

the considerable variability in these neurobiological correlates between patients can be translated into the clinical setting.

Objectives We aimed to identify neuroimaging predictors of clinical course in patients with schizophrenia. Combined with the identification of genetically determined markers of schizophrenia risk, our studies aimed to elucidate the biological basis and the clinical relevance of inter-individual variability between patients.

Methods We included over 150 patients with schizophrenia and 279 healthy volunteers across five neuroimaging centers in the framework of the IMAGEMEND project [1]. We performed multiple studies on MRI scans using random forests and ROC curves to predict clinical course. Data from healthy controls served to normalize the data from the clinical population and to provide a benchmark for the findings.

Results We identified ensembles of neuroimaging markers and of genetic variants predictive of clinical course. Results highlight that (i) brain imaging carries significant clinical information, (ii) clinical information at baseline can considerably increase prediction accuracy.

Conclusion The methodological challenges and the results will be discussed in the context of recent findings from other multi-site studies. We conclude that brain imaging data on their own right are relevant to stratify patients in terms of clinical course; however, complementing these data with other modalities such as genetics and clinical information is necessary to further develop the field towards clinical application of the predictions.

Disclosure of interest Giulio Pergola is the academic supervisor of a Hoffmann-La Roche Collaboration grant that partially funds his salary.

Reference

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S120

Neuroimaging findings in ADHD and the role of genetics

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ADHD is frequently diagnosed in children and adults. The disorder is highly heritable. However, the genetic architecture of ADHD is complex, with multiple genetic variants of individually small effect size contributing to disease in most patients.

In our own studies as well as in the large mega-analyses of the ENIGMA ADHD Working Group, we have investigated the brain substrates of ADHD. We find the disorder to be characterized by delayed sub-cortical and cortical growth of gray matter in childhood, which gradually normalizes in adulthood: sub-cortical volumes as well as cortical thickness and surface area are smaller in children with ADHD, but become indistinguishable from healthy individuals in adulthood. The situation looks different for white matter connectivity: both in childhood and adulthood, widespread differences in the major white matter tracts are found. The pattern of findings suggests that alterations in myelination might lie at the basis of such case-control differences. Since the disorder and many brain structural measures affected in ADHD are highly heritable, we investigated the overlap of genetic risk factors for ADHD with genetic factors involved in brain volume. This resulted in the identification of several genetic variants contributing to disease risk as well as ADHD-related brain phenotype.

In conclusion, we find ADHD to be a disorder of delayed brain maturation in terms of gray matter, but of persistently altered white matter connectivity across the lifespan. Genetic factors influencing both disease risk and brain measures might improve our understanding of disease etiology and persistence.

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S121

Cortical and Sub-cortical volumetric abnormalities in bipolar disorder

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Previous MRI studies of bipolar disorder (BD) are often limited by small sample sizes and heterogeneity exists with regard to neuroimaging markers. To address these limitations, the ENIGMA Bipolar Disorder Working Group collected the largest BD neuroimaging data set ever studied ($n=6,500$). Here, we review findings from sub-cortical volume and cortical thickness and area analyses.

ENIGMA harmonized analysis methods were applied to 28 international pooled study samples of MRI data and involved sub-cortical and cortical imaging analyses. We assessed differences between BD and healthy controls (HC) using both mega and meta-analytic multiple linear regression models, adjusting for standard covariates (age, sex, etc.), and correcting for multiple comparisons.

Sub-cortical volume analysis revealed we found consistent volumetric reductions in BD patients for hippocampus and thalamus and enlarged lateral ventricles in patients. In BD, cortical gray matter was thinner in frontal, temporal and parietal regions of both brain hemispheres. BD had large general effects on mean gray matter thickness in both left and right brain hemispheres. Further we found that psychopharmacological treatment showed significant associations with cortical thickness and surface area.

The ENIGMA pipeline allows for identification of brain MRI abnormalities in BD in the largest analysis ever conducted. The results suggest a pattern of brain structure abnormalities, which provide novel insight in pathophysiology of BD, and potential effects of mood stabilizing agents.

Disclosure of interest Received speaker's honorarium from Lundbeck, Lilly, Otsuka

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Symposium: Schizophrenia and clinical psychopathology: From research to clinical practice

S122

Are deficits in social cognition differentiating between schizophrenia and affective disorders

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Over the last decades, in matters of the assessment of psychopathology and its clinical consequences, there has been an increased interest in neurocognitive function including non-social and social cognition.

Classic psychopathology –as represented e.g. by the standardized AMDP system– focuses on pathognomonic signs for the categorization of syndromes [1] and differentiates between disturbances

of perception, concentration, memory retention and long-term memory. A recent short screen for cognitive impairment in psychiatry (SCIP) has addressed five domains of cognitive function: verbal learning–immediate, working memory, verbal fluency, verbal learning–delayed and processing speed [2].

Using the SCIP in admissions from a defined catchment area in the southwest of Vienna we confirm the presence of cognitive deficits in schizophrenic patients and to a lesser degree in bipolar patients. The deficits were present in all five domains and no discriminatory pathognomonic signs could be found between schizophrenia and bipolar disorder.

Recently, possibly selective deficits in social cognition have been described in schizophrenic patients [3]. We review the evidence on the specificity of social impairment to schizophrenia.

Disclosure of interest The authors declare that they have no competing interest.

Reference

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S123

From (Psycho) pathology to diagnosis: psychiatry nosology beyond dichotomy

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As in all medical disciplines, diagnosis in clinical psychiatry should be reached in a step-wise approach: after assessing the chief complaint of the patient, a careful examination of the psychopathology follows e.g. by using the AMDP system [1] to preliminarily conclude the process with a syndromal classification [2]. This syndromal classification is of great importance as it guides the initiation of therapy in daily life practice. After gaining additional information (e.g. investigation in the course of the disease, brain imaging, thorough assessment of cognitive function, exclusion of organic causes) a final diagnosis is possible. Unfortunately, a premature jumping to diagnosis is not uncommon (with the potential consequence of incorrect therapies).

In addition to these difficulties, recent neurobiological research has shown that nosologic assignments through conventional diagnostic classifications are far less specific than assumed, revealing a large overlap between diagnostic categories [3,4], e.g. between Schizophrenia and affective disorders. Consequences of this finding are discussed both for the construction of future classification systems and for therapy.

Disclosure of interest The authors declare that they have no competing interest.

Reference

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estimated from genome-wide SNPs. *Nature genetics* 45.9 2013:984–99.

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Symposium: Autism spectrum disorders: From the neurobiology to interventions

S124

Psychosis and autism spectrum disorders

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Autism spectrum disorders (ASD) and schizophrenia were separated into different diagnostic categories in the late 1970's (DSM-III) having previously been considered as related diagnostic entities. Since then, several lines of evidence have indicated that these disorders show clinical and cognitive overlaps as well as some common neurobiological characteristics. Furthermore, there is a group of patients presenting with ASD and psychotic experiences who pose particular diagnostic and management challenges and may represent a subgroup of ASD more closely linked to psychosis. Evidence from a study of the first empirically derived classification of children with ASD in relation to psychosis based on three underlying symptom dimensions, anxiety, social deficits and thought disorder, will be presented. Further phenomenological, genetic and neuroimaging research on the clinical boundaries and overlapping pathophysiology of ASD and psychosis may help better define their relationship and lead to more effective interventions. Understanding this relationship will also provide a framework of working with patients with mixed clinical presentations.

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S125

Neurobiology of autism spectrum disorders

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Autism Spectrum Disorders (ASD) is a group of neurodevelopmental disorders with heterogeneous etiology characterized by deficits in social cognition, communication, and behavioral flexibility. Disturbances on molecular and cellular level in early brain development incl. intercellular communication, an unbalanced ratio between certain neuronal populations and maturation/differentiation process, oxidative stress, happening in embryonal stages, might be promising candidates to explain the development of autistic symptoms.