DERMATOGLYPHICS PATTERNS ABNORMALITIES AS PUTATIVE MARKERS OF PSYCHOMETRIC-RISK FOR SCHIZOPHRENIA

Clément Dondé1,2,3, Thierry D’Amato1,2,3,4,5 & Romain Rey1,2,3,4,5

1Inserm U1028, CNRS UMR 5292, VR2 Team, Lyon Neuroscience Research Center, Lyon, France
2University of Lyon, Lyon, France
3University of Lyon 1, Villeurbanne, France
4Centre Hospitalier Le Vinatier, Bron, France
5Fondation FondaMental, Créteil, France

Dear editor,

It is commonly theorized that schizophrenia (SZ) stems from neurodevelopmental disruption arising from interactions between genetic risk factors and environmental insults. This process is expressed across a dynamic continuum of related psychotic manifestations, ranging from high-risk state for psychosis to schizotypy to full-blown clinical SZ. In line with this model, identifying reliable phenotypic markers of attenuated forms of SZ is an important issue as a significant proportion of these patients will go on develop full-blown SZ in a short period (Cannon 2008). Notably, it has been ascertained that patients with SZ display significant dermatoglyphic patterns abnormalities compared to healthy controls in a recent meta-analysis (Golembo-Smith et al. 2012). Dermatoglyphics are epidermal lines of the skin that can be observed on fingers and handprints. Dermatoglyphics abnormalities (DA) are thought to be underpinned by both genetics and environmental prenatal contributors to vulnerability for psychosis (i.e. prenatal maternal infections, stress experiences) that disrupt the embryo’s ectodermal tissue from which develop both the epidermis and brain structures that are impaired in SZ (King 2009). Dermatoglyphics measures show many advantages as they can be performed in a non-invasive, inexpensive and quickly fashion, they are not prone to recall bias and provide continuous data.

However, in order to stand as a reliable marker of risk for SZ, DA should be present at all psychosis continuum stages that are thought to be etiologically related to SZ, such as high-risk state for psychosis and schizotypy. Furthermore, relevant biomarkers for early stages of SZ are still missing. To better demonstrate the value of DA as a marker for these attenuated forms of SZ and see if DA hold along a clinical continuum of psychotic manifestations, we conducted a systematic review of the PubMed database using the following headings: (Dermatoglyphic*) AND (Psychos* OR Schizotyp* OR Prodrom*). The criteria for inclusion were: (i) patients with psychometrically-identified high-risk state for psychosis and schizotypy, (ii) dermatoglyphic measures, (iii) comparison with healthy controls.

The search returned 41 records from which 9 plus 1 from citation lists were reviewed. Extracted data are provided in Table. Table 2. Two main types of dermatoglyphics measures were conducted: counts of various dermatoglyphic features and degree of fluctuating dermatoglyphic asymmetry (the absolute difference of dermatoglyphic features between counts of left and right hand). A higher degree of dermatoglyphic asymmetry was observed in subjects with ultra high risk of SZ (Russak et al. 2016, Mittal et al. 2012). Results provided for patients with schizotypy remain heterogeneous, with only few studies showing overall significant differences in dermatoglyphic features bet samples (Weinstein et al. 1999, Chok et al. 2005, Chok & Kwapil 2005). However, “negative forms” of schizotypy with higher levels of physical and social anhedonia show a strong tendency to exhibit more DA than other clinical presentations of the disease (Barrantes-Vidal et al. 2003, Rosa et al. 2004, Chok et al. 2005), which is consistent with the higher association of negative symptoms with global developmental deviance (van Os et al. 1998). Significant differences between controls and schizotypy may be gender-related, likely related to sex differences seen in psychosis and neurodevelopment (Daly et al. 2008). Interestingly, there was no significant difference between schizotypy and conduct disorder/other personality disorder, which raises the hypothesis that neurodevelopmental disruption is also implicated in such disorders (Weinstein et al. 1999) (Table 1).

Our review helps to delineate DA as a potentially reliable marker of psychometric-risk for SZ, although large samples studies are still required to use such measure as diagnostics. We encourage the measure of dermatoglyphics patterns in predisposed sample to detect at-risk subjects for SZ and help reducing the duration of untreated psychosis, especially in cases with high anhedonic symptoms. Finally, we think DA show relevance to better elucidate etiological processes involved in SZ-spectrum.
Table 1. Summary of studies comparing dermatoglyphic features between predisposed samples for schizophrenia and controls.

<table>
<thead>
<tr>
<th>Study</th>
<th>Stage</th>
<th>Measure of psychotic symptoms</th>
<th>Sample</th>
<th>Dermatoglyphics measures</th>
<th>Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Studies comparing degree of fluctuating dermatoglyphic asymmetry</td>
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</tr>
<tr>
<td>Russak et al. 2016</td>
<td>HR</td>
<td>SCID-P, SIPS</td>
<td>51 P, 45 controls</td>
<td>TFRC</td>
<td>HR &gt; C</td>
</tr>
<tr>
<td>Mittal et al. 2012</td>
<td>HR</td>
<td>PQ-B</td>
<td>18 P, 205 controls</td>
<td>TFRC</td>
<td>HR &gt; C</td>
</tr>
<tr>
<td>Daly et al. 2006</td>
<td>schizotypy</td>
<td>PAS, MIE, SAS, PAHs</td>
<td>166 social anhedonia, SzP, 220 controls</td>
<td>a-b, et al. TFRC</td>
<td>no significant difference between SzP male and C male</td>
</tr>
<tr>
<td>Chok et al. 2005</td>
<td>schizotypy</td>
<td>PAS, MIE</td>
<td>51 SzP, 63 controls</td>
<td>a-b, et al. TFRC</td>
<td>no significant difference between C and SzP</td>
</tr>
<tr>
<td>Rosa et al. 2004</td>
<td>schizotypy</td>
<td>PAS, PAHs, SAS</td>
<td>250 randomly selected students</td>
<td>a-b</td>
<td>FA associated with ‘negative’ SzP</td>
</tr>
<tr>
<td>Weinstein et al. 1999</td>
<td>schizotypy</td>
<td>SCID-P</td>
<td>16 SzP, 10 conduct disorder or other P disorder, 26 controls</td>
<td>TFRC</td>
<td>no significant difference between C and SzP</td>
</tr>
<tr>
<td>Studies comparing counts of dermatoglyphic features</td>
<td></td>
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</tr>
<tr>
<td>Daly et al. 2006</td>
<td>schizotypy</td>
<td>PAS, MIE, SAS, PAHs</td>
<td>166 social anhedonia, SzP, 230 controls</td>
<td>a-b</td>
<td>SzP male &lt; C male, no significant difference between SzP female and C female, no significant difference between SzP total and C total, no significant difference between SzP and C</td>
</tr>
<tr>
<td>Chok &amp; Kwapi et al. 2005</td>
<td>schizotypy</td>
<td>PAS, MIE, SAS, PAHs</td>
<td>197 SzP, 135 controls</td>
<td>ET</td>
<td>SzP &gt; C, ‘negative’ SzP &gt; ‘positive’ SzP</td>
</tr>
<tr>
<td>Langley et al. 2005</td>
<td>HR</td>
<td>PSE</td>
<td>55 P, 26 controls</td>
<td>a-b, ridges number of whorls, radial and ulnar loops, dermatoglyphic complexity (whorls minus arches)</td>
<td>HR &gt; C, p-value not provided</td>
</tr>
<tr>
<td>Chok et al. 2005</td>
<td>schizotypy</td>
<td>PAS, MIE</td>
<td>51 SzP, 63 controls</td>
<td>a-b, et al. TFRC</td>
<td>SzP &lt; C, SzP &gt; C, no significant difference between C and SzP</td>
</tr>
<tr>
<td>Bannantine et al. 2003</td>
<td>schizotypy</td>
<td>PAS, SAS, PAHs</td>
<td>36 negative SzP, 56 high SzP</td>
<td>a-b</td>
<td>‘negative’ SzP &gt; C, no significant differences between C and ‘high’ or ‘positive’ SzP</td>
</tr>
<tr>
<td>Farkhander et al. 1994</td>
<td>schizotypy</td>
<td>PAS, SAS</td>
<td>A pair of 13 y.o. female</td>
<td>TFRC</td>
<td>SzP twin &gt; C twin</td>
</tr>
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<td></td>
<td></td>
<td></td>
<td>MZ twins discordant for SzP</td>
<td></td>
<td>SzP twin &lt; C twin</td>
</tr>
</tbody>
</table>

- Subjects: HR = high-risk state for psychosis, SzP = schizotypy, C = controls.
- Clinical form of schizotypy: ‘high’ SzP = SAS score + PHS score, ‘negative’ SzP = PAHs or SAS score > PASS score,‘social anhedonia’ SzP: SAS score > 12 SD, ‘positive’ SzP = PASS score > SAS or PAHs score.
- Dermatoglyphic measures: a-b = palmar a-b ridge count: number of ridges that cross the line drawn between the middle point of three ridges (called a triradius) at the base of the index (triradius a) and major triradius (b) fingers, a-d = a-d angle: angle formed by lines drawn from triradius a to triradius b, the most distal axial triradius near the base of the palm, TFRC = total finger ridge count: total number of ridge that cross the line drawn between the triradius of the fingers and its corresponding core of the finger pattern, for all 10 fingers, ET = extratrigeminal triradius, the finger tip’s triradius is absent or not visible because it does not fit on the ridge area of the finger tip, rd = ridge dissociation: short broken and disorganized segments of lines that cover the palmar pattern.
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References

5. Daly MP, Gooding DC, Jessen HM, Auger AP: Indicators of developmental deviance in individuals at risk for schizophrenia. Schizophr Res 2008; 101:152–60

Correspondence:
Clément Dondé; MD
INSERM U1028, CNRS UMR 5292, ΨR2 Team,
Lyon Neuroscience Research Center; Centre Hospitalier Le Vinatier
CH Le Vinatier, Batiment 416, 95 boulevard Pinel,
BP 300 39; 69 678 Bron cedex, France
E-mail: clement.donde-coquelet@ch-le-vinatier.fr