever married*. Finally, due to the nature of the data we controlled for *the number of FSIR's we obtained* for each individual during FSIR construction; and for *the number of complete years they were enrolled* in Medicare parts A and B (which included a 12 month period, or a partial period if they aged in or died during the year), as this affected the at-risk period for having a BHD claim.

Sample Construction

Sample construction proceeded in several steps. First, we created a subsample of individuals for whom we had adequate FSIR data. We constructed FSIR's for a total of 5,262 pedigrees, including 219,998 distinct individuals. We required each pedigree to contain at least 1,000 members, including at least two suicides. While cross-validating these records with the CMS denominator data, we removed several individuals that appeared to have inconsistent data or were under age 65 in 2009. The end FSIR subsample then consisted of 208,860 egos, with FSIR's ranging from 1.002 to 6.527.

We then constructed a separate subsample of egos for calculating PD/E and the control variables. We considered those egos for which we knew the sex and year of birth. We also required the birth years of both parents, and adequate follow-up data for ego and parents. We further excluded any egos that were adopted or where the ego or ego's parents were involved in a polygamous relationship. Merging this subsample with the FSIR subsample yielded a new subsample of 181,567 individuals.

Next, we merged the CMS carrier claims data with the CMS denominator data. We considered any claims during the years 1992-2009 where any reported ICD9 code matched those listed in Table 1 to be indicative of a BHD. When merged with denominator data for those who were enrolled in Medicare parts A and B for the full year, we obtained another subsample of

187,337 persons. Finally, we merged all these subsamples together to obtain a final sample of N=163,295 egos.

Analytic Plan

First, we performed simple descriptive analyses of the data. Then, we performed a series of logistic regression models. Logistic regression enables unbiased testing of G x E interactions through the inclusion of interaction terms (Mukherjee et al. 2012). We extended this methodology to F x E interactions, as they are conceptually similar, though not identical, phenomena. Model 1 examined the basic associations between PD/E, FSIR and diagnosis with BHD, including controls. Model 2 added the F x E interaction, as measured by PD/E x FSIR. This two-model sequence was repeated twice—once using the mean FSIR, and once using the maximum FSIR calculated for the ego. Note the FSIR variables were centered about the mean to protect against multicollinearity that can be introduced with continuously-measured interaction terms.

FINDINGS

Table 2 displays the descriptive statistics for sample. In the interest of space, we do not discuss them in great detail here. But, note that 32.08% of this Utah sample had at least one BHD claim during the 1992-2009 study period. And, there were more females than males in the sample, which is to be expected given the sample had to survive to at least age 65, and it is well-known men have higher mortality at younger ages.

Table 3 displays the log-odds coefficients and p-values for the variables in the logistic regressions, with the mean FSIR as a predictor variable. The positively signed and statistically significant log-odds coefficient of 0.047 for PD/E (p=.004) suggests that PD/E is in fact a risk factor for BHD's in older adulthood. Further, the positively signed 0.076 coefficient for centered mean FSIR (p=.000) suggests that familial susceptibility to suicide is also a risk factor for

BHD's in older adulthood. In model 2, these values are substantially similar. But, the coefficient for their interaction is not statistically significant (0.729). Thus, we found no evidence for an F x E interaction. Table 4 shows a similar trend when using the maximum FSIR. While the 0.046 coefficient for the centered maximum FSIR in model 1 is smaller in strength, it is still statistically significant (p=.000). And, the F x E interaction term is not significant (p=.534).

Amongst the controls, being male is protective against BHD's in older adulthood, as is religiosity and having been married. Note also that lower SES as measured by the Nam Powers score appears to be a risk factor for BHD's. Interestingly, children of farmers seem less likely to have BHD's in older adulthood.

PROPOSED ADDITIONS TO THE STUDY

While we feel this draft presents important findings, it is a work in progress. In particular, we still want to address the following:

1. As the findings suggest, the experiences of males and females in terms of BHD claims are quite different. It would be informative to consider each sex separately.
2. The BHD's utilized represent a diverse array of phenotypes. Clustering them into one group of ever/never had a BHD claim is a crude first approach. It might be informative to group the BHD's into more meaningful categories, and consider each as a separate dependent variable.
3. While we control for year of birth in this study, we do not feel this controls well for age, as we cannot be certain at what age each individual entered the study period. For example, the experiences of 65-year old individuals may differ greatly from 90-year old individuals in the 1992 claims data. Given our sample size, it could be informative to better measure age.

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Table 1 - ICD9 Codes for Behavioral Health Disorder (BHD) Phenotypes

ICD9 Code	Phenotype
296.XX	Affective disorders
303.XX	Alcohol dependence
305.0X	Alcohol abuse (non-dependent)
291.XX	Alcohol-induced mental disorders
300.XX	Anxiety states
304.XX	Drug dependence
305.2X-305.9X	Drug abuse (non-dependent)
307.10, 307.50, 307.51	Eating disorders
301.XX	Personality disorders
295.XX	Schizophrenic disorders
307.4X, 327.XX, 333.94	Sleep disorders
V62.84	Suicidal Ideation
E950-E958.9	Self-harm behavior

Table 2 – Descriptive Statistics for Final Sample

Categorical Variables	**Frequency**	**Percentage**
N	163,295	100.00%
Male	76,714	46.98%
At least one Behavioral Health Disorder Claim	52,382	32.08%
At Least One Parental Death	22,193	13.59%
Baptized	115,441	70.69%
Maternal Suicide	176	0.11%
Paternal Suicide	934	0.57%
Ego Ever Married	140,166	85.84%
Nam Powers Score [a]		
76-99	22,591	13.83%
51-75	33,195	20.33%
26-50, excluding 40	21,782	13.34%
1-25	11,098	6.80%
40 (Farming)	45,236	27.70%
Missing	29,393	18.00%
Continuous Variables		
	Mean	**Std. Dev.**
Number of FSIR's Obtained	4.82	3.07
Mean FSIR	1.53	0.32
Maximum FSIR	1.92	0.58
Number of Complete Years Enrolled	9.17	5.31
Ego's Birth year	1925.18	10.64
Maternal Age	28.50	6.50
Paternal Age	32.19	7.63
a - The greater of the ego's mother's or father's Nam Powers Score obtained from parent's death certificate		

Table 3 – Logistic Regressions of Ever Having a Behavioral Health Disorder Claim, as a Function of Early Parental Death (E), Mean FSIR (F), the F x E Interaction, and Controls

N=163,295	Model 1		Model 2	
Key Variables	**Log Odds**	**P-Value**	**Log Odds**	**P-Value**
At Least One Parental Death	0.047	0.004	0.047	0.004
Mean FSIR (centered)	0.076	0.000	0.079	0.000
Parental Death x FSIR			-0.017	0.729
Controls				
Male	-0.587	0.000	-0.587	0.000
Ego's Birth year	0.010	0.000	0.010	0.000
Baptized	-0.209	0.000	-0.209	0.000
Ego Ever Married	-0.069	0.000	-0.069	0.000
Maternal Age	0.000	0.957	0.000	0.956
Paternal Age	0.000	0.737	0.000	0.736
Maternal Suicide	0.242	0.135	0.243	0.134
Paternal Suicide	0.117	0.102	0.117	0.101
Number of Complete Years Enrolled	0.098	0.000	0.098	0.000
Number of FSIR's Obtained	0.005	0.017	0.005	0.017
Nam Powers Score [a]				
51-75	0.017	0.137	0.017	0.137
26-50, excluding 40	0.042	0.002	0.042	0.002
1-25	0.062	0.001	0.062	0.001
40 (Farming)	-0.057	0.000	-0.057	0.000
Missing	-0.034	0.006	-0.034	0.006

a - The greater of the ego's mother's or father's Nam Powers Score obtained from parent's death certificate

Table 4 - Logistic Regressions of Ever Having a Behavioral Health Disorder Claim, as a Function of Early Parental Death (E), Maximum FSIR (F), the F x E Interaction, and Controls

N=163,295	Model 1		Model 2	
Key Variables	**Log Odds**	**P-Value**	**Log Odds**	**P-Value**
At Least One Parental Death	0.047	0.004	0.047	0.004
Maximum FSIR (centered)	0.046	0.000	0.049	0.000
Parental Death x FSIR			-0.017	0.534
Controls				
Male	-0.587	0.000	-0.587	0.000
Ego's Birth year	0.009	0.000	0.009	0.000
Baptized	-0.210	0.000	-0.210	0.000
Ego Ever Married	-0.069	0.000	-0.069	0.000
Maternal Age	0.000	0.969	0.000	0.963
Paternal Age	0.000	0.781	0.000	0.773
Maternal Suicide	0.240	0.138	0.242	0.136
Paternal Suicide	0.115	0.106	0.116	0.104
Number of Complete Years Enrolled	0.098	0.000	0.098	0.000
Number of FSIR's Obtained	0.002	0.368	0.002	0.374
Nam Powers Score [a]				
51-75	0.018	0.125	0.018	0.125
26-50, excluding 40	0.042	0.002	0.042	0.002
1-25	0.062	0.001	0.062	0.001
40 (Farming)	-0.057	0.000	-0.057	0.000
Missing	-0.034	0.006	-0.034	0.006

a - The greater of the ego's mother's or father's Nam Powers Score obtained from parent's death certificate