the Paranoia Checklist), negative affect and the employment of specific ER strategies (e.g. reappraisal, acceptance, expressive suppression and rumination). Cross-sectional group differences were examined by conducting ANOVAs. Multilevel analysis was used to analyze the longitudinal data.
Results: Assessment and analysis of the interim data are currently in progress and will be completed by the date of the congress.
Conclusion: The insight into the mechanisms leading from negative affect to paranoia is likely to provide a basis for novel interventions, such as the training of functional ER strategies and would therefore corroborate the ongoing development of an interventionist-causal model approach to persecutory delusions targeting factors that have been shown to be causal to delusions.
Policy of full disclosure: The study has been funded by the German Research Foundation (DFG LI 1298/8-1).

S-29-004
Insomnia and paranoia: an experimental study with analysis of mediating mechanisms

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Objective: Our view is that insomnia may be a causal factor in the occurrence of psychotic experiences such as paranoia and hallucinations. However, the causal relationship is not established. The aim of the study was to assess the effect of insomnia on psychotic experiences via a sleep restriction manipulation (three nights of four hours sleep).
Methods: The study was a within-subjects crossover design that included a planned mediation analysis. 68 non-clinical volunteers completed a self enforced reduced sleep (insomnia) condition and a standard sleep (control) condition in randomised order in two consecutive weeks, with a weekend washout period. Psychotic experiences (paranoia, hallucinations, grandiosity, and cognitive disorganisation) and candidate mediating variables (negative affect and related processes, working memory, decision making, and perceptual processing) were assessed before and after each condition.
Results: Actigraphy verified an average of 5 h 15 min of sleep in the insomnia condition, versus 6 h 58 min in the control condition. After the insomnia condition, relative to the control condition, participants reported significant increases in paranoia, hallucinations and cognitive disorganisation, with no significant changes in grandiosity. The insomnia condition was also associated with significant increases in negative affect, negative self and other cognitions, worry, and working memory impairment. Mediation analyses indicated that changes in psychotic experiences were mediated by changes in negative affect and related processes, but not memory impairment.
Conclusion: The conclusion is that reduced sleep has a causal role in the occurrence of certain psychotic experiences in the general population, and that a key route is via negative affect.
Policy of full disclosure: None.

O-01 Factors impacting the risk of developing psychosis

O-01-001
Clinical high risk symptoms and criteria in the community: prevalence, clinical significance and risk factors for their occurrence

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Objective: In clinical samples, symptomatic ultra-high risk (UHR) criteria and the basic symptom criterion "cognitive disturbances" perform well in predicting psychosis, and best when both approaches are combined. However, little-to-nothing is known about clinical high risk (CHR) and their constituent symptoms in the community. Therefore, we studied the prevalence, clinical relevance, and moderators of CHR criteria and symptoms in the community.
Methods: Regression analyses involved 2683 community participants (age 16–40 years; response rate: 63.4%). Semi-structured telephone interviews were performed by well-trained psychologists.
Results: Lifetime and current CHR symptoms were reported by 21.1 and 13.8% of interviewees. Frequency of symptoms was mostly low, only 2.4% met any CHR criterion. A stepwise relationship underlay the association of the two types of CHR symptoms and criteria with the presence of mental disorders and functional deficits, with odds ratios being highest (7.4–31.8) when UHR and basic symptoms occurred together. Report of a family history of mental disorder generally increased risk for CHR symptoms. While younger age increased risk for basic symptoms, lifetime substance misuse and trauma increased risk for UHR symptoms.
Conclusion: Prevalence of CHR criteria was within the to-be-expected range from prevalence rates of psychoses. Clinical relevance of both CHR symptoms and criteria increased in a stepwise manner from basic symptoms via UHR symptoms to their combined presence, reinforcing the clinical utility of their combined use. The risk factors selectively associated with basic and UHR symptoms support developmental models relating basic symptoms to neurobiological and UHR symptoms to psychological factors.
Policy of full disclosure: None.

O-01-002
The role of parenting and family functioning in the development of psychotic experiences in adolescence (TRAILS)

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Objective: Psychotic experiences occur relatively frequent during adolescence in the general population, but generally disappear over time. The potential persistence of psychotic experiences is dependent on the presence of other risk factors. The current study aims to investigate the predictive value of parenting and family functioning on the development of psychotic experiences during adolescence in a population and clinical-referred sample.
Methods: The participants were 2059 Dutch children who participate in the 'Tracking Adolescents' Individual Lives Survey' (TRAILS), from a clinical ($n = 416$) and population ($n = 1643$) based sample. At age 11 family functioning (Family Assessment Device; Epstein, Baldwin and Bishop 1983), parental stress (Parenting Stress Index; Abidin 1983) and expressed emotion (perceived warmth, rejection and over protection, EMBU; Markus, Lindhout, Boer, Hoogendijk and Arrindell 2003) by both parents was assessed. At age 16 psychotic experiences (Community Assessment of Psychic Experiences; Stefanis et al. 2002) were measured. Linear regression models were computed to assess the predictive value of family functioning, parental stress and expressed emotion at age 11 on the reporting of psychotic experiences at age 16.
Results: In the population based sample, over protection from the mother at baseline (age 11) predicted both the frequency ($B = -.11$, $t(1574) = 4.45$, $p < .001$) and distress ($B = -.13$, $t(1498) = 2.38$, $p < .01$) of psychotic experiences at age 16. In the clinical sample, parental stress at baseline (age 11) predicted the frequency of psychotic experiences at age 16 ($B = -.05$, $t(410) = 2.64$, $p < .01$).