Neural and Behavioral Correlates of Empathy for Pain in Tourette Syndrome

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Background & Objective
The defining features of Gilles de la Tourette Syndrome (GTS) are motor and vocal tics. Researched less are diverse echophenomena, coprophenomena and emotional dysregulation, as well as difficulties in social cognition tasks. Echophenomena are “automatic imitative actions without explicit awareness” and are present in 11% to 44% of patients.

Research Hypothesis
Echophenomena and other social symptoms can be explained by increased mirroring of others. We test this using somatosensory mu suppression as a marker for the mirror neuron system (MNS) and test group differences in an empathy for pain paradigm.

Can altered social behaviors, specifically echophenomena, in GTS be explained by an overactive mirror neuron system?

Methods
Participants
- N = 50 (n = 25 GTS, n = 25 HC) adults
- healthy controls (HC) are age-, gender- and education matched
- clinical testing of GTS symptoms, depression, OCD, ADHD and IQ

Empathy for pain paradigm
- 160 trials in 4 blocks, participants gave ratings in 20% of the trials
- paradigm includes two factors: Action (neutral vs. painful stimulus), randomized within each block and Sensitivity (normal vs. enhanced pain sensitivity of the actor), randomized blockwise

we measured ratings, EEG & EDA

Results
Increased level of echophenomena in GTS

Performance in Echometer
- 30 videos of actors performing facial movements
- “Echos” = exact replications of observed movement OR movements in the same body part as observed movement

No behavioral group differences in empathy for pain paradigm

- both groups show an interaction of action x sensitivity for the rating of their own supposed pain (F(1,43) = 197.2, p < .001)
- no significant group differences

Conclusions
- Our results do not support the predicted heightened empathy for pain in GTS. On the contrary, GTS patients showed less pain-related mu suppression compared to controls, while there were no behavioral group differences.
- Our results question the hypothesis of an overactive MNS in GTS and highlight the need for more research into social cognition in patients with GTS.

References

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