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Neurodevelopmental Disorders (NDD) without boundaries: research and interventions beyond classifications

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Abstract :

On June 2022, the 2nd Webinar “Neurodevelopmental Disorders (NDD) without boundaries took place at the Imagine Institute in Paris and was broadcasted live and in replay. The aim of this webinar is to address NDD in a dimensional rather than in a categorical approach. Several speakers were invited to present their researches on the subject. Classifications in NDD were discussed: irritability in NDD, involvement of the immune system in neurodevelopment, nutrition and gut microbiota modulate brain inflammation and neurodevelopment, co-occurring conditions in autistic adolescents and adults without intellectual disability. Classifications in psychiatric disorders were asked : mapping the effect of genes on cognition and autism risk, epigenetics and symptomatic trajectory in neurodevelopmental disorders, the autism-schizophrenia continuum in two examples: minor neurological signs and EEG microstates, the cerebellum in schizophrenia and autism: from imaging to intervention perspectives. Both genetic and environmental factors, along with clinical and imaging features, argue toward a continuum between NDD but also with adult psychiatric presentations. This new paradigm could modify the therapeutic strategy, with the development of large-spectrum treatments or new psychotherapies addressing co-occurring symptoms. The complexity and the heterogeneity of NDD apply well to the next scientific and political challenges: developing international convergence to push back the frontiers of our knowledge.

This article is a summary of the 2nd webinar “Neurodevelopmental Disorders (NDD) without boundaries: research and interventions beyond classifications” sponsored by the French National Academy of Medicine, the autism and neurodevelopmental disorders scientific interest group (GIS), the International Research Network Dev-O-Psy and the French Institute of Psychiatry (GDR3557). Oral presentations are available as a replay on the following website (in French):

<https://autisme-neurodev.org/evenements/2022/04/12/tnd-sans-frontieres-la-recherche-et-les-interventions-au-dela-des-classifications/>

The webinar was broadcasted from the Imagine Institute in Paris, with the participation of :

- Mrs Claire Compagnon, Interministerial Delegate for the National Strategy for Autism and Neurodevelopmental Disorders
- Prof. Jean-François Allilaire, Permanent Secretary of the French national Academy of Medicine
- Prof. Stanislas Lyonnet, Director of the Imagine Institute, Paris, France
- Prof. Catherine Barthélémy, Director of the GIS
- Prof. Marie-Odile Krebs, Coordinator of the IRN Dev-O-Psy and of the Institute of Psychiatry, GDR 3557, France

Speakers:

- Diane Purper-Ouakil (CHU Montpellier, France)
- Pierre Ellul (INSERM U959, Immunology, Immunopathology, Immunotherapy/Excellence Centre for Autism & Neurodevelopmental Disorders, France)
- Quentin Leyrolle (Nutrineuro, Bordeaux)
- Julien Dubreucq (Child and Adolescent Psychiatry Department, Saint-Etienne University Hospital, France)
- Sébastien Jacquemont (Sainte-Justine UHC, Montreal, Canada)
- Boris Chaumette (Institute of Psychiatry and Neuroscience of Paris, INSERM U1266, France)
- Anton Iftimovici (Institute of Psychiatry and Neuroscience of Paris, INSERM U1266, France)
- Charles Laidi (Child Mind Institute, New-York, USA & Univ. Paris-Est Créteil, Assistance Publique des Hôpitaux de Paris, GHU Henri-Mondor, INSERM U955 IMRB, Neurospin CEA Saclay)

Moderator :

- Paul Olivier, Project manager of the GIS

INTRODUCTION :

The aim of this webinar is to address NeuroDevelopmental Disorders (NDD) – Autism Spectrum Disorder (ASD), Attention Deficit Hyperactivity Disorder (ADHD), specific learning disorders, intellectual disabilities... - in a dimensional rather than in a categorical approach (Claire Compagnon). Facing the immense diversity of situations of persons with NDD, the broadest national and international scientific studies must be implemented. The research must be transdisciplinary, with a lifelong vision, involving public and private stakeholders. The scientific challenge is even more complex, because NDDs do not correspond to distinct disorders. Moreover, the frequency of comorbidities (co-occurring disorders) lead to question the definition of NDDs. A paradigm shift must be implemented in order to understand the variety of functional expressions (Jean-François Allilaire). NDDs are a major public health issue: at the Imagine Institute, NDD stands for half of the consultations (32,000 consultations/year). Functional profiles of persons with NDDs spread all over a wide spectrum with

similarities and specificities. The cross-talk between the rare and the frequent presentations therefore seems inevitable to aim personalized and appropriate interventions (Stanislas Lyonnet).

NDD, beyond classifications

1) Irritability in NDD - Towards new therapeutic approaches, Diane Purper-Ouakil

Regarding emotional functioning, a distinction can be made between emotional reactivity (reaction to a stimulus, analysis of a stimulus in its emotional valence) and emotional regulation (modulation of physiological and behavioural responses to emotions). Irritability is defined behavioural reactions that are inappropriate to the environment, unsuited to the stimulus and difficult to modify. Irritability is the main behavioural manifestation of emotional dysregulation. There are 2 types of irritability: phasic irritability defined by frequent and high intensity tantrums inappropriate to developmental age and context (Brotman & al, 2017; Stringaris & al, 2018), more specifically associated with ADHD (Cardinale & al, 2021); tonic irritability defined by the period between tantrums when the person manifests a persistent moodiness (Brotman & al, 2017; Vidal-Ribas & al, 2016). Irritability is transnosographic and occurs frequently in NDD, anxiety disorders, depression and in lesions of the central nervous system. The DSM-5 now identifies irritability as a core dimension of a specific disorder, the disruptive mood dysregulation disorder (DMDD). This new diagnostic category was aimed to reduce the number of diagnoses of paediatric bipolar disorders and longitudinal studies have highlighted that irritability is likely to develop into both internalizing and externalizing disorders, but not specifically bipolar disorder (Hawes & al, 2020). Emotional hyperreactivity, is the main emotional deficit in ADHD, and can worsen with age but children with ADHD also show other facets of emotional dysregulation such as emotional recognition and regulation difficulties (Graziano & al, 2016). New therapeutic approaches have been proposed to cope with irritability. The objective is to move from external strategies implemented by parents/primary caregivers (based on anticipation and contingency management) suited to a younger public to personal strategies using transnosographic therapeutic approaches such as emotional socialization programs, multisystemic therapy, combined treatments (expert drug treatment combined with behavioural treatment) (de la Cruz & al, 2015; Towbin & al, 2020; Lawrence & al, 2016; Gonzalo Salazar de Pablo & al, 2022).

2) Involvement of the immune system in neurodevelopment, Pierre Ellul

Neurodevelopmental disorders are now considered in a dimensional manner. Indeed, neurodevelopmental trajectories are influenced by different pre- and post-natal risk factors that will give rise to a spectrum of neurodevelopmental difficulties. The objective of many research projects is to study these different risk factors in order to better understand and potentially modify these trajectories. Among the environmental risk factors, prenatal maternal immune activation (such as infections or auto-immunity) can have a negative impact on the fetal neurodevelopment. In the postnatal period, it has also been shown that early infections may impact normal brain development. Thus, we hypothesized that early and repeated inflammatory insults may alter the neurodevelopment. We used a cohort of 184 subjects with childhood autoimmune and autoinflammatory diseases (ARTEMIS) and studied their impact on the neurological development. The combination of early onset, repeated inflammation and potential delay in diagnosis could represent factors associated with childhood autoimmune and auto-inflammatory diseases leading to NDD. The aim of the Artemis cohort was to isolate a time window in which these inflammatory conditions could disrupt the neurodevelopmental trajectory. We showed that, there is an age-dependent impact of systemic inflammation on neurodevelopment. As the age of onset of the immune systemic diseases increases, the risk of developing NDD decreases. Importantly, the brain seems to have a certain resilience to inflammation, as early introduction of disease modifying therapy (in the first 5-6 months) can rescue the risk of developing NDD. It is therefore crucial to systematically screen for NDDs in child immune systemic disorder, especially when they have an early onset.

3) Nutrition and gut microbiota modulate brain inflammation and neurodevelopment, Quentin Leyrolle

Maternal malnutrition is a well-known risk factor for NDD. The composition of the gut microbiota is found to be different in children with NDD. On the other side, microglial cells are involved in brain development, particularly in the establishment of functional neural networks. Altering the microbiota alters the maturation of microglia, which results in impaired neural network development. A deficiency in omega-3 fatty acids lead to a loss of the homeostatic signature of the microglia, associated with an increased phagocytic activity (*Madore & al, 2020*). The consequence being a dysfunction of the neuronal network and subsequent behavioral deficits. In individuals with such nutritional deficiencies, the overproduction of omega-6 derivative also leads to an increased microglial phagocytic activity. Moreover, omega-3 deficiency is responsible for a delay in the maturation of oligodendrocytes and myelination deficit. This adverse outcome is reversible with the administration of clemastine (a remyelinating anti-histaminic drug) (*Leyrolle & al, 2021, Leyrolle & al, 2022*). A cumulative effect of omega-3 deficiency and prenatal inflammation on neuronal network dysfunction has been observed (*Labrousse & al, 2018*). Furthermore, metabolites produced by the gut microbiota appear to have an important role in brain development. The disappearance of beneficial bacterial metabolites (including hippurate) seems to be deleterious for normal neuronal development (*Vuong & al, 2020*). In mice, preliminary data have shown that hippurate prevents some of the behavioural and metabolic effects of social stress.. Conversely, the increase in deleterious metabolites, in particular the microbial metabolite 4-ethylphenyl sulfate (4-EPS), is associated with impaired brain myelination in individuals with ASD (*Needham & al, 2022*). In adolescents with ASD, a pharmacologic strategy aiming at avoiding 4-EPS intestinal absorption (sequestering 4-EPS at this level) induced a decrease in 4-EPS plasmatic concentration, associated with decreased anxiety, irritability and gastrointestinal symptoms. Together, these data suggest that it would be interesting to identify target populations for nutritional or metabolite supplementation to prevent poor neurodevelopmental outcome.

4) Co-occurring conditions in autistic adolescents and adults without intellectual disability: from diagnosis to interventions, Julien Dubreucq

Co-occurring conditions in autism are frequent but represent an important challenge for psychotherapeutic interventions. Emotional dysregulation explains 50% of the variance in anxiety in autism and could underlie other co-occurring health conditions. Suicidal risk is increased by 4.2 times for autistic individuals and the risk of suicidal recurrence is increased by 5 times. Access to health care for autistic people is often difficult, which explains why they are often treated in emergency rooms (anticipated stigma from health providers, difficulties in communicating with untrained health professionals). Offering psychosocial interventions to autistic people should be based on their strengths and aspirations. Several objectives may be targeted: optimizing the match between the person and the environment, promoting the social inclusion, improving the quality of life. Interventions should be adapted to the presence of co-occurring health conditions. The treatment of co-occurring conditions can be done through a more nuanced use of compensation strategies, and psychotherapies such as Cognitive Behavioral Therapy (CBT) or mindfulness.

Psychiatric disorders, beyond classifications

1) Mapping the effect of genes on cognition and autism risk, Sébastien Jacquemont

Deletions and duplications account for between 5 and 15% of patients referred for NDD. Deletions are commonly responsible with decreased gene activity, while duplication are associated with increased gene activity. 200 genes have been identified to be associated with autism and 500 genes with intellectual disability and, more broadly, with human

cognition. Precise estimation of the quantitative effects of these rare genetic variations is crucial. Additionally, the effects of common variants are beginning to be incorporated into predictive models (Mollon & al, submitted). Many variants are currently known to be deleterious, but their quantitative effect is poorly understood. It would be relevant to build statistical models capable of predicting the effect of any deletion or duplication on cognition, on the risk of autism, or on other psychiatric disorders (Jacquemont & al, 2022). As first results, a meta-analysis highlights that a single deletion of any "loss-of-function intolerant genes" is consistently associated with a loss of 2.6 Intelligence Quotient (IQ) points (and 0.81 IQ points for a duplication) in the general population and in autistic population cohorts. Additive models predict the effects of 47 recurrent CNVs on IQ with 78% accuracy <https://cnvprediction.urca.ca/>.

Cognition is a polygenic trait, even in NDD related with rare variants. There is no single gene involved in more than 10% of the effect of the 16p11.2 deletion. Furthermore, the role of the DYRK1A gene does not exceed 30% of the cognitive effects of CNV in Down syndrome. The more loss-of-function intolerant genes the CNV affects, the greater the impact on IQ. But half of the genome appears to be included in cognitive processes. With such a heterogeneity, it may be difficult to prioritize the mechanisms of cognition since a large range of different mechanisms seems to be involved.

Similarly, almost all brain regions have an impact on cognition. Brain regions that are very sensitive to chromosomal deletions are those that are less sensitive to duplication, and vice versa. There is a mirror effect deletions and duplications on the cerebral cortex and the cognitive functions (Sydnor & al, 2021). For many years, it was thought that one gene was responsible for a particular disorder. Current knowledge shows that many genes are regulators of thousands of other genes expression. These complex polygenic phenomena must be taken into account in developing future therapeutic strategies.

2) Epigenetics and symptomatic trajectory in neurodevelopmental disorders, Boris Chaumette

The concept of neurodevelopmental disorders refers to a variety of clinical presentations with the comorbidities being the rule, not the exception. This phenotypic overlap involves neuropsychiatric disorders as the same genes code for different diseases: "the genes have not read the DSM". For instance, polymorphisms have shown that ADHD correlates with bipolar disorder, with depression, with autism, or that schizophrenia correlates with autism and bipolar disorder (*Brainstorm Consortium – Science, 2018*). Rare variants, especially Copy Number Variants, lead to different neurodevelopmental and psychiatric phenotypes (Collins & al, 2021). There is also a variable expression over the lifetime, leading to different symptomatic trajectory. Every patient does not have the same phenotype at different ages of life (Jonas & al, 2014) suggesting a 'lifelong' neurodevelopment. Psychiatric disorders can therefore be the adult continuum of NDD. The delayed expression of psychiatric disorders in some individuals may be the effect of epigenetics through two phenomena, DNA methylation or microRNA expression. For example, in the 22q11 deletion, the effect of stress may change the DNA methylation in genes that have been well identified as involved in cognition, social communication, etc. Also, genes that are deregulated by microRNA expression are involved in neurodevelopment. Determining these changes could help to predict the evolution of subjects at genetic risk by explaining the variable penetrance. It may open the way to targeted therapeutic measures as well as preventive approaches to avoid "over-accident".

3) The autism-schizophrenia continuum in two examples: minor neurological signs and EEG microstates, Anton Iftimovici

ASD and schizophrenia are frequently comorbid. This is explained by an overlap of genetic risk for both disorders (Lee & al, 2019), while neuroanatomically, they also share the same morphological anomalies compared to healthy controls (*ENIGMA Writing Committees, 2021*). Here, we present two other modalities of this continuum, neurophysiological and neurocognitive. In resting-state EEG, microstates have been described as unstable states of whole-brain polarisation, lasting

around 100 ms, which correspond to different modes of information processing: phonological, visual, self-referential and interoceptive, and attentional processes. EEG microstates are hypothesized to correspond to continuous shifts of the global neural workspace randomly changing between these different modes in order to be constantly in a state adapted to treat any type of information (*Michel & al, 2018*). Having measured the imbalance of microstates C and D (microstate C represents the self-referential mode ; microstate D represent the attentional mode), we found that both in psychosis and in ASD, there seems to be more self-referential and less attentional modes, suggesting subjects consider more interoceptive information and are focused on their own thoughts rather than being outward looking. In a second experiment, subjects watched a movie for assessment of social cognition in schizophrenia and ASD, and we measured clinical dimensions such as neurological soft signs (NSS include motor coordination, motor integration, sensory integration, abnormal movements, lateralization, extrapyramidal signs), the autism quotient, and the thought, language and communication scale. While social cognition was more affected on average in ASD than in schizophrenia, causal mediation analysis showed that this effect was mainly mediated, transdiagnostically, by NSS. Since NSS reflect a neurodevelopmental burden of dysexecutive functioning, we may hypothesize a continuum between executive and social functions that are impaired along the autism-schizophrenia spectrum.

4) The cerebellum in schizophrenia and autism: from imaging to intervention perspectives, Charles Laidi

The cerebellum represents 15% of the total volume of the brain but contains more than 50% of the total number of neurons. Through intricate neural networks, the functioning of the cerebellum is closely connected to the brain. The role of the cerebellum is not limited to motor coordination. As illustrated by Schmahmann's syndrome (psychiatric pathology such as schizophrenia or ASD, which the etiology is an ischemic or tumoral lesion of the posterior region of the cerebellum), the cerebellum is involved in a very large number of cognitive tasks (*Andreasen & al, 1993; Van Overwalle & al, 2015*) and may play a role in different NDD. In ASD, altered connectivity between the cognitive component of the cerebellum and the superior temporal region is observed using resting-state functional MRI (Magnetic Resonance Imaging). However, there is no atypicality in the structural anatomy of the cerebellum in ASD (*Laidi & al, 2022*). Schizophrenia seems to be associated with a decreased volume of the cerebellar cognitive area (*Laidi & al, 2015, 2019*). This neurostructural abnormality would lead to a decreased connectivity between the cerebellar vermis and the prefrontal brain cortical areas. Clinical improvement following rTMS (repetitive Transcranial Magnetic Stimulation) treatment to ameliorate negative symptoms of schizophrenia correlates with modified connectivity between the cerebellar vermis and prefrontal cortex (*Brady & al, 2019*). There is therefore interest in continuing large-scale studies to elucidate the link between cerebellar anatomy and mental health.

CONCLUSION :

NDD are an umbrella of overlapping conditions. Rather than a categorical definition, a dimensional approach may be a more appropriate view. Indeed, both genetic and environmental factors, along with clinical and imaging features, argue toward a continuum between NDD but also with adult psychiatric presentations (i.e. schizophrenia). This new paradigm could modify the therapeutic strategy, with the development of large-spectrum treatments or new psychotherapies addressing co-occurring symptoms. The complexity and the heterogeneity of NDD demands harmonized and joint international effort, as illustrated by our recent webinar which involved French, Canadian and US researchers and clinicians. Thus, the motto "Neurodevelopmental Disorders without boundaries" applies well to the next scientific and political challenges: developing international convergence to push back the frontiers of our knowledge.

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