Genetic Risk Assessment in Psychiatry

Holly Landrum Peay

Center for Newborn Screening, Ethics, and Disability Studies, RTI International, Research Triangle Park, North Carolina 27703, USA

Correspondence: hpeay@rti.org

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Most psychiatric disorders of pediatric and adult onset are caused by a complex interplay of genetic and environmental risk factors. Risk assessment in genetic counseling is correspondingly complicated. Outside of neurodevelopmental conditions, genetic and genomic testing has not achieved clinical utility. Genetic counselors most often base risk assessment on the client's medical and family history and empiric recurrence risk data. In rare cases significant familial risk may arise from variants of large effect. New approaches such as polygenic risk scores have the potential to inform diagnosis and management of affected individuals and risk status for at-risk individuals. Research on the genetic and environmental factors that increase risk for schizophrenia and etiologically related disorders are reviewed, guidance in determining and communicating risks to families is delivered, and new opportunities and challenges that will come with translating new research findings to psychiatric risk assessment and genetic counseling are anticipated.

Psychiatric disorders are common and associated with significant functional impairment (Whiteford et al. 2013). Our knowledge about the complex etiology of psychiatric disorders is rapidly evolving. Strong evidence implicates genetic risk factors associated with psychiatric disorders of both pediatric and adult onset (Sullivan and Geschwind 2019). Genetic heritability estimates range from 40% to >80% (Polderman et al. 2015) with the heritability of bipolar disorder and schizophrenia at the high end of the range (Nöthen et al. 2010). Genome-wide association studies (GWASs) have identified common single nucleotide polymorphisms (SNPs) and rare copy-number variants (CNVs) for many psychiatric disorders of childhood and adult onset (Psychiatric GWAS Consortium Coordinating Committee et al. 2009). There is also consistent evidence for genetic risk factors that are shared across different diagnoses, including across childhood- and adolescent- or adult-onset disorders (Rasic et al. 2014; Martin et al. 2018; Sullivan and Geschwind 2019).

A wide range of environmental risk factors also contribute to etiology and may moderate genetic risk, including traumatic brain injury at birth, maternal prenatal infection or viral infection during the lifetime, environmental toxins, childhood trauma, stressful life events, head injury, and substance abuse (Peay and Austin 2011; Uher 2014; Janoutová et al. 2016; Marangoni et al. 2016; Al-Haddad et al. 2019; Bölte et al. 2019; Estrada-Prat et al. 2019). Unfortunately, with the exception of substance use, these

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factors are largely outside of the control of at-risk individuals. Though this is an oversimplification of the complex interplay among the risk factors, it is useful to consider genetic risk as conferring vulnerability to environmental stressors (Peay and Austin 2011).

There is some evidence that psychiatric diagnoses represent extreme ends on a continuous spectrum of related population traits (Rössler et al. 2007; Martin et al. 2018). Under this model, as genetic and/or environmental risk increases across a population, the associated traits become more extreme until a threshold is reached. After reaching the threshold, individuals would have a level of symptomatology that is sufficient to receive a psychiatric diagnosis.

And yet, even given the rapid pace of research progress in psychiatry, risk assessment is not yet meaningfully informed by genetic or genomic testing for the majority of clients. Most identified genetic risk factors play a small role in etiology (Mitchell et al. 2010; Sullivan and Geschwind 2019), and even when genetic risk factors are aggregated, they do not yet explain a clinically relevant portion of risk (Zheutlin and Ross 2018; Martin et al. 2019). Neurodevelopmental disorders are the exception (see Blesson and Cohen 2019).

The application of psychiatric genomic research to inform individual-level risk is further complicated by the current diagnostic classification, which does not define clear biological entities and thus the relationship between diagnostic categories and genomic results is probabilistic (Smoller et al. 2019). Eventual improvements in understanding the etiology are anticipated to lead to new, etiologically based diagnostic categories (Bipolar Disorder and Schizophrenia Working Group of the Psychiatric Genetics Consortium 2018; Zheutlin and Ross 2018; Sullivan and Geschwind 2019). Clinical applications related to diagnosis and management of symptomatic individuals will likely precede the ability to forecast risk in at-risk relatives.

Regardless of these limitations, genetic counseling about etiology and recurrence for psychiatric disorders is of value to clients. In most cases, risk assessment continues to be based on personal and family history. Clients have varied reasons for seeking consultation that may or may not include a desire for a quantified assessment of risk (Borle et al. 2018). This highlights the importance of contracting prior to engaging in risk assessment and customizing the counseling to client needs. Clients seek psychiatric genetic counseling for multiple indications (e.g., based on personal history, with concerns about personal or familial recurrence; based on family history with concerns about personal and/or family risk; in situations of adoption) (Peay et al. 2017; also see Austin 2019).

This review prepares genetic counselors to engage in risk assessment by providing an overview of research on the etiology of schizophrenia as an illustrative example, delivering guidance for determining and communicating risk based on family history, and anticipating benefits and challenges to translating new research findings to psychiatric genetic counseling. The focus is on risk assessment for disorders that traditionally have onset in adolescence or adulthood. Here we assume that psychiatric risk in the family is, or has become, the primary focus of the genetic counseling session, and the genetic counselor has sufficient time to engage in targeted risk assessment and counseling.

OVERVIEW OF THE ETIOLOGY OF SCHIZOPHRENIA

Schizophrenia is among the most severe of the major psychiatric disorders and is associated with considerable disability (Whiteford et al. 2013). Schizophrenia has been the focus of considerable research to elucidate specific risk factors and evidence of causative overlap with other psychiatric conditions. This research is briefly summarized below.

Common Variants with Small Effect Sizes

The mapping of genes for schizophrenia and other complex disorders is conducted using GWASs, which provides an assessment of variation across the genome through comparison of millions of polymorphisms across cases and controls. Hundreds of loci implicated in schizophrenia risk have been identified using GWASs,

with enrichment of brain-expressed genes, genes associated with immune function, and genes implicated in synaptic plasticity and function (Avramopoulos 2018; Bearden and Forsyth 2018; Coelewij and Curtis 2018; Prata et al. 2019).

The variants found in GWASs can be used to develop polygenic risk scores (PRSs). PRSs provide an analytic approach to explore how genetic risk is manifested in individuals across different study populations, and thus how phenotypes are related to one another (Bipolar Disorder and Schizophrenia Working Group of the Psychiatric Genetics Consortium 2018; Mistry et al. 2018; Nakahara et al. 2018). PRSs in schizophrenia reflect a broad phenotypic spectrum, with studies finding association with factors such as cognitive ability, emotional identification, verb reasoning, and memory (Nakahara et al. 2018).

Copy-Number Variants (CNVs)

Although rare, CNVs can confer significant risk for schizophrenia (Marshall et al. 2017), and the identification of such variants may have clinical utility for medical management and genetic counseling. An example is deletion 22q11.2 (velocardiofacial syndrome [VCFS]), which confers an increased risk of psychosis. A deletion at 22q11.2 has been estimated to be present in 0.3% of those diagnosed with schizophrenia (Marshall et al. 2017). Importantly, though the genetic variations directly cause VCFS, having the 22q11.2 deletion is neither necessary nor sufficient to result in psychiatric illness.

In a large recent case-control study, 22q11.2 and seven additional CNVs achieved genome-wide significance (Marshall et al. 2017). Most of these high-risk variants are hypothesized to be de novo because of the negative selection associated with the phenotypic outcomes, which include intellectual disability and dysmorphic features as well as schizophrenia (Marshall et al. 2017; Avramopoulos 2018). Taken together, CNVs are expected to account for 2% of patients with schizophrenia (Bearden and Forsyth 2018; Coelewij and Curtis 2018). Additional research is needed to assess the lifetime risk for psychiatric outcomes associated with emerging CNV findings.

Rare Variants

There is evidence for rare, damaging variants that contribute substantially to schizophrenia risk (Avramopoulos 2018; Coelewij and Curtis 2018). Additional variants are likely to be identified through ongoing genomic sequencing studies (Bearden and Forsyth 2018).

Genetic Risk Factors Replicated across Approach

Several functional categories of genes have been implicated by studies identifying both common and rare variants. These include genes involved in central nervous system development, immunity, synaptic transmission, *N*-methyl-D-aspartate receptors, activity-regulated cytoskeletal complex, and fragile-X-related protein (Avramopoulos 2018; Bearden and Forsyth 2018).

Relationship of Schizophrenia to Other Psychiatric Disorders

Schizophrenia and mood disorders have a high degree of genetic overlap (e.g., ~70% for schizophrenia and bipolar disorder) (Lee et al. 2013; Zheutlin and Ross 2018), and family studies indicate that first-degree relatives of individuals with schizophrenia are at risk for psychotic and for mood disorders (Lichtenstein et al. 2009; Rasic et al. 2014). The Bipolar Disorder and Schizophrenia Working Group of the Psychiatric Genetics Consortium reported shared genetic loci across schizophrenia, bipolar disorder, major depressive disorder, autism, and attention deficit hyperactivity disorder (Ruderfer et al. 2014). A recent GWAS study of schizophrenia and bipolar disorder (Bipolar Disorder and Schizophrenia Working Group of the Psychiatric Genetics Consortium 2018) reported more than 100 loci that contributed to both schizophrenia and bipolar disorder, with enrichment for genes involved in response to potassium ions. The study also identified four loci that distinguished between the two disorders (Bipolar Disorder and Schizophrenia Working Group of the Psychiatric Genetics Consortium 2018).

Environmental and Paternal Risk Factors

There is evidence that a number of environmental factors contribute to risk for schizophrenia including prenatal and perinatal factors such as maternal malnutrition, birth trauma, and season of birth; exposure to infections; residing in an urban environment; exposure to stressful life events; and substance abuse (Mitchell et al. 2010; Peay et al. 2017; Avramopoulos 2018). Advanced paternal age has also been implicated in increased risk and earlier onset of symptoms (Wang et al. 2015; Fond et al. 2017). A recent study supported an independent role of both advanced paternal age and of very young paternal age in increasing risk for early-onset schizophrenia in offspring (Wang et al. 2019).

Summary of Schizophrenia Etiology

Schizophrenia is caused by a complex etiology wherein genetic risk originates from hundreds to thousands of genes. Although genetic risk is attributed to both common and rare variants, a large proportion of heritable risk is caused by interactions among common allele variants that each contribute small effects to one or more psychiatric conditions (Avramopoulos 2018; Bearden and Forsyth 2018; Coelewij and Curtis 2018). No evidence supports a common variant of large effect, which is likely due to the low reproductive fitness among those with schizophrenia (Power et al. 2013). Genetic risk factors likely confer a lifelong biological vulnerability (Bearden and Forsyth 2018) with the individual's mental health status ultimately determined by interactions with environmental factors.

APPLICATION OF GENOMIC RESEARCH FINDINGS TO CLIENT RISK ASSESSMENT

This overview of schizophrenia research provides background to facilitate an understanding of the complex etiology of psychiatric disorders and also a context in which to consider applying research data to individual risk assessment. There are three primary ways that genomic data can be applied to genetic counseling. The first is explanatory, in which genomic data is used to understand causation and/or confirm or reject a particular diagnosis for a person with psychiatric symptoms (Lázaro-Muñoz et al. 2018). Currently, this is limited to genes of significant effect such as well-defined CNVs. In the future, use of individual-level genomic and clinical data may be compared to others with similar PRS profiles to inform diagnostic classification and predict course of illness (Bipolar Disorder and Schizophrenia Working Group of the Psychiatric Genetics Consortium 2018).

The second is to inform the treatment and management of an affected individual. Currently available pharmacogenetic testing (Lister 2016; Routhieaux et al. 2018) aims to predict medication efficacy and risk of side effects and adverse events. Specific panels are available to inform psychotropic medication selection, such as the Assurex GeneSight Psychotropic Test (https://genesight.com/). A systematic review (Health Quality Ontario 2017) of the Assurex panel found improved patient response to depression treatment and greater response in measures of depression, but no differences in rates of complete remission (based on low to very-low quality evidence). The optimal timing of pharmacogenetic testing and extent to which results should inform treatment recommendations are still under debate (Lister 2016; Routhieaux et al. 2018). The American Psychiatric Association recently concluded that there is insufficient data to support the widespread use of pharmacogenetic testing (Zeier et al. 2018). In the future, pharmacogenetics will very likely play a role in personalized medicine through the optimization of psychotropic medication selection.

The use of polygenic risks scores also holds promise for informing treatment and management. For example, a recent study used PRSs to anticipate lithium response on people with bipolar disorder (International Consortium on Lithium Genetics et al. 2018). Research is advancing our ability to compare individual-level genetic and clinical data against others with similar profiles and thereby to inform treatment approaches that have been effective in etiologically similar patients (Bipolar Disorder and

Schizophrenia Working Group of the Psychiatric Genetics Consortium 2018).

Finally, genetic/genomic data could be applied to the prediction of risk for unaffected individual(s). Currently this risk assessment is based on personal and family history (as described in the next section) for a large majority of clients. In rare cases, the client's clinical or family history may indicate that a medical genetics evaluation and/or testing for rare variants of large effect (e.g., syndromic causes) are warranted. The identification of such variants has significant implications for psychiatric risk to unaffected relatives, although that risk may be dwarfed by other health-related or developmental risks associated the variant.

Common variants have little or no predictive value independently but could be combined with a large number of other common and/or with rare variants to result in a more stable and clinically meaningful predictive value (Mitchell et al. 2010; Bearden and Forsyth 2018). This is the concept behind PRSs that provide a sum of known risk alleles weighted by the magnitude of risk each variant is expected to confer (Mistry et al. 2018). Because PRSs result in a single value representing an individual's overall genetic risk, they have theoretical value in allowing prediction and possibly risk mitigation efforts for unaffected individuals (Zheutlin and Ross 2018) and individuals with emerging or subclinical symptoms (Calafato et al. 2018). Although PRSs could be applied to unaffected individuals at population risk, genetic counselors are likely to first use PRSs for assessing risk to individuals with a family history. Yet we are likely years from achieving this outcome in clinical psychiatric genetic counseling. PRSs are limited by insufficiently powered GWASs (e.g., in what is considered to be a very large GWAS study, only 7% of the variance in schizophrenia was explained [Schizophrenia Working Group of the Bipolar Disorder and Schizophrenia Working Group of the Psychiatric Genetics Consortium 2014]) and further limited by the lack of ancestral diversity among participants. Notably, PRSs for psychiatric disease are not yet considered to be clinically meaningful (Zheutlin and Ross 2018; Martin et al. 2019).

APPLICATION OF CLIENT FAMILY HISTORY TO RISK ASSESSMENT

Against the backdrop of rapidly advancing psychiatric genomic research that has not yet achieved clinical utility, family history–based risk assessment is still the most appropriate course for the majority of families. Individuals who seek out psychiatric genetic counseling often have multiple affected individuals in their families and have significant concerns for recurrence (Peay and Austin 2011; Peay et al. 2014; Borle et al. 2018). Risk of recurrence is often overestimated by family members (Meiser et al. 2007; Austin et al. 2012), and there is evidence that clients' perceived risk of recurrence for psychiatric disorders influences their psychological well-being and behaviors, including reproductive decision-making (Austin and Honer 2005; Meiser et al. 2007; Peay et al. 2014).

Obtaining the Family and Personal History

Because psychiatric risk assessment is rooted in the client's personal and family histories, genetic counselors have an opportunity to engage in bidirectional sharing of information to the benefit of both parties. Genetic counselors need detailed information from the client, and clients can be helped to understand psychiatric genetics through a transparent questioning process. For example, asking questions about the age of onset for a disorder provides an opportunity to discuss that increased risk may be associated with early symptom onset; and queries about relevant environmental exposures provide an opportunity to understand client perceptions of nongenetic risk factors and sets the stage for counseling about complex etiology. The implications of shared etiology across diagnoses, which include the clustering of different diagnoses in families and risk that extends beyond the diagnosis made in the proband to etiologically related conditions (e.g., if the proband has bipolar disorder, close relatives are also at increased risk for major depression; see Table 1), provide another area for shared information gathering and learning. Because the genetic counselor is reliant upon the client for an accurate and complete family and personal history, and because the client's lived

Table 1. Absolute empiric recurrence risks for selected psychiatric disorders

	Schizophrenia	Bipolar disorder	Major depressive disorder
General population	1%	0.8%–1.6%	2%–23% (female) 1%–15% (male)
Affected parent, for same diagnosis	5%–13%	6%–15%	7%–26%
Affected parent, for either schizophrenia, bipolar, or depression	27% for offspring age 20 or older	27% for offspring age 20 or older	48% for offspring age 20 or older
Affected parent, for any diagnosed mental disorder	45% for offspring age 20 or older	58% for offspring age 20 or older	65% for offspring age 20 or older
Affected sibling	9%–16%	5%–20%	5%–30%
Affected second-degree relative (pooled)	2%–8%	5%	—
Risks for additional mental disorders (not comprehensive)	Schizoaffective disorder, personality disorders, bipolar disorder, major depression, substance use	Major depression, substance use, schizophrenia, schizoaffective disorder, anxiety disorder, ADHD	Anxiety disorders, substance use, disruptive disorders, dysthymia, ADHD

Contents of table adapted from Peay et al. 2017 and Rasic et al. 2014.
(FDRR) First-degree relative recurrence risk, (ADHD) attention deficit hyperactivity disorder.

experience and perception of the illness will impact how he/she responds to the risk assessment data, the mutuality in this experience provides trust and a strong basis for client-centered counseling.

During this process, genetic counselors should obtain the client's personal history and at least three generations of psychiatric family history. Specific information to obtain includes the following (adapted from Peay and Austin 2011; Peay et al. 2017):

- Psychiatric and substance abuse diagnoses made at any time in the lifespan and the age (s) at symptom onset and at diagnosis
- Potentially important environmental exposures
- Symptomatic, undiagnosed individuals and/or undiagnosed individuals treated with a psychiatric medication or by a mental health provider
- Developmental history of relatives and achievement of normal milestones for age (i.e., independent living and employment for adults)
- History of suicide and/or self-harm
- Birth defects, mental retardation, or learning disabilities and unusual medical conditions
- The age and sex of unaffected family members

Family History–Based Risk Assessment

Family and personal histories represent both environmental and genetic risk, which cannot be reasonably separated for risk assessment. The following factors are associated with increased risk to family members when they pertain to the affected or symptomatic individual(s) in the family (adapted from Peay and Austin 2011; Peay et al. 2017):

1. Risk for recurrence in the family increases with the number of affected relatives, regardless of the type of major psychiatric disorder clustering in the family (see Table 1).
2. Individuals who have particularly severe illness, unusually early age at onset, and/or who are of the less-commonly affected sex may represent a higher load of genetic risk, and their relatives may face a higher risk.

3. Assortative mating increases the risk of recurrence in subsequent generations, whether between a couple with concordant or discordant diagnoses.
4. Comorbid developmental disability, birth defects, or unusual medical history increases suspicion of rare CNVs with significant effect.

There are also important risk determinants related to the at-risk individual(s) in the family:

1. At-risk individuals have different degrees of risk based on their current age (Rasic et al. 2014). Young individuals still have all years of greatest disease risk ahead of them, whereas older individuals have "lived through" a portion of their risk.
2. Individuals with existing subthreshold psychiatric symptoms or unusual behaviors or beliefs are at greater risk (Fusar-Poli et al. 2013).
3. Exposure to known environmental triggers may increase the risk to at-risk individuals, although environmental exposures are difficult to identify and evaluate (Peay et al. 2017).

Family history–based risk assessment relies on empiric recurrence risks (see Table 1). A recent meta-analysis of family studies provides a useful set of recurrence risks for schizophrenia, bipolar disorder, and major depressive disorder and highlights the cross-disorder risk (Rasic et al. 2014). Empiric recurrence risks are limited in that they are not specific to any one individual or family but rather reflect aggregated risks for recurrence among large numbers of families that have been evaluated in research studies. Recurrence risks can be reported as relative risks or absolute risks in the literature, which is a critical distinction for disorders with a high population prevalence. Studies consistently demonstrate that recipients of health risk information understand absolute risks more accurately than relative risks (Fagerlin et al. 2011).

Even given those limitations, empiric recurrence risks provide a useful indication of average risk based on the degree of relatedness to an affected individual. Genetic counselors can evaluate the empiric risks for "fit" to any particular family (Peay et al. 2017)—for example, does the family history indicate that your at-risk client faces the same magnitude of risk as the average person who has an affected parent? The available empiric risks can be tailored, to the extent possible, based on the determinants outlined above in the family and personal history. These determinants provide evidence to raise or decrease the familial risk as determined by empiric risks. In some cases, a range of empiric risks is available and tailoring can indicate if the upper or lower end of the range may best fit the family history. In other cases, the personal and/or family history may be sufficiently compelling that the population-based empiric risks should not be seen as representing a valid measure of risk for that family. In those situations, the risk provided to the client may include the empiric risk as a lower limit while including a range that spans well above.

The primary consideration for tailoring empiric risks is the number and pattern of affected (diagnosed or symptomatic) individuals in the family in relation to unaffected individuals in the family. Empiric risks may also need to be adjusted when the proband's psychiatric history indicates an increased risk (e.g., based on early symptom onset) or when there is assortative mating, which can result in substantially elevated risks.

A final consideration is related to the status of the at-risk individual(s) for whom risk assessment is being conducted. As described above, the age of the at-risk individual should be compared to the typical age at onset for the condition. Risks of disorder manifestation are reduced considerably once at-risk individuals live beyond the expected age of onset, although, of course, late age at symptom onset is possible. Genetic counselors must be aware that risk typically extends beyond the diagnosis in the proband to etiologically related conditions (see Table 1).

The rare situations in which there are multiple affected individuals across several generations should raise red flags related to the risk of recurrence. In those situations, even without a developmental/medical history that suggests an underlying genetic syndrome, high risks of re-

currence are possible. This may be due to a rare variant of significant effect or to an unusually large number of risk factors of small effect that are segregating in the family. Either situation calls into question the use of a standard empirical risk ranges and indeed suggests risks that mimic a dominant trait with reduced penetrance. Empiric risks are not available to guide assessment of such "loaded" families, and genetic counselors should use their clinical expertise and judgement to determine whether to provide a quantitative risk range and, if so, what risks to provide.

Provision of Risks

Counselors should customize their approach to risk communication to facilitate achieving the client's objectives for the genetic counseling session and making meaning of etiology and risk information. It is important to convey that genetic factors confer susceptibility through interaction with environmental factors rather than directly causing psychiatric conditions. This discussion may be facilitated through the use of an explanatory model such as the jar model (Peay and Austin 2011; also discussed in Austin 2019).

Genetic counselors should take care to explain the limitations associated with empiric recurrence risks. We simply cannot provide truly personalized risks for the majority of clients who seek psychiatric genetic counseling. In most cases, it is preferable to present a range of empiric risks that reflect the ambiguity associated with the estimates. Empiric risks should be compared to the population risk for the disorder in question to facilitate understanding and interpretation of the data. Genetic counselors should check in with the client to discuss the extent to which presented risks were in alignment with expected risks (Inglis et al. 2017).

Clients who have a psychiatric diagnosis or who are at high risk for psychiatric diagnoses may enter genetic counseling with a predisposition to respond negatively to stressful information, including predictive risk information (Peay et al. 2017). This should not be interpreted as a reason to withhold information. It is instead a relevant contextual factor that should result in a supportive approach to risk counseling.

CONCLUDING REMARKS

Currently, genetic risk assessment for psychiatric disorders is primarily based on family history. The addition of large consortia and large study populations, methodological improvements, and advancements in approaches such as transcriptome, methylome, and neuroimaging studies will allow future research to more thoroughly explicate the relationships of protective and risk factors to phenotypes (Arslan 2018; Avramopoulos 2018; Punzi et al. 2018). These data can be integrated into PRSs and other approaches that may provide clinically meaningful information to clients with psychiatric disorders and to individuals at risk. Although PRSs that achieve clinical utility may first be used in affected individuals to inform diagnosis and treatment, it is naive to imagine that their use in at-risk family members will lag far behind.

Genetic counselors are particularly well-positioned to participate in determinations of clinical and personal utility for PRS and to educate mental health professionals. They are natural leaders for the needed research on the implementation of PRSs and related approaches first in research settings and later in clinical settings. Important research topics for the future include an assessment of the impact of PRSs on knowledge, self-concept, symptom burden, and treatment adherence for affected individuals. For at-risk individuals, studies may evaluate knowledge and risk perception, the positive and negative psychological and social impact of learning the risk information, and any resulting behavior changes for participants.

Longer-term, effective risk-reduction interventions will increase our motivation to apply PRSs to unaffected, at-risk individuals (Hirschhorn 2009) to allow targeting of early intervention approaches (Calafato et al. 2018; Martin et al. 2019). Additional research will be needed to assess health and well-being outcomes for at-risk individuals who engage in risk-reduction interventions (Martin et al. 2019). If these efforts are successful, routine use of PRSs for psy-

chiatric disorders may extend to those at population risk. Genetic counselors should also anticipate the return of incidental findings that have implications for psychiatric outcomes if there are effective risk-reducing interventions. These outcomes would increase the relevance of psychiatric genetic counseling to counselors who work in other specialty areas.

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