

Using Network Models in Person-Centered Care in Psychiatry: How Perspectivism Could Help To Draw Boundaries

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- 17 Abstract
- 18 In this paper, we explore the conceptual problems arising when using network analysis in person-
- 19 centered care (PCC) in psychiatry. Personalized network models are potentially helpful tools for PCC,
- 20 but we argue that using them in psychiatric practice raises boundary problems, i.e., problems in
- 21 demarcating what should and should not be included in the model, which may limit their ability to
- 22 provide clinically-relevant knowledge. Models can have explanatory and representational boundaries,
- 23 among others. We argue that we can make more explicit what kind of questions personalized network
- 24 models can address in PCC, given their representational and explanatory boundaries, using perspectival
- 25 reasoning.

26 1 Introduction

Mental disorders often dominate the lives of people who experience them¹. It stands to reason that to 27 28 understand these conditions, it is crucial to not only focus on symptoms, but also to recognize and 29 examine an individual's personal experience and situational context. For instance, an individual's 30 experience may be influenced by *biological* factors, such as fighting an infection, being malnourished 31 or one's microbiome (Allen et al., 2017); social factors such as unemployment and lack of social 32 support, and *psychological* factors such as their personality type and resilience. These personal, contextual factors could influence what symptoms someone develops and how they experience their 33 34 condition². 35 Despite the recognition that personal and contextual factors play an important role in psychopathology, clinical research has increasingly moved away from focusing on these types of factors. A prime 36 37 example of this is the impressive proliferation of neuroscientific research in the last three decades, that 38 has given neurobiological factors a privileged explanatory status in psychopathology. As a result, today 39 it is not uncommon to hear phrases such as "you are your brain" or to encounter headlines like "the 40 [adjective] brain", where the brackets are filled in with categories like "female/male", "teenage", 41 "addicted", "hyper-active" and so on. This trend is known as neuroessentialism: the idea that denotes 42 the brain as the essence of a person, with the brain being synonymous with concepts like the 'self' 43 (O'Connor et al., 2012). The former director of the National Institute of Mental Health (NIMH), 44 Thomas Insel, even claimed that mental disorders are no more than brain disorders (Insel and Cuthbert, 45 2015). Evidently, the brain is a fundamental organ for the mind, which among other things is reflected 46 by the fact that brain damage is associated with impoverished perceptual and cognitive abilities. 47 However, equating mental disorders to brain dysfunction neglects these other personal, contextual 48 factors that play an important role in understanding psychopathology. Moreover, it has been argued

¹ Throughout this article, we will use the term 'patient' ('the one who suffers') to refer to people who seek therapy for their mental health problems. We are aware that the use of this term is contested by some who have been given a mental health diagnosis. For instance, some argue that the term 'client' better reflects their experiences. However, each term comes with its own advantages and disadvantages, and the term patient is most suited for the setting that we want to address (i.e., psychiatric, clinical practice). Similarly, we will use 'mental disorder' to refer to the mental health problems that people experience and are treated for in clinical practice, whilst acknowledging that not everyone who has been diagnosed will resonate with this term.

² The sociocultural and historical context in which an individual operates also plays an important role in the diagnosis and treatment of mental disorders. Among others, it influences what is considered pathological. To illustrate, homosexuality was considered a mental disorder by the Diagnostic and Statistical Manual of Mental Disorders (DSM) until 1973. Fortunately, homosexuality is not in the DSM anymore, but it is likely that the disease classification will have influenced people's conception of their homosexuality in the past. However, quantifying sociocultural and historical influences in scientific models is far from straightforward. Hence, these factors will not be discussed explicitly in the remainder of the article (we would like to thank one of the reviewers for putting this point forward).

that our theories and models are heuristic strategies meant to describe phenomena, facilitate manipulation and future predictions, and ultimately, make a phenomenon intelligible (de Regt, 2015; Glas, 2019). Neuroessentialism, as a theory of psychopathology, ignores the web of relationships between an individual and their context, relationships that co-determine their identity. By ignoring these aspects of psychopathology, the neuroessentialist approach may be obscuring the phenomenon that is a mental disorder instead of making it more intelligible to clinicians, patients, and researchers.

55 Accompanying this development, we have seen a decreased emphasis on the subjective aspects of 56 mental disorders. For instance, neuroessentialism further implies that neuroscientific data alone 57 provides an exhaustive insight into the objective, truer core of psychopathology, while personal 58 experience is merely a subjective reflection of this fundamental biological core. Hence, according to 59 this view, knowledge about the pathogenesis of disease belongs to the objective core, whereas values, 60 patient interests, and clinical intuitions belong to the soft margins surrounding that core. The separation 61 between objective and subjective aspects of being ill is also related to the birth of evidence-based 62 medicine (EBM). EBM emerged as a new paradigm for clinical care in medicine and psychiatry. It 63 states that psychiatrists should conscientiously, explicitly, and judiciously use the current best scientific 64 evidence in making decisions for patient care (Sackett et al., 1996, 71). EBM created a hierarchy of 65 evidence where meta-analyses of randomized clinical trials were at the top, while clinical intuition and 66 personal experience were placed at the bottom. However, both neuroessentialism and EBM are 67 inadequate for diagnosing and treatment of mental health problems, chiefly because these approaches 68 neglect the personal and contextual factors that play an equally important role in a mental disorder.

As a reaction to these methodological and conceptual shortcomings of neuroessentialism and EBM, person-centered care (PCC) arose as a guiding vision on how to diagnose and treat an individual. PCC has traditionally been used in nursing, especially in geriatrics (Morgan and Yoder, 2012). Its aim is to respectfully care for an individual considering their preferences, needs, and values, and ensuring that these aspects guide all clinical decisions (Morgan and Yoder, 2012; Håkansson Eklund et al., 2019). In this way, the alliance between a therapist and a patient is emphasized. Mezzich (2011, 335) gives the following definition of person-centered medicine (PCM), which we think applies well to PCC:

"PCM is the medicine *of* the person (of the totality of the person's health, including its ill and positive aspects), *for* the person (promoting the fulfilment of the person's life project), *by* the person (with clinicians extending themselves as full human beings, well-grounded on science and with high ethical aspirations) and *with* the person (working respectfully, in collaboration and in an empowering manner 80 through a partnership of patient, family, and clinicians). The person here is conceptualized in a fully
81 contextualized manner."

82 What role does scientific evidence play in PCC? PCC does not reject the use of scientific evidence in 83 psychiatry. Rather, it aims to place it in a framework that is sensitive to the patient's experience, 84 context, and personal values (Glas, 2019). However, integrating these personal and contextual factors 85 into a scientific framework is no easy task. How can we use scientific methods in a way that captures 86 PCC's tenets and is fruitful for both patient and therapist? As we will argue in this paper, psychiatry is 87 finding new avenues to do so with the help of recent developments in network analysis. However, it is important to consider whether network models that do justice to the person-, context- and value-88 89 dependency of mental disorders could provide clinically-relevant knowledge. Indeed, network models 90 could be used to represent almost anything, and making network models personalized and context-91 sensitive may make decisions on what should or should not be included in the model less principled. 92 This lack of boundaries may limit the epistemic power of such models in clinical practice. In this paper, 93 we examine where epistemic boundaries arise when using network models as tools for PCC, and 94 address how perspectivism can be used to define these boundaries. The paper is structured as follows. 95 In section 2, we discuss the network approach to mental disorders in more detail and examine why 96 network models could be used as tools for PCC. In section 3, we discuss how boundary problems arise 97 when using personalized network models of mental disorders in PCC. In section 4, we assess what kind 98 of knowledge about mental disorders personalized network models can provide by examining their 99 representational and explanatory boundaries. In section, 5, we examine perspectivism and how it can 100 help us demarcate personalized network models. In section 6, we address how perspectival reasoning 101 can shed light on the relevant explanation-seeking questions that personalized network models could 102 afford in clinical practice.

103 2 The network approach to mental disorders

What is network analysis, and why could it be used as a tool for PCC? Network analysis is inspired by principles of graph theory, which state that a network is a system whose elements are connected and mathematically represented as a graph. A graph is a set of nodes (elements of the network) and edges (connections between the nodes) (van den Heuvel and Sporns, 2013). The nodes may represent any kind of variable and the edges represent any kind of connection between them. We can use network analysis to quantify the connectivity patterns in a graph. These mathematically quantifiable connectivity patterns are called *topological properties* (see Box 1 for more information). Network

111 analysis has been applied to numerous fields like telecommunications, economics, city planning, 112 semantics, biology, neuroscience, and social sciences. In the past years, network analysis has also been 113 applied to the study of mental disorders. Indeed, proponents of the network approach to mental 114 disorders (e.g., Borsboom, 2017; Borsboom et al., 2019) argue that mental disorders should be 115 conceptualized as networks of interconnected symptoms. In this approach, non-symptom factors (such 116 as adverse life events, inflammation, abnormal brain functioning, or genetic mutations) are either 117 considered to be part of the 'external field' of factors affecting the symptom network (Borsboom, 2017) 118 or as constitutive of symptoms or symptom relations (Borsboom et al., 2019). So, network 119 analysis could provide a different means of conceptualizing mental disorders.

120 Proponents of the network approach also argue that in order to obtain better insight into mental 121 disorders, we should study symptom networks empirically. What role could such quantitative network models play in clinical practice? Of course, scientific models are not able to address all questions 122 123 pertaining to clinical practice: there are many (epistemic) aspects of clinical practice that are not best 124 addressed by scientific models (e.g., tacit knowledge). However, there are various reasons why network 125 models may be suitable scientific tools for clinical practice in general, and for PCC more specifically. 126 First, the network approach to PCC emphasizes that mental disorders involve a multitude of factors 127 instead of one root cause, thereby moving away from reductionistic (neuroessentialist) interpretations. 128 So, network models could be suitable tools for PCC because they promote a multidimensional view of 129 the nature of mental disorders. Also, network models can be construed in ways that do justice to 130 relevant characteristics of an individual, their disorder, and their context. Novel data collection methods 131 allow us to obtain such personalized data based on which personalized network models can be 132 estimated. For instance, recent developments in experience sampling methods (ESM; Larson and 133 Csikszentmihalyi, 1983) allow people to report on their thoughts, feelings, behaviour, and environment 134 using apps on their electronic devices. This modern form of ESM is called ambulatory assessment 135 (Timmons et al., 2017) and allows researchers to get insight into relevant patterns of someone's daily 136 life. It has been argued that ESM "enables a more detailed understanding of psychiatric 137 phenomenology" that may provide useful information for treatment targets (Myin-Germeys et al., 138 2009, 1534). Indeed, various studies have investigated whether estimating personalized symptom 139 networks based on ESM data could provide new insights and tools for treatment for therapists (e.g., 140 Bak et al., 2016; Fisher et al., 2017; Rubel et al., 2018). The types of personalized network models that 141 are most commonly used are vector-autoregressive (VAR) models. In VAR modelling, networks are 142 based on time series data, in which nodes represent symptoms and the edges denote (partial)

correlations between symptoms.³ VAR models can be used to estimate *temporal networks* (in which 143 144 edges represent how one variable predicts another at a later measurement window) and *contemporaneous networks* (in which edges represent the partial correlations between variables in the 145 146 same measurement window after controlling for the other variables in the same measurement window 147 and all variables at the previous measurement window) (for more information on how to estimate and 148 interpret such VAR models, see Epskamp et al., 2018a, 2018b). These quantitative models can be 149 construed on the basis of time series data of one person, and could include clinical, physiological and 150 contextual data, amongst others. Hence, whereas many statistical methods rely on larger samples of subjects, these models could be construed on an individual basis. Because of this, the construction of 151 152 personalized networks could allow for the incorporation of the patient's experiences and values, which may provide better insight into their clinical picture. Therefore, network models, due to their potential 153 154 to be personalized, could be a tool for PCC.

155 Another way that network models could be adapted to be in line with the principles of PCC is to add 156 salutogenic, or health-promoting factors. Salutogenesis refers to the study of the origins of health (Latin 157 salus = health, Greek genesis = origin) (Antonovsky, 1979). Indeed, salutogenesis is considered one of the principles of PCC: we cannot fully understand someone with a mental disorder diagnosis if we 158 159 do not include factors that may promote their well-being. As the World Health Organization (WHO) 160 stated almost fifty years ago, health is not merely the absence of disease or infirmity (Callahan, 1973). 161 If psychiatric practice and our models of mental disorder only focus on symptom reduction, this 162 implicitly adheres to the definition that health is the absence of disease. Moreover, it has been 163 demonstrated that simply decreasing negative mental states does not necessarily increase positive mental states (Bradburn, 1969; Keyes et al., 2002). So, from the perspective of PCC, it makes sense to 164 165 include health-promoting factors in our models of mental disorders. In fact, various authors have 166 emphasized that we need to have an open methodology of what to place in a network model in order to 167 truly capture an individual's condition (Köhne, 2020). It has been suggested – in line with PCC – that 168 the focus of network models on symptomatic factors only, without including health-promoting factors 169 is a missed opportunity (de Haan, 2020, 42): there is nothing in the network model that poses this 170 limitation, and including them would make sense from a clinical perspective. Since in principle

³ VAR network models should not be confused with dynamical system models, which are based on sets of differential equations and may provide directed (causal) relations between variables (e.g., causal loop diagrams). So, it is important to note that the claims we make with respect to the epistemic potential and boundaries of VAR models do not necessarily extend to dynamical system models.

- 171 anything can be represented as a node in a network model, network models in principle allow to include
- 172 health-promoting factors.

173 Network analysis has already been applied to the study of well-being. For instance, empirical studies 174 have examined the structure of well-being (Giuntoli and Vidotto, 2020), and subjective well-being in 175 people with mental health diagnoses (e.g., autism spectrum disorder) (Deserno et al., 2017a). However, 176 in line with PCC, it is also possible to integrate health-promoting factors into symptom networks. How 177 can we perform network analysis in such a way that it incorporates and/or does justice to the 178 interrelations between symptoms, contextual influences, and health-promoting promoting factors? This could either be done by simply incorporating these different components as variables into the analysis 179 180 (Deserno et al., 2017b), or by making use of more advanced network analysis methods such as multi-181 layer networks (Bianconi, 2018) that could do justice to the difference between these psychometric 182 items. These network models could combine the different factors using cross-sectional data. However, 183 in line with the principles of PCC, it is also possible to construct personalized VAR network models 184 that incorporate both symptoms, health-promoting factors, and contextual factors (Kroeze et al., 2017; 185 Lutz et al., 2018). However, if we allow our network models to be personalized by including healthpromoting and other contextual factors, does this not amount to drawing the boundary too broad for 186 clinicians, patients and researchers to make sensible inferences on their basis? Attempts to move 187 188 beyond symptoms inevitably give rise to questions concerning what factors (not) to include⁴. We will 189 discuss this problem in more detail in the following section.

190 **3** Network models: how to draw their boundaries?

191 What are the boundaries of network models, and what are the epistemic consequences of how we define

the boundary of these models? A boundary, in its most basic definition, is present when an entity is somehow demarcated from something else (Varzi, 2013). However, deciding how to demarcate an entity from its surroundings is not always straightforward. Boundary problems arise where there is a lack of consensus or principled reasons for demarcating a system, i.e., deciding what elements we should consider as being part of the system and as being external to it. It has been argued that such

⁴ The appearance of a boundary issue when including environmental factors in network accounts of psychopathology has already been emphasized by de Boer et al. (2021, 6). It is important to note that issues related to system demarcation and the epistemic consequences of where we draw the boundaries of our models, are not specific to psychopathology and/or network models. In fact, as one of the reviewers pointed out, boundary issues may be widespread in modelling practices. However, we argue that the specific questions concerning system/model demarcation and the consequences it bears, will differ per model and context in which the model is used. Hence, in this article, we focus on how boundary problems play out with respect to personalized network models in PCC.

197 difficulties inevitably arise when we deal with phenomena that are constituted or influenced by multiple 198 factors: even physical systems rarely have clearly defined boundaries (Meadows, 2008). Why is this 199 an issue for the use of personalized network models in PCC? This problem with system demarcation 200 translates directly to problems in model demarcation. For network models, this means that uncertainties 201 on how to define a system of interest will affect our node selection, i.e., selecting the variables that we 202 want to include in our model. This has important implications for the types of explanations, predictions, 203 and knowledge that personalized network models can provide. Node selection may strongly influence 204 the kind of topological properties that we find in network models, which could further impact the 205 conclusions we draw based on our findings (Forbes et al., 2017; Hallquist et al., 2019). For instance, 206 the value of the topological measure betweenness centrality, i.e., the relative number of shortest paths 207 passing through a specific node (Freeman, 1977), is highly influenced by the other nodes that are 208 included in the network (Bringmann et al., 2019). This means that removing or including one additional 209 factor in the network can have a great impact on the betweenness centrality values of individual nodes 210 (see Figure 1 for an illustration of this phenomenon). Another reason why node selection is important 211 is that models serve as epistemic tools that guide our reasoning about and understanding of the 212 phenomena they represent: they make complex phenomena more intelligible and manageable 213 (Knuuttila, 2009, 2011). This is of particular importance in clinical practice since models are able to 214 partially determine how both the therapist and the patient reason about the latter's condition. Hence, 215 where we draw the boundary of personalized network models (i.e., what nodes we select) has important 216 epistemic (and clinical) consequences. How, then, should we decide where to draw and how to justify 217 the boundary of personalized network models of mental disorders? In the next section, we will examine 218 in more detail how the use of VAR-based personalized network models could constrain the type of 219 knowledge that these models can provide in clinical practice.

220 4 The representational and explanatory boundaries of personalized network models

What boundaries do personalized VAR network models provide? More specifically, what features of
these models constrain the knowledge about mental disorders that we can obtain when using them?
Here, we will discuss two types of boundaries that these models afford: representational and
explanatory boundaries.

First, the statistical techniques that are used in estimating personalized network models will influence how the network is represented, and hence what kind of interpretations of mental disorders the model affords. These types of boundaries can be referred to as representational boundaries, i.e., constraints

228 that are related to the model's representation and its construction. Network representations themselves 229 do not provide many constraints on what can be represented. Network models typically capture global 230 and very abstract features of a system, whereas, for instance, mechanistic models capture more fine-231 grained and local features (Darrason, 2018; Kostić, 2018b, 2018a, 2019a, 2019b, 2020; Rathkopf, 232 2018; Kostić and Khalifa, 2021, 2022). However, nodes and edges can in principle represent anything. So, it could be argued that network models are representationally boundless: they do not provide 233 inherent constraints on what nodes can be included and can be extended indefinitely in size or scale. 234 235 However, network models in general, and VAR personalized network models more specifically, do 236 provide some, albeit limited representational constraints. For instance, VAR models cannot represent 237 how the structural relations between these variables will change over time (Molenaar, 2004), nor how 238 the variables in the network may be related to each other on other timescales. So, making use of VAR 239 network models does provide some representational constraints, and thereby influences the type of 240 information that these models can provide.

241 Relatedly, personalized network models seem limited to providing only certain types of explanations. 242 Explanatory boundaries concern the constraints provided by the types of explanations that a particular 243 model can provide. It is commonly agreed that models in general (Gelfert, 2018; Massimi, 2019; 244 Serban, 2020), and network models of mental disorders in particular (Epskamp and Fried, 2018) have 245 an *exploratory* function: they can be used as exploratory tools for estimating potential network 246 structures from psychological data, or as methods to generate hypotheses about the development and 247 treatment of mental disorders. However, network models of mental disorders may also provide 248 explanations. What types of explanations of mental disorders could VAR models provide? The first 249 possibility is that these models provide topological explanations, i.e., explanations that are based on 250 the topological properties of a network. We argue that this is the most promising explanatory potential 251 of these models because network models in general are particularly suited to provide such explanations (Huneman, 2010; Jones, 2014; Darrason, 2018; Rathkopf, 2018; Kostić, 2019a, 2020, forthcoming; 252 253 Kostić and Khalifa, 2021, 2022; Khalifa et al., 2022). What criteria should personalized network 254 models of mental disorders meet in order to provide topological explanations? As argued by (Kostić, 255 2020), this requires that the topological properties and empirical properties that feature in it are 256 approximately true, and also stand in an appropriate counterfactual dependence relation to each other 257 (see section 6). Second, could personalized network models provide mechanistic explanations? 258 Mechanistic explanations show how the working parts of a phenomenon that are organised into a 259 mechanism either cause a phenomenon of interest or constitute a phenomenon that is at a higher level

260 (Craver, 2007; Bechtel, 2008). For instance, some philosophers have argued that if network models 261 provide any explanation at all, it is a mechanistic one (Craver, 2016). According to this view, 262 mechanistic explanations show how the working parts that are organised into a mechanism either cause 263 the phenomenon of interest or constitute a phenomenon that is at a higher level (think of how the 264 macro-physical property of hardness is constituted by the micro-physical atomic structures). Given 265 this, personalized network models will not provide mechanistic explanations if any of the following mechanistic conditions are violated: 1) nodes and edges in a network model denote working parts of a 266 267 mechanism, 2) the explanandum (what is to be explained) is at a higher level than the explanans (what 268 does the explaining), and, 3) topological properties are causally responsible for the explanandum 269 (Kostić and Khalifa, 2022). Since the nodes and edges in personalized network models will likely 270 violate conditions 1 and 3, they do not provide mechanistic explanations. More precisely, the first condition is violated because the time series and correlations between them that are represented in 271 272 VAR models are not spatiotemporal working parts of a mechanism (they are merely conventional). 273 The third, causal responsibility condition is violated because the topological properties that are 274 explanatory in VAR models do not precede the phenomenon they explain (they are simultaneous): 275 since causation requires that causes precede their effects, it is not justified to claim that topological 276 properties in these VAR models cause mental disorders. So, it is unlikely that VAR models can provide 277 mechanistic explanations.

278 Finally, are VAR models able to provide causal explanations? On the one hand, it has been argued that 279 the edges in the temporal network provide temporal predictions or *Granger causality* (Granger, 1969), 280 which can be considered an approximation or potential indication of causal relations. However, it is 281 unclear whether VAR network models of mental disorders can provide *causal explanations* (Olthof et 282 al., 2020). For instance, it is unlikely that these models will satisfy interventionist criteria for causality 283 (Woodward, 2003; de Boer et al., 2021; Kostić and Khalifa, 2022). So, whereas personalized network 284 models could provide topological explanations, it is less clear whether they provide mechanistic or 285 causal explanations.

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Here, we see how making use of VAR personalized network models provides representational and explanatory constraints, and thereby limits the type of knowledge that these models can provide. To what extent do these considerations inform node selection? Arguably, the boundaries do not only constrain the type of explanations of mental disorders we can obtain based on personalized network models; they also constrain the model itself, i.e., what factors we decide to include. Indeed, the

292 explanatory potential of network models depends on what nodes and edges represent (Craver, 2016). 293 As aforementioned, the explanatory power of personalized network models will depend on whether 294 the topological and empirical properties in question are 'approximately true' (Kostić, 2020), which is 295 not limited to representational accuracy of nodes and edges, but also includes justification of particular 296 measurement approaches that are used to obtain and analyse data (Bringmann et al., 2022). Hence, if 297 we want personalized network models to provide explanations, this may constrain node selection. 298 However, to what extent will this consideration inform node selection in clinical practice? Assessing 299 these criteria is often difficult in clinical practice, especially because it does not give us information on 300 what kind of factors the model should include. In the next section, we argue that perspectivism could 301 help us provide such constraints on node selection in PCC.

302 **5** Perspectivism

303 As we already discussed, PCC affords certain aims, values, and goals for the therapist and the patient. Here, we argue that it is justified that such perspectival considerations influence node selection. 304 305 Perspectivism is a philosophical position that emphasizes the importance of pragmatic factors in 306 (scientific) theorizing and inquiry. It acknowledges that we cannot study the world in a way that is 307 independent of our own perspective, and that each system can be characterized by multiple perspectives 308 (Wimsatt, 2007, 222). Perspectivism presupposes that our theories and models serve specific goals of 309 interest. They each have a limited range, so the ones that researchers will use depend on their research 310 questions and goals at hand. Hence, perspectivism allows for - and even promotes - the use of a 311 plethora of diverse models to examine complex phenomena, such as mental disorders. In other words, 312 it could be argued that perspectivism promotes explanatory pluralism.

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314 It makes sense to examine personalized network models in light of perspectivism. Indeed, clinical 315 practice is inherently perspectival, and PCC brings the perspectival character to the fore. From a PCC 316 perspective, symptoms are no longer the central focus, but the individual with the disorder, their coping 317 with the disorder and everything that comes along with it. They can enter clinical practice with various 318 goals in mind: feeling better, functioning better, improving of agency, and finding the right balance 319 between dependence and independence (of help). Moreover, clinical goals serve as a guide for the 320 questions that the patient and therapist want to address, given a particular individual with a particular 321 disorder in a particular context. For instance, 'How can I feel better?', 'How can I function better (in 322 different domains of functioning)?', 'What can I do myself in order to improve my condition?' and What kind of help do I need?' Hence, in order to be suitable for clinical practice, network modelsshould help us to address these perspectival goals and questions.

325 These perspectival considerations can also play an important role in deciding what nodes should be 326 included in personalized network models. If we want clinical goals to constrain our node selection, the 327 nodes included should be 1) of relevance to the patient and their situational context, 2) able to guide 328 treatment, and/or 3) monitor clinical development. This means that node selection will be determined 329 by the specific problem that the patient wants to address – as decided in collaboration with the therapist 330 - or the symptoms they consider most burdensome (Bringmann et al., 2022). For instance, if it is 331 hypothesized that someone's depressive symptoms may be aggravated by their stressful job, this factor 332 should be included in the model. It may also limit nodes to ones on which it could be intervened 333 (Frumkin et al., 2021), or to items that are most relevant in monitoring whether treatments are effective 334 (Helmich et al., 2021), or predicting the risk of relapse (Smit et al., 2019). Moreover, various authors 335 have emphasized that how we build network models of mental disorders should be informed by clearly 336 defined research questions (and hypotheses) that are of personal and clinical relevance (Bastiaansen et 337 al., 2020; Borsboom et al., 2021; Bringmann et al., 2022). So, the clinical setting from which we start 338 our inquiry can provide constraints on node selection.

339 Does this mean, however, that any variable can in principle be included in personalized network models 340 as long as it is of relevance to the patient and clinician? A general worry is that perspectivism invokes 341 relativism by making node selection too dependent on contingent factors: the inquirer's background 342 knowledge, preferences, or contingent facts about personal circumstances (Giere, 2006; Mitchell and 343 Dietrich, 2006; Massimi, 2018; Massimi and McCoy, 2020). One may argue that if this is the case, this 344 may limit the robustness of personalized network models and hence their ability to provide useful 345 knowledge about a patient's condition. This issue is even more pressing if we take personal and 346 contextual factors into account, as would be advocated by PCC. One means by which we could ensure 347 that our models provide knowledge is by getting more clarity into the clinical questions that 348 personalized network models would actually be able to address. In other words, we should ensure that 349 the clinical questions we want personalized network models to address at least do justice to their representational and explanatory boundaries. In the next section, we will explore how perspectival 350 351 reasoning could help with that.

352 6 Perspectival reasoning and topological explanation in personalized network models

- How can we get more insight into the clinical questions that personalized network models could help 353 354 us answer? To illustrate how this can be done, we can use insights from perspectival (or erotetic) 355 reasoning. According to perspectival reasoning, questions can be conclusions in arguments. More 356 specifically, perspectival reasoning demonstrates how we can logically derive questions from the sets 357 of propositions (which may include hypotheses) about a model, and empirical observations (Hintikka, 358 1981; Wiśniewski, 1996). So, we can start from a set of propositions and derive relevant questions 359 based on the syntax (structure) and semantics (meaning) of those statements. To illustrate this, we can 360 use a toy example inspired by Wiśniewski, (1996, 2): 361 (1) If Mary writes three books in one year, then she is a nun, single, or she has a very patient partner. 362 (2) Mary writes three books in one year. 363 (3) Is Mary a nun, single, or does she have a very patient partner? 364 This example demonstrates that we can derive a relevant question – and space of possible answers to 365 366 that question – by observing what is the case (Mary writes three books in one year), and by keeping in 367 mind the possible explanations of what is the case (she either is a nun, single or has a patient partner). 368 Whilst perspectival reasoning cannot help us to determine the answer to this question, it does make it
- 369 clear what questions are sensible to ask given the available knowledge⁵.
- 370 How could perspectival reasoning be of use for our case at hand, i.e., determining what knowledge
- 371 personalized network models could provide in PCC? We argue that perspectival reasoning allows us
- 372 to formulate relevant explanation-seeking questions. To illustrate this claim, we will focus on the
- 373 topological explanatory potential of these models.
- 374 What criteria should be met before personalized network models are able to provide topological
- 375 explanations? We already discussed this briefly in section 4, but here we will explore this in more

⁵ This example differs from more familiar examples of deductive arguments in two ways. First, whereas traditional deductive arguments derive a conclusion which is also a proposition, this argument derives a question. Second, perspectival reasoning requires a disjunction of hypothetical propositions in the first premise, where any of the disjuncts could be true. The second premise specifies more closely what is the case. And based on that we are able to derive a relevant question, which also implies a space of possible answers. For the technical details of the logic of this type of arguments, see (Groenendijk and Stokhof, 1994; Wiśniewski, 1995, 2013; Millson, 2019, 2020).

- 376 detail using the account of topological explanations developed by Kostić (Kostić, 2020, forthcoming;
- 377 Kostić and Khalifa, 2021, 2022). Kostić's account provides necessary and sufficient conditions under
- 378 which a network model provides a genuine topological explanation and does so by explicitly
- 379 incorporating perspectival criteria. Kostić formulates his account as follows:
- 380 *a's* being *F* topologically explains why *a* is *G* if and only if:
- 381 (T1) *a* is *F* (where *F* is a topological property);
- 382 (T2) a is G (where G is an empirical property);
- 383 (T3) Had *a* been F' (rather than F), then *a* would have been G' (rather than G);
- 384 (T4) a is F is an answer to the question why is a G?

385 What do these criteria entail? T1 states that a system should have a certain network connectivity pattern, 386 expressed as a topological property (see Box 1 for examples of topological properties). T2 states that 387 a system should have an empirical property, e.g., it displays certain behaviour. T3 describes the 388 counterfactual dependence between a system's topological and empirical property: the behaviour of 389 the system should depend on the presence of the topological property. Topological explanations hence 390 concern a counterfactual dependence. However, if we combine these criteria, there is still something 391 missing: we do not yet know based on these criteria whether the topological property is an answer to 392 the relevant explanation-seeking question. That is why Kostić's account provides the perspectival 393 criterion T4: in order for a topological property to be an explanation for an empirical property, it should 394 be an answer to the relevant explanation-seeking question. This shows how asking the relevant 395 questions makes it intelligible why some empirical property G counterfactually depends on a network 396 connectivity pattern, which is expressed as its topological property F.

397 Let us now apply these considerations to an example that is relevant for the use of personalized network 398 models in PCC. Various studies have examined the global topological property *network density* to 399 personalized symptom networks to predict whether someone is vulnerable to developing (or relapsing 400 into) a mental disorder. In line with the idea that mental disorders behave like complex dynamic 401 systems (Wichers, 2014; Cramer et al., 2016; Olthof et al., 2020), it is supposed that we are complex 402 systems that may shift from a healthy into a disordered state following perturbations to the system. 403 Perturbations to the healthy state may not have any effects until a tipping point is reached and the 404 system (abruptly) shifts to a disordered state. Researchers have suggested that an increase in the 405 network density (i.e., the strength of associations between symptoms) may predict this transition from 406 a healthy to a disordered state (Wichers et al., 2011; van de Leemput et al., 2014). This hypothesis has been examined in simulation studies (Cramer et al., 2016) and in small samples of time-series data of
individuals with a major depressive disorder diagnosis (Wichers et al., 2011, 2020). Hence, if someone
has a symptom network that is more strongly connected, they are more likely to develop a mental
disorder.

We can use Kostić's scheme to formulate what criteria should be met before we can claim that a strongly connected symptom network can serve as an explanation for this vulnerability. Here, a refers to an individual, F refers to high symptom network density, and G as being vulnerable to developing a

414 mental disorder (i.e., entering a disordered state). Hence, the example can be unpacked as follows:

- 415 An individual *a* having high symptom network density explains why they are vulnerable
- 416 towards developing a mental disorder if and only if:
- 417 (T1) an individual *a* has a high symptom network density (which is topological property *F* in the
 418 schema above);
- 419 (T2) an individual *a* is vulnerable to developing a mental disorder (which is an empirical property
 420 *G* in the schema above)
- 421 (T3) had an individual *a* had a low symptom network density (rather than a high symptom
- 422 network density), then the individual *a* would not have been vulnerable to developing a
- 423 mental disorder
- 424 (T4) an individual having a high symptom network density is the relevant answer to the question425 why the individual is vulnerable to developing a mental disorder.

426 How can we examine whether T4 is the case by making use of the principles of perspectival reasoning? 427 By assessing whether being vulnerable towards developing a mental disorder counterfactually depends 428 on high symptom network density, and combining this with the observation that an individual is 429 vulnerable towards developing a mental disorder. However, starting with a statement about what it is 430 for an individual to be vulnerable to developing a mental disorder, and the empirical finding that the 431 individual is more vulnerable to developing a mental disorder, we can also come up with a relevant 432 explanation-seeking question. The argument itself provides a space of possible answers. It makes it 433 intelligible why appealing to a dependency between network density and vulnerability counts as an 434 explanation of why the mental disorder has developed (with a particular collection of symptoms), but 435 also why appealing to different topological properties or even non-topological properties does not, i.e., 436 it is because different topological properties or even non-topological properties are not included in the 437 space of possible answers (Lange, 2018). Here, we can see how the principles of perspectival reasoning

- 438 can help us in dealing with the boundaries of personalized network models in clinical practice: it can
- help us derive questions that are epistemically fruitful for both explanatory and clinical purposes. It
 also suggests that we should limit our personalized network models to nodes about which we have
- 441 specific (topological) hypotheses.

442 **7** Conclusion

443 In this paper, we have provided a conceptual analysis of the boundary problems that arise when using 444 personalized network models in PCC. PCC focuses on individuals and considers disorders as highly 445 context-dependent. There are various aspects of network models that make them suitable as tools for 446 PCC, including their ability to be personalized by making use of ESM data and their ability to 447 accommodate a variety of different personal and/or contextual factors. However, the type of knowledge 448 that these models can provide for clinical practice is influenced by how we draw the model's boundary. 449 We have argued that the use of personalized network models influences the interpretations and 450 explanations of mental disorders that we can provide. Perspectivism can help us to determine what 451 nodes should be included in the model, and perspectival reasoning can help us make the explanations 452 that these models could provide more intelligible.

453 Using personalized network models in PCC will inevitably invoke problems in node selection and 454 model demarcation. However, our analysis has shown that we can justify our decisions on what factors 455 (not) to include, although this does not mean that the use of network models in PCC is straightforward. 456 One of the important issues in this application is how to determine the relevance of the *patterns* that 457 are found and that aim at gaining a better understanding of the patient and how they deal with their 458 condition. Moreover, the relevance that a therapist attributes to a pattern may differ from the relevance 459 that a patient attributes to it, for both stakeholders may have different values attributed to these findings. 460 Clinical practice is messy, and there will not be a one-on-one translation of our proposal into clinical 461 guidelines. However, our account may suffice as an example of how network demarcation could work 462 in practice. At last, our account emphasizes the importance of making a patient's context and clinical 463 goals explicit, for this may constrain the range of relevant why-questions that personalized network 464 models could address and could guide these in the right direction.

465 8 Conflict of Interest

466 The authors declare that the research was conducted in the absence of any commercial or financial 467 relationships that could be construed as a potential conflict of interest.

468 9 Author Contributions

469 All authors contributed to the conception and design of the study. MR wrote summaries of the literature

470 on PCC and networks; NSdB and DK wrote the section on networks; NSdB developed and together

471 with DK wrote the section on boundary problems; DK developed and together with NSdB wrote the

472 section on perspectivism; LdB and GG wrote parts of the introduction and the conclusion; DK and LdB

473 coordinated the project.

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Box 1: A non-exhaustive overview of topological properties that can be used in network psychometrics. A network is a collection of nodes and edges. A node is a variable within a network (e.g., anhedonia could be a node in a symptom network), and an edge is a connection between nodes in a network (e.g., a partial correlation in a psychometric network). In weighted networks, edges can have signs (positive or negative relation) and weights (the strength of the relation).

Figure 1: A hypothetical example to illustrate the influence of node selection on local topological properties in a network. In (A), we see a hypothetical network that consists of six nodes. (B) demonstrates that node 3 has the highest node degree, closeness centrality, and betweenness centrality. (C) shows the same network in which node 3 is removed. (D) shows the influence of this removal on the network's centrality measures. Now, nodes 4-6 have the highest node degree, and node 4 has the higher closeness and betweenness centrality. Moreover, the betweenness centrality values of nodes 5 and 6 have strongly increased.