Background

Description of the condition

A personality disorder is "an enduring pattern of inner experience and behaviour that deviates markedly from the expectations of the person's culture, is pervasive or inflexible, has an onset in adolescence or early adulthood, is stable over time and leads to distress or impairment" (DSM-IV-TR (APA 2000)). Narcissistic personality disorder (NPD) is part of the category of "cluster B" (or "dramatic, emotional, or erratic") personality disorders, that are linked by theory and phenomenology to Axis I and impulse control disorders (APA 2000).

NPD is characterised by DSM-IV-TR (301.81; APA 2000) as "a pervasive pattern of grandiosity (in fantasy or behaviour), need for admiration, and lack of empathy, beginning by early adulthood and present in a variety of contexts." It is diagnosed by the presence of five or more of the following:

1. has a grandiose sense of self-importance (for example, exaggerates achievements and talents, expects to be recognized as superior without commensurate achievements);
2. is preoccupied with fantasies of unlimited success, power, brilliance, beauty or ideal love;
3. believes that he or she is "special" and unique and can only be understood by, or should associate with, other special or high-status people (or institutions);
4. requires excessive admiration;
5. has a sense of entitlement, i.e., unreasonable expectations of especially favourable treatment or automatic compliance with his or her expectations;
6. is interpersonally exploitatence, i.e., takes advantage of others to achieve his or her own ends;
7. lacks empathy: is unwilling to recognize or identify with the feelings and needs of others;
8. is often envious of others or believes that others are envious of him or her;
9. shows arrogant, haughty behaviours or attitudes.

The International Classification of Mental and Behavioural Disorders (ICD-10, WHO 1992a) lists NPD under F60.8 "other specific personality disorders" without further specification beyond the general set of personality disorder criteria (F60). "These are severe disturbances in the personality and behavioural tendencies of the indivi
d not directly resulting from disease, damage, or other insult to the brain, or from another psychiatric disorder, usually involving several areas of the personality; nearly always associated with considerable personal distress and social disruption; and usually manifest since childhood or adolescence and continuing throughout adulthood (WHO 1992b).

Epidemiologic show prevalence rates of DSM-IV NPD to be below 1% in the general population (Samuels 2002; Cost 2006; Lenzenweger 2007). The lifetime prevalence of NPD was found to be 6.2% in a recent epidemiologic survey (Simons 2008). Research findings consistently show that NPD is more common in men if women (Fossati 2000; Torgersen 2001; Simons 2008). In general, NPD is mostly prevalent among younger adults, but NPD in early adulthood does not always turn long-term condition (Ekstrom 2001; Crawford 2005). In one study, about 50% of young adults with NPD no longer qualified for the diagnosis after a three year follow (Ronningstam 1995). NPD rates are higher amongst younger people and among separated, widowed, divorced and never married individuals (Simons 2008). This is consistent with finding that NPD is associated with impaired emotional functioning (Simons 2008) and also with costs experienced by relatives of the persons concerned i.e., distress and functional impairment (Miller 2007). Levels of income and education, however, are not linked to NPD (Simons 2008).

To date, there is a consensus among researchers that, as for any mental disorder, biological, psychological and social factors contribute to the development of personality disorders (Paris 2006). A genetic influence is underlined by the findings of a twin study (Torgersen 2000), indicating that heritability is higher in NPD compared with other personality disorders. Although neurochemical and neuroimaging studies have focused on borderline personality disorder and antisocial personality disorder, it is probable that their findings of neurobiological dysfunction and abnormalities in brain structures are not specific to these conditions but are also applicable to other cluster B personality disorders such as NPD (Coccaro 2005). Peculiarities include the function of monoaminergic systems (for example, serotonin, norepinephrine, vasopressin for impulsivity and aggression, possibly acetylcholine for mood reactivity) and brain structures related to emotional behaviour and information processing (orbitofrontal cortex, amygdala). From a developmental perspective, both retrospective and prospective studies show that there are associations of adult NPD with childhood sexual and emotional abuse and neglect (Johnson 2005). Little is known about social factors contributing to NPD development. Some hypothesis have contrast to traditional societies with high social cohesion and fixed social roles, modernity forces people to develop their own identities and social roles, which may exacerbate the risk of personality disorders (Paris 1996).

Despite low overall prevalence rates in the general population, NPD is of high importance in clinical settings and, due to frequent comorbidity with Axis I disorders, is prevalent in clinical samples. Estimates range from 2.3% in psychiatric outpatients (Zimmerman 2005) to 36% in a mixed sample of both psychiatric inpatients and outpatients (Fossati 2000). Common comorbid conditions include substance use disorders, mood disorders, anxiety disorders and other personality disorders (Skodol 2006; Simons 2008). These may have been partially caused by NPD (according to the "vulnerability model", for example, Klein 1993). NPD symptoms are usually ego-syntonic, i.e., those affected do often not recognise that most others may consider their behaviour exploitative, arrogant or showing contempt. This often leads to interpersonal difficulties and disturbed relationships, which, in turn, causes suffering to those affected and to their relatives. Due to very fragile self-esteem, they are prone to take on narcissistic injury in situations people without NPD would not, resulting in serious suicidal crises (Ronningstam 1998). In contrast to people who have other cluster B personality disorders and attempt suicide, people with NPD who attempt suicide have been shown to have lower levels of impulsivity but higher levels of completed suicide (Blasco-Fontecillas 2009).

More care and prolonged care is required if Axis I and Axis II disorders co-occur, with greater and more widespread levels of impairment (Jackson 2002; Skodol 2000 more chronicity (Hart 2001; Grilo 2005) and an overall poorer response to treatment (Skodol 2005; Newton-Howe 2006). The suicidal risk is, as in other conditions, raised in times of comorbid depression (Perry 1996).
Description of the intervention

Since personality disorders are defined as syndromes, different patients with NPD may experience different facets of pathology, indicating different drug treatment options. Comorbid conditions may also require psychotropic treatment or indicate a certain class of drugs due to overlapping symptoms. Thus, people with NPD may receive a broad range of pharmacological treatments; however, agents aiming to stabilise affective dysregulative symptoms (i.e., labile, depressive, angry, anxious moods) are especially relevant for people with NPD.

How the intervention might work

In personality disorders, psychotropic medication is given assuming that modifying effects on neurotransmitter functions may mediate expressions of state symptom (such as angry or aggressive mood (Soloff 1998)), but also appease trait vulnerabilities that are related to personality dimensions (Soloff 2000). The appropriate use medication may also facilitate psychotherapy by stabilising a patient through a process of change. A certain drug is chosen for personality disorder treatment under the assumption that symptoms may be mediated, in part, by the same neurotransmitter systems as affect the phenomenologically-similar symptoms of Axis I disorders.

Why it is important to do this review

There has been increasing interest in developing and evaluating pharmacological treatments for personality disorders in recent years, suggesting that a systematic review is now timely to assess the quality and conclusions of available evidence for NPD. In addition, this is a condition that causes life-threatening situations for those concerned and caregivers need to know which treatment options are helpful.

Objectives

This review aims to evaluate the potential beneficial and adverse effects of pharmacological interventions for people with NPD.

Methods

Criteria for considering studies for this review

Types of studies

Controlled trials in which participants have been randomly allocated. We will include all relevant randomised controlled trials with or without blinding of the assessors.

Types of participants

People who are 18 years or over with a diagnosis of NPD defined by any operational criteria such as DSM-IV. We will include studies of people diagnosed with comorbid personality disorders or other mental health problems other than the major functional mental illnesses (i.e., schizophrenia, schizoaffective disorder or bipolar disorder). The decision to exclude people with comorbid major functional illness is based on the rationale that the presence of such disorders (and the possible confounding effect of any associated management or treatment) might obscure whatever other psychopathology (including personality disorder) might be present.

Types of interventions

Any drug(s) with psychotropic properties, including those falling within the following classes of pharmacological interventions (as defined by the British National Formulary (BNF 2010)):

1. hypnotics, anxiolytics and barbiturates;
2. antipsychotic drugs (including depot injections);
3. antimanic drugs;
4. antidepressant drugs: tricyclic and related, monoamine-oxidase inhibitors, SSRIs and related, and other antidepressant drugs;
5. central nervous system stimulants;
6. antiepileptics/mood stabilising agents;
7. drugs used in substance dependence.

If sufficient studies are found, we plan to group outcome measures by class of drug, with possible subgroup analysis with classes of type of drug (for example, tricyclic antidepressants analysed separately from SSRIs).

Types of outcome measures

Primary and secondary outcomes are listed below in terms of single constructs. We anticipate that a range of outcome measures will have been used in the studies included in the review, for example, relevant outcomes may be measured by self-report scales or by an external observer. We will report long-term follow-up data for relevant outcomes without restriction on the period of follow-up.

Primary outcomes

- Narcissistic symptoms: improvement in narcissistic pathology as measured on validated clinical scales such as the Structured Clinical Interview for Axis II Personality Disorders (SCID-II) (First 1997), the Structured Interview for DSM-IV Personality (SIDP-IV) (Pfohl 1997) or self-report measure such as the Personality Diagnostic Questionnaire-4 (PDQ-4) (Hyder 1994), NEO Personality Inventory (NEO-PI-R)/NEO Five-Factor Inventory (NEO-Costa 1992), the Narcissistic Personality Inventory (NPI) (Raskin 1981).
- Global state/functioning: measured through improvement on the Global Assessment of Functioning numeric scale (GAF) (APA 2000), Global Assess Scale (GAS) (Endicott 1976), Clinical Global Impression of Severity scale (CGI-S) (Guy 1976), Symptom Checklist-90 (SCL-90R) (Derogatis 1984) or similar validated scales.
- Social functioning: measured through improvement on the Social Adjustment Scale (SAS-SR) (Weissman 1976), the Social Functioning Questionnaire (SFQ) (Tyrer 2005) or similar validated scales.
- Adverse events: measured as incidence of patients with at least one adverse event and of the three most common adverse events in particular; this dichotomous outcome, measured as numbers reporting.

Secondary outcomes

- Quality of life: self-reported improvement in overall quality of life measured through improvement in scores on the European Quality Of Life instrument (EuroQol) (EuroQol 1990), SF36 (Ware 1993) or similar validated scales.
- Engagement with services: health-seeking engagement with services measured though improvement on the Service Engagement Scale (SES) (Tait 2002) or similar validated scales.
- Anxiety symptoms: improvement in anxiety symptoms as measured on the State-Trait Anxiety Inventory (STAI) (Spielberger 1983) or similar validated scales.
- Depressive symptoms: improvement in depressive symptoms as measured on the Hamilton Depression Rating Scale (HAMD) (Hamilton 1960), the Inventory Depression (BDI) (Beck 1961) or similar validated scales.
Satisfaction with treatment: measured through improvement in scores on the Client Satisfaction Questionnaire (CSQ-8) (Attkisson 1982) or similar validated scales.

Leaving the study early: measured as a proportion of participants discontinuing treatment from the point of randomisation.

Employment status: measured as number of days in employment over the assessment period.

Search methods for identification of studies

Electronic searches

We will search the electronic databases listed below:

- Cochrane Central Register of Controlled Trials (CENTRAL), part of the Cochrane Library
- MEDLINE
- EMBASE
- CINAHL
- PsycINFO
- ASSIA
- BIOSIS
- Dissertation Abstracts
- National Criminal Justice Reference Service Abstracts
- Science Citation Index (SCI)
- Social Sciences Citation Index (SSCI)
- Sociological Abstracts
- ZETOC (Conference search)
- metaRegister of Controlled Trials

Electronic searches will be based on the following MEDLINE search strategy which includes the Cochrane highly sensitive search strategy for identifying randomised trials (Lefebvre 2008). The strategy includes search terms for all types of personality disorder as this is one of a series of PD reviews. Search terms and syntax will be modified as necessary for other databases.

1 exp Personality Disorders/
2 (moral adj2 insanity).tw.
3 (DSM and (axis and II)).tw.
4 (ICD and (F60 or F61 or F62)).tw.
5 (code$ or eccentric$ or dramatic$ or anxious$ or fearful$) adj5 cluster$.tw.
6 ("Cluster A" or "Cluster B" or "Cluster C").tw.
7 (aggressiv$ or anxious$ or borderline$ or dependent$ or emotional$ or passiv$ or unstable) adj5 personalit$.tw.
8 (anankastic$ or asocial$ or avoidant$ or antisocial$ or compulsiv$ or dissocial$ or histrionic$ or narciss$ or obsessiv$ or paranoid$ or psychopath$ c sadist$ or schizoid$ or schizotyp$ or sociopath$).tw.
9 (personalit$ adj5 disorder$).tw.
10 character disorder$.tw.
11 (an$ adj (personalit$ or character$ or retentiv$)).tw.
12 or/1-11
13 randomized controlled trial.pt.
14 controlled clinical trial.pt.
15 randomised.ab.
16 placebo.ab.
17 drug therapy.fs.
18 randomly.ab.
19 trial.ab.
20 groups.ab.
21 13 or 14 or 15 or 16 or 17 or 18 or 19 or 20
22 exp animals/ not humans.sh.
23 21 not 22
24 12 and 23

Searching other resources

Hand searching

We will search the reference lists of included and excluded studies for additional relevant trials. We will examine bibliographies of systematic review articles published in the last five years to identify relevant studies.

Contacting authors

We will also contact authors of relevant studies to enquire about other sources of information and the first author of each included study for information regarding unpublished data. We will contact a representative from all major pharmaceutical companies to request information about any published/unpublished trials.

There will be no restriction by language. Where we are unable to translate a paper, we will use the resources of the Cochrane network to identify Cochrane collaborators and staff who can read the paper in its original language, assess the paper against the review inclusion and exclusion criteria and, if required, translate the paper.

Data collection and analysis

Selection of studies

This review is one of a series of reviews about personality disorders. We will carry out the selection of studies in two stages. Two members of the review team will screen titles and abstracts independently to identify all studies carried out with participants with personality disorder, regardless of any specific personality disorder(s) diagnosis. Two members of the review team will then assess full copies of studies identified against the inclusion criteria. We will identify not only trials with participants diagnosed with NPD, but also trials with participants having a mix of PDs for which data on a subgroup with NPD may be available.

We will only include studies with two treatment conditions in which the relevant participants form a small subgroup if the trial investigators randomised at least five participants with HPD. The rationale is that variance and standard deviation cannot be calculated in samples of two or less, and a two-condition study that randomises fewer than relevant participants will have at least one arm for which variance or standard deviation cannot be calculated.
We will resolve uncertainties concerning the appropriateness of studies for inclusion through consultation with a third review author.

**Data extraction and management**

Two review authors will extract data independently using a data extraction form. We will enter data into Review Manager 5 (Review Manager 2011). Where data are available in the published trial reports, we will contact the authors and ask them to supply the missing information.

**Assessment of risk of bias in included studies**

For each included study, two review authors will independently complete the Cochrane Collaboration’s tool for assessing risk of bias (Higgins 2008a, section 8.5.1). Disagreement will be resolved through consultation with KL. We will assess the degree to which:

- the allocation sequence was adequately generated (‘sequence generation’);
- the allocation was adequately concealed (‘allocation concealment’);
- knowledge of the allocated interventions was adequately prevented during the study (‘blinding’);
- incomplete outcome data were adequately addressed;
- reports of the study were free of suggestion of selective outcome reporting;
- the study was apparently free of other problems that could put it at high risk of bias (for example, substantial differences in trial design, baseline outcomes, or loss to follow up).

Each domain will be allocated one of three possible categories for each of the included studies: low risk of bias, or high risk of bias or unclear risk of bias where the risk bias is uncertain or unknown.

**Measures of treatment effect**

For dichotomous (binary) data, we will use the odds ratio (OR) with a 95% confidence interval to summarise results within each study. The OR has been chosen because it has statistical advantages relating to its sampling distribution and its suitability for modelling, and is a relative measure so can be used to combine studies.

For continuous data, we will compare the endpoint mean scores between the treatment groups and standardise them to a uniform scale by dividing by the pooled standard deviation of participants’ outcomes (standardised means difference, SMD). The SMD is advantageous in making the combination of outcome data assess different instruments possible. We will provide 95% confidence intervals. Where possible, we will make these comparisons at specific follow-up periods: (1) within the first month; (2) between one and six months; and (3) between six and twelve months. Where possible, we will report point and interval data. Where only change data are available, we will use them instead but analyse separately.

We will report continuous data that are skewed in a separate table and will not calculate treatment effect sizes to minimise the risk of applying parametric statistics to data that depart significantly from a normal distribution. We will define skewness as occurring when, for a scale or measure with positive values and a minimum value of zero, the mean is less than twice the standard deviation (Altman 1996).

**Unit of analysis issues**

**Cluster-randomised trials**

We will follow the guidance on statistical methods for cluster-randomised trials described in the Cochrane Handbook for Systematic Reviews of Interventions (Higgins 2008b, Section 16.3). We will seek direct estimates of the effect (for example, an odds ratio with its confidence interval) from an analysis that properly accounts for the clustering (Altman 1996). This requires information on an intraclass correlation coefficient (ICC), which describes the relative variability in outcome within and between clusters. We will extract this information from the articles if available; otherwise, we will contact the authors or use external estimates obtained from similar studies. We will find closest matching scenarios with regard to both outcome measures and types of clusters from existing databases of ICCs (Ukoumunne 1999) and if we are unable to identify any, we will perform sensitivity analyses using a high ICC of 0.1, a moderate ICC of 0.01 and a small ICC of 0.001. We recognise that these values are relatively arbitrary but prefer to use them to adjust the effect estimates and their standard errors due to the implausibility that the ICC is actually zero. Subsequently, we will combine the estimates and their corrected standard errors from the cluster-randomised trials with those from parallel designs using the generic inverse variance method in Review Manager 5 (RevMan 2011) (Donner 2001).

**Cross-over trials**

When conducting a meta-analysis combining the results of cross-over trials, we plan to use the inverse variance methods recommended by Ukoumunne 2002. Where c presented from a cross-over trial are restricted (and more information is not available from the original investigators), we plan to use the presented data within the first six months only, up to the point of cross-over.

**Multi-arm trials**

We will include all eligible outcome measures for all trial arms in this review. Where there are more than two arms of the trial that meet the inclusion criteria and each refers to a different treatment, we will include them as different comparisons (Higgins 2008b). If two or more arms refer to the same type treatment, we will combine ! groups to create a single pair-wise comparison (Higgins 2008b).

**Dealing with missing data**

**Missing observations**

In spite of using comprehensive search strategies, we cannot rule out the possibility of missing whole studies leading to a presence of publication bias and we will address this as described below (see ‘Assessment of reporting bias’).

We will discuss the possibility of obvious presence of missing outcomes of primary studies and rate this in the risk of bias tables, under ‘Selective reporting bias’. As concerns missing outcomes of individual patients, we will report missing data and dropouts for each included study and will also report the number of participants included in the final analysis as a proportion of all participants in each study. We will contact the original investigators to request information on whether or not it be assumed to be ‘missing at random’. Where missing data might be assumed to be ‘missing at random’ then it may be appropriate to analyse only the available data.

We will assess the extent to which the results of the review could be altered by the missing data by, for example, a sensitivity analysis based on consideration of the ‘best-case’ and ‘worst-case’ scenarios (Gamble 2005). Here, the ‘best-case’ scenario is that where all participants with missing outcomes in the experimental condition have good outcomes, and all those with missing outcomes in the control condition had poor outcomes, and the ‘worst-case’ scenario is the converse (Higgins 2008b).

**Missing statistics**

If relevant statistics are missing, we will ask the study authors to provide them. If the data cannot be obtained, we will try to find an adequate way of imputation of the missing data with replacement values if, after seeking statistical advice, to do so is deemed practical and appropriate (for example, last observation carried forward, imputing an assumed outcome such as assuming all were poor outcomes). In any case, we will make the assumptions of the coping methods explicit and discuss potential impacts on the findings of the review. If there is no advisable way of imputation, we will provide a qualitative summary.

**Assessment of heterogeneity**

We will assess the extent of between-trial differences and the consistency of results of any meta-analysis in three ways: by visual inspection of the forest plots, by performing the $\chi^2$ test of heterogeneity (where a significance level less than 0.10 will be interpreted as evidence of heterogeneity), and by examining the $I^2$ statistic.
The I\(^2\) statistic describes approximately the proportion of variation in point estimates due to heterogeneity rather than sampling error. We will consider values of less than 40% as where heterogeneity may not be important, values in the range 30% to 60% may represent moderate heterogeneity, values in the range to 50% may represent substantial heterogeneity and 75% to 100% considerable heterogeneity. We will attempt to identify any significant determinants of heterogeneity categorised at moderate or high. We cannot state in advance what our preferred method of dealing with heterogeneity, if present, would be as this would be contingent on the data. However, we would first check that the data had been correctly entered. There are a number of methods that can be used for handling heterogeneity and if include the following. The studies can be examined to explore the reason for heterogeneity but this is not recommended where there are few studies. A random-effects model could be used. Where a study paper is an outlier, sensitivity analysis can be carried out with and without the study. Other strategies include ignoring the heterogeneity, not performing a meta-analysis and changing the effect measure. We will seek statistical advice before using any of these methods.

Assessment of reporting biases
We will draw funnel plots (effect size versus standard error) to assess publication bias if sufficient studies are found. Asymmetry of the plots may indicate publication although they may also represent a true relationship between trial size and effect size. If such a relationship is identified, we will further examine the clinical diversity studies as a possible explanation (Egger 1997).

Data synthesis
We will undertake a quantitative synthesis of the data using both fixed- and random-effects models. Meta-analyses may be conducted to combine comparable outcome measures across studies. In carrying out meta-analysis, the weight given to each study will be the inverse of the variance so that the more precise estimates (from larger studies with more events) are given more weight. We will use random-effects models because studies may include somewhat different treatments or populations. W group outcome measures by length of follow-up.

Subgroup analysis and investigation of heterogeneity
If sufficient studies are found, we will undertake subgroup analysis to examine the effect on primary outcomes of:

1. participants’ principal diagnosis, (for example, personality disorder, axis-I disorder);
2. setting (inpatient, outpatient);
3. type of drug within class.

Sensitivity analysis
If there is sufficient data, we will undertake sensitivity analyses to investigate the robustness of the overall findings in relation to certain study characteristics. We plan to conduct sensitivity analyses for:

1. concealment of allocation;
2. blinding of outcome assessors;
3. extent of dropouts.

Acknowledgements
We would like to thank the editorial team of the Cochrane Developmental, Psychosocial and Learning Problems Group (CDPLPG), Belfast, especially Jane Dennis, Champion, Laura MacDonald, Jo Abbott, Margaret Anderson and Geraldine Macdonald, and the German Cochrane Centre for supporting this work. We are grateful to Gerd Antes, director of the German Cochrane Centre, who made contact with CDPLPG and helped in gaining grants for financing this work. We are also grateful to I Schumacher, director of the Institute of Biostatistics and Medical Informatics at the University Medical Center Freiburg, who gave support in application submission.

We are grateful to the German Ministry of Education and Research (BMBF; grant no. 01KG0812), and the research committee of the University Medical Center Mair supporting this work.

History

Contributions of authors
Jutta Stoffers wrote the protocol.
Michael Ferriter, Birgit A Vollm, Simon Gibbon, Hannah Jones, Najat Khalifa, Klaus Lieb and Conor Duggan helped write and revise the protocol.

Declarations of interest
- Jutta Stoffers - none known.
- Nick Huband - none known.
- Michael Ferriter - none known.
- Birgit A Vollm - none known.
- Simon Gibbon - none known.
- Hannah Jones - none known.
- Conor Duggan - advisor to a current randomised controlled trial of schema focused therapy at Ashworth Special Hospital, UK; investigator in a completed randomised controlled trial of social problem solving therapy plus psychoeducation for people with personality disorder.
- Klaus Lieb - chair, Department of Psychiatry and Psychotherapy, University Medical Center, Mainz; advisor to a planned randomised controlled trial of schema therapy in patients with personality disorders.

Sources of support
Internal sources
- Nottinghamshire Healthcare NHS Trust, UK.

External sources
- Federal Ministry of Education and Research, Germany.
  Grant no. 01KG0812
- NHS Cochrane Collaboration Programme Grant Scheme, UK.
References

Additional references

Altman 1996


APA 2000


Coid 2006


Coccaro 2005


Crawford 2005


Crawford 2009


Crawford 2000


Costa 1992


Costa 1990


Cridland 2002


Cronbach 1951


Cronbach 1957


Cronbach 1957


Cronbach 1957


Cronbach 1957


Cronbach 1957


Cronbach 1957


Cronbach 1957


Cronbach 1957


Cronbach 1957


Cronbach 1957


Cronbach 1957


Cronbach 1957


Cronbach 1957


Cronbach 1957


Cronbach 1957


Cronbach 1957


Cronbach 1957


Cronbach 1957


Cronbach 1957


Cronbach 1957


Cronbach 1957


Cronbach 1957


Cronbach 1957


Cronbach 1957


Cronbach 1957


Cronbach 1957

Pharmacological interventions for people with narcissistic personality disorder – The Cochrane Library – Stoffers – Wiley Online Library

Johnstone 2005


Klein 1993


Lefebvre 2008


Lenzenweger 2007


Miller 2007


Newton-Howes 2006


Paris 1996


Paris 2005


Perry 1990


Pfohl 1997
