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Recent Advances in the Neurobiology of Bipolar Disorder

Thereasa Cronan*

Department of Psychology, San Diego State University, California, USA

Introduction

An important contributor to global impairment is bipolar disorder (BD). Its biological cause is unclear, and current therapies are ineffective. Here, we examine two recent advancements. With an emphasis on voltage-gated calcium channels as a component of the illness process and as a potential treatment target, let's start with the finding of risk genes and their consequences. Second, it is becoming more and more clear, thanks to new technology, that the bipolar phenotype is more complicated and nuanced than just recurrent manic and depressed episodes. Persistent mood instability is one of these characteristics, and research is being done to better understand its causes and potential medical applications. BD demonstrates how modern neuroscience, genetics, and digital methods are transforming psychiatry.

Discussion

Diagnostic categories from the nineteenth century continue to be heavily used in psychiatry. These are managed with medications that were inadvertently found a few decades ago and are based on clusters of symptoms rather than biological markers. This unfavourable situation is typified by BD. Its primary characteristics, as well as how it is evaluated and treated, have hardly changed despite the name change [manic depression was its previous term]. Beyond its well-established high heredity, a significant contributor to this stagnation has been the absence of any serious progress into its underlying biology and causes. Although there is evidence of altered structural and functional brain connectivity as well as alterations in markers of oxidative stress, mitochondrial function, inflammation [1], circadian rhythms, and dopamine, it is still challenging to integrate these contradictory findings and to distinguish between changes that are directly related to the disorder and its treatment from those that are secondary to it.

The problem is finally getting better. There are genuine chances for a change in our perception of BD and how it is diagnosed and treated, albeit optimism must be restrained by a grasp of the many difficulties. In this article, we focus on two current topics of interest: the identification of the first BD risk genes and their implications, as well as the use of cutting-edge technologies that have the potential to clarify or redefine the BD phenotype. These advancements serve as an example of how genomics, neurology, and digital technology are bringing about a new age in psychiatry [2].

Bipolar disorder by genetic inheritance

According to twin studies, the heritability of BD is between 0.7 and 0.8, with the chance of getting it in a child of an affected parent being roughly ten times higher. Mendelian inheritance or significant effect genes are not supported by any data. Instead, genome-wide association studies (GWAS) are starting to find many susceptibility loci, each of small effect, as with the majority of psychiatric diseases. Since 2007, several GWAS have been conducted, along with associated meta-analyses. lists the loci and genes that have been implicated so far. By GWAS standards, the combined sample sizes are still small, and more loci need to be found. In fact, the Psychiatric Genomics Consortium analysis, which will be released soon and includes more than 20,000 BD cases and 30,000 controls [3], found 19 significant loci, including 12 novel ones. Initial exome and genome sequencing data point to the presence of rare detrimental mutations in some cases of BD, but their identity and overall impact on the condition are yet unknown. There is a small amount of clinicogenetic variation within BD, for instance, based on the predominating symptoms or between the bipolar I and bipolar II subtypes. There is, however, limited evidence for BD-specific genes; joint GWAS analyses reveal large overlaps in the risk loci for BD and schizophrenia, as well as with other serious mental conditions and intermediate phenotypes, such as circadian characteristics. One difference between BD and schizophrenia is the extent of copy number variation in the latter [4].

Though genomics for BD is still in its infancy, attempts have started to comprehend the biological underpinnings of the relationships found so far. Due to what was already known about the activities of two genes (CACNA1C and ANK3), interest has been focused on them. Below, CACNA1C is addressed in more detail. Ankyrin G, which is encoded by ANK3, connects axonal voltage-gated sodium channels to the cytoskeleton and also plays a part in dendrites and glia. Another risk gene, TRANK1, also encodes ankyrin G and may have some common activities because it includes numerous ankyrin repeat domains. The first attempts have been made to discover the pathways that these particular risk genes influence, complementing the focus on these genes [5]. Reported six pathways including hormones, second messengers, calcium and glutamate signalling, and connection with BD that could be replicated. Together, these results suggest that BD may be an ion channelopathy, at least in part, with abnormal calcium signalling playing a key role.

Conclusion

It is frustrating how little we still know about BD. It will continue to be a descriptive syndrome since we don't know enough about it to be able to characterise it or conceptualise it based on a mechanism or aetiology. Undoubtedly, there are still many unanswered questions (see Outstanding Questions). There are, however, causes for optimism. First, the identification of some of the BD risk genes has the potential to fundamentally alter our knowledge of the pathophysiology and neurobiology of the disease. Second, a more quantitative, longitudinal approach to the BD phenotype is currently possible because to the employment of digital technology and remote sensors along with cutting-edge analyses of the associated data. This increases the possibility for more accurate clinical course prediction and offers a more complex phenotype for behavioural and biological research. The growing significance of "big data" in both of these fields, whether in terms of genetic investigations or the multidimensional data streams acquired by digital gadgets, is a common trait. Third, although it is not covered in this article, structural and functional brain imaging are being used to assist identify the important neuronal circuits in BD and may be useful for prognostic and diagnostic purposes.

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***Address for Correspondence:** Thereasa Cronan, Department of Psychology, San Diego State University, California, USA, E-mail: cronan@threasa.in*

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