of patient subgroups mapping on distinct cognitive and clinical features.
Policy of full disclosure: None.

S-38-004
How much does working memory contribute to learning impairments in schizophrenia?

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Objective: The cause of learning deficits in schizophrenia remains unclear. While many studies have investigated potential sources in impaired striatal dopaminergic mechanisms, other neuro-cognitive systems might be responsible. In particular, prefrontal-dependent working memory is well known to be impaired in patients, and might be accountable for slowed learning. We aim at parsing out the contributions of these different systems to learning impairments.
Methods: Medicated patients (N = 49) and matched healthy controls (N = 36) performed a reinforcement learning task with varying degrees of cognitive demand. We designed a hybrid computational model that allowed us to extract the independent contributions to behavior of fast, capacity-limited working memory from the contributions of slow reinforcement learning.
Results: Patients showed strong learning impairments compared to healthy controls. Our hybrid model accounted well for both groups' behavior, showing that their learning resulted from at least two separable processes. Model fitting showed significant differences between groups in the working memory parameters, but not in the reinforcement learning process.
Conclusion: Our results support the idea that an important part of schizophrenia patients' learning impairment stems from working memory deficits, rather than from the incremental reinforcement learning process implemented in the dopaminergic and striatal system. This study highlights the fact that multiple brain mechanisms contribute to simple single behaviors, and that careful experimental design and computational mechanisms are required to disentangle their contributions to behavior. This is essential to understanding the underlying neurobiological processes of pathologies.
Policy of full disclosure: Consulting for Roche.

S-39 Epidemiological and clinical aspects of psychosis risk syndromes and psychotic experiences in children, adolescents and adults

S-39-001
Prevalence, familial liability and clinical correlates of psychotic experiences and symptoms in children of the general population: the Copenhagen Child Cohort 2000

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Objective: Psychotic experiences (PE) in individuals of the general population are hypothesized to mark the early expression of the pathology underlying psychosis. This notion of PE as an intermediate phenotype is based on the premise that PE share genetic liability with psychosis. The study aimed to examine whether PE in childhood was predicted by a family history of mental disorder with psychosis rather than a family history of non-psychotic mental disorder; and whether this association differed by severity of PE.
Methods: We examined 1632 children from a general population birth cohort assessed at age 11–12 years by use of a semi-structured interview covering 22 psychotic symptoms. The Danish national registers were linked to describe the complete family history of hospital-based psychiatric diagnoses. Uni- and multivariable logistic regressions were used to test whether a family history of any mental disorder with psychosis, or of non-psychotic mental disorder, versus no diagnoses, was associated with increased risk of PE in offspring (hierarchical exposure variable).
Results: The weighted lifetime prevalence of PE at age 11–12 years was 10.9 % (CI 9.1–12.7). The majority of children with PE (n = 172) either had a DSM-IV-mental disorder or sub-threshold subjective difficulties. The risk of PE increased with emotional and neurodevelopmental disorders and problems. The occurrence of PE in offspring was significantly associated with a history of psychosis among the first-degree relatives (adjusted RR = 3.29, 95 %CI 1.82–5.93). The risk increased for combined hallucinations and delusions (adjusted RR = 5.90, 95 %CI 2.64–13.16). A history of non-psychotic mental disorders in first-degree relatives did not contribute to the risk of PE in offspring, nor did any mental disorder among second-degree relatives.
Conclusion: Our findings support the notion of PE as a vulnerability marker of transdiagnostic psychosis. The effect of psychosis in first-degree relatives may operate through shared genetic and environmental factors.
Policy of full disclosure: None.

S-39-002
Follow-up findings of the Bern Epidemiologcial At-Risk (BEAR) study

F. Schultze-Lutter (University of Bern, Child & Adolescent Psychiatry, Bern, Switzerland; C. Michel, B. G. Schimmelmann)

Objective: In clinical samples of early detection services, both ultra-high risk and basic symptom criteria are associated with a 2- to 3-year conversion rate of roughly 30 %. Yet, their prevalence and course outside help-seeking samples is largely unknown and therefore studied in the BEAR study that is funded by two independent project grants of the Swiss National Foundation (SNF).
Methods: At baseline, 25 % of the young adults from the community (16–40 years) reported any lifetime risk symptom, but only 3 % met any risk criterion. After 2.5 years, those with any lifetime risk symptom (RISK) and a control group (CONTROL) are being re-interviewed. At the time of writing, 274 follow-ups were conducted: in 143 RISK and 131 CONTROL. In regression analyses, the association of baseline status with various outcomes and predictors of report of any risk symptom at follow-up were examined.
Results: Three RISK (2 %), but no CONTROL reported a meanwhile development of first-episode psychosis. Furthermore, RISK were significantly more likely than CONTROL to report presence of any risk symptom within the follow-up period (33 % vs. 6 %; OR = 7.52, 95 % CI 3.39–16.70); and lifetime report of risk symptoms at baseline was the sole predictor of their report at follow-up. Altogether 8 % (11 % in RISK and 5 % CONTROL) met criteria for a non-psychotic axis-I disorder at or within follow-up without report of risk symptoms being a significant predictor of psychiatric morbidity.
Conclusion: This indicates that risk symptoms might frequently be not just fleeting experiences but tend to persist. Thereby, they do not seem to generally increase the likelihood of developing any full-blown mental disorder but—should the result of psychotic disorders

developing solely in RISK hold—might indeed predispose to the development of psychotic symptoms.
Policy of full disclosure: None.

S-39-003
Age as a source of heterogeneity in psychosis high risk research

S. Ruhrmann (University of Cologne, Psychiatry and Psychotherapy, Cologne, Germany; S. Schmidt, F. Schultze-Lutter)

Objective: Objective: Currently available reports on conversion rates show a broad heterogeneity. Several reasons have been discussed, including a lacking consideration of the dynamic character of the interaction of environmental factors with the individual vulnerability/resilience. One aspect which may reflect this dynamic is the interaction between risk indicators and age. The transitory phase from early adolescence to adulthood is associated with a whole bunch of biological, cognitive and social changes. The increasing risk is demonstrated by the rise of first manifestations from up to 1 % of all schizophrenia cases below age of 13 to up to one-third of all cases below age of 18. On this background, it seems noteworthy that the age structure of high risk differs markedly. Age effects, however, may remain undetected, as samples may not span the critical range or lack sufficient statistical power. We therefore analyzed age effects on conversion rates and outcome of intervention studies across samples.
Methods: Two meta-analyses were performed (http://www.europsy.net/publications/guidance-papers/), one on prediction, finally including 45 studies, one on prevention, finally including 15 studies. Categories for age distribution included: almost entirely minors ($\leq$18 years; CAD), almost entirely adults (minimum age 18 years or mean >18 with lower sd only spanning $\geq$18 years; ADULT), $\geq$50 % minors (median or mean age $\leq$18 years or mean $\leq$18 with upper sd still spanning $\leq$18 years; YOUTH).
Results: When ultra-high risk criteria defined inclusions, the two-year conversion rates in CAD were more than 50 % lower than those in ADULT ($p < .05$). Regarding intervention studies, effects were less clear, yet no CAD group was available.
Conclusion: Age composition of samples seems to be an important source of heterogeneity. CHR criteria should be used in children and early adolescents, but only with utmost care. Primarily psychosis preventive interventions in this age range are not supported by current evidence.
Policy of full disclosure: None.

S-39-004
Social environment as a risk factor for psychosis proneness

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Objective: To test whether spatial and social neighbourhood patterning of people at ultra-high risk [UHR] of psychosis differs from first episode psychosis [FEP] participants or controls, to determine whether exposure to different social environments is evident before disorder onset.
Methods: We tested differences in the spatial distributions of representative samples of FEP, UHR and control participants, and fitted two-level multinomial logistic regression models, adjusted for individual-level covariates, to examine group differences in neighbourhood-level characteristics.
Results: The spatial distribution of controls (n = 41) differed from UHR (n = 48; $p = 0.04$) and FEP participants (n = 159; $p = 0.01$), whose distribution was similar ($p = 0.17$). Risk in FEP and UHR groups was associated with the same neighbourhood-level exposures: proportion of single-parent households (FEP adjusted odds ratio [aOR]: 1.56 95 %CI 1.00–2.45; UHR aOR: 1.59; 95 %CI 0.99–2.57), ethnic diversity (FEP aOR: 1.27; 95 %CI 1.02–1.58; UHR aOR: 1.28; 95 %CI 1.00–1.63), and multiple deprivation (FEP aOR: 0.88; 95 %CI 0.78–1.00; UHR aOR: 0.86; 95 %CI 0.76–0.99).
Conclusion: Similar neighbourhood-level exposures predicted UHR and FEP risk, whose residential patterning was closer to each other's than controls. Adverse social environments are associated with psychosis before FEP onset.
Policy of full disclosure: None.

S-40 Current state and perspectives of policies for schizophrenia care

S-40-001
Quality assurance in schizophrenia treatment and care: state of the art and policy developments

W. Gaebel (Heinrich-Heine-University, Department of Psychiatry and Psychotherapy, Düsseldorf, Germany)

Objective: This lecture will address the relevance of quality assurance and different quality assurance tools in mental healthcare for people suffering from schizophrenia. Requirements for the evaluation and assurance of quality in mental healthcare will be outlined as well as the need to include quality assurance on policy agendas.
Methods: Quality assurance programs and instruments in mental healthcare for people suffering from schizophrenia will be reviewed. Specific examples of such programs and instruments as well as their methodological characteristics and goals will be outlined.
Results: In general, a first step to assure quality in mental healthcare for people suffering from schizophrenia is to include quality assurance in political agendas and plans in order to outline its scope and foster the development of adequate instruments. Quality assurance can address the structures, processes and outcomes of care. It is approached by different stakeholders, such as policy-makers, health insurers, care providers and people with schizophrenia themselves. Quality assurance tools, such as quality indicators and clinical practice guidelines, should be developed in systematic processes and on the basis of evidence-based literature.
Conclusion: In order to optimize and assure qualitative care delivery a systematic development, implementation and evaluation of quality assurance programs and instruments is necessary. Not only indicators on the structures and processes of care inform about quality but also outcome measures that focus on patients'perspectives, expectations and needs.
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S-40-002
Bridging the gap between neuroscience and policy care in schizophrenia

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Abstract: Psychiatry has profited considerably by new developments in molecular biology and imaging over the last 20 years. Our