disorder were recruited and randomly allocated to one of two rehabilitation programs: Social Skill Training (SST) + Computerized Cognitive Training (CCT) (Group A) and usual rehabilitation activities of the Department (Group B). The active treatment phase lasted 6 months. Psychopathological aspects, as well as psychosocial and neurocognitive functioning, were assessed both before and after treatment. Group A subjects participated in two one-hour sessions of CCT and one two-hour session of SST. Group B patients spent an equivalent amount of time in the usual rehabilitation activities.

The two groups did not differ on baseline clinical, neurocognitive and psychosocial variables.

At the end of treatment, a worsening of the negative dimension was observed in group B, but not in group A, in which a significant improvement of two psychosocial indices (participation in family life and availability to work) was found.

The experimental program (SST+CCT) was more effective than usual rehabilitation activities of the departments.

S08.03

Improvement of prefrontal brain function in schizophrenia under atypical neuroleptic treatment

A.J. Fallgatter, M.J. Herrmann, A.C. Ehlis. *Department of Psychiatry and Psychotherapy, University of Wuerzburg, Wuerzburg, Germany*

Background and Aims: Various cognitive activation tasks in schizophrenic patients have demonstrated an altered function of the anterior cingulate cortex (ACC) interconnecting the prefrontal cortex with limbic areas. This prefrontal dysfunction is a main target of antipsychotic treatment, as it is considered to be involved in both negative symptoms and cognitive dysfunction.

Methods: Two- (NoGo-anteriorization; NGA) and three-dimensional topographical measures (source locations with the Low Resolution Electromagnetic Tomography; LORETA) of the event-related potentials elicited during the execution (Go) and the inhibition (NoGo) condition of the Continuous Performance Test allow an assessment of anterior cingulate function with extraordinary high inter-individual stability and retest reliability.

Results: These methods revealed a significant brain electrical hypoactivity in the ACC of schizophrenic patients as compared to age- and gender-matched controls. Both a neuropsychological index of ACC performance and the proposed electrophysiological measure of this region have been shown to be improved in patients treated with atypical but not with typical antipsychotics.

Conclusions: These results support the notion that a functional deficit of the ACC during response control is a core feature in schizophrenias, which can be improved by atypical antipsychotic treatment.

S09. Symposium: NEUROBIOLOGICAL FACTORS IN ANTISOCIAL DISORDERS: RESEARCH, CLINICAL AND ETHICAL IMPLICATIONS (Organised by the AEP Section on Forensic Psychiatry)

S09.01

Genetics and forensic psychiatric nosology

H. Anckarsater. *Forensic Psychiatric Clinic, Malmo University Hospital, Lund University, Malmo, Sweden*

Background: Research on genetic mechanisms involved in human behaviour stands before a huge discrepancy. One the one hand, twin research has shown that strong genetic effects are involved in creating individual differences in virtually all human behaviour patterns, including aggression. On the other hand, molecular genetic research has not been able to identify gene variants associated with such traits.

Aims: To review the current state of genetic research on aggression, psychopathy and criminality.

Method: Systematic literature searches for aggression, psychopathy, criminality, antisocial, conduct disorder and ADHD vs. gene/genetic, following both the epidemiological and the molecular strands.

Results: Genetic effects explain a considerable part the variance in aggression. No molecular genetic variant specifically involved in this causation has been identified, even if there are some promising findings.

Conclusion: Genes are important but the mechanisms involved are enigmatic and most certainly unspecific.

S09.02

Neurobiological markers in conduct disorder

S.H. Herpertz [1], B. Herpertz-Dahlmann [2]. [1] *Department of Psychiatry and Psychotherapy, Rostock University, Rostock, Germany* [2] *Department of Child Psychiatry and Psychotherapy, Aachen RWTH, Aachen, Germany*

Aggressive behavior in mental disorders may occur in childhood in the context of conduct disorder or in adulthood as a leading feature of personality disorders. Those children, who meet the criteria for conduct disorder already in early life (''early starters'') tend to exhibit high levels of aggression throughout development and continuation of violence in adulthood. They exhibit autonomic underarousal and low autonomic responses which have been shown to be predictive of adult antisocial behavior and which have been suggested to act as biological mediators through which genetic influences operate on antisocial behavior. This predictor is stronger in boys without psychosocial disadvantages compared to those boys with unfavorable social backgrounds and may therefore particularly reflect the biology-antisocial behavior relationship. There are several lines of evidence that aggressive behavior at any age is closely related to an individual's capability to regulate emotions. Emotions of anger or fear trigger reactive, impulsive aggression whereas a failure to experience fear, empathy or guilt facilitates instrumental aggression. Brain structures significantly involved in affect regulation, such as the amygdala and hippocampus, have been found to be smaller in early-onset CD boys compared to healthy controls. In emotional challenge tasks these boys exhibited increased amygdalar hyperresponsiveness to emotional stimuli; this finding might reflect a time-limited mechanism of compensation for smaller amygdala volumina in the maturating brain. Data from neuroimaging and electrophysiology will be forwarded to clarify the neurobiological underpinnings of children with conduct disorders and their risk of being the fledging adult violent offenders.

S09.03

Neurobiological correlates of antisocial personality traits - research findings and treatment implications

B.A. Vollm, M. Dolan, P. Richardson, S. McKie, R. Elliott, S. Williams, I. Anderson, J.F.W. Deakin. *Neuroscience and Psychiatry Unit, University of Manchester, Manchester, United Kingdom*

Background and Aims: There is increasing evidence for a neurobiological basis of antisocial personality disorder (ASPD),

https://doi.org/10.1016/j.eurpsy.2007.01.057 Published online by Cambridge University Press