UNIVERSIDAD COMPLUTENSE DE MADRID
FACULTAD DE PSICOLOGÍA

TESIS DOCTORAL

Análisis de redes en el estudio de la psicopatología y su aplicación en el estudio de la dinámica de las creencias paranoides

Network analysis in the study of psichopathology and its application to the study of the dynamics of paranoid beliefs

MEMORIA PARA OPTAR AL GRADO DE DOCTORA

PRESENTADA POR

Alba María Contreras Cuevas

DIRECTORA

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Dir: Dra. Carmen Valiente Ots

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-Facultad de Psicología-

"*Caminante no hay camino, se hace camino al andar*"

Antonio Machado.

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RESUMEN

Introducción

Las ideas paranoides son el tipo de creencia más común y estudiada dentro del espectro de los trastornos psicóticos (Jorgensen & Jensen, 1994). La investigación, hasta el momento, ha mostrado evidencias de que el pensamiento paranoide se encuentra presente en población general, lo que ha dado lugar a la conceptualización de este fenómeno como un proceso multifactorial continuo (Bebbington et al., 2013; Freeman, 2016; Garety & Hemsley, 1997). En la actualidad, existen varios modelos teóricos que señalan los posibles factores psicológicos implicados en el desarrollo y mantenimiento de estas creencias. Uno de los modelos más influyentes, la teoría del *sesgo auto-sirviente*, ha señalado la importancia de tener en cuenta los niveles y fluctuaciones de los procesos asociados a las creencias paranoides (Bentall et al., 2001), ya que su dinámica temporal puede afectar su gravedad (Murphy et al., 2018). Aunque existen algunos trabajos que han estudiado cómo pueden afectar a la paranoia las fluctuaciones de algunos procesos que intervienen en ella como la autoestima, el afecto negativo o la evitación experiencial, (Thewissen et al., 2011; Udachina et al., 2014), los estudios que prestan atención a la dinámica asociada a la creencia paranoide desde una perspectiva multifactorial son escasos.

Recientemente ha emergido un nuevo enfoque en la investigación en psicopatología, conocido como la *teoría de redes* (Borsboom & Cramer, 2013). Esta perspectiva propone una visión de los problemas mentales alternativa a las proporcionadas hasta el momento por los modelos vigentes en psicopatología. Así, la teoría de redes en psicología conceptualiza los problemas mentales como interacciones dinámicas entre elementos que se influyen mutuamente (Borsboom, 2017), lo que puede permitir el estudio de la creencia paranoide como proceso dinámico y multidimensional.

Objetivos

El objetivo principal de la presente tesis es investigar el estado actual del análisis de la teoría de redes en el campo de la psicopatología y analizar su aplicación al estudio de la dinámica de las creencias paranoides. Esperamos que los conocimientos desarrollados durante el presente trabajo contribuyan a vislumbrar la proliferación de trabajos sobre esta teoría en la investigación psicopatológica y contribuir al avance en el conocimiento sobre cómo las fluctuaciones de los mecanismos involucrados en las creencias persecutorias pueden afectar el desarrollo y mantenimiento de las mismas.

Resultados

Los resultados encontrados sugieren que el análisis de redes no sólo es una alternativa teórica a los modelos tradicionales de causa común, sino que también proporciona herramientas que permiten investigar la creencia paranoide como un proceso dinámico y multifactorial. De manera específica, la aplicación de un modelo vectorial multinivel autorregresivo (mlVAR) para datos longitudinales recogidos con metodología de muestreo de experiencias (ESM), supera las limitaciones de otros tipos de análisis de redes previos basados en datos transversales, que han sido considerados estáticos. Los resultados muestran que las interacciones entre autoestima, afecto negativo, evitación experiencial y sentimiento de cercanía a los demás fluctúan en individuos con vulnerabilidad al pensamiento paranoide y éstas fluctuaciones son distintas cuando se tienen en cuenta diferentes momentos temporales. Además, los resultados señalan un posible papel protector del sentimiento de cercanía a los demás frente a la paranoia, ya que predice niveles bajos de paranoia en el siguiente momento temporal.

Conclusión

El análisis de redes ha surgido en psicopatología como una estrategia de investigación clínica alternativa a los modelos tradicionales en psicopatología,

permitiendo la visualización y el análisis de patrones complejos de una gran variedad problemas mentales. La contribución más prometedora de este enfoque es que nos permite pensar en formas dinámicas de estudiar la salud mental en general, y las creencias persecutorias en particular. Este enfoque alternativo puede ayudar dilucidar las interacciones entre los mecanismos centrales implicados en las creencias paranoides y ofrecer nuevas direcciones hacia la identificación de objetivos para la prevención y tratamiento de la paranoia.

SUMMARY

Introduction

Paranoid ideation is the most common and studied type of belief in the spectrum of psychotic disorders (Jorgensen & Jensen, 1994). To date, research has shown that paranoid thinking is present in the general population, which has led to the conceptualization of this phenomenon as a multifactorial and continuum process (Bebbington et al., 2013; Freeman, 2016; Garety & Hemsley, 1997). Currently, there are several theoretical models accounting for psychological factors involved in the development and maintenance of these beliefs. One of the most influential, the *self-serving bias theory*, has pointed out the importance of taking into account not only levels, but also fluctuations in the processes associated with paranoid beliefs (Bentall et al., 2001). Thus, it has been hypothesized that fluctuations in paranoia-related processes might affect the severity of these beliefs (Murphy et al., 2018). Although there are several works that have studied how the fluctuations in some processes involved in paranoia, such as self-esteem, negative affect or experiential avoidance, can affect it (Thewissen et al., 2011; Udachina et al., 2014), research paying attention to the dynamics of these processes from a multifactorial perspective is scarce.

Recently, a new approach to psychopathology research has emerged, known as *network analysis theory* (Borsboom & Cramer, 2013). This perspective proposes an alternative vision of mental problems to those provided so far by current models in psychopathology. From this approach, mental problems are conceptualised as dynamic interactions between elements that influence each other (Borsboom, 2017), which seems to fit well the study of paranoid belief as a dynamic and multidimensional process.

Objectives

The main objective of the present dissertation was to investigate the current state

of network analysis in the field of psychopathology and to analyse its application to the study of the dynamics of paranoid beliefs. We hope that the knowledge developed in the present work will contribute to clarify the rapid proliferation of network analysis theory in psychopathological research and contribute to the advancement of knowledge on how the fluctuations of paranoid thinking mechanisms can affect the development and maintenance of these beliefs.

Results

The results suggest that network analysis is not only a theoretical alternative to traditional models of common cause, but also provides appealing methodological tools to investigate paranoid belief as a dynamic and multifactorial process. Specifically, the application of a multilevel vector autoregressive model (mlVAR) for longitudinal data collected with Experience Sampling Methodology (ESM) overcomes the limitations from previous cross-sectional network studies, considered more static. Our findings show that the different levels and interactions between self-esteem, negative affect, experiential avoidance and the feeling close to others fluctuate when different time frames are taken into account. In addition, the results point to a possible protective role for feeling close to others, since it predicts low levels of paranoia in the next temporal moment.

Conclusion

Network analysis has emerged as an alternative research strategy to traditional models in psychopathology allowing the visualisation and analysis of complex dynamic patterns of a wide variety of mental problems. The most promising contribution of this approach is that it allows us to think of dynamic ways to study mental health in general, and persecutory beliefs in particular. This alternative approach can help elucidate the interactions between the central mechanisms involved in paranoid beliefs and offer new directions towards identifying targets for the prevention and treatment of paranoia.

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PREAMBLE

> "Psychosis should be seen as just part and parcel of human variation, rather than as an illness"
>
> (Bentall, 2004, p.104).

We are social beings who live in constantly changing communities, shared with other individuals. Among the many decisions we make every day, one of the most frequent is whether or not we could trust other people. This judgment can be learned indirectly, for instance, by watching others, but there are also elements that can vary at the individual level, such as previous life events or possible difficulties in discerning the intentions of others, which can also influence our judgments about trust. When these judgments are founded to be suspicious, then we are moving along the what is known the spectrum of paranoia (Freeman & Garety, 2014; Freeman, 2016).

Although paranoid thinking has been identified as a characteristic psychotic symptom within the so-called spectrum of schizophrenia and other psychotic disorders, it is currently considered an individual experience across a continuum in the general population. The prevalence of paranoid cognitions has been reported to vary from approximately 2-42% in the general population (Freeman, 2006; Statham et al., 2019). Existing data, also suggest that one-sixth of the population spends a lot of time wondering whether they could trust their friends or co-workers, while 10% of the population sometimes feels that others are looking at them with deliberate intent to harm them (Bebbington et al., 2013). On this basis, our paranoid thoughts can be located within a broad range that goes from fear of others rejection, feelings of being the centre of other people's conversations, to the idea that there is a direct threat to the individual intended by others. This latter extreme would correspond to what is commonly termed in current

research as persecutory delusion, that is, the most severe clinical presentation within the spectrum of paranoia (Freeman & Garety, 2000).

To date, paranoid beliefs (and persecutory delusion at their most severe manifestation) are psychological problems of enormous importance and high significant clinical impact (Freeman, 2016). First of all, paranoid ideation is the most common abnormal belief in schizophrenia (American Psychiatric Association., 2013; Jorgensen & Jensen, 1994). For instance, it is present in the 70-90% of people with a first psychotic episode (Coid et al., 2013; Moutoussis et al., 2007), often resulting in hospital admission (Castle et al., 1994). Furthermore, it has been pointed out that this prevalence has transcultural validity, since it occurs both in Western countries and in other parts of the world (Jorgensen & Jensen, 1994; Stompe et al., 1999).

On the other hand, paranoid ideation is not exclusive to the spectrum of schizophrenia, but it is common to a wide range of other mental disorders (Bell et al., 2006). For example, people with anxiety or depression have been shown to have high scores on measures of persecutory ideation (Van Os et al., 1999). In addition, persecutory beliefs have been shown to be the most common manifestation of the delusions present in approximately 15% of individuals with unipolar depression (Horwath et al., 1992; Rosen et al., 2012) and around 12-29% of people with Post-Traumatic Stress Disorder (Butler et al., 1996; Buckley et al., 2009). Lastly, paranoia is also a common experience in paranoid personality disorders (Freeman, 2007) and neurological disorders such as dementia (Flint, 1991; Mendez et al., 2008) or epilepsy (Trimble, 1992; Gaitatzis et al., 2004).

Undoubtedly, current data on its clinical impact make evident the benefits of studying paranoia, not only in affected individuals, but also in the society at large (Statham et al., 2019). In people with persecutory thinking, the presence of these beliefs

is often accompanied by anxiety (Hartley et al., 2013), depression (Vorontsova et al., 2013), sleep disturbances (Freeman et al., 2009), as well as suicidal ideation (Freeman et al., 2011). Along with generating distress, they are associated with emotional and avoidant coping, negative attitudes towards emotional expression and submissive behaviours (Freeman et al., 2005). Furthermore, about half of the individuals with persecutory delusions show lower levels of psychological well-being and quality of life than the general population (Freeman et al., 2014; Valiente et al., 2019). Moreover, given the interpersonal nature of the content of paranoid thinking, it is not surprising that its presence has repercussions at the social level. Paranoid beliefs are associated with negative beliefs about others (Lamster et al., 2017) and the subjective perception of social exclusion (Westermann et al., 2012).

Recently, the importance of studying not only the factors involved in paranoid processes, but also their dynamics has been pointed out. That is, factors involved such as self-representations or emotional regulation processes vary over time, and these temporal fluctuations may play a central role in the maintenance and severity of persecutory beliefs (Murphy et al., 2018). Consequently, some researchers now advocate focusing on paranoid belief as a complex multifactorial process interrelated with other processes in a dynamic way, in which interpersonal factors cannot be neglected.

In short, conceptualised as a transdiagnostic feature (Bentall et al., 2009) paranoid beliefs are a therapeutic target, not only for individuals with psychotic disorders but also in a wide range of mental disorders and psychological problems (Freeman, 2007; Lincoln et al., 2013). Advances in our understanding of the mechanisms involved and their dynamic associations over time could provide evidence of the potential causal factors involved in the paranoid ideation and, thus, guide the development of new therapeutic interventions (Blackwood et al., 2001). Given all this notions, the main objective of the

present dissertation is to investigate the dynamics of several core factors associated with paranoia in individuals with paranoid vulnerability and to identify possible causal relationships between them that may predict paranoia over time. To do so, we depart from a recently emerged approach in the study of psychopathology, known as Network Analysis theory (NA). From this approach, a temporal network analysis model is applied to longitudinal data, which allows us to study the temporal dynamics of paranoid thinking and associated core elements. The specific objectives pursued in each chapter are set out below (see Table 1):

Table 1.
Summary of specific objectives pursued in the present dissertation

Chapter	Specific aim
Chapter 1. Paranoia, paranoid beliefs and persecutory delusions.	-To review the theoretical background regarding paranoid thinking, evolution of the definition and current conceptualization.
Chapter 2. Psychological mechanisms and current psychological models of paranoid (persecutory) thinking.	-To provide an overview about the empirical evidence of the core mechanisms associated to paranoid beliefs. -To review the main current theoretical explanatory models accounting for the development and maintenance of these beliefs.
Chapter 3. Current classification systems in psychopathology: network analysis (NA) as an alternative.	-To describe current proposed limitations about current traditional approach in psychopathology. -To offer a theoretical background of the new emerged network analysis theory as an alternative approach.
Chapter 4. The study of psychopathology from the network analysis perspective: a systematic review.	-To systematically review all empirical literature applying network analysis in the study of psychopathology. -To identify current state, strengths, limitations and challenges of network analysis from empirical evidence.
Chapter 5. A temporal network approach to study paranoia.	-To conceptualize paranoid beliefs from the network approach as dynamical complex system. -To overcome actual limitations from cross-sectional studies by using a network analysis approach applied to longitudinal data. To study fluctuations of persecutory beliefs and other core factors across different time-frames.
Chapter 6. General discussion and conclusion.	To provide a summary of the main findings, a general discussion about the most important considerations, limitations and future challenges and a conclusion of the present dissertation.

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CHAPTER 1. PARANOIA, PARANOID BELIEFS AND PERSECUTORY DELUSION

"Since time immemorial, delusion has been taken as the basic characteristic of madness. To be mad was to be deluded"

(Jasper, 1963, p. 93).

Throughout history, there has been considerable confusion between the term´s paranoia, paranoid beliefs and persecutory delusion. These terms are often used interchangeably but there are clear differences. The term paranoia was first coined by the Greeks (*pará*, which expresses the idea of being alien; *nous*: which means mind) to refer to "madness" or being "out of one´s mind" (Lewis, 1970). About 4,000 years ago, psychotic manifestations were conceived by simplistic theories as acts of madness, whose aetiology and recovery were accounted by magical, religious or mystical explanations (Read et al., 2004). Nonetheless, it was the German physician, S.G. Vogel in the 18th century, who first proposed to use the term "paranoia" to refer to all types of delusional experiences and later on, E. Lasegue in 1852 would introduce the notion of persecutory delusion to refer only to individuals who experience being subjected to persecution (Berrospi et al., 2003).

During the 19th century, a conceptual transition took place, away from the notion of paranoid delusions towards the conceptualisation of paranoid thinking as a belief. This idea had progressively gained acceptance in the scientific community and promoted the individualised study of paranoid belief as a symptom (Valiente, 2010). Then, although paranoia has been conceptualised as a symptom of psychosis within the schizophrenia spectrum, this new perspective has led to recent evidence pointing out that paranoia is also a common experience in the general population (Freeman et al., 2011). That is why paranoia nowadays is understood as a continuum in which paranoid beliefs may vary from interpersonal worries to the most severe form, identified as paranoid or persecutory

delusions (Freeman, 2007). In this chapter, we take a brief look at the many efforts over three centuries to address the problems involved in this concept, culminating in the current definition of paranoid beliefs.

1.1. First conceptions of paranoia

In order to clarify what paranoia is, we must first understand the concept of delusion. Delusions, together with hallucinations, are considered two of the main psychological phenomena defining a psychotic experience. Karl Jaspers, a German psychiatrist and philosopher, has been one of the most influential individuals in shaping our current understanding of delusion. According to Jaspers (1946) a delusion is a belief based on inadequate foundations which is held with great conviction despite evidence or rational arguments to the contrary and which contains strange content, as it is not shared by individuals from the same culture or educational background (Díez-Alegría et al., 2001; Valiente, 2010). Today, current definitions of DSM-5 define delusions as 'fixed beliefs that cannot be changed in light of conflicting evidence' (APA, 2013). While delusion is a term used to refer to all types of delusional beliefs (e.g., erotomanic, grandiose or nihilist delusion –see definitions below), the term paranoid or persecutory[1] delusion refers to a specific content of the delusional experience that is concerned with the idea that 'one is going to be harmed, harassed, and so forth by an individual, an organisation, or other group' (American Psychiatric Association., 2013). It is also worth mentioning that the term delusion has been used in the literature with two different meanings. During the 19th century and after the development of Descriptive Psychology, delusion was used to describe a global disorder, originated by biological causes and with

[1] Note that the terms paranoia and persecution are used here as synonyms, both of which are identified in the literature to refer to prejudice content.

a chronic evolution (Berrios, 1997; Lewis, 1970). A century later and in line with current trends, however, the study of the phenomenology of the delusion gives rise to a conception of it as an isolated symptom (Berrios, 1997).

There are many classical case-descriptions of delusions existing in the literature. One prototypical example is the case of Paul Daniel Schreber, a German judge who published in his biography *Memoirs of my Nervous Illness*, (1903/1955) his own psychotic experience. Among his many delusional beliefs, it is striking that his firm conviction was that his doctor, Dr. Flechsig, was trying to transform him into a woman in order to abuse him (Bentall et al., 2001). Undoubtedly, this case became well-known due to the influence of Sigmund Freud (1911), one of the most relevant figures in Psychopathology, who used Schreber's autobiography to formulate his own theory about paranoia (Bentall, 2004). From Freud's interpretation, his delusional beliefs would come from repressed homosexual impulses of the individual, which struggled to manifest. Since then, delusions have been considered from the psychoanalytic perspective as an expression of the individual's premature fantasies and desires (Baños & Belloch, 2008).

Nonetheless, Emil Kraepelin (1899/1990) reintroduced the term paranoia to describe a type of delusional disorder. Considered the father of Psychiatry, Kraepelin was one of the pioneers in developing a classification of mental disorders that has shaped the study of mental illness (Lewis, 1970). He was particularly interested in the study of the most severe forms of "madness" (i.e., where individuals lose touch with reality), which could give rise to three types of disorders or 'psychosis', namely *dementia-praecox*, *manic-depressive illnesses*, and *paranoia*[2].

[2] In the eighth edition of the *Textbook* published in 1915, Kraepelin identified three types of psychoses, namely *dementia praecox, manic-depressive illness* and *paranoia*. The first major

Based on the medical model, Kraepelin proposed that these diseases will produce prototypical symptom patterns with a specific pathological anatomy and an identical aetiology, and that a complete knowledge of these three groups would lead to a uniform classification of mental diseases (Berrios & Hauser, 1988). Thus, the term paranoia was then used as a diagnostic label for a chronic, persistent and incurable disease, characterised by the presence of delusions (Bentall, 2004). In the eighth edition of Kraepelin´s treatise in 1915, he defined paranoia as "a delusion of insidious onset and chronic course, originated from internal causes, devoid of hallucinations and without personality deterioration" (Olivos, 2009).

After studying Kraepelin's proposal, the well-known Swiss psychiatrist Eugen Bleuler (1950) proposed to replace the term "dementia praecox", and to incorporate paranoia into the generic umbrella term "schizophrenia" (Berrospi et al., 2003). He argued that the illness did not always result in an extreme form of deterioration (i.e., there was not always *dementia*) and could arise in adult life (i.e., it was not always *praecox*). According to Bleuler, the so-called schizophrenia would be a disorder composed of four fundamental manifestations (i.e., "four A´s"; loss of association, ambivalence, autism and inappropriate affect) (Bentall, 2004; Bentall et al., 1988). What is most remarkable about Bleuler's work is that he excluded delusions as one of these fundamental symptoms, conceiving them as merely *accessory symptoms*, that is, not a by-product of the disorder.

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type was related to ‘senility of the young’, and included *catatonia* (i.e., a disorder characterised by stupor and abnormal postures), *hebephrenia* (i.e., a disorder that appeared during adolescence and lead to a rapid deterioration of mental functions) and *dementia paranoia* (i.e., a disorder that leads to a rapid deterioration, but characterised by bizarre fears of persecution). The second mental illness category was characterised by ‘recurrent’ mood disorders in which episodes of illnesses were followed by periods of normal functioning. Finally, *paranoia* was a term used to refer to a chronic illness characterised by delusional beliefs in the absence of significant changes to the personality of the individual (Bentall, 2004).

This would be one of the first conceptualisations of delusion as a symptom or experience shared by all humans and as a phenomenon with a psychological origin (Ruiz-Ogara & Barcia-Salorio, 1998).

During the late 19th century, a conceptual distinction between "knowledge¨ and "belief" took place and the conception of delusion as a false belief began to become more popular (Berrios, 1991). "Knowledge" was related to scientific certainty and, therefore, required evidence while "belief" was a subjective experience and was redefined in terms of probabilistic knowledge and mental attitudes (Baños & Belloch, 2008). Accordingly, *to know* was to be sure of the existence or truth of something, whereas *to believe* was to have something as true or existing, but without being sure of it (Díez-Patricio, 2011). Given that the content of delusion is difficult to adjust to the epistemological standards of science, the idea that delusions were an abnormal belief was strengthened. However, some authors have argued against the use of the concept of belief and proposed that delusion might be better conceptualised as affirmations or judgments, as they are subjective experiences more consistent with the notion of "knowing" than with "believing" (Maher & Spitzer, 1993). Likewise, Berrios and Fuentenebro (1996) conceptualised delusions as brain events called "empty speech acts". They would be 'empty' because they do not provide information about the brain module where they were formed and their content do not refer to the patient's world or sense of self (Valiente, 2010). In other words, delusions would be fragments of information that have been trapped at the moment of their crystallisation, which have no meaning, but can be said, like any other act of language. From this biological perspective, delusions would be like any other acts of declarative speech, whose origin would be chance or neurobiological events (Berrios, 1991, 1996). Nevertheless, delusion as a belief has been present for most

of the 20th century, despite the criticism received and the limitations to distinguish it from other psychopathological phenomena.

It was also during the 20th century, when phenomenology of delusion emerges with prominent figures like Karl Jaspers seeking to describe mental phenomena with the greatest accuracy as they are experienced by the patient (Spitzer, 1992). Jaspers reconsidered how to study mental illness and outlined, in his book *General Psychopathology* (1975), where the dividing line between normal and abnormal belief could be found (Walker, 1991). On the one hand, a normal belief (i.e., *delusion-like* or *secondary delusion*) would be an 'understandable' phenomenon that occurs in the normal psychic life of the individual, as a consequence of the attempt to explain an abnormal experience or a morbid affective state. In contrast, delusional (i.e., *abnormal* or *primary delusion*) would be an intrusive experience 'not understandable' in terms of the individuals' cultural and educational background, a reflection of an "abnormal" state of mind (Walker, 1991). Then, this *primary delusion*, mainly experienced by psychotic patients, would be characterised by: (a) being held with extraordinary conviction; (b) being resistant to counter-arguments or contradictory evidence; and (c) having bizarre or impossible content (Jasper, 1963). Later, Jaspers himself recognises that these criteria were simply based on "external characteristics" and that the essential criterion to properly distinguish whether a belief is really a delusion or not is the concept of understanding (Valiente, 2010). Thus, an *abnormal* or *primary delusion* would be a direct manifestation of a pathological process, which is incomprehensible because it appears suddenly without any context and has no precedent in the history of the patient's experience or personality (Walker, 1991). Jasper described *primary delusions* themselves in detail by dividing them into: *delusional perception*, where there is an immediate change in the meaning of a particular perception, although the perception itself remains unchanged; *delusional ideas*

or *notion*, that appear as sudden notions, new aspects and new meanings of remembered life experiences and; *delusional awareness*, which is characterised by knowledge of an event without a clear idea or sensory perception (Garety & Hemsley, 1997).

This breakdown of delusions into three groups was later shortened into two by the hand of the German psychiatrist Kurt Schneider (1959). Schneider abbreviated Jaspers subgroups of delusions into *delusional mood or notion* (i.e., an idea provoked by a perception); and *delusional perception* which would be the true delusions (i.e., an attribution of a new meaning, usually self-referent to a normally perceived object) (Koehler, 1979). This way, the *delusional perception* cannot be understood as arising from the patient´s affective state or previous attitudes (Bland & Orn, 1980; Koehler, 1979). It has been suggested that the severity of delusional perception would be the phenomena that better represent the progression of the schizophrenia (Bland & Orn, 1980; Garety & Hemsley, 1997). And so, analogous to the fundamental and accessory symptoms of Bleuler, Schneider developed the so-called *first-rank symptoms* that would constitute the core of the schizophrenia disorder (Bentall, 2004) and include *delusional perception*. Then, these primary experiences need to be differentiated from the *second-order symptoms*, where *delusional notion* would belong to, along with disturbances of mood (Lake, 2012).

The abundant psychopathological literature has therefore conditioned the way paranoia is defined as a disorder (e.g., Kraepelin), as an accessory symptom (e.g., Bleuler) or as a core psychotic manifestation of the schizophrenia disorder (e.g., Schneider). Changes in its conceptualisation are also reflected in the various editions of the Diagnostic and Statistical Manual of Mental Disorders of the American Psychiatric Association (see Table 1). For example, Kraepelin´s paradigm was adopted by the DSM-III (Bentall, 2004) where paranoia is viewed as a ‘paranoid disorder’, a disorder that would become a

‘delusional (paranoid) disorder’ in the III-R version. Latter versions such as DSM-IV and DSM-IV-TR, paranoia is no longer of such substantive importance as an entity and is reintroduced as a characteristic psychotic symptom of schizophrenia and other psychotic disorders. In these latest versions mentioned, paranoid delusion, would be defined as an erroneous belief that usually implies a misinterpretation of perceptions or experiences, with persecution content being one of the most frequent.

Table 1.
Evolution of diagnostic categories of schizophrenia and other psychotic disorders across different versions of the DSM

El DSM-III (1980)	DSM-III-R (1987)	DSM-IV (1994), DSM-IV-TR (2000)	DSM-5 (2013)
Schizophrenic disorders	**Schizophrenia**	**Schizophrenia and other psychotic disorders**	**Schizophrenia spectrum and other psychotic disorders**
- Disorganized	- Catatonic	- Schizophrenia	- Schizotypal Personality
- Catatonic	- Disorganized	- Schizophreniform	- Delusional
- Paranoid	- Paranoid	- Schizoaffective	- Brief psychotic
- Undifferentiated	- Undifferentiated	- Delusional	- Schizophreniform
- Residual	- Residual	- Brief psychotic	- Schizophrenia
Paranoid disorders	**Delusional disorder (paranoid)**	- Shared psychotic	- Schizoaffective
- Paranoia	**Psychotic disorders not elsewhere classified**	- Psychotic due to medical illness	- Substance/Medication induced psychotic
- Shared paranoid	- Brief reactive psychosis	- Substance-induced psychotic	- Psychotic due to another medical condition
- Acute paranoid	- Schizophreniform	- Unspecified Psychotic	- Catatonia
- Atypical paranoid	- Schizoaffective		- Other specified schizophrenia spectrum and other psychotic.
Psychotic disorders not elsewhere classified	- Induce psychotic		- Unspecified schizophrenia spectrum and other psychotic
- Schizophreniform	- Atypical psychosis		
- Brief reactive psychosis			
- Schizoaffective			
- Atypical psychosis			

Note. DSM= Diagnostic and Statistical Manual of Mental Disorders; III= third version; III-R= third version revised; IV= fourth version; IV-TR fourth version revised; V= fifth version.

1.2. Current conceptualisation: paranoia as an individual experience

"Mental health is a matter of degree"

(Read, 2004, p. 52).

As we have seen, during the 20th century, the notion that a delusion might be considered a belief was gradually established. But, the difficulty to identify when a belief is delusional is present throughout the literature (Lewis, 1970). When paying attention to persecutory delusion in particular, the literature is quite scarce and the study of paranoid content had been neglected by psychologists until somewhat recently (Oltmanns & Maher, 1988). However, in the 1990s, there was an upsurge in interest and research in the field of psychotic symptoms by a group of British researchers (Valiente, 2010). These researchers promoted the study of the psychosocial processes underlying this symptom and put the study of paranoia under the microscope. This impetus has been maintained throughout the 21st century and the study of the persecutory delusion has been paramount, particularly given its high prevalence when compared to other types of delusional beliefs. This has resulted in a move away from classical psychiatric classifications and towards an integrative conceptualisation of paranoid beliefs as an individual experience.

1.2.1. Back to the symptom

A classification system based on Kraepelin's assumptions of chronicity, clear delineation between disorders and overly biological is clearly outdated (Bentall et al., 1988; Read et al., 2004). This disjunctive classification system is still in use today, and it has been criticised from clinical and research professionals as inadequate for the study of paranoia for several reasons (a more detailed review of critics of current approaches is provided in Chapter 3). First, despite great efforts to develop precise operational criteria, there are inconsistencies in agreement as to who does or does not meet a diagnosis (Beck

et al., 1962; Spitzer & Fleiss, 1974) and has shown low reliability for schizophrenia (Bentall, 2004). Second, the fact that different individuals could have totally different symptoms and still all have the same "disorder" has also evidenced its low diagnostic usefulness and validity (Bentall, 2004; Bentall et al., 1988). Hence, the set of symptoms associated with schizophrenia might not be enough evidence for a single diagnostic entity (Read et al., 2004).

Alternatively, it has been argued that the study of the symptom, rather than that of global entities, would be a more useful method for the acquisition of knowledge about the processes of psychosis (Bentall, 1993). Shifting the focus to psychotic symptoms rather than schizophrenia shows several advantages: a) it makes psychotic behaviour more understandable and closer to ordinary behaviours and experiences (Bentall, 1993) and thus enhancing the understanding and treatment of difficult experiences for which individuals require help (Freeman & Garety, 2014); b) it would allow us to focus our attention on the phenomenon of interest from a normal-pathological continuity that is more valid than the categorical perspective (Vázquez, Valiente & Díez-Alegría, 1999; Vázquez, 1990); c) it ensures that researchers study similar phenomena (Freeman & Garety, 2000) and; d) it would facilitate the study of these phenomena from a transdiagnostic approach as psychotic behaviours are present in other disorders (Bentall et al., 2008).

Within the schizophrenia spectrum of diagnoses there are multiple experiences, such as paranoia, hallucinations, ideas of grandiosity, disorganised thinking or anhedonia. So, paranoid thinking, like other psychopathological experiences, must be considered as an individual psychotic experience (Freeman, 2016; Freeman & Garety, 2014). But first, we should start by defining clearly what paranoid belief is on its own. As we will see in the next section, although paranoia has been traditionally conceptualised as a symptom

of schizophrenia, there is also evidence that it is a common experience in the general population (Freeman, 2006). Thus, the term paranoia nowadays is used to refer to the complete range of paranoid ideation (Freeman, 2016). That is why, in the literature, the qualification of paranoid or persecutory accompanies not only the noun of delusion, but also that of thought, belief or cognition (e.g., paranoid 'delusion', 'thinking', 'belief' or paranoid 'cognitions'), to refer to the wide paranoia spectrum. More precisely, Freeman (2016) proposed that the term paranoia refers to two essential components, namely ideas of *reference* and of *persecution*. On the one hand, *persecutory* thinking refers to 'unfounded ideas that harm will be produced and that the persecutor has a deliberate intention' (Freeman, 2016), that is, there is a threat to the individual and an intentionality on the part of others. On the other hand, ideas of *reference* are a little less specific (i.e., unfounded cognitions of being observed, followed, discussed, or the subject of messages or communications). In other words, although reference ideas are often the grounds for persecutory beliefs (i.e., the threat is *self-centred*), they can also serve to fund other thinking that may occur in other clinical pictures. For example, ideas of reference may serve to substantiate ideas of grandiosity or may also be part of the difficulties of self-centred attention observed in social anxiety (Clark & Wells, 1995). Unlike persecutory ideation, referential ideation can be experienced in the absence of others' malicious intent to cause harm, what is a specific and defining feature of paranoia (Fenigstein & Vanable, 1992; Freeman, 2016). Moreover, it has been found that persecutory thoughts are more common among clinical individuals, while ideas of reference are more common among non-clinical individuals as opposed to those experiencing psychosis (Green et al., 2008; Ibáñez-Casas et al., 2015).

In addition, a further distinction must be made, in this case, between paranoid thinking and conspiracy mentality. The conspiracy mentality is considered a widespread

political attitude by which individuals follow conspiracy theories that explain complex world events, referring to secret plots hatched by powerful groups (Imhoff & Bruder, 2014). As mentioned, along with others intentionality, paranoia implies the belief that there is harm to the individual. In paranoid thinking, this threat is considered *self-centred* and not as a broader social threat as it might be the case in conspiracy theories. Individuals with a conspiracy mentality believe that there are invisible, intentional forces linked to a paranoid style (Oliver & Wood, 2014), but these ideas do not necessarily have a self-referential component.

1.2.2. Paranoid beliefs classifications (Form vs Content)

Many efforts have been made to classify delusions. One of the most relevant and simple method involves the distinction between form (i.e., the modality in which the belief is presented), and content (i.e., the information contained within the belief) (Bentall et al., 2001). For example, as we have seen above, the distinctions from the point of view of form come from authors such as Jasper and Schneider. They differentiated between *primary delusions* or *delusional perceptions* (respectively) and *secondary delusions* or *delusional notions* (respectively), depending on their (in)comprehensibility (see previous section). Furthermore, within *primary delusions*, four types have been postulated according to their form: a) *delusional intuition*, which would be indistinguishable from any idea that suddenly assails us, its content is usually self-referential and of great importance to the individual; (b) *delusional perception*, which is the delusional interpretation of a normal perception; (c) *delusional atmosphere*, which consists of the subjective experience that the world has changed in a way that is difficult or impossible to define and is often accompanied by a delusional mood, as the individual feels uncomfortable and even perplexed and; d) *delusional memories*, which consist of the

delusional reconstruction of a real memory, or that the individual suddenly remembers something that is clearly delusional (Baños & Perpiñá, 2009).

However, attempts to classify delusions according to their content have proved more successful and reliable when compared to form-based systems (Bentall, 2004). The content (or 'topic') of delusions can be quite varied and there is a wide diversity of content-based classifications. For instance, according to Kraepelin, there are six content subtypes of delusions: sin or guilt, persecution, influence, grandiosity, sexual, and reference ideas (Bentall, 2001; Kraepelin, 1991). Following current classifications such as the one provided by DSM-5, "the content of delusions may vary and they tend to cover a small number of topics" (American Psychiatric Association, 2013, p. 87). The DSM-5 manual (2013) considers several types of delusions according to their content: persecutory, referential (i.e., belief that certain gestures, comments, environmental cues, and so forth are directed at oneself), grandiose (i.e., when an individual believes that he or she has exceptional abilities, wealth, or fame), erotomanic (i.e., when an individual believes falsely that another person is in love with him or her), nihilistic (i.e., the conviction that a major catastrophe will occur) and somatic delusions (i.e., preoccupations regarding health and organ function). Among these, persecutory delusions, defined as "belief that one is going to be harmed, harassed, and so forth by an individual, organisation, or other group", is referred as the most common delusion (American Psychiatric Association, 2013, p. 87). Consistently, empirical studies has shown evidence in favour of persecutory delusions as the most common content-based type of delusion (Garety et al., 1988). For instance, Jorgensen & Jensen found delusion of persecution was the most common among first admitters to a psychiatric hospital, followed by delusions of reference, influence, guilt and others (Jorgensen & Jensen, 1994). Furthermore, this

observation appears to be present across different cultural backgrounds (Stompe et al., 1999).

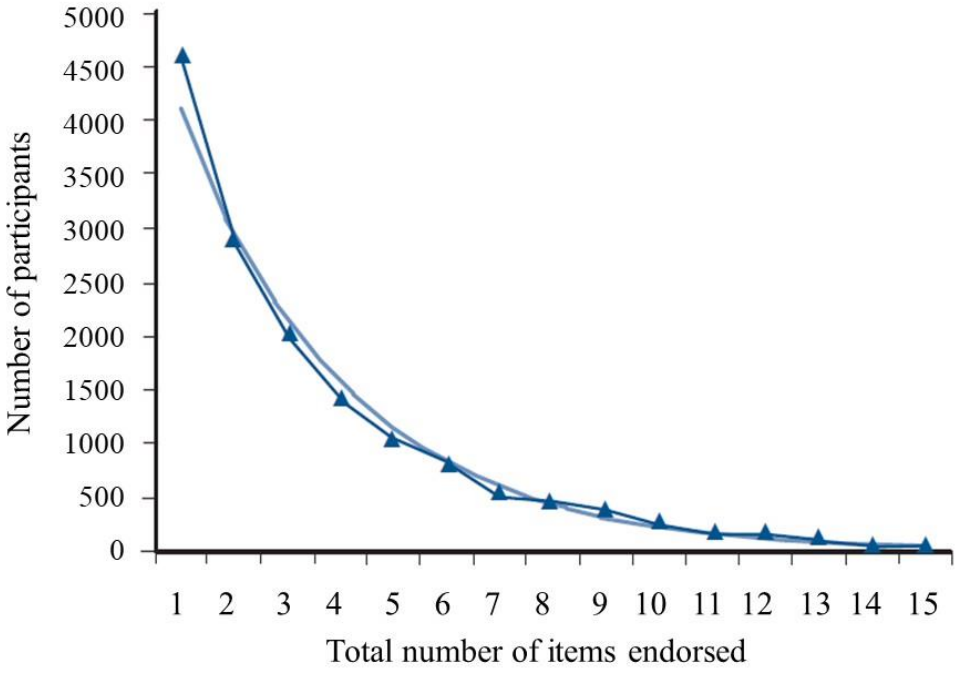
More recently, several authors such as Bebbington (2013) or Freeman (2016) have suggested that the structure of paranoia spectrum contains a number of common factors. By using confirmatory analysis, they provided strong evidence that the structure of paranoia includes four common paranoid cognitions, which are: a) interpersonal sensitivity (i.e., concerns about rejection or about being vulnerable); b) mistrust (i.e., being suspicious or no confidence in others intention); c) ideas of reference (i.e., ideas that others are, for instance, watching or talking about them) and; d) persecution or the idea that there is a threat or harm intentionally caused by others (Freeman, 2016; Bebbington 2013).

1.2.3. Paranoia as a continuum multidimensional process

As we can infer from the previous sections, definitions of delusion are not able to accurately capture where the boundaries between a normal belief and a delusional belief may be (Bentall, 2004). A German philosopher and psychologist, Erwin Strauss (1969) is well-known for proposing, in a novel way, an understanding of delusions by locating these beliefs on a continuum. To date, it has been pointed out that there is an exponential distribution of paranoid beliefs, also in the general population (see Figure 1). At least 10-15% of the general population experiences paranoid thoughts (Freeman, 2007), which is associated with poorer physical and mental health (e.g., suicidal ideation, worry, anxiety), weaker social cohesion and lower quality of life (Bebbington et al., 2013; Esterberg & Compton, 2009; Freeman & Garety, 2014). These findings have led to propose that the spectrum of paranoia may exist in the general population and, therefore, paranoia should

be understood as a continuum ranging from normal and ordinary everyday beliefs to strange and impossible beliefs (Raihani & Vaughan, 2019).

Figure 1.
Distribution of paranoia scores in the general population



Note. Adapted from "The structure of paranoia in the general population" (p. 422), by Bebbington et al. (2013). *British Journal of Psychiatry, 202* (6). In a sample of 8,576 individuals, paranoia was measured by using 4 items of the Psychosis Screening Questionnaire (PSQ; Bebbington & Nayani, 1996) and 11 items of the Structured Clinical Interview for DSM-IV Personality disorders (SCID-II; First et al., 1997) The total number of paranoia items could range from 0 to 15.

Bentall (2004) stated that a clear line cannot be drawn between mental illness and normal functioning and that it seems reasonable to assume, as a general principle, that abnormal behaviour and experiences exist continuously with normal behaviours and experiences. This principle of continuity could be formally stated as follows:

"Abnormal behaviours and experiences are related to normal behaviours and experiences by continua of frequency (the same behaviours and experiences occur less frequently in non-psychiatric populations), severity (less severe forms of the behaviours and experiences can be identified in non-psychiatric populations) and phenomenology

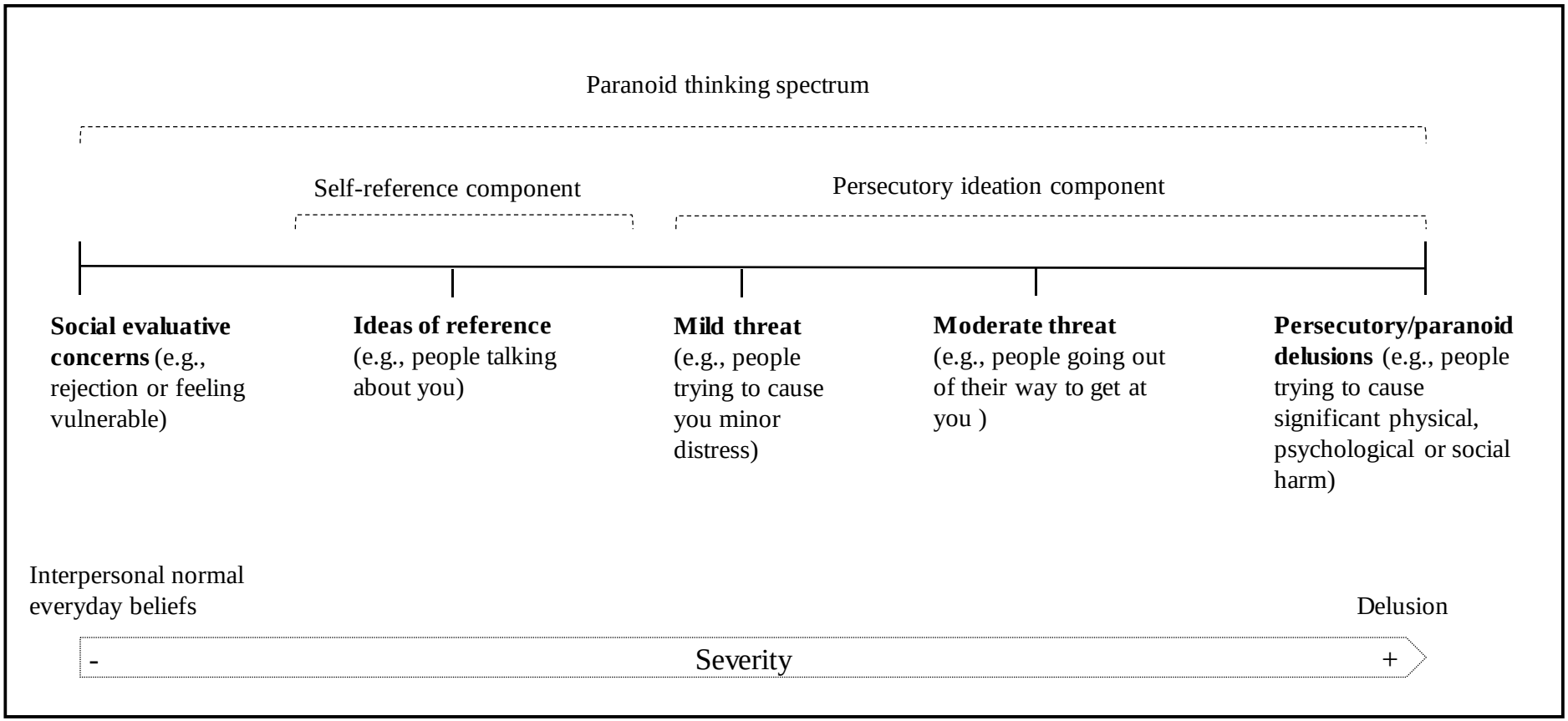
(non-clinical analogues of the behaviours and experiences can be identified as part of normal life"

(Bentall, 2004, p.115).

Thus, the content of our beliefs may vary from social evaluative concerns such as "the fear of rejection" to more severe threats like "people trying to cause significant physical, psychological or social harm (Freeman & Garety, 2014). As it can be seen in Figure 2, paranoid thinking on this continuum would be present to a greater or lesser degree in the general population and persecutory delusion (i.e., the individual believes that harm is occurring, or will occur, to him or her, and that the persecutor intends to cause it) would represent the more severe extreme (Freeman, 2007). That is, when that persecutory thought is sustained with a high degree of certainty, we would be experiencing persecutory delusions (Freeman, 2016).

This conceptualisation of paranoid beliefs as a continuum has been supported by empirical research reporting that some processes are common in the general population, subclinical and people with psychosis (Elahi et al., 2017). Overall, the evidence suggests that people experiencing psychotic symptoms without seeking treatment outnumber those who do seek treatment by about ten to one (Read et al., 2004). For instance, in a survey of 7,076 people, it was estimated that 3.3% of the sample reported delusions and 8.7% had delusions that were not clinically relevant (Van Os, Hanssen, Bijl, & Ravelli, 2000). Therefore, the continuum approach suggests that persecutory delusions do not differ qualitatively from normal beliefs, but simply represent a more extreme mental phenomenon (van Os, & Verdoux, 2003).

Figure 2.
Paranoid thinking continuum



Note. Adapted from "Advances in understanding and treating persecutory delusions: A review" (p. 1181), by Freeman & Garety (2014), *Social Psychiatry and Psychiatric Epidemiology*, *49* (8).

Then, if paranoid beliefs are presented on a continuum, there could be certain characteristics of the beliefs that might help us locate them within this continuum. This is why it has been considered that a more productive way of understanding paranoia would be to apprehend it as a complex multidimensional phenomenon (McGorry et al., 1998). At present, the dimensional approach suggests that symptoms quantitatively differ from normal experiences and behaviours (Johns & Van Os, 2001). Accordingly, it is assumed that delusions may vary with regard to a number of descriptive features that should be studied in their totality, and not as an all-or-nothing phenomenon (Valiente, 2010). For instance, Oltmann and Maher (1988) carried out a review and listed the main characteristics contained in most of the definitions of delusions (Baños & Belloch, 2008). These authors stressed that, when assessing the presence of delusions, some dimensions should be taken into account, none of which are necessary or sufficient, but they may lead to produce greater confidence in their presence. Accordingly, the more unfounded, strongly held, not shared by others, distressing and worrying a belief is, the more likely it is to be considered a delusion (Freeman, 2007).

There is some empirical support that delusional beliefs, like ordinary beliefs and attitudes, vary through a number of characteristics or dimensions such as the duration, frequency, conviction with which they are held (i.e., delusional conviction), the degree to which the individual is concerned about them, and the degree to which they lead to distress (i.e., delusional distress) (Garety et al., 1988; Johns & Van Os, 2001; Kendler et al., 1983; Lincoln, 2007). These dimensions have clinical significance, for instance paranoid thoughts that are more frequent, distressing, and appraised with more conviction and preoccupation has been suggested to be more common among clinical populations (Green et al., 2008; Ibáñez-Casas et al., 2015). In fact, distress, conviction, and preoccupation associated with persecutory thinking have been pointed out as the

determinants of how 'delusional-like' a belief is (Peters et al., 2004). Finally, Trower and Chadwick (1995) suggested that the degree of 'deservedness' is also a factor to take into account. These authors have differentiated between 'poor me' paranoia (i.e., individuals who see themselves as hapless and angry victims so they perceived the persecution as unjust or underserved) and 'bad me' paranoia (i.e. individuals who see themselves as bad and blame-worthy and thus, perceived the persecution as a deserved consequence of an individual's actions) (Trower & Chadwick, 1995). To date, research has provided evidence in favour of 'poor me' paranoia as more common among individuals with psychosis related diagnosis (Melo et al., 2009; Melo & Bentall, 2013).

The identification of reliable dimensions of delusions has several advantages in both, clinical and research practice. First, identifying these characteristics helps to better grasp individual variability in these dimensions, which does justice to the complexity of paranoid phenomena (Freeman, 2007; Oltmanns & Maher, 1988). Delusions are common among general population but may differ from the symptoms experienced by persons with schizophrenia diagnosis. The multidimensional approach is able to capture the high degree of heterogeneity and variation of persecutory beliefs across the continuum (Esterberg & Compton, 2009; Lincoln, 2007). Second, the use of continuous/dimensional instead of dichotomous/categorical variables adds greater statistical power with respect to statistical procedures (Kraemer & Noda, 2004). Finally, a dimensional approach favours the predictive validity of clinical symptoms (Peralta et al., 2002). For instance, it allows to ascertain clinical information necessary to make appropriate recommendations regarding treatment (van Os & Verdoux, 2003).

In short, paranoid ideation is present in the general population, and can be adaptive in some situations but can become a clinical problem when it is excessive, exaggerated, distressing or interferes with functioning (Bebbington et al., 2013). The current

definitions of paranoid belief as a multidimensional individual experience within a continuum might solve many of the problems in defining paranoia to date (Bentall, 2004). However, since a large number of research has neglected the dimensional nature of delusional phenomena, more studies are needed in order to better understand each of these dimensions (Freeman, 2007).

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CHAPTER 2. PSYCHOSOCIAL MECHANISMS AND CURRENT PSYCHOLOGICAL MODELS OF PARANOID (PERSECUTORY) THINKING

Up until now, there has been a great deal of research into possible causes of positive psychotic symptoms (Pickard, 2011). The classical debate between genes and environment has been a theme in this area, with the widespread assumption that schizophrenia (and thus, their symptoms) is a genetic condition of the brain. Thus, bio-research has monopolised resources and scientific attention, with clear clinical implications on how psychotic symptoms have been conceived and approached. Unfortunately, the methodological biases of the genetic literature have led to an overestimation of the importance of heritability in schizophrenia, which has nevertheless, failed to identify specific genes of vulnerability to this disorder (Bentall et al., 2007).

A problematic premise is that high rates of inheritance entails that the environment is excluded as one of the main determinants of this condition. Therefore, most brain and genetic research have either ignored the psychosocial causes of psychosis or relegated them to a secondary role (i.e., triggering or exacerbating a vulnerability) (Read et al., 2009). A recent study with 31,524 twins has estimated that heritability of schizophrenia and other psychotic disorders in the spectrum is 73%, but the concordance rate in monozygotic twins is 33% (Hilker et al., 2018). Thus, although research points to considerable genetic risk, it reveals that the vulnerability of this condition is not determined solely by genetic factors (Hilker et al., 2018; Van Os et al., 2010), indicating that the environment can also play an equally important role in the occurrence of persecutory beliefs (Freeman & Garety, 2014).

This chapter provides a brief overview of psychosocial mechanisms associated with paranoid beliefs as well as current theoretical models accounting for their formation and/or maintenance.

2.1. Psychosocial mechanisms involved in persecutory beliefs

In general, beliefs can be acquired in many different ways, such as through lived experiences, observation, vicarious learning, cultural examples or an interaction between cognitive processes and the environment (Bentall et al., 1994). Hence, the formation of a belief is not an automatic process, but rather a dynamic and multifactorial development where many mechanisms might be involved. Symptom-focused psychopathological research has made some progress in the understanding of persecutory beliefs and several systematic reviews of the literature have pointed to the presence of a variety of psychosocial factors associated to these types of beliefs (Bentall et al., 2001; Freeman & Garety, 2014; Vázquez et al., 2012). The following section tries to address them briefly.

2.1.1. Psychosocial risk

A rich body of literature has consistently supported the role of several antecedents in the onset of psychosis, especially if experienced during childhood, which has led to the suggestion that these experiences could be understood as an adaptation to social context (Bentall et al., 2012).

a) Adverse events. The association between childhood trauma and psychosis is well-established in the empirical literature. A sound meta-analytic review has highlighted that childhood adversity is strongly associated with increased risk for psychosis (Varese et al., 2012). Specifically, these authors revealed that child maltreatment (which include neglect, sexual abuse, physical abuse and emotional/psychological abuse), significantly increased the risk for psychosis 2.90, 2.38, 2.95 and 3.40 times, respectively (Varese et al., 2012). Besides, experiences of peer victimisation (e.g., bullying) have also been stressed as a potential childhood adverse event, which is associated with a 2.39 fold increased odds of having psychosis (Varese et al., 2012). Another adverse event found to

significantly increase the risk of psychosis is parental separation during childhood (i.e., including death of parents or early long-term separation from one or both parents) (Varese et al., 2012). In the latter, compared to controls, people with psychotic experiences are approximately three times more likely to have one parent die and two times more likely to have experienced a long-term separation from one or both parents before age 17 (Stilo et al., 2013).

Interestingly, research on childhood adversity has revealed a certain degree of specificity (Bentall et al., 2012). For instance, while sexual abuse (rape in this case) has been found to be associated with hallucinations only (with an odd ratio = 8.9), paranoia (persecution) is specifically associated with separation experiences (Bentall et al., 2012). In this respect, Bentall et al., (2012) found that individuals who have been raised in an institutional care facility are 11.08 times more likely to experience paranoia. Furthermore, insecure attachment has been found to predict in particular the propensity for paranoia, but not hallucinations. This indicates that specific disruptions of early relationships may be especially important in conferring vulnerability to paranoid beliefs (Bentall et al., 2014; Pickering et al., 2008).

b) *Urbanicity.* There is some empirical evidence indicating that psychotic experiences are more common in densely populated areas and neighborhoods (Allardyce & Boydell, 2006; Freeman et al., 2015; Kirkbride et al., 2014; Pedersen & Mortensen, 2006). A meta-analysis comprising of 46,820 cases with psychosis has revealed that the risk of schizophrenia in the urban environment was 2.37 times higher than in the rural environment (Vassos et al., 2012). Besides, it has been found that the effect of increasing population density on increasing risk of schizophrenia is particularly relevant for adolescents with both, poor social and poor cognitive functioning, which have been considered expressions of pre-existing vulnerabilities to the disorder (Weiser et al., 2007).

According to Weiser et al., (2007), this 'urbanicity effect' does not necessarily imply causation, but still is a very important risk marker (Weiser et al., 2007). In this vein, a recent review has suggested that the association between urbanicity and psychosis is heterogeneous and driven by multiple risk and protective factors (e.g., social and economic stressors) that seem to act differently in different ethnic groups and countries (Fett et al., 2019).

c) Socio-economic disadvantage. Indicators of socio-economic disadvantage and deprivation have received a great deal of attention as an explanation of psychosis risk, especially in urban areas (Croudace et al., 2000). Incidence of schizophrenia is higher in more deprived communities, where social and economic difficulties are likely to be more profound (Kirkbride et al., 2014).

Along with low levels and/or absence of educational opportunities (Freeman et al., 2011; Vázquez et al., 2012), research has found a very robust relationship between first-episode psychosis and social disadvantage (Stilo et al., 2013). For instance, Stilo et al., (2013) found an odd ratio for living alone of 1.19, while being unemployed increased 3.40 times the risk of a first-episode. Interestingly, some research has additionally calculated an index of cumulative social disadvantages and has found that individuals with first episode psychosis were around nine times more likely than healthy controls to report at least two disadvantages (Stilo et al., 2013). Similarly, Wickham et al (2014) replicated that multiple deprivation factors significantly predicted paranoid ideation, a relationship that was partially mediated by stress and trust. Of particular relevance is the fact that individuals who experienced social disadvantage in childhood (in the form of separation, as mentioned before), are between 1-2 times more likely to report social disadvantage in adulthood, suggesting that disadvantage is already well established

before the onset of first symptoms and may be especially toxic in childhood (Stilo et al., 2013).

Finally, a very interesting piece of research conducted in East London neighbourhoods found that social deprivation, social fragmentation and income inequality were associated with increased incidence of (non-affective) psychoses (Kirkbride et al., 2014). Some results indicated that separation from one's ethnic group or group ethnic density were also associated with the risk of psychosis (Kirkbride et al., 2014).

d) Discrimination. In conjunction with what has been said above, there are claims that the association between ethnic group and psychosis is confounded by racial discrimination. For example, it has been suggested that perceived discrimination, especially for people of African and Caribbean origin, induces delusional thinking and contributes to the high rates of psychotic experiences observed in minority populations (Janssen et al., 2003). The paranoid beliefs, in particular, have also been linked to a subjective perception of social exclusion (Westermann et al., 2012).

e) Migration. A personal or family history of migration is also an important risk factor for schizophrenia (Selten et al., 2007). A meta-analysis of 18 studies by Cantor-Graae & Selten (2005) found an increased risk for schizophrenia among first and second-generation migrants. The risk of developing schizophrenia is 2.7 times higher for first generation migrants, while for second generation migrants the risk is 4.5 times higher. Interestingly, subgroup comparisons yielded significantly greater effect sizes for migrants from developing versus developed countries. Besides, they also found a particularly high risk for migrants from countries where the majority of the population was black (with a relative risk of 4.8 points) versus white and neither black nor white (Cantor-Graae & Selten, 2005).

2.1.2. Cognitive processes

During the last two decades, considerable amount of research has attempted to explain paranoid beliefs in terms of cognitives mechanisms that may be implicated in these types of beliefs (Bentall et al., 2001; Freeman & Garety, 2014).

a) Schemas about oneself, the world and others. Cognitive schemas refer to an individual's perception of self, surroundings and others (Ziller et al., 1969). First, according to self-concept (e.g., self-esteem), findings have showed inconsistences. While some studies have reported evidence in favour of a negative self-esteem in people with persecutory beliefs, others have reported a well-preserved self-esteem (Bentall et al., 2001; Freeman, 2007, 2016). Moreover, research in paranoia has also supported the existence of discrepancies between explicit and implicit self-esteem (Bentall et al., 2001; Valiente et al., 2011). It is argued that these inconsistencies can be explained in part because research does not take into account the dynamic aspects of the self, understanding that instability of the self is a crucial aspect related to persecutory ideation (Bentall et al., 2001).

Second, paranoid beliefs have been found to be related with particular schemes about the world. Specifically, these schemes are associated with a hostile attitude towards the world as a reaction to external threats, and a hypervigilance towards possible threats to one' s physical or psychological integrity (Vázquez et al., 2012). Third, research has also evidenced an association between a negative view of others and paranoid beliefs (Garety & Freeman, 2013). Particularly, negative interpersonal schemata have been found to mediate the relationship between loneliness and paranoia, suggesting a potential role of these schemas in the formation and maintenance of paranoia (Lamster et al., 2017).

b) Attentional and recall biases. Individuals with paranoia are highly sensitive to threat and have shown a biased pattern towards material related to personal threat (Bentall et al., 2001). For instance, individuals with paranoia detect and address threatening facial expressions like anger more quickly than other types of expressions (Vázquez). In fact, processes involved in the collection and retrieval of information, such as attentional biases towards threatening information and memory biases towards threatening situations have been found to be related with persecutory ideation (Bentall et al., 2001).

c) *Information processing biases.* Some biases in information processing have been highlighted to be particularly prominent for individuals with paranoid thinking. Especially, biases in probabilistic reasoning like 'jumping to conclusions' (JTC), that is the tendency to make hasty decisions with certainty on the basis of little evidence (Jolley et al., 2014). The presence of JTC has been found to be more common in people with persecutory delusions than in non-clinical populations (Freeman & Garety, 2014)

d) Attributional biases. Since it was proposed that paranoid ideation could be associated with abnormal attributional processes (Bentall et al., 1994), this process has been, along with self-esteem, one of the most studied in relation to the development and maintenance of paranoid beliefs. The most consistent findings in this area indicate the presence of an external and personalising attributional biases where individuals with paranoid ideas attribute responsibility for negative events to others (Bentall et al., 2001; Murphy et al., 2018).

e) *Metacognitive performance.* There is increasing empirical support for the potential role of metacognition in the vulnerability and maintenance of psychotic experiences (Morrison et al., 2011). For instance, an impaired source monitoring (i.e., the ability to distinguish internally from externally generated experiences) in individuals with both, chronic and first-episode delusions has been found (Moritz et al., 2006; Morrison et

al., 2011). For paranoid beliefs in particular, Freeman and Garety (1999) showed that there is a tendency to experience "meta-worry", defined as worry about their ability to control their delusive thinking (Freeman & Garety, 1999; Valiente et al., 2012). Furthermore, research has shown that negative and positive beliefs about paranoia were predictive of the experience of paranoia (Morrison et al., 2005). Experiencing positive beliefs about paranoia (e.g., consider paranoia as a survival strategy to avoid aversive experiences) have been found to predict the frequency of paranoid thinking, while negative beliefs about paranoia (e.g., consider paranoia as a negative result of a negative social experience) predicted distress associated with the ideation (Morrison et al., 2005).

2.1.3. Affective processes

The current state of research in paranoia indicates that there is a large direct contribution of affective processes in both clinical and non-clinical populations (Freeman, 2007).

a) Anxiety. The presence of anxiety symptoms has been found to be related to paranoid beliefs, increasing, almost 10 times, the probabilities of the most severe form of these beliefs, that is, persecutory delusions (Freeman et al., 2011). Meta-analytical work has also supported that anxiety is significantly associated, not only with the severity, but also with the distress and the content of the delusions (Hartley et al., 2013). In addition, research has also shown that social anxiety is associated with paranoid ideation (Combs & Penn, 2004; Martin & Penn, 2001).

b) Worry. It has been suggested that worry in paranoid ideation can be understood in terms of catastrophisation, similar to that shown by people with generalised anxiety disorder (Startup et al., 2007). This catastrophic worry refers to the individual's persistent iteration of the problematic features of his or her concern, causing the person to perceive

systematically worse results (Startup et al., 2007). Adittionaly, empirical research has supported this claim for people with persecutory delusions (Freeman & Garety, 2014; Garety & Freeman, 2013). Furthermore, worry has also been found to predict the persistence of non-clinical paranoia (Freeman et al., 2013) and to predict new paranoid thoughts 18 months later (Freeman et al., 2012).

c) Depressive symptoms. Symptoms of depression as well as related processes such as rumination (i.e., repetitive, negative thinking about one's symptoms) have been found to be common in people with persecutory thinking (Vorontsova et al., 2013). The presence of depressive symptoms may lead to an increase in paranoia as it has been associated with a 7-fold increased risk of experiencing the most severe form of paranoid beliefs (Freeman et al., 2011; Freeman & Garety, 2014). Besides, the aforementioned systematic review conducted by Hartley et al., (2013) revealed that depression, along with anxiety, is also associated with the distress and the content of the delusions.

d) Emotional regulation. It is defined as 'a set of processes where people seek to redirect the spontaneous flow of their emotions' (Koole, 2009, p. 6). Westerman et al., (2011) assessed six types of emotion regulation strategies (i.e., non-acceptance of emotional responses, difficulties engaging in goal-directed behaviour, impulse control difficulties, lack of emotional awareness, limited access to emotion regulation strategies and lack of emotional clarity) and found all of them to be moderately correlated with the frequency and degree of convicction of paranoia. In addition, these authors found that only non-acceptance of emotional responses strategy was exclusively associated with distress related to paranoid ideation (Westermann & Lincoln, 2011). Non-acceptance is similar to the concept of experiential avoidance (EA), which has also been associated to paranoia (Udachina et al., 2014). EA is defined as 'unwilling to remain in contact with particular private experiences (e.g., bodily sensations, thoughts, and emotions)' and

attempts to eliminate such experiences (Hayes et al., 2004, p. 554). It has been shown that individuals with paranoid tendencies reported higher levels of EA than participants without paranoia, which might be particularly damaging at high levels of stress (Udachina et al., 2009). Interestingly, Valiente et al., (2015) found that, in people with paranoid schizophrenia, the combination of high insight and high EA was associated with lower satisfaction with life while those with low insight and high EA had the highest level of satisfaction with life (Valiente et al., 2015).

2.2. Current models accounting for persecutory beliefs

Given the multiple ways in which a belief can be acquired and the many processes that might be involved, it is not surprising that there are several explanatory models of the aetiology of persecutory beliefs (Valiente, 2010). The following are the current psychological accounts that incorporate different combinations of the processes mentioned above to explain the formation and/or maintenance of persecutory beliefs.

2.2.1. Two-factor model

This model proposed by Langdon & Colheart (2000) has its origins in cognitive neuropsychiatry and, although it proposes an explanation of the presence of delusions in general (i.e., all types of delusions and not persecutory in particular), we are compelled to mention it because it is one of the first multifactorial proposals in the literature. The model belongs to what has been called 'belief-positive models' or those models that explain delusions as a breakdown of normal belief formation[3] (Bell et al., 2006).

[3] Bell et al. (2006) differentiated between 'belief-positive models' explained above, 'beliefs-negative models' or approaches that attempt to only explain the pathological process and make little reference to a normal beliefs formation mechanisms, and 'continuum view' or models that consider delusions as one end of a distribution of anomalous mental phenomena.

Accordingly, delusions would occur when there is a disturbance in the normal cognitive system, through which beliefs are generated, evaluated and acquired (Langdon & Colheart, 2000).

To understand the Langdon & Colheart two-factor model, we must first mention Maher's ideas about delusions. Maher proposed that delusions are false beliefs that arise as normal responses to abnormal experiences that are subjected to a wide range of neuropsychological anomalies, such as endogenous neural activation or unrecognised defects in the sensory system (Maher, 1999; Maher, 1974). Hence, delusion might be the result of attempts to make sense of anomalous perceptual experiences. According to Maher, the main difference between delusional and non-delusional beliefs would be the nature and intensity of the experience that is being explained (Maher, 1999). Conversely, Langdon & Colheart (2000) claim that Maher's proposal was not enough to account for a delusional belief, and proposed that a second factor is required in order to explain the transition from unusual experience to delusional belief (Davies et al., 2001).

Then, to explain the presence of delusions, this model distinguishes between two factors or deficits that may be present in the cognitive system (Langdon, & Coltheart, 2000). The first factor refers to the presence of a perceptual aberration, caused by a deficit in the sensory or attentional mechanisms. For these authors, different types of unusual experiences would lead to different types of delusions. The perceptual alteration would influence the nature of what is perceived and would cause delusion which, together with the presence of cognitive biases such as attribution, would be responsible for the bizarre content (Langdon, & Coltheart, 2000). But, as mentioned before, none of these processes, separately or together, would be sufficient to explain the presence of delusional beliefs. A second factor to explain the etiology of delusions would be required, which, unlike the first, would be common to all individuals and types of delusions. This second deficit in

the model is described as a loss of the ability to reject a belief because of its implausibility and its inconsistency with everything else the patient knows (Davies et al., 2001). In other words, it would correspond to a difficulty to discard potentially impossible ideas with the previous individual's knowledge and beliefs that would contribute the person not to reject the delusional belief, despite the presence of evidence against it. It has also been argued that this alteration could be generated by a reasoning deficit, associated with the right frontal cortex (Coltheart et al., 2007).

2.2.2. Theory of Mind

When individuals accurately infer mental states (e.g., beliefs, desires, attitudes, feelings or intentions) in oneself or others, they are said to possess 'Theory of Mind' (ToM; Frith & Corcoran, 1996). Based on a neuropsychological formulation, Frith & Corcoran (1996) argued that individuals with schizophrenia reflect an impairment in the ability to infer others' mental states. For instance, a lack of ability to understand the ideas, thoughts and intentions of others may lead subjects to believe that others have malevolent intentions (Corcoran et al., 1995). Thus, persecutory delusions could be explained as difficulties in accurately inferring intentions of others, that would result in subjects assuming that others are hiding those intentions and, therefore, they must be bad (Bentall et al., 2001).

Research in paranoia has found mixed evidence in relation to ToM. On the one hand, individuals with persecutory beliefs have been found to show worse performance on ToM tasks than control participants (Corcoran et al., 1995), individuals in remission (Corcoran et al., 1997; Randall et al., 2003), and people with schizophrenia without persecutory beliefs (Langdon et al., 2005), as they tend to assume that they are hidden and malicious (Brüne, 2005). On the other hand, it has been found in people with psychosis and that deficits in ToM correlate with negative rather than positive symptoms

(Garety & Freeman, 1999). Therefore, this theory is still contested in paranoia, given that studies point to a dysfunction in the ToM but there are no specific associations between these deficits and persecutory beliefs (Bentall et al., 2001; Brüne, 2005).

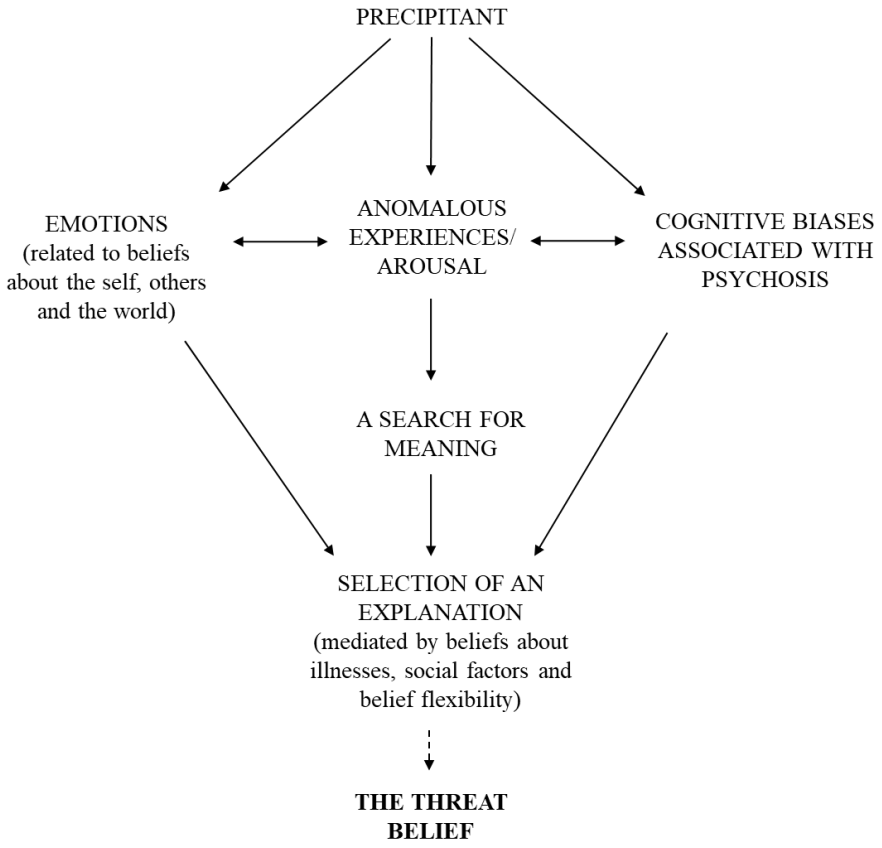
2.2.3. Cognitive model of persecutory beliefs

Freeman and colleagues (2002) propose a multifactorial model of persecutory beliefs that places a special emphasis on anxiety related processes, in combination with psychotic processes (such as associated cognitive bias), pre-existing beliefs of the individual, and the environment. Understanding persecutory delusion as threat belief or that others intend to harm you, these authors in their original proposal, indicated different factors in the formation and maintenance of persecutory beliefs (Freeman et al., 2002).

2.2.3.1 Formation of persecutory beliefs

The vulnerability stress model assumes that the presence of symptoms is the by-product of the individual's vulnerability (that may arise from genetic, biological, psychological or social factors) and stress, whose origin may also be biological, psychological or social (Freeman et al., 2002). Accordingly, it is hypothesised that the onset of a threatening belief like persecutory would begin with a precipitant (e.g., a life-event) that may generate internal or external anomalous or emotionally significant experiences for the individual (e.g., perceptual anomalies). As it can be seen in Figure 1, the authors suggest that this unusual experience could be directly generated by the precipitant, but also can be indirectly generated through other two additional routes, that is, through the individuals' emotional disturbance, or through the activation of cognitive biases associated with psychosis (Freeman et al., 2002). Following the proposed account of persecutory beliefs formation in Figure 1, the individual would then initiate a searching process, in order to find an explanation for the unusual experience.

Figure 1.
Mechanisms underlying the formation of persecutory beliefs



Note. Figure adapted from "A cognitive model of persecutory delusions" (p. 334), by Freeman et al. (2002), *British Journal of Clinical Psychology, 41* (4).

The selection of the explanation might be influenced, again, by the two routes mentioned above. First, the individual's beliefs are related to premorbid levels of anxiety and depression, and they may be reflected in the content of the beliefs. Hence, a persecutory belief is likely to be formed if individuals believe they are vulnerable, they deserve to be harmed, or if they see other people or the world as hostile. Particularly, it is hypothesised that anxiety is the key emotional component in the formation of persecutory beliefs. This is because anxiety and persecutory delusion share thematic content as both refer to the anticipation of danger as the main element. The second influential factor in the formation of a persecutory belief is the presence of cognitive processes associated with psychosis (Figure 1). For instance, cognitive bias such as JTC may limit the amount

of data gathered to support an explanation, whereas attribution bias may cause a tendency to blame others for events (see cognitive biases implicated in psychotic experiences in the first section of the chapter). Finally, the model proposed that the selection of the explanation would also be mediated by several factors such as: previous beliefs about mental illness (e.g., individuals attribute this experience to the individual or to a world situation), social factors (e.g., if the person is isolated or reluctant to talk to others, threatening ideas are more likely to be thrived) and/or belief inflexibility, or the limited ability to consider alternatives may increase the likelihood that the persecutory explanation will be accepted (Freeman et al., 2002).

2.2.3.2 Maintenance of persecutory beliefs

According to Freeman's 2002 model, once a persecutory belief is formed, there are two key factors implicated in their maintenance by selecting evidence that confirm persecutory beliefs. The first factor refers to biases that may affect the process of finding confirmatory evidence for the belief. For example, attentional biases towards threatening information may lead to threatening interpretations of ambiguous events, while the presence of memory biases may lead to frequent presentations in the individual's mind of evidence in favour of the belief. Then, the person may react to his or her belief in a way that provokes hostility or isolation (e.g., being aggressive or treating others suspiciously), which may result in others acting differently towards the person and thus, confirming the persecutory ideas. The second factor involved in the maintenance of persecutory beliefs is related to the processes that make it difficult for the subject to find evidence against his/her belief (non-confirmatory evidence). For example, through security behaviours such as avoidance, individuals take steps to reduce the feared situation rather than processing that the harm was not going to happen in any case. Thus, individuals can reason that their safety behaviours have prevented the potential harm. In addition,

individuals may attribute non-confirmatory evidence to the failure of others' intentions (Freeman et al., 2002) and thus reason that the threat has not yet occurred because the others have not been successful.

Finally, the model includes the hypothesis that emotional distress associated with the belief may appear in two ways: a) beliefs are associated with emotional distress that is reflected in the content of the delusion, which, in turn, feeds back into the belief and increases the emotional distress and; b) assessment of the content of the belief may increase negative emotional reactions (e.g., 'persecution is a sign of failure or evil', 'I am vulnerable in a hostile and dangerous world') (Freeman et al., 2002).

2.2.3.3. Current version of cognitive model

In 2016, Freeman updated their proposal due to the proliferation of new research in the field of paranoid thinking (e.g., Freeman, 2007; Garety and Freeman, 2013). In this version, they grouped the processes involved in both the development and maintenance of threatening (persecutory) beliefs in six categories explained hereafter (see Figure 2).

Figure 2.
The six processes implicated in persecutory beliefs

Note. Adapted from "Persecutory delusions: a cognitive perspective on understanding and treatment" (p. 687), by Freeman (2016), *The Lancet Psychiatry, 3* (7).

1) *Worry*. In this current version of Freeman' model (2016), worry, in terms of catastrophising (Startup et al., 2007), keeps a pivotal role in the formation of persecutory beliefs. Worry shares with persecution the same thematic content, which may bring to mind implausible fearful ideas. Once the belief is founded, the presence of worry would exacerbate the distress and predict the persistence of persecutory delusion (Freeman et al., 2002; Freeman, 2016).

2) *Negative self-beliefs*. Developed in the course of the individual's interpersonal experiences, individuals might have a negative perception of his/her worth. The presence of negative self-beliefs that may lead individuals to feel inferior, different to others, and, therefore, vulnerable, which may not only increase the likelihood of a persecutory belief, but also reinforce the belief once it is founded.

3) *Anomalous experiences*. The presence of anomalous sensations and perceptions can give rise to fearful explanations and thus, to paranoid beliefs. Accordingly, it is common, for example, for the individual to feel a high level of physiological arousal associated with anxiety and this can be interpreted as a sign that indicates an external threat.

4) *Sleep dysfunction*. Following Freeman (2016), sleep disturbances such as insomnia, hypersomnia, circadian rhythm disorder, or nightmares can be considered as a maintaining factor of a persecutory belief through many routes. In short, the presence of sleep disturbances might elevate levels of negative emotion, affect mood dysregulation and elevate abnormal perceptions, which may reinforce the belief once it is generated. In addition, disturbances in sleep can also reduce the individuals' cognitive resources, and thus, prevent the individual to review the original interpretation of an ambiguous situation.

5) *Reasoning biases*. The presence of cognitive flexibility (explained in an earlier version of the model, 2002) or JTC (explained in the first section of the chapter), may prevent the processing of alternative explanations of events. For example, a belief inflexibility can limit the generation of alternative explanations to the threat while JTC may exacerbate such inflexibility given the reduced number of evidence collected.

6) *Safety behaviours*. Defined as actions taken by individuals to reduce a threat. They include avoidance, escape, within-situation behaviours, compliance and aggression. Following this version of the cognitive model (2016), the most common type of safety behaviour in individuals with persecutory ideation is avoidance. The consequence of using these types of actions is that, as explained before, they actually prevent the processing of evidence that confirms the actual absence of the threat. The use of avoidance behaviour will, therefore, have the effect of limiting the amount of potentially reliable evidence to disconfirm the belief.

2.2.4. Defence model or self-serving bias theory

The defence model, also known as the self-serving bias model, is one of the most influential motivational models of persecutory beliefs (Bentall et al., 2001). The model has evolved over more than two decades, during which several versions have been proposed and modified as new evidence has come to light (Bentall, 2004).

In their first version, the authors proposed an integrative model based on the hypothesis that individuals with paranoid beliefs could reflect an exaggeration of cognitive biases observed in a normal population (Bentall et al., 1994). The explanation of the origin and maintenance of beliefs relies on two assumptions. First, since persecutory beliefs imply a concern about relationships with other people, the first assumption proposes that social attribution could be a mechanism involved. The process of attribution refers to the generation of a causal explanation (i.e., an attribution) at the

presence of an event. Usually, the majority of people show a "self-serving bias", where we tend to make an internal attribution if the event is positive (i.e., the cause of the event is due to the individual himself), while we make an external attribution in the presence of a negative event, (i.e., the cause of the event is related to factors external to the person) (Read et al., 2004). Based on findings from several studies (Kaney et al., 1992; Kaney & Bentall, 1989), the authors propose that individuals with paranoid beliefs show an exaggeration of this bias. Particularly, it is suggested that self-serving bias is especially evident when the individual feels threatened or confronted with self-referential material, being prone to blame others (i.e., external-personal attributions) rather than circumstances or chance (i.e., external-situational attributions) (Campbell, & Sedikides, 1999; Kinderman, & Bentall, 1997). Accordingly, the occurrence of self-serving bias when information is particularly threatening may be explained due to the presence of attention and recall bias to the material related to personal threat (Bentall et al., 1994).

The second assumption points to beliefs about oneself as a process involved in paranoid beliefs. Since self-serving bias can be considered a homeostatic mechanism that many of us use to regulate our self-esteem (Bentall, 2004), the authors infer that people with persecutory beliefs may be struggling excessively to protect themselves from the presence of negative self-esteem. Specifically, in this first version of the model, self-esteem is conceptualised as a discrepancy between the *actual-self* and the *ideal-self*. This distinction is made based on the classification from Higgins (Higgins, 1987) between *actual-self* (i.e., how we actually are), *the ideal-self* (i.e., how we would like to be) and the *parent-actual self* (i.e., how we believed our parents see us). In this way, the reasoning that gives name to the theory is that paranoid beliefs would have a defensive function. In other words, individuals would show external and personal attribution to protect their self-esteem, trying not to see the disparity between what they are and what they would

like to be (Bentall et al., 1994). However, the presence of a discrepancy between the self and others would increase the sense of interpersonal threat, causing the individual to perceive that others think negatively about them. That is, if we believe that the negative events that happen to us are due to the bad intentions of others, it is not surprising that we believe that others hate us (Valiente, 2010). In a later version of the model, an alternative conceptualisation of self-esteem discrepancy is offered (see Table 1).

Table 1.
Processes involved and key predictions of the defensive model of persecutory delusions

First account (1994): *'the defence model'*	**Revised version (2001):** *'the attribution-self-representation cycle'*
1. People with persecutory beliefs show an exaggerated *self-serving bias* (external and personal attribution of negative events).	1. People with persecutory beliefs show an exaggerated *self-serving bias* (external and personal attribution of negative events).
2. Persecutory beliefs imply a *self-esteem discrepancy* between actual-self and ideal-self.	2. Persecutory beliefs imply a *self-esteem discrepancy* between explicit and implicit self-esteem.
3. Self-serving bias minimises discrepancies between self-esteem, maintaining positive self-concept, but causes *negative beliefs about others*.	3. Self-serving bias prevents low implicit self-esteem to become conscious or explicit, having positive and negative effects on self-esteem, but causes *negative beliefs about others*.
	4. Attribution and self-esteem mutually influence each other, leading to a dynamic process that result in an inherent *instability of self-esteem* that will increase the severity of persecutory delusions.

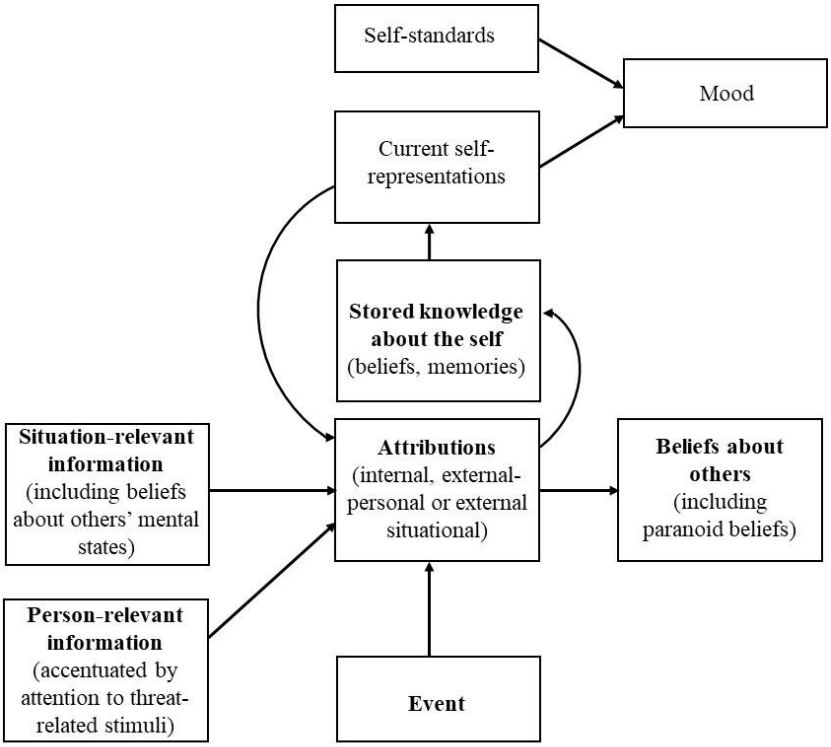
Note. This table summarises the processes and predictions of both versions of the defensive model of persecutory delusions. The content has been adapted from "The self, attributional processes and abnormal beliefs: Towards a model of persecutory delusions" by Bentall et al., (1994), *Behaviour Research and Therapy, 32* (3); "Persecutory delusions: A review and theoretical integration" by Bentall et al., (2001), *Clinical Psychology Review 21* (8) and; "The paranoia as defence model of persecutory delusions: a systematic review and meta-analysis" by Murphy et al., (2018), *The Lancet Psychiatry 5* (11).

Given that self-esteem could involve unconscious and automatic self-evaluations, a distinction is made between explicit and implicit self-esteem (Bentall et al., 2001; Kinderman, & Bentall, 2000). Explicit self-esteem refers to a cognitive mode achieved through the conscious and rational processing of self-relevant information, whereas implicit self-esteem concerns to an automatic and intuitive mode (Valiente et al., 2011). The updated prediction is that individuals with paranoid beliefs would show low levels of implicit self-esteem and that, through external attribution, they would prevent implicit self-esteem from reaching consciousness or becoming explicit (see Table 1).

But undoubtedly, the most interesting aspect of this updated version is its novel proposal on the dynamic relations between the processes involved in persecutory beliefs. The authors propose the *attribution-self-representation cycle* (see Figure 3) to reflect that the defensive function of paranoia has a dynamic nature, where the processes of attribution and self-esteem influence each other (Bentall et al., 2001).

In this cyclical process, the attribution would have a direct influence on one's representations of oneself and an indirect influence on the individual's mood through these representations. This constant reciprocal influence would result in an inherent instability of self-esteem, meaning that self-esteem scores would fluctuate over time, and would be associated with the severity of persecutory belief.

Figure 3.
The Attribution-Self-Representation Cycle



Note. Figure adapted from "Persecutory delusions: A review and theoretical integration" (p. 1168), by Bentall et al., (2001), *Clinical Psychology Review*, *21* (8).

The large number of studies motivated by this new proposal has culminated in a recent meta-analysis that provides evidence in favour of several of the predictions of both versions of the model (Murphy et al., 2018). Specifically, they reported a moderate amount of evidence supporting the idea that the severity of paranoia is positively correlated with the degree of attributional bias, and in favour of the presence of a discrepancy between implicit and explicit self-esteem. In addition, there is strong evidence supporting that the instability of self-esteem correlates with the severity of paranoia in people with psychosis. However, according to this meta-analysis, supporting evidence in favour of this instability of self-esteem is based on only four investigations. Therefore, despite the large number of studies that pay attention to the attribution and self-esteem discrepancy, very few have shown it to be unstable in people with paranoia.

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CHAPTER 3. CURRENT CLASSIFICATION SYSTEMS IN PSYCHOPATHOLOGY: NETWORK ANALYSIS (NA) AS AN ALTERNATIVE [4]

"From the remotest period in the history of the world it has been seen that organic beings resemble each other in descending degrees, so that they can be classified in groups which are subordinated to each other. This classification is not arbitrary, like the grouping of stars into constellations. The existence of groups would have been of simple significance if one group had been adapted exclusively to living on land and another to living in water; one to feeding on meat and another on vegetable matter, and so on; but the case is very different, since it is notorious that, very commonly, different members of even the same subgroup have different customs".

(Darwin, 1895)

The task of classification is not only inherent to any scientific activity, but to the very need of human beings to find and give order to the events that occur in the world (Vázquez, 1995). As with other phenomena in nature, there have been several attempts to classify psychopathological behaviour. In the field of mental health, the classification of disorders has traditionally been based on categorical systems, as reflected in schemes such as the Diagnostic and Statistical Manual of mental disorders (DSM), by the American Psychiatric Association, or the International Classification of Diseases (ICD), by the World Health Organization. In recent decades, the usefulness and validity of these models have been called into question by a significant part of the scientific and clinical practice community (Berrios, 1996; Fava, 2014; Insel et al., 2010). Consequently, new

[4] Part of the information contained in this chapter corresponds to the published article: Blanco, I., **Contreras, A.**, Valiente, C., Espinosa, R., Nieto, I., & Vázquez, C. (2019). El análisis de redes en psicopatología: conceptos y metodología. *Psicología Conductual*, *27*(1), 87-106.

alternatives on how to approach the psychological phenomena have emerged, among them, the so-called Network Analysis (NA) as one of the most promising perspectives (Borsboom & Cramer, 2013). This chapter describes the main characteristics of the traditional classification approach in psychopathology, the received criticisms in recent years and it describes the theoretical account of mental health problem from the novel alternative known as Network Analysis.

3.1. Classification systems in Psychopathology

For a long time, research and clinical practice in clinical psychology has been dominated by a model of thinking inspired by medical sciences (López & Costa, 2014). The influence of the work to classify mental disorders by Kraepelin and Bleuler (described in Chapter 1) remains influential to the present day, conditioning the study and treatment of mental disorders and their symptoms, specially so for psychotic phenomena (e.g., considering schizophrenia to be a chronic illness and outside the spectrum of normality, conferring unjustified pessimism on the chances of recovery or ignoring personal, family or social factors) (Read et al., 2004).

The approach most commonly adopted in the study of psychopathology is the well-known categorical system, which proposes that there are separate categories into which mental disorders can be classified. Consequently, for each category a number of criteria are made explicit, so that it is possible to identify with certainty whether or not individuals belong to that particular category (Vazquez et al., 2014). For example, the concept of "schizophrenia," introduced in 1950 as a disorder, was accompanied by a list of symptoms (or behaviours) that, when experienced together, made up that diagnostic category (Read et al., 2004). Moreover, such diagnostic categories should be mutually exclusive and independent, so that an individual classified into one category could not share characteristics of the other. One of the most relevant expression of this

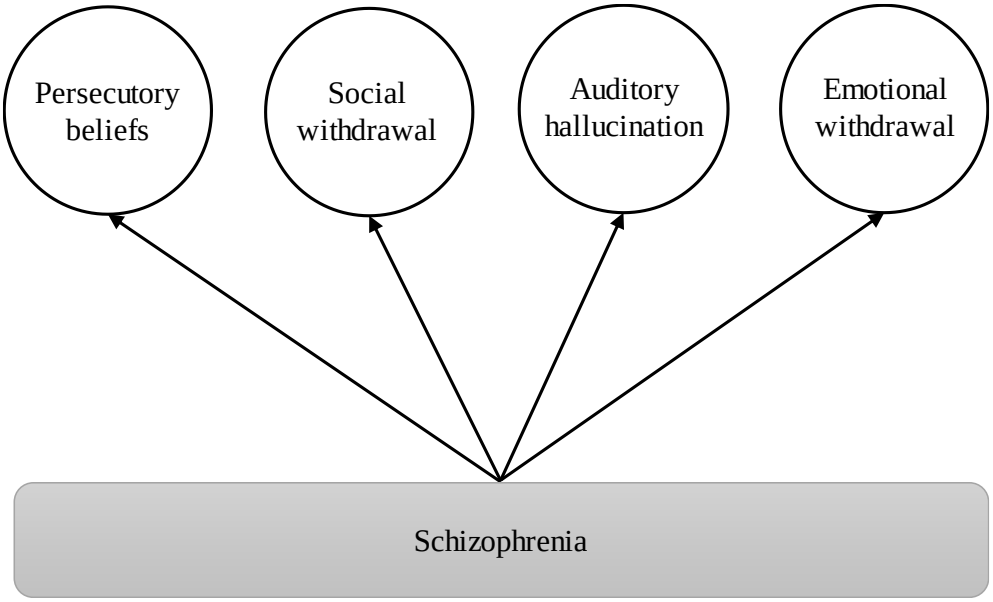
compartmentalized view of mental disorders is reflected in current diagnostic systems such as the DSM, since 1980 and the ICD, since 1992, in which the existence of operational criteria aims to reliably assign an individual to a given diagnostic category to which he or she is alleged to belong.

It is remarkable that the categorical approach attributes a binary character to each category (i.e., there is presence or absence of disorder). Furthermore, as in the field of medicine, it is assumed that there are underlying entities that would give rise to the existence of the symptoms, that is, it is assumed that there is a latent variable that constitutes the disorder. In this vein, similarly to the medical model, it is assumed that an unobservable entity is causing the presence of such mental symptoms. So, by generating a circular argument, the symptoms exist because such disorder "exists" (Guze, 1992).

Imagine the case of C., a 35-year-old female who shows a set of symptoms characteristic of the diagnosis of schizophrenia. She has developed persecutory beliefs following a violent relationship with a former partner. C. believes that her ex-partner is following her as he pretends to rape and humiliate her. She believes that this person is on every single street corner, trying to get information about her and commanding others to hurt her too. She shows lack of involvement in daily life’s activities because she focuses all her energy in her perceived sense of threat and avoids leaving the house. She shows a diminished interest in social interactions due to a lack of energy and fear, as she believes that others are conspiring against her, what leads to isolation. Probably connected in part to her extreme isolation and a disrupted sleep patterns due to her sense of extreme threat and hypervigilance, C. has also started to hear voices commenting on her actions and

criticizing her life[5]. Following the categorical mode, the fact that C. "has" schizophrenia, is what would explain the display and co-occurrence of persecutory beliefs, auditory hallucinations, social and emotional withdrawal (see Figure 1). Thus, these symptoms would not appear by previous experiences or circumstances, but by a series of biological mechanisms driven by that underlying disorder (latent entity).

Figure 1.
Conceptualisation of mental problems from the categorical model



Note. Adapted from "Network Analysis: An Integrative Approach to the Structure of Psychopathology" (p. 94), by Borsboom & Cramer (2013), *Annual Review of Clinical Psychology*, *9*. The figure conceptualises the example of C. case from the categorical approach. Schizophrenia would represent the latent entity (the disorder) while persecutory delusions, social withdrawal, auditory hallucinations, and emotional withdrawal are its observable symptoms. Accordingly, the presence of the schizophrenia (an unobserved variable) is causing the presence of the symptoms (observable variables).

[5] This example has been adapted from the case study presented in the book "Acceptance and commitment therapy and mindfulness for psychosis" (chapter 3) by Morris, Johns & Oliver (2013).

Along with the categorical system, dimensional models are also common conceptualization systems in psychology, and particularly in the field of personality (Kupfer, 2005). Unlike the categorical, the dimensional approach does not assign individuals to categories according to an inclusion criterion, but rather classifies them along a set of dimensions. This classification is quantitative and uses a continuous criterion of the degree to which a certain characteristic appears or is represented in the individual. This allows the combination of several clinical attributes at the same time, where psychopathology and normality can be interpreted as extremes of the same continuum and not as separated phenomena (Vazquez et al., 2014). The current version of the DSM-5 (American Psychiatric Association, 2013) emphasizes the value of adopting a dimensional perspective, both for clinical and research purposes, including a dimensional approach to some disorders to complement the categorical diagnosis. For instance, psychotic symptoms are now conceptualised as non-exclusive to schizophrenia and the diagnostic category is re-labeled as *Schizophrenia Spectrum Disorder and other Psychotic Disorders.* The manual also indicates that the dimensional assessment can better capture the variation in symptom severity and provides a scale to quantify the severity of the primary symptoms of psychosis[6] (American Psychiatric Association, 2013).

On a theoretical level, a classification system, whether categorical or dimensional, should serve the following purposes to be worthwhile (Blashfield, 1984). To begin with, the nomenclature and the use of common terms (e.g., operational criteria and diagnostic

[6] By using the scale *Clinician-Rated Dimensions of Psychosis Symptom Severity* (see section III, Assessment Measures in DSM-5, APA, 2013), each of the primary symptoms of psychosis (i.e., delusional ideas, hallucinations, disorganized speech, abnormal psychomotor behaviour and negative symptoms) can be classified by its current severity, on a 5-point scale (0=absent; 1=equivocal; 2=present but mild; 3=present and moderate and; 4=present and severe).

labels) should facilitate the communication among mental health professionals. People working in the field need a common language, with basic terms to refer to the wide variety of psychological manifestations they work with. Second, the existence of a classification should guide the management of relevant clinical information. For example, knowing that a person has a specific diagnosis could be used to help the clinician know where to look for related information (e.g., probable symptomatology), to make predictions about the prognosis, course or options for therapeutic intervention (Goekoop & Goekoop, 2014). Third, the descriptive information provided about diagnosis should help moving forward in the study of the origin of the mental health problem. That means that the presence of covariation among the symptoms within each category should facilitate the study of the aetiology of mental health problems in each subgroup of individuals within the category.

3.2. Criticisms of classification systems

Although traditional classifications systems serve the functions identified by Blashfield (1984), the debate on its utility is still on the rise. Whether categorical or dimensional, the current diagnostic classification system has been heavily criticized and termed as inadequate for the study of mental disorders (and schizophrenia in particular) for several reasons we convey hereafter (Bentall et al., 1988; Insel et al., 2010; Kraemer et al., 2004; López & Costa, 2014; Read et al., 2004; Vazquez et al., 2014).

First, diagnostic labels are theoretical constructs aiming to organize a wide range of psychological manifestations, but they do not represent "the reality". This implies, on the one hand, the absence of objective criteria to support the presence of a diagnosis, unlike in the medical field (Vazquez et al., 2014). For instance, the term schizophrenia, is used to "label" the presence of a similar group of symptoms, but there is no irrefutable evidence that supports the diagnosis. On the other hand, the symptoms are not equivalent, so the sum-score strategy is questioned. That is, two individuals “with schizophrenia”

may have different symptoms and might not represent the same reality. As it was conveyed by Darwin quoted in the opening of this chapter, the existence of a classification would make sense if all members of a category shared the same characteristics. In nature, however, it is observed that members of the same group (i.e., category) may exhibit different habits. Obviously, Darwin was referring to organic beings (e.g., plants or animals) but the same is applicable to human beings, where the repertoire of psychological manifestations (e.g., symptoms) is very varied within a category (i.e., diagnosis). Therefore, the existence of different presumably equivalent criteria limits the study of schizophrenia, since different diagnoses do not offer coinciding results (Cuesta et al., 2007).

Secondly, existing diagnostic systems have shown less consistency and usefulness in making a proper diagnosis than was previously assumed (Spitzer & Fleiss, 1974). For instance, in terms of *diagnostic reliability* (or *inter-judge reliability)* that is the degree of agreement that subjects are classified in the same category (Vazquez et al., 2014), evidence has shown that the rate of diagnosis concordance is 54% (Beck et al., 1962) and 57% for the diagnosis of schizophrenia in particular (Spitzer & Fleiss, 1974). Interestingly, reliability has also showed a wide cross-cultural variability (Copeland et al., 1971; Sartorius et al., 1978). In terms of *diagnostic validity*, that is accuracy in assigning individual with similar experiences to the same category, four relevant areas have been identified, namely, the utility in identifying clinical characteristics, aetiology, course of the disorder and treatment (Spitzer & Fleiss, 1974). Unfortunately, the diagnostic label of "schizophrenia" is not an useful category for any of these validity areas and, therefore, this diagnosis does not help predicting the specific symptoms experienced by a particular individual, nor the cause of the symptoms, the course of the clinical

picture, or the appropriate treatment for that person (Bentall, 2004; Goekoop & Goekoop, 2014).

Third, these systems include signs and symptoms, but may disregard other clinical variables. For instance, in relation to delusions, core cognitive processes involved in the origin and maintenance of persecutory beliefs do not appear as relevant information in current diagnostic classification, despite their empirical evidence (see Chapter 2). It is essential to evaluate other relevant areas (such personal, motivational, cognitive or social) in order to make a comprehensive clinical formulation and sound treatment decisions (Vazquez et al., 2014).

Fourth, there are very high diagnostic comorbidity rates, probably due to many reasons such as the very nature of mental disorders, the use of broad and imprecise criteria, as well as the overlapping symptoms present in different disorders. According to data from the National Comorbidity Study, in 79% of people who have a mental disorder, there exists, has existed in the past, or will exist in the future, another diagnosable mental disorder using DSM criteria (Kessler et al., 1996). Comorbidity does not only occur between disorders within the same category, but also between disorders in different categories (Kessler et al., 1996). Categorical diagnostic systems have not been successful in addressing the very common phenomena of comorbidity (Cramer et al., 2010).

Fifth, criteria and definitions of psychological disorders change over time. From one edition of the DSM or ICD to another, new labels are introduced, and others disappear (see Table 1 in Chapter 1). Despite great efforts to improve traditional diagnosis, the current perspective continues to assume the presence of an unobservable latent entity, making scientific and clinical progress in this field difficult (Vazquez et al., 2014).

Finally, it is inevitable to associate a mental disorder category with a diagnostic label which entails serious negative social and emotional consequences, including social

stigma and self-stigma imposed on the individual who receives it (Corrigan & Kleinlein, 2005). Diagnosis have the potential to become self-fulfilling prophecies that have strong effects on the person receiving the label (Blashfield, 1984).

To sum up, during the last decade, there is a growing consensus among mental health experts that these limitations are a constraint on the knowledge acquisition of psychopathology and have been interpreted as a failure of the medical model in psychopathology (Insel, 2013). Logically, these limitations have also burst into the study of schizophrenia. In this area, it has been argued that there is no evidence of schizophrenia as single diagnostic entity (Bentall, 1993; Bentall et al., 1988) which has led to advocacy for the study of their symptoms as a better method of knowledge acquisition in psychosis (see Chapter 1).

3.3. Alternatives to classification systems

The crisis of confidence in traditional diagnostic systems has stimulated several movements by the scientific community in order to address the identified limitations. The National Institute of Mental Health (NIMH), one of the main research funding agencies in the United States, has prompted to discourage the use of the DSM in their submitted research projects (Insel, 2013) and thus, other alternatives have begun to be contemplated. For instance, complementary to the traditional classification, the Research Domain Criteria (RDoC) has been proposed by NIMH. Indeed, it is not considered a classification model that could replace the current diagnostic system, but rather a complementary research strategy for systematically organizing findings into basic dimensions of functioning (Tamayo, 2018). In order to establish a taxonomy of mental processes in consonance with the evidence, the RDoC perspective proposes a matrix of elements. This matrix is dynamic, as it evolves according to new findings, and integrates different levels of information to explore the range of behaviour from normal to abnormal. To this aim,

the matrix rows contemplate six domains of human functioning (i.e., systems responsible for responses to aversive situations; positive situations; cognitive systems; social processes systems; arousal/regulatory systems and; sensorimotor systems) while the columns contain different levels of analysis such as genetic, molecular, or behavioural (an in-depth desciption is elsewhere, Morris & Cuthbert, 2012; Sanislow et al., 2010).

However, according to Cuthbert & Insel, (2010), the rationale for the RDoC framework is to facilitate the translation of modern molecular biology, neuroscience, and behavioural approaches to explain the pathophysiology of disorders. It, therefore, maintains a conceptualization of mental disorder as a brain disorder (Insel et al., 2010). These notions have led some authors to question the possible implications of the RDoC in the study of schizophrenia, as it may lead to a refinement of Kraepelin faulty assumptions or the emergence of new entities defined by genetics or neurobiology (Cuthbert & Insel, 2010).

3.3.1. Network Analysis; Concepts and methodology

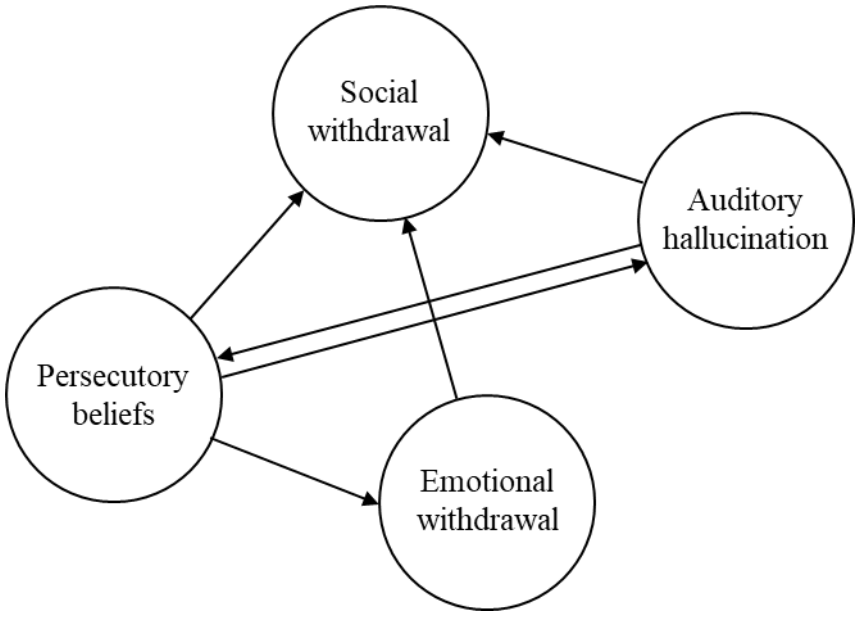
> "What are mental disorders in the first place?"
>
> (Borsboom and Cramer, 2013, p. 92).

Despite the current lack of objective evidence on the diagnosis and possible causes of mental disorders, it is blatantly obvious that the manifestations of mental problems create patterns of covariance (Borsboom & Cramer, 2013). Indeed, the symptoms of, for example, a delusional disorder, are more associated with other delusional experiences than with symptoms of another disorder. While the current model in psychopathology explains this association between symptoms by addressing a supposedly common underlying disorder, the so-called *Network Analysis* (Borsboom & Cramer, 2013) has emerged as a promising alternative approach to explain the existence of such covariation

patterns and well as to provide an appealing way of thinking about the nature of psychological phenomena (Borsboom, 2017; McNally, 2016).

According to the network model in psychopathology, the presence and symptomatic covariation in mental health would not be a consequence of the presence of a latent entity (Borsboom & Cramer, 2013). Conversely, "life problems" are the result of dynamic and causal interactions between the symptoms themselves (Borsboom, 2017). Following the example of C. described in the previous section, the symptoms showed by C. do not reflect necessarily any underlying entity but rather, the symptoms themselves are the problem (see Figure 2). Therefore, what we usually call "mental disorder", would actually consists on a complex system of symptoms that cause each other and also other variables that are possibly dynamically self-reinforcing (Cramer et al., 2010). Thus, symptoms constitute the disorder (McNally, 2016).

Figure 2.
Conceptualisation of mental problems from the Network Analysis theory



Note. Hypothetical network structure of C. case example. The circles represent the symptoms (observable variables) which constitutes the “disorder” itself.

The theory proposed by the network perspective is not new, but its use in the field of psychopathology is. The construction of complex networks has its roots in disciplines of physics and mathematics (Erdös & Rényi, 1959) and in recent decades, have provided strategies and research methods in several scientific fields for studying complex organizations of elements such as neuroscience or sociology (Barabási, 2016). Lately, it has generated considerable acceptance by the psychopathological scientific community (Borsboom & Cramer, 2013; Contreras et al., 2019; Fried et al., 2017; Robinaugh et al., 2019).

The official appearance of Network Analysis in the psychopathology field has been dated in 2008 at a conceptual level and in 2010 at an empirical level (Fried et al., 2017). And, from the Network Analysis theory has derived the so-called "Network psychometrics", which involves all the methodological tools to study mental problems as complex organizations of elements (Epskamp, Borsboom, et al., 2018). The process of building a psychological network model call upon statistical packages incorporated to the *R* and *Rstudio* software (Team, 2013). By doing so, the researcher will use different *R* packages to estimate different types of network depending on whether data is cross-sectional (i.e., data collected at one time point, in which cases are independent), temporally ordered datasets (i.e., single case or multiple cases with several time points measures) (Epskamp, Waldorp, et al., 2018). Thus, the network estimation would depend on the type of variable and the aim of the study. One difference to highlight between networks in psychopathology and in other disciplines, is that the associations between variables are not observable, but rather need to be estimated (Epskamp, Borsboom, et al., 2018; Epskamp & Fried, 2015). The next sections offer a more detailed description of the novel theoretical and methodological proposals from this approach.

3.3.1.1. The nomenclature

Mental problems are theoretically conceptualised as networks of symptoms that mutually interact, but a researcher can estimate the statistical relationships between the variables forming the network structure and visualize it. The visualization and exploration of network properties in psychology is based on *Graph theory*, a branch of mathematics and computer science that studies the properties of what is called a "graph", or a visual representation of a data structure (Barabási, 2016).

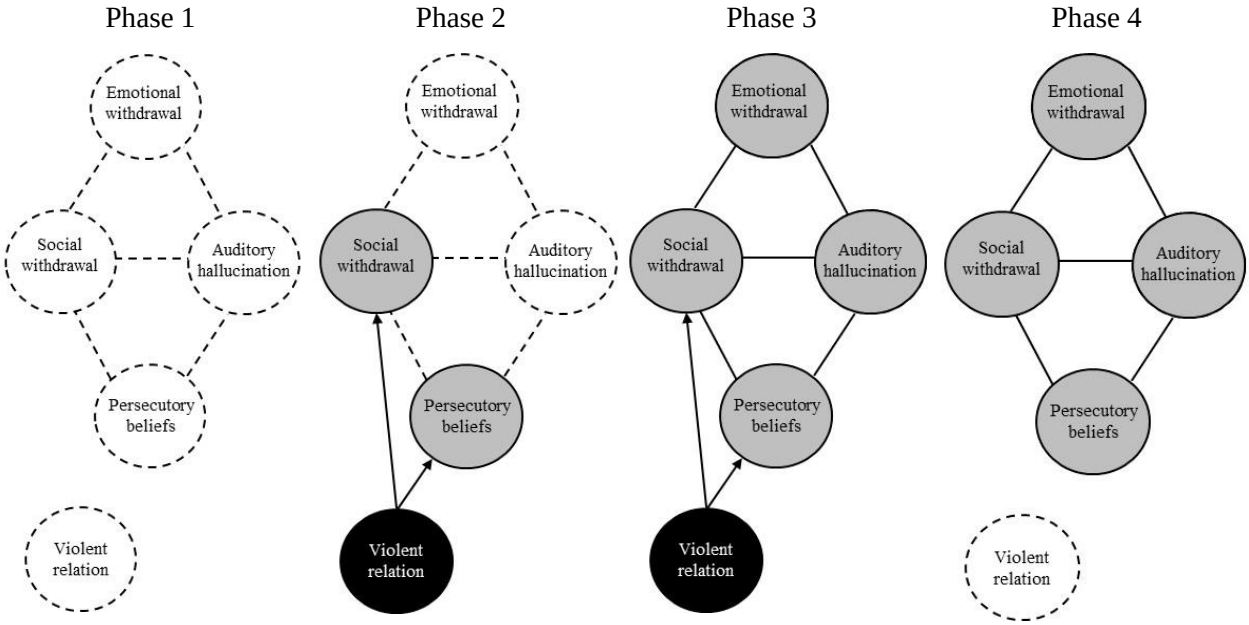
In this way, in psychological networks two elements are distinguished. The first component is the variables under study, which are represented as *nodes* (circles) (see Figure 2). In most cases, nodes would depict symptoms, but they can certainly represent any observable variable. That is, network analysis allows the inclusion of other clinically relevant variables and study how they interact with other elements in the network. Furthermore, since the word "symptom" refers to the medical model, it has been proposed to abandon it and replace it with the concept of "element" (Hofmann et al., 2016). The second component of a network are *edges*, or links (the line connecting two nodes) that represent the association (statistical relationship) between a pair of nodes. The valence of the association is usually depicted with colours, with red lines for negative associations and green (sometimes blue) lines indicating positive associations. In the last section of this chapter we will see that statistical relations represented in edges might be different depending upon the type of network estimated.

3.3.1.2. Hysteresis, vulnerability and resilience

According to the network perspective, an episode of a "disorder" can occur whenever a certain number of symptoms are activated for a sufficient amount of time, while remission occurs when the symptoms are deactivated, the links between them dissipates, or both (McNally, 2016). Thus, mental problems could be understood as an

activation processes between symptoms even after the triggering cause is no longer in place (Borsboom, 2017). This maintenance of activation has been called *hysteresis* and would be observed mainly in strongly connected symptom networks (see Figure 3).

Figure 3.
The hysteresis phases



Note. Adapted from "A network theory of mental disorders" (p. 9) by Borsboom (2017), *World Psychiatry, 16* (1). Phases in the development of a mental problem from the network perspective. Following the example of case C., Phase 1 reflects an asymptomatic state, when the violent episode has not appeared yet. Phase 2 reflects how an external event such as the violent relation has activated some of the symptoms, specifically persecutory beliefs and social withdrawal. In Phase, 3 symptoms activated in previous stages are now activating other symptoms. If the network is strongly connected, the removal of the event (C. does not have a relationship with her ex-partner anymore), does not lead to recovery. In Phase 4 it is observed how the network is self-sustaining in an active state, event after the element that cause the activation in no longer present.

Then, following the notion of hysteresis, network theory offers a new perspective to comprehend psychological phenomena such as vulnerability and resilience. For example, the phenomenon of hysteresis could explain the activation of strongly interconnected networks that make them particularly vulnerable because the activation of

one node can easily lead to the activation of neighbouring nodes. Thus, the concept of vulnerability would be understood as the arrangement of strongly connected networks in the face of a disturbance in the external field. However, the network will be resilient if the connections between symptoms are not strong enough to become autonomous and gradually recovers and returns to its asymptomatic state. A resilient network is, therefore, defined as one with the tendency of weakly connected networks to quickly return to their stable state of mental health (Borsboom, 2017). This comprehensive analysis of the patterns of interconnection and activation between symptoms may help to better understand some aspects of mental problems such as relapses, recurrences or coping.

3.3.1.3. Centrality

If we consider a mental problem as an interaction of symptoms (nodes), the notion of centrality proposed by the network theory allows to identify those nodes with greater relevance within the interacting network structure (Bringmann et al., 2019). Unlike the traditional view, that assumes that symptoms are equivalent, Network Analysis allows quantifying the importance of each specific node within the network (Fried et al., 2017). McNally (2016) has made a great compilation of metrics that allow the estimation of the different types of centrality from the network structure:

a) Degree Centrality. The degree centrality of a node is the number of edges linked to it. The higher the degree centrality, the greater the number of nodes that are connected to it. Accordingly, this central node would be clinical importance due to the high degree of interaction with the other symptoms (nodes).

b) Strength Centrality and Expected Influence. In networks where edges represent the magnitude of the associations between the nodes (see section types of networks), the strength centrality do not take into account the number, but the magnitude of all associations in the network. Then, this metric is the sum of the weights (e.g., correlation

coefficients) of the edges connected to a node. This would represent the probability that the activation of that node is followed by the activation of other symptoms (i.e., the more likely it is that other nodes will be activated). For directed networks (i.e., those that depict the direction of the association), two different types of strength index can also be calculated: *in-strength centrality* (or the magnitude of all association a node received from other nodes); and *out-strength centrality* (or the magnitude of all association a node emanate to other nodes in the network).

However, it has been suggested that strength centrality index might have a limitation. Given that is calculated from the 'absolute' magnitude, this metric works as long as the networks structure show only positive edges. Strength index does not discriminate between positive and negative edges and it may not properly assess the strength of a node's influence within the network (Robinaugh et al., 2016). For this reason, *Expected Influence* measure has been proposed to better assess the strength of the associations, as it takes into account both, positive and negative edges (Robinaugh et al., 2016).

c) Closeness Centrality. The closeness of a node is the average distance of that node from all other nodes in the network. It is, then, the shortest path between a certain node to the rest. A simple example to understand this metric would be the logic of the well-known *Google maps application* for smartphones, where from one location to another, it calculates the shortest path.

d) Betweenness Centrality. This index refers to the number of times a node is on the shortest path between two other nodes. In order to calculated it, first, the length of the shortest path between each pair of nodes in the network is calculated. Then, betweenness centrality would be the number of times a node is on the shortest path between two other nodes.

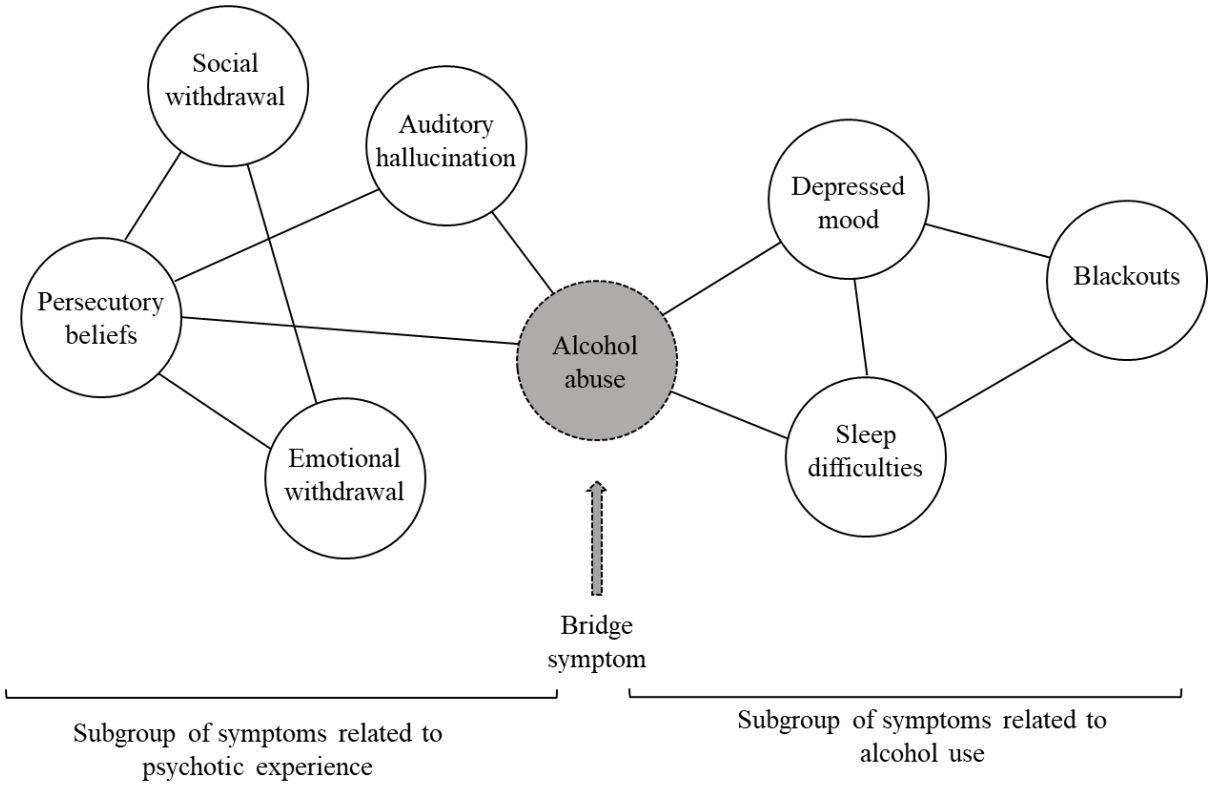
At a theoretical level, centrality metrics have clinical implications. If we can identify a node with high importance in the network propagation, targeting the symptom could prevent the spreading and, therefore, weakening 'the problem'. For this reason, centrality has been suggested as "identifier of therapeutic targets". That is, high scores in centrality would allow us to identify potential variables in the psychological network that may require early intervention (Borsboom, 2017; McNally, 2016). In fact, network literature has suggested that the strength centrality could be especially important in psychology, since it reflects the probability that the activation of one node is followed by the activation of other elements in the network (McNally, 2016). However, as we will discuss in next chapter, the current evidence is scarce and inconsistent, suggesting that the proposed clinical implications are premature and more research is needed on this matter (Bringmann et al., 2019; Fried et al., 2018; Haslbeck & Waldorp, 2018).

3.3.1.4. Comorbidity and bridge symptoms

As mentioned in the first part of the chapter, the rate of occurrence of symptoms of two or more diagnoses in the same individual is very high and traditional classifications systems have not been able to properly address this phenomenon. Comorbidity has usually been explained by the presence or covariance of two latent variables (i.e., two disorders). Alternatively, from the network perspective, comorbidity is expected when two subsets of networks (i.e., two subgroups of elements) are connected by the same symptoms. Remember the example of C., who has beliefs about others trying to hurt her. We mentioned before that due to her sense of extreme threat, C. starts having an emotional withdrawal, isolate, and hear voices commenting and criticizing her. Imagine that C. has begun to drink alcohol as an attempt to avoid hearing the voices and the distress associated with the threatening belief, which is also fuelling a more disrupted sleep pattern and a

depressed mood. Similarly, she has also started to feel blackouts, that is, the disruption of her memory for events during drinking episodes (see Figure 4).

Figure 4.
Comorbidity phenomena explained from the network perspective



Note. Following the example of C., the grey node depicts the bridge symptom (i.e., alcohol abuse) that acts as a link between the two subsets of symptoms.

In this case, comorbidity could be understood as two subgroups of symptoms (e.g., a subnetwork of symptoms related to delusional experience and a subnetwork of symptoms related to alcohol use) connected by certain symptoms (e.g., alcohol intake). This latter symptom would serve as a link between the two subnetworks, called bridge symptoms.

In short, the activation of the bridge symptom, may activate the symptoms of another subgroup of symptoms but not because there is an underlying connection between two essential or latent entities (i.e., schizophrenia and substance use disorder in this case). As stated by Borsboom (2017), although symptom relationships may be more active

within the symptom set of a particular syndrome/disorder, they need not be confined to any DSM diagnosis. Finally, as proposed by the network theory, the identification of bridging symptoms might help identifying potential target to prevent the activation of a second subgroup of symptoms, and thus, prevent the development of comorbidities (Cramer et al., 2010).

3.2.1.5. Types of networks

Although the objective of this thesis is not methodological (a detailed methodological description are very well-documented somewhere else, Bringmann et al., 2013; Epskamp, Borsboom, et al., 2018; Epskamp, Waldorp, et al., 2018) the following section briefly compiles the main types of networks that can be estimated as well as their main characteristics. We consider this information is of vital importance since it determines the type of conclusions that can be drawn after applying Network Analysis (e.g., depending on the types of data and the estimated networks that are obtained). Table 1 shows a summary of all the most important types of existing networks and packages used in psychology so far.

Table 1.
Summary of existing types of psychological network

Directionality	**Type of data**	**Type of network**	**Edge description**	**R packages**
A) Undirected networks (magnitude)	Cross-sectional	*Association*	Correlations.	*qgraph*
	Cross-sectional	*Concentration*	Partial correlations.	*qgraph, EstimateIsing*
	Cross-sectional	*Regularized partial correlation network*	Regularized partial correlations (after taking into account the rest of the variables in the network).	*qgraph, IsingFit*
	Time-series (N=1, N>1)	*Contemporaneous*	Regularized partial correlations between variables in the same measurement occasion (after taking temporal effects into account). *Can be directed or undirected.	*GraphicalVAR* (GVAR), *StructuralVAR* (SVAR), *multilevelVAR* (mlVAR)
	Time-series (N>1)	*Between-subjects*	Regularized partial correlations (after taking into account the rest of variables in the network) between means of different persons during a specific period of time (i.e., one-week assessment).	*GraphicalVAR* (GVAR), *StructuralVAR* (SVAR), *multilevelVAR* (mlVAR)
B) Directed networks (magnitude, directionality)	Cross-sectional	*Relative importance network*	The importance of one symptom in predicting another (after controlling for the effect of other symptoms on the network).	*PcAlg, relaimpo*
	Cross-sectional	*Bayesian network*	Probabilistic prediction from one node to another.	*PcAlg*
	Time-series (N=1, N>1)	Temporal network	Regularized partial correlation (after controlling for all other variables at the previous measurement occasion) between different measurement occasions.	*GraphicalVAR* (GVAR), *StructuralVAR* (SVAR), *multilevelVAR* (mlVAR)

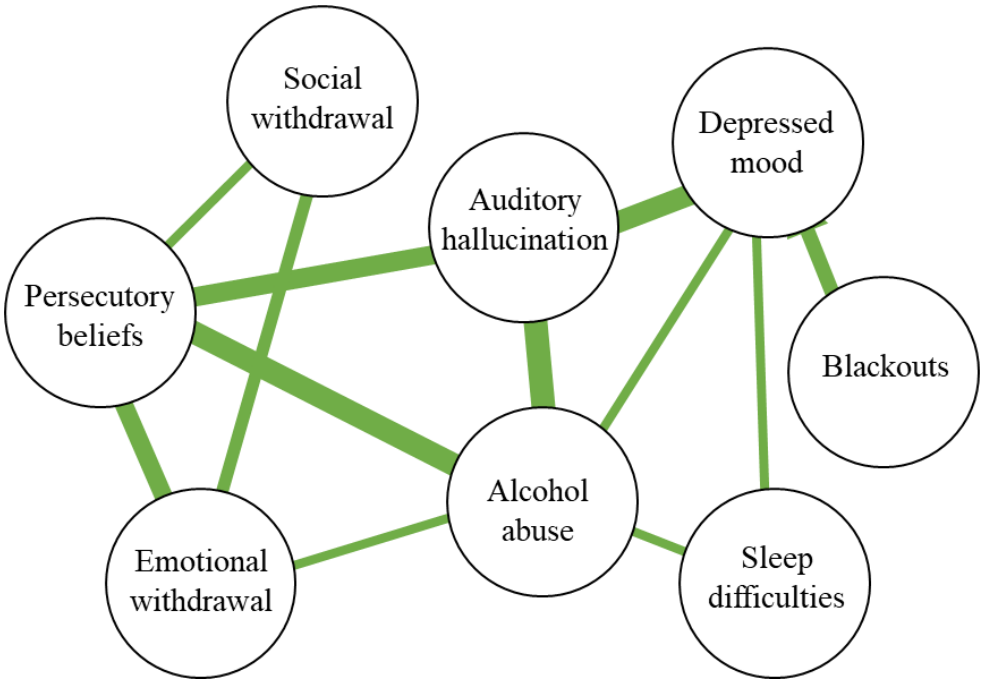
Note. Cross-sectional= a dataset composed of multiple subjects measured at a single moment in time; N=1= a dataset composed of a single case measured at several time points; N>1 a dataset composed of multiple subjects measured several time points.

A) Non-directed networks

These are network models that take into account the magnitude of the associations (i.e., degree of correlation) and the type of correlation (i.e., positive or negative), between the nodes constituting the network, but not their directionality (the nodes are connected by edges without an arrowhead). Undirected models are often based on Gaussian Graphical Models (GGM), which estimated edges as partial correlation coefficients, that is, the associations between variables after controlling for other nodes in the network (Jordan et al., 2020). In these types of networks, the thickness of the edge (i.e., thinner or thicker lines) is used to indicate the magnitude of the association between nodes. When the association between the variables is equal to zero, there is no connection between those nodes (Fried, 2017, personal communication). As shown in Table 1, we can estimate different types of undirected networks.

Association network. This is the simplest type of network, where nodes represent the variables under study (i.e., elements) and edges represent a correlation between them. Thus, the thickness of each edge indicates the strength of the correlation between pairs of nodes (see Figure 5). This type of network is very useful to see initially what symptoms are more or less associated with others. By using algorithms included in *qgraph* R package (Epskamp et al., 2019), such as the *Fruchterman-Reingold* algorithm, the researcher can place the network elements with stronger correlations in the centre of the plot and those with weaker correlations in the periphery (Jones et al., 2018). The central or peripheral placement according to the strength of the correlations gives an idea of the importance of each symptom in the network.

Figure 5.
Hypothetical association network of the C. case example



Note. Nodes represent the elements, and edges represent correlation between nodes. Green edges depict positive correlation and the thickness of each edge indicates the strength of the correlation between pairs of nodes.

Partial correlation and Regularized Partial correlation networks. A partial correlation network (also known as *concentration network*) provides information on the relationship between each pair of symptoms, after controlling for the remaining symptoms in the network. By controlling for all other symptoms, this network reveals what is known as *conditional independence* relationships (i.e., or how two symptoms are related after controlling for the remaining). Then, the coefficients represented by the edges range from -1 to 1 and the absence of a link between two variables would mean that they are independent conditional on the presence of other nodes (Fried, 2017, personal communication).

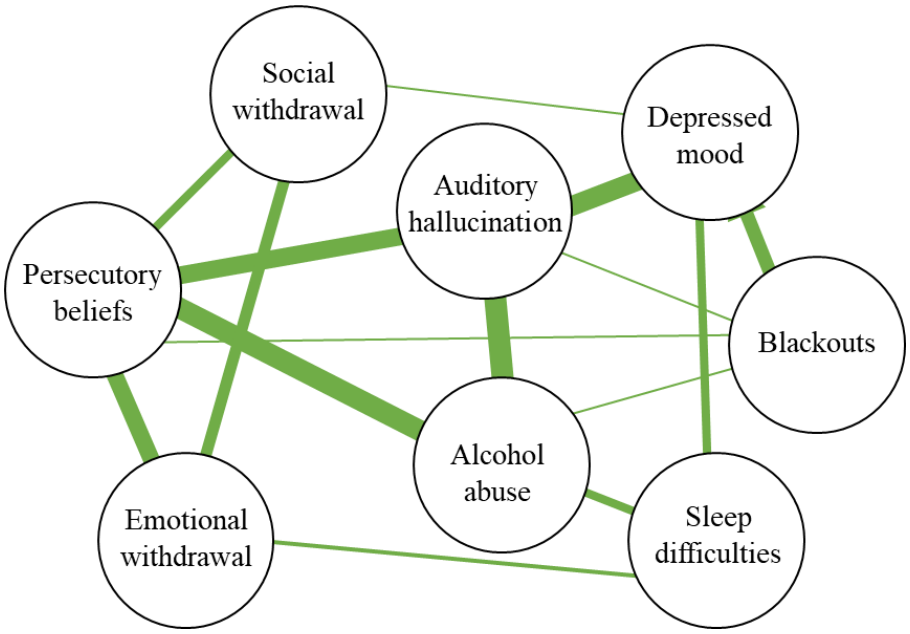
However, when estimating this type of network, very small partial correlations can arise. These would be relationships between symptoms that are actually produced by

third variables and can represent possible false positives. In order to avoid these spurious connections and get a more parsimonious network, Epskamp, Borsboom et al., (2018) proposed the *regularization* technique. For example, the researcher can find in *qgraph* R package the called *'Least Absolute Shrinkage and Selection Operator'* (LASSO) as a regularization procedure. LASSO uses a fitting parameter (known as the *Extended Bayesian Information Criterion* or EBIC) that allow the researcher to control the degree of regularization. The application of the regularization technique returns a network model called in the network literature *Regularized Partial correlation network* (or *sparse network)*, which is much more interpretable (Epskamp & Fried, 2015). Hence, regularized partial correlations networks are necessary to reveal false alarms (see Figure 6) and it is the most used type of network in psychopathology to explain the covariation structure of the data (Epskamp, Borsboom, et al., 2018).

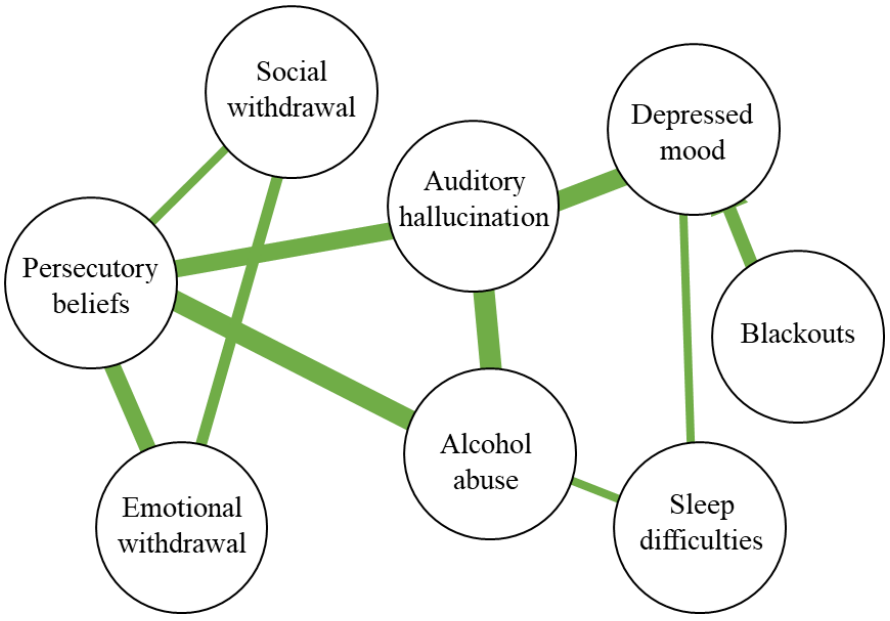
Contemporaneous network. This network can be estimated from temporally ordered data (i.e., time-series) and it is defined as a within-subject network model of effects between variables in the same measurement occasion, after taking temporal effects into account (Epskamp, Waldrop, et al., 2018). This type of network provides information about how variables covariate at the same temporal moment. Contemporaneous network can also be directed, but it is included in the undirected network classification since they are the most prevalent type in the literature (a visual example is provided in Chapter 5).

Figure 6.
Hypothetical Partial correlation network (upper panel) and Regularized Partial correlation network (lower panel) of the C. case example

Partial correlation network



Regularized Partial correlation network



Note. Edges represent partial correlation between nodes, after controlling for the remaining nodes. Observe the removal of some spurious association in the Regularized Partial correlation (lower) when comparing to the Partial correlation network (upper). For instance, after the regularization, spurious connections between emotional withdrawal and sleep difficulties have been removed.

Between-subject network. Alike the contemporaneous, between-subject networks are estimated from time-series data. These network model explain the (co)variation between stationary means of different persons (Epskamp, Waldorp, et al., 2018). That is, it provides information on how, on average, all the variables are related in an average time, established by the design of the time-series study. For example, if information is collected on different subjects during a week, the between-subject network shows the average covariation of all variables during that week (a visual example is provided in Chapter 5).

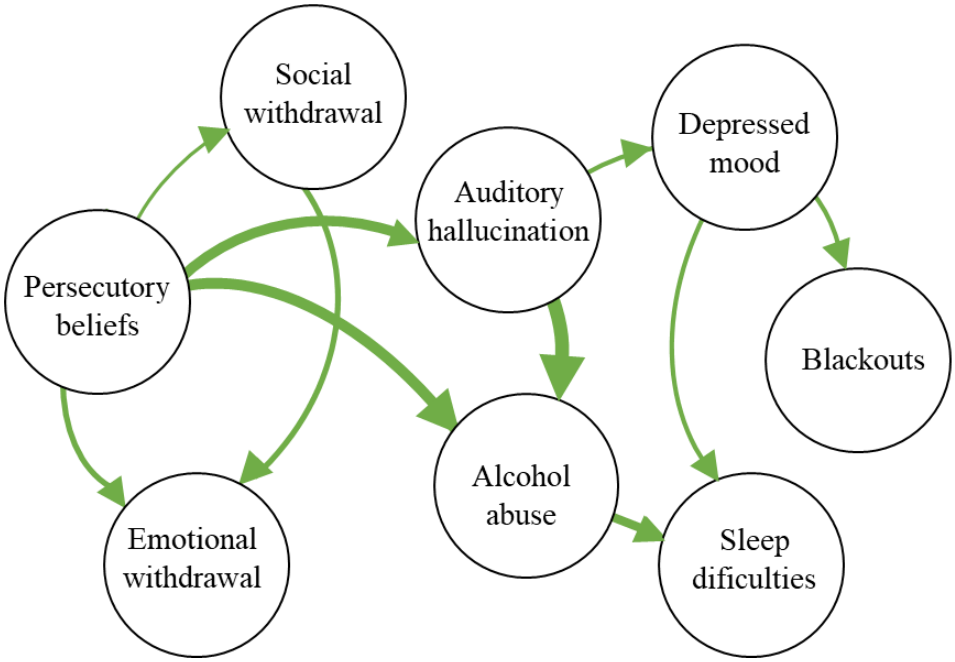
B) Directed networks

Directed networks take into account both, the magnitude and the directionality of the relationships between nodes (see table 1). Directed networks are relevant because, as it has been suggested (McNally, 2016), they represent an approach to potential causal relationship between symptoms, by suggesting that one symptom precedes another. These predictive relationships are represented in the network by an arrowhead link, and we can identify several types:

Relative importance networks. Relative importance indicates the proportional contribution of each node as a predictor in the network structure. In this type of network, edges are represented by arrows that start from the predictive symptom to the symptom that is predicted (see Figure 7). Each edge reflects the importance of symptom A in predicting B, taking into account both the effect of A on B and of B on A, after controlling for the effect of the other symptoms in the network. In this case, not only the effect of third symptoms is controlled as in the partial correlation networks, but also the strength and direction of the prediction is described. The metrics ranges from 0 to 1, quantifying the amount of explained variance assignable to each predictor (node). Relative

importance networks are considered as a step beyond directed networks as they not only provide the strength but also the direction of the prediction (Heeren & McNally, 2016).

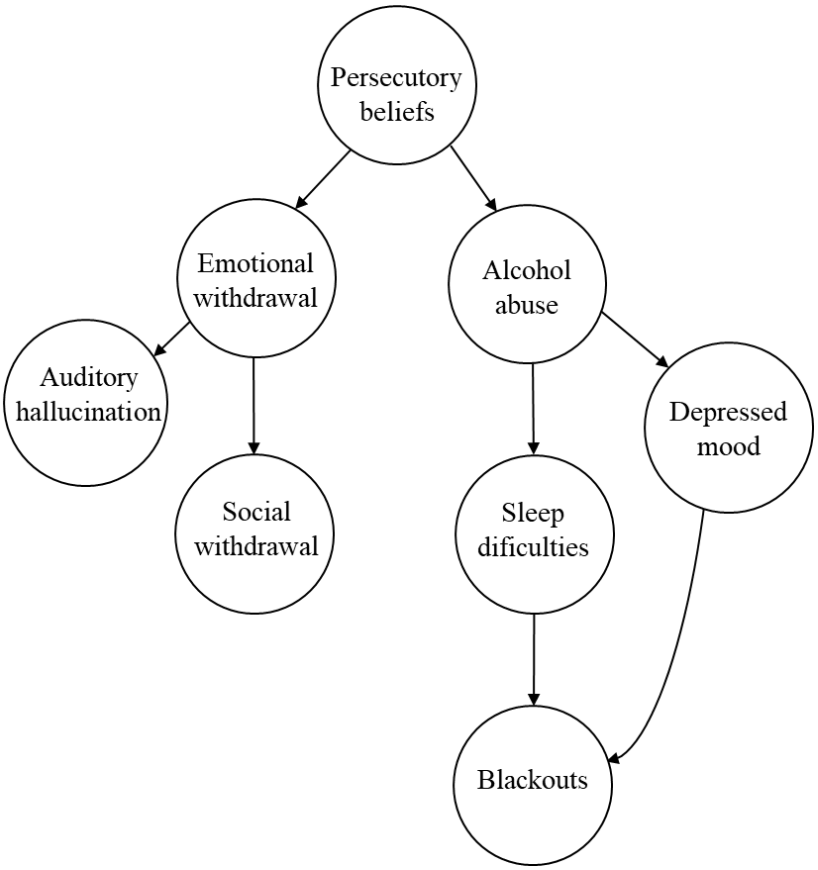
Figure 7.
Hypothetical Relative importance network of the C. case example



Bayesian networks. These networks are also directional but acyclical. That is, they are directional because each edge points to the direction of prediction and possible causality, but they are acyclical because activation from one node to another does not produce the flow activation of the rest of the network (see Figure 8). This network is also known in network literature as Directed Acyclic Graph (DAG), or a directed network in which one node doesn't point to itself (Epskamp, Waldorp, et al., 2018). This type of network can be built through an iterative process (of repetition) in which edges are added, eliminated and reverted until a specific setting value is found and return a causal structure of the relations between nodes base on their probabilistic dependencies (Heeren et al., 2020). The advantage of this procedure is that can we know the importance of edge in the network, which is very useful for creating models from which to generate hypotheses of the potential causal relationships between symptoms. However, the result is still a

probabilistic prediction of one symptom over the appearance of another, but without being able to assure causality (McNally, Heeren, et al., 2017; McNally, Mair, et al., 2017; McNally, 2016).

Figure 8.
Hypothetical Directed Acyclic Graph of the C. case example



Temporal network. The last type of directed network can be estimated from longitudinal data (e.g., time-series data). A temporal network is a within-subject network model of effects between different measurement occasions, showing temporal prediction or potential causal pathways (Epskamp, Waldorp, et al., 2018). It is estimated by combining a lagged variable y *t-1* (i.e., at previous time) and current variable y *t* into a single node, connected with directed edges which are weighted according to the regression parameters contained. Thus, an edge in the temporal network indicates that a node predicts another node (or itself in the common case of self-loops) at the next

measurement occasion, after controlling for all other variables at the previous measurement occasion. Temporal networks may thus highlight potential causal pathways (a visual example is provided in Chapter 5).

3.4. Conclusions

In a context of current crisis of the traditional classification system in psychopathology, network analysis has emerged as a promising alternative. The conceptualisation of mental problems proposed by the network theory can be conceptually linked to classic functional analysis of behaviour according to which the essence of the disorders are the problems or symptoms themselves (Virués-Ortega & Haynes, 2003), but not an underlying entity. In addition, it is assumed that the symptoms are activators of other symptoms and relationships between symptoms can be revealed by computational procedures. The emergence of the theory of network analysis has meant, at a theoretical level, a new and radical understanding of the nature of psychological problems, thinking of mental problems as something fluid and offering alternative explanations for heterogeneity within the diagnoses. This novel vision has promoted the development of a wide range of methodological resources that allow the uncovering and visualization of association patterns (McNally et al., 2017). However, this research perspective is still in its infancy and several questions remain unanswered yet. What is the viability of the application of network analysis to the empirically study mental disorders? Does the empirical application of the theory show real clinical implications?

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CHAPTER 4. THE STUDY OF PSYCHOPATHOLOGY FROM THE NETWORK ANALYSIS PERSPECTIVE: A SYSTEMATIC REVIEW[7]

As described in Chapter 3, in recent years, the alternative conceptualization of psychopathological phenomena from the Network Analysis approach (NA) has been gaining popularity. Network perspective can provide some useful conceptual and analytical tools to describe psychopathology and to explore issues related to the network structure of psychological problems. Consequently, a big amount of empirical research has been mounting in this area during the last years. Yet, there is no study that systematically review the empirical NA evidence so far, in order to make sense of these growing amount of scientific production. Systematic reviews are a good first step to link theory with evidence, untangle scientific findings about the applications of NA in psychopathology, synthesize conclusions and implications, and thus explain their significance for theory and future research (Siddaway et al., 2019).

This chapter contains a study, whose aim was to provide a systematic review of empirical research (i.e., using clinical or general population data) applying NA to study psychopathology. Given the growing number of studies in the field, it seems timely to provide a general overview of the potential strengths and limitations of this approach in the clinical psychology field. The review aimed to summarise the network studies on psychopathology in terms of their main characteristics (i.e. sample type and characteristics, instruments used to assess psychological variables or nodes, type of network estimated, robustness of the analysis) and, sharing of software codes and/or

[7] Part of the information contained in this chapter corresponds to the published article: Contreras, A., Nieto, I., Valiente, C., Espinosa, R., & Vazquez, C. (2019). The study of psychopathology from the network analysis perspective: a systematic review. *Psychotherapy and psychosomatics*, *88*(2), 71-83.

databases were also coded (given the current debate of replicability in psychology, Open Science Collaboration, 2015).

4.1. Method

4.1.1. Search strategy

Following PRISMA guidelines (Moher et al., 2009), a systematic literature search was carried out using PubMed and PsycINFO databases. Specific keywords are fully described in Figure 1. Eating disorders were excluded since there has been a recent overview of NA studies in this field (Smith et al., 2018). The search was restricted to peer-reviewed studies published in English.

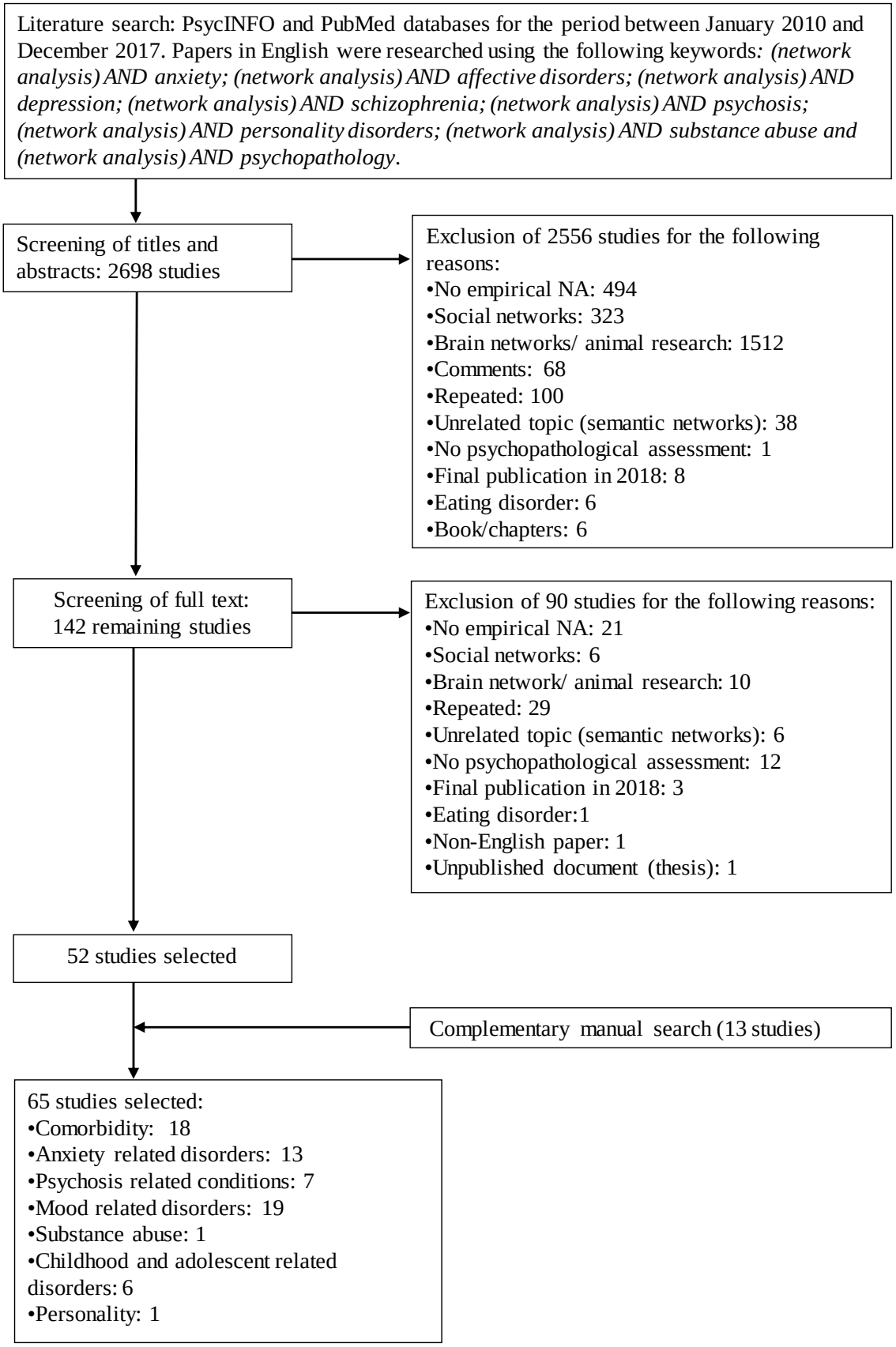
4.1.2. Study selection process

The selection process was carried out by two of the authors (AC and IN), applying the following eligibility criteria: a) empirical studies; b) paper version published from January 2010 to December 2017); and c) inclusion of measures of psychopathological symptoms (in general or clinical population). Network studies that did not focus on clinical symptoms were excluded (e.g. well-being). In addition, the bibliography of relevant papers was manually revised in order to complete the search (see Figure 1).

4.1.3. Data extraction

For the studies included, the following information was independently collected by AC and IN: a) sample characteristics; b) network elements and instruments used to assess psychopathology; c) type of data (cross-sectional or longitudinal data); d) network analysis information provided (type of network estimated, centrality metrics and robustness analyses); d) availability of software code or syntax (e.g. R script) and/or data

Figure 1.
Process of literature search strategy of Network Analysis (NA) studies in psychopathology



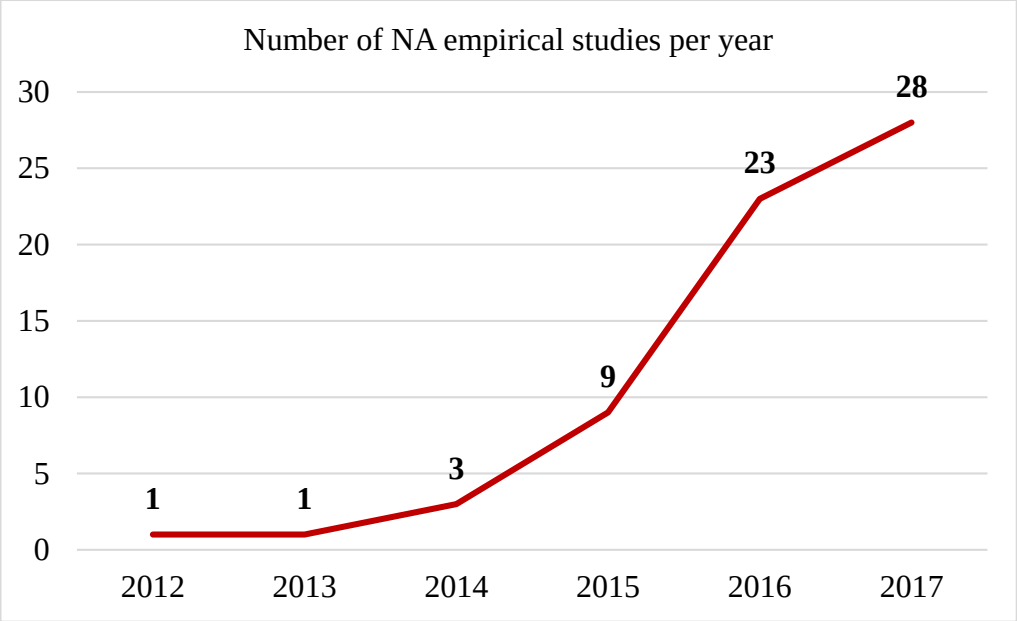
(i.e. correlation matrix or datasets) in the published papers. A more detailed description about the data extraction is provided in Appendix 1).

4.2. Results

4.2.1. Study selection and characteristics

A total of 52 studies met the criteria and were selected for the current revision. Moreover, 13 relevant articles were added from manual complementary cross-referencing search. As a result, a total of 65 studies were included. Their characteristics are described in the Appendix section (Tables A1-A3). The majority of the literature reviewed has been published recently (see Figure 2).

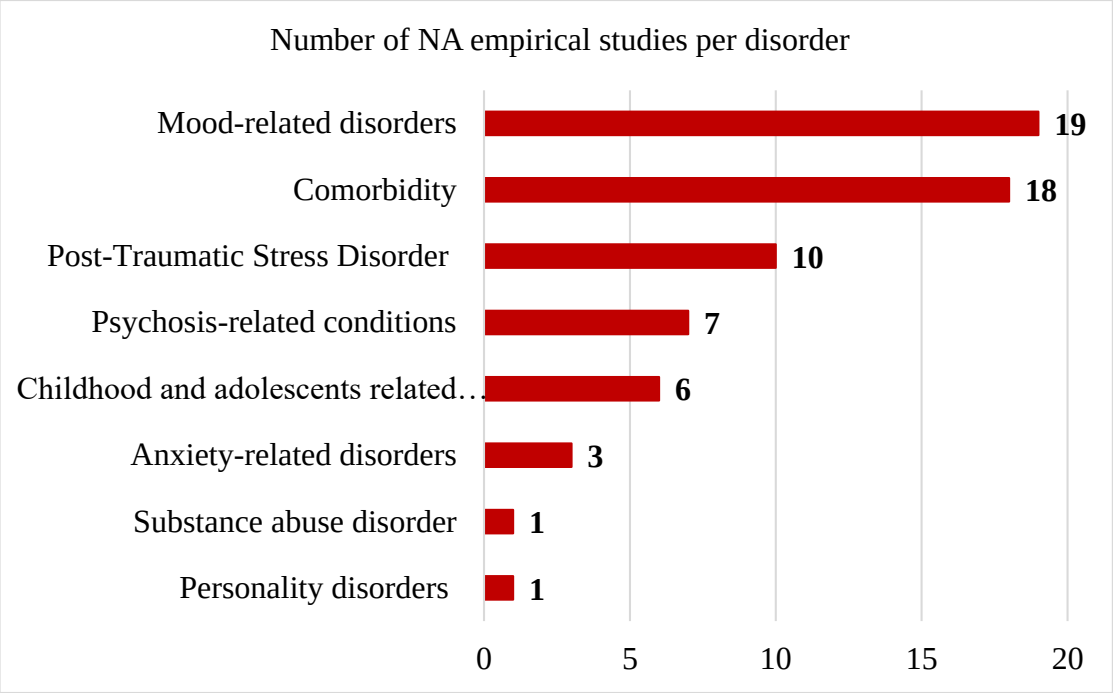
Figure 2.
Number of NA empirical studies published per year, from 2010 to 2017



Overall, most of the studies used adult samples; only 8 studied NA in a childhood and/or adolescent population. In terms of the type of data, 46 studies used cross-sectional data, while only 19 studies used longitudinal data (7 of them using Experience Sampling Methodology, ESM). Regarding the type of NA, 23 studies constructed a Directed Network while the remaining used association networks or Regularised Partial

Correlation Networks, and 55 studies calculated centrality indexes. Only 21 studies assessed robustness or quality of estimated parameters (stability and/or accuracy of results) but tools to carry out these procedures have been available recently (Epskamp, Borsboom, et al., 2018). Finally, a low number of studies shared their data in the published paper (i.e. 18 shared correlation matrixes and only 6 shared both data and scripts). An overview of the number of empirical papers from 2010 to 2017 studying specific disorder is shown in Figure 3.

Figure 3.
Number of NA research studying specific mental health disorder, from 2010 to 2017



4.2.2. Results of NA for specific mental disorders

4.2.2.1. Post-traumatic stress Disorder (PTSD)

The literature identified 10 NA on PTSD (detailed data is provided in Appendix, Table A1). Across studies, results disclosed strong associations among avoidance symptoms (McNally et al., 2015; Sullivan et al., 2016) and between hypervigilance and startle response (Armour et al., 2017; Birkeland & Heir, 2017; Bryant et al., 2017;

McNally et al., 2015; Spiller et al., 2017). Particularly, McNally et al. (McNally et al., 2017) found that physiological responses to reminders of trauma predicted symptoms such as ‘being upset’ ‘flashbacks’ and ‘traumatic dreams’, in a sample of adults who reported histories of childhood sexual abuse. According to these authors, physiological reactivity, which is associated in NA with being female (Birkeland & Heir, 2017), may play a potential causal role in the activation of the PTSD network. Moreover, NA has also found some unexpected interconnections between anger, sleep and concentration problems, suggesting that difficulties in the regulation of emotions and attention may arise from sleep-related problems in trauma-related problems (McNally et al., 2015). Likewise, Glück et al. showed that ’trait anger’, ‘rumination anger’ and ‘emotional abuse’ may play an important role in persons who suffered childhood trauma, highlighting the possible clinical implications of including anger in trauma interventions (Glück et al., 2017).

Interestingly, one study showed that connections may vary in different age groups, that is, symptoms such as ‘amnesia’ and ‘numbness of negative affect’ were more strongly associated in children than in the adolescent network (Russell et al., 2017). In addition, in the only longitudinal study that compared networks across time, Bryant et al. (2017) found that, after one year, re-experiencing symptoms were more strongly connected than during the acute phase and that physiological reactivity was strongly associated with startle response which, in turn, was associated with hypervigilance. This strong "reactivity association", according to these authors, could be conceptualised as a circuit of fear that becomes increasingly more sensitive to long-term threats.

NA has also shown PTSD symptom connections with other relevant elements and clinical problems, such as depression symptoms (Armour et al., 2017) or ‘low social

support after the exposure', which has been found to be connected with sleep disturbances in individuals with PTSD (Birkeland & Heir, 2017).

Finally, NA has revealed mixed results regarding central symptoms in PTSD. For instance, 'physiological reactivity' and 'flashbacks' have been found to be central symptoms in veteran and earthquake survivors (Armour et al., 2017; McNally et al., 2015). However, central symptoms in terrorist attack witnesses are 'emotional numbness' and 'concentration difficulties' (Birkeland & Heir, 2017) whereas, intrusive thoughts and anger were highly central in a sample of witnesses from a shooting event (Sullivan et al., 2016). Again, after a year from a traumatic injury, 're-experiencing' persisted as a core symptom (Bryant et al., 2017) whereas 'emotional cue reactivity' is the most important node in a sample of refugees (Spiller et al., 2017). Lastly, Jayawickreme et al. (Jayawickreme et al., 2017) found that war social problems were more important than the traumatic event in a sample of war survivors.

In sum, PTSD results provide relevant information about elements interactions, about variables that may affect symptoms connections and have allowed the finding of some unexpected associations among symptoms (McNally et al., 2015) that deserve to be further explored. Findings also illustrates potential pathways between traumatic events and symptoms (Jayawickreme et al., 2017). However, the heterogeneity of results, which could be due to differences in the type of trauma experiences or the size and type of samples included in the studies (e.g. nonclinical samples, different trauma exposure), hinders the integration of results and should impose caution about the generalizability of the results.

4.2.2.2. Anxiety-related disorders

Two studies focussed on Social Anxiety (SA) symptoms interaction and with other non-symptoms variables (see Appendix, Table A1). Tsuruta et al. (2017) innovatively examined the association among SA symptoms and cognitive functioning

and found that orienting attention to non-emotional material was linked to fear of social situations, which impacted the social experience by triggering avoidance social behaviours. NA has also been used to study less known anxiety syndromes such as olfactory reference syndrome. Tsuruta et al. (2017) revealed that SA may play a key role in the onset of fear to bodily odours.

Finally, one study explored anxiety related to death in patients suffering from cancer (Vehling et al., 2017), disclosing that most central concern related to death is 'running out of time'. The authors also identified nodes that act as a bridge between two death-related anxiety clusters (i.e. one related to practical fears regarding the process of dying, and other to existential concerns). These findings suggest that psychology interventions should be aimed at targeting those central symptoms in order to alleviate anxiety related to death.

4.2.2.3. Mood-related disorders

The literature search revealed a total of 19 studies on mood symptom networks: 16 on depression, 1 on suicide attempters, 1 on bipolar disorder and 1 on alexithymia (see Appendix, Table A1).

Bringmann and colleagues (2013) found that positive emotions were negatively associated with negative mood variables, that the presence of one symptom predicted the occurrence of the same symptom in the future and that depressed females showed stronger associations than controls (Bringmann et al., 2013). Also, using ESM methods, Pe et al. (2015) have shown that, compared to healthy controls, participants with major depression had higher networks density of negative emotions but there were no differences in regard to the density of positive emotions. These results suggest that, in depression, previous negative emotions have a greater influence on the next negative emotional states which overall make negative emotions more resistant to change in this disorder (Pe et al., 2015).

NA has also been used to study the interaction of depressive symptoms over time. It was found that patients with persistent depression showed stronger associations between depressive symptoms than those in remission, supporting the idea that the strength of connections is associated to vulnerability (Borkulo et al., 2015). Likewise, Madhoo and Levine (2016) reported that the connectivity among symptoms significantly diminished after an intervention. Several studies have used Experience Sampling Methods (ESM) to collect time-series data in depressed participants, although, the outcomes assessed are heterogeneous. For example, Dejonckheere et al. (Dejonckheere et al., 2017) found that the perception of social pressure to feel good instigated increases in slowed-down symptoms of depression (e.g. hypersomnia, motor retardation).

More interesting, from a comprehensive view of psychopathology, is that several studies added non-symptom variables in the network (see Appendix, Table A1). Hoorelbeke et al. (2016) studied the potential role of risk and protective factors and found that resilience was the principal hub in the network and it could be a key factor in the remission of depression. Cramer et al. (2012) found specific connections between depressive symptoms (e.g. 'feelings of worthlessness') and stressful life events such as 'ending of a romantic relationship'.. In a sample of women in their third trimester of pregnancy, Santos et al. (2017) assessed the relationship between stress and reproductive biomarkers (e.g. cortisol) and depression. Interestingly, all biomarkers showed very small associations with symptoms of depression which, according to the authors, may cast some doubts about their role as causal candidates of symptoms of depression (Santos et al., 2017).

Three studies have also explored depression and personal losses. Robinaugh et al. (2014) and Fried et al. (2016) found associations between loss of a spouse and depression symptoms. Specifically, 'loneliness' was found to activate other symptoms through its

association with loss (Fried et al., 2015) and to connect to risk factors such as a lack of instrumental social support (Robinaugh et al., 2014). Maccallum et al. (2017) analysed the networks of depressive and complicated grief symptoms regarding two different types of losses (i.e. death of a spouse or a parent) in general population. The results showed that both types of losses produced very similar networks being the link between yearning and emotional pain the strongest connection. Remarkably, avoidance had a peripheral situation in both networks.

Regarding centrality, Fried et al. (2016) found that DSM-5 criteria symptoms (e.g. sad mood) were not more central than non-DSM symptoms (e.g. anxiety). In clinical, as well as general populations, some authors have found that 'concentration problems' and 'feeling sad' were central symptoms (Boschloo, Borkulo, et al., 2016; Borkulo et al., 2015). These results are in line with the findings of the study by McWilliams et al. (2017) in participants suffering from chronic pain, showing that difficulty in concentrating, loss of interest, depressed mood and fatigue were the most important symptoms.

De Beurs et al. (2017) analysed suicide ideation in a sample of patients following a suicide attempt, revealing that 'desire for an active attempt' was the most central symptom of the network for the entire sample. One study focussed on bipolar disorder (Koenders et al., 2015) finding that 'loss of energy' was highly central in bipolar patients with different levels of severity and patterns of symptoms . Yet, there were specific variations, in terms of centrality, in different subtypes of patients. The highest degree of centrality was 'increased speech' and 'loss of interest' in the minimally impaired group, 'decreased self-esteem' and 'slowness' in the depressed group and 'restlessness' and 'suicidality' in the cycling group (Koenders et al., 2015). Watters et al. (2016) investigated the basic components of alexithymia and their mutual interaction. They found that the strongest associations were between difficulties in identifying and

describing feelings, and that both components were also the most central ones in alexithymia (Watters et al., 2016).

Some preliminary findings on the connectedness of symptoms in depression are worth mentioning. For instance, the finding that depressed individuals show a strong connection of symptoms of depression (Santos et al., 2017) or emotions (Pe et al., 2015) could be relevant to explore the organisation of symptoms in depression.

4.2.2.4. Psychosis-related conditions

The literature search revealed a total of 7 studies that focussed on *psychosis-related symptoms* (see Appendix, Table A1). A "transdiagnostic network approach" was used to study psychosis and to analyse multiple domains of psychopathology (Rooijen et al., 2017; Wigman et al., 2016). Associations were found within each psychotic domain which were especially strong for negative symptoms (Wigman et al., 2017) and within different domains. For example, Wigman et al. (2017) there are interconnections between some positive symptoms (e.g. 'being persecuted') and anxiety items (e.g. 'worried about panic'), negative symptoms (e.g. 'lack of energy'), and depression items (e.g. 'feeling tense'). Their NA topology discloses the possibility that, once the delusion is activated, it triggers anhedonia symptoms, which in turn activates depressive symptoms or vice versa.

Using ESM data in a single participant through different disorder course moments, Bak et al. (2016) found that when the patient has not relapsed yet, 'feeling down' and 'paranoia' fuelled each other (i.e. the lower the mood, the higher the paranoia in the next moment). However, this association was weaker in the full relapse group, while paranoia was directly connected to hearing voices (Bak et al., 2016). NA can also be used to explore changes in patters of symptoms before and after interventions. Levine and Leucht (2016), study negative symptoms in 3 different moments (i.e. before, after and during an intervention). They identified a "negative symptoms severity system" in which symptoms severity are grouped in four clusters (e.g. 'affect', 'poor responsiveness', 'lack of interest'

and ‘apathy-inattentiveness’). They also highlighted two central symptoms (‘decreases spontaneous movement’ and ‘speech’) as potential future treatment targets, due to the fact they remained central after and during intervention, respectively. Similarly, Esfahlani et al. (2017) compared the symptoms network at baseline and at 18 months’ follow-up of a trial of antipsychotics in individuals with psychosis. Results showed that the treatment responsive group had more densely connected symptoms after the treatment, while global connectivity of the treatment resistant group is not affected by the treatment (Esfahlani et al., 2017).

Two of the studies reviewed in this section added adversity variables in the networks (Isvoranu et al., 2016; Isvoranu et al., 2017). In a sample of general population, developmental trauma was found to be connected to psychotic expression and somatisation (Isvoranu et al., 2016). Further, the authors found that drug use might play a mediating role between trauma experienced and the onset of psychotic symptoms (Isvoranu et al., 2016). Moreover, in a sample of people diagnosed with a psychotic disorder, childhood trauma was associated with positive and negative symptoms only through some general psychopathology symptoms, such as anxiety, suggesting that there may be different pathways between trauma and psychosis (Isvoranu et al., 2017).

Both cross-sectional (van Rooijen et al., 2017; Wigman et al., 2017) and longitudinal studies (Bak et al., 2016; Levine & Leucht, 2016) seem to be pointing to the significant role that negative affect plays in paranoia, as well as, the possibility to map transdiagnostic symptoms of psychopathology (Wigman et al., 2017).

4.2.2.5. Personality disorders

A study on Borderline Personality Disorder (BPD) was identified in the present literature review (see Appendix, Table A1). Richetin et. Al (2017) compared the relation of nine characteristics of BPD in two different samples (university students and clinical population). Although ‘affective instability’, ‘identity disturbances’ and ‘effort to avoid

abandonment' appeared to have a central role in both samples, some edges were unique for the clinical sample (i.e. suicidal behaviour and unstable relationship), what highlights particular connections between symptoms in severe manifestations of BPD (Richetin et al., 2017).

4.2.2.6. Substance abuse disorders

The literature search revealed only 1 NA study in a sample of people who reported 'having used substances more than six times in their lifetime' (see Appendix, Table A1). Rhemtulla et al. (2016) found that 'using a substance more than planned' was strongly connected to 'tolerance'. Besides, a common association between 'being unable to stop' and 'hazardous use' was found when comparing symptoms connections across different substances. However, some correlations changed across substance networks. For example, hazardous use and legal consequences was strongly connected when using sedatives, but it was not for the opioids, cocaine or hallucinogens networks. Regarding centrality, 'substance used more than planned' was the most central symptom in the general substance use network, as well as in the cocaine, cannabis and stimulants networks, which may indicate that losing control over a drug may precipitate a host of other types of abuse and dependence symptoms (Rhemtulla et al., 2016).

4.4.3. Results from NA in psychopathology and/or comorbidity

The literature search revealed a total of 18 studies on general psychopathology and/or comorbidity (see Appendix, Table A2).

4.4.3.1. Comorbidity with depression

Out of the 9 studies that focused on comorbidity with depressive symptoms, some have analysed comorbid Major Depressive Disorder (MDD) and Generalized Anxiety Disorder (GAD)(Beard et al., 2016; Bekhuis et al., 2016; Curtiss & Klemanski, 2016; Fisher et al., 2017). All of them found a highly connected network in which no symptom remained isolated from the others. Regarding centrality, studies have reached different

conclusions. While one study found that ‘worry’ and ‘sad mood’, the main diagnostic criteria for GAD and MDD, respectively, were the most central in a cross-sectional network (Beard et al., 2016), another study found that these symptoms were the least central in a temporal network (Fisher et al., 2017). However, both studies pointed out ‘anhedonia’ and ‘guilt’ as highly central in their comorbid networks.

Two studies analysed depression and PTSD (Afzali et al., 2017; Choi et al., 2017) and found that symptoms of depression and PTSD formed separated clusters but they were also connected by bridge symptoms, such as ‘sleep disruption’ or ‘concentration difficulties’ (see Appendix, Table A2).

Regarding comorbid depression and psychosis, some authors found that symptoms formed two separated clusters, being only the symptom of paranoia more closely related to depression (Jaya et al., 2017). Using longitudinal data, Wigman et al. (2015) studied the dynamics of five mental states (i.e., cheerful, insecure, content, down and suspicious) in individuals with a diagnosis of depression, psychosis and no diagnosis. The main difference between the clinical groups was that there were many connections between positive and negative emotions in the depression group, while they formed two separate clusters in the psychosis group. The highest centrality indexes were down momentary mental states for the psychosis group and the positive momentary mental states for the healthy controls (Wigman et al., 2015).

Finally, studying comorbid depression and Obsessive-Compulsive Disorder (OCD), McNally et al. (2017) found that both clusters of symptoms were connected through ‘sadness’, but sleep and appetite symptoms were not connected to either depression or OCD. These types of results illustrate that not all symptoms play the same role when developing comorbid disorders.

4.4.3.2. Comorbidity between different conditions

The reviewed literature identified 9 NA studies that focused on comorbidity and psychopathology (see Appendix, Table A2). Two of them used temporal networks to explore the dynamics of emotions with longitudinal data. It was found that people with high neuroticism and healthy people had denser emotion networks in comparison with people low in neuroticism or depressed people, respectively. Authors tried to see the relationship between neuroticism and the centrality in the networks and their variability, but they reached different conclusions (Bringmann et al., 2016; De Vos et al., 2017), remaining unclear how this trait influences emotional changes in time.

Some studies have used NA to explore the association between symptoms classified within different diagnostic categories (Anker et al., 2017; Boschloo et al., 2015; Goekoop & Goekoop, 2014; Knefel et al., 2016). The main findings have consistently been that some symptoms cluster together (e.g. symptoms of depression) (Goekoop & Goekoop, 2014), but are also connected by bridging symptoms. For example, 'internal avoidance' and 'identity disturbance' are related in comorbid PTSD and BPD (Knefel et al., 2016), while 'drinking to cope' and 'subjective stress' could explain comorbid Alcohol Use Disorder and anxiety and depression (Anker et al., 2017). These results may be important in terms of identifying individuals at risk to develop two or more disorders and also to be used in treatment to reduce current and future comorbidity.

Finally, NA has allowed exploring the relationship between psychopathological symptoms and non-symptom variables. Using longitudinal data, it was found that environmental factors (i.e., childhood trauma, urbanicity, cannabis use and discrimination) increased symptom connectivity (Guloksuz et al., 2016) and that 'self-criticism' could play a central role in the relationship between rumination and executive control (Bernstein et al., 2017). Further, personality variables such as extroversion and openness to experience were left outside the cluster of anxiety and depression, but

extrovert personality together with symptoms such as ‘worry’, ‘perceived distress’ or ‘low energy’ were found to be the most central in the network (Pereira-Morales et al., 2017).

In sum, comorbidity is an interesting filed to be studied using NA in order to discover how symptoms considered from different disorders are related. These findings have direct implications in their use in clinical interventions, as will be further explained in the discussion section.

4.2.4. Results from NA in childhood and adolescents related disorders

The literature search revealed a total of 6 NA studies that focussed on disorders related to childhood or adolescence (see Appendix, Table A3) in which both, samples and outcomes studied were heterogeneous. Anderson et al. (Anderson et al., 2015) studied autistic traits in a sample of children with Pervasive Developmental Disorder. They found that ‘usual eye contact’ and’ facial expression directed towards others’ were the most important nodes. Moreover, they showed that anxiety was more central in males, and that social nodes were more central in low functioning children. Ruzzano et al. (2014) studied the comorbidity between autism and OCD symptoms, revealing that compulsion symptoms may be the bridge between autism and OCD (Ruzzano et al., 2014).

Two studies focused on Attention Deficit Hyperactivity Disorder (ADHD) symptoms interactions. Martel et al. (2016) found that ADHD symptoms changed across time, although symptoms such as ‘often easily distracted’ and ‘difficulty sustaining attention’ remained central over time. Likewise, Smith et al. (2017) showed an association between ADHD and Oppositional Defiant Disorder (ODD) symptoms and also that anger was the most important symptom in pre-schoolers with ODD.

Boschloo et al. (Boschloo, Schoevers, et al., 2016) examined the empirical network of 95 emotional and behavioural problems in a sample of adolescents. Findings

revealed strong connections within psychopathological domains (i.e. externalising, internalising, attention, thought and social problems), but also connections between domains. Finally, Hasmi and colleagues (2017) studied whether the dynamic of emotions in daily life differed across different levels of genetic liability and exposure to childhood trauma in a general population mixed-gender twin sample. NA confirmed that negative emotions are associated to genetic risk factors (Hasmi et al., 2017).

4.3. Discussion

In the current context of crisis of traditional classification systems within mental health, NA research has substantially increased in recent years. The systematic review revealed evidence that highlights NA as a promising psychopathology research tool to study symptom connections, although it must have clinical utility and, ultimately, being sensitive to changes in the clinical population. Also, several important cautions emerge that will be addressed.

In relation to the wide-range of psychopathological phenomena, NA has been used to study many psychological disorders (see Figure 2). The systematic review reveals that a great part of NA research has been conducted in an exploratory way of which Mood disorders, PTSD, psychosis related conditions and comorbidity phenomena are the most studied areas. Comparing results of NA within a given domain of psychopathology may be problematic as the critical mass of studies using these tools is still scarce and studies are very heterogeneous in regard to sample characteristics, types of measures, or other relevant variables. In this way, part of the problem with current research in NA is that it is too descriptive, results are rather diverse. Given the exponential growth of the literature in this field, and the heterogeneity of the results, it is expected that there will be specific systematic reviews for each psychopathology domain. Therefore, a systematic review may provide a good main entrance to this field of studies. Although NA in certain cases

provides expected results, like the fact that sadness is an almost ubiquitous central symptom in depression (Boschloo, Borkulo, et al., 2016; Madhoo & Levine, 2016; Van Borkulo et al., 2015), the results of the present review supports the idea that NA adds information that the traditional classification models do not incorporate (Boschloo, Schoevers, et al., 2016). For instance, our review shows the potential capacity of NA to identify unexpected associations between symptoms (McNally et al., 2015). In the case of depression, NA has shown that a low level of energy often emerges as a core symptom in mood-related disorders, which may predict the onset of major depression (Boschloo, Borkulo, et al., 2016), albeit it is important to consider that the centrality of symptoms may vary at different time points through the course of the problem (Madhoo & Levine, 2016). In addition, despite 'fatigue' or 'loss of energy' are not DSM symptoms, they emerged as central symptoms in depression (Boschloo, Borkulo, et al., 2016) and bipolar patients (Koenders et al., 2015). In the case of PTSD, NA studies have identified a potential causal interconnection of anger with sleep and concentration problems, pointing the possibility that these associations may affect the regulation of emotions and attention (McNally et al., 2015).

Although most of the extant NA research has focussed on symptom-to-symptom interactions, there is evidence showing that other non-symptom should be meaningfully incorporated in psychopathology networks. Our results bear out that adding non-symptom element can enhance the understanding of important aspects of psychopathology. The range of these elements included in NA vary a great deal, from attention bias in social anxiety networks (Heeren, & McNally, 2016) to biomarkers (Santos et al., 2017), resilience in depressive networks (Hoorelbeke et al., 2016), or the so-called external field variables (Fried & Cramer, 2017), like life events (Cramer et al., 2012). For instance, the study by Isvoranu et al. (2017) revealed multiple potential pathways between childhood

trauma and psychosis, either through a pathway of emotional distress or through general psychopathology. As the field of NA matures, it is desirable that it reflects the complex interactions between different components that, beyond symptoms, are involved in the aetiology and maintenance of psychopathology (Borsboom, 2017). This implies that it is not possible to attribute casual or explanatory priority to psychological or biological levels, but rather that a holistic research strategy is needed (Borsboom et al., 2018), in which all those factors that psychopathology has evidence so far should be taken into account.

NA may also make contributions to some of the classification and conceptual dilemmas. For instance, the transdiagnostic network approach use by Wigman et al. (2017) puts psychosis in a continua where the dividing lines between different disorders and sanity seem arbitrary such as it has been shown by epidemiological data (Johns & Van Os, 2010). Also, Isvoranu's studies (Isvoranu et al., 2016; Isvoranu et al., 2017) confirm that environmental factors, such as childhood trauma, are associated to network symptoms in schizophrenia. Taking together, these findings are a good example of how NA can provide attractive novel symptoms information. Nonetheless, different subtypes of disorders may offer different topological configuration of symptoms, what make very complex the generalization of results. Thus, these results should ineludibly be confirmed with additional experimental and clinical studies as well as additional research to cover the wide variety of mental disorders.

Another potential contribution of NA to the field of psychopathology is provided by centrality metrics. Finding central symptoms departs from the core idea in current diagnostic systems that symptoms are interchangeable, within a diagnostic category, to yield a diagnosis (Parnas, 2015). The aim of finding centrality indexes may be highly relevant for this enterprise as they inform of the relative importance of nodes in the

network. Central nodes are largely interconnected to other nodes and, therefore, their variations are more likely to affect the other nodes in the network. It has been proposed that identifying central nodes would be relevant not only to reveal which symptoms are more important in a psychopathological condition but also to guide clinicians on which symptoms should be given priority in interventions (Dejonckheere et al., 2017; Kivelä, et al., 2014; Madhoo & Levine, 2016; McNally, 2016; Moher et al., 2009; Vehling et al., 2017). Moreover, centrality indexes may also be helpful making predictions about recovery and prognosis. For instance, they have been suggested as indicators of prognosis for people with MDD (Boschloo, van Borkulo, et al., 2016), as well as indicators of different courses of the disorder (Borkulo et al., 2015). Thus, it could be possible that NA may have some added value to identify clinically relevant symptoms than earlier procedures used in traditional psychometrics. This is especially interesting in the study of comorbid disorders. Centrality indexes and the identification of bridge symptoms have potential applications in clinical interventions to predict and prevent the development of comorbidity, as well as to target those symptoms to reduce each condition.

However, results on centrality must be critically examined as our review reveals that central symptoms differ across studies. For example, in the field of PTSD, psychopathological responses may vary due to differences on the types of traumatic events as well as their relative impact on survivors (Birkeland & Heir, 2017; Sullivan et al., 2016). Given the disparity of results among studies, it would premature to defend that central variables found in specific studies should automatically become new interventions targets (Fried et al., 2018). Since centrality has been considered a relative metric, predictability analysis, which provides an absolute measure of interconnections in the network, has been suggested, in order to overcome these problems (Fried et al., 2018). While some authors have proposed that centrality metrics in psychopathology, such as

betweenness and closeness, seem inadequate as measures of importance to the nodes (Bringmann et al., 2019) there is still no agreement on which the best indexes of centrality in network analyses could be (Lü et al., 2016).

A central feature of the network perspective is its promising capacity to study comorbidity (see Appendix, Table A2) (Boschloo et al., 2015; Cramer, et al., 2010; Fried & Cramer, 2017). However, researchers using NA tools should be cautious when deciding what variables to include in the network, pondering on whether two nodes really represent different things or are measuring the same construct. The idea of topological overlap (i.e. combining overlapping variables into one node) has been proposed to address this issue (Fried & Cramer, 2017). Thus, future research is needed to identify and validate potential bridge symptoms and to test whether topological overlap offers an opportunity to guide decisions about what nodes should be included in the network (Fried et al., 2017).

The literature reviewed also identified several methodological issues in NA. Many authors have mentioned the use of cross-sectional data as a limitation and point out the need to carry out longitudinal studies to be able to discern the directionality of the associations in the network (Bak et al., 2016; Fried, et al., 2015; Levine, & Leucht, 2016; Robinaugh et al., 2014; Borkulo et al., 2015) as well as temporal prediction (Epskamp, Waldorp, et al., 2018). For example, in our review there are only three studies using longitudinal data of psychotic symptoms of which only one use ESM measures (see Appendix, Table A1). This situation is unfortunate as using longitudinal studies data could be extremely useful in clinical practice to understand predictions and symptoms that may play a role in the onset and maintenance of mental disorders (Bak et al., 2016; Bringmann et al., 2016; Wichers & Groot, 2016). Thus, the analysis of data measure over different time points may disclose temporal patterns of symptoms as well as it may

provide insights about the dynamics of psychopathology and how is related to intra and inter individual differences (Bringmann et al., 2013).

In relation to robustness and replicability of results, it has been recommended that NA research reports should include the assessment of the quality or accuracy of network parameters and measures (e.g. how accurate edge-weight are estimated, or how stable centrality metrics are (Epskamp, Borsboom, et al., 2018). Of note, tools to carry out these procedures have been made available recently (Epskamp, Borsboom, et al., 2018; Haslbeck & Fried, 2017) and very few studies have used them and confirmed their validity. Besides, some authors have criticized the lack of replicability of network analyses (Forbes et al., 2017), which represents an important theoretical and technical challenge, that could be enhanced if proper analytical methods are used (Borsboom, 2017; Fried et al., 2018). In addition, authors should also share information on, for example, R scripts and data matrices (Epskamp, Borsboom, et al., 2018). Our results reveal that few studies directly shared their data while only four provided R scripts (see Appendix, Table A1-A3) in the publication. Furthermore, sensitivity analysis of networks has a great technical complexity that may exceed the clinicians' methodological capacity and, therefore, we believe that future NA procedures should not only be available but also need more friendly statistical programs and more accumulation of knowledge on networks and psychopathological symptoms using larger and more varied samples.

A more important limitation of NA is that the vast majority of the published NA studies have used existing datasets (see Appendix, Table A1-A3) based on a categorical approach (e.g. studying symptoms associations patterns in studies based on disorders diagnosed with the DSM or ICD). In other words, most NA studies depend upon the limitations of previous databases and types of gathered data (e.g. instruments based on categorical approach that are used to measure the symptoms which relationship is

estimated) rather than being based on specific designs aimed at testing specific hypotheses. Furthermore, the aim of challenging current diagnostic systems (Borsboom et al., 2011) on NA based almost exclusively on the extant data gathered with those symptoms seems tautological. In fact, there is a debate, within experts in the field, on to what extent NA should be simply used as an additional analytic tool to explore constellations of elements (signs, symptoms, stressors, etc.) rather than using it as tool to criticize diagnostic systems (Bringmann & Eronen, 2018; Wichers et al., 2017). Thus, so far, NA has been using a highly limited type of information which surely restricts the utility of this approach to validate descriptive and etiological models of psychopathology. It is still soon to see if NA will be able to provide sound responses to these important issues and whether it can apprehend the complexities and nuances of the dynamics of psychopathology, which go beyond measuring symptoms (Fava et al., 2012).

In conclusion, to date, the network perspective represents a promising challenge to the usual way of thinking in the field of clinical psychology and psychopathology (Borsboom et al., 2018) and is already providing novel ways of considering the importance (i.e., centrality) of symptoms or connections between symptoms. As a clinical tool, it could be possible that NA might offer, in the future, information to the clinician in two different manners. On one hand, knowing the type of main complains that a patient may have (e.g., delusions and sadness), based on previous NA on similar individuals (van Rooijen et al., 2017), a clinician could select the order of intervention on these symptoms. On the other hand, following the principles of personalized interventions, it could be possible that if there were repeated measures of symptoms of the same patient (i.e., longitudinal data gathering) the clinician might assess the dynamics of symptoms over time which could contribute to enhance the treatment (Bak et al., 2016). Yet, these

promises cannot be taken for granted and must be further tested in sound clinical and experimental programmatic research.

Also, it will be important to develop rigorous analytic methods that allow exploring the reliability of the networks. As Dejonckheere et al. (2017) have pointed out, indexes as those related to centrality may be highly idiosyncratic for each study as minor variations. Thus, it seems crucial that, to favour the replicability in science, developments in NA allow the comparability of different studies and the analysis of commonalities in networks NA research must be guided by hypotheses and the already remarkable amount of extant evidence on the aetiology of psychopathology rather than conducting blind exploratory analyses based on the sophisticated analytical tools that NA provides (Wichers et al., 2017). There is some risk of overuse of NA with no clear theory-driven plan of research and the risk that the use of this tool in psychopathology becomes a fad. NA developers and users must be aware that new tools, however apparently sophisticated, must have clinical utility and, ultimately, being sensitive to changes in the clinical population (Fava & Belaise, 2005). The heterogeneity of both the existing analytic tools and results, so far, should favour caution over a mindless use of NA in the clinical field.

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CHAPTER 5. A TEMPORAL NETWORK APPROACH TO STUDY PARANOIA. [8]

As we have seen in Chapter 1 and 2, the symptom-specific approach in the psychosis field has turned out in a large amount of research shedding light about psychological components involved in paranoid (persecutory) beliefs and other positive symptoms. In particular, among these processes, self-esteem (SE) has caught a lot of the attention of research. SE is considered a dynamic process, that is, its levels fluctuate over time due to a variety of factors, personal or contextual (Thewissen et al., 2008). Fluctuations in SE are common in many mental health disorders, especially where negative self-views are involved, such as in persons with paranoid thinking (Barrowclough et al., 2003; Thewissen et al., 2008). For instance, for an individual with poor self-evaluation, negative daily experiences can trigger fluctuations in the emotional state of individuals with persecutory beliefs (Murphy et al., 2018).

Accordingly, a large number of studies on the psychological mechanisms of paranoia have indicated that it is pivotal to take into account the dynamic aspects of SE and that its fluctuations are a more important determinant of paranoia than the level of SE per se (Jasper Palmier-Claus et al., 2011). In the same line, prominent psychological models of persecutory thinking like the *self-serving bias theory* stressed the importance SE variations over time (Bentall et al., 2001). Meta-analytic evidence has revealed that the degree of SE instability is significantly and positively correlated with paranoia severity which supports the self-serving model of paranoia (Murphy et al., 2018). However, the research on SE instability is scarce. There is only a few studies that support that paranoia is characterized by fluctuations in SE (Erickson & Lysaker, 2012; Palmier-Claus et al., 2011; Thewissen et al., 2008; Udachina et al., 2012).

[8] Part of the information contained in this chapter corresponds to the following manuscript: **Contreras, A.,** Valiente, C., Heeren, A. & Bentall, R. (2020, *accepted*). A temporal network approach to paranoia: a pilot study. *Frontiers.*

Furthermore, SE fluctuations in paranoia may vary over time due to other factors such as beliefs about others or emotional regulation difficulties in the presence of negative events among other things. However, the few studies focusing on SE fluctuations and paranoia have not taken into account other core paranoia-related processes that might interact with each other. Hence, it is pivotal to study SE fluctuations, not only in the context of daily life, but also taking into account other related processes. As some authors has pointed out, only a multifactorial understanding of symptom development and maintenance adequately reflects this phenomenon (Freeman et al., 2002) and we should bear in mind that these processes may vary over time and affect each other.

The network approach (NA) presented in Chapter 3, allows us to conceptualized paranoid thinking and related processes as a network of interacting elements, taking into account the influence of each of these elements with each other. From the NA one can expect that relevant paranoia-related processes (e.g., self-esteem, mood, emotional regulation or beliefs about others) are embedded within a network system wherein they trigger one another over time. Previous NA research has relied on cross-sectional network models to study psychotic symptoms (Isvoranu, Boyette, Guloksuz, & Borsboom, 2018; Isvoranu et al., 2017; van Rooijen et al., 2017) but only a few studies included paranoid thinking as an element in the network (Bell & O'Driscoll, 2018; Hajdúk et al., 2019; Isvoranu et al., 2016). All these studies were cross-sectional, which do not allow to study changes over time and thus, precluding strong inference regarding the causal (e.g., Maurage et al., 2013) as well as the temporal relationships among paranoia-related processes (e.g., Bos et al., 2017). According to findings from the study presented in Chapter 4, whereas cross-sectional networks are good at describing associations between the average scores on the variables of interest, they fall short of explaining how these variables dynamically trigger each other over time. Hereafter, we believe conceptualizing

paranoia-related processes as a temporal network, rather than restricting it to cross-sectional associations between processes, may offers clues to generating new hypotheses about the temporal dynamic interplay of self-esteem and paranoia-related processes.

A dynamic conceptualization of paranoia-related processes can be done by generating network models from intensive time-series data collected via Experience Sampling Methodology (ESM; for a review see Myin-Germeys et al., 2018). ESM allows assessing psychological constructs repeatedly in daily life. This approach offers a great advantage as it allows taking into account information arising from both the intra- and inter-individual level (e.g., Epskamp, Borsboom, et al., 2018a; Epskamp, Waldorp, et al., 2018). Some ESM studies have shed light on time-lagged associations between paranoia and core processes like self-esteem, negative affect or experiential avoidance (Ben-Zeev et al., 2011; Kramer et al., 2014; Thewissen et al., 2008; 2011; Udachina 2009; 2014). Although the aforementioned variables have been well established in previous ESM research, a network perspective provides a different approach. Temporal network analysis call upon a multilevel vector autoregressive (mlVAR) approach that allows the estimations of three types of networks taking into account three different time frames (i.e., the same measurement time, different measurement occasion and one-week average; Epskamp et al., 2018). The advantage of multilevel temporal network model is that it offers the possibility of considering the intra- and the inter-individual level of information and create network models that control for all other variables and temporal effects (e.g., Epskamp, Borsboom, & Fried, 2018; Epskamp, Waldorp, Mõttus, & Borsboom, 2018). In this way, by using a multilevel vector autoregressive (VAR) analysis from the network approach in combination to the ESM data, one may disentangle the temporal sequence of the dynamic interaction between the variables of interest (Bringmann et al., 2016; Epskamp, Borsboom, et al., 2018; Epskamp, Waldorp, et al., 2018; Hoorelbeke et al.,

2019). Although there is a growing interest in applying time-series network analysis in psychology, only one single case study has, to date, included paranoia as an element in a temporal network (Bak et al., 2016).

This chapter provides the information related to the study 2, whose aim was to examine the temporal dynamics of paranoia-related processes from a multifactorial perspective by conducting time-series network analyses on ESM data.

5.1. Methods

5.1.1. Open Science Practice

The de-identified data, ESM items as well as R code are publicly available via the Open Science Framework and can be accessed at https://osf.io/7tk4b/.

5.1.2. Participants

Participants were recruited from a larger randomized controlled trial registered at ClinicalTrials.gov (https://clinicaltrials.gov/ct2/show/NCT04476771), aiming at testing the impact of psychological group-intervention for people with paranoid tendencies (Valiente et al., 2019). They were attending the Psychology Clinic of the Complutense University of Madrid for clinical psychological distress[9] (i.e., mood, anxiety, interpersonal or non-specified problems) and they were referred by their therapist. The current paper reports findings from the assessment phase that preceded the treatment protocol.

Eligibility criteria were as follows: a) be over 18 years old b) scoring at least one SD above the population mean in the subscales for paranoid ideation and/or interpersonal

[9] Participants are users who attend the University Psychology Clinic for different problems. They have significant clinical problems that have led them to seek psychotherapy to the clinic, this does not mean that all of them meet a diagnostic criteria of a mental health disorder. Additionally, in line with the transdiagnostic nature of paranoid thinking, they all score high on paranoia, but do not meet criteria for any psychotic disorder (i.e., subclinical paranoia).

sensitivity of the validated Spanish-version of the Symptom Checklist-90-Revised (SCL-90-R; Derogatis, 2001). This is a widely used scale, especially as a screening tool, to assess psychological and psychopathological symptoms in both, clinical and normal populations (Derogatis, 2001). The latter criterion was used to broaden the range of paranoid experience included in the study, as previous research has revealed that interpersonal sensitivity is associated with paranoid thinking (Bebbington et al., 2013; Isvoranu et al., 2016; Meisel et al., 2018), and paranoid ideation can be considered as an extension of such concerns (Freeman & Garety, 2014). Following the screening procedure, 64 patients were enrolled in the study and thus participated in the ESM assessment. Of this sample, we only included data from participants providing over 21 valid responses (i.e., 1/3 of potential total number of responses; a cut-off based on prior research combining network analysis and ESM methodology (Aalbers et al., 2019; Greene, Gelkopf, Fried, Robinaugh, & Pickman, 2019). The resulting 23 participants who were included in the analyses (82.6% females) completed an average of 28.48 measurements (SD = 6.58). Participants demographics and clinical characteristics are depicted in Table 1. Note that included and excluded participants did not differ on any demographic or clinical variables (see Appendix, Table A4).

Table 1.
Participants' demographic and clinical characteristics

	Participants *n*=23
Demographic characteristics	
Age in years, mean (SD)	23.78 (6.17)
Sex: Women, n (%)	19 (82.6)
Single status, n (%)	22 (95.7)
Education, n (%)	
Secondary School	3 (13)
Post-secondary	20 (87)
Employed, n (%)	
Unemployed	15 (65.2)
Part-time employment	5 (21.7)
Full-time employment	3 (13)
Clinical characteristics	
SCL-90-R paranoid ideation, mean (SD)	1.24 (0.92)
SCL-90-R interpersonal susceptibility, mean (SD)	1.57 (0.86)
SCL-90-R anxiety, mean (SD)	1.10 (0.46)
SCL-90-R depression, mean (SD)	1.93 (0.74)
Participants Diagnosis: n (%)	
Not meeting criteria for diagnosis	9 (39.1)
Major depression disorder	5 (21.7)
Anxiety disorder (includes PD and GAD)	3 (13)
Post-Traumatic Stress Disorder	2 (8.7)
Dysthymia	3 (13)
Trichotillomania	1 (1.3)
ESM observations	
Number of completed observations, mean (SD)	28.48 (6.58)
Number of missing beeps, mean (SD)	30.83 (13.90)
Number of missing data, mean (SD)	10.70 (12.95)

Note. SCL-90-R=Symptom Checklist-90-R (Cronbach's α= 0.79-0.90); SD=Standard Deviation; PD=Panic Disorder; GAD= Generalized Anxiety Disorder; Number of missing beeps=the final amount of missing notifications due to technical problems; Number of missing data=notifications that participants did not response.

5.1.3. Experience Sampling Methodology (ESM)

The study of psychological phenomena in daily life and in their natural context has been made possible thanks to the emergence of the ESM. Larson and Csikszentmihalyi, (1983) developed the methodology in the US while, in Europe, its

application in the field of mental health begins with the work Dutch Maastricht researchers (Palmier-Caus et al., 2019). The ESM is a daily self-reported assessment, that functions as a diary, which allows the appraisal of a variety of internal mental phenomena (i.e. mood, thoughts, symptoms) (Myin-Germeys et al., 2003). To carry out the daily assessment the individuals carry with them an electronic device (e.g., wristwatches or notepads) throughout the day and they respond to a set of evaluations previously scheduled (i.e. several times per day, during a number of days) according to the objectives of the research. Nowadays, with advances in technology, participants are asked to report their momentary assessment when their prompted by "a beep" in their smartphones (Myin-Germeys et al., 2018). The monitoring of numerous measures of life experiences allows to assess variability and to better study dynamics nature of psychological phenomena (Palmier-Caus et al., 2019). The use of ESM presents several advantages: a) it evaluates psychological phenomena as they occur, reducing potential retrospective recall biases; b) the assessment is more accurate as it offers an understanding of how symptoms evolve in daily life over time; c) it allows to directly examine the emotional experience and its relation with subsequent activities and behaviors; d) it allows stronger causal inferences than cross-sectional data and e) given that the phenomena is measured in the context of daily life, it has an increased ecological validity.

In the current study, a time-contingent ESM design was used as recommended by Myin-Germeys et al., (2018) whereby participants received ten notifications a day between 9:00AM and 10:00PM over a 7-day period. We used a stratified schedule wherein, each day, ten notifications were delivered between intervals of, at least, 30 minutes between each signal. The assessment was also programmed to cease to be available 15 minutes after it beeps, as there are evidence that reports completed after this interval are less reliable and less valid (Delespaul, 1995). The study used part of a larger

ESM protocol which included 33 items. For the present study, we only focused on paranoia-related processes, which include:

a) *Negative affect*. We use one item to measure sadness ("At this moment, I feel sad"). This ESM item has been previously used to study negative affect (e.g., Palmier-Claus, 2011).

b) *Self-esteem*. We adapted two item from the Rosenberg Self-esteem Scale (RSES; Rosenberg, 1989) ("At this moment, I feel useful" and "At this moment, I feel I can manage issues well"). The internal consistency of this scale was α=.745;

c) *Closeness to others*. As persecutory beliefs are also associated with negative beliefs about others as well as social exclusion, we designed one item to assess how close individuals perceived others ("At this moment, I feel close to others");

d) *Experiential avoidance*, as an emotional regulation strategy. We adapted one item from the Acceptance and Action Questionnaire-II (AAQ-II; Hayes et al., 2004) ("Since the last beep, I have tried to avoid negative thoughts and feelings") and;

e) *Persecutory beliefs*. Following the Persecutory Ideation Questionnaire (PIQ; McKay, R., Langdon, R., & Coltheart, 2006) we adapted three items ("Since the last beep, I have had the impression that I cannot trust people", "I have had the impression that people have tried to harm me" and "I have had the impression that people have criticized me"). The internal consistency of this scale was α=.737.

All items were presented on a 9-point Likert-type scale and a careful translation was carried out, following the latest indications (e.g., Wild et al., 2005). Please note that items were translated into English for publication and exposition purpose, for original Spanish items (see Appendix, Table A5). ESM items were expressed in "colloquial" terms rather than technical terms, so they closely represent how individuals from the target population may describe their own experiences (Palmier-Claus et al., 2019).

Furthermore, the ESM item wording was adjusted to assess relevant experiences that might occur beyond the exact assessment points (Palmier-Claus et al., 2019). That is, the time frame used to assess sadness, closeness to others and self-esteem is amended to provided "truly momentary assessments" at a given time point (i.e., "At this moment…"). Conversely, the time frame used to assess experiential avoidance and paranoid beliefs differ, as they are amended to be "retrospective reports of experiences" (i.e., "Since the last beep…"). Although the retrospective reports may involve the retrieval and retrospective evaluation, they are often extremely useful due to their ability to capture relevant event that might not be possible to assess in a given time point. In other words, if the individual is, for instance, struggling with experiences of avoidance or persecution, it may be difficult to respond in that given time (Palmier-Claus et al., 2019).

5.1.4. Procedure

As a part of a broader clinical trial project, the ESM data reported here was collected during the baseline assessment of the trial (Valiente et al., 2019). At the outset, we used PACO APP (The Personal Analytics Companion; https://pacoapp.com), a free and open-source application for building and running ESM studies. However, this application ceased to be accessible in midway through the research, at which point we switched to the Qualtrics platform (www.qualtrics.com). Note that in both platforms everything was made identical and there were no significant differences in terms of demographic and clinical characteristics between people who received the ESM-procedure via the first versus the second platform (see Table 2).

Table 2.
Differences test for demographic, clinical and ESM characteristics between participants who use app1 (PACO App) and app2 (Qualtrics)

	app1 (n=51)	app2 (n=13)	t/x^2	*p*
Demographic characteristics				
Age in years, mean (SD)	23.27 (4.80)	24.15 (5.90)	-0.56	0.57
Sex: Women, n (%)	43 (84.3)	10 (76.9)	0.39	0.52
Single status, n (%)	50 (98)	12 (92.3)	1.12	0.28
Education, n (%)			0.03	0.84
Secondary School	9 (17.6)	2 (15.4)		
Post-secondary	42 (82.4)	11 (84.6)		
Employed, n (%)			0.62	0.73
Unemployed	32 (62.7)	9 (69.2)		
Part-time employment	13 (25.5)	2 (15.4)		
Full-time employment	6 (11.8)	2 (15.4)		
Clinical characteristics, mean (SD)				
SCL-90-R paranoid ideation	1.26 (0.88)	0.93 (0.76)	1.24	0.21
SCL-90-R interpersonal susceptibility	1.72 (0.79)	1.25 (0.80)	1.89	0.06
SCL-90-R anxiety	1.25 (0.64)	1.01 (0.49)	1.26	0.21
SCL-90-R depression	1.98 (0.76)	1.81 (0.56)	0.76	0.44

Note. SCL-90-R= Symptom Checklist-90-R (Cronbach's α= 0.79-0.90); SD= Standard Deviation; *p-value<0.05; ** p-value<0.01; *** p-value<0.001.

An initial one-on-one instructional session was organized with each participant. During this session, the ESM-platform use was demonstrated and turned on the notification's parameters of the participant's mobile phone. Participants were given an email address to contact in case of questions or technical problems with the application.

The study was approved by the Complutense University of Madrid's Institutional Review Board and conducted according to the Declaration of Helsinki (see Appendix). All participants provided written informed consent and were fully debriefed at the end of the study.

5.1.5. Data analysis

5.1.5.1. Identifying redundant items

Because some ESM variables were measured with more than one item, we use a data driven approach to identify potential conceptual overlap between them. Following

previous research (Bernstein et al., 2019), we first tested that the correlation matrix was positive definite, using the *is.positive.definite* function within the *corpcor* R package (Schäfer et al., 2017). Secondly, we identify nodes which most likely measure the same underlying construct with goldbricker function within *networktools* R package (Jones et al., 2019). The results of the function suggested to reduce self-esteem (2 items) and paranoia (3 items). Therefore, we calculate the average of these items before including in the analysis.

5.1.5.2. Assumptions check

The Multilevel Vector Autoregressive (mlVAR) model has two main assumptions. The first assumption is normality. We used the Shapiro-Wilk Normality Test to test whether each variable was normally distributed. Besides, mlVAR analysis employed to analyse the data assumes that the mean and variance of a variable do not change as a function of time - i.e., the assumption of stationarity (Aalbers et al., 2019; Bringmann et al., 2016). To test the second assumption, we used the *Kwiatkowksi-Phillips-Schmidt-Shin unit root test* (KPSS Test for Stationarity) for each variable of each participant, as implemented in the R package *tseries* (Trapletti, & Hornik, 2019).

5.1.5.3. Network estimation and visualization

To analyse the dynamic relationships between the variables, we used the VAR model on the ESM data, implemented in *mlVAR* R package (version 0.4.3; Epskamp, Deserno, et al., 2019). Within this model, we estimate three network structures (Epskamp, Waldorp, et al., 2018): 1) A contemporaneous network, which is a Gaussian Graphical Model (GGM) that depicts within-time-window edges (associations between nodes) corresponding to a multilevel partial correlation network, after controlling for temporal associations; 2) A temporal network, which is a directed network of regression coefficients that depicts the lagged associations between nodes from one measurement

point to the next after controlling for all other variables at the previous measurement point; and 3) Finally, a between-subject network, which is a GGM that depicts regularized partial correlations (after taking into account the remaining variables in the network) between individual's means during a specific period of time (Epskamp, Borsboom, et al., 2018; Epskamp, Waldorp, et al., 2018). Thus, while contemporaneous inform about association at the same timeframe, the between-subject network model reveals, on average (i.e., one-week assessment), the variance-covariance structure of participants' means.

Decomposing the variance in these three distinct networks provides different but complementary insights in the covariation and potential dynamics of the constructs of interest. First, the contemporaneous network shows whether deviations from a person's means on two variables predict one-another at the same measurement occasion (Epskamp, van Borkulo, et al., 2018; Greene et al., 2018). Second, the temporal network indicates whether a deviation from a person's mean predicts a deviation from that person's mean in another variable at the next measurement occasion (Bringmann et al., 2016; Epskamp, van Borkulo, et al., 2018). Finally, the between-subjects network mirrors the covariation between means of participants (Epskamp, van Borkulo, et al., 2018; Greene et al., 2018) and, in this way, allows for comparison with previous cross-sectional studies (Epskamp, Borsboom, et al., 2018; Epskamp, Waldorp, et al., 2018).

The *mlVAR* package calls upon the qgraph package (version 1.6.3; Epskamp, Costantini, et al., 2019) to plot the estimated coefficients as graphical network models. For the contemporaneous and between-subject networks, we used the conservative "AND-rule" approach in retaining significant edges—that is, an edge was retained if both regressions on which the edge was based were significant ($\alpha = 0.05$). For each network model, blue lines on the graphical representations show positive associations, whereas

red lines show negative ones. The strength of the connectivity is visually represented by thickness of the edges. A thicker edge denotes a larger association. In the temporal network, arrows are used as edges to illustrate the direction of effects. Interpretations regarding centrality of nodes rely on visual inspection of the obtained network structures, given that standardized centrality indices are not ideal for multilevel VAR-models (Bringmann et al., 2016; Thewissen et al., 2008).

5.2. Results

5.2.1. Descriptive statistics

Means and standard deviations of within-participants means and within-participants standard deviations of each variable are depicted in Table 3.

Table 3.
Means and Standard Deviations of within-participants Means and Within-participant Standard Deviations for all ESM variables

ESM variable	*M (SD)*	*SD (SD)*
Sad	3.92 (1.59)	1.73 (0.50)
Closeness to others (Others)	4.73 (1.36)	1.59 (0.62)
Self-esteem (SE)	4.95 (1.12)	1.27 (0.40)
Experiential Avoidance (EA)	4.21 (1.79)	1.77 (0.61)
Paranoia	2.01 (1.03)	0.78 (0.50)

Note. Following previous research and transparency guidelines, we provided this data as they are the input to the network model estimation, as explained in the Method sections. *M*=Mean; *SD*=Standard Deviation. All variables were measured using a 9-point Likert scale (range 0-9).

5.2.2. Assumptions checks

Shapiro-Wilk tests indicated that no variable was normally distributed (see Table 4). As we aimed to account for temporal variability, we conducted the analysis as planned regardless this assumption. Kwiatkowski-Phillips-Schmidt-Shin unit root tests suggested stationary data for all variables in all participants.

Table 4.
Shapiro-Wilk normality test values

Variable	Statistic	*p*
Sad	0.91173	0.000***
SE	0.98427	0.000***
Others	0.96034	0.000***
EA	0.90518	0.000***
Paranoia	0.75771	0.000***

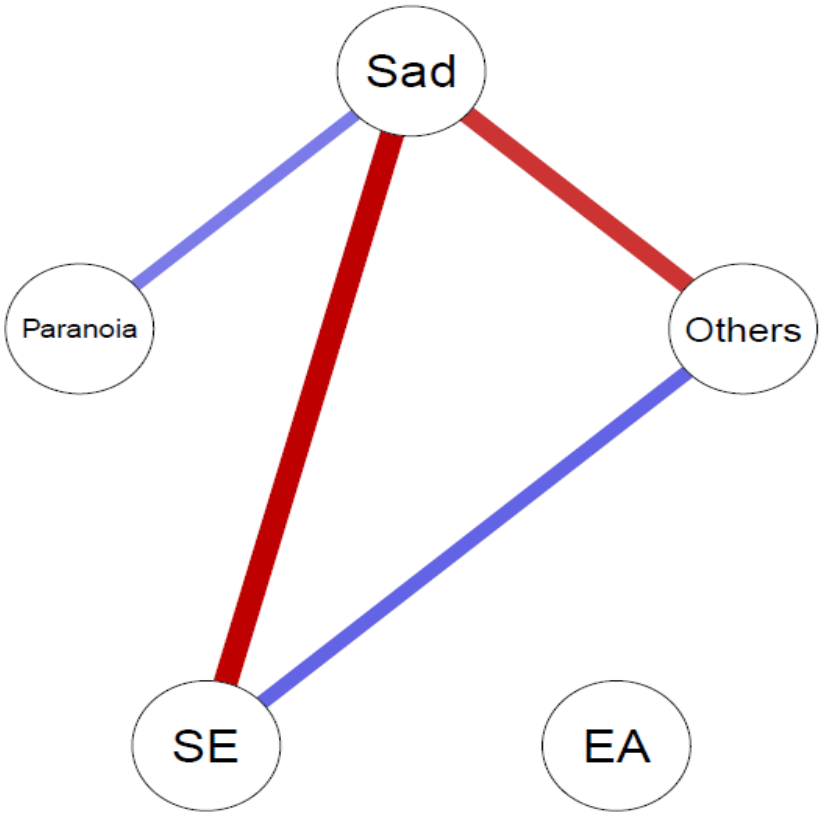
Note: *p-value<0.05; ** p-value<0.01; *** p-value<0.001

5.2.3. Network estimation and visualization

5.2.3.1. Contemporaneous network

Figure 1 depicts the contemporaneous network— i.e. the associations between the variables within the same timeframe after controlling for all other temporal and contemporaneous relations. A few connections stand out. Paranoia and sadness are positively associated, suggesting that higher scores in paranoia are associated with higher of levels of sadness during the same timeframe. Likewise, sadness is negatively associated with both self-esteem and feeling close to others. In other words, the higher the sadness, the lower the levels of self-esteem and feeling close to others at the same moment. Moreover, self-esteem and feeling of being close to others are positively associated. Finally, experiential avoidance is not connected to any other variable in this analysis (correlation matrix of the data is provided in the Appendix section).

Figure 1.
Contemporaneous network model



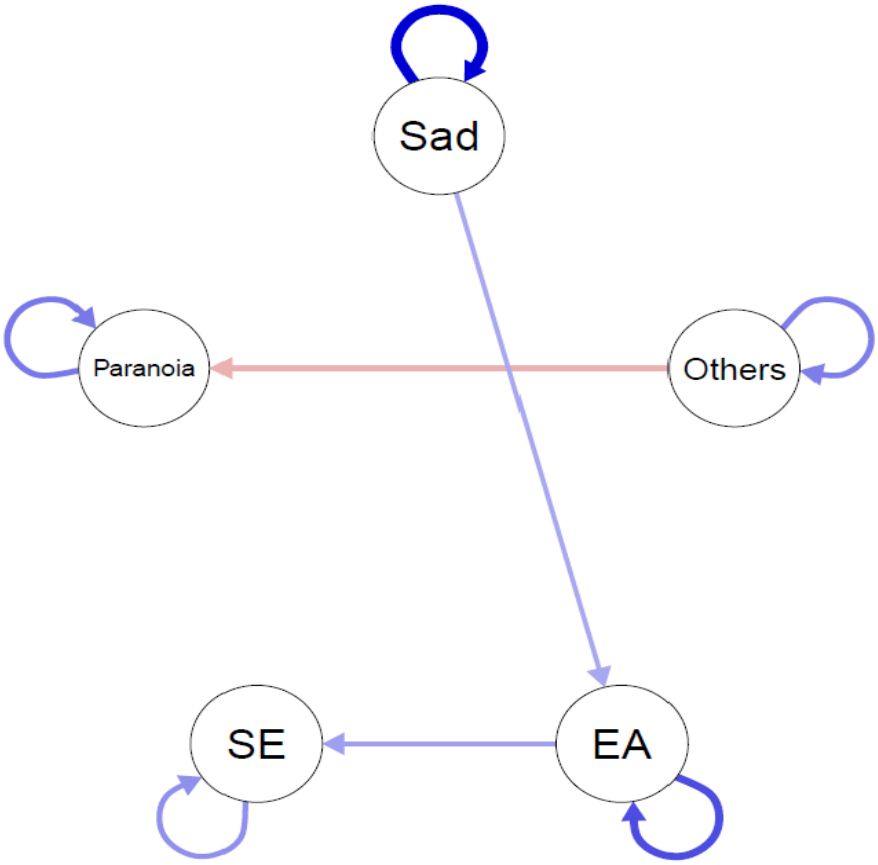
Note. Edges represent associations between the variables within the same timeframe after controlling for temporal associations. Blue lines depict positive associations and red lines depict negative associations between variables. SE=Self-Esteem, EA=Experiential Avoidance, Others=Closeness to others.

5.2.3.2. Temporal network

Figure 2 depicts the temporal network, which represents the extent to which nodes predicted themselves (i.e., autoregression) and each other from one timeframe (*t*) to the next (*t+1*). The arrow depicts the direction of the prediction and this analysis is much more informative about potential causal mechanisms. Unsurprisingly, all nodes show positive autocorrelation over time; with sadness being particularly autoregressive; these findings simply show the relative stability of these variables over relatively short time frames. Much more importantly, feeling of being close to others negatively predicted paranoia at the next time point and sadness positively predicted experiential avoidance,

which, in turn, positively predicted self-esteem at the next time point (correlation matrix of the data is provided in the Appendix section).

Figure 2.
Temporal network model



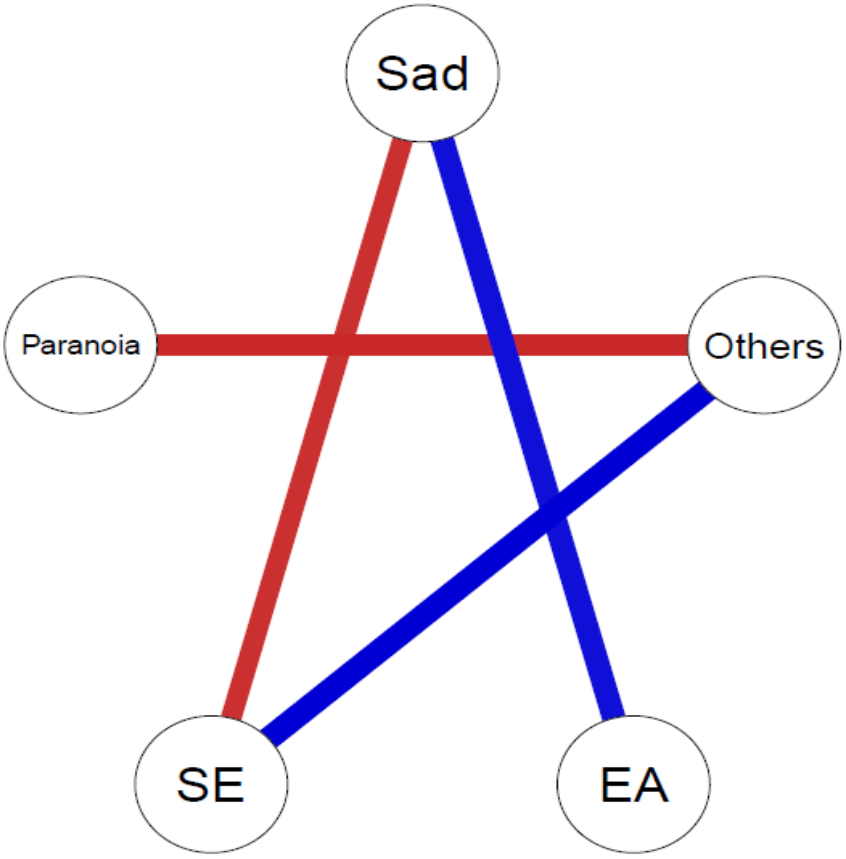
Note. Edges represent prediction between nodes from one measurement point to the next that remain after controlling for all other variables at the previous measurement point. Blue lines depict positive associations and red lines depict negative associations between ESM variables. SE=Self-Esteem, EA=Experiential Avoidance, Others=Closeness to others.

5.2.3.3. Between-subjects network

The between-subjects network shows the correlations between intra-individual mean levels of the nodes over the entire testing week. That is, the associations between individual's means during the overall ESM week. As shown in Figure 3, the mean levels of closeness to others was negatively associated with the mean levels of paranoia and positively associated with mean levels of self-esteem. The mean level of sadness was

negatively associated with the mean levels of self-esteem and positively associated with mean levels of experiential avoidance (correlation matrix of the data is provided in the Appendix section).

Figure 3.
Between-subject network model



Note. Edges represent correlations between intra-individual mean levels, after taking into account the remaining variables in the network. Blue lines depict positive associations and red lines depict negative associations between ESM variables. SE=Self-Esteem, EA=Experiential Avoidance, Others=Closeness to others.

5.3. Discussion

In this study, we aimed at unfolding the temporal interplay between sadness, experiential avoidance, self-esteem, feeling of being close to others, and paranoia during one ESM week among 23 participants with high scores in paranoia. We applied temporal network analysis and computed three network models to examine within and between

individual differences over time. The contemporaneous and between-subjects networks consider the way that the variables of interest covary, at the same time point and on average, respectively; the latter in effect replicates cross sectional analyses previously reported in the literature but with longitudinal data (Epskamp, van Borkulo, et al., 2018; Epskamp, Waldorp, et al., 2018). The temporal network allows us to move closer to identifying the potential causal interplay between the variables by considering how events at one-time point predict what happens at the next. It is worth noting that, because of the timing of the ESM assessments, the temporal model can only detect potential causal associations that take place over a number of hours (Greene et al., 2018). Note also that all nodes show self-loops, indicating that all variables predict themselves at the next time frame, which may point to a degree of stability in the variables. Some previous ESM studies have used lagged data to identify how changes in specific variables lead to changes in others, for example how low self-esteem (Thewissen et al., 2008), experiential avoidance (Udachina et al., 2014) and momentary attachment insecurity (Sitko et al., 2016) relate to paranoia, but network analysis allows the interplay between all of these variables to be considered at the same time. Together these networks provide insights into the dynamical nature of paranoid beliefs when taking into account different timeframes.

Perhaps the most surprising finding is a negative one: paranoia was not directly related to self-esteem in any of the three networks. Hence, although there has been consistent evidence supporting the role of self-esteem in paranoia from previous studies (Kesting & Lincoln, 2013; Murphy et al., 2018), the current findings do not replicate this effect, and therefore call into question psychological models which afford self-esteem a central role, for example the attributional model of paranoia proposed by Bentall et al. (2001). Several explanations could account for this unexpected lack of association, other than the possibility that it does not, in fact, exist.

First, the relationship between the two variables might have become non-significant when controlling for the remaining variables in the model. Sadness is a candidate variable in this respect. Both the contemporaneous and between-subjects networks reported a negative association between sadness and self-esteem consistent with numerous previous time-series studies of both depression (Orth & Robins, 2013) and paranoia (Thewissen et al., 2011). Second, the association between paranoia and self-esteem might be mediated by other processes. In fact, our contemporaneous network model evidences that the relation between paranoia and self-esteem is conditionally independent, given the presence of sadness. These findings are consistent with previous cross-sectional research that shows associations between paranoia, low mood, and low self-esteem (e.g., Thewissen et al., 2011). This result indicate that sadness could have a mediating role on a small timescale, supporting previous findings that point to depression as a significant mediator in the relationship between paranoia and self-esteem (Ben-Zeev et al., 2009). These findings are also consistent with previous cross sectional research which shows associations between paranoia, low mood and low self-esteem (e.g., Thewissen et al., 2011). Accordingly, the current findings can be interpreted as in line with the cognitive perspective of paranoia, which assumes that low self-esteem affects paranoia largely through depressed and anxious symptomatology (Freeman, 2016).

The most important positive finding of this study is that closeness to others is directly and negatively associated with paranoia in the between-subjects network, and the findings from the temporal network show a similar effect, raising the possibility that this effect is causal. This observation aligns with an attachment framework for understanding paranoia (Bentall & Sitko, 2020) and previous studies showing that paranoia can be triggered by interpersonal factors such as negative interpersonal schemata (Lincoln et al., 2010), momentary attachment insecurity (Sitko et al., 2016), perceived social exclusion

(Westermann et al., 2012), and impoverished social network (Gayer-Anderson & Morgane, 2013). Furthermore, our results reveal that closeness to others is positively associated with self-esteem in both, the between-subject and the contemporaneous network. Again these findings are completely consistent with previous cross-sectional research which shows associations between positive beliefs about the self and secure attachment (Gayer-Anderson & Morgane, 2013).

Finally, the networks are informative about the role of experiential avoidance. Experiential avoidance refers to the need to avoid distressing mental contents, and has been hypothesised to play an important role in maintaining psychopathology (Hayes et al., 2004). Previous studies have reported high experiential avoidance in paranoid patients and non-patients. In studies with analogue (Udachina et al., 2009) and patient samples (Udachina et al., 2014) some positive direct effect of experiential avoidance on paranoia was found, as well as an indirect effect through lowered self-esteem; these findings were interpreted as paranoia arising from failed attempts to avoid negative thoughts about the self. In our analyses of the present data, however, no direct association between experiential avoidance and paranoia was found. Moreover, the association between experiential avoidance and sadness is consistent in the between-subjects and temporal network. In the temporal network, sadness provoked experiential avoidance which in turn led to improved self-esteem, an effect that is entirely consistent with the original conceptualization of the experiential concept by Hayes et al. (2014). Overall, these findings suggest a complex relationship between experiential avoidance and mood but, at most, a distant and very indirect role for experiential avoidance in paranoid thinking.

5.3.1. Clinical implications

Our findings yield clinical implications. Overall, our results underscore the importance of taking into account the dynamic nature of psychological phenomena.

Temporal network analysis might be useful to identify potential therapeutic target that may change the dynamic in the network. Specifically, based on the results from the temporal network model, it may be hypothesized that intervening on attachment related cognitions may reduce paranoid thinking over time (Bentall, & Sitko, 2020). It is possible that clinical effects will be enhanced by focusing on the positive aspect of social relations, instead of focusing on their deficit (Wykes, Steel, Everitt, & Tarrier, 2008). This idea is in line with the recent increased awareness in the need for a positive movement in psychology, focused on positive psychosocial factors and well-being (Jeste, Palmer, & Saks, 2017). Positive psychology intervention for psychosis have encouraged the enhancement of positive social relationships as a protective factor (Slade, Brownell, Rashid, & Schrank, 2016; Slade, 2010), and accordingly, our findings suggest that targeting interpersonal processes might be beneficial for people with vulnerability to paranoia.

5.3.2. Limitations and strengths

This is a pilot study and several issues require further examination in follow-up research. First, the compliance to the ESM-protocol was low, resulting in a small final sample size (n= 23). Although the number of measurements per person is satisfactory, the sample is modest for temporal network analysis and replications would benefit from a larger group of participants. Several explanations may explain this issue. To begin with, because the use of both ESM-apps relies on internet access, one cannot exclude that whenever participants were not connected to a proper internet network, they were not properly notified and, in turn, missed the beeps (see Appendix, Table A4). Another potential explanation could be that people with high paranoid tendencies do not rely on devices or do not feel comfortable sharing their experiences via an app. However, although prior research has made efforts to cast light on potential predictors of compliance

and did not found demographic or clinical variables to be related to lack of compliance (Hartley et al., 2014), the study of the adherence to an ESM-protocol among people with paranoid tendency has yet to be done. Second, our three network models are low dimensional (i.e., few nodes relative to the number of participants). Network analyses, like any statistical tool, can only examine variables that are entered into a model. Therefore, though the current graphs are parsimonious with only five nodes and based on current prominent models of paranoia, there could be important variables left out. For instance, the external explanation for negative events and a distinction between explicit and implicit self-esteem are important components in one of the most influential model of paranoia (Bentall et al., 2001; Murphy et al., 2018). Thus, we encourage future research to compare empirical data model to theory models (Haslbeck et al., 2019). Third, as this sample was made of participants with paranoia vulnerability (i.e., subclinical population), the mean levels of paranoia are low. We consider that replications of the current study in a population with higher paranoid severity are needed. Fourth, our data did not meet normality assumptions. Such an approach is common in psychological sciences and has been reported in previous temporal network studies (e.g., Aalbers et al., 2018). Some authors have highlighted that assuming normally distributed parameters can be problematic because it imposes that subjects cannot differ on the structure of the network (Epskamp, Waldorp, et al., 2018). However, it is still unclear how robust time-series analysis is to these violations, and results should be interpreted cautiously. We consider this issue to be an essential direction for future work on temporal network analysis. In addition, it is pivotal to state that the obtained results are useful for generating hypotheses about the causality of paranoia-related processes, but not sufficient to draw robust conclusions about true causality. Finally, there are procedures available to test the robustness and accuracy of estimated parameters from cross-sectional data (Epskamp,

Borsboom, et al., 2018), but unfortunately there is no tools available for time-series data and mlVAR (Aalbers et al., 2019). Hence, we encourage future research to develop methodological procedures to assess the quality of temporal networks.

Despite these constraints, an important quality of the current study is that we have applied a complex methodology and provide all material to replicate the study via Open Science Framework. We also share potential methodological issues that future research may encounter and should be aware of in order to move forward in the understanding of this methodology in the paranoia field.

5.3.3. Conclusion

The current study provided important contributions to paranoia field by identifying how certain psychological mechanisms such as self-esteem, sadness, feeling close to others and experiential avoidance are meaningfully related to paranoia. In addition, we provided evidence that psychopathology can be conceptualized as a complex dynamical system and that temporal (time-series) network analysis as a useful approach to provide novel insight about the complexity of mechanisms implicated in paranoia.

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CHAPTER 6. GENERAL DISCUSSION AND CONCLUSION

The aim of the current dissertation was to investigate the 'state of art' of Network Analysis (NA) in the psychopathology field and to analyse its application in the study of the dynamics of paranoid thinking.

In **Chapter 1** we have seen how the evolution of the concept of paranoid thinking has led to its current definition as a multifactorial and continuous process. There have been significant efforts in the field of psychosis research to abandon traditional conceptualizations of mental disorders from a common cause model and this trend has been most enlightening in the study of paranoid thinking as a symptom. In **Chapter 2**, we have compiled the evidence that research has so far yielded on the main psychological factors related to paranoid (persecutory) thinking. In parallel, we have provided an overview about the main current theoretical explanatory models accounting for the development and maintenance of these beliefs. In this context, it has been pointed out the importance of studying, not only the processes associated with persecutory thinking, but also their fluctuations over time. Thus, it has been proposed that instability or fluctuations in paranoia related process may influence the presence and severity of paranoid beliefs (Bentall et al., 2001; Murphy et al., 2018). According to Bentall (2001 p.1145): "change is the hallmark of psychopathology, and symptoms rarely stay constant". And, the evidence so far seems to support this claim (Erickson & Lysaker, 2012; Palmier-Claus et al., 2011; Thewissen et al., 2008; Udachina et al., 2012). Although paranoid thinking is conceptualized as a multidimensional process, there is little research including the various related processes and how their fluctuations might affect each other over time.

In **Chapter 3** we describe how the emergence of network analysis has burst into psychopathological research in recent years. Its conceptualization of mental problems as interactions between elements influencing each other, seems to fit the needs of studying

paranoid thinking as a multidimensional process. However, the advance of what network theory may entail in our field cannot be understood without studying its applicability at an empirical level. This leads to **Chapter 4**, where we present the findings of a systematic review up to 2017 of all the empirical literature that has applied network analysis to the study of mental health disorders. The results of the review suggest that network analysis is not only a theoretical alternative to the common-cause model, but it also provides tools that allow us to investigate paranoid thinking as a dynamic process. In **Chapter 5**, we have used a temporal network analysis approach and apply multilevel Vector Auto-regressive model (mlVAR) to time-series data (collected via Experience Sampling Methodology-ESM) of multiple subjects with vulnerability to paranoia. This combination of network analysis and time-series enables us to study the fluctuations of psychological phenomena related to paranoid thinking intra and between subjects. The results show that paranoia related processes such as self-esteem, negative affect, experiential avoidance and feeling close to others fluctuate over time and their associations with paranoid thinking differ when taking into account different time frames. The following is a summary of the main contributions of this dissertation, as well as potential future lines of research.

6.1. General discussion

The accelerated growth of the methods provided by the *Network Psychometrics* (Epskamp, Borsboom, et al., 2018) has led to an increase in the empirical literature in psychopathology, making evident the need to analyse these findings in a systematic way. Given the novelty of these techniques, traditional methodologies of meta-analysis cannot be applied so far (Robinaugh et al., 2019). We believe, therefore that the systematic review discussed in Chapter 4 can be considered a good first step towards: a) obtaining a comprehensive view on what the network literature offers to the psychopathology field

and, b) linking the network theory with the empirical evidence (Siddaway et al., 2019). The results show the applicability of network models for the investigation of a wide range of mental problems, as well as its usefulness in providing a plausible explanation of the patterns of association between psychopathological elements.

However, one question that still remains unclear is *what elements should be included in the network* depending upon the problem under scope (Bringmann & Eronen, 2018). The theory proposes that symptoms may be activated by factors external to the person such as a traumatic event (e.g., Isvoranu et al., 2017), or may arise through processes within the person such as the misinterpretation of auditory sensations (e.g., Borsboom et al., 2019; Wigman et al., 2017). Aiming to move towards a holistic research strategy (van Bork et al., 2018), several authors have proposed that the associations in mental problems cannot be understood without including the content of mental states such as beliefs, desires, emotions and intentions, as well as possible historical and cultural variations given that, psychological manifestations differ across contextual factors and cultures (Borsboom et al., 2019). In this vein, although they differ in their assumptions and conceptualizations, the NA reasoning is similar to that proposed by the RDoC initiative, which highlights the importance of incorporating, in addition to symptoms, different units of analysis from different domains (NIMH). The results of the systematic review show that the inclusion of non-symptoms enhance our understanding of mental phenomena (e.g., Heeren & McNally, 2016; Santos et al., 2017). The inclusion of other relevant clinical variables, along with doing justice to the complexity of mental problems, comes closer to more recent arguments that have point out elements (such as cognitive processes or genetic factors, etc.) as more defining characteristics units of mental problems than diagnostic criteria (Vazquez et al., 2014).

But, *how do we measure these elements?* The decision about which instruments should be used to measure the constructs that make up the network can influence the estimation and interpretation of the results (Robinaugh et al., 2019). Our findings show that, while some studies include single items (e.g., Fried et al., 2015), others include a compound scores (Hoorelbeke et al., 2016), and still others investigate the same construct, by using two different assessment instruments (e.g., Bryant et al., 2017; McNally et al., 2017). Additionally, it should be noted that most psychopathology networks have been estimated from data using scales within categorical diagnostic systems, such as the DSM. Although this approach based on DSM data has proven to be useful at the exploratory level and as a hypothesis generator, it could lead, contrary to the initial purpose of network theory, to remain within the common cause model (Borsboom et al., 2019). Furthermore, the fact that the same construct is measured with different instruments may explain the great heterogeneity of results found (Robinaugh et al., 2019). In view of all these notions, future research should work on psychometric strategies that could optimize the evaluation of the components to be included in the network structure (Bringmann & Eronen, 2018). These above-mentioned handicaps could also be overcome by adopting a theory driven approach, where the design of the network study is based on existing theories of the psychological phenomena under study (Haslbeck et al., 2019). This strategy could foster a rich exchange between theoretical and empirical research, in which NA would contribute to the advancement of theories and a greater understanding of psychological phenomena as a causal system (e.g., Heeren et al., 2020).

Finally, *how to share and disseminate NA in psychopathology?* We must emphasize that empirical research on networks is characterized by a great exchange of data and statistical tools (e.g., scripts) that are currently available in several ways (i.e., supplementary materials, open science platform, tutorials, website blogs, etc.). These

practices foster their reproducibility, that is, another analyst can re-perform the same analysis with the same code (Guloksuz et al., 2017; Patil et al., 2016). The present dissertation, in line with this initiative, shares the data and scripts used for the study reported in Chapter 5. Still, besides reproducibility, another fundamental characteristic of scientific studies is also its replicability (Patil et al., 2016). A study in psychology is said to be replicable when, given a population, hypothesis, experimental design, and analysis plan we get consistent estimates when recollecting data and redo the analysis (Patil et al., 2016). In this case, although researchers in the field are doing great efforts (Espkamp, Borsboom, et al., 2018), network studies do not seem to fit so well. Rather, the defining feature of the current state of the network findings is its heterogeneity, regarding their study designs, population studied, tools and materials used. In the future, we believe that it will be necessary for researchers to evaluate which of these exploratory findings are replicable and generalizable which could contribute to the survival of this research approach in the area of psychopathology.

6.1.1. Towards a dynamic perspective

Network theory proposes that mental problems emerge from the causal interaction between symptoms (Borsboom et al., 2019). Nevertheless, without longitudinal data, it is not possible to make statements about temporality, let alone causality (Guloksuz et al., 2017; Robinaugh et al., 2019). Although models using cross-sectional data to estimate psychological networks are necessary as first exploratory steps, they are considered static models (Bringmann & Eronen, 2018), given its limited ability to uncover dynamic relationships between variables (Jordan et al., 2020). Conversely, in clinical research, temporal network models (i.e., estimated from longitudinal data) enable us to detect direct influences that symptoms have on one another over time (Jordan, 2020). Thus, one can

draw conclusions about the dynamics of psychological phenomena closer to potential causality, since they take into account the time factor (Bringmann & Eronen, 2018).

Towards a dynamic perspective develops the study presented in Chapter 5, in which a temporal network model is applied to longitudinal data (i.e., time-series collected via ESM) to study the dynamic of paranoia-related processes in a non-psychotic clinical sample. The decision of including the selected mechanisms related to paranoia (i.e., self-esteem, experiential avoidance, closeness to others, sadness and paranoia) was based on prior ESM multilevel studies and existing theories accounting for paranoia (see Chapter 2). Although the aforementioned variables have been well established in previous ESM research (Ben-Zeev et al., 2011; Kramer et al., 2014; Thewissen et al., 2008, 2011; Udachina et al., 2009, 2014), the temporal model presented in Chapter 5 provides a different approach that might be more informative in comparison to other ESM data analysis approaches, such as multilevel without autoregressive vectors. The temporal network analysis conducted in Chapter 5 call upon a multilevel vector autoregressive (mlVAR) approach that allows the estimations of three types of networks models taking into account three different time frames (Epskamp, Waldorp, et al., 2018): *contemporaneous* model (the same measurement time), *temporal* model (different measurement occasions) and *between-subject* network model (one-week average). These three network models provide different but complementary insights about the fluctuations over time of the selected processes. The advantage of mlVAR is that it offers the possibility of considering the intra- and the inter-individual level of information and create network models that control for all other variables and temporal effects (e.g., Epskamp, Borsboom, 2018; Epskamp, Waldorp, et al., 2018). In this way, one may disentangle the temporal sequence of the dynamic interaction between more than one variable of interest (Bringmann et al., 2016; Epskamp, Borsboom, et al., 2018; Epskamp,

Waldorp, et al., 2018; Hoorelbeke et al., 2019). This enables to investigate paranoid thinking from a multifactorial perspective, as suggested by authors in the field (Freeman et al., 2002). Our findings revealed that processes dynamically interact with each other and that there are differences in the associations between paranoia-related mechanism when different measurement time are taking into account. Therefore, by combining temporal network analysis and time-series data, one can obtain a vision about the natural flow of psychological processes that may attends to more subtle fluctuations in individuals with vulnerability to paranoia (Wigman et al., 2015) and, without assuming the presence of latent variable (Jordan, 2020).

As far as we know, the study presented in Chapter 5 is pioneering in its way of conceptualizing paranoid thinking as a complex dynamic system, so we belief that it offers important and original contributions in the field of paranoia. Importantly, the study must be considered exploratory, since it is the first applying the temporal network methodology to study associations between all these processes at once. Hence, we believe this study can serve as a precedent in generating hypothesis about potential temporal causal relationships when taking into account these processes together. However, this empirical work is a pilot study and is therefore not exempt from limitations that are mentioned below.

6.1.2. Limitations and challenges for future

On the one hand, some aspect related to the ESM deserve attention. The use of ESM is a high-demanding method for individuals, which may increase the chance of low compliance and affect the quality of the data, as we saw in Chapter 5. Research on ESM has not found yet any significant demographic or clinical variables related to lack of compliance (Hartley et al., 2014). However, a recent study has pointed out that individuals with psychosis are less compliant than individuals without psychosis (Rintala et al.,

2019). In our study, although the sample is not made up of individuals with psychosis, a possible explanation for the low compliance could be the difficulty of measuring a mental state at the time it occurs (i.e., paranoid thinking), as this experience might be incompatible with filling out a questionnaire. Interestingly, a recent study has pointed out some other elements may decrease the likelihood of compliance such as feeling disturbed by a "beep" or the use of medication (Rintala et al., 2020). We believe these elements should be taken into account in future research in order to improve compliance rates in different types of ESM designs, both in the general and clinical population (Hartley et al., 2014; Rintala et al., 2019). An additional consideration is that there are no specific ESM measures designed for Network Analysis, which makes it difficult for us to know their psychometric properties (Levinson et al., 2018). In addition to adapting ESM items following existing guidelines (Granholm et al., 2008; Myin-Germeys et al., 2018; Palmier-Claus et al., 2019), we believe that it would be extremely interesting to open a new line of research to validate ESM items, following a similar process to traditional assessment instrument validation. By doing so we would better capture the psychological constructs or phenomena that we want to analyse from NA.

On the other hand, the mlVAR model shows some challenges in relation to its application and interpretation of results. Power calculation for these models is not yet available (Greene et al., 2018) which would be necessary to estimate the sample size and number of observations needed to draw robust conclusions. While some simulation studies have suggested that using 50 momentary observations for an 8 element network would be adequate (i.e., an average of 6.25 observations per node; Oreel et al., 2019), the vast majority of the existing literature has followed the recommendations indicated by the ESM research (Kimhy et al., 2012; Myin-Germeys et al., 2018). It is clear that a large sample size with many evaluation points and fewer nodes would be the ideal situation

(Epskamp, Waldorp, et al., 2018; Levinson et al., 2018), but there is a lack of specific guidelines. In our study, although the number of observations per subject is satisfactory according to prior research (Aalbers et al., 2019; Greene et al., 2018), the sample size was less than ideal. Thus, we believe that future studies replicating our study are needed, with larger sample size and number of observations, as well as a broader representation of the paranoia as a continuum.

Another issue regarding mlVAR model is related to its assumptions and robustness. As discussed in Chapter 5, mlVAR must meet the assumptions of *normality* and *stationarity* (Aalbers et al., 2019; Bringmann et al., 2016), but there is currently a great deal of debate regarding whether or not these assumptions are necessary. Accepting normality is common in psychological sciences, but some authors have highlighted that assuming normally distributed parameters with network studies would mean that the individuals do not showed variability in their observations (Epskamp, Waldorp, et al., 2018). Similarly, it has been proposed that stationarity may not be plausible for network models. If our objective is to evaluate the dynamics, we would expect our data to vary over time (van Bork et al., 2018). Therefore, it is still unclear how robust time-series analysis is when these assumptions are violated, and results should be interpreted cautiously (van Bork, 2019).

Finally, we believe it is pivotal to stress that we have identified factors that could be *causally* relevant to paranoid beliefs, but we cannot extract claims of causality forcefully (Fried & Cramer, 2017; Jones et al., 2017). It might be easy to misinterpret findings from the temporal network model as causal interaction, but we believe this is not the case. The temporal network approach conducted in the current dissertation only satisfy one of the many criteria required to draw conclusions about causality, that is, the temporal requirement for Granger's causality (Aalbers et al., 2019; Epskamp, Waldorp, et al.,

2018). According to Granger causality, if one variable predicts another over time, we can conclude that one causes the other (Granger, 1969). Hence, we believe the obtained results are useful for generating hypotheses about potential temporal causality between processes, but it is not sufficient to draw robust conclusions about true causality.

In summary, this approach is at its infancy and much remains to be done to know with certainty what methodological criteria should be followed to carry out research with power and accuracy. We consider and encourage network researchers to design guidelines with methodological specifications on the number of observations, what to do if assumptions are violated, as well as to develop methods to assess the accuracy and stability of estimates with the VAR model which are currently only available for cross-sectional data (Aalbers et al., 2019; Groen et al., 2019; Johnson & Hoffart, 2018). This could guide the design of future longitudinal network studies and encourage more robust conclusions.

6.1.3. Clinical implications

First at all, findings from the present work underline the importance of studying paranoia from a transdiagnostic perspective. Identifying the specific processes that underpin the wide range of clinical presentations of paranoid thinking could effectively guide interventions, not only for people with psychotic disorders, but also in a broad range of people who may experience paranoid thinking (Bentall et al., 2009; Lincoln et al., 2013).

In addition, the current study provides evidence in favor of both current conceptualizations of paranoia, as a continuum (Freeman, 2016) and as a dynamic process that fluctuates over time (Erickson & Lysaker, 2012; Murphy et al., 2018; Palmier-Claus et al., 2011; Thewissen et al., 2008; Udachina et al., 2012). Therefore, the identification of the mechanisms that can affect the content of our thoughts on a daily basis provides

information that nourishes possible intervention targets to avoid their severity within the severity continuum. Specifically, our findings indicate the positive effect that social connection might have on people with paranoia vulnerability. Our results are in line with recent clinical research that highlights the importance of, not only reducing symptoms and/or functioning, but also expanding the range of potential therapeutic targets such as the different dimensions of well-being and quality of life (Seligman et al., 2006; Slade et al., 2016; Valiente et al., 2019). From this Positive Psychology perspective, positive social relationships have been suggested as a protective factor in paranoia (Slade, 2010; Slade et al., 2016). Similarly, this observation also aligns studies that have tried to understand paranoia from the attachment framework (Bentall & Sitko, 2020) and previous studies showing that paranoia can be triggered by interpersonal factors such as negative interpersonal schemata (Lincoln et al., 2010), momentary attachment insecurity (Sitko et al., 2016), perceived social exclusion (Westermann et al., 2012), and impoverished social network (Gayer-Anderson & Morgane, 2013). Consequently, based on our results, it can be argued that tailoring interventions that focus on interpersonal factors (such belonging, attachment or social support) can change the structure of the paranoid network and show beneficial consequences by deactivating the strength of this network and promoting pro-social behaviours that can activate well-being networks.

This intervention proposal must, however, be considered as a hypothesis. As we mentioned before, the current work identified *potential* relevant elements in the network structure, but no truly causality. To this end, we consider necessary studies that include the experimental manipulation of the identified variables to test network hypothesis (Fried & Cramer, 2017). To our knowledge, there are still no NA studies that have included the manipulation interventions of network elements with the aim of evaluating their impact on the remaining symptoms interrelationships. We consider this type of

studies of vital importance for the survival of this approach in psychopathology, as it may provide robust evidence on causality and benefits of intervening on the symptoms.

6.2. Conclusion

In the current context of increasing polarization between the common cause model and network analysis (Bringmann & Eronen, 2018), we could consider Network Analysis in psychopathology as a clinical research strategy that allows visualization and analysis of complex patterns of mental problems rather than as a new model to replace traditional approaches. However, the question of whether the network approach is here to stay remains unresolved, although its survival is surely conditioned by the extent to which it is able to incorporate current theoretical models and the quality of network datasets. The most promising contribution from this approach is that it allows us to think in dynamic ways of studying mental health and paranoid thinking in particular. In this thesis we hope to contribute to the advancement of knowledge about how the fluctuations and mechanisms involved can affect the development and maintenance of paranoid thinking. We also hope that the knowledge developed during this thesis will help to clarify the proliferation of studies on network psychopathology and emphasize the importance of building bridges between the work of clinicians, theorists, and network experts in order to build a solid and clinically useful NA methodology together.

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APPENDIX

Systematic review, data extraction

The data extraction was independently performed and double-checked by two authors (AC and IN). For the studies included, the following information was collected, as it can be seen in Tables A1-A3:

a) Sample characteristics: subsamples or total sample size, gender (percentage of males), type of participants (e.g. current or past clinical disorder) and age (mean and standard deviation).

b) Network elements and instruments: symptoms and/or relevant psychological variables that have been represented in the network as a node (e.g. symptoms of the disorder analysed) and instruments used to assess the outcome studied.

c) Type of data: cross-sectional or longitudinal data. Note that longitudinal data have been considered cross-sectional when authors only took measures at one data point for the network estimation.

d) Network analysis information provided:

-*Type of network estimated*. We can identify several types of networks. In an *Association Network* (AN), each edge represents weighted correlation (positive or negative) between symptoms while *Concentration Networks* (CN) use partial correlation (i.e. edges have been adjusted for the influence of all other symptoms). CNs can use cross-sectional and time-series data (Epskamp, Waldorp, et al., 2018). Although a large sample size is recommended to compute a network, regularisation techniques allow reliable estimations, leading to *Regularised Partial Correlation Networks* (RPCN) (Epskamp, Borsboom et al., 2018). In addition, some types of networks also reflect, not only the magnitude of the association, but also the directionality of it. For instance, the relative importance of symptoms as predictors of another symptom can be depicted with a

Relative Importance Network, which represents strength and direction of the prediction but not causation (McNally, 2016). *Bayesian Networks*, based on parametric methods that produce directed acyclic graphs (DAGs), identify the direction of the prediction and possible causation (McNally, 2016). Finally, *temporal networks* are also directed and estimated from time-series data. From now on, Relative Importance, Bayesian and Temporal networks will be referred as *Directed Networks* (DN) (Epskamp, Waldorp, et al., 2018).

-*Centrality analysis*. We assess whether any centrality metric was reported (i.e. strength, closeness and betweenness) (Fried et al., 2016; Fried et al., 2017).

-*Network parameters robustness*. We assess according to Fried et al. guidelines (Epskamp, Borsboom et al., 2018), whether robustness analyses of estimated data were done (i.e. measures of stability of centrality indices and/or accuracy of estimated edge-weight).

e) Availability of materials in the published paper: software code or syntax (e.g. R script) and/or data (i.e. correlation matrix or dataset).

Table A1. Main characteristics of network analysis studies in specific psychopathological conditions

Authors, year	Group(s), N (% male)	Mean age (SD)	Network symptoms or elements	Assessment of symptoms or elements	Type of data	Network analysis					Availability	
						AN	RPCN	DN	CA	Robustness analysis	Syntax	Data
Post-traumatic stress disorder												
McNally et al., 2015	Survivors of an earthquake, 362 (26.5)	44.8 (10.9)	PTSD symptoms	PCL-C	CS	√	√	√	√	N/A	N/A	N/A
Sullivan et al., 2016	Witnesses of a shooting event, 4639 (45.4)	21.8 (4.4)	PTSD symptoms	TSQ	CS	N/A	√	√	√	√	N/A	NA
Armour et al., 2017	Veterans with PTSD, 221 (86.7)	54.0 (14.8)	PTSD symptoms, depression, anxiety, suicidal ideation, quality of life	THS, PCL-5, PHQ-4, PHQ-9, SF-8	CS	N/A	√	N/A	√	√	√	√
Birkeland & Heir, 2017	Witness of a terrorist attack, 190 (N/A)	44.7 (11.9)	PTSD symptoms, social support, neuroticism	PCL-S, BFI (neuroticism), CSS (4 items)	CS	N/A	√	N/A	√	√	N/A	N/A
Bryant et al., 2017	Survivors of a traumatic injury from the AIVS, 1138 (73.6)	37.9 (13.6)	PTSD symptoms	CAPS	L	N/A	√	√	√	√	N/A	√
Glück et al., 2017	Survivors of institutional abuse, 220 (59.8)	57.9 (9.5)	PTSD symptoms, anger, aggression, rumination, traumatic events, shame	CTQ, DAQ, LEC-5, ITQ, STAXI	CS	√	√	N/A	√	√	N/A	N/A
Jayawickreme et al., 2017	Survivors of the Sri Lankan civil war, 337 (54.9)	43.41 (13.7)	Trauma exposure, war-related problems, stressful life events and psychopathology	PRPWPQ	CS	√	√	N/A	√	√	N/A	√
McNally et al., 2017	Adults who reported childhood sexual abuse, 179 (33.3)	41.2 (12.4)	PTSD symptoms	PCL-C	CS	N/A	√	√	√	√	√	√
Russell et al., 2017	Exposed to a natural disaster (49): 388 children, 388 adolescents	8-13 (N/A) 14-18 (N/A)	PTSD Symptoms	UCLA PTSD-RI	CS	N/A	√	N/A	√	N/A	N/A	N/A
Spiller et al., (2017)	Refugees with and without formal posttraumatic stress disorder, 151 (70)	41.9 (9.8)	Trauma exposure, PTSD symptoms	HTQ; DSM-IV, V (PCL, third part)	CS	N/A	√	N/A	√	√	√	N/A

Authors, year	Group(s), N (% male)	Mean age (SD)	Network symptoms or elements	Assessment of symptoms or elements	Type of data	Network analysis					Availability	
						AN	RPCN	DN	CA	Robustness analysis	Syntax	Data
Anxiety-related disorders												
Heeren et al., 2016	Social anxiety disorder, 61 (19.6)	25.9 (9.1)	Social anxiety, depression, attention bias, social reactivity	LSAS, STAI, BDI, ANT, LM, SUDS, BASA	CS	√	√	√	√	N/A	N/A	N/A
Tsuruta et al., 2017	General population, 1360 (0)	19.6 (1.1)	Social anxiety, preoccupations with body part odor, subjective halitosis, olfactory ideas of reference	Ad hoc scale for social anxiety (7 items), halitosis (10 items) and olfactory ideas (7 items)	CS	N/A	N/A	√	N/A	N/A	N/A	N/A
Vehling et al. 2017	Patients with advanced cancer, 382 (40.3)	58.7 (11.4)	Death anxiety	DADDS	CS	N/A	√	N/A	√	N/A	N/A	√
Mood disorders												
Cramer et al., 2012	Depressive symptoms, from VATSPUD study, precipitated by a SLE: Stress 710 (57.46); Loss 528 (49.62); Health 371 (44.47); Conflict 487 (43.94)	N/A	Depression, stressful life events	VATSPUD interview, LEDS	CS	√	N/A	N/A	√	N/A	N/A	√
Bringmann et al., 2013	Residual depressive symptoms from a RCT: MT, 63 (N/A) CG, 66 (N/A)	 44.6 (9.7) 43.2 (9.5)	Positive and negative affect, neuroticism	ESM, NEO- FFI	L	N/A	N/A	√	√	N/A	√	√
Robinaugh et al., 2014	Loss of a spouse, from the CLOC, 265 (14.7)	70.2 (6.9)	Persistent complex Bereavement Disorder, depression, risk factors	DSM-5 (13 items) CES-D, ITDS (9 items), NEO-FFI (11 items)	L	√	N/A	√	√	N/A	N/A	N/A
Bringmann et al., 2015	MDD from a RCT: 99 CT (20); 83 IT (36)	 40 (12) 41 (12)	Depressive symptoms	BDI-II	L	N/A	N/A	√	√	N/A	N/A	N/A
Fried et al., 2015	With and without loss of spousal, 515 (14.6)	73.3 (6.5)	Depression, spousal loss	CES-D (11-item)	CS	N/A	√	N/A	N/A	N/A	N/A	N/A

Authors, year	Group(s), N (% male)	Mean age (SD)	Network symptoms or elements	Assessment of symptoms or elements	Type of data	Network analysis					Availability	
						AN	RPCN	DN	CA	Robustness analysis	Syntax	Data
Koenders et al., 2015	Bipolar disorder, mildly impaired 47 (42.6); predominantly depressed 42 (40.5); cycling 36 (36.1)	50.6 (11.2); 53.1 (10.8); 51.9 (11.7); 45.8(10)	Bipolar disorder, depression, mania	YMRS, QIDS-SR, LCM-R	L	√	N/A	N/A	√	N/A	N/A	√
Pe et al., 2015	MDD, 53 (28.3); CG, 53 (32.1)	25.4 (6.4); 28.2 (6.4)	Negative affect	ESM	L	N/A	N/A	√	N/A	N/A	N/A	N/A
van Borkulo et al., 2015	MDD from NESDA: 262 in remission (N/A) and 253 persistent (N/A)	40.9 (12.1)	Depressive symptoms	IDS	CS	N/A	√	N/A	√	√	N/A	N/A
Boschloo, van Borkulo et al., 2016a	Individuals with no lifetime depressive or anxiety disorder from the NESDA, 501 (N/A)	N/A	Depressive symptoms	IDS (12 items)	CS	N/A	√	N/A	√	N/A	N/A	N/A
Fried et al., 2016	Single or recurrent nonpsychotic MDD from the STAR*D, 3463 (37)	41 (13)	Depressive symptoms	IDS-C	CS	N/A	√	N/A	√	√	N/A	N/A
Hoorelbeke et al., 2016	MDD history and being in (partial) remission for at least six months, 69 (33.3)	47.1 (11.4)	Depression, cognitive control, emotional regulation, resilience	INQUISIT, RS, BRIEF-WM, RDQCERQ, PASAT	CS	√	√	√	√	N/A	N/A	√
Madhoo et. al., 2016	Nonpsychotic MDD from the STAR*D: baseline, 2862 (N/A); endpoint, 2585 (N/A); change, 2578 (N/A)	18-75 (N/A)	Depressive symptoms	QIDS-SR	CS	N/A	√	N/A	√	N/A	N/A	N/A
Watters et al., 2016	Psychiatric out and inpatient, community, medical outpatients and healthy participants 842 (36.6)	38.6 (13.1)	Alexithymia	TSIA	CS	√	√	N/A	√	√	N/A	N/A
Wichers et al., 2016	History of multiple episodes of MD, 1 male	57 (N/A)	Mental unrest, cognitive context, negative and positive affect	ESM	L	N/A	N/A	√	N/A	N/A	N/A	N/A

Authors, year	Group(s), N (% male)	Mean age (SD)	Network symptoms or elements	Assessment of symptoms or elements	Type of data	Network analysis					Availability	
						AN	RPCN	DN	CA	Robustness analysis	Syntax	Data
De Beurs et al., 2017	Suicide attempters: repeaters at 15-month follow-up 94 (46.80); non-repeaters 272 (41.91)	35 (13.6)	Suicide symptoms	SSI	CS	√	√	N/A	√	N/A	N/A	N/A
Maccallum et al., 2017	Loss of a spouse 193 (33.5); Loss a parent, 180 (55)	57.04 (6.58); 42.35(10.94)	Grief, depression	PG-13 CES-D	CS	N/A	√	N/A	√	√	N/A	N/A
Dejonckheere et al., 2017	High scores in depression, 112 (52)	34.27 (9.7)	Social expectancies, depressive symptoms	SEDAS DSM-5	L	N/A	N/A	√	√	N/A	N/A	N/A
Santos et al. 2017	Women at the second trimester of pregnancy: 245 with symptoms of depression, 270 with no depressive symptoms	24.6 (5.8)	Depression, stress, reproductive biomarkers	CES-D-20, blood sample	CS	N/A	√	N/A	√	√	√	√
McWilliams et al., 2017	MDD with chronic pain, 225 (38)	47.06 (N/A)	Depressive symptoms	PHQ-9	CS	N/A	√	N/A	√	N/A	N/A	N/A
Psychosis-related conditions												
Bak et al., 2016	Paranoid schizophrenia, 1 female	46 (N/A)	Schizophrenia, positive and negative affect	ESM	L	N/A	N/A	√	√	N/A	N/A	N/A
Isvoranu et al., 2016	General population, 3012 (N/A)	14-24 (N/A)	Anxiety, depression, somatization, OCD, phobia, hostility, risk factors, psychosis	N/A	CS	√	N/A	N/A	N/A	N/A	N/A	N/A
Levine et al., 2016	Chronic schizophrenia, 437 (65.3)	34.04 (9.4)	Negative symptoms	SANS	L	N/A	√	N/A	√	N/A	N/A	N/A
Esfahlani et al., 2017	Psychotic disorders from CATIE: 733 TV (N/A), 316 TR (N/A)	N/A	Positive and negative syndrome, general psychopathology	PANSS	L	√	N/A	N/A	√	N/A	N/A	N/A
Isvoranu et al., 2017	Participants with psychotic disorder from GROUP, 552 (75.7)	30.8 (7.2)	Psychosis, childhood trauma	PANSS, CTQ-SF	CS	N/A	√	N/A	√	N/A	N/A	N/A

Authors, year	Group(s), N (% male)	Mean age (SD)	Network symptoms or elements	Assessment of symptoms or elements	Type of data	Network analysis					Availability	
						AN	RPCN	DN	CA	Robustness analysis	Syntax	Data
van Rooijen et al., 2017	Non-affective psychotic disorder from GROUP, 408 (100)	27.4 (7,5)	Manic and depressive syndrome, delusions, avolition, alogia hallucinations, formal thought, inattention, catatonic and affective symptoms	CASH	CS	N/A	√	N/A	√	√	N/A	N/A
Wigman et al., 2017	Individuals with and without hallucinations, 293 (41)	18.9 (0.4)	Verbal and auditory hallucinations, anxiety, depression, stress	CAPE, DASS-21	CS	N/A	√	N/A	√	N/A	N/A	N/A
Personality disorders												
Richetin et al., 2017	Healthy students, 1317 (25.9); Psychiatric and/or personality disorder, 96 (39.5)	22.56 (4.05) 37.75 (10.5)	Borderline personality symptoms	BPDCL	CS	N/A	√	N/A	√	N/A	N/A	√
Substance abuse												
Rhemtulla et al., 2016	Frequent life-time substance abusers (from the VATSPSUD), 2405 (65)	34.7 (7.3)	Substance Abuse	SCID	CS	N/A	√	N/A	√	N/A	N/A	N/A

Note. SD=Standard Deviation; **Network analysis initials**: AN=Association Network; CA=Centrality Analysis; CS = Cross-Sectional; Data=sharing database; DN=Directed Network; L=Longitudinal; N/A=Not Applicable; Robustness analysis=stability and/or accuracy; RPCN=Regularized Partial Correlation Network; Syntax=sharing analysis codes**. Rest of initials:** AIVS=Australian Injury Vulnerability Study; ANT=Attentional Network Task; BASA=Behavioral Assessment of Speech Anxiety; BDI=Beck Depression Inventory; BFI=Big Five Inventory; BPDCL=Borderline Personality Disorder Checklist; BRIEF-WM=Behavior Rating Inventory of Executive Function Adult version; CAPE=Community Assessment of Psychic Experiences; CAPS=Clinician Administered PTSD Scale; CASH=Comprehensive Assessment of Symptoms and History; CATIE=Clinical Antipsychotic Trials of Intervention Effectiveness study; CES-D=Center for Epidemiologic Studies Depression scale; CERQ=Cognitive Emotion Regulation Questionnaire; CG=Control Group; CLOC= Changing Lives of Older Couples study; CSS= Crisis Support Scale; CT=Cognitive Therapy; CTQ/SF=Childhood Trauma Questionnaire/ Short Form; DADDS=Death and Dying Distress Scale; DAQ=Displaced Aggression Questionnaire; DASS-21=Depression, Anxiety and Stress Scale; DSM-IV/V=Diagnostic and Statistical Manual of Mental Disorder (fourth/fifth edition); EPSI=Eating Pathology Symptoms Inventory; ESM=Experience Sample Methodology; HTQ=Harvard Trauma Questionnaire; IDS/SR/C=Inventory of Depressive Symptomatology/ Self Report version/ Clinician version; INQUISIT=Cognitive computer task (Millisecond software package); IT=Interpersonal Therapy; ITDS=Interpersonal Dependency Sale; ITQ=International Trauma Questionnaire; LCM-R=Retrospective Life Chart Method; LEC-5=Life Events Checklist for DSM-5; LEDS=Life Events and Difficulties Measure; LM=Laboratory Measures (of attention bias); LSAS=Liebowitz social Anxiety

Scale; NEO-FFI=Big Five Personality Questionnaire; NESDA=The Netherlands Study of Depression and Anxiety; PASAT=Paced Auditory Serial Addition Task; PTSD=Posttraumatic Stress Disorder; MDD=Major Depressive Disorder; MT=Mindfulness Therapy; THS=Trauma History Screen; TSIA=Toronto Structured Interview for Alexithymia; TR=Treatment Resistant; TSQ=Trauma Screening Questionnaire; TV=Treatment Responsive; OCD=Obsessive Compulsive Disorder; PANSS=Positive and Negative Syndrome Scale; PCL-C=Posttraumatic Checklist-Civilian (Mandarin Chinese version); PCL-5=Posttraumatic Stress Disorder Checklist from DSM-5; PG-13=Prolonged Grief-13; PHQ-4=Patient Health Questionnaire-4; PHQ-9=Patient Health Questionnaire-9; PRPWPQ=Penn/RESIST/Peradeniya War Problems Questionnaire; QIDS/SR=Quick Inventory of Depressive Symptomatology/ Self-Report); RCT=Randomised Control Trial; RDQ=Remission of Depression Questionnaire; RS=Resilience Scale; SANS=Scale for the Assessment of Negative Symptoms; SCID=Structured Clinical Interview for DSM; SEDAS: Social expectancies about Depression and Anxiety Scale; SF-8=Short Form-8 Health Survey; SLE=Stressful Life Events; SSI: Beck Scale for Suicide Ideation; STAI=State Trait Anxiety Inventory; STAR*D=Sequenced Treatment Alternatives to Relieve Depression study; STAXI=State-Trait Anger Expression Inventory; SUDS=Subjective Units of Discomfort Scale; UCLA PTSD-RI=University of California, Los Angeles Posttraumatic Stress Reaction Index for Children; VATSPUD=Virginia Adult Twin Study of Psychiatric and Substance Use Disorder; YMRS=Young Mania Rating Scale.

Table A2. Main characteristics of studies with NA and psychopathology or comorbidity

Authors, Year	Group(s), N (% male)	Mean age (SD)	Network symptoms or elements	Assessment of symptoms or elements	Type of data	Network analysis					Availability	
						AN	RPCN	DN	CA	Robustness analysis	Syntax	Data
Goekoop et al., 2014	Individuals with acute mental disorders, 192 (39)	54.3 (6.1)	Psychopathology	CPRS	CS	√	N/A	N/A	√	N/A	N/A	√
Boschloo et al., 2015	General population from the NESARC, 34.653 (42)	49.1 (17.3)	Major depressive episode, dysthymia, (hypo)mania, GAD, social specific phobia, panic, agoraphobia, PTSD, ADHD and substance disorders	AUDADIS-IV	CS	N/A	√	N/A	N/A	N/A	N/A	√
Wigman et al., 2015	Past diagnosis and current mild depression, 129 (24); psychotic disorder, 263 (68); CG, 207 (48)	44.1 (9.3); 35.5 (11.0); 34.3 (11.6)	Negative and positive affect, psychosis	ESM	L	N/A	N/A	√	√	N/A	N/A	√
Beard et al., 2016	Mood, anxiety, personality and psychotic disorders, 1029 (48)	35 (13.8)	MDD, GAD	MINI, PHQ-9, GAD-7	L	N/A	√	N/A	√	√	N/A	√
Bekhuis et al., 2016	918 prior history of depressive and/or anxiety disorder (N/A); 1441 with past diagnosis (N/A);	41.7 (13.1)	GAD, MDD, Somatisation	IDS-SR, 4DSQ	CS	N/A	√	N/A	N/A	N/A	N/A	√
Bringmann et al., 2016	General population students from two datasets: 95 (38); 79 (37)	19 (1); 24 (8)	Positive and negative affect, neuroticism	ESM, NEO-FFI	L	N/A	N/A	√	√	N/A	N/A	N/A
Curtiss et al., 2016	GAD, 70 (27); MDD, 41 (39)	35.1 (12.3); 33.2 (14.4)	MDD, GAD	BDI-II, STAI-T, CSR	CS	N/A	√	N/A	√	N/A	N/A	N/A
Guloksuz et al., 2016	EDSP, 3021 (50.7); NAMESIS-1, 7076 (46.6)	18.26 (3.3) 41.16 (12.1)	Psychopathology, environment exposure	SCL-90-R, CIDI (1 item)	L	N/A	N/A	√	N/A	N/A	N/A	N/A
Knefel et al., 2016	(Complex) PTSD, borderline personality disorder, MDD, GAD, and alcohol-related disorders, 219 (59.8)	57.9 (9.5)	PTSD, borderline personality disorder	ICD-TQ, CTQ/SF, LEC-5, SCID	CS	N/A	√	N/A	√	√	N/A	N/A

Authors, Year	Group(s), N (% male)	Mean age (SD)	Network symptoms or elements	Assessment of symptoms or elements	Type of data	Network analysis					Availability	
						AN	RPCN	DN	CA	Robustness analysis	Syntax	Data
Jaya et al., 2017	General population from MTurk: time 1, 289 (32.5); time 2, 155 (53.5); time 3, 151 (58.9); time 4, 171 (42.7)	 37.6 (12.7) 36.5 (11.6) 34.07 (11.3) 37.06 (12.6)	Psychosis, MDD, loneliness	R-UCLA (3 items), CES-D, subscale of CAPE	CS	N/A	√	N/A	√	N/A	N/A	N/A
Afzali et al., 2017	PTSD and MDD, 909 (28)	43.8 (15.1)	PTSD, MDD	WMH CIDI	CS	N/A	√	N/A	√	N/A	N/A	N/A
Anker et al., 2017	Alcohol dependence and anxiety disorder, 362 (62)	39.3 (10.2)	GAD, depression, social anxiety, panic, agoraphobia, perceived stress, drinking to cope, coping self-efficacy, alcohol craving and drinking behaviour	PSWQ, BDI SPS, PDSS, MIA, OCDS, TLFBI PSS, IDS-100, SCQ	CS	N/A	√	N/A	√	√	N/A	N/A
Bernstein et al., 2017	General population, 91 (43)	23.48 (4.5)	Rumination and executive control	N-back task, emotional flanker task, internal shift task, stressor task, 5 state rumination questions	L	N/A	√	√	√	√	N/A	√
Choi, K. et al., 2017	HIV negative, 296 (100)	38.0 (11.7)	PTSD, depression and sexual risk behaviour	CES-D, DTS, self-report of sexual risk behaviour	CS	N/A	√	N/A	N/A	√	N/A	N/A
de Vos et al., 2017	MDD, 27 (26); CG, 27 (26)	34.7 (9.9); 34.0(9.0)	Positive and negative affect	14 self-report items of positive and negative affect	L	N/A	N/A	√	√	N/A	N/A	N/A
Fisher et al., 2017	MDD, GAD or both 40 (35)	18-60 (N/A)	MDD, GAD, positive and negative affect, rumination, behavioural avoidance and reassurance seeking	HARS, HRSD, ESS	L	N/A	√	√	√	N/A	N/A	N/A
McNally et al., 2017	OCD and MDD, depression or dysthymia, 408 (47.3)	31.1 (12.2)	OCD, MDD	Y-BOCS-SR, QIDS-S-SR	CS	N/A	√	√	√	√	√	√

Authors, Year	Group(s), N (% male)	Mean age (SD)	Network symptoms or elements	Assessment of symptoms or elements	Type of data	Network analysis					Availability	
						AN	RPCN	DN	CA	Robustness analysis	Syntax	Data
Pereira-Morales et al., 2017	General population, 334 (25.4)	21.7 (4.8)	Psychological distress, sleep problems, alcohol use, perceived self-efficacy/coping, childhood trauma, suicidal ideation and personality traits	PHQ-9; HADS-D; PSS; item 9 of the OSQ; AUDIT; CTQ-brief version; BFI-S	CS	N/A	√	N/A	√	√	N/A	N/A

Note. SD=Standard Deviation; **Network analysis initials**: AN=Association Network; CA=Centrality Analysis; CS=Cross-Sectional; Data=sharing database; DN=Directed Network; L=Longitudinal; N/A=Not Applicable; Robustness analysis=stability and/or accuracy; RPCN=Regularised Partial Correlation Network; Syntax=sharing analysis codes**. Rest of initials:** ADHD=Attention Deficit/Hyperactivity Disorder; AUDADIS-IV=The Alcohol Use disorder and Associated Disabilities Interview Schedule; AUDIT=Alcohol Use Disorders Identification Test; BDI=Beck Depression Inventory; BFI-S=Big Five Inventory; CAPE=Community Assessment of Psychic Experiences; CES-D=Center for Epidemiologic Studies Depression Scale; CG=Control Group; CIDI=Composite International Diagnostic Interview; CPRS=Comprehensive Psychopathological Rating Scale; CSR=Clinical Severity Rating; CTQ=Childhood Trauma Questionnaire; CTQ/SF=Childhood Trauma Questionnaire/Short Form; DTS=Davidson Trauma Scale; EDSP=the Early Developmental Stages of the Psychopathology study; ESM=Experience Sample Methodology; ESS=Experience Sample Survey; GAD=Generalised Anxiety Disorder; GAD-7=Generalised Anxiety Disorder Scale; HADS-D=Hospital Anxiety and Depression Scale; HARS=Hamilton Anxiety Rating Scale; HRSD=Hamilton Rating Scale for Depression; ICD-TQ=International Classification of Disorders Trauma Questionnaire; IDS-SR=Inventory of Depressive Symptomatology/Self Report version; IDS-100=Inventory of Drinking Situations; LEC-5=Life Events Checklist for DSM-5; MDD=Major Depressive Disorder; MIA=Mobility Inventory for Agoraphobia; MINI=Mini-International Neuropsychiatric Interview; MTurk=the Amazon´s Mechanical Turk;NAMESIS-1=the Netherlands Mental Health Survey and Incidence Study; NEO-FFI=Big Five Personality Questionnaire; NESARC=The National Epidemiologic Survey on Alcohol and Related Conditions study, OCD=Obsessive compulsive disorder; OCDS=Obsessive Compulsive Drinking Scale; OSQ=Oviedo Sleep Questionnaire; PDSS=Panic Disorder Severity Scale; PHQ-9=Patient Health Questionnaire-9; PSS=Perceived Stress Scale; PSWQ=Penn State Worry Questionnaire; PTSD=Posttraumatic Stress Disorder; QIDS/SR=Quick Inventory of Depressive Symptomatology/ Self-Report; R-UCLA=Loneliness scale;; SCID=Structured Clinical Interview for DSM; SCL-90-R=Symptom Checklist-90-Revised; SCQ=Situational Confidence Questionnaire; SPS=Social Phobia Scale; STAI=State Trait Anxiety Inventory; TLFBI=Time Line Follow-Back Interview; WMH CIDI=World Mental Health Composite International Diagnostic Interview; Y-BOCS-SR=Yale Brown Obsessive Compulsive Scale Self Report; 4DSQ=Four Dimensional Symptom Questionnaire.

Table A3. Main characteristics of network analysis studies in childhood and adolescent related disorders

Authors, year	Group(s), N (% male)	Mean age (SD)	Network symptoms or elements	Assessment of symptoms or elements	Type of data	Network analysis					Availability	
						AN	RPCN	DN	CA	Robustness analysis	Syntax	Data
Anderson et al., 2015	PDD, 301 (82.4); CG, 176 (68.8)	114.8 (49.6); 127.9 (43.1)	Autism	ADOS	CS	N/A	√	N/A	√	N/A	N/A	N/A
Ruzzano et al., 2015	Autism, OCD, ADHD, Tourette syndrome, ODD, conduct disorder, 213 (86)	9.2 (1.9)	Autism, OCD	ADI-R, PCR-S P-DISC-IV	CS	√	N/A	√	√	N/A	N/A	N/A
Boschloo, Schoevers et al., 2016	Community sample of preadolescents, 2175 (49.1)	11.1 (0.55)	Internalising and externalising behaviour, attention, thought and social problems	YSR	CS	N/A	√	N/A	N/A	N/A	N/A	√
Martel et al., 2016	Preschoolers, 109 (64): ADHD, 61, CG, 48; Children, 548 (59): ADHD, 302, CG, 246; Adolescents, 357 (59): ADHD, 144, CG, 213; Adults, 406 (49): ADHD, 145, CG, 261	3-6 (N/A) 6-12 (N/A) 13-17 (N/A) 18-37 (N/A)	ADHD	ADHD-RS	CS	√	N/A	N/A	√	N/A	N/A	N/A
Hasmi et al., 2017	Population-based twin pairs and siblings, 704 (40)	17.6 (3.7)	Positive and negative affect	ESM	L	N/A	N/A	√	√	N/A	N/A	N/A
Smith et al., 2017	Preschoolers with: ADHD, 18 (N/A); ODD, 18 (N/A); both, 43 (N/A); CG, 30 (N/A)	4.34 (1.08)	ODD, ADHD	DBRS, K-DBDS	CS	√	N/A	N/A	√	N/A	N/A	N/A

Note. SD=Standard Deviation; **Network analysis initials**: AN=Association Network; CA=Centrality Analysis; CS=Cross-Sectional; Data=sharing database; DN=Directed Network; L=Longitudinal; N/A=Not Applicable; Robustness analysis=stability and/or accuracy; RPCN=Regularised Partial Correlation Network; Syntax=sharing analysis codes**.** **Rest of initials:** ADHD=Attention Deficit Hyperactivity Disorder; ADHD-RS=Attention Deficit Hyperactivity Disorder Rating Scale; ADI-R=Autism Diagnostic Interview-Revised; ADOS=Autism Diagnostic Observation Schedule; DBRS=Disruptive Behavior Rating Scale; ESM=Experience Sampling Method; HCG=Healthy comparison group; K-DBDS=Kiddie Disruptive Behavior Disorders Schedule; OCD=Obsessive Compulsive Disorder; ODD=Oppositional Defiant Disorder; PCR-S=Perceived Causal Relationships Scale; PDD=Pervasive Developmental Disorder; P-DISC-IV= Diagnostic Interview Schedule for Children for DSM-IV, parent version's=Youth Self-Report.

Ethical approval from University Complutense of Madrid board

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A quien corresponda,

Leído el proyecto de investigación presentado por la **Dra. Carmen Valiente Ots**, con el título "EVALUACIÓN Y TRATAMIENTO DEL SESGO AUTO-SIRVIENTE EN LA PARANOIA: WELLFOCUS-PPT Y NUEVAS TECNOLOGÍAS" la Comisión Deontológica de la Facultad de Psicología de la UCM emite **informe favorable** sobre los aspectos éticos relacionados con el estudio, toda vez que la Investigadora Responsable se compromete a respetar las normas deontológicas del Colegio Oficial de Psicólogos y las Sociedades Científicas de Psicología durante el desarrollo del proyecto. En particular, la Investigadora Responsable se compromete a obtener una autorización firmada de cada uno de los participantes, previa información escrita y comprensible sobre (i) los objetivos y del procedimiento al que van a someterse, (ii) el carácter voluntario de su participación y (iii) su derecho a abandonar el estudio en cualquier etapa del mismo si así lo desean. La Investigadora Responsable también se compromete expresamente a respetar el carácter confidencial de la información obtenida y a custodiarla conforme a la legislación vigente.

Madrid, a 13 de octubre de 2016

Fdo. Rocío Alcalá Quintana
Presidenta de la Comisión Deontológica de la Facultad de Psicología
Vicedecana de Investigación, Infraestructuras y Doctorado

FACULTAD DE PSICOLOGIA
DECANATO

Table A4. Differences test for demographic, clinical and ESM characteristics between included and excluded participants

	Included (*n*=23)	Excluded (*n*= 41)	*t/x²*	*p*
Demographic characteristics				
Age in years, mean (SD)	23.78 (6.17)	23.27 (4.29)	0.359	0.69
Sex: Women, n (%)	19 (82.6)	34 (82.9)	0.001	0.97
Single status, n (%)	22 (95.7)	40 (97.5)	0.17	0.67
Education, n (%)			0.43	0.51
Secondary School	3 (13)	8 (19.5)		
Post-secondary	20 (87)	33 (80.5)		
Employed, n (%)			0.06	0.97
Unemployed	15 (65.2)	26 (63.4)		
Part-time employment	5 (21.7)	10 (24.4)		
Full-time employment	3 (13)	5 (12.2)		
Clinical characteristics, mean (SD)				
SCL-90-R paranoid ideation,	1.24 (0.94)	1.17 (0.85)	0.31	0.75
SCL-90-R interpersonal susceptibility	1.57 (0.88)	1.66 (0.80)	-0.41	0.67
SCL-90-R anxiety	1.10 (0.47)	1.27 (0.70)	-1.11	0.27
SCL-90-R depression	1.93 (0.76)	1.93 (0.73)	-0.15	0.88
ESM observations, mean (SD)				
Number of completed observations	28.48 (6.58)	8.39 (6.45)	11.86	0.00***
Number of missing beeps	30.83 (13.90)	48.79 (19.16)	-3.93	0.00***
Number of missing data	10.70 (12.95)	12.71 (17.33)	-4.85	0.62

Note. SCL-90-R=Symptom Checklist-90-R (Cronbach's α=0.79-0.90); *SD*=Standard Deviation; **p*-value<0.05; ** *p*-value<0.01; *** *p*-value<0.001; Number of missing beeps=the final amount of missing notifications due to technical problems; Number of missing data=notifications that participants did not response.

Table A5. ESM Spanish and English items

Negative affect: -"*En este momento me siento triste"* ["At this moment, I feel sad"]
Clossenes to others: -*"En este momento, me siento cercano a los demás"* ["At this moment, I feel close to others"]
Experiential Avoidance: -*"Desde el último pitido, he tratado de quitarme de la cabeza pensamientos y sentimientos negativos"* ["Since the last "beep", I have tried to avoid negative thoughts and feelings"]
Paranoid beliefs: -*"Desde el último pitido, he tenido la sensación de que no se puede confiar en la gente"* ["Since the last "beep", I have had the impression that I cannot trust people"] -*"Desde el último pitido, he tenido la sensación de que la gente ha intentado fastidiarme"* ["Since the last "beep", I have had the impression that people have tried to harm me"] -*"Desde el último pitido, he tenido la sensación de que la gente me ha criticado"* ["Since the last "beep", I have had the impression that people have criticized me"]
Self-esteem: -*"En este momento, me siento útil"* ["At this moment, I feel useless"] -*"En este momento, siento que me enfrento bien a los problemas"* ["At this moment, I feel I can manage issues well"]

Correlation matrix of the data

a) Contemporaneous network model

	Sad	Others	EA	SE	Paranoia
Sad	0.0000000	-0.2505747	0	-0.3127817	0.1627861
Others	-0.2505747	0.0000000	0	0.1910085	0.0000000
EA	0.0000000	0.0000000	0	0.0000000	0.0000000
SE	-0.3127817	0.1910085	0	0.0000000	0.0000000
Paranoia	0.1627861	0.0000000	0	0.0000000	0.0000000

b) Temporal network model

	Sad	Others	EA	SE	Paranoia
Sad	0.3151423	0.0000000	0.1074770	0.0000000	0.00000000
Others	0.0000000	0.1591497	0.0000000	0.0000000	-0.0961123
EA	0.0000000	0.0000000	0.2215667	0.1190074	0.00000000
SE	0.0000000	0.0000000	0.0000000	0.1389711	0.00000000
Paranoia	0.0000000	0.0000000	0.0000000	0.0000000	0.16652858

c) Between-subject network model

	Sad	Others	EA	SE	Paranoia
Sad	0.0000000	0.0000000	0.6050973	-0.5232103	0.0000000
Others	0.0000000	0.0000000	0.0000000	0.6434132	-0.5412957
EA	0.6050973	0.0000000	0.0000000	0.0000000	0.0000000
SE	-0.5232103	0.6434132	0.0000000	0.0000000	0.0000000
Paranoia	0.0000000	-0.5412957	0.0000000	0.0000000	0.0000000

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