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First-degree relatives of suicide completers may have impaired decision-making but functional cognitive control

A. Hoehne [a], S. Richard-Devantoy [a, b], Y. Ding [a], G. Turecki [a], F. Jollant [a, c, *]

[a] McGill University, Department of Psychiatry & Douglas Mental Health University Institute, McGill Group for Suicide Studies, Montréal, Québec, Canada
[b] Laboratoire de Psychologie des Pays de la Loire, UPRES EA 4638, Université d'Angers, Angers, France
[c] Department of Psychiatry, CHU de Nîmes, France

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ABSTRACT

Background: The heritability of suicide is well established. Transmission of risk appears to follow traits more than disorders like depression. In the present project, we aimed at investigating the potential for transmission of cognitive deficits previously observed in suicide attempters, specifically impaired decision-making and cognitive control.

Methods: Seventeen healthy first-degree relatives of suicide completers with no personal history of suicidal act were compared to 18 first-degree relatives of individuals with major depressive disorder but no family history of suicidal act, and 19 healthy controls. Decision-making was assessed with the Iowa Gambling Task, and cognitive control with the Stroop Task, the Hayling Sentence Completion Test, and the Trail-Making Test.

Results: Both suicide and depressed relatives showed lower gambling task net scores than healthy controls. However, there were trends toward lower learning abilities in suicide than depressed relatives (interaction: $p = 0.07$), with more risky choices at the end of the test. Suicide relatives also showed a higher number of self-corrected errors relative to the total number of errors in the Stroop colour test compared to both control groups, with no difference in interference scores. There was no group-difference for any other cognitive tests.

Conclusion: Our findings suggest that decision-making impairment may be found in healthy relatives of suicides and represent a cognitive endophenotype of suicidal behaviour. Normal cognitive control (or self-corrected deficits) may protect relatives against suicidal acts. Impairments in value-based and control processes may, therefore, be part of the suicide vulnerability and represent potential targets of preventative interventions.

1. Introduction

Suicidal behaviour is often modelled as the interaction between proximal stressful events such as job loss or marital conflicts, and vulnerability factors (Mann, 2003). Understanding this vulnerability is important in the hopes of improving preventative interventions. Family, twin and adoption studies have provided robust evidence of a familial transmission of the diathesis for suicidal behaviour with a heritability of suicidal behaviour estimated at 45–50% (Statham et al., 1998; Brent and Mann, 2005). There is also evidence that the transmission of suicide risk co-occurs with the transmission of particular personality traits, more than with categorical diagnoses such as major depressive disorder (Brent and Mann, 2005; Mcgirr et al., 2009). Given the relatively high heritability of suicidal behaviour, there is a growing interest in studying vulnerability factors of suicidal behaviour as endophenotypes i.e. heritable traits that are found both in patients and unaffected family members (Courtet et al., 2011). First-degree relatives of suicides represent a particularly interesting population to investigate toward this end.

Mounting evidence from a growing body of literature points to a number of neurocognitive impairments in suicide attempters that cannot be attributed to comorbid psychopathologies (Jollant et al., 2011). A recent meta-analysis of neuropsychological studies in mood disorders confirmed an association between suicide vulnerability and several cognitive deficits (Richard-Devantoy et al., 2014).

* Corresponding author. Douglas Mental Health University Institute, Frank B. Common Building, 6875 LaSalle Boulevard, Montréal, Québec H4H1R3, Canada.
E-mail address: fabrice.jollant@mcgill.ca (F. Jollant).

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This was notably the case of value-based decision-making (often measured by the Iowa Gambling Task) and cognitive control processes (particularly the interference effect measured by the Stroop test). This led us to a model of neurocognitive vulnerability to suicidal behaviour as being the combination of alterations in value-based and control processes (Jollant et al., 2011). These deficits may be partly heritable, and therefore represent endophenotypes of suicidal acts.

Previous studies have indeed suggested that cognitive skills are under substantial genetic modulation and are heritable. For instance, variants of genes expressed in the prefrontal cortex including COMT, dopamine receptor genes and BDNF, were previously associated with cognitive function (Savitz et al., 2006). Moreover, in a systematic review of literature, Hermo et al. (2014) found that different cognitive domains differed in their heritability in healthy individuals. This heritability and genetic modulation apply to cognitive measures previously linked to suicidal behaviour. For instance, performance in the Stroop test was found to be influenced by genetic variants (Stins et al., 2004). We also found that variants of several genes coding for the serotonergic system modulate decision-making in suicide attempters (Jollant et al., 2007a). Furthermore, a twin study using a behavioural economic design assessed the heritability of risk attitude at 57%, a measure indirectly related to decision-making performance in the Iowa Gambling Task (Zhong et al., 2009). Understanding the heritability of neurocognitive impairments previously found in suicide attempters would shed light on mechanisms of neurocognitive vulnerability to suicidal acts.

To our knowledge, only one study of cognitive function in relatives of suicide completers has been published to date. Mcgirr et al. (2010) reported that first-degree relatives of suicides, in comparison to healthy controls with no family history of mental disorders or suicidal behaviour, exhibited impaired cognitive inhibition but only following a psychosocial stress paradigm. In another article on the Wisconsin Card Sorting Test in the same sample, Mcgirr et al. (2013) found that first-degree relatives of suicides made more perseverative errors and had a lower level of conceptual responses. Evidence from this study, together with previous findings in healthy populations, suggests that neurocognitive impairments including deficits in cognitive inhibition and decision-making may represent cognitive endophenotypes of suicidal behaviour.

In the current paper, we investigated cognitive deficits in first-degree relatives of suicides with no personal history of suicidal acts. We recruited a new and slightly larger sample and, contrary to the previous study, we included a control group of first-degree relatives of individuals with major depressive disorder to distinguish cognitive deficits associated with suicidal behaviour from those associated with the vulnerability to depression. We hypothesized that the same cognitive deficits previously observed in suicide attempters, specifically in decision-making and cognitive control, would be found in first-degree suicide relatives.

2. Methods

2.1. Population

Three groups of participants aged between 18 and 55 years old were recruited through newspaper advertisement:

1) 17 biological first-degree relatives of suicide completers (*suicide relatives*). The suicide completers had suffered from major depressive disorder but not schizophrenia, bipolar disorder or unknown disorders. The relatives (participants) had no personal history of suicide attempt.
2) 18 biological first-degree relatives of individuals with major depressive disorder (*patient relatives*) with no personal and (second-degree) family history of suicidal acts.
3) 19 *healthy controls* with no (second-degree) family history of suicidal behaviour or major mental disorders.

Additional non-inclusion criteria for all participants included, alcohol and substance dependence or abuse within the last 12 months, major comorbid psychiatric disorders such as schizophrenia and bipolar disorder, lifetime history of severe head trauma or central nervous system disorder. All participants were right-handed as checked by the Edinburgh handedness inventory (Oldfield, 1971) and normothymic at time of participation as checked by the Structured Clinical Interview for Axis I DSM-IV (SCID-I).

Participants in the suicide relative group had at least one first-degree biological relative who committed suicide, commonly defined in the literature as an act carried out with some intent to die and having led to death (Mann, 2003). Suicide was assessed by the FIGS following information given by the relative (https://www.nimhgenetics.org/interviews/figs/). Unclear cases (e.g. if there is a doubt about an accident) were not included.

This study was carried out in accordance with the latest version of the Declaration of Helsinki. Written informed consent was obtained from all participants. This study was conducted at Douglas Mental Health University Institute, Montreal, and has been approved by the local ethics committee.

2.2. Clinical assessment

Diagnoses were made with the structured Clinical Interview for Axis I DSM-IV (SCID-I) (First and Spitzer, 2002) and Axis II DSM-IV (SCID-II) (First and Gibbon, 1997). Level of depression was rated with the 21-item Hamilton Rating Scale for Depression (HAMD-21) (Hamilton, 1960) and the Beck Depression Inventory (BDI) (Beck et al., 1961). The anxiety level was assessed with the Spielberger State Trait Inventory (Spielberger, 1983). The Buss-Durkee Hostility Inventory (BDHI) (Buss and Durkee, 1957), the Brown-Goodwin Assessment of Lifetime History of Aggression (BGLHA) (Brown and Goodwin, 1986), and the Barratt's Impulsivity Scale (BIS-11) (Barratt, 1965) were used to assess traits of hostility, aggression and impulsivity, respectively.

2.3. Neuropsychological assessment

The following neuropsychological domains were evaluated: 1) Verbal IQ with the National Adult Reading Test (NART) (Mackinnon et al., 1999); 2) Cognitive inhibition with the Stroop Colour Test (Stroop, 1935), the Trail Making Test (TMT), and the Hayling Sentence Completion Test (Burgess and Shallice, 1997); 3) Verbal fluency with the FAS verbal fluency test (Benton and Hamsher, 1976); 4) Working memory with the Digit Span number part 1 and 2 of the Weschler Adult Intelligence Scale 4th edition (WAIS-IV) (2008); and 5) decision-making with the Iowa Gambling Task (IGT) (Bechara et al., 1999). The order of the tasks was randomized.

In the first part of the *Stroop colour test*, (the “naming” sheet), participants are asked to name the colour of 100 coloured rectangles as fast as they can, without making any mistakes. In the second part (the “lecture” sheet), participants are asked to read words printed in black, all words naming colours. In the third part (the “interference” sheet), subjects are asked to name the colour of the ink of the words written on the page, all words naming colours that do not correspond to the colour of their ink (e.g. the word “green” printed in blue). We calculated an interference time index score corresponding to the time to read interference sheet minus naming