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Review

Sensitive and critical periods during neurotypical and aberrant neurodevelopment: A framework for neurodevelopmental disorders

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ABSTRACT

During sensitive and critical periods, the brain undergoes significant plasticity from the level of individual synapses and neuronal networks up to the level of behaviour. Both sensitive and critical periods during neurotypical development of the young animal provide a framework to the early temporally-regulated modifications that occur in the nervous system.

In neurodevelopmental disorders (NDD), notably autistic syndromes and intellectual disability, children exhibit developmental delays in motor, social and sensory processes and often miss key developmental milestones. In corresponding genetic NDD mouse models, recent data reveal temporally-regulated and in some cases, transient impairments in many neuronal and behavioural phenotypes during development. However, the mechanisms underlying these impairments in NDDs and their potential links with neurobiological mechanisms governing neurotypical development are not fully investigated. This article highlights the potential for the use of known critical and sensitive periods during vertebrate development to investigate and advance our understanding of the neural bases underlying impairments in these developmental disorders of the nervous system.

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Contents

1. Introduction

Building a brain, a complex neuronal computer with a repertoire of behaviours and the potential to learn, requires a carefully choreographed sequence of steps. The process of brain development is determined by distinct developmental stages of gene expression, intrinsic neuronal activity and molecular guidance cues (Chilton, 2006; Marin et al., 2010), combined with the influence of external factors including resources from the mother during embryonic stages (Zimmerman and Connors, 2010). Time-limited developmental stages in neuronal migration, circuit formation and synaptic refinement are key to form the adult brain. Using the

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rodent brain as a model system, much is known about the neural basis of these distinct developmental stages, known as critical or sensitive periods. To-date, research has largely focused on elucidating the neural mechanisms underlying critical and sensitive periods of normal or neurotypical brain development and behaviour (Hensch, 2004; Knudsen, 2004). Clear evidence is shown for the existence of critical and sensitive periods across many vertebrate species, including pre- and postnatal stages in the primate brain comparable to those observed in the rodent (Workman et al., 2013).

More recently, animal models for monogenic neurodevelopmental disorders have shown specific impairments during well-established critical periods. These data have led to the notion that impairments underlying neurodevelopmental brain disorders have their onset during restricted critical or sensitive periods (Kroon et al., 2013; LeBlanc and Fagiolini, 2011; Martin and Huntsman, 2012; Meredith et al., 2012; Wang et al., 2014). This mini-review will outline the idea that our existing knowledge of sensitive and critical periods can be used as a framework to investigate potential mechanisms underlying NDDs and as a guide for elucidating the developmental time periods during which misregulation may first occur.

1.1. Critical and sensitive periods for brain development

During the development of an organism, critical and sensitive periods are time windows during which the system is most subject to change (Hensch, 2004, 2005; Johnson, 2005; Michel and Tyler, 2005). During these time windows, plasticity of specific physiological or behavioural phenotypes are heightened relative to other developmental stages. A critical period is a restricted time window during which the system is most responsive for an essential developmental change to occur, its absence causing a permanent modification in brain and behaviour. Critical periods are limited during a specific time window and after their closure, the phenotype is classically thought not to be malleable – but see, e.g. (Oberlaender et al., 2012; Pizzorusso et al., 2002) for somatosensory and visual manipulations in adulthood. The distinction between critical and sensitive periods can be subtle: Historically, critical periods have been used to describe brain circuit-based phenotypes including ocular dominance in the visual system or synaptic plasticity in the developing somatosensory cortex (Fox et al., 2000; Hensch, 2004). Sensitive periods, on the other hand, are often referred to as time windows during which exposure of the organism to external factors or experience modulates the emergence of specific behaviours. Classic examples include filial imprinting in young birds whereby they form a social attachment to their mother or equivalent during the first few days post-hatching (Horn, 2004; Lorenz, 1935).

These restricted time periods occur along different temporal trajectories from filial imprinting within the first few days of life in the precocial chick, to visual development in humans, where poor vision in the condition amblyopia can be corrected over a period of months and even years but only during childhood. Critical and sensitive periods provide a framework to map out time windows for great change and plasticity of the brain as it grows normally. However, many now believe that these periods also represent points of particular vulnerability in the developing brain. A small change in gene expression, external growth factor or altered neuronal activity pattern in the nervous system due to intrinsic or extrinsic sources can have a major influence upon the developmental trajectory of the organism, and can potentially lead to specific neurodevelopmental impairments.

1.2. Developmental aspects of NDDs

Neurodevelopmental disorders (NDDs) are caused by impairments during growth of the nervous system that cause or lead to dysfunction at neuronal and sensory or behavioural levels (Goldstein and Reynolds, 1999). NDD impairments usually manifest at birth or during infancy and can affect multiple functions including cognitive processing, language, emotion and motor control (Zoghbi and Bear, 2012). Many syndromes are classified as NDDs; for the purposes of this article, focus will be limited to intellectual disability (ID) and autism spectrum disorders (ASD). However, the hypotheses discussed may well apply to other disorders with clearly dysregulated developmental profiles. For ID and ASDs, the onset and progression of the disorder can be striking: parents and clinicians report a series of missed developmental milestones during the first few years of life including speech impairments, motor delays and irregular social interactions for some children (Geschwind and Levitt, 2007; Kau et al., 2000). For example, hypotonia and early onset delays in motor skills are characteristic for many monogenic NDDs including Angelman and Fragile X syndromes (Clayton-Smith and Laan, 2003; Kau et al., 2000; Williams et al., 2006). Furthermore, deficits in speech development and difficulties with social communication & interaction are common to both Angelman and Fragile X syndromes, either characterised as core symptoms or as part of an associated ASD (Amiet et al., 2008; Gillberg and Billstedt, 2000). Even if transient in nature, the effects of these developmental delays may also extend beyond the initial appearance to cause later disruptions. This concept, known as 'sleeper effects' can be seen in the visual system where early transient impairments in vision caused by cataracts disrupt the normal patterned activity necessary for aspects of visual perception later on in adulthood (Maurer et al., 2007).

It is not just syndromic NDDs with a monogenic cause that show clear developmental onset and progression: other nonsyndromic NDDs or those induced by environmental insults delineate similar dependence upon brain developmental stages. For example, significantly higher than normal incidences of spina bifida and ASD diagnosis occur in children whose mothers took the anti-epileptic drug valproic acid (VPA) during pregnancy (Christensen et al., 2013). Specifically, incidence of malformations in foetal valproate syndrome is highest if exposure occurs during the first trimester (Lindhout and Omtzigt, 1992). These effects are verified in a rodent model of VPA exposure where injection of VPA into the mother at embryonic day (E)12.5 causes an increased incidence of neural tube closure difficulties in the offspring and later emergence of autistic phenotypes in the form of stereotypy and hyperactivity (Dawson et al., 2006; Schneider and Przewlocki, 2005).

Regardless of the underlying genetic or environmental cause for an NDD, the early developmental onset and delays in progression are common across syndromic and nonsyndromic conditions. By focusing on the developmental misregulation during specific critical periods, this article proposes that the known neural mechanisms regulating these critical periods may guide investigation into the neural changes that may underlie the misregulated phenotypes in NDDs.

1.3. Synaptic basis of NDDs

Many NDDs are heterogeneous disorders, with heritable but also multiple de novo mutations involved in ID and ASD (Neale et al., 2012; O'Roak et al., 2012; Sanders et al., 2012). Of those genes known from monogenic syndromes and those from large genome-wide-association-studies for ASD, a significantly high proportion of candidate targets are located at the pre- or postsynaptic compartments or are known to directly regulate synaptic functions in neurons (Kang et al., 2011; Ruano et al., 2010; van Bokhoven, 2011; Voineagu et al., 2011). These observations have led to the term 'synaptopathies' to describe the multitude of conditions, including ID and ASD, that directly affect synaptic processing and plasticity (Brose et al., 2010). At the anatomical level, these synaptic effects

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