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Improving Mental Health in Persons with Intellectual Disability – From Science to Practice

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Entitled 'From Science to Practice: Improving Mental Health in Persons with Intellectual Disability', the 2021 EAMHID congress brings together a range of stakeholders and champions the implementation of scientific findings to improve people's daily lives and clinical practice. The various contributions from the invited speakers gathered in this book will further stimulate the transfer of knowledge from research to practice and provide the option for an in-depth engagement with the different topics. We hereby aim to further promote the exchange of knowledge in terms of evidence-based methods and best-practice examples throughout Europe.

A central question is the understanding (or not understanding) of people with developmental disabilities. Hereby, different approaches are chosen from the authors to address this fundamental question in its epidemiological, developmental, psychological, therapeutic and educational facets. What idea do we have of the other person? And how can we support her or him to live a good and healthy life? When reading through the different contributions, the relationship between the person with a disability, their families, social environments and the professionals seems to be key. Different options to shape and consider interactions and communications will be presented by the international colleagues, and it will take a lot out of the reader to implement these demanding tasks into their daily life.

Social inclusion, structured social activities and participation are considered significant strategies for improving mental health. Supporting individuals with intellectual and developmental disabilities thus leads to system changes in the larger context. This can be impacted upon by organizational and structural aspects of service providers, but also the development of medical standards in treatment and care.

The topics addressed by the authors were arranged according to *basic science*, *mental health*, *treatment and care*, and *living in the community.* All contributions are cutting-edge and many propose innovative ways of support and service provision. We hereby want to inspire the readership and paint the thrilling picture of moving forward – side by side with the persons, their families and your colleagues.

One chapter stands alone for itself as it gives us insights into one of the darkest chapters of German history, the Nazi regime and the devastating T4 action – the murder of persons with disabilities. In the light of the future-oriented goals of the

congress we want to keep the memories alive to all the victims and cruelties that happened that time. It must not be forgotten, not today, and not in the future.

We finally want to thank all the authors for their additional work they put into the writing of the chapters or the given interviews. And now, we invite you to dive into the exiting read. Take care and stay safe – wherever you are!

Tanja Sappok and Reinhard Burtscher

Berlin, June 2021

Focus on the brain: how individual genes, prenatal epigenetic characteristics and early interaction experiences shape resilience and risk

Nicole Strüber

Abstract

The individual genetic makeup of people, their prenatal programmed epigenetic characteristics and early interaction experiences influence the chemistry and the neuronal networks in the brain. This in turn affects how people deal with high demands later in life, how effective they are in regulating their emotions, and whether they feel comfortable in relationships.

Numerous research results show that traumatic experiences can shape the development of the brain in such a way that the risk of developing mental illnesses is increased. Secure attachment experiences, on the other hand, can create resources that help people to deal with later traumatic or chronic stress experiences appropriately. Secure attachment experiences are therefore the basis for resilience. Later in life, psychotherapy, but also other factors, can reduce the influence of an unfavorable early imprint on behaviour through effects on the brain.

Introduction

A child's genes and early experiences shape the development of its brain, its temperament and its later personality. These factors shape whether the child develops into a shy or even socially inhibited person, an impulsive person or even a person willing to take risks. Genes and experiences also influence the ability to cope with stress and the risk of developing mental illnesses or behavioural disorders.

But how can genes affect brain development? And how can something as immaterial as experiences affect the brain with its cells and molecules, and how can cells and molecules give rise to temperament and mental characteristics? To explain this, let us start with an even more fundamental question: how do human brains differ in the first place?

Individual nerve cell networks and individual substance systems

Whenever we feel, think or act, and even when we (mistakenly) think we are doing nothing at all, numerous networks of nerve cells are active in the brain. Circuits from distant and neighbouring cells are activated by all that is going on inside and around us. Sensory information from the environment is connected with information from inside the body, with information about habitual behaviour patterns or explicit memories of previous situations. The information is often transmitted from one cell to the next via chemical synapses. Many cells use fast-acting neurotransmitters such as glutamate, glycine or GABA for this chemical communication.

People differ not only in how their nerve cells are interconnected, but also in the extent to which the communication between their nerve cells is influenced by other substances called neuromodulators. These substances are released, for example, when the environment or our own needs require the brain to react quickly or persistently, because something is particularly important and needs our attention and motivation. This could be a complicated traffic situation, a threatening interpersonal situation, a sporting competition, the cooking of a delicious dessert or whatsoever.

If the brain detects such a situation, these modulatory substances are released by specialised cells, often localised in structures of the brainstem or midbrain, and usually distributed via their nerve fibres to different areas of the brain. Once there, the substances bind to their binding sites, their so-called receptors, and influence the activity of other nerve cells - those nerve cells that are currently occupied with the world around us and our own needs. The substances modulate how we process the information. Thus, in dangerous situations, for example, the brain can be put into a very reactive state, which we call 'alert'. The substances make us alert, attentive, motivated, flexible or euphoric.

Qualities such as alertness, attentiveness or for instance the motivation to engage with others in social interactions are – as we all know – not equally present in different individuals. One reason for this is that people differ in the effectiveness of these substances. People differ in how well these substances are broken down or transported back into the cell after they have taken effect, and also in the number of binding sites for these substances. One person then develops a highly efficient cortisol system, another an outstandingly functioning oxytocin system and a third person both.

Genes and experiences form the basis for individual brain development

But how do these differences arise? First candidate: the genes. The various processes in the body (including those in the brain) are, as we know, controlled by genes. The genes code for the various proteins. These include proteins that bind the neuromodulators described and mediate their effect, and that transport these substances back into the cell or break them down, for example.

The genes coding for these proteins often exist in different forms (polymorphisms). Therefore, in some people more and in others less or different forms of these special proteins are built. The processes controlled by the proteins then function more or less effectively.

Let us look at the frequently described gene for the serotonin transporter. Serotonin is a modulatory substance whose effects include making us react less impulsively and instead flexibly and goal-oriented (Worbe, Savulich, de Wit, Fernandez-Egea, & Robbins, 2015). If serotonin is released in the brain into the synaptic cleft between the nerve cells, it must then be transported back into the cell via a transporter protein. A region of the gene coding for this transporter can exist in different forms. People therefore differ in how much serotonin transporter protein is produced, how efficiently the return transport works and how much serotonin remains in the cleft for an effect. And via this mechanism, how well serotonin modulates brain activity and our behaviour in everyday life.

In addition, serotonin has a significant effect on brain development, both on the development of the serotonergic system itself and on the tissues where the fibres of the serotonin-releasing cells end (e.g. Gaspar, Cases, & Maroteaux, 2003). This effect on development in particular seems to be responsible for the finding that a certain gene variant for the serotonin transporter is related to mental illness (Ansorge, Hen, & Gingrich, 2007). It has been observed that people with a specific allele of the gene for the serotonin transporter are more likely to react to negative life events with depression or suicidal thoughts than individuals who have inherited the other gene variant from both parents (Caspi et al., 2003). Through this effect, one's genes influence a person's tendency to develop mental illnesses as a result of stressful events.

The same is true for other genes of the serotonin system as well as for genes of other substance systems. The genes determine how well the substance systems function. And the substance systems influence brain development or even acute brain activity, experience, behaviour and the risk for mental illnesses and behavioural disorders. A variant for the oxytocin receptor, for example, influences how

more loving care in their early childhood that restrains their stress system and steers it in the right direction. If this happens, these children seem to be able to make good use of the reactivity of their stress system in order to cope with the high demands of the environment.

Beyond early childhood

Individual genes and early experiences thus influence how nerve cells are interconnected and how the brain is influenced by substance systems. The person who experienced a secure attachment relationship and correspondingly little stress in early childhood has a good chance of developing a well-functioning oxytocin system with sufficient receptors as well as a well-functioning stress system (for an overview see Strüber, 2016; 2019).

The positive influence of early attachment experiences on neuronal connections and the long-term setting of modulatory substance systems may explain why securely attached children often develop so well. In the often-cited longitudinal study from Minnesota, for example, it was shown that school children who can look back on a secure early attachment relationship are very independent and self-reliant, can regulate their emotions well, are less anxious but at the same time more empathetic towards others, respond to their environment in a curious, inquisitive and flexible way, and have high self-esteem (Sroufe, 2005).

An increasing number of studies also show that adult attachment representations are related to stress coping and emotion regulation skills (for reviews, see Gander and Buchheim, 2015). When adult subjects are exposed to a social stress situation (Trier Social Stress Test), those with a secure-autonomous attachment representation report little perceived stress. They respond to the situation with a high release of oxytocin and a moderate release of cortisol. Individuals with other attachment classifications, suggesting an unprocessed less optimal attachment history, experience greater subjective stress in this situation. Their oxytocin release in the stressful situation is reduced and their cortisol response is also not the same as that of subjects with a secure attachment representation (Pierrehumbert, Torrisi, Ansermet, Borghini, & Halfon, 2012).

Early experiences may also influence one's parenting skills, contributing to the transmission of attachment classifications from one generation to the next. Adults who have developed a secure internal model of attachment as a result of their genes and their own attachment history respond to being with their infants with a higher oxytocin response (Strathearn, Fonagy, Amico, & Montague, 2009). Oxytocin also has a relationship-enhancing effect in the parental brain. In the parents' brain, oxytocin strengthens the connection of infantile stimuli to the reward system and thus supports that being together with the child is experienced

as pleasant. In addition, it reduces the parents' own aversive reactions to the child (e.g. in the face of persistent crying) via an effect on the amygdala and promotes the empathic reaction of the mother via an influence on the anterior insula. The child also reacts to the sensitive and loving care with a high release of oxytocin and establishes a secure attachment relationship with its parents. Accompanied by correspondingly optimal emotional brain development, this in turn forms the ideal basis for later parenting skills of its own (for an overview see Rilling and Young 2014).

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Neurobiology of dissociation: a stress perspective

Kim Hinkelmann

Abstract

The term dissociation describes an interruption of the normally integrative functions of consciousness, memory, identity or perception of the environment. Dissociative phenomena are quite common and also occur in everyday life. The development of dissociation is multifactorial, i.e. caused by an interaction of biological, life and learning history as well as genetic factors.

Dissociative symptoms are multifaceted and range from feeling confused to the loss of pain sensation and the ability to act, with functional neurological symptoms such as movement disorders or seizures counted among them in ICD-10. However, 'psychological' dissociative symptoms are often reported in the context of traumatization, so that a connection with the stress response is obvious.

Introduction

This chapter will use examples from animal and human studies to provide an overview of various neurobiological factors that play a role in dissociation, as well as illustrating possible mech-anisms of dissociation based on findings from stress research.

The term 'dissociation' describes a disruption in the usually integrated functions of consciousness, memory, identity, or perception of the environment (American Psychiatric Association, 2013). Dissociative symptoms are manifold and range from, 'being beside yourself' to the loss of the ability to feel pain and or to move. Mild dissociative phenomena are very common: in the general population, up to 6% of people report relevant dissociative phenomena or symptoms (Seedat et al., 2003). More severe dissociative phenomena, such as derealisation (the feeling that

the world around you seems unreal) or depersonalisation (the feeling of stepping out of one's own body or observing a situation from above) often occur in traumatic situations. Dissociative experiences are part of the diagnostic criteria for acute stress disorder and post-traumatic stress disorder (PTSD) as well as borderline personality disorder (American Psychiatric Association, 2013). However, dissociative symptoms are seen in many psychiatric disorders (Lyssenko et al., 2018) and thus may seem to be non-specific at a first glance. Historically, the occurrence of dissociation as well as dissociative disorders was closely related to the concept of trauma. Current concepts of dissociation discuss a multifactorial genesis, i.e. dissociation stems from a combination of genetic, biographical and biological factors.

Concepts of dissociation: the diathesis-stress model

The diathesis-stress model is a framework for understanding the development of psychological disorders, assuming that, 'etiologic factors underlying psychiatric illness can be divided into those that are present from an early age and are temporally stable in their effect (diathesis) and those that are temporally discrete, occurring close in time to disorder onset (stress)' (Kendler, 2020). The current diathesis-stress model of dissociation assumes that dissociation is a reaction to stress (Butler et al., 1996). If a person with a vulnerability (i.e., a higher tendency to dissociate) is exposed to early-life stress, e.g. abuse in childhood, he or she will react more frequently with dissociation. Later on in life, this 'practice' will enhance the probability to react with dissociation even to minor stresses or strains in everyday life.

How do we respond to stress: the concept of fight, flight and freeze

A plethora of preclinical studies revealed three essential behavioural patterns in response to stress: fight, flight and freeze. In situations of imminent threat, these defensive responses are rapidly selected and executed. Furthermore, those defensive behaviours can be rapidly switched and are dependent on contextual factors like the existence of escape routes and proximity of the predator.

These behavioural patterns are considered to be highly conserved in the course of evolution and seem to be advantageous for survival (Gross and Canteras, 2012; Tovote et al., 2016).

When a threat is detected (e.g. when a mouse spots a cat), stress-response systems are activated, thus (nor-) adrenaline and cortisol are released leading to

adaptive processes like an increase in blood pressure and heart rate as well as providing energy to enhance the probability of a successful flight (or fight). If flight or fight are no longer an option, there are various freezing or immobility reactions to increase the chance of survival. Freezing is a state of attentive immobility increasing chances of survival when facing predator threat, as the predator may have greater difficulty detecting a rigid and still prey animal (Roelofs, 2017; Volchan et al., 2017). Tonic immobility is another, later reaction in the defence cascade in response to a severe threat (e.g., predator attacking). Here the prey seems motionless, rigid, sometimes remaining in bizarre postures (playing dead). This behavioural response may be advantageous as many predator species do not eat rotten carcasses and lose interest in the prey. Both the distance from the predator as well as the availability of escape routes seem to drive the selection of defence behaviours: more distant threats seem to evoke freezing reactions, whereas imminent threats and lack of escape routes induce tonic immobility (Blanchard et al., 2011).

Neural correlates of defensive behaviours, especially their key structures within the amygdala and periaqueductal grey (PAG), are relatively well studied in mammals. In both, stimulation evokes freezing and blocking (via lesions) result in absence of freezing reactions (Roelofs, 2017). Both structures are closely connected. Furthermore, opioid-mediated pathways from the PAG towards the spinal cord mediate analgesia (Roelofs, 2017). Both, sympathetic and parasympathetic branches of the autonomic nervous system are activated in defensive behaviours. Sympathetic activity leads to heart rate acceleration ('palpitations' in face of danger) and the fight-flight response is mainly sympathetically-driven. Freezing is associated with parasympathetic dominance and a decrease in heart rate ('fear bradycardia') (Fanselow, 1994). Fear-associated bradycardia can result in syncopes. The PAG slows the sympathetic driven arousal down and thus keeps the fight-flight reactions on hold (Roelofs, 2017). The PAG therefore has a central role in switching between fight-flight and freeze responses.

Endocrine responses are closely related to fight-flight and freeze behaviour. In response to stress, the HPA-axis is activated and cortisol (in humans) or corticosterone (mammals) is released (Joels and Baram, 2009). Extensive work from Kalin and others have shown that HPA-axis stimulation increases and blockage of the HPA-axis decreased stress-induced freezing (Kalin & Shelton, 1989; Kalin, 1993; Kalin et al., 1998).

Fight-flight-freeze has mostly been studied in rodents and non-human primates. The high anatomical similarity of neural structures involved in fear processing (i.e. amygdala, hypothalamus, PAG) across all mammals suggests that they also have a similar function in humans (Gross & Canteras, 2012). There is an ongoing debate whether dissociative symptoms, especially derealization and depersonalisation, could be linked to freezing behaviour.

New insights on psychopathological assessment in persons with intellectual disability and low-functioning autism spectrum disorder

Marco O. Bertelli and Annamaria Bianco

Abstract

Intellectual disability (ID) and autism spectrum disorder (ASD) are associated with a broad vulnerability to concomitant health issues, especially psychiatric disorders, with higher prevalence rates compared to the general population. ID and ASD often co-occur, and their differentiation may be difficult, especially in the context of increasing severity of cognitive impairment.

In persons with ID and/or low-functioning autism spectrum disorder (LF-ASD) the presentation of psychopathological symptoms and syndromes can considerably vary from that of the general population, for a number of reasons including specific cultural factors, cognitive and communicative impairments, developmental peculiarities, and neuro-autonomic vulnerability. Psychiatric symptomatology can present scarcely defined, chaotic, mixed, intermittent, atypical or masked. Even key elements of some syndromes, such as delusions, hallucinations or suicidal ideation may be hard to recognize, especially in persons with low or absent verbal communication skills who may only be able to express themselves through changes in behaviour.

Some problem behaviours have been identified as symptoms or groups of symptoms, specific to some psychiatric disorders, taking the name of 'behavioural equivalents', especially in persons with low functioning and low adaptive skills. These behavioural equivalents have to be carefully distinguished from other problem behaviours, based on some characteristic, such as onset, development, maintenance and extinction, especially in respect to other concurrent possible symptoms of a psychiatric disorder. Some behavioural equivalents have been included in the last adaptations of the DSM and ICD diagnostic criteria for the general population.

Instrumental assessment can usefully support clinicians in the identification of behavioural equivalents, although available tools show considerable differences in structural and psychometric characteristics. In this chapter, a new comprehensive set of tools is presented, whose automatic score reporting includes odds indicators of behavioural equivalence for every significant behavioural change, specifically 'relative syndromic weight', 'syndromic specificity', and syndromic clinical relevance. This new tool battery, called SPAIDD (Systematic Psychopathological Assessment for persons with Intellectual and Developmental Disabilities), has been designed to meet all the different practical needs related to every phase of the clinical intervention (general psychopathological screening, diagnostic categorical specification, dimensional diagnosis, and symptoms monitoring) and can be used by mental health professionals with different background and by the whole multidisciplinary team working with people with ID and LF-ASD. Authors of this tool system also tried to overcome the other main limits of previous tools, such as impossibility of being used across the range of cognitive and communication impairments, misalignment with DSM or ICD, lack of some main symptoms or syndromes, lack of chronological criteria, and high time expenditure.

The increasing availability of personal digital devices equipped with sensors offers new opportunities to continuously and passively measure human behaviour in relation to mental health states and environmental context and may help to increase our understanding of behavioural symptoms of different psychiatric disorders.

Prevalence of psychiatric disorders and epidemiological biases

All types of psychiatric disorders are common in people with intellectual disability (ID) and low-functioning autism spectrum disorder (LF-ASD), which is perhaps not surprising, given the complex mix of biological factors, psychological and social disadvantages, and additional developmental factors that people with ID/LF-ASD have and are exposed to (Bertelli et al., 2015; Bertelli et al., 2009; Luckasson et al., 2002).

Overall, the prevalence rates of mental disorders are higher than in the general population (Mazza et al., 2020; Cooper et al., 2007). Psychiatric disorders are frequently overlooked in people with ID for a variety of reasons, including diagnostic overshadowing, complexity and multi-morbidity, reliance on caregiver reports, and, above all, presentations that are quite different from those in the general population. A previously undiagnosed mental disorder can be found in around 30% of persons, especially in those with more severe cognitive and communication impairments (Peña-Salazar et al., 2020). Failure to identify additional

disorders results in delayed treatment, which has serious consequences for the illness's course and outcome.

Psychiatric assessment in persons with ID is difficult since psychiatric disorders frequently occur in conjunction with other neurodevelopmental disorders, such as ASD or attention deficit hyperactivity disorder, and physical disorders, impairments, and disabilities such as epilepsy. Individual tests demonstrate that practically all adults with ID suffer from multi-morbidity (two or more disorders) (Kinnear et al., 2018). Other research has found that even when the conditions sought were limited to a small number of physical and psychiatric disorders receiving input from general practitioners, the majority of people had multi-morbidity, with the extent of this in young 20-year-old adults being comparable to that of 50-year-olds in the general population (Cooper et al., 2015; Carey et al., 2016). Some physical disorders relate to the persons underlying cause of ID, but lifestyle and environmental factors, sub-optimal support and healthcare are also important contributors (Cooper et al., 2017).

The frequency with which several neurodevelopmental and behavioural abnormalities coexist is so high that a specific term has been coined to describe it, that is ESSENCE (Early Symptomatic Syndromes Eliciting Neurodevelopmental Clinical Examination), which includes major problems in motor skills, general development, speech and language, social interaction and communication, behaviour, hyperactivity or impulsivity, hypoactivity, inattention, and sleep or feeding difficulties. The ESSENCE clustering is increasingly supported by genetic data, also in reference to a broader group of conditions including some neurologic (i.e., epilepsy) and psychiatric disorders (Gillberg, 2010).

Because of this high multimorbidity, people with ID and/or LF-ASD are subjected to a variety of pharmacological therapies, the side effects of which might resemble psychopathological symptoms, leading to a mistaken diagnosis of co-occurring psychiatric disorders and, in some cases, the need for further medication.

The prevalence of psychiatric disorders reported by studies varies depending on the methodologies used. Differences in reported rates are mostly related to the disorders classified as mental disorders, such as whether or not problem behaviours and ASD are included among other psychiatric disorders, as well as the size of the ID population examined. The types of assessments conducted and diagnostic criteria employed, as well as whether data was collected from existing records such as general practitioner health records, which are likely to underreport the prevalence of mental illnesses, represent other factors that significantly affect study outcomes. Given that the boundary between mild ID and the general population merges, particularly in adulthood, some studies report rates separately for people with mild ID from people with moderate-to-profound ID. Rates vary also by the age ranges included in the studies.

Some authors have attempted to synthesize data from studies of the prevalence of psychiatric conditions in children and adolescents (Einfeld et al., 2011) and adults (Buckles et al., 2013; Mazza et al., 2020), but these syntheses have inherent limitations as the authors acknowledge. Reported rates of psychiatric disorders in children and young people with ID range from 30% (Rutter et al., 1970; Birch et al., 1970) to 50% (Dekker et al., 2002), though it should be noted that these rates include Problem Behaviours (PB). A study examining robust data from UK private household surveys, reported a prevalence of 36% in 641 children and young people (aged 5-16 years) with ID, mostly of mild degree (Emerson & Hatton, 2007). More recently, the first study on a population-representative sample of US adolescents found that 65% met lifetime criteria for at least one psychiatric disorder (Platt et al., 2019).

For adults with ID, reported prevalence ranges from 14.5%, excluding PB, ADHD, ASD, dementia, personality disorders, people aged 65 and over, and people with severe ID (Deb et al., 2001), to 43.8%, in reference to adults with moderate-to-profound ID only (Bailey, 2007). The conditions covered in studies and the quality assessment used seem to represent the main drivers of prevalence variability in adults (Deb et al., 2001; Cooper et al., 2007). The largest adult population-based prevalence study in which each person (aged 16 years and over) was individually assessed, included 1,023 adults with ID (Cooper et al., 2007). It found a point prevalence of psychiatric disorders of 28.3% using rigorous techniques (or 40.9% if problem behaviours are also included). People with mild ID had a prevalence of 25.4%, whereas adults with moderate-to-profound ID had a rate of 30.2% (Cooper et al., 2007).

The effect of ID severity on prevalence of psychiatric disorders is controversial, with some findings indicating higher rates in persons with severe/profound ID (He et al., 2018; Peña-Salazar et al., 2020), and some others indicating the opposite (Axmon et al., 2018; Mazza et al., 2020).

Prevalence of psychiatric disorders in persons with both intellectual disability and autism spectrum disorder

Persons with both ID and ASD present several symptoms and deficits which are not seen in those with ID or ASD alone, and a different frequency of co-occurrent disorders (Boucher et al. 2008).

Research shows an inverse relationship between IQ and severity of ASD, with rate of PB in ASD getting lower as long as IQ increases (Totsika et al., 2011; Kurzius-Spencer et al., 2018). Conversely, severity of ASD symptoms – and not that of ID – resulted directly associated with the rates of stereotypies (Matson & Kozlowski, 2011).

Video-feedback intervention to support parents with intellectual disabilities

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Abstract

More and more people with intellectual disabilities are starting a family of their own, often based on the intense desire to become a parent. However, when we are looking at the choice to become a parent, people with intellectual disabilities encounter many challenges. The parties to the United Nations Convention on the Rights of Persons with Disabilities (2006) have seen it as pertinent to affirm the right of persons with disabilities to start a family and call for appropriate assistance to people with intellectual disabilities performing their child-rearing responsibilities.

Several studies underpin that parents with intellectual disabilities are able to learn parenting skills and can improve parenting behaviour. Three important factors are contributing to this result: parents are willing to ask for support and accept the support offered; parents are provided with effective parenting support programs and parents have access to a supportive social network.

In the Netherlands, from 2008 until 2017, the consortium, 'What works for parents with intellectual disabilities' (VU – Amsterdam) conducted research based on these protective factors. An evidence-based video feedback intervention for the general population, based on attachment and coercion theory, was tailored and tested for parents with intellectual disabilities (Video feedback Intervention on Positive Parenting for Parent with Learning Difficulties; VIPP-LD) in a randomized controlled trial and demonstrated to alleviate parenting stress for the whole group and the group parents with low adaptive functioning improved in parenting behaviour. At the moment, this intervention is continuing to be implemented. Professionals are in training for VIPP-LD intervention and start to support families headed by parents with intellectual disabilities with this intervention in settings where families live independently in their own houses and in families living in special family support homes.

In this chapter, we will show how the results of the research done are transferred to practice. We take you into the process of implementation, the way to introduce this evidence based intervention as a valuable treatment in families headed by parents with intellectual disabilities, the way to get governments interested, involved and supportive (including funding) and the training of professionals. We take you to the best practices but also to the lessons learned. And last but not least, we will share the stories of parents themselves, the way they feel supported in their parenthood.

Introduction

Ask people what they consider important in their lives and they will often mention: a child, being a parent, or forming a family. People with intellectual disabilities have similar dreams and desires as those without disabilities. Becoming a parent is an important and intense transition in a person's life, and in the context of being an adult is often seen as a highly regarded social role. (Baumeister, Vohs, Aaker, & Garbinsky, 2013). However, parenthood is challenging for many people with intellectual disabilities, as we can see in the high rate of child removal by child welfare authorities and several sociocultural and economic factors like health problems and poverty (Booth, Booth, & McConnell, 2005a, 2005b; Emerson & Brigham, 2013; Hindmarsh, Llewellyn, & Emerson, 2015; McConnell, Feldman, Aunos, & Prasad, 2011; Willems, De Vries, Isarin, & Reinders, 2007).

The choice of becoming a parent is a human right outlined in the United Nation Convention on the Rights of People with Disabilities (2006). Article 23 states:

> *'States Parties shall take effective and appropriate measures to eliminate discrimination against persons with disabilities in all matters relating to marriage, family, parenthood and relationships, on an equal basis with others. In paragraph 2 of this article the Convention mentions that states are bound to 'render appropriate assistance to persons with disabilities in the performance of their child-rearing responsibilities.'*

Article 26 underpins the responsibilities of professionals. They need to work according to the latest evidence base of their profession and keep themselves updated. Part 3 also mentions that knowledge and experiences need to be used to develop methods and programs especially designed for or tailored to the needs of people with (intellectual) disabilities. The convention is now ratified in 182 countries.

Thus, parenthood is seen as a fundamental right. The right to parenting brings the duty to take care of and educate a child. Several studies underpin that parents with intellectual disabilities are able to learn parenting and caring skills, and can improve these skills (Feldman, 1994, 2004; Wade, Lewellyn, & Matthews, 2008).

In the Netherlands the consortium *What works for parents with intellectual disabilities?* (2008-2017, VU – Amsterdam) carried out different studies on parents with intellectual disabilities. In one of these studies an evidence-based video feedback intervention for the typical population, based on attachment and coercion theory (VIPP-SD; Juffer, Bakermans-Kranenburg, & Van IJzendoorn, 2008) was tailored and tested for parents with intellectual disabilities resulting in the VIPP-LD: Video feedback Intervention on Positive Parenting for Parent with Learning Difficulties (Hodes, Meppelder, Schuengel, & Kef, 2014).

This paper starts with a description of some of the major characteristics of the group of parents with intellectual disabilities and then addresses the features of effective support for this group. Following that, we describe the development of the VIPP-LD, the training and schooling and the place of this intervention in parenting support programs. A case study illustrates the clinical work. We mention future implications.

Parents with intellectual disabilities: definition and prevalence

The American Association on Intellectual and Developmental Disabilities (AAIDD; Schalock, Luckasson, & Tassé, 2021) defines intellectual disability as 'characterized by significant limitations both in intellectual functioning and in adaptive behaviour, as expressed in conceptual, social and practical adaptive skills. This disability originates during the developmental period, which is defined operationally as before the individual attains age 22.' Adaptive skills are skills needed to perform adequately in everyday life and assessed based on the individual's typical performance at home, school, work and leisure.

Parents with intellectual disabilities are mainly represented in the group of persons with a mild intellectual disability (IQ from 50/55-70) and in the group of persons with borderline intellectual functioning (IQ 71-85), both in combination with significant limitations in adaptive behaviours. In this article, the term intellectual disability will be used for the total group of people with mild intellectual disabilities and borderline IQ functioning.

Quite a few people with intellectual disabilities become parents. A clear estimation is not easy to obtain. There is no official registration of people with intellectual disabilities nor of people with intellectual disabilities who become mothers or fathers.

The estimates in the Netherlands are that about 1.5% of persons with intellectual disabilities will have children based on research by Willems et al. (2007). In Sweden, research reported a percentage of 2% (Weiber, Berglund, Tengland, & Eklund, 2011), an American study estimated 1% (Parish, Mitra, Son, Bonardi, Swoboda, & Igdalsky, 2015) and out of the 1.2 million people with mild intellectual disabilities in the United Kingdom, about 7% would become parents (Emerson & Hatton, 2014).

Parents with intellectual disabilities are facing multiple risk factors like health problems, stress, depression, and histories of non-parental or institutional upbringing. These risks appear to influence their chances of succeeding as parents. Compared to the general population, parents with intellectual disabilities are more likely to live in deprived neighbourhoods, have low incomes and report low levels of social support (Emerson & Brigham, 2014; Hatton & Emerson, 2003; Hindmarsh et al., 2015; Willems et al., 2007).

Parents with intellectual disabilities are overrepresented as clients of child welfare and child protection services. Often, they do not have access to proper health services. Health care information, but also general information about parenting is often too complicated, resulting in poor or wrongly understood support. Providers of services report that they lack knowledge to help these families properly.

Parenting with intellectual disabilities

Several researchers mentioned that parenting and the well-being of the child is influenced by an interplay of different child, parent, family and environmental factors (Feldman & Aunos, 2010; Feldman & Tahir, 2016; Knowles, Machalicek, & Van Norman, 2015; Wade, Llewellyn, & Matthews, 2015; Willems et al., 2007). Intellectual disability as a single factor was not found to be a determinant of maladaptive parenting. Poverty seemed to be an important risk factor (Emerson & Brigham, 2014). Willems et al. (2007) reported that one third of the parents with intellectual disabilities in the Netherlands showed good enough parenting (as defined by the lack of involvement of child protection services and the absence of concerns about abuse and neglect). However, not one single factor determines the quality of the parenting. A balance model, based on the interplay of protective and risk factors (risk factors need to be compensated for by protective factors; protective factors alleviate risk factors) better explained this quality. Three important protective factors were uncovered as associated with 'good enough' parenting: asking and accepting support, effective intervention strategies and the presence of a supportive social network.

Parents with intellectual disabilities do benefit from parenting support and are able to learn important skills to secure their child's safety, health, cognitive devel-