Socioeconomic Indicators and Psychopathology in Children with Chronic Physical Illness

by
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Authors Declaration

This thesis consists of material all of which I authored or co-authored: see Statement of Contributions included in the thesis. This is a true copy of the thesis, including any required final revisions, as accepted by my examiners.

I understand that my thesis may be made electronically available to the public.

Statement of Contributions

This thesis was conducted by Jothi Khaira in collaboration with her supervisor, Dr. Mark Ferro, and with guidance from advisory committee members, Dr. John Mielke and Dr. Martin Cooke.

Abstract

Background: Children with a chronic physical illness (CPI) have an increased risk of developing psychopathology. The combination of chronic stress and socioeconomic disadvantage often contributes to poor mental health in this vulnerable population of children.

Research Questions: This study addresses the following questions: (1) Are neighborhood-level socioeconomic indicators worse among families of children with CPI compared to the general population? (2) Are these socioeconomic indicators associated with psychopathology among children with CPI over a 48-month period? (3) Does child stress moderate associations between socioeconomic indicators and child psychopathology over time?

Methods: Data come from the Multimorbidity in Children and Youth across the Life-course (MY LIFE) study, which followed 263 children aged 2-16 years who had been diagnosed with a CPI, for 48 months. A one-sample t-test compared mean differences in socioeconomic indices, measured by the Ontario Marginalization Index (ON-MARG), between MY LIFE and the general population. Linear mixed models (LMMs) examined associations between ON-MARG dimensions and psychopathology symptoms, as measured with the Emotional Behavioural Scales. Moderation by child HCC was investigated using a time-varying product-term interaction.

Results*:* At baseline, the study included 263 children with a CPI (mean age: 9.4 years, 53% male). Children in MY LIFE resided in areas characterized by greater socioeconomic disadvantage relative to the general population (M= -0.38- -0.20, p= <0.001). Material resources (β=0.13*,* p=0.03) and the two-way interaction between households and dwellings and time (β=0.02, p=0.04) were significantly associated with parent-reported externalizing symptoms. Households and dwellings (β=0.09, p=0.02) and the three-way interaction between age and labour force, time, and child stress (β=0.02, p=0.04) were significantly associated with child-reported externalizing symptoms. No significant associations were noted between ON-MARG dimensions and parent- and child-reported internalizing symptoms.

Conclusion*:* Children with CPI were more likely to live in areas of high socioeconomic disadvantage with housing and material deprivation predicting elevated psychopathology

symptoms. LMMs showed that time-varying child stress may have a moderating effect on indicators of socioeconomic disadvantage, particularly related to households and dwellings and material resources. Differences between parent- and child-reported models reinforce the importance of multi-informant perspectives. Findings highlight the need for integrated, equity-informed interventions targeting both socioeconomic and physiological drivers of child psychopathology among children with CPI.

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Table 1. Demographic and Health Related Characteristics of the MYLIFE Sample.

Characteristic	Response Option	Baseline		48-months	
		n	**(%[a])**	**n**	**(%[a])**
Child (n=263)					
Sex	Male	138	52.7	138	52.7
Age (years)	<10	144	54.8	61	23.2
	≥10	119	45.2	202	76.8
Country of birth	Canada	246	93.9	246	93.9
Ethnicity/Race	White	232	88.2	232	88.2
	Latin American	3	1.14	3	1.14
	Black	8	3.04	8	3.04
	Arabic	11	4.18	11	4.18
	South Asian	1	0.38	1	0.38
	Southeast Asian	1	0.38	1	0.38
	First Nations	7	2.66	7	2.66
Diagnosis	Dermatological	23	8.7	23	8.7
	Endocrine	38	14.4	38	14.4
	Gastroenterological	34	12.9	34	12.9
	Hematological	29	11	29	11
	Neurological	12	4.6	12	4.6
	Respiratory	54	20.5	54	20.5
	Rheumatological	73	27.8	73	27.8
Years since diagnosis	<1.0	63	25		
	1.0 to <2.5	37	14.7		
	2.5-<5.0	54	21.4	67	25.5
	5.0 to <7.5	46	18.3	70	26.6
	≥7.5	52	20.6	126	47.9
Corticosteroid Use	Yes	53	20.2	32	15.8
	No	206	78.3	171	84.2
Frequency of Healthcare Professional Visits	0	233	88.9	150	75
	1-3	12	4.6	11	5.5
	4-7	9	3.4	20	10
	8-11	3	1.1	7	3.5
	12+	5	2.0	12	6
Parent (n=263)					
Sex	Female	235	89.7	235	89.7
Country of Birth	Canada	222	84.7	222	84.7
Marital status	Married	202	77.1	157	78.1
	Common-law	26	9.9	19	9.5
	Widowed	2	0.8		
	Separated	12	4.6	10	5
	Divorced	10	3.8	7	3.5
	Never married	10	3.8	8	4

Table 1 is continued on the following page.

Parental Education	Some secondary	7	2.7	7	2.7
	Secondary graduate	46	17.6	46	17.9
	Vocational/Technical graduate	10	3.8	10	3.8
	Post-secondary graduate	150	57.3	150	57.2
	Graduate/Professional school graduate	49	18.7	49	18.7
Household Income	≤ $29,999	23	8.8	6	3
	$30 000- $59 999	29	11.2	14	7.1
	$60 000- $89 999	52	20	31	15.7
	$90 000- $119 999	35	13.5	27	13.7
	$120 000- $149 999	46	17.7	18	9.1
	≥$150 000	85	32.7	101	51.3

Table 2. Internalizing and Externalizing Score Summary at Baseline and 48-months

Informant	Time	Internalizing Score (Mean ± SD)	Externalizing Score (Mean ± SD)	Internal Consistency (α)
Parent	Baseline	n= 262	n=262	0.84-0.96
		11.0 ± 9.18	7.74 ± 6.70	
	48-months	n= 202	n= 193	
		10.0 ± 9.56	6.52 ± 11.9	
Child	Baseline	n= 116	n= 116	0.90-0.94
		12.2 ± 8.78	7.38 ± 5.34	
	48-months	n= 144	n=144	
		13.8 ± 11.9	7.50 ± 6.46	

Table 2 displays the mean and standard deviation of parent and child-reported internalizing and externalizing scores at baseline and 48-months.

Table 3. ON-MARG Dimension Analysis: Sample vs. General Population

ON-MARG Dimension	Sample Mean	Std. Deviation	t	P	Effect Size
Households and Dwellings	-0.38	0.79	-7.74	<0.001**	-0.48
Material Resources	-0.34	0.90	-6.08	<0.001**	-0.38
Age and Labour Force	-0.22	0.95	-3.64	<0.001**	-0.23
Racialized and Newcomer Populations	-0.20	0.68	-4.69	<0.001**	-0.29

*Table 3 displays the summary of results of the one-sample t-test conducted to compare the MYLIFE sample vs. the general population. All four dimensions were significantly lower than the population average (M= 0, S.D.=1), indicating greater socioeconomic disadvantage in the study sample. Significance Codes are as follows: *p<0.05, **p<0.01, ***p<0.001*

Table 4. Fixed and Random Effects Summary of LMMs for Socioeconomic Indicators and Parent-Reported Internalizing Symptom Scores.

	Unadjusted Model		**Adjusted Model**		**Moderation Analysis**	
Fixed Effect	**Estimate (β) (S.D.)**	**P**	**Estimate (β) (S.D.)**	**P**	**Estimate (β) (S.D.)**	**P**
Time (months)	-3.20e-3 (0.02)	0.04*	-4.48e-3	0.02*	-0.08 (0.06)	0.24
HCC					0.02 (0.08)	0.81
Households and Dwellings	-5.20e-3 (0.09)	0.95	0.02 (0.09)	0.81	0.04 (0.29)	0.88
Material Resources	0.08 (0.08)	0.28	0.08 (0.08)	0.28	0.02 (0.28)	0.95
Age and Labour Force	7.60e-3 (0.08)	0.92	0.01 (0.07)	0.86	0.27 (0.25)	0.28
Racialized and Newcomer Populations	-0.10 (0.10)	0.30	-0.08 (0.10)	0.43	2.00e-3 (0.27)	0.99
Child Age			-9.38e-3 (0.01)	0.45	-0.01 (0.01)	0.66
Child Sex			0.22 (0.10)	0.03*	0.19 (0.11)	0.08
Parent Income ≥ $90k			0.04 (0.07)	0.52	0.07 (0.08)	0.36
Parent Marital Status						
Common-law			0.03 (0.14)	0.84	-0.06 (0.16)	0.72
Widowed			0.61 (0.40)	0.12	0.75 (0.41)	0.07
Divorced			0.07 (0.19)	0.70	0.05 (0.23)	0.84
Separated			0.09 (0.17)	0.61	0.17 (0.20)	0.39
Never Married			0.26 (0.19)	0.17	0.24 (0.22)	0.27
Corticosteroid Use (Yes)			-9.18e-3 (0.08)	0.91	-0.01 (0.09)	0.90
Frequency of Healthcare Professional Visits						
1-3			0.37 (0.18)	0.04*	0.62 (0.22)	0.01
4-7			0.24 (0.14)	0.08.	0.28 (0.17)	0.10
8-11			-0.01 (0.17)	0.94	0.08 (0.19)	0.66
12+			0.32 (0.17)	0.04*	0.37 (0.21)	0.09
*Households and Dwellings*Time*	-3.08e-3 (2.01e-3)	0.14	-3.95e-3 (2.24e-3)	0.08.	-0.03 (0.08)	0.67
*Material Resources*Time*	1.83e-3 (1.85e-3)	0.32	2.71e-3 (1.99e-3)	0.18	0.05 (0.08)	0.55
*Age and Labour Force*Time*	2.20e-3 (2.04e-3)	0.29	2.33e-3 (2.18e-3)	0.29	-0.09 (0.07)	0.22
*Racialized and Newcomer Populations*Time*	1.35e-3 (2.30e-3)	0.55	1.08e-3 (2.34e-3)	0.65	-0.04 (0.08)	0.62
*HCC*Time*					-0.01 (0.03)	0.67
*Households and Dwellings*HCC*					0.03 (0.10)	0.80
*Material Resources*HCC*					0.06 (0.10)	0.58
*Age and Labour Force*HCC*					-0.12 (0.08)	0.15
*Racialized and Newcomer Populations*HCC*					-0.05 (0.09)	0.57
*Households and Dwellings*Time*HCC*					-0.01 (0.03)	0.73
*Material Resources*Time*HCC*					-0.02 (0.03)	0.51
*Age and Labour Force*Time*HCC**					0.05 (0.03)	0.06
*Racialized and Newcomer Populations*Time*HCC*					0.03 (0.03)	0.41

Random Effects	**Variance**	**S.D.**	***r***	**Variance**	**S.D.**	***r***	**Variance**	**S.D.**	***r***
Intercept	0.74	0.86		0.67	0.82		0.81	0.90	
Time	2.20e-4	0.01	-0.30	1.90e-4	0.01	-0.39	0.02	0.15	-0.56
Residual	0.26	0.51		0.28	0.53		0.28	0.53	

*Table 4 summarizes fixed and random effect results of linear mixed models analyzing parent-reported internalizing symptoms over 48 months. Significance codes are as follows; *p<0.05, **p<0.01, ***p<0.001.*

Table 5. Fixed and Random Effects Summary of LMM for Socioeconomic Indicators and Parent-Reported Externalizing Symptom Scores.

	Unadjusted Model		Adjusted Model		Moderation Analysis	
Fixed Effect	**Estimate (β) (S.D)**	**P**	**Estimate (β) (S.D)**	**P**	**Estimate (β) (S.D)**	**P**
Time (months)	-0.30 (1.40e-3)	0.04*	-0.04 (0.02)	0.04*	-0.02 (0.06)	0.78
HCC					0.05 (0.07)	0.49
Households and Dwellings	-0.13 (0.08)	0.12	0.17 (0.10)	0.08	0.03 (0.25)	0.91
Material Resources	0.15 (0.07)	0.03	0.13 (0.08)	0.03*	-0.01 (0.25)	0.96
Age and Labour Force	3.54e-3 (0.07)	0.96	0.02 (0.08)	0.79	-0.19 (0.22)	0.39
Racialized and Newcomer Populations	-0.06 (0.09)	0.49	-2.84e-3 (0.12)	0.98	0.08 (0.24)	0.73
Child Age			-8.49e-3 (0.01)	0.45	-0.01 (0.01)	0.64
Child Sex			-0.19 (0.10)	0.04*	-0.21 (0.11)	0.04*
Parent Marital Status						
Common-law			0.12 (0.14)	0.39	0.15 (0.15)	0.33
Widowed			0.83 (0.35)	0.02*	0.94 (0.37)	0.01*
Divorced			0.12 (0.17)	0.48	0.16 (0.21)	0.45
Separated			0.11 (0.16)	0.47	0.18 (0.18)	0.33
Never Married			0.26 (0.17)	0.13	0.21 (0.20)	0.30
Corticosteroid Use (Yes)			0.05 (0.07)	0.51	0.04 (0.08)	0.67
Frequency of Healthcare Professional Visits						
1-3			0.12 (0.16)	0.51	0.25 (0.19)	0.19
4-7			0.28 (0.13)	0.03*	0.23 (0.15)	0.13
8-11			-0.03 (0.15)	0.83	-0.03 (0.17)	0.86
12+			0.31 (0.15)	0.04*	0.40 (0.19)	0.04*
*Households and Dwellings*Time*	2.07e-3 (1.8e3-3)	0.26	0.02 (0.02)	0.04*	-0.01 (0.02)	0.24
*Material Resources*Time*	9.89e-4 (1.62e-3)	0.95	9.74e-3 (0.02)	0.61	-0.08 (0.07)	0.28
*Age and Labour Force*Time*	-7.34e-4 (1.80e-3)	0.68	-0.01 (0.02)	0.59	0.08 (0.07)	0.22
*Racialized and Newcomer Populations*Time*	-4.44e-4 (2.00e-3)	0.82	-0.02 (0.02)	0.42	0.08 (0.07)	0.79
*HCC*Time*					-0.02 (0.07)	0.58
*Households and Dwellings*HCC*					-0.09 (0.10)	0.34
Material Resources HCC*					0.08 (0.09)	0.34
*Age and Labour Force*HCC*					0.09 (0.07)	0.23
*Racialized and Newcomer Populations*HCC*					-0.03 (0.08)	0.74
*Households and Dwellings*Time*HCC*					0.05 (0.03)	0.08.
*Material Resources*Time*HCC*					-0.04 (0.03)	0.14
*Age and Labour Force*Time*HCC**					-0.04 (0.03)	0.13
*Racialized and Newcomer Populations*Time*HCC*					-0.01 (0.03)	0.83

Random Effects	**Variance**	**S.D.**	***r***	**Variance**	**S.D.**	***r***	**Variance**	**S.D.**	***r***
Intercept	0.64	0.80		0.73	0.85		0.72	0.85	
Time	1.53e-4	0.01	-0.30	0.02	0.14	-0.47	0.02	0.14	-0.47
Residual	0.20	0.45		0.20	0.45		0.21	0.46	

*Table 5 summarizes fixed and random effect results of linear mixed models analyzing parent-reported externalizing symptoms over 48 months. Significance codes are as follows; *p<0.05, **p<0.01, ***p<0.001.*

Table 6. Fixed and Random Effects Summary of LMM for Socioeconomic Indicators and Child-Reported Internalizing Symptom Scores

	Unadjusted Model		Adjusted Model		Moderation Analysis	
Fixed Effect	**Estimate (β) (S.D)**	**P**	**Estimate (β) (S.D)**	**P**	**Estimate (β) (S.D)**	**P**
Time (months)	1.55e-4 (2.28e-3)	0.95	-2.63e-3 (2.28e-3)	0.36	4.83e-3 (0.09)	0.96
HCC					0.11 (0.13)	0.40
Households and Dwellings	-0.12 (0.12)	0.34	-0.06 (0.13)	0.63	0.47 (0.46)	0.31
Material Resources	0.01 (0.10)	0.92	0.03 (0.11)	0.76	-0.29 (0.42)	0.49
Age and Labour Force	0.10 (0.09)	0.28	0.11 (0.09)	0.24	0.30 (0.39)	0.45
Racialized and Newcomer Populations	-3.10e-4 (0.13)	0.98	0.06 (0.13)	0.65	-0.54 (0.46)	0.24
Child Age			-4.72e-3 (0.02)	0.84	7.69e-3 (0.02)	0.76
Child Sex			0.47 (0.13)	<0.001**	0.52 (0.14)	2.78e-4 ***
Parent Marital Status						
Common-law			0.20 (0.21)	0.36	0.16 (0.22)	0.48
Widowed			0.63 (0.43)	0.15	0.76 (0.45)	0.08.
Divorced			0.24 (0.24)	0.31	0.38 (0.29)	0.19
Separated			0.67 (0.28)	0.02*	0.53 (0.34)	0.12
Never Married			-0.09 (0.28)	0.74	-0.04 (0.31)	0.90
Corticosteroid Use (Yes)			-0.15 (0.13)	0.27	-0.13 (0.15)	0.37
Frequency of Healthcare Professional Visits						
1-3			0.34 (0.24)	0.16	0.63 (0.28)	0.03*
4-7			0.10 (0.18)	0.57	0.19 (0.21)	0.35
8-11			0.33 (0.21)	0.11	0.42 (0.24)	0.08.
12+			0.37 (0.26)	0.16	0.58 (0.36)	0.11
*Households and Dwellings*Time*	2.08e-4 (3.00e-3)	0.95	-2.02e-3 (3.26e-3)	0.54	-0.11 (0.12)	0.35
*Material Resources*Time*	4.66e-4 (2.62e-3)	0.86	5.95e-4 (2.95e-3)	0.84	0.09 (0.12)	0.43
*Age and Labour Force*Time*	-6.54e-5 (2.93e-3)	0.98	1.77e-3 (3.26e-3)	0.59	-0.05 (0.12)	0.67
*Racialized and Newcomer Populations*Time*	5.53e-4 (3.18e-3)	0.86	1.56e-3 (3.40e-3)	0.65	0.12 (0.12)	0.32
HCC*Time					-0.04 (0.04)	0.35
*Households and Dwellings*HCC*					-0.20 (0.19)	0.30
*Material Resources*HCC*					0.12 (0.16)	0.44
*Age and Labour Force*HCC*					-0.10 (0.16)	0.54
*Racialized and Newcomer Populations*HCC*					0.21 (0.17)	0.21
*Households and Dwellings*Time*HCC*					0.02 (0.06)	0.70
*Material Resources*Time*HCC*					-0.03 (0.05)	0.61
*Age and Labour Force*Time*HCC*					0.04 (0.05)	0.42
*Racialized and Newcomer Populations*Time*HCC*					-0.03 (0.05)	0.61

Random Effects	**Variance**	**S.D.**	***r***	**Variance**	**S.D.**	***r***	**Variance**	**S.D.**	***r***
Intercept	0.64	0.80		0.54	0.74		0.77	0.80	
Time	2.33e-4	0.02	-0.17	1.96e-4	0.01	-0.14	0.04	0.20	-0.58
Residual	0.31	0.56		0.35	0.59		0.34	0.59	

*Table 6 summarizes fixed and random effect results of linear mixed models analyzing child-reported internalizing symptoms over 48 months. Significance codes are as follows; *p<0.05, **p<0.01, ***p<0.001.*

Table 7. Fixed and Random Effects Summary of LMM for Socioeconomic Indicators and Child-Reported Externalizing Symptom Scores

	Unadjusted Model		**Adjusted Model**		**Moderation Analysis**	
Fixed Effects	**Estimate (β) (S.D)**	**P**	**Estimate (β) (S.D)**	**P**	**Estimate (β) (S.D)**	**P**
Time (months)	-2.86e-4 (1.69e-3)	0.87	-7.60e-3 (0.03)	0.77	0.01 (0.07)	0.84
HCC					0.08 (0.09)	0.38
Households and Dwellings	-0.33 (0.10)	1.18e-3	-0.39 (0.13)	3.00e-3**	-0.47 (0.34)	0.17
Material Resources	0.12 (0.08)	0.16	0.10 (0.11)	0.38	-0.11 (0.31)	0.72
Age and Labour Force	0.02 (0.08)	0.76	-0.03 (0.09)	0.78	0.39 (0.29)	0.17
Racialized and Newcomer Populations	-0.13 (0.10)	0.20	-0.14 (0.13)	0.27	-0.24 (0.33)	0.47
Child Age			-0.02 (0.02)	0.20	-0.03 (0.02)	0.13
Child Sex			-0.11 (0.12)	0.34	-0.10 (0.12)	0.40
Parent Marital Status						
Common-law			0.10 (0.11)	0.34	0.13 (0.24)	0.34
Widowed			0.23 (0.43)	0.22	0.46 (0.27)	0.06.
Divorced			0.44 (0.14)	0.41	0.24 (0.31)	0.21
Separated			0.58 (0.16)	0.04*	0.45 (0.25)	0.07.
Never Married			-0.08 (0.18)	0.54	-0.03 (0.28)	0.81
Corticosteroid Use (Yes)			-0.20 (0.11)	0.06.	-0.21 (0.21)	0.30
Frequency of Healthcare Professional Visits						
1-3			0.39 (0.19)	0.04*	0.49 (0.20)	0.02*
4-7			0.08 (0.14)	0.58	0.26 (0.15)	0.09.
8-11			0.11 (0.16)	0.50	0.22 (0.18)	0.21
12+			0.36 (0.21)	0.07.	0.50 (0.28)	0.08.
*Households and Dwellings*Time*	2.92e-3 (2.25e-3)	0.08.	0.03 (0.03)	0.36	0.08 (0.09)	0.39
*Material Resources*Time*	1.80e-4 (1.94e-3)	0.71	0.01 (0.03)	0.65	0.09 (0.09)	0.31
*Age and Labour Force*Time*	8.20e-4 (2.20e-3)	0.71	0.03 (0.03)	0.29	-0.15 (0.09)	0.09.
*Racialized and Newcomer Populations*Time*	3.62e-4 (2.35e-3)	0.88	8.05e-3 (0.03)	0.80	0.01 (0.09)	0.87
*HCC*Time*					-0.02 (0.03)	0.44
*Households and Dwellings*HCC*					0.08 (0.14)	0.58
*Material Resources*HCC*					0.08(0.12)	0.47
*Age and Labour Force*HCC*					-0.21 (0.12)	0.08.
*Racialized and Newcomer Populations*HCC*					0.05 (0.12)	0.69
*Households and Dwellings*Time*HCC*					-0.04 (0.04)	0.33
*Material Resources*Time*HCC*					-0.03 (0.04)	0.37
*Age and Labour Force*Time*HCC**					0.09 (0.04)	0.02*
*Racialized and Newcomer Populations*Time*HCC*					-7.79e-3 (0.04)	0.85

Random Effects	**Variance**	**S.D.**	***r***	**Variance**	**S.D.**	***r***	**Variance**	**S.D.**	***r***
Intercept	0.46	0.68		0.43	0.65		0.52	0.72	
Time	1.03e-4	0.01	3.00e-3	0.01	0.12	-0.17	0.02	0.16	-0.40
Residual	0.19	0.44		0.20	0.45		0.17	0.41	

*Table 7 summarizes fixed and random effect results of linear mixed models analyzing child-reported externalizing symptoms over 48 months. Significance codes are as follows; *p<0.05, **p<0.01, ***p<0.001.*

Table 8. Model Fit Indices for Unadjusted, Adjusted, and Moderation Analysis for Parent-Reported and Child-Reported Internalizing and Externalizing Symptoms

	REML Criterion	AIC	BIC
Parent-Reported Internalizing			
Unadjusted Model	2645.9	2673.9	2744.9
Adjusted Model	2413.6	2467.6	2601.6
Moderation Analysis	1902.9	1976.9	2150.3
Parent-Reported Externalizing			
Unadjusted Model	2365.4	2393.4	2464.4
Adjusted Model	2116.4	2168.4	2297.2
Moderation Analysis	1717.1	1789.1	1957.4
Child-Reported Internalizing			
Unadjusted Model	1578.7	1606.7	1669.2
Adjusted Model	1365.6	1417.6	1528.9
Moderation Analysis	1111.2	1417.6	1528.9
Child-Reported Externalizing			
Unadjusted Model	1289.9	1317.9	1380.4
Adjusted Model	1105.5	1147.52	1237.49
Moderation Analysis	910.4	972.4	1098.1

Table 8 summarize model fit indices for all 12 models analyzed using both parent and child-reported outcomes. Lower values across REML, AIC, and, BIC indicate improved model fit, with the moderation models consistently demonstrating the best fit for most outcomes, particularly parent-reported symptoms. Child-reported internalizing symptoms showed minimal improvement in AIC/BIC, thought REML indicated better variance estimation in the moderation model.

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Appendix 5. Theoretical LMM for both Parent and Child-reported Internalizing and Externalizing Symptom Outcomes.

Figure 1. Analysis Depiction of Socioeconomic Indicators and their Associations to Psychopathology Symptoms in Children with a CPI over time.

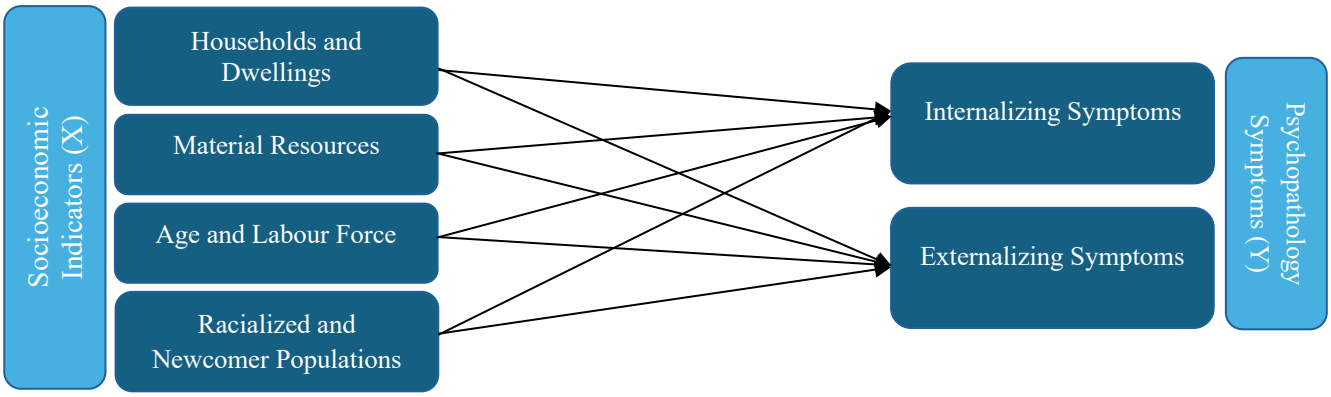


Figure 1 displays the theoretical model between ON-MARG dimensions as the measure of socioeconomic indicators and their associations to outcomes of internalizing and externalizing symptoms.

Figure 2. Moderation Analysis Depiction of Child Stress in the Association between Socioeconomic Indicators and Psychopathology Symptoms in Children with a CPI over time.

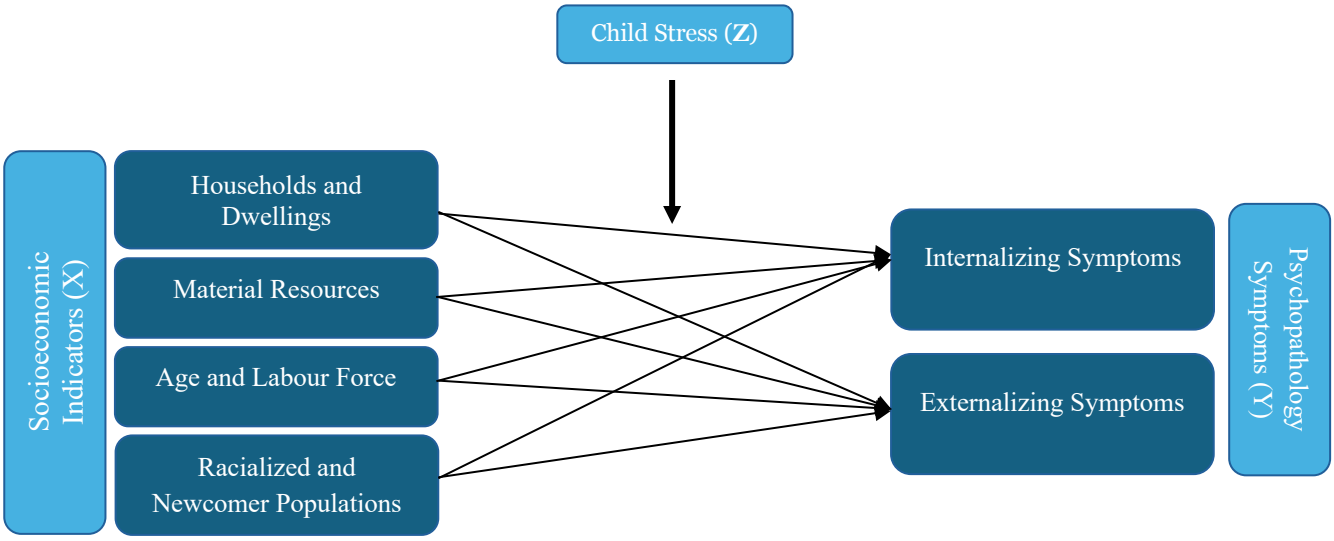


Figure 2 displays the theoretical model between ON-MARG dimensions as the measure of socioeconomic indicators and their associations to outcomes of internalizing and externalizing symptoms, while adding child stress as a time-varying interaction term.

Figure 3. Moderating Effect Plots for Each ON-MARG Dimension using Parent and Child-Reported Internalizing and Externalizing Symptoms.

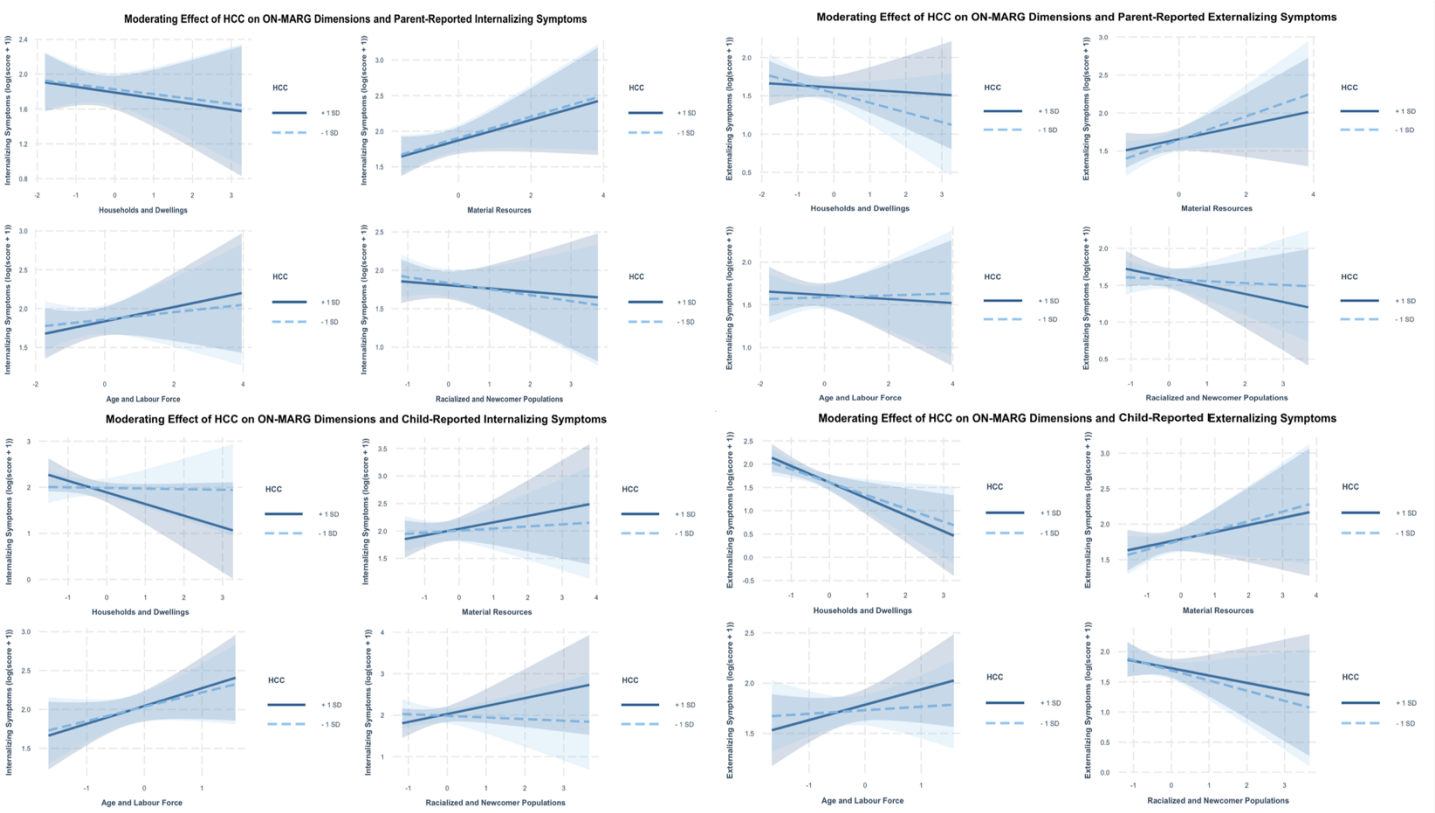


Figure 3 depicts the moderating effect of child stress, measured by child HCC, on the relationship between ON-MARG dimensions and parent and child-reported internalizing and externalizing symptoms, as analyzed in objective 3. Overall, higher HCC (+1 SD) amplified the association between ON-MARG and greater symptom severity, particularly for externalizing symptoms and in domains such as Households and Dwelling, Material Resources, and Age and Labour force. Crossover effects were evident, with higher symptom scores observed at high child HCC and higher ON-MARG index scores. Moderation patterns were consistent for parent-reported outcomes than child-reported.

List of Abbreviations

ADHD	Attention-deficit Hyperactivity Disorder
CD	Conduct Disorder
CPI	Chronic Physical Illness
GAD	Generalized anxiety disorder
HCC	Hair Cortisol Concentration
HPA	Hypothalamic-Pituitary-Adrenal
ON-MARG	Ontario Marginalization Index
EBS	Emotional Behavioural Scales
MDD	Major Depressive Disorder
MI	Multiple Imputation
MNAR	Missingness Not at Random
ODD	Oppositional defiant disorder
SAD	Social Anxiety Disorder
SES	Socioeconomic Status
SoP	Social Phobia

Background

1.1 Introduction

Children with a chronic physical illness (CPI; e.g., juvenile arthritis, diabetes, asthma, epilepsy) can experience substantial levels of stress, and are at a greater risk of developing psychopathology.[1] Psychopathology is characterized by the dysfunction of biological, psychological, or developmental processes causing significant disturbances to behaviors, cognition, and emotional regulation.[2] Psychopathology in this population can arise from stress related to environmental or familial burdens, treatment plans, social isolation, or reoccurring ruminative thoughts.[3, 4] These challenges predispose children with a CPI to developing psychopathology symptoms.[5,6] The co-occurrence of a CPI and a mental disorder is known as physical-mental multimorbidity and is a rising public health concern. Research suggests that there is a wide range of multimorbidity prevalence, where 5-25% of children with at least one CPI also have at least one mental disorder.[7,8,9,10] While individual and clinical factors contribute to multimorbidity, growing evidence highlights the importance of social and economic conditions in shaping child vulnerability to psychological distress.[11,12] Children facing the burden of a CPI are often socioeconomically disadvantage, which can intensify stress and limit access to protective resources such as stable housing, supportive caregiving, or access to mental health services, underscoring the need to examine broader contextual influences on their psychopathology.[13,14] The high prevalence of childhood multimorbidity supports the need to assess the influences of societal, economical, and neighborhood-level factors on physiological stress responses to better inform future interventions.[8,15,16,17]

1.2 Socioeconomic Indicators and Child Psychopathology

Investigating the intersection of individual and neighborhood-level influences, such as, sociodemographic characteristics, geographical areas, and marginalization factors is key to developing a more complete understanding of the determinants of psychopathology in children.[13] Marginalization is defined as the inhibition of participation in societal activities, involving neighborhood and individual health, due to potential barriers, including accessing satisfactory housing, employment, education, or access to health services.[18] Marginalized communities often experience structural and systematic barriers that manifest through measurable socioeconomic conditions. Specific indicators of marginalization, particularly those related to material

deprivation or household instability, offer a concrete way to assess the extent and impact of socioeconomic disadvantage amongst communities. Indicators of socioeconomic disadvantage, such as access to sufficient housing, sufficient material resources, and dependency ratios, may provide evidence to understand the level of socioeconomic disadvantage in a specified geographical area.[18] Disadvantage is defined as a state of shortcoming under socially acceptable levels compared to an individuals' peers or community.[19] Socioeconomic disadvantage may be the result of communities and families among low socioeconomic status (SES). Socioeconomic status has been defined as the level of social and material deprivation of an individual living in society.[19] Socioeconomic gradients, from indicators such as unemployment rates or minimal education, may have both direct causes to differences in nutrition, hygiene and, exposure to toxins, and indirect causes such as influences on stress levels and immune responses.[20] Those residing in socioeconomically disadvantaged communities within low social strata of communities are less likely to benefit from advances in healthcare, ultimately influencing prevention and treatment of various physical illnesses and psychopathology.[21]

Psychopathology can significantly impact various aspects of human life including personal relationships, educational success, employment opportunities, and overall wellbeing.[22] Psychopathology can often arise from traumatic events related to negative familial experiences, including low SES, strained marital relationships, and limited access to supports.[22,23] Some studies suggest that individuals who are socioeconomically disadvantaged may experience higher levels of psychopathology due to individual and neighborhood-level factors including substandard housing quality, housing tenure, poor neighbourhood quality, unemployment, and other financial difficulties.[24,25,26] These factors are likely to contribute to cumulative burden that exacerbate psychopathology vulnerabilities, therefore reinforcing socioeconomic gradients.[27] Canadians who have low income are three to four times more likely to report poor to fair mental health compared to those in higher income groups, who report good mental health.[28] Children in the lowest-income families are also more likely to show symptoms of psychopathology, including high levels of hyperactivity and emotional disorders.[29] Furthermore, socioeconomically disadvantaged communities that include a large population of immigrants may be a greater risk of negative psychopathology.[29] Negative societal experiences for these populations may arise from the lack of societal incorporation and sense of neighborhood

belonging and pose larger threats on overall well-being and directly impact child health, both internally and externally.[29,30,31]

The overall prevalence of psychopathology, including behavioural or emotional problems, in children between the ages of 4-11 and 12-17 are 18.1% and 21.8%, respectively.[32] Symptoms of behavioural and emotional problems are commonly characterized into two categories, internalizing and externalizing.[33] Internalizing problems are identified by constant worrying, anxiety, depression, or social disconnect. Externalizing problems, on the other hand, include behaviours of hyperactivity, aggression, or impulsivity.[33] It is important to consider internalizing and externalizing symptoms as distinct outcomes, as they may be shaped by different social and environmental determinants and often require tailored interventions and supports.[32,33]

Childhood adversity has been associated with an increased risk of both internalizing and externalizing symptoms in children between the age of 14-17 years.[34] Experiences of poor housing, increased family stress, and lower SES have been previously associated with increased internalizing disorders.[35,36] Additionally, factors such as low household income, limited parental education, and reduced access to educational and health resources has been associated with an increased risk of externalizing symptoms.[36] Current evidence exploring child dependency ratios and unowned dwellings and proportions of ethnic minorities and their connections to neighborhood-level influences on internalizing and externalizing symptoms in children, especially those with a CPI, are inadequate. Although studies examining this relationship are limited, socioeconomic disadvantage has been previously associated to physiological stress dysregulation, which may also contribute to the development of psychopathology symptoms. Understanding the differences between internalizing and externalizing symptom outcomes in seriocomically disadvantage communities will contribute to improving individual health outcomes and inform evidence-based policies and mental health initiatives across Canada.

1.3 Impacts of Stress on Physiological Responses

In response to psychosocial stressors, such as socioeconomic disadvantage, children are at an increased odds of experiencing dysregulation in biological responses.[15,37] Chronic stress can trigger an imbalance in regulation processes of the sympathetic nervous system, often causing physiological effects such elevation of stress hormones such as epinephrine and cortisol.[38,39, 40, 41]

Hypothalamic-pituitary-adrenal (HPA)-axis activity involves communication between the hypothalamus, pituitary gland, and adrenal glands that release cortisol within the body in response to stress. Cortisol has various roles within the body, but primarily mediates stress and inflammatory responses, as well as immune functions.[42] Cortisol can be collected from saliva, blood, hair, and urine and is an adaptive measure of the release of glucocorticoids.[43]

Hair cortisol concentration (HCC) has become an increasingly popular biomarker to document longitudinal fluctuations of cortisol while eliminating burdens of other invasive approaches (e.g., blood sampling).[44] Because hair grows at approximately 1 cm per month, HCC allows retrospective estimates of chronic stress exposure and is a suitable marker for understanding how early-life socioeconomic disadvantage contributes to the development of psychopathology.[45,46] Many existing studies use self-reported measures to understand experiences of chronic stress; however, HCC may eliminate the potential of recall bias. Therefore, HCC offers an objective method to explore the long-term effects of stressors on child well-being and development.

Socioeconomic disadvantage may contribute to perceived stress in early-childhood and may trigger elevated physiological stress responses. [37] Neighborhood factors have been associated with increased HCC mediated by early-life chronic stress.[47, 48, 49] Elevated HCC was also seen in children coming from racialized and vulnerable groups who may be more susceptible to differences in socioeconomic diversity, lack of maternal supports, or developmental delays.[49,50] These stress-related factors may exacerbate psychopathology and exhibit positive correlations with increased cortisol.[44,51,52,53] There is little evidence on how physiological stress may impact associations between socioeconomic indicators and psychopathology. Existing studies show that factors such as emotional responses and cognitive flexibility may moderate associations between socioeconomic disadvantage and psychopathology.[54] These responses have been linked to elevated baseline cortisol and blood pressure levels, often causing reactivity in HPA-axis activity.[54] The moderating effect of a hormonal biomarker, such as cortisol, is yet to be explored and can provide valuable information to determine how the strength of the relationship between neighborhood-level factors and psychopathology differs based on stress-related exposures in early-life adverse experiences. Elevated HCC levels may provide evidence of prolonged exposures to stress, particularly related to socioeconomic disadvantage in children with CPI and expose factors intensifying the development of internalizing or externalizing symptoms.[42,55] By

assessing child stress, measured by HCC, this study assesses vulnerability and resilience to psychopathology symptoms to better shape existing policies and interventions.

1.4 Socioeconomic Disadvantage, Stress, and Psychopathology in Children with CPI

Children with a CPI are at an increased risk of experiencing dysregulations in HPA-axis activity due to chronic physiological stress related to environmental or familial factors and health-related challenges.[8,56] This results in an elevated risk of developing physical-mental multimorbidity within this population. Although psychopathology is relatively common, affecting 20-40% of children with a CPI, very few studies have studied the mechanisms by which neighborhood-level factors influence individual-level mental health.[8,57,58] Socioeconomic indices are commonly used to explore socioeconomic disadvantage in population health outcomes, and are commonly connected to disease prevalence rates, life expectancies, and general health outcomes.[59,60] It is vital to explore how individual experiences and socioeconomic circumstances may become biologically embedded during the sensitive period of child development.[61]

The occurrence of multimorbidity is heavily influenced by the social circumstances in which an individual is born, works, lives, and grows.[62] The combination of environmental and individual factors may increase the risk of developing of multimorbidity in children who experience societal deprivation.[63] The majority of existing studies exploring multimorbidity in socioeconomically disadvantaged groups have been conducted in adult populations. In Ontario, the prevalence of multimorbidity is prominently higher in those residing in more deprived areas, where environmental factors such as residential instability or material deprivation may be more predominant.[64] Increased multimorbidity has been seen in groups with lower SES, lower educational qualifications, and among ethnic minorities.[65,66] Associations between household income or area-level deprivation differed in age groups and multimorbidity type.[66] Improving understanding of neighborhood-level socioeconomic indicators as social determinants of multimorbidity could better inform strategies to limit its burden. It is unknown if this evidence can generalize to children with a CPI.

1.5 Theoretical Framework: Gene-environment Interaction (Diathesis-stress Model)[69]

To understand the interplay of environmental and individual characteristics, it is important to consider the role of gene-environmental interaction (GEI) on disease etiology.[67,68,69,70] Gene-environmental interaction is defined as the phenomenon whereby the impacts of environmental exposures on disease risk are dependent on individual genotype.[67,68] Advances in biological research have revealed that genetic influences are highly complex, with some disease outcomes clustering within families while others fall inconsistent with expected patterns of inheritance.[69]

To understand the development of psychopathology, the diathesis-stress model provides support in understanding the degree of inherent vulnerability in an individual.[69,70] This framework suggests onset of a psychological disorder can be triggered by an environmental stress.[70] Guided by this model, children with CPI may possess intrinsic vulnerabilities due to ongoing health challenges, treatment demands, and developmental disruptions.[4,71] These vulnerabilities may interact with environmental factors, such as socioeconomic disadvantage, to influence the emergence of psychopathology symptoms.[71] Furthermore, elevated physiological stress may also moderate associations between socioeconomic disadvantage and psychopathology, reflecting the biological embedding of adversity.[68,69] By integrating socioeconomic and biological perspectives, this study leverages the GEI framework to deepen our understanding of multimorbidity and the interplay between CPI, social context, genetic susceptibility and physiological stress outcomes among children.

1.6 Gaps in Literature

Despite the growing research on physical-mental multimorbidity, research exploring this phenomenon in children, particularly those experiencing socioeconomic disadvantage, remains limited. While socioeconomic gradients have been broadly linked to child psychopathology, few studies have explored neighborhood-level indicators such as material deprivation, residential mobility rates, or proportion of labour force participation. Additionally, current literature does not investigate internalizing and externalizing symptoms separately as outcomes of socioeconomic disadvantage in children with CPI. It is also important to note, existing studies focus mainly on adult populations, with only one longitudinal study in Quebec evaluating chronic adversity and its associations with child psychopathology and child HCC.[72] This study

found non-linear associations between adversity and HCC, with moderate HCC levels associated with lower adversity, suggesting a complex physiological response to chronic stress.[72]

To date, there are no known studies that have explored child stress, measured by HCC, as a moderator between neighborhood-level socioeconomic indicators and psychopathology in pediatric CPI populations. Most studies consider HCC as an outcome or general biomarker of acute stress, and overlook how chronic stress may amplify psychological effects of socioeconomic disadvantage. Addressing these gaps is essential to understanding the interactions between environmental, psychological, and biological factors to better target both individual and neighborhood-based health interventions.

Study Rationale and Hypotheses

Although there is a plethora of literature exploring socioeconomic indicators in Canadian populations, the need to explore multimorbidity is necessary. The findings of this study expose neighborhood-level factors influencing psychopathology by providing insight on potential neighborhood-level socioeconomic indices exacerbating chronic stress in children with a CPI. These results are obtained by comparing the experiences of children with a CPI with children in the general population of Canada. The first research question provides a comparison of experiences of children with a CPI versus the general population of Ontario across four dimensions of socioeconomic indicators. It was hypothesized that children with a CPI will reside in communities that exhibit higher socioeconomic disadvantage compared to the general population.[73]

The second research question combines socioeconomic experiences with outcomes of psychopathology by differentiating experiences associated with internalizing and externalizing symptoms. Existing literature provides evidence to state that experiences of early-life socioeconomic adversity may play a significant role in the development of physical-mental multimorbidity, however very few studies have differentiated the outcomes specific to internalizing or externalizing symptoms.[74] Exploring these symptom categories separately exposes the need for existing policies and interventions to focus on the impacts of the most influential socioeconomic indicators predicting behavioral outcomes in children. Although a positive association between socioeconomic disadvantage and psychopathology is expected, findings that contradict expected outcomes have been reported within the MYLIFE sample. For instance, a recent study examining school belonging in youth, >10 years of age, found no decline in perceptions of school belonging during the transition to secondary school.[75] This was an unexpected result given that existing literature typically reports reduced school belonging during this period.[76] This discrepancy underscores the complexity of developmental and social experiences in children with CPI and highlights the importance of continued examination of these associations to inform both research and clinical practice.[76]

Furthermore, the third research question aims to assess stress as a moderator in the relationship between socioeconomic disadvantage and psychopathology, which is yet to be explored in literature. Only one study has explored the outcomes of socioeconomic adversity on psychopathology and child stress measured by HCC.[72] Conducting this research will provide a

holistic approach in understanding the psychopathology in children with CPI by recognizing biological stress responses alongside individual and neighborhood-level social or psychological factors. Exploring stress as a moderator, measured by HCC, can reveal if the effects of socioeconomic disadvantage on psychopathology are stronger or weaker dependent on individual long-term biological stress responses. It was hypothesized that higher levels of stress will amplify the association between socioeconomic disadvantage and increased internalizing and externalizing symptom severity in children with a CPI overtime. This interaction is expected to reflect a diathesis-thesis stress model, in which children with heightened biological sensitivity to context may be more vulnerable to the psychological impacts of socioeconomic adversity.[77] Based on this expectation, it is hypothesized that children with higher long-term physiological stress (i.e., elevated HCC) will be more vulnerable to the psychological effects of socioeconomic disadvantage, leading to more severe internalizing and externalizing symptoms over time. In this context, biological sensitivity to stress may amplify the risk associated with social adversity. Clarifying this interaction can guide the development of integrated healthcare policies and interventions that address not only environmental inequities, but also individual susceptibility to psychological distress in children with chronic conditions.

Research Questions

This study investigated neighborhood-level socioeconomic indicators and psychopathology in children aged 2-16 years diagnosed with CPI who received outpatient services at a pediatric hospital in Ontario. The specific research questions are:

(1) Are indicators of neighborhood-level socioeconomic disadvantage higher among families of children with CPI compared to the general population?

(2) Are these socioeconomic indicators associated with psychopathology among children with CPI over time?

(3) Does child stress moderate associations between socioeconomic indicators and child psychopathology over time?

Methods

4.1 MY LIFE Study Data

Sample data come from the Multimorbidity in Children and Youth Across the Life-course (MY LIFE) study.[78] This longitudinal study was conducted to estimate the prevalence of mental disorder in children and youth with CPI and explore biological and psychosocial factors that influence multimorbidity.

4.2 Sample Recruitment

The MY LIFE study includes 263 children and adolescents with one CPI between the age of 2 to 16 years, and their parent. Families were recruited from pediatric outpatient clinics at McMaster Children's Hospital in Hamilton, Ontario.[78] The outpatient clinics participating in study recruitment were those in the divisions of endocrinology, gastroenterology, hematology and oncology, immunology, allergy, and dermatology, neurology, respirology, and rheumatology.[78] For this study, CPI is defined as a physician diagnosed condition that is expected to persist for at least 12 months. These diagnoses can result in one or more of the following: (1) limitations on physical functions, (2) dependence on external aids for physical limitations, and (3) the continued use of healthcare services.[79] Inclusion criteria for the selected sample was intentionally broad to increase the generalizability for early-life CPI diagnoses and allow adequate follow-up of the cohort through continued pediatric care.[80] In the case that a child/youth was experiencing multiple medically complex physical conditions, their participation from the study was excluded as their experiences with psychopathology may differ. Participants were also required to have sufficient English language skills as not all measures included in the MY LIFE study have been validated in other languages.[78] Participants and their parent were followed for a total of 48 months, with assessment occurring at five time-points: recruitment (baseline), 6, 12, 24, and 48 months.[78]

4.3 Study procedures

Health professionals worked with research assistants (RAs) to collect necessary data. Children and their parents interested in participating spoke to research staff who verified participant eligibility and obtained written informed consent from all parents and children who were at least16 years of age, written assent from children between the age of 7-15 years, and oral

assent from children younger than seven years.[78] The study coordinator then contacted parents to schedule data collection around each time point. Data was collected using structured interviews, self-reported measures, and biological samples in offices within the hospital by an RA before the COVID-19 pandemic. During the pandemic, data was collected through phone interviews and mail-in packages to minimize exposure between research staff and participants.[78] To minimize missing data and data entry and coding errors, data collection was computer-assisted. Ethics approval for this study was obtained from the Hamilton Integrated Research Ethics Board (2797; April 2017) and Waterloo Human Research Ethics Board (ORE-31010; June 2017). Additional details on study procedures are available elsewhere.[78]

4.4 Data Collection and Study Measures

4.4.1 Neighborhood-Level Socioeconomic Indicators

To assess socioeconomic indicators within the population, this study used indicator measures from the 2016 Ontario Marginalization Index (ON-MARG) in specified geographical areas.[81] The ON-MARG is a validated, empirically derived index that uses principal component analysis to generate continuous factor scores. This metric provides data indicating surrounding multiple axes of neighborhood-level factors in Ontario, including indicators of economic, ethno-racial, social, and age-based marginalization. The ON-MARG was developed using factor analysis of 42 potential indicators (18 were retained) found in the 2001 Canadian census which are grouped within four dimensions; households and dwellings, material resources, age and labour force, and racialized and newcomer populations.[81]

Households and dwellings include indicators that measure the availability of residential accommodations, family structure, and characteristics including proportions of the population living alone and homes unowned.[82] Material resources include indicators that measure the access to basic material needs including the percentage of the population that are unemployed or have not obtained a high school diploma. Age and labour force includes indicators describing the percentage of seniors (≥ 65 years) within the population, those not participating in the labour work force, and the dependency ratio of seniors and children to the population between ages 15-64. The fourth dimension, racialized and newcomer populations, includes indicators measuring the percentage of recent immigrants in the population as well as those self-identifying as visible minorities.[83]

Using postal codes, participants were assigned factor scores on an interval scale that corresponded to their specified dissemination area. Dissemination areas are the smallest relatively stable geographic units in Canada, each containing about 400-700 people, for which census data is collected.[81] Postal codes were linked to participants using the 2017 Postal Code Conversion File (PCCF+) created by Statistics Canada. This standardized score of each dimension is a mean value of 0 and a standard deviation of 1, where a higher score indicates higher socioeconomic inequities.[81] These factor scores have demonstrated strong temporal reliability, with 81-96% of dissemination areas remaining in the same quintile across census years, supporting their stability and consistent measurement over time.[84] As such, ON-MARG provides a robust and interpretable metric of neighborhood-level socioeconomic disadvantage in epidemiological research.[84]

4.4.2 Child Psychopathology

Child psychopathology was measured using the Emotional Behavioral Scales (EBS). The EBS is a 52-item self-reported checklist, with high clinical reliability and validity, that measures symptoms of mental disorders over the past six months and is aligned with the Diagnostic and Statistical Manual of Mental Disorders (DSM-5).[32] It uses a three-point Likert scale where each item presents a score of 0, 1, or 2 indicating responses of 'never or not true', 'sometimes or somewhat true' and 'often or very true', respectively.[32] Analyses used parent-reported responses, and child-reported responses for child participants ≥10 years of age, to assess differences in effects.[78] The responses of each child provides dimensional and categorical measurement of symptoms associated with major depressive disorder (MDD), generalized anxiety disorder (GAD), social phobia (SoP), social anxiety disorder (SAD), oppositional defiant disorder (ODD), attention-deficient hyperactivity disorder (ADHD) and conduct disorder (CD), which were then categorized as externalizing (ODD, ADHD, CD) and internalizing (MDD, GAD, SoP, SAD).[85] The internal consistency for the internalizing and externalizing scales, using child-reported data, was α=0.90-0.94, and α=0.84-0.96 for parent-reported data over all time-points.[80]

4.4.3 Child Stress

Child HCC was used as a measure of physiological stress. Hair samples were collected from each child at each time point using a standardized procedure. Each participant provided

approximately 80 strands of hair extracted from the posterior vertex of the head, as close as possible to the scalp, by research staff and by parents post-COVID.[78] Samples were then attached to a piece of folded cardstock using a paperclip, where direction of growth was indicated by an arrow drawn on the page. The prepared samples were placed in a plastic bag and mailed to investigators alongside a completed study survey. Parents completed a questionnaire regarding specific variables that are hypothesized to influence HCC, including hair washing routine, chemical treatments, medications, exposure to smoking, hair colour, and other relevant information.[78]

The first 3 cm of hair, closest to the scalp, were analyzed using enzyme-linked immunosorbent assay (ELISA).[78] Hair typically grows at a rate of 1cm per month, therefore the 3 cm sample was reflective of the three months prior to data collection.[86] Prior research found no significant difference between HCC measurements between the collection of samples at home versus in clinic.[87] Hair samples were processed following a standardized procedure encompassing hair washing, steroid extraction, and cortisol assay. The proximal 3 cm segment of hair was cut and placed in a 50mL Falcon conical centrifuge tube.[88] The sample was then washed with 12mL isopropanol twice and shaken manually for 2 minutes. The isopropanol was then discarded, and the tubs were left open to air dry for the subsequent 48 hours to ensure complete evaporation of the liquid.[88]

The dried hair samples were placed into a grinding jar with four stainless steel ball bearings and pulverized using a Retsch CryoMill that operated at 25 Hz for 3 minutes.[88] Between 30-35 mg of the ground hair powder was then weighted and transferred into a 2 mL Eppendorf tube. The powder was then mixed manually with 1 mL of 100% ethanol and rotated at room temperature at a speed of 45 rpm on an RPI Mix-All Laboratory Tube Mixer for 24 to 72 hours. After incubation, the sample was vortexed for 5 seconds and centrifuged for 15 minutes at a speed of 2800 rpm. Then, 0.8 mL of the supernatant was collected into a fresh 2 mL Eppendorf tube (supernatant tube) and left to air dry for 48 hours.[88]

After the vertexing and centrifugation, 1 mL of the supernatant was combined with the previously collected supernatant and left to dry for 48 hours.[88] The dried residue was reconstituted with 150 µL of Salimetrics Salivary Cortisol Assay Diluent, vortexed for 5 seconds, then centrifuged for 10 minutes. Cortisol concentration was then measured in duplicate by ELISA using the High Sensitivity Salivary Cortisol Immunoassay Kit (Cat# 1-3002,

Salimetrics, USA). Measurements were reported using picograms of cortisol per milligram of hair (pg/mg).[88]

4.4.4 Sociodemographics and Health Related Sample Characteristics

To collect child and family characteristics, parent participants completed questionnaires with items obtained from population-based surveys created by Statistics Canada. Data collected included age, sex at birth, years since diagnosis, corticosteroid use, frequency of healthcare professional visits per year, ethnicity, immigration status of both the child and parent, marital status (common law, widowed, separated, divorced, never married), household income (≤$29,999- ≥$150,000), and, parental education (some secondary education, secondary graduate, vocational/technical graduate, post-secondary graduate, graduate/professional school graduate).[78,80]

Statistical Analysis

Data analysis was conducted using R Studio software (Version 4.5.1) with a significance value of $\alpha = 0.05$. This conventional significance threshold was applied as it is widely accepted in psychosocial research for balancing the likelihood of type I error with the need to detect meaningful effects in observational studies.[89]

4.5.1 Research question 1: Socioeconomic disadvantage compared to the general population using one-sample t-tests

Mean factor scores of each dimension of the ON-MARG index were used to compare socioeconomic indicators in children with a CPI versus those in the general population. Scores for each of the four dimensions were compared to general population data, using one-sample t-tests, where the mean ON-MARG=0 and SD=1.[81] This method controlled for the multiple dimensions of the index, and provided a comparison of significance.

A power analysis was performed, for each ON-MARG dimension, to determine the appropriate sample size required to obtain a statistically significant effect (if one exists) compared to the general population. The calculation assumed a type I error of $\alpha = 0.05$ and desired power of 80%. Two-tailed hypothesis tests were completed to account for possible deviations in either direction of the hypothesized mean values. The mean z-score results were as

follows: households and dwellings (-0.38. SD= 0.78), material resources (-0.34, SD= 0.90), age and labour force (-0.22, SD= 0.95), and racialized and newcomer population (-0.20, SD= 0.68). With the given sample size (child: n=183, parent: n=260), the statistical power is >100%, demonstrating adequate power to detect anticipated effect sizes with a high degree of confidence. Additional details can be found in Table 3.

4.5.2 Research question 2: Socioeconomic indicators and associations to psychopathology symptoms using linear mixed models

Associations between neighborhood-level socioeconomic indicators and child psychopathology were estimated using descriptive statistics and linear mixed-effects models (LMMs) with random slopes and intercepts. The use of LMMs allowed for repeated measurements of psychopathology symptoms over time and controlled for between-child variability to capture the dynamic relationship between the various ON-MARG dimensions and internalizing and externalizing symptoms (*see Appendix 5*). Analyses were conducted using the lme4 and lmerTest packages in R.

Descriptive statistics summarized EBS scores, for both internalizing and externalizing symptoms, and mean ON-MARG index scores at each time point. Additionally, four unadjusted models were analyzed to identify the change of parent-reported and child-reported internalizing and externalizing symptoms. Each model explored the continuous natural log (internalizing/externalizing symptom score + 1) using ON-MARG dimensions, as fixed effects variables, and child baseline variability and trajectories over time, as random effects. Model assumptions for LMMs were evaluated by inspecting the distribution of residual and fitted values to ensure approximate normality and homoscedasticity. Additionally, random effects structures were assessed to confirm that variability in baseline scores and symptom trajectories over time across individuals. The outcome was log-transformed to address positive skewness in the distribution of residuals of symptom scores and to improve the interpretability and stability of model estimates.[90] Theoretical model details can be found in Appendix 7.

Eight adjusted models, using parent and child reports, were created to consider various sociodemographic and clinical covariates expected to influence the trajectory of psychopathology symptoms including child age (continuous), child sex (binary), household income (binary), parent marital status (categorical), parent education (categorical), use of

corticosteroids (binary), and frequency of health care visits (categorical). Details have been summarized in Table 1.[78,80,83,91,92] Covariates were added using stepwise addition, where sociodemographic variables were added first, followed by clinical variables. This approach allowed for the examination of the independent contributions of sociodemographic factors before accounting for potential confounding by clinical variables. Various models were analyzed to determine the best fitting model using various fit indices including the Restricted Maximum Likelihood (REML) criterion, Akaike Information Criterion (AIC), and the Bayesian Information Criterion (BIC). Lower values of all indices were considered indicative of better model fit, reflecting a balance between model accuracy and complexity. Control variables were selected based on both theoretical considerations and model fit indices to ensure robust and meaningful results. The theoretical model for this research question can be found in Figure 1.

A power analysis was performed for all four unadjusted models using ON-MARG dimensions and outcomes of psychopathology using parent- (n=263, INTobs=1198, EXTobs= 1189) and child-reported (n=185, INTobs=645, EXTobs=645) symptoms, to determine if the results of this objective will obtain a statistically significant effect. Using the SAS macro, unconditional models with a random intercept were created using the MYLIFE data.[93] Each model used the following variables for the analysis: n= 263, observations= 5, simulations= 100. After running the analysis, the null hypothesis is rejected as the objective has a power of >99%. This suggested that the analysis is highly likely to detect a statistically significant interaction (if one exists).

4.5.3 Research question 3: Moderation analysis using child stress measured by HCC

To investigate the moderating effect of child stress, natural log (child HCC +1) was added as a continuous variable and as time-varying fixed effect into the best-fitting adjusted LMMs. To account for positive skewness and to stabilize variance, child HCC was natural log-transformed prior to inclusion (*see Appendix 1-4*). Interaction terms between child stress and each ON-MARG dimension (*ON-MARG dimension*child HCC*) were included, as well as an interaction between both predictor variables and time (*ON-MARG dimension*child HCC*time*). The effect outputs of both fixed and random effects were analyzed, alongside model fit indices to understand the relationship of interest. The theoretical model for this research question can be found in Figure 2.

A power analysis was performed, using the InteractionPoweR Shiny app for analytical power, for the moderation analysis of child stress in the association between socioeconomic indicators and child psychopathology.[94] Correlation values were calculated for each ON-MARG dimension, internalizing and externalizing symptoms, and child stress, presenting a range of small effect sizes (0.024-0.21). Using a minimum sample size of n=130, the output suggests that the moderation analysis is underpowered (23%). The InteractionPoweR Shiny app is designed for cross-sectional data, so the estimated power may be an underestimate of the power for this longitudinal study. This would suggest that the results of this research question are considered exploratory and will potentially help identify important interactions for future research.

4.6 Missing data

The MYLIFE data set was analyzed to determine if missing data are missing at random (MAR), missing completely at random (MCAR), or missing not at random (MNAR). To handle missing symptom data and other available variables, multiple and mean imputation methods were used. To assess item-level missingness, the frequencies of complete-case subscale scores were compared to those that used mean imputation. Complete-case scores were calculated only when all items in the subscale were answered, whereas imputed scores were generated when at least 50% of items were completed, with missing values replacing the mean of available items. This comparison allowed for the identification of the extent to which item-level missingness occurred across the sample. Missing item-level data for self-reported questionnaires were <5% at each assessment point. In the case of missing ON-MARG index values, factor scores could not be imputed for dissemination areas where data was unavailable, therefore postal codes were assigned to the closest dissemination area available.[81]

Results

5.1 Sociodemographic and Health Related Sample Characteristics

At baseline, the sample included 263 children with a CPI (mean age: 9.4 years, 53% male). The age distribution shifted markedly over time; while 54.8% of children were under 10 years old at baseline, only 23.2% remained in that group at 48-months, reflecting natural aging within the sample. Majority of children were born in Canada (93.9%) and identified as White (88.2%), with small representations from Latin Americans (1.14%), Black (3.04%), Arabic (4.18%), South Asian (0.38%), Southeast Asian (0.38%), and First Nations (2.66%) backgrounds. Rheumatological conditions were the most common diagnosis (27.8%), followed by respiratory (20.5%), endocrine (14.4%), and gastroenterological (12.9%) disorders. Over time, the duration since diagnosis increased, with 47.9% of children having lived with their condition for at least 7.5 years by the 48-month mark, compared to the 20.6% at baseline. Corticosteroid use declined from 20.2% to 15.8%, and healthcare professional visits became more frequent. While 88.9% of children had no visits at baseline, only 75% reported none at 48 months. Participating parents were predominantly female (89.7%) and Canadian-born (84.7%). Marital status shifted, with a slight increase in married parents (77.1% to 78.1%), while common-law, separated, divorced, and never-married relationships remained mostly consistent. Parental education remained stable, with 57.3% of parents holding post-secondary degrees and 18.7% having graduate-level education. Household income showed upward mobility, with the proportion of families earning ≥$150,000 rising from 32.7% to 51.3% at 48 months, while lower-income brackets declined. Additional details can be found in Table 1.

Mean parent-reported internalizing and externalizing symptoms scores remained relatively stable from baseline (Int= 11.0 ± 9.18, Ext= 7.74 ± 6.70) to 48-months (Int= 10.0 ± 9.56, Ext= 6.52 ± 11.9), whereas child-reported internalizing symptoms increased from baseline (12.2 ± 8.78) to 48-months (13.8 ± 11.9). Additional details can be found in Table 2.

5.2 Socioeconomic Disadvantage in children with CPI vs. General Population

Analysis showed that children in the MYLIFE sample were living in areas with higher socioeconomic disadvantage compared to the general population (M=0, SD= 1). On average, scores were lowest for the households and dwellings dimension (M = -0.38, p= <0.001), followed by material resources (M = -0.34, p= <0.001), age and labour force (M = -0.22, p=

<0.001), and racialized and newcomer populations (M = -0.20, p= <0.001). Additional details can be found in Table 3.

5.3 Parent-Reported Internalizing Symptoms

In the unadjusted model, parent-reported internalizing symptoms in children slightly decreased over time (β = -0.0032, p = 0.04), and this pattern remained significant after adjusting for child and family characteristics (β = -0.0045, p = 0.02). Results of socioeconomic indicators in all 4 dimensions and their interactions with time were non-significant.

After stress was added as a moderator, the time effect was no longer significant (β = -0.08, p = 0.24), and child stress itself was not associated with outcomes of parent-reported internalizing symptoms (β = 0.02, p = 0.81). None of the ON-MARG dimensions and their interactions with child stress or time were statistically significant. The three-way interaction between age and labour force, time, and child stress (*age and labour force*time*child HCC*, β = 0.05, p = 0.06) was marginally significant, suggesting a potential moderating effect that requires further exploration. Moderating effects on each ON-MARG dimension can be seen in Figure 3.

Random effect results indicated that the variability between children remained substantial between both adjusted and unadjusted models. After adjustment, the intercept variance decreased slightly from 0.74 to 0.67, and residual variance increased marginally from 0.26 to 0.28. The variance of random slopes was small in both models (2.20e-4 unadjusted, 1.90e-4 adjusted), with moderate negative correlations between random intercepts and slopes (-0.30 unadjusted, -0.39 adjusted), which may suggest that individuals with higher baseline symptoms tended to experience slightly less decline in internalizing symptoms overtime. In the moderation model, the intercept variance increased to 0.81 and slope variance increased to 0.02, accompanied by a stronger negative correlation (-0.56). Residual variance was similar across models (0.28), reflecting stable within-person variability. Additional details can be found in Table 4.

Model fit improved with the addition of covariates and moderation terms. The best fit was observed in the moderation analysis model, indicating that the inclusion of child stress and its interaction terms provided the most optimal model for explaining the trajectories of parent-reported internalizing symptoms in children with a CPI. Details of specific REML, AIC, and BIC reports can be found in Table 8.

5.4 Parent-Reported Externalizing Symptoms

In the unadjusted model, parent-reported externalizing symptoms significantly decreased over time (β = -0.30, p = 0.04). This pattern remained significant in the adjusted model, though with an attenuated effect size (β = -0.04, p = 0.04). Between all four dimensions of socioeconomic indicators, material resource indicators were positively associated with higher externalizing symptoms in both the unadjusted (β = 0.15, p =0 .03) and adjusted models (β = 0.13, p = 0.03). Interaction effects between all socioeconomic indicators and time were mostly nonsignificant, with the exception of the interaction between households and dwellings and time (β = 0.02, p = .04). This suggested a slight increase in externalizing symptoms over time for children in areas of lower quality households and dwellings. Moderating effects on each ON-MARG dimension can be seen in Figure 3.

When child stress was added as a moderator, the effect of time was no longer significant (β = -0.02, p = 0.78) and the effect of stress was not associated with externalizing symptoms (β = 0.05, p = 0.49). No three-way interactions between socioeconomic indicators, time, and child stress were significant.

Consistent with the fixed effects, random effects analyses revealed meaningful variability in symptom trajectories across individuals. In the adjusted model, variance in intercepts slightly increased to 0.73 compared to 0.64 in the unadjusted model. Residual variance remained stable across models. Variance for random slopes of time was small in both models though slightly higher in the adjusted model with a value of 0.02. Negative correlations between random intercepts and slopes of -0.30 in the unadjusted model and -0.47 in the adjusted model were observed, indicating that individuals with higher baseline externalizing symptoms tended to show less decline over time. In the moderation analysis, similar results were produced for the random intercept and slope variance, as well as the intercept-slope correlation. Residual variance remained stable indicating similar levels of within-person variability. A summary of results can be found in table 5.

Model fit progressively improved through all three models. The unadjusted model demonstrated the poorest fit, whereas the moderation analysis model demonstrated the best fit. This suggests that the inclusion of child stress, measured by child HCC, and its interactions with ON-MARG dimensions provided the most optimal model for explaining the trajectories of parent-reported externalizing symptom trajectories. Additional details can be found in table 8.

5.5 Child-Reported Internalizing Symptoms

In the unadjusted model, the effect of time was not significant (β = 0.0002, $p = 0.95$) and remained nonsignificant in the adjusted model controlling for covariates (β = -0.0026, $p = 0.36$). Socioeconomic indicators and their interactions with time remained nonsignificant in both the unadjusted and adjusted models.

After the addition of child stress as a moderator, both time and stress presented nonsignificant results, indicating no association to child-reported internalizing scores. There were no significant interactions between socioeconomic indicators in all 4 dimensions with time or child stress, and amongst the three-way interactions (*ON-MARG dimensions*time*child HCC*). Moderating effects on each ON-MARG dimension can be seen in Figure 3.

Random effect analyses indicate moderate variability over time. In the unadjusted model, the random intercept variance of 0.64 decreased to 0.54 in the adjusted model. This suggests that some of the between-person variability was accounted for by covariates. The correlation of random slope variate also reduced slightly from -0.17 to -0.14, indicating that participants with higher baseline symptoms tended to show slightly less steep changes over time. Residual variance increased marginally, indicating the remainder of moderate unexplained variability after the addition of covariates. In the moderation model, the random intercept and slope variance increased to 0.77 and 0.04 respectively and displayed a stronger negative intercept-slope correlation. Residual variance remained stable, reflecting consistent levels of within-person variability. A summary of results can be found in Table 6.

Model fit indices improved through all 3 models. After adjusting for covariates, model fit improved with lower fit indices. The moderation analysis model further improved fit of the REML criterion. However, AIC and BIC values for the moderation model remained consistent with the adjusted model, suggesting that the addition of the moderation terms improved the model's ability to explain variance in internalizing symptoms without substantially increasing model complexity. Additional details can be found in Table 8.

5.6 Child-Reported Externalizing Symptoms

In the unadjusted model, households and dwellings were significantly associated with lower externalizing symptom scores (β = -0.33, $p = 0.001$), and this association remained significant after adjusting for covariates (β = -0.39, $p = 0.003$). Interactions between

socioeconomic indicators in all 4 dimensions and time were insignificant, even after the addition of covariates.

After the addition of child stress as a moderator, the association of households and dwellings was no longer significant ($\beta = -0.47$, $p = 0.17$). A significant three-way interaction was observed between age and labour force, time, and child stress (*age and labour force*time*child HCC*, $\beta = 0.09$, $p = 0.02$). This suggests that the relationship of age and labour force indicators and child-reported externalizing symptom trajectories over time differed depending on child HCC. Moderating effects on each ON-MARG dimension can be seen in Figure 3.

Random effects estimates highlighted that the random intercept variance in the unadjusted to adjusted model slightly decreased. This indicated moderate variability in baseline externalizing symptom scores across all participants in both models. The random slope variance in the unadjusted model was very small suggesting minimal variability in individual rates of change in externalizing symptoms over time. The random slope variance remained small for the adjusted model, however had a correlation of -0.17. This suggests that in this model, participants with higher baseline externalizing symptoms tended to have slightly less steep trajectories over time. Residual variance slightly increased from the unadjusted model to the adjusted model, indicating a slightly increase in unexplained within-person variability after adjusting for covariates. When child stress was added as a moderator, the random intercept and slope variance both increased, with a stronger negative intercept-slope correlation. Residual variance remained stable, indicating consistent within-person variability. A summary of results can be found in Table 7.

The stepwise reduction in REML, AIC, and BIC values across all three models indicated improved model fit with the inclusion of covariates and interaction terms. This suggests that both sets of variables contributed meaningfully to explain variability in child-reported externalizing symptom trajectories over time. Additional details can be found in Table 8.

Discussion

The results of this study provided important insights on the influences of environmental factors on child psychopathology in vulnerable populations. Guided by the GEI model, findings contributed to our understanding on how socioeconomic disadvantages and stress may interact in the development of internalizing and externalizing symptoms among children with CPI.[70] The use of both parent and child informants allowed for a deeper understanding of symptom expression and revealed diverging patterns that underscore the complexity of child psychopathology. Key socioeconomic and clinical variables were included to account for potential confounding, ensuring robust estimates across all models. The integration of child stress, measured by HCC, as a time-varying moderator provided a novel lens to examine how biological sensitivity to stress may amplify or buffer the psychological impact of socioeconomic adversities. These methodological choices positioned this study to make important contributions to understanding the interplay between environmental and biological determinants of mental health in medically vulnerable child populations.

6.1 Socioeconomic Disadvantage in children with CPI vs. General Population

To contextualize the environment of the MYLIFE sample, baseline comparisons were conducted to assess the levels of socioeconomic disadvantage relative to the general population. Analyses revealed that children in the sample reside in areas characterized by significantly higher levels of socioeconomic disadvantage. The most pronounced disadvantage was observed in the households and dwellings dimension, suggesting that children with a CPI live in dissemination areas with greater household instability, crowding, or residential mobility, factors known to be associated to poorer child health outcomes.[95] Low scores in material resources highlight potential barriers to basic needs such as income, transportation, and educational attainment. These factors have been linked to increased psychological stress in children and present potential for unmet health needs in children with CPI.[96,97] Similarly, disadvantage in age and labour force may reflect limited employment opportunities or economic participation in the surrounding communities, factors that can increase household stress and reduce access to supportive services.[11] Although racialized and newcomer populations scored slightly higher relative to other domains, the negative mean still points to the overrepresentation of children living in ethnically diverse and potentially socioeconomically disadvantaged neighborhoods.

Collectively, these findings explain the socioeconomic disadvantage faced by children with CPI and provide important contextual grounding for examining how environmental adversity may contribute to elevated psychopathology symptoms over time.[98,99]

6.2 Parent-Reported Internalizing Symptoms

Parent-reported internalizing symptoms are frequently used in pediatric research as they offer valuable insights into child emotional and behavioural function across home and clinical settings.[100] However, parental reports may be influenced by their own perceptions, stress levels, or mental health status, which may affect the accuracy and consistency of reporting, particularly for internalizing symptoms that are less observable.[101] Within the MYLIFE sample, parent-reported internalizing symptoms declined modestly over time; however, none of the socioeconomic indicators were significantly associated with the outcome. After adjusting for relevant covariates, socioeconomic indicators in all four ON-MARG dimensions remained insignificant as well. It is possible that none of the ON-MARG dimensions are directly related to parent-reported internalizing symptoms in this sample, suggesting that neighborhood-level disadvantage may not exert a straightforward effect on these outcomes. While children with a CPI are living in areas of higher socioeconomic disadvantage, this may not translate into elevated internalizing symptoms. Instead, individual and family-level factors, such as child sex and healthcare utilization, showed significant associations. This suggests that while neighborhood-level socioeconomic disadvantage may shape the broader environment for children with CPI, its direct impact on parent-reported internalizing symptoms may operate through more proximal mechanisms such as family dynamics, individual stress regulation, or other unmeasured contextual factors.[11,102] Existing evidence indicates that living in disadvantaged neighborhoods often affects internalizing symptoms through influences on family and caregiving processes; for instance, higher neighborhood deprivation may be associated with increased parental stress and disrupted parenting behaviours, which in turn elevates child anxiety, depression and withdrawal.[103] Furthermore, studies suggest that neighborhood disadvantage may have a stronger effect on externalizing than internalizing symptoms, partly because internalizing difficulties are less visible and more influenced by child temperament and parental mental health.[104,105] Thus, null interactions in this study may reflect that neighborhood-level disadvantage exerts its effects indirectly, through stress processes and caregiving pathways,

rather than producing a direct or additive associations with parent-reported internalizing symptoms. Together, this highlights that parent-reported internalizing symptoms may not always directly mirror disadvantage, but may be influenced by parental stress, caregiving dynamics, and child stress susceptibility.

Importantly, the random effects structure revealed considerable variability in both baseline symptoms and trajectories across individual children, supporting the decision to model both random intercepts and slopes.[106] This variability reflects the heterogenous nature of symptom presentation in children with CPI and emphasizes the value of account for within-child change over time. There were no significant interactions between socioeconomic indicators, parent-reported internalizing symptoms, and child stress. The relationship between age and labour force, time, and child stress suggests a possible conductional effect worth further exploration in future research. For example, younger children may be more insulated by parental buffering, while older children and adolescents may be more directly exposed to stressors linked to socioeconomic disadvantage, such as reduced neighborhood resources or parental employment instability.[107,108] Together, these findings highlight the need to consider developmental timing and within-child variability when examining how neighborhood-level socioeconomic disadvantage shapes the trajectories of child-reported internalizing symptoms in children with CPI.

6.3 Parent-Reported Externalizing Symptoms

Externalizing symptoms, such as aggression, hyperactivity, and oppositional behaviours, are commonly observed among children with CPI and are often reported by parents due to their disruptive and observable nature.[6] Understanding how these behaviours are shaped by socioeconomic indicators and familial contexts is critical, as externalizing symptoms are linked to long-term challenges in academic, social, and health outcomes.[109] The MY LIFE sample showed significant associations between the material resources dimension and parent-reported externalizing symptoms. This finding may suggest that children living in areas with lower access to economic and material supports, such as affordable housing, recreation programming, or nutritious diets may exhibit more disruptive or outward-directed behaviours.[27] This aligns with existing literature indicating that financial strain and unmet material needs can contribute to externalizing behaviours through increased family stress and reduced access to protective

factors.[13,110] While neighborhood-level material resource disadvantage emerged as a significant predictor of parent-reported externalizing symptoms, its interaction with time suggested that its effects remained relative stable over a 48-month period. In contrast, the significant interaction between households and dwellings and time points to a dynamic relationship in which housing-related disadvantage may have a growing or cumulative influence on parent-reported externalizing symptoms. Experiences from overcrowding, instability, or frequent residential moves, may experience an accumulation of environmental stress that exacerbates externalizing symptoms over time.[111,112] Unstable housing can disrupt routines, limit access to supporting resources, such as neighborhood programs or school continuity, and increase caregiver stress, all which may contribute to escalating behavioural dysregulation as children age.[113,114] This may be particularly vital amongst children with CPI, were consistent care and stability and especially crucial.

In the moderation model, no three-way interactions with child stress were significant. Random effects showing moderate intercept variance indicated that between-child differences, whereas minimal slope variance and a negative intercept-slope correlation suggested flatter trajectories among children with higher initial symptoms when stress is considered. These findings should be interpreted alongside evidence of low agreement between parent and child reports of externalizing symptoms, as multi-informant discrepancies are common and reflect differences in perception across settings and reporters.[100,113,114,]

6.4 Child-Reported Internalizing Symptoms

Children with a CPI may internalize stress responses, often leading to symptoms such as anxiety, withdrawal, and low mood, which may all be shaped by both individual vulnerability and environmental adversity.[115,116] Examining child-reported internalizing symptoms is essential, as children's subjective perceptions of distress may diverge from parent reports and offer unique insights into the psychosocial impacts of socioeconomic disadvantage.[100,117] Despite theoretical expectations that internalizing symptoms would fluctuate overtime in children with a CPI experiencing socioeconomic adversity, there were no significant longitudinal changes observed. This suggests relative temporal stability in child-reported internalizing symptoms across 48-months. This stability in symptoms may stem from children adapting to the ongoing demands of

CPI and socioeconomic adversity overtime, leading to the stabilization of psychopathology symptoms rather than escalation.[118]

The lack of association between socioeconomic indicators and child-reported internalizing symptoms may reflect that neighborhood-level disadvantage often influences children indirectly, operating through parent stress, caregiving quality, or family dynamics rather than shaping child symptom reports directly.[104,105] It is also possible that neighborhood-level socioecnomic indicators, as captured by the ON-MARG, are not directly related to child-reported internalizing symptoms in this sample, and any potential effects may primarily emerge through proximal family- or individual-level pathways.

Children with CPI frequently report high levels of internalizing symptoms related to illness burden, treatment demands, or feelings of social isolation, which may overshadow or mask additional effects of socioeconomic disadvantage.[6,119] Additionally, children may underreport internalizing symptoms due to limited emotional insights, developmental differences in self-reporting capacity, or social desirability bias, particularly where mental health stigma may be present.[100,120] These factors may obscure more subtle or internalized impacts of socioeconomic indicators that are not easily captured through child self-reports.

Notably, the inclusion of child stress as a moderator, while not statistically significant, improved model fit and altered the pattern of random effect variances. These changes support the relevance of incorporating biological sensitivity into psychosocial models. Existing literature shows that children's susceptibility to psychopathology is shaped by the interplay of biological and psychosocial factors.[121] For instance, heightened biological reactivity to stress, including elevated HPA axis activity, has been associated to stronger associations between socioeconomic adversity and child-reported internalizing symptoms, including anxiety and depression.[77,122] Similarly, psychosocial factors such as harsh or inconsistent parents, exposure to interparental violence and parental depression or anxiety are disproportionately concentrated in socioeconomically disadvantaged households, where chronic financial strain and limited resources exacerbate family stress.[11,123] These adversity-related stressors are consistently linked to heightened risk for internalizing symptoms as children in such contexts are more likely to experience insecure attachment relationships, reduced emotional support, and higher levels of family conflict.[113,124]

Importantly, children with elevated biological sensitivity to context may be especially susceptible to these dynamics, showing stronger associations between socioeconomic disadvantage, family stress, and the emergence of internalizing symptoms.[125,126] This aligns with the GEI model, as a CPI may function as both a biological and psychosocial diathesis, whereby children predisposed to internalizing symptoms respond differently to varying levels of external stress from socioeconomic indicators. The absence of interaction effects may reflect the complex, multifactorial nature of these pathways, emphasizing that internalizing problems do not arise solely from economic deprivation, but from the convergence of disadvantage-related psychosocial stresses and individual biological stress responsivity.

6.5 Child-Reported Externalizing Symptoms

Children with a CPI may exhibit behavioural dysregulation in response to chronic stress, resulting in outward facing symptoms such as irritability, defiance, and impulsivity. Child-reported externalizing symptom reports can be valuable, as children's self-assessments may be different from parent reports and offer distinct insights into how socioeconomic disadvantage and stress sensitivity contribute to outward behavioural functioning in this population.[127] In this study, indicators of socioeconomic disadvantage amongst households and dwellings were significantly associated to child-reported externalizing symptoms, indicating that areas with poorer housing conditions predicted higher externalizing symptoms in both the unadjusted and adjusted models. This finding reinforces a growing body of literature linking residential instability, overcrowding, and unsafe housing environments with increased behavioural difficulties in children, particularly those managing chronic illness.[27,104,128] Although the other dimensions of socioeconomic indicators were not significantly associated with the outcome, the housing-specific effect suggests that the physical living environment may be especially influential to day-to-day behavioural experiences.[129,130] For instance, residential instability has been shown to disrupt child routines, limit continuity of peer and school relationships, and increase stress exposure, which can manifest in externalizing behaviours such as irritability, aggression, and defiance.[131,132] Overcrowded or unsafe housing conditions can also intensity family conflict and reduce opportunities for privacy or self-regulation, further contributing to heightened child-reported externalizing symptoms.[27] For children with CPI, these stressors may compound the challenges of disease management, as chaotic or unstable living environments can

undermine treatment adherence, increase caregiver stress, and amplify frustration, all of which may be expressed outwardly in behavioural difficulties.[6,133]

When considering child stress as a moderator, a significant interaction emerged between age and labour force, time, and stress (*age and labour force*time*child HCC)*. This finding suggests that children experiencing elevated stress responses over time were more behaviourally affected by neighborhood-level economic disengagement, particularly through factors related to dependency ratios and reduced availability of stable adult role models or employment opportunities in surrounding environments.[134] This supports the GEI model and extends on literature on biological embedding by showing that stress physiologically does not act in isolation but interactions dynamically with socioeconomic disadvantage to shape escalating symptom trajectories.[27,67] Children exposed to chronic socioeconomic disadvantage demonstrate heightened activation of HPA axis activity, which has been linked to greater difficulties in emotional regulation and increased risk of externalizing behaviours such as aggression, impulsivity, and opposionality.[27] Furthermore, neighbourhoods with limited employment opportunities and weakened adult role models often foster environments with reduced supervision, inconsistent social norms, and heighten exposure to conflict, all of which are all known to exacerbate child-reported externalizing symptoms.[104,135] Together, these findings show that children's biological stress responses become biologically embedded within adverse structure environments, resulting in more pronounced trajectories of externalizing symptoms as reported by children themselves.

Random effects analyses further contextualize these findings. Across all models, changing intercept variance indicated meaningful between-child differences in baseline externalizing symptoms, while minimal slope variance suggested relatively stable trajectories over time. It is important to note, that in the moderation model, the intercept-slope correlation became more negatively associated, indicating that children with higher baseline symptoms exhibited flatter symptom trajectories once child stress was accounted for. This may reflect a form of stress-related behavioural rigidity, whereby chronic physiological challenges limit behavioural changes over time. These findings reinforce the value of incorporating child perspectives alongside biological and socioeconomic factors to understand externalizing behaviours.[136] Child self-reports can capture behavioural expressions, emotional states, and stress experiences that may not be fully recognized or reported by parents or caregivers,

providing a more direct account of their lived experiences.[100] For example, children may perceive peer conflict, unsafe neighbourhood conditions, or their own stress reactivity differently than adults, and these subjective experiences often have unique predictive value for externalizing symptoms.[137] By integrating child perspectives, researchers can more accurately trace how socioeconomic disadvantage and biological stress responses translate into behavioural outcomes, reducing single-informant bias and highlighting pathways that might otherwise remain obscured. They also highlight the need for interventions that not only target socioeconomic adversity, such as housing or employment instability, but also address individual-level stress vulnerability amongst children with a CPI.[138.139]

Strengths, Limitations, and Mitigation Strategies

Findings from this study advance gaps in current literature. This study was able to capture experiences of children from various CPI from many speciality clinics, to better understand the manifestation of psychopathology in Canadian pediatric populations. Additionally, the use of both parent and child reports strengths this study by capturing multiple perspectives on internalizing and externalizing symptoms, which may vary dependent on the informant's context and awareness. The use of multiple perspectives captures both observable behaviours and child internal subjective experiences to better understand detrimental experiences through socioeconomic adversity. This dual perspective also reduces single-informant bias and enhances the validity of symptom reporting, particularly for internalizing symptoms that may be underrecognized by parents but meaningfully experienced by children.

It is also important to consider the limitations of this study. First, participant recruitment comes from a paediatric hospital in one Canadian city in Southern Ontario. Included participants were also required to have sufficient English language skills which may limit the generalizability of the study results. It is also important to note the nature of the ON-MARG index. The PCCF+ relies on matching postal codes to dissemination area boundaries which are generally less accurate in rural areas. This may cause assignment of rural postal codes to multiple dissemination areas. The ON-MARG index is unable to provide factor scores for dissemination areas with incomplete data, due to incomplete census enumerations, and so this study used the assignment of the closest possible dissemination area. This may cause underrepresentation of rural and Indigenous populations. It is also important to note that each ON-MARG measure was made based on the current postal code, however this gives us no information on the length of exposure. This may underestimate associations between socioeconomic disadvantage and psychopathology outcomes. Another limitation to consider is that the MY LIFE sample is a clinical sample recruited from children actively receiving outpatient services at a pediatric hospital. This indicates that the characteristics of this sample may not reflect the general population of children who are experiencing a CPI. Thus, the sample may not be the best to understand the manifestation of psychopathology outcomes specific to children with a CPI.

Several strategies were used to mitigate the limitations of this study. Although recruitment was limited to a single pediatric hospital, the inclusion of children from diverse CPIs across multiple specialty clinics enhanced the representativeness and clinical relevance of the

sample. To address the geographic limitations of the ON-MARG index, particularly for rural areas, this study used the assignment of the closest possible dissemination area when direct postal codes were unavailable. This allowed for geographic coverage to be preserved. Additionally, incorporating all four ON-MARG dimensions allowed for a more comprehensive and multidimensional assessment of socioeconomic indicators amongst the sample. While exposure duration to socioeconomic disadvantage could not be assessed, the longitudinal design of this study over 48 months provided a robust understanding of the temporal trajectory of psychopathology symptoms.

Implications and Future Direction

The findings of this study offer several important implications for clinical practice, public health policy, and future research aimed at preventing and supporting the psychopathology of children with CPI living within socioeconomically marginalized contexts. The use of socio-biological frameworks demonstrates how socioeconomic disadvantage may interact with child stress to influence the outcome of internalizing and externalizing symptom trajectories.[134] This supports the GEI model that emphasizes that children with heightened biological sensitivity may be more vulnerable to the effects of environmental adversity.[127,] The incorporation of reports from both parents and children identified distinct and occasionally discrepant patterns between informants, thereby underscoring the necessity of employing multi-informant methodologies in clinical and research contexts to mitigate single informant bias'.[100]

It is also valuable to note that the use of all four ON-MARG dimensions and their associations with outcomes of psychopathology supports the idea that mental health interventions should not adopt a one-size-fits-all approach, but rather tailor prevention and support strategies to address the distinct challenges faced by children in socioeconomically disadvantaged neighborhoods.[141] Even though some indicators were not significantly associated with psychopathology in this study, their presence may capture contextual risks that could become more salient in other samples, developmental stages, or health contexts, underscoring the importance of continuing to consider all dimensions in future research and intervention planning. Evidence from targeted, context-specific programs shows clear links to child psychopathology outcomes. For example, home-visiting programs such as the Nurse-Family Partnership have reduced rates of childhood behavioral problems, externalizing symptoms, and later substance use by providing tailored nurse-led support to high-risk families living in areas of higher socioeconomic disadvantage.[142] Place-based models such as Promise Neighborhoods emphasize integrated educational, social, and mental health resources adapted to local contexts to address risk for emotional or behavioral symptoms.[143] Housing mobility experiments such as Moving to Opportunity, demonstrate that changes in the neighborhood environment can alter psychopathology trajectories, showing decreased depression and conduct disorder in girls but increased behavioral problems in boys, highlighting the importance of considering individual-specific implementation.[144,145] Environmental interventions, such as neighborhood greening, have also been shown to significantly reduce depressive symptoms in both youth and adults in

material resource-low areas, underscoring that addressing socioeconomic inequities, through housing stability, access to local services, and income support, can improve psychopathology outcomes.[146] Together, these examples highlight that neighborhood-level interventions can meaningfully alter the developmental course of emotional and behavioral problems, but their success depends on tailoring strategies to the specific socioeconomic, demographic, and biological contexts of the children they aim to support.

By advancing knowledge on neighborhood socioeconomic experiences, prevention and treatment for psychopathology can be enhanced using adequate screening tools and better addressed resource allocation. Furthermore, the results of this study will further reinforce the use of biological markers in child health research to understand the experiences of children that are more likely to develop internalizing or externalizing symptoms.[122,147] Using HCC as a measure of stress will contribute the limited existing Canadian research indicating increased levels of cortisol in young children may be due to social adversities including socioeconomic diversity, unemployment, material deprivation, or racialized and newcomer population concentration experiences.[148.149.150] This research will continue to pillar the implementation of HPA-axis activity assessment tools and focus on providing evidence of HCC being an effective, reliable, and non-invasive measure in child health research. Interpretations of these findings in future studies must consider children taking corticosteroids or other medications that disrupt HPA-axis function, as such treatments can alter cortisol levels dependent of psychosocial stress and potentially influence observed relationships between socioeconomic disadvantage and psychopathology outcomes. By integrating geospatial, psychosocial, and biological data, this study offers a scalable model for future research and surveillance programs aiming to identify at-risk subgroups and inform targeted, developmentally sensitive interventions.[151]

Conclusion

This study revealed that children with CPI disproportionately reside in neighborhoods characterized by greater socioeconomic disadvantage compared to the general population. Findings of this study suggest that socioeconomic indicators, mainly in disadvantage related to households and dwellings and material resources, are pervasive in predicting internalizing and externalizing symptoms amongst children with CPI. LMM analyses provide evidence to support the interplay of individual health, sociodemographic, and environmental factors in the outcomes of psychopathology. The inclusion of time-varying child stress, using HCC, further illuminated how physiological reactivity may moderate the relationship between socioeconomic indicators and symptom expression. Differences observed between parent and child reports also emphasized the value of multi-informant approaches in accurately capturing children's emotional and behavioral health. Taken together, these findings support the use of societal, economical, and environmental factors on physiological stress responses to better understand pediatric psychopathology and call for targeted, context-sensitive interventions.

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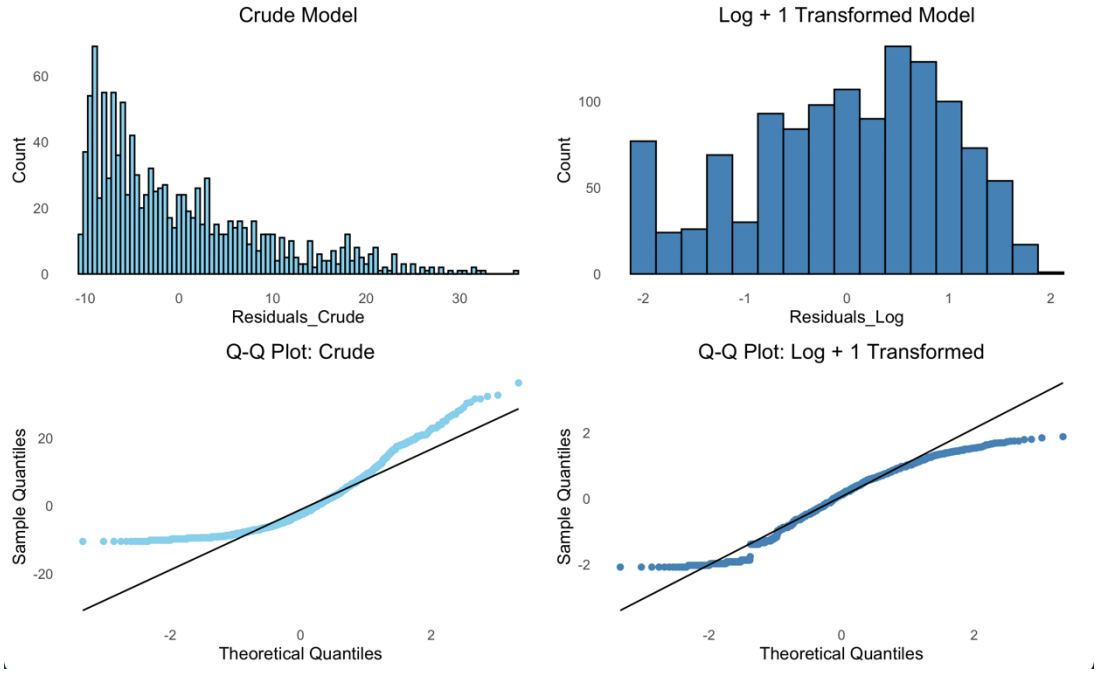
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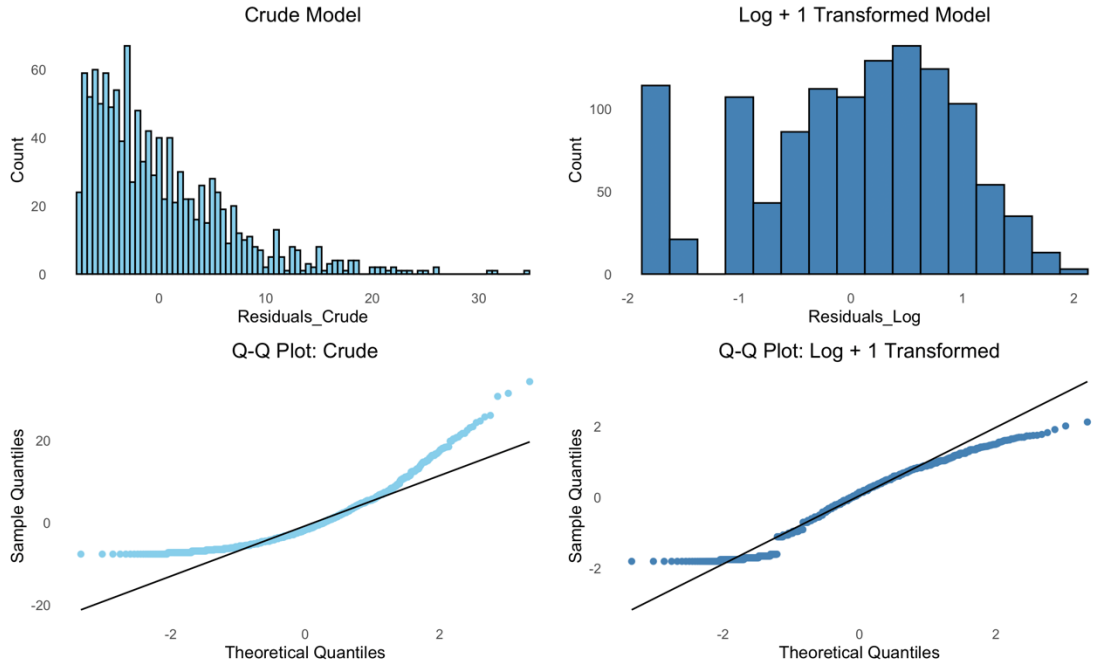
Appendix

Appendix 1. Distribution of Residuals in Crude and Log-Transformed Parent-Reported Internalizing Symptom scores.



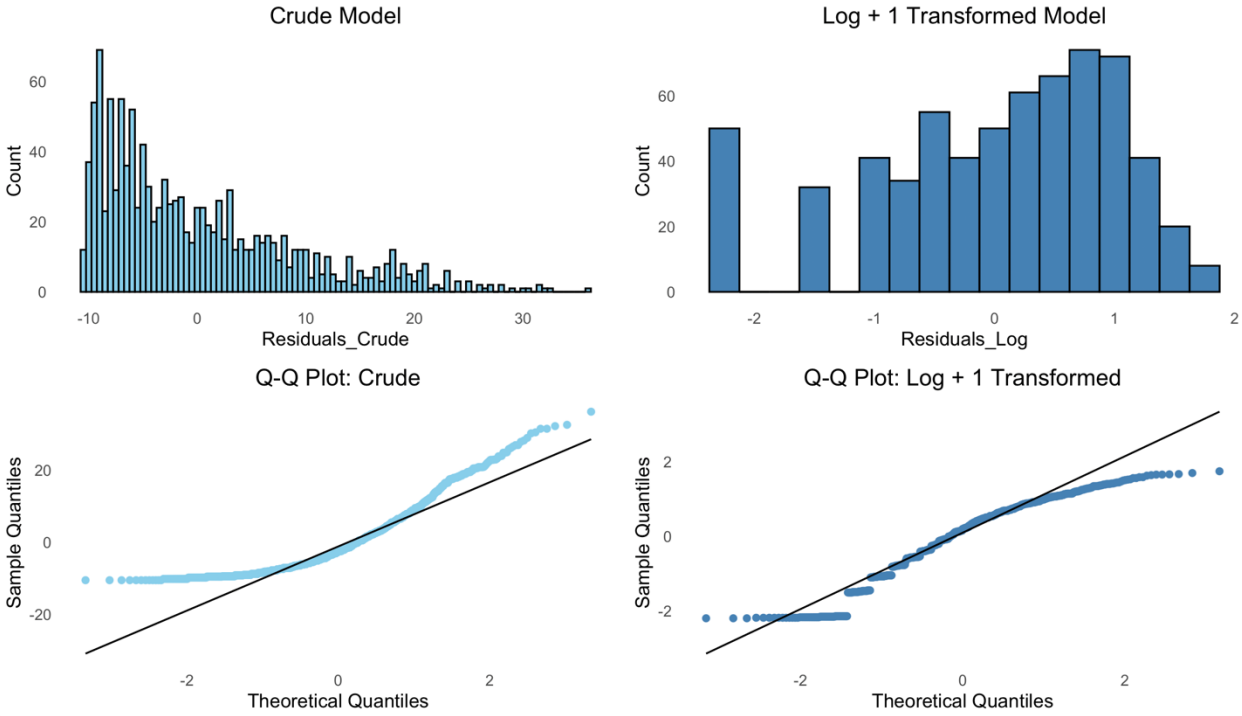
Histograms and Q-Q plots displaying the distribution of residual from crude and log-transformed LMMs for parent-reported internalizing symptom scores. Log transformation improved normality of residuals and reduced right skew, supporting its use to meet model assumptions of homoscedasticity and normal error distribution.

Appendix 2. Distribution of Residuals in Crude and Log-Transformed Parent-Reported Externalizing Symptom scores.



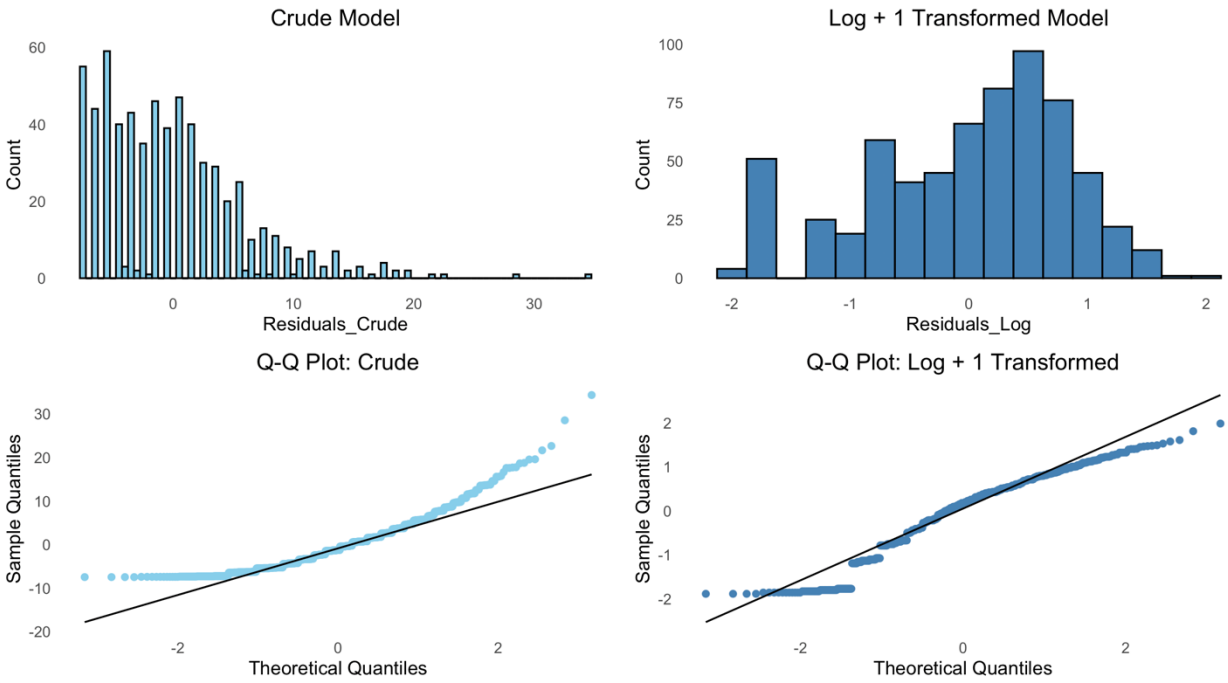
Appendix 2 shows histograms and Q-Q plots displaying the distribution of residual from crude and log-transformed LMMs for parent-reported externalizing symptom scores. Log transformation improved normality of residuals and reduced right skew, supporting its use to meet model assumptions of homoscedasticity and normal error distribution.

Appendix 3. Distribution of Residuals in Crude and Log-Transformed Child-Reported Internalizing Symptom scores.



Appendix 3 shows histograms and Q-Q plots displaying the distribution of residual from crude and log-transformed LMMs for child-reported internalizing symptom scores. Log transformation improved normality of residuals and reduced right skew, supporting its use to meet model assumptions of homoscedasticity and normal error distribution.

Appendix 4. Distribution of Residuals in Crude and Log-Transformed Child-Reported Externalizing Symptom scores.



Appendix 4 shows histograms and Q-Q plots displaying the distribution of residual from crude and log-transformed LMMs for child-reported externalizing symptom scores. Log transformation improved normality of residuals and reduced right skew, supporting its use to meet model assumptions of homoscedasticity and normal error distribution.

Appendix 5. Theoretical LMM for both Parent and Child-reported Internalizing and Externalizing Symptom Outcomes

$$Y_{ij} = \beta_{0i} + \beta_1(Visit)_j + \beta_2(Households\ and\ Dwellings)_i + \beta_3(Households\ and\ Dwellings\ x\ Visit)_{ij} + \beta_4(Material\ Resources)_i + \beta_5(Material\ Resources\ x\ Visit)_{ij} + \beta_6(Age\ and\ Labour\ Force)_i + \beta_7(Age\ and\ Labour\ Force\ x\ Visit)_{ij} + \beta_8(Racialized\ and\ Newcomer\ Populations)_i + \beta_9(Racialized\ and\ Newcomer\ Populations\ x\ Visit)_{ij} + u_{0i} + u_{1i}(Visit)_j + \epsilon_{ij}$$

Where,

Y_{ijt}= natural log-transformed internalizing or externalizing psychopathology symptom score for individual i at time point j, derived from either parent or child

β_{0i} = individual-specific intercept (baseline psychopathology score) for individual i.

β_{1j} = the effect of time (visit 1-5) on the outcome, averaged across all individuals.

$\beta_{2i,}\ \beta_{4i,}\ \beta_{6i,}\ \beta_{8i}$ = main effect of ON-MARG dimensions (*households and dwellings, material resources, age and labour force, racialized and newcomer populations*) on psychopathology score for individual i over time.

$\beta_{3i,}\ \beta_{5i,}\ \beta_{7i,}\ \beta_{9i}$ = the time-varying effect of ON-MARG dimensions (*households and dwellings, material resources, age and labour force, racialized and newcomer populations*) on psychopathology score for individual i over time.

$u_{0i} \sim N(0, \sigma^2_{u0})$ = the subject-specific random intercept capturing unobserved heterogeneity in baseline symptoms of sample.

$u_{1i} \sim N(0, \sigma^2_{u1})$ = the subject-specific random slope for time, allowing variability in symptom trajectories over time.

$\epsilon ij \sim N(0, \sigma^2)$ = residual error term capturing intra-individual variation not explained by fixed or random effects, as the model is assumed to be independent and normally distributed.