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Attention, memory and verbal learning and their relation to schizotypal traits in unaffected parents of schizophrenic patients

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ABSTRACT. The main objective of this *ex post facto* study is to compare the differences in cognitive functions and their relation to schizotypal personality traits between a group of unaffected parents of schizophrenic patients and a control group. A total of 52 unaffected biological parents of schizophrenic patients and 52 unaffected parents of unaffected subjects were assessed in measures of attention (Continuous Performance Test- Identical Pairs Version, CPT-IP), memory and verbal learning (California Verbal Learning Test, CVLT) as well as schizotypal personality traits (Oxford-Liverpool Inventory of Feelings and Experiences, O-LIFE). The parents of the patients with schizophrenia differ from the parents of the control group in omission errors on the Continuous Performance Test- Identical Pairs, on a measure of recall and on two contrast measures of the California Verbal Learning Test. The associations between neuropsychological variables and schizotpyal traits are of a low magnitude. There is no defined pattern of the relationship between cognitive measures and schizotypal traits.

KEYWORDS. Schizotypy. Schizophrenia. Attention deficit. Memory. Relatives of schizophrenic patients. *Ex post facto* study.

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RESUMEN. El objetivo principal de este estudio *ex post facto* es comparar las diferencias de las alteraciones cognitivas y su relación con los rasgos de la personalidad esquizotípica entre un grupo de padres no afectados de pacientes esquizofrénicos y un grupo control. Se han evaluado 52 padres biológicos no afectados de pacientes esquizofrénicos y 52 padres no afectados de sujetos no afectados en medidas de atención (*Continuous Performance Test- Identical Pairs Version, CPT-IP*), memoria y aprendizaje verbal (*California Verbal Learning Test, CVLT*) así como en rasgos de personalidad esquizotípica (*Oxford-Liverpool Inventory of Feelings and Experiences, O-LIFE*). Los padres de los pacientes con esquizofrenia se diferencian de los padres del grupo control en los errores de omisión del *Continuous Performance Test- Identical Pairs*, en una medida de recuerdo y en dos índices de contraste del *California Verbal Learning Test*. No existe un patrón definido de la relación entre las medidas cognitivas y los rasgos de esquizotipia.

PALABRAS CLAVE. Esquizotipia. Esquizofrenia. Déficit atencional. Memoria. Familiares de pacientes esquizofrénicos. Estudio *ex post facto*.

Despite the efforts, the aetiology of schizophrenia remains unknown. A specific cause has not yet been found and it is usually accepted that it has a multifactorial aetiology with a complex interaction between genetic and environmental factors. The genetic bases of schizophrenia have been broadly confirmed through studies of family (Gottesman and Erlenmeyer-Kimling, 2001; Kety *et al.,* 1994; Tsuang and Vanderley, 1986), of twins (Franzek and Beckman, 1998; Kallman, 1994; Kringlen, 1991), of adoption (Cornblatt and Keilp, 1994; Erlenmeyer-Kimling *et al.,* 1995; Kety, Rosenthal, Wender, and Schulsinger, 1968) and high risk studies (Erlenmeyer-Kimling *et al.,* 2000; Mednick, Parnas, and Schulsinger, 1987; Wolf and Cornblatt, 1996). The high risk studies are strongly linked to the neurodevelopmental theory (Murray, 1994; Weinberger, 1987). In order to know which aetiological and pathophysiological mechanisms play a role in schizophrenia, an important part of the research has been focused on the detection of vulnerability markers in high risk subjects. This vulnerability, known as 'schizotypy' (Claridge, 1994, 1997; Gruzelier, 1995; Meehl, 1962, 1989; Rado, 1953; Venables, 1995), can be identified in unaffected subjects with a genetic disposition towards the development of schizophrenia.

Another area which has experienced important growth in recent years concerns the neuropsychological aspects of schizophrenia. The aim of research in this area has been to study the relationship between cerebral functioning and behaviour. These studies have provided for, first of all, the creation of models of neural dysfunction based on schizophrenia (Andreasen, Paradiso, and O'Leary, 1998; Weinberger, 1987); secondly, the interpretation of schizophrenic symptomatology as an underlying defect in specific aspects of cognitive functioning (Frith, 1987; Hemsley, 1987); and finally, the attempt to integrate cognitive and neuronal aspects, *i.e.* the cognitive processes related to the signs and symptoms of schizophrenia and the relationship between these specific cognitive processes and distinct cerebral systems (Frith, 1995).

Still today, neither the role played by neuropsychological deficits in the aetiology of the disorder, nor the extent to which these may be genetic markers or indicators of

schizophrenia are very well known (Wolf and Cornblatt, 1996). The identification of neuropsychological indicators of risk for schizophrenia is an important step in linking these two areas of study. In order to qualify study variables as risk indicators they should: a) be present and relatively stable in patients with schizophrenia, b) be less common in patients with other psychiatric disorders, and c) be present, to a lesser extent, in those subjects at risk for schizophrenia (Kremen *et al.,* 1994).

The need to identify bio behavioural indicators of the schizophrenic genotype is widely recognized by researchers (Cornblatt and Keefe, 1991; Erlenmeyer-Kimling, and Cornblatt, 1992). Experimental studies have found that schizophrenic subjects, compared to normal subjects, perform more poorly in tasks that require alertness, rapid responses or sustained attention (Nuechterlein and Dawson, 1984). It has also been demonstrated that this attentional dysfunction is not a simple secondary effect of an acute state of schizophrenia, nor the result of chronicity, nor of the seriousness of the illness, nor of the length of hospitalisation; attentional deficit is as evident during acute episodes as it is during periods of remission and, consequently, considered to be a trait of the disorder or a marker of vulnerability towards it (Cornblatt and Keilp, 1994; Nuechterlein, 1991).

Attentional capacity measured with the Continuous Performance Test (CPT) (Rosvold, Mirsky, Sarason, Bransome, and Beck, 1956) has been widely established in the literature as a good indicator of genetic vulnerability for schizophrenia. Research in the field shows that altered performance in the CPT at an early age can be a predictor of the disorder. This conclusion is based on three facts: a) the children of schizophrenic parents show global attentional deficit, b) there is a very close relationship between attentional dysfunction in infancy and behavioural disorders in adolescence among subjects at risk of schizophrenia, and c) continual attentional deficit is only characteristic in subjects at risk for the disorder (Cornblatt and Erlenmeyer-Kimling, 1985). The results of successive research (Cornblatt, Lenzenweger, Dworkin, and Erlenmeyer-Kimling, 1992; Erlenmeyer-Kimling *et al.,* 1993; Freedman, Rock, Roberts, Cornblatt, and Erlenmeyer-Kimling, 1998) suggest that attentional problems are vulnerability markers in infants for disorders of the schizophrenia spectrum and that, in individuals with a possible vulnerability towards schizophrenia, chronic attentional deficit seems to be related to a lack of interpersonal sensitivity and to social indifference together with a tendency to avoid others in adulthood.

Other research studies carried out with non-psychotic relatives of schizophrenic patients also show that they have poorer attentional performance than a control group (Franke, Maier, Hardt, Hain, and Cornblatt, 1994; Goldberg *et al.,* 1995; Keefe *et al.,* 1997; Toomey *et al.,* 1998). In conclusion, data demonstrate that the relationship between CPT performance and being a relative of a schizophrenic patient confirms that CPT attentional deficit may be considered a genetic marker of vulnerability for schizophrenia. Along these same lines, results obtained through the psychometric high risk strategy show that attentional deficit is also found in subjects defined as schizotypal on psychometric tests (Chen, Hsiao, and Lin, 1997; Chen *et al.,* 1998; Condray and Steinhauer, 1992; Lenzenwerger and Moldin, 1990, Obiols *et al.,* 1992; Obiols, García-Domingo, De Trinchería, and Doménech, 1993; Obiols, *et al.,* 1997).

Another neurocognitive variable that has been studied in populations at risk for schizophrenia is the memory function. Some studies have found that relatives of schizophrenic patients perform more poorly on recognition tasks (Rutschmann, Cornblatt, and Erlenmeyer-Kimling, 1980), have worse results in short- and long-term memory tasks (Faraone *et al.,* 1995; Golberg *et al.,* 1995; Toomey *et al.,* 1998) and in free recall tasks (Sponheim, Steele and McGuire, 2004), have worse discriminability, and use semantic clusters less as a learning strategy (Lyons *et al.,* 1995). Volgmaier, Seidman, Salisbury, and McCarley (1997) and Bergman *et al.* (1998) found that schizotypal subjects also commit more errors in memory tasks.

The main objective of this *ex post facto* study (Montero and León, 2007) is to identify vulnerability markers in unaffected parents of schizophrenic patients and to determine how they relate to schizotypal traits compared with normal parents of normal subjects. There have been few research studies exclusively of samples of parents of schizophrenic patients (Appels, Sitskoorn, Westers, Lems and Kahn, 2003; Keefe *et al.,* 1994; Grosh, Docherty and Wexler, 1995; Ross *et al.,* 1998; Harris, *et al.,* 1996). One of the advantages of studying subjects who have passed the age of risk for developing schizophrenia is that the deficits identified can be part of a vulnerability towards the disorder and, in this way, confusion with those deficits that are real premorbid indicators of schizophrenia is avoided.

Method

Participants

The subjects taking part in this study were unaffected parents of schizophrenic patients and normal parents of normal subjects (for more information about the selection procedure see Caparrós, Barrantes-Vidal and Obiols, 2001). Each group was made up of 26 pairs of parents, 52 parents of schizophrenics and 52 normal control parents, for a total of 104 subjects. Due to the characteristics of the study in which genetics plays an important role, and with the aim of being able to count on the complete genotype of both parents, all the cases in which it was not possible to assess both parents were rejected. Requirements for inclusion in the group of parents of schizophrenic patients were: a) to have a child (or children) with schizophrenia diagnosed by medical experts; b) the absence of mental illness in each of the parents, that is, neither the father nor the mother can have been diagnosed, treated or hospitalised for any mental pathology, in order that the results are not affected by the presence of mental illness (psychotic or not); and c) having adequate intellectual and literacy levels to be able to correctly understand and respond to the administered tests. The requirements for inclusion in the control group were exactly the same, with the obvious exception of the first, as it was not required to have schizophrenic children or a family history of mental disorders. The age of the parents of the schizophrenic patients varied between 43 and 78 years ($M = 56.5$; $SD = 8.21$) and that of the parents of the normal subjects between 45 and 74 years ($M = 57.63$; $SD = 7.69$), there were no significant difference between the means of the two groups. Both groups of parents participated voluntarily in the study after being informed of its objectives.

Measures

The personal data questionnaire was prepared with the objective of obtaining information about demographic characteristics such as age, sex, education level and profession. The subjects were also asked if they had suffered from any physical or mental illnesses, if they had been treated or hospitalised for any of those disorders, if they used any drugs or if they had any relatives with mental disorders. This information was useful to include or exclude subjects from the study according to whether or not they met the previously mentioned requirements.

In order to evaluate the level of intellectual capacity and in this way create homogenous groups for this variable, Raven's Test of Progressive Matrices (PM-56), General Scale (Raven, 1969) was used.

The multidimensional assessment of schizotypy was carried out using the Oxford-Liverpool Inventory of Feelings and Experiences (O-LIFE by Mason, Claridge, and Jackson, 1995), a questionnaire that assesses 4 factors: *Unusual Experiences*, *Cognitive Disorganisation*, *Introvertive Anhedonia* and *Impulsive Nonconformity*.

The Continuous Performance Test - Identical Pairs Version (CPT-IP by Cornblatt, Risch, Faris, Friedman, and Erlenmeyer-Kimling, 1988) was used to assess sustained attention. We used the computerized digital version (Obiols and Padró, 1991). This programme provides data about omission errors, commission errors, distraction errors, the discrimination index (d), the b index and reaction time.

The California Verbal Learning Test (CVLT by Delis, Kramer, Kaplan, and Ober, 1987) was used to assess memory and verbal learning. The CVLT quantifies the following parameters: levels of total recall and recognition in all the trials, semantic and serial learning strategies, the serial position effect (effect of primacy and recency), the proportion of learning across the trials, the consistency of the recalled items across the trials, the degree of vulnerability to proactive and retroactive interference, short- and long-term information retention, improvement in recall performance by guided categories and in recognition assessment, recognition performance indexes (discrimination and response bias), recall persistence and intrusions, and false positives in recognition.

Procedure

First of all, the schizophrenic patients were identified, having been diagnosed by expert psychiatrists. All the patients met criteria DSM-III-R and DSM-IV and were derived from the mental health services of Girona and Barcelona (Spain). The second step consisted of contacting the parents. All the parents who agreed to collaborate in the study and met the requirements were assessed on the various tasks. The subjects who met the requirements to participate in the control group and collaborated voluntarily in the study were also assessed in the same way.

Unaffected parents of schizophrenic patients were compared with parents of normal subjects on each one of the neuropsychological variables with the *t* test comparison of means with a level of significance of $p<0.05$. We used Pearson's method of significance (χ^2) to analyse the differences between groups in the variables related to studies and intellectual level. Pearson's correlation coefficient (*r*) was used to analyse the relationship between the quantitative variables.

The present document was edited according to the norms established by Ramos-Álvarez, Valdés-Conory, and Catena (2006).

Results

Level of studies and intellectual level

Table 1 contains data relative to the level of studies and the intellectual level of the group of parents of schizophrenic patients (from hereon the index group) and of the group of normal parents of normal subjects (the control group). The majority had a high level of primary studies and their performance in the Raven Test of Progressive Matrices was average. Although some subjects from both the index and control groups obtained a score below the mean score for their age group, the subjects had sufficient capacity to carry out the tasks required in the study because their level of literacy, although low, was adequate.

TABLE 1. Studies and intellectual level of the index and control groups.

	Index group (n = 52)		Control group (n = 52)		χ^2	p
	n	%	*n*	%		
Studies completed					2.56	.278
Primary	29	55.80	33	63.5		
Secondary	15	28.80	16	30.8		
University	8	15.40	3	5.80		
Raven's Progressive Matrices					2.20	.531
Intellectually superior	1	1.90	1	1.90		
Definitely above average	9	17.30	4	7.70		
Average	34	65.40	38	73.10		
Definitely below average	8	15.40	9	17.30		

Attentional deficit

Comparing both groups on the CPT-IP task, it can be seen (Table 2) that the index group does not differ in global performance in the test from the control group (d), although the parents of schizophrenic patients obtain lower scores than the parents of the control group. The only significant difference between the index group and the control group is in the value related with omission errors.

TABLE 2. Comparison of the results of the CPT-IP between
the index and control groups.

	Index group (n = 51) Mean (SD)	Control group (n = 52) Mean (SD)	t
d'	1.20 (1.18)	2.14 (.88)	-.710
β	3.35 (3.57)	2.85 (3.11)	.755
Reaction time	508.33 (107.01)	483.46 (112.92)	1.147
Omission errors	9.29 (6.41)	6.62 (4.07)	2.537*
Commission errors	3.25 (4.06)	4.37 (3.77)	-1.439
Distraction errors	3.16 (3.33)	3.27 (4.80)	-.138

*$p < .05$

Memory and verbal learning

As seen in Table 3, there are significant differences between the index group and the control group in the number of words recalled from the interference list, the index group having worse results.

TABLE 3. Comparison of the CVLT recall measures
between the index and control groups.

	Index group (n = 52) Mean (SD)	Control group (n = 51) Mean (SD)	t
Recall measures			
First list (list A) total trials 1-5	46.62 (10)	47.9 (9.23)	-.306
trial 1	5.96 (2.03)	6.06 (1.50)	-.276
trial 2	8.00 (2.10)	8.78 (2.15)	-1.876
trial 3	10.10 (2.70)	10.12 (2.35)	-.043
trial 4	10.86 (2.54)	10.78 (2.43)	.166
trial 5	11.54 (2.48)	11.33 (2.45)	.422
Total Interference list (List B)	4.88 (1.60)	5.96 (2.37)	-2.70**
List A, short-delay free recall	9.87 (2.66)	9.51 (2.52)	.696
List A, short-delay cued recall	10.75 (2.39)	11.02 (1.76)	-.650
List A, long-delay free recall	10.17 (2.83)	10.37 (2.37)	-.388
List A, long-delay cued recall	11.31 (2.73)	10.92 (2.25)	.782

**$p<.01$

The learning slope regression line does not show any differences between the groups; that is, both groups learn the same number of words across the trials. Likewise, there are no significant differences between the groups regarding either strategies used to evoke the words on the first list in trials 1 to 5 or regarding the position of the recalled words. The means show that both the index group and the control group use the serial more than the semantic cluster. Although there are no significant differences between the groups, the index group tends to recall a higher percentage of words in the recent region than the control group. There are not any significant differences regarding

the results related to recall consistency; both groups have the same percentage of recalled words across the trials. Neither do the results show significant differences between groups regarding recall persistence and intrusion errors made. Differences have not been found between the groups in recognition memory (Table 4).

TABLE 4. Comparison of the CVLT learning across essays, characteristics of learning, recall errors and recognition between the index and control groups.

	Index group (n=52) Mean (SD)	Control group (n=51) Mean (SD)	t[1]
Learning across essays list A trials 1-5			
Learning (slope regression line)	1.40 (.63)	1.29 (.53)	.987
Percent recall consistency across trials 1-5	78.29 (11.29)	78.55 (10.02)	-.127
Characteristic of learning, list a trials 1-5			
Semantic cluster	1.52 (.54)	1.53 (.60)	-.145
Serial cluster	2.55 (1.41)	2.75 (1.50)	-.697
Primacy region (%)	29.56 (4.84)	30.87 (6.06)	-1.212
Middle region (%)	41.64/(8.25)	42.74 (6.58)	-.750
Recent region (%)	28.23/(5.80)	26.35 (5.38)	1.701
Recall errors (lists a and b)			
Perseverations (free and cued recall total)	7.65 (4.55)	8.63 (5.80)	-.949
Intrusions (free recall)	1.12 (1.93)	1.02 (1.62)	.273
Intrusions (cued recall)	.27 (.79)	.25 (.56)	.106
Total intrusions	1.39 (2.51)	1.27 (2.04)	.244
Recognition measures			
Correct target recognitions (hits)	13.96 (1.89)	14.33 (1.70)	-1.048
False positives	.98 (1.70)	1.06 (1.56)	-.236
Discriminability	93.14 (6.56)	93.80 (6.17)	-.531
Response bias	-.12 (.34)	-.09 (.30)	-.525

[1] no significant differences between the groups.

As far as contrast measures are concerned (Table 5), there are appreciable differences between the parents of schizophrenic patients and the parents of the control group. A significant difference can be seen in the number of words recalled from the interference list compared to the number of words recalled from the first list in trial 1. The values show that the parents of schizophrenic patients recall significantly fewer words from the interference list compared with the first trial from List A (proactive interference) than the parents of the control group. Likewise, there is also a significant difference between the groups in terms of the comparison of words recalled in long- and short-delay free recall.

TABLE 5. Comparison of the CVLT contrast measures
between the index and control groups.

	Index group (n=52) Mean (SD)	Control group (n=51) Mean (SD)	t
Contrast measures			
List B compared with list A trial 1	-14.04 (28.19)	.17 (37.25)	-2.186*
Short-delay free recall compared with list A trial 5	-14.13 (15.52)	-15.80 (12.78)	.597
Long-delay free recall compared with short-delay free recall	5.50 (18.74)	-1.41 (11.22)	2.267*
Recognition hits compared with long-delay free recall	49.45 (57.81)	44.16 (32.42)	.572

*$p < 0.05$

Relationship between schizotypal traits and neuropsychological variables

This section presents the most relevant correlations found between vulnerability for schizophrenia (using the O-LIFE schizotypy traits) and the neuropsychological variables in the index and control groups.

In order to carry out this correlation analysis, the level of intelligence variable, assessed with Raven's test, has been controlled. This procedure was followed because in the exploratory analysis a tendency was observed towards a relation between the level of intelligence and some of the personality variables studied in the total sample. We found negative correlations between the level of intelligence and the scale of unusual experiences ($r = -.25$; $p < .05$), cognitive disorganization ($r = -.12$; $p < .05$), introvertive anhedonia ($r = -.17$; $p < .05$) and impulsive nonconformity ($r = -.05$; $p < 0.05$). These results indicate that higher scores in these personality characteristics are associated with scores that are also higher on Raven's Test of Progressive Matrices.

We only show the significant correlation between schizotypy factors and cognitive variables for index group and control group (Table 6).

TABLE 6. Significant correlations between schizotypal traits and
neuropsychological variables in the index and control groups.

Index group
Unusual experiences
CVLT
Trial 1 list A ($r = -.29*$)
Trial 3 ($r = -.30*$)
Semantic cluster ($r = -.26*$)
Total list B ($r = -.30*$)
Cognitive disorganisation
CVLT
Short delay free recall ($r = -.27*$)
Short delay cued recall ($r = -.35**$)
Long delay cued recall ($r = -.32*$)
Slope of learning ($r = -.27*$)

TABLE 6. Significant correlations between schizotypal traits and
neuropsychological variables in the index and control groups. *(Cont.)*

Introvertive anhedonia
            CVLT
                        Total list A (r = -.33**)
                        Trial 1 (r = -.43***)
                        Trial 2 (r = -.33*)
                        Recognition (r = -.29*)
Impulsive nonconformity
            CPT
                        Commission errors (r = .36**)
                        d' (r = -.44**)
            CVLT
                        Trial 1 (r = -.39**)
                        Trial 2 (r = -.27*)
                        Short delay free recall (r = -.31*)

Control group

Unusual Experiences
            CPT
                        Omission errors *(r = -.33**)*
Introvertive anhedonia
            CVLT
                        Short delay cued recall (r = -.36**)
                        Long delay free recall (r = -.27*)

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*p<.05; **p<.01; ***p<.001

Discussion

As has been seen in the results section, there is a tendency in the index group to perform more poorly than the control group on the CPT-IP. This is reflected in the lower score obtained in the global index of task (d'). Even though the difference is not significant, it is important to point it out. These data support the idea defended by other authors (Cornblatt *et al.,* 1992; Cornblatt and Keilp, 1994; Franke *et al.,* 1994) that attentional deficit is a valid phenotypic indicator of the schizophrenic genotype and that it can be detected in clinically unaffected risk populations. Evidently, it is hoped that the visible attentional dysfunctions in unaffected relatives of schizophrenic patients, when compared with those of the control group, are much more subtle than those shown by the schizophrenic patients themselves. Related to this fact, some studies have also found a tendency (although not significant) among the relatives of the patients to perform different attentional tests more poorly than the control groups (Toomey *et al.,* 1998). The response pattern shown by the subjects of our study does not coincide with that found by Keefe *et al.* (1997), in whose study the relatives of schizophrenic patients commit more commission errors than those of the control group and do not show differences between groups with respect to omission errors. These differences could be explained because in the study by Keefe *et al.,* as they themselves recognise, a relatively

simple version of the CPT (version 3-7) is used. It could be that this version is not sensitive enough to assess the subtle attentional dysfunctions that risk populations show as is the case of relatives of patients with schizophrenia. On the other hand, these results do coincide with those found by Obiols *et al.* (1993) showing that the schizotypal subjects commit significantly more omission errors than the control group. The version of the CPT was the same as was used in this work, the version of identical pairs, sensitive enough to evaluate the subtle dysfunctions in populations at risk (Cornblatt *et al.,* 1988).

As for memory and verbal learning, the results are fairly similar in both groups. In the measurements of short- and long-term immediate recall from list A, even though the scores of the index group are a little lower than those of the control group, they do not differ significantly. These data do not corroborate those found in other studies (Faraone *et al.,* 1995; Toomey *et al.,* 1998) which show more dysfunction in long-term immediate recall in relatives of schizophrenics. A significant difference is only found in the number of words recalled from list B. The parents of the index group recall fewer words presented from list A after five continual trials. In addition, the results of the contrast measures indicate that the parents of the index group, compared to the control group, recall significantly fewer words in list B than in the first trial of list A. This means there is proactive interference in the index group, *i.e.* the material learned previously interferes in the recall of new material. In the review of studies of relatives of schizophrenics, there are no data referring to proactive interference. But in the study conducted by Kareken, Moberg and Gur (1996) with schizophrenic patients found that the patients showed less proactive inhibition in comparison with those of the control group, *i.e.* they were incapable of keeping old information from interfering in the memorization of new elements. Therefore, the results of our study would suggest that this dysfunction is also seen in relatives of schizophrenic patients, and that proactive interference is possibly a differentiating marker of subjects with vulnerability.

Apart from the previously mentioned difference in proactive interference, a significant difference is also found between the groups in the contrast measure of long-term free recall versus short-term free recall. Both groups have better long-term (after 20 minutes) than short-term recall, that time has no effect on omission. And the control group has better long-term than short-term recall in comparison with the index group. This finding does not have a clear explanation, but it could be because the subjects are asked, after the short-term free recall, to perform a guided recall and are explicitly offered the categories in which the words are included.

Summarising, we can say that indicators of memory and verbal learning generally do not differentiate the group of unaffected parents of schizophrenic patients from the group of parents of normal subjects. We could only point out that the proactive interference measurement is possibly a good marker of subjects with vulnerability towards the disorder.

As for the relationship between schizotypal traits and neuropsychological variables, bearing in mind that the 'intellectual level' variable has been controlled, we have been able to observe that the associations are of a low magnitude and that the differences between the groups do not follow a clearly determined pattern. Nevertheless, in the

index group there are more associations than in the control group. The largest degree of schizotypy traits is associated with a poorer performance on the memory and verbal learning tasks. The most remarkable data concerning this point refer to the fact that positive and impulsive schizotypy is related, above all, to a poorer global performance in the attentional task and to making more commission errors. The negative factor of schizotypy is related to the lower number of recalled words. In the control group, the associations are found, above all, between the indexes of the mnesic task. Although there are some associations between the variables, it is difficult to see a specific pattern. Therefore, the results suggest that the subtle neuropsychological dysfunctions found in the relatives of the schizophrenic patients are other manifestations of vulnerability not associated to a schizotypal vulnerability demonstrated behaviourally, through the personality traits. These data corroborate, on the other hand, the findings of studies such as those carried out by Condray and Steinhauer (1992), Keefe *et al.* (1994), and Pogue-Geile, Garret, Brunke, and Hall (1991). Following the thesis of Keefe *et al.* (1997), the personality and the neuropsychological patterns are possibly two different phenotypes related with schizophrenia. Consequently, these different phenotypes can be used independently in the identification of vulnerability in relatives of schizophrenic patients. We should also keep in mind, as these authors point out, that the relationship found between the dimensions of schizotypy and the neuropsychological deficits has been found in some studies carried out, above all, on clinical populations, and we should not forget that the subjects who took part in this study are unaffected subjects; furthermore, as we have been able to see, there are not many significant differences between the two groups in the majority of personality indexes.

References

Andreasen, N.C., Paradiso, S., and O'Leary, D.S. (1998). "Cognitive Dysmetria" as an integrative theory of schizophrenia: A dysfunction in cortical-subcortical-cerebellar circuitry? *Schizophrenia Bulletin*, *24,* 203-218.

Appels, M.C.M., Sitskoorn, M.M., Westers, P., Lems, E., and Kahn, R. (2003). Cognitive dysfunction in parents of schizophrenic patients parallel the deficits found in patients. *Schizophrenia Research*, *63*, 285-293.

Bergman, A.J., Harvey, P.D., Lees-Roitman, S., Mohs, R.C., Marder, D., Silverman, J.M., and Siever, L. (1998). Verbal learning and memory in schizotypal personality disorder. *Schizophrenia Bulletin, 24*, 635-641.

Caparrós, B., Barrantes-Vidal, N., and Obiols, J. (2001) Características de personalidad del espectro esquizofrénico y locus de control en padres no afectados de pacientes esquizofrénicos. *Revista de Psicopatología y Psicología Clínica, 6,* 105-119.

Chen, W.J., Hsiao, C.K., and Lin, C.C.H. (1997). Schizotypy in community samples: The three-factor structure and correlation with sustained attention. *Journal of Abnormal Psychology*, *106*, 649-654.

Chen, W.J., Liu, S.K., Chang, C.J., Lien, Y.J., Chang, Y.H., and Hwu H.G. (1998). Sustained attention deficit and schizotypal Personality Features in nonpsychotic relatives of schizophrenic probands. *American Journal of Psychiatry*, *155*, 1214-1220.

Claridge, G. (1994). Single indicator of risk for schizophrenia: Probable fact or likely myth? *Schizophrenia Bulletin*, *20,* 158-167.

Claridge, G. (1997). Theoretical background and issues. In G. Claridge (Ed.), *Schizotypy. Implications for Illness and Health* (pp. 3-18). Oxford: Oxford University Press

Condray, R. and Steinhauer, S. (1992). Schizotypal personality disorder in individuals with and without schizophrenic relatives: Similarities and contrasts in neurocognitive and clinical functioning. *Schizophrenia Research*, *7*, 33-41.

Cornblatt, B.A. and Erlenmeyer-Kimling, L. (1985). Global attentional deviance as a marker of risk for schizophrenia: Specificity and predictive validity. *Journal of Abnormal Psychology*, *94*, 470-486.

Cornblatt, B.A. and Keefe, R.S.E. (1991). The genetics of mental illness. *Social Biology*, *38*, 163-218.

Cornblatt, B.A. and Keilp, J.G. (1994). Impaired attention, genetics and the pathophysiology of schizophrenia. *Schizophrenia Bulletin*, *20*, 31-46.

Cornblatt, B.A., Lenzenweger, M.R., Dworkin, R.H., and Erlenmeyer-Kimling, L. (1992). Childhood attentional dysfunctions predict social deficits in unaffected adults at risk for schizophrenia. *British Journal of Psychiatry*, *161 (Supl. 18)*, 59-64.

Cornblatt, B. A., Risch, N. J., Faris, G., Friedman, C., and Erlenmeyer-Kimling, L. (1988). The Continuous Performance Test, Identical Pairs version (CPT-IP): I. New findings about sustained attention in normal families. *Psychiatry Research, 26,* 223-238.

Delis, D.C., Kramer, J.H., Kaplan, E., and Ober, B.A. (1987). *California Verbal Learning Test. Manual.* New York: Psychological Corporation.

Erlenmeyer-Kimling, L. and Cornblatt, B.A. (1992). A summary of attentional findings in the New York High-Risk Project. *Journal of Psychiatry Research*, *26*, 405-426.

Erlenmeyer-Kimling, L., Cornblatt, B.A., Rock, D., Roberts, S., Bell, M., and West, A. (1993). The New York High-Risk Project: Anhedonia, attentional deviance and psychopathology. *Schizophrenia Bulletin*, *19*, 141-153.

Erlenmeyer-Kimling, L., Rock, D., Roberts, S.A., Janal, M., Kestenbaum, C., Cornblatt, B., Adamo, U.H., and Gottesmann, I.I. (2000). Attention, memory and motor skills as childhood predictors of schizophrenia-related psychoses: The New York High-Risk Project. *American Journal of Psychiatry, 157*, 1416-1422.

Erlenmeyer-Kimling, L., Squires-Wheeler, E., Adamo, U.H., Bassett, A.S., Cornblatt, B.A., Kestenbaum, C.J., Rock, D., Roberts, S.A., and Gottesman, I.I. (1995). The New York High-Risk Project: Psychoses and cluster A personality disorders in offspring of schizophrenic parents at 23 years of follow-up. *Archives of General Psychiatry*, *52*, 857-865.

Faraone, S.V., Seidman, L.J., Kremen, W.S., Pepple, J.R., Lyons, J., and Tsuang, M.T. (1995). Neuropsychological functioning among the nonpsychotic relatives of schizophrenic patients: A diagnostic efficiency analysis. *Journal of Abnormal Psychology, 104*, 286-304.

Franke, P., Maier, W., Hardt, J., Hain, C., and Cornblatt, B.A. (1994). Attentional abilities and measures of schizotypy: Their variation and covariation in schizophrenic patients, their siblings and normal control subjects. *Psychiatry Research*, *54*, 259-272.

Franzek, E. and Beckman, H. (1998). Different genetic background of schizophrenia spectrum psychoses: A twin study. *American Journal of Psychiatry, 155*, 76-83.

Freedman, L.R., Rock, D., Roberts, S.A., Cornblatt, B.A., and Erlenmeyer-Kimling, L. (1998). The New York High-Risk Project: Attention, anhedonia and social outcome. *Schizophrenia Research, 30,* 1-9.

Frith, C.D. (1987). The positive and negative symptoms of schizophrenia reflect impairments in the perception and initiation of action. *Psychological Medecine*, *17*, 631-648.

Frith, C.D. (1995). *La esquizofrenia. Un enfoque neuropsicológico cognitivo*. Barcelona: Ariel.

Goldberg, T.E., Torrey, E.F., Gold, J.M., Bigelow, L.B., Ragland, R.D., Taylor, E., and Weinberger, D.R. (1995). Genetic risk of neuropsychological impairment in schizophrenia: A study of

monozygotic twins discordant and concordant for the disorder. *Schizophrenia Research*, *17*, 77-84.

Gottesman, I.I. and Erlenmeyer-Kimling, L. (2001). Family and twin strategies as a head start in defining prodromes and endophenotypes for hypothetical early-interventions in schizophrenia. *Schizophrenia Research, 51*, 93-102.

Grosh, E.S., Docherty, N.M., and Wexler, B.E. (1995). Abnormal laterality in schizophrenics and their parents. *Schizophrenia Research, 14,* 155-160.

Gruzelier, J. (1995). Syndromes of schizotypy: Patterns of cognitive asymmetry, arousal and gender. In A. Raine, T. Lencz, and S. A. Mednick (Eds.), *Schizotypal Personality* (pp. 329-351). Cambridge: University Press.

Harris, J.G., Adler, L.E., Young, D.A., Cullum, C.M., Rilling, L.M., Cicerello, A., Intemann, P. M., and Freedman, R. (1996). Neuropsychological dysfunction in parents of schizophrenics. *Schizophrenia Research, 20*, 253-260.

Hemsley, D.R. (1987). An experimental psychological model for schizophrenia. In H. Hafner, W. Gattaz, and W. Janzaric (Eds.), *Search for the causes of schizophrenia* (pp. 179-188). Heidelberg: Springer-Verlag.

Kallmann, F.J. (1994). The genetic theory of schizophrenia. An analysis of 691 schizophrenic twin index families. *American Journal of Psychiatry, 151,* 189-198.

Kareken, D.A., Moberg, P.J., and Gur, R.C. (1996). Proactive inhibition and semantic organization: Relationship with verbal memory in patients with schizophrenia. *Journal of International Neuropsychological Society, 2,* 486-493.

Keefe, R.S.E., Silverman, J.M., Lees Roitman, S.E., Harvey, P.D., Duncam, M.A., Alroy, D., Siever, L.J., Davis, K.L., and Mohs, R.C. (1994). Performance of nonpsychotic relatives of schizophrenic patients on cognitive tests. *Psychiatry Research*, *53*, 1-12.

Keefe, R.S.E., Silverman, J.M., Mohs, R.C., Siever, L.J., Harvey, P.D., Friedman, L., Lees Roitman, S.E., DuPre, R.L., Smith, C.J., Schmeidler, J., and Davis, K.L. (1997). Eye tracking, attention, and schizotypal symptoms in nonpsychotic relatives of patients with schizophrenia. *Archives of General Psychiatry*, *54*, 169-176.

Kety, S.S., Rosenthal, D., Wender, P.H., and Schulsinger, F. (1968). The types and prevalence of mental illness in the biological and adoptive families of adopted schizophrenics. In D. Rosenthal and S.S. Kety (Eds.), *The Transmission of Schizophrenia* (pp. 345-362). Elmsford, NY: Pergamon Press.

Kety, S.S., Wender, P.H., Jacobsen, B., Ingraham, L.J., Janson, L., Faber, B., and Kinney, D.K. (1994). Mental illness in the biological and adoptive relatives of schizophrenic adoptees. Replication of the Copenhagen Study in the rest of Denmark. *Archives of General Psychiatry*, *51*, 442-455.

Kremen, W.S., Seidman, L.J., Pepple, J.R., Lyons, M.J., Tsuang, M.T., and Faraone, S.V. (1994). Neuropsychological risk indicators for schizophrenia: A review of family studies. *Schizophrenia Bulletin*, *20*, 103-119.

Kringlen, E. (1991). Adoption studies in functional psychosis. *European Archives of Psychiatry and Clinical Neurosciences*, *240*, 307-313.

Lenzenweger, M.F. and Moldin, S.O. (1990). Discerning the latent structure of hypothetical psychosis proneness through admixture analysis. *Psychiatry Research*, *33*, 247-257.

Lyons, M.J., Toomey, R., Seidman, L.J., Kremen, W.S., Faraone, S.V., and Tsuang, M.T. (1995). Verbal learning and memory in relatives of schizophrenic: Preliminary findings. *Biological Psychiatry*, *37*, 750-753.

Mason, O., Claridge, G., and Jackson, M. (1995). New scales for the assessment of schizotypy. *Personality and Individual Differences*, *1,* 7-13.

Mednick, S.A., Parnas, J., and Schulsinger, F. (1987). The Copenhagen high-risk project, 962-1986. *Schizophrenia Bulletin*, *13*, 485-495.

Meehl, P.E. (1962). Schizotaxia, schizotypy, schizophrenia. *American Psychology*, *17*, 827-838.

Meehl, P.E. (1989). Schizotaxia revisited. *Archives of General Psychiatry*, *46*, 935-944.

Montero, I. and León, O. (2007). A guide for naming research studies in Psychology. *International Journal of Clinical and Health Psychology, 7*, 847-862.

Murray, R.M. (1994). Neurodevelopmental schizophrenia: The rediscovery of dementia praecox. *British Journal of Psychiatry*, *165*, 6-12.

Nuechterlein, K.H. (1991). Vigilance in schizophrenia and related disorder. In S. Steinhauer (Ed.), *Handbook of schizophrenia: Neuropsychology, Psychophisiology and Information Processing 5* (pp. 397-433). Amsterdam: Elsevier Science Publishers.

Nuechterlein, K.H. and Dawson, M.C. (1984). Information processing and attentional functioning in the developmental course of schizophrenic disorders. *Schizophrenia Bulletin*, *10*, 160-203.

Obiols, J.E., Clos, M., Corberó, E., García-Domingo, M., De Trinchería, I., and Doménech, E. (1992). Sustained attention deficit in young schizophrenic and schizoptypic men. *Psychological Reports*, *71,* 1131-1136.

Obiols, J.E., García-Domingo, M., De Trinchería, I., and Doménech, E. (1993). Psychometric schizotypy and sustained attention in young males. *Personality and Individual Differences*, *14*, 381-384.

Obiols, J.E. and Padró, F. (1991). Aspectos generales y técnicos de un instrumento –el CPT-IP- para la evaluación de la atención sostenida en psicopatología. *Clínica y Salud*, *2*, 207-212.

Obiols, J.E., Serrano, F., Barrantes, N., García-Marimón, M., Gras, E., Bosch, S., Caparrós, B., and Carandell, F. (1997). Frontal dysfunction and psychosis proneness in CPT-linked vulnerable adolescents. *Personality and Individual Differences, 23*, 677-683.

Pogue-Geile, M.F., Garret, A.H., Brunke, J.J., and Hall, J.K. (1991). Neuropsychological impairments are increased in siblings of schizophrenic patients. *Schizophrenia Research, 4,* 390.

Rado, S. (1953). Dynamics and classification of disordered behavior. *American Journal of Psychiatry*, *110*, 406-416.

Ramos-Álvarez, M.M., Valdés-Conory, B., and Catena, A. (2006). Criteria of the peer-review process for publication of experimental and quasi-experimental research in Psychology. *International Journal of Clinical and Health Psychology, 6,* 773-783.

Raven, J.C. (1969). *PM56-Matrices Progresivas. Escala General.* Madrid: Mepsa.

Ross, R.G., Harris, J.G., Olincy, A., Randant, A., Adler, L.E., and Freedman, R. (1998). Familial transmission of two independent saccadic abnormalities in schizophrenia. *Schizophrenia Research*, *30*, 59-70.

Rosvold, H.E., Mirsky, A., Sarason, I., Bransome, E.D., and Beck, L.H. (1956). A continuous performance test of brain damage. *Journal of Consulting and Clinical Psychology*, *20,* 343-350.

Rutschmann, J., Cornblatt, B., and Erlenmeyer-Kimling, L. (1980). Auditory recognition memory in adolescent at risk for schizophrenia: Report on a verbal continuous recognition task. *Psychiatry Research*, *3*, 151-161.

Sponheim, S.R., Steele, V.R., and McGuire, K.A. (2004). Verbal memory process in schizophrenia patirnts and biological relatives of schizophrenia patirnts: Intact implicit memory, impaired explicit recollection. *Schizophrenia Research*, *71*, 339-348.

Toomey, R., Faraone, S.V., Seidman, L.J., Kremen, W.S., Pepple, J.R., and Tsuang, M.T. (1998). Association of neuropsychological vulnerability markers in relatives of schizophrenic patients. *Schizophrenia Research*, *31*, 89-98.

Tsuang, M.T. and Vanderley, R. (1986). Critical review of recent adoption, twin and family studies of schizophrenia: Behavioral genetics perspectives. *Schizophrenia Bulletin*, *2,* 360-398.

Venables, P.H. (1995). Schizotypal status as a developmental stage in studies of risk for schizophrenia. In A. Raine, T. Lencz, and S.A. Mednick (Eds.), *Schizotypal Personality* (pp. 107-131). Cambridge: University Press.

Weinberger, D.R. (1987). Implications of normal brain development for the pathogenesis of schizophrenia. *Archives of General Psychiatry, 44, 660-669.*

Wolgmaier, M.M., Seidman, L.J., Salisbury, D., and McCarley, R.W. (1997). Neuropsychological dysfunction in schizotypal personality disorder: A profile analysis. *Biological Psychiatry, 41,* 530-540.

Wolf, L.E. and Cornblatt, B.A. (1996). Neuropsychological functioning in children at risk for schizophrenia. In C. Pantelis, H. E. Nelson, and T.R.E. Barnes (Eds.), *Schizophrenia. A neuropsychological perspective* (pp. 161-182). Chichester: Wiley.