The glue of (ab)normal mental life: Networks of interacting thoughts, feelings and behaviors
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Chapter 1

Introduction

Major depression is caused by a gene, which causes serotonin shortage in the brain. True or false? Likewise: extraversion causes party-going in individuals. Again: true or false? These and similar statements are not a figment of my imagination, these are real statements in either the scientific literature or in the popular press; and these statements depict a deceptively simple picture of what psychological constructs such as major depression and extraversion are. Small and inconsequential as such statements may seem, they speak volumes of how people—scientists and laypeople alike—(implicitly) think about psychological constructs in general: for example, 1) they are ultimately reducible to specific (neuro)biological properties (e.g., disorder X is caused by gene A), 2) they operate in the minds of individual people (e.g., personality trait Y is somewhere in my brain), and 3) we know for a fact how these constructs, in your and in my mind, operate to cause a constellation of (pathological) thoughts, feelings, and behaviors (e.g., we know how disorder X causes symptoms B and C). One of the main forces that drove the inception of this dissertation was the realization that these statements are most likely false. Let us consider some of the facts: despite tremendous efforts to find it, there is no gene (or constellation of genes) that explains more than a fraction of the phenotypic variance in major depression, or any other psychological construct (Kendler, 2005a; Stefanis, 2008); antidepressants, which aim to augment available serotonin levels in the brain, do not work in all patients with major depression (e.g., Lacasse & Leo, 2005); there is no single experiment that has ever shown how exactly extraversion causes party-going behavior in individual people (Cramer, van der Sluis, et al., 2012); etc. That is, given the actual findings, it is at best premature to claim to know what psychological constructs are and how they are caused, let alone presupposing that biological reductionism should be the ultimate goal of psychological science. At worst, we are essentially clueless regarding the nature of psychological constructs.

There is one thing that we do know, which forms the starting point of this dissertation: undisputed and consistent, perhaps the number one fact in clinical


and personality psychology is that some (pathological) thoughts, feelings, and behaviors co-occur more often with one another than with other thoughts, feelings and behaviors. For example, liking parties and liking people tend to co-occur in individuals while liking parties and sorting one’s socks by color do not. Likewise in psychiatry, since the early 20th century, psychiatrists such as Kraepelin (1923) and Lewis (1934) have observed that, for example, depressed mood and feelings of guilt tended to co-occur more frequently with one another than, say, depressed mood and having panic attacks. Now, the number one question is, or should be, what causes these specific patterns of covariation? What is the glue that holds together some (pathological) thoughts, feelings and behaviors?

For decades the answer has been: different underlying causes. That is, in personality psychology, the dominant idea is that, for instance, liking parties and liking people co-occur because they share the same underlying cause, namely extraversion. Liking parties and sorting one’s socks by color, in contrast, covary to a lesser extent because they have different underlying causes: liking parties is caused by extraversion while sorting socks by color is caused by conscientiousness. Likewise in psychiatry, depressed mood and feelings of guilt co-occur frequently because they are caused by the same underlying disorder: major depression. In contrast, depressed mood and panic attacks do not co-occur as frequently because they are caused by different disorders: depressed mood is caused by major depression while panic attacks are caused by panic disorder. In particular for mental disorders, the idea of disorders being common causes of their symptoms probably has its roots in the successful paradigm of Western medicine (Hyland, 2011). For quite some medical diseases, it makes sense to postulate that disorders cause their respective symptoms: for example, a lung tumor that, because of its physical presence in someone’s lungs, causes shortness of breath, chest pains and coughing up blood. This medical disease model is exactly what has fueled the quest for the analogy of the ‘tumor’ in the case of mental disorders: some pathophysiological correlate that, like a lung tumor, causes the symptoms of a particular mental disorder. For instance, in the case of major depression, a plethora of work appears to show that major depression is associated with a host of pathophysiological correlates: e.g., serotonin depletion, allelic variants of certain genes that appear to predict treatment outcome (Ogilvie et al., 1996; Serretti, Kato, De Ronchi, & Kinoshita, 2007; Wong, Dong, Andreev, Arcos-Burgos, & Licinio, 2012), and atrophy in brain areas such as the hippocampus (MacQueen & Frodl, 2011; Sheline, Wang, Gado, Csernansky, & Vannier, 1996).

The problem, however, with many of these findings is at least fourfold: 1) specificity: abnormalities in the serotonin reuptake function, for example, are not only implicated in the etiology of major depression but in that of obsessive-compulsive disorder, substance abuse and anxiety disorders (Nakamura, Ueno, Sano, & Tanabe, 2000); 2) explained variance: when combining all possible candidate genetic variants, they still explain only a very small portion of the variance in major depression (Wong et al., 2012); 3) cause or effect: for hippocampal atrophy, for example, it is not clear whether this is a cause or effect of (repeated episodes of) major depression (MacQueen et al., 2003); and 4) no omnipresence: serotonin depletion, for example, is not present in a substantial proportion of patients with major depression (Lacasse & Leo, 2005). As such, given these problems, the com-
mon causes that correspond to mental disorders (the ‘tumors’) either do not exist or else are very hard to find. There are two ways in which one can respond to such a gap between theory and empirical evidence. One way, the road that has usually been taken in the past years, is that we should look harder. With more participants, better research equipment and ever more intricate ways of analyzing the data, we will eventually find the ‘tumor’ equivalent and its associated physiological and genetic abnormalities. The other road, and the one we, and others (McGrath, 2005; Kendler, Zachar, & Craver, 2011) have taken, is to accept these findings as an indication that we may need to rethink the nature of mental disorders, and of psychological constructs in general.

So if not common causes, what then is the glue of (ab)normal mental life? In this dissertation, I will argue that direct interactions between (pathological) thoughts, feelings and behaviors in a network are the glue. As such, liking parties and liking people do not covary because they are both caused by extraversion; they covary because they directly interact with one another: e.g., enjoying the company of other people prompts a person to seek out environments in which one can mingle with people, for example at parties. Likewise, depressed mood and feelings of guilt do not co-occur frequently because they are both caused by major depression; they covary because of a direct relation: e.g., depressed mood causes a person to feel guilty towards friends and family for being so blue all the time. In Chapter 2, this network approach will be explicated and exploratively tested for mental disorders, in particular for comorbidity between major depression and generalized anxiety disorder. Also, the network model will be conceptually contrasted to the mathematical formalization of the common cause idea, namely latent variable models. In Chapter 3, I respond to various commentaries that were written in reply to Chapter 2. In Chapter 4, I will show that the network model explains the phenomenon that various stressful life events (e.g., the loss of a loved one) influence different symptoms of major depression; a finding that is not easily accommodated by latent variable models. In Chapter 5, I present a mathematical formalization of the network model in which the probability of a symptom becoming activated is a logistic function of the activation of its neighboring symptoms in the network. Additionally, I show that this model is able to explain empirical phenomena such as spontaneous recovery, and that the model accommodates taxonomic as well as continuous views on major depression. In Chapter 6, I outline a network perspective on normal personality traits and show how taking this perspective might change our outlook on the concept of a ‘trait’, the relationship between genes and traits, and between personality and psychopathology. In Chapter 7, I respond to various commentaries that were written in reply to Chapter 6. Finally, Chapter 8 provides a practical guide to construct and analyze networks.