

Familial Risk Across Three Generations and Psychosocial Correlates for Developing Psychopathology in a Changing World

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Received date: January 2, 2014, Accepted date: April 08, 2014, Published date: April 15, 2014

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Abstract

Background: Human development is a highly complex, environment-dependent process including mental health and psychopathology among its outcomes. Thus, it needs to be studied comprehensively in order to identify correlations and interactions among different biological, economical and psychosocial variables across time and generations.

Objective: Is familial psychopathology risk across three generations significantly associated with ongoing psychosocial variables reported by probands?

Methods: The study was designed as a household survey on a representative sample of the adult population aged 18 - 65 years in Mexico City. The family-history method was used to obtain information about psychiatric antecedents on their parents and a validated screening instrument was used to evaluate caseness in their offspring. Lifetime prevalence of psychiatric disorders on probands was evaluated using the Composite International Diagnostic Interview. Morbid risk in terms of the odds ratio and prevalence ratio were calculated using logistic regression with fixed effects as well as with generalized estimating equations (GEE) population-averaged models with an exchangeable structure. Additionally, the population attributable risk percent was also calculated for selected variables associated with the outcome in the complete models.

Results: The strength of the association of familial psychiatric antecedents is moderate between generations in either direction: backwards in regards of proband's parents or downwards in regards of their children. However, when interaction between the two previous generations was considered, the strength of the association was higher, crude OR=7.8, and showing significant variations when controlling for the effect of the variables and especially for probands' psychosocial correlates. The population attributable risk percent for these variables across generations is sometimes higher than the obtained for familial risk.

Conclusions: Findings suggest that besides psychiatric familial risk, socioeconomic and psychodynamic risk factors have been interacting across time and generations contributing to the high prevalence of psychiatric disorders recently reported on youth Mexican population.

Keywords: Socioeconomic; Family stress; Psychopathology; Three generation study; Cultural characteristics; Diathesis-stress; Childhood; Adolescence; G x E; CBT: Cognitive-behavior therapy for depressed.

Introduction

All common psychiatric disorders where a genetic basis is suspected correspond to the so called "complex disorders". These are the result of the interaction between genetic liability and environmental factors. Both epidemiologist and genetic scientists are interested in these disorders but there is a need for a common methodology. Intergenerational studies have become of special interest, as heritable risk and early childhood biological markers of emotional disorder may be passed across consecutive generations (e.g., temperamental variables, elevated cortisol levels) [1, 2]. In a like manner, psychosocial characteristics that increase the risk of emotional and conduct disturbances may be passed from parent to child via processes such as modeling and direct communications [3]. To the extent that these

genetic, biological, and psychosocial characteristics— and their attendant risk—are transmitted from one generation to the next, an intergenerational mediation model may best characterize the development of psychopathology in general as well as for specific disorders (i.e., G1, G2, and G3).

The continuity of mental health and adjustment problems is not limited to two generations as it has been documented to occur at least across three generations [3-11]. However, a major unanswered question is whether the relationship between multiple mental health problems across generations is a result of the continuity of underlying problems that are genetically transmitted across generations, or is the result of gene-environmental interactions [12].

Human development is a highly complex, environment-dependent process including mental health and psychopathology among its outcomes. Cicchetti [13] signaled that in order to develop a thorough and comprehensive understanding of maladaptive and adaptive functioning, it is important that developmental scientists increasingly

incorporate multiple levels of analysis and multiple domains into their research investigations. Gene-environment correlations (rGE) concern genetic influences on individual variations in people's exposure to particular sorts of environments. There are several types of rGE that play a substantial role in influencing environmental risk exposure, but their impact is best understood through the effects of parent and child behaviors in shaping and selecting environments [14]. Studies exploring the role of family social economic status (SES) as a moderator of genetic and environmental influences on general cognitive ability have provided evidence that socioeconomic circumstances differentially impact the heritability of cognitive abilities [15]. Furthermore, as SES is correlated with differences in life stress and family resources (e.g. fewer financial resources increases parental stress, negatively affecting child development through its effects on parenting behavior), Hackman et al [16] suggested that prenatal factors, parent-offspring interactions and cognitive stimulation partly underlie the effects of SES, corroborating the hypothesis of the family stress model, whereby economic disadvantage affects children's well-being through its effects on the parent. Family income and other measures of socioeconomic status (SES) are public health indicators closely related to health in general as well as with developmental outcomes in children [17]. Differences in families across the continuum of SES may create different experiences of stress and environmental complexity, potentially affecting neuropsychological development, cognition and mental health as a whole. Stress and environmental conditions are two primary experiential influences on brain development as well for psychological development. For example, a recent report found a direct negative effect of financial resources on grey matter cerebral volume and total cerebral volume [18].

As most of the studies published are based on Caucasian populations and in developed countries, there is the need for data from studies in other countries with different ethnic, cultural and socio-economic conditions. A distinguishing characteristic of the developing world is the fast pace of social change from one generation to the next in terms of economic conditions, communication, globalization, changing gender roles, secularization, weakening family ties, improvements in educational attainment levels but there are still unfavorable economic conditions for many, all of which may have a profound effect upon exposure to childhood adversities [19]. A recent study on Mexican youth population [20] found that almost 40% of adolescents reported a 12-month disorder, and the difference between these results and the 25% median prevalence estimate in developed regions was discussed in terms of the accelerated rate of social change and social adversity.

Several countries have experienced notable economical adversities with potential impact for the mental health status on their populations in recent years. The goal of the present paper is to bring out information that may contribute to a better understanding of how socioeconomic changes correlate with mental health outcomes in populations experiencing rapid economical adjustments, using data from a sample collected among the Mexican population during a year of significant social and economic turmoil.

The adoption of the neoliberal economic model in Mexico in the early 1990s induced several social adjustments and disparities. Six months before the field-work of this study began, in 1995, the greatest Mexican financial crisis occurred. The peso-to-dollar exchange rate increased from 3.49 to 9.42 pesos per dollar, with an inflation rate of 225% and significant reduction of actual acquisitive power of 35% [21].

Hence, the objective for this paper is to answer the following questions:

Are socio-demographic characteristics such as gender, age, household income and proband's labor status associated with the development of psychopathology between and across generations?

Are probands' psychosocial characteristics associated with the development of psychopathology between and across generations?

Is familial psychopathology risk across three generations significantly associated with ongoing psychosocial variables reported by probands?

Materials and Methods

The study was designed as a household survey on a representative sample of the adult population aged 18 - 65 years in Mexico City [22]. Briefly, a standardized assessment for adults' lifetime prevalence of psychiatric disorders was obtained via an amended version of the Composite International Diagnostic Interview, CIDI. 1.1. The ICD-10 diagnostic categories included were: Anxiety disorders (i.e., agoraphobia, social phobia, specific phobias, generalized anxiety, panic and obsessive-compulsive disorder), Affective disorders (i.e., depressive episodes, dysthymia, hypomania and mania), as well as Substance Use disorders (including alcohol, sedatives, tranquilizers, stimulants, analgesics, inhalants, marijuana, cocaine, hallucinogens and heroin).

Response rate was 60.4%. The total sample size was 1932 adult subjects (probands, Generation 2). In addition to the CIDI, all respondents provided information on the psychiatric history of their parents (Generation 1) about anxiety, affective and substance-use disorders following the Family-history research criteria [23-25]. Also, 925 respondents with children aged 4-16 years living in the same household were interviewed (providing data about 1686 children and adolescents, Generation 3) using a standardized screening questionnaire for assessing psychopathology, the Brief Screening and Diagnostic Questionnaire (CBTD for its initials in Spanish). The CBTD is a 27-item questionnaire answered by the parents of the child, which explore symptoms frequently reported as motives for seeking attention at the outpatient mental health services. Presence of the symptom requires that each item has to be reported as "frequently" presented. The internal consistency of the questionnaire showed a Cronbach's alpha of 0.81, range: 0.76 to 0.85 (26). Diagnostic algorithms in order to define probable DSM-IV disorders in children were created based on data from this epidemiological study [27].

For children and adolescents, caseness was defined based on the questionnaire score for those at the 9th decil and with 5 or more symptoms [26]. Concurrent validity with any DSM-IV diagnosis using the E-MiniKid standardized interview showed a positive predictive value of 88% (95% [confidence interval] CI: 83.7%, 91.5%) and the Area under the Curve (AUC) obtained by Receiver Operating Characteristic Curves (ROC) analysis was 0.78 (95% CI: 75%, 81%) [28].

To estimate the familial morbid risk for children and adolescents, interaction of familial psychopathology across generations was defined as follows: Psychiatric history only in grandparents (G1); psychiatric history only in proband (mother or father, G2); psychiatric history on both previous generations (G1 and G2).

Analyses were carried out controlling for the effect of different potential confounding variables. The first group included: proband's gender and age, children's gender and age, proband's labor status, and household income. The estimated household income was divided into 5 levels: 26.04% were at the bottom, 27.35%, 26.09%, 16.94% in subsequent levels, and only 3.6% at the top. The second group included the following proband's psychosocial variables: relationship with spouse, stress over work, couple's total work hours per week, worries between home and work, perception of family support (not living at the same household), perception of community support, own health's perception, and social isolation. On these, the survey included specific sections with questions and scales that were used by Dr. Kessler at the first National Co-morbidity Study and described elsewhere [29]. Scores, arranged so that a higher punctuation indicated more adversity, were converted into dummy variables using quartiles.

Analyses were done using the Stata 12.0 program. Variance and confidence interval estimation accounted for the complex stratified sampling of the survey, as well as for the clustering by family units that has been extensively described elsewhere [7, 29].

Morbid risk in terms of the odds ratio were calculated using logistic regression with fixed effects as well as with generalized estimating equations (GEE) population-averaged models with an exchangeable structure. GEE is an iterative procedure, using quasi-likelihood to estimate the regression coefficients; the relationships between the variables of the model at different time-points are analyzed simultaneously. Because the repeated observations within one subject are not independent of each other, a correction must taken into account for these within-subject correlations choosing a correlation structure. In an exchangeable structure, as used in this study, the correlations between subsequent measurements are assumed to be the same, irrespective of the length of the time interval [30].

As a multi-nominal sampling was used, and the odds ratios are always an over-estimation of the real population's relative risk, GEE population-averaged analysis with Poisson regression was performed based on the results of the most complete previous models (31) in order to obtain prevalence ratios. Also, as the data were representative of the population and a wide comprehensive approach of psychosocial variables was included, the population attributable risk percent was also obtained for selected variables from the complete models using the following equation:

$$PAR\% = \frac{(Pe) (RR - 1)}{1 + (Pe) (RR - 1)} \times 100$$

Where Pe is the exponentiated prevalence prediction obtained from the model. The relative risk, RR, is the ratio in the exposed population to the risk in the unexposed population. The population attributable risk percent indicates the percentage of total risk that is due to significant variables associated with the outcome [32].

Results:

Characteristics of the sample are shown on Table 1. There were 1932 probands (G2), 45.1% of whom were males; overall, 28.6% met caseness criteria. Proband's reported data on 2743 of their parents (G1), 48.8% corresponding to males; and 23.6% meeting caseness criteria. There were 925 probands who had children 4-16 years old, for a total of 1686 children and adolescent data reported (G3), 51.3% of whom were males; and with 16% of G3 meeting caseness criteria for a mental problem. Proband's mean age was 34.8 years old, 55% had one offspring, 30.5% had two, 11.3% had three, and 3.2% had up to six offspring. Children mean age was 9.7 years; and the distribution by age groups was as follows: 4-5 years old 16.3%, 6-8 years old 25.5%, 9-12 years old 30.9%, and 13-16 years old 27.4%.

	Males	%	Females	%	Total	Cases	%	Non cases	%
Proband's (Gen 2)	871	45.1	1061	54.9	1932	552	28.6	1380	71.4
Grandparents (Gen 1)	1339	48.8	1404	51.2	2743	647	23.6	2096	76.4
Proband's (Gen 2) with offspring aged 4 to 16 years	381	41.2	544	58.8	925	292	31.6	633	68.4
Children and adolescents (Gen 3)	865	51.3	821	48.7	1686	269	16	1416	84

Table 1: Sample Characteristics

The results on Table 2 show that the strength of the association between any psychiatric antecedents in the previous generation (G1) and the presence of psychopathology on probands (G2) did not vary considerably across models with progressive inclusion of covariates. Females were less likely to report any lifetime psychopathology. Proband's on-going correlates significantly associated with psychopathology were tension over work, perceiving less support from family (not living at the same household) as well as poorer perception of own's health.

	Model 1		Model 2		Model 3	
	OR	p	OR	P	OR	p
Stress over work						

	95% CI		95% CI		95% CI	
Non cases	1		1		1	
Any familial psychiatric antecedents	2.2	<0.001	2.5	<0.001	2	<0.001
	1.6, 3.2		1.8, 3.6		1.4, 2.7	
Female			0.5	<0.001	0.3	<0.001
			0.3, 0.6		0.2, 0.5	
Stress over work					1.7	0.019
					1.1, 2.6	

Perception of support by non-nuclear family				1.5	<0.001
				1.2, 1.7	
Perception of health status				1.4	<0.001
				1.2, 1.7	
No. observations	1682		1653	1551	
F	21.23		9.27	10.47	
GI	1		4	12	
P	0.0001		0.0001	0.0001	
Adjusted for possible confounding variables **	None		a	a, b	

Table 2: Association* between familial psychiatric antecedents (Gen 1) and proband's psychopathology (GEN 2) and psychosocial correlates.

*Logistic Regression with fixed effects ("classic").

** a) proband's gender and age, proband's labour status, and household income; b) proband's psycho-social variables: relationship with spouse, stress at work, couple's total work hours per week, worries between home and work, perception of family support (not living at the same household), perception of community support, own health's perception, and social isolation.

Table 3 conveys the odds ratio across different models indicating the strength of the association between probands' psychiatric disorders and psychopathology in their offspring. Here, the estimated association was higher than the crude Odds Ratio when controlling for demographic variables, but it was practically the same when further controlling for probands' psychosocial characteristics. Notice that main effects rely on psychiatric familial antecedents as well as in female gender. Among probands' psychosocial variables, poorer perception of community support as well as from the non-nuclear family and worse perception of owns health were significantly associated with report of psychopathology in their children.

	Model 1		Model 2		Model 3	
	OR	p	OR	p	OR	p
	95% CI		95% CI		95% CI	
Non cases	1		1		1	
Proband's psychopathology	2.7	0.001	3.5	<0.001	2.7	<0.001
	2.0, 3.7		2.5, 4.9		1.9, 3.9	
Mother			2.7	<0.001	2.2	<0.001
			1.8, 4.0		1.5, 3.3	
Daughter			0.7	0.005	0.6	0.002
			0.5, 0.9		0.5, 0.8	
Children's Age			1.2	0.015	1.2	0.033

			1.0, 1.3		1.0, 1.3	
Perception of support by non-nuclear family					1.2	0.014
					1.1, 1.5	
Perception of community support					1.2	0.034
					1.0, 1.4	
Perception of health status					1.2	0.035
					1.0, 1.4	
Tension over work					1.2	0.452
					0.8, 1.7	
No. observations	1682		1652		1553	
X2	42.29		84.4		97.12	
GI	1		7		14	
p	<0.001		<0.001		<0.001	
Adjusted for possible confounding variables **	None		a		a, b	

Table 3: Association* between psychopathology on GEN 3 and proband's psychiatric antecedents (Gen 2) and psychosocial correlates

*Logistic Regression with generalized estimated equations

** a) proband's gender and age, children's gender and age, proband's labour status, and household income; b) proband's psycho-social variables: relationship with spouse, stress at work, couple's total work hours per week, worries between home and work, perception of family support (not living at the same household), perception of community support, and own health's perception.

The strength of the association between psychopathology in the offspring (G3) and the interaction with familial psychopathology across generations, controlling for the effect of socio-demographic variables as well as for on-going psychosocial correlates at the time of the study, are shown in Table 4. The odds ratio were higher when there were psychiatric familial antecedents on both previous generations and controlling for sociodemographic variables. When proband's psychosocial correlates were further adjusted for, the odds ratio diminished notably, although it increased for G1 and especially for G2. Proband's gender was associated with psychopathology in the offspring on the second model, but as other variables were incorporated to the analysis, the observed significant association vanished. The estimated household income that was inversely associated (Model 3, p=0.039) became no longer significant (Model 4, p= 0.234) Psychosocial correlates significantly associated with the report of psychopathology in the offspring indicated that proband's tension over work as well as less perceived support from family (not living at the same household) were important variables in the process.

Antecedents	Model 1	Model 2	Model 3	Model 4
	OR (95% CI)	OR (95% CI)	OR (95% CI)	OR (95% CI)

G 1	2.9 (1.5, 5.8)	2.7 (1.4, 5.4)	2.9 (1.4, 5.9)	3.2 (1.5, 6.5)
G 2	2.4 (0.9, 6.0)	2.6 (1.0, 6.6)	2.8 (1.1, 7.3)	3.5 (1.3, 9.2)
G 1 and G 2	7.8 (3.9, 15.4)	8.7 (4.3, 17.4)	9.5 (4.6, 19.3)	6.6 (2.8, 15.5)
Mother		2.2 (1.2, 4.0)	1.9 (0.9, 3.7)	1.6 (0.8, 3.5)
Household income			0.8 (0.6, 1.0)	0.8 (0.6, 1.1)
Tension over work				2.2 (1.2, 4.2)
Perception of support by non-nuclear family				1.4 (1.1, 2.0)
Worries between home and work				0.5 (0.3, 0.9)
Observations	739	739	728	699
Groups.	419	419	413	397
X2	35.26	40.29	44.04	73.48
gl.	3	7	9	16
P	<0.001	<0.001	<0.001	<0.001
Adjusted for confounding variables *	None	a, b, c, d	a, b, c, d, e, f	a, b, c, d, e, f, psychosocial**

Table 4: Association* between psychopathology on Gen 3 and antecedents of psychiatric disorders on previous generations and psychosocial correlates.

**Logistic Regression with generalized estimated equations

a) proband's sex; b) children's sex; c) children's age; d) proband's age; e) socio-economic level; f) proband's work status;** proband's psychosocial variables: relationship with spouse, stress over work, couple's total work hours per week, worries between home and work, perception of family support (not living at the same household), perception of community support, own health's perception, and social isolation.

Familial risk for developing psychopathology between and across generations in terms of the prevalence ratios and population attributable risk percent for each of the significant variables associated with the outcome on previous analyses, are presented on Table 5. Between probands and their parents, one fifth of the total risk was attributable to psychopathology in the previous generation. However, psychosocial variables had considerably high attributable risks, specially for tension over work and perceiving less support from non-nuclear family, which exceeded the population attributable risk of familial psychopathology in the highest quartiles (top of Table 5).

Complementing results, the prevalence ratio of any psychiatric disorder in the probands for developing psychopathology in their offspring (middle Table 5) was slightly higher than in the previous analysis, although the population attributable risk percent was also slightly lower. Notably, perceiving less support from family (not living at the same household) contributed up to 64%in the worst situations.

Tension over work was not significantly associated with the outcome and its contribution in the worst cases was similar to the percent obtained for the lower population attributable risk percent between G1 and G2.

Finally, familial risk for developing psychopathology across generations was higher on the third one when there were psychiatric antecedents on both previous generations explaining 36% of the population attributable risk. The prevalence ratios of psychosocial correlates - tension over work and less perceived support from family - not living at the same household- were both found to be highly contributing as shown by the population attributable risk percent. Less worries between home and work showed a relatively little protective effect (bottom Table 5).

Generations/Variables	Prevalence ratio	Population attributable risk %
	95% CI	
In G2 by G1		
Any psychiatric disorder	1.6 (1.2, 2.0)	20.8
Females	0.5 (0.4, 0.6)	-14.6
Tension over work	1.3 (1.1, 1.5)	15.4 to 34.4*
Perception of support by non-nuclear family	1.3 (1.1, 1.4)	11.4 to 26.2*
Perception of health status	1.2 (1.1, 1.4)	9.5 to 18.7*
In G3 by G2		
Any psychiatric disorder	2.1 (1.6, 2.8)	17.1
Mother	1.8 (1.3, 2.5)	12.9
Female children	0.7 (0.5, 0.8)	-4.6
Children's age	1.1 (1.0, 1.3)	2.5 to 7.1**
Perception of support by non-nuclear family	1.2 (1.0, 1.4)	4.3 to 64.0*
Perception of community support	1.1 (1.0, 1.3)	1.3 to 12.2*
Perception of health status	1.1 (1.0, 1.3)	8.2 to 18.4*
Tension over work	1.2 (1.0, 1.5)	2.7 to 17.3*
In G3 across generations		
Only in grandparents	2.7 (1.4, 5.0)	18.4
Only in father or mother	2.9 (1.3, 6.6)	15.4
On both previous	4.6 (2.3, 9.4)	36
Tension over work	1.7 (1.1, 2.8)	1.8 to 44.0*
Perception of support by non-nuclear family	1.4 (1.1, 1.7)	4.2 to 28.3*
Worries between home and work	0.6 (0.4, 0.9)	-1.9 to -4.3

Table 5: Prevalence ratio and population attributable risk for psychopathology between and across generations.

* Range by quartiles: referent first quartile. **Range by age-groups: referent 4-5 years-old; subsequent: 6-8; 9-12; 13-16 years-old.

Discussion:

Results from this epidemiological study in the general population of Mexico City have shown that, in terms of the odds ratios controlling for the effect of socio-demographic and on-going proband's psychosocial variables, the strength of the association of familial psychiatric antecedents is moderate on either direction: backwards in regards of proband's parents, OR=2.0, or downwards in regards of their children, OR=2.7. The strength of the association is similar to the results obtained for specific disorders between members of two generations in the general population of the United States [25]. However, when interaction between the two previous generations was considered, the strength of the association was higher, crude OR=7.8, and showing significant variations when controlling for the effect of the rest of the variables and especially for probands' psychosocial correlates.

On the first specific question for this report, do socio-demographic variables such as gender, age, household income and proband's labor status play a role for the development of psychopathology between and across generations? In summary, results indicated that between G1 and G2 proband's female gender was the only socio-demographic variable that was inversely associated with developing psychopathology. However, being a female offspring of a G2 case was observed to be associated with almost two-fold higher odds of developing psychopathology, while female gender in G3 was inversely associated with being a probable case. Also children's age was positively associated with developing psychopathology. In sum, when socio-demographic variables were incorporated to the analyses only female gender and children's age were found significantly associated with developing psychopathology between generations. The odds ratio indicating the strength of the association between the psychiatric familial antecedents on the previous generation as related to the next, were only slightly higher between parent's, G2, and their offspring, G3, when these variables were taken into account. Across generations these variables seemed to have a stronger association only when there are psychiatric familial antecedents on both previous generations.

Mother's figure is very important in familial relationships, particularly in Latino cultures. An intergenerational study [6] have presented very interesting data on how major depressive disorder (MDD) in grandmothers had an effect on grandchildren by maternal, G2, chronic interpersonal stress. In the Mexican culture, most grandmothers help their descendants in nurturing and raising grandchildren, especially if both parents have to work, or when their offspring are single parents.

On the second question, does proband's psychosocial variables play a role for the development of probable psychopathology between and across generations? The results from this study have shown that between and across generations the most consistent variable associated with the outcome is the perception of less support from family (not living at the same household).

On one hand, perception of available support from family and from other persons in the social networks has been identified as an important factor helping people cope with their problems, although studies have not confirmed that support actually exists; consequently, the answers to these questions have been considered as subjective and thus related to subjects' personality [33]. However, effects of environmental risks are mediated by individual vulnerabilities that are expressed on personality traits [34], and as related to the contents of the questionnaire used (see appendix) this variable could indicate

personality dispositions where individuals experience themselves as helpless, distrustful and misunderstood. On other hand, as commented by Benjet et al. [19], "Mexican culture is a distinct family systems organization in which families tend to be more cohesive, extended, interdependent, and group-oriented, a cultural value sometimes called familism or familialism which has both potentially positive and negative consequences, the positive being that families provide greater social support and collective resources, and the negative being that the problems of one of its members are shared by all and can lead to greater caretaker burden among family members". Anyhow, this variable has both an implicit psychodynamic nature as well as a socio-cultural value that should be contemplated more carefully and in-depth.

The other variable significantly associated with the outcome between, although not across generations, was having a poor perception of own's health. This is important and closely related to cultural values, as previously discussed, although not exclusive of it. If own's health is weak, caring for others is difficult, and thus feeling sick can be a source of stress by itself, a manifestation of the caretaker burden, or both. Health status is one of the six indicators proposed by Blank [35] for the operationalization of SES using multiple components.

In addition, tension over work emerged as a very interesting socio-economic variable, significantly associated with the outcome between G1 and G2 and across generations, but not between G2 and G3. This finding suggests that there may be a moderator effect [36]. A plausible explanation resides on the family systems organization, previously mentioned and the accumulated burden of economic responsibilities as inflation during the 80s heavily eroded pensions and acquisitive power [37].

Anyway, it is important to note that SES in terms of household income in the final model was not significantly associated with the outcome; rather it was tension over work. On this, decades ago, studies suggested a complex set of intervening variables between economic life change and symptoms in workers employed in two closing industrial facilities. Findings suggested that by the time a discharged worker actually leaves a job, he or she may already have paid the price in symptoms due to adaptations in self-esteem and standard of living as well as to the stress of job seeking [38].

Finally, does familial psychopathology risk across three generations is significantly influenced by ongoing psychosocial variables reported by probands? Morbid risk measures presented in terms of prevalence ratios showed that between G1 and G2 the morbid risk was 1.6 (95% CI: 1.3, 2.1), between G2 and G3 was 2.1 (95%CI: 1.6, 2.8) and notably increased across generations to become G1= 2.7 (95% CI: 1.4, 5.0), G2 = 2.9 (95% CI: 1.3, 6.6) and for G1 and G2 = 4,6 (95% CI: 2.3, 9.4). The increased morbid risk on G1 alone may be that, as already mentioned, grandparents are expected to help their descendants in nurturing and raising grandchildren especially if both parents have to work, or when their offspring are single parents, so that the effect on grandchildren may be the result of interpersonal stress between parental and grandparental figures or directly between grandparents and grandchildren [6].

Among the general population the PAR% of any psychiatric disorder between generations was practically the same, 20.8% and 17.1%, respectively, across generations. When there were psychiatric antecedents on both G1 and G2, the PAR% was practically twofold,

36.0%, as compared to just having psychopathological antecedents on either G1 or G2, thus suggesting a possible genetic component.

The PAR% of perceiving less support by non-nuclear family of G2 as related to G1 was between 11.4% and 26.2%, varied considerably and increased in relation to G3, from 4.3% to 64%, maybe indicating the urgency of helping demand that the economic crisis imposed. When interaction across generations was considered the PAR% of perceiving less support by non-nuclear family was between 4.2% and 28.3%, thus suggesting that most of the effect was already expressed as related to G1.

Among the general population the PAR% of proband's tension over work as related to G1 was between 15.4% and 34.4%, and from 2.7% to 17.3% as related to G3, and when interaction across generations was considered the PAR% extended between 1.8% and 44%. These results suggest that most of the burden and worries pending on the adult population may have been already present at the time of the study and related to the parents rather than to the offspring, and the economical crisis increased it. The relevance of tension over work can be better understood by considering that the contribution of this variable to the mental health status of children and adolescents exceeded the population PAR% of having psychopathological antecedents on G1 and G2.

Consistent with our observation, on the impact of SES on child development, McLoyd's [17] posited "that these problems do not necessarily arise directly from low SES but from the impact it has on the parents of the children, which in turn, influences the socio-emotional development of the children". She also pointed out that "studies suggest that poverty and economic stress elevate socio-emotional problems in children partly by increasing parents' tendency to discipline children in a punitive and inconsistent manner and to ignore children's dependency needs," a finding we have also corroborated in primary care services [39]. "Likewise, overwhelming evidence exists that these parenting behaviors stem partly from an overabundance of negative life events and conditions that confront poor adults. Moreover, persistent poverty, not transitory poverty, is consistently associated with more harmful effects on children's socio-emotional functioning" [17].

Limitations of the present study include the fact that only one adult per household was selected, and so the present analyses of familial risk across generations is lacking on data about one parent in two-parent households. However, the sample was representative of the adult population aged 18 to 64 years, and morbid risk was calculated using GEE averaged-population models, in which the interest is the population and not the individual's risk.

Family history method has shown to be highly specific although with a moderate sensitivity as compared to personal interviews in validity studies. Thus, results tend to attenuate rather than exaggerate familial aggregation [23,24]. Predictive validity of both methods has been demonstrated and it has been concluded that reports based on the family history method could be seen as another assessment tool for familial psychiatric disorders, having potential utilities as well as important limitations, rather than a poor substitute of personal interviews [25].

Information about children and adolescents was obtained from the parent and not by direct interviews so that reliability and validity could be an issue. On this, it has been recommended that all epidemiological information should be considered as "informant's specific" [40, 41], although on a long-term follow-up study Hofstra et al. [42] found a

considerable stability on parents' reports in a period that extended for 14 years.

Although the study was only based on adult probands information it has the advantage of minimal bias as related to help-seeking, reference and use of services.

Notwithstanding these limitations, our results have shown that among the Mexican population psycho-social variables, mainly stress over work and an adverse perception of family support, are associated with developing psychopathology across generations. The first is a socio-economical factor and the second a psychodynamic factor, both possibly interacting across time with psychiatric familial risk for the outcome. It is our hope that these results may help explain and understand the high complexity of mental health problems in populations exposed to heavy economical changes and the need for developing policies and programs in accordance to human needs.

Acknowledgments

This study was funded by The National Council of Science and Technology (CONACYT), award 2077-H9302. Dr. Fernando A. Wagner offered valuable suggestions to a later version of this manuscript. To the Editor for the invitation to prepare this manuscript and to Reviewers for their useful comments.

References

1. Ashman SB, Dawson G, Panagiotides H, Yamada E, Wilkinson CW (2002) Stress hormone levels of children of depressed mothers. *Dev Psychopathol* 14: 333-349.
2. Goldsmith HH, Buss KA, Lemery KS (1997) Toddler and childhood temperament: expanded content, stronger genetic evidence, new evidence for the importance of environment. *Dev Psychol* 33: 891-905.
3. Pettit J, Olin T, Robert E, Seeley J, Lewinsohn P (2008) Intergenerational Transmission of Internalizing Problems: Effects of Parental and Grandparental Major Depressive Disorder on Child Behavior. *J Clin Child Adol Psychol* 37: 640-650.
4. Brook JS, Whiteman M, Zheng L (2002) Intergenerational transmission of risks for problem behavior. *J Abnorm Child Psychol* 30: 65-76.
5. Thornberry TP, Freeman-Gallant A, Lizotte AJ, Krohn MD, Smith CA (2003) Linked lives: the intergenerational transmission of antisocial behavior. *J Abnorm Child Psychol* 31: 171-184.
6. Hammen C, Shih JH, Brennan PA (2004) Intergenerational transmission of depression: test of an interpersonal stress model in a community sample. *J Consult Clin Psychol* 72: 511-522.
7. Caraveo-Anduaga JJ, Nicolini-Sánchez H, Villa-Romero A, Wagner FA (2005) [Psychopathology across three family generations: an epidemiological study in Mexico City]. *Salud Publica Mex* 47: 23-29.
8. Weissman MM, Wickramaratne P, Nomura Y, Warner V, Verdelli H, et al. (2005) Families at high and low risk for depression: a 3-generation study. *Arch Gen Psychiatry* 62: 29-36.
9. Warner V, Wickramaratne P, Weissman MM (2008) The role of fear and anxiety in the familial risk for major depression: a three-generation study. *Psychol Med* 38: 1543-1556.
10. Caraveo-Anduaga J (2011) Intergeneration familial risk and psychosocial correlates for anxiety syndromes in children and adolescents in a developing country. Ágnes Szirmai (edn.), *Anxiety and related disorders*. INTECH open Access Publisher 49-68.
11. Leventhal AM, Pettit JW, Lewinsohn PM (2011) Familial influence of substance use disorder on emotional disorder across three generations. *Psychiatry Res* 185: 402-407.
12. Taylor A, Kim-Cohen J (2007) Meta-analysis of gene-environment interactions in developmental psychopathology. *Dev Psychopathol* 19: 1029-1037.

13. Cicchetti D (2002) The impact of social experience on neurobiological systems: illustration from a constructivist view of child maltreatment. *Cognitive Development* 17: 1407-1428.
14. Rutter M, Moffitt TE, Caspi A (2006) Gene-environment interplay and psychopathology: multiple varieties but real effects. *J Child Psychol Psychiatry* 47: 226-261.
15. Hart SA, Soden B, Johnson W, Schatschneider C, Taylor J (2013) Expanding the environment: gene \times school-level SES interaction on reading comprehension. *J Child Psychol Psychiatry* 54: 1047-1055.
16. Hackman DA, Farah MJ, Meaney MJ (2010) Socioeconomic status and the brain: mechanistic insights from human and animal research. *Nat Rev Neurosci* 11: 651-659.
17. McLoyd VC (1998) Socioeconomic disadvantage and child development. *Am Psychol* 53: 185-204.
18. Marcus Jenkins JV, Woolley DP2, Hooper SR3, De Bellis MD (2013) Direct and Indirect Effects of Brain Volume, Socioeconomic Status and Family Stress on Child IQ. *J Child Adolesc Behav* 1.
19. Benjet C, Borges G, Medina-Mora ME, Zambrano J, Cruz C, et al. (2009) Descriptive epidemiology of chronic childhood adversity in Mexican adolescents. *J Adolesc Health* 45: 483-489.
20. Benjet C, Borges G, Medina-Mora ME, Zambrano J, Aguilar-Gaxiola S (2009) Youth mental health in a populous city of the developing world: results from the Mexican Adolescent Mental Health Survey. *J Child Psychol Psychiatry* 50: 386-395.
21. Fernández-Torres J E (2005) La crisis financiera de 1994-1995 y el TLCAN a diez años.
22. Caraveo J, Colmenares B, Saldívar H (1999) Morbilidad psiquiátrica en la Ciudad de México: prevalencia y comorbilidad en la vida. *Salud Mental* 22 :62-67.
23. Andreasen NC, Endicott J, Spitzer RL, Winokur G (1977) The family history method using diagnostic criteria. Reliability and validity. *Arch Gen Psychiatry* 34: 1229-1235.
24. Andreasen NC, Rice J, Endicott J, Reich T, Coryell W (1986) The family history approach to diagnosis. How useful is it? *Arch Gen Psychiatry* 43: 421-429.
25. Kendler KS, Davis CG, Kessler RC (1997) The familial aggregation of common psychiatric and substance use disorders in the National Comorbidity Survey: a family history study. *Br J Psychiatry* 170: 541-548.
26. Caraveo-Anduaga J (2006) Cuestionario Breve de tamizaje y diagnóstico de problemas de salud mental en niños y adolescentes, CBT-D: confiabilidad, estandarización y validez de construcción. *Salud Mental*. 29: 65-72.
27. Caraveo-Anduaga J (2007) Cuestionario Breve de Tamizaje y Diagnóstico de problemas de salud mental en niños y adolescentes: algoritmos para síndromes y su prevalencia en la Ciudad de México. *Salud Mental* 30: 48-55
28. Caraveo-Anduaga JJ, López-Jiménez JL, Soriano-Rodríguez A, López-Hernández JC, Contreras-Garza A, et al. (2011) [Concurrent validity and efficiency of the CBT-D for the surveillance of mental health on children and adolescents at a primary health care center in Mexico]. *Rev Invest Clin* 63: 590-600.
29. Caraveo J, Martínez N, Rivera E (1998) Un modelo para estudios epidemiológicos sobre la salud mental y la morbilidad psiquiátrica. *Salud Mental*. 21: 48-57
30. Twisk J W R (2003) *Applied longitudinal data analysis for epidemiology*. Cambridge University Press, Cambridge, UK
31. McNutt LA, Wu C, Xue X, Hafner JP (2003) Estimating the relative risk in cohort studies and clinical trials of common outcomes. *Am J Epidemiol* 157: 940-943.
32. Jekel JF, Elmore JG, Katz DL (1996) *Epidemiology, biostatistics, and preventive medicine*. W.B. Saunders Company, Philadelphia, USA
33. Kessler RC, House JS, Williams DR, Anspach RR (1994) Social psychology and health. In: Cook K, Fine G, House JS (edn.). *Sociological perspectives on social psychology*. Neeham Heights, MA: Allyn & Bacon. 548-570
34. Paris J (1999) *Nature and nurture in psychiatry. A predisposition-stress model of mental disorders*. American Psychiatric Press, Washington D.C.
35. Blank RM (2008) Presidential address: How to improve poverty measurement in the United States. *J Policy Anal Manage* 27: 233-254.
36. Bradley RH, Corwyn RF (2002) Socioeconomic status and child development. *Annu Rev Psychol* 53: 371-399.
37. Lustig NC and Székely M (1997) México: Evolución económica, pobreza y desigualdad.
38. Catalano R, Dooley CD (1977) Economic predictors of depressed mood and stressful life events in a metropolitan community. *J Health Soc Behav* 18: 292-307.
39. Caraveo-Anduaga J, Soriano RA, Erazo PJ (2013) Anxiety syndromes and their correlates in children and adolescents: A two-year-follow-up study at primary health care in Mexico City. In: Durban F (edn.). *New insights into anxiety disorders*. INTECH open Access Publisher, 233-258.
40. Boyle MH, Offord DR, Racine Y, Szatmari P, Fleming JE, et al. (1996) Identifying thresholds for classifying childhood psychiatric disorder: issues and prospects. *J Am Acad Child Adolesc Psychiatry* 35: 1440-1448.
41. Offord DR, Boyle MH, Racine Y, Szatmari P, Fleming JE, et al. (1996) Integrating assessment data from multiple informants. *J Am Acad Child Adolesc Psychiatry* 35: 1078-1085.
42. Hofstra MB, Van der Ende J, Verhulst FC (2000) Continuity and change of psychopathology from childhood into adulthood: a 14-year follow-up study. *J Am Acad Child Adolesc Psychiatry* 39: 850-858.