

**Taking Stock and Moving Forward: Synthesizing Ethnic-Racial Diversity in Canadian  
Social Genomics Research**

Yao Zheng, Ph.D., Zachary Meyer, M.S., Yiqun Wu, B.S.

Department of Psychology, University of Alberta, AB, Canada

December 17<sup>th</sup>, 2023

Correspondence concerning this article should be addressed to Yao Zheng, Ph.D., Department of Psychology, University of Alberta, P-217 Biological Sciences Building, Edmonton, AB, Canada T6G 2E9. (tel) 780-492-0187. Email: [yao.zheng@ualberta.ca](mailto:yao.zheng@ualberta.ca)

This work is funded by a Knowledge Synthesis Grant from the Social Sciences and Humanities Research Council of Canada in partnership with Genome Canada (Genome Alberta).

None of the authors have any potential conflicts of interest, real or perceived, or financial disclosures to report.

## Table of Contents

<b>Executive Summary</b> .....	4
<b>Background and Objectives</b> .....	4
<b>Results</b> .....	4
<b>Key Messages</b> .....	4
<b>Methodology</b> .....	5
<b>Background</b> .....	6
<b>Social genomics</b> .....	6
<b>The importance of racial-ethnic diversity in social genomics</b> .....	8
<b>Immigration and evolving ethnic-racial compositions in Canada</b> .....	10
<b>Objectives</b> .....	11
<b>Methods</b> .....	11
<b>Registration, search strategy, inclusion and exclusion criteria</b> .....	11
<b>Data extraction and quality assessment</b> .....	12
<b>Results</b> .....	13
<b>Implications</b> .....	16
<b>Study characteristics: common designs and outcomes</b> .....	17
<b>Geographic and demographic trends in Canadian social genomics research</b> .....	19
<b>Conclusions</b> .....	20
<b>Knowledge Mobilization Activities</b> .....	21

Ethnic-Racial Diversity in Canadian Social Genomics Research	3
<b>Bibliography</b> .....	22
<b>Appendices</b> .....	34

## **Executive Summary**

### **Background and Objectives**

Social genomics can identify modifiable environmental targets to offset genetic risks, prevent adverse socioemotional outcomes, and promote well-being and resilience among vulnerable populations. Contemporary literature on human genetics research, including social genomics, has predominantly centered on populations of European descents. The inclusion of diverse ethnic/racial populations into social genomics could further elucidate the mechanisms that contribute to socioemotional developmental outcomes across the life cycle under various environmental experiences and broader societal or cultural contexts. This project aims to perform a systematic review to survey ethnic/racial diversity in Canadian social genomics research.

### **Results**

Only a small number of studies examined outcomes that fell into the broad definition of social genomics. The majority of them focused on psychiatric symptoms. Barely any studies explored how different environmental experiences and broader societal or cultural contexts could amplify genetic potentials or suppress genetic risks. Most studies examined White populations, regardless of study design, province origin, study outcome, or sample age. Many studies failed to report any relevant information on ethnic/racial composition at all. It is more challenging to obtain intersectionality information (e.g., ethnic/racial minority women). Regardless of study designs or other study characteristics, the average quality of ethnicity/race reporting as well as the quality rating across studies was low to medium at best.

### **Key Messages**

There is currently a limited number of Canadian social genomic research over the past three decades, demonstrating a major research gap compared to research conducted in the US.

We urgently need more research attention in future pertinent studies that extend from merely investigating psychopathology and focus on a wide range of socioemotional and behavioral outcomes. Particularly, research attention is sorely needed in understanding how different broader societal and cultural contexts could modify genetic potentials among different groups. Moreover, future research should take a developmental and life cycle approach to better understand how genes and environment contribute to human development with the aid of longitudinal designs. Marginalized ethnic/racial minority groups are poorly represented in extant literature. Future research must extend from investigating predominantly White adult populations onto ethnically/racially diverse child, adolescent, and adult populations. Rather than indiscriminately aggregating marginalized ethnic/racial minority groups into the larger White samples for analysis, more research should set out to specifically include and understand marginalized racial/ethnic minority groups, in recognition of their differences in genetic predispositions as well as their unique sociocultural systems and environmental experiences. Greater transparency and intentionality are needed in ethnicity/race and intersectionality reporting in future Canadian social genomics research.

### **Methodology**

A systematic literature review was performed across Web of Science Core Collection, Web of Science BIOSIS Citation Index, PubMed, Medline, PsycINFO, Embase, and Scopus. Included studies had to be published, peer-reviewed empirical investigations that met pre-established relevancy criteria on the study population, research method, and outcomes assessed, examining relevant social genomics outcomes using either quantitative or molecular genetics designs. Only studies containing unique samples were retained for review across study designs.

## **Background**

### **Social genomics**

Different from medical genetics that focuses more on the evaluation, diagnosis, and treatment of medical genetic diseases (e.g., birth defects, cancer genetics, prenatal diagnosis) or agricultural genomics (e.g., food systems and consumptions), social genomics aims to clarify the interplay between social experiences and broader societal and cultural contexts and genetic potentials within people (Mills & Tropf, 2020; Robinson et al., 2005; Shanahan, 2013).

Centering on people, social genomics primarily focuses on socioemotional and behavioral outcomes (e.g., self-esteem, parent–child relationship, substance use) that largely fall outside the medical or agricultural domains. Social genomics can identify modifiable environmental targets (e.g., parenting) to offset genetic risks, to prevent adverse outcomes (e.g., substance abuse), and to promote well-being and resilience among vulnerable populations (Leve et al., 2018).

Particularly, social genomics can elucidate how social processes (e.g., institutional, cultural norms and beliefs, systemic racism and discrimination) can suppress or augment underlying genetic potentials (Shanahan & Hofer, 2005).

Decades of accumulated evidence from social genomics research has robustly demonstrated that genes influence every aspect of our lives, ranging from emotional, cognitive, psychological to behavioral processes (Plomin et al., 2016; Polderman et al., 2015).

Consequently, a large portion of the commonly found associations in family studies and social sciences more broadly (e.g., harsh discipline and child externalizing problems; Rothbaum & Weisz, 1994) can be attributed to genetic confounding: parents who are genetically at risk for externalizing problems also tend to use harsh discipline and pass on genes to their offspring. In the absence of true experimental manipulation (i.e., randomization), as in the case of the majority

of social sciences, the twin design represents a natural quasi-experiment with the capacity to disentangle genetic effects from environmental effects by comparing similarity in various traits between identical twins, who share all of their genes, and fraternal twins, who share on average half of their segregating alleles (Leve et al., 2018; Pingault et al., 2018; Plomin et al., 2016). Thus, the twin design represents an ideal way to bring us one step closer toward causal inference in the role of genes and environment by simultaneously controlling for genetic and family-level environmental confounding (Pingault et al., 2018; Plomin et al., 2016). Besides twin designs, social genomics also employs various other techniques and designs. For instance, genome-wide association studies (GWAS) use hypothesis free association testing to detect links between genetic variants (single nucleotide polymorphisms, SNPs) across the entire genome and traits in large samples (Tam et al., 2019). Candidate gene approaches involve identifying and testing the influences of a few selected or pre-specified genetic variants on a given trait (Tabor et al., 2002). Polygenic score approaches compile genetic variants linked with a given trait derived from GWAS studies into a single score to test their cumulative influences (Anderson et al., 2019).

Extant Canadian social genomics research has made tremendous contributions to advancing our understanding in gene-environment interplay in a wide variety of socioemotional and behavioral outcomes that carry profound societal impacts. For instance, twin studies have indicated that unique (i.e., person-specific) environmental influences primarily contribute to the development of health anxiety in adult Canadian twins, suggesting that health anxiety is predominately a learned outcome rather than being genetically transmitted (Taylor et al., 2006). Candidate gene research has implicated polymorphisms in the SNAP25 gene with susceptibility towards hyperactive and inattentive behaviors in Canadian families (Feng et al., 2005). Polygenic score research exploring the interaction between early cannabis use and genetic risk

for schizophrenia on brain maturation suggests that early cannabis use hastens cortical thinning in Canadian adolescent males who are at high polygenic risk for schizophrenia (French et al., 2015).

### **The importance of racial-ethnic diversity in social genomics**

Much like the social sciences in general, contemporary literature on human genetics research has predominantly centered on populations of European descent (Polderman et al., 2015). Canadian social genomics research is no exception. For instance, in the aforementioned candidate gene study (Feng et al., 2005), approximately 95% of the sample were White, while the sample in the polygenic study (French et al., 2015) were entirely White. Taylor et al. (2006) even failed to report the ethnic-racial composition of their twin sample.

Scant social genomics research has systematically included and represented Canadian marginalized ethnic-racial populations. One “implicit” (mis)assumption is that findings from White populations of European descent “naturally” generalize to marginalized ethnic-racial populations. Such a misassumption ignores the differences in genetic predispositions of marginalized ethnic-racial populations, as well as their unique sociocultural systems and environmental experiences that could dynamically modify the expression of genetic predispositions. Specifically, racial-ethnic diverse populations follow different genetic ancestries and possess distinct inherited genetic predispositions, which could create differential genetic vulnerabilities in different environmental contexts for evolutionary purposes. For instance, many East Asians develop facial flushing due to the buildup of acetaldehyde as a result of genetic variations responsible for faulty production of enzymes that process alcohol (Lee et al., 2014). Additionally, genetic variations that grant resistance to malaria can be found in sub-Saharan regions where malaria is prevalent (Hedrick, 2011). Moreover, the substantial sociocultural



differences between racial-ethnic diverse populations could differentially amplify or offset their genetic predispositions. For instance, considering that parent–child and peer relationships are heavily influenced by culturally influenced socialization processes (Chen & French, 2008), cross-cultural differences in these social processes may result in distinct gene–environment interplay patterns in ethnic-racial diverse populations.

The inclusion of diverse ethnic-racial populations into social genomics could further elucidate the mechanisms that contribute to social developmental outcomes across the lifecycle under various environmental experiences and broader societal contexts. Human genetics research has been increasingly calling for greater racial-ethnic diversity initiatives to address this research disparity (Sirugo et al., 2019). Given the unique environmental experiences (e.g., systemic racism and discrimination) that marginalized ethnic-racial populations encounter on a daily basis, the US National Institute of Health has been increasingly funding more genomics research that specifically targets marginalized ethnic-racial populations.

A small yet burgeoning body of literature has made progress accordingly in these regards. For instance, examinations of the interplay between polygenic risk for alcohol use disorder and experienced discrimination on alcohol use problems suggest greater representation in social genomic research is needed to understand the role of gene–environment interactions in alcohol use problems in Black youth (Su et al., 2022). Moreover, polygenic risk for depression has been associated with elevated risk for major depression disorder in Mexican-origin adolescents, demonstrating the utility of polygenic approaches in identifying genetically at-risk Latino youth (Rabinowitz et al., 2020). Recent studies have also revealed distinct genetic and environmental contributions to the development of Asian adolescents' ADHD, anxiety, and depressive symptoms (Zheng et al., 2016, 2019) from typical findings in European populations.

Notably, the relative environmental contributions originated from the shared family, neighborhood, and community experiences are larger in Asian populations who follow a collectivistic culture that emphasize interpersonal relationships, family harmony, and parent–child relationships (Chen & French, 2008; Chen et al., 2015) as opposed to the individualistic culture in most Western countries. These findings suggest that it is imperative to specifically include marginalized ethnic-racial populations in social genomics to better promote well-being among these vulnerable groups.

### **Immigration and evolving ethnic-racial compositions in Canada**

Contrary to the growing emphasis on social genomics research in racial-ethnic diverse populations advocated in the US, there is comparably a lack of concerted effort to promote such initiatives in Canadian marginalized ethnic-racial populations. One looming and crucial literature gap concerns the relative lack of information regarding the racial-ethnic diversity in extant Canadian social genomics research. It is imperative to take stock of the strengths and limitations in the pertinent literature, to steer the next generation of Canadian social genomics research.

Canada is in a unique position to tackle such research gaps compared to the US. Immigration constitutes an evolving global phenomenon that constantly and rapidly shapes the Canadian demographic landscape, notably its ethnic-racial composition, which has far-reaching implications for Canadian society. Contemporary Canadian immigration and multicultural integration policies are comparatively more supportive than those in many Western societies (Trebilcock, 2019). Pro-immigration attitudes and initiatives have resulted in substantial increases in immigrant populations in recent years (Hiebert, 2016). Roughly 23% of census respondents in 2021 reporting being either a landed immigrant or permanent resident (Statistics Canada, 2022a). This rising trend is only expected to continue, as the proportion of immigrants

in Canada are estimated to increase to approximately 25–30% by 2041 (Statistics Canada, 2022a). Since 2010, immigrants coming to Canada have primarily come from Asia (62%), followed by Africa (15%), Europe (10%), and Central and South America (9%; Statistics Canada, 2022a). Marginalized ethnic-racial populations currently constitute a sizeable portion of the Canadian population: South Asian (2.6 million people; 7.1% of the population), Indigenous (1.8 million; 5.0%), Chinese (1.7 million; 4.7%), and Black (1.5 million; 4.3%). The remaining 9.1% of visible minorities being: Filipino, Arab, Latin American, Southeast Asian, West Asian, Korean, and Japanese (Statistics Canada, 2022b, 2022c).

### **Objectives**

To break systemic barriers in Canadian social genomics research, to include, incorporate, and invite marginalized ethnic-racial populations historically excluded from these research foci into the modern research agenda, synthesized evidence on the ethnic-racial diversity in extant Canadian social genomics research is sorely needed. Hence, the primary research question of the current study is to understand the extent to which marginalized ethnic-racial populations are included and represented by extant Canadian social genomics research, especially in the last two decades. Specially, what is the proportion (%) of various marginalized ethnic-racial populations in each empirical study and over all studies? How do these proportions intersect with other key factors (e.g., sex and age)? Are there any inequities and biases in investigated social outcomes (e.g., marginalized ethnic-racial populations are disproportionately examined from a deficit [vs. competence] approach on negative and adverse [vs. positive and beneficial] outcomes)?

### **Methods**

#### **Registration, search strategy, inclusion and exclusion criteria**

The current systematic review was preregistered with the International Prospective

Register of Systematic Reviews (PROSPERO) network and was assigned the registration number CRD4202343140. The systematic review adhered to the recommendations and guidelines of the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA-S) statement, which requires transparent reporting of systematic reviews and meta-analyses (Rethlefsen et al., 2021). A comprehensive search strategy was performed through seven databases: the Web of Science Core Collection, Web of Science BIOSIS Citation Index, PubMed, Medline, PsycINFO, Embase, and Scopus. Studies considered for inclusion had to be published, peer-reviewed empirical investigations that met pre-established relevancy criteria on the study population, research method, and outcomes assessed, examining relevant social genomics outcomes (e.g., self-esteem, parenting–child relationship, substance use) using either quantitative (e.g., twin) or molecular (e.g., GWAS, candidate gene, polygenic risk) genetics designs.

Exclusion criteria for the current systematic review included studies focusing on medically relevant outcomes (e.g., congenital disabilities, cancer genetics) and studies examining Canadian samples residing abroad. As the review focused on assessing ethnic/racial inclusion, only studies containing unique samples were retained for review across study designs, as duplicate samples would not provide additional relevant information. Key terms included in database search queries included “Canada” or “Canadian,” province-specific search terms (e.g., Ontario, Quebec, Alberta) and “gene” or “genomics” or “twin,” “GWAS” “candidate genes” or “polygenic.”

### **Data extraction and quality assessment**

Figure 1 displays the number of studies screened at each review stage and the reasons for excluded articles. Two independent reviewers assessed all articles that could be relevant for their

titles and abstracts. Full-text copies were obtained for all articles that met the initial inclusion criteria, and any discrepancies between reviewers were resolved first through discussion among reviewers or by a third reviewer if necessary. Both reviewers conducted the data extraction process independently, followed by consensus agreement and subsequent approval by the principal investigator. The studies eligible for review had various data extracted, including sample characteristics such as mean age, age ranges, sex/gender distribution, ethnic-racial composition (e.g., White, Asian, Black, Latino, Indigenous), geographic regions covered, study design and outcomes, as well as years of publication and total sample sizes.

Since there was no pre-established protocol suitable for the purpose of the current study, a specific rating framework was created to evaluate ethnic-racial inclusivity in included genetically informed studies. Four criteria were considered important to assess the quality of the studies with respect to sample demographic reports practices, including the reporting quality of 1) sample characteristics, 2) racial-ethnic compositions, 3) sample representativeness, and 4) considerations of potential intersections between ethnicity/race and sample characteristics in the study design (e.g., ethnicity by gender). Criteria were rated on a 3-point scale (1 = inadequate reporting/consideration, 2 = partial reporting/consideration, 3 = comprehensive reporting/full consideration). Both reviewers independently scored each criterion and an average score across reviewers was created for each criterion. The scores across the four criteria were then averaged to produce a composite quality assessment score for each study within the framework of the rating system.

## **Results**

During the literature identification phase of the review, a total of 2,290 studies were initially retrieved across the seven databases. Among them, 1,254 studies were identified as

duplicates by Covidence and additional 10 studies were deemed duplicates and removed manually by reviewers, leaving 1,206 studies eligible for title and abstract screening. Of these, 936 studies did not meet eligibility criteria during title and abstract screening and were subsequently excluded, leaving 90 studies for full-text review. Following the full-text review process, 63 studies were excluded, primarily due to wrong study designs or duplicate samples, as indicated in Figure 1. Ultimately, a total of 29 studies were deemed eligible for inclusion in the current review. It is important to note that some of the studies included for review incorporated multiple study designs (e.g., GWAS/polygenic risk) and pooled samples across multiple provinces (e.g., Ontario and Quebec), as such, counts of study design, geographic region, and outcomes are reported across each design where applicable.

Candidate gene studies were the most frequently employed research design ( $n = 15$ ; see Figure 2) with an average sample size of 837 and sample range of merely 77 to 4,386. Candidate gene studies were conducted across many geographic regions, including Alberta ( $n = 3$ ), British Columbia ( $n = 1$ ), Newfoundland ( $n = 1$ ), Ontario ( $n = 4$ ), and Quebec ( $n = 7$ ). Most studies focused on outcomes in adulthood ( $n = 9$ ; see Figure 3). Study outcomes included ADHD ( $n = 2$ ), opioid use disorder ( $n = 1$ ), mania and depression ( $n = 1$ ), bipolar disorder ( $n = 3$ ), anxiety ( $n = 1$ ), suicide completion ( $n = 1$ ), social orientation ( $n = 1$ ), pathological gambling ( $n = 1$ ), depressive symptoms ( $n = 1$ ), schizophrenia ( $n = 1$ ), dyslexia ( $n = 1$ ), and food addiction ( $n = 1$ ). On average, the reporting of ethnicity/race composition and study quality were medium ( $M_s = 2.27$  and  $2.02$ , respectively).

Seven studies employed genome-wide association designs (Figure 2) with an average sample size of 3,237 and sample range of 624 to 16,361. Frequency of GWAS studies by geographic region included national ( $n = 1$ ), Newfoundland ( $n = 1$ ), Ontario ( $n = 2$ ), Quebec ( $n =$

2), and Not reported ( $n = 1$ ). Genome-wide association studies most commonly assessed adult samples as well ( $n = 5$ ; Figure 3). Study outcomes included suicide completion ( $n = 1$ ), anxiety and pain problems ( $n = 1$ ), food addiction ( $n = 1$ ), reading disabilities and genetic risk for neurodevelopmental disorders ( $n = 1$ ), autism spectrum disorder ( $n = 1$ ), bipolar ( $n = 1$ ), and PTSD ( $n = 1$ ). Average ethnicity/race reporting quality was medium ( $M = 2.2$ ) and average study quality was low ( $M = 1.8$ ). An additional study was not formally classified into a specific category given its unique study design using an epigenome wide approach, which assessed epigenomic indices of aging as related to peer victimization, depression, and suicidal ideation in children from Quebec. Ethnicity/race reporting and study quality for this study were low ( $M_s = 1$  and  $1.5$ , respectively).

Seven studies utilized the polygenic risk/score designs (Figure 2) and had an average sample size of 2,848 and sample range of 145 to 16,361. Polygenic risk geographic region frequencies were as follows: national ( $n = 1$ ), Manitoba ( $n = 2$ ), Ontario ( $n = 2$ ), Quebec ( $n = 2$ ). Like candidate gene research (Figure 3), most polygenic risk studies focused on outcomes in adulthood ( $n = 4$ ). Study outcomes included reading disabilities and neurodevelopmental disorders ( $n = 1$ ), PTSD ( $n = 1$ ), bipolar and schizophrenia ( $n = 1$ ), ADHD ( $n = 1$ ), multiple sclerosis and depression ( $n = 1$ ), psychiatric comorbidity in irritable bowel disease ( $n = 1$ ), social withdrawal and genetic risk for mental health disorder ( $n = 1$ ). Ethnicity/race reporting quality was low ( $M = 1.57$ ), and average study quality was also low ( $M = 1.54$ ).

Three twin studies with unique samples were included for review (Figure 2) with an average sample size of 1,163 and sample range of 614 to 1,578 and used samples from British Columbia ( $n = 1$ ), national ( $n = 1$ ), and Quebec ( $n = 1$ ). Unlike other study designs, adolescent and adult developmental periods (Figure 3) were equally assessed ( $n = 2$ ) for both periods,

respectively. Reactive and proactive aggression ( $n = 1$ ), antisocial personality traits and alcohol misuse ( $n = 1$ ), and obsessive compulsive ( $n = 1$ ) outcomes were assessed across twin studies, among many other relevant social genomic outcomes examined in other excluded studies using the same samples (due to sample duplication). Ethnicity/race reporting and overall quality score for twin studies were both medium ( $M_s = 2$  and  $2.08$ , respectively).

### **Implications**

The current study represents the first effort to quantify the extent to which marginalized ethnic/racial populations have been systematically included and considered in Canadian social genomics research in different types of genetically informed research designs that has been conducted on Canadian populations over the last three decades. Besides highlighting prominent gaps in the literature, the current study provides insights into the demographic and geographic trends present in contemporary Canadian social genomics research. Moreover, the current review reveals the current poor state of sample reporting practices across various genetically informed designs, highlighting the need for greater transparency and more detailed ethnicity/race reporting practices in future research. The current findings offer several important implications to policy and research as discussed in the following sections.

First and foremost, there is currently a limited number of Canadian social genomics research over the past three decades, demonstrating a major research gap compared to research conducted in the US. By directly focusing on proximal psychosocial processes as well as distal societal and cultural contexts, social genomics has the capacity to promote well-being and resilience, and prevent adverse outcomes, especially among vulnerable populations. Therefore, we urge more policy in directing research funding and priorities to social genomic research. Moreover, despite growing calls for more racial-ethnic diversity initiatives in human genetics



research (Sirugo et al., 2019), few social genomics studies intentionally incorporate marginalized ethnic/racial groups as a part of their research focus. Moreover, marginalized ethnic-racial populations are often simply aggregated into the larger White samples for analysis. Quantifying the extent to which racial/ethnic minorities have been systematically included into contemporary social genomics literature constitutes a first step to addressing the growing diversity initiatives within the field. The current findings further highlight the severe lack of relevant social genomic research that specifically or exclusively focus on ethnic-racial minority populations. We call for more relevant policies and funding priorities that especially encourage and promote social sciences research with genetically informative designs to target marginalized ethnic-racial groups.

### **Study characteristics: common designs and outcomes**

Despite an initial large return of literature research through seven databases, only a small number of studies examined outcomes that fell into the broad definition of social genomics. Among those studies, only 29 Canadian social genomics studies used unique and independent Canadian samples that were eligible for subsequent inclusion and analysis. Candidate gene (i.e., single gene approach) studies represented the most frequently used study design. Studies using polygenic risk score designs (i.e., multiple genes) and genome-wide association designs (i.e., whole genome sequencing) were used to a lesser degree. Only three studies used the twin design with unique Canadian samples, and one study used a design that could not be categorized into the other study designs (i.e., epigenome-wide methylation). Sample sizes in these studies tended to be large compared to common social science studies, on average exceeding one thousand participants. Only one twin study design employed longitudinal designs where they followed participants over multiple years. Genetically informative longitudinal designs offer the unique

opportunity to unravel how genes and environment contribute to the onset, persistence, and desistence of psychosocial development with a lifecycle approach. Thus, much more longitudinal research is needed in future research practices.

Regardless of study designs, the majority of included studies focused on psychiatric or psychopathology symptoms as their investigated outcomes, included ADHD, PTSD, anxiety and depression, schizophrenia, substance use disorder, problem gambling, suicide, neurodevelopmental disorders, obsessive-compulsive disorder, bipolar disorder, among others. These investigated outcomes only covered a rather small and limited section of potential outcomes that are frequently investigated in social genomics research, leaving out numerous other important and socially-relevant outcomes, such as parent-child relationships and peer relationships, parenting, self-esteem, personality, and academic achievement. In addition, a focus on these adverse or negative outcomes are congruent with the conventional research practices originated from a deficit view. We strongly encourage a competence-based perspective towards genetic and environmental influences as implemented in social genomics to actively elucidate how genes and environment could bring out competence, capacity, and prosperity in human development. Moreover, and more importantly, few studies explored how different environmental experiences and broader societal or cultural contexts could amplify genetic potential or suppress genetic risks associated with these outcomes, the exception being in studies using twin designs. This is a severe under-utilization of one of the major advantages of social genomic research. We encourage more research practices in taking a social determinant perspective to examine modifiable environmental targets that could inform future intervention in both harm reduction as well as wellness promotion.

**Geographic and demographic trends in Canadian social genomics research**

Most extant Canadian social genomics studies focused on populations from Quebec, a predominately European founder population. Other Canadian provinces that contributed eligible studies included Ontario, Alberta, British Columbia, Manitoba, and Newfoundland. Relevant studies using national data or samples pooled across multiple Canadian provinces constitute a minority of social genomics studies conducted in Canada. It would greatly enhance population inclusion and representation if future research comes more from other less or rarely investigated provinces or areas. National collaboration or consortium efforts in this regard would achieve such a goal. In addition, the included studies overwhelmingly examined adult populations, with much less attention given to earlier developmental periods (e.g., early childhood, adolescence) except for studies using twin designs. People experience different environmental contexts across their life stages, which could trigger or suppress the expression of different genetic predispositions. Hence, a developmental and lifecycle approach towards social genomic research could greatly serve the research community as well as the society.

Relating to one of the main goals of the current study, the current findings revealed that most extant studies examined White populations, regardless of study design, province origin, study outcome, or sample age. Furthermore, many studies failed to report any relevant information on ethnic/racial composition at all, making it more challenging to draw meaningful insights about the extent to which demographic intersections (e.g., ethnic/racial minority women) were intentionally addressed in contemporary social genomics research. Regardless of specific study designs or other study characteristics, the average quality of ethnicity/race reporting, as well as the quality ratings across studies were low to medium, suggesting that the reporting of informative sample characteristics (e.g., age, sex, ethnicity) are often missing or only partially

included at best. These findings reveal major gaps in research practices in the inclusion of, as well as the accurate reporting of ethnic-racial minority groups into Canadian social genomic research, and highlight important areas and directions that research policy should prioritize.

### **Conclusions**

There is currently a limited number of Canadian social genomic research over the past three decades, demonstrating a major research gap compared to research conducted in the US. We urgently need more research attention in future pertinent studies that extend from merely investigating psychopathology and focus on a wide range of socioemotional and behavioral outcomes. Particularly, research attention is sorely needed in understanding how different broader societal and cultural contexts could modify genetic potentials among different ethnic-racial groups. Moreover, future research should take a developmental and life cycle approach to better understand how genes and environment contribute to human development with the aid of longitudinal designs. Marginalized ethnic/racial minority groups are poorly represented in extant literature. Future research must extend from investigating predominantly White adult populations onto ethnically/racially diverse child, adolescent, and adult populations. Rather than indiscriminately aggregating marginalized ethnic/racial minority groups into the larger White samples for analysis, more research should set out to specifically include and understand marginalized racial/ethnic minority groups, in recognition of their differences in genetic predispositions as well as their unique sociocultural systems and environmental experiences. Greater transparency and intentionality are needed in ethnicity/race and intersectionality reporting in future Canadian social genomics research.

**Knowledge Mobilization Activities**

Meyer, Z., Wu, Y., & Zheng, Y. (2024, April). *Examining ethnic-racial diversity in Canadian sociogenomic studies: Insights into adolescent research*. Poster to be presented at the annual meeting of the Society for Research on Adolescence, Chicago, IL, USA.

Meyer, Z., Wu, Y., & Zheng, Y. (2024, April). *Taking stock and moving forward: Synthesizing ethnic/racial diversity in Canadian social genomics research*. Poster to be presented at the annual meeting of the Royce-Harder Research Conference at University of Alberta, Edmonton, AB, Canada.

Zheng, Y., Meyer, Z., & Wu, Y. (2024, January). *Taking stock and moving forward: Synthesizing ethnic/racial diversity in Canadian social genomics research*. Oral presentation at the virtual Forum on Shifting Dynamics of Privilege and Marginalization by the Social Sciences and Humanities Research Council of Canada.

Zheng, Y., Meyer, Z., & Wu, Y. (2023, November). *Taking stock and moving forward: Synthesizing ethnic/racial diversity in Canadian social genomics research*. Evidence brief submitted to the Social Sciences and Humanities Research Council of Canada.

Meyer, Z., Wu, Y., & Zheng, Y. (2023, October). *Taking stock and moving forward: Synthesizing ethnic/racial diversity in Canadian social genomics research*. Poster presentation at the Women & Children's Health Research Institute (WCHRI) Research Day, Edmonton, AB.

### Bibliography

- Anderson, J. S., Shade, J., DiBlasi, E., Shabalin, A. A., & Docherty, A. R. (2019). Polygenic risk scoring and prediction of mental health outcomes. *Current Opinion in Psychology*, 27, 77–81. <https://doi.org/10.1016/j.copsyc.2018.09.002>
- Barr, C. L., Wigg, K., Malone, M., Schachar, R., Tannock, R., Roberts, W., & Kennedy, J. L. (1999). Linkage study of catechol-O-methyltransferase and attention-deficit hyperactivity disorder. *American Journal of Medical Genetics*, 88(6), 710–713. [https://doi.org/10.1002/\(SICI\)1096-8628\(19991215\)88:6<710::AID-AJMG23>3.0.CO;2-Q](https://doi.org/10.1002/(SICI)1096-8628(19991215)88:6<710::AID-AJMG23>3.0.CO;2-Q)
- Bawor, M., Dennis, B. B., Tan, C., Pare, G., Varenbut, M., Daiter, J., Plater, C., Worster, A., Marsh, D. C., Steiner, M., Anglin, R., Desai, D., Thabane, L., & Samaan, Z. (2015). Contribution of BDNF and DRD2 genetic polymorphisms to continued opioid use in patients receiving methadone treatment for opioid use disorder: An observational study. *Addiction Science & Clinical Practice*, 10(1), 1–9. <https://doi.org/10.1186/s13722-015-0040-7>
- Boies, S., Mérette, C., Paccalet, T., Maziade, M., & Bureau, A. (2018). Polygenic risk scores distinguish patients from non-affected adult relatives and from normal controls in schizophrenia and bipolar disorder multi-affected kindreds. *American Journal of Medical Genetics Part B: Neuropsychiatric Genetics*, 177(3), 329–336. <https://doi.org/10.1002/ajmg.b.32614>
- Boivin, M., Brendgen, M., Dionne, G., Ouellet-Morin, I., Dubois, L., Pérusse, D., Robaey, P., Tremblay, R. E., & Vitaro, F. (2019). The Quebec newborn twin study at 21. *Twin Research and Human Genetics*, 22(6), 475–481. <https://doi.org/10.1017/thg.2019.74>
- Brendgen, M., Vitaro, F., Boivin, M., Dionne, G., & Pérusse, D. (2006). Examining genetic and

environmental effects on reactive versus proactive aggression. *Developmental Psychology*, 42(6), 1299–1312. <https://doi.org/10.1037/0012-1649.42.6.1299>

Bureau, A., Beaulieu, J. M., Paccalet, T., Chagnon, Y. C., & Maziade, M. (2017). The interaction of GSK3B and FXR1 genotypes may influence the mania and depression dimensions in mood disorders. *Journal of Affective Disorders*, 213, 172–177.

<https://doi.org/10.1016/j.jad.2017.02.023>

Chagnon, Y. C., Maziade, M., Paccalet, T., Croteau, J., Fournier, A., Roy, M.-A., & Bureau, A. (2020). A multimodal attempt to follow-up linkage regions using RNA expression, SNPs and CpG methylation in schizophrenia and bipolar disorder kindreds. *European Journal of Human Genetics*, 28(4), 499–507. <https://doi.org/10.1038/s41431-019-0526-y>

Chen, X., & French, D. C. (2008). Children's social competence in cultural context. *Annual Review of Psychology*, 59, 591–616. <https://doi.org/10.1146/annurev.psych.59.103006.093606>

Chen, J., Yu, J., Zhang, J., Li, X., & McGue, M. (2015). Investigating genetic and environmental contributions to adolescent externalizing behavior in a collectivistic culture: A multi-informant twin study. *Psychological Medicine*, 45(9), 1989–1997.

<https://doi.org/10.1017/S0033291714003109>

De Lima, R. M. S., Barth, B., Arcego, D. M., De Mendonça Filho, E. J., Clappison, A., Patel, S., Wang, Z., Pokhvisneva, I., Sassi, R. B., Hall, G. B. C., Kobor, M. S., O'Donnell, K. J., Bittencourt, A. P. S. D. V., Meaney, M. J., Dalmaz, C., & Silveira, P. P. (2020). Amygdala 5-HTT gene network moderates the effects of postnatal adversity on attention problems: Anatomic-functional correlation and epigenetic changes. *Frontiers in Neuroscience*, 14, 1–18.

<https://doi.org/10.3389/fnins.2020.00198>

Del Zompo, M., De Luca, V., Severino, G., Ni, X., Mulas, S., Congiu, D., Piccardi, M. P., &

- Kennedy, J. L. (2007). Haplotype association study between DRD1 gene and bipolar type I affective disorder in two samples from Canada and Sardinia. *American Journal of Medical Genetics Part B: Neuropsychiatric Genetics*, *144B*(2), 237–241.  
<https://doi.org/10.1002/ajmg.b.30445>
- Ernst, C., Wanner, B., Brezo, J., Vitaro, F., Tremblay, R., & Turecki, G. (2011). A deletion in tropomyosin-related kinase B and the development of human anxiety. *Biological Psychiatry*, *69*(6), 604–607. <https://doi.org/10.1016/j.biopsych.2010.10.008>
- Feng, Y., Crosbie, J., Wigg, K., Pathare, T., Ickowicz, A., Schachar, R., Tannock, R., Roberts, W., Malone, M., Swanson, J., Kennedy, J. L., & Barr, C. L. (2005). The SNAP25 gene as a susceptibility gene contributing to attention-deficit hyperactivity disorder. *Molecular Psychiatry*, *10*(11), 998–1005. <https://doi.org/10.1038/sj.mp.4001722>
- French, L., Gray, C., Leonard, G., Perron, M., Pike, G. B., Richer, L., Séguin, J. R., Veillette, S., Evans, C. J., Artiges, E., Banaschewski, T., Bokde, A. W. L., Bromberg, U., Bruehl, R., Buchel, C., Cattrell, A., Conrod, P. J., Flor, H., Frouin, V., ... Paus, T. (2015). Early cannabis use, polygenic risk score for schizophrenia and brain maturation in adolescence. *JAMA Psychiatry*, *72*(10), 1002–1011. <https://doi.org/10.1001/jamapsychiatry.2015.1131>
- Fiori, L. M., Zouk, H., Himmelman, C., & Turecki, G. (2011). X chromosome and suicide. *Molecular Psychiatry*, *16*(2), 216–226. <https://doi.org/10.1038/mp.2009.132>
- Gross, J. A., Bureau, A., Croteau, J., Galfalvy, H., Oquendo, M. A., Haghighi, F., Mérette, C., Giegling, I., Hodgkinson, C., Goldman, D., Rujescu, D., Mann, J. J., & Turecki, G. (2015). A genome-wide copy number variant study of suicidal behavior. *PLoS One*, *10*(5), e0128369.  
<https://doi.org/10.1371/journal.pone.0128369>
- Hedrick, P. W. (2011). Population genetics of malaria resistance in humans. *Heredity*, *107*(4),



283–304. <https://doi.org/10.1038/hdy.2011.16>

Hiebert, D. (2016). What's so special about Canada? Understanding the resilience of immigration and multiculturalism. *Migration Policy Institute*, 1–21.

Ishii, K., Masuda, T., Matsunaga, M., Noguchi, Y., Yamasue, H., & Ohtsubo, Y. (2021). A reexamination of the effects of culture on dopamine D4 receptor gene interaction on social orientation. *Psychologia*, 63(2), 137–150. <https://doi.org/10.2117/psysoc.2021-B014>

Jang, K. L. (2013). The University of British Columbia Twin Project: Still figuring out what personality is and does. *Twin Research and Human Genetics*, 16(1), 70–72.

<https://doi.org/10.1017/thg.2012.70>

Jang, K. L., Vernon, P. A., & Livesley, W. J. (2000). Personality disorder traits, family environment, and alcohol misuse: A multivariate behavioural genetic analysis. *Addiction*, 95(6), 873–888. <https://doi.org/10.1046/j.1360-0443.2000.9568735.x>

Kelsoe, J. R., Spence, M. A., Loetscher, E., Foguet, M., Sadovnick, A. D., Remick, R. A., Flodman, P., Khristich, J., Mroczkowski-Parker, Z., Brown, J. L., Masser, D., Ungerleider, S., Rapaport, M. H., Wishart, W. L., & Luebbert, H. (2001.). A genome survey indicates a possible susceptibility locus for bipolar disorder on chromosome 22. *Proceedings of the National Academy of Sciences*, 98(2), 585–590. <https://doi.org/10.1073/pnas.98.2.585>

Kennedy, J. L., Xiong, N., Yu, J., Zai, C. C., Pouget, J. G., Li, J., Liu, K., Qing, H., Wang, T., Martin, E., Levy, D. L., & Lin, Z. (2016). Increased nigral SLC6A3 activity in schizophrenia patients: Findings from the Toronto–McLean cohorts. *Schizophrenia Bulletin*, 42(3), 772–781. <https://doi.org/10.1093/schbul/sbv191>

Kowalec, K., Fitzgerald, K. C., Salter, A., Dolovich, C., Harder, A., Bernstein, C. N., Bolton, J., Cutter, G. R., Graff, L. A., Hägg, S., Hitchon, C. A., Lu, Y., Lublin, F., McKay, K. A., Patten,

- S. B., Patki, A., Tiwari, H. K., Wolinsky, J. S., & Marrie, R. A. (2023). Polygenicity of comorbid depression in multiple sclerosis. *Neurology*, *101*(5), 1–28.  
<https://doi.org/10.1212/WNL.0000000000207457>
- Lee, H., Kim, S. S., You, K. S., Park, W., Yang, J. H., Kim, M., & Hayman, L. L. (2014). Asian flushing: Genetic and sociocultural factors of alcoholism among east Asians. *Gastroenterology Nursing*, *37*(5), 327–336.  
<https://doi.org/10.1097/SGA.0000000000000062>
- Leve, L. D., Neiderhiser, J. M., Harold, G. T., Natsuaki, M. N., Bohannon, B. J., & Cresko, W. A. (2018). Naturalistic experimental designs as tools for understanding the role of genes and the environment in prevention research. *Prevention Science*, *19*(1), 68–78.  
<https://doi.org/10.1007/s11121-017-0746-8>
- Li, Y., Bernstein, C. N., Xu, W., & Hu, P. (2022). Polygenic risk and causal inference of psychiatric comorbidity in inflammatory bowel disease among patients with European ancestry. *Journal of Translational Medicine*, *20*(1), 1–10. <https://doi.org/10.1186/s12967-022-03242-9>
- Lobo, D. S. S., Souza, R. P., Tong, R. P., Casey, D. M., Hodgins, D. C., Smith, G. J., Williams, R. J., Schopflocher, D. P., Wood, R. T., el-Guebaly, N., & Kennedy, J. L. (2010). Association of functional variants in the dopamine D2-like receptors with risk for gambling behaviour in healthy Caucasian subjects. *Biological Psychology*, *85*(1), 33–37.  
<https://doi.org/10.1016/j.biopsycho.2010.04.008>
- Mascheretti, S., Forni, D., Lampis, V., Fumagalli, L., Paquin, S., Andlauer, T. F. M., Wang, W., Dionne, G., Brendgen, M. R., Vitaro, F., Ouellet-Morin, I., Rouleau, G., Gouin, J.-P., Côté, S., Tremblay, R. E., Turecki, G., Garon-Carrier, G., Boivin, M., & Battaglia, M. (2023).

- Adolescent anxiety and pain problems: A joint, genome-wide investigation and pathway-based analysis. *PLoS One*, *18*(5), e0285263. <https://doi.org/10.1371/journal.pone.0285263>
- McCaffery, J. M., Duan, Q. L., Frasure-Smith, N., Barhdadi, A., Lespérance, F., Théroux, P., Rouleau, G. A., & Dubé, M.-P. (2009). Genetic predictors of depressive symptoms in cardiac patients. *American Journal of Medical Genetics Part B: Neuropsychiatric Genetics*, *150B*(3), 381–388. <https://doi.org/10.1002/ajmg.b.30824>
- Mills, M. C., & Tropf, F. C. (2020). Sociology, genetics, and the coming of age of sociogenomics. *Annual Review of Sociology*, *46*(1), 553–581.
- Morneau-Vaillancourt, G., Andlauer, T. F. M., Ouellet-Morin, I., Paquin, S., Brendgen, M. R., Vitaro, F., Gouin, J., Séguin, J. R., Gagnon, É., Cheesman, R., Forget-Dubois, N., Rouleau, G. A., Turecki, G., Tremblay, R. E., Côté, S. M., Dionne, G., & Boivin, M. (2021). Polygenic scores differentially predict developmental trajectories of subtypes of social withdrawal in childhood. *Journal of Child Psychology and Psychiatry*, *62*(11), 1320–1329. <https://doi.org/10.1111/jcpp.13459>
- Mundo, E., Zai, G., Lee, L., Parikh, S. V., & Kennedy, J. L. (2001). The 5HT1D $\beta$  receptor gene in bipolar disorder: A family-based association study. *Neuropsychopharmacology*, *25*(4), 608–613. [https://doi.org/10.1016/S0893-133X\(01\)00259-7](https://doi.org/10.1016/S0893-133X(01)00259-7)
- Orri, M., Boivin, M., Chen, C., Ahun, M. N., Geoffroy, M., Ouellet-Morin, I., Tremblay, R. E., & Côté, S. M. (2021). Cohort profile: Quebec longitudinal study of child development (QLSCD). *Social Psychiatry and Psychiatric Epidemiology*, *56*(5), 883–894. <https://doi.org/10.1007/s00127-020-01972-z>
- Ouellet-Morin, I., Wigg, K. G., Feng, Y., Dionne, G., Robaey, P., Brendgen, M., Vitaro, F., Simard, L., Schachar, R., Tremblay, R. E., Pérusse, D., Boivin, M., & Barr, C. L. (2008).

- Association of the dopamine transporter gene and ADHD symptoms in a Canadian population-based sample of same-age twins. *American Journal of Medical Genetics Part B: Neuropsychiatric Genetics*, *147B*(8), 1442–1449. <https://doi.org/10.1002/ajmg.b.30677>
- Pedram, P., Zhai, G., Gulliver, W., Zhang, H., & Sun, G. (2017). Two novel candidate genes identified in adults from the Newfoundland population with addictive tendencies towards food. *Appetite*, *115*, 71–79. <https://doi.org/10.1016/j.appet.2017.01.004>
- Perret, L. C., Boivin, M., Morneau-Vaillancourt, G., Andlauer, T. F. M., Paquin, S., Langevin, S., Girard, A., Turecki, G., O'Donnell, K., Tremblay, R. E., Côté, S. M., Gouin, J., Ouellet-Morin, I., & Geoffroy, M. (2023). Polygenic risk score and peer victimisation independently predict depressive symptoms in adolescence: Results from the Quebec longitudinal study of children development. *Journal of Child Psychology and Psychiatry*, *64*(3), 388–396. <https://doi.org/10.1111/jcpp.13706>
- Perret, L. C., Geoffroy, M.-C., Barr, E., Parnet, F., Provencal, N., Boivin, M., O'Donnell, K. J., Suderman, M., Power, C., Turecki, G., & Ouellet-Morin, I. (2023). Associations between epigenetic aging and childhood peer victimization, depression, and suicidal ideation in adolescence and adulthood: A study of two population-based samples. *Frontiers in Cell and Developmental Biology*, *10*, 1–14. <https://doi.org/10.3389/fcell.2022.1051556>
- Petronis, A., Bassett, A. S., Honer, W. G., Vincent, J. B., Tatuch, Y., & Sasaki, T. (1996). Search for unstable DNA in Schizophrenia families with evidence for genetic anticipation. *American Journal of Human Genetics*, *59*(4), 905–911.
- Pingault, J. B., O'Reilly, P. F., Schoeler, T., Ploubidis, G. B., Rijdsdijk, F., & Dudbridge, F. (2018). Using genetic data to strengthen causal inference in observational research. *Nature Reviews Genetics*, *19*(9), 566–580. <https://doi.org/10.1038/s41576-018-0020-3>

- Plomin, R., DeFries, J. C., Knopik, V. S., & Neiderhiser, J. M. (2016). Top 10 replicated findings from behavioral genetics. *Perspectives on Psychological Science, 11*(1), 3–23.  
<https://doi.org/10.1177/1745691615617439>
- Polderman, T. J., Benyamin, B., De Leeuw, C. A., Sullivan, P. F., Van Bochoven, A., Visscher, P. M., & Posthuma, D. (2015). Meta-analysis of the heritability of human traits based on fifty years of twin studies. *Nature Genetics, 47*(7), 702–712. <https://doi.org/10.1038/ng.3285>
- Price, K. M., Wigg, K. G., Feng, Y., Blokland, K., Wilkinson, M., He, G., Kerr, E. N., Carter, T., Guger, S. L., Lovett, M. W., Strug, L. J., & Barr, C. L. (2020). Genome-wide association study of word reading: Overlap with risk genes for neurodevelopmental disorders. *Genes, Brain and Behavior, 19*(6), e12648. <https://doi.org/10.1111/gbb.12648>
- Rabinowitz, J. A., Campos, A. I., Benjet, C., Su, J., Macias-Kauffer, L., Méndez, E., Martínez-Levy, G. A., Cruz-Fuentes, C. S., & Rentería, M. E. (2020). Depression polygenic scores are associated with major depressive disorder diagnosis and depressive episode in Mexican adolescents. *Journal of Affective Disorders Reports, 2*, 100028.  
<https://doi.org/10.1016/j.jadr.2020.100028>
- Rethlefsen, M. L., Kirtley, S., Waffenschmidt, S., Ayala, A. P., Moher, D., Page, M. J., & Koffel, J. B. (2021). PRISMA-S: An extension to the PRISMA statement for reporting literature searches in systematic reviews. *Systematic Reviews, 10*(1), 1–19.  
<https://doi.org/10.1186/s13643-020-01542-z>
- Robinson, G. E., Grozinger, C. M., & Whitfield, C. W. (2005). Sociogenomics: Social life in molecular terms. *Nature Reviews Genetics, 6*(4), 257–270. <https://doi.org/10.1038/nrg1575>
- Rothbaum, F., & Weisz, J. R. (1994). Parental caregiving and child externalizing behavior in nonclinical samples: A meta-analysis. *Psychological Bulletin, 116*(1), 55–74.

<https://doi.org/10.1037/0033-2909.116.1.55>

Rushton, J. P., Bons, T. A., Ando, J., Hur, Y.-M., Irwing, P., Vernon, P. A., Petrides, K. V., & Barbaranelli, C. (2009). A general factor of personality from multitrait–multimethod data and cross–national twins. *Twin Research and Human Genetics*, *12*(4), 356–365.

<https://doi.org/10.1375/twin.12.4.356>

Sengupta, S., Xiong, L., Fathalli, F., Benkelfat, C., Tabbane, K., Danics, Z., Labelle, A., Lal, S., Krebs, M.-O., Rouleau, G., & Joobert, R. (2006). Association study of the trinucleotide repeat polymorphism within SMARCA2 and schizophrenia. *BMC Genetics*, *7*(1), 34.

<https://doi.org/10.1186/1471-2156-7-34>

Shanahan, M. J. (2013). Social genomics and the life course: Opportunities and challenges for multilevel population research. In L. J. Waite, & T. J. Plewes (eds.), *New directions in the sociology of aging* (pp. 255–276). The National Academies Press.

Shanahan, M. J., & Hofer, S. M. (2005). Social context in gene-environment interactions: Retrospect and prospect. *The Journals of Gerontology Series B: Psychological Sciences and Social Sciences*, *60*(1), 65–76. [https://doi.org/10.1093/geronb/60.Special\\_Issue\\_1.65](https://doi.org/10.1093/geronb/60.Special_Issue_1.65)

Shink, E., Harvey, M., Tremblay, M., Raymond, C., Labbé, M., Gagné, B., & Barden, N. (2005). Exclusion of non-synonymous SNPs and a polyglutamine tract in SMRT/N-CoR2 as common deleterious mutation for bipolar disorder in the Saguenay-Lac-St-Jean population: N-Association of SMRT/N-CoR2 with bipolar disorder. *American Journal of Medical Genetics Part B: Neuropsychiatric Genetics*, *134B*(1), 10–12. <https://doi.org/10.1002/ajmg.b.30145>

Sirugo, G., Williams, S. M., & Tishkoff, S. A. (2019). The missing diversity in human genetic studies. *Cell*, *177*(1), 26–31. <https://doi.org/10.1016/j.cell.2019.02.048>

Spence, M. A., Flodman, P. L., Sadovnick, A. D., Bailey-Wilson, J. E., Ameli, H., & Remick, R.

- A. (1995). Bipolar disorder: Evidence for a major locus. *American Journal of Medical Genetics*, 60(5), 370–376. <https://doi.org/10.1002/ajmg.1320600505>
- Statistics Canada. (2022a). *Immigrants make up the largest share of the population in over 150 years and continue to shape who we are as Canadians*. <https://www150.statcan.gc.ca/n1/daily-quotidien/221026/dq221026a-eng.htm>
- Statistics Canada. (2022b). *The Canadian census: A rich portrait of the country's religious and ethnocultural diversity*. <https://www150.statcan.gc.ca/n1/daily-quotidien/221026/dq221026b-eng.htm>
- Statistics Canada. (2022c). *Indigenous population continues to grow and is much younger than the non-Indigenous population, although the pace of growth has slowed*. <https://www150.statcan.gc.ca/n1/daily-quotidien/220921/dq220921a-eng.htm>
- Su, J., Trevino, A. D., Kuo, S. I., Aliev, F., Williams, C. D., Guy, M. C., & Dick, D. M. (2022). Racial discrimination and alcohol problems: Examining interactions with genetic risk and impulsivity among African American young adults. *Journal of Youth and Adolescence*, 51(8), 1552–1567. <https://doi.org/10.1007/s10964-022-01609-1>
- Tabor, H. K., Risch, N. J., & Myers, R. M. (2002). Candidate-gene approaches for studying complex genetic traits: Practical considerations. *Nature Reviews Genetics*, 3(5), 391–397. <https://doi.org/10.1038/nrg796>
- Tam, V., Patel, N., Turcotte, M., Bossé, Y., Paré, G., & Meyre, D. (2019). Benefits and limitations of genome-wide association studies. *Nature Reviews Genetics*, 20(8), 467–484. <https://doi.org/10.1038/s41576-019-0127-1>
- Taylor, S., Thordarson, D. S., Jang, K. L., & Asmundson, G. J. (2006). Genetic and environmental origins of health anxiety: A twin study. *World Psychiatry*, 5(1), 47–50.

Taylor, S., Jang, K. L., & Asmundson, G. J. G. (2010). Etiology of obsessions and compulsions: A behavioral-genetic analysis. *Journal of Abnormal Psychology, 119*(4), 672–682.

<https://doi.org/10.1037/a0021132>

Tozzi, F., Manchia, M., Galwey, N. W., Severino, G., Del Zompo, M., Day, R., Matthews, K., Strauss, J., Kennedy, J. L., McGuffin, P., Vincent, J. B., Farmer, A., & Muglia, P. (2011).

Admixture analysis of age at onset in bipolar disorder. *Psychiatry Research, 185*(1–2), 27–32.

<https://doi.org/10.1016/j.psychres.2009.11.025>

Trebilcock, M. (2019). The puzzle of Canadian exceptionalism in contemporary immigration policy. *Journal of International Migration and Integration, 20*(3), 823–849.

<https://doi.org/10.1007/s12134-018-0633-6>

Tzenova, J., Kaplan, B. J., Petryshen, T. L., & Field, L. L. (2004). Confirmation of a dyslexia susceptibility locus on chromosome 1p34-p36 in a set of 100 Canadian families. *American Journal of Medical Genetics, 127B*(1), 117–124. <https://doi.org/10.1002/ajmg.b.20139>

Viechtbauer, W. (2010). Conducting meta-analyses in R with the metafor package. *Journal of Statistical Software, 36*(3), 1–48. <https://doi.org/10.18637/jss.v036.i03>

Woodbury-Smith, M., Paterson, A. D., O'Connor, I., Zarrei, M., Yuen, R. K. C., Howe, J. L., Thompson, A., Parlier, M., Fernandez, B., Piven, J., Scherer, S. W., Vieland, V., & Szatmari, P. (2018). A genome-wide linkage study of autism spectrum disorder and the broad autism phenotype in extended pedigrees. *Journal of Neurodevelopmental Disorders, 10*(1), 20.

<https://doi.org/10.1186/s11689-018-9238-9>

Xu, W., Cohen-Woods, S., Chen, Q., Noor, A., Knight, J., Hosang, G., Parikh, S. V., De Luca, V., Tozzi, F., Muglia, P., Forte, J., McQuillin, A., Hu, P., Gurling, H. M., Kennedy, J. L., McGuffin, P., Farmer, A., Strauss, J., & Vincent, J. B. (2014). Genome-wide association study



of bipolar disorder in Canadian and UK populations corroborates disease loci including SYNE1 and CSMD1. *BMC Medical Genetics*, 15(1), 2. <https://doi.org/10.1186/1471-2350-15-2>

Zai, C. C., Cheema, S. Y., Zai, G. C., Tiwari, A. K., & Kennedy, J. L. (2022). Post-traumatic stress disorder in the Canadian Longitudinal Study on Aging: A genome-wide association study. *Journal of Psychiatric Research*, 154, 209–218. <https://doi.org/10.1016/j.jpsychires.2022.07.049>

Zheng, Y., Pingault, J. B., Unger, J. B., & Rijdsdijk, F. (2019). Genetic and environmental influences on attention-deficit/hyperactivity disorder symptoms in Chinese adolescents: A longitudinal twin study. *European Child & Adolescent Psychiatry*, 29(2), 205–216. <https://doi.org/10.1007/s00787-019-01346-0>

Zheng, Y., Rijdsdijk, F., Pingault, J. B., McMahon, R. J., & Unger, J. B. (2016). Developmental changes in genetic and environmental influences on Chinese child and adolescent anxiety and depression. *Psychological Medicine*, 46(9), 1829–1838. <https://doi.org/10.1017/S003329171600031>

## **Appendices**

Figure 1. PRISMA flow diagram.

Figure 2. Study counts, average ethnicity reporting and quality scores.

Figure 3. Developmental periods of samples across study designs.

Table 1. Study characteristics and quality assessment.

Figure 1. PRISMA Flow Diagram

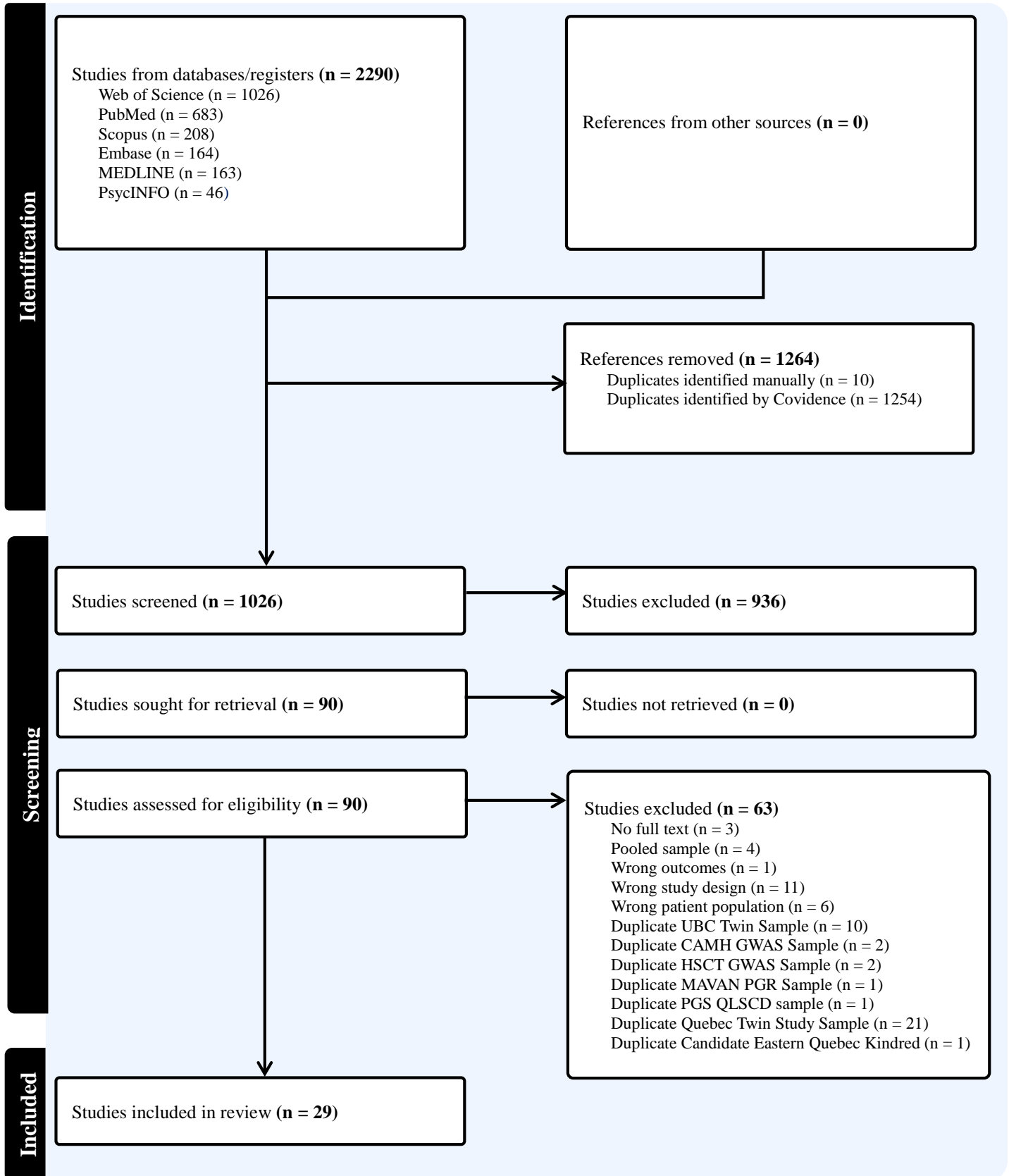


Figure 2. Study counts, average ethnicity reporting and quality scores.

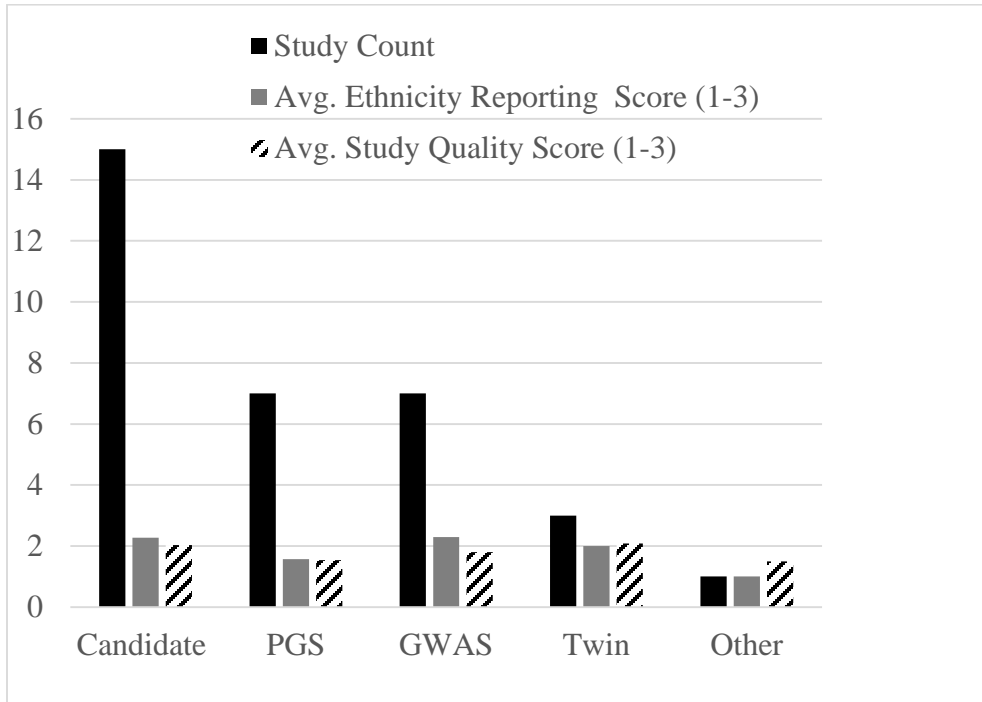


Figure 3. Developmental periods of samples across study designs.

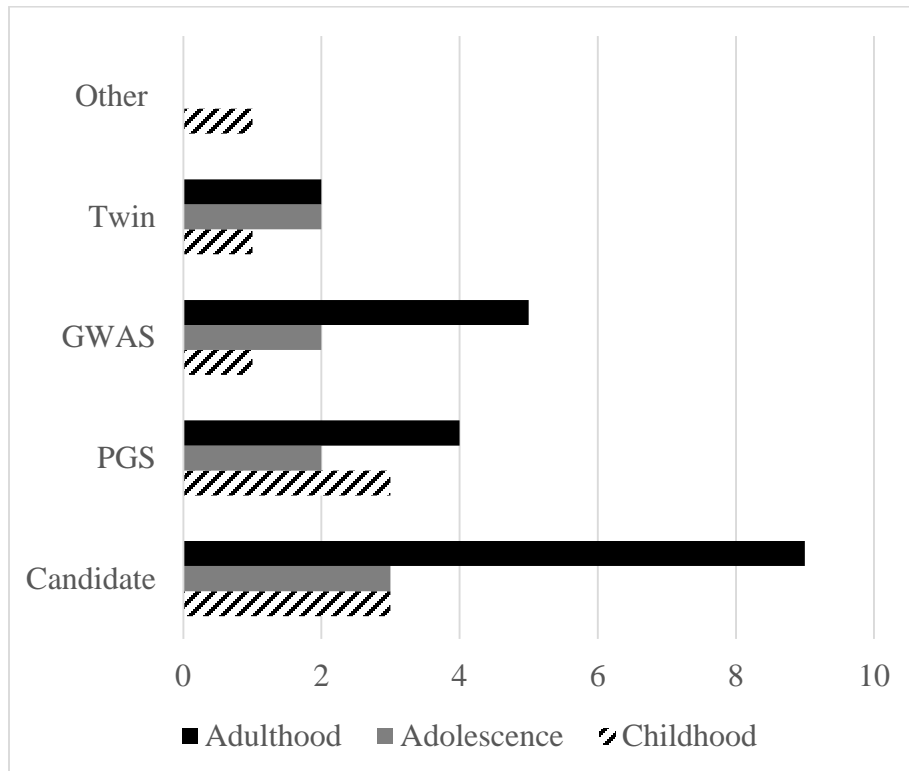


Table 1. Study characteristics and quality assessment.

Public ation Details	Study Design		Study Characteristics							Findings			Quality Assessment				
	Study	Genetic Design	Study Method	Sample	Geographic Region	N	Ethnic/Racial Composition	Age Range	Developmental Period	Sex	Study Outcome	Main Results	Sample Characteristics Reporting	Ethnic Composition Reporting	Representativeness	Ethnic × Sample Characteristics	Average Score
Barr et al. (1999)	Candidate Gene	Cross-sectional	Hospital for Sick Children, Toronto Ontario (HSCT)	Ontario	77 (Families)	96% European Caucasian descent 5 non-Caucasian (African Canadian & Native Canadian)	– 7-16	Middle Childhood - Middle Adolescence	–	ADHD	No evidence for linkage of Catechol-O-Methyltransferase and ADHD	2	3	2	1	2	Medium
Bawor et al. (2015)	Candidate Gene	Cross-sectional	Canadian Addiction Treatment Centres (CATC)	Ontario	240	European 84.6% Native North/South America 7.9% Asian .8% Persian .4%	37.1 –	Adulthood	Female (40%)	Opioid use disorder	No effect of BDNF and DRD2 on continued opioid use in patients treated with methadone	3	3	3	1	2.5	High
Bureau et al. (2017)	Candidate Gene (G x G)	Cross-sectional	Schizophrenia and Bipolar Disorder Eastern Quebec	Quebec	384	Caucasian French-Canadian	– –	Adulthood	–	Mania and depression in mood disorders	Interaction of GSK3B and FXR1 genotypes	1	2	2	2	1.75	Low

			Kindred Study								influence mania and depression levels						
Del Zompo et al. (2007)	Candidate Gene (Haplotype Association)	Cross-Sectional	Families with type 1 bipolar disorder from Cagliari & Toronto areas	Ontario	<b>Toronto Sample:</b> 229 (Trios)	-	35.46 -	Adulthood	Female (58.95%)	Bipolar Type 1 Affective Disorder	DRD1 polymorphisms considered risk factor for BP1	3	1	1	1	1.5	Low
Ernst et al. (2011)	Candidate Gene	Longitudinal	Longitudinal cohort of Quebec general population	Quebec	640	-	- -	Childhood Adulthood	Female (57%)	Anxiety	Base pair deletion in TrkB significantly associated with increases in anxiety traits during childhood and development of anxiety disorders in adulthood	2	1	3	1	1.75	Low
Fiori et al. (2011)	(1) Gene-association (2) Candidate Gene	Cross-sectional	(1) Clinical patients/Quebec general population (2) Quebec Suicide Brain Bank	Quebec	(1) <b>Suicide completors:</b> 333 <b>Non-suicide controls:</b> 389 (2) <b>Depressed suicide completors:</b>	French Canadian	(1) <b>Suicide completors:</b> 41.2 - <b>Nondepressed suicide completors:</b> 39.3 -	Adulthood	Male (100%)	Suicide completion	6 genes within two significant regions in X chromosome were differentially expressed in suicide	3	2	2	2	2.25	Medium

					14 <b>Nondepressed suicide completors: 8</b>  <b>Control: 13</b>  <b>Total: 757</b>		(2) <b>Depressed suicide completors: 34.9 –</b>  <b>Nondepressed suicide completors: 34.8 –</b>  <b>Control: 36.8 –</b>				complete rs						
Ishii et al. (2021)	Candidate Gene	Cross-sectional	Undergraduate students from Kobe (Japan) and Alberta (Canada)	Alberta	<b>Alberta Sample: 368</b>	European Canadian	19.46 –	Late Adolescence	Female (67.4%)	Social orientation	Dopamine D4 receptor gene does not interact with culture to predict social orientation	3	3	2	3	2.75	High
Lobo et al (2010)	Candidate Gene	Cross-sectional	Leisure, Lifestyle, and Lifecycle Project	Alberta	242	European Caucasian	46.57 –	Adulthood	Female (62.81%)	Pathological gambling	Functional variants in dopamine D2 like receptors associated with risk for gambling behavior	3	3	2	2	2.5	High
McCaffery et	Candidate Gene	Cross-sectional	(1) POLYMO RPHISME	Quebec	(1) 416	French Canadian	59.29 –	Adulthood	Female (21.	Depressive symptoms	Genetic variation relevant	3	3	3	3	3	High



al. (2009)			(2) Epidemiological Study of Acute Coronary Syndromes and the Pathophysiology of Emotions (ESCAPE)		(2) 561 <b>Total:</b> 977				29% )	in cardiac patients	to endothelial dysfunction and platelet aggregation contributes to the expression of depressive symptoms in cardiac patients						
Ouellet-Morin et al. (2008)	Candidate Gene	Cross-sectional	Quebec Newborn Twin study	Quebec	6-year-old group: 789 7-year-old group: 838 <b>Total:</b> 1,627	Caucasian 88.3% Asian 1.9% Black 1.1% Mixed ethnicity .6% Caucasian-Native North American (1 twin pair) Caucasian-Black (1 twin pair) Other 2.2% No report 5.9%	- 6-7	Middle Childhood	6-year-olds: VNT R: Female (49%) Rs27072: Female (48%) 7-year-olds: VNT R: Female (48%) Rs27072 Female	ADHD	No association between VNTR and ADHD symptoms. Rs27072 significantly associated with ADHD symptoms	3	3	3	1	2.5	High

									(48%)								
Sengupta et al. (2006)	Candidate Gene	Cross-sectional	Clinical patients and families from Montreal & Ottawa	Quebec, Ontario	<p><b>French Canadian Triads:</b> Case: 100 Control: 102</p> <p><b>Montreal/Ottawa Triads:</b> Case: 122 Control: 125</p> <p><b>Total:</b> 449</p>	French Canadian European	- -	- -	-	Schizophrenia	Family-based and case-control association analyses show no association between SMARCA2 polymorphism and schizophrenia	1	2	1	1	1.25	Low
Shink et al. (2005)	Candidate Gene	Cross-sectional	Bipolar and case-control participants from Saguenay-Lac-St-Jean Population	Quebec	<p><b>Case-BP1:</b> 182</p> <p><b>Case-BP2:</b> 31</p> <p><b>Control:</b> 214</p> <p><b>Total:</b> 427</p>	French Canadian	(Age of Onset) <b>Case-BP1:</b> 28 -	Adulthood	<p><b>Case-BP1:</b> Female (60%)</p> <p><b>Case-BP2:</b> 27 -</p> <p><b>Control:</b> -</p> <p><b>Control:</b> -</p>	Bipolar disorder	No significant associations found between polymorphisms and bipolar phenotype	3	2	1	1	1.75	Low
Spence et al. (1995)	Candidate Gene	Cross-sectional	Bipolar 1, 2 and major depression index cases from UBC	British Columbia	<p><b>Probands:</b> 487</p> <p><b>Relative:</b> 3,899</p>	Caucasian Central/Northern European Ancestry	-	-	-	Bipolar disorder	A single dominant mendelian major locus was the best	2	3	1	1	1.75	Low

			University Hospital		<b>Total:</b> 4386						fitting models for BP1 and BP2 probands						
Tzenova et al. (2004)	Candidate Gene/Genetic Linkage Analysis	Cross-sectional	Families of dyslexic children from Calgary	Alberta	<b>Dyslexia Cases:</b> 895 <b>Control:</b> 112 <b>Total:</b> 1007	European Ancestry and 4 families with 1 parent non-European	>8	-	-	Dyslexia	Evidence for dyslexia susceptibility locus on chromosome 1p34-36	2	2	1	1	1.5	Low
Mascheretti et al. (2023)	Genome-Wide Association & Pathway Analysis	Cross-sectional	(1) Quebec Newborn Twin Study (2) Longitudinal Study of Child Development in Quebec	Quebec	(1) 1,002 (2) 1,437 <b>Total:</b> 2439	French Canadian	- 12-14	Early-Middle Adolescence	-	Adolescent anxiety and pain problems	Pathway involved in regulation of myotube differentiation found to be associated with pain problems and anxiety symptoms across samples	2	2	2	1	1.75	Low
Pedram et al. (2017)	(1) Genome-Wide Screening (2) Candidate Gene Association	Cross-sectional	Complex Diseases in the Newfoundland population : Environment and Genetics (CODING)	Newfoundland	<b>Stage 1:</b> 24 <b>Stage 2:</b> 752 <b>Overall:</b> 752	-	<b>Stage 1:</b> FAO: 37.4 - NFO: 40.5 - <b>Control:</b> 44.5 - <b>Stage 2:</b>	Adulthood	<b>Stage 1:</b> Female (100%) <b>Stage 2:</b> FAO : Female (70%)	Food addiction	Two genes associated with addictive tendencies towards food identified: DRD2 & TIRAP	3	1	1	1	1.5	Low

							FAO: 43.5 – NFO: 45.5 – Control 44 –		NFO: Female (47.35%)  Control: Female (78.78%)								
Price et al. (2020)	(1) Genome-Wide Association  (2) Polygenic Risk	Cross-sectional	Hospital for Sick Children in Toronto (HSCT)	Ontario	624	European Caucasian	9.83 –	Late Childhood	Female (41%)	Reading disabilities & genetic risk for neurodevelopmental disorders	Findings suggest shared genetic risk between word reading, educational outcomes and autism spectrum disorder	3	3	1	1	2	Medium
Woodbury-Smith et al. (2018)	Genome-Wide Linkage Study	Cross-sectional	Families with Autism Spectrum Disorder	–	529	Northern European Ancestry	35.3 15-56	Middle Adolescence-Adulthood	–	Autism spectrum disorder & broad autism phenotype	Results suggest multiple susceptibility variants for ASD across pedigrees with potential overlapping loci between ASD and BAP	2	2	1	1	1.5	Low
Xu et al. (2014)	Genome-Wide	Cross-Sectional	(1) Centre for Addiction	Ontario	(1) Case: 431	(1) Northern/Western	(1) 45.5 –	Adulthood	(1) Case :	Bipolar disorder	Findings support numerous	3	3	1	1	2	Medium

	Association		and Mental Health (CAMH)  (2) Independent BP Family Trios Toronto		<b>Control:</b> 440  <b>Total:</b> 871  (2) <b>Trio Families:</b> : 229	European Ancestry  (2) 97.9% Caucasian 1.4% Asian .7% Native American <i>*Mundo et al., 2001</i>	<i>*Tozzi et al., 2011</i>  (2) 36.3 – <i>*Mundo et al., 2001</i>		Female (62.87%)  <b>Control:</b> Female (60%)  (2) <b>Trio Families:</b> Female (61%) <i>*Mundo et al., 2001</i>	s genes previously implicated in etiology of BD: CSMD1, SYNE1 as well as new genes: ADCY2, NCALD, WDR60, SCN7A & SPAG16							
Zai et al. (2022)	(1) Genome-Wide Association  (2) Polygenic Risk	Cross-sectional	Canadian Longitudinal Study on Aging (CLSA)	National	<b>PTSD:</b> 796  <b>No PTSD:</b> 15,565  <b>Total:</b> 16,361	European Ancestry	<b>PTSD:</b> 59.6 – <b>No PTSD:</b> 63.2 –	Adulthood	<b>PTSD:</b> Female (66%)  <b>No PTSD:</b> Female (50%)	PTSD	No significant findings for GWAS analyses; PRS showed variable levels of association between PTSD items with various problem outcomes e.g., depression,	3	3	1	1	1.75	Low

											educational attainment, insomnia						
Perret et al. (2023)	Epigenome-Wide DNA Methylation	Longitudinal	(1) Quebec Longitudinal Study of Child Development  (2) 1958BBC (UK)	Quebec	Quebec Sample 149	French Canadian	10.47 9.7-11.3	Late Childhood	Female (57.8%) <i>*Orr et al., 2021</i>	Epigenetic aging, childhood peer victimization, depression & suicidal ideation	Peer victimization not associated with epigenetic indices in Canadian cohort. Higher pediatric-buccal-epigenetic aging and slower pace of aging predicted higher depressive symptom scores.	3	1	1	1	1.5	Low
Boies et al. (2018)	Polygenic Risk	Cross-sectional	Schizophrenia and Bipolar Disorder Easter Quebec Kindred Families	Quebec	Subjects from SZ/BD Families : 333 Control: 894 Total: 1,227	-	-	Adulthood	-	Bipolar Disorder & Schizophrenia	Significant PRS difference between SZ and BD affected subjects and NAARs and controls	1	1	2	2	1.5	Low
De Lima et al. (2020)	Polygenic Risk	Cross-sectional	(1) Maternal Adversity, Vulnerability, and Neurodeve	Quebec, Ontario	MAVAN Sample: 145	-	Maternal age at birth: 30.60 -	Early Childhood	Female (50.3%)	ADHD problems	PGR reflecting amygdala 5-HTT gene network	2	1	1	1	1.25	Low

			lopment (MAVAN) (2) GUSTO(Singapore)				<b>Gestational age (weeks):</b> 39.18 -				functions moderate the effects of postnatal adversity on attention problems						
Kowalec et al. (2023)	Polygenic Risk	Cross-sectional	(1) Manitoba Multiple Sclerosis Cohort (2) UK Biobank (3) CombiRx Trial (US)	Manitoba	<b>Manitoba Sample</b> 370	-	<b>MS/Dep:</b> 51 - <b>MS/No Dep:</b> 51.8 - <b>Dep/No Immune:</b> 46.4 - <b>Healthy:</b> 44 -	Adulthood	<b>MS/Dep:</b> Female (83.3%) <b>MS/No Dep:</b> Female (80.3%) <b>Dep/No Immune:</b> Female (80.2%) <b>Healthy:</b> Female (63%)	Comorbid depression in multiple sclerosis	Individuals with MS and depression had higher depression on PGS compared to MS without depression and health controls	3	1	1	1	1.5	Low
Li et al. (2022)	Polygenic Risk	Cross-sectional	Manitoba Inflammatory Bowel Disease (IBD) Cohort	Manitoba	<b>IBD without PC:</b> 146 <b>IBD with PC:</b> 94 <b>Total:</b> 240	-	≥18 -	Late Adolescence – Adulthood	<b>IBD without PC:</b> Female (51.4%)	Psychiatric comorbidity (PC) in IBD	Polygenic risk significantly associated with PC status in IBD patients	2	1	1	1	1.25	Low

									IBD with PC: Female (64.9%)								
Morneau-Vaillancourt et al. (2021)	Polygenic Risk	Longitudinal	(1) Quebec Newborn Twin Study (2) Quebec Longitudinal Study of Child Development	Quebec	971	French Canadian	– 6-12	Middle Childhood- Early Adolescence	–	Social withdrawal and genetic risk for mental health disorders	Polygenic risk for loneliness predicted membership to high trajectory for social wariness. Genetic risk for general mental health associated with high-chronic solitude	2	1	2	1	1.5	Low
Boivin et al., (2019) *Demographic information from Brendgen et al., (2006)	Twin	Cross-sectional	Quebec Newborn Twin Study	Quebec	1,296	84% European 3% African 2% Asian 2% Native American 9% N/A	6 –	Middle Childhood	Female (51%)	Reactive & Proactive Aggression	Reactive & proactive aggression primarily influenced by socialization experiences specific to aggression type. Genetic influences are relatively small	3	3	3	3	3	High



Jang (2012) *Demographic information from Jang et al., (2000)	Twin	Cross-sectional	(1) UBC Twin Registry (2) Volunteer Sample from London Ontario	British Columbia Ontario	(1) 1318 (2) 260 <b>Total:</b> 1578	-	(1) 32.05 18-86 (2) 25.68 18-43	Late Adolescence Adulthood	(1) Female (63.3%) (2) Female (77%)	Antisocial personality traits and alcohol misuse	Common genetic factors found between antisocial personality traits and alcohol misuse	3	1	1	1	1.5	Low
Taylor et al. (2010)	Twin	Cross-sectional	Community sample of MZ DZ twins	National	614	Mostly Caucasian	40 17-81	Late Adolescence-Adulthood	Female (78%)	Obsessive compulsive symptoms	M = 49% of variance for OC-related symptoms due to genetic factors, the rest due to nonshared environment	3	2	1	1	1.75	Low