

**SCHIZOPHRENIC PSYCHOSES AND VIOLENT BEHAVIOUR  
AETIOLOGICAL AND THERAPEUTIC ISSUES FROM A PREVENTIVE POINT OF  
VIEW**

**PhD Theses**

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## Contents

Introduction.....	2
Study 1: Minor physical anomalies in homicidal versus non-violent schizophrenia patients.....	4
Study 2: Symptoms of Traumatization, Guilt and Shame in the Post-Offensive Period .....	8
Study 3: Symptom profiles and parental bonding in two groups of schizophrenia patients.....	14
Tables and graphs .....	18
Summary of new observations.....	29
List of publications.....	30
Acknowledgements.....	32

## **Introduction**

### **Schizophrenia and Violent Behaviour**

In my thesis I am exploring possible connections between schizophrenic psychoses and violent, especially homicidal behaviour. The first two chapters offer an overview of the literature relevant to his subject.

In the theoretic introduction, I am examining possible contributing factors to the aetiology of homicidal behaviour. We can establish that the presence of delusions, thoughts of persecution as well as the trait hostility are only strongly correlated with later violent behaviour. Further contributing factors include comorbid substance abuse, lack of insight and compliance with treatment. In many cases, however, the homicidal behaviour is not directly connected to psychotic symptoms. Comprehensive approaches regard homicidal behaviour as a process: the history of antisocial traits and conduct disorder in childhood significantly increase the likelihood of later violent behaviour.

I discuss the preventive approach to schizophrenia in a separate chapter. Following a review of the premorbid and prodromal symptoms of the illness, as well as the neuro-developmental approach, I propose the introduction of a staging model. In this model, a poly-genetic alteration leads to a progressive delay in neuronal maturation. This delay later leads to early deficits in neuromotor functioning, sustained attention and working memory and a dysfunction of the context module. These deficits and dysfunctions are signs of a deficient prefrontal-orbitofrontal-limbic integration and later lead to marked cognitive, emotional dysfunction. This chapter promotes the identification of early pmorbid signs of schizophrenia that may be targeted in preventive interventions.

The general hypothesis of our three studies is that homicidal behaviour in schizophrenia is an indication of more severe psychopathology.

In our first study, we tested this hypothesis from a neuro-developmental perspective. For the first time in international research, we compared the number and the topological profile of minor physical anomalies in homicidal schizophrenia patients, non-violent matched control schizophrenia patients as well as healthy control subjects.

In our second study, we explored emotions, attitudes, post-offence behaviour and coping mechanisms after committing homicide. In this case, we used a different control group to

compare with homicidal schizophrenia patients: non-clinical perpetrators of homicide serving their prison sentences. This was also a pioneering study, which addressed the following issues. Are emotional reactions and coping mechanisms experienced and applied after the crime different in offenders diagnosed with schizophrenia from the ones experienced and applied by offenders without a psychiatric diagnosis? What is the possible role of shame, a moral emotion largely neglected in previous studies, in the post-offence reactions of perpetrators?

The main question of our third study was how parental bonding, including care and overprotection perceived by the child was related to psychotic symptoms and homicidal behaviour.

In the concluding chapter of my thesis, I discussed the experience gathered in the therapeutic work with psychotic patients who had committed a violent crime. Beyond the questions of psychotherapies with psychotic patients in general we have to consider the dilemmas of guilt versus illness as well as curative versus punitive aspects of treatment.

## **Study 1: Minor physical anomalies in homicidal versus non-violent schizophrenia patients**

### **Introduction**

Minor physical anomalies (MPAs) are mild and insignificant errors of morphogenesis which have a prenatal origin and may bear major informational value for diagnostic, prognostic and epidemiological purposes. The presence of minor physical anomalies is a sensitive physical indicator of embryonic development. Since both the central nervous system and the skin derived from the same ectodermal tissue in utero, minor physical anomalies may be external markers of abnormal brain development. Minor physical anomalies are considered to develop during the first and/or early second trimester of gestation (Aase 1990, Méhes 1985,1988, Pinsky 1985, Tényi et al 2004,2009) and represent potentially valuable indices of disturbances in early neurodevelopment. Once formed they persist into adult life and are readily detected on visual examination of the particular body area. Minor physical anomalies have been found with increased frequency in various neuropsychiatric illnesses (Waldrop et al 1971, Méhes 1988, Csábi et al 2008), schizophrenia (Gualtieri et al 1982, Lohr & Flynn 1993, Lane et al 1997, Trixler et al 1997,2001, Akabaliev & Sivkov 2007, Sivkov & Akabaliev 2003, Weinberg 2007) and affective disorders (Tényi et al 2009). We must make a clear distinction between minor malformations and phenogenetic variants. Minor malformations are always abnormal and are qualitative defects of embryogenesis, which arise during organogenesis. All malformations are developmental field defects and usually they are all-or-none anomalies. In contrast phenogenetic variants are quantitative defects of final morphogenesis and arise after organogenesis. Morphologically phenogenetic variants are the exact equivalents of normal anthropometric variants. Using a list of minor physical anomalies containing 57 minor signs collected by Méhes (1988). In our study, we used the scale developed by Méhes (1988), containing a total of 57 minor physical anomalies.

As no study has been published yet on the prevalence of MPAs in schizophrenia patients with the history of homicide, the aim of the present study was to investigate the rate and topological profile of minor physical anomalies in patients with schizophrenia with the history of committed or attempted homicide comparing them to patients with schizophrenia without homicide in their history and to normal control subjects.

The following hypotheses have been tested:

(1) Minor physical are more common in homicidal schizophrenia patients compared to non-homicidal schizophrenia patients and normal controls, which could support a stronger neurodevelopmental component of etiology in this subgroup of schizophrenia,

(2) a higher rate of minor physical anomalies is found predominantly in the head and facial regions in schizophrenia patients with the history of committed or attempted homicide compared to non-homicidal schizophrenia patients and normal controls, pointing at a more pronounced role of aberrant brain development in the case of homicidal schizophrenia.

## **Study**

### **Material and methods**

Using a list of 57 minor physical anomalies collected by Méhes (1988), 44 patients with the diagnosis of schizophrenia were examined with the history of committed or attempted homicide. All of these patients were treated in the Institute of Forensic Psychiatry (Igazságügyi Megfigyelő és Elmegyógyító Intézet, IMEI) in Budapest, Hungary during the evaluation of the study. As a comparison, 22 patients with the diagnosis of schizophrenia were examined, without the history of any kind of homicide and violence, while 21 normal control subjects were also observed for minor physical anomalies. The diagnoses of the patients were evaluated independently by two experienced psychiatrists according to the DSM-IV-TR (Diagnostic and Statistical Manual of Mental Disorders, 2000). All available clinical information and data were obtained from structured clinical interviews. In the compared schizophrenia group and the normal control group persons with antisocial behavior, impulse control disorder, personality disorder or any other neuropsychiatric disease were excluded.

#### Examination of minor physical anomalies

We have used the Méhes Scale for evaluation of minor physical anomalies, which includes 57 minor signs (Trixler et al, 2001, Tényi et al, 2009). The evaluated minor physical anomalies are shown in Table 1, also minor physical anomalies are connected to body regions for comparison and analysis of data. All participants gave informed consent, the study was performed in accordance with the Declaration of Helsinki and was evaluated following institutional guidelines. Two examiners (Gy.Cs., T.T.) investigated all the patients and controls separately. The examination of minor physical anomalies was done qualitatively (present or

absent) without scores being used, but where it was possible, measurements were taken with callipers and tape to improve the objectivity of examination.

### Statistical analysis

Before the statistical analyses interrater reliability was tested and the kappa coefficient was  $> 0,75$  for all items. Statistical analyses were carried out by applying the Kruskal Wallis-test for the simultaneous comparison of the three groups, Mann - Whitney U-test for the comparison of two groups with each other. Comparing the three groups by body regions z-test was introduced, because of multiple comparisons with Bonferroni correction. 2-sided Fisher's exact tests were used to compare two groups with each other by body regions, the level of significance chosen was  $p < 0,025$ , Odds ratios were calculated with 95% confidence intervals. For the analysis of the frequency of each individual minor physical anomalies the two-sided Fisher's exact probability test was used, the level of significance chosen was  $p < 0,025$ . All the statistical analyses were done by the use of SPSS Version 21.

### Results

We should consider as a robust finding that between the three groups a statistically highly significant difference rate could be detected in the prevalence of minor physical anomalies using the Kruskal-Wallis test ( $p < 0,001$ ). Also the comparison of two groups with the Mann-Whitney-U-test showed significant differences between them (homicidal schizophrenia patients versus non-homicidal schizophrenia patients,  $p < 0,001$ , homicidal schizophrenia patients versus normal controls,  $p < 0,001$ , non-homicidal schizophrenia patients versus normal controls,  $p < 0,001$ , see TABLE 1). The z-test comparison of the three groups by body regions confirmed that concerning the ear region, the two schizophrenia groups differed from the control group but not from each other, on the results of the head region the homicidal schizophrenia group was different from both the non-homicidal schizophrenia group and the normal control group, concerning the mouth the homicidal group was different from the non-homicidal schizophrenia group, analysing the trunk data the non-homicidal group differed from the normal control group, while concerning the legs the homicidal group was different from the normal group. Homicidal schizophrenia patients showed a higher frequency of MPAs in the head and the mouth regions compared to non-homicidal patients, higher rates of MPAs could be seen in the ear, head, eye and leg regions among homicidal schizophrenia patients compared to normal control subjects, while non-homicidal schizophrenia patients showed a higher frequency of MPAs in the ear region compared to normal controls. Two

minor malformations (flat occiput, double posterior hair whorl) were more prevalent in the homicidal schizophrenia group compared to the schizophrenia patients without aggression and violence, four minor malformations (flat occiput, double posterior hair whorl, furrowed tongue, Sydney line) appeared more frequently among the homicidal schizophrenia patients compared to normal control subjects, while two minor malformations (furrowed tongue, Sydney line) were more common in the non-homicidal schizophrenia group compared to the normal control group. One minor malformation (earlobe crease) was more frequent in the non-homicidal group compared to the homicidal schizophrenia group (see TABLE 2).

### **Discussion**

Our results are in line with results from neuroimaging studies, which have repeatedly shown structural abnormalities in violent and aggressive schizophrenia patients (Soyka 2011). This study also confirmed previous reports on the higher prevalence of MPAs among patients with schizophrenia (see the meta-analysis by Weinberg et al 2007), while the statistically more common appearance of MPAs in the homicidal schizophrenia group is the first such observation in literature.

Based on previous results (Green et al 1989, O'Callaghan et al 1991, Hata et al 2003, Trixler et al 2001, Gourion et al 2004) the higher rate of MPAs found predominantly in the head and mouth regions in schizophrenia patients with the history of committed or attempted homicide compared to non-homicidal schizophrenia patients, confirms the possibility of a more seriously aberrant brain development in the case of homicidal schizophrenia.

It is also important to mention the few reports on the association of the higher prevalence of MPAs with later violent behavior among individuals without psychoses (Raine 2002).

We can conclude that MPAs are more common in homicidal schizophrenia patients compared to non-homicidal schizophrenia patients and normal controls, which could support a stronger neurodevelopmental component of etiology in this subgroup of the disease.

Our results can be viewed as a major contribution to the predictive-preventive approach, since minor physical anomalies can be regarded as early detectable markers of pathological psychological development and behaviour. They may, hence, become central in interventions aimed at the prediction and the prevention of later disorders.

## **Study 2: Symptoms of Traumatism, Guilt and Shame in the Post-Offensive Period: a Comparative Study of Perpetrators With and Without Severe Mental Illness**

### **Introduction**

Based on criminal record surveys and retrospective accounts made by offenders, committing homicide in most cases involves an elevated level of stress (Stone 1992, Porter et al 2001).

Perpetrators of homicide often report feelings of anxiety, guilt and shame, even traumatism and/or grief (Fraser 1988, Harry and Resnick 1986, Thomas, Adshead and Mezey 1994, Pollock 1999)

Symptoms of traumatism were found to be comparably high in samples of perpetrators with a severe mental illness (SMI)(Papanastassiou et al 2004). Severity of PTSD-symptoms was significantly correlated with the severity of guilt-related cognitions as well as closeness to the victim (Crisford, Dare and Evangelis 2008)

Most studies addressed the role of offence-related guilt. It is, however, possible that shame, being another significant moral emotion, may also play an important part in the emergence of PTSD symptoms. The symptoms of shame and traumatization show a significant overlap (Wright and Gudjonsson 2007).

Studies show that homicidal offenders with a diagnosis of SMI show differences from those without such a diagnosis in the antecedents and motivations of the crime, and acute psychotic symptoms largely determine their appraisal of the entire situation and their behaviour (Hakkanen and Laajasalo 2006).

In our study, the following hypotheses were tested:

- 1) Perpetrators of homicide with a diagnosis of SMI apply different coping mechanisms in the post-offensive period than perpetrators without such a diagnosis.
- 2) Reported level of stress, symptoms of traumatism, guilt and shame coping mechanisms (habitual and post-offensive), experienced stress, shame, guilt and mechanisms of coping are interrelated.

## **Study**

### **Subjects, material and method**

All of our 80 subjects were, at the time of the study, incarcerated for either homicide (n=66) or attempted homicide (n=15). 57 subjects (prisoners) were recruited from three Hungarian high-security prisons, they had no history of psychiatric illness or treatment. 24 subjects (NGRIs) were recruited from Institute of Forensic Psychiatry (Igazságügyi Megfigyelő és Elmegyógyító Intézet, IMEI) in Budapest, Hungary. Members of this group were found not guilty by reason of insanity (NGRI) based on their severe mental illness (with a diagnosis of either schizophrenia, schizo-affective disorder or delusional disorder) and ordered to involuntary psychiatric treatment. Participants of the two groups were matched on sex, age and level of education. All participants gave informed consent. For descriptive data on our subjects, see TABLE 3.

State- and trait-anxiety were assessed using the Hungarian version of the State-Trait Anxiety Inventory (STAI) (Sipos, Sipos, & Spielberger 1988). We modified the time-frame in the instruction from previous week to time period after the perpetration of the crime.

For the assessment of preferences in coping strategies in anxiety-provoking situations, we used the Coping Preferences Questionnaire (CPQ, Oláh 1986). Here, we also modified the time-frame in the instruction to the time after the perpetration of the crime.

We used the Offence-Related Shame and Guilt Scale (ORSGQ, Wright & Gudjonsson 2007) to assess feelings of guilt and shame related to the homicidal act.

We applied Kruskal-Wallis ANOVA and paired-sample t-test to compare the scores of the groups.

SPSS 17.0 software was used for the statistical analysis.

## **Results**

### **Scales**

Mean trait-anxiety in the two groups is within the average range for the Hungarian population. Mean state-anxiety following the offence (M=63.04; SD=13.810) in both groups was significantly higher than before the offence and significantly higher than the average in the Hungarian population (Male sample: M=38.47; SD=10.66; Female sample: M=42.64;

SD=10.79). There were no significant differences in the anxiety scores between the prisoner and the NGRI groups.

In the prisoner group, the following coping mechanisms showed a decline following the crime: problem-centric reaction, social support seeking, pressure control, distraction and emotion focus. Anxiety and self-punishment, as mechanisms to cope with the situation, on the other hand, increased after the offence in the prisoner group.

In the NGRI group, problem-centric reaction, social support seeking, pressure control and emotional focus declined, while anxiety and self-punishment increased after the homicidal act (see TABLE 4).

among homicide perpetrators in the general context is Pressure control (M=46.056; SD=6.106) (see Table 2). Distraction (M=33.371; SD=6.989) and Problem centric reaction (M=32.528; SD=5.262) are also preferred. Deference (M=11.458; SD=3.293) and Self-punishment (M=11.739; SD=3.009) are the least preferred coping mechanisms. Emotion focus (M=27.371; SD=5.598), Social support seeking (M=19.096; SD=5.442) and Emotion discharge (M=14.653; SD=3.311) are in the middle range. These results suggest that, in the general context, perpetrators of homicide try to stabilize their personality and seek distraction from focusing on the threat to the self and pursue less threatening activities. They may also exit the situation and/or procrastinate action in the first place while try to change the situation and solve the problem because they are unable to defer and accept disturbing situations. In the general context they do not tend to blame themselves for the problems.

After the homicidal act, Pressure control (M=39.642; SD=7.627), Distraction (M=30.597; SD=7.171) and Problem centric reaction (M=27.686; SD=7.072) are the mechanisms most commonly used by perpetrators. Deference (M=11.714; SD=3.418), Self-punishment (M=13.903; SD=4.159) and Emotion discharge (M=14.197; SD=3.786) are the least preferred coping mechanisms in the post-offence phase. Emotion focus (M=24.559; SD=5.703) and Social support seeking (M=15.254; SD=5.736) are in the middle range. These results suggest that the overall pattern of preferred coping mechanisms does not change after the homicidal act, perpetrators apply their habitual means in coping with the heightened level of anxiety.

Mean scores of offence-related PTSD among homicide offenders (M=37.063; SD=11.158) were between „medium” and „medium-severe” related symptoms. 7 individuals (11.1%) fell in the „severe” category, 19 (29.7%) in the „medium-severe” category, 23 (40.6%) in the „medium” category, 15 (23.6%) in the „mild” category (Foa, 1995). These results suggest

that homicide offenders do experience traumatization symptoms related to their homicidal act, moreover, four perpetrators out of ten experience severe or medium-severe symptoms. Offence-related guilt ( $M=14.729$ ;  $SD=4.488$ ) was found to be significantly higher than offence-related shame ( $M=11.174$ ;  $SD=4.608$ ).

#### Comparing post-offensive reactions and coping mechanisms of convicted and NGRI samples

Independent sample t-tests were conducted to compare scores of post-offence reactions and coping mechanisms of the prisoner and the NGRI subjects for the following scales: STAI Post-offensive State, CPQ scales, crime related PTSD Scale to compare level of homicide-related post-traumatic symptoms in convicted and NGRI perpetrators, for scales of ORSGQ to compare level of post-offensive guilt and shame. There were no significant differences found on any of the scales between the scores of convicted and NGRI perpetrators. More detailed t-tests conducted on each item, however, showed significant differences between the two groups. Participants of the NGRI group were, eg, more likely to agree that, following the crime they felt more relaxed ( $t=-1.322$ ,  $p=0.001$ , NGRI mean=1.90, NGRI SD=1.261, prisoner mean=1.57, prisoner SD=0.838), rested ( $t=-2.562$ ,  $p=0.000$ , NGRI mean=1.76, NGRI SD=1.136, prisoner mean=1.24, prisoner SD=0.612), joyful ( $t=0.968$ ,  $p<0.05$ , NGRI mean=1.23, NGRI SD=0.685, prisoner mean=1.11, prisoner SD=0.320), pressure-free ( $t=-0.965$ ,  $p<0.05$ , NGRI mean=1.41, NGRI SD=0.959, prisoner mean=1.24, prisoner SD=0.547), having a good time ( $t=-1.289$ ,  $p<0.05$ , NGRI mean=1.43, NGRI SD=0.870, prisoner mean=1.21, prisoner SD=0.567), etc than did participants from the prisoner group.

#### Correlation between symptoms of traumatising, offence-related shame (ORS) offence-related guilt (ORG) and post-offence anxiety

In the full sample, symptoms of offence-related traumatising (OR PTSD) were correlated with post-offence anxiety ( $r=0.615$ ;  $p<0.05$ ), and shame ( $r=0.585$ ;  $p<0.05$ ). We found correlations between offence-related shame and guilt ( $r=0.544$ ;  $p<0.05$ ), offence-related shame and post-offence anxiety ( $r=0.535$ ;  $p<0.05$ ) and offence-related guilt and post-offence anxiety ( $r=0.483$ ;  $p<0.05$ ).

In the prisoner sample, OR PTSD was strongly correlated with offence-related guilt ( $r=0.681$ ;  $p<0.01$ ), offence-related shame ( $r=0.678$ ;  $p<0.01$ ) and post-offence anxiety ( $r=0.656$ ;  $p<0.01$ ). We found a correlation between offence-related shame and offence-related guilt ( $r=0.777$ ;

$p < 0.01$ ), offence-related shame and post-offence anxiety ( $r = 0.526$ ;  $p < 0.01$ ), offence-related guilt and post-offence anxiety ( $r = 0.728$ ;  $p < 0.01$ ).

In the NGRI sample, OR PTSD was correlated with post-offence anxiety ( $r = 0.616$ ;  $p < 0.05$ ) and with offence-related shame ( $r = 0.585$ ;  $p < 0.05$ ). There was a correlation between offence-related shame and offence-related guilt ( $r = 0.544$ ;  $p < 0.05$ ), offence-related shame and post-offence anxiety ( $r = 0.535$ ;  $p < 0.05$ ), and offence-related guilt and post-offence anxiety ( $r = 0.483$ ;  $p < 0.05$ ). For detailed results, see TABLE 5.

## **Discussion**

The anxiety-scores of homicide perpetrators are within the average range of the Hungarian population. However, the anxiety experienced after homicide showed a significant increase. There was no difference in the levels of post-offence anxiety for convicted and NGRI perpetrators. This confirms that homicide is a very stressful and anxiety-provoking event for most perpetrators.

Parallel to increased anxiety, most perpetrators apply different coping mechanisms in the post-offence period than before. Problem-centric, rational methods, the ability to escape the situation diminish and procrastination becomes more frequent. Perpetrators withdraw socially, experience a higher level of self-blaming thoughts and feelings that they find difficult to control.

Contrary to our hypothesis, we found no significant differences in the post-offence emotional reactions and coping mechanisms between convicts and NGRI participants on any scale. An item-level comparison, however, indicates emotional responses in the NGRI group that seem inadequate following the homicidal act (relaxed, calm, joyful feelings). While there may be several explanations to this, the presence of acute psychotic symptoms seems plausible. Deficiencies in reality functioning, disorganised behaviour and emotions may appear. As a result of these, the perpetrator acts against his own interest. Frequent post-offence reactions in the prisoner group, on the other hand (worries, making plans, struggle) reflect a more realistic appraisal of the situation, aimed at self-defence.

Confirming previous results, we found homicide to be traumatic for a high percentage of offenders. Some 40% of our full sample reported medium-severe or severe traumatisation.

Offence-related guilt, shame, post-offence anxiety are interrelated. Traumatisation after the homicidal act is particularly strongly associated with the level of anxiety and shame.

Our results raise the demand to explore the moral emotions of violent offenders more in depth. In the case of perpetrators with SMI, it is essential to consider their difficult emotional situation and address issues of rehabilitation and the relapse-prevention.

### **Study 3: Symptom profiles and parental bonding in two groups of schizophrenia patients**

#### **Introduction**

Characteristics of attachment and bonding between the parent and the child are essential issues in all psychiatric disorders. These constructs are, however, for a number of reasons, difficult to assess.

A widely accepted tool to measure parental bonding is the Parental Bonding Instrument, the PBI (Parker, Tupling és Brown 1979, Hungarian version: H-PBI, Tóth and Gervai 1999).

The PBI is a self-administered questionnaire that assesses maternal and paternal behaviours and attitudes as experienced by the child in the first 16 years of life on a four-point Likert-scale. It consists of the scales Care vs Rejection and Overprotection vs Encouragement of Autonomy/Independence. The H-PBI has been shown to be a reliable (test-retest Pearson coefficients between 0.88 and 0.93 on the three scales for both parents) and valid tool to assess subjects' perception of parental bonding.

Earlier studies have found lower Care and frequently higher Overprotection-scores in patients diagnosed with schizophrenia compared to non-clinical subjects. Patients with severe (predominantly borderline) personality disorder have given similar PBI-profiles (Gomes et al 2015, Willinger et al 2002). The characteristics of parental bonding in schizophrenia patients with a history of homicide have, however, to our knowledge, not yet been explored.

In our study, we assessed the intensity of psychotic symptoms as well as the components of Care and Overprotection in parental bonding in a sample of male schizophrenia patients with the history of committed or attempted homicide and in a sample of matched male schizophrenia patients with no history of violent behaviour. Care and Overprotection were also assessed in a third group of matched male control subjects with no history of psychiatric disorders or violent behaviour.

We hypothesized that homicidal behaviour in the sample of schizophrenia patients is an indication of more severe psychopathology. We therefore expected more intense positive psychotic symptoms in homicidal schizophrenia patients than in non-violent patients. Our second hypothesis was that the more severe psychopathology in homicidal patients is correlated with lower perceived parental Care and higher Overprotection as compared to non-

violent schizophrenia patients and non-violent healthy controls. This pattern indicates a more negatively perceived parental bonding in the subsample of homicidal schizophrenia patients.

## **Study**

### **Subjects and method**

The first group of schizophrenia subjects (SCH-HOM, n=22) consisted of male individuals diagnosed with schizophrenia with a history of committed or attempted homicide. They were subjected to involuntary psychiatric treatment at the Institute of Forensic Psychiatry (Igazságügyi Megfigyelő és Elmegyógyító Intézet, IMEI) in Budapest.

The second group (SCH-nonHOM, n=19) consisted of male subjects diagnosed with schizophrenia matched to the first group by sex, age and education, with no history of violent behaviour.

The third group (NORM, n=20) consisted of healthy, non-violent male control individuals matched to the first two groups by sex, age and education. (For descriptive data of our subjects, see TABLE 6, for relevant clinical characteristics, see TABLE 7, for data on antipsychotic medication, see TABLE 8).

We used the Positive and Negative Syndrome Scale (PANSS, Kay, Fiszbein and Opler 1987) to measure the severity of positive, negative and general psychopathology symptoms of schizophrenia.

For the assessment of parental bonding, we used the Hungarian version (Szülői Bánásmód Kérdőív, H-PBI, Tóth and Gervai 1999) of the Parental Bonding Instrument in all three groups.

We used Kruskal-Wallis ANOVA between the three groups and the Tukey HSD-test for the post hoc comparisons of two groups where differences were significant. Statistical analyses were carried out using STATISTICA Version 10.0.

### **Results**

We found significant differences on all three subscales of the PANSS between the homicidal and the non-violent patient groups, including the Positive Scale ( $p=0.000143$ , mean SCH-HOM=32.44, SD=6.33, mean SCH-nonHOM=23.04, SD=7.36), the Negative Scale ( $p=0.000435$ , mean SCH-HOM=32.87, SD=4.51, mean SCH-nonHOM=25.75, SD=6.89), and

the General Psychopathology Scale ( $p=0.002575$ , mean SCH-HOM=66.37, SD=9.11, mean SCH-nonHOM=55.07 SD=13.39). Confirming our hypotheses, members of the violent group scored significantly higher on all three subscales than members of the non-violent group. We found the largest difference between the scores of the two groups on the items Delusions ( $p=0.000132$ ), Suspiciousness/persecution ( $p=0.00097$ ) and Hostility ( $p=0.00071$ ) (see GRAPH 1 and 2, TABLE 9).

In the case of paternal bonding, we found no significant differences on any scale or item between the members of the three groups.

As to the patterns of maternal bonding, we found significant differences between the members of the three groups on the scales Care ( $p=0.001077$ ) and Overprotection ( $p=0.030936$ , as well as on items 1 (*Spoke to me in a warm and friendly voice*,  $p=0.0041$ ), 4 (*Seemed emotionally cold to me*,  $p=0.0009$ ), 5 (*Appeared to understand my problems and worries*,  $p=0.0034$ ), 6 (*Was affectionate to me*,  $p=0.0063$ ), 8 (*Did not want me to grow up*,  $p=0.016$ ), 9 (*Tried to control everything I did*,  $p=0.0042$ ), 11 (*Enjoyed talking things over with me*,  $p=0.00083$ ), 12 (*Frequently smiled at me*,  $p=0.0015$ ), 14 (*Did not seem to understand what I needed or wanted*,  $p=0.0013$ ), 16 (*Made me feel I wasn't wanted*,  $p=0.00013$ ), 17 (*Could make me feel better when I was upset*,  $p=0.042$ ), 18 (*Did not talk with me very much*,  $p=0.0046$ ), 19 (*Tried to make me feel dependent on her/him*,  $p=0.00095$ ), 20 (*Felt I could not look after myself unless she/he was around*,  $p=0.021$ ), 24 (*Did not praise me*,  $p=0.00012$ ). On all of the above mentioned items members of the non-violent schizophrenia group gave significantly lower Care- (items 1, 2, 4, 5, 6, 11, 12, 14, 16, 17, 18 and 24) and significantly higher Overprotection-scores (items 8, 9, 19 and 20) than members of the other two groups. There were no significant differences between the scores of homicidal schizophrenia patients and normal controls on any of the above listed items.

Non-violent schizophrenia patients (the SCH-nonHOM group) scored significantly lower on the maternal Care scale (mean=19.53, SD=10.55) than homicidal schizophrenia patients (mean=28.45, SD=5.05), and normal controls (mean=28.05, SD=3.83). There was no significant difference between the scores of homicidal schizophrenia patients and normal controls. Similarly, on the scale of maternal Overprotection, non-violent schizophrenia patients scored significantly higher (mean=10.89, SD=6.39), than homicidal schizophrenia patients (mean=7.14, SD=4.28) and normal controls (mean=7.15, SD=4.20). We found no

significant differences between the scores of homicidal schizophrenia patients and normal controls (see GRAPH 3, TABLE 10).

## **Discussion**

Our results support the hypothesis that schizophrenia patients who had committed or attempted homicide suffer from a more severe condition involving more intense psychotic symptoms.

Considering that severe positive, negative and general psychopathology symptoms are connected to committing or attempting homicide, it is important to register such symptoms as possible markers of later violent behaviour.

Possible differences in paternal bonding among the members of the three groups – in case there are any – do not seem relevant to later homicidal behaviour or the severity of psychotic symptoms.

The pattern of maternal bonding perceived by our subjects contradicts our hypothesis. Our findings raise the possibility that, unlike violent offenders without the diagnosis of SMI, in the case of psychotic perpetrators, negatively perceived parental bonding does not play a significant role. Biological, neuro-developmental factors may be more important in this subgroup, as indicated by our own previous study (Tényi et al 2015). As I have discussed more in detail in the first study of this thesis, the greater number and special topological profile of minor physical anomalies point at a more seriously aberrant development of early neurodevelopment in the case of homicidal schizophrenia patients as compared to non-violent patients.

**TABLE 1 – Occurrence of MPAs in the different body regions among the three groups**

<b>Homicidal schizophrenia group</b>	<b>Non-homicidal schizophrenia group</b>	<b>Fisher's exact-test results (2-sided)</b>	<b>Odds ratios with 95% confidence interval</b>
MPAs in the head region	MPAs in the head region	p<0,001	OR:13,650 CI(3,821, 48,768)
MPAs in the mouth region	MPAs in the mouth region	p<0,001	OR:11,083 CI(3,282 37,657)
<b>Homicidal schizophrenia group</b>	<b>Normal controls</b>	<b>Fisher's exact-test results (2-sided)</b>	<b>Odds ratios with 95% confidence interval</b>
MPAs in the ear region	MPAs in the ear region	p=0,001	OR:18,261 CI(2,250, 148,181)
MPAs in the head region	MPAs in the head region	p=0,001	OR:33,150 CI(7,940, 138,922)
MPAs in the eye region	MPAs in the eye region	p=0,025	OR: 9,333 CI(1,136 76,690)
MPAs in the leg region	MPAs in the leg region	p=0,001	OR:15,200 CI(1,871 123,517)
<b>Non-homicidal schizophrenia group</b>	<b>Normal controls</b>	<b>Fisher's exact-test results (2-sided)</b>	<b>Odds ratios with 95% confidence interval</b>

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MPAs in the ear region	MPAs in the ear region	p=0,009	OR:13,846
			CI(1,612 122,584)

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**TABLE 2 – Comparison between individual MPAs in the three groups**

<b>Minor malformations</b>	<b>Homicidal schizophrenia group,number of MPAs</b>	<b>Non-homicidal schizophrenia group,number of MPAs</b>	<b>Statistical significance (p-values of Fisher's exact test,two-sided)</b>
Earlobe crease	0	4	p=0,010
Flat occiput	26	5	p=0,008
Double posterior hair whorl	16	0	p=0,001
<b>Phenogenetic variants</b>	<b>Homicidal schizophrenia group,number of MPAs</b>	<b>Non-homicidal schizophrenia group,number of MPAs</b>	<b>Statistical significance (p-values of Fisher's exact test,two-sided)</b>
High arched palate	18	1	p=0,002
Abnormal philtrum	18	1	p=0,002
<b>Minor malformations</b>	<b>Homicidal schizophrenia group,number of MPAs</b>	<b>Normal controls,number of MPAs</b>	<b>Statistical significance (p-values of Fisher's exact test,two-sided)</b>
Flat occiput	26	0	p<0,001
Double posterior hair whorl	16	0	p=0,001

Furrowed tongue	18	0	p<0,001
Sydney line	10	0	p=0,024
<b>Phenogenetic variants</b>	<b>Homicidal schizophernia group,number of MPAs</b>	<b>Normal controls,number of MPAs</b>	<b>Statistical significance (p-values of Fisher's exact test,two-sided)</b>
Abnormal philtrum	18	0	p<0,001
High arched palate	18	0	p<0,001
Clinodactyly	12	0	p=0,006
Wide distance between 1 and 2 toes	15	1	p=0,013
<b>Minor malformations</b>	<b>Non-homicidal schizophrenia group,number of MPAs</b>	<b>Normal controls,number of MPAs</b>	<b>Statistical significance (p-values of Fisher's exact test,two-sided)</b>
Furrowed tongue	6	0	p=0,021
Sydney line	9	0	p=0,001

**TABLE 3 – Descriptive data of subjects**

	<b>Convicts</b>	<b>NGRI patients (involuntary treatment)</b>
Participants	N = 57	N = 24
Age at the time of homicide	t = 28.43 (SD 9.84)	t = 33.46 (SD 10.62)
Time elapsed since homicide (months) **	t = 106.3 (SD 57.79)	t = 77.54 (SD 49.72)
Length of time incarcerated	t = 103.89 (SD 60.41)	t = 79.5 (SD 47.95)
Attempted homicide	5	10
Homicide	52	14
<b>Education</b>		
Less than primary school	5	0
Primary school	25	7
Profession without high- school qualification	15	3
Secondary school	7	9
University, college	1	4
<b>Marital status</b>		
Single	21	15
Non-marital relationship	0	1
Married, common-law marriage	31	2
Divorced	3	6
<b>Residence</b>		
Capital	13	5
County centre	11	3
Town	14	8
Village	16	6
Farmstead	1	0

**TABLE 4 – Data of scales used in our study**

	N	Minimum	Maximum	Mean	SD	Cronbach $\alpha$
STAI TRAIT	77	39.00	57.00	45.078	2.928	0.366
PO STAI State	68	24.00	80.00	63.044	13.180	0.600
PO CPQ Problem Centric Reaction	70	13.00	41.00	27.686	7.072	0.834
PO CPQ Social Support Seeking	71	7.00	32.00	15.254	5.736	0.846
PO CPQ Pressure Control	67	20.00	57.00	39.642	7.627	0.721
PO CPQ Distraction	67	17.00	50.00	30.597	7.171	0.784
PO CPQ Emotion Focus	68	12.00	40.00	24.559	5.703	0.713
PO CPQ Emotion Discharge	71	8.00	24.00	14.197	3.786	0.581
PO CPQ Self-Punishment	72	5.00	20.00	13.903	4.159	0.834
PO CPQ Deference	70	5.00	20.00	11.714	3.418	0.623
OR PTSD	64	18.00	62.00	37.063	11.158	0.888
OR Guilt	70	5.00	20.00	14.729	4.488	0.803
OR Shame	69	5.00	20.00	11.174	4.608	0.845

**TABLE 5 – Results of correlation coefficients, PTSD, offence-related shame (ORS) and guilt (ORG), post-offence anxiety (PO STAI State)**

	PO PTSD	PO STAI State	ORG
PO PTSD			
PO STAI State	<b>,656**</b>		
	<b>,616*</b>		
ORG	<b>,681**</b>	<b>,728**</b>	
	<b>,366</b>	<b>,483*</b>	
ORS	<b>,678**</b>	<b>,526**</b>	<b>,777**</b>
	<b>,585*</b>	<b>,535*</b>	<b>,544*</b>

\*\* . Correlation is significant at the 0.01 level (2-tailed).

\* . Correlation is significant at the 0.05 level (2-tailed).

b. Cannot be computed because at least one of the variables is constant.

Convicts sample; **NGRI Sample**

**TABLE 6 – Mean age, duration of illness and duration of hospitalization in the homicidal and the non-violent schizophrenia group**

	SCH-HOM mean (SD)	SCH-nonHOM mean (SD)
Age	37.60 (4.62)	39.40 (3.88)
Duration of illness (month)	36.51 (7.67)	42.42 (6.51) ***
Duration of hospitalization (month)	3.1 (2.73)	4.3 (3.45) ***

Number of psychotic episodes prior to hospitalization (mean)	1.3 (3.34)	1.1 (2.46)

\*\*\*=p<0.05

**TABLE 7 – Clinical characteristics of the homicidal and the non-violent schizophrenia group**

	SCH-HOM mean (SD)	SCH-nonHOM mean (SD)
Age of onset of schizophrenia (years)	22.5 (3.84)	24.7 (4.02)
History of comorbid substance abuse (%)	45.45 (3.61)	40.00 (2.59)
Family history of diagnosed psychiatric disorder (%)	59.09 (4.67)	55.00 (2.85)
History of childhood abuse (%)	13.63 (6.88)	10.00 (5.27)

\*\*\*=p<0.05

**TABLE 8 – Antipsychotic medication received by the homicidal and the non-violent schizophrenia group during their current treatment**

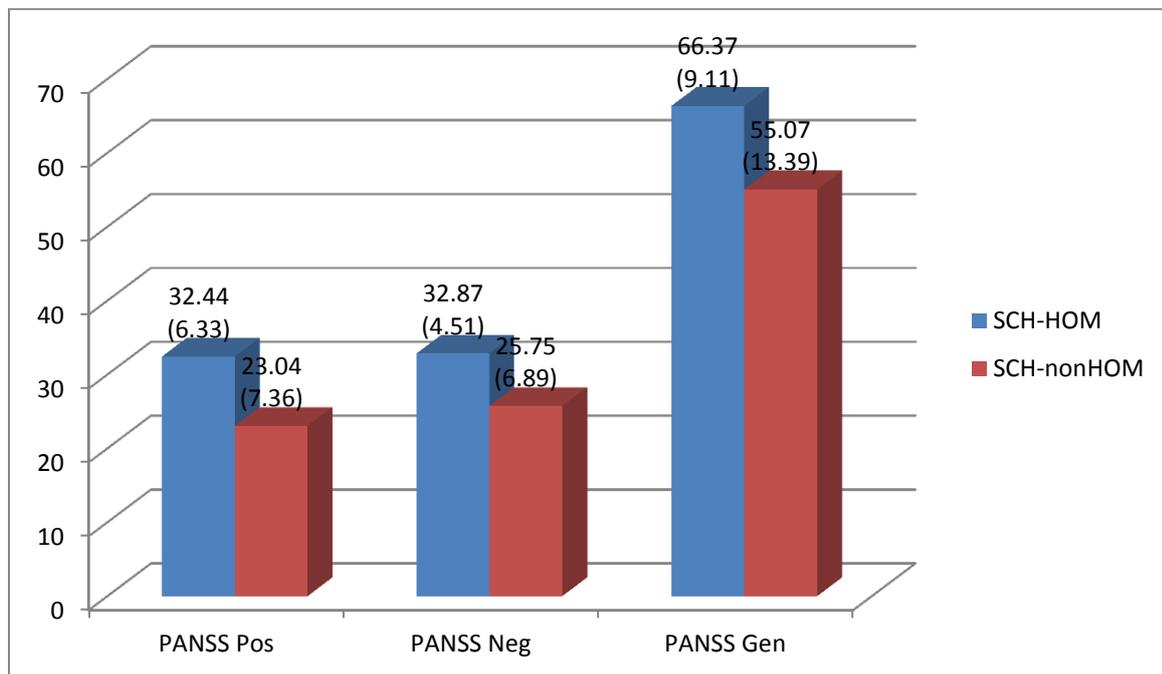
	SCH-HOM n=22	SCH-nonHOM n=19	Total n=41
Monotherapy Typical	15 (68.18%)	3 (15.78%)	18 (43.90%)
Monotherapy Atypical	0 (0%)	11 (57.89%)	11 (26.83%)
Polytherapy	7 (31.81%)	6 (31.58%)	(34.15%)

**TABLE 9 – Symptom profiles, symptom severity in the homicidal and the non-violent schizophrenia group**

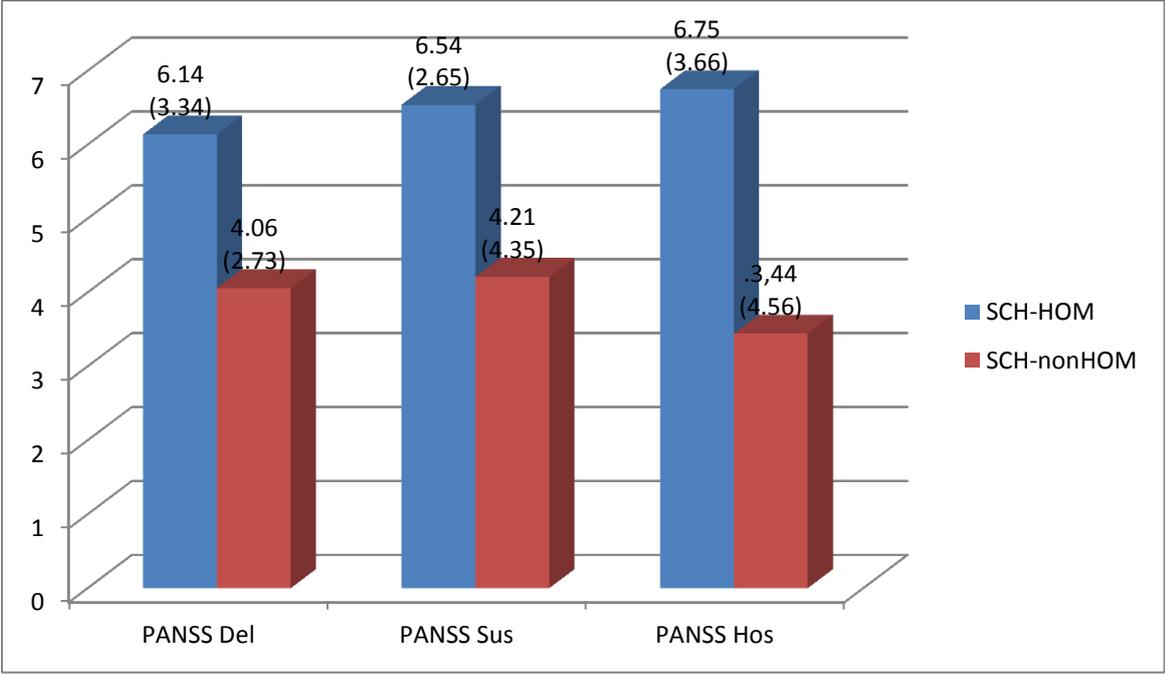
	SCH-HOM n=22 mean (SD)	SCH- nonHOM n=19 mean (SD)	F	dF	p

PANSS Positive Scale	32.44 (6.33)	23.04 (7.36)	17.68	40	0.00014
PANSS Negative Scale	32.87 (4.51)	25.75 (6.89)	14.71	40	0.00044
PANSS General Psychopathology Scale	66.37 (9.11)	55.07 (13.39)	10.34	40	0.00257
PANSS Delusions	6.14 (3.34)	4.06 (2.73)	9.67	40	0.00013
PANSS Suspicion/persecution	6.54 (2.65)	4.21 (4.35)	11.65	40	0.00097
PANSS Hostility	6.75 (3.66)	3.44 (4.56)	10.67	40	0.00071

**GRAPH 1 – Symptom profiles, symptom severity in the homicidal and the non-violent schizophrenia group – PANSS Scales**



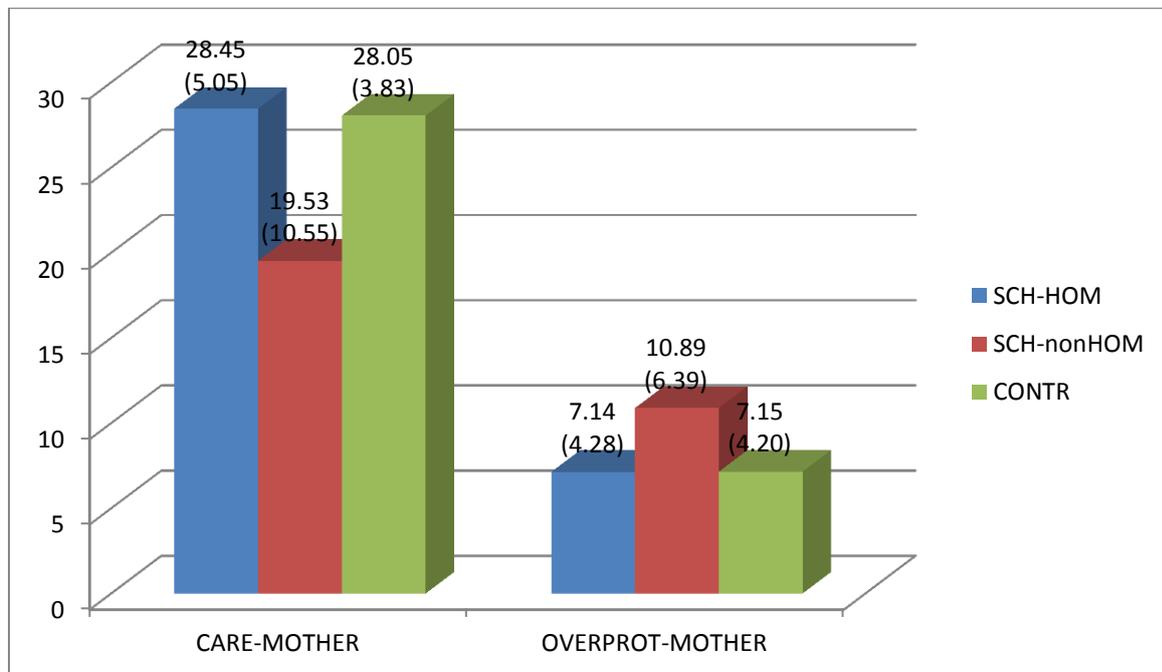
**GRAPH 2 – Symptom profiles, symptom severity in the homicidal and the non-violent schizophrenia group – PANSS Items**



**TABLE 10 – Care and Overprotection scores in the homicidal vs non-violent schizophrenia vs normal control group**

	SCH-HOM (n=22) mean (SD)	Sch-nonHOM (n=19) mean (SD)	NORM (n=20) mean (SD)	F	dF	p
Care_Mother	28.45 (5.05)	19.53 (10.55)	28.05 (3.83)	14.71	59	0.001077
Overprot_Mother	7.14 (4.28)	10.89 (6.39)	7.15 (4.20)	5.01	59	0.030936

**GRAPH 3 – Care and Overprotection scores in the homicidal vs non-violent schizophrenia vs healthy control group**



### **Summary of new observations**

- We found a higher prevalence of minor physical anomalies in homicidal schizophrenia patients than non-violent patients and healthy control subjects, especially in the head and mouth region – this points to a more significant neurodevelopmental component in this subgroup of schizophrenia patients.
- Most homicide offenders experience intense anxiety and traumatisation following the crime. Perpetrators with a severe mental illness are more likely to give inadequate emotional reactions and irrational behaviour in the post-offence period than perpetrators with no psychiatric diagnosis.
- Feelings of shame, guilt and anxiety in the post-offence period are interrelated. Post-offence traumatisation is especially strongly connected to post-offence anxiety and shame in perpetrators with a severe mental illness as well as in those without a psychiatric diagnosis.
- We found more severe positive, negative psychotic and general psychopathology symptoms in homicide offenders with the diagnosis of schizophrenia than those patients who have not committed a violent crime. Thoughts of persecution and suspicion as well as hostility seem especially severe in this subgroup.
- We found more Care and less Overprotection and, hence, a more favourable pattern of maternal bonding than patients without a history of violence. We found no difference in paternal bonding patterns between the members of the two groups.

## **Publications**

### **Publications related to the thesis**

**Halmi T**, Tényi T, Gonda X. (2017). Symptom profiles and parental bonding in homicidal versus non-violent male schizophrenia patients. *Ideggyógyászati Szemle*, 70: 43-52. **IF (2015): 0,376.**

Lehoczki Á, **Halmi T**. (2016). Homicide Offenders With or Without Psychotic Disorder: Post-Traumatic Symptoms, Guilt and Shame, and Coping in the Post-Offence Period. *Annals of Forensic Research and Analysis*, 3: 1031-6.

Tényi T, **Halmi T**, Antal A, Benke B, Jeges S, Tényi D, Tóth LÁ, Csábi Gy. (2015). Minor physical anomalies are more common in schizophrenia patients with the history of homicide. *Psychiatry Research*, 225: 702-5. **IF (2015): 2,466.**

Uzonyi A, Deli É, Gyollai Á, **Halmi T**, Lehoczki Á, Lukács-Miszler K, Magyar E, Magyar J. (2015). „Kényszergyógykezelők” – Terápiás lehetőségek egy tekintélyelvű szervezetben. *Pszichoterápia*, 24: 114-21.

**Halmi T**, Tényi T. (2014). Pszichózis és erőszak – szakirodalmi áttekintés preventív nézőpontból. *Psychiatria Hungarica*, 29: 35-47.

**Halmi T**, Tényi T. (2013). Psychosis as a process – new implications of staging models of schizophrenia. *Ideggyógyászati Szemle*, 66: 383-90. **IF (2013): 0,343**

**Halmi T**. (2011). Pszichózis, emberölés – és a tabu. *Pszichoterápia*, 20:161-7.

**Impact factor: 3,185**

### **Abstracts related to the thesis**

Tényi T, Hajnal A, **Halmi T**, Simon M, Varga E, Herold R, Csábi Gy. (2015). Endophenotypic Markers in the Relatives of Schizophrenia Patients: Systematic Reviews of Theory of Mind and Informative Morphogenetic Variant Studies. *European Psychiatry*, 30, Suppl.1, Article 0909. **IF: 3,21.**

**Halmai T**, Csábi Gy, Jeges S, Antal A, Benke B, Tényi T. (2012). Szkizofrén pszichózisok és erőszakos viselkedés – biológiai és pszichoszociális markerek. *Neuropsychopharmacologia Hungarica* 14 Suppl 1, 22-23.

### **Further publications**

Berecz H, Csábi Gy, Jeges S, Herold R, Simon M, **Halmai T**, Trixler D, Hajnal A, Tóth ÁL, Tényi T. (2017). Minor physical anomalies in bipolar I and bipolar II disorders – Results with the Méhes Scale. *Psychiatry Research*, 249: 120-4. **IF (2015):2,466.**

**Halmai T**, Tényi T. (2017). Személyiségzavarok – igazságügyi pszichiátriai vonatkozások. In: Tényi T. (szerk). *Személyiségzavarok*. Budapest: Medicina, 267-295.o.

Hajnal A, Csábi Gy, Herold R, Jeges S, **Halmai T**, Trixler D, Simon M, Tóth ÁL, Tényi T. (2016). Minor physical anomalies are more common among the first-degree unaffected relatives of schizophrenia patients – Results with the Méhes Scale. *Psychiatry Research*, 237, 224-228.**IF (2015): 2,466.**

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**Impact factor with abstracts: 11,760**

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