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What risk factors for Developmental Language Disorder can tell us about the neurobiological mechanisms of language development

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ABSTRACT

Language is a complex multidimensional cognitive system that is connected to many neurocognitive capacities. The development of language is therefore strongly intertwined with the development of these capacities and their neurobiological substrates. Consequently, language problems, for example those of children with Developmental Language Disorder (DLD), are explained by a variety of etiological pathways and each of these pathways will be associated with specific risk factors. In this review, we attempt to link previously described factors that may interfere with language development to putative underlying neurobiological mechanisms of language development, hoping to uncover openings for future therapeutical approaches or interventions that can help children to optimally develop their language skills.

1. Introduction

Language acquisition involves learning the meaning and use of words, phrases and sentences, and the rules to compose and combine them. It is a multidimensional process that entails the development of expressive and receptive skills in oral or signed, and later written, modalities, as well as learning to use language for communicative purposes. Despite the complexity of language, children unravel its structure with relative ease (Saffran et al., 2001). Most children successfully use it for communication within the first few years of their lives, which is highly important for further linguistic development, as well as for development in other domains, including social competence (Longobardi et al., 2016) and academic skills (Bleses et al., 2016).

Language acquisition is robust, but there are also substantial individual differences (Donnelly and Kidd, 2020). Some children experience language difficulties that are secondary to other conditions, such as autism spectrum disorder (ASD) or traumatic brain injury, but there are also children who have severe language problems in the absence of a clear cause (Leonard, 2014). This latter group of children, estimated at 5–7% of the population (Calder et al., 2022; Norbury et al., 2016; Tomblin et al., 1997), is diagnosed with a Developmental Language Disorder (henceforth DLD; previously often referred to as Specific Language Impairment (Bishop et al., 2017)). These children experience challenges in daily communication and in societal participation throughout their lifespan (Botting et al., 2016; Johnson et al., 2010). Problems of children with DLD are thus persistent, even though speech and language therapy interventions are found to be effective (Heidlage et al., 2020; Law et al., 2015, 2003).

Although little is yet known about its etiology, DLD is best defined as a complex neurodevelopmental disorder that involves the interaction of multiple genetic and environmental risk factors (Bishop, 2009). Recognition of DLD in young children can be problematic due to the large variation in typical language development, as well as the heterogeneity within the DLD population, with symptoms varying from child

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to child. Next to the most prominent linguistic deficits, which can be observed in all language domains and modalities, children with DLD have been found to show weaknesses in auditory perception (de Wit et al., 2018; van Bijnen et al., 2019), motor skills (Hill, 2001; Sanjeevan et al., 2015) and several higher-order cognitive abilities such as working memory (Vugs et al., 2013), declarative and procedural long-term memory (Lum and Conti-Ramsden, 2013), statistical learning (Evans et al., 2009), and sustained attention (Ebert and Kohnert, 2011). While DLD may primarily affect language, it is thus also associated with (subclinical) deficits in nonlinguistic domains (Bishop et al., 2017). A number of theoretical, neurocognitive accounts have tried to integrate the linguistic and nonlinguistic weaknesses of children with DLD. Deficits in cognitive and perceptual mechanisms that are important for the acquisition of language, including auditory processing (Bishop, 2007; Tallal, 2004), memory (Gathercole and Baddeley, 1990; Ullman and Pierpont, 2005) and executive functioning (Kapa and Plante, 2015), have been proposed to explain the language problems of children with DLD.

The development of language difficulties thus depends on complex and dynamic interactions between a wide variety of internal and external factors, in which timing is critical. A better understanding of the neurobiological mechanisms that underlie the impact of these factors will help in accounting for cascading effects, as well as interindividual variation in developmental trajectories. In the end, understanding the neurobiological processes that underlie language acquisition is necessary to optimally support the language development of young children, prevent language delays and disorders, and further develop effective interventions aimed at vulnerable populations and children with language problems, including children with DLD.

2. Organization of this review

Although an integral understanding of the neurobiological mechanisms of language development is currently missing, many studies have identified or suggested risk factors that may disturb children's language development and that could be associated with DLD. Importantly, in this review it is not our aim to examine such risk factors themselves or weigh the evidence in favor or against them. Instead, we attempt to link risk factors described in the literature to putative underlying neurobiological mechanisms of spoken language development. Subsequently, we aim to evaluate to what extent the neurobiological evidence we present converges with existing neurocognitive accounts of DLD. This endeavor is ambitious, and we certainly do not presume to offer a definitive account. Nonetheless, we believe that this is an important exercise that will enhance our understanding of the complex etiology of DLD, and could potentially inform the development of successful interventions.

We identified potential risk factors for DLD and related language problems in the available literature through a thorough (though not systematic) literature search, using the key words 'Specific Language Impairment', 'Developmental Language Disorder', and 'risk factors'. We selected risk factors that are frequently mentioned in the literature and explored how these risk factors could reveal possible underlying mechanisms of language impairment. We acknowledge that the list of potential risk factors in this review is not complete and may include factors for which evidence is uncertain. For a thorough overview of the evidence supporting these risk factors, we refer the reader to previous excellent reviews (e.g. Bishop, 2006; Calder et al., 2022; Diepeveen et al., 2017; Prathanee et al., 2007; Reilly et al., 2010; Rudolph, 2017; Stanton-Chapman et al., 2002; Whitehouse et al., 2014).

We noticed that there are only few neurobiological mechanisms that seem to impact language development specifically. That is, the mechanisms underlying most risk factors appear to disadvantage brain development and neurocognition in general, affecting brain activity and gene expression patterns during neurodevelopment and, as a result, impacting the acquisition of language. In this review, we will discuss the following risk factors for which this is the case: sex, family composition, exposure to toxic substances (specifically alcohol and chemicals in plastics), nutrition, maternal health problems, and viral infections in both mother and child (Section 2). In addition, we identified three risk factors that are, among others, associated with a neurobiological mechanism that potentially explains how specifically language development is affected: maternal smoking, preterm birth, and low sleep quality. These risk factors will be discussed separately in Section 3. It is worth mentioning that our distinction between general and specific mechanisms reflects the knowledge that is currently available, but it is neither clear-cut nor definitive.

When discussing possible underlying neurobiological mechanisms, we combine results from human and animal studies. Most of what we know about the complex neurobiological processes during brain development comes from animal studies. Human studies on the neurobiology of language acquisition can at best be correlational; multiple potential risk factors interact and their effects are therefore difficult to disentangle. Animal models, which allow for a high level of experimental control, may help unraveling the precise effects and mechanisms of potential risk factors for language problems in children. Of course, some potential risk factors cannot be satisfactorily modeled in animals, but important components of pre- and postnatal conditions, the effects of environmental factors, and even cognitive stimulation can certainly be captured. Furthermore, animal studies can provide cellular and molecular details, which are largely inaccessible in human studies. In the current review, we specifically include studies on rodents and songbirds. The strength of rodent studies lies in the detailed genetic and molecular control and comparability of mammalian brain development, whereas the strength of songbirds as a model is their vocal learning ability, auditory-motor integration and associated neural adaptations.

This review consists of six parts. In the *first* part, we briefly describe key neurobiological mechanisms of brain development and discuss how environmental factors may disturb these processes. In the *second* part, we discuss potential mechanisms of risk factors that appear to compromise brain development in general and may affect multiple brain functions in parallel. In specific brain regions, the effects of these factors may be so small as to even go unnoticed, but their accumulated effects can contribute to the emergence of a deficit in a range of cognitive and social abilities, including learning and using language. This may well be exacerbated by the interdependencies between various brain functions relevant for language. In the *third* part, we describe potential mechanisms of three risk factors (maternal smoking, preterm birth, sleep) that appear to be specifically associated with impaired language development mechanisms. That is, they may interfere with the development of a specific neural (sub)system that is a critical component of the neurocognitive architecture needed for language learning.

To be able to identify possible neurobiological pathways and understand the underlying mechanisms of risk factors that have been described in the literature, we discuss each risk factor separately. However, it is important to note that none of the individual risk factors are sufficient or necessary to explain impaired language development or DLD, in line with the description of DLD as a multifactorial neurodevelopmental disorder (2nd paragraph of introduction). Moreover, risk factors are also typically related. Notably, several of the risk factors we discuss tend to co-occur in children growing up in families with low socioeconomic status (SES), including prenatal exposure to drugs, early life stress, or infections. Interactions between risk factors can also result from overlap between underlying neurodevelopmental mechanisms or brain structures involved. In the *fourth* part of the review, we therefore elaborate on the multifactorial and interactive nature of the underlying mechanisms of risk factors that we discuss in the second and third part. In the *fifth* part, we will discuss the possible relation between the neurobiological mechanisms and neurocognitive accounts of DLD and, finally, in the *sixth* part, we will provide a perspective on future research.

3. Neurobiological processes underlying early language development

The neurobiological processes underlying the early development of brain structure and function are highly complex. During early fetal development, different types of neurons are generated in different brain areas, and they need to migrate to the right location and then form connections with other neurons through axonal outgrowth and synapse formation (Stage 1 in Fig. 1A). These processes are driven by specific genetic programs (as will be expounded in section 3.1.1), but this does not entail that the development of the neuronal networks is fully under genetic control. Activation of developing neuronal networks by external stimuli plays a critical role. After developing neurons have made initial synaptic connections, the activity and the connectivity of a brain circuit is further optimized for the type of information the circuit is processing (Stage 2 in Fig. 1A). Sensory input (from the ears, eyes, etc.) and intrinsic excitability of the immature neural networks engender the neuronal activity that directs synaptic changes in the developing brain. Especially during early stages of development, external input is crucial for the fine-tuning of brain circuits (Hensch, 2005). In each of these processes, timing is critical: foundational input activity needs to be available at the time at which specific circuits emerge (section 3.1.2).

3.1. Genetics

Language problems and DLD often run in families, which implies a genetic component. However, familial risks are ambiguous, as they could point to effects of shared genes and/or a shared environment. Certainly, not all language-related problems can be connected to genetics. For example, poor nonword repetition (i.e., a measure of phonological short-term memory) was found to be highly heritable, but poor auditory processing was not (Bishop, 2002). Also, expressive language skills are highly heritable, whereas receptive skills are not (Law et al., 2009). With respect to DLD, there are several findings that suggest that genetic factors contribute to DLD (den Hoed and Fisher, 2020), such as a higher prevalence in boys than girls, and in monozygotic twins than dizygotic twins (Bishop et al., 1995). However, the observation that DLD has strong hereditary components does not necessarily imply that specific gene variations are responsible for the language problems. As described above, the development of the brain areas that mediate language follows a complex developmental trajectory and involves many genetic programs in parallel that are mutually coordinated. This complexity facilitates successful language development despite the wide genetic variations that occur between individuals. It is therefore not surprising that monogenetic causes of DLD appear rare, and that in most cases complex interactions between different genes and environmental factors account for the observed language problems (Barry et al., 2007; Mets and Brainard, 2017; Mountford et al., 2019; Onnis et al., 2018).

There are specific genetic variations that have been linked to language development in humans (and also song development in songbirds), and some of these variants have been associated with DLD and other language-related disorders such as dyslexia, childhood apraxia of speech, and autism spectrum disorder (Devanna et al., 2018; Graham and Fisher, 2015; Sriganesh and Ponniah, 2018). Most of the identified

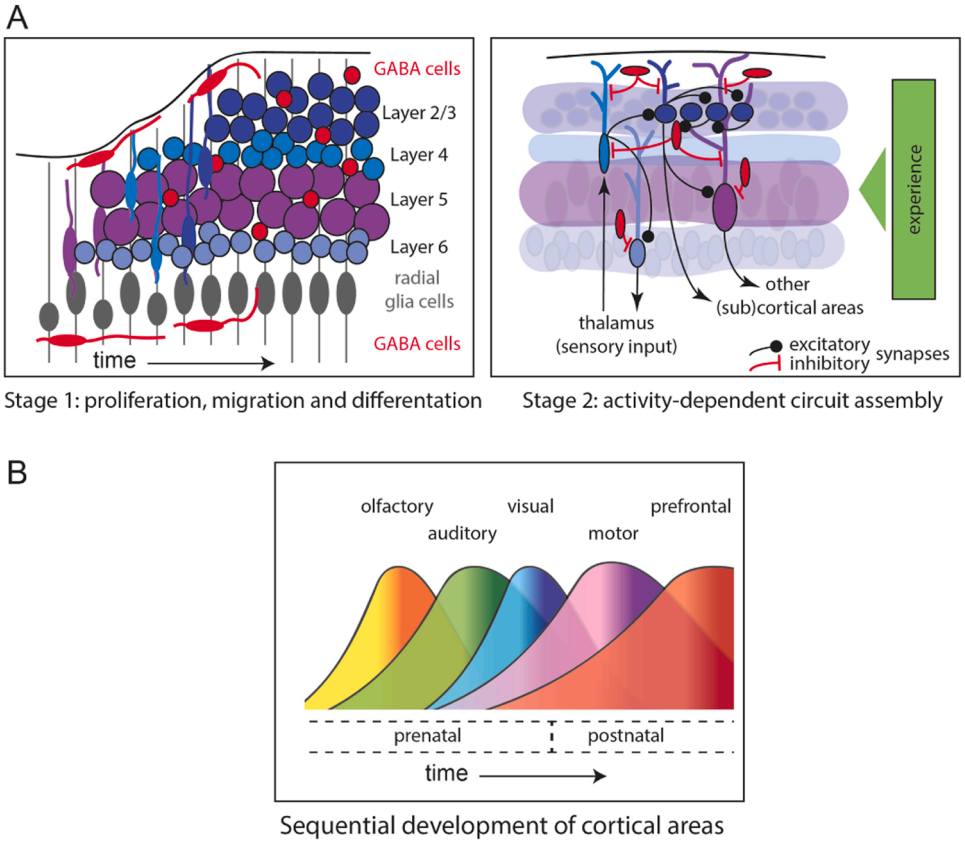

Fig. 1. Sequential development of cortical areas. A: The development of a cortical circuit consists of two stages. During stage 1, principal (excitatory) neurons are generated from radial glia cells and migrate to the cortical layers (migrating cells are depicted with elongated leading neurites). Later born neurons migrate through layers of early born neurons. GABAergic cells are generated outside of the cortex and migrate tangentially into the developing cortex. During stage 2, the activity of the neurons within a circuit shapes the excitatory and inhibitory connections to optimize circuit function to process incoming signals. This stage is strongly modulated by experience and is therefore often referred to as a 'sensitive' or 'critical' period. B: Schematic representation of the sequential development of different cortical areas. The olfactory and auditory cortex are relatively well-developed at birth, while the motor cortex and prefrontal cortex undergo extensive postnatal development. Full development of the human brain takes ~20 years.

genes involve transcription or epigenetic factors that influence the expression of other genes (some with an unknown function), consequently leading to a wide variety of symptoms that are shared among various neurodevelopmental disorders (Graham and Fisher, 2015). It is beyond the scope of this review to provide an overview of the genes that have been associated with DLD (for a recent review see den Hoed and Fisher, 2020). Below, we mention two monogenetic examples to illustrate the broad range of potential genetic mechanisms underlying language disorders, emphasizing that these are neither necessary nor sufficient to cause DLD in its full complexity. We focus on these two, as their mechanisms have been relatively well-described. It is important, however, to point out that our current knowledge of genetic factors underlying language impairment is far from complete. More large-scale research is necessary to adequately assess the role of high-risk single variants in DLD.

3.1.1. Examples: FOXP2 and USH2A

A relatively well-known monogenetic etiology is related to *FOXP2*. *FOXP2* variations are associated with problems in speech production (e.g., in childhood apraxia of speech) (den Hoed and Fisher, 2020), phonological working memory (Schulze et al., 2018), sequence learning (Bolhuis et al., 2010; Graham and Fisher, 2015; Onnis et al., 2018), and receptive and expressive language (Fisher and Scharff, 2009; Onnis et al., 2018). *FOXP2* is widely expressed in sensory, limbic and motor areas in the brain, and there is a strong functional correspondence between expression patterns in songbirds and humans (Vargha-Khadem et al., 2005). *FOXP2* is a transcription factor that regulates the expression of other genes including *CNTNAP2* and *VLDLR* (Graham and Fisher, 2015; Onnis et al., 2018). Variation in FOXP2 target genes or in binding partners of FOXP2 has been linked to language problems (Bates et al., 2011; den Hoed et al., 2021; Wang et al., 2015; Whitehouse et al., 2011). FOXP2-related variations likely affect cell migration, axon guidance, and development of the GABAergic system in the developing brain, which could ultimately result in altered neural plasticity of the motor-speech circuit.

Another gene which may be related to language difficulties is *USH2A* (Perrino et al., 2020), which acts via a completely different mechanism compared to *FOXP2*. Homozygotic pathogenic *USH2A* mutations cause congenital hearing loss. However, heterozygous carriers are not diagnosed with hearing loss, but can develop (sometimes unnoticed) deficiencies in auditory processing, which increase the risk of language problems. In this case, the source of the altered auditory processing is likely in the cochlea and not in the brain (Perrino et al., 2021).

3.2. Input and timing in brain development

As mentioned, the interaction between brain activity, synaptic development, and environmental input is essential for the optimization of neural connections and the refinement of brain function. Indeed, numerous studies have shown that animals that are raised with no or limited sensory input show long-lasting, perhaps permanent, deficits in sensory processing, due to reduced or improper synaptic connectivity in essential brain areas (Kang et al., 2013; Kreile et al., 2011). Early life experience is thus highly important in shaping cognitive abilities later in life (Bijlsma et al., 2022; Jones et al., 1996).

Importantly, different brain areas mature at different times during pregnancy and postnatal development (Hensch, 2005; Stiles and Jernigan, 2010; Tau and Peterson, 2010). For instance, when babies are born, they already possess a well-developed sense of smell, their auditory acuity is reasonably good, but their vision is rather poor. This corresponds to the degree of maturation of the corresponding brain areas (i.e., the olfactory cortex develops before visual cortex; Fig. 1B). Furthermore, brain areas that process primary sensory input mature before brain areas which process more complex information, or which combine information from multiple senses ('association' areas). As a result, the developing brain gradually becomes sensitive to more complex aspects of sensorimotor experience. The successive maturation of specific brain regions results in so-called 'sensitive' (or 'critical') periods in the early life of a child, when the developing neuronal circuits are particularly sensitive to external input. In this way, early life experiences have a strong influence on brain development, with enduring consequences for adult brain function. An overview of the main brain areas that are involved in language development is given in Fig. 2.

Consistent with the critical role of external input in brain development, child-external environmental factors are crucial for acquiring language. Children's language learning mechanisms are sensitive to the amount of language they hear and use, as well as to the richness of language input (e.g., lexical diversity, number of different speakers (Hoff, 2006)) and own output (Blom et al., 2023; Bohman et al., 2010). The spoken language that young children are exposed to co-determines the structure of the neural circuits that are involved in speech perception (Kuhl, 2010; Romeo et al., 2018). For example, Pierce and others found that the neural processing of French in monolingual French children who were adopted from China was affected by exposure to Chinese in the first years of life (Pierce et al., 2015). Timing of input appears essential, as is illustrated by the finding that internationally adopted children experience difficulties in acquiring the language spoken by their adoptive parents. These children have lower language outcomes (though not reaching clinical levels) than their non-adopted peers, even when they were adopted early in life (Delcenserie and Genesee, 2014; Scott et al., 2011). In addition, children who are born deaf can develop normal language skills when they receive cochlear implants at an early age. However, when implantation takes place at a later age, the brain has already developed in absence of auditory input and cannot fully adjust anymore (Szagun and Schramm, 2016).

3.3. Summary

Complex interactions between biological (including genetic) and environmental factors are essential for the development of brain areas that underlie language acquisition. The complex and multidimensional character of language acquisition requires proper and properly timed development of multiple brain areas and functions, indicating that different neurocognitive mechanisms and processes are involved (Donnelly and Kidd, 2020). If some of those are disturbed, impairing potential routes towards successful language development, a child will develop severe and persistent language problems (Bishop, 2006).

4. Risk factors: general impact on brain development

Risk factors may affect several brain functions in parallel and therefore can have a general effect on neurodevelopment. Even if risk factors affect specific neurotransmitter systems or specific brain regions, their local effects (e.g., within a brain area or perception modality) may be small and can stay unnoticed. The suboptimal functioning or small defects in information processing may be well tolerated or perhaps compensated. However, as alluded to in the previous section, the development of different brain functions and structures is tightly interdependent. This means that the accumulated effect across the entire brain can result in the emergence of global deficiencies in cognitive and social abilities, including learning and using language. Indeed, language development depends on proper functioning of many general neurocognitive functions such as perception, motor control, memory, attention and executive functions. Altered, suboptimal performance in one or more of these functions can then culminate in language defects without a traceable underlying cause. In this section, we will describe risk factors associated with underlying mechanisms that impact brain development in general, and we will specifically focus on their impact on language development.

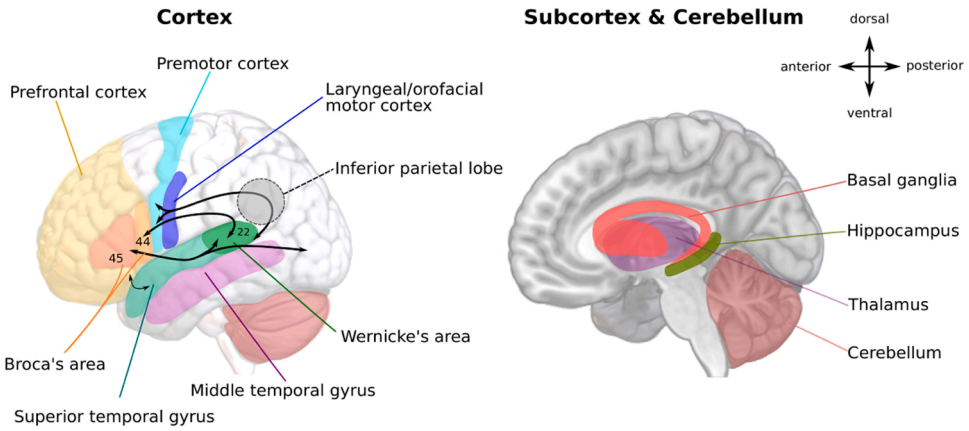

Fig. 2. Brain regions involved in language. Human brain areas relevant for speech and language processing. Left: Cortical language regions in the left hemisphere are connected through multiple pathways (black arrows) (Friederici and Gierhan, 2013). The most dorsal pathway connects the premotor cortex (PMC) with the superior temporal gyrus (STG) and is involved in speech repetition. The second dorsal pathway connects posterior Broca's area (Brodmann 44) with Wernicke's area and supports complex syntactic processing. The ventral pathway between frontal operculum and STG is involved in more basic syntactic processing (finite state and local phrase structure). The second ventral pathway connects frontal, temporal and occipital regions and is associated with lexico-semantic processing. The prefrontal cortex regulates executive functioning (Barde et al., 2012), affecting 'lower-level' processes associated with language and speech. Right: Regions in the subcortex and cerebellum relevant for language, speech, sequence processing and memory. Basal ganglia (including putamen and caudate nucleus) are important for sequential learning, fine motor control, working memory and executive control (De Diego-Balaguer et al., 2008). The thalamus and hippocampus are relevant for memory and sleep (memory consolidation, strengthening connectivity (Paller et al., 2021). The cerebellum is involved in a variety of speech, language and motor functions, particularly through the coordination and modulation of cortical functions (Mariën and Borgatti, 2018). For a more detailed description of the development of brain regions that are involved in language, we refer the reader to (Skeide and Friederici, 2016).

4.1. Sex differences

4.1.1. Link with language development

Males are at greater risk for neurodevelopmental cognitive deficits, including (persisting) language delays and being diagnosed with DLD, compared to females (Bale, 2016; Cheuk and Wong, 2005; Dale et al., 2003; Harrison and McLeod, 2010; Law et al., 2009; Mossabeb et al., 2012; Prathanee et al., 2007; Stanton-Chapman et al., 2002; Whitehouse, 2010; Whitehouse et al., 2012b; Wilson et al., 2013). Also, in typical language development (TD), there are differences between male and female children. In the first year, male infants have been reported to produce higher amounts speech-like vocalizations than female infants (Oller et al., 2020). However, when examining children's scores on a variety of language measures, females often outperform males (Bornstein et al., 2004; Chen et al., 2009; Lutchmaya et al., 2002b; Whitehouse, 2010), although this difference may decrease with age (Lange et al., 2016).

4.1.2. Possible mechanisms

Male children tend to be less social (e.g., make less eye contact) than females (Chapman et al., 2006; Lutchmaya et al., 2002a), and participate less in joint attention (Olafsen et al., 2006; Tomasello et al., 2005). Male infants' slight disinclination to engage with other people may affect how parents and peers interact with them, potentially affecting input quantity and quality. However, parenting behavior directed to male or female infants does not appear to differ (Endendijk et al., 2016) and mothers do not speak to female infants more frequently than to male infants (Huttenlocher et al., 1991).

Sex-dependent differences in brain development may underlie sex-dependent vulnerability for genetic and environmental risk factors (McCarthy and Wright, 2017). The difference between the development of male and female brains is largely determined by sex hormones (Kelava et al., 2022; McCarthy, 2016a). Hormonal surges during early development called 'minipuberty' regulate brain masculinization and other developmental processes (Bale, 2016; McCarthy and Wright, 2017). Male but not female fetuses secrete large amounts of testosterone starting at approximately three months of pregnancy and between one and three months after birth (Huhtaniemi, 1989; McCarthy, 2016b). In females, several early estrogen waves occur during the first six months (Lanciotti et al., 2018). Accordingly, sex-dependent differences in language and brain development have been associated with differences in the levels of sex hormones circulating in the blood. For example, phonological discrimination responses were found in male and female 4-week-old infants with low levels of testosterone, but not in male infants with high testosterone (Friederici et al., 2008). Articulatory skills and phoneme discrimination in 5-month-old infants, and 4-year-old children's sentence comprehension are negatively correlated with their concurrent testosterone and positively correlated with their estradiol levels (Quast et al., 2016; Schaadt et al., 2015). Sex differences in language may be related to the observation that testosterone levels are negatively, and estradiol levels are positively associated with grey matter volume (GMV) in the left inferior frontal gyrus (including Brodmann area 44; see Fig. 2), a brain area important for language (Witte et al., 2010). The influence of circulating sex hormones on neural structure was clearly demonstrated in a study in which testosterone was given to female-to-male transsexuals. This was found to reduce GMV specifically in Broca's and Wernicke's areas and enhance the white matter tracts between the two areas (Hahn et al., 2016), which are part of the ventral language pathway (see Fig. 2). Fetal testosterone levels during pregnancy have been associated with differences in GMV, both between the sexes and within males, particularly in brain regions that overlap with Wernicke's area (Lombardo et al., 2012; Marrocco and McEwen, 2016; van de Beek et al., 2004).

Animal research supports the role of steroid hormones in neuroanatomical and behavioral changes. Grey matter differences may be explained by sex-specific differences in the neural microstructure, for instance in the number of synapses between male and female rats (McCarthy, 2016a). Testosterone affects neurite outgrowth, branching and synapse formation via stimulating GABA synthesis and enhancing the depolarizing action of GABA (McCarthy et al., 2009, 1997; VanRyzin et al., 2019), which is an important factor directing early brain development (Peerboom and Wierenga, 2021). In songbirds, a set of brain nuclei specialized for song learning (the 'song system') often, although not always, differs between sexes (Remage-Healey et al., 2010). The development of these regions (and thus behavior) is guided by a combination of genetic differences and sex steroids produced within the

brain and in the gonads. Sex hormones (e.g., steroids) affect brain plasticity for song development (including timing of the sensitive periods), where they can induce growth or shrinkage of brain nuclei that control song (Ball and Macdougall-Shackleton, 2001; Simpson and Vicario, 1991). Effects on brain plasticity include neurogenesis, neuron migration, apoptosis, increased cell soma (Wade and Arnold, 2004), dendritic spines (Vellema et al., 2019), sensorimotor connections (Wade and Arnold, 2004), and extracellular matrix components (e.g., perineuronal nets) related to sensitive periods (Balmer et al., 2009; Cornez et al., 2018). Evidence from songbirds also shows that white matter changes are dependent on hormones, as local estrogen production regulates synaptic connectivity between song system nuclei responsible for learnt vocal production (Holloway and Clayton, 2001).

At the behavioral level, animal studies report findings relevant to language acquisition. Auditory experience itself induces hormonal surges (estrogen) in the juvenile male songbird's brain, which in turn affect auditory behavior (Maney and Pinaud, 2011). Auditory learning is present also in non-singing females, but specific sex differences exist in auditory processing (Giret et al., 2015; Gobes et al., 2009; Krentzel and Remage-Healey, 2015) and auditory learning may be affected independent of production learning (Vahaba et al., 2020). Effects of hormones on production quality and quantity of song have been found (Meitzen et al., 2007; Van Hout et al., 2012). Testosterone can also promote singing motivation in songbirds (Alward et al., 2013; Shevchouk et al., 2017). Possibly, the higher abundance of babbling production in male human babies could be explained by similar hormonal influences, but this link has not been studied as far as we know.

4.1.3. Summary and conclusion

Males are at greater risk for language problems and DLD compared to females. There are important sex-dependent differences in brain development and brain structure, which can affect the specific vulnerability for risk factors. Sex differences largely depend on the presence (or absence) of the sex hormones, which affect many specific aspects of cellular and synaptic development. Interactions between timing of hormonal surges, genetic expression and (auditory) input may guide the timing of developmental processes relevant for language learning.

4.2. Family composition

4.2.1. Link with language development

Children who are born as the second or later child in the family are more likely to be diagnosed with DLD than firstborns (Diepeveen et al., 2017; Harrison and McLeod, 2010; Prathanee et al., 2007; Stanton-Chapman et al., 2002). Later-born TD children have been found to perform worse on standardized language tests than firstborns (Kampouri et al., 2018; Prathanee et al., 2007; Reilly et al., 2010). This underperformance does not seem specific to language, as similar effects of birth order are also found in other domains of neurocognitive development (Kristensen and Bjerkedal, 2007). Interestingly, being born as a middle or lastborn child increases the risk of expressive language difficulties, whereas receptive language skills may be protected by having older siblings (Harrison and McLeod, 2010). Family size has also been identified as a risk factor for DLD: a higher number of children in the household increases the risk of children having language problems (Choudhury and Benasich, 2003).

4.2.2. Possible mechanisms

There are multiple explanations for the abovementioned findings. We highlight two possibilities: (1) Children born later may have more adverse prenatal and perinatal factors as a result of increased parental age; (2) Children born earlier in the family or children from smaller families may receive more stimulation from their parents (Black et al., 2018; Kristensen and Bjerkedal, 2007; Stanton-Chapman et al., 2002).

4.2.2.1. Parental age. Some studies found that increased maternal age (>30 years) is associated with a higher risk of DLD, although effect sizes were small (Choudhury and Benasich, 2003; Diepeveen et al., 2017). Other studies did not find this association (Cheuk and Wong, 2005; Mossabeb et al., 2012; Stanton-Chapman et al., 2002; Whitehouse et al., 2014). There is stronger evidence that older fathers (>40 years) increase the risk of DLD (Cheuk and Wong, 2005; Choudhury and Benasich, 2003). Increased paternal age has been associated with sperm and DNA abnormalities, epigenetic changes, increased risk of spontaneous abortions, preterm birth and stillbirth, and neurocognitive disorders in offspring, including autism and schizophrenia (Sharma et al., 2015). However, in a large cohort study, increased paternal age was found to be associated with lower maternal education, which is a risk factor for DLD (Stanton-Chapman et al., 2002) and maternal education can thus be a confounding factor (Edwards and Roff, 2010). More research is needed to determine the role of paternal age on the risk of DLD in children.

4.2.2.2. Social environment and stimulation. A study by Kristensen and Bjerkedal (2007), who investigated the relationship between intellectual abilities and birth order by looking at children's biological rank versus their social rank, underlines the important role of home environment as a possible underlying mechanism for the effects of birth order. In this study, the children's biological rank was defined as the order they were born in, whereas social rank was their order in the family. For example, children who are born as a second child have a biological rank of a second child, but not necessarily the social rank of a second child if their older sibling passed away (which can result in a situation in which they are more raised like the eldest than a second child). Kristensen and Bjerkedal found an association between intelligence and social rank, and not with biological rank. Given the influence of social rank on children's social environment and parental stimulation, this suggests that postnatal environmental differences are important when explaining the relation between birth order and neurocognitive abilities (Kristensen and Bjerkedal, 2007).

Birth order effects may account for differences in the social environment and stimulation of children with DLD in comparison with TD peers, although this has not yet been studied directly. For example, children with DLD are less likely to have attended day care, which can be a stimulating environment, than TD children (Law et al., 2009). Moreover, children with DLD are less likely to have parents who read to them, tell them stories, discuss daily activities or feelings, or teach the alphabet (Hammer et al., 2001). Children with DLD also experience negative interactions with their parents (e.g., disciplining and time-out) more often than TD children (Hammer et al., 2001). As children with DLD are more often later-borns (see 2.2.1.) and as parents on average invest less in later-borns (Black et al., 2018), these environmental differences could potentially be related to, and in part explain, the effects of birth order.

Regarding the role of family size, relevant research in male zebra finches shows that nests with larger brood size induced lower accuracy in 'birdsong syntax' learning (sequence of notes) and lower consistency of song motif duration. Both may be a result of competition in their social environment and restrictions in their early nutritional environment (Holveck et al., 2008). Within-nest competition for resources may result in stress and therefore have a negative influence on song learning and song quality. Something similar has been suggested in humans, as having more children in the family results in competition for parental attention (and thus less stimulation), food resources, and space (e.g., in home crowding) (Black et al., 2018; Kampouri et al., 2018; Law et al., 2009).

4.2.3. Summary and conclusion

Being born later in the family is associated with a higher risk of DLD. Differences in the home environment of firstborns versus later-borns may play a role in this association, as being born later in the family can mean more limited parental stimulation and resources.

4.3. Nutrition: breastfeeding

4.3.1. Link with language development

Children with DLD are less often, or for a shorter period of time, breastfed than unaffected children (Diepeveen et al., 2017; Harrison and McLeod, 2010; Prado and Dewey, 2014; Prathanee et al., 2007; Tomblin et al., 1997). In the general population, exclusive breastfeeding, and to a lesser extent partial breastfeeding, is associated with higher scores on tests for cognition, communication, social interaction, and motor development at later ages (Choi et al., 2018; Oddy et al., 2011; Park et al., 2016; Tomblin et al., 1997; Vestergaard et al., 1999). Next to breastfeeding, there are also other nutritional effects on neurocognitive development. For example, childhood malnutrition has been widely studied, and is known to depress motor, language and cognitive skills later in life (Galler et al., 1984; Grantham-McGregor, 1995; Khandelwal et al., 2020; Laus et al., 2011). Moreover, many animal studies have shown that the gut environment plays an important role in neuronal development (Morel et al., 2023) which may also impact cognition. In the current section, we focus on breastfeeding, as this has been most clearly related to DLD in the literature.

4.3.2. Possible mechanisms

Below, we discuss two different mechanisms that may explain the link between breastfeeding and neurocognitive development, including language. First, children who are breastfed may have a stronger bond with their mother and may be less easily distracted than children who are not breastfed. Second, breast milk contains important nutrients that support neurodevelopment.

4.3.2.1. Mother-child communication and child-internal factors. Breast-feeding facilitates face-to-face communication, strengthening the emotional bond between mother and child. The breast-feeding induced bonding effect has been demonstrated in both young children and adolescents (Choi et al., 2018; Diepeveen et al., 2017; Linde et al., 2020; Prado and Dewey, 2014). A stronger bond between mother and child may ultimately lead to enhanced social skills, in humans as well as in other mammals (Mogi et al., 2011). When this bond cannot be formed, for example in mice that are weaned during the lactation period, stress responses occur and myelin formation in the brain and neurogenesis are impaired (Mogi et al., 2011).

The most prominent reasons for feeding with formula, instead of breastfeeding, is that infants are either unable to latch on properly due to anatomical constraints or are too eager (i.e., infants are too enthusiastic and get too easily distracted) (Feenstra et al., 2018). Children with DLD often have comorbid attention deficits (Kovac et al., 2001; McGrath et al., 2008), which may be linked to difficulties with breastfeeding. To our knowledge, there has not yet been a retrospective study that has investigated specific reasons why mothers with children with DLD opted for formula-feeding more often than mothers with TD children.

4.3.2.2. Nutrition in breast milk. Breast milk contains specific nutrients, growth factors, hormones, and prebiotics (compounds that stimulate beneficial microorganisms) that support neurodevelopment. Also, during pregnancy, the maternal iron, vitamin A, iodine, thyroid hormones, zinc, choline, thiamine (vitamin B1), and vitamin B12 status are important for the offspring's neuron proliferation, axonal and dendritic growth, synapse formation, pruning, and function, and myelination, which ultimately affects cognitive ability (Park et al., 2016; Prado and Dewey, 2014).

The importance of these nutrients is illustrated by the fact that supplementation of many nutrients (including polyunsaturated fatty acids, vitamin B12, iodine, choline and zinc) for two years in groups at risk for neurological impairment or cerebral palsy has shown to affect cognitive and language development to an extent that was clinically relevant, although not statistically significant (Andrew et al., 2018a, 2018b). With the disclaimer that some observations come from extreme cases and may not necessarily generalize to normal dietary variations, we will discuss what is known about the most important mechanisms for nutrients in breast milk below.

4.3.2.2.1. Polyunsaturated fatty acids. Breast milk contains polyunsaturated fatty acids (PUFAs) and essential fatty acids (EFAs). PUFAs are required for membrane phospholipid synthesis, myelin synthesis, and synaptic maturation during neurogenesis (Ferguson and Molfese, 2007; Prado and Dewey, 2014). EFAs are acquired through dietary intake and needed for the production of arachidonic acid (AA) and docosahexaenoic acid (DHA), which are important in cellular signaling (van Elst et al., 2014; Wallis et al., 2002). Enrichment of formula with PUFAs or EFAs stimulates the maturation of the auditory pathway in the brainstem in the first weeks of life, resulting in better vocabulary, executive functioning, and speech processing at later ages compared to non-enriched formula (Colombo et al., 2013; Ünay et al., 2004). While promising, enriched formula is still less beneficial for neurodevelopment than breastfeeding and its benefits are dose-dependent (Colombo et al., 2013; Ferguson and Molfese, 2007; Molfese, 2000; Molfese and Molfese, 1985). There is a risk as well: increasing the blood omega-6/omega-3 PUFA ratio too strongly results in developmental delay (i.e., lower bodyweight, delayed puberty) (van Elst et al., 2018). Thus, simply including more PUFAs is not the key. Maintaining a proper fatty acid homeostasis appears to be most important during development.

4.3.2.2.2. Iron. Infants with chronic iron deficiencies score lower on motor, expressive and receptive language, and communication tasks (Beltrán-Navarro et al., 2012; Prado and Dewey, 2014). Iron is required for enzymes that regulate cell division, the maturation and efficacy of dendrites and synapses, and in dopamine and norepinephrine (receptor) metabolism (Prado and Dewey, 2014). Furthermore, anemic mothers tend to be less responsive to their infants due to symptoms such as depression, fatigue, irritability, and poor concentration, which affect mother-child bonding (Ludwig and Kathrin, 2001; Perez et al., 2005) and can, in turn, impact neurocognitive development (Faisal-Cury et al., 2022).

4.3.2.2.3. Iodine and thyroid hormones. Both low and high prenatal iodine intake increases risk for persistent language delays (Abel et al., 2017; Markhus et al., 2018; Zhou et al., 2019). Interestingly, language delays associated with iodine intake correlate with the presence of attention deficit hyperactivity disorder (ADHD) symptoms, a comorbidity often found in children with DLD (Abel et al., 2018; Miniscalco et al., 2006). In general, iodine is used to produce thyroid hormones, needed for proper cell metabolism. Research in zebra finches suggests that thyroid hormone levels in the brain increase during early development in a sex-dependent manner (Yamaguchi et al., 2017). Furthermore, thyroid-related gene expression is high in song regions in the brain during song learning (Raymaekers et al., 2017). Prenatal and early postnatal iodine deficiency results in lower brain weight and lower cell numbers, impairs cell migration in humans and animal models, and leads to reduced dendritic branching, synaptic density, and myelination in animal models (Prado and Dewey, 2014). Myelination and thyroid hormones have both been associated with sensitive periods for learning (McGee et al., 2005; Yamaguchi et al., 2012).

4.3.2.2.4. Vitamines. Vitamin A (retinol). Higher levels of vitamin A in TD infants are associated with better motor skills at two years of age (Chen et al., 2009). Vitamin A may affect language development through its role in the production of retinoic acid. More specifically, retinoic acid is important during neurogenesis and in specific forms of synaptic plasticity (Sarti et al., 2013; Shearer et al., 2012; Zhong et al., 2018). In adult zebra finches, exposure to high doses of retinoic acid during song learning results in less stable songs (Wood et al., 2008).

Vitamin D. Maternal vitamin D insufficiency is linked to persistent language impairment in children (Whitehouse et al., 2012a), and poor motor and social development (Darling et al., 2017; Whitehouse et al., 2012a). It also increases the risk for diseases such as asthma and psychiatric disorders such as ASD (Kaushal and Magon, 2013; Kočovská

et al., 2012; Vinkhuyzen et al., 2017). However, some studies did not find an association between maternal vitamin D concentration and child behavioral or affective disorders, school achievements, growth, and neurodevelopment (Strøm et al., 2014; Wang et al., 2018). Vitamin D has been associated with cortical thickness, mitosis, apoptosis, neurogenesis, neurotrophic factors, and gene expression. Similar to iron deficiency, maternal vitamin D deficiency affects the bonding between mother and child due to less maternal care as shown in a rat study (Yates et al., 2018).

Vitamin B1 (thiamine). Infants who were fed with a thiamine-deficient formula for at least one month before the age of 1 year (due to an error at the company producing the formula) experienced many nonspecific symptoms such as cardiomyopathy and neural hyperintensities, but also motor and (particularly syntactic) language impairments (Fattal et al., 2011; Fattal-Valevski et al., 2009, 2005). It is thought that thiamine deficiency causes neurodegeneration and oxidative stress (Y. Liu et al., 2017; D. Liu et al., 2017). Early life thiamine deficiencies in rats also affect GABA and glutamate levels in the thalamus, hippocampus, and prefrontal cortex (de Freitas-Silva et al., 2010).

Folic acid. It is well-established that deficiencies in folic acid during pregnancy are associated with neural tube defects, anencephaly, and spina bifida (Prado and Dewey, 2014). Prenatal supplementation with folic acid is also positively associated with children's neurodevelopment, vocabulary, and receptive and expressive language development (Gao et al., 2016; Roth et al., 2011).

4.3.3. Summary and conclusion

Breastfeeding is beneficial for language development (and neurocognitive development in general) and lowers the risk for DLD. This effect may be linked to the mother-child bond, child-internal factors and nutrients in the breast milk. With respect to the latter, the effect cannot be traced back to one specific ingredient. Maternal and infant early deficiencies in nutrients can have long-lasting effects on cognitive, social, and language development, but do not seem specific to language.

4.4. Exposure to toxic substances

4.4.1. Alcohol

Prenatal exposure to drugs such as alcohol, cocaine, heroin, and marijuana has been associated with persisting deficits in cognition, hearing, receptive language, semantic skills, phonological processing abilities, syntactic maturity, and locomotor development (Bandstra et al., 2011; Buckingham-Howes et al., 2013; D'Apolito, 1998; Delaney-Black et al., 2000; Lehikoinen et al., 2016; Lewis et al., 2013). Drug abuse during pregnancy affects global neurodevelopment through interactions with many different neurotransmitter systems and often affects the stress system. However, effect sizes are often small after adjusting for confounders such as consumption of multiple drugs together, tobacco usage, or socio-economic status (Buckingham-Howes et al., 2013). In the first part of this section, we focus on prenatal alcohol exposure, for which the most information is available. Exposure to tobacco smoke can occur pre- and postnatally and may have a specific risk for auditory processing. We therefore consider that the mechanism underlying exposure to tobacco smoke can be specific for language impairment and this will be discussed in Section 5.2.

4.4.1.1. Link with language development. High prenatal alcohol exposure will perturb anatomical, cognitive, and language development, as is clear from the extreme case of fetal alcohol spectrum disorder (FASD) (Cone-Wesson, 2005), which leads to a broad range of deficits in receptive and expressive language skills, including vocabulary, grammar, and narrative skills (Church and Kaltenbach, 1997; Mattson et al., 2019; McGee et al., 2009; Terband et al., 2018; Wyper and Rasmussen, 2011). Results of studies on language development after exposure to more moderate levels of alcohol have been inconsistent, which is most likely due to variations in exposure quantity. Some studies report that children who are exposed to alcohol prenatally are more likely to have speech and language impairments (Coggins et al., 2007; Mattson and Riley, 1998; Terband et al., 2018; Weinberg, 1997), especially in combination with postnatal risk factors such as neglect (Coggins et al., 2007). Other studies do not find such associations (Greene et al., 1990; Stanton-Chapman et al., 2002; Tomblin et al., 1997; Whitehouse et al., 2014; Wilson et al., 2013). The findings on children's neuropsychological outcomes after low to moderate prenatal alcohol exposure are also inconclusive (Comasco et al., 2018; Gray et al., 2009). Although exposure at any time during pregnancy may delay neurobehavioral development (D'Apolito, 1998; May and Gossage, 2011; Sarman, 2018), the second half of pregnancy may be most critical for language (Mattson and Riley, 1998). Children who were exposed to alcohol only during the first trimester showed increased thrill-seeking behavior, but no cognitive or language deficits (Halliday et al., 2017; Nulman et al., 2004).

4.4.1.2. Possible mechanisms. Alcohol does not have a specific receptor within the brain and therefore does not appear to affect one brain area more than others. Alcohol can perturb DNA methylation, which regulates expression of many genes involved in neuronal differentiation, axon guidance, neuronal excitability, neuroinflammation, neurodegeneration, and cell adhesion (Frey et al., 2018). Alcohol also modifies several neurotransmitter systems. For instance, alcohol is a potent inhibitor of NMDA receptors (Hoffman et al., 1990), which are essential for (developmental) synaptic plasticity (Naassila and Pierrefiche, 2019). Furthermore, prenatal alcohol exposure has a life-long effect on the density of specific GABAergic interneurons in a cell- and brain region-specific manner (Kenton et al., 2020; Smiley et al., 2019). In addition, alcohol can damage outer hair cells in the cochlea, affecting sensory development (Church and Kaltenbach, 1997; Cone-Wesson, 2005; Sarman, 2018).

4.4.2. Phtalates

Developing children may also be exposed to toxic substances that come from their living environment. For instance, young children may be exposed to metals (e.g., from lead-containing paint on the walls of their home), to poisonous chemicals from car fumes from traffic near their homes, or to chemicals in their food or water. Many of these toxins interfere with synaptic transmission or directly induce cell death (e.g. Ramírez Ortega et al., 2021). In the second part of this section we focus on a special type of chemicals called phthalates, which are currently widely used to make polyvinyl chloride (PVC) plastics more flexible. Children can get exposed through food items that come in plastic containers, but phthalates can also be present in pesticides (and end up in food), and in the domestic environment (e.g., vinyl flooring). Phthalates remain relatively unknown as risk factors, but we discuss here how they appear to specifically affect endocrine function during neural development.

4.4.2.1. Link with language development. Both prenatal exposure to phthalates via maternal ingestion and subsequent crossing of the placenta (Saillenfait et al., 1998) and postnatal exposure via breast milk and food (Bornehag et al., 2018) have been associated with suboptimal neurobehavioral development. Findings include increased externalizing problems in children (e.g., aggression), increased internalizing problems (e.g., decreased attention, increased depressive symptoms), a higher prevalence of conduct problems, psychomotor delays, and cognitive difficulties (e.g., lower IQ, slower processing speed, lower scores on perceptual reasoning, working memory, and verbal comprehension) (Engel et al., 2010; Factor-Litvak et al., 2014; Whyatt et al., 2012; Yolton et al., 2011). However, some studies did not find any associations with cognitive development (Huang et al., 2015; Polanska et al., 2014; Téllez-Rojo et al., 2013). The strongest correlation with language development defects have been reported when exposure to phthalates

during the third trimester was considered (Bornehag et al., 2018; Jensen et al., 2019; Olesen et al., 2018).

4.4.2.2. Possible mechanisms. Phthalates have been shown to increase oxidative stress in a dose-dependent manner (Ma et al., 2015; Tang et al., 2015). In cell cultures, they suppress neurocyte proliferation and alter neuronal differentiation (Chen et al., 2011). The administration of antioxidants, such as vitamin E and melatonin, reduce oxidative stress and can reduce the adverse effects of these neurotoxic factors (Ma et al., 2015; Tang et al., 2015). The effects depend on the type of phthalates and are sex-dependent. Some studies report a specific susceptibility for male infants (Kim et al., 2011; Weiss, 2012; Yolton et al., 2011), while others report stronger effects in females (Téllez-Rojo et al., 2013; Whyatt et al., 2012). Animal studies may provide insight here. They show that phthalate exposure during gestation and lactation reduces testosterone production in the fetal testes in rats, reducing masculinization of the brain and reducing aromatase activity, which converts androgens into estrogens (Andrade et al., 2006; Weiss, 2012). This appears to be different for different types of phthalates (Chen et al., 2014). Other potential mechanisms include interference with thyroid hormone production, and disruption of dopaminergic activity (Matsuda et al., 2012; Weiss, 2012). Although the link with language development is not directly clear, alterations in the hormonal regulation of brain development may change vulnerability for language problems later in life.

4.4.3. Summary and conclusion

Alcohol exposure appears most harmful during the second half of the pregnancy, particularly when dosage is high. Alcohol affects global neurodevelopment through interactions with many different neurotransmitter systems and DNA methylation, thereby changing expression levels of many genes that are involved in neuronal development. These interactions interfere with normal neurodevelopment including speech and language development.

Chemical compounds in the environment can have negative effects on a child's behavioral, cognitive, and language development. Exposure to plasticizers, notably phthalates, during the third trimester of pregnancy is specifically linked to impaired language development. Males are more susceptible to environmental toxicity, possibly via dysregulation of sex hormones.

4.5. Maternal health problems

In this section, we will consider physical health, mental health, and the use of antidepressant drugs of the mother. Depression in adults is often medicated with antidepressants, including Selective Serotonin Reuptake Inhibitors (SSRIs). Maternal prenatal depression and antidepressant medication both affect postnatal language development in the child, but via different underlying mechanisms. Therefore, these mechanisms will be discussed separately.

4.5.1. Maternal physical health

4.5.1.1. Link with language development. Maternal physical health during pregnancy has been shown to have a potential impact on a child's language development. For example, maternal high pre-pregnancy Body Mass Index (BMI), diabetes during pregnancy, preeclampsia, and hypertension increase the risk for persistent language deficiencies, and delays in motor and social-behavioral development (Adane et al., 2016; Battin et al., 2018; Chen et al., 2009; Dionne et al., 2008; Jo et al., 2015; Perna et al., 2015; Torres-Espínola et al., 2015). However, such associations were not observed in two studies investigating the risk for DLD (Diepeveen et al., 2017; Whitehouse et al., 2014).

4.5.1.2. Possible mechanisms. One factor that has been suggested to play a role in maternal disorders such as diabetes during pregnancy and preeclampsia, and subsequent effects in infants, is placental insufficiency. In these disorders, the development of the placenta is disrupted and the transfer of oxygen and nutrients to the fetus is reduced (Meakin et al., 2017). Although the exact mechanisms are unclear, fetal hypoxia and malnutrition alter neural growth and affects the maturation of the fetal auditory system (Kisilevsky, 2016; Perna et al., 2015; Werker and Hensch, 2015). This may impede infants' learning from (prenatal) speech input, as it has been found that growth restricted fetuses, fetuses from diabetic mothers, and from hypertensive mothers are less capable of recognizing their mother's voice *in utero* and postnatally (Kisilevsky, 2016). Delays in auditory development also interfere with coordinated development across brain regions which is required for language development (Werker and Hensch, 2015).

4.5.2. Maternal mental health

4.5.2.1. Link with language development. High levels of maternal stress (due to, e.g., adverse life events, anxiety, depression) during the first two trimesters is associated with increased risk for delayed cognitive functioning, and delayed expressive and receptive language development in the infant (Harrison and McLeod, 2010; Ibanez et al., 2015; Kawai et al., 2017; King and Laplante, 2005; Skurtveit et al., 2014). Kaplan and colleagues found a delay in expressive language but not in receptive language or general cognitive ability (Kaplan et al., 2014). Research furthermore showed that prenatal stressors in animals, especially in the first trimester, affect offspring's motor, attention, and memory development (King and Laplante, 2005; van den Berg et al., 2017), in line with studies with humans (e.g., Graignic-Philippe et al., 2014), illustrating that stress has a broader impact beyond language problems.

4.5.2.2. Possible mechanisms. First, prenatal stress, anxiety and depression increase cortisol levels, which have a broad range of effects in the fetus. Prenatal maternal stress in animals, in particular around the time of maximal brain growth, heightens postnatal cortisol responses to stress in the offspring (Hartman et al., 2018; Kapoor et al., 2006; Ornoy, 2017; Sohr-Preston and Scaramella, 2006). Increased postnatal stress activation interferes with processes that are important for learning, such as attention, executive functioning, and memory (Sohr-Preston and Scaramella, 2006). For instance, it impairs the filtering of repeating auditory stimuli (i.e., auditory gating), which would otherwise be considered redundant and not facilitating the learning process (Maxwell et al., 2006). These findings suggest that children of mothers who were anxious or depressed during pregnancy may thus be impaired in postnatal attending to and memorizing of auditory stimuli relevant for learning language.

Second, mothers with prenatal depression or anxiety are likely to continue to have depressive symptoms after birth (Ibanez et al., 2015; Sohr-Preston and Scaramella, 2006; Stein et al., 2008), resulting in stress in their babies. In animal studies, prolonged increased levels of the stress hormone glucocorticoid in early life has life-long effects on learning and memory (Derks et al., 2016; Xiong et al., 2016). In addition, postnatal depression reduces a mother's inclination to stimulate her child. Depressed parents engage less with their children, use less infant-directed speech and read less to their children (Paulson et al., 2009; Sohr-Preston and Scaramella, 2006). This affects the language and cognitive abilities of their children and increases the risk of DLD (Ahun et al., 2017; Castelli et al., 2015; Ibanez et al., 2015; Kawai et al., 2017; La Paro et al., 2004; D. Liu et al., 2017; Y. Liu et al., 2017; Paulson et al., 2009; Quevedo et al., 2011; Reck et al., 2018; Valla et al., 2016). Interactions may be especially important up to 6 months of age, when children acquire fundamental language-related perceptual skills (Werker and Hensch, 2015). Some studies suggest that it is possible to prevent negative effects of parental depression on language development with a highly stimulating home environment (Ahun et al., 2017; Piteo et al., 2012; Quevedo et al., 2011), as children who experience

prenatal stress are more susceptible to environmental influences and thus are likely to benefit from environmental support (Pluess and Belsky, 2011).

A final possible mechanism implicates a disturbance in the sequence of specific aspects of language development (Werker and Hensch, 2015). As described in Section 1, language acquisition requires coordinated developmental changes in multiple brain regions (Werker and Hensch, 2015), and each brain specialization has its own sensitive period (Hensch, 2005) (Fig. 1B). An important early postnatal brain specialization required for language is the transition from a broad sensitivity to all possible speech sounds to a sensitivity narrowed down to the phoneme inventory of the native language (i.e., perceptual narrowing or attunement) (Werker, 2018). Maternal depression delays the closure of the sensitive window for this specialization, presumably due to hormonal changes or a reduced prenatal exposure to engaging speech (Weikum et al., 2012). A delay in recognizing speech sound distinctions that are relevant in the native language (i.e., phonemic) will interfere with (further) language development.

4.5.3. Antidepressants

4.5.3.1. Link with language development. Long-term prenatal exposure to SSRIs, taken by depressed mothers, has been associated with lower language abilities in children, independent of maternal depressive symptoms (Skurtveit et al., 2014), although not all studies have found this association (Diepeveen et al., 2017; Ornoy, 2017).

4.5.3.2. Possible mechanisms. Antidepressant medication, SSRIs, can interfere with the opening and closure of sensitive periods in the fetus. In contrast to maternal depression, which may delay the closure of the sensitive window for perceptual attunement, prenatal exposure to SSRIs accelerates and shortens the period for specific brain specialization (Weikum et al., 2012; Werker and Hensch, 2015). SSRIs increase the levels of serotonin in the brain, and serotonin plays an important role in promoting the maturation of GABAergic connections. Increased serotonin levels in the developing brain via SSRIs therefore enhance inhibitory signaling, which leads to a precocious opening of the sensitive period and enhanced plasticity early in development (Edagawa et al., 2001; Fagiolini et al., 2004; Hensch, 2005; Kojic et al., 2000; Robinson et al., 2003; Werker and Hensch, 2015). More generally, neonates prenatally exposed to SSRIs display functional hyperconnectivity in the auditory network (Rotem-Kohavi et al., 2018). Besides GABAergic signaling, serotonin signaling is also important for other aspects of early neurodevelopment (Olivier et al., 2011), which, in turn, could influence language development. For example, enhanced levels of serotonin during development negatively impact working memory, which depends on the integrity of the prefrontal cortex (Witteveen et al., 2013) and results in alterations in the dopaminergic system (Garcia et al., 2019).

A child's genetic background determines how much they are affected by maternal depression or SSRIs. For example, the gene *SLC6A4*, which encodes the serotonin reuptake transporter that is blocked by SSRIs, has two variants: a long variant resulting in high transporter expression and a short variant which yields low transporter levels. Children with two long *SLC6A4* variants are highly susceptible to the effects of maternal depression. In particular their executive functioning, potentially implicated in language learning, is affected: severe maternal depressive symptoms are inversely correlated with their executive functioning performance (Weikum et al., 2013). In contrast, children with one or two short variants are protected after being exposed to SSRIs prenatally.

4.5.4. Summary and conclusions

Poor physical health of the mother can cause hypoxia and malnutrition, which may affect language development due to altered maturation of the fetal auditory system. Maternal stress affects neural development in utero, but may also affect social interaction with the infant after birth. Maternal depression and antidepressants can affect serotonin signaling, in turn affecting early plasticity during a sensitive phase for phonological development.

4.6. Infections

4.6.1. Link with language development

Viral infections at young age can be dangerous when they lead to encephalitis, causing a broad range of symptoms including sensory, motor, cognitive, language, and speech problems (Tsai et al., 2017). Several viral infections have been associated with speech and language problems in young children (<6 years), including enterovirus infections (EV, virus entering via the intestine) (Hung et al., 2018), Mycoplasma pneumonia (MP, a mild version of pneumonia) (Tsai et al., 2017) and congenital cytomegalovirus (cCMV, a member of the herpes family) (Korndewal et al., 2017). The EV genus is a diverse group of viruses, targeting different areas of the brain (Rhoades et al., 2011). Maternal EVs have also been shown to affect neural development of the fetus (Rhoades et al., 2011). The risk of developing speech and language disorders after an EV or MP infection was reported to be highest when the infection occurs in the first years after birth (Hung et al., 2018; Tsai et al., 2017). Population studies on EV, MP and cCMV did not report specific language or speech disorders (Hung et al., 2018; Korndewal et al., 2017; Tsai et al., 2017). In the remainder of this chapter, we focus on EV, MP and cCMV, but we do not exclude the possibility that other infections may have similar effects, especially if they cause encephalitis.

4.6.2. Possible mechanisms

It is unclear how early life viral infections may lead to speech and language disorders, but there are several candidate neuropathological mechanisms. For example, cCMV has been associated with developmental, sensory and neurological symptoms such as microcephaly, seizures, and sensorineural hearing loss. Neuroimaging studies and animal studies suggest that infections may cause neurological problems via neuronal damage directly resulting from invasion or neurotoxicity, virus-induced apoptosis and neuro-inflammation, as well as disturbed development of neural stem cells (Cheeran et al., 2009; Rhoades et al., 2011). EV and MP (but not cCMV) can induce growth retardation in birds (McNulty et al., 1990), but effects on song learning have not been reported. Human neuroimaging studies of cCMV infections in utero (Hoffmann et al., 2010), as well as adult studies with another herpes virus (Frisch et al., 2015; Utley et al., 1997), suggest that infections are associated with reduced temporal lobe volumes. Several auditory and language-related areas are located in the temporal lobes (see Fig. 2). Severe immune reaction in the (developing) temporal lobes might therefore increase the risk of language disorders.

Infections can elicit autoimmune reactions targeting brain cells or causing vascular problems reducing blood and oxygen supply to the brain (Fu et al., 1998; Tsai et al., 2017). Such autoimmune reactions might also play a role in the link between autoimmune diseases, and increased familial risk for DLD (Choudhury and Benasich, 2003), as has been suggested for neurodevelopmental disorders and ASD (Bilbo et al., 2018; Ellul et al., 2023), especially in male infants (McCarthy, 2019). Evidence from individuals with ASD suggests that altered immunological activation impacts brain development, including neuronal and glial cell migration, differentiation, and synaptic maturation (Bilbo et al., 2018). These alterations might lead to a malformed or malfunctional language network, but how they could induce specific language disorders is not yet clear.

The severity of effects of a cCMV infection strongly depends on gestational age: the earlier the infection, the more severe the effects are. Children who sustained cCMV infections in the first trimester had smaller temporal lobes, compared to healthy controls. Infections in the second trimester resulted in a similar but weaker effect and third trimester infected patients did not differ significantly from controls (Hoffmann et al., 2010). Notably, some cases did not show overt

neuropathology (such as cysts), yet they had smaller temporal lobes than controls (Hoffmann et al., 2010). Children without overt neuropathology can still show long-term neurodevelopmental effects (Yaniv et al., 2016).

4.6.3. Summary and conclusions

Viral infections, particularly when they lead to encephalitis, can cause neurocognitive deficits, including language problems. Infections during early pregnancy have the strongest impact on language. Although the precise molecular mechanisms remain unclear, these findings suggest that brain development involves interactions with the immune system.

4.7. The importance of timing

In the previous sections, we described mechanisms linked to risk factors that have a general impact on brain development, and we sketched how these mechanisms may lead to impaired language development. The effects of the risk factors associated with general mechanisms that we discussed may become specific to language in interaction with time of onset during pregnancy.

As described above (Section 1), different brain regions develop at their own pace. Disadvantageous circumstances during the early life of a child can perturb brain development, but the specific consequences will likely depend on which brain area was most vulnerable during the time when these circumstances occurred. In line with this hypothesis, the effects on language development of some of the factors discussed here are reported to be time-dependent. We found that exposure to alcohol, prenatal maternal stress and infections appear most harmful when occurring early during pregnancy (Section 2). On the other hand, for phthalates, the strongest correlation with impaired language development was when exposure occurred during the third trimester (section 2.4) (Bornehag et al., 2018; Jensen et al., 2019; Olesen et al., 2018). In the third trimester of pregnancy, the development of the prefrontal cortex is still at an early stage (stage 1 in Fig. 1) and may therefore be more susceptible to toxicity compared to other brain areas during this period. Damage, or suboptimal conditions, during the third trimester of pregnancy may cause permanent alterations in the prefrontal cortical structure with potential life-long impact on cognitive capacities, including executive functions and language processing. The same exposure to toxicity at an earlier time point of pregnancy would mostly affect earlier developing brain areas and therefore result in neurodevelopmental problems that are distinct from language deficits.

The primary auditory cortex (in the temporal lobe), on the other hand, is structurally mature by the end of the second trimester (Hensch, 2005; Huttenlocher and Dabholkar, 1997; Monson et al., 2018). However, a large developmental reorganization of connections in this part of the cortex occurs during the third trimester (stage 2 in Fig. 1), which is strongly modulated by (auditory) experience. Exposure to stress, inflammation, malnutrition or hypoxia during this period may affect these plasticity processes and therefore alter the development of the auditory system, which will have a lasting impact on auditory processing later in life. Circumstances during the third trimester of pregnancy appear especially important for the acquisition of prosodic language components (Ragó et al., 2014), and inability to acquire these skills may aggravate poor language development later on.

Thus, adverse conditions that perturb the general development of the brain can have specific impact on language development if they are present at the time when specific brain regions that support processes crucial to language, such as auditory processing and executive functioning, go through significant developmental changes.

5. Risk factors: specific mechanisms of impaired language development

The mechanisms of risk factors discussed in the previous section are associated with many brain functions and influence neurodevelopment in general, with potential effects on language development. In this section, we identify three risk factors that seem to interfere with the development of a specific neural (sub)system that is a critical component of the neurocognitive architecture needed for language learning. We focus on preterm birth, maternal smoking, and sleep problems. Children who are born preterm develop outside of the womb in the third trimester. As explained in the previous section, the third trimester may be specifically vulnerable for language development, as crucial developmental processes occur during this period in brain regions that are involved in language. Maternal smoking exposes the fetus to toxic substances in the tobacco smoke, including nicotine. One reason for why we classify the effect of prenatal exposure to nicotine specific to language is that brain cells express specific receptors for nicotine, the nicotinergic acetylcholine receptor, and the cholinergic system is crucial during development of the auditory system and for experience-dependent adaptation of neuronal circuits. Sleep problems are a rather understudied risk factor for language difficulties, but available research suggests specific pathways by which low sleep quality, and consequent perturbation of specific neurophysiological processes, can have a crucial negative effect on memory consolidation essential for language learning.

5.1. Preterm birth

There are many aspects of a child's birth that can severely impact their well-being later in life, including difficulties during labor, perinatal asphyxia, intra-uterine growth restriction, and premature birth (i.e., born before week 37 of gestation). Many of these have been found to have an impact on a child's future language skills (e.g. (Diepeveen et al., 2013; Law et al., 2009; Prathanee et al., 2007; Stanton-Chapman et al., 2002). In this section, we focus on preterm birth, as this neonatal condition is most frequently studied in relation to language skills.

5.1.1. Link with language development

Around 20–25% of infants born preterm develop language problems (Müller et al., 2018; Reidy et al., 2013) and the more preterm a baby is born, the poorer their language outcome typically will be (Müller et al., 2018; Prathanee et al., 2007; van Noort-van der Spek et al., 2022). Children born preterm are more likely to visit speech-language pathology services than full-terms (Harrison and McLeod, 2010; Knuijt and Sondaar, 2001). Moreover, they are at a high risk of DLD and other cognitive impairments, e.g., in general intellectual ability and executive functioning (da Costa Ribeiro et al., 2016; Gozzo et al., 2009; Knuijt and Sondaar, 2001; Largo et al., 1990; Müller et al., 2018; Sansavini et al., 2010; Woodward et al., 2012). Preterm-born children display a wide variety of simple and complex expressive and receptive speech-language difficulties (Gonzalez-Gomez and Nazzi, 2012; Rand and Lahav, 2014; Reidy et al., 2013; van Noort-van der Spek et al., 2012), such as articulation deficits, stuttering, difficulty producing grammatically correct sentences, and poor receptive vocabulary (da Costa Ribeiro et al., 2016; Gozzo et al., 2009; Ionio et al., 2016; Largo et al., 1990; Mossabeb et al., 2012; Rabie et al., 2015; Sansavini et al., 2010; Vohr, 2014; Woods et al., 2014; Woodward et al., 2012). Deficits in receptive vocabulary have been found to occur at a young age and, as preterm infants become older, difficulties integrating multiple language components, including the meaning of complex concepts and structure of complex sentences, become more apparent (van Noort-van der Spek et al., 2012). However, some studies do not find language deficits in preterm infants (Diepeveen et al., 2017, 2013; Harrison and McLeod, 2010; Knuijt and Sondaar, 2001; Pérez-Pereira et al., 2016, 2014). Although preterm-born children score persistently lower in language assessments compared to matched full-term peers during the first 3 years of life (Sansavini et al., 2014) and their language processing remains affected (Barnes-Davis et al., 2018), their lower scores on language tests may still be within the normal range (Barde et al., 2012; Pérez-Pereira et al., 2016; Reidy et al., 2013; Sansavini et al., 2014; Woods et al., 2014).

5.1.2. Possible mechanisms

The mechanisms underlying language difficulties in preterm children are only partially understood and most likely multifactorial. Preterm birth often co-occurs with additional risk factors for developing language deficits (e.g., dysmaturity, need for prolonged ventilation, brain injury, and infections (Müller et al., 2018; Pérez-Pereira et al., 2016)). For example, one study showed that preterm born infants with a very low birth weight may only show language deficits if they have a history of bronchopulmonary dysplasia, a chronic lung disease in which (prolonged) mechanical ventilation during neonatal care is a risk factor (Singer et al., 2001). Below, we discuss and evaluate mechanisms that have been proposed in the literature.

5.1.2.1. Impaired prosodic and motor development. One possible cause for language difficulties in preterm born children is a shorter exposure to linguistic rhythm, intensity, and pitch (prosodic features of spoken language) *in utero*. The recognition and discrimination of such features has been shown to be delayed in preterm infants (Bosch, 2011; Peña et al., 2010), while phonotactic acquisition (i.e., learning constraints about phoneme combinations), which for the most part takes place postnatally, is not affected (Gonzalez-Gomez and Nazzi, 2012; Perszyk et al., 2018). These findings suggest that circumstances during the third trimester of pregnancy are especially important in the acquisition of prosodic language components (Ragó et al., 2014), and inability to acquire these skills may aggravate poor language development later on. Prosodic features are thought to support speech segmentation into words (Johnson and Jusczyk, 2001) and have been argued to be fundamental for the detection of grammatical structures later in life (Christophe et al., 2003; Soderstrom et al., 2003).

Another possible cause may be impaired motor development, which is common in children born preterm (Müller et al., 2018; Ullman and Pierpont, 2005). Assessments of early motor skills of preterm infants correlate with later language and cognitive performances (Oudgenoeg-Paz et al., 2015; Zuccarini et al., 2017). Motor difficulties may result in less manual and oral exploration, which is known to be important for subsequent cognitive and language development (Oudgenoeg-Paz et al., 2015; Zuccarini et al., 2017).

5.1.2.2. Effects of the NICU. Both the inability to acquire prosodic features on time and impaired motor development (previous section) might also be related to preterm infants staying for a long period at neonatal intensive care units (NICU). NICUs tend to be noisy. The sounds in NICUs often come from machines (Best et al., 2018; Ionio et al., 2016) and parents are unable to stay in the NICU for extended periods of time, resulting in a lack of language and speech stimulation of their child (Best et al., 2018; Maitre et al., 2013). These conditions are neither optimal for learning prosodic features, nor for social interactions in general, which are of special importance to language development (Kuhl, 2007). Also, babies in the NICU often are in a recumbent position for a long period, which may impede motor development (Sansavini et al., 2014), and the loud sounds in NICUs may cause stress, which, in turn, negatively impacts early brain development. For this reason, NICU environments are made increasingly more quiet, but this may also not be optimal for a child's language development due to reduced linguistic input (Rand and Lahav, 2014). The exposure to the NICU environment during the first months after birth may thus partially explain the language difficulties of preterm children and the association between prematurity and DLD.

5.1.2.3. Brain structure abnormalities. At birth, preterm brains display grey matter (GM) and white matter (WM) abnormalities in comparison to full-term brains, which can last until at least early adulthood (Allin et al., 2006). Brain volume is relatively low (Barnes-Davis et al., 2018), subarachnoid space is enlarged, gyrification is immature (Inder et al., 2003), and the cerebellum often shows abnormalities (Gano and Barkovich, 2019; Salvan et al., 2017; Stipdonk et al., 2021; Tam, 2018). In adulthood, volumes of subcortical and medial temporal cortices are reduced. In contrast, frontal and lateral parieto-temporal cortices are found relatively unaffected (Karolis et al., 2017).

Moderate to severe white matter (WM) abnormalities have been found in ~20% of preterm infants, including delayed myelination and enlarged ventricles (Constable et al., 2008; Counsell et al., 2008; Howard et al., 2011; Inder et al., 2003; Maalouf et al., 2001; Schafer et al., 2009). WM abnormalities in preterm infants have been associated with lower performance in tasks tapping into phonological awareness, semantics, grammar, discourse and expressive language, and presence of such abnormalities increase the risk for language delays (Howard et al., 2011; Reidy et al., 2013; Woodward et al., 2012). The more severe the WM abnormalities are, the worse neurocognitive outcomes are, whereas absence of WM abnormalities has been associated with performance similar to full-terms (Woodward et al., 2012). WM abnormalities in preterm infants have been found in left frontal and bilateral temporal areas, intrahemispheric fiber tracts and connected regions (Constable et al., 2008; Mullen et al., 2011; Schafer et al., 2009). Male preterm infants run a higher risk than female preterm infants to have WM abnormalities in the uncinate fasciculus, which is part of the ventral language pathway (Constable et al., 2008). Also, abnormalities are found in the left arcuate fasciculus (AF), one of the WM tracts connecting Wernicke's and Broca's area, two brain areas of primary importance for language (see Fig. 2). AF abnormalities are negatively associated with linguistic abilities (Salvan et al., 2017).

Preterm infants also have a reduced number of inhibitory interneurons in the prefrontal cortex, and similar observations are reported in mouse models of neonatal brain injury (Lacaille et al., 2019; Stolp et al., 2019). Migration of interneurons to the frontal lobe occurs during the final weeks of gestation, rendering this process vulnerable to injury during this period. Impaired prefrontal functioning due to loss of interneurons may fundamentally alter cognitive abilities, including language abilities.

5.1.2.4. Differences in brain function. Some studies have found functional neural differences between preterm and full-term children in the absence of structural abnormalities (Barnes-Davis et al., 2018). Different cortical language areas and a broader neural network are activated during semantic and phonological processing in preterm children than in full-term peers (Barde et al., 2012; Barnes-Davis et al., 2018; Frye et al., 2010; Ment et al., 2006; Schafer et al., 2009; Wilke et al., 2014). For instance, preterm children show greater recruitment of the prefrontal cortex when processing long sentences than full-terms (Barde et al., 2012). Activation in the frontotemporal language network in preterm children is less left-lateralized than in full-terms (Baldoli et al., 2015; Barnes-Davis et al., 2018; Mürner-Lavanchy et al., 2014). This pattern of activation may be related to a delay in white matter maturation in thalamocortical pathways and has been associated with neurodevelopmental outcome (Baldoli et al., 2015). Moreover, connectivity in the ventral pathway seems to be negatively affected in preterm infants, similar to what has been found in children with DLD (Gozzo et al., 2009; Ment et al., 2006; Mullen et al., 2011; Northam et al., 2012; Peterson et al., 2002; Schafer et al., 2009). These abnormalities appear associated with functional outcomes. For instance, at age 8, the (prefrontal) brain areas that children born full-term use when processing phonological information, are instead used for semantic information processing by children born preterm (Peterson et al., 2002). This activation pattern is associated with poor language comprehension.

5.1.3. Summary and conclusions

Preterm born infants have weaker language skills than full-term peers and are at a higher risk of DLD. In the third trimester, they develop outside of the womb, which affects crucial developmental processes in brain regions that are involved in language. The NICU environment may play a role in the association between prematurity and

language problems. Both structural and functional brain abnormalities have been related to the language difficulties of preterm children.

5.2. Smoking

5.2.1. Link with language development

Mixed findings are presented in the literature regarding the effect of prenatal exposure to tobacco smoke on cognitive and language development in typically developing children (Harrison and McLeod, 2010). A recent systematic review reported that 57% of the 14 included studies found a direct effect of smoking on language development, 35% found indirect effects (e.g., through SES, maternal IQ or parental age) and 7% found no effect (Peixinho et al., 2022). The effect has also been shown to depend on the amount of smoking. Higher levels of maternal smoking and active, rather than passive, smoking are associated with worse outcomes (Eicher et al., 2013; Makin et al., 1991), although also low levels and second-hand maternal smoking have been related to lower language outcomes in the offspring (Hernández-Martínez et al., 2017). With respect to the relation between parental smoking during pregnancy and DLD, findings are also mixed. Some studies report no associations (Diepeveen et al., 2017; Tomblin et al., 1998), whereas other studies found an increased risk for DLD if the parents had smoked (Calder et al., 2022; Law et al., 2009; Rudolph, 2017; Tomblin et al., 1997).

5.2.2. Possible mechanisms

5.2.2.1. Effects on brain structure and function. Tobacco smoke contains a large amount and variety of harmful substances of which the primary component is nicotine. Nicotine has been studied extensively in relation to neurodevelopment. Exposure to nicotine increases neuronal death, decreases the number of neurons, and suppresses synaptogenesis, resulting in long-term alterations of the hippocampus, somatosensory and prefrontal cortex (England et al., 2017; Ren et al., 2022). MRI studies in human infants and adolescents, as well as rodents, reveal that prenatal exposure to nicotine is associated with reduced brain volumes in the frontal lobe, lateral ventricular system, and the cerebellum tracts (Bublitz and Stroud, 2012; Jacobsen et al., 2007). Furthermore, it is associated with the thinning of frontal, parietal, and temporal cortices, disruption of the microstructure, and reduced processing efficiency of major thalamic and cortical white matter tracts (Bublitz and Stroud, 2012; Jacobsen et al., 2007). Activity in the frontal and temporal brain areas have been linked to language processing, specifically to the representation of sentence meaning, analysis of syntactic relations, and bottom-up acoustic processing (Fedorenko et al., 2016; Skeide and Friederici, 2016). Thus, prenatal exposure to tobacco smoke, specifically nicotine, affects global neural development, also in language-related areas.

5.2.2.2. Nicotine's effect on the cholinergic and dopaminergic system. Nicotine affects a multitude of neurotransmitter systems (Bublitz and Stroud, 2012), but its primary effect is activation of nicotinergic acetylcholine (ACh) receptors in the brain. In week 18–22 of (human) pregnancy, cholinergic fibers arise from the basal forebrain (Kanold and Luhmann, 2010). Nicotine exposure during pregnancy induces down-regulation of cortical ACh receptors which alters activity patterns in the developing brain (Kanold and Luhmann, 2010; Slotkin et al., 2007), which is likely to impair auditory processing (Kanold and Luhmann, 2010; King et al., 2003; Liang et al., 2006). For example, maternal smoking during pregnancy delays the onset of the fetus's response to the mother's voice in the womb (Cowperthwaite et al., 2007), which suggests that nicotine-exposed fetuses begin to process auditory stimuli later than those not exposed to nicotine. In the adult brain, neural responses after auditory stimulation are strongly modulated by cholinergic activity (Bublitz and Stroud, 2012; Kable et al., 2009; Morley, 2005; Peck et al., 2010) via specialized inhibitory circuits (Kuchibhotla et al., 2017). Disruption of this modulation by prenatal nicotine exposure may interfere with the ability to encode auditory information (Bublitz and Stroud, 2012). Thus, by affecting the cholinergic system during early development, nicotine may interfere with the ability of the auditory cortex to learn later in life.

The development of the auditory cortex is important for the development of spoken language. Auditory cortical maturation and auditory processing early in life predict speech segmentation and language ability later in life in both typically developing children and children with an elevated risk of dyslexia (Kwok et al., 2018; Molfese, 2000; Molfese and Molfese, 1985; Skeide and Friederici, 2016). Defects in auditory processing are also prevalent in children with DLD (Key et al., 2007; Kidd et al., 2017; Rocha-Muniz et al., 2015; Rosen, 2003), although a direct causal relationship between auditory processing and language deficits has not yet been established (Kidd et al., 2017; Rosen, 2003).

In addition to cholinergic systems, the dopaminergic system also appears sensitive to perinatal nicotine exposure (Oliff and Gallardo, 1999). Release of dopamine in the prefrontal cortex is strongly reduced in mice that have been prenatally exposed to nicotine (Alkam et al., 2017; Zhu et al., 2012) and nicotine exposure alters gene expression patterns in the VTA (McGill et al., 2023), the brain region where dopaminergic neurons are located. Mice that were exposed to nicotine are hyperactive (Zhu et al., 2012) and have reduced self-control (Pinheiro et al., 2015). These effects are partially sex-dependent (Dwyer et al., 2019).

5.2.3. Summary and conclusion

Observed relationships between prenatal parental smoking and delayed or disordered language development may stem from neuronal impairments in brain areas that are affected by nicotine. Nicotine impacts on acetylcholine receptor expression and activity patterns in the developing auditory pathway. This, in turn, may interfere with auditory processing and learning later in life, possibly limiting the auditory processing abilities of children with DLD. In addition, nicotine exposure interferes with the normal development of dopaminergic system.

5.3. Sleep

5.3.1. Link with language development

Sleep impacts language learning in developing young children. Naps have been shown to improve word learning (Friedrich et al., 2015; Horváth et al., 2015; Horváth and Plunkett, 2016), the detection of grammatical patterns (non-adjacent dependency learning) (Hupbach et al., 2009), and abstraction of grammatical rules in infants (Gómez et al., 2006) through memory consolidation. The positive effects of sleep on language learning may be reduced in individuals with DLD. For example, adults with DLD show reduced consolidation of speech sounds during sleep (Earle et al., 2018). Studies assessing if children with DLD have sleep difficulties have so far yielded mixed results. Some children with communication disorders, including DLD, have sleeping patterns that are poor relative to patterns of TD children (Botting and Baraka, 2018). Children with communication disorders experience more semantic/pragmatic language difficulties if they needed more time to fall asleep, and in particular more expressive and receptive difficulties if they tend to sleep for shorter duration (Botting and Baraka, 2018). However, a study based on self-report indicated that TD children reported more sleep problems than children with DLD (Arkkila et al., 2011). A systematic review on sleep and language problems discussed three papers in which sleep quality is associated with lower language scores, yet still within normal range (McGregor and Alper, 2015). Contradictory findings in studies using self-report to measure sleep quality may be explained by a discrepancy between perceived and actual sleep quality. Sleep disruptions may only be evident from EEG signals during sleep without altering sleep behavior. It is thus possible that children who experience only minor sleep problems may actually have relatively poor sleep quality. In addition, timing may be an important

factor, as less mature sleep at 6 and 18 months of age, but not at 30 months, is associated with lower vocabulary scores (Dionne et al., 2011).

5.3.2. Possible mechanisms

Reduced sleep quality, rather than a limited amount of sleep, may be relevant for understanding language difficulties. A healthy sleeping episode consists of several cycles, with alternating phases of deep, slow wave sleep (SWS) and rapid eye movement (REM) sleep. High sleep quality is usually defined as mostly undisturbed sleep, at regular times, containing sufficient sleep in all stages. However, it is also important that the proper physiological and metabolic processes occur during sleep. Regarding language and speech, undisrupted neurophysiological patterns during slow wave sleep appear to be particularly relevant. Slow wave sleep is important for memory consolidation and synaptic plasticity (Chen and Wilson, 2017; Paller et al., 2021). During slow wave sleep, specific synchronized oscillatory brain activity occurs (slow oscillations in the cortex, 'sharp wave ripples' (SWR) in the hippocampus and 'spindles' in the thalamus and cortex; Fig. 2), which reflects thalamocortical and hippocampal-neocortical interactions. The hippocampus-neocortex interactions are particularly important for declarative memory (e.g., word learning), whereas thalamocortical interactions are involved in procedural memory (e.g., grammar) (Lee et al., 2020; Ullman et al., 2020). Animal studies have shown that neurons that were active when the animal was showing awake behavior (e.g., vocalizing) are reactivated during slow wave sleep (Chen and Wilson, 2017; Shank and Margoliash, 2009; Wilson and McNaughton, 1994), suggesting that memories are 'played back' to support consolidation. This replay coincides with hippocampal SWR (Paller et al., 2021).

Although SWRs have not been identified in songbird hippocampus (Beckers and Rattenborg, 2015; Meij et al., 2020), very similar sleep states and substates have been found in birds and in mammals, and replay activity has been suggested to occur in sensory-motor areas (Dave and Margoliash, 2000). Sleep spindles are thought to improve the formation and retention and generalization of lexical-semantic information (Chen and Wilson, 2017; Friedrich et al., 2015). In starlings, sleep improved auditory memory (Brawn et al., 2013) and sleep-related vocal changes have been shown in juvenile zebra finches during song learning (Derégnaucourt et al., 2005; Shank and Margoliash, 2009). In addition, lateralization of brain activity during sleep (stronger activity in the left compared to the right hemisphere) correlated with song imitation performance by the juvenile (Moorman et al., 2015). It is therefore likely that poor sleep quality has a negative effect on memory consolidation processes required for language learning (Dionne et al., 2011; James et al., 2017).

5.3.2.1. Abnormal or epileptiform EEG during sleep. Epileptiform sleep waves are a possible indication of poor sleep quality. In nocturnal epileptic disorders, disruption of sleep has been shown to affect children's language development, suggesting that abnormal activation of the brain during sleep interferes with language learning (Ballaban-Gil and Tuchman, 2000; Monjauze et al., 2005; Overvliet et al., 2010; Scabar et al., 2006). Nocturnal epileptic surges that are associated with these disorders may interfere with the functional organization of brain areas involved in language (Monjauze et al., 2011; Overvliet et al., 2010). Similar abnormal or epileptiform nocturnal EEG discharges have also been found in some children with DLD (Billard et al., 2009; Dlouha et al., 2020; Mehta et al., 2015; Nasr et al., 2001; Overvliet et al., 2010; Parry-Fielder et al., 2009; Venkateswaran and Shevell, 2008). However, other studies did not find a correlation between epileptiform activity during sleep and language difficulties (Lajunen et al., 2023; Systad et al., 2017). Although a general relation between language ability and abnormal EEG was not found, two region-specific relations were observed: right lateralized epileptiform activity was found to be negatively correlated with naming speed, and centrotemporal activity was negatively correlated with phonology and orthography performance (Systad et al., 2017). Daytime epileptiform discharges have also been reported in some children with DLD, but nighttime discharges seem more prevalent, predominantly during non-REM sleep (SWS) (Dlouha et al., 2020). The similarities between the nocturnal spikes of children with DLD and children with more severe epileptic disorders suggest a continuum of nocturnal epileptic activity, with DLD on the lower end of the spectrum (Billard et al., 2009). Unfortunately, the current experimental evidence does not yet allow us to draw strong conclusions and more research with larger samples (also including healthy controls) will be necessary. Furthermore, more attention should go out to disentangling differences in specific EEG abnormalities and their anatomical origin.

5.3.2.2. Brain abnormalities related to nocturnal EEG discharges. The involvement of the gene *GRIN2A* may be of importance in children with speech or language impairments and nocturnal epilepsy. Around 20% of children who have a speech impairment and some that suffer from DLD with a severe type of nocturnal epilepsy have shown to carry gene defects in *GRIN2A* (Chen et al., 2017; Lesca et al., 2013). This gene encodes for the GluN2A subunit of NMDA receptors (Carvill et al., 2013; Lemke et al., 2013; Lesca et al., 2013). NMDA receptors are important regulators of neural plasticity, and are for instance required during the memorization phase of song learning in zebra finches (Basham et al., 1996). In these birds, *GRIN2A* is specifically expressed in song system nuclei, suggesting a region and function specific effect (Wada et al., 2004). Mice in which *GRIN2A* was knocked out display epileptiform discharges during early postnatal development as well as transient microstructural abnormalities in the thalamocortical system, hippocampus, and the corpus callosum later in life (Salmi et al., 2018). As the thalamocortical circuitry is specifically associated with slow wave sleep (Salmi et al., 2018), this suggests a direct link between EEG discharges and maturation of thalamocortical circuits involved in sleep. Other studies have suggested that *GRIN2A* knockout can also affect the maturation of inhibitory cells in the hippocampus, which are crucial for SWR activity (Cardis et al., 2018; Sun et al., 2018) and may interfere with replay and memory consolidation during sleep. Furthermore, disrupted maturation may negatively affect the sensitive period for language acquisition and thus restrict or delay language learning and memory.

5.3.3. Summary and conclusion

Children with DLD may experience low quality of sleep, including disturbed neurophysiological processes, which may negatively affect memory consolidation. In a subset of these children, epileptiform discharges during sleep (possibly brain region specific) may be the cause of poor sleep quality. Epileptiform discharges during sleep may be related to maturation of thalamocortical or hippocampal circuits involved in sleep and memory consolidation, and, consequently, in language development.

6. Multifactorial causes

Until now, we have discussed risk factors separately to pinpoint specific pathways, but it is often the case that multiple factors are involved, which may depend on each other or which could converge on a common outcome. Below, we describe how such interaction and additive effects can result from overlap between underlying neurodevelopmental mechanisms or brain structures. We furthermore discuss some of the interactions between risk factors for DLD that have been reported in the literature. These are examples where one risk factor potentially moderates the effect of another risk factor. In addition, we delve deeper into the role of socioeconomic status (SES) and mediating factors.

6.1. Mechanisms of multifactoriality

Risk factors may interact and lead to additive effects at the level of the neural and neurocognitive systems that support language acquisition. One instantiation of this is that a particular risk factor impacts separate systems (networks, neurotransmitter systems) that are each critically involved in language learning. Prenatal exposure to nicotine is an example, as it may affect the cholinergic system in the auditory system, as well as the dopaminergic system in the striatum. A second instantiation is that different risk factors impact the same neuronal system. Examples are reduced sleep quality, genetic factors (*FOXP2* mutations), and nicotine exposure, which may each impact structural and functional development of the basal ganglia and may thus reinforce one another. A third instantiation is that risk factor(s) impact a system that is involved in multiple functions relevant for language learning. For example, the basal ganglia are critical for sequential learning (supporting acquisition of phonotaxis, word segmentation, grammatical patterns, articulating), working memory, and executive control (De Diego-Balaguer et al., 2008). Thus, any factor that impacts the structural and functional development of the basal ganglia may impact language development through different routes.

Risk factors may also interact in a correlative manner. For example, children that have difficulty articulating, which is linked to a genetic risk factor (*FOXP2* mutation), may engage less in verbal interaction and as result receive less input from their caretakers. Also, a child that carries a genetic risk factor is likely to have a parent with the same risk factor who may, as a result, communicate less or differently with the child. In this way, the genetic factor harms the child both directly and indirectly.

6.2. Examples of interactions between risk factors for DLD

One risk factor for DLD which is described to be involved in interactions with several other risk factors is breastfeeding. First, the child's sex: benefits of breastfeeding on neurocognitive development are reported to be larger for male than for female infants (Oddy et al., 2011). Breastfeeding also interacts with smoking, as mothers who smoke produce less milk, feed for a shorter time per lactation, and have lower quality of milk, as its beneficial nutrient content is reduced (Napierala et al., 2016). Complex interactions are found between breastfeeding, smoking, and sleep. Infants who are breastfed shortly after the mother smoked, were found to sleep less overall and have poorer quality of sleep (i.e., less time spent in active sleep). The amount of nicotine delivered via breast milk was inversely related with sleep quality (Mennella et al., 2007). Nicotine may alter the release of neurotransmitters that regulate the sleep-wake cycle (Guzmán-Marín et al., 2001) or stimulate wake-promoting neurons (Guzmán-Marín et al., 2001; Saint-Mleux et al., 2004; Sigalas et al., 2015). In addition, the amount and quality of sleep of the mother during pregnancy influences neonatal auditory responses to emotional sounds (Lavonius et al., 2020).

Genetic factors often reflect genetic dispositions or sensitivity towards other risk factors and this is also true for language development (Sriganesh and Ponniah, 2018). For instance, limited parental stimulation has more pronounced negative consequences for language development in children with CNTNAP2 risk alleles (Onnis et al., 2018). A twin study has shown that the ratio between day and night sleep duration is highly heritable and that language delays at the age of 5 were correlated with less mature sleep patterns at 6 and 18 months (Dionne et al., 2011). After the age of 18 months, sleep ratio is mainly affected by individual differences in environment rather than by genetics, but sleep ratio still predicts language development (Dionne et al., 2011).

Sex and sex hormones can interact with the expression of genetic risk factors for DLD. The exact relation can be highly complex and depends on several factors including child age and brain region. For example, testosterone treatment can lead to increased levels of expression of GRIN2A (Heinrich et al., 2002), whereas testosterone induces increased or decreased FOXP2 levels depending on brain region (Michael Bowers et al., 2014).

6.3. Socioeconomic status

Low socioeconomic status (SES) is a risk factor for poor health in general and associations between low SES and poor developmental outcomes have been well studied. SES, as a factor in developmental science, is typically indexed via measures of caretakers' / parents' education level, family income, or parental occupation (Bradley and Corwyn, 2002; Ensminger and Fothergrill, 2003). Children with low SES backgrounds are at risk of developmental delays of vocabulary, grammar and phonology (Arriaga et al., 1998; Hart and Risley, 1995; Huttenlocher et al., 2002; Rescorla and Alley, 2001; Schwab and Lew-Williams, 2016) as well as literacy skills (Pace et al., 2017). Moreover, studies that investigated the effects of SES on multiple neurocognitive systems showed the largest effects on language development and processing (Farah et al., 2006; Hackman et al., 2010; Noble et al., 2005). Of the various SES measures, maternal education correlates most strongly with child language outcomes (Hoff et al., 2012; Magnuson et al., 2009). Low maternal education has been identified as a risk factor for DLD (Rudolph, 2017). A recent scoping review describes low SES as a predictor of DLD, but with relatively low predictive power compared to other predictors such as family history and early language skills (Sansavini et al., 2021).

The effects of low SES on children's language development are mediated by some of the risk factors discussed in Sections 2 and 3. The large body of literature that examined how and why SES is related to child language outcomes points to a limited number of specific mediating factors, in which three overlapping clusters can be identified: (1) prenatal factors affecting children's language processing from early on; (2) factors related to parenting and parent-child interactions; (3) factors related to cognitive stimulation and richness of environment (Hackman et al., 2010; Pace et al., 2017).

6.3.1. Prenatal factors

SES differences in efficiency of language processing are already evident at 18 months (Fernald et al., 2013). It is as yet unclear whether infants from lower SES backgrounds start off with less efficient processing skills, or incur delays during early postnatal development (Pace et al., 2017). It is likely, however, that several factors impact on neurocognitive development prenatally. Exposure to toxic substances may be among these, as low SES tends to be related to higher levels of parental smoking, drinking, and drug abuse (Newacheck et al., 2003). Hackman and colleagues (2010) point out that low SES is related to maternal stress, infections, and poor nutrition during pregnancy, which in turn are associated with prematurity and suboptimal fetal growth. In summary, low SES is associated with a high prevalence of prenatal risk factors.

6.3.2. Parent-child interactions and parenting

Children from low SES backgrounds on average receive less verbal input from their parents than their higher-SES peers (Hart and Risley, 1995; Huttenlocher et al., 1991). Yet, there is wide variability in input quantity within SES strata (Sperry et al., 2019; Weisleder and Fernald, 2013) and qualitative features of the input were found to be more important than sheer quantity (Rowe, 2012; Rowe and Snow, 2019). Specific aspects of input quality that mediate effects of SES on children's language outcomes are the length of parents' sentences (Hoff, 2003), and lexical and syntactic diversity (Huttenlocher et al., 2010). Other studies point to effects of parenting style, and parental sensitivity and responsivity (Perkins et al., 2013; Shahar and Raviv, 2004). These covary with parental health and general well-being, which tend to be poorer in low-SES families.

6.3.3. Cognitive stimulation and richness of environment

Children from low SES backgrounds are often raised in less

stimulating environments (e.g., fewer books and linguistically and cognitively challenging toys) than children from high SES backgrounds. They also have fewer opportunities to go on trips and to visit places that offer new experiences, such as zoos, museums, or high-quality libraries, which has repercussions for their neurocognitive and language development (Hackman et al., 2010); (Paceet al., 2017). Animal studies have demonstrated that environmental enrichment has a positive influence on brain development by enhancing the synaptic plasticity which is necessary to optimize brain function for the rest of the life span (Berardi et al., 2015; Sale, 2018), and the same may hold for humans. Songbird studies have shown that within-nest competition for resources (e.g., attention, food, space) may result in stress and therefore have a negative influence on song learning and song quality (Holveck et al., 2008). Such domestic crowding and competition for resources is more likely to occur in low SES families (Melki et al., 2004) and, analogous to the songbird case, these factors may contribute to children's language delays.

7. Neurocognitive accounts of DLD in relation to neurobiological mechanisms

In this review, we have so far discussed risk factors for DLD and suboptimal language development and we have explored how these risk factors can compromise the intricate sequence of neurodevelopmental processes that support the development of speaking and understanding. In this section, we evaluate to what extent theories about the etiology of DLD converge with the neurobiological evidence we presented. DLD, just as any other neurodevelopmental disorder, is thought to be the outcome of a complex interaction of numerous genetic and environmental risk factors (Bishop, 2009). This perspective would explain why 'grand unification' theories of DLD, which propose a single-base deficit, have consistently been followed by counter-evidence (Kapa and Plante, 2015). It would also explain why the notion of DLD itself is elusive: the population of children diagnosed with the condition (as a rule by excluding putative causes of their language difficulty) is diverse, in terms of severity, affected modalities and language domains. Consequently, we think a realistic perspective on DLD is that it covers a continuum of language disorders, each of which may result from several distinguishable, but potentially partly overlapping pathological pathways. A number of such pathways have been proposed at the neurocognitive level, i.e., they construe DLD as the result of a dysfunction in one of the perceptual or cognitive systems that are part of the neurocognitive backbone of (spoken) language acquisition. Three of these accounts, which are not mutually exclusive, are discussed below, in particular to see if they converge with the neurobiological evidence discussed in this review. This is an attempt at building bridges between relatively disparate types of work, which we consider important in advancing neurocognitive theorizing on language acquisition and furthering our understanding of the complex etiology of DLD.

7.1. Auditory processing deficits

Several theories propose that difficulties with auditory processing, or deficits in the discrimination and processing of speech and nonspeech sounds, underlie the language problems of children with DLD (e.g. Tallal, 2004 and Bishop, 2007). The common thread is that a central auditory processing deficiency leads to significant delays in the acquisition of the phoneme inventory of the native language, which is of paramount importance to learning a (spoken) language. Such delays have cascading effects on word learning as well as on the acquisition of grammar, as grammatical features and relations are frequently encoded in subtle phonological distinctions. According to some accounts, such auditory processing deficiencies may be part of a broader auditory perception difficulty, affecting the ability to segregate speech from ambient noise and attending to a stream of speech (de Wit et al., 2018). These characterizations are suggestive of a connection with executive functioning (see below; Section 5.3). There is evidence supporting an association between early auditory processing deficits and DLD (e.g. Benasich and Tallal, 1996; Tallal and Benasich, 2002), but, importantly, there are also many children with DLD who do not have such deficits (Protopapas, 2014; Rosen, 2003).

We identified several mechanisms that potentially disrupt auditory development. Timing, as discussed in section 2.7, seems to be a crucial factor. Development of the auditory cortex (in the temporal lobe) starts well before birth and continues to develop after birth throughout the language learning period. Auditory exposure also starts before birth and auditory experience affects neural plasticity. This sensitive, and therefore vulnerable, period in the development of the auditory system strongly determines processing of auditory signals later in life. We found that exposure to alcohol and phthalates were most detrimental in the second half of pregnancy when they might interfere with auditory cortex development.

Developmental timing of auditory experience and brain maturation is particularly relevant for children born prematurely, as a misalignment may occur between stages of high plasticity and exposure to formative sound stimuli. The impact of preterm birth on auditory development can also be related to exposure to the NICU soundscape, which is drastically different from that in the womb. As input-driven proliferation of connections (Stage 2 in Fig. 1) in the auditory cortex is still underway, this aberrant auditory stimulation is likely to influence the child's processing of speech sounds.

Next to preterm birth, prenatal exposure to nicotine may compromise auditory development. Nicotine affects the cholinergic system in the auditory cortex: Acetylcholine receptor expression is reduced, and this modifies activity patterns in the developing auditory pathway, which may limit auditory processing modulation and thus interferes with the ability of the auditory cortex to learn later in life. This may well explain the risk of developing a language disorder in children prenatally exposed to nicotine.

We note that, in addition to preterm birth and nicotine exposure, some of the risk factors we classified as 'general' may impact development of the auditory system as well. That is to say, within the broad domain of impact associated with these factors, a specific pathway linked to audition may be plausible. Poor maternal physical health is a case in point. Fetal hypoxia and malnutrition alters neural growth and affects the maturation of the fetal auditory system (Kisilevsky, 2016; Perna et al., 2015; Werker and Hensch, 2015). This may impede infants' learning from (prenatal) speech input, as it has been found that growth restricted fetuses / infants, and fetuses / infants from diabetic or hypertensive mothers have difficulty recognizing their mother's voice *in utero* and postnatally (Kisilevsky, 2016). The sensitive phase for postnatal attunement to native phonemes may be affected by maternal depression. Delays in auditory development also interfere with coordinated development across brain regions which is required for language development (Werker and Hensch, 2015).

7.2. Memory

It has been proposed that deficits in short-term (phonological) memory or long-term memory are a causal factor in the emergence of DLD. According to the phonological short-term memory deficit account, children with DLD have a reduced capacity for temporarily storing sequences of phonological material, which in turn impacts word learning (Baddeley et al., 1998; Gathercole and Baddeley, 1990). As word learning, by all accounts, is a gateway to acquiring grammar (which comprises morphology and syntax), a general deficit will ensue. The procedural/declarative model developed by Ullman and colleagues (e.g. Ullman, 2004; Ullman et al., 2020; Ullman and Pierpont, 2005) invokes *long-term* memory functions. The model's basic tenet is that word learning is supported by the declarative memory system, while grammar learning is a function of the procedural system. The declarative and procedural memory systems are domain-general components of long-term memory (Squire and Knowlton, 2000), which are functionally

distinct. Procedural learning, associated with the striatum and cortico-striatal connections, underlies the formation of habits and skills. Ullman argues that DLD, with grammatical difficulties as a core feature, is an outgrowth of a deficiency of the procedural system. The procedural learning circuitry is also assumed to be the substrate of sequential statistical learning, which recent research implicates as a mechanism critically involved in young children's learning of grammar (Krishnan et al., 2016; Ullman et al., 2020). Several studies have indeed reported that children with DLD perform more poorly than their TD peers on tasks assessing procedural learning (Lum et al., 2014), as well as on tasks probing statistical learning (Lammertink et al., 2017), although there is large heterogeneity (West et al., 2021).

Short-term (or working) memory and long-term memory are mediated by separate neural systems. Working memory (related to storage and manipulation of information) is mostly considered an executive function of the prefrontal cortex (see below; Section 5.3), while long-term memory is mediated by a larger cortico-hippocampal network. A mechanism discussed in this review that is evidently associated with long-term memory is disrupted consolidation during sleep. Sleep is crucial for experience-dependent plasticity and learning, in particular during a child's early development. During sleep, brain activity associated with experiences during the day are 'replayed'. Sleep facilitates the formation of neural connections during consolidation of procedural as well as declarative memory. Sleep disruptions therefore interfere with learning in general but may specifically affect language development in some children (Deak et al., 2011; Indefrey, 2011). Together, these observations make a case for a causal connection between poor sleep quality and deviant language learning in childhood. Again, this cannot be the only causal mechanism, as there is no mutual implicational relation between poor sleep quality and DLD.

Regarding procedural memory, prenatal nicotine exposure may interfere with dopaminergic function. Dopamine plays a crucial role in reward processing and dopamine is the primary neurotransmitter in cortico-striatal networks, which have been associated with procedural learning and memory. Nicotine may exert negative effects on language development through this dopamine pathway as well (Eicher et al., 2013; Ullman et al., 2020).

7.3. Executive functioning

A third theoretical account focuses on executive functioning (Kapa et al., 2017; Kapa and Plante, 2015). Executive functions are the higher-level cognitive functions that regulate lower-level cognitive processes to effectuate goal-oriented behavior (Friedman and Miyake, 2017). Executive functions are themselves also organized from lower to higher level skills, with sustained attention as a fundamental building block required for later development of working memory, inhibition and, finally, attention shifting (Garon et al., 2008; Kapa et al., 2017). In this context, the neurocognitive mechanisms and processes mentioned thus far (auditory processing, phonological memory, procedural memory) are the 'lower-level' cognitive processes that are (partly) under control of the executive functions. For certain, executive functions, and specifically (sustained) attention, are critical for auditory processing, short-term memory, and learning (either procedural or declarative). How DLD and executive functioning are related is an issue of ongoing debate (Gooch et al., 2016; Kapa and Plante, 2015), but there are indications that children with DLD are often outperformed by their TD peers on a wide range of executive functioning measures (for reviews, see Ebert and Kohnert, 2011). Sustained attention impairments of children with DLD may be more severe than impairments in working memory, inhibition and attention shifting (Kapa et al., 2017). Furthermore, sustained attention has been found to explain lower grammar, vocabulary and narration outcomes of children with DLD (Blom and Boerma, 2016; Boerma et al., 2017). This strengthens the conclusion that sustained attention deficits, and possibly also deficits in other executive functions, underpin the language difficulties of children with DLD, although it is unlikely that this holds for all children with DLD.

The prefrontal cortex is still undergoing significant structural development in the third trimester of pregnancy (Hensch, 2005; Huttenlocher and Dabholkar, 1997), and neurons throughout the area are still immature (Kolb et al., 2013; Kubo et al., 2017) (stage 1 in Fig. 1). Preterm birth may specifically interfere with this early circuit formation in the prefrontal cortex, disturbing the development of executive functions. Suboptimal conditions during the third trimester of pregnancy, for example caused by toxicity, may also lead to suboptimal development of the prefrontal cortex, affecting executive functioning. The same holds for prenatal exposure to nicotine, as this has been associated with, among others, thinning of the prefrontal cortex. In particular, altered development of the dopaminergic or cholinergic systems in the brain after maternal smoking will be crucial in determining executive functions later in life. Dopaminergic modulation of the prefrontal cortex is important for working memory and reward processing, while cholinergic modulation mediates attention. Perinatal exposure to nicotine is associated with impaired working memory and attention (Heath and Picciotto, 2009; Zhang et al., 2018), increased impulsivity (Fitzpatrick et al., 2014) and reduced self-control (Pinheiro et al., 2015). Working memory defects are also reported after prenatal exposure to antidepressant drugs (Meurer et al., 2021), after thiamine deficiency (Kipp et al., 2021), and exposure to stress (Sohr-Preston and Scaramella, 2006).

7.4. Summary

In this section, we tried to converge the neurobiological mechanisms of risk factors that we identified in this review onto three neurocognitive accounts of DLD. As mentioned, these accounts and mechanisms are by no means mutually exclusive, but may each contribute to a better understanding of DLD. It is important to stress that not all risk factors and mechanisms highlighted in this review can be accounted for by specific neurocognitive models. This is because language skills depend on proper functioning of many neurocognitive functions including perception, motor control, memory, and attention. Suboptimal performance in one or more of these functions can culminate in language defects without a traceable underlying cause. Why in some cases this leads to language defects and in some cases to other cognitive defects remains an open question. One possible explanation may be that processing of linguistic sequences demands longer sustained attention and larger memory space than some non-sequential skills such as object recognition. This is in line with the finding that processing of musical sequences also seems to be delayed in children with DLD (Sallat and Jentschke, 2015). Future research will be necessary to further investigate this possibility. In the next section, we will provide a broader perspective for future research directions.

8. Future perspectives

This review aimed to link previously described risk factors that may interfere with language development to putative underlying neurobiological mechanisms. It was not our aim to thoroughly examine or weigh the evidence in favor or against these risk factors, but this review clearly shows that language development in children can be negatively impacted by a wide range of environmental and innate factors. As has been mentioned previously, none of these individual risk factors are necessary or sufficient to explain impaired language development. However, elimination or avoidance of these factors will *reduce the risk* of impairment. For some of these factors this is generally acknowledged. The dangers of exposing young or unborn children to toxins, drugs or stress are widely recognized. Maternal smoking is also recognized as a risk factor and generally discouraged. The potential impact of some other factors may currently be less well-known. For example, infections during early development may be an underestimated or even overlooked risk factor for DLD, as these may occur without any acute

neuropathological problems, and the consequences may only show years later when language problems emerge at school age. Another DLD risk factor that has been given relatively little attention is poor sleep quality. It is well possible that not all sleep irregularities in children with DLD are currently being recognized, particularly those that are associated with nocturnal EEG irregularities tend to go unnoticed. More research into sleep problems and nocturnal EEG irregularities in children with DLD is important, as sleep characteristics may inform early diagnosis and treatment, even before severe language problems have manifested themselves. It will also be interesting to examine the specific contribution of different sleep stages (REM, NREM) to cognitive and linguistic processing in children with DLD.

It is also important to note here that even though elimination or avoidance of risk factors for DLD (and suboptimal development in general) appears to be a straightforward remedy, reality may prove to be more stubborn, as many of these risk factors tend to accumulate in families that are low on societal and economical capital. For such families, elimination of risk factors may be less a matter of personal choice than of societal and political conditions.

For a number of the risk factors discussed here, plausible biological mechanisms are emerging. An example in point is preterm birth, which interferes with maturation and structural growth of regions in the cerebral cortex known to support neurocognitive functions that are critical for language development. We were able to link some of the biological mechanisms to existing neurocognitive theories of DLD. This information can be used to test specific hypothesis on how detailed steps of these pathways are effected. For example, phonemic discrimination may be affected by auditory processing and/or memory consolidation, and can mechanistically be disrupted at different levels of processing (from cochlear level, STG, up to IFG; see Fig. 1).

For other risk factors, the mechanisms by which they affect language development are not yet well understood. For instance, the biological pathway to explain the enhanced vulnerability to DLD of male compared to female children remains largely unresolved. A better understanding of differential genetic and hormonal modulation of brain development will help resolve this in the future. In this review, we show that timing and interactions between factors may determine when language development is (or is not) affected. Future research is necessary to further explore this, for example by comparing children with and without language problems who have all been 'exposed' to a risk factor for which the mechanism is not well understood.

As described in this review, different risk factors are likely associated with specific phases of prenatal and postnatal neurodevelopment and, furthermore, they affect neurobiological mechanisms that interact with one another directly, or that are involved in intricate feedback (and feedforward) loops. Generally, to further elucidate the pathways that enhance the risk for DLD, it would seem necessary to invest in large-scale longitudinal studies on both population samples and clinical samples that incorporate a wide range of variables pertaining to DLD risk factors. Specifically, our understanding of the etiology and pathology of DLD would benefit from studies that include systematic genetic screening, anamnesis and monitoring of sleep patterns, and repeated assessments of auditory processing, memory and executive functions, and that incorporate, ultimately, structural and functional brain imaging. Importantly, detailed behavioral analysis of the individual deficits are necessary in such studies to disentangle potential clusters in disorders that can be tied to specific mechanisms. Furthermore, obtaining a complete picture of the neurobiological mechanisms would require that we relate systemic mechanisms and functional outcomes to underlying molecular processes. Adequate animal models can play a role here, provided that we can identify cross-species methods that can be safely and responsibly used with animals and human children.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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