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Mood and Anxiety Disorders in Australia and New Zealand's Indigenous Populations: A Systematic Review and Meta-Analysis

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Abstract

The Indigenous populations of Australia and New Zealand are considered at higher risk of mood and anxiety disorders but many studies do not include direct comparisons with similar non-Indigenous controls. We conducted a systematic search of relevant electronic databases, as well as snowballing and targeted searches of the grey literature. Studies were included for meta-analysis if they compared rates of mood and anxiety disorders between Indigenous and non-Indigenous Australians or Maori. Seven Australian and 10 NZ studies were included. Overall, Indigenous people in both countries did not have significantly higher rates of disorder. However, in terms of specific disorders, there were differences in risk by gender, country (Australia or NZ), disorder type, and prevalence (current, 12-month or lifetime). For instance, Indigenous Australians and Maori both had significantly lower rates of simple phobias (current prevalence) and Maori participants had significantly lower rates of both lifetime simple phobia and generalised anxiety disorders. By contrast, Indigenous Australians had significantly higher rates of bipolar affective disorder and social phobia (current prevalence). Generalisations regarding the risk of psychiatric disorders in Indigenous people cannot therefore be made as this varies by several factors. These include disorder type, sociodemographic factors, Indigenous origin and study method.

Keywords: Australia; New Zealand; Indigenous; Maori; Aboriginal and Torres Strait Islander peoples; psychiatric disorders.

1. INTRODUCTION

Australia and New Zealand's (NZ) Indigenous populations, comprising Aboriginal and Torres Strait Islander (Indigenous Australians) and Maori peoples (respectively), have experienced significant disadvantage over the last 200 years. Indigenous Australians form approximately 3.0% of Australia's population (Australian Bureau of Statistics, 2013), while Maori people form 14.9% of NZ's population (Statistics New Zealand, 2013). These populations fare worse than their non-Indigenous counterparts in terms of both health (Australian Bureau of Statistics, 2014; Ministry of Health, 2015) and life expectancy (Australian Institute of Health and Welfare, 2011; Statistics New Zealand, 2015). Further, both Indigenous Australian and Maori populations have higher rates of suicide (Australian Bureau of Statistics, 2012; Ministry of Health, 2011) and psychological distress (Australian Institute of Health and Welfare, 2011; Cunningham and Paradies, 2012; Gubhaju et al., 2013; Ministry of Health, 2015), resulting in a disproportionately high use of mental health services (Abas et al., 2003; Abas et al., 2008; Australian Institute of Health and Welfare, 2011).

Given these findings, it is also likely that there are higher rates of formal psychiatric diagnoses in these groups. Whilst the physical health of these groups is well-understood, mental health is not as well-researched however. Previous reviews have attempted to identify rates of psychiatric disorders in Indigenous Australians (Black et al., 2015) and New Zealanders (Baxter, 2008), with varying findings. For instance, Black et al. found wide variability in the rates of psychiatric disorder for Indigenous Australians, with some studies reporting high rates (up to approximately 50% of participants) for anxiety and mood disorders. This variability was possibly due to varying samples (for example, community, medical, or corrections samples), as well as differences in design and methodology, measurement, and prevalence type (i.e., point, one year, or lifetime). No direct comparison was made between Indigenous Australians and similar non-Indigenous controls.

Baxter's (2008) review of Maori mental health literature determined that Maori also experienced high rates of disorder. Baxter presented studies making comparisons between Maori and non-Maori New Zealanders, finding that Maori were more likely to experience mood and anxiety disorders (some of which were significantly higher, depending on the disorder and study). However results were reported separately for the reviewed studies, and no statistical pooling was undertaken to determine generalisability across studies.

Neither of these reviews quantitatively analysed the data to determine if rates of psychiatric disorder were statistically greater than in similar non-Indigenous controls. This review aimed to address this gap by examining all available literature to determine the prevalence of common psychiatric (i.e., mood and anxiety) disorders in Maori and Indigenous Australian populations as compared to their non-Indigenous counterparts, with statistical pooling as appropriate.

2. METHOD

2.1. Inclusion and Exclusion Criteria

Common Mental Disorders (CMDs) were examined. These include major depression, Generalised Anxiety Disorder (GAD), panic disorder, Obsessive-Compulsive Disorder (OCD), Post-Traumatic Stress Disorder (PTSD), and simple phobias (Kendrick and Pilling, 2012). Due to the focus on mood disorders, Bipolar Affective Disorder (BPAD) and dysthymic disorder were also included for analysis. These disorders were defined in accordance with diagnostic criteria specified by: the Diagnostic and Statistical Manual of Mental Disorders, 5th Edition (DSM-5; American Psychiatric Association, 2013) or earlier versions of this manual; and the International Classification of Diseases, 10th Edition (ICD-10; World Health Organization, 1992).

Studies were included for analysis if they met the following criteria:

1. English-language, peer-reviewed empirical journal articles or government reports.
2. Prevalence data was cited for common psychiatric disorders (mood and anxiety). This could be lifetime, 12 month, or current prevalence.
3. The sample comprised Indigenous Australian or Maori populations of any age, with a non-Indigenous comparison group from Australia or NZ (respectively).

2.2. Search strategy

Preferred Reporting Items for Systematic reviews and Meta-Analyses (PRISMA; see Figure 1; Moher et al., 2009) guidelines were followed. Articles published from 1994 to 2016 were included; this time frame was selected so as to be broad enough to capture as many papers as possible, and be consistent with the use of DSM-IV (American Psychiatric Association, 1994). Databases searched included PubMed, MedLine, CINAHL, Scopus, Web of Science, ScienceDirect, ProQuest Research Library, PsycInfo, PsycArticles, and the

Informit Indigenous and Health Collections (comprising the following databases: AMI; APAIS-Health; ATSIHealth; AUSPORT; AusportMed; CINCH-Health; DRUG, Health & Society; HIVA; Health Collection; RURAL; AEI-ATSIS; AGIS-ATSIS; AHB-ATSIS; AIATSIS; APAIS-ATSIS; Indigenous Australia; CINCH-ATSIS; FAMILY-ATSIS; FNQ; Indigenous Collection; and MAIS-ATSIS). Keyword searches were conducted 'Across All Fields' in electronic databases, and included the following:

1. MEdical Subject Headings (MESH) term search: 'Australia' AND 'Indigenous Population' AND 'Mental Disorders'
2. MESH: 'New Zealand' AND 'Indigenous Population' AND 'Mental Disorders'
3. 'Indigenous' AND 'Mental Health' AND 'Prevalence' AND 'Australia'
4. 'Maori' AND 'Mental Health' AND 'Prevalence' AND 'New Zealand'
5. 'Aboriginal' AND 'Psychiatric' AND 'Prevalence' AND 'Australia'
6. 'Maori' AND 'Psychiatric' AND 'Prevalence' AND 'New Zealand'

All searches were conducted in 2016, with the most recent search conducted on 12th July 2016. Retrieved results were imported into an Endnote x7 database (Thomson Reuters, 2013). Duplicate entries were removed with Endnote's 'Find Duplicates' function, followed by a manual search through all papers to identify missed duplicates. Screening was then undertaken by reviewing titles and abstracts. Papers were then full text reviewed, and snowballing undertaken from the reference lists of included papers. Separate, targeted searches were also undertaken of the grey literature for relevant Australian and NZ government reports, as well as a search of the Cochrane Library. Papers retrieved through these targeted searches and snowballing were full text reviewed to determine eligibility for inclusion. Titles, abstracts, and papers were independently reviewed by two reviewers, as was data extraction and study quality (see below).

2.3. Study quality

The methodology of included studies was assessed using the model of Loney, Chambers, Bennett, Roberts, and Stratford (1998) . This model is designed to assess epidemiological studies examining prevalence or incidence of a given condition, and uses an eight point scale covering several methodological areas (e.g.: sampling method, frame, and size; response rate; and measures employed). The higher the score, the stronger the methodology.

In addition to assessing methodological quality according to Loney et al.'s (1998) method, possible socio-demographic differences between Indigenous and non-Indigenous participants were also considered. Where possible, subgroup analyses were undertaken to reduce potential confounding, and Indigenous/non-Indigenous sociodemographic characteristics reported in studies were inspected; details are reported in subsequent sections.

2.4 Statistical analysis

Review Manager, version 5.3 (The Cochrane Collaboration, 2014), was used for statistical pooling of data extracted from studies. As studies were predominantly cross-sectional, odds ratios for dichotomous variables were calculated. Heterogeneity was assessed using the I^2 statistic; this indicates how widely the effect sizes vary. An I^2 outcome of $\geq 50\%$ indicates likely heterogeneity, and scores in the range of 75 – 100% indicate considerable heterogeneity (Higgins and Green, 2011). As there was heterogeneity in some of the analyses, a random effects model was employed (as suggested by Higgins and Green, 2011). Heterogeneity was explored in subgroup analyses of individual disorders, gender, country/Indigenous origin, differences in comparison groups (NZ only), and study quality (with an outcome score of ≥ 5 on Loney et al.'s 1998 criteria used to indicate better quality studies); each individual study was also omitted in turn.

Where there were an adequate number of studies ($n \geq 10$), publication bias was assessed with two measures: funnel plot asymmetry and the fail-safe *N* statistic. These were calculated using Win-Pepi, *version 11.63* software (Abramson, 2011).

3. RESULTS

3.1. Included studies

Seventeen papers were suitable for analysis (see Figure 1 for PRISMA outcomes). Samples sizes varied greatly across studies, ranging from $N = 146$ to $N = 40{,}333$. Seven papers reported on Indigenous Australian participants (Table 1), and 10 papers on Maori (Table 2). Sample types also varied, with studies of: incarcerated people ($k = 7$, where k = number of studies; five of these were Australian studies); pregnant or postpartum women ($k = 3$); primary health care consumers ($k = 3$; all of these were NZ studies); the general population ($k = 1$); people with diabetes ($k = 1$); Vietnam veterans ($k = 1$); and a birth cohort ($k = 1$).

Included studies predominantly reported crude prevalence rates, and two reported both unadjusted and adjusted rates. Excepting two studies, differences in sociodemographic characteristics of Indigenous and non-Indigenous participants were considered. Typically, this was age and/or gender, although some studies reported greater detail (for example, marital status, education, employment status, household income, number of children, or offence history in prison samples). The studies that didn't report these details were not intended as epidemiological studies: one aimed to validate a measure (Evans et al., 2010), and the other retrospectively examined PTSD within veterans (MacDonald et al., 1997).

Specific to NZ, three studies compared Maori outcomes against NZ European participants (Evans et al., 2010; Waldie et al., 2015; Webster et al., 1994); all other NZ studies used 'non-Maori' comparison groups, with three of these reporting that the non-Maori comparison group was predominantly NZ European.

Study quality also varied widely, evidenced by methodology ratings from 1 – 8 (where 1 = minimum score and 8 = maximum score; Tables 1 - 2). Lower scores were sometimes because papers were not necessarily designed as epidemiological studies or to specifically measure the prevalence of CMDs. A range of diagnostic tools (n = 11) were used including structured and unstructured interviews, as well as questionnaires to assess specific disorders (such as depression; Tables 1 and 2). Fifteen studies reported on mood disorders, eight on anxiety disorders and five reported on any psychiatric disorder; varying prevalence rates (current, 12 month, or lifetime) were however reported by different studies. The different CMDs are reported by their varying prevalence rates in Table 3.

3.2. Meta-analyses

3.2.1. Current prevalence.

Eleven studies reported on the current prevalence of CMDs; the majority of these were for major depression (k = 10) (Table 3). Typically, there were no significant differences between Indigenous and non-Indigenous people in rates of current disorders, although Indigenous people were significantly more likely to have a diagnosis of social phobia than controls, and significantly *less* likely to have a diagnosis of simple phobia (Table 3). Most of the comparisons showed evidence of heterogeneity apart from the following anxiety disorders where scores were less than 50%: OCD, panic disorder, and agoraphobia. Major depression however

had significant heterogeneity (I^2 = 86%). When studies on perinatal depression or depression comorbid with diabetes were excluded, the findings remained the same but heterogeneity was no longer significant.

Sensitivity analyses were undertaken on a range of variables. Table 4 shows the results of subgroup analysis by gender. Indigenous females did not have higher rates of psychiatric disorder than non-Indigenous controls apart from one study that reported on broader classes of 'Any Mood Disorder' or 'Any Psychiatric Disorder' (Butler et al., 2007). Indigenous males were significantly more likely to have a diagnosis of Social Phobia than non-Indigenous males, but not any of the other disorders. None of these results showed heterogeneity.

We also undertook a sensitivity analysis of studies with a better methodology (Arroll et al., 2009; Butler et al., 2007; Indig et al., 2010; Simpson et al., 2003); findings remained the same.

Subgroup analysis was also undertaken on Indigenous origin (Maori or Indigenous Australian). Indigenous Australians were significantly more likely to have a diagnosis of BPAD (*OR* = 3.21, 95%CI = 1.34 – 7.69, p <0.01) or social phobia (*OR* = 8.48, 95%CI = 1.54 – 46.5, p <0.01), but not other disorders. Maori people did not significantly differ from controls in rates of mood and anxiety disorders although one study however found that Maori participants were significantly more likely to have 'Any Psychiatric Disorder' than non-Maoris.

Some of the NZ studies used different non-Indigenous populations as controls and so we investigated the effect of this on the results (Table 2). This was only possible in the case of major depression and when the comparison was restricted to just those of Caucasian origin (k = 3), Maori participants did have a significantly higher rate of major depression (*OR* = 1.88, 95%CI = 1.05 - 3.38, p <0.05). Another sensitivity analysis on major depression involved examining perinatal depression (k = 3). This determined that Indigenous women were significantly more likely to experience depression in the perinatal period (*OR* = 2.31, 95%CI = 1.90, 2.80, p <0.01).

Finally, subgroup analysis was undertaken on studies that used prison samples (k = 6). Findings remained the same.

3.2.2. Twelve month prevalence.

Only three studies reported data on 12 month prevalence of CMDs (Table 3); all disorders that included more than one study had considerable heterogeneity (BPAD, Any Mood Disorder, Any Anxiety Disorder, and Any Psychiatric Disorder). Whilst Indigenous Australians and Maori were significantly more likely to have 'Any Anxiety Disorder' (Table 3), this was not reflected in rates of individual anxiety disorders, and sensitivity analysis by Indigenous origin determined that this was actually not significant within Maori or Indigenous Australian populations separately. This is likely to be a function of low numbers of included studies within subgroup analysis.

One study determined that Maori people were more likely to have 'Any Psychiatric Disorder' than controls (*OR* = 1.75, 95%CI = 1.58, 1.94, *p* <0.01) (Baxter et al., 2006). Again, this however was not reflected in rates of individual disorders. Baxter et al.'s measurement of 'Any Psychiatric Disorder' included mood, anxiety, eating, and substance use disorders; rates reported for substance use disorders were higher for Maori participants than controls, which may account for this discrepancy.

No further subgroup comparisons were able to be undertaken, due to the low number of included studies for 12 month prevalence (see Table 3 for numbers of included studies per disorder).

3.2.3. Lifetime prevalence.

Indigenous people did not have significantly higher rates of any lifetime CMD than non-Indigenous people (Table 3). PTSD and 'Any Psychiatric Disorder' comparisons had considerable heterogeneity, whilst other anxiety and all mood disorders had nil or negligible variations (see Table 3 footnote). Due to this heterogeneity, sensitivity analysis was undertaken on studies with a higher methodology rating and Indigenous origin.

Sensitivity analysis of better quality studies (Arroll et al., 2009; Baxter et al., 2006; Butler et al., 2007; Marie et al., 2008; Simpson et al., 2003) revealed that Maori participants were significantly less likely to have simple phobia (*k* =1; *OR* = 0.75, 95%CI = 0.57, 1.00; *p* = 0.05) or GAD (k = 1; *OR* = 0.38, 95%CI = 0.14, 0.99; *p* = 0.05) than controls, according to Simpson et al. (2013). In contrast, 'Any Anxiety Disorder' was significantly elevated in Maori people (k = 1; *OR* = 1.59, 95%CI = 1.04, 2.44; *p* < 0.05), according to Marie et al. (2008). These discrepant results are likely to be a function of the different samples employed (Evans et al.

recruited incarcerated Maori males; Marie et al. examined young people within a birth cohort of young people), different diagnostic instruments used, and different anxiety disorders assessed.

Subgroup analysis by Indigenous origin determined that Indigenous Australians did not differ from controls in disorder prevalence. Heterogeneity was present in disorders that included more than one study for comparisons: 'Any Anxiety Disorder', I^2 = 70%, and 'Any Psychiatric Disorder', I^2 = 94%. Analysis by Maori origin resulted in findings reported in the preceding paragraph (i.e., significantly lower rates of GAD and simple phobia, yet significantly higher rates of 'Any Anxiety Disorder').

3.3. Publication Bias

Major depression (current prevalence) was the only CMD with sufficient studies to assess for publication bias. The fail-safe *N* statistic indicated that 20 studies with null findings would be required in order to reduce the overall ratio to a negligible size (i.e., a weighted odds ratio of 1.1). The funnel plot did not have significant asymmetry (p = 0.47). Given that double the current studies are required to make the current effect size negligible and the large p value, it is likely these results were relatively safe from publication bias.

4. DISCUSSION

To our knowledge, this is the first meta-analysis that compares rates of common psychiatric disorders in Indigenous populations across Australia and New Zealand with those of non-Indigenous people from the same two countries. We only included research that assessed both groups within the same study, using the same instruments and methodology. It was unexpected that mood and anxiety disorders in general were not elevated for Indigenous Australian and Maori populations, given the context of elevated rates of mental health service usage (Abas et al., 2003; Abas et al., 2008; Australian Institute of Health and Welfare, 2011), psychological distress (Australian Institute of Health and Welfare, 2011; Cunningham and Paradies, 2012; Gubhaju et al., 2013; Ministry of Health, 2015), alcohol and/or substance use (Baxter et al., 2006; Indig et al., 2010; Indig et al., 2011; Marie et al., 2008; Simpson et al., 2003), hospitalised rates of self-harm (Ministry of Health, 2011; Steering Committee for the Review of Government Service Provision, 2014), and suicide in these groups (Australian Bureau of Statistics, 2012; Ministry of Health, 2011). Whilst unexpected, these findings are consistent with research outcomes in other Indigenous populations, where similar or lower rates of CMDs have also been found. For example, a large study recruiting two Native American tribes (N = 3,084) found noticeably

lower rates of major depression as compared to the general American population (Beals et al., 2005). Another study found that after adjusting for covariates (such as gender and parental education), Canadian Aboriginal status was not significantly linked to depressed mood in a large sample of young people (N= 4,093; Lemstra et al., 2008). These results may therefore reflect genuine outcomes where Indigenous people are not at significantly higher risk of some psychiatric disorders than other groups. It may be that despite significant disadvantage, Indigenous culture actually forms a protective factor against psychiatric disorders: research has found that for Maori people, connection to culture is linked to greater resilience (Muriwai et al., 2015) and subjective well-being (Houkamau and Sibley, 2011). Further, research has found that living in areas of higher ethnic density is linked to better health outcomes, such as lower diagnosed psychiatric disorders and higher self-reported health in Maori people (despite deprivation in these regions; Bécares et al., 2013) and less psychological distress in Indigenous Australian people (Cunningham and Paradies, 2012). Interestingly, Bécares et al.'s study found that psychological distress was highest in areas of higher deprivation- these areas also happened to have higher Maori density, and lower rates of psychiatric disorders. Therefore, Indigenous people may experience higher rates of psychological distress and associated variables due to factors unrelated to ethnicity, but potentially lower rates of disorders as a result of cultural connection or density. This hypothesis may also account for the sensitivity findings that Indigenous Australians had significantly higher rates of some disorders, whilst Maori people had significantly lower rates of some disorders. Maori people comprise a larger proportion of NZs' population than Indigenous Australians in Australia's population (14.9% vs 3.0% , respectively; Australian Bureau of Statistics, 2013; Statistics New Zealand, 2013). However, it is also possible that differences in cultures, customs, and histories could also influence these outcomes.

Another explanation for the lack of difference in psychiatric morbidity may be that the assessment tools used in these studies, or diagnoses derived from ICD or DSM, may not accurately measure psychiatric symptoms in Indigenous populations. For instance, sadness and low mood may be more typical of anxiety rather than depressive disorders in Indigenous young people (Thomas et al., 2010). Depression may also present differently with anger or substance misuse predominating over low mood and somatic symptoms (Brown et al., 2012; Thomas et al., 2010). Anger and reckless behaviour, in the absence of low mood or somatic symptoms, may explain both the significantly higher rates of BPAD in this group, and the equivalent or significantly lower

rates of major depression in Indigenous participants. Further exploration of how CMDs present in Maori and Indigenous Australians is warranted to clarify potential cultural differences in CMD presentation and how this influences diagnostic prevalence.

Another factor influencing prevalence may be the types of samples recruited. Included studies typically consisted of specific samples, such as incarcerated people, primary health care attendees, and pregnant/postpartum women. Only two studies could be considered representative of the community: both of these were in NZ, with one epidemiological study and one birth cohort study. Sensitivity analysis indicated that findings generally remained consistent by setting, although Maori and Indigenous Australian women in the perinatal period were significantly more likely to experience depression than their non-Indigenous counterparts. However, in the case of social phobia such an analysis was not possible: there was only one study and this was of a prison sample. It is therefore possible that the significantly higher rates of social phobia may be an artefact of that particular setting. Given these specific samples, it is unclear whether the results of the current study are applicable to the broader Indigenous Australian and Maori communities.

It is also unclear why instruments would fail to detect disorders such as major depression, but not others such as social phobia. Complicating this picture further is evidence that psychotic disorders are also elevated in Indigenous Australians (Butler et al., 2007; Indig et al., 2011) and Maori (Evans et al., 2010), as compared to controls. Differences may therefore be disorder-specific.

In summary, several explanations may account for the current study's findings, including cultural differences, differences between internalising/externalising disorders, and genuine outcomes. What makes this picture more complex however is that findings were not uniform, so that no explanation accounts for all outcomes. Different patterns in findings were obtained across type of prevalence assessed, which may be due to different studies and methods being included for current, 12 month, and lifetime prevalence. This is further supported by high heterogeneity in some of the comparisons. When this was explored further in subgroup analyses, possible explanations included differences by Indigenous group (Indigenous Australian or Maori), gender, and individual disorders measured and included. Twelve month prevalence was also not as well-researched as current and lifetime prevalence, with a lesser number of included studies.

Finally, the type of comparison group employed may also influence research outcomes. Whilst conducted on current prevalence only, a sensitivity analysis found that Maori people had higher rates of major depression when compared to only NZ European participants, but not when the comparison group comprised other ethnicities; this occurred even when the comparison group was predominantly NZ European. This indicates that another ethnic group may be influencing these outcomes. For example, research has identified that Pacific Islanders have higher rates of psychiatric disorder than the general NZ population (Foliaki et al., 2006); it may be that including this population in non-Maori comparison groups obscures potential significant outcomes. Future research therefore needs to consider the ethnic make-up of comparison groups when researching Maori and Indigenous mental health.

4.1 Limitations

This study had several limitations. Firstly, although Indigenous and non-Indigenous participants came from the same setting in each study, it is possible that sociodemographic differences between these populations (such as age, gender, employment, educational status, or even differences in the comparison group recruited) could confound these results. Further, included studies typically used specific populations, which may not be representative of the broader Indigenous communities. This risk was managed by undertaking subgroup analyses on a range of variables; and, given the marginalisation of many Indigenous Australian and Maori people, any bias would be in the direction of increasing their risk of psychiatric disorders. This does not explain several findings that the risk was often the same between groups, or even significantly lower for some disorders.

Some results showed heterogeneity, which was explored further in several sensitivity analyses. Differences in sociodemographic variables within the Indigenous and comparison groups influenced outcomes. Omitting each study in turn made little difference to heterogeneity levels. Accordingly, a random-effects model was used throughout to incorporate heterogeneity into the analyses. Despite attempts to minimize heterogeneity effects, analyses where this was present should still be treated cautiously. Finally, publication bias was only able to be assessed in current major depression; whilst this was low risk for bias, the same may not be true of other comparisons.

4.2. Conclusions

Generalisations regarding risk of psychiatric disorders cannot be made for Indigenous people as whole, as risk varies by disorder type, sociodemographic factors, Indigenous origin, study method, and even comparison group recruited. Research that further attempts to elucidate the impact of such variables on CMD rates is indicated. Another factor that may influence outcomes is culture itself. For instance, resilience may be enhanced when Indigenous people live in close proximity with numbers of other Indigenous people. Epidemiological research that examines variations in rates of CMDs by region, ethnic density, and cultural connection is warranted.

This review also identified a lack of representative community studies, particularly for Australia. Epidemiological research within Maori and Indigenous Australian community samples is required in order to gain accurate understanding of disorder rates.

Finally, current measurement of psychiatric disorders may not accurately capture the expression of these disorders in Indigenous populations, explaining why some analyses found non-significant, or significantly lower rates of some disorders in Indigenous people. Future research is therefore required that examines the cross-cultural validity of psychiatric constructs and diagnostic instruments, in order to avoid misdiagnosis and false negatives.

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Figures

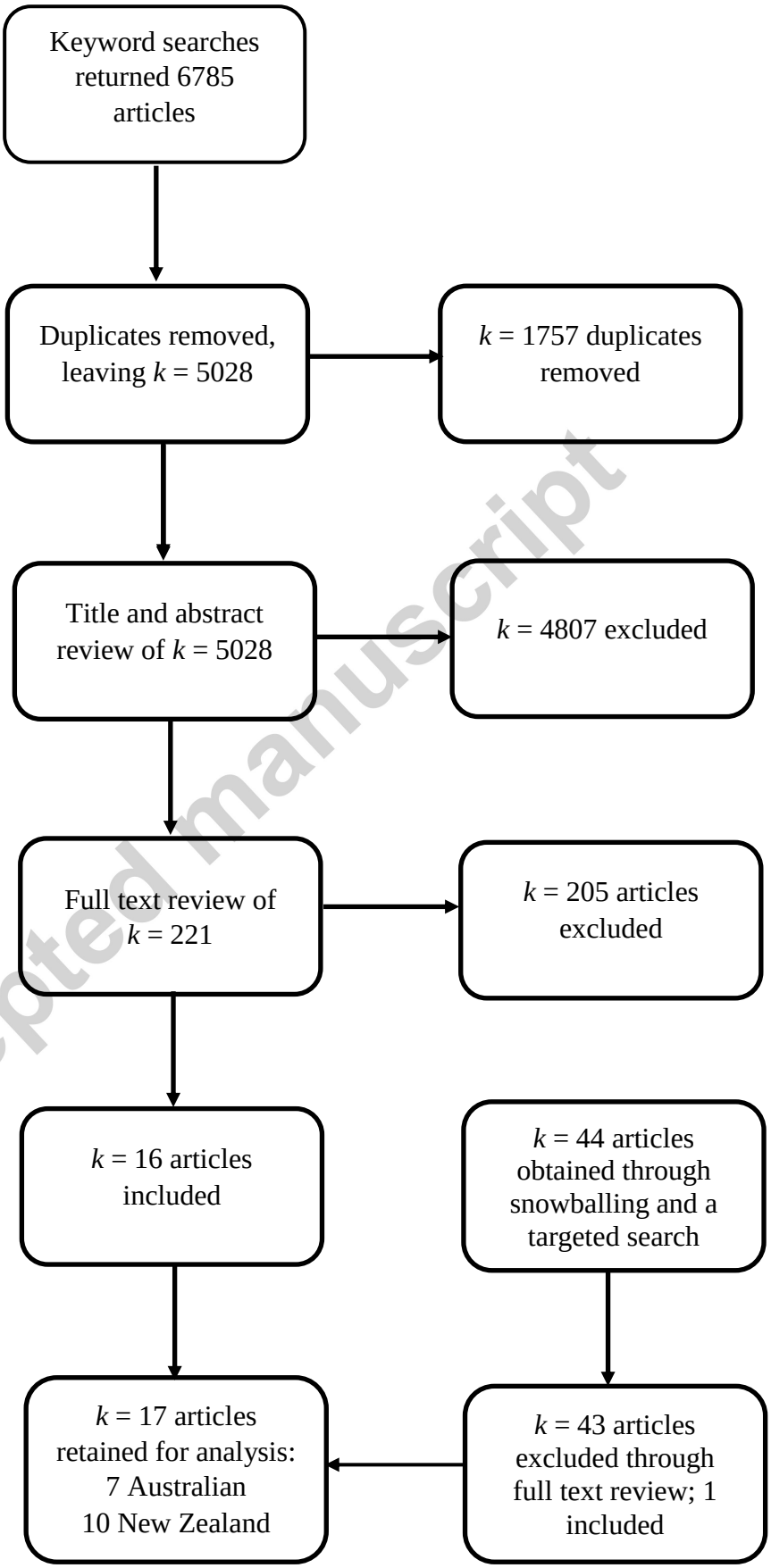

Fig 1. Literature Search Strategy.

Table 1 *Studies reporting prevalence of common psychiatric disorders for Indigenous People of Australia*

Study	*N*	Assessment[a]	Sample Characteristics[b]	Sampling strategy [c]	Methodology rating[d] and Limitations: A. Study-identified B. Review-identified
Butler et al. (2007)	1,470	A range of measures (not all diagnostic); notably, the Composite International Diagnostic Interview (CIDI; World Health Organization, 1993).	Mixed sample of Indigenous and non-Indigenous Australian prisoners (sentenced and in reception). Indigenous participants (*n* = 277), with 81.6% male (*n* = 226), and 18.4% female (*n* = 51). Data on sentenced prisoners came from the 2001 Inmate Health Survey that was also used by Indig et al (2010; see below) but studies reported on different outcomes	Study occurred across all 29 Correctional centres in New South Wales (NSW). Stratified random sample of sentenced prisoners, and convenience sample of reception prisoners. Recruitment rate of 85%.	5/8 **A.** Authors discuss the population sampling intended of reception prisoners not feasible (due to a variety of factors), so relied on convenience sampling in this area (not representative). Authors queried the validity of the OCD diagnosis in Indigenous Australians. **B**. Psychosis screener used, therefore not diagnostic. Measures not validated for Indigenous people. Very specific population (in detention) means results not generalisable to broader community. There was no information on demographic differences between ethnic groups. Results were reported

[a] This column considers how the disorders were assessed or measured, and the tools used for this.
[b] Sample characteristics described, including ethnicity, gender, age (range and mean), and location.
[c] The sampling and recruitment strategy used by the study are briefly described, as well as the location of the study and sample type.
[d] This was done using Loney et al.'s criteria, which assign a score out of 8. Criteria used include: design and sampling method, size, and frame; response rate; participant description; measurement of the health outcome, and potential for bias; and the provision of confidence intervals and subgroup detail when prevalence or incidence is reported.

					separately for males and females.
Davis et al. (2015)	900	Nine-item Personal Health Questionnaire (PHQ-9)	People with diabetes living in the community n = 52 Indigenous Australian participants completed the PHQ-9 (overall study had n = 107). Anglo-Celt n = 793; although n = 772 completed the PHQ-9.	Anyone in the study catchment area with a clinician-verified diagnosis of diabetes was eligible. Recruitment was through public hospital clinics, laboratories, clinician referrals, advertisements in pharmacies and local media, as well as mail-outs through the Australian National Diabetes Supply Scheme and National Diabetes Register. In addition, an Aboriginal health worker identified and recruited indigenous patients.	4/8 **A**. Low numbers of Indigenous participants **B**. The sample was restricted to people with diabetes and depressed mood was assessed by a questionnaire.
Doolan et al. (2012)	3,705	Diagnostic Interview based on ICD-10 criteria administered by experienced and trained clinicians, and discussed with Consultant Psychiatrist during a team meeting.	Indigenous Australians formed 46% of sample (*n* = 1696); the rest were non-indigenous participants. Indigenous females were 25.4% (*n* = 430) of Indigenous sample, males 74.6% (*n* = 1266). Age range 10 - 21 years.	Recruited from a Youth Detention Centre from those referred to the Mental Health Alcohol Tobacco and Drugs service. Only 1091 Indigenous Australian participants attended the diagnostic interview: Males (78.1%; *n* = 852); Females (21.9%; *n* = 239). Response rate for males = 69.2%, females = 60.1%	4/8 **A**. Only people referred to the service were assessed; this could lead to bias as potentially there are people with psychiatric disorders who may not be referred to the service. Attrition also present in sample through people being released prior to interview. **B**. Very specific population (in detention) means results not generalisable to broader community. Specific diagnoses not reported. Lower than ideal response rate. There was no information on demographic differences between ethnic groups. Results were reported

					separately for males and females.
Fleming et al. (2012)	146	Participants self-reported treatment for diagnoses on the survey instrument (administered via interview).	Participants were recruited from two WA maximum security prisons. 43 participants were Indigenous Australian, with 21 females (49%) and 22 males (51%). Further Indigenous-specific demographics not reported.	Participants in the study approximated 18% of the population in the two prisons. Information sources such as peer support officers and flyers promoted the project; participants volunteered and were then screened for inclusion.	1/8 **A**. Small number of participants acknowledged as not being generalisable. Identified that Indigenous Australians were under-represented in the study. **B**. Small number of Indigenous participants means results are limited. Self-report data can be biased; study was not diagnostic. Sampling strategy could also bias results. There was no information on demographic differences between ethnic groups. Results were reported separately for males and females.
Indig et al. (2010)	996 914 789	Computer-assisted telephone interview; physical health examination; and a range of questionnaires, notably Beck Depression Inventory- II (BDI-II; Beck et al., 1996).	2009 Inmate Health Survey - Mixed sample. Of incarcerated Indigenous Australians (*n* = 312), 83% were male (*n* = 259) and 17% female (*n* = 53). Six percent identified as TSI. Average age was 35.5 years, with females (*M* = 31.1 years) and males	Stratified (by age, gender, and Indigenous status) random sampling occurred across all correctional centres in NSW (*n* = 30): 26 male centres and 4 female centres. Response rate was 85.4% (1128 inmates were approached).	6/8 **A.** The authors note that only a small sample of Indigenous women were recruited, so these results are not representative. **B**. There was no information on demographic differences between ethnic groups other

			(*M* = 34.1 years).		than the Indigenous group in the 2009 Inmate Health Survey was significantly younger. Results were reported separately for males and females.
		As above	2001 Inmate Health Survey 1996 Inmate Health Survey		
Indig et al. (2011)	361	A health survey with physical examination, and psychological assessments, including the Kiddie Schedule for Affective Disorders for Children-Present and Lifetime version.	2009 Young People in Custody Health Survey. Overall, 174 Indigenous young people participated; however only 140 completed the psychological assessments. Age range was 13 – 21 years (M = 16.7 years).	Survey conducted at 8 juvenile justice centres and 1 juvenile correctional centre in NSW. Over-sampling used at female centre to increase number of female participants. All incarcerated young people on the first day at the location were approached. Response rate was 95%. The total sample represented 80% of all incarcerated young people.	4/8 **A.** Parental component of diagnostic schedule not administered. Interrater reliability not assessed. Study relied on self-report rather than using a range of strategies (e.g., medical records, parent report). Possible fatigue effects are acknowledged due to the battery of measures. Small number of females recruited. Not all measures have been validated for Indigenous populations. **B.** The Indigenous group was a small sample. There was no information on demographic differences between ethnic groups
Milgrom et al. (2008)	40,333	Edinburgh Postnatal Depression Scale, along with other screening measures. This assessed both Antenatal and Postnatal Depression; only the Antenatal rates are reported due to Indigenous statistics not being reported for Postnatal Depression.	Prospective Australian sample of pregnant women, assessed antenatally and 6 weeks postpartum for depression. In the Antenatal Depression sample: Non-Indigenous n = 12,139 Indigenous Australians n = 222	Midwives in hospitals across Australia recruited participants. Details not reported.	3/8 **A**. Not all pregnant women participated in the study; and there was an attrition rate of just over 50% for women followed up (for those that received follow-up). **B**. Use of 10 item screening tool for diagnosis. Lacks recruitment details. Prevalence rates across Indigenous/ non-Indigenous groups only reported for antenatal

	depression, not postnatal depression.

Table 2 *Studies reporting prevalence of common psychiatric disorders in Indigenous People of New Zealand*

Study	*N*	Assessment[a]	Sample Characteristics[b]	Sampling strategy[c]	Methodology rating[d] and Limitations: A. Study-identified B. Review-identified
Arroll et al. (2009)	7432	Computerised CIDI PHQ also completed by one third of sample	People accessing their GP (N = 7432). Non-Maori n = 6711. Maori n = 721 Maori mean age was 39 years, with 62% of sample female (n = 449).	General Practitioners (GPs) in Auckland were recruited to ask if their patients were interested in the study. GPs needed to work above 0.4FTE and have an interview room available for the study. Patients were then approached in the waiting room, informed consent sought, and CIDI interview conducted.	5/8 **A**. Medical practices were not randomly chosen- they required available interviewing rooms for the study. Study may be underpowered. CIDI has been criticised. Different prevalence rates obtained to prior research in primary care. **B**. As participants recruited from primary care, which the authors acknowledge have higher prevalence rates than in the community, findings not representative of Maori generally.
Arroll et al. (2002)	253	BDI	People attending their GP (N = 253); ranged in age from 16 - 95 years. Maori n = 64 Non-Maori n = 189	Conducted at a medical centre in Auckland. GPs asked all consecutive patients aged ≥16years if they would complete a survey about their health and mood post-consultation. Those who consented were referred to the study's interviewer.	3/8 **A**. Authors state that use of BDI is a weakness, as the gold standard for measuring depression is psychiatrist interview. **B**. Small sample that is specific to one medical practice. Interviewer not blinded to GP's opinion as to whether patient depressed or not. Questionnaire self-administered, which relies on literacy skills.
Baxter et al. (2006)	12,992 (Part 1) 7435 (Part 2)	CIDI, broken into two parts to reduce participant burden. Part 1 involved screening of all mental disorders, diagnostic assessment of mood and anxiety disorders (excepting PTSD, OCD). Those with no disorders at the end of Part 1 were not invited back to complete Part 2, except for a probability subsample.	The New Zealand Mental Health Survey 2003-2004. General population ≥16 years Maori n = 2595; of which females 53.4%, males 46.6% Pacific Islanders n = 2236 'Other' (predominantly NZ European origin) n	Face to face survey of households; nationally representative.	8/8 **A**. The authors suggest that using standardised western tools cross-culturally may not appropriately capture all expressions of the disorder. **B**. Part 1 and Part 2 findings reported in an unclear manner so it is unknown which sample formed the findings at any given time.

[a] This column considers how the disorders were assessed or measured, and the tools used for this.
[b] Sample characteristics described, including ethnicity, gender, age (range and mean), and location.
[c] The sampling and recruitment strategy used by the study are briefly described, as well as the location of the study and sample type.
[d] This was done using Loney et al.'s criteria, which assign a score out of 8. Criteria used include: design and sampling method, size, and frame; response rate; participant description; measurement of the health outcome, and potential for bias; and the provision of confidence intervals and subgroup detail when prevalence or incidence is reported.

Study	*N*	Assessment[a]	Sample Characteristics[b]	Sampling strategy [c]	Methodology rating[d] and Limitations: A. Study-identified B. Review-identified
			= 8161		
		Part 2 involved assessing PTSD, OCD, and SUDs.			
Evans et al. (2010)	1292 screens completed 530 MINI completed	Initial screening measures included the Brief Jail Mental Health Screen and English Mental Health Screen. All positive screens, and a proportion of negative screens, completed the Mini International Neuropsychiatric Interview.	New male prisoners admitted to Christchurch's Men's Prison, Auckland Central Remand, and Mount Eden Prison. Of the screens completed, n = 546 were NZ European and n = 440 were Maori. Of the 530 MINI completed, n = 229 NZ European and n = 187 Maori.	All new admissions assessed with the brief screens. Those with positive results, and 20% of negative screens were invited to participate in the MINI (which had a 92% response rate).	4/8 **A**. Only male prisoners recruited. Structured diagnostic tool used instead of an experienced clinician may undermine validity of the measure. Self-harm in/out of prison not differentiated. **B**. Unclear if lay interviewers were used instead of clinicians, and if MINI interviewers were blinded to the screen or not. Small number of Maori who completed the diagnostic measure.
MacDonald et al. (1997)	756	Self-report questionnaires employed: the Mississippi Scale (for PTSD); the BDI (Depression); and subscale of the State-Trait Anxiety Inventory (Anxiety). Demographic, military experience, and trauma exposure questions also included.	Known Vietnam veterans invited to participate. 22% were Maori (n = 166) 78% non-Maori (n = 590)	Questionnaires were mailed to known Vietnam veterans on a random basis from a Ministry of Defence list; however this was approximately 50% of this population, due to not having a national registry of veterans.	0/8 **A**. Low response rate and therefore not able to determine sample bias present; non-random sample; relies on retrospective reporting. Assessment of PTSD not the standard (i.e., self-report questionnaire used). Number of Maori soldiers thought to be under-represented in the sample; although Maori *n* is oversampled when compared to community Census data. **B**. Findings not representative of veterans due to sampling frame limitations and low response rate. Low number of Maori participants.
Marie et al. (2008)	984	CIDI supplemented by questions using DSM-IV criteria for a range of disorders	Birth cohort up to the age of 25 years; part of the Christchurch Health and Development Study. Non-Maori n = 875	Initial cohort comprised all births in Christchurch in a certain time period (N =1310; 1265 agreed to be part of the study). 80% of the original sample retained for this study.	6/8 **A**. Birth cohort specific to one region and time frame, therefore generalisability unclear. Small number of Maori participants. **B**. Given the young age of

Study	*N*	Assessment[a]	Sample Characteristics[b]	Sampling strategy[c]	Methodology rating[d] and Limitations: A. Study-identified B. Review-identified
			Sole Maori identity n = 50 Maori/Other identity n = 59		the sample, this won't indicate prevalence of disorders across the lifespan. Information lacking on administration of measures and by whom.
Simpson et al. (2003)	1287	CIDI and the Personality Diagnostic Questionnaire for DSM-IV	National Study on Psychiatric Morbidity in NZ Prisons Maori over-represented. Maori n = 622 Pacific Islander n = 107 NZ European n = 398 Other n = 158	All prisons in NZ sampled- all female prisoners and all male remand prisoners approached. For sentenced male prisoners, a random sample was approached.	6/8 **A**. Risk of committing cross-cultural error in diagnosis as diagnostic tool not validated for Maori. Maori response rates not recorded (refuser characteristics not documented). **B**. Variety of interviewers employed (including experienced clinicians, psychology interns, and lay interviewers). Inter-rater reliability not assessed.
The MaGPIe Research Group (2005)	786	Initial screen with GHQ by GP; subset (n = 786) invited back to complete CIDI	Primary care patients aged ≥ 18 years. GHQ score determined who was invited to participate in CIDI. Maori n = 81 Non-Maori n = 705	70 randomly selected GPs from a region in NZ were invited to be part of the study. 50 consecutive patient GHQ screens completed on people about to consult their GP. GHQ score used to select CIDI sample.	4/8 **A**. Low number of Maori participants; diagnostic interview not validated for use with Maori people; not representative of a community sample. **B**. Study methodology lacks detail.
Waldie et al. (2015)	5664	EPDS, to assess for antenatal depression	Pregnant women participating in the Growing Up in NZ longitudinal cohort study. Recruited from a region in NZ with due dates from 2009-2010. Ethnicity data available for N = 5656. Maori n = 747 NZ European n = 3168 Pacific Islander n = 726 Asian n = 802 Other n = 213	Participants initially invited to participate by their midwife, physician, or obstetrician. Media and community organisations used to inform eligible women of the study. Recruiters also attended hospitals and antenatal clinics.	3/8 **A**. Measure able to be employed were limited by feasibility within the large sample. Diagnostic validity of EPDS within sample is lacking. **B**. Use of a 10 item screening measure for diagnosis of depression.
Webster, Thompson, Mitchell,	206	EPDS, to assess postnatal depression	Women who gave birth to a live baby in hospital.	All women who had a baby in Auckland over a 1 week period; they	2/8 **A**. Time frame used may

Study	*N*	Assessment[a]	Sample Characteristics[b]	Sampling strategy[c]	Methodology rating[d] and Limitations: A. Study-identified B. Review-identified
and Werry (1994)		Small, random subsample also completed a semi-structured interview based on DSM-III depression criteria.	Maori n = 42 NZ European n = 163	were assessed at four weeks postpartum.	have resulted in women with resolving adjustment disorders being diagnosed with depression, and may have missed other cases that later went on to develop depression. **B**. Use of a 10 item screening measure to diagnose depression. Small number of Maori participants.

Note. BDI = Beck Depression Inventory; CIDI = Composite International Diagnostic Interview; PHQ = Patient Health Questionnaire; GHQ = General Health Questionnaire; EPDS = Edinburgh Postnatal Depression Scale.

Table 3 *Current, 12 month, and lifetime prevalence of common psychiatric disorders in Indigenous people of Australia and New Zealand*

	Current			12 month			Lifetime		
	k^a	*N*	Odds Ratio [M-H, Random, 95% CI]	*k*	*N*	Odds Ratio [M-H, Random, 95% CI]	*k*	N	Odds Ratio [M-H, Random, 95% CI]
Mood Disorder									
Major depression	10	25,826	1.21 [0.89, 1.65]	1	1,478	0.71 [0.48, 1.06]	6	20,789	1.09 [0.90, 1.32]
Dysthymic disorder	2	2,656	0.83 [0.34, 2.05]	1	1,478	1.14 [0.68, 1.90]	1	1,178	0.95 [0.57, 1.57]
BPAD/mania/hypomania	3	3,072	1.45 [0.68, 3.10]	2	4,220	1.92 [0.98, 3.74]	2	1471	0.81 [0.42, 1.56]
Any Mood Disorder	1	1,478	1.06 [0.74, 1.51]	3	13,020	1.86 [0.92, 3.76]	1	293	0.86 [0.50, 1.48]
Anxiety Disorder									
GAD	2	2,656	0.51 [0.09, 2.97]	1	1,478	1.17 [0.82, 1.67]	2	1,471	0.56 [0.27, 1.19]
Panic disorder	2	2,656	1.13 [0.71, 1.80]	1	1,478	1.27 [0.83, 1.95]	1	1,178	0.67 [0.41, 1.10]
OCD	2	2,656	1.18 [0.70, 1.98]	1	1,478	1.09 [0.47, 2.52]	2	1,471	0.86 [0.56, 1.33]
Agoraphobia	2	2,656	1.13 [0.47, 2.74]	1	1,478	1.65 [0.84, 3.25]	1	1,178	1.65 [0.89, 3.07]
Social phobia	1	1,478	8.48 [1.54, 46.51]**	1	1,478	1.29 [0.42, 3.99]	1	293	1.56 [0.48, 5.03]
PTSD	2	2,656	0.94 [0.60, 1.47]	1	1,478	1.11 [0.83, 1.50]	3	2,227	1.46 [0.59, 3.65]
Simple phobia	1	1,178	0.63 [0.40, 0.99]*	0	-	-	2	1,471	0.77 [0.58, 1.01]
ADNOS	0	-	-	0	-	-	1	293	1.38 [0.36, 5.24]
Any anxiety disorder	1	1,478	1.11[0.83, 1.47]	3	13,020	1.76 [1.14, 2.72]**	3	1,423	1.16 [0.66, 2.03]
Any psychiatric disorder	2	1,894	1.22 [0.83, 1.78]	2	12,234	1.40 [0.87, 2.24]	2	2,584	1.10 [0.22, 5.60]

[a] *k* = number of studies

Note. * $p \leq .05$; ** $p \leq .01$; *** $p \leq .001$

$I^2 \leq$ 50% for: Current OCD, panic disorder, and agoraphobia; 12 month BPAD/mania/hypomania; Lifetime GAD, OCD, agoraphobia, social phobia, simple phobia, panic disorder, ADNOS, major depression, BPAD/mania/hypomania, dysthymic disorder, Any Mood Disorder.

BPAD = Bipolar Affective Disorder; *GAD* = Generalised Anxiety Disorder; *OCD* = Obsessive Compulsive Disorder; *PTSD* = Post-Traumatic Stress Disorder; *ADNOS* = Anxiety Disorder Not Otherwise Specified.

Table 4 *Gender differences in current prevalence of common psychiatric disorders*

Males	Females

	k[10]	*N*	Odds Ratio [M-H, Random, 95% CI]	*k*	*N*	Odds Ratio [M-H, Random, 95% CI]
Mood Disorders						
Major depression	4	6,794	0.96 [0.74, 1.25]	6	21,712	1.38 [0.96, 1.97]
Dysthymic disorder	1	1,208	0.97 [0.50, 1.90]	1	270	2.23 [0.84, 5.95]
BPAD/mania/ hypomania	2	1,624	1.56 [0.80, 3.04]	1	270	3.16 [0.93, 10.76]
Any Mood Disorder	1	1,208	0.73 [0.46, 1.15]	1	270	2.22 [1.18, 4.18]**
Anxiety Disorders						
GAD	1	1,208	0.84 [0.52, 1.36]	1	270	1.33 [0.64, 2.76]
Panic disorder	1	1,208	1.18 [0.58, 2.41]	1	270	1.30 [0.45, 3.78]
OCD	1	1,208	0.62 [0.18, 2.08]	1	270	3.67 [0.51, 26.60]
Agoraphobia	1	1,208	1.77 [0.77, 4.06]	1	270	0.50 [0.06, 4.17]
Social phobia	1	1,208	13.20 [1.37, 127.47]*	1	270	3.62 [0.22, 58.77]
PTSD	1	1,208	1.00 [0.66, 1.51]	1	270	1.44 [0.80, 2.61]
Any anxiety disorder	1	1,208	0.98 [0.70, 1.37]	1	270	1.45 [0.81, 2.58]
Any psychiatric disorder	2	1,624	1.11 [0.61, 2.01]	1	270	1.90 [1.05, 3.44]*

Note. * $p \leq .05$; ** $p \leq .01$; *** $p \leq .001$
$I^2 \leq$ 50% for male rates of BPAD/mania/hypomania, and Any Psychiatric Disorder.
BPAD = Bipolar Affective Disorder; *GAD* = Generalised Anxiety Disorder; *OCD* = Obsessive Compulsive Disorder; *PTSD* = Post-Traumatic Stress Disorder.

Highlights

[10] *k* = number of studies

- Overall, mood and anxiety disorders were not elevated in the Indigenous populations examined
- Disorder risk varied by several factors, such as gender, disorder type, and measurement
- Indigenous people had significantly lower rates of Simple Phobias
- Indigenous Australians had higher rates of Social Phobia and Bipolar Affective Disorder