MAJOR RESEARCH PROJECT

Theories of addiction and their clinical implications

Submitted by **Daniel Casey** to the University of Exeter as a thesis for the degree of **Doctor of Clinical Psychology**, August 2024

I certify that all material in this thesis which is not my own work has been identified and that any material that has previously been submitted and approved for the award of a degree by this or any other University has been acknowledged.

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The impact of the disease model of addiction on the self-perception and recovery potential of substance users: A systematic review

Trainee: Daniel Casey

Main Supervisor: Professor Lee Hogarth, Associate Professor of Experimental Psychology, School of Psychology, Faculty of Health and Life Sciences, University of Exeter

Secondary Supervisor: Dr Fiona Warren, DClinPsy Research Director, School of Psychology, Faculty of Health and Life Sciences, University of Exeter

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Abstract

Scientific discourse about addiction might have implications for the way that problematic substance users see themselves and their recovery journey. Proponents of the brain disease model of addiction (BDMA) argue that emphasising the biological determinants of addiction should reduce problematic substance users' negative self-perception (blame, shame) and enhance their potential for recovery. Critics of the BDMA predict the opposite, arguing that emphasising biological determinants is likely to increase problematic substance users' negative self-perception and reduce their recovery potential. The current systematic review collated studies that have tested these predictions.

Systematic searches of PsycINFO, PubMed, and Web of Science Core Collection were conducted in January 2024. Eligible studies included correlational and/or experimental framing designs that measured/manipulated substance users' beliefs in a disease construct of addiction to detect correlations/effects on their negative self-perception or recovery potential. Risk of bias was assessed using the Quality Assessment Tool for Quantitative Studies. Synthesis was conducted through vote counting based on direction of correlations/effects and summarised narratively due to significant heterogeneity in study design.

After title and abstract screening, full-text review, and reference list checks, a total of 16 studies were included in the review. Nine studies were correlational designs with mostly treatment-engaged participants whereas the other seven were experimental framing designs with harmful substance users. A variety of outcomes indexing recovery potential were identified including relapse status, substance use severity, self-efficacy, treatment attendance, treatment motivation, and problem

recognition. Indices of negative self-perception included self-blame, perceived discrimination to self, and shame.

Seventy-seven effects were extracted from the 16 studies and vote counted. Forty-two relationships were identified in correlational studies: 19 null, 10 beneficial, and 12 iatrogenic for recovery potential, and one null for negative self-perception. Thirty-five effects were identified in framing studies: 20 null, one beneficial, and seven iatrogenic on recovery potential, and six null, zero beneficial, and one iatrogenic on negative self-perception.

Null effect was the most common finding but the significant effects indicated limited support for the iatrogenic hypothesis. However, a systematic research program is required before strong claims can be made about whether scientific discourse emphasising the brain disease model of addiction affects substance users' negative self-perception and recovery potential. This finding was interpreted in light of conceptual ambiguities in defining a disease model of addiction and methodological difficulties inherent in quantifying the precise aspects of beliefs about the causes and nature of addiction that are most pertinent to substance users' negative self-perceptions and recovery potential. Additionally, ideologically driven epistemic grandiosity and associated economic incentives towards a more reductionist view of science were implicated, along with opposition to such trends.

Recommendations for future research into substance users' beliefs about addiction include a greater emphasis on qualitative methodologies alongside a shift towards within-subject in-person designs for quantitative research. Additionally, the focus on addiction aetiology is questioned, and a shift towards research that focuses on the processes, subjective and objective, that lead substance users towards recovery from addiction is advocated.

Introduction

Perspectives on the nature and causes of addiction

Within healthcare the ideal function of diagnosis is to identify and classify a pathology of the body linked to a clearly defined aetiology. Medical professionals can then use this information to provide a reliable prognosis and prescribe a course of treatment (Maitland, 2010). This can be a relatively straightforward process for many illnesses and injuries of the body, such as infections or broken bones, but becomes more complex when a health problem is associated with the human mind and behaviour. There has been debate around this issue for decades in the field of mental health where understanding of aetiology remains elusive (Clark et al., 2017). While some factors have been strongly associated with mental health problems including social inequalities (Perry, 1996), and both individual (Dalvie et al., 2021) and intergenerational traumatic stress (Yehuda & Lehrner, 2018), specific causal pathways for most mental health difficulties remain ill-defined and have been summarised in jest as “everything causes everything” (Johnstone & Boyle, 2018, p. 148). Due to these circumstances, current diagnostic practice is based on descriptions of symptom clusters without reference to aetiology. The resulting diagnostic categories vary considerably in both validity and reliability (Kapadia et al., 2020) and although effective treatments can be identified for certain difficulties like anxiety disorders (Bandelow et al., 2017), for many problems treatment is minimally effective (Leichsenring et al., 2022).

Addiction and the myriad diagnostic labels associated with it, such as alcohol use disorder, are no exception to these issues. In fact, the very term *addiction* lacks a universally agreed definition and is often used ambiguously (Rosenthal & Faris, 2019). Some argue that substance and behavioural addictions are very similar

phenomena (Alavi et al., 2012; Griffiths, 2017) whereas others see them as possessing critical differences relevant to treatment (Yau & Potenza, 2015). Such disagreements occur in a context of conceptual disarray within addiction research resulting in contradictions such as a strong research focus on biomolecular aetiology for a problem that is defined primarily in behavioural terms (Hammer et al., 2013).

A variety of explanatory theories have emerged within this conceptual and aetiological vacuum in an attempt to define the nature and causes of addiction. One of the earliest explanatory theories of addiction, commonly described as the moral model, was strongly influenced by Christian values i.e. *"Wine is a mocker, strong drink a brawler, and whoever is led astray by it is not wise*" (Proverbs 20:1, English Standard Version). The moral model emphasised personal responsibility for addiction due to a lack of moral character and suggested moral development as the pathway to recovery.

In the early 19th century, as the prevailing religious ideas of the time were being eclipsed by a growing scientific worldview, the medical model arose and challenged the moral model. This model strongly emphasised loss of control over substance use (Crocq, 2007) and the need for medical intervention. A famous proponent of this model was the physician Thomas Trotter (1813) who critiqued "the priesthood [for] pour[ing] forth its anathemas from the pulpit" (p. 3) and argued for a medical, philosophical and chemical understanding of addiction. As advances in scientific and medical knowledge continued over the 19th and early 20th centuries, this medical conceptualisation became crystalised when the World Health Organisation added alcoholism to the International Classification of Diseases (ICD) in 1948, shortly followed by its inclusion in the DSM-I in 1952. A variety of addictions

to other substances and behaviours (e.g., gambling) have been classified as diagnosable clinical states in subsequent iterations of the DSM and ICD.

As scientific and technological development continued into the late 20th century, the medical model became increasingly influenced by the emerging multidisciplinary field of neuroscience. The brain disease model of addiction (BDMA) emerged in this context with proponents stating that substance addiction is "a chronic, relapsing disease that results from the prolonged effects of drugs on the brain" (A. Leshner, 1997, p. 46). The purpose of the BDMA was to improve understanding of the biological aetiology of addiction, develop better medical treatments for those struggling with addiction, and to reduce blame and stigma by defining addiction as outside individuals' control. Its largest advocates, the National Institutes of Drug Abuse (NIDA) and Alcohol Abuse and Alcoholism (NIAAA) in the US received annual research budgets of over $2 billion in 2023 (NIAAA, 2023; NIDA, 2023).

While the aims of the BDMA are noble, many argue that its intentions have not been realised and unintended harms or costs have resulted (Hall et al., 2015a). For example, the translation of neuroscientific research into simplistic terms in the media has led the public towards an essentialist view (Haslam, 2011), i.e., that an individual's risk of addiction is attributable to their bio-psychological constitution, promoting marginalisation of people as "neurobiological others" (p. 65), and increasing social divisions (Buchman et al., 2011). Stanton Peele (2016) argues that the BDMA is actively iatrogenic because it convinces people they are helpless and "undercuts the self-efficacy required to achieve freedom from addiction" (p. 100). Finally, Nick Heather (2017) suggests that the longstanding assumption that the BDMA reduces stigma is a false assumption for which some supporting evidence

has been published (Kelly et al., 2021b; Pescosolido et al., 2010; Wild et al., 2021). The level of activity surrounding this debate is attested to in a voluminous book that includes a range of authors' evaluations of the BDMA (Heather et al., 2022)

A variety of alternative models to the BDMA have been put forward by researchers that focus more prominently on the role of psychosocial factors in addiction aetiology. Heyman (2013; 2021) posits that addiction is a disorder of choice driven by behavioural psychological principles of reward learning such as the matching law, melioration, and hyperbolic discounting. This behavioural-economic model is linked more directly to sociological factors by Hogarth (2022) who argues that the choice to continue using substances or engaging in addictive behaviours is powerfully motivated by socioeconomic deprivation-related factors (Shuai et al., 2022) significantly increasing the value of addictive substances/behaviours relative to other sources of reward.

A slightly different approach is taken by Lewis (2015; 2017) who focuses on the neurobiology of addiction, arguing for a developmental-learning model which suggests brain changes in addiction are similar to those observed when recurrent, highly motivated goal seeking results in the development of deep habits including normative 'compulsive' behaviours like exercising, and falling in love. Peele's life-process model (2016; Peele et al., 1991) takes a similar approach in framing addiction as a normative habitual response and source of gratification and security for people that can be understood only in the context of social relationships and experiences. Finally, Alexander (2000; 2010) takes a broader approach and considers addiction through an "adaptive paradigm". He argues that what we call addiction is in fact an adaptive response to the global expanse of western free-market capitalist societies which dislocate people from their traditional sources of

psychological, social, and spiritual support. He indicates the high prevalence of addiction in western societies and in indigenous societies subject to colonisation as evidence for his position.

These alternative theories suggest that addiction is a strong but otherwise normal form of motivation to adapt to life's challenges suffered disproportionately by disadvantaged individuals (Alexander, 2023), rather than a constitutional aberration of brain function. Consequently, an individual's ability to overcome addiction depends on augmenting coping strategies, pursuing values and purpose in life, repairing relationships, and expressing personal agency – all of which require a transition away from a biomedical approach.

The effect of scientific discourse about addiction on substance users

It is important to acknowledge that explanatory theories of addiction which originate in academia do not just guide research paradigms but also perform "cultural work" (Hammer et al., 2013, p. 2). For instance, in healthcare contexts academic theories shape the beliefs and discourse that treatment providers and their clients deploy to explain and understand their conditions; including the impact on their life, causes, prognosis, and how they should be remedied (Jack et al., 2019). Such beliefs can influence people's behaviour in either health-promoting or health-damaging ways and, for this reason, beliefs are integral to many models of health behaviour. Brennan's (2018) model of psychological adjustment posits a person's basic assumptions as the critical factor in their ability to adapt and respond to a health problem. Similarly, Leventhal's Common Sense Model (2016) includes the role of specific beliefs about health problems such as their identity, cause, timeline, consequences, and curability as having a significant impact on coping behaviours.

Evidence of the impact of health beliefs on health behaviours has been found in a variety of conditions. Higher self-efficacy, higher perceived severity of illness, and lower perceived barriers to change have been associated with increased hypertension medication adherence (Al-Noumani et al., 2019). In chronic pain, beliefs about pain equating to bodily damage have been associated with higher physical disability (Jensen et al., 1994). Cognitive-behavioural treatments, which assume a causal roles for beliefs, have been found to decrease beliefs around the disabling nature of pain and increase beliefs about control over pain, leading to symptomatic improvement (Jensen et al., 2021). With regards to substance use, increased efficacy beliefs about harm reduction strategies was found to decrease alcohol consumption in young people (De Leon et al., 2023), and in smokers, higher perceived severity of smoking-related diseases, higher self-efficacy, and fewer perceived barriers to quitting were associated with increased likelihood of quitting smoking (Kaufman et al., 2018). It is therefore important to understand how prevalent explanatory theories influence the health beliefs and behaviours of people experiencing substance addictions.

The explanatory theories favoured by clinicians and providers of addiction treatment services have been studied to some extent, and systematically reviewed by Barnett et al. (2018). They concluded that while the disease concept of addiction had relatively strong endorsement, theories of addiction were highly variable across the workforce such that “service users may experience multiple and potentially contradictory explanations of addiction” (p. 717). There have also been studies on the views of explanatory theories of addiction in scientists (Hammer et al., 2012; Ochterbeck & Forberger, 2022), adult children of people with addiction problems (Järvinen, 2015), the general public (Crawford & Heather, 1987; Meurk, Carter, Hall,

et al., 2014; Meurk, Carter, Partridge, et al., 2014) and students (Henderson & Dressler, 2017). The results obtained with these groups were similar to the Barnett et al. (2018) study with treatment providers, namely a general endorsement of the disease concept simultaneously alongside a multitude of other views of addiction. That is, people tend not to be strongly wedded to any particular aetiological theory of addiction, but rather, endorse the full list of proposed risk variables with little discrimination. What is noteworthy in this literature, is the paucity of research on the addiction theories utilised by people with substance addictions, and the potential impact of these beliefs on their self-perception and recovery.

The benefits and harms of a disease model of addiction

One hotly debated issue among addiction scientists is whether scientific discourse around the brain disease model of addiction (BDMA), with its emphasis on biological determinants and medical solutions, is likely to impact on substance users' negative self-perception and recovery potential. For instance, the principal exponents of the BDMA, Nora Volkow and George Koob, have argued against the idea that the BDMA might harm substance users. In their response to an article criticising the BDMA, they asserted that "the mere framework of [the] BDMA has benefits in treatment as it significantly diminishes the stigma attached with addiction and gives hope for recovery to those fighting this devastating disease" (2015, p. 677). It is noteworthy that they did not reference any published study to support this claim, suggesting it was an ideological rather than an empirical assertion, which may or may not be true. Whatever the case, Volkow and Koob have stated a testable prediction that belief in the BDMA should decrease substance users' negative self-perception and increase their recovery potential.

Nick Heather and Stanton Peele have made equally strong assertions in the opposite direction. In an article about the launch of the Addiction Theory Network, an organisation whose mission statement is “to oppose the dominant influence of the BDMA, and to collaborate on developing alternative ways of understanding and responding to addiction”, Nick Heather stated: “While this biology-based definition of addiction aims to alleviate the moral judgement, discrimination and stigma associated with drug use, evidence suggests that the BDMA has only furthered the stigma associated with addiction, leaving addicts increasingly vulnerable to exclusion and marginalisation” (2018, p. 250). Likewise, in a review article critiquing the BDMA and its emphasis on pharmacological treatment Stanton Peele claimed that “telling yourself that you can't quit your addiction without the drug undercuts the self-efficacy required to achieve freedom from addiction” (2016, p. 100). However, these assertions were also ideological rather than empirical. To support their claims, Heather cited one of his own earlier review articles, and Peele provided no reference at all. Despite the absence of references, Heather and Peele are nevertheless on record in stating their predictions in opposition to Volkow and Koob that belief in the BDMA should increase substance users’ negative self-perception and decrease their potential for recovery.

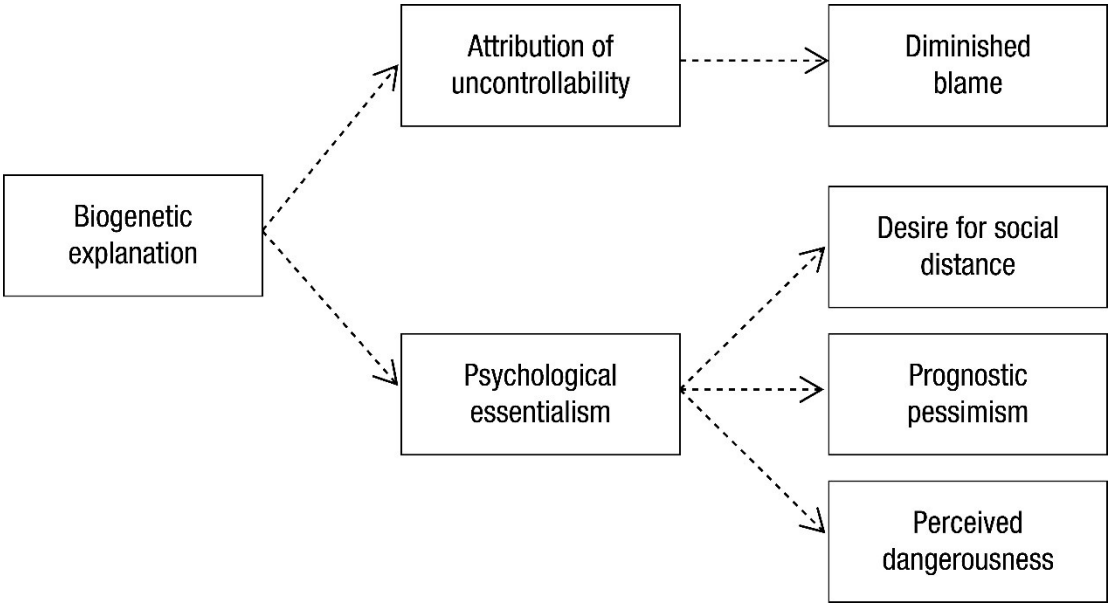
What evidence is there for these contradictory claims? Indirectly linked research testing the impact of neurobiological and genetic explanations of mental health provides some support to both sides. A meta-analysis of correlational studies by Kvaale, Gottdiener, et al. (2013) found that lay people who hold biogenetic explanations for mental disorders tend to blame effected persons less for their problems, but perceive them as more dangerous and desire more distance from them. Another meta-analysis by the same authors (Kvaale, Haslam, et al., 2013)

looked at experimental studies and included professionals and individuals affected by psychological problems in addition to lay people. They found that biogenetic explanations reduced blame but induced prognostic pessimism.

In a theoretical review summarising these findings, Haslam and Kvaale (2015) described biogenetic explanations as a mixed blessing (Figure 1). They argue that blame is reduced on the basis of attribution theory (Weiner, 1993) which suggests people are more sympathetic when undesirable behaviours are perceived as uncontrollable. This is reinforced by neuroscience-based mechanistic explanations which diminish the notion of freewill and lead people to be seen as less blameworthy (Shariff et al., 2014). However, they proposed that mechanistic thinking also has negative consequences in that it leads to psychological essentialism - the belief that fixed, hidden, and identity-based properties (e.g., DNA or brain structure) generate an observed behaviour or characteristic. This essentialist attribution of addiction to the individual's constitution is argued to drive the effect of biogenetic explanations on increased desire for social distance, perceived dangerousness, and prognostic pessimism.

Figure 1

The mixed blessing model (Haslam & Kvaale, 2015)



Within the field of addiction, there appears to be a relatively small and heterogeneous set of studies which have explored the effect of BDMA beliefs on stigma and recovery. Moreover, to this authors' knowledge, no previous attempt has been made to systematically identify and synthesize such evidence to arrive at an evidence-based conclusion concerning the impact of such discourse, which is a significant omission given that contradictory claims in this space are central to the debate.

Aims and objectives

The aim of the current systematic review is to address the claims made about the benefits and harms of the disease model of addiction by advocates and critics. Using Haslam and Kvaale's (2015) mixed blessing model as a framework, this review seeks to answer the following questions:

(1) Is belief in a disease model of addiction linked to an increase or decrease in substance users' negative self-perception?

(2) Is belief in a disease model of addiction linked to an increase or decrease in substance users' recovery potential?

If the 'iatrogenic hypothesis' proposed by Heather (2018) and Peele (2016) to criticise the BDMA is correct, then belief in a disease model of addiction should be correlated with, or experimentally induce, increased negative self-perception and reduced recovery potential in substance users. By contrast, if the 'beneficial hypothesis' proposed by Volkow and Koob (2015) is correct, then belief in a disease model of addiction should be correlated with, or experimentally induce, decreased negative self-perception and increased recovery potential in substance users. This work has implications for how scientific addiction theories are described in clinical and public discourse and whether they promote desirable outcomes for substance users.

Method

This systematic review is reported in line with the standards of the 'Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) 2020' (Page et al., 2021). The aims and methodology of this review were pre-registered via PROSPERO [CRD42024504264].

Search strategy

Systematic searches of PsycINFO via Ovid, PubMed, and Web of Science Core Collection were conducted on 13 January 2024. These databases represented the most relevant repositories for addiction research across the life and social sciences. PsycINFO includes psychological, behavioural and social science articles, PubMed includes life and behavioural science articles, and Web of Science includes social science, humanities, and multidisciplinary articles. The Electronic Theses

Online Service (EThOS) and Open Access Theses and Dissertations (OATD) were also searched to reduce publication bias through the identification of grey literature.

Search terms were developed that aimed to achieve an adequate balance of sensitivity and specificity and formulated using the 'PI(E)COS' approach: (i) population: people using substances i.e. drugs and/or alcohol; (ii) intervention/exposure: disease model of addiction; (iii) outcomes: indices of negative self-perception (e.g., shame, self-blame) and/or recovery potential (e.g., self-efficacy, abstinence); (iv) comparator: not applicable; and (v) study design: correlational or experimental framing studies. Search terms were created for PsycINFO via Ovid, PubMed, and Web of Science (Table 1) and no date limits were set because no review of this topic had been conducted previously.

Table 1

Search strategy

Population
'heroin', 'cocaine', 'amphetamine', 'methamphetamine', 'marijuana', 'cannabis', 'alcohol', 'ketamine', 'smoking', 'tobacco', 'nicotine', 'substance abuse*', 'addict*'
Intervention/Exposure
'disease', 'entity', 'fixed', 'binary', 'compuls*', 'belief*', 'fram*', 'model', 'etiolog*'
Comparator
not applicable
Outcomes
'blame', 'stigma', 'self-efficacy', 'efficacy', 'recovery', 'relapse', 'help-seek*', 'recognition', 'motivation'
Study design (exclusionary terms)
'systematic review', 'meta-analysis', 'review'

* wildcard term e.g. abuse, abuser, abusers

Study selection

The retrieved articles were uploaded to Rayyan and screened against inclusion and exclusion criteria (Table 2) in three phases: (i) title and abstract screening, (ii) full-text review, and (iii) reference list review. Figure 2 provides an

overview of the selection process including reasons for exclusion at each stage. A random sample of 25% of the articles at full-text and reference list review were screened by a second rater with an inter-rater agreement of 88%. Cohen's κ was 0.75 which is considered to represent 'excellent' agreement beyond chance (Cicchetti & Sparrow, 1981). All discrepancies were resolved through discussion with the second rater. In total 16 articles were identified as eligible for inclusion in the systematic review.

The review collated studies which recorded substance users' belief in a disease construct of addiction and related these to indices of negative self-perception and recovery potential (broadly defined). The review also collated studies which had experimentally manipulated substance users' beliefs in a disease construct of addiction that used a framing design to test the causal effect of disease framing on indices of negative self-perception and recovery potential.

A disease construct of addiction was defined as any explanatory construct that pertained to the neurobiological, medical, or genetic causes of addiction and its fixed, entity-like, or categorical nature. Measures of belief included any standardized measures of belief in a disease construct of addiction e.g. the Addiction Belief Scale (Schaler, 1995), and non-standardized measures that demonstrated validity and reliability. Manipulations of belief included any written, visual, or auditory means of attempting to influence a participant to adopt a disease construct of addiction.

The outcomes that were deemed to be plausibly relevant to a person's negative self-perception included, but were not limited to: self-blame, self-perceived discrimination and shame. The outcomes that were deemed to be plausibly relevant to a person's recovery potential included, but were not limited to: relapse status,

substance use quantity, self-efficacy, locus of control, readiness to change, treatment motivation, and problem recognition.

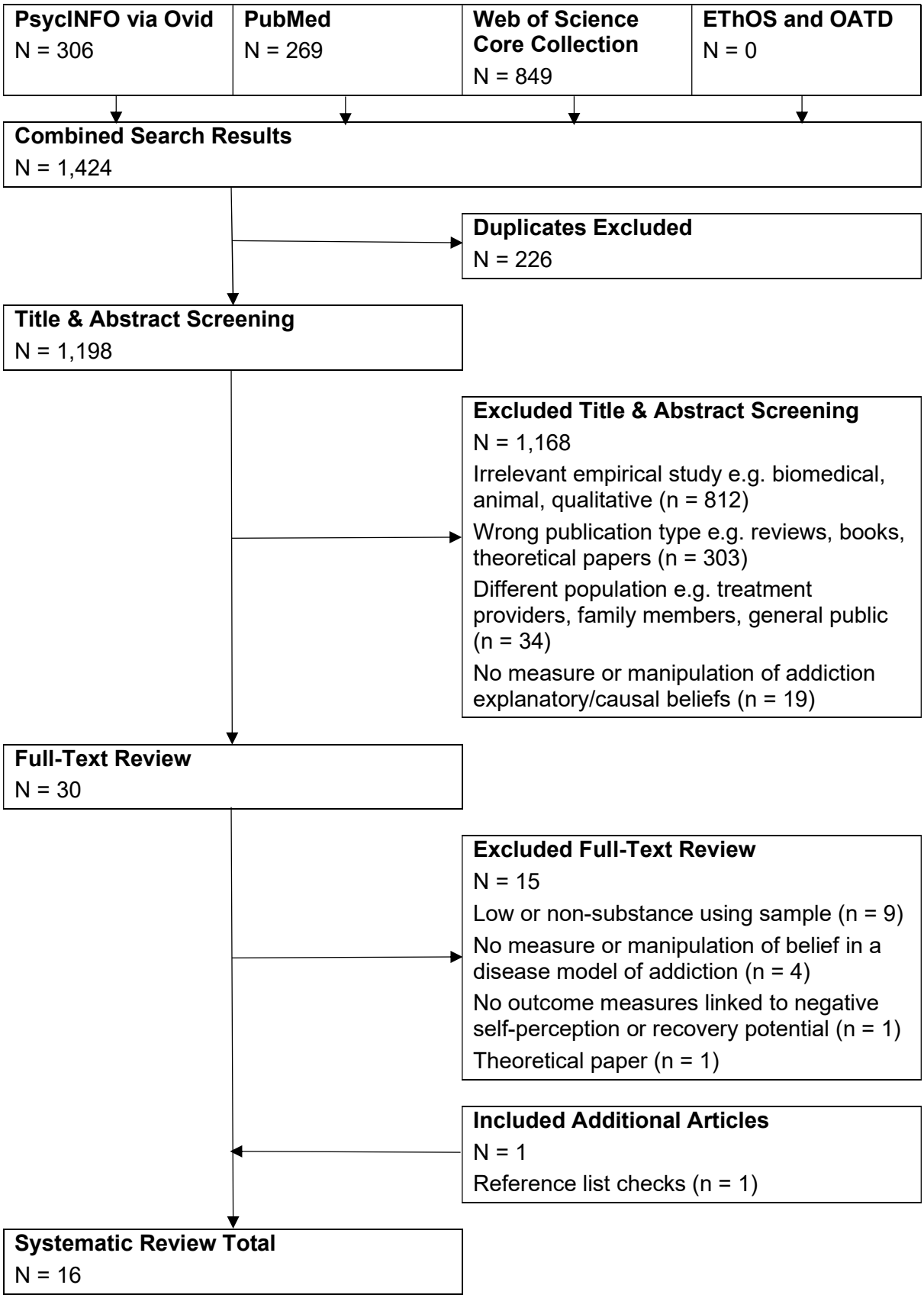
Table 2

Search inclusion and exclusion criteria

Inclusion criteria
Studies that have recruited human participants who use substances i.e. drugs and/or alcohol at a level the researchers deem to be risky, harmful, or hazardous which differentiates them from a general sample who may use substances at low risk levels or not at all.
Studies that measure and/or manipulate substance users' beliefs in a disease construct of addiction including related explanatory constructs that pertain to the neurobiological, medical, or genetic causes of addiction and its fixed, entity-like, or categorical nature.
Studies that include either of the following two outcome measures. 1. Outcome measures that are thought to tap into substance users' negative self-perception arising from their substance use, or label as a member of a substance user group, i.e. the extent to which they believe themselves to be personally responsible or feel negatively about themselves for their substance use problems. And/or 2. Outcome measures that are thought to tap constructs related to substance users' potential for recovery from or control over their substance use, for example: relapse, substance use level, self-efficacy, locus of control, treatment motivation, or problem recognition.
Quantitative studies including both correlational designs measuring these constructs and experimental (within- and between-participants designs) which manipulate disease beliefs to measure effects on the outcomes.
Exclusion criteria
Studies that have recruited participants with behavioural addiction problems i.e. gambling, sex, internet usage, and gaming.
Studies that only include participants indirectly related to people with substance use problems including, but not limited to; family members, general public, clinicians, treatment providers, addiction scientists, and researchers.
Studies with only qualitative data.
Studies not available in the English language.

Figure 2

Study selection process



Quality appraisal

Quality appraisal of all included articles was undertaken using the Quality Assessment Tool for Quantitative Studies (Thomas et al., 2004). This tool was chosen because it was designed specifically for assessing the study quality of health research, including substance abuse, and is applicable across a variety of quantitative study designs. The tool provides guidance to assess methodological quality across the domains of selection bias, study design, confounders, blinding, data collection methods, and withdrawals and dropouts. Each of the six domains is rated as ‘strong’, ‘moderate’, or ‘weak’ based on domain specific criteria. A global rating of ‘strong’ is given to a study if none of its six domains are rated as ‘weak’, a global rating of ‘moderate’ is given if one of its six domains are rated as ‘weak’, and a global rating of ‘weak’ is given if two or more of its six domains are rated as ‘weak’.

To reduce bias and ensure inter-rater reliability, a second rater critically appraised a random sample of 50% of the included articles with an inter-rater agreement of 88%. Cohen’s κ was 0.75 which is considered to represent ’excellent’ agreement beyond chance (Cicchetti & Sparrow, 1981). All discrepancies were resolved through discussion with the second rater.

Data extraction and synthesis

Data extraction from the 16 included papers was undertaken by the main author. The following information was extracted to define the nature of the study: (i) design type: correlational or experimental framing study segmented into separate tables; (ii) study features and sample characteristics; (iii) measures used to index negative self-perception; (iv) measures used to index recovery potential.

The vote counting of study results was used to evaluate evidence for the iatrogenic versus beneficial hypothesis of the disease model (see introduction). From

the correlational studies (shown in Table 3), the significance and direction were noted for each regression analysis which tested whether an index of disease belief was associated with the index of negative self-perception or recovery potential. A significant result was labelled an ‘Iatrogenic relationship’ if greater disease beliefs were associated with increased negative self-perception or decreased recovery potential or labelled ‘Beneficial relationship’ if the significant association was in the opposite direction. Non-significant associations were labelled as ‘No relationship’.

Interpretation of correlations is complex, as disease beliefs might be confounded (covary) with a wide range of other factors, such as dependence severity, familiarity with Alcoholics Anonymous literature and other factors, which might be responsible for any association with negative self-perception and recovery potential. Some studies have addressed this issue by employing adjusted regression models, to test the unique association between disease beliefs and outcomes controlling for such covariates. Table 3 labels the relationships as “Adjusted” or “Unadjusted” to signal when covariates were controlled, to provide insight into the specificity of the associations reported. Similarly, some studies used difference scores to index greater endorsement of disease over control beliefs (e.g., free-will beliefs) revealing greater specificity of the relationship, or they used absolute scores on a disease belief scale which were less specific. The method used for indexing disease beliefs as a “Difference score” or “Absolute score” is noted in the disease measure column of Table 3. Even where optimal methods were used (i.e., disease belief difference scores entered into adjusted regression models), causal inferences cannot be drawn from associations. However, correspondence between correlational and causal effects from experimental framing studies would strengthen conclusions

as to whether disease model beliefs affects substance users' self-perception and recovery potential.

From experimental framing studies (shown in Table 4), significance and direction were noted for each statistical analysis which tested whether the disease belief induction group differed from the control group with respect to the index of negative self-perception or recovery potential. A significant result was labelled an 'Iatrogenic effect' if the disease belief induction group reported increased negative self-perception or decreased recovery potential or labelled 'Beneficial effect' if the significant difference was in the opposite direction. Non-significant groups effects were labelled as 'No effect'. Subgroup analyses were also noted for correlational and framing studies if these relationships/effects were reported within a sample subgroup.

Due to the heterogeneity in the designs, participants, measures, and analytic methods of the included studies, a meta-analysis of effect estimates was not possible. Synthesis was instead undertaken through vote counting based on the number and direction of effects and summarised narratively in accordance with SWiM guidelines (Campbell et al., 2020).

Table 3

Correlational studies

Study and sample characteristics	Measures indexing belief in a disease model of addiction	Measures indexing negative self-perception	Measures indexing recovery potential	Correlations between disease beliefs and negative self-perception	Correlations between disease beliefs and recovery potential	QA tool global rating
Aharon (2000) PhD thesis. Prospective cohort study of Canadian adults (N = 213) with mixed alcohol or drug use problems assessed via the Substance-Abuse Subtle Screening Inventory-2 (SASSI-2). Recruited from a private addiction rehabilitation hospital in Toronto, Canada and followed-up over 3-months. Age (M = 38.7, SD = 11.2) Male (77.2%) Canadians (69.1%) Employed (66.7%) Alcohol (64.1%) Single substance (53.6%)	Predictor label = "Belief in the disease model of addiction". Difference score. Unadjusted. Instrument = Addiction Belief Scale (ABS) 18-items e.g. "The fact that alcoholism runs in families means that it is a genetic disease" Scale = 5-point Likert scale from "strongly disagree" to "strongly agree" with higher scores equating to greater disease model endorsement and lower scores to greater free-will model endorsement.	N/A	Outcome label = "Recovery status". Instrument = Level of recovery. Scale = High recovery (abstinent or up to 3 short lapses) or low recovery (some reduction in frequency and/or quantity of use or unimproved). Outcome label = "Support use". Unadjusted. Instrument = Aftercare and/or 12-step attendance. Scale = High use (1 or 2 times per month up to 3 or more times per month) or low use (started then dropped out or never started).	N/A	**No relationship** Disease belief not associated with recovery status $p > .05$. Effect size not calculable due to no SD reported. **Beneficial relationship** Greater disease beliefs were associated with greater support use $p < .01$. $d = 0.53$.	Strong

Brzostek (2000) PhD thesis. Prospective cohort study of US adolescents (N = 82) with mixed alcohol and/or drug use problems assessed via the substance abuse portion of the Structured Clinical Interview for Diagnoses (SCID). Recruited from a private addiction rehabilitation camp in California, USA. 10th & 11th grade (61.0%) Male (56.1%) Caucasian (70.7%) Cannabis (37.8%) Heroin (22.0%) No prior treatment (61.0%)	Predictor label = "Belief in the disease model of addiction". Absolute score. Instrument = Short Understanding of Substance Abuse Scale (SUSS) disease model subscale 7-items e.g.: "If an alcoholic has a drink, or if an addict takes a hit, they lose control and are unable to stop from getting drunk or high". Scale = 5-point Likert scale from "strongly disagree" to "strongly agree" with the higher total score indicating greater belief in the disease model of substance addiction.	N/A	Outcome label = "Readiness to take steps towards change" Adjusted. Instrument = Stages of Change Readiness and Treatment Eagerness Scale (SOCRATES) taking steps subscale 8-items e.g.: "I am working hard to change my drinking/drug use". Scale = 5-point Likert scale from "strongly disagree" to "strongly agree". Outcome label = "Number of abstinent days". Adjusted. Instrument = Timeline Followback (TLFB). Scale = Number of days using substances over the past 7 days.	N/A	**Beneficial relationship** Greater disease beliefs were associated with greater readiness to change $p < .01$. $r = .31$. **Beneficial relationship** Greater disease beliefs were associated with greater number of abstinent days $p < .01$. $r = .30$.	Strong
Colon and Massey (1989) Published article. Prospective cohort study of US adults (N = 59) with mixed alcohol	Predictor label = "Agreement with disease concept of addiction". Absolute score.	N/A	Outcome label = "Treatment compliance" Unadjusted.	N/A	**Beneficial relationship** Agreement with the disease concept of addiction was positively correlated with treatment compliance at	Moderate

and/or drug use problems requiring detoxification treatment. Recruited from a large public hospital in the Northeastern USA. Age (M = 33.0, SD = 9.3) Male (74.6%) White (81.4%) Employed (53.4%) Years of Education (M = 12.1, SD = 2.2) Polydrug use (59.3%) Prior treatment (M = 3.3, SD = 6.7)	Instrument = Causes of Illness Inventory modified to fit detox units treatment philosophy 5-items e.g.: “running in the family, bad blood, allergy, a chemical imbalance, and being born with it”. Scale = Yes/no responses.		Instrument = Ordinal categories of treatment compliance. Scale = Full (attending aftercare and remained drug and alcohol free), partial (either drug and alcohol free or attending aftercare treatment but not both), non (neither drug and alcohol free or attending aftercare treatment).		30-days $p < .05$. rho = .28. **Beneficial relationship** Agreement with the disease concept of addiction was positively correlated with treatment compliance at 60-days $p < .05$. rho = .27. **Beneficial relationship** Agreement with the disease concept of addiction was positively correlated with treatment compliance at 90-days $p < .05$. rho = .24.	
Grand (2001) PhD thesis. Randomized controlled trial of US adults (N = 117) with mixed alcohol and/or drug dependence assessed via structured clinical interview with a certified social worker or addiction counsellor using the Addiction Severity Index (ASI) and DSM-IV criteria checklist and separately by medical	Predictor label = “Implicit theory about addiction”. Difference score. Instrument = Implicit Theory Questionnaire (ITQ) 3-items e.g.: “You have a particular tendency to abuse alcohol and/or other drugs, and you can’t really do much to change it”. Scale = 6-point Likert scale from “strongly agree” to “strongly	N/A	Outcome label = “Confidence in treatment”. Adjusted. Instrument = Treatment Motivation Questionnaire (TMQ) 3-items. Scale = 5-point Likert scale. Outcome label = “Confidence in self-efficacy”. Adjusted.	N/A	**Iatrogenic relationship** Entity theorists had lower confidence in self-efficacy $p < .01$. $d = 0.55$. **Iatrogenic relationship** Entity theorists had higher cost of change $p < .05$. $d = 0.48$. **No relationship** Entity and incremental theorists did not differ in confidence in treatment $p > .05$. $d = 0.38$.	Strong

staff to determine the need for detoxification. Recruited from a private drug and alcohol abuse treatment facility in New York City, USA. Age (M = 39.97, SD = 11.19) Male (82.1%) Caucasian (58.1%) Employed (77.8%) Years of Education (M = 14.94, SD = 3.22) Alcohol only (42.7%) Alcohol and drugs (31.6%) Drugs only (25.6%) Previous substance treatment (52.4%)	disagree" with the mean score indicating entity belief (3 or less) or incremental belief (4 or more) and scores between 3.1 and 3.9 neutral belief (excluded from the study).	Instrument = Competence Scale (CS) 5-items. Scale = 7-point Likert scale. Outcome label = "Cost and benefit of change" Adjusted. Instrument = Alcohol and Drug Consequences Questionnaire (ADCQ) 29-items. Scale = 5-point Likert scale. Outcome label = "Session attendance". Adjusted. Instrument = Number of sessions attended. Scale = Total count.	**No relationship** Entity and incremental theorists did not differ in benefit of change $p > .05$. $d = 0.00$. **No relationship** Entity and incremental theorists did not differ in session attendance at 2-weeks $p > .05$. $d = 0.16$. **No relationship** Entity and incremental theorists did not differ in session attendance at 4-months $p > .05$. $d = 0.18$. **Beneficial effect** Entity theorists had greater session attendance at 2-weeks in the GMI group compared to the control group $p < .05$. $d = 0.61$. **No relationship** Entity and incremental theorists did not differ on session attendance between the GMI and control groups at 4-months $p > .05$. $d = 0.11$.

McClure (1999) PhD thesis. Prospective cohort study of US adults (N = 76) with a DSM-IV diagnosis of alcohol dependence. Recruited from inpatient, residential, and outpatient treatment programs in California, USA. Age (M = 39.20, SD = 9.08) Male (77.6%) Caucasian (73.6%) High school or college educated (60.5%)	Predictor label = "Belief in the disease model of addiction". Difference score. Instrument = Addiction Belief Scale (ABS) 18-items e.g. "The fact that alcoholism runs in families means that it is a genetic disease" with a 3-factor structure of power, dichotomous-thinking, and way-of-coping-with-life. Scale = 5-point Likert scale from "strongly disagree" to "strongly agree" with higher scores equating to greater disease model endorsement and lower scores to greater free-will model endorsement.	N/A	Outcome label = "Relapse status at 90 days post-treatment" Unadjusted. Instrument = Dichotomous relapse. Scale = Maintained abstinence or not. Instrument = Continuous relapse. Unadjusted. Scale = Post-treatment days of drinking, days of heavy drinking, and days to first drink. Outcome label = "Locus of control" Unadjusted. Instrument = Drink Related Internal-External (DRIE) 25-items e.g.: "As far as drinking is concerned, most of us are victims of forces we can neither understand or control". Scale = Forced choice between paired statements with only the external items scored.	N/A	**No relationship** Belief in the disease model was not correlated with dichotomous relapse $p > .05$. $r = .10$. **Iatrogenic relationship** Belief in the disease model was weakly positively correlated with post-treatment days of heavy drinking $p < .01$. $r = .33$. **No relationship** Belief in the disease model was not correlated with days of drinking $p > .05$. $r = .16$. **No relationship** Belief in the disease model was not correlated with days to first drink $p > .05$. $r = -.12$. **Iatrogenic relationship** Belief in the disease model was weakly positively correlated with participant external locus of control $p < .05$. $r = .24$. **Iatrogenic relationship** Belief in the disease model was weakly	Strong

			Outcome label = "Index of drinking severity". Unadjusted. Instrument = 13-items e.g.: "Experiencing shakes, loss of memory, previous detoxification". Scale = Total number of items endorsed.		positively correlated with the index of drinking severity $p < .01$. $r = .36$.	
Miller et al. (1996) Published article. Prospective cohort study of treatment engaged US adults (N = 122) recruited from outpatient services at the University of New Mexico Center on Alcoholism, Substance Abuse and Addictions (CASAA). Severity of alcohol dependence was assessed using the Alcohol Dependence Scale (ABS) with score cut-offs of 14-21 for moderate, 22-30 for substantial, and 31-47 for severe alcohol dependence. Age (M = 33.5)	Predictor label = "Belief in the disease model". Absolute score. Instrument = Understanding of Alcoholism Scale (UAS) disease model subscale 23-items e.g.: "If an alcoholic has a drink, he or she loses control and is unable to keep from getting drunk". Scale = 5-point Likert scale from "strongly disagree" to "strongly agree" with the higher total score indicating greater belief in the disease model of alcohol addiction.	N/A	Outcome label = "Relapse status". Unadjusted. Instrument = Ordinal categories of relapse status. Scale = 0. completely abstinent, 1. slipped (drinking after at least 4 days of abstinence), 2. relapsed (heavy drinking after at least 4 days of abstinence), and 3. continuous drinkers (no period of 4 or more days of abstinence).	N/A	**No relationship** Pre-treatment belief in the disease model of alcoholism was not correlated with relapse at 2-months $p > .05$. $r = .17$. **Iatrogenic relationship** Weakly positively correlated with relapse at 4-months, $p < .05$. $r = .23$. **Iatrogenic relationship** Weakly positively correlated with relapse at 6-months $p < .01$. $r = .25$. **No relationship** Not correlated with relapse at 8-months $p > .05$. $r = .21$.	Strong

Male (68.9%) Non-Hispanic white (51%) Unemployed (64%) ABS Scores (M = 21.5)					**No relationship** Not correlated with relapse at 10-months p > .05. r = .21. **No relationship** Not correlated with relapse at 12-months p > .05. r = .14. **Iatrogenic relationship** Disease model beliefs were assessed again at six months and found to be weakly positively correlated with relapse at 6-months p < .001. r = .37. **Iatrogenic relationship** Moderately positively correlated with relapse at 8-months p < .001. r = .41. **Iatrogenic relationship** Weakly positively correlated with relapse at 10-months p < .05. r = .29. **No relationship** Not correlated with relapse at 12-months p > .05. r = .19.	
Morphett et al. (2018) Published article.	Predictor label = "Beliefs about the role	N/A	Outcome label = "Desire to quit".	N/A	**Beneficial relationship**	Moderate

Cross-sectional cohort study of Australian adults (N = 1538) who were regular smokers i.e. smoke daily and have smoked at least 100 cigarettes in their lifetime. Recruited from a commercial online research panel. Age (M = 43.0, SD = 16.1) Male (54.0%) Born in Australia (75.3%) Non-university educated (68.7%) Cigarettes per day (M = 15.0, SD = 9.6)	of neurobiology in smoking" Absolute score. Instrument = Non-standardized 2-items e.g.: "Smoking changes the chemistry of the brain" and "Smoking is a brain disease". Scale = 4-point Likert scale from "strongly disagree" to "strongly agree" with scores split into a binary of "agreed" or "disagreed".		Unadjusted. Instrument = Non-standardized 1-item e.g.: "How much do you want to give up smoking?". Scale = 4-point Likert scale from "not at all" to "very much" with scores split into a binary of "not at all" or "to some extent". Outcome label = "Quitting self-efficacy". Unadjusted. Instrument = Non-standardized 1-item e.g.: "If you decided to give up smoking completely in the next six months, how sure are you that you would succeed?". Scale = 5-point Likert scale from "not at all sure" to "extremely sure" with scores split into a binary of "low" or "moderate/high".		Participants who agreed that smoking changes the chemistry of the brain had a higher desire to quit $p < .001$. Effect size not calculable due to no SD reported. **No relationship** Participants who agreed and disagreed that smoking changes the chemistry of the brain did not differ on quitting self-efficacy $p > 05$. Effect size not calculable due to no SD reported. **Beneficial relationship** Participants who agreed that smoking is a brain disease had higher desire to quit $p < .001$. Effect size not calculable due to no SD reported. **Beneficial relationship** Participants who agreed that smoking is a brain disease had higher quitting self-efficacy $p < .001$. Effect size not calculable due to no SD reported.	
West and Power (1995)	Predictor label = "Disease model".	Outcome label = "Personal	Outcome label = "Personal	**No relationship**	**No relationship**	Moderat e

Published article. Cross-sectional cohort study of UK adults (N = 61) attending inpatient and outpatient alcohol treatment unit in the south of England. Age under 45 (63%) Male (62%) Unemployed (51%) Previous treatment (69%) Alcohol use per week in units (M = 51.4)	Absolute score. Instrument = Non-standardized 2-items from an 18-item questionnaire e.g.: “Do you believe problem drinking is a disease?” Scale = 5-point Likert scale from “disagree” to “agree”.	responsibility for causing addiction”. Unadjusted. Instrument = Non-standardized 2-items from an 18-item questionnaire e.g.: “How far do you feel that you, personally, have been responsible for your drinking problems? Scale = 5-point Likert scale from “disagree” to “agree”.	responsibility for recovery”. Unadjusted. Instrument = Non-standardized 2-items from an 18-item questionnaire e.g.: “How far do you feel that solving your drinking problems is up to you?”. Scale = 5-point Likert scale from “disagree” to “agree”. Outcome label = “Value of treatment”. Unadjusted. Instrument = Non-standardized 3-items from an 18-item questionnaire e.g.: “Do you feel that recovery from problem drinking depends on good treatment?”. Scale = 5-point Likert scale from “disagree” to “agree”. Outcome label = “Importance of motivation”. Unadjsuted. Instrument = Non-standardized 2-items	Endorsement of the disease model was not correlated with personal responsibility for causing addiction $p > .05$. $r = .08$.	Endorsement of the disease model was not correlated with personal responsibility for recovery $p > .05$. $r = -.23$. **No relationship** Endorsement of the disease model was not correlated with valuing treatment $p > .05$. $r = .07$. **Iatrogenic relationship** Endorsement of the disease model was weakly negatively correlated with importance of motivation $p < .05$. $r = -.26$.

			from an 18-item questionnaire e.g.: "Do you feel that you could quit drinking if you made up your mind to?". Scale = 5-point Likert scale from "disagree" to "agree".			
Zeldman et al. (2004) Published article. Prospective cohort study of US adults (N = 74) attending an outpatient methadone maintenance program in the USA followed-up over 6 months. Age (M = 41.24, SD = 7.14) Male (51.4%) Caucasian (52.7%) Unemployed (78.4%) Years of Education (M = 12.11, SD = 2.30) Years of drug use (M 21.39, SD = 9.05) Days in treatment (M = 595.45, SD = 185.87)	Predictor label = "Belief that addiction is an entity i.e. a fixed part of the self". Absolute score. Instrument = Addiction Entitization Scale (AES) 7-items. Scale = 7-point Likert scale from "strongly disagree" to "strongly agree" with the higher total score indicating greater entity belief about addiction.	N/A	Outcome label = "Treatment adherence". Unadjusted. Instrument = Relapse. Scale = Percentage of random urine tests that are positive during treatment. Unadjusted. Instrument = Low attendance. Scale = Percentage of missed appointments at supportive services. Unadjusted. Instrument = Take-home status. Scale = Staff estimate of number of days until take-home methadone dose is achieved.	N/A	**No relationship** Greater entity belief did not predict relapse p > .05. r = -.24. **Iatrogenic relationship** Greater entity belief was significantly predictive of low attendance p < .01. r = -.28. **No relationship** Greater entity belief did not predict take-home status p > .05. r = -.23.	Strong

Table 4

Experimental studies

Study and sample characteristics	Manipulation of addiction beliefs	Measures indexing negative self-perception	Measures indexing recovery potential	Effects of disease beliefs on negative self-perception	Effects of disease beliefs on recovery potential	QA tool global rating
Burnette et al. (2019) Published article. Between-subjects study of US adults (N = 214) with probable substance dependence to alcohol and/or drugs assessed via the CAGE Adapted to Include Drugs (CAGE-AID). Recruited through Amazon's Mechanical Turk (MTurk) online web-survey service. Female (68%) Caucasian (75%) Age (M = 34.09, SD = 9.89)	Participants randomly assigned to one of two crafted newspaper style articles about addiction based on common media discourses. Disease-fixed model which combines the idea of addiction as a biomedical disease with the literature on mindsets to emphasize the fixed and unchanging nature of addiction. Compensatory-growth model which combines the view of addiction as a psychosocially influenced choice with the literature on mindsets to emphasize the malleability of addiction and the possibility of personal growth and change.	Outcome label = "Onset blame". Instrument = Non-standardized 1-item e.g.: "How responsible are you personally for becoming addicted to drugs/alcohol? That is, how much do you feel that your addiction is/was a result of choices you made, rather than something you can't control?". Scale = 7-point Likert scale from "not at all responsible" to "very responsible".	Outcome label = "Offset efficacy". Instrument = Non-standardized 6-items e.g.: "The harder I work at managing my addiction, the better I will be at it". Scale = 7-point Likert scale from "strongly disagree" to "strongly agree".	**No effect** The framing groups (disease-fixed vs. compensatory-growth) did not differ in onset blame $p > .05$. Effect size not calculable due to no M or SD reported.	**Iatrogenic effect** The disease-fixed group had significantly lower offset efficacy compared to the compensatory-growth group $p < .001$. $d = 0.38$.	Strong
Leonhard et al. (2022)	Participants randomly assigned to one of four	N/A	Outcome label = "Problem recognition"	N/A	**No effect**	Moderate

Published article. Between-subjects study of German adults (N = 488) with risky alcohol use assessed via AUDIT-C. In addition, direct or indirect (work or family) experience with alcohol use disorder (AUD) was assessed via yes/no questions. Recruited online using SoSciSurvey. Age 18-24 (66.1%) Female (77.1%) AUDIT-C (M = 2.99, SD = 2.14) Previous AUD experience (57.9%)	video vignettes about a person's alcohol use across two symptom-narrative conditions: continuum and dichotomous models. The dichotomous narrative emphasized alcohol use is an all or nothing problem meaning risky drinking and severe alcohol abuse are qualitatively different while the continuum narrative emphasized that alcohol use is a spectrum meaning risky drinking and severe alcohol abuse are qualitatively similar but quantitatively different.		Instrument = Stages of Change Readiness and Treatment Eagerness Scale (SOCRATES) 4-items emphasizing drinking severity from the ambivalence and recognition subscales e.g.: "There are times when I wonder if I drink too much". Scale = 5-point Likert scale from "strongly disagree" to "strongly agree".		Symptom-narrative groups did not differ in their level of problem recognition p > .05. d = 0.08.	
Lipkus et al. (2015) Published article. Between-subjects study of US adults (N = 128) who were light smokers i.e. smoke at least one cigarette per week but less than 5 cigarettes per day. This study was included as any amount of smoking is considered hazardous (WHO, 2013).	Participants exposed to neurobiological and genetic risk information about smoking and then randomly assigned to one of three groups regarding their genetic risk for nicotine dependence (based on rs16969968 allele): above average versus average risk.	N/A	Outcome label = "Abstinence self-efficacy beliefs". Instrument = Non-standardized 3-items e.g.: "How confident are you that you can stop smoking in the next month". Scale = 7-point Likert scale from "not at all" to "extremely".	N/A	**No effect** The average and above average risk groups did not differ in abstinence self-efficacy beliefs p > .05. Effect size not calculable due to no SD reported. **Beneficial effect** The above average versus average risk group reported greater desire to quit p < .05. d = 0.53.	Moderate

Recruited from seven college campuses in North Carolina, USA. Age (M = 18.95, SD = 0.99) Male (54.4%) White (60.1%) Cigarettes per day (M = 0.75, SD = 2.12)			Outcome label = "Desire to quit". Instrument = Non-standardized 1-item e.g.: "How strong is your desire to quit smoking right now?". Scale = 7-point Likert scale from "not at all" to "very". Outcome label = "Cessation at 1-month follow-up". Instrument = Non-standardized 2-items e.g.: "Have you smoked a cigarette, even a puff, during the last 30 days? If yes, have you smoked a cigarette, even a puff, during the last 7 days?". Scale = Yes/no response.		**No effect** The average and above average risk groups did not differ on 30 day cessation p > .05. Effect size not calculable due to no SD reported.	
Morris et al. (2020) Published article. Between-subjects study of UK adults (N = 597) with harmful alcohol use assessed via AUDIT-C scores of ≥8 for women and ≥9 for men. In addition,	Participants randomly assigned to one of three video vignettes about alcohol use across three belief-type conditions: control (personal account of alcohol problems), continuum model, and binary disease model.	N/A	Outcome label = "Problem recognition" Instrument = Stages of Change Readiness and Treatment Eagerness Scale (SOCRATES) recognition subscale 7-items e.g.: "My	N/A	**Iatrogenic effect** Harmful drinkers without addiction experience in the binary disease group reported significantly lower problem recognition compared to the	Strong

direct or indirect (work or family) addiction experience was assessed via yes/no questions. Recruited using Qualtrics survey software via Facebook and Twitter advertisements. Age (M = 37.21, SD = 13.58) Male (52.9%) British (89%)	Binary disease model refers to the belief that alcohol use and other related issues are dichotomous i.e. all or nothing problems. Continuum model refers to the belief that alcohol use and associated issues exist across a broad spectrum of severity.		drinking is causing a lot of harm". Scale = 5-point Likert scale from "strongly disagree" to "strongly agree".		continuum group p < .001. d = 0.82. **No effect** Harmful drinkers with addiction experience in the binary disease group did not differ from the continuum group on problem recognition p > .05. d = 0.06. **No effect** Harmful drinkers without addiction experience in the binary disease group did not differ from the control group on problem recognition p > .05. d = 0.33. **No effect** Harmful drinkers with addiction experience in the binary disease group did not differ from the control group on problem recognition p > .05. d = 0.20.	
Morris et al. (2022) Published article. Between-subjects study of UK adults (N = 244) with harmful alcohol use assessed via AUDIT-C scores of	Participants randomly assigned to one of six written vignettes about alcohol use across three belief conditions: control, continuum, and binary disease model, and two stigma	The presence or absence of stigmatizing language within the three belief-condition written vignettes.	Outcome label = "Problem recognition" Instrument = Stages of Change Readiness and Treatment Eagerness Scale (SOCRATES)	N/A	**Iatrogenic effect** Participants in the binary disease group exposed to stigmatizing language had significantly lower problem recognition	Strong

≥8 for women and ≥9 for men. In addition, direct or indirect (work or family) addiction experience was assessed via yes/no questions and only participants without addiction experience were included in the study. Recruited using Qualtrics survey software via Facebook and Twitter advertisements. Age (M = 29.98, SD = 16.93) Male (54%) British (96%)	conditions: with or without stigmatizing language. Binary disease model refers to the belief that alcohol use and other related issues are dichotomous i.e. all or nothing problems. Continuum model refers to the belief that alcohol use and associated issues exist across a broad spectrum of severity.	recognition subscale 7-items e.g.: “My drinking is causing a lot of harm”. Scale = 5-point Likert scale from “strongly disagree” to “strongly agree”.	compared to the stigma exposed continuum group $p < .05$. $d = 0.41$. **Iatrogenic effect** Participants in the binary disease group exposed to stigmatizing language had significantly lower problem recognition compared to the stigma exposed control group $p < .05$. $d = 0.25$. **Iatrogenic effect** Participants in the binary disease group exposed to stigmatizing language had significantly lower problem recognition compared to those in the binary disease group without stigmatizing language $p < .05$. $d = 0.34$. **No effect** Participants in the binary disease group without exposure to stigmatizing language did not differ from the non-stigma continuum group on problem recognition $p > .05$. $d = 0.01$. **No effect**

					Participants in the binary disease group without exposure to stigmatizing language did not differ from the non-stigma control group on problem recognition p > .05. d = 0.29.	
Waters et al. (2019) Published article. Between-subjects study of US adults (N = 392) who self-identified as smokers. Recruited from public locations in Saint Louis, Missouri, USA. Age (M = 44.3, SD = 13.2) Female (65.1%) White (52.9%) Vocational or lower education (51.6%)	Participants randomly assigned to receive one of two news-style articles about smoking: one about the genetics of smoking or another about pharmacies decisions to stop selling smoking products (control).	N/A	Outcome label = “Intentions to quit smoking”. Instrument = Non-standardized 1-item e.g.: “Are you planning to quit smoking?” Scale = 4-point Likert scale from “not planning to quit” to “in the next month”. Outcome label = “Self-efficacy of quitting smoking”. Instrument = “Smoking Self-Efficacy Questionnaire (SEQ-12) 12-items e.g.: “ability to refrain from smoking when celebrating something” Scale = 4-point Likert scale from “not at all sure” to “absolutely sure”.	N/A	**No effect** The genetic and control groups did not differ on intentions to quit p > .05. d = 0.19. **No effect** The genetic and control groups did not differ on self-efficacy p > .05. d = 0.06. **No effect** The genetic and control groups did not differ on worry about being unable to quit smoking p > .05. d = 0.13. **No effect** The genetic and control groups did not differ on perceived risk of having a gene making it harder to quit smoking p > .05. d = 0.10.	Strong

			Outcome label = "Worry about being unable to quit smoking". Instrument = Non-standardized 1-item e.g.: "How worried are you about being unable to quit smoking?". Scale = 4-point Likert scale from "no worry" to "a great deal of worry". Outcome label = "Perceived risk of having a gene making it harder to quit smoking". Instrument = Non-standardized 1-item e.g.: "How sure are you that you might have a gene that makes quitting harder?". Scale = 4-point Likert scale from "not sure" to "very sure".			
Wiens and Walker (2015) Published article. Between-subjects study of US adults (N	Between-subjects design with participants randomly assigned to read 15 addiction framing statements for either a disease model,	Outcome label = "Personal feelings of stigma and shame". Instrument = Perceived	Outcome label = "Personal perceptions of agency". Instrument = Free Will and Scientific Determinism Plus	**No effect** The framing groups (disease vs. psychosocial) did not differ on	**No effect** The framing groups (disease vs. psychosocial) did not differ on belief in free will $p > .05$. Effect size	Moderate

= 81) with mild to moderate alcohol problems assessed via the Modified Semi-Structured Assessment for the Genetics of Alcoholism II (SSAGA II). Recruited through Amazon's Mechanical Turk (MTurk) online web-survey service. Male (67%) Caucasian (79%) Age (M = 29.8, SD = 8.1)	psychosocial model, or neutral control (US geography). Disease and psychosocial models of addiction based on text extracts taken from two papers that advocate these models (Milam & Ketcham, 1981, for the disease model; Fingarette, 1988, for the psychosocial model).	Devaluation–Discrimination Scale (PDD) 12-items e.g.: "Most people would willingly accept someone with a former alcohol addiction as a close friend." Scale = 6-point Likert scale from "strongly disagree" to "strongly agree". Instrument = Self-Stigma of Mental Illness Scale (SSMIS) 40-items e.g.: "Because I have an alcohol addiction I cannot be trusted". Scale = 9-point Likert scale from "strongly disagree" to "strongly agree". Instrument = State Shame and Guilt Scale (SSGS) shame subscale 5-items e.g.: "I feel like I am a bad person".	(FAD-Plus) 27-items e.g.: "People's biological makeup determines their talents and personality". Scale = 5-point Likert scale from "totally disagree" to "totally agree". Instrument = Multidimensional Health Locus of Control (MHLC–Form C) 18-items e.g.: "I am directly responsible for my condition getting better or worse." Scale = 6-point Likert scale from "strongly disagree" to "strongly agree". Instrument = Brief Approach/Avoidance Coping Questionnaire (BACQ) 12-items e.g.: "I make an active effort to find a solution to my problems." Scale = 5-point Likert scale from "strongly disagree" to "strongly agree". Instrument = Controlled Drinking Self-Efficacy Scale	perceived discrimination p > .05. Effect size not calculable due to no M or SD reported. **No effect** The framing groups (disease vs. psychosocial) did not differ on self-stigma p > .05. d = 0.04. **No effect** The framing groups (disease vs. psychosocial) did not differ on shame p > .05. d = 0.11. **No effect** The disease and control groups did not differ on perceived discrimination p > .05. Effect size not calculable due to no M or SD reported. **Iatrogenic effect** The disease group had	not calculable due to no M or SD reported. **Iatrogenic effect** The disease group had significantly lower locus of control compared to the psychosocial group p < .05. d = 0.63. **No effect** The framing groups (disease vs. psychosocial) did not differ on coping style p > .05. Effect size not calculable due to no M or SD reported. **No effect** The framing groups (disease vs. psychosocial) did not differ on drinking self-efficacy p > .05. d = 0.43. **No effect** The framing groups (disease vs. psychosocial) did not differ on addiction entitisation p > .05. d = 0.51. **No effect** The disease and control groups did not differ on belief in free will p > .05. Effect size not

		Scale = 5-point Likert scale from “not feeling this way at all” to “feeling this way strongly”.	(CDSES) 20-items e.g.: “How confident are you that you can stop drinking alcohol at least three days a week?”. Scale = 11-point Likert scale from “0%” to “100%”. Instrument = Alcohol Addiction Entitisation Scale (AAES) 6-items e.g.: “You can learn new strategies, but you can’t really overcome your alcohol use/addiction” Scale = 6-point Likert scale from “strongly disagree” to “strongly agree”.	significantly higher self-stigma compared to the control group $p < .05$. $d = 0.64$. **No effect** The disease and control groups did not differ on shame $p > .05$. $d = 0.53$.	calculable due to no M or SD reported. **No effect** The disease and control groups did not differ on locus of control $p > .05$. $d = 0.27$. **No effect** The disease and control groups did not differ on coping style $p > .05$. Effect size not calculable due to no M or SD reported. **Iatrogenic effect** The disease group had significantly lower drinking self-efficacy compared to the control group $p < .05$. $d = 0.76$. **No effect** The disease and control groups did not differ on addiction entitisation $p > .05$. $d = 0.14$

Results

Database searches retrieved 1,198 studies after removal of duplicates. Of these, 1,168 were excluded at title and abstract screening with the most common exclusion reason that studies were irrelevant to the review question. Thirty papers were subjected to full-text review with 15 studies excluded because they included low or non-substance using participants (Cunningham et al., 1994; Heberlein et al., 2014; Henderson & Dressler, 2017; Klingemann et al., 2017; Lebowitz & Appelbaum, 2017; Racine et al., 2017; Rather, 1991; Ricardo et al., 2022; Wood, 2019), had no measure or manipulation of belief in a disease model of addiction (DeHardt, 2000; Jang et al., 2019; Sanderson & Wardle, 2005; Wartel, 2017), had no outcome measures linked to recovery potential (Hughes, 2009), or were theoretical papers (Ramanathan & Reischl, 1999). Reference list checks of the 15 eligible studies identified only a single study that was not already included in the review. This resulted in a total of 16 eligible studies being included in the systematic review.

A total of 77 effects were identified and extracted from the 16 studies using vote counting based on direction of effect (Table 5). 42 effects were extracted from the correlational studies, and 35 effects from the experimental studies.

Table 5

Total vote count of effects and their directions

Study design	Negative self-perception			Recovery potential		
	Nulls	Beneficial	Iatrogenic	Nulls	Beneficial	Iatrogenic
Correlational	1	0	0	19	10	12
Experimental	6	0	1	20	1	7
Total	7	0	1	39	11	19

Correlational studies

There were a total of nine studies identified with correlational data, six with prospective cohort designs, two with cross-sectional designs, and one RCT. The

studies took place mostly in the USA between 1989 and 2018 and included 2,342 participants (Mdn = 82, IQR = 48), the majority of whom were white (66.2%) male (64.6%) adults. Five of the studies had been published in peer-reviewed journals and the other four were PhD theses. Eight of the studies included treatment-engaged participants for mixed drugs and alcohol (four studies), alcohol (three studies), and heroin (one study). The remaining study included smokers. The overall quality of the studies was good with six studies rated strong and three as moderate. There was significant heterogeneity in study participants, measures of addiction belief, and outcomes related to negative self-perception and recovery potential.

Negative self-perception

There was only one outcome from a single study that related to negative self-perception (Table 6). The outcome indicated no relationship which supports the null hypothesis. However, making any kind of claim about the impact of the disease model on substance users' negative self-perception based on such limited evidence is questionable.

Table 6

Vote count of negative self-perception relationships by study quality rating

Quality rating	Negative self-perception		
	Nulls	Beneficial	Iatrogenic
Moderate	1	0	0
Strong	0	0	0

Recovery potential

A total of 41 outcomes were identified across the nine studies. Key outcomes included relapse status after various periods of follow-up, substance use severity, self-efficacy, treatment attendance, and treatment motivation. The relationships of disease beliefs to these outcomes were synthesised through vote counting based on significance and direction.

Nineteen outcomes indicated no relationship, 10 indicated a beneficial relationship, and 12 indicated an iatrogenic relationship. The most common finding was no relationship which might be true negatives, i.e., disease beliefs are not related to recovery potential, or false negatives, i.e., disease beliefs are related to recovery potential, but the study design or the measures employed were not optimised to detect this relationship. There were marginally more significant iatrogenic versus beneficial relationships with the advantage becoming more pronounced when effects were split according to study quality (Table 7). This complex mixture of findings should not be surprising given the significant heterogeneity between studies in terms of participants, index of addiction beliefs, outcomes, and analytical approaches. This field is clearly at its inception and more work is needed for confident conclusions to be drawn about disease beliefs and substance users' recovery potential.

Table 7

Vote count of recovery potential relationships by study quality rating

Quality rating	Recovery potential		
	Nulls	Beneficial	Iatrogenic
Moderate	3	6	1
Strong	16	4	11

Experimental studies

There were a total of seven experimental studies identified, all with between-subjects designs, that took place between 2015 and 2022 mostly in the USA, and included 2,154 participants (Mdn = 244, IQR = 269) the majority of whom were white (66.8%) female (54.6%) adults. All seven studies had been published in peer-reviewed journals. There was less heterogeneity in study participants compared with the correlational studies with five studies including risky alcohol users and two including smokers recruited from the general population. The overall quality of the

studies was good with four studies rated strong and three moderate. There was also slightly less heterogeneity in outcomes with three studies focusing exclusively on problem recognition for example.

Negative self-perception

A total of seven outcomes were identified across the seven studies. These outcomes included self-blame, perceived discrimination, shame, and self-stigma. The effects were synthesised through vote counting based on significance and direction. Six outcomes indicated no effect, zero outcomes indicated beneficial effects, and one outcome indicated an iatrogenic effect (Table 8). As with correlational studies, the nulls could either be true or false negatives.

Table 8

Vote count of negative self-perception effects by study quality rating

Quality rating	Negative self-perception		
	Nulls	Beneficial	Iatrogenic
Moderate	5	0	1
Strong	1	0	0

Recovery potential

With regards to recovery potential, a total of 28 outcomes were identified across the seven studies. Self-efficacy was the most common outcome in four studies, followed by problem recognition in three studies. Other outcomes included desire to quit smoking, smoking cessation rate, and treatment attendance. These effects were synthesised through vote counting based on significance and direction.

Twenty outcomes showed no disease framing effect, one outcome showed a beneficial effect, and seven outcomes showed iatrogenic effects (Table 9). Although the most common outcome was no effect, again, these could represent either true or false negatives. Focusing just on significant effects and comparing their frequency supports the iatrogenic over the beneficial hypothesis. This suggests that Heather

(2018) and Peele (2016) may be correct and Volkow and Koob (2015) wrong in their assertions, but this support for the iatrogenic hypothesis is limited. As with the correlational studies, more high-quality framing studies are required to determine with confidence whether disease discourse is harmful or beneficial for substance users' recovery potential.

Table 9

Vote count of recovery potential effects by study quality rating

Quality rating	Recovery potential		
	Nulls	Beneficial	Iatrogenic
Moderate	11	1	2
Strong	9	0	5

Discussion

The balance of significant effects identified in this review provide limited support for the iatrogenic hypothesis of BDMA critics (Heather et al., 2018; Peele, 2016) over the beneficial hypothesis of BDMA advocates (Volkow & Koob, 2015) on substance users' negative self-perception (one versus zero) and recovery potential (19 versus 11). However, null effects were by far the most common identified in this review for both negative self-perception (seven nulls versus one effect) and recovery potential (39 nulls versus 30 effects).

These findings are understandable given ambiguous conceptualisations of addiction as a disease. For example, biomedically-oriented research institutions, such as NIDA, promote a neuroscience-based disease model that involves "uncovering the molecular targets and circuits underlying addiction" and treating them with "effective medications" and "stimulation techniques such as transcranial magnetic stimulation (TMS)" (Volkow & Koob, 2015, pp. 677-678). A very different but equally influential disease model is promoted by 12-step mutual aid organisations. This spiritually-oriented version requires "accepting addiction as a

disease that can be arrested but never eliminated" and promotes a path to recovery by "enhancing individual maturity and spiritual growth, minimizing self-centeredness, and providing help to other individuals who are addicted" (Donovan et al., 2013, p. 315).

Further to these basic conceptual issues, different aspects of the ambiguous disease model were hypothesized to affect substance users' negative self-perception and recovery potential. Several studies in this review operationalized the disease model's active component as encouragement towards categorical over dimensional thinking. Examples include binary-disease versus continuum (Leonhard et al., 2022; Morris et al., 2020; Morris et al., 2022), disease-fixed versus compensatory-growth (Burnette et al., 2019), and entity versus incremental (Grand, 2001; Zeldman et al., 2004). In other studies, the active component was operationalized as freewill i.e. voluntary choice and control versus loss of control. The most common addiction belief measure identified in this review, the Addiction Belief Scale (Schaler, 1995), used this framing. Related to loss of control as the active ingredient in the disease model was the concept of locus of control and whether recovery from addiction comes from within or without. An internal locus of control was identified in two studies (McClure, 1999; Wiens & Walker, 2015) as being undermined by a disease model, yet in other studies the disease model, associated with an external locus of control, was associated with better treatment engagement (Aharon, 2000; Brzostek, 2000; Colon & Massey, 1989). Given these ambiguities about precisely what aspects of a disease concept are helpful or harmful to substance users' recovery, it is unsurprising that researchers have emphasised different constructs and have attempted to operationalize them heterogeneously.

Beyond these conceptual and methodological difficulties, it is arguable that finding mostly null results overall illustrates the power of ideology and politics in this area of science. The largest addiction research institutes in the world, NIAAA and NIDA, both strongly support the BDMA (A. Leshner, 1997; Volkow & Koob, 2015), and received $596.6 million (NIAAA, 2023), and $1,843 million (NIDA, 2023) respectively in research funding from the US congress in 2023. The situation is similar in the UK, albeit with significantly less financial clout. Between 2019 and 2021, biomedical addiction research received £4,909,745 from the Medical Research Council (MRC) compared to £177,750 for socioeconomic addiction research from the Economic and Social Research Council (ESRC), a 28 times difference in budget allocation (Hogarth, 2022).

This substantial funding bias towards medical and neurobiological research has been argued to arise from the "seductive allure of neuroscience" (Racine et al., 2017, p. 1) and the resulting scientific authority this provides irrespective of empirical justification. For example, Weisberg et al. (2015) found that the presence of irrelevant neuroscientific information made participants rate a poor explanation of psychological phenomenon as more satisfying compared to the same poor explanation without neuroscientific information. This has also been described as the "reductive allure" (Hopkins et al., 2016) within the sciences where disciplines are seen to exist in a hierarchy. This means that explanations from physics and chemistry are more fundamental than explanations from the social sciences, which are more superficial (Kaiser, 2011; Midgley, 2003). Within this paradigm, brain-based explanations are the "parts" that explain the "whole" of human behaviour. This gives neuroscientific models more scientific authority than psychological or sociological models. This has led some to conclude that neurobiological addiction theories like

the BDMA have been endorsed not because of their scientific validity, but simply because they secure more research funding (Room, 2021).

From this position of scientific authority, it is easy to see how disease model proponents could become inclined to make strong claims without empirical justification. The critical psychologist Thomas Teo provides a useful perspective regarding the construction of scientific knowledge and the dangers inherent in making strong claims to objectivity, often associated with more reductionist approaches to science:

> The belief that one can assume a point from nowhere, that history, culture, and society do not play a role in epistemic subjectivity, that "I" am objective, whereas others are not, may lead to a feeling of epistemic grandiosity, whereas the assumption that "my" knowledge is always fragile, even when "I" attempt to be objective, might inspire epistemic modesty. (Teo, 2019, p. 33)

Teo's concept of epistemic grandiosity provides a useful framework for understanding why the poorly supported claims this review explored may have occurred. Epistemic grandiosity is said to result from selected scientific observations being oversold as representing value-free objective truth. This grandiosity encourages interpretative speculation about scientific data in the human sciences to be presented as factual "knowledge" which is used explicitly or implicitly to construct an essentialised "Other" from the social groups such knowledge is about. These groups, such as people with addictions, then become stigmatised and marginalised in wider society (Zwick et al., 2020). This process of scientific knowledge contributing to social harms is labelled epistemic violence by Teo (2008).

Through the lens of epistemic grandiosity and violence, the counter-claims made by the critics of the BDMA can be viewed not as scientific criticism, but as

opposition to an ideologically dominant discourse that obscures the sociocultural and economic realities that contribute to addiction (Hart, 2017; Heyman et al., 2019; Hogarth, 2022; Shuai et al., 2022). The findings of this review now provide some very limited empirical support for these claims.

Limitations and future directions

A significant limitation of this review is the use of a vote-counting approach to synthesize the evidence. The interpreted ratio between significant and non-significant results is highly influenced by sample size variation and the differential statistical power across the included studies, and does not fully consider effect sizes.

A further limitation of the evidence synthesized in this review is the methodological difficulties associated with quantifying and operationalizing the nebulous concept of the disease model of addiction. This challenges whether quantitative methods are the best tools for understanding the role of substance users' beliefs about addiction in their recovery. More exploratory research using qualitative methods is needed to elaborate substance users' views on addiction to contribute to greater depth and breadth of theory generation. Quantitative methods may then be in a better position to answer more precise questions about the role of substance users' addiction theories on recovery processes, in much the same way the role of beliefs has been investigated in other areas of health (Jensen et al., 2021). Future quantitative research on the role of substance users' addiction theories on recovery may also be improved through within-subjects designs conducted in-person rather than by online survey. By having substance users act as their own controls, any contrasting effects between different conceptualizations of addiction on recovery-related outcomes are more likely to be detected, and collecting data directly from participants will improve its quality.

Another significant limitation of the evidence in this review is that the basic research assumptions underlying it are premised primarily on an academic debate about addiction aetiology that appears to have limited baring on substance users' recovery potential. This may represent a clash between academia and its focus on basic science and generating knowledge about underlying processes, and healthcare research and its applied science focus on generating knowledge that has clinical utility. An emerging area of research within the field of addiction that may provide a more fruitful direction in terms of clinical utility is recovery-informed theory (Brown & Ashford, 2019). This theoretical framework is an attempt to develop a science of recovery that positions the subjectivity and experiences of people with addictions as central to the research agenda. Its proponents argue that through "promoting the role of subjective recovery experience in the formulation of the study of recovery, it may be possible to summon new ideas, metrics, and strategies that can directly address substance use disorders in society" (p. 2). A shift away from a focus on problems and aetiology, towards a focus on solutions and recovery, may be the most promising direction for future research in the field of substance addiction.

Conclusion

This review aimed to investigate evidence for claims made by proponents and critics of the disease model of addiction. It collated and evaluated evidence of the 'beneficial hypothesis' expressed by proponents of the disease model which suggested that adoption of this understanding by substance users would decrease their negative self-perception and increase their recovery potential. It also collated and evaluated evidence of the 'iatrogenic hypothesis' expressed by critics of the disease model that argued substance users' adoption of this understanding would decrease their recovery potential and increase their negative self-perception. The

balance of significant effects identified in the review provided limited support to the iatrogenic hypothesis over the beneficial hypothesis for both negative self-perception and recovery potential. However, the most common finding overall was null effect.

These findings were interpreted in light of conceptual ambiguities in defining a disease model of addiction and methodological difficulties inherent in quantifying precisely what aspects of beliefs about the causes and nature of addiction are most pertinent to substance users' stigma towards themselves and their potential for recovery. Additionally, ideological driven epistemic grandiosity and associated economic incentives towards a more reductionist view of science were implicated, along with opposition to this trend.

Recommendations for future research into substance users' beliefs about addiction include a greater emphasis on qualitative methodologies alongside a shift towards within-subject in-person designs for quantitative research. Additionally, the focus on addiction aetiology is questioned, and a shift towards research that focuses on the processes, subjective and objective, that lead substance users towards recovery from addiction is advocated.

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Appendices

Appendix A – Search Strategy

Search Strategy for PsycINFO via Ovid:

#1. heroin.mp.
#2. cocaine.mp.
#3. amphetamine.mp.
#4. methamphetamine.mp.
#5. marijuana.mp.
#6. cannabis.mp.
#7. alcohol.mp.
#8. ketamine.mp.
#9. smoking.mp.
#10. tobacco.mp.
#11. nicotine.mp.
#12. (substance adj abuse*)
#13. 1 or 2 or 3 or 4 or 5 or 6 or 7 or 8 or 9 or 10 or 11 or 12
#14. addict*
#15. disease.mp.
#16. entity.mp.
#17. fixed.mp.
#18. binary.mp.
#19. compuls*.mp.
#20. 15 or 16 or 17 or 18 or 19
#21. belief*.mp.
#22. fram*.mp.
#23. model.mp.
#24. etiolog*.mp.
#25. 21 or 22 or 23 or 24
#26. blame.mp.
#27. stigma.mp.
#28. self-efficacy.mp.
#29. efficacy.mp.
#30. recovery.mp.
#31. relapse.mp.
#32. help-seek*.mp.
#33. recognition.mp.
#34. motivation.mp.
#35. 26 or 27 or 28 or 29 or 30 or 31 or 32 or 33 or 34
#36. systematic review
#37. meta-analysis
#38. review
#39. 36 or 37 or 38
#40. (13 and 20 and 25 and 35) not 39
#41. 14 and 40
#42. limit 41 to (human and english language)

Search Strategy for PubMed:

("heroin" OR "cocaine" OR "amphetamine" OR "methamphetamine" OR "marijuana" OR "cannabis" OR "alcohol" OR "ketamine" OR "smoking" OR "tobacco" OR "nicotine" OR "substance abuse*") AND ("disease" OR "entity" OR "fixed" OR "binary" OR compuls*) AND ("belief*" OR "fram*" OR "model" OR etiolog*) AND ("blame" OR "stigma" OR "self-efficacy" OR "efficacy" OR "recovery" OR "relapse" OR help-seek* OR "recognition" OR "motivation") NOT (systematic review[Publication Type] OR meta-analysis[Publication Type] OR review[Publication Type]) AND addict* AND (humans[Filter]) AND (english[Filter])

Search Strategy for Web of Science Core Collection:

(((((ALL=("heroin" OR "cocaine" OR "amphetamine" OR "methamphetamine" OR "marijuana" OR "cannabis" OR "alcohol" OR "ketamine" OR “smoking” OR “tobacco” OR “nicotine” OR “substance abuse*”)) AND ALL=("disease" OR "entity" OR "fixed" OR "binary" OR compuls*)) AND ALL=("belief*" OR "fram*" OR "model" OR etiolog*)) AND ALL=("blame" OR "stigma" OR "self-efficacy" OR "efficacy" OR "recovery" OR "relapse" OR help-seek* OR "recognition" OR "motivation")) NOT TI=(systematic review OR meta-analysis OR review)) AND ALL=(addict*)

Appendix B – Quality Assessment Tool for Quantitative Studies

QUALITY ASSESSMENT TOOL FOR QUANTITATIVE STUDIES

EPHPP
Effective Public Health Practice Project

COMPONENT RATINGS

A) SELECTION BIAS

(Q1) Are the individuals selected to participate in the study likely to be representative of the target population?

1 Very likely
2 Somewhat likely
3 Not likely
4 Can't tell

(Q2) What percentage of selected individuals agreed to participate?

1 80 - 100% agreement
2 60 – 79% agreement
3 less than 60% agreement
4 Not applicable
5 Can't tell

RATE THIS SECTION	STRONG	MODERATE	WEAK
See dictionary	1	2	3

B) STUDY DESIGN

Indicate the study design

1 Randomized controlled trial
2 Controlled clinical trial
3 Cohort analytic (two group pre + post)
4 Case-control
5 Cohort (one group pre + post (before and after))
6 Interrupted time series
7 Other specify _____
8 Can't tell

Was the study described as randomized? If NO, go to Component C.

No Yes

If Yes, was the method of randomization described? (See dictionary)

No Yes

If Yes, was the method appropriate? (See dictionary)

No Yes

RATE THIS SECTION	STRONG	MODERATE	WEAK
See dictionary	1	2	3

1

C) CONFOUNDERS

(Q1) Were there important differences between groups prior to the intervention?

1 Yes
2 No
3 Can't tell

The following are examples of confounders:

1 Race
2 Sex
3 Marital status/family
4 Age
5 SES (income or class)
6 Education
7 Health status
8 Pre-intervention score on outcome measure

(Q2) If yes, indicate the percentage of relevant confounders that were controlled (either in the design (e.g. stratification, matching) or analysis)?

1 80 – 100% (most)
2 60 – 79% (some)
3 Less than 60% (few or none)
4 Can't Tell

RATE THIS SECTION	STRONG	MODERATE	WEAK
See dictionary	1	2	3

D) BLINDING

(Q1) Was (were) the outcome assessor(s) aware of the intervention or exposure status of participants?

1 Yes
2 No
3 Can't tell

(Q2) Were the study participants aware of the research question?

1 Yes
2 No
3 Can't tell

RATE THIS SECTION	STRONG	MODERATE	WEAK
See dictionary	1	2	3

E) DATA COLLECTION METHODS

(Q1) Were data collection tools shown to be valid?

1 Yes
2 No
3 Can't tell

(Q2) Were data collection tools shown to be reliable?

1 Yes
2 No
3 Can't tell

RATE THIS SECTION	STRONG	MODERATE	WEAK
See dictionary	1	2	3

F) WITHDRAWALS AND DROP-OUTS

(Q1) Were withdrawals and drop-outs reported in terms of numbers and/or reasons per group?

1 Yes
2 No
3 Can't tell
4 Not Applicable (i.e. one time surveys or interviews)

(Q2) Indicate the percentage of participants completing the study. (If the percentage differs by groups, record the lowest).

1 80 -100%
2 60 - 79%
3 less than 60%
4 Can't tell
5 Not Applicable (i.e. Retrospective case-control)

RATE THIS SECTION	STRONG	MODERATE	WEAK	
See dictionary	1	2	3	Not Applicable

G) INTERVENTION INTEGRITY

(Q1) What percentage of participants received the allocated intervention or exposure of interest?

1 80 -100%
2 60 - 79%
3 less than 60%
4 Can't tell

(Q2) Was the consistency of the intervention measured?

1 Yes
2 No
3 Can't tell

(Q3) Is it likely that subjects received an unintended intervention (contamination or co-intervention) that may influence the results?

4 Yes
5 No
6 Can't tell

H) ANALYSES

(Q1) Indicate the unit of allocation (circle one)

community organization/institution practice/office individual

(Q2) Indicate the unit of analysis (circle one)

community organization/institution practice/office individual

(Q3) Are the statistical methods appropriate for the study design?

1 Yes
2 No
3 Can't tell

(Q4) Is the analysis performed by intervention allocation status (i.e. intention to treat) rather than the actual intervention received?

1 Yes
2 No
3 Can't tell

GLOBAL RATING

COMPONENT RATINGS

Please transcribe the information from the gray boxes on pages 1-4 onto this page. See dictionary on how to rate this section.

A	**SELECTION BIAS**	**STRONG**	**MODERATE**	**WEAK**	
		1	2	3	
B	**STUDY DESIGN**	**STRONG**	**MODERATE**	**WEAK**	
		1	2	3	
C	**CONFOUNDERS**	**STRONG**	**MODERATE**	**WEAK**	
		1	2	3	
D	**BLINDING**	**STRONG**	**MODERATE**	**WEAK**	
		1	2	3	
E	**DATA COLLECTION METHOD**	**STRONG**	**MODERATE**	**WEAK**	
		1	2	3	
F	**WITHDRAWALS AND DROPOUTS**	**STRONG**	**MODERATE**	**WEAK**	
		1	2	3	Not Applicable

GLOBAL RATING FOR THIS PAPER (circle one):

1	STRONG	(no WEAK ratings)
2	MODERATE	(one WEAK rating)
3	WEAK	(two or more WEAK ratings)

With both reviewers discussing the ratings:

Is there a discrepancy between the two reviewers with respect to the component (A-F) ratings?

No Yes

If yes, indicate the reason for the discrepancy

1	Oversight
2	Differences in interpretation of criteria
3	Differences in interpretation of study

Final decision of both reviewers (circle one):

1 STRONG
2 MODERATE
3 WEAK

Study	QA Tool	A - Selection Bias	B - Study Design	C - Confounders	D - Blinding	E - Data Collection Methods	F - Withdrawals & Drop-outs	*Global Rating of Study Quality*		
Aharon (2000)	Q1	2	Prospective cohort	1	2	1	1	*STRONG*		
	Q2	5		2	3	1	1			
	Rating	2	2	2	2	1	1			
Brzostek (2000)	Q1	2	Prospective cohort	1	2	1	1	*STRONG*		
	Q2	5		2	3	1	1			
	Rating	2	2	2	2	1	1			
Colon and Massey (1989)	Q1	1	Prospective cohort	2	3	3	1		*MODERATE*	
	Q2	1		1	3	3	1			
	Rating	1	2	1	2	3	1			
Grand (2001)	Q1	2	Randomized controlled trial	1	2	1	1	*STRONG*		
	Q2	1		1	2	1	1			
	Rating	1	1	1	1	1	1			
McClure (1999)	Q1	2	Prospective cohort	1	2	1	1	*STRONG*		
	Q2	5		1	2	1	2			
	Rating	2	2	1	1	1	2			
Miller et al. (1996)	Q1	2	Prospective cohort	1	2	1	1	*STRONG*		
	Q2	5		1	2	1	1			
	Rating	2	2	1	1	1	1			
Morphett et al. (2018)	Q1	2	Cross-sectional cohort	1	2	1	1		*MODERATE*	
	Q2	2		1	2	1	1			
	Rating	2	3	1	1	1	1			
West and Power (1995)	Q1	2	Cross-sectional cohort	1	2	1	1		*MODERATE*	
	Q2	2		1	2	1	1			
	Rating	2	3	1	1	1	1			
Zeldman et al. (2004)	Q1	2	Prospective cohort	1	2	1	1	*STRONG*		
	Q2	5		1	3	1	1			
	Rating	2	2	1	2	1	1			

Study	QA Tool	A - Selection Bias	B - Study Design	C - Confounders	D - Blinding	E - Data Collection Methods	F - Withdrawals & Drop-outs	*Global Rating of Study Quality*		
Burnette et al. (2019)	Q1	1	Between-subjects	1	2	1	1	*STRONG*		
	Q2	1		1	2	1	1			
	Rating	1	1	1	1	1	1			
Leonhard et al. (2022)	Q1	2	Between-subjects	2	2	1	1		*MODERATE*	
	Q2	3		1	2	1	2			
	Rating	3	1	1	1	1	2			
Lipkus et al. (2015)	Q1	1	Between-subjects	3	2	1	1		*MODERATE*	
	Q2	2		4	2	1	1			
	Rating	2	1	3	1	1	1			
Morris et al. (2020)	Q1	1	Between-subjects	1	2	1	1	*STRONG*		
	Q2	1		1	2	1	1			
	Rating	1	1	1	1	1	1			
Morris et al. (2022)	Q1	2	Between-subjects	1	2	1	1	*STRONG*		
	Q2	1		1	2	1	1			
	Rating	1	1	1	1	1	1			
Waters et al. (2019)	Q1	1	Between-subjects	1	2	1	1	*STRONG*		
	Q2	1		1	2	1	1			
	Rating	1	1	1	1	1	1			
Wiens and Walker (2015)	Q1	1	Between-subjects	1	2	1	1		*MODERATE*	
	Q2	1		1	2	1	3			
	Rating	1	1	1	1	1	3			

EMPIRICAL STUDY

The impact of explanatory theories of addiction on the perceived recovery potential of treatment-engaged substance users

Trainee: Daniel Casey

Primary Supervisor: Professor Lee Hogarth, Associate Professor of Experimental Psychology, School of Psychology, Faculty of Health and Life Sciences, University of Exeter

Secondary Supervisor: Dr Fiona Warren, DClinPsy Research Director, School of Psychology, Faculty of Health and Life Sciences, University of Exeter

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Abstract

Significant time, effort, and financial resources are invested into neurobiological addiction research, influenced by the seductive allure of theories like the brain disease model of addiction (BDMA). Critics of this approach suggest it has not delivered effective treatments and argue that such research *about* substance users results in *epistemic violence*, turning substance users into “neurobiological others” which both marginalises them from wider society and undermines their potential for recovery. Alternative addiction theories attempt to broaden the research focus towards psychological and social factors, promoting a more social justice approach to addiction.

This study investigated whether emphasising biological over social causal factors in addiction has a negative impact on substance users’ potential for recovery, with the exception of reducing self-blame. A within-subjects design was used to explore the impact of simplified biological and social addiction theories on ten outcome variables that tapped constructs linked to participants perceived recovery potential from addiction. Participants were 34 working age adult service users from a drug and alcohol service in Southern England, UK.

The study hypotheses were mostly supported with worse scores on measures of therapeutic alliance, social support, recovery optimism, belief in the probability of relapsing, and self-predicted substance use, and a better score on the measure of self-blame under the framing of the biological compared to the social addiction theory. No support was found for any difference between the two addiction theories on measures of self-efficacy and coping, resilience, treatment motivation, or negative affect about self.

These findings were interpreted using the mixed blessing model of biogenetic explanations (Haslam & Kvaale, 2015) which suggests blame is reduced through attributions of uncontrollability at the cost of increasing prognostic pessimism through psychological essentialism. By contrast, the social theory presented in the study offered an inversed mixed blessing, increasing prognostic optimism at the cost of increasing participants blame towards themselves. The inclusion of genetic vulnerability within the biological theory presented in the study may be the reason such a mixed blessing was found (Loughman & Haslam, 2018).

The results of the study provide support for the value of in-depth exploration of substance users understandings of their addiction as a critical aspect of treatment. In clinical psychology, professional and service user knowledge and experience can be integrated into formulations of addiction that emphasise “what's happened to you?” over “what's wrong with you?” (Harper & Cromby, 2022), and suggest pathways to recovery that fit with substance users’ needs and capabilities. This emphasis on shared understanding and collaboration is a direct challenge to scientific knowledge generated *about* substance users and instead promotes scientific knowledge that is both *for* and *from* those struggling with addiction. Such an ethical position is essential in the human sciences generally, but is particularly important in the applied science of clinical psychology and related mental health professions. It is hoped that the findings of this study will be of use to those struggling with addiction and the people supporting them on their journey towards recovery.

Introduction

Addiction is characterized by powerful urges to engage in behaviours that produce experiences of reward despite negative consequences (NHS, 2021). Addictive behaviours typically involve consumption of substances like alcohol but also other behaviours such as gambling. Irrespective of the involvement of substances, as an issue of behavioural health, recovery from addiction requires behaviour change and this is influenced by the beliefs a person holds about their addiction. However, the beliefs people hold about the causes and nature of addiction can be quite varied because, despite being relatively simple to understand on the surface, addiction is causally multifactorial i.e. biopsychosocial in nature (Marlatt et al., 1988). This makes it conceptually complex and challenging to explain (Alexander, 2023; Shaffer et al., 2004) so scientific accounts simplify the complexity by focusing on some factors to the neglect of others leading to a multiplicity of theories emphasising different aspects of biopsychosocial causation (Green et al., 2021).

The biomedicalization of addiction

The most widely adopted explanatory theory of addiction is the brain disease model (BDMA) (A. I. Leshner, 1997; Volkow & Koob, 2015) which is championed by the National Institutes of Drug Abuse (NIDA) and Alcohol Abuse and Alcoholism (NIAAA) in the United States. NIDA and NIAAA fund the majority of addiction research in the world and had annual budgets of $596.6 million (NIAAA, 2023) and $1,843 million (NIDA, 2023) in 2023. The NIDA website provides an succinct summary of the BDMA which defines addiction as “a chronic, relapsing disorder characterized by compulsive drug seeking and use despite adverse consequences

[and] a brain disorder, because it involves functional changes to brain circuits involved in reward, stress, and self-control." (NIDA, 2020).

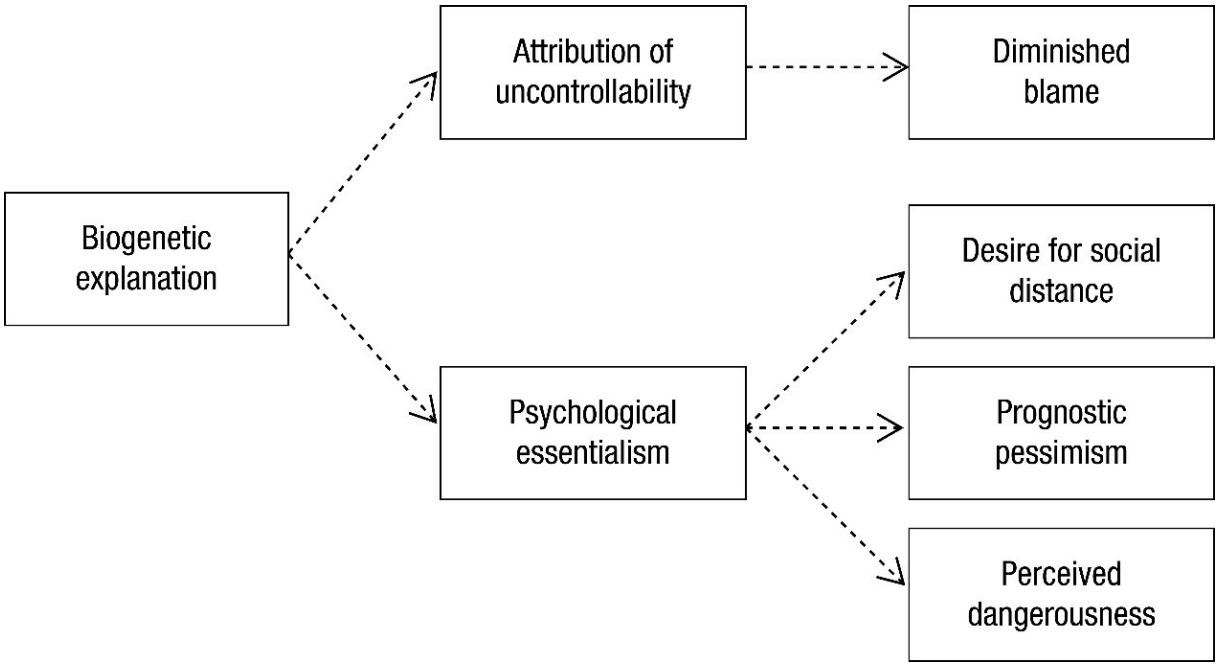
Though widely endorsed and well-funded, the BDMA is not without its critics (Hall et al., 2015a, 2015b; Heather et al., 2022), and can be said to perform "cultural work" (Hammer et al., 2013, p. 2) beyond its clinical application to addiction research and treatment. Buchman et al. (2011) argue that despite intentions to improve treatment, reduce stigma, and promote compassion, the BDMA and its characteristic emphasis on neuroscientific explanation leads the public to view people struggling with addiction as "neurobiological others". For example, Kelly et al. (2021a) found that describing a person with substance use problems as having a 'chronically relapsing brain disease' decreased prognostic optimism, and increased the perceived need for continuing care and danger compared to describing the same person's substance use as a 'problem'. Nick Haslam (2011) argues that findings like this arises from media portrayals of the neuroscience of mental health problems (including addiction), which lead the public to essentialise addiction and other problems i.e. attribute them to individuals' biological and psychological constitution. This results in the discrimination and marginalisation of vulnerable people but also, when those struggling with addiction essentialise themselves, undermines the very self-efficacy they need to recover from addiction (Peele, 2016).

Research from the broader field of mental health about the relationship between biogenetic explanations and stigma provides some support to these criticisms, but also suggests a more complex picture. Based on the findings from two meta-analyses that reviewed how people with mental health problems are viewed by the public, by themselves, and by clinicians (Kvaale, Gottdiener, et al., 2013; Kvaale, Haslam, et al., 2013), Haslam and Kvaale (2015) described biogenetic explanations

as a mixed blessing (Figure 1). In their theoretical review they appeal to attribution theory (Weiner, 1993) to suggest that people are more sympathetic, to themselves and to others, when harmful behaviours are perceived as uncontrollable. This attribution of uncontrollability is then reinforced by neuroscience-based mechanistic explanations which diminish blame by undermining the notion of free will (Shariff et al., 2014). However, the mixed blessing model proposes that attributions of uncontrollability and mechanistic thinking also lead to psychological essentialism. This is the belief that fixed, hidden, and identity-based properties, like DNA or brain structure, generate an observed behaviour or characteristic. This essentialized perspective is argued to mediate the relationship between biogenetic explanations and increased prognostic pessimism (and desire for social distance and perceived dangerousness from the perspective of others).

Figure 1

The mixed blessing model (Haslam & Kvaale, 2015)



The purpose of explanatory theories

Discussing biomedical addiction theories and the criticism they provoke begs the question, what do we want addiction theories to do? The answer of course depends on who you're asking. For addiction scientists, the answer is a comprehensive explanatory account of the causality of addiction that can attract research funding, lead to high impact publications, and ultimately achieve impact on real world outcomes. For substance users, an assumed answer might be to know how to recover from addiction. For clinicians/treatment providers, the answer is likely to mirror substance users' in wanting to know how to be effective in supporting their recovery. This implies that explanatory theories are more reflective of values and priorities than they are scientific truth.

Within the field of psychological science, Barbara Held (2020) describes how psychological knowledge is often constructed *about* people and argues that this opens the door to the objectification and othering of specific groups of people such as those struggling with addiction. She contrasts this with knowledge that is constructed *for* and *from* people and how this approach makes the values underlying knowledge creation more explicit and makes the knowledge itself more useful to people. Although the natural sciences seek to practice objectivity, the knowledge generated by researchers will always come from a culturally-influenced perspective with unacknowledged norms and values (Midgley, 2003). Unless these biases are recognised, the generation of scientific knowledge could lead to social harms or waste, with a mere selection of favoured observations being reported as an overarching narrative (Bishop, 2020). Thomas Teo (2019) coined the term *epistemic grandiosity* to describe this tendency of selected observations being oversold as representing value-free "objective truth". He argues that such grandiosity about

scientific knowledge can lead to social groups such knowledge is *about* becoming essentialised and marginalised. He calls this process of scientific knowledge contributing to social harm *epistemic violence* (Teo, 2008). Specifically, *epistemic violence* refers to the way interpretative speculation about scientific data in the human sciences is presented as factual "knowledge" which is then used explicitly or implicitly to construct an "Other" who is negatively impacted by such "knowledge". The case par excellence for this is scientific race theory and its use in the justification of slavery and racist political policies with a legacy that still profoundly impacts the world today (Bryan et al., 2022; Ward, 2022).

Returning to the BDMA, a great deal of the criticism levelled at this theory can be framed in terms of *epistemic violence* through the creation of "neurobiological others" (Buchman et al., 2011) out of people struggling with addiction through essentialising their difficulties as being primarily about their biological constitution (Haslam & Kvaale, 2015). This can also be seen as a form of *epistemic grandiosity* that contributes to social injustice (Hart, 2017) and iatrogenic harms (i.e. harmful medical interventions) by "undercut[ing] the self-efficacy required to achieve freedom from addiction" (Peele, 2016).

Alternative theories of addiction

There are many alternative explanatory theories that emphasise other aspects of the biopsychosocial causal continuum. For example, Lewis (2015; 2017), Heyman (2013; 2021), and Peele (2016; Peele et al., 1991) all propose very similar views based on behavioural psychology that, far from being an illness or disease, addiction is a normal part of human experience albeit at the extreme ends. These de-pathologizing perspectives suggest that an individual's ability to overcome addiction depends on developing coping strategies, pursuing values and purpose, repairing

relationships, and expressing personal agency – all of which are a normal part of being human.

Other theories go beyond the individual and use epidemiological research on social determinants to reveal how exposure to a wide range of social adversities increases the risk of addiction (Marmot, 2015) and addiction-related harms (Boyd et al., 2022; Heyman et al., 2019). For example, Alexander's (2000, 2023) sociological perspective argues that what we call addiction is in fact an adaptive response to the global expanse of western free-market societies which dislocate people from their traditional sources of psychological, social and spiritual support. He points to the high prevalence of addiction in indigenous societies subject to colonisation (Urbanoski, 2017) and in western societies (Mounteney et al., 2016) as evidence for this "adaptive paradigm".

Additionally, Hogarth (2022) argues that the choice to continue using substances or engage in addictive behaviours is powerfully motivated by socioeconomic deprivation-related factors (Shuai et al., 2022) significantly increasing the value of addictive substances or behaviours relative to other sources of reward. A very similar perspective is presented by Acuff and colleagues (2023) who emphasise the contextual-dependence of the reinforcing nature of addiction i.e. ease of access to addictive substances in the environment versus other healthier sources of reward, and whether people have the means to engage in these healthier activities. These socially-motivated choice theories of addiction broaden the view of addiction beyond the individual and promote a stance of social justice by emphasising inequity and suggesting the need for intervention at a broader societal level (Engemann et al., 2019).

Addiction framing studies

Within the field of healthcare, the impact of beliefs on behaviours has been established in conditions such as chronic pain where beliefs about pain implying damage to the body are associated with higher physical disability (Jensen et al., 1994) and challenging these beliefs through cognitive-behavioural therapy improves symptoms (Jensen et al., 2021). In the case of substance addictions, increased belief in the efficacy of harm reduction strategies can decrease alcohol consumption in young people (De Leon et al., 2023), and smokers have a greater likelihood of quitting if they have greater self-efficacy, believe smoking-related diseases are more severe, and believe that there are fewer barriers to quitting (Kaufman et al., 2018).

In the last decade researchers have begun to explore substance users beliefs about the causes and nature of addiction and test empirically whether the theories adopted by substance users have an impact on metrics of recovery and self-stigma. These studies use framing designs that compare biological (i.e. disease, genetic, medical, neurobiological) and psychological/social-based addiction theories on various clinically relevant constructs linked to recovery and self-stigma in different populations.

Wiens and Walker (2015) conducted an online between-subjects study of US adults (N = 81) with mild to moderate alcohol problems who were randomly assigned to read 15 addiction framing statements for either a disease model, psychosocial model, or neutral control (US geography). Personal feelings of stigma and shame were measures using scales of perceived discrimination, self-stigma, and shame. The control condition was found to have lower self-stigma compared to both the disease ($p < .05$) and psychosocial groups ($p < .05$), and the psychosocial group was found to have higher shame compared to the control group ($p < .05$). No other

statistically significant differences were found between the conditions. Personal perceptions of agency were measures using scales of free will, locus of control, approach/avoidance coping, drinking self-efficacy, and addiction entitisation i.e. the idea that addiction is categorical. The psychosocial condition was found to have higher locus of control compared to both the disease ($p < .05$) and control groups ($p < . 05$). The disease group was found to have lower drinking self-efficacy compared to the control group ($p < .05$). Finally, the psychosocial group was found to have lower addiction entitisation compared to the control group ($p < .05$). No other statistically significant differences were found between the conditions. The findings indicate that a disease framing has some benefits in reducing shame but at the cost of reducing perceptions of agency.

Burnette et al. (2019) conducted another online between-subjects study of US adults (N = 214) with probable substance dependence to alcohol and/or drugs who were randomly assigned to one of two crafted newspaper style articles based on common media discourses about addiction. Their disease-fixed model combined a biomedical view with the mindset literature to emphasize the fixed and unchanging nature of addiction. Their compensatory-growth model combined a psychosocially influenced choice view with the mindset literature to emphasize the malleability of addiction and the possibility of personal growth. They measured blame for the onset of addiction and efficacy for offsetting addiction i.e. confidence in recovery, using non-standardized items. They found no statistically significant difference in onset blame between conditions but found the disease-fixed group had lower offset efficacy compared to the compensatory-growth group ($p < .001$). Additionally, they found that the compensatory-growth group had higher intentions to pursue counselling ($p = .006$) and CBT ($p = .009$) compared to the disease-fixed group but

neither condition differed on intentions to pursue pharmacological treatment. The findings show that alternatives to a disease framing can improve self-efficacy without also increasing blame.

Morris et al. (2020) conducted an online between-subjects study of UK adults (N = 597) assessed for harmful alcohol use and addiction experience who were randomly assigned to one of three video vignettes that described alcohol misuse as a continuum (existing across a broad spectrum of severity), a binary-disease (an all or nothing problem), or control (personal account of alcohol problems). Problem recognition was measured for each condition and participants in the binary-disease group who were harmful drinkers without addiction experience were found to have lower problem recognition compared to those in the continuum group ($p < .001$). Morris et al. (2022) followed this up with another online between-subjects study of adult harmful alcohol users without addiction experience (N = 244) who were randomly assigned to one of six written vignettes about alcohol use across continuum, binary-disease, and control conditions with or without stigmatizing language. They found that those in the binary-disease group exposed to stigmatizing language had lower problem recognition compared to both the stigmatized continuum ($p < .05$) and control groups ($p < .05$) along with the non-stigmatized binary-disease group ($p < .05$). These findings suggest that a disease framing leads those who may be at risk of addiction to deny the significance of their substance use, an effect that is strengthened by stigma.

These studies provide some evidence, albeit inconsistent, that biological framings can be harmful to substance users' sense of agency and problem recognition, and in the context of stigma, contribute to harmful substance users denying the extent of their problems. However, there is a lack of consistency in how

biological theories have been defined with terms such as essentialist, genetic, and disease being used across studies alongside a mix of alternative theories i.e. psychosocial, compensatory-growth, and continuum models. This makes it difficult to know precisely what aspects of theories are most influential on substance users. The studies also focused mostly on risky alcohol users so we do not know whether any of their findings translate to more severely addicted substance users who use different substances and are engaged in services. Additionally, all the studies used between-subjects designs making it more challenging to detect effects due to natural variation between participants.

Despite these issues there is enough evidence to suggest that biologically-focused theories are likely to be harmful to treatment-engaged substance users and that other addiction theories may have benefits in supporting their recovery. Conversely, there is evidence to suggest that biologically-focused theories may have some limited benefit regarding substance users self-blame. This gap in the literature was the focus of his study which aimed to address it through use of a within-subjects design that contrasted clearly defined addiction theories and measured the impact of these on plausible indices of recovery potential in people experiencing more severe forms of substance addiction who were engaged with services.

Study aim

The aim of this study was to test whether biologically-focused theories, like the BDMA, are more harmful to recovery processes than socially-focused theories to treatment-engaged substance users, with the exception of self-blame. To answer this question we ran an experimental study using a within-subjects design with working age adult service users from a drug and alcohol service in Southern England, UK.

We presented biological and social addiction theories to participants (in counterbalanced order within-subjects) and asked them to imagine attending a group early in their recovery journey where they would be discussing each of these theories. We then measured their level of agreement with each theory along with their perceived recovery potential within each imaginary therapeutic group. Ten single-item measures tapped into different constructs linked to their perceived recovery potential within those groups: (1) facilitator support, (2) peer/member support, (3) recovery optimism, (4) confidence to solve problems, (5) relapse likelihood, (6) quitting effort, (7) resilience to difficulties, (8) self-blame, (9) negative affect abut self, and (10) self-predicted substance use. By conducting this study our hope was to generate knowledge *for* and *from* people directly impacted by addiction.

Study hypothesis

The main study hypotheses were that scores on the measures of perceived recovery potential would be worse under the framing of the biological theory of addiction compared to social theory of addiction, with the exception of self-blame which would be better. We predicted facilitator support, peer/member support, recovery optimism, confidence to solve problems, quitting effort, and resilience to difficulties to have lower scores, and relapse likelihood, negative affect about self, and self-predicted substance use to have higher scores in the biological theory condition compared to the social theory condition. We predicted self-blame would not follow this pattern and instead have higher scores in the biological theory condition compared to the social theory condition

No additional hypotheses were made, but moderating factors of the hypothesised main effects were explored including participants' level of agreement with addiction theories, and demographic and treatment history variables.

Methods

Research assumptions

The philosophy of science underpinning this study is critical realism (Pilgrim, 2020), with its axioms of ontological realism and epistemological relativism. Ontological realism is the idea that the world exists independently of human consciousness. Epistemological relativism asserts that human knowledge is mediated through concepts and language in interaction with others, making our understanding of the world indirect and value-laden.

Reflexivity

I approached this research from the position of having alcohol addicted grandparents which has strongly influenced my desire to understand addiction in greater depth and conduct research that aims to be useful for people experiencing addiction. It was important that I did not let my personal experience obscure the very real power differential between myself and those who participated in my research who were some of the most marginalized people in society. As a trainee clinical psychologist, I was in a position of power to influence the perspectives of participants and I had a duty of care to ensure I respected participants' views. To manage this, I consulted the drug and alcohol service's co-production group about the study design and ensured that I presented the two views of addiction within the study as simplifications, emphasizing that the study was about knowing which of the views, if any, were useful to the participant and not about either of them being right or wrong.

Design

This study was exploratory in nature with its main goal to determine whether different explanatory theories differentially impact the perceived recovery potential of treatment-engaged substance users'. A quantitative methodological framework was

chosen to achieve this goal because it allowed for comparisons to be made between addiction theories and inferences to be drawn about whether they led to differences on measures of substance users' perceived recovery potential. It also allowed for associations to be explored between substance users' level of agreement with addiction theories and different aspects of their perceived recovery potential.

Due to the complexity of different addiction theories, and evidence that the public (Meurk, Carter, Hall, et al., 2014), and addiction scientists and clinicians (Bell et al., 2014) hold a complex mixture of views, we developed two simplified theories and named them the biological view and the social view to participants. This decision was based on the tendency for theories to lean towards either biological accounts that emphasise genetic vulnerabilities and structural and functional neurobiological changes, or social accounts which variously emphasise socioeconomic deprivation, interpersonal trauma, learned coping strategies, and cultural and political contexts.

A within-subjects design was used for the study with one independent variable of addiction theory (biological vs. social) and ten dependent variables consisting of single-item outcome measures of participants perceived recovery potential. This design was chosen to reduce the necessary sample size given the difficulties recruiting participants from substance using populations, it also had the added benefit of reducing natural error associated with individual differences. Further, some of the drawbacks of this design, such as participant fatigue and order effects, were mitigated against by having the presentation of the explanatory theories counterbalanced between participants and through keeping the study procedure as short as possible (30-minutes). Increased liability for demand characteristics was managed by explicitly briefing participants that people differ on which kind of addiction theories they find helpful and that this research was about determining

which, if any, of the two views being presented to participants was helpful to them or not.

Procedure

Ethical approval

In accordance with ethical guidelines, approval (Ref: 1003462) was obtained through the School of Psychology ethics committee at the University of Exeter (Appendix A). The approved ethics application was then provided to the drug & alcohol service's research panel who gave their own approval for the study to proceed.

Participants and recruitment

The study took place at a drug & alcohol service in Southern England, UK. The organisation is the main provider of substance misuse services in the county and has several regional hubs. A total of 34 people with lived experience of substance use problems participated in the study. This sample size was calculated a priori through a power analysis using an α of 0.05, a β of 0.20, and a predicted effect size (*d*) of 0.50 which was estimated based on studies with comparable designs (Appendix B).

Inclusion criteria for the study were (1) aged 18 or over, (2) able to understand and speak English without an interpreter, and (3) is currently in treatment for alcohol and/or drug (prescription, illegal or combination) use or is in recovery but has remained engaged in the service as a peer mentor or volunteer to maintain their recovery. Exclusion criteria included (1) being intoxicated at the time of the study determined by asking participants directly and visible signs such as poor motor coordination, physical agitation, bloodshot eyes, dilated pupils, and/or inability to follow researcher instructions, and (2) a peer mentor or volunteer that had never

received treatment for alcohol and/or drug use i.e. recovered without formal treatment.

Recruitment took place between March and November 2023 using an opportunistic sampling strategy at the drug and alcohol service's Exeter and Newton Abbot hubs, in community centres in Axminster and Tiverton, and from online referrals from recovery workers. The first stage of recruitment involved the distribution of participant information sheets (Appendix C) and consent forms (Appendix D), in written (paper and digital) and audio formats, to team leaders for them to circulate amongst recovery workers. Staff then identified potential participants from among their service users and gained consent to pass on their contact details. The second stage involved me attending several staff team meetings to promote the study and encourage workers to identify suitable participants. In the third stage I attended drop-in cafés and recovery groups at the service's regional hubs and community centres and recruited from amongst the attendees. Participants were given the option to engage in the study in-person or online via Microsoft Teams in order to maximise inclusivity and provide sufficient flexibility to ensure the required sample size was achieved.

Data collection and measures

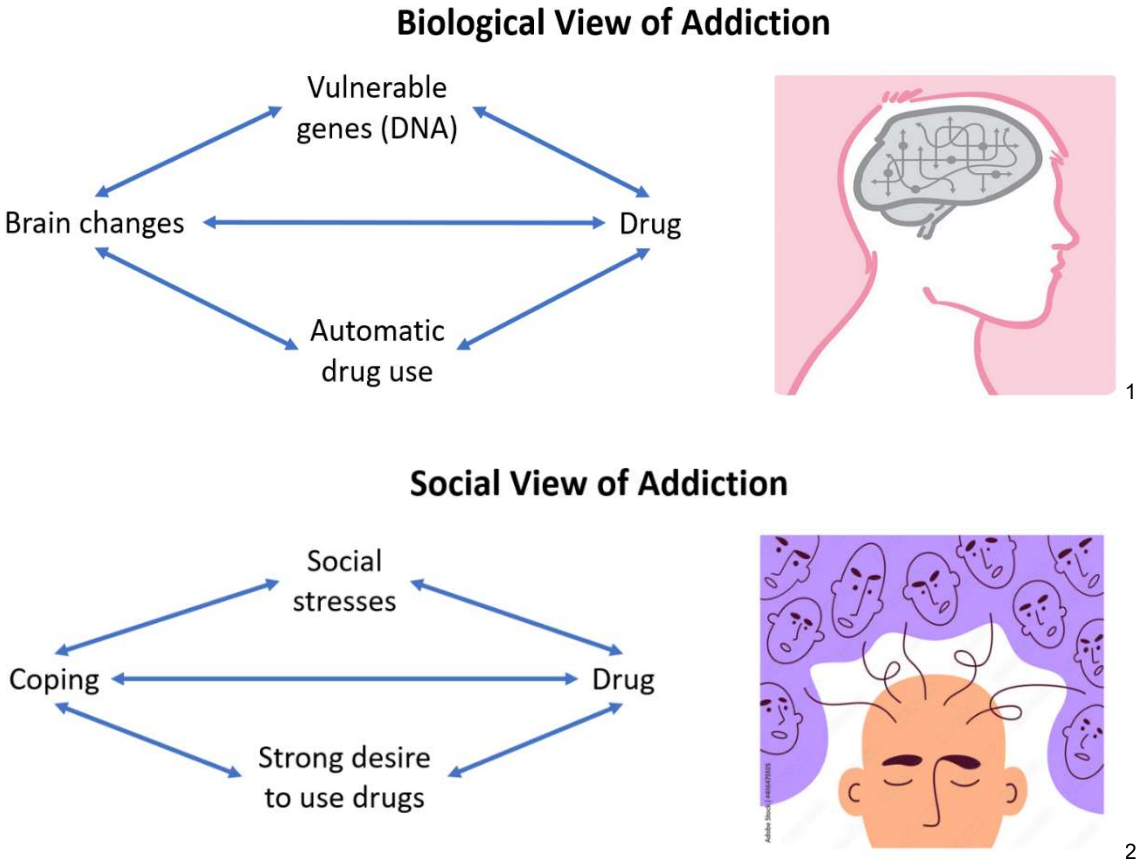
At the beginning of the study I confirmed participants had read or listened to the information sheet and consent form and responded to any questions they had. Once I was clear they understood the nature of the study I gathered their signed consent form (either physical or digital) and then briefed them about study rationale and that I would be exploring two common scientific views of addiction and asking which, if any, they would find most helpful at the beginning of their recovery journey. The full transcript for the study procedure can be found in Appendix E.

I went through a set of questions to gather demographic and background data which were developed to align with data collected routinely as part of the National Drug Treatment Monitoring System (Office for Health Improvement & Disparities, 2023). I asked each participant their age, gender, ethnicity, which substance/s they were in treatment for/recovery from, how long they had been receiving support from the drug and alcohol service, how many days in the past four weeks they had used substances and how many, whether they were using any prescribed medications for their substance use, how many days in the past four weeks they had been in employment/volunteering/training, whether they were experiencing severe housing issues i.e. homelessness, their rating of their psychological health, physical health, and quality of life, their goal for treatment, and whether they had ever had pharmacological, psychological, and/or socially-oriented treatment and, if so, how effective they rated it. Full details of each question and their response options can be found in Appendix F.

I then started the first experimental manipulation by explaining one of two simplified views of addiction (Figure 2), and followed this by asking participants to describe the view in their own words as a manipulation check. All participants gave adequate responses that indicated, to the best of the researcher's knowledge, that they had understood the view described to them (for both experimental manipulations). The first male and female participants were randomly allocated to receive either the biological or social view first, with participants alternated thereafter based on their gender for counterbalancing. After this I asked participants "How much do you agree with the biological/social view of addiction we just discussed?" and asked them to rate their agreement on a 7-point Likert scale from 1 "strongly disagree" to 7 "strongly agree".

Figure 2

Diagrams of simplified addiction theories used for experimental manipulations



Following this I asked participants to imagine themselves right at the beginning of their recovery journey as if they were attending a support group where they would be talking about addiction. I explained that the view of addiction we had just discussed would be the focus of the group and then proceeded to collect the first set of outcome measures.

No pre-validated measures were a good fit for the study context so, based on a review of the literature on treatment outcomes and recovery from substance addictions, a variety of constructs were identified that were linked both positively and negatively with recovery. Ten single-item measures were developed to tap these

[1] Biological view image – https://newsinhealth.nih.gov/2015/10/biology-addiction
[2] Social view image – https://stock.adobe.com/fr/images/social-pressure-manipulation-criticism-bullying-human-faces-give-advice-psychological-influence-emotional-stress-mental-addiction-of-people-opinion-info-or-news-overload-vector-illustration/416475105

constructs in a straightforward face-valid way and tailored to fit the study. The use of bespoke single-item rating scales raises concerns around both reliability and validity, but research in psychometrics has shown that such measures can be just as valid and reliable as multi-item measures (Ahmad et al., 2014; Ang & Eisend, 2018). In addition, single-item measures have the benefit of being quicker to administer, easier for respondents, and less ambiguous in their measurement of a construct of interest (Allen et al., 2022) i.e. they have good face validity (Holden, 2010). The single-item measures developed for this study, including the constructs they are intended to tap, are as follows:

(1) “If the leader or facilitator of the group talked a lot about this view of addiction, how supported would you feel by them?” on a 5-point Likert scale from 1 “not at all supported” to 5 “extremely supported”. This taps into the therapeutic alliance (Kelly et al., 2016).

(2) “If other members of the group talked a lot about this view of addiction, how supported would you feel by them?” on a 5-point Likert scale from 1 “not at all supported” to 5 “extremely supported”. This taps into social support (Nikmanesh et al., 2016).

(3) “As part of this group which talked a lot about this view of addiction, how optimistic would you be about your likelihood of recovering control over your substance use?” on a 5-point Likert scale from 1 “not at all optimistic” to 5 “extremely optimistic”. This taps into recovery optimism (Provost et al., 2022).

(4) “As part of this group which talked a lot about this view of addiction, how confident would you be in your capacity to solve problems and improve your quality of life?” on a 5-point Likert scale from 1 “not at all confident” to 5 “extremely

confident". This taps into self-efficacy and coping skills (Ciraulo et al., 2003; Nikmanesh et al., 2016).

(5) "As part of this group which talked a lot about this view of addiction, how likely do you think you would be to relapse to substance use?" on a 5-point Likert scale from 1 "not at all likely" to 5 "extremely likely". This taps into belief in probability of relapsing (Mohammadpoorasl et al., 2012).

(6) "As part of this group which talked a lot about this view of addiction, how much effort would you put into trying to quit?" on a 5-point Likert scale from 1 "no effort at all" to 5 "a great deal of effort". This taps into treatment motivation (Ciraulo et al., 2003; Shaul et al., 2019).

(7) "As part of this group which talked a lot about this view of addiction, how resilient would you feel in being able to cope with difficulties in your life?" on a 5-point Likert scale from 1 "not at all resilient" to 5 "extremely resilient". This taps into resilience (Rudzinski et al., 2017).

(8) "As part of this group which talked a lot about this view of addiction, how much would you blame yourself for your addiction?" on a 5-point Likert scale from 1 "not at all to blame" to 5 "a great deal to blame". This taps into self-blame (Snoek et al., 2021).

(9) "As part of this group which talked a lot about this view of addiction, how negatively would you feel about yourself?" on a 5-point Likert scale from 1 "not at all negative" to 5 "extremely negative". This taps into negative affect about self (Ciraulo et al., 2003; Hogarth, 2020).

(10) "As part of this group which talked a lot about this view of addiction, how many days per week do you think you would use substances?" on a 5-point Likert

scale from 1 “0 days” to 5 “7 days”. This taps into self-predicted substance use (Acuff et al., 2019).

After participants had given their responses to the ten outcome measures in the first experimental manipulation, I then began the second experimental manipulation by explaining the alternative view of addition. I completed another manipulation check, asked them to rate their level of agreement with the second view of addiction, and finally collected their responses to the ten outcome measures for a second time. The materials used for the experimental manipulations are included in Appendix G.

Once both experimental manipulations and data collection were complete I had a warm-down chat with participants and asked how they experienced taking part in the study and answered any questions that arose for them. I then provided a full debrief about the rationale for the study and explained my beliefs about the value of developing collaborative understandings of addiction with people. I engendered an optimistic view of participants recovery potential to induce a mood uplift and they were thanked for taking part and given their payment voucher electronically via email or physically in the post. They were also given the options of attending a post-study group discussion meeting and to receive a copy of the completed study.

Analytic plan

Paired-sample t-tests were used to contrast the scores on the ten single-item outcome measures (facilitator support, peer/member support, recovery optimism, confidence to solve problems, relapse likelihood, quitting effort, resilience to difficulties, self-blame, negative affect about self, and self-predicted substance use) between the biological and social addiction theory conditions to determine any differences.

Spearman correlation coefficients were also generated to explore any associations between agreement with the biological and social addiction theories, participant demographic and treatment history, and the ten single-item outcome measures. Spearman was chosen over Pearson because the data from the addiction theory agreement and the ten single-item outcomes measures was ordinal rather than interval.

Results

Participant characteristics

All 34 participants recruited for the study successfully completed it and none withdrew their data during the one month post-study period prior to anonymization.

Table 1

Participant demographics and characteristics

Demographics and characteristics	
Age in years	*M* = 41.67, SD = 9.92
Gender	Male (67.7%), Female (29.4%), Non-binary (2.9%)
Ethnicity	White British, Irish or other (100%), Mixed (0%), Asian or Asian British (0%), Black or Black British (0%), Other ethnic group (0%)
Substance in treatment for	Alcohol (32.4%), Poly substance use (32.4%), Heroin (23.5%), Other drugs e.g. cocaine (11.8%)
Length of current treatment (months)	*M* = 26.29, *SD* = 26.54
Number of previous attempts at treatment	None (47.1%), One (14.7%), Two (5.9%), Three (11.8%), Four or more (20.6%)
Days with substance use in past 4 weeks	*M* = 8.76, *SD* = 11.79
Number of substances used in past 4 weeks	None (44.1%), One (29.4%), Two (14.7%), Three (11.8%)
Currently prescribed medication for addiction	Yes (50.0%)
Days in employment, education, training, or volunteering in past 4 weeks	*M* = 7.88, *SD* = 9.50
Severe housing problems or homelessness	No (73.5%)
Mental health (0 ‘poor’ to 20 ‘good’)	*M* = 11.68, *SD* = 4.48
Physical health (0 ‘poor’ to 20 ‘good’)	*M* = 11.38, *SD* = 3.55
Overall quality of life (‘poor’ to 20 ‘good’)	*M* = 12.03, *SD* = 3.61
Treatment goals	Abstinence (79.4%), Controlled use (11.8%), Other goal e.g. abstinence for one substance, controlled use for another (8.8%)
Ever received pharmacological intervention	Yes (58.8%)
Length of pharma intervention (months)	*M* = 28.90, *SD* = 21.62
Pharma effectiveness (1 ‘not at all’ to 5 ‘extremely’)	*M* = 3.60, *SD* = 0.88
Ever received psychological intervention	Yes (64.7%)
Length of psych intervention (months)	*M* = 21.36, *SD* = 22.35
Psych effectiveness (1 ‘not at all’ to 5 ‘extremely’)	*M* = 3.18, *SD* = 1.26
Ever received social intervention	Yes (79.4%)
Length of social intervention in months	*M* = 23.41, *SD* = 20.59
Social effectiveness (1 ‘not at all’ to 5 ‘extremely’)	*M* = 3.96, *SD* = 0.85

Descriptive and inferential statistics

The descriptive and inferential statistics for theory agreement and the ten outcomes are shown in Table 2. Mean difference scores are shown in Figure 3.

Table 2

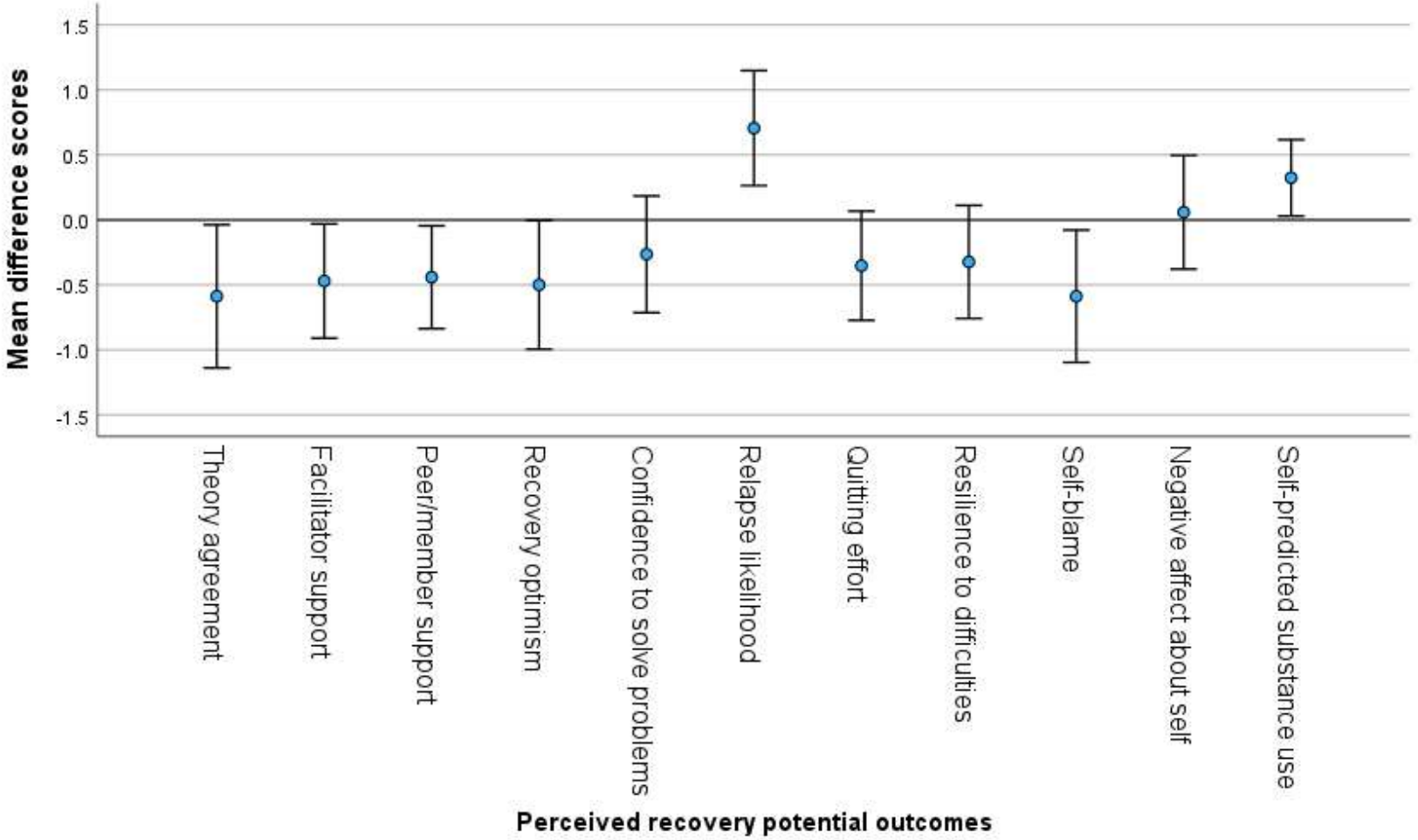
Descriptive and inferential statistics

Perceived recovery potential outcomes	Biological		Social		Paired sample t-tests					Wilcoxon signed-rank tests		
	M	*SD*	*M*	*SD*	*Mdiff*	*SD*	*t*(33)	*p*	d	*T*	*z*	*p*
0. Theory agreement	**5.35**	**1.20**	**5.94**	**1.04**	**-0.59**	**1.58**	**-2.173**	**.037***	**-0.37**	**221.00**	**2.07**	**.038***
1. Facilitator support	**2.97**	**1.09**	**3.44**	**0.79**	**-0.47**	**1.26**	**-2.176**	**.037***	**-0.37**	**233.00**	**1.96**	**.050**
2. Peer/member support	**3.03**	**1.03**	**3.47**	**0.79**	**-0.44**	**1.11**	**-2.270**	**.030***	**-0.39**	**205.50**	**2.14**	**.032***
3. Recovery optimism	**2.35**	**1.20**	**2.85**	**0.99**	**-0.50**	**1.42**	**-2.054**	**.048***	**-0.35**	**171.00**	**1.98**	**.048***
4. Confidence to solve problems	2.29	1.09	2.56	0.96	-0.27	1.29	-1.200	.239	-0.21	159.00	1.09	.278
5. Relapse likelihood	**3.24**	**1.10**	**2.53**	**0.83**	**0.71**	**1.27**	**3.246**	**.003****	**0.56**	**59.00**	**-2.87**	**.004****
6. Quitting effort	3.03	1.11	3.38	0.96	-0.35	1.20	-1.711	.097	-0.29	149.00	1.69	.091
7. Resilience to difficulties	2.21	1.01	2.53	0.99	-0.32	1.25	-1.511	.140	-0.26	156.00	1.45	.146
8. Self-blame	**3.12**	**1.45**	**3.71**	**1.19**	**-0.59**	**1.46**	**-2.351**	**.025***	**-0.40**	**160.00**	**2.12**	**.034***
9. Negative affect about self	2.91	1.22	2.85	1.26	0.06	1.25	0.274	.786	0.05	82.50	-0.52	.603
10. Self-predicted substance use	**3.41**	**1.35**	**3.09**	**1.46**	**0.32**	**0.84**	**2.238**	**.032***	**0.38**	**30.00**	**-2.13**	**.033***

*$p < .05$ **$p < .01$ two-tailed.

Figure 3

Error bar of mean difference scores showing 95% confidence intervals (biological theory compared to social theory of addiction)



Note. Values above the line indicate larger scores for the biological theory compared to the social theory. Values below the line indicate larger scores for the social theory compared to the biological theory. Confidence intervals overlapping the line signal no statistically significant difference in scores between the biological and social theory conditions.

Participants tended to agree with both addiction theories but favoured the social view, which had a mean score closer to six, or the “moderately agree” category (7-point Likert scale from 1 “strongly disagree” to 7 “strongly agree”), compared with the biological view which had a mean score closer to five, or the “slightly agree” category.

Participants mean scores on the outcome measures of facilitator support, peer/member support, recovery optimism, confidence to solve problems, quitting effort, resilience to difficulties, and self-blame were all lower under the biological condition compared to the social condition. Additionally, mean scores on the outcome measures of relapse likelihood, negative affect about self, and self-predicted substance use were all higher under the biological condition compared to the social condition, though negative affect about self was only marginally higher.

Paired sample t-tests were performed to compare the biological condition to the social condition on the measure of theory agreement and the ten single-item measures of perceived recovery potential. Statistically significant differences were found for theory agreement (d = -0.37), facilitator support (d = -0.37), peer/member support (d = -0.39), recovery optimism (d = -0.35), relapse likelihood (d = 0.56), self-blame (d = -0.40), and self-predicted substance use (d = 0.38). The effect sizes for all results were small apart from relapse likelihood which was moderate. For the outcome measures of confidence to solve problems, quitting effort, resilience to difficulties, and negative affect about self, no statistically significant difference was found.

For the purpose of graphing, difference scores were calculated between the biological and social condition for each of the ten outcomes, by subtracting the response to the biological condition from the response to the social condition, such

that positive scores reflect a greater response to the biological condition. Figure 3 shows the 95% confidence intervals for the mean differences scores of each variable.

Due to some of the variables not meeting the assumptions for parametric tests a non-parametric Wilcoxon signed-rank test was performed which mostly confirmed the results of the paired-sample t-tests. The only difference was that the result for facilitator support became marginally non-significant (p = .050). However, facilitator support was one of the variables that had met parametric assumptions so the results of the paired sample t-test were given primacy.

A post-hoc power analysis was conducted using the mean effect size, *d* = .33, of the ten single-item measures. This produced a figure of .46 which was much lower than expected due to actual effect sizes being smaller than predicted.

Correlations

Multiple exploratory correlations were conducted so a Holm-Bonferroni correction was performed to adjust the alpha level for statistical significance and control for familywise Type I error.

Table 3 shows the correlation coefficients for participants level of agreement with the biological and social addiction theories and their demographic and treatment variables. The only variable that showed a significant correlation was days in employment, training, or volunteering which was moderately positively correlated with social theory agreement (r_s = .621).

Table 4 shows the correlation coefficients for participants level of agreement with the biological and social addiction theories and their responses to the ten single-item measures of perceived recovery potential. Biological theory agreement was moderately positively correlated with facilitator support (r_s = .462) and peer/member

support (r_s = .503), although facilitator support was marginally non-statistically significant (p = .054). Social theory agreement was moderately positively correlated with facilitator support (r_s = .504), peer/member support (r_s = .559), recovery optimism (r_s = .532), and confidence to solve problems (r_s = .558). The significance of these correlations in relation to the findings of the statistical tests of difference will be evaluated in the discussion section. All statistical outputs from SPSS are included in Appendix H.

Table 3

Spearman's correlation matrix of theory agreement and demographic and treatment history variables

Variables	1	2	3	4	5	6	7	8	9	10	11	12	13	14
Biological theory agreement	.024	-.086	.028	.324	.159	.035	.200	.025	.249	-.220	-.071	.006	.208	.226
Social theory agreement	-.227	.297	.075	**.621****	.240	-.014	.047	-.011	.092	.089	.041	-.125	.325	.255

*$p < .05$ **$p < .01$ two-tailed.

1 = age, 2 = gender, 3 = housing problems (poor quality housing/risk of eviction/homelessness), 4 = days in employment/training/volunteering, 5 = psychological health, 6, physical health, 7 = overall quality of life, 8 = days of substance use in past months, 9 = length of current treatment, 10 = number of previous treatments, 11 = currently prescribed medication for substance use, 12 = ever had pharmacological treatment, 13 = ever had psychological treatment, 14 = ever had social treatment

Table 4

Spearman's correlation matrix of theory agreement and outcome measures of perceived recovery potential

Variables	1	2	3	4	5	6	7	8	9	10
Biological theory agreement	.462	**.503***	.250	.277	-.176	.165	.096	-.032	.073	.105
Social theory agreement	**.504***	**.559***	**.532***	**.558***	.102	.353	.426	-.091	.063	.106

*$p < .05$ **$p < .01$ two-tailed.

1 = facilitator support, 2 = peer/member support, 3 = recovery optimism, 4 = confidence to solve problems, 5 = relapse likelihood, 6 = quitting effort, 7 = resilience to difficulties, 8 = self-blame, 9 = negative affect about self, 10 = self-predicted substance use

Note. The correlations in Table 4 represent the relationship between participants' level of agreement with each addiction theory and the ten outcome measures immediately after each corresponding theory of addiction had been described.

Discussion

The findings of this study provide some indication that emphasising biological versus social causal factors in addiction has differential impacts on substance users' potential for recovery. In support of the study hypothesis, the results show a benefit to the social over the biological theory of addiction on measures of therapeutic alliance, social support, recovery optimism, belief in the probability of relapsing, and self-predicted substance use. It is important to note that the effect sizes were small, with a mean Cohen's *d* of *d* = 0.41. Also in support of the study hypothesis, a benefit for the biological theory over the social theory was found for the measure of self-blame. Again, the effect size for this result was small, with a Cohen's d of d = 0.40.

No support was found for the study hypothesis on the measures of self-efficacy and coping, resilience, treatment motivation, or negative affect about self which did not differ between biological and social theories. Additionally, the correlation matrix indicated no association between the level of agreement with either addiction theory and the measures of belief in probability of relapsing, self-predicted substance use, and self-blame. This suggests participants believed they were more likely to relapse and use substances but were less likely to blame themselves under the framing of the biological theory regardless of their level of agreement with either addiction theory. However, it is important to recognize that these effects were small.

These findings contribute further evidence towards the mixed blessing model (Haslam & Kvaale, 2015) of biogenetic explanations of mental health and addiction. The biological theory presented to participants in the study emphasised genetic vulnerability and brain changes, both of which are outside the control of the individual. It is therefore plausible that participants made attributions of

uncontrollability about their substance use which diminished their self-blame under the framing of the biological theory over the social theory. Furthermore, participants attributions of uncontrollability and the mechanistic thinking implied by the focus on genetics and neurobiology may have led them to essentialized themselves under the biological theory framing, contributing to them feeling less optimistic about their recovery, to believe they were more likely to relapse, and predict they would use more substances after attending the imaginary therapeutic group. Conversely, under the framing of the social theory participants experienced an inverse mixed blessing, with the emphasis on social stresses and coping going beyond the individual to recognise the context in which addiction occurs and implying more choice and control for participants. It may be this increased sense of choice and control that led participants to experience greater prognostic optimism at the cost of feeling more to blame for their difficulties.

In an attempt to understand the specifics of the mixed blessing model, Loughman and Haslam (2018) conducted a meta-analysis of studies that examined the effect of neurobiological explanations, without reference to genetics, on prognostic pessimism, desire for social distance, perceived dangerousness, and blame (Loughman & Haslam, 2018). The findings showed that neurobiological explanations, similarly to biogenetic explanations, were associated with greater prognostic pessimism, desire for social distance, and perceived dangerousness. However, unlike explanations involving genetics, the neurobiological explanations were not associated with reduced blame. Therefore, if the biological theory utilised in this study had only made reference to brain changes and not genetic vulnerability, it is possible that the beneficial effect would not have occurred.

In terms of overall endorsement of the two theories, the results indicated that treatment-engaged substance users possess complex views of addiction, agreeing that both biological and social factors play important roles. This is similar to the views of addiction treatment providers (Barnett et al., 2018) who have been found to endorse disease, moral, free-will, and social addiction theories simultaneously, and addiction researchers (Hammer et al., 2012; Ochterbeck & Forberger, 2022) who emphasize complex biopsychosocial causation.

However, participants expressed greater agreement with the social theory over the biological theory as reflected in the statistically significant t-test. This preference had an influence on recovery processes, with statistically significant positive correlations found between social theory agreement and the measures of therapeutic alliance, social support, recovery optimism, and self-efficacy and coping. Interestingly, biological theory agreement was not correlated with many of the measures of recovery potential, but statistically significant positive correlations with therapeutic alliance and almost statistically significant with social support were found. The fact the level of agreement with both theories was associated with therapeutic alliance and social support, under their respective framings, provides empirical support to the idea that alignment in understanding of addiction between substance users, their peers, and treatment providers has value for treatment engagement and retention (Dacosta-Sánchez et al., 2022).

Clinical implications

Alignment in understanding about addiction is important for all helping professions involved in addiction services, but has particular relevance for clinical psychology and its practice of formulation. A simple definition of formulation is “a tool used by clinicians to relate theory to practice” leading to “hypotheses to be tested”

(Johnstone & Dallos, 2014). It can be seen as a form of narrative with the aim of creating new understandings that provide targets for therapeutic intervention. A formulation is not an expert pronouncement like a medical diagnosis, but instead is a plausible account of how a person has come to have the difficulties they do (Johnstone & Boyle, 2018; Kinderman, 2019).

What is considered a plausible account will differ between people and be determined by substance users' experiences. For example, the number of days participants were in employment, training, or volunteering was moderately positively correlated with their social theory agreement. This may be because employed participants had experience of coping with work stress using substances, making such social causation more obvious to them. Alternatively, some of the participants were peer mentors who stated that their engagement in the role was an important part of their recovery journey. This may have led them to place more value in the social theory because, in addition to social causation, it implied the use of social solutions in recovery from addiction of which they had direct experience.

Therefore, such accounts need to be developed collaboratively with substance users, integrating their knowledge and experience alongside professionals, if they are going to be useful in promoting healthy behaviour change. They also need to be dynamic and open to change as the meaning of an individual's addiction, and the possible pathways for overcoming it, develop over the course of their recovery journey.

Strengths and limitations

A significant strength of this study over previous research was the inclusion of treatment-engaged substance users as participants in an experimental study. To the author's knowledge, such participants had only previously been included in

observational studies. This places the study on firmer ground regarding causal claims about the clinical impact of different addiction theories. Additionally, the use of an interview-style approach with participants, both online and in-person, was a significant improvement in terms of data quality over previous studies that had used online surveys to gather data. This was further supported by the very simple and specific conceptualisations of biological and social addiction theories in the study which were easy for participants to understand, providing greater confidence in the experimental manipulations.

Furthermore, the within-subjects design reduced noise in the data by having participants act as their own controls, and the use of simple face-valid outcome measures meant the study could be conducted within 30-minutes, reducing fatigue and practice effects. Based on the authors' experience recruiting participants and consultation with drug and alcohol service staff, a longer study procedure and/or the use of more linguistically complex and time-consuming outcome measures would have significantly reduced the representativeness of the participants, many of whom would have struggled to meaningfully engage. The combination of these different improvements over previous research may be why such clear effects between experimental conditions were found.

One limitation of the study was the small sample size, which, while sufficient for the main analysis, was not suitable for sub-analyses such as exploring differences between the substances participants were in treatment for. Furthermore, the participant demographics mirrored much of the previous research in the field by including mostly white males. Although this is understandable given substance misuse is twice as common in men (McManus et al., 2016), it would have been interesting to explore as research suggests gender differences exist i.e. accelerated

onset from harmful to disordered use (Fonseca et al., 2021). It would also be useful to understand if ethnicity interacted with substance use and how the addiction theories presented in the study fit, or not, with different cultural narratives about addiction.

Finally, it was challenging recruiting treatment-engaged substance users early in their recovery journey meaning 55.9% of participants were not in their first episode of treatment and the mean length of current treatment was 26 months. This meant that the study reflected participants' perception about the utility of theoretical discourse in recovery generally, rather than in early recovery specifically.

Future research directions

Future research on the effects of translating scientific narratives into clinical discourse on recovery processes should utilise qualitative methodologies to generate more in-depth understanding of how substance users make sense of addiction. This would elaborate the study finding that participants broadly agreed with both addiction theories presented to them. This richer exploration might also lead to a more precise understanding of the mixed blessing model and suggest ways to avoid it, giving substance users greater optimism about their recovery without feeling blamed. Future quantitative research should look to replicate the findings from Loughman and Haslam's (2018) meta-analysis by separating the neurobiological and genetic components of the biological theory presented in the study. This would determine whether the beneficial effect on self-blame found in the study was due to the inclusion of genetic vulnerability within the biological theory.

Inclusion of more women and gender non-confirming people, alongside participants from a wider range of ethnic backgrounds would also enrich the field as this might point to demographic differences that have a baring on recovery. Finally, a

rebalancing of research priorities away from a problem-focus on addiction aetiology towards a solution-focus on recovery (Brown & Ashford, 2019) is recommended. The is particularly important for healthcare research and its applied focus on clinical utility over a more general understanding of basic processes.

Conclusion

This study aimed to investigate whether biologically-focused addiction theories are more harmful and less useful than more socially-focused theories to treatment-engaged substance users. A within-subjects study was conducted with 34 working age adult service users from a drug and alcohol service in Southern England, UK. The study explored the impact of simplified biological and social addiction theories on ten outcome variables that tapped constructs linked to participants perceived recovery potential from addiction.

The study hypotheses were mostly supported with worse scores on outcome measures of therapeutic alliance, social support, recovery optimism, belief in the probability of relapsing, and self-predicted substance use, but a better score on the measure of self-blame under the framing of the biological compared to the social theory. No support was found for any difference between the two addiction theories on measures of self-efficacy and coping, resilience, treatment motivation, or negative affect about self. These findings were interpreted using the mixed blessing model of biogenetic explanations (Haslam & Kvaale, 2015) which suggests blame is reduced through attributions of uncontrollability at the cost of increasing prognostic pessimism through psychological essentialism. By contrast, the social theory presented in the study offered an inversed mixed blessing, increasing prognostic optimism at the cost of increasing participants blame towards themselves. The inclusion of genetic

vulnerability within the biological theory presented in the study may be the reason such a mixed blessing was found (Loughman & Haslam, 2018).

The results of the study provide support for the value of in-depth exploration of substance users understandings of addiction as a critical aspect of treatment. This is firstly to get alongside substance users and engage them in treatment, and secondly to help identify interventions that make sense in light of substance users understandings. In clinical psychology, professional and service user knowledge and experience can be integrated into formulations of addiction that emphasise “what's happened to you?” over “what's wrong with you?” (Harper & Cromby, 2022), and suggest pathways to recovery that fit with substance users’ needs and capabilities.

This emphasis on shared understanding and collaboration is a direct challenge to the privileging of scientific knowledge generated *about* substance users and promotes the value of scientific knowledge that is both *for* and *from* those struggling with addiction. Such an ethical position is essential in the human sciences generally, but is particularly important in the applied science of clinical psychology and related mental health professions. It is hoped that the findings of this study will be of use to those struggling with addiction and the people supporting them on their journey towards recovery.

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Appendices

Appendix A – Ethical Approval

Research Ethics Committee Review Outcome

Dear DANIEL CASEY

Ethics Application ID: 1003462

Title: Perspectives on Addiction: The impact of different explanatory theories of addiction on the recovery potential of treatment-engaged substance users

(Version: 1.1)

Proposed Project Duration: 6 Mar 2023 - 4 Mar 2024

Your research study ethics application submitted above on 23 Jan 2023, 12:40 has been reviewed by the FHLS Psychology Ethics Committee.

Outcome decision by Research Ethics committee: Approved

Decision Date: 17 Feb 2023, 08:31*

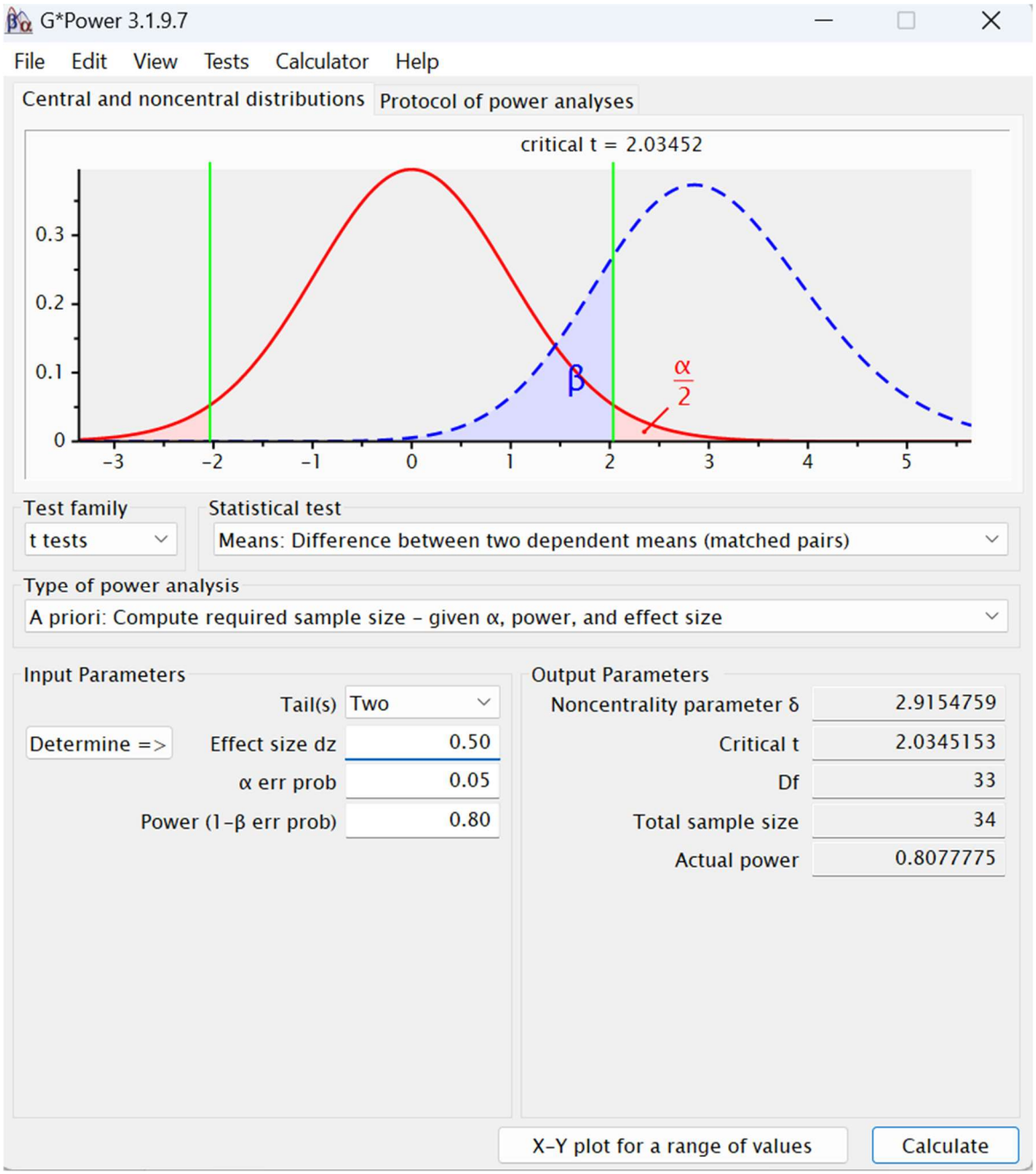
You can only start your research once you have received an **Approved outcome. The start date of your research will be no sooner than the Ethics Committee Approval decision date above.*

Research Ethics Committee Approval End Date:

Regards,

FHLS Psychology Ethics Committee

Appendix B – Sample Size Calculation



Appendix C – Participant Information Sheet (Paper & Digital)

Title of Project: Relationship between service users' understanding of addiction and their recovery potential

Researchers: Dan Casey & Dr Lee Hogarth

Invitation and brief summary:
You have been invited to take part in a study (either in person or online) about how different scientific explanations of addiction might impact on people struggling with addiction who are early in their recovery journey. You should only participate if you want to. Before you decide whether you want to take part, it is important for you to read the following information carefully to understand why the research is being done and what it will involve. Please ask the researcher if there is anything that is not clear or if you would like more information before deciding to take part.

Purpose of the research:
Scientists and clinicians differ in their understanding of addiction but we do not know whether this matters to people struggling with addiction. We therefore want to run a study with service users like yourself to get your perspective on how different understandings of addiction might impact on your potential to recover from your addiction problems.

Why have I been approached?
You have been approached to participate in this study because you are currently seeking treatment for addiction and are still relatively early in your recovery journey. We are hoping to recruit between 30-40 people like yourself to take part in this study.

What would taking part involve?
You will be invited, either in-person at the CONFIDENTIAL Drug & Alcohol Service CONFIDENTIAL hub or online on Microsoft Teams, where I will talk through some common scientific views of addiction with the help of some diagrams. I will then ask you some questions about your beliefs in your potential for recovery. This will all occur in one sitting and will take approximately 30-60 minutes.

What are the possible benefits of taking part?
You will be contributing to the understanding of how the way we explain and talk about addiction impacts on the people who are struggling with addiction problems. This may then lead to changes in clinical practice and influence what kind of research is prioritised in the future.

What are the possible disadvantages and risks of taking part?
There are no significant disadvantages or risks associated with taking part in this study. The only potential risk could be that hearing about different scientific explanations of addiction might be confusing or distressing to you. However, you will be given a verbal briefing before undertaking the study and a detailed debrief once the study is complete to fully inform you of the rationale and clarify any confusion or concerns the study may have brought up for you.

What will happen if I don't want to carry on with the study?
You can decide to withdraw from the study at any time. If you decide you do not want to carry on during the middle of the study, we will stop the study and any data collected up to that point will be deleted.

How will my information be kept confidential?
The University of Exeter processes personal data for the purposes of carrying out research in the public interest. The University will endeavour to be transparent about its processing of your personal data and this information sheet should provide a clear explanation of this. If you do have any queries about the University's processing of your personal data that cannot be resolved by the research team, further information may be obtained from the University's Data Protection Officer by emailing dataprotection@exeter.ac.uk or at www.exeter.ac.uk/dataprotection.

The data collected from you during the study includes the following two categories: (1) demographic and background information such as age, gender, ethnicity, current/historic substance use, health/social functioning, and current/historic addiction treatment, and (2) outcome measure

responses. The two sets of data will be linkable back to you for a period of 1 month from the date of taking part in the study. This will be done through a record of your name which will be stored on a password protected Microsoft Excel file. After 1 month I will delete the reference to your name making the data anonymous.

Will I receive any payment for taking part?
For your time and effort in participating in the study you will be reimbursed with a £10 digital voucher which will be emailed to you.

What will happen to the results of this study?
The study will be written up in an academic paper which will be submitted for publication in an academic journal in order to reach the widest academic and clinical audience. For study participants, I intend to run a group meeting to have an open discussion about the study and discuss the results. CONFIDENTIAL Drug & Alcohol Services will also disseminate the results of the study through their marketing team within their organisation.

Who is organising and funding this study?
This study is being undertaken as part of the researchers Doctorate in Clinical Psychology and is funded by the School of Psychology in the Faculty of Health and Life Sciences at the University of Exeter.

Who has reviewed this study?
This project has been reviewed and approved by the Research Ethics Committee at the University of Exeter (Reference Number: 1003462) and Clinical Governance at CONFIDENTIAL Drug & Alcohol Services.

Further information and contact details
If you would like to take part in the study and/or have any questions or concerns you can contact the researcher using the below details:
Dan Casey – Trainee Clinical Psychologist (dc674@exeter.ac.uk)

If you wish to raise a concern about the study or your participation in it that can't be directed to the researcher you can contact the project supervisor:
Dr Lee Hogarth – Associate Professor of Experimental Psychology (l.hogarth@exeter.ac.uk, 01392 724613)
or the Research Ethics and Governance Manager:
Gail Seymour (g.m.seymour@exeter.ac.uk, 01392 726621)

Thank you for your interest in this project.

Participant Information Sheet

Title of Project: Relationship between service users' understanding of addiction and their recovery potential

Researchers: Dan Casey & Dr Lee Hogarth

Invitation and brief summary

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or the Research Ethics and Governance Manager:
Gail Seymour (g.m.seymour@exeter.ac.uk, 01392 726621)

Thank you for your interest in this project.

Appendix D – Consent Form (Paper & Digital)

Title of Project: Relationship between service users' understanding of addiction and their recovery potential

Researchers: Dan Casey & Dr Lee Hogarth

1. I confirm that I have read the information sheet dated 01.12.2022 (version no 1.00) for the above project. I have had the opportunity to consider the information, ask questions and have had these answered satisfactorily. ☐
2. I understand that my participation is voluntary and that I am free to withdraw at any time without giving any reason and without my legal rights being affected. ☐
3. I understand that relevant sections of the data collected during the study, may be looked at by members of the research team, individuals from the University of Exeter, where it is relevant to my taking part in this research. I give permission for these individuals to have access to my records knowing they will be anonymised. ☐
4. I understand that taking part involves anonymised demographic, substance use, health/social functioning, and treatment history information and question responses to be used for the purposes of a report published in an academic publication. ☐
5. I understand that 1 month from the date of taking part in the study, the record of my name linking me to my data will be deleted making my data anonymous and meaning I can no longer withdraw my data from the study. ☐
6. I understand that anonymised data will be registered and archived at the University of Exeter's ORE repository in order to make them available to other researchers in line with current data sharing practices. ☐
7. I understand that data from the study will be used in reports in academic publication, media publication, and other publications but that no data or responses will be published in which I can be identified individually. ☐
8. I agree to take part in the above project. ☐

_____	_____	_____
Name of Participant	Date	Signature
_____	_____	_____
Name of researcher taking consent	Date	Signature

Consent Form

Title of Project: Relationship between service users' understanding of addiction and their recovery potential

Researchers: Dan Casey & Dr Lee Hogarth

I confirm that I have read the information sheet for the above project. I have had the opportunity to consider the information, ask questions and have had these answered satisfactorily.

I understand that my participation is voluntary and that I am free to withdraw at any time without giving any reason and without my legal rights being affected.

- ○ Yes
- ○ No

I understand that relevant sections of the data collected during the study, may be looked at by members of the research team, individuals from the University of Exeter, where it is relevant to my taking part in this research. I give permission for these individuals to have access to my records knowing they will be anonymised.

- ○ Yes
- ○ No

I understand that taking part involves anonymised demographic, substance use, health/social functioning, and treatment history information and question responses to be used for the purposes of a report published in an academic publication.

- ○ Yes
- ○ No

I understand that 1 month from the date of taking part in the study, the record of my name linking me to my data will be deleted making my data anonymous and meaning I can no longer withdraw my data from the study.

- ○ Yes
- ○ No

I understand that anonymised data will be registered and archived at the University of Exeter's ORE repository in order to make them available to other researchers in line with current data sharing practices.

- ○ Yes
- ○ No

I understand that data from the study will be used in reports in academic publication, media publication, and other publications but that no data or responses will be published in which I can be identified individually.

- ○ Yes
- ○ No

I agree to take part in the above project. To proceed, you must select ONE of the boxes below:

- ○ I HAVE READ THE CONSENT FORM ABOVE AND FULLY UNDERSTAND IT. I GIVE MY CONSENT TO PARTICIPATE IN THIS STUDY. CLICKING THIS OPTION WILL BE TREATED AS YOUR SIGNATURE.
- ○ I DO NOT GIVE MY CONSENT TO PARTICIPATE IN THIS STUDY.

Please enter your name below:

Appendix E – Study Procedure Transcript

Stage 1 – Gaining informed consent

1. ***Confirm participant has read or listened to the participant information sheet and consent form and has digitally signed them on Qualtrics:***

   Hi, my name is Dan Casey and I'm a Trainee Clinical Psychologist from the University of Exeter. Can I confirm that you have read or listened to the participant information sheet and consent form and have ticked the box to sign them?

   Great, before we get started did you have any questions or concerns about the study?

2. ***Inform participants they can withdraw from the study at any time and remind them where my contact details are located:***

   Please remember that once the study has started you can stop at any point if you change your mind and do not want to take part anymore and I will delete any data recorded.

   The information sheet and consent form you have signed have all the details about the study if you need to remind yourself at any point. It also has my contact details should you wish to discuss anything. Are you okay to start?

Stage 2 – Study brief including demographic data collection

3. ***Brief participant about the study, giving an overview of the background and reason for requesting their participation:***

   Thank you for agreeing to participate in this study. The purpose of the study is to explore your views about addiction and how they relate to different scientific views of addiction. For example, some scientists focus on the brain and genes, while others focus on a social stresses and ways of coping. The way scientists think about addiction can be quite complicated and hard even for

other scientists to understand let alone the public. This means that scientific explanations often get turned into simplified stories in the media, on TV, in newspapers, on the internet and these explanations can affect the way people dealing with addiction see themselves and think about their recovery.

The idea for this study is that we want your perspective on which views of addiction you would find most helpful at the beginning of your recovery journey. I will talk you through two views of addiction that are very influential amongst scientists and then ask you to respond to some questions based on how each approach makes you feel about your recovery journey and your engagement with treatment.

4. ***Gather information on participant demographics, substance use history, and treatment history:***

   Before we start the study, I just need to ask you some background questions such as your age, gender, and employment status and also about your use of substances and treatment history.

   All this information is completely confidential and anonymous and stored safely and securely on my password protected university computer. Are you okay to answer my questions? [Go through Qualtrics demographic form with participant].

Stage 3a – First experimental manipulation and data collection (Biological View 1st)

5. ***Show PowerPoint diagram [Slide 1] of the biological view of addiction and talk it through with participants:***

   The most important thing to know about the science of addiction is that it's quite complex and there are lots of different theories. To make this easier to

understand we will focus on two broad views of addiction that scientists tend to fall into.

The first view of addiction is called the biological view. According to this view, some people have genes that make them more vulnerable to becoming addicted. This vulnerability then means that when a person takes drugs or uses alcohol this has a more powerful effect on how the brain works so that eventually the persons' drug or alcohol use becomes automatic and they lose control over it [point to arrows connecting these elements on slide 1].

To make sure I've explained this clearly, could you describe the biological view of addiction in your own words? There's no right or wrong here, it's more about making sure we have a similar enough understanding of what this view means.

Thank you for sharing your understanding with me. Now I would like to know how much do you agree or disagree with the biological of addiction we just discussed? [Question asked while showing slide 2 which shows the question in written form and a visual representation of the response scale].

Great, I'm now going to ask you some follow up questions about this view of addiction.

6. ***Brief participants about the process for the first experimental manipulation [while showing slide 3]:***

So now I want you to imagine yourself near the beginning of your recovery journey. Pretend you have just reached out to CONFIDENTIAL Drug and Alcohol Services for support and have been invited to an initial support group to help you make sense of your struggles with addiction. In the group you will talk about what addiction is and what leads people to become addicted. I want

you to imagine that in this group they focus on the biological view of addiction we just discussed which means the group talks about vulnerable genes, changes in the brain and how this leads to drug and alcohol use becoming automatic. Okay, so try as best as you can to imagine being part of this group. [Participant to be shown single image of biological view of addiction on slide 3 and I will point to the diagram in concert with my verbal explanation above].

I now want you to answer some questions about how you might feel being a part of this group.

7. ***Ask participants to complete the outcome measures by reading out the items and recording their responses [participants will also be able to simultaneously read each question and see the response scale on slides 4-13].***

Stage 3b – First experimental manipulation and data collection (Social View 1st)

5. ***Show PowerPoint diagram [Slide 1] of the social view of addiction and talk it through with participants:***

The most important thing to know about the science of addiction is that it's quite complex and there are lots of different theories. To make this easier to understand we will focus on two broad views of addiction that scientists tend to fall into.

The first view of addiction is called the social view. According to the social view of addiction, some people experience very difficult social situations which means they're more likely to have a lot of problems and stresses in their lives. Some of these people learn to cope with their problems by using drugs or alcohol to block it out. As a result of struggling with all these social problems and using drugs or alcohol to cope, this leads to a strong desire to

keep using drugs or alcohol over time [point to arrows connecting these elements on slide 1].

To make sure I've explained this clearly, could you describe the social view of addiction in your own words? There's no right or wrong here, it's more about making sure we have a similar enough understanding of what this view means.

Thank you for sharing your understanding with me. Now I would like to know how much do you agree or disagree with the social of addiction we just discussed? [Question asked while showing slide 2 which shows the question in written form and a visual representation of the response scale].

Great, I'm now going to ask you some follow up questions about this view of addiction.

6. ***Brief participants about the process for the first experimental manipulation [while showing slide 3]:***

So now I want you to imagine yourself near the beginning of your recovery journey.

Pretend you have just reached out to CONFIDENTIAL Drug and Alcohol Services for support and have been invited to an initial support group to help you make sense of your struggles with addiction. In the group you will talk about what addiction is and what leads people to become addicted. I want you to imagine that in this group they focus on the social view of addiction we just discussed which means the group talks about people experiencing a lot of social stresses, using drugs or alcohol to cope, and how this leads to a strong desire to keep using drugs or alcohol. Okay, so try as best as you can to imagine being part of this group.

I now want you to answer some questions about how you might feel being a part of this group.

7. ***Ask participants to complete the outcome measures by reading out the items and recording their responses [participants will also be able to simultaneously read each question and see the response scale on slides 4-13].***

Stage 4a – Second experimental manipulation and data collection (Biological View 1st)

8. ***Show PowerPoint diagram [Slide 1] of the social view of addiction and talk it through with participants:***

The second scientific view of addiction is called the social view. According to the social view of addiction, some people experience very difficult social situations which means they're more likely to have a lot of problems and stresses in their lives. Some of these people learn to cope with their problems by using drugs or alcohol to block it out. As a result of struggling with all these social problems and using drugs or alcohol to cope, this leads to a strong desire to keep using drugs or alcohol over time [point to arrows connecting these elements on slide 1].

To make sure I've explained this clearly, could you describe the social view of addiction in your own words? There's no right or wrong here, it's more about making sure we have a similar enough understanding of what this view means.

Thank you for sharing your understanding with me. Now I would like to know how much do you agree or disagree with the social of addiction we just

discussed? [Question asked while showing slide 2 which shows the question in written form and a visual representation of the response scale].

Great, I'm now going to ask you some follow up questions about this view of addiction.

9. ***Brief participants about the process for the second experimental manipulation [while showing slide 3]:***

   So now I want you to imagine yourself near the beginning of your recovery journey. Pretend you have just reached out to CONFIDENTIAL Drug and Alcohol Services for support and have been invited to an initial support group to help you make sense of your struggles with addiction. In the group you will talk about what addiction is and what leads people to become addicted. I want you to imagine that in this group they focus on the social view of addiction we just discussed which means the group talks about people experiencing a lot of social stresses, using drugs or alcohol to cope, and how this leads to a strong desire to keep using drugs or alcohol. Do you best to imagine yourself being part of this group.

   I now want you to answer some questions about how you might feel being a part of this group.

10. ***Ask participants to complete the outcome measures a second time by reading out the items and recording their responses [participants will also be able to simultaneously read each question and see the response scale on slides 4-13].***

Stage 4b – Second experimental manipulation and data collection (Social View 1st)

8. ***Show PowerPoint diagram [Slide 1] of the biological view of addiction and talk it through with participants:***

The second scientific view of addiction is called the biological view. According to the biological view, some people have genes that make them more vulnerable to becoming addicted. This vulnerability then means that when a person takes drugs or uses alcohol this has a more powerful effect on how the brain works so that eventually the persons' drug or alcohol use becomes automatic and they lose control over it [point to arrows connecting these elements on slide 1].

To make sure I've explained this clearly, could you describe the biological view of addiction in your own words? There's no right or wrong here, it's more about making sure we have a similar enough understanding of what this view means.

Thank you for sharing your understanding with me. Now I would like to know how much do you agree or disagree with the biological of addiction we just discussed? [Question asked while showing slide 2 which shows the question in written form and a visual representation of the response scale].

Great, I'm now going to ask you some follow up questions about this view of addiction.

9. ***Brief participants about the process for the second experimental manipulation [while showing slide 3]:***

So now I want you to imagine yourself near the beginning of your recovery journey. Pretend you have just reached out to CONFIDENTIAL Drug and Alcohol Services for support and have been invited to an initial support group to help you make sense of your struggles with addiction. In the group you will

talk about what addiction is and what leads people to become addicted. I want you to imagine that in this group they focus on the biological view of addiction we just discussed which means the group talks about vulnerable genes, changes in the brain and how this leads to drug and alcohol use becoming automatic. Okay, so try as best as you can to imagine being part of this group. [Participant to be shown single image of biological view of addiction on slide 3 and I will point to the diagram in concert with my verbal explanation above].

I now want you to answer some questions about how you might feel being a part of this group.

10. ***Ask participants to complete the outcome measures a second time by reading out the items and recording their responses [participants will also be able to simultaneously read each question and see the response scale on slides 4-13].***

Stage 5 – Study debrief and payment

11. ***Post-study warm down chat to respond to any initial questions or concerns they might have about their experience of the study in preparation for the full debrief:***

    That's the study complete. Thank you so much for your participation. What was it like for you hearing about and comparing those different views of addiction? Do you have any questions at all now it's over?

12. ***Post-study debrief to explain the full rationale for the study and engender an optimistic perspective on the different approaches discussed to induce a mood uplift. Also an opportunity for the participant to have an open conversation about the study and their perspective on it***:

So this study is all about understanding how different scientific explanations of addiction actually effect people who are dealing with addiction. You might have noticed that after I asked you to imagine being in a support group that talked about addiction in those two different ways, I asked you all sorts of questions about things like your how much you agreed with the views, how supported you would feel, how likely you thought you would be to recover control over your substance use, and several others. The reason I asked you about those things is that it's my belief, along with other scientists, that the way people make sense of their addiction is an important part of the process of recovering from addiction. As I told you earlier, addiction is very complex and people have different preferences in how they understand it. That's why I also asked you to rate your agreement with the different approaches. It's not that one approach is right or wrong, it's more that some people feel more hopeful and confident about their recovery when they hear certain explanations of addiction whereas other explanations make them less hopeful and confident. What are your thoughts about my predictions now the study is over?

I just want to make it clear that it doesn't seem to matter in what way you understand addiction, as long as the way you understand it helps you to feel hopeful about your chances for recovery and motivated to keep engaging with support from services. How are you feeling now you know more about what I'm hoping to achieve with this study?

Lastly, because you now know what the study is all about, it's very important you don't talk to anyone else about what I've just told you until after

I've written up the study or it could influence how other people behave in the experiment, which may affect the results.

13. ***Pay participant with a digital voucher sent to their email address.***
14. ***Invite participant to post-study group debrief meeting where we will explore in a more open way how everyone found being in the study and their thoughts, feelings, and perspectives on it. Participants will be informed they have the option of receiving a copy of the study if they wish:***

    Once I have finished the study and am ready to share the results I will organize a group meeting where you and everyone else who participated will be invited to attend. In this group meeting I'll talk about the results of my study and we can have a conversation about how everyone found being in the study and all your thoughts and feelings about the results. Would you be interested in coming to this group?

    Great, in that case can I take down some contact details for you, either a phone number or email address which I will keep completely separate from the study data so I can reach you in the future. Also, if you would like I would be happy to send you a copy of the study once it is written up. [Enter participants contact details on separate Qualtrics form and tick the box for whether they are interested in the post-study group and/or receiving a copy of the study].

Appendix F – Demographics and Background Questions

ID. Participant identification number

Q0. Who is your current/past recovery worker?

Q1. What is your age?

Q2. What is your gender?

- ○ Male
- ○ Female
- ○ Non-binary / third gender
- ○ Prefer not to say

Q3. What is your ethnicity?

- ○ White (British, Irish, Any other White background)
- ○ Mixed (White and Black Caribbean, White and Black African, White and Asian, Any other mixed background)
- ○ Asian or Asian British (Indian, Pakistani, Bangladeshi, Any other Asian background)
- ○ Black or Black British (Caribbean, African, Any other Black background)
- ○ Other Ethnic Groups (Chinese, Any other ethnic group)
- ○ Prefer not to say

Q4. What substance/s are you currently/previously in treatment for?

Q5. How long have you been receiving support from [redacted] for?

Q26. Is this the first time you have received support for your problems with drugs and/or alcohol? If not, how many times have you received support in the past?

Q6. How many days in the last 4 weeks have you used the following substances?

	Days (0-28)
Alcohol	
Opiates/Opioids (including street heroin and non-prescribed medications)	
Crack	
Cocaine	
Amphetamines	
Cannabis	
Any other street drug	

Q7. Are you using any prescribed medications?

- ○ Yes
- ○ No

Q8. How many days in the past 4 weeks have you been in paid employment, volunteering, or training/education?

0 3 6 8 11 14 17 20 22 25 28

Days

Q9. Are you currently experiencing any problems with housing including poor quality housing, at risk of being evicted, or currently experiencing homelessness?

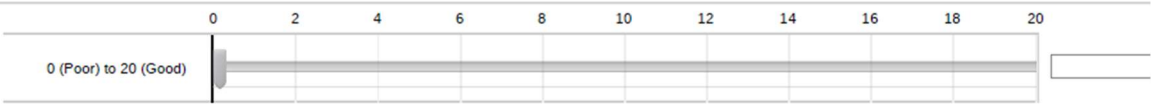
- ○ Yes
- ○ No

Q10. How would you rate your psychological and emotional health?

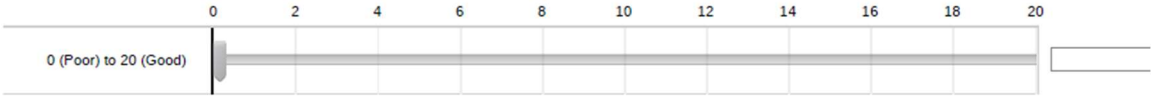
0 2 4 6 8 10 12 14 16 18 20

0 (Poor) to 20 (Good)

Q11. How would you rate your physical health?



Q12. How would you rate your overall quality of life?



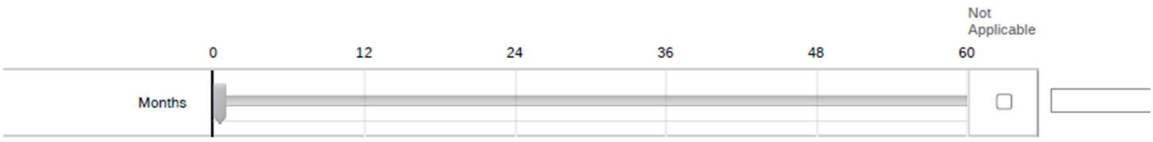
Q13. What is your current/previous goal for treatment?

- ○ Abstinence
- ○ Controlled Use
- ○ Other

Q14. Have you ever been prescribed any medications as treatment for your addiction problems? E.g. Benzodiazepine, Diazepam, Naltrexone, Buprenorphine, Methadone etc.

- ○ Yes
- ○ No

Q14a. If yes, how long have you been/were you on them?

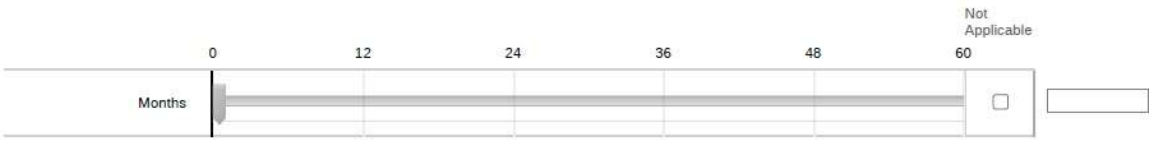


Q14b. If yes, how effective do you think they are/were?

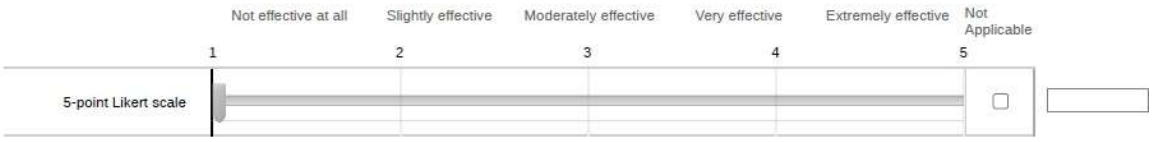
Q15. Have you ever had any one-to-one support, talking therapy or group support for your addiction problems? E.g. CBT, counselling, one-to-one peer support, 12-step programme etc.

- ○ Yes
- ○ No

Q15a. If yes, how long have you been receiving/did you receive 1:1/therapy/group support for?



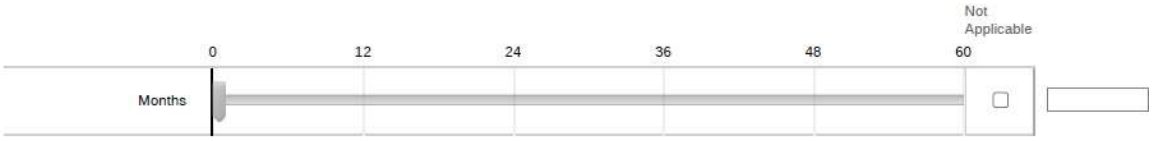
Q15b. If yes, how effective do you think it is/was?



Q16. Are you currently receiving any social support for your addiction problems? E.g. peer support, family/parenting support, housing support, employment support, education/training support etc.

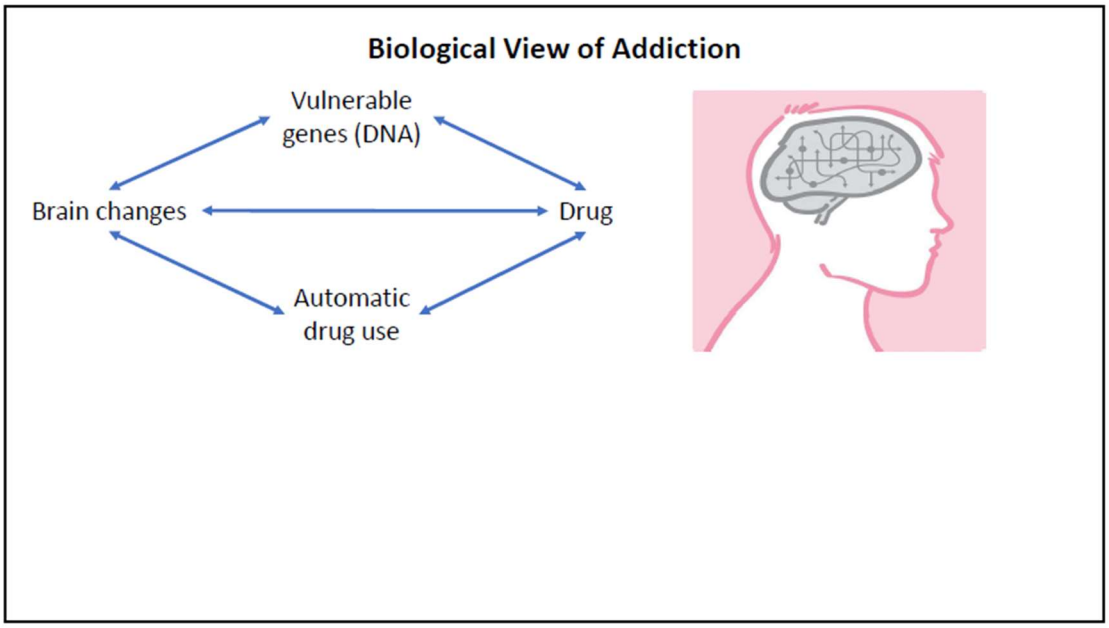
- ○ Yes
- ○ No

Q16a. If yes, how long have you been receiving/did you receive support for?

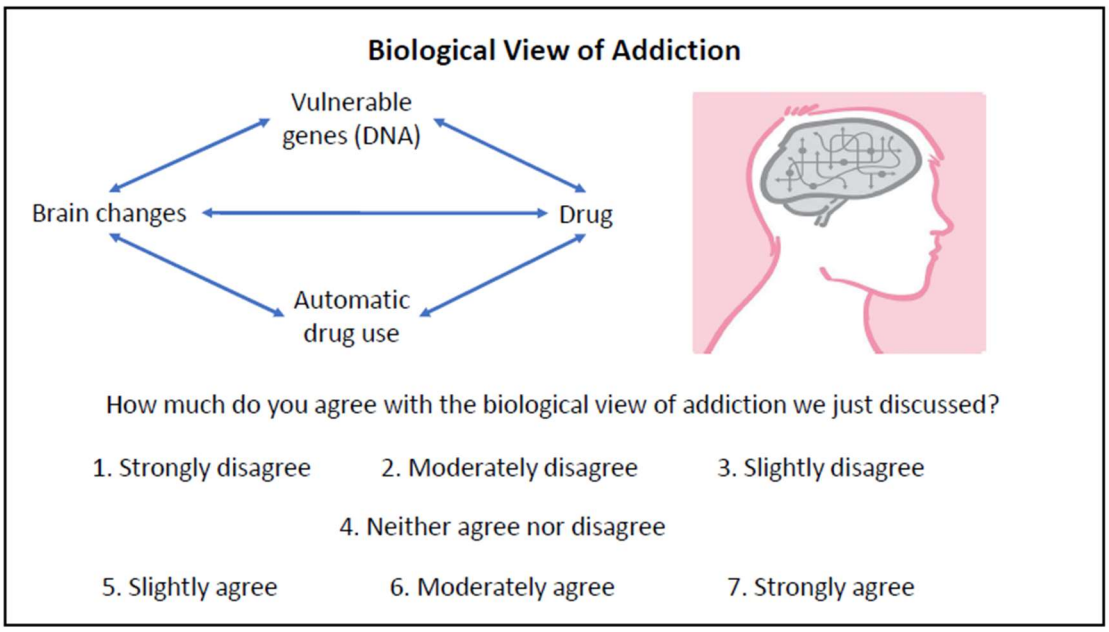


Q16b. If yes, how effective do you think it is/was?

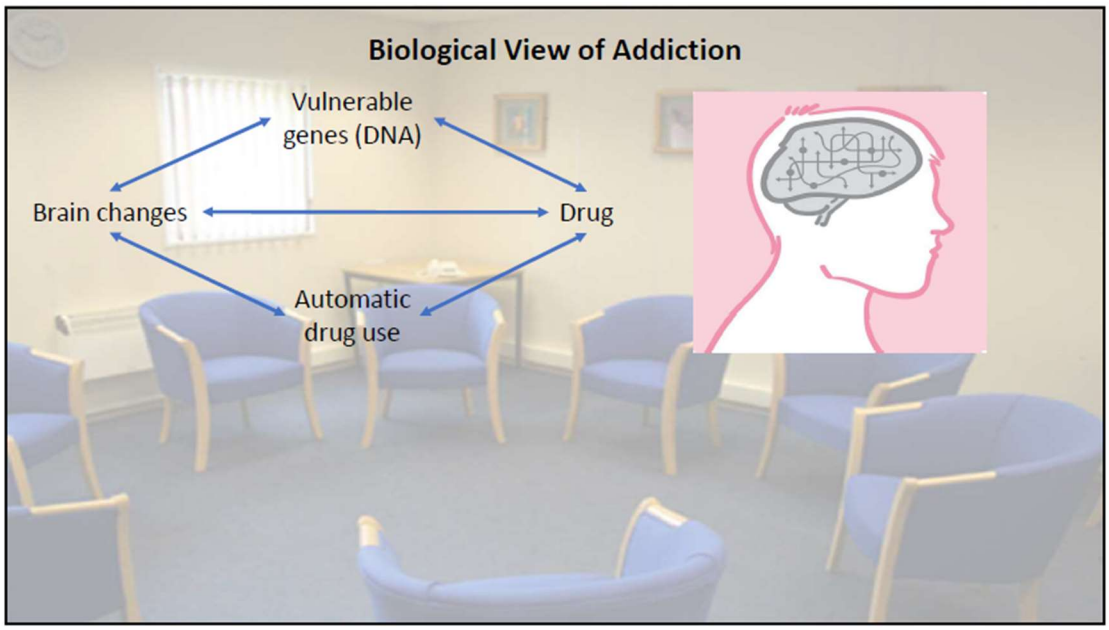
Appendix G – Experimental Manipulation Materials



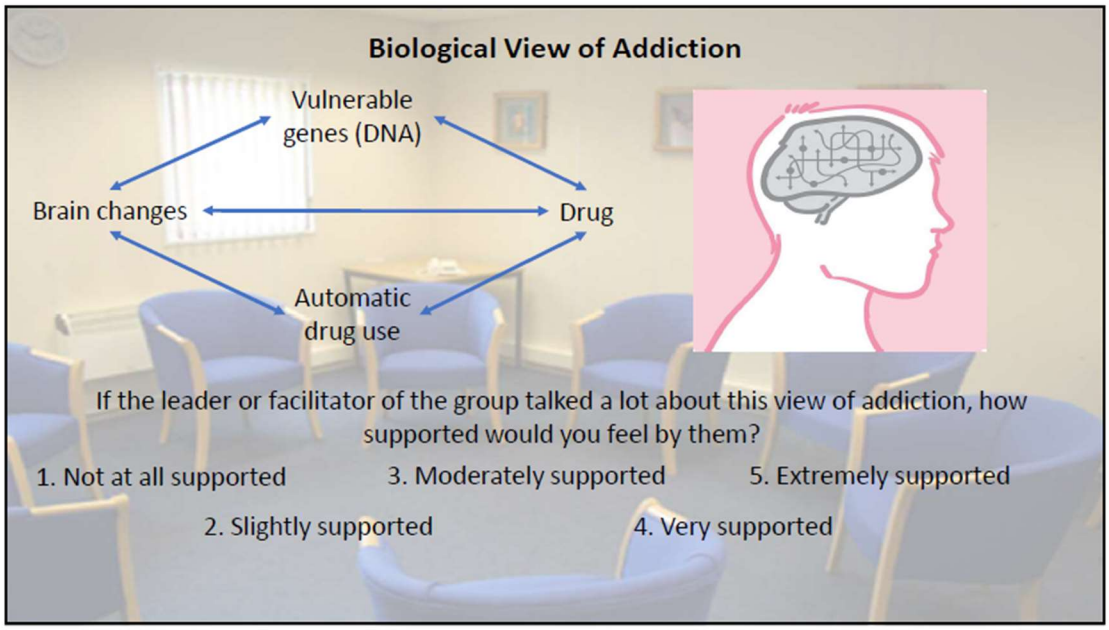
1



2


Biological View of Addiction
Vulnerable genes (DNA)
Brain changes
Drug
Automatic drug use

3


Biological View of Addiction
Vulnerable genes (DNA)
Brain changes
Drug
Automatic drug use
If the leader or facilitator of the group talked a lot about this view of addiction, how supported would you feel by them?
1. Not at all supported
2. Slightly supported
3. Moderately supported
4. Very supported
5. Extremely supported

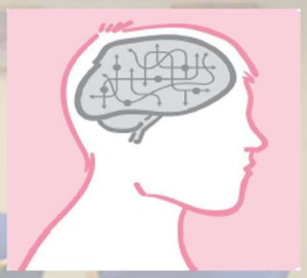
4

Biological View of Addiction

Vulnerable genes (DNA)

Brain changes ↔ Drug

Automatic drug use



If other members of the group talked a lot about this view of addiction, how supported would you feel by them?

1. Not at all supported
2. Slightly supported
3. Moderately supported
4. Very supported
5. Extremely supported

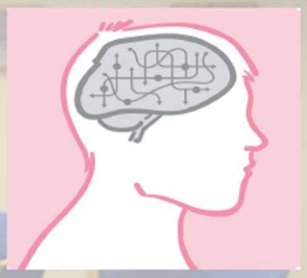
5

Biological View of Addiction

Vulnerable genes (DNA)

Brain changes ↔ Drug

Automatic drug use

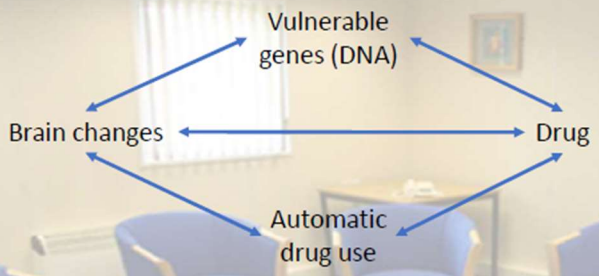


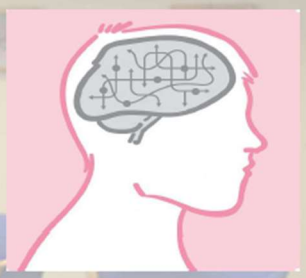
As part of this group which talked a lot about this view of addiction, how optimistic would you be about your chances of recovering control over your substance use?

1. Not at all optimistic
2. Slightly optimistic
3. Moderately optimistic
4. Very optimistic
5. Extremely optimistic

6

Biological View of Addiction



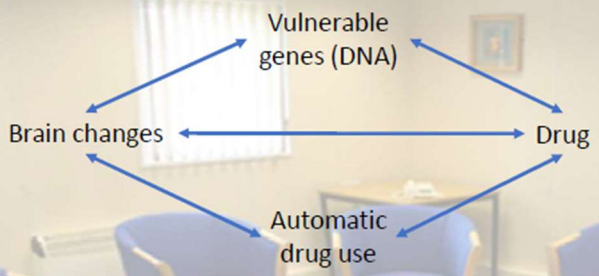


As part of this group which talked a lot about this view of addiction, how confident would you be in your ability to solve problems and improve your quality of life?

1. Not at all confident
2. Slightly confident
3. Moderately confident
4. Very confident
5. Extremely confident

7

Biological View of Addiction



As part of this group which talked a lot about this view of addiction, how likely do you think you would be to relapse to substance use?

1. Not at all likely
2. Slightly likely
3. Moderately likely
4. Very likely
5. Extremely likely

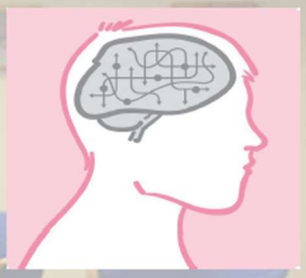
8

Biological View of Addiction

Vulnerable genes (DNA)

Brain changes ↔ Drug

Automatic drug use



As part of this group which talked a lot about this view of addiction, how much effort would you put into trying to quit?

1. No effort at all
2. A little effort
3. A moderate amount of effort
4. A lot of effort
5. A great deal of effort

9

Biological View of Addiction

Vulnerable genes (DNA)

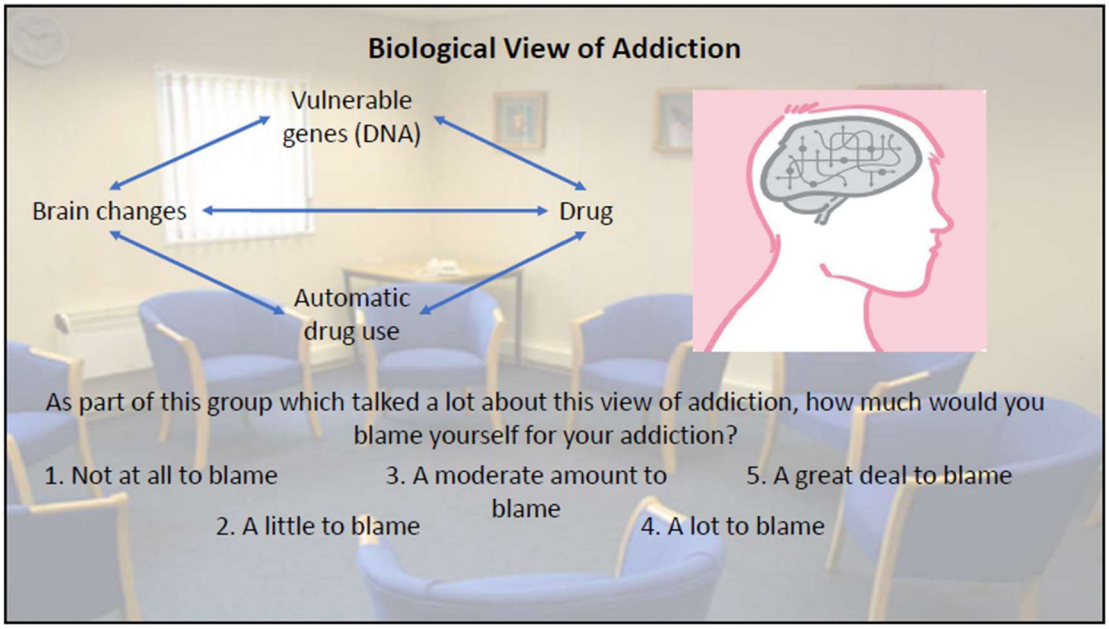
Brain changes ↔ Drug

Automatic drug use

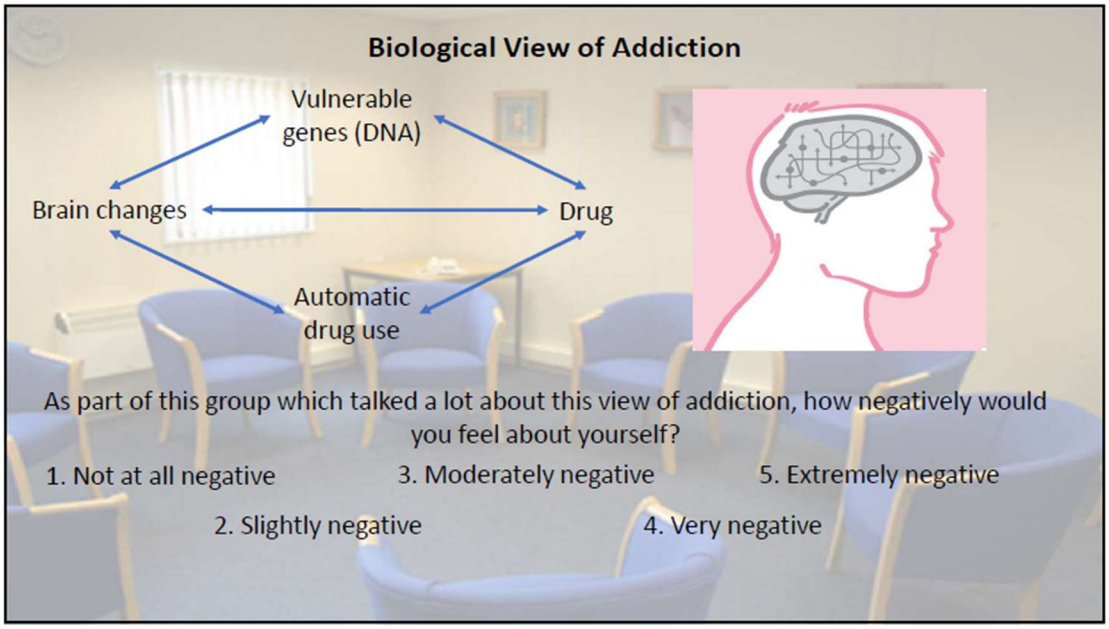
As part of this group which talked a lot about this view of addiction, how resilient would you feel in being able to cope with difficulties in your life?

1. Not at all resilient
2. Slightly resilient
3. Moderately resilient
4. Very resilient
5. Extremely resilient

10

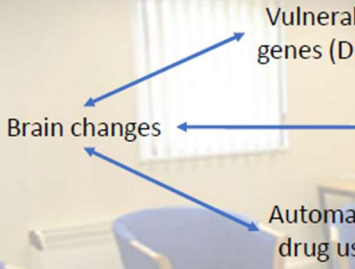

Biological View of Addiction
Vulnerable genes (DNA)
Brain changes
Drug
Automatic drug use
As part of this group which talked a lot about this view of addiction, how much would you blame yourself for your addiction?
1. Not at all to blame
2. A little to blame
3. A moderate amount to blame
4. A lot to blame
5. A great deal to blame

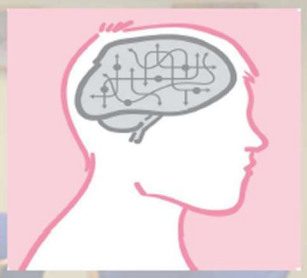
11


Biological View of Addiction
Vulnerable genes (DNA)
Brain changes
Drug
Automatic drug use
As part of this group which talked a lot about this view of addiction, how negatively would you feel about yourself?
1. Not at all negative
2. Slightly negative
3. Moderately negative
4. Very negative
5. Extremely negative

12

Biological View of Addiction



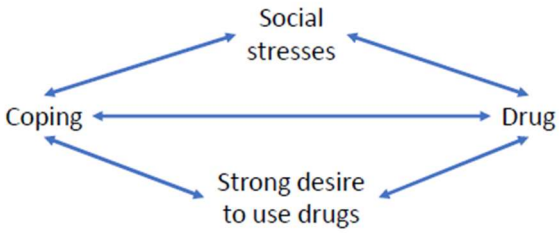


As part of this group which talked a lot about this view of addiction, how many days per week do you think you would use substances?

1. 0 Days
2. 1-2 Days
3. 3-4 Days
4. 5-6 Days
5. 7 Days

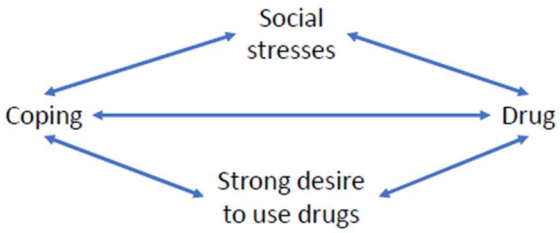
13

Social View of Addiction



1

Social View of Addiction



How much do you agree with the social view of addiction we just discussed?

1. Strongly disagree
2. Moderately disagree
3. Slightly disagree
4. Neither agree nor disagree
5. Slightly agree
6. Moderately agree
7. Strongly agree

2

Social View of Addiction

Social stresses

Coping ↔ Drug

Strong desire to use drugs

3

Social View of Addiction

Social stresses

Coping ↔ Drug

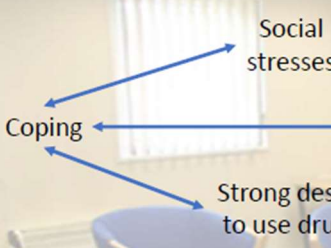
Strong desire to use drugs

If the leader or facilitator of the group talked a lot about this view of addiction, how supported would you feel by them?

1. Not at all supported
2. Slightly supported
3. Moderately supported
4. Very supported
5. Extremely supported

4

Social View of Addiction

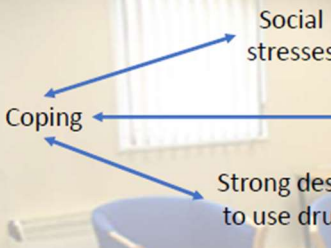


If other members of the group talked a lot about this view of addiction, how supported would you feel by them?

1. Not at all supported
2. Slightly supported
3. Moderately supported
4. Very supported
5. Extremely supported

5

Social View of Addiction

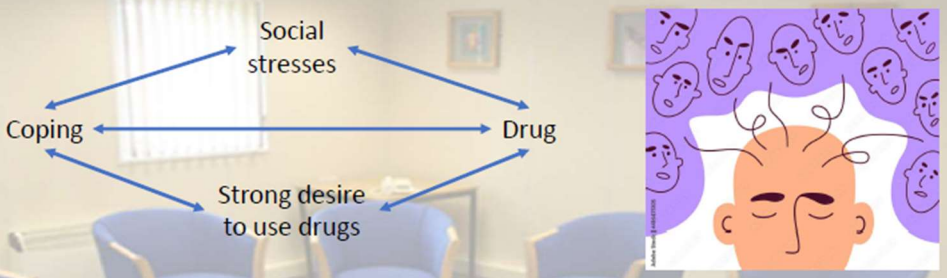


As part of this group which talked a lot about this view of addiction, how optimistic would you be about your chances of recovering control over your substance use?

1. Not at all optimistic
2. Slightly optimistic
3. Moderately optimistic
4. Very optimistic
5. Extremely optimistic

6

Social View of Addiction

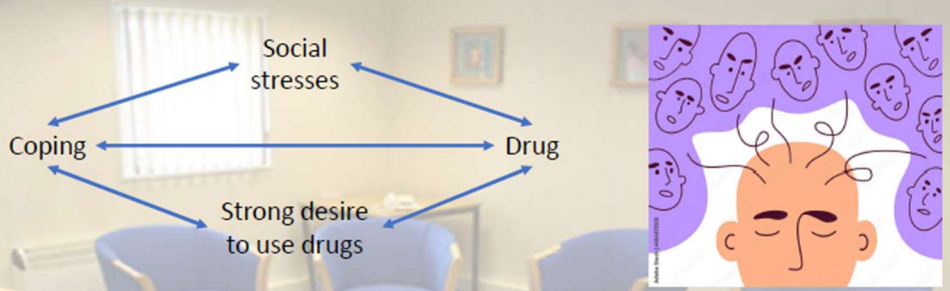


As part of this group which talked a lot about this view of addiction, how confident would you be in your ability to solve problems and improve your quality of life?

1. Not at all confident
2. Slightly confident
3. Moderately confident
4. Very confident
5. Extremely confident

7

Social View of Addiction

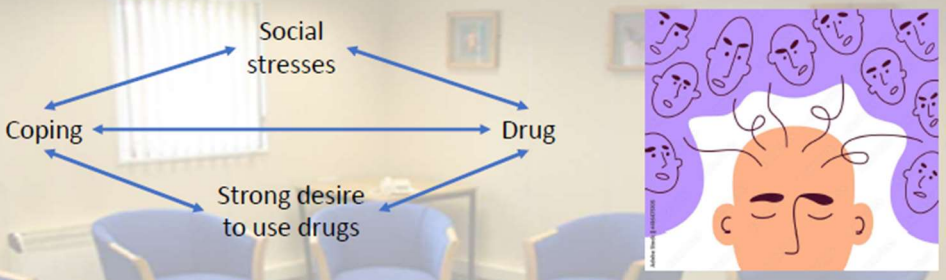


As part of this group which talked a lot about this view of addiction, how likely do you think you would be to relapse to substance use?

1. Not at all likely
2. Slightly likely
3. Moderately likely
4. Very likely
5. Extremely likely

8

Social View of Addiction

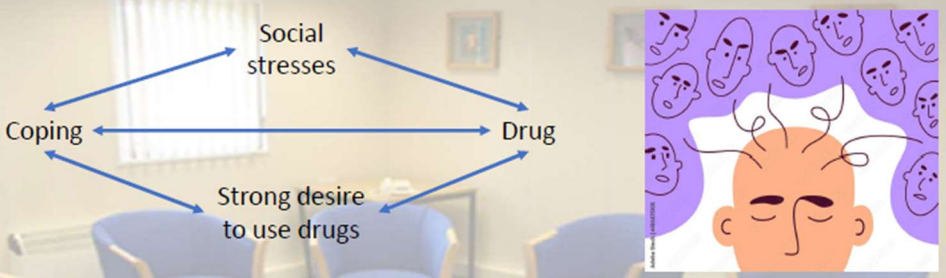


As part of this group which talked a lot about this view of addiction, how much effort would you put into trying to quit?

1. No effort at all
2. A little effort
3. A moderate amount of effort
4. A lot of effort
5. A great deal of effort

9

Social View of Addiction

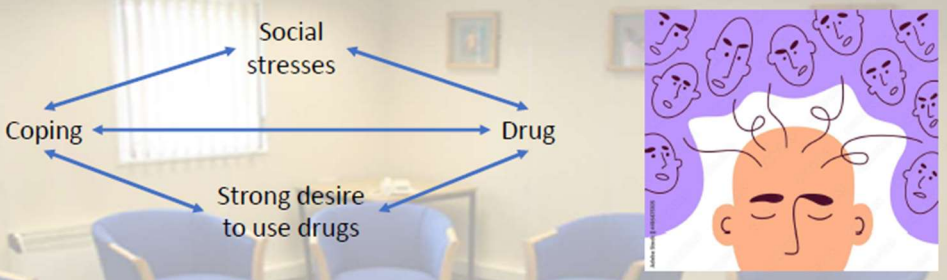


As part of this group which talked a lot about this view of addiction, how resilient would you feel in being able to cope with difficulties in your life?

1. Not at all resilient
2. Slightly resilient
3. Moderately resilient
4. Very resilient
5. Extremely resilient

10

Social View of Addiction

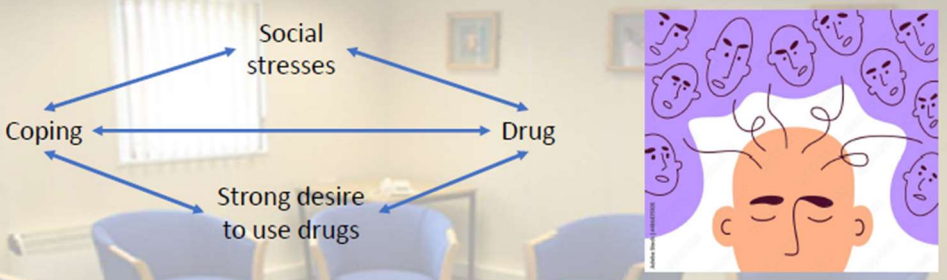


As part of this group which talked a lot about this view of addiction, how much would you blame yourself for your addiction?

1. Not at all to blame
2. A little to blame
3. A moderate amount to blame
4. A lot to blame
5. A great deal to blame

11

Social View of Addiction

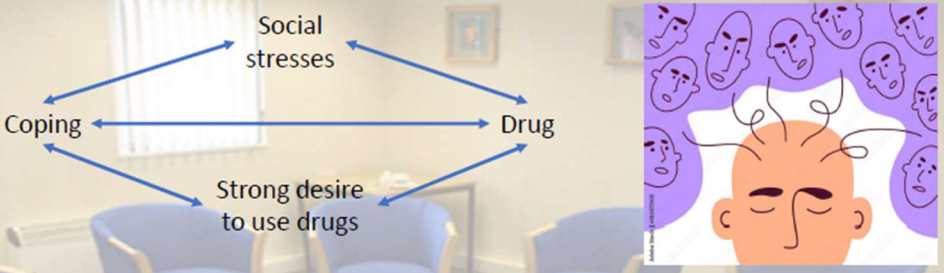


As part of this group which talked a lot about this view of addiction, how negatively would you feel about yourself?

1. Not at all negative
2. Slightly negative
3. Moderately negative
4. Very negative
5. Extremely negative

12

Social View of Addiction



As part of this group which talked a lot about this view of addiction, how many days per week do you think you would use substances?

1. 0 Days
2. 1-2 Days
3. 3-4 Days
4. 5-6 Days
5. 7 Days

13

Appendix H – Statistical Output from SPSS

Prior to conducting the inferential statistical analysis, the data was analysed to determine whether parametric assumptions had been met. Outliers were screened for using boxplots and z-scores and none were identified, with all z-scores falling below 3.29. Indications of normality were mixed with statistically significant K-S test results for the mean difference scores of theory agreement and the ten outcomes (theory agreement and outcomes 1-4 $p < .01$, outcome 5 $p < .05$, and outcomes 6-10 $p < .001$).

Further analysis was undertaken and z-scores were computed for skewness and kurtosis which were found to be non-significant for the majority of variables. The exception to this was self-blame which was negatively skewed z = 2.55, negative affect about self which was negatively skewed z= 2.24 and leptokurtic z = 2.84, and self-predicted substance use which was positively skewed z = -2.28 and leptokurtic z = 2.57. In addition, histograms and Q-Q plots were reviewed and appeared normal. Despite this mixed evidence, the central limit theorem (Kwak & Kim, 2017) suggests that the study sample size of 34 was reasonable grounds for accepting that the assumption of normality had been met.

Order effects were tested for using independent t-tests with addiction theory presentation order as the predictor. The majority of results were statistically non-significant with three exceptions. When the biological view had been presented first, participants scored higher on social condition negative affect about self (*MDiff* = 0.88, *SD* = 0.41), $t(32) = 2.154$, $p = .039$, and lower on biological condition recovery optimism (*MDiff* = -0.82, *SD* = 0.39), $t(32) = -2.096$, $p = .044$, and biological condition resilience to difficulties (*MDiff* = -0.88, *SD* = 0.32), $t(32) = -2.804$, $p = .009$. However, given this only reflected a small minority of the results it did not significantly compromise the validity of the study.

Case Processing Summary

	Cases					
	Valid		Missing		Total	
	N	Percent	N	Percent	N	Percent
Biological_Agree	34	100.0%	0	0.0%	34	100.0%
Biological_FacilitatorSupport	34	100.0%	0	0.0%	34	100.0%
Biological_MembersSupport	34	100.0%	0	0.0%	34	100.0%
Biological_RecoveryOptimism	34	100.0%	0	0.0%	34	100.0%
Biological_ConfidencetoSolveProbs	34	100.0%	0	0.0%	34	100.0%
Biological_LikelyRelapse	34	100.0%	0	0.0%	34	100.0%
Biological_QuitEffort	34	100.0%	0	0.0%	34	100.0%
Biological_ResilianceToDifficulties	34	100.0%	0	0.0%	34	100.0%
Biological_BlameYouself	34	100.0%	0	0.0%	34	100.0%
Biological_NegativeAboutSelf	34	100.0%	0	0.0%	34	100.0%
Biological_SubstanceUseDaysPerWeek	34	100.0%	0	0.0%	34	100.0%
Social_Agree	34	100.0%	0	0.0%	34	100.0%
Social_FacilitatorSupport	34	100.0%	0	0.0%	34	100.0%
Social_MembersSupport	34	100.0%	0	0.0%	34	100.0%
Social_RecoveryOptimism	34	100.0%	0	0.0%	34	100.0%
Social_ConfidencetoSolveProbs	34	100.0%	0	0.0%	34	100.0%
Social_LikelyRelapse	34	100.0%	0	0.0%	34	100.0%
Social_QuitEffort	34	100.0%	0	0.0%	34	100.0%
Social_ResilianceToDifficulties	34	100.0%	0	0.0%	34	100.0%
Social_BlameYouself	34	100.0%	0	0.0%	34	100.0%
Social_NegativeAboutSelf	34	100.0%	0	0.0%	34	100.0%
Social_SubstanceUseDaysPerWeek	34	100.0%	0	0.0%	34	100.0%

Descriptives

			Statistic	Std. Error
Biological_Agree	Mean		5.35	.206
	95% Confidence Interval for Mean	Lower Bound	4.93	
		Upper Bound	5.77	
	5% Trimmed Mean		5.40	
	Median		5.50	
	Variance		1.447	
	Std. Deviation		1.203	
	Minimum		2	
	Maximum		7	
	Range		5	
	Interquartile Range		2	
	Skewness		-.519	.403
	Kurtosis		.167	.788
Biological_FacilitatorSupport	Mean		2.97	.186
	95% Confidence Interval for Mean	Lower Bound	2.59	
		Upper Bound	3.35	
	5% Trimmed Mean		2.97	
	Median		3.00	
	Variance		1.181	
	Std. Deviation		1.087	
	Minimum		1	
	Maximum		5	
	Range		4	
	Interquartile Range		2	
	Skewness		.061	.403
	Kurtosis		-.398	.788
Biological_MembersSupport	Mean		3.03	.177
	95% Confidence Interval for Mean	Lower Bound	2.67	
		Upper Bound	3.39	
	5% Trimmed Mean		3.03	
	Median		3.00	
	Variance		1.060	
	Std. Deviation		1.029	
	Minimum		1	
	Maximum		5	
	Range		4	
	Interquartile Range		2	
	Skewness		-.239	.403
	Kurtosis		-.193	.788
Biological_RecoveryOptimism	Mean		2.35	.206
	95% Confidence Interval for Mean	Lower Bound	1.93	
		Upper Bound	2.77	
	5% Trimmed Mean		2.28	

			Statistic	Std. Error
	Median		2.00	
	Variance		1.447	
	Std. Deviation		1.203	
	Minimum		1	
	Maximum		5	
	Range		4	
	Interquartile Range		2	
	Skewness		.590	.403
	Kurtosis		-.470	.788
Biological_ConfidencetoSolveProbs	Mean		2.29	.187
	95% Confidence Interval for Mean	Lower Bound	1.91	
		Upper Bound	2.67	
	5% Trimmed Mean		2.24	
	Median		2.00	
	Variance		1.184	
	Std. Deviation		1.088	
	Minimum		1	
	Maximum		5	
	Range		4	
	Interquartile Range		2	
	Skewness		.417	.403
	Kurtosis		-.443	.788
Biological_LikelyRelapse	Mean		3.24	.189
	95% Confidence Interval for Mean	Lower Bound	2.85	
		Upper Bound	3.62	
	5% Trimmed Mean		3.26	
	Median		3.00	
	Variance		1.216	
	Std. Deviation		1.103	
	Minimum		1	
	Maximum		5	
	Range		4	
	Interquartile Range		1	
	Skewness		-.210	.403
	Kurtosis		-.027	.788
Biological_QuitEffort	Mean		3.03	.191
	95% Confidence Interval for Mean	Lower Bound	2.64	
		Upper Bound	3.42	
	5% Trimmed Mean		3.01	
	Median		3.00	
	Variance		1.242	
	Std. Deviation		1.114	
	Minimum		1	

			Statistic	Std. Error
	Maximum		5	
	Range		4	
	Interquartile Range		2	
	Skewness		.358	.403
	Kurtosis		-.959	.788
Biological_ResilianceToDifficulties	Mean		2.21	.173
	95% Confidence Interval for Mean	Lower Bound	1.85	
		Upper Bound	2.56	
	5% Trimmed Mean		2.14	
	Median		2.00	
	Variance		1.017	
	Std. Deviation		1.008	
	Minimum		1	
	Maximum		5	
	Range		4	
	Interquartile Range		2	
	Skewness		.690	.403
	Kurtosis		.373	.788
Biological_BlameYouself	Mean		3.12	.249
	95% Confidence Interval for Mean	Lower Bound	2.61	
		Upper Bound	3.62	
	5% Trimmed Mean		3.13	
	Median		3.00	
	Variance		2.107	
	Std. Deviation		1.452	
	Minimum		1	
	Maximum		5	
	Range		4	
	Interquartile Range		2	
	Skewness		-.217	.403
	Kurtosis		-1.312	.788
Biological_NegativeAboutSelf	Mean		2.91	.208
	95% Confidence Interval for Mean	Lower Bound	2.49	
		Upper Bound	3.34	
	5% Trimmed Mean		2.90	
	Median		3.00	
	Variance		1.477	
	Std. Deviation		1.215	
	Minimum		1	
	Maximum		5	
	Range		4	
	Interquartile Range		2	
	Skewness		-.037	.403

			Statistic	Std. Error
	Kurtosis		-.927	.788
Biological_SubstanceUseDaysPerWeek	Mean		3.41	.232
	95% Confidence Interval for Mean	Lower Bound	2.94	
		Upper Bound	3.88	
	5% Trimmed Mean		3.46	
	Median		3.00	
	Variance		1.825	
	Std. Deviation		1.351	
	Minimum		1	
	Maximum		5	
	Range		4	
	Interquartile Range		2	
	Skewness		-.350	.403
	Kurtosis		-.938	.788
Social_Agree	Mean		5.94	.179
	95% Confidence Interval for Mean	Lower Bound	5.58	
		Upper Bound	6.31	
	5% Trimmed Mean		5.99	
	Median		6.00	
	Variance		1.087	
	Std. Deviation		1.043	
	Minimum		4	
	Maximum		7	
	Range		3	
	Interquartile Range		2	
	Skewness		-.729	.403
	Kurtosis		-.550	.788
Social_FacilitatorSupport	Mean		3.44	.135
	95% Confidence Interval for Mean	Lower Bound	3.17	
		Upper Bound	3.72	
	5% Trimmed Mean		3.43	
	Median		3.50	
	Variance		.618	
	Std. Deviation		.786	
	Minimum		2	
	Maximum		5	
	Range		3	
	Interquartile Range		1	
	Skewness		-.192	.403
	Kurtosis		-.337	.788
Social_MembersSupport	Mean		3.47	.135
	95% Confidence Interval for Mean	Lower Bound	3.20	
		Upper Bound	3.75	

			Statistic	Std. Error
	5% Trimmed Mean		3.49	
	Median		4.00	
	Variance		.620	
	Std. Deviation		.788	
	Minimum		2	
	Maximum		5	
	Range		3	
	Interquartile Range		1	
	Skewness		-.688	.403
	Kurtosis		-.385	.788
Social_RecoveryOptimism	Mean		2.85	.170
	95% Confidence Interval for Mean	Lower Bound	2.51	
		Upper Bound	3.20	
	5% Trimmed Mean		2.84	
	Median		3.00	
	Variance		.978	
	Std. Deviation		.989	
	Minimum		1	
	Maximum		5	
	Range		4	
	Interquartile Range		1	
	Skewness		.111	.403
	Kurtosis		.158	.788
Social_ConfidencetoSolvePr obs	Mean		2.56	.165
	95% Confidence Interval for Mean	Lower Bound	2.22	
		Upper Bound	2.89	
	5% Trimmed Mean		2.53	
	Median		2.00	
	Variance		.921	
	Std. Deviation		.960	
	Minimum		1	
	Maximum		5	
	Range		4	
	Interquartile Range		1	
	Skewness		.588	.403
	Kurtosis		-.004	.788
Social_LikelyRelapse	Mean		2.53	.142
	95% Confidence Interval for Mean	Lower Bound	2.24	
		Upper Bound	2.82	
	5% Trimmed Mean		2.53	
	Median		2.50	
	Variance		.681	
	Std. Deviation		.825	

			Statistic	Std. Error
	Minimum		1	
	Maximum		4	
	Range		3	
	Interquartile Range		1	
	Skewness		.073	.403
	Kurtosis		-.404	.788
Social_QuitEffort	Mean		3.38	.169
	95% Confidence Interval for Mean	Lower Bound	3.04	
		Upper Bound	3.73	
	5% Trimmed Mean		3.40	
	Median		3.00	
	Variance		.971	
	Std. Deviation		.985	
	Minimum		1	
	Maximum		5	
	Range		4	
	Interquartile Range		1	
	Skewness		-.257	.403
	Kurtosis		-.212	.788
Social_ResilianceToDifficulties	Mean		2.53	.170
	95% Confidence Interval for Mean	Lower Bound	2.18	
		Upper Bound	2.88	
	5% Trimmed Mean		2.50	
	Median		3.00	
	Variance		.984	
	Std. Deviation		.992	
	Minimum		1	
	Maximum		5	
	Range		4	
	Interquartile Range		1	
	Skewness		.112	.403
	Kurtosis		-.081	.788
Social_BlameYouself	Mean		3.71	.205
	95% Confidence Interval for Mean	Lower Bound	3.29	
		Upper Bound	4.12	
	5% Trimmed Mean		3.76	
	Median		4.00	
	Variance		1.426	
	Std. Deviation		1.194	
	Minimum		1	
	Maximum		5	
	Range		4	
	Interquartile Range		2	

			Statistic	Std. Error
	Skewness		-.522	.403
	Kurtosis		-.847	.788
Social_NegativeAboutSelf	Mean		2.85	.216
	95% Confidence Interval for Mean	Lower Bound	2.41	
		Upper Bound	3.29	
	5% Trimmed Mean		2.84	
	Median		3.00	
	Variance		1.584	
	Std. Deviation		1.258	
	Minimum		1	
	Maximum		5	
	Range		4	
	Interquartile Range		2	
	Skewness		-.191	.403
	Kurtosis		-1.160	.788
Social_SubstanceUseDaysPerWeek	Mean		3.09	.255
	95% Confidence Interval for Mean	Lower Bound	2.57	
		Upper Bound	3.61	
	5% Trimmed Mean		3.10	
	Median		3.00	
	Variance		2.204	
	Std. Deviation		1.485	
	Minimum		1	
	Maximum		5	
	Range		4	
	Interquartile Range		3	
	Skewness		.135	.403
	Kurtosis		-1.462	.788

Tests of Normality

	Kolmogorov-Smirnov[a]			Shapiro-Wilk		
	Statistic	df	Sig.	Statistic	df	Sig.
Biological_Agree	.205	34	<.001	.899	34	.004
Biological_FacilitatorSupport	.195	34	.002	.920	34	.016
Biological_MembersSupport	.224	34	<.001	.909	34	.008
Biological_RecoveryOptimism	.204	34	.001	.882	34	.002
Biological_ConfidencetoSolveProbs	.183	34	.005	.882	34	.002
Biological_LikelyRelapse	.239	34	<.001	.887	34	.002
Biological_QuitEffort	.234	34	<.001	.877	34	.001
Biological_ResilianceToDifficulties	.228	34	<.001	.875	34	.001
Biological_BlameYouself	.199	34	.002	.879	34	.001
Biological_NegativeAboutSelf	.168	34	.016	.915	34	.012
Biological_SubstanceUseDaysPerWeek	.174	34	.010	.881	34	.001
Social_Agree	.258	34	<.001	.820	34	<.001
Social_FacilitatorSupport	.261	34	<.001	.855	34	<.001
Social_MembersSupport	.338	34	<.001	.788	34	<.001
Social_RecoveryOptimism	.236	34	<.001	.901	34	.005
Social_ConfidencetoSolveProbs	.279	34	<.001	.875	34	.001
Social_LikelyRelapse	.239	34	<.001	.870	34	<.001
Social_QuitEffort	.205	34	<.001	.907	34	.007
Social_ResilianceToDifficulties	.241	34	<.001	.889	34	.002
Social_BlameYouself	.215	34	<.001	.867	34	<.001
Social_NegativeAboutSelf	.201	34	.001	.887	34	.002
Social_SubstanceUseDaysPerWeek	.209	34	<.001	.855	34	<.001

a. Lilliefors Significance Correction

T-Test

Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	Biological_Agree	5.35	34	1.203	.206
	Social_Agree	5.94	34	1.043	.179
Pair 2	Biological_FacilitatorSupport	2.97	34	1.087	.186
	Social_FacilitatorSupport	3.44	34	.786	.135
Pair 3	Biological_MembersSupport	3.03	34	1.029	.177
	Social_MembersSupport	3.47	34	.788	.135
Pair 4	Biological_RecoveryOptimism	2.35	34	1.203	.206
	Social_RecoveryOptimism	2.85	34	.989	.170
Pair 5	Biological_ConfidencetoSolveProbs	2.29	34	1.088	.187
	Social_ConfidencetoSolveProbs	2.56	34	.960	.165
Pair 6	Biological_LikelyRelapse	3.24	34	1.103	.189
	Social_LikelyRelapse	2.53	34	.825	.142
Pair 7	Biological_QuitEffort	3.03	34	1.114	.191
	Social_QuitEffort	3.38	34	.985	.169
Pair 8	Biological_ResilianceToDifficulties	2.21	34	1.008	.173
	Social_ResilianceToDifficulties	2.53	34	.992	.170
Pair 9	Biological_BlameYouself	3.12	34	1.452	.249
	Social_BlameYouself	3.71	34	1.194	.205
Pair 10	Biological_NegativeAboutSelf	2.91	34	1.215	.208
	Social_NegativeAboutSelf	2.85	34	1.258	.216
Pair 11	Biological_SubstanceUseDaysPerWeek	3.41	34	1.351	.232
	Social_SubstanceUseDaysPerWeek	3.09	34	1.485	.255

Paired Samples Correlations

		N	Correlation	Significance One-Sided p	Significance Two-Sided p
Pair 1	Biological_Agree & Social_Agree	34	.017	.462	.924
Pair 2	Biological_FacilitatorSupport & Social_FacilitatorSupport	34	.122	.246	.492
Pair 3	Biological_MembersSupport & Social_MembersSupport	34	.244	.082	.164
Pair 4	Biological_RecoveryOptimism & Social_RecoveryOptimism	34	.172	.165	.330
Pair 5	Biological_ConfidencetoSolveProbs & Social_ConfidencetoSolveProbs	34	.215	.111	.222
Pair 6	Biological_LikelyRelapse & Social_LikelyRelapse	34	.159	.185	.370
Pair 7	Biological_QuitEffort & Social_QuitEffort	34	.348	.022	.044
Pair 8	Biological_ResilianceToDifficulties & Social_ResilianceToDifficulties	34	.221	.105	.209
Pair 9	Biological_BlameYouself & Social_BlameYouself	34	.405	.009	.017
Pair 10	Biological_NegativeAboutSelf & Social_NegativeAboutSelf	34	.487	.002	.004
Pair 11	Biological_SubstanceUseDaysPerWeek & Social_SubstanceUseDaysPerWeek	34	.827	<.001	<.001

Paired Samples Test

		Paired Differences			
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the ... Lower
Pair 1	Biological_Agree - Social_Agree	-.588	1.579	.271	-1.139
Pair 2	Biological_FacilitatorSupport - Social_FacilitatorSupport	-.471	1.261	.216	-.911
Pair 3	Biological_MembersSupport - Social_MembersSupport	-.441	1.133	.194	-.837
Pair 4	Biological_RecoveryOptimism - Social_RecoveryOptimism	-.500	1.420	.243	-.995
Pair 5	Biological_ConfidencetoSolveProbs - Social_ConfidencetoSolveProbs	-.265	1.286	.221	-.714
Pair 6	Biological_LikelyRelapse - Social_LikelyRelapse	.706	1.268	.217	.263
Pair 7	Biological_QuitEffort - Social_QuitEffort	-.353	1.203	.206	-.773
Pair 8	Biological_ResilianceToDifficulties - Social_ResilianceToDifficulties	-.324	1.249	.214	-.759
Pair 9	Biological_BlameYouself - Social_BlameYouself	-.588	1.459	.250	-1.097
Pair 10	Biological_NegativeAboutSelf - Social_NegativeAboutSelf	.059	1.254	.215	-.379
Pair 11	Biological_SubstanceUseDaysPerWeek - Social_SubstanceUseDaysPerWeek	.324	.843	.145	.029

Paired Samples Test

		Paired ... 95% Confidence Interval of the ... Upper	t	df	Significance One-Sided p
Pair 1	Biological_Agree - Social_Agree	-.037	-2.173	33	.019
Pair 2	Biological_FacilitatorSupport - Social_FacilitatorSupport	-.031	-2.176	33	.018
Pair 3	Biological_MembersSupport - Social_MembersSupport	-.046	-2.270	33	.015
Pair 4	Biological_RecoveryOptimism - Social_RecoveryOptimism	-.005	-2.054	33	.024
Pair 5	Biological_ConfidencetoSolveProbs - Social_ConfidencetoSolvePrObs	.184	-1.200	33	.119
Pair 6	Biological_LikelyRelapse - Social_LikelyRelapse	1.148	3.246	33	.001
Pair 7	Biological_QuitEffort - Social_QuitEffort	.067	-1.711	33	.048
Pair 8	Biological_ResilianceToDifficulties - Social_ResilianceToDifficulties	.112	-1.511	33	.070
Pair 9	Biological_BlameYouself - Social_BlameYouself	-.079	-2.351	33	.012
Pair 10	Biological_NegativeAboutSelf - Social_NegativeAboutSelf	.496	.274	33	.393
Pair 11	Biological_SubstanceUseDaysPerWeek - Social_SubstanceUseDaysPerWeek	.618	2.238	33	.016

Paired Samples Test

		Significance
		Two-Sided p
Pair 1	Biological_Agree - Social_Agree	.037
Pair 2	Biological_FacilitatorSupport - Social_FacilitatorSupport	.037
Pair 3	Biological_MembersSupport - Social_MembersSupport	.030
Pair 4	Biological_RecoveryOptimism - Social_RecoveryOptimism	.048
Pair 5	Biological_ConfidencetoSolveProbs - Social_ConfidencetoSolveProbs	.239
Pair 6	Biological_LikelyRelapse - Social_LikelyRelapse	.003
Pair 7	Biological_QuitEffort - Social_QuitEffort	.097
Pair 8	Biological_ResilianceToDifficulties - Social_ResilianceToDifficulties	.140
Pair 9	Biological_BlameYouself - Social_BlameYouself	.025
Pair 10	Biological_NegativeAboutSelf - Social_NegativeAboutSelf	.786
Pair 11	Biological_SubstanceUseDaysPerWeek - Social_SubstanceUseDaysPerWeek	.032

Paired Samples Effect Sizes

			Standardizer[a]	Point Estimate	95% ... Lower
Pair 1	Biological_Agree - Social_Agree	Cohen's d	1.579	-.373	-.718
		Hedges' correction	1.616	-.364	-.701
Pair 2	Biological_FacilitatorSupport - Social_FacilitatorSupport	Cohen's d	1.261	-.373	-.718
		Hedges' correction	1.291	-.365	-.702
Pair 3	Biological_MembersSupport - Social_MembersSupport	Cohen's d	1.133	-.389	-.735
		Hedges' correction	1.160	-.380	-.719
Pair 4	Biological_RecoveryOptimism - Social_RecoveryOptimism	Cohen's d	1.420	-.352	-.696
		Hedges' correction	1.453	-.344	-.680
Pair 5	Biological_ConfidencetoSolveProbs - Social_ConfidencetoSolveProbs	Cohen's d	1.286	-.206	-.544
		Hedges' correction	1.317	-.201	-.532
Pair 6	Biological_LikelyRelapse - Social_LikelyRelapse	Cohen's d	1.268	.557	.191
		Hedges' correction	1.298	.544	.187
Pair 7	Biological_QuitEffort - Social_QuitEffort	Cohen's d	1.203	-.293	-.635
		Hedges' correction	1.231	-.287	-.620
Pair 8	Biological_ResilianceToDifficulties - Social_ResilianceToDifficulties	Cohen's d	1.249	-.259	-.599
		Hedges' correction	1.278	-.253	-.585
Pair 9	Biological_BlameYouself - Social_BlameYouself	Cohen's d	1.459	-.403	-.750
		Hedges' correction	1.493	-.394	-.733
Pair 10	Biological_NegativeAboutSelf - Social_NegativeAboutSelf	Cohen's d	1.254	.047	-.290
		Hedges' correction	1.283	.046	-.283
Pair 11	Biological_SubstanceUseDaysPerWeek - Social_SubstanceUseDaysPerWeek	Cohen's d	.843	.384	.033
		Hedges' correction	.863	.375	.032

Paired Samples Effect Sizes

			95% ... Upper
Pair 1	Biological_Agree - Social_Agree	Cohen's d	-.022
		Hedges' correction	-.022
Pair 2	Biological_FacilitatorSupport - Social_FacilitatorSupport	Cohen's d	-.023
		Hedges' correction	-.022
Pair 3	Biological_MembersSupport - Social_MembersSupport	Cohen's d	-.038
		Hedges' correction	-.037
Pair 4	Biological_RecoveryOptimism - Social_RecoveryOptimism	Cohen's d	-.003
		Hedges' correction	-.003
Pair 5	Biological_ConfidencetoSolveProbs - Social_ConfidencetoSolveProbs	Cohen's d	.136
		Hedges' correction	.132
Pair 6	Biological_LikelyRelapse - Social_LikelyRelapse	Cohen's d	.915
		Hedges' correction	.894
Pair 7	Biological_QuitEffort - Social_QuitEffort	Cohen's d	.052
		Hedges' correction	.051
Pair 8	Biological_ResilianceToDifficulties - Social_ResilianceToDifficulties	Cohen's d	.085
		Hedges' correction	.083
Pair 9	Biological_BlameYouself - Social_BlameYouself	Cohen's d	-.051
		Hedges' correction	-.049
Pair 10	Biological_NegativeAboutSelf - Social_NegativeAboutSelf	Cohen's d	.383
		Hedges' correction	.374
Pair 11	Biological_SubstanceUseDaysPerWeek - Social_SubstanceUseDaysPerWeek	Cohen's d	.730
		Hedges' correction	.713

a. The denominator used in estimating the effect sizes.
Cohen's d uses the sample standard deviation of the mean difference.
Hedges' correction uses the sample standard deviation of the mean difference, plus a correction factor.

Hypothesis Test Summary

	Null Hypothesis	Test	Sig.[a,b]
1	The median of differences between Biological_Agree and Social_Agree equals 0.	Related-Samples Wilcoxon Signed Rank Test	.038

Hypothesis Test Summary

	Decision
1	Reject the null hypothesis.

a. The significance level is .050.

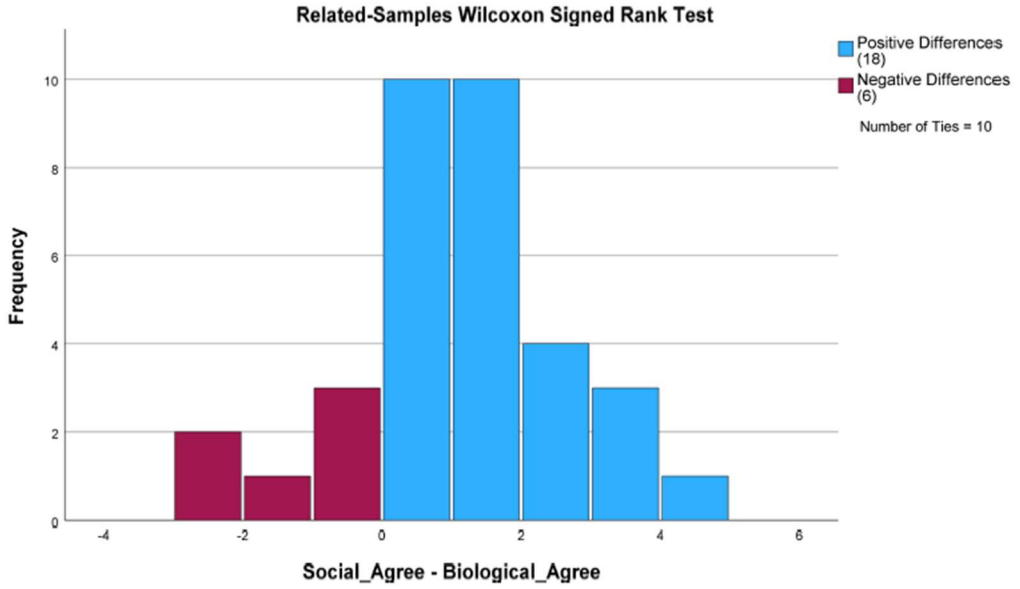
b. Asymptotic significance is displayed.

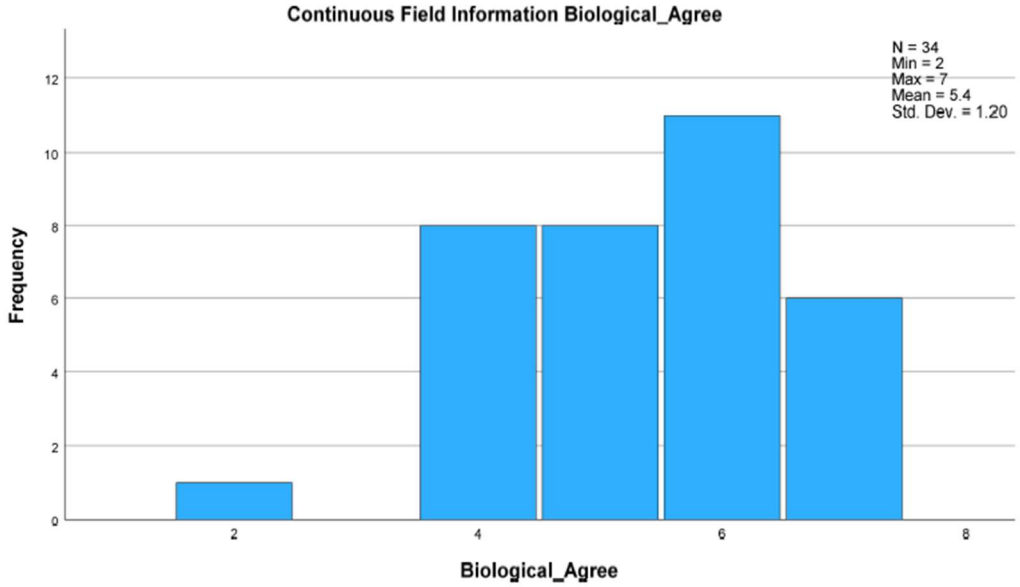
Related-Samples Wilcoxon Signed Rank Test

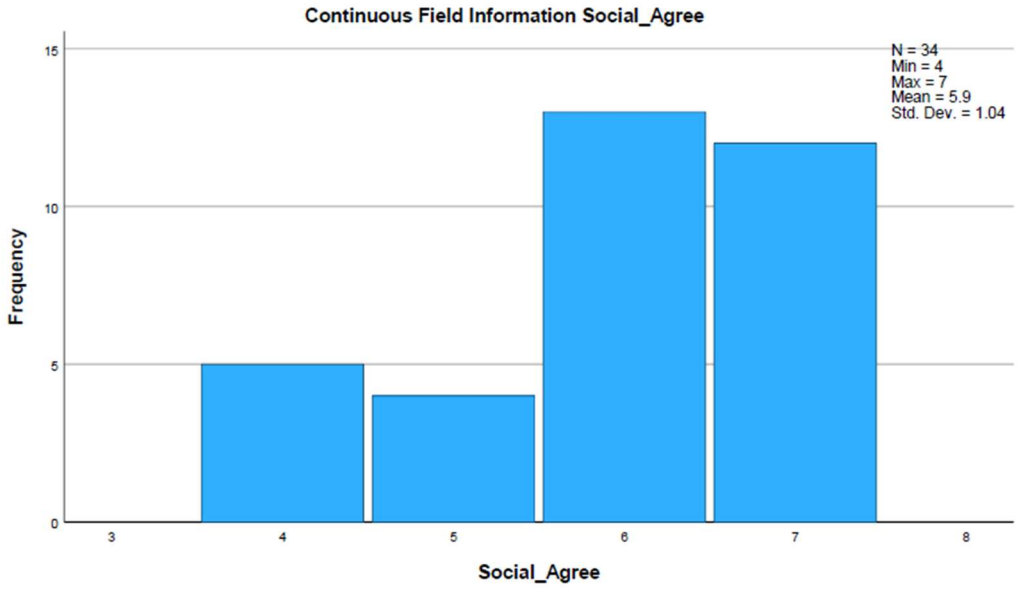
Biological_Agree, Social_Agree

Related-Samples Wilcoxon Signed Rank Test Summary

Total N	34
Test Statistic	221.000
Standard Error	34.271
Standardized Test Statistic	2.072
Asymptotic Sig.(2-sided test)	.038


Related-Samples Wilcoxon Signed Rank Test
Positive Differences (18)
Negative Differences (6)
Number of Ties = 10
Frequency
Social_Agree - Biological_Agree


Continuous Field Information Biological_Agree
N = 34
Min = 2
Max = 7
Mean = 5.4
Std. Dev. = 1.20
Frequency
Biological_Agree



Nonparametric Tests

Hypothesis Test Summary

	Null Hypothesis	Test	Sig.[a,b]
1	The median of differences between Biological_FacilitatorSupport and Social_FacilitatorSupport equals 0.	Related-Samples Wilcoxon Signed Rank Test	.050

Hypothesis Test Summary

	Decision
1	Retain the null hypothesis.

a. The significance level is .050.

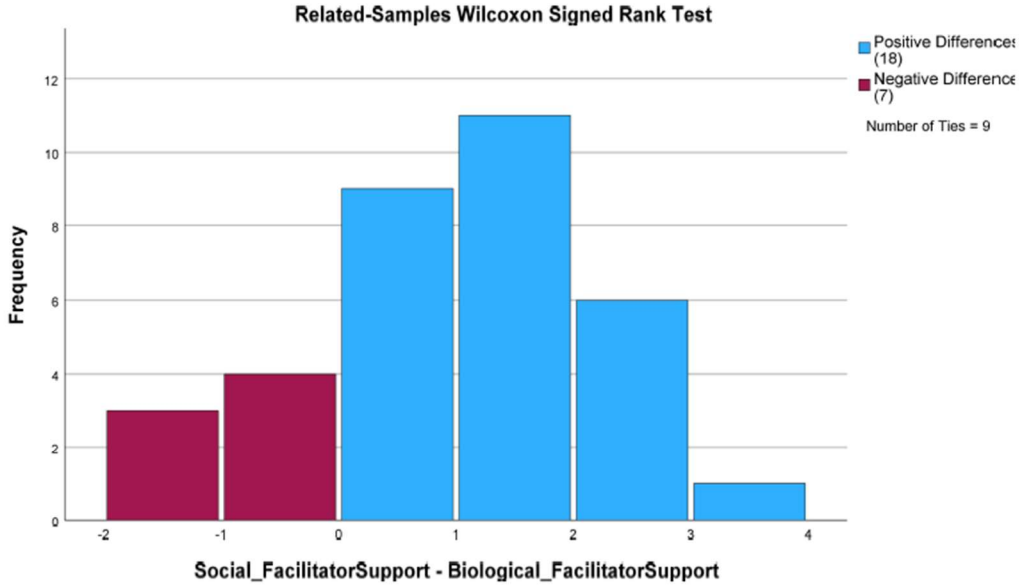
b. Asymptotic significance is displayed.

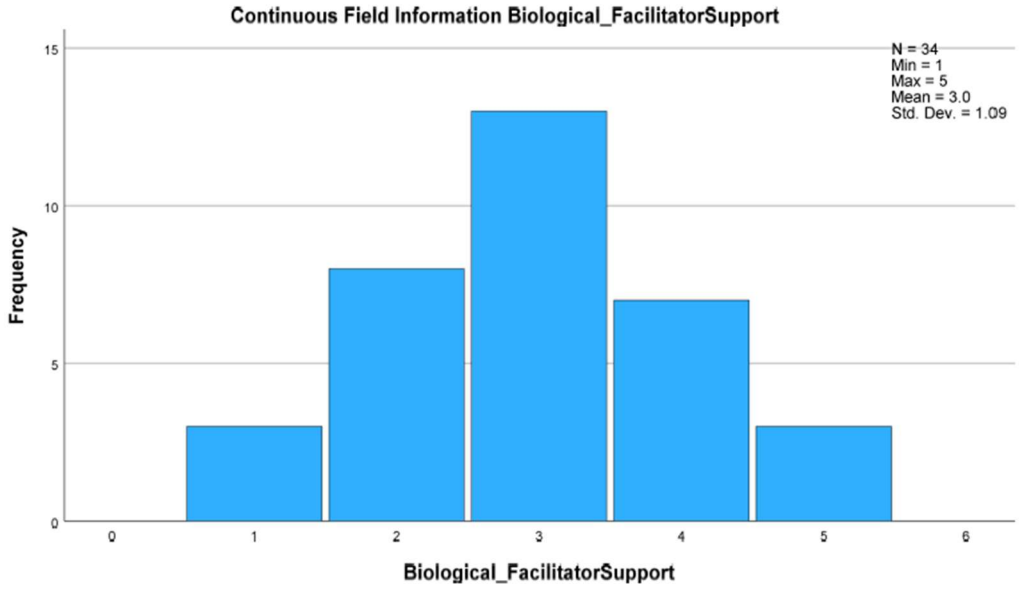
Related-Samples Wilcoxon Signed Rank Test

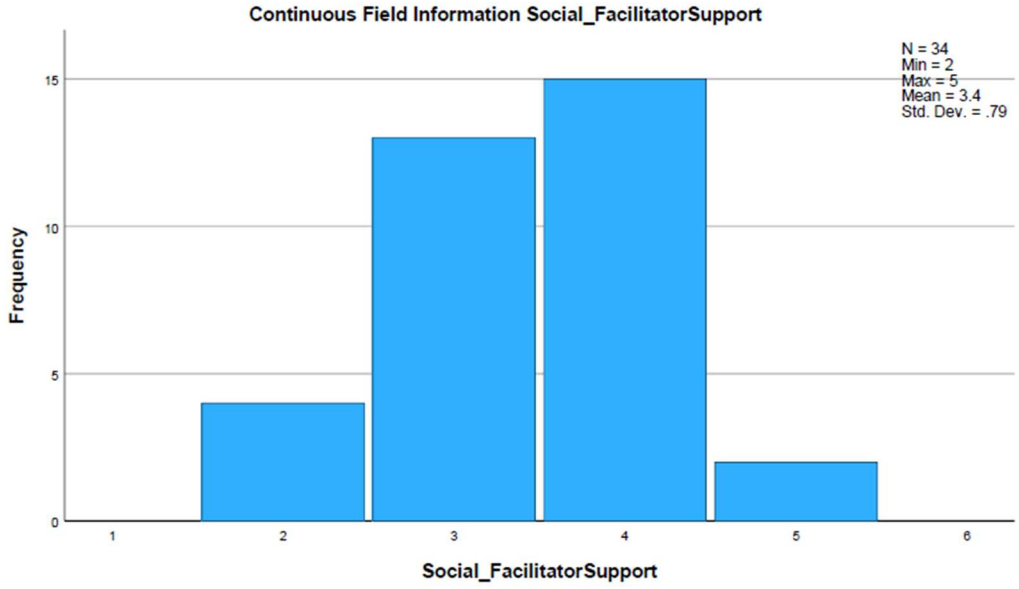
Biological_FacilitatorSupport, Social_FacilitatorSupport

Related-Samples Wilcoxon Signed Rank Test Summary

Total N	34
Test Statistic	233.000
Standard Error	36.003
Standardized Test Statistic	1.958
Asymptotic Sig.(2-sided test)	.050







Nonparametric Tests

Hypothesis Test Summary

	Null Hypothesis	Test	Sig.[a,b]
1	The median of differences between Biological_MembersSupport and Social_MembersSupport equals 0.	Related-Samples Wilcoxon Signed Rank Test	.032

Hypothesis Test Summary

	Decision
1	Reject the null hypothesis.

a. The significance level is .050.

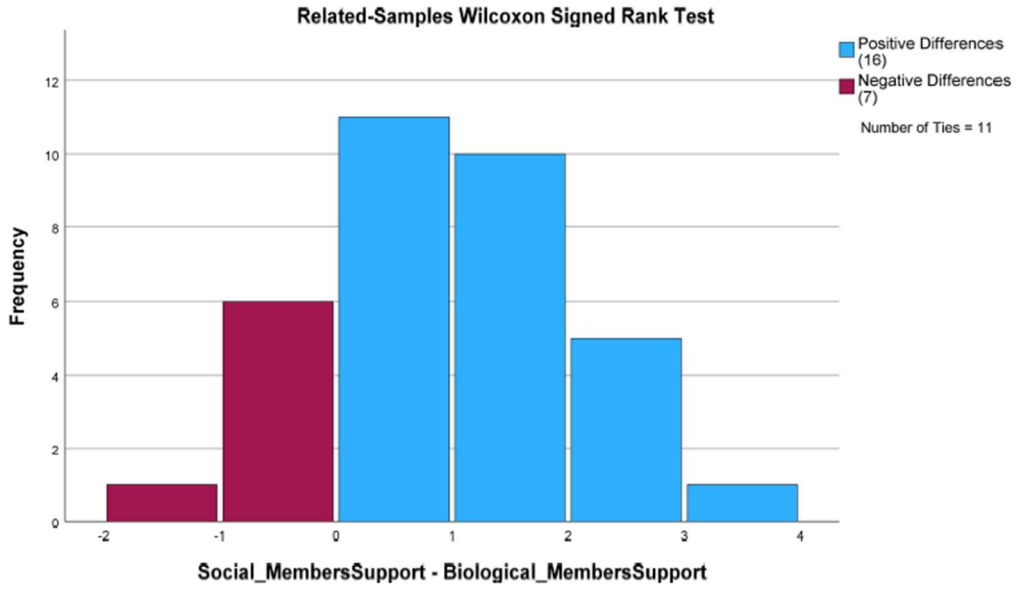
b. Asymptotic significance is displayed.

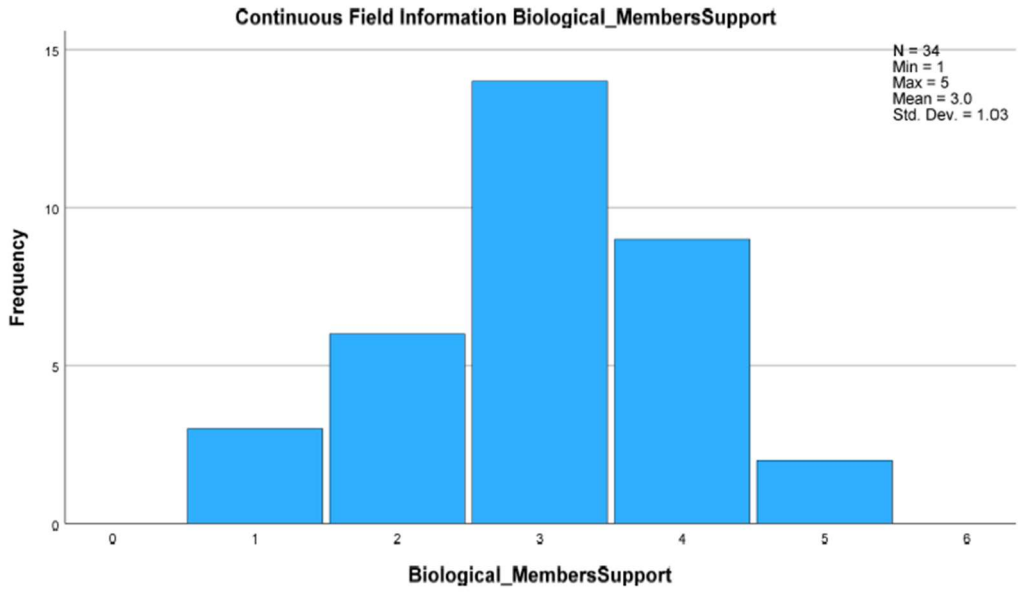
Related-Samples Wilcoxon Signed Rank Test

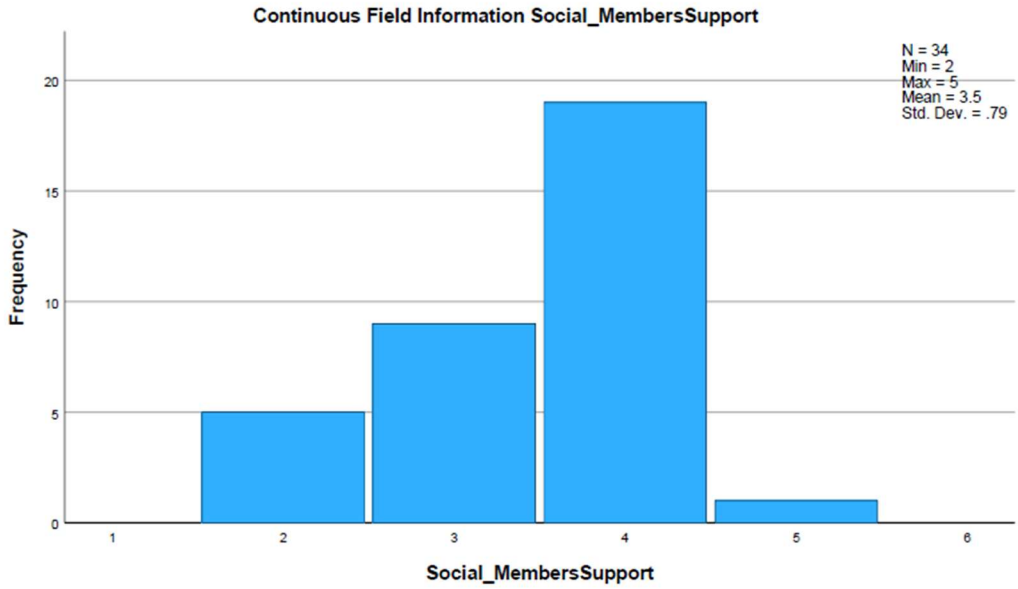
Biological_MembersSupport, Social_MembersSupport

Related-Samples Wilcoxon Signed Rank Test Summary

Total N	34
Test Statistic	205.500
Standard Error	31.490
Standardized Test Statistic	2.144
Asymptotic Sig.(2-sided test)	.032







Nonparametric Tests

Hypothesis Test Summary

	Null Hypothesis	Test	Sig.[a,b]
1	The median of differences between Biological_RecoveryOptimism and Social_RecoveryOptimism equals 0.	Related-Samples Wilcoxon Signed Rank Test	.048

Hypothesis Test Summary

	Decision
1	Reject the null hypothesis.

a. The significance level is .050.

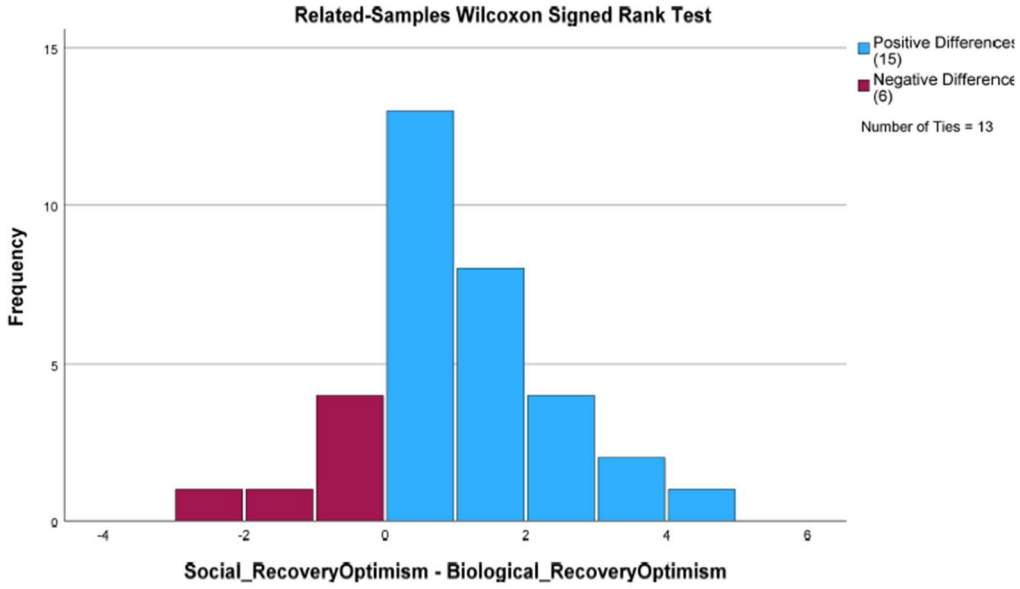
b. Asymptotic significance is displayed.

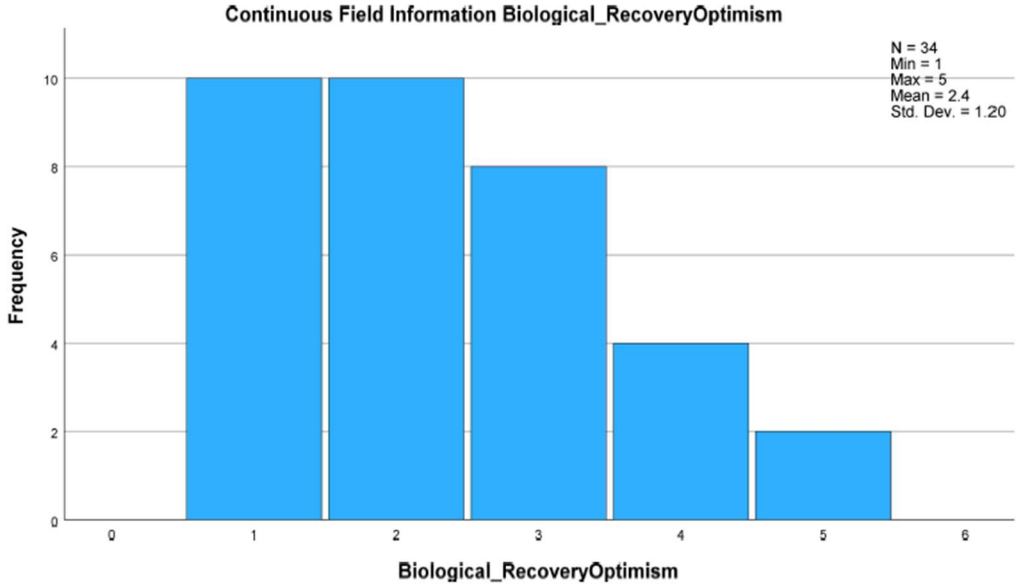
Related-Samples Wilcoxon Signed Rank Test

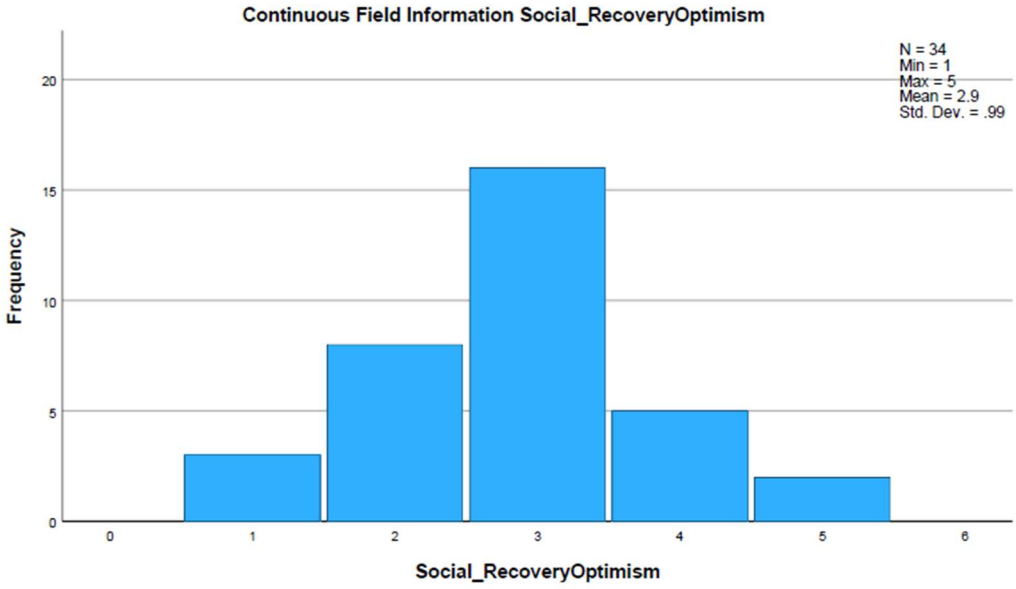
Biological_RecoveryOptimism, Social_RecoveryOptimism

Related-Samples Wilcoxon Signed Rank Test Summary

Total N	34
Test Statistic	171.000
Standard Error	28.089
Standardized Test Statistic	1.976
Asymptotic Sig.(2-sided test)	.048







Nonparametric Tests

Hypothesis Test Summary

	Null Hypothesis	Test	Sig.[a,b]
1	The median of differences between Biological_ConfidencetoSolveProbs and Social_ConfidencetoSolveProbs equals 0.	Related-Samples Wilcoxon Signed Rank Test	.278

Hypothesis Test Summary

	Decision
1	Retain the null hypothesis.

a. The significance level is .050.

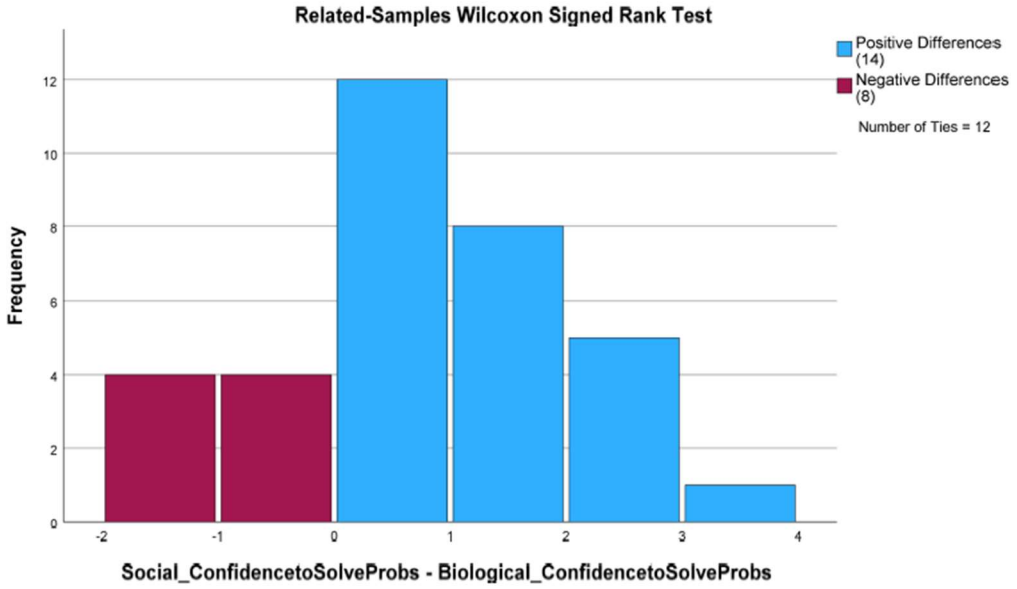
b. Asymptotic significance is displayed.

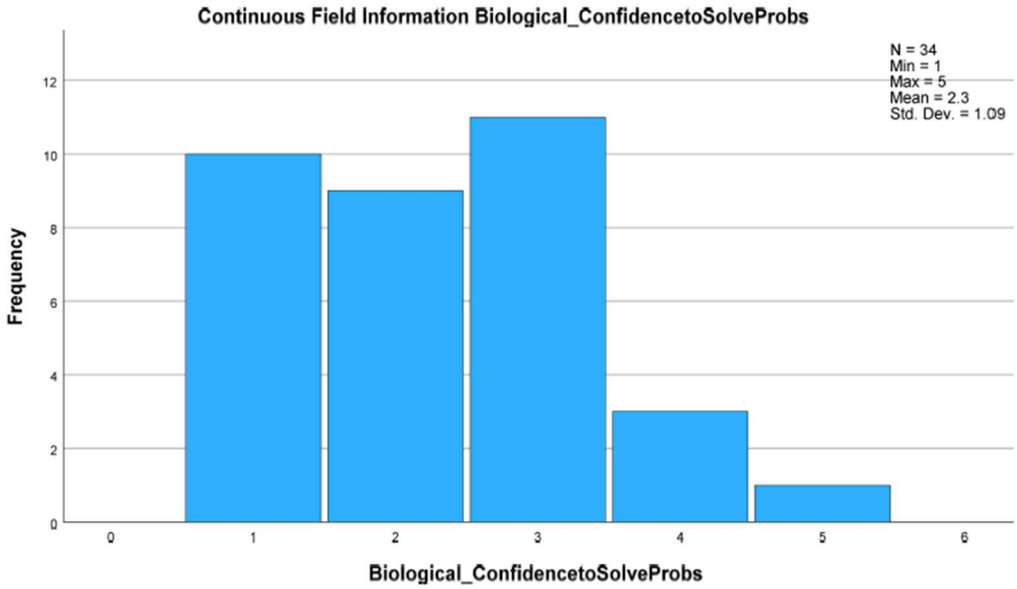
Related-Samples Wilcoxon Signed Rank Test

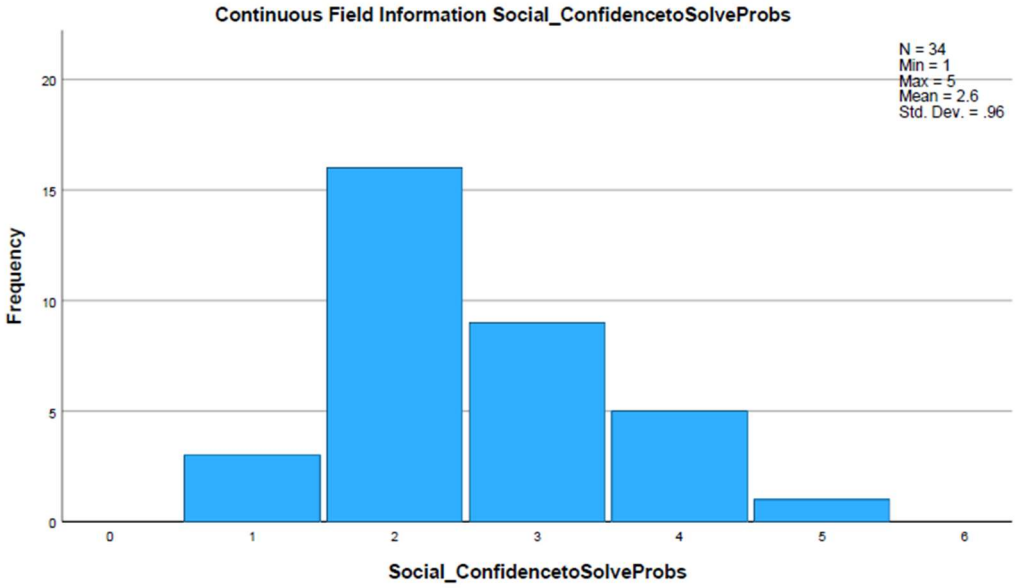
Biological_ConfidencetoSolveProbs, Social_ConfidencetoSolveProbs

Related-Samples Wilcoxon Signed Rank Test Summary

Total N	34
Test Statistic	159.000
Standard Error	29.967
Standardized Test Statistic	1.085
Asymptotic Sig.(2-sided test)	.278







Nonparametric Tests

Hypothesis Test Summary

	Null Hypothesis	Test	Sig.[a,b]
1	The median of differences between Biological_LikelyRelapse and Social_LikelyRelapse equals 0.	Related-Samples Wilcoxon Signed Rank Test	.004

Hypothesis Test Summary

	Decision
1	Reject the null hypothesis.

a. The significance level is .050.

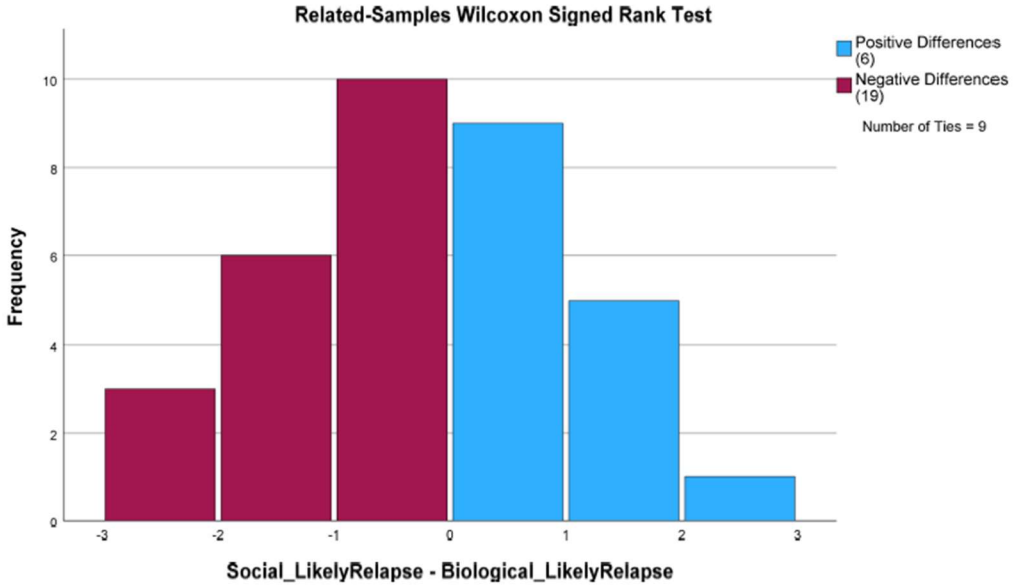
b. Asymptotic significance is displayed.

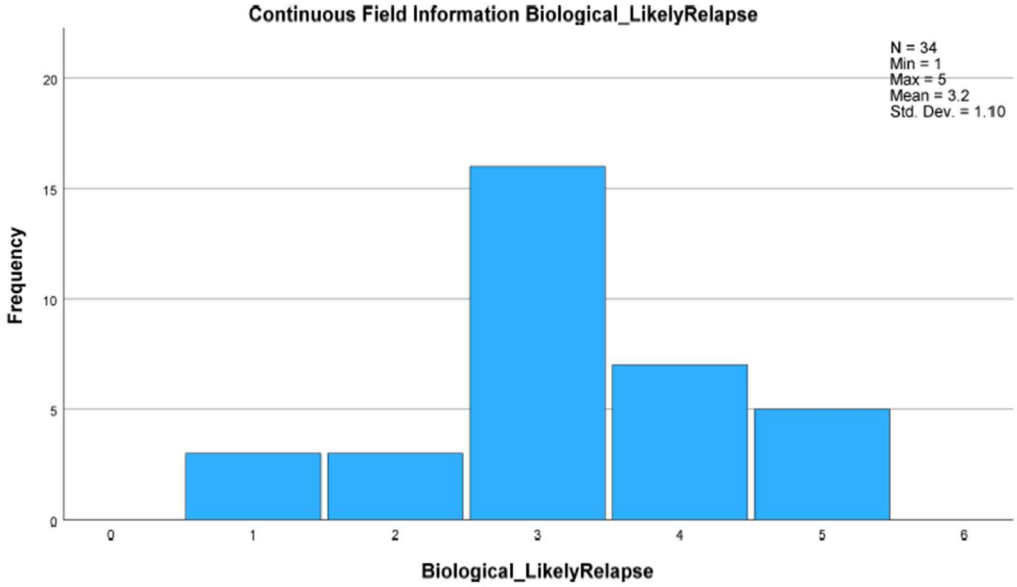
Related-Samples Wilcoxon Signed Rank Test

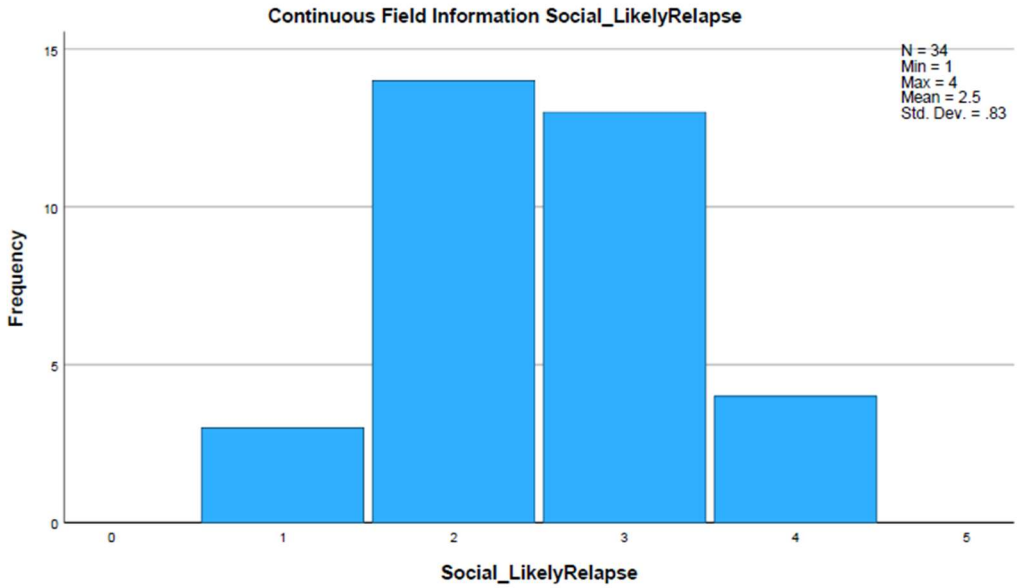
Biological_LikelyRelapse, Social_LikelyRelapse

Related-Samples Wilcoxon Signed Rank Test Summary

Total N	34
Test Statistic	59.000
Standard Error	36.107
Standardized Test Statistic	-2.866
Asymptotic Sig.(2-sided test)	.004







Nonparametric Tests

Hypothesis Test Summary

	Null Hypothesis	Test	Sig.[a,b]
1	The median of differences between Biological_QuitEffort and Social_QuitEffort equals 0.	Related-Samples Wilcoxon Signed Rank Test	.091

Hypothesis Test Summary

	Decision
1	Retain the null hypothesis.

a. The significance level is .050.

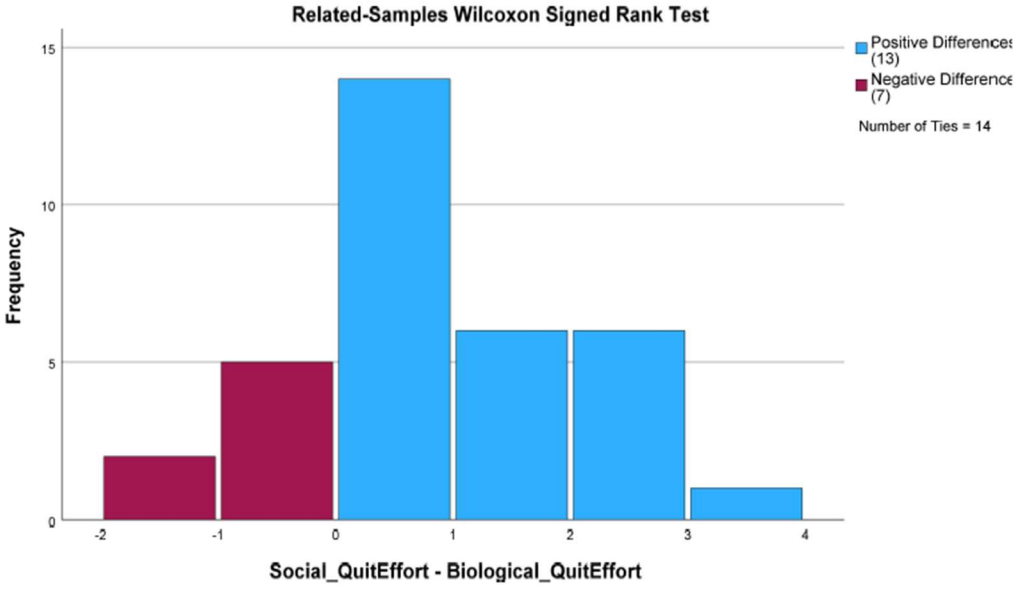
b. Asymptotic significance is displayed.

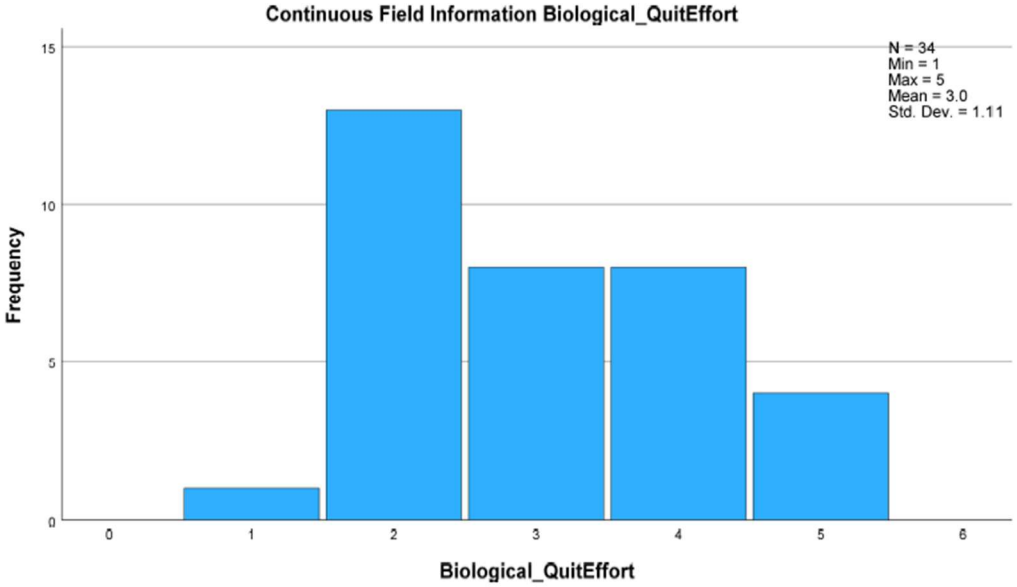
Related-Samples Wilcoxon Signed Rank Test

Biological_QuitEffort, Social_QuitEffort

Related-Samples Wilcoxon Signed Rank Test Summary

Total N	34
Test Statistic	149.000
Standard Error	26.067
Standardized Test Statistic	1.688
Asymptotic Sig.(2-sided test)	.091


Related-Samples Wilcoxon Signed Rank Test
Positive Differences (13)
Negative Differences (7)
Number of Ties = 14
Frequency
15
10
5
0
-2
-1
0
1
2
3
4
Social_QuitEffort - Biological_QuitEffort


Continuous Field Information Biological_QuitEffort
N = 34
Min = 1
Max = 5
Mean = 3.0
Std. Dev. = 1.11
Frequency
15
10
5
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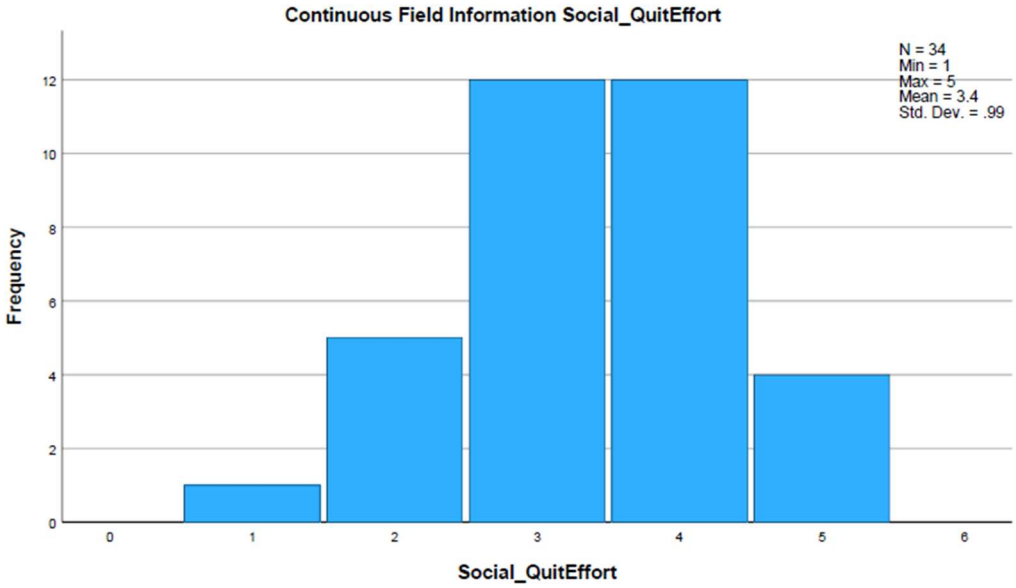
1
2
3
4
5
6
Biological_QuitEffort



Nonparametric Tests

Hypothesis Test Summary

	Null Hypothesis	Test	Sig.[a,b]
1	The median of differences between Biological_ResilianceToDifficulties and Social_ResilianceToDifficulties equals 0.	Related-Samples Wilcoxon Signed Rank Test	.146

Hypothesis Test Summary

	Decision
1	Retain the null hypothesis.

a. The significance level is .050.

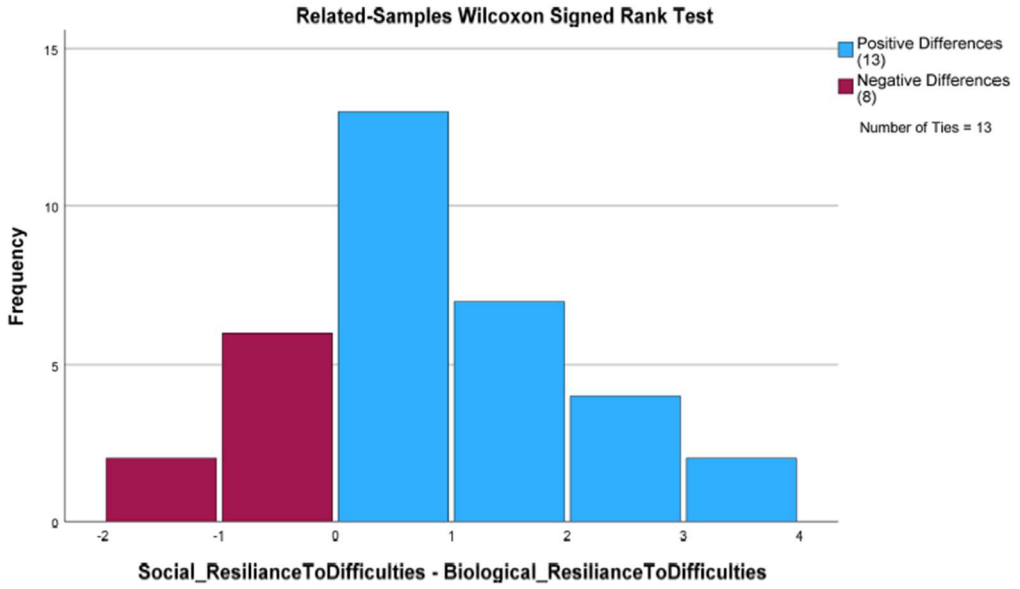
b. Asymptotic significance is displayed.

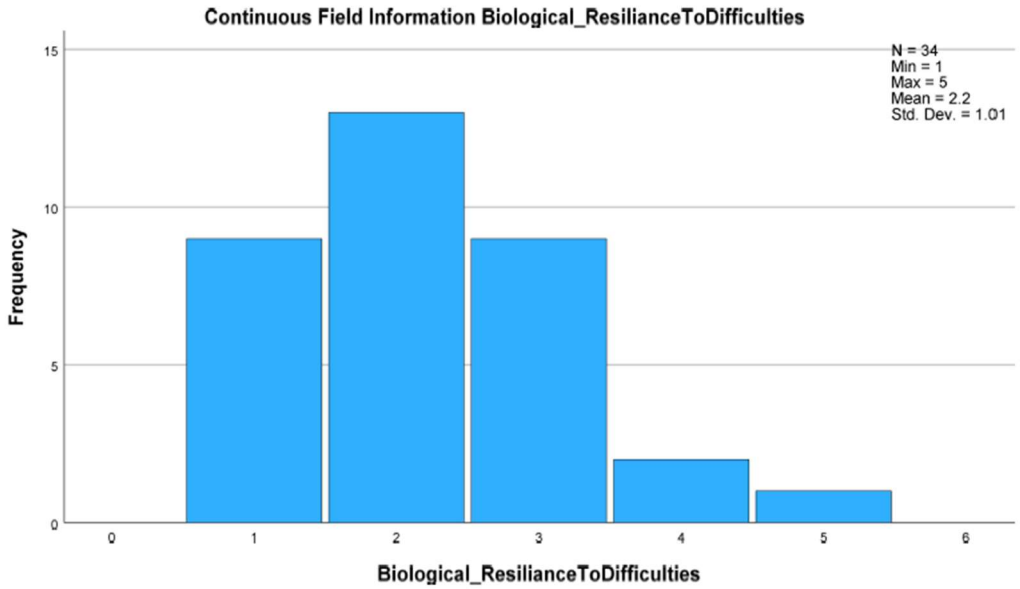
Related-Samples Wilcoxon Signed Rank Test

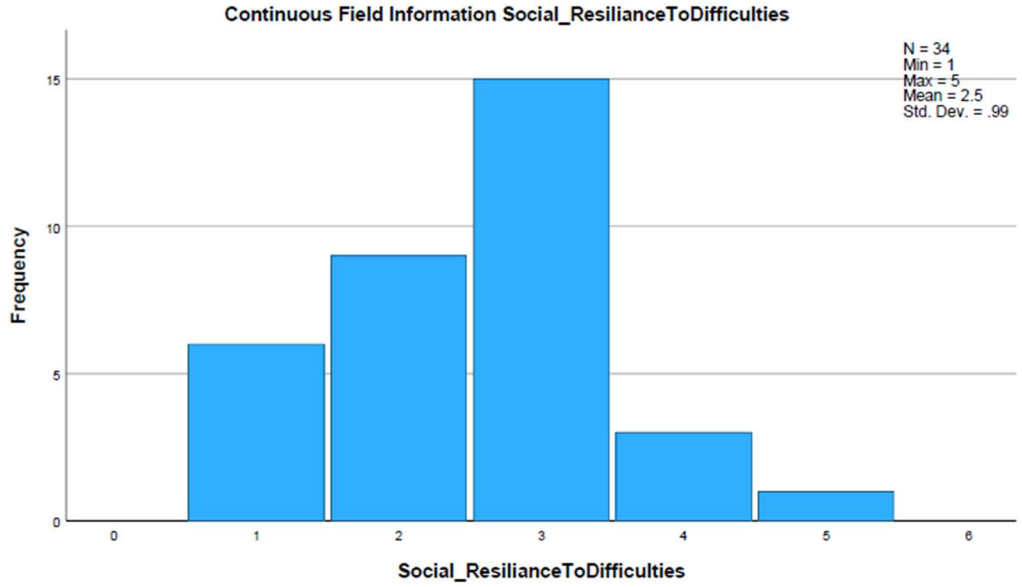
Biological_ResilianceToDifficulties, Social_ResilianceToDifficulties

Related-Samples Wilcoxon Signed Rank Test Summary

Total N	34
Test Statistic	156.000
Standard Error	27.888
Standardized Test Statistic	1.452
Asymptotic Sig.(2-sided test)	.146







Nonparametric Tests

Hypothesis Test Summary

	Null Hypothesis	Test	Sig.[a,b]
1	The median of differences between Biological_BlameYouself and Social_BlameYouself equals 0.	Related-Samples Wilcoxon Signed Rank Test	.034

Hypothesis Test Summary

	Decision
1	Reject the null hypothesis.

a. The significance level is .050.

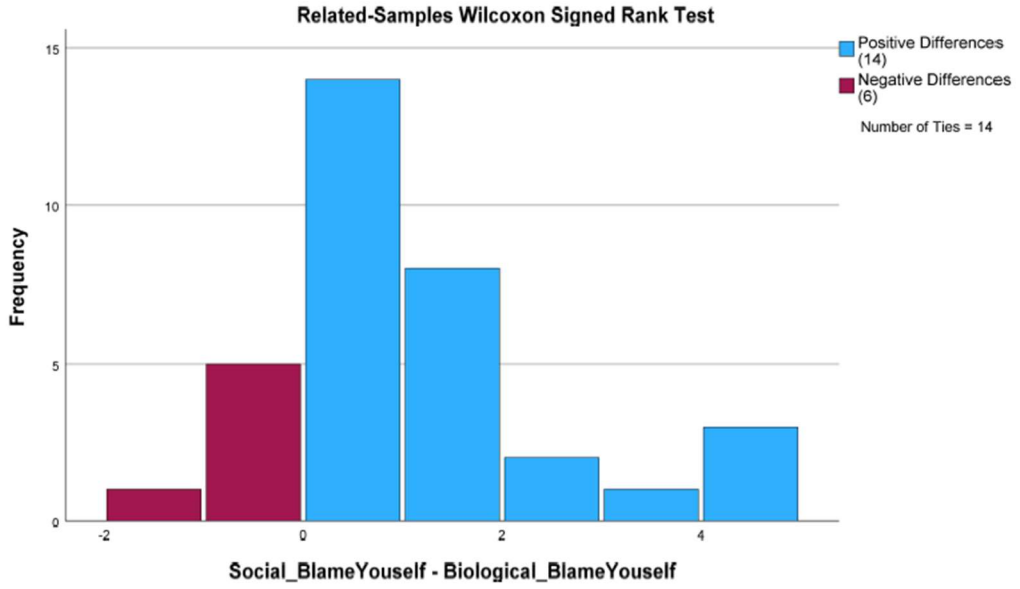
b. Asymptotic significance is displayed.

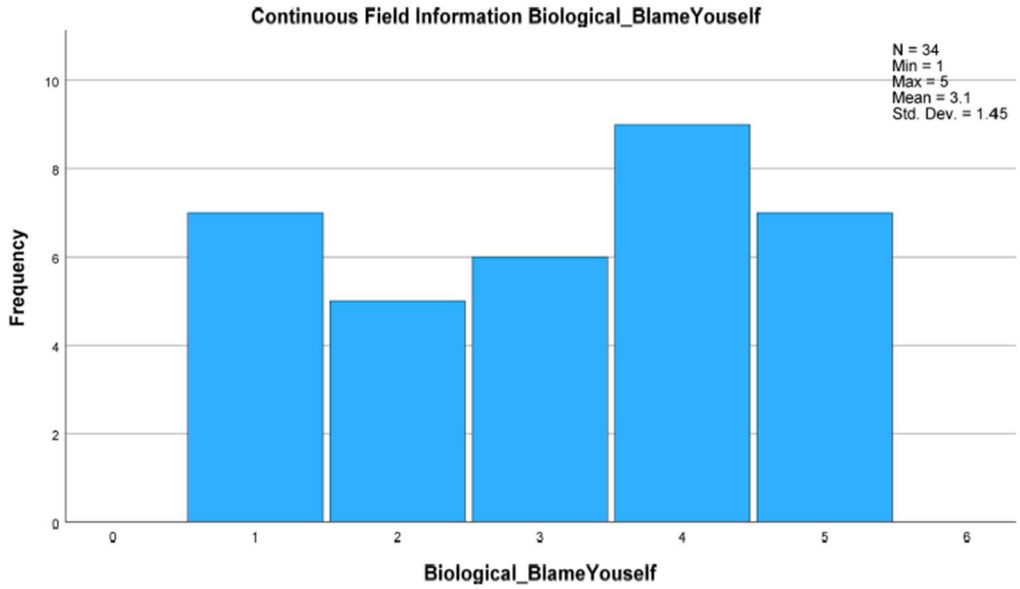
Related-Samples Wilcoxon Signed Rank Test

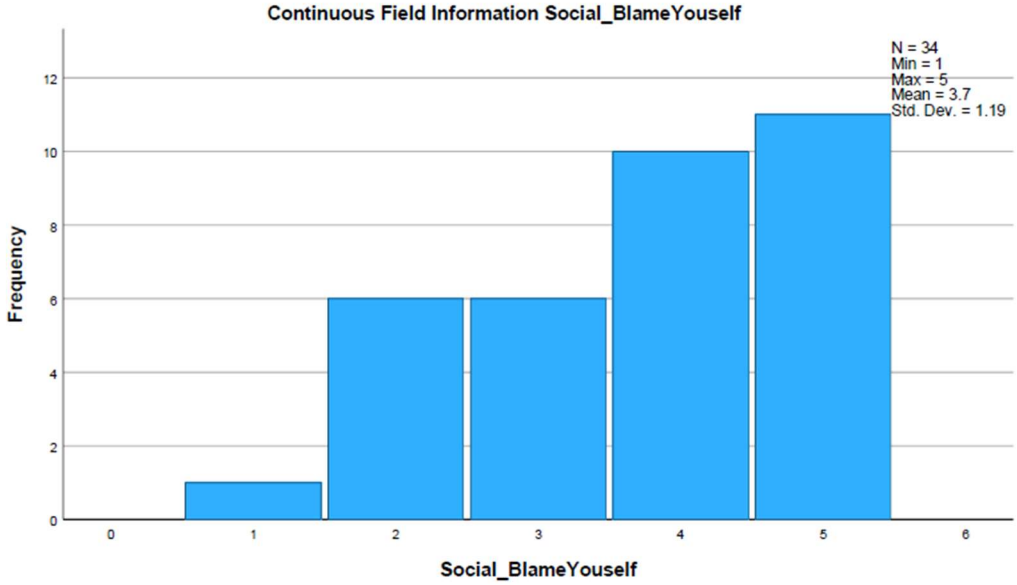
Biological_BlameYouself, Social_BlameYouself

Related-Samples Wilcoxon Signed Rank Test Summary

Total N	34
Test Statistic	160.000
Standard Error	25.904
Standardized Test Statistic	2.123
Asymptotic Sig.(2-sided test)	.034







Nonparametric Tests

Hypothesis Test Summary

	Null Hypothesis	Test	Sig.[a,b]
1	The median of differences between Biological_NegativeAboutSelf and Social_NegativeAboutSelf equals 0.	Related-Samples Wilcoxon Signed Rank Test	.603

Hypothesis Test Summary

	Decision
1	Retain the null hypothesis.

a. The significance level is .050.

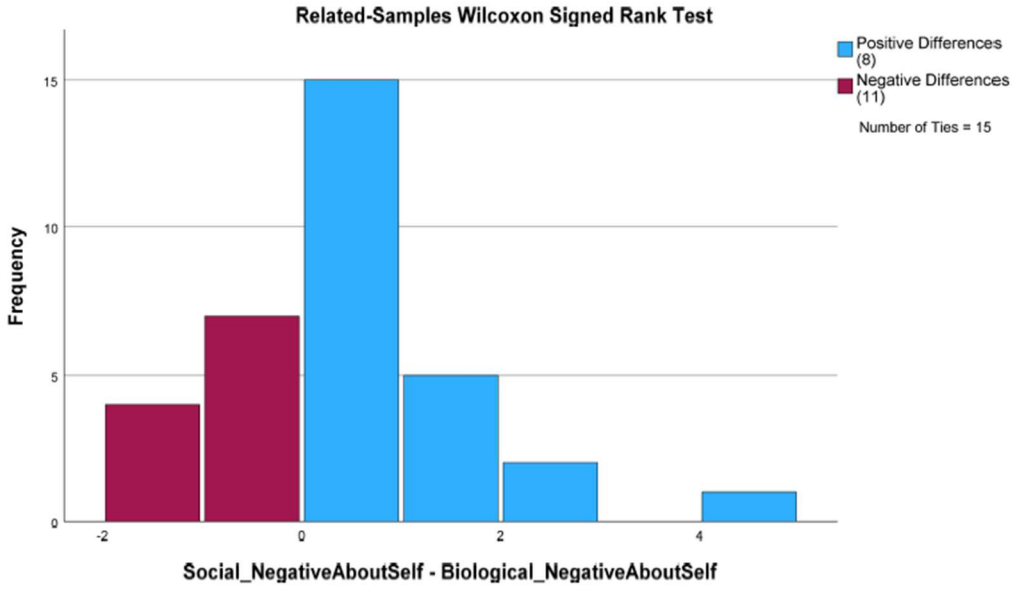
b. Asymptotic significance is displayed.

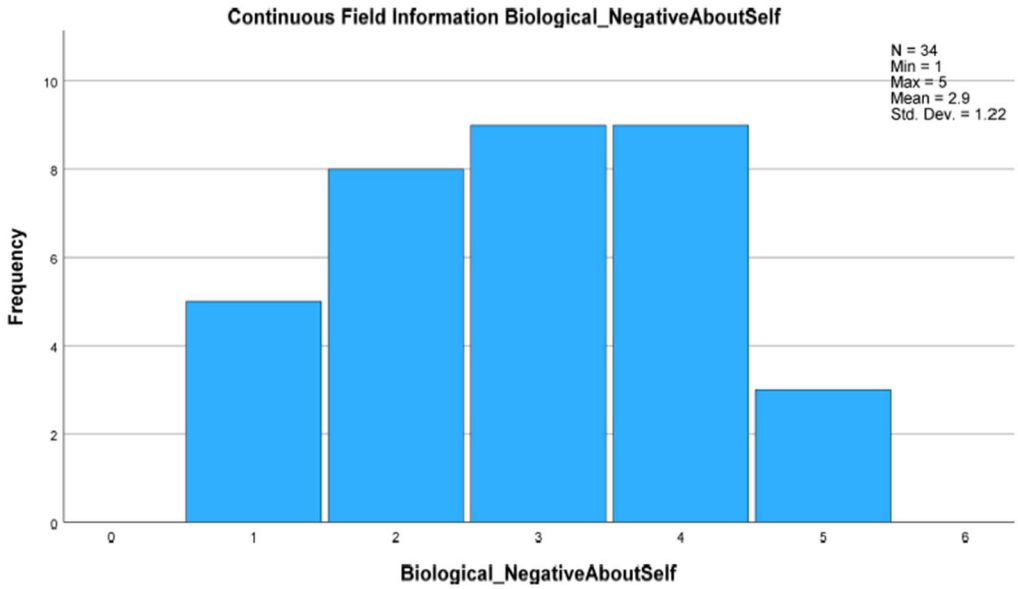
Related-Samples Wilcoxon Signed Rank Test

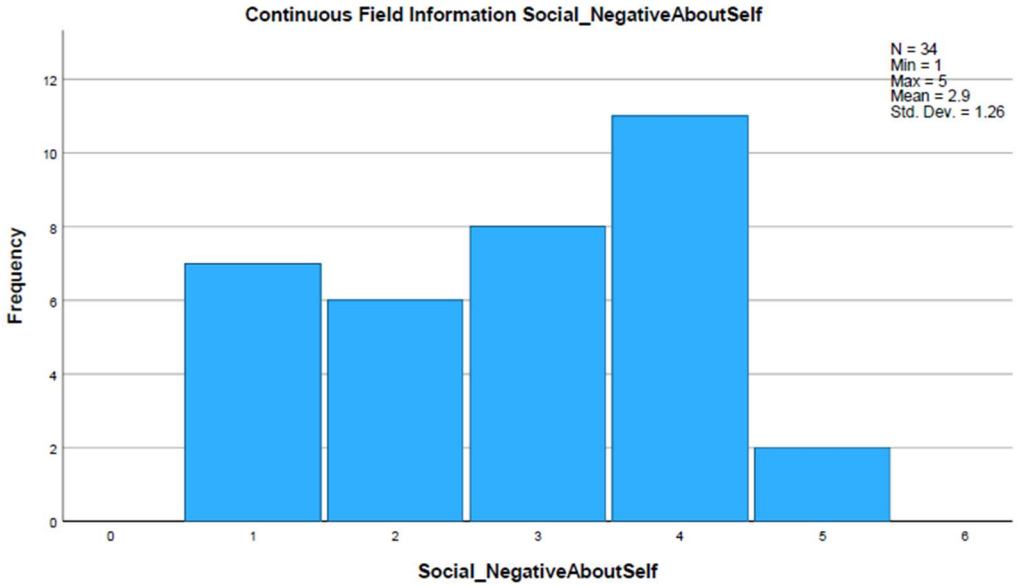
Biological_NegativeAboutSelf, Social_NegativeAboutSelf

Related-Samples Wilcoxon Signed Rank Test Summary

Total N	34
Test Statistic	82.500
Standard Error	24.029
Standardized Test Statistic	-.520
Asymptotic Sig.(2-sided test)	.603







Nonparametric Tests

Hypothesis Test Summary

	Null Hypothesis	Test	Sig.[a,b]
1	The median of differences between Biological_SubstanceUseDaysPerWeek and Social_SubstanceUseDaysPerWeek equals 0.	Related-Samples Wilcoxon Signed Rank Test	.033

Hypothesis Test Summary

	Decision
1	Reject the null hypothesis.

a. The significance level is .050.

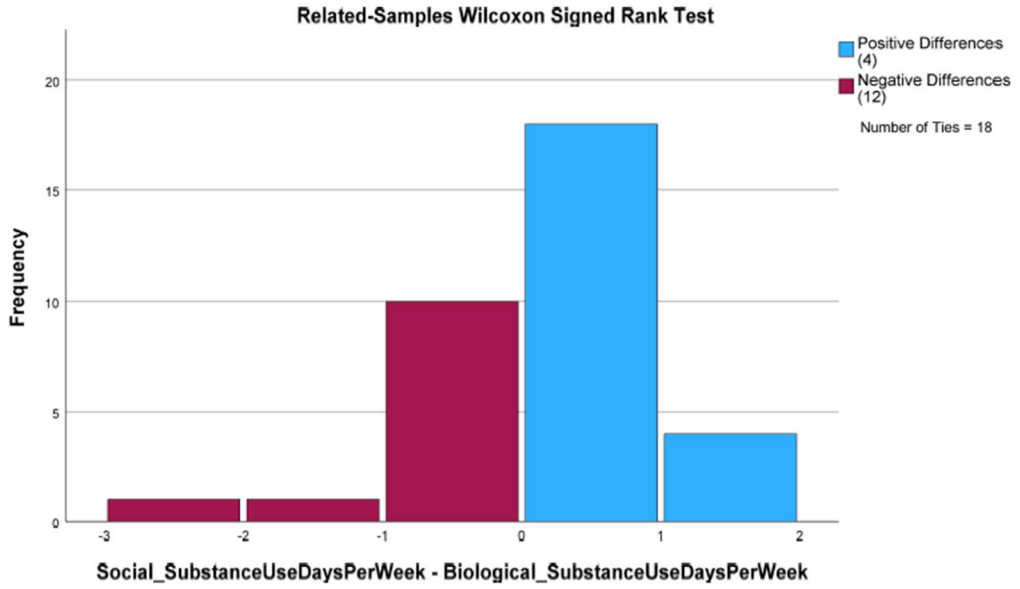
b. Asymptotic significance is displayed.

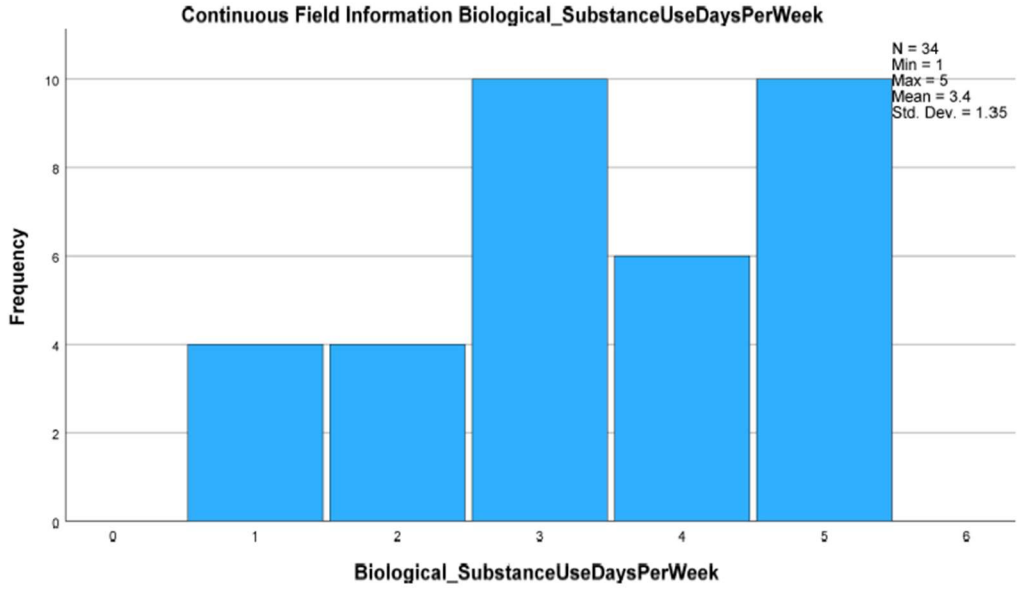
Related-Samples Wilcoxon Signed Rank Test

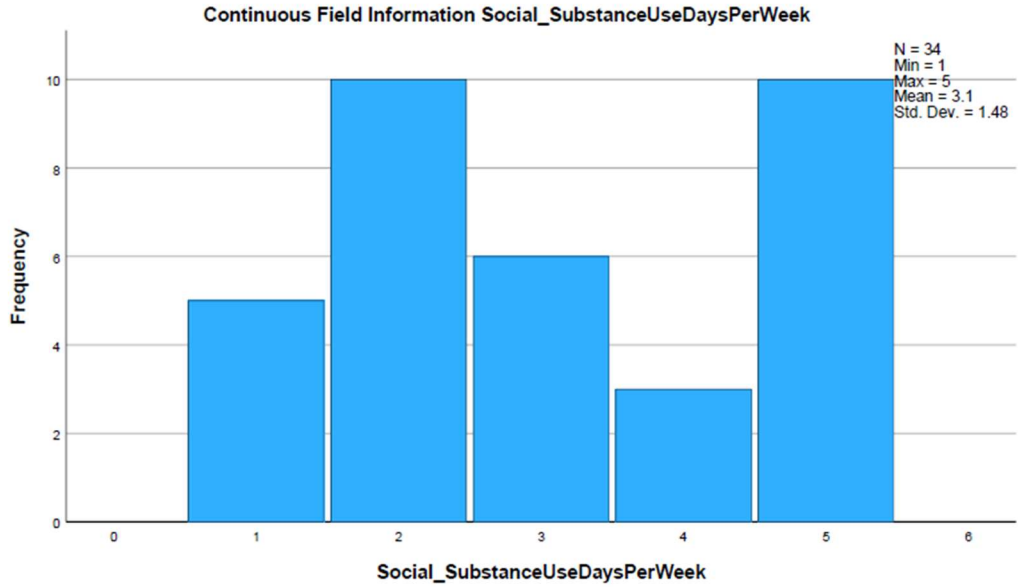
Biological_SubstanceUseDaysPerWeek, Social_SubstanceUseDaysPerWeek

Related-Samples Wilcoxon Signed Rank Test Summary

Total N	34
Test Statistic	30.000
Standard Error	17.808
Standardized Test Statistic	-2.134
Asymptotic Sig.(2-sided test)	.033






Continuous Field Information Social_SubstanceUseDaysPerWeek
N = 34
Min = 1
Max = 5
Mean = 3.1
Std. Dev. = 1.48
Frequency
10
8
6
4
2
0
0
1
2
3
4
5
6
Social_SubstanceUseDaysPerWeek

Nonparametric Correlations

Correlations

			What is your age?	What is your gender?	Are you currently experiencing any problems with housing including poor quality housing, at risk of being evicted, or currently experiencing homelessness?
Spearman's rho	Biological_Agree		.024	-.086	.028
			.893	.628	.875
			34	34	34
	Social_Agree		-.227	.297	.075
			.197	.088	.672
			34	34	34

Correlations

			How many days in the past 4 weeks have you been in paid employment, volunteering, or training/education? - Days	How would you rate your psychological and emotional health? - 0 (Poor) to 20 (Good)	How would you rate your physical health? - 0 (Poor) to 20 (Good)
Spearman's rho	Biological_Agree		.324	.159	.035
			.062	.369	.844
			34	34	34
	Social_Agree		.621	.240	-.014
			.000	.172	.939
			34	34	34

Correlations

			How would you rate your overall quality of life? - 0 (Poor) to 20 (Good)	PastMonthSubstanceUseDays	treatmentLength N
Spearman's rho	Biological_Agree		.200	.025	.249
			.257	.886	.155
			34	34	34
	Social_Agree		.047	-.011	.092
			.791	.952	.604
			34	34	34

Correlations

			treatmentTimes n	Are you using any prescribed medications? - Selected Choice	Have you ever been prescribed any medications as treatment for your addiction problems? E.g. Benzodiazepine, Diazepam, Naltrexone, Buprenorphine, Methadone etc.
Spearman's rho	Biological_Agree		-.220	-.071	.006
			.211	.689	.972
			34	34	34
	Social_Agree		.089	.041	-.125
			.616	.817	.480
			34	34	34

Correlations

			Have you ever had any one-to-one support, talking therapy or group support for your addiction problems? E.g. CBT, counselling, one-to-one peer support, 12-step programme etc.	Are you currently receiving any social support for your addiction problems? E.g. peer support, family/parenting support, housing support, employment support, education/training support etc.
Spearman's rho	Biological_Agree		.208	.226
			.239	.198
			34	34
	Social_Agree		.325	.255
			.061	.146
			34	34

Nonparametric Correlations

Correlations

			Biological_Agree	Biological_FacilitatorSupport	Biological_MembersSupport
Spearman's rho	Biological_Agree		1.000	.462	.503
			.	.006	.002
			34	34	34

Correlations

			Biological_RecoveryOptimism	Biological_ConfidencetoSolvePrObs	Biological_LikelyRelapse
Spearman's rho	Biological_Agree		.250	.277	-.176
			.153	.113	.321
			34	34	34

Correlations

			Biological_QuitEffort	Biological_ResilianceToDifficulties	Biological_BlameYouself
Spearman's rho	Biological_Agree		.165	.096	-.032
			.350	.588	.857
			34	34	34

Correlations

			Biological_NegativeAboutSelf	Biological_SubstanceUseDaysPerWeek
Spearman's rho	Biological_Agree		.073	.105
			.682	.556
			34	34

Nonparametric Correlations

Correlations

			Social_Agree	Social_FacilitatorSupport	Social_MembersSupport
Spearman's rho	Social_Agree		1.000	.504	.559
			.	.002	.001
			34	34	34

Correlations

			Social_RecoveryOptimism	Social_ConfidencetoSolveProbs	Social_LikelyRelapse
Spearman's rho	Social_Agree		.532	.558	.102
			.001	.001	.567
			34	34	34

Correlations

			Social_QuitEffort	Social_ResilianceToDifficulties	Social_BlameYouself
Spearman's rho	Social_Agree		.353	.426	-.091
			.040	.012	.610
			34	34	34

Correlations

			Social_NegativeAboutSelf	Social_SubstanceUseDaysPerWeek
Spearman's rho	Social_Agree		.063	.106
			.724	.552
			34	34