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Explanations for Elevated Psychiatric Vulnerability in Nonheterosexuals: Environmental Stressors, Genetics, and the HPA and HPG Axes

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1. Introduction

In Western societies, around 2-5% of men and women identify as gay, lesbian, or bisexual (i.e. nonheterosexual), and at least 10% report at least some attraction to same-sex individuals (Sell et al. 1995; Kendler et al. 2000; Kirk et al. 2000). Nonheterosexuality is not limited to today's Western societies, but has been commonly observed in other cultures today and throughout history (e.g. Hirsch 1992; Murray and Roscoe 1998; Vasey and VanderLaan 2007). In this chapter I firstly outline extensive and robust evidence that sexual minorities are at greater risk of psychiatric disorder than are heterosexuals (Section 2). In Section 3, I explore possible explanations for why this might be the case. I discuss the dominant 'minority stress hypothesis' (Section 3.1); lifestyle factors associated with nonheterosexuality that may exacerbate psychiatric vulnerability (Section 3.2); and 'common cause' explanations involving genetic factors (Section 3.3.1) and adverse childhood experiences (Section 3.3.2), which appear to predispose to both nonheterosexuality and psychiatric disorder. I also propose a novel neurohormonal explanation to account for how the same genetic factors and childhood experiences might affect both sexual orientation and psychiatric risk (Sections 3.3.1.3 and 3.3.2). In Section 4 I conclude and draw attention to areas in which much more research is needed.

2. Nonheterosexuals are at elevated risk of psychiatric disorder

Prior to 1973, homosexuality was classified by the American Psychiatric Association as a disorder in and of itself. However, in that year homosexuality was removed from the DSM II and subsequent editions, and is not now considered a disorder in the psychiatric community (American Psychiatric Association 1973). This chapter describes and proposes explanations for robust findings that nonheterosexuals are at greater risk of psychiatric disorder than the general population. However, it cannot be emphasized strongly enough that this should in no way pathologise nonheterosexuality itself, any more than we should pathologise non-right-handedness, for example, which is also associated with higher rates of psychiatric disorder (Elias et al. 2001; DeLisi et al. 2002).

Several recent large-scale studies have indicated that nonheterosexuals are at elevated risk for many psychiatric symptoms and disorders, including mood disorders (e.g., major depression, bipolar disorder), anxiety disorders (e.g., generalized anxiety disorder, phobic disorders, obsessive compulsive disorder), eating disorders, conduct disorder, substance misuse, suicidal ideation, and suicide attempts (Fergusson et al. 1999; Sandfort et al. 2001; Meyer 2003; Mills et al. 2004; Sandfort et al. 2006; King et al. 2008; Frisell et al. 2010; Bolton and Sareen 2011). These studies have used population samples in several different countries, so the results are unlikely to be due to sampling bias, and the size of the samples and the numerous replications suggests that the findings are not due to sampling error. Furthermore, the size of the effects are by no means trivial – for example, a recent meta-analysis revealed that, compared to heterosexuals, nonheterosexuals are at approximately twice the risk of major depressive disorder (depression) and anxiety disorders, deliberate self harm and attempted suicide (King et al. 2008).

Some studies, particularly earlier ones, suffered from the problems associated with convenience sampling, potentially biasing the estimates of psychiatric risk. However, larger and more representative samples have confirmed the findings. **Table 1 shows results of major population-based studies comparing psychiatric risk in heterosexuals and nonheterosexuals** in which odds ratios (in comparison with heterosexuals) are provided.

Fergusson et al. 1999	<p><u>Men and women pooled</u> N= 1007 (2.8% identify nonheterosexual i.e. gay/lesbian/bisexual)</p> <p>Major depression - OR 4.0; 95%CI 1.8-9.3</p> <p>Generalized anxiety disorder - OR 2.8; 95%CI 1.2-6.5</p> <p>Conduct disorder - OR 3.8; 95%CI 1.7-8.7</p> <p>Nicotine dependence - OR 5.0; 95%CI 2.3-10.9</p> <p>Other substance abuse and/or dependence - OR 1.9; 95%CI 0.9-4.2</p> <p>Multiple disorders - OR 5.9; 95%CI 2.4-14.8</p>
Cochran and Mays 2000	<p>N= 9,908 (2.0% had same-sex partner in previous year)</p> <p><u>Men</u></p> <p>Major depression - 2.94 (1.38-6.28)</p> <p>Generalized anxiety disorder - 2.32 (0.55-9.70)</p> <p>Agoraphobia - 4.85 (0.94-25.17)</p> <p>Panic attack - 4.30 (1.53-12.13)</p> <p>Drug dependency - 2.05 (CI 0.86-4.93)</p> <p>Alcohol dependency - 1.33 (0.55-3.22)</p> <p>Any psychiatric syndrome - 2.26 (1.34-3.89)</p> <p><u>Women</u></p> <p>Major depression - 1.79 (0.74-4.32)</p> <p>Generalized anxiety disorder - OR 1.54 (CI 0.49-4.86)</p> <p>Agoraphobia - 1.41 (0.52-3.84)</p> <p>Panic attack - 1.78 (0.40-8.33)</p> <p>Drug dependency - 3.27 (1.23-8.70)</p> <p>Alcohol dependency - 2.85 (1.16-6.98)</p> <p>Any psychiatric syndrome - 1.63 (CI 0.76-3.48)</p>

(Sandfort et al. 2001)	<p><u>Men</u> N=2,878 (2.8% had same-sex partner in previous year)</p> <p>Mood disorders - 3.11(1.91-5.05)</p> <p>Major depression - 2.35 (1.39-3.97)</p> <p>Dysthymia 2.33 (0.94-5.75)</p> <p>Bipolar disorder 7.27 (2.85-18.52)</p> <p>Anxiety disorders 2.67 (1.62-4.41)</p> <p>Panic disorder 4.21 (1.65-10.77)</p> <p>Agoraphobia (without panic) 4.54 (1.79-11.53)</p> <p>Simple phobia 3.61 (1.94-6.74)</p> <p>Social phobia 2.29 (1.17-4.50)</p> <p>Generalized anxiety disorder 2.88 (0.82-10.18)</p> <p>Obsessive compulsive disorder 6.20 (2.03-18.90)</p> <p>Substance use disorders total 0.79 (0.48-1.32)</p> <p>Alcohol abuse 0.48 (0.24-0.95)</p> <p>Alcohol dependence 1.23 (0.62-2.44)</p> <p>Drug abuse 1.34 (0.45-4.01)</p> <p>Drug dependence 2.47 (0.82-7.45)</p> <p><u>Women</u> N=3,120 (1.4% had same-sex partner in previous year)</p> <p>Mood disorders 2.41 (1.26-4.63)</p> <p>Major depression 2.44 (1.26-4.72)</p> <p>Dysthymia 1.62 (0.65-4.02)</p> <p>Bipolar disorder 0.92 (0.12-6.97)</p> <p>Anxiety disorders 0.96 (0.46-1.97)</p> <p>Panic disorder 0.75 (0.18-3.20)</p> <p>Agoraphobia (without panic) 1.36 (0.41-4.56)</p> <p>Simple phobia 1.27 (0.51-2.97)</p> <p>Social phobia 1.81 (0.79-4.14)</p> <p>Generalized anxiety disorder 0.84 (0.11-6.28)</p> <p>Obsessive compulsive disorder --</p>
Sandfort et al. 2001 (continued)	<p>Substance use disorders total 3.43 (1.60-7.33)</p> <p>Alcohol abuse 2.01 (0.60-6.79)</p> <p>Alcohol dependence 3.59 (1.16-11.18)</p> <p>Drug abuse 1.88 (0.23-15.33)</p> <p>Drug dependence 8.04 (2.49-25.91)</p>

Gilman et al. 2001	<p><u>Men</u> N=2,384 (3.1% had same-sex partner in previous year)</p> <p>Anxiety disorders 1.3 (0.8-2.4)</p> <p style="padding-left: 20px;">Agoraphobia 1.1 (0.3-3.9)</p> <p style="padding-left: 20px;">Generalised anxiety disorder 2.8 (1.0-8.0)</p> <p style="padding-left: 20px;">Panic 1.2 (0.2-6.5)</p> <p style="padding-left: 20px;">Social phobia 1.6 (0.7-3.5)</p> <p style="padding-left: 20px;">Simple phobia 1.0 (0.4-2.5)</p> <p style="padding-left: 20px;">Post-traumatic stress disorder 1.1 (0.5-2.5)</p> <p>Mood disorders 1.3 (0.8-2.4)</p> <p style="padding-left: 20px;">Major depression 1.5 (0.7-3.0)</p> <p style="padding-left: 20px;">Dysthymia 1.1 (0.4-2.8)</p> <p>Substance use disorders 1.5 (0.8-2.8)</p> <p style="padding-left: 20px;">Alcohol abuse 1.2 (0.7-2.3)</p> <p style="padding-left: 20px;">Alcohol dependence 1.4 (0.6-3.0)</p> <p style="padding-left: 20px;">Drug abuse 2.8 (1.6-5.1)</p> <p style="padding-left: 20px;">Drug dependence 2.4 (1.2-4.8)</p> <p><u>Women</u> N=2,526 (2.0% had same-sex partner in previous year)</p> <p>Anxiety disorders 1.8 (1.2-2.8)</p> <p style="padding-left: 20px;">Agoraphobia 1.1 (0.2-5.9)</p> <p style="padding-left: 20px;">Generalised anxiety disorder 3.2 (1.4-7.3)</p> <p style="padding-left: 20px;">Panic 2.6 (0.9-7.7)</p> <p style="padding-left: 20px;">Social phobia 1.5 (0.7-3.3)</p> <p style="padding-left: 20px;">Simple phobia 1.8 (1.2-2.9)</p> <p style="padding-left: 20px;">Post-traumatic stress disorder 2.7 (1.2-6.1)</p> <p>Mood disorders 2.0 (1.1- 3.5)</p> <p style="padding-left: 20px;">Major depression 1.9 (1.0-3.3)</p> <p style="padding-left: 20px;">Dysthymia 1.9 (0.8-4.4)</p> <p>Substance use disorders 2.4 (1.3-4.4)</p> <p style="padding-left: 20px;">Alcohol abuse 1.8 (0.7-4.5)</p> <p style="padding-left: 20px;">Alcohol dependence 2.2 (0.9-5.6)</p> <p style="padding-left: 20px;">Drug abuse 4.4 (2.4-8.1)</p> <p style="padding-left: 20px;">Drug dependence 1.7 (0.5-5.5)</p>
Chakraborty et al.	<p><u>Men and women pooled</u> N=7,403 (8.8% identify nonheterosexual i.e. gay/lesbian/bisexual)</p> <p>Depressive episode - 1.80 (1.13–2.87)</p> <p>Generalised anxiety disorder - 1.49 (1.03–2.15)</p> <p>Obsessive–compulsive disorder - 2.24 (1.18–4.27)</p> <p>Phobic disorder - 1.91 (1.07–3.39)</p> <p>Probable psychosis - 3.75 (CI 1.76–8.00)</p> <p>Drug dependence - 1.70 (1.06–2.73)</p> <p>Alcohol dependence - 2.05 (1.45–2.90)</p>

<p>Bolton and Sareen 2011 * Using the same dataset, Bostwick et al (2010) show the elevation in psychiatric risk differs depending on the measure of sexual orientation used.</p>	<p><u>Men</u> N=14,449 (1.3% identify gay, 0.6% bisexual, 0.5% not sure) Any mood disorder [Gay] 2.98 (2.11–4.20) [Bi] 2.36 (1.44–3.88) [Not sure] 1.93 (1.04–3.59) Any anxiety disorder 2.66(1.78–3.97); 2.09(1.20–3.64) ; 2.07 (1.06–4.03) Any substance use disorder 1.77 (1.25–2.51) ; 1.30 (0.71–2.36) ; 0.77 (0.41–1.45) Any personality disorder 1.82 (1.19–2.77) ; 1.66 (0.98–2.82) ; 2.46 (1.34–4.51) Schizophrenia, psychotic illness, or episode 2.85 (1.57–5.15) ; [–] ; 3.80 (1.44–10.04) <u>Women</u> N=19,896 (0.7% identify lesbian, 0.8% bisexual, 0.5% not sure) Any mood disorder [Lesbian] 1.60 (1.05–2.44) [Bi] 2.66 (1.83–3.89) [Not sure] 1.23 (0.70–2.16) Any anxiety disorder 1.53 (1.01–2.32); 3.09 (2.04–4.68); 1.17 (0.69–1.99) Any substance use disorder 1.37 3.41 (2.13–5.44); 3.90 (2.64–5.75); 1.88 (1.01–3.49) Any personality disorder 1.74 (1.13–2.69); 2.12 (1.43–3.13); 1.65 (0.87–3.15) Schizophrenia, psychotic illness, or episode [–] ; 3.18 (1.78–5.69); [–]</p>
<p>Zietsch et al. in press</p>	<p>N= 9,884 <u>Men</u> (4.3% identify nonheterosexual i.e. gay/lesbian/bisexual) Lifetime depression 2.8 (2.0-3.9) <u>Women</u> (3.3% identify nonheterosexual i.e. gay/lesbian/bisexual) Lifetime depression 2.7 (1.9-3.7)</p>

Table 1. Major population-based studies comparing psychiatric risk in heterosexuals and nonheterosexuals. Odds ratios are with reference to rates in heterosexuals (i.e. for whom OR=1), and are accompanied by 95% confidence intervals in brackets. Where applicable, odds ratios presented are those adjusted for possible confounds. NB: We focus here on psychiatric risk and do not include several studies on suicidality.

3. What might explain the elevated psychiatric vulnerability in nonheterosexuals?

3.1 The minority stress hypothesis

Perhaps the first explanation that comes to mind is that nonheterosexuality is stigmatised in many societies, and that it must be stressful and depressing to be frequently subject to prejudice and discrimination. Indeed, this is the basis of the “minority stress” hypothesis, the dominant explanation for explaining elevated psychiatric vulnerability in nonheterosexuals (Meyer 1995; Mays and Cochran 2001; Meyer 2003; Lehavot and Simoni 2011). Meyer (2003) describes a number of stress processes that may increase psychiatric risk in nonheterosexuals. These include the experience of prejudice events, expectations of rejection, hiding and concealing, internalized homophobia, and ameliorative coping processes. Some experiences of prejudice may be institutionalised discrimination, such as legal bans on gay marriage or religious intolerance of homosexuality, but many are likely to be everyday experiences of negativity, rejection, and labelling (Meyer 1995). Hatzenbuehler

(2009) describes a theoretical framework for understanding psychological mechanisms by which these stigma-related experiences may cause psychopathology.

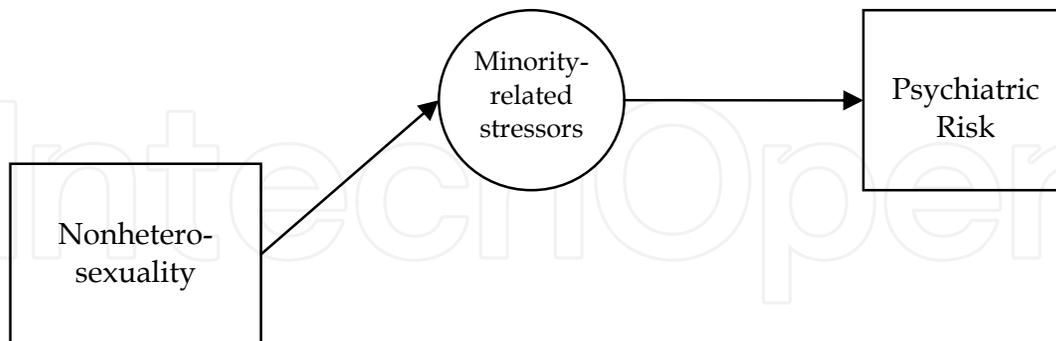


Fig. 1. Nonheterosexuality leads to exposure to minority-related stressors, which leads to elevated psychiatric risk.

Two studies provide strong evidence that homophobic prejudice and discrimination are indeed involved in elevated psychiatric risk in nonheterosexuals (Mays and Cochran 2001; Frisell et al. 2010). In community-based samples, both studies confirmed that nonheterosexuals reported more prejudice and discrimination than heterosexuals (e.g. being fired from a job, or threatened, harassed, or insulted), and found that controlling for reported levels of discrimination attenuated the relationship between sexual orientation and psychiatric disorder.

While it is tempting to dust off the hands and proclaim the mystery solved, **there are indications that minority stress may not be a complete explanation.** Firstly, in both studies mentioned above (Mays and Cochran 2001; Frisell et al. 2010), even after controlling for reported levels of prejudice and discrimination there remained considerable elevation in psychiatric risk in nonheterosexuals. Secondly, heterosexual relatives of nonheterosexuals are also at greater psychiatric risk than the general population (Frisell et al. 2010; Zietsch et al. in press), suggesting that familial (i.e. genetic or family environmental) factors other than minority stress are also important. Thirdly, studies show the relationship between sexual orientation and mental health is just as strong in The Netherlands, where there has long been greater cultural acceptance of homosexuality than in other countries (Sandfort et al. 2001; Lewis 2009). Fourth, racial-ethnic minorities in the US appear to have *lower* psychiatric risk than the majority white population, despite their experiences of minority-related prejudice and discrimination (Breslau et al. 2006). Nonheterosexuals' minority status probably impacts differently on their mental health because they normally have heterosexual family members who may also stigmatise nonheterosexuality. However, it appears that individuals with developmental physical disabilities (who may also be stigmatised and not have similarly affected family members) also have similar or lower risk of psychiatric disorder than the general population (Australian Bureau of Statistics 1998; Hagiliassis et al. 2005; Ostlie et al. 2010), reinforcing the suggestion that minority stress does not necessarily elevate psychiatric risk.

As such, **there may be additional mechanisms generating the link between sexual orientation and psychiatric risk.** This does not imply that I am skeptical of the role of minority stress, only that *additional* explanations warrant investigation. To date, additional

explanations have hardly even been discussed, let alone empirically investigated. Below I describe some of these possible additional explanations, and any relevant evidence.

3.2 Lifestyle factors associated with nonheterosexuality

It has been suggested that lifestyle factors associated with nonheterosexuality may explain some of its link with elevated psychiatric risk (Bailey 1999). This is a broad explanation that depends firstly on understanding how the lifestyles of nonheterosexuals tend to differ from those of heterosexuals, and secondly identifying those lifestyle differences that could be relevant to psychiatric risk. Below I outline a number of lifestyle factors for which are likely to differ with sexual orientation and may also be related to psychiatric risk, but the list is not intended to be exhaustive.

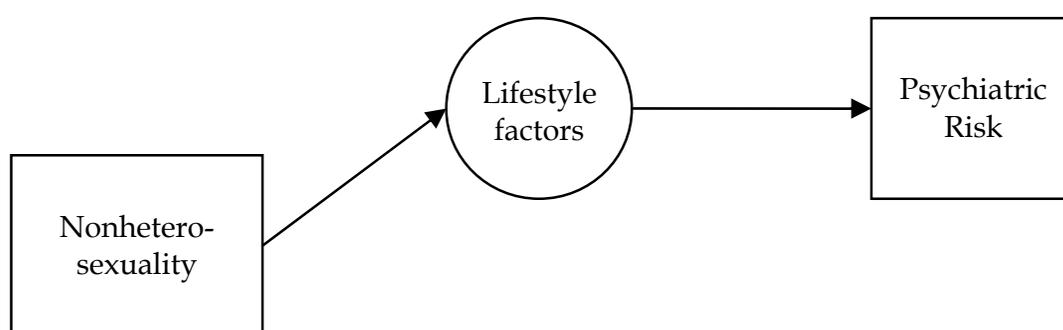


Fig. 2. Nonheterosexuality leads to associated lifestyle factors, which lead to elevated psychiatric risk.

3.2.1 Relationship stability

It appears that gay and lesbian relationships tend to be less stable than heterosexual relationships; in a 5-year study, both gay and lesbian cohabiting couples were more likely to breakup than were heterosexual married couples (Kurdek 1998). (Note that this could be related to legal barriers to a) homosexuals wishing to get married and b) heterosexual married couples wishing to break up.) If homosexual relationships are less stable, it could pose a risk to mental health to nonheterosexuals given that relationship dissolution is a major life stressor that can provoke psychiatric problems (Chung et al. 2002).

3.2.2 Difficulty of having children

An important lifestyle consideration distinctive to homosexual couples is that, without the aid of a third party, they cannot have children. In addition, homosexual couples wishing to have or adopt a child may face legal obstacles in many countries. The little evidence there is suggests that lesbian women's desire to have children is at least as strong as their heterosexual counterparts (Bos et al. 2003). The difficulty of fulfilling this desire could be a psychiatric risk factor, especially in light of findings infertile women have elevated risk of psychiatric disorder (Noorbala et al. 2009). In the absence of evidence to the contrary, it can only be assumed that the same issues apply to gay men.

3.2.3 Substance use

Substance use and dependence is more prevalent among nonheterosexuals (McKirnan and Peterson 1989a; Fergusson et al. 1999; Cochran and Mays 2000; Chakraborty et al. 2011), and

is also a risk factor for psychiatric disorder (Semple et al. 2005). It is unclear if drug use is more often part of nonheterosexuals' chosen lifestyles, and/or if increased drug use is a coping mechanism for the stressors and psychiatric difficulties nonheterosexuals are more likely to encounter. There is evidence that the latter is true (McKirnan and Peterson 1989b), but if the former is also true (e.g. Mansergh et al. 2001), one way to reduce mental health risk in nonheterosexuals may be to target recreational drug use in the gay and lesbian communities. Other mechanisms are also possible (e.g. common causes, see Section 3.3. below).

3.2.4 Sexually transmitted disease and general health

Sexually transmitted diseases, including AIDS, are more common in male nonheterosexuals than in male heterosexuals, due to higher incidence of risky sexual behaviours (Cates and Panel Am Social Hlth Assoc 1999; Stolte and Coutinho 2002). Acquiring sexually transmitted diseases is associated with distress (Cochran and Mays 2007), and AIDS is certainly a major life stressor. Both male and female nonheterosexuals report poorer physical health in general than heterosexuals (Sandfort et al. 2006; Cochran and Mays 2007), though it is unclear whether this is due to lifestyle choices, minority-related stressors, or some other mechanism (e.g. common causes, see Section 3.3. below).

3.2.5 Body image

Nonheterosexual men have greater concerns about body image and greater incidence of eating disorders than do heterosexual men; on the other hand, nonheterosexual women actually have fewer problems than heterosexuals with body image and eating disorders (Herzog et al. 1992; Gettelman and Thompson 1993; French et al. 1996). This may be because men are the sexual targets of homosexual men and heterosexual women, with men placing higher importance on physical appearance than women (Buss 1989).

3.3 Common causes of nonheterosexuality and psychiatric disorder

It is obvious to ask if the link between nonheterosexuality and psychiatric risk is due to factors that cause both, but it is difficult to answer in practice due to our limited understanding of the causes of either psychiatric disorder or, especially, nonheterosexuality. Here I will focus on genetic factors and adverse childhood experiences, which are associated with both sexual orientation and psychiatric risk.

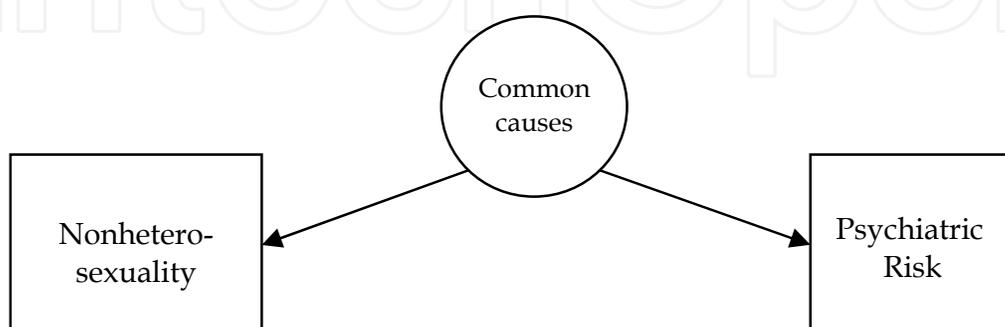


Fig. 3. Common causes lead to both nonheterosexuality and psychiatric risk.

3.3.1 Genetic factors

It is well established that **genetic factors influence sexual orientation**. Early family studies in men (Bailey and Pillard 1991) and women (Bailey et al. 1993) revealed that biological siblings (twins) were much more concordant in their sexual orientation than were adoptive siblings, suggesting that genes or intrauterine effects were more important than factors associated with the family environment. They also showed that identical (monozygotic; MZ) twins, who share 100% of their genes, were more concordant than nonidentical (dizygotic; DZ) twins, who share on average 50% of their genes – this indicated that the familial effects were at least partly genetic; statistical modeling procedures (Neale and Cardon 1992; Posthuma et al. 2003) yielded estimates that genetic factors accounted for at least half of the variance in sexual orientation (i.e. heritability > 50%).

These early studies were criticized for using convenience samples (Lidz 1993), which may have biased the heritability estimates. Indeed, the numerous subsequent studies using very large, representative, community-based twin samples, have often yielded somewhat lower heritability estimates (≈20-50%), but they have consistently replicated the finding that MZ twins are more concordant in their sexuality than DZ twins (Bailey et al. 2000; Kendler et al. 2000; Santtila et al. 2008; Zietsch et al. 2008; Langstrom et al. 2010). **Table 2 summarizes the findings of major twin studies on sexual orientation.**

Bailey and Pillard 1991	<p>Recruitment nonheterosexual probands recruited through ads in gay-oriented publications</p> <p>Classification [homosexual/bisexual] vs heterosexual</p> <p><u>Men</u> 52% MZ concordance (N=56 pairs) 22% DZ concordance (N=54 pairs) 11% adoptive brothers concordance (N=57 pairs)</p>
Bailey et al. 1993	<p>Recruitment nonheterosexual probands recruited through ads in gay-oriented publications</p> <p>Classification [homosexual/bisexual] vs heterosexual</p> <p><u>Women</u> 48% MZ concordance (N=71 pairs) 16% DZ concordance (N=37 pairs) 6% adoptive sisters concordance (N=35 pairs)</p>
Kendler et al. 2000	<p>Recruitment national twin sample</p> <p>Classification [homosexual/bisexual] vs heterosexual</p> <p><u>Men and women combined</u> .68 MZ twin pair correlation (N=324 pairs) .43 DZ twin/sibling pair correlation (1806 pairs)</p>

Zietsch et al. 2008 *Bailey et al (2000) analyzed the same dataset but used a more complex, composite measure of sexual orientation, which yielded lower heritability estimates of female sexual orientation.	Recruitment community-based twin sample Classification [Kinsey attraction scale ≥ 1] vs [Kinsey attraction scale < 1] <u>Men</u> .57 (95%CI: .33-.74) MZ twin pair correlation (N=312 pairs) .20 (95%CI: -.11 -.49) DZ twin pair correlation (N=185 pairs) <u>Women</u> .47 (95%CI: .33-.74) MZ twin pair correlation (N=667 pairs) .37 (95%CI: .33-.74) DZ twin pair correlation (N=377 pairs) <u>Opposite sex twin pairs</u> -.01 (95%CI: .33-.74) DZ twin pair correlation (N=366 pairs)
Santtila et al. 2008	Recruitment community-based twin sample Classification potential for homosexual response: 6-point scale; quite impossible - very likely <u>Men</u> (N=3152 individuals) .53 (SE=.08) MZ twin pair correlation .23 (SE= .11) DZ twin pair correlation <u>Women</u> (N=6001 individuals) .53 (SE=.04) MZ twin pair correlation .26 (SE=.05) DZ twin pair correlation
Langstrom et al. 2010	Recruitment national twin sample Classification [any same-sex sexual partners] vs [no same-sex sexual partners] <u>Men</u> .39 MZ twin pair correlation (N=807 pairs) .19 DZ twin pair correlation (N=517 pairs) <u>Women</u> .36 MZ twin pair correlation (N=1513 pairs) .27 DZ twin pair correlation (N=989 pairs)
Zietsch et al. in press	Recruitment community-based sample Classification [homosexual/bisexual] vs heterosexual <u>Men</u> .50 (95%CI: .25-.60) MZ twin pair correlation (N=633 pairs) .27 (95%CI: -.12-.60) DZ twin pair correlation (N=503 pairs) <u>Women</u> .53 (95%CI: .33-.70) MZ twin pair correlation (N=1079 pairs) .33 (95%CI: .04-.57) DZ twin pair correlation (N=811 pairs) <u>Opposite-sex pairs</u> .43 (95%CI: .12-.66) DZ twin pair correlation (N=866 pairs)

Table 2. Tetrachoric correlations (i.e. twin similarity) for sexual orientation in identical (MZ) and nonidentical (DZ) twin pairs in major studies.

Many large twin studies have also consistently shown that **psychiatric disorders are substantially influenced by genetic factors** (see Sullivan et al. 2000; Bouchard and McGue 2003; Sullivan et al. 2003 for reviews), with a wide range of heritabilities from around 40% for depression to around 80% for schizophrenia. Many psychiatric disorders are highly comorbid (i.e. they are often found together in the same individuals), particularly those related to anxiety and depression, including phobias, generalised anxiety disorder, major depressive disorder, obsessive compulsive disorder, and substance use disorders (Rohde et al. 1991; Kendler et al. 1992). As well as estimating genetic influence on variation in a psychiatric disorder, twin studies can also be used to estimate the genetic influence on covariation between different disorders, by modelling cross-twin cross-trait correlations. If the cross-twin cross-trait correlation is greater in MZ pairs than in DZ pairs it suggests a genetic correlation between the traits, and modelling can estimate what proportion of the total correlation between the traits is due to this genetic correlation (as opposed to correlation of environmental factors); see Neale and Cardon (1992) or Posthuma et al (2003) for details. This type of multivariate genetic analysis has revealed that comorbidity between the various anxiety-related psychiatric disorders is largely due to overlapping genetic influences (Kendler et al. 1992; Hettema et al. 2005). This suggests that the genetic factors involved are pleiotropic (i.e. have multiple effects, e.g. predispose to multiple disorders), though it should be noted that these genetic factors could influence the different disorders via different genes that are in linkage disequilibrium (i.e. genes that tend to be inherited together).

Given that we know that **genetic factors influence both psychiatric disorder and sexual orientation**, the next question is, **do these genetic factors overlap?** A genetic correlation would be necessary for a genetic 'common causes' explanation to be plausible. My colleagues and I (Zietsch et al. 2011) first tested this possibility in a large twin sample (N=4904) by investigating the relationship between sexual orientation and Eysenck's personality traits Neuroticism and Psychoticism, putative measures of vulnerability to internalizing (e.g. depressive and anxiety) disorders and psychotic disorders (e.g. schizophrenic), respectively (Eysenck 1967; Eysenck and Eysenck 1976). Neuroticism has been shown to be a premorbid predictor of depression (Kendler et al. 1993; Ormel et al. 2004), accounts for comorbidity with anxiety (Andrews 1996; Khan et al. 2005), and is also associated with eating disorders (Cassin and von Ranson 2005). Similarly, Psychoticism is a premorbid predictor of schizophrenia and other psychotic disorders (Claridge et al. 1983; Laurent et al. 2002) and is related to antisocial behavior and conduct problems (Tranah et al. 1998; Miller and Lynam 2001; Cale 2006). Both Neuroticism and Psychoticism are related to suicidal behavior (Nordstrom et al. 1995). We found that nonheterosexual men and women scored significantly higher on both Neuroticism and Psychoticism than their heterosexual counterparts, consistent with their greater psychiatric risk found elsewhere. Using bivariate genetic models, we showed that the association of sexual orientation with both Neuroticism and Psychoticism was due primarily to significant genetic correlations, whereas the corresponding environmental correlations were not significant. This provides some support for the idea that overlapping genetic factors act as a common cause of both sexual orientation and psychiatric vulnerability.

In a second study testing this idea, my colleagues and I (Zietsch et al. in press) investigated the relationship between sexual orientation and depression in a large twin sample (N=9,884). We found that nonheterosexuals were at significantly greater risk of depression

and again we found that a significant genetic correlation was primarily responsible, accounting for 60% of the total correlation between sexual orientation and depression.

Since it appears that pleiotropic genetic factors may act as a common cause of nonheterosexuality and psychiatric risk, it is natural to ask: **what mechanisms might confer this shared genetic risk?** Below I describe why explanations involving specific genes or general mutation load are unlikely to be viable. I then propose a more plausible theory involving interaction between the hypothalamic-pituitary-gonadal (HPG) axis and the hypothalamic-pituitary-adrenal (HPA) axis.

3.3.1.1 Specific genes

The recent availability of high-density genotype information has enabled studies that test for association between hundreds of thousands of genetic variants (single nucleotide polymorphisms; SNPs) and any given trait. These genome-wide association (GWA) studies have yielded very few robustly replicated associations between specific genes and complex psychological traits, including psychiatric disorder (Manolio et al. 2009; Sullivan et al. 2009; Verweij et al. 2010). Genes previously 'identified' using earlier approaches (linkage, candidate gene association) as influencing traits such as personality (e.g. Ebstein et al. 1996), depression (e.g. Ogilvie et al. 1996), or substance use (see Agrawal and Lynskey 2009) have generally failed to replicate using these more powerful methods (Verweij et al. 2010; Bosker et al. 2011; Verweij et al. 2011). This suggests publication bias towards positive findings and replications may have resulted in a high proportion of the reports being false positives.

Early findings of linkage of a gene on the X-chromosome with sexual orientation (Hamer et al. 1993; Hu et al. 1995) failed to consistently replicate in subsequent studies (Rice et al. 1999; Mustanski et al. 2005). Sexual orientation has not yet been subject to GWA analysis (it is very expensive and requires very large samples), but **it seems highly unlikely that variation in sexual orientation could be explained with a small number of genes of large effect** given that this is not the case for any other psychological trait that has been studied with GWA methodology (Manolio et al. 2009).

Studies examining the combined effects of thousands of SNPs on complex traits suggest that 'missing heritability' (the discrepancy between high heritability estimates from twin-family studies and inability to identify the specific genetic variants responsible) is partly because of the extremely small effect sizes of any individual genetic variant, and partly because many of the genetic variants involved are rare in the population (conventional GWA studies cannot detect rare variants) (Manolio et al. 2009; The International Schizophrenia Consortium 2009; Yang et al. 2010). Variation in both sexual orientation and depression is likely to be due to thousands of genetic variants of tiny effect size. If this is the case, **the large genetic correlation between sexual orientation and depression could not be due to a few key genes.**

3.3.1.2 Mutation load

Bailey (1999) suggested that elevated rates of nonrighthandedness that have been observed in both nonheterosexuals and the mentally ill may reflect a biological factor that manifests in unusual laterality, sexual orientation, and psychiatric problems.

Mutation load is an individual's aggregate of mildly deleterious mutations acquired in recent generations. It is thought to be related to psychiatric disorder via increased developmental instability (Yeo et al. 1999; Keller and Miller 2006). In theory, a high mutation load could predispose to both depression and nonheterosexuality via multiple downstream

effects of the destabilising mutations. Indeed, both nonheterosexuals and those with psychiatric disorders are more likely to be nonrighthanded (Hicks and Pellegrini 1978; Elias et al. 2001; DeLisi et al. 2002; Lippa 2003), which is thought to be an indicator of developmental instability (Yeo and Gangestad 1993; Yeo et al. 1993).

However, traits affected by mutation load are also expected to be associated with advanced paternal age, since the sperm of older men carry more de novo mutations (Crow 2000). Accordingly, advanced paternal age has been associated with higher risk of autism, bipolar, schizophrenia, and lower IQ (Auroux et al. 1989; Malaspina et al. 2001; Reichenberg et al. 2006; Frans et al. 2008; Saha et al. 2009). In contrast, paternal (or maternal) age appears to have no effect on either sexual orientation or depression (Zietsch et al. in press). Furthermore, previous studies found no correlation between sexual orientation and fluctuating asymmetry (Mustanski et al. 2000; Rahman and Wilson 2003b; Rahman 2005a), the best-documented marker of developmental instability (Gangestad and Simpson 2000). Therefore, **mutation load does not seem a viable explanation for the genetic correlation between sexual orientation and depression.**

3.3.1.3 Interaction between the HPA and HPG axes

I propose a novel and more plausible explanation involving a neurohormonal mechanism, but it is speculative and requires direct testing in future research. Its basis is that (unknown) genetic factors affect both sexual orientation and psychiatric risk via effects on the hypothalamic-pituitary-gonadal (HPG) axis, which stimulates sex hormone production (primarily, testosterone in men and estrogen in women). Though the details are far from clear, sex-atypical levels of gonadal hormones during development are thought to be involved in sexual orientation (Gooren 2006; Swaab and Garcia-Falgueras 2009), and the most robust correlates of nonheterosexual orientation relate to sex-atypicality in behavior, cognition, and brain structure (Bailey et al. 2000; Rahman and Wilson 2003a; Swaab 2008). Therefore, via low gonadal hormone levels during development, genetically low activity of the HPG axis might have organizing effects on the brain and predispose to a nonheterosexual orientation. Meanwhile, low gonadal hormone levels are also involved in the etiology of depressive and anxiety disorders. Depressed women have lower plasma levels of estrogen and depressed men have lower testosterone levels (Young et al. 2000; Swaab et al. 2005; Walf and Frye 2006; Zarrour et al. 2009), and administering testosterone and estrogen has been found to reduce depression in men and women, respectively (Soares et al. 2001; Walf and Frye 2006; Zarrour et al. 2009). Animal models suggest the effect in males is due to testosterone's suppression of the hypothalamic-pituitary-adrenal (HPA) axis (i.e. the stress system; Evuarherhe et al. 2009), whereas less established mechanisms in females appear to involve estrogen receptors in the hippocampus and amygdala, which in turn regulate HPA activity (Walf and Frye 2006). Also, gonadal hormone levels during development have programming effects affecting adult HPA reactivity (Evuarherhe et al. 2009; Romeo 2010). Thus, **genetic factors associated with low HPG axis activity (either throughout development or in critical phases) might predispose to development of both nonheterosexual orientation and vulnerability to disorders related to the stress response.**

3.3.2 Adverse childhood experiences – risky family environment and sexual abuse

Along with genetic factors, other possible common causes of both nonheterosexuality and psychiatric risk are adverse childhood experiences such as 'risky' family environment and sexual abuse.

Risky family environments are those characterized by conflict and relationships that are cold, unsupportive, and neglectful (Repetti et al. 2002). Such family environments are known to be associated with a range of psychiatric problems, including aggression, conduct disorder, delinquency and antisocial behaviour, anxiety, depression, and suicide (Felitti et al. 1998; Kendler et al. 2002; Repetti et al. 2002; Kendler et al. 2006). These associations are partly a function of genetic predispositions that underlie the damaging parental behaviours and are inherited by the children (Plomin 1994), but other research (including longitudinal studies) suggests that risky family environment also plays a direct role in predisposing to disorder (O'Connor et al. 1998; Johnson et al. 2001). Childhood sexual abuse has also been strongly linked to psychiatric risk (Felitti et al. 1998; Kendler et al. 2002; Nelson et al. 2002; Kendler et al. 2004; Kendler et al. 2006; Fergusson et al. 2008). Though the mechanisms involved are not yet entirely clear, there is mounting evidence that these associations between adverse childhood experiences and psychiatric risk are mediated by permanent effects of repeated stressors on HPA axis reactivity (Heim et al. 2008; Pariante and Lightman 2008; Lupien et al. 2009; Romeo 2010; Young and Korszun 2010).

Several studies have shown elevated rates of childhood sexual abuse and risky family environment in nonheterosexuals (Cameron and Cameron 1995; Lenderking et al. 1997; Fergusson et al. 1999; Paul et al. 2001; Tomeo et al. 2001; Balsam et al. 2005; Arreola et al. 2008; Alanko et al. 2009; Zietsch et al. in press). A meta-analysis of studies examining the prevalence of childhood sexual abuse in nonheterosexuals (Rothman et al. 2011) yielded higher rates than corresponding rates in a meta-analysis of studies of the general population (Stoltenborgh et al. 2011). A problem with most studies comparing rates of childhood sexual abuse by sexual orientation is that nonheterosexual samples are usually selected based on their sexual orientation (e.g. recruiting in gay-oriented magazines), leading to unpredictable biases on the results. To my knowledge only one study has directly compared rates of childhood sexual abuse in heterosexuals and nonheterosexuals in a large population-based sample selected without reference to sexual orientation or child abuse (Zietsch et al. in press) – it found that rates of childhood sexual abuse (and risky family environment) were significantly higher (odds ratios $\approx 2-3$) in nonheterosexuals than heterosexuals and were also associated with higher rates of depression. These dual effects (i.e. childhood risky family environment and sexual abuse) contributed to the elevated rates of depression in nonheterosexuals, but only to a modest degree (their combined effect accounted for around 16% of the sexual orientation-depression link).

It is not at all clear how adverse childhood experiences might affect adult sexual orientation, and indeed the prevailing scientific view is that sexual orientation is fixed before birth (Rahman 2005b; Swaab and Garcia-Falgueras 2009). **I propose that the aforementioned interaction of the HPA and HPG axes also provides a possible mechanism for stressful childhood experiences to influence sexual orientation** in already predisposed individuals. Again, this is speculative and the hypothesis requires direct empirical testing.

Persistent or repeated stress exposure (activation of the HPA axis) inhibits activity in the HPG axis (Kirby et al. 2009), so repeated stressors during development may chronically disrupt gonadal hormone levels (low testosterone and estrogen levels in males and females, respectively). The deficiency in gonadal hormones (either throughout development, or in critical stages such as puberty, when gonadal hormone levels surge; Romeo 2010) may have organizing effects on the brain (van Goozen et al. 2002; Gooren 2006; Neufang et al. 2009; Peper et al. 2010), decreasing sexual differentiation and increasing the likelihood of same-

sex attraction. This could explain the effect of childhood risky family environment and sexual abuse on sexual orientation.

An alternative version of this explanation could involve prenatal transmission of maternal stress, and this would fit better with the conventional wisdom that sexual orientation is largely determined before birth. As well as directly posing problems for a developing child, a risky family environment may also impact the child before it is born via the impact of family dysfunction on the mothers' stress levels during pregnancy. Maternal stress during certain prenatal periods may predispose the offspring increased HPA axis reactivity (Tollenaar et al. 2011) and to various psychiatric disorders (Koenig et al. 2002; Beydoun and Saftlas 2008). Animal models suggest that maternal stress can decrease behavioural sexual differentiation (Goel and Bale 2009) and increase homosexual behaviour (Meek et al. 2006). There is also some human evidence that maternal stress may have (modest) effects on the offspring's adult sexual orientation (Ellis and Cole-Harding 2001), though other negative findings on child gender-role behaviour casts doubt on this (Hines et al. 2002). Both the human and animal literature is complicated by findings that maternal stress has different effects in males and females (Ellis and Cole-Harding 2001; Weinstock 2007). A further problem, particularly for the human studies, is the difficulty of distinguishing the prenatal effects of maternal stress from genetic predispositions or postnatal stressful experiences of the child (Beydoun and Saftlas 2008). Nevertheless, whether directly or via prenatal maternal effects it is possible that stressors during development may have an impact on both sexual orientation and psychiatric risk through the interactions of the HPA and HPG axes.

4. Conclusion

There is good evidence to suggest that minority stress increases the psychiatric risk of nonheterosexuals, but other evidence suggests that additional mechanisms also contribute. These may involve lifestyle factors associated with nonheterosexuality, and also common causes of both nonheterosexuality and psychiatric risk. These common causes appear to involve both genetic factors and adverse childhood experiences. Of course, it is likely that numerous factors are responsible, and the importance of each factor probably differs across specific psychiatric disorders. For example, while minority stress appears to play an important role in depression, it could not be responsible for the elevated risk of eating disorders in nonheterosexual men, given that lesbian women are at lower risk than heterosexual women.

Very importantly, most of the findings described above involve great uncertainty regarding causation and specific mechanisms involved. For example, for simplicity I have assumed that the association of adverse childhood experiences with adult sexual orientation is due to the former predisposing to the latter, but there are many other causal possibilities, some more plausible than others. Longitudinal studies, especially those with genetically informative samples, may help to resolve some of these alternative explanations. Indeed, there is enormous scope for future work in this area, given the importance of the problem and how much is unknown. In particular, the neurohormonal mechanism I proposed above is potentially important, but is speculative and requires rigorous empirical testing and theoretical development.

It is crucially important to understand these and other issues so we can know how best to improve the psychiatric wellbeing of nonheterosexual individuals. Of course, the most sure-

fire steps involve reducing the stigma, prejudice, and discrimination that sexual minorities face in everyday life.

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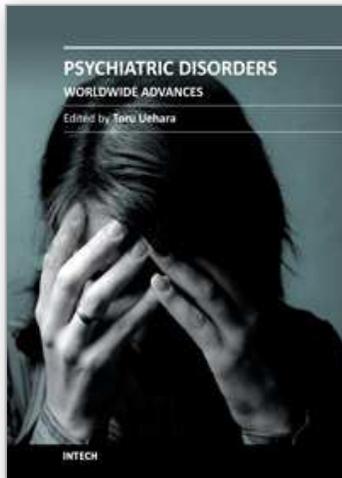
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A psychiatric disorder is defined as any complex condition that involves the impairment of cognitive, emotional, or behavioral functioning. Aside from knowing the physical organic factors, its causal pathology has remained a mystery. Regarding recent advances in psychiatry and neurosciences, psychiatric disorders have been closely associated with socio-cultural, psychological, biochemical, epigenetic or neural-networking factors. A need for diverse approaches or support strategies is present, which should serve as common knowledge, empathetic views or useful skills for specialists in the field. This book contains multifarious and powerful papers from all over the world, addressing themes such as the neurosciences, psychosocial interventions, medical factors, possible vulnerability and traumatic events. Doubtlessly, this book will be fruitful for future development and collaboration in "world psychiatry".

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