Altered perception-action binding modulates inhibitory control in Gilles de la Tourette syndrome

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Abstract

Background: Gilles de la Tourette Syndrome is a multi-faceted neuropsychiatric developmental disorder with onset in childhood or adolescence and frequent remissions in early adulthood. A rather new emerging concept of this syndrome suggests that it is a disorder of purposeful actions, in which sensory processes and their relation to motor responses (actions) play a particularly important role. Thus, this syndrome might be conceived as a condition of altered 'perception-action binding'. In the current study, we test this novel concept in the context of inhibitory control.

Methods: We examined N=35 adolescent Gilles de la Tourette patients and N=39 healthy controls in a Go/Nogo-task manipulating the complexity of sensory information triggering identical actions; i.e. to inhibit a motor response. This was combined with event-related potential recordings, EEG data decomposition and source localization.

Results: Gilles de la Tourette patients showed worse performance compared to controls and larger performance differences when inhibitory control had to be exerted using uni-modal visual compared to bi-modal auditory-visual stimuli. This suggests increased binding between bi-modal stimuli and responses leading to increased costs **of switching between** responses instructed by bi-modal and those instructed by uni-modal stimuli. The neurophysiological data showed that this was related to mechanisms mediating between stimulus evaluation and response selection; i.e. perception-action binding processes in the right inferior parietal cortex (BA40).

Conclusions: **Gilles de la Tourette patients have difficulties to flexibly use different sensory stimuli to trigger behavioral inhibition. It is possible that this reflects altered (stronger) stimulus-action binding and stimulus-action inhibition binding in these patients.**

Introduction

Gilles de la Tourette Syndrome (GTS) is a multi-faceted neuropsychiatric developmental disorder with onset in childhood or adolescence characterized clinically by multiple motor and vocal tics [1]. GTS is associated with a number of structural and functional changes predominantly in fronto-striatal circuits [2,3]. GTS is typically considered a movement disorder, which, however, is not undisputed. It has in fact been suggested that GTS might be viewed as a disorder of purposeful actions, in which sensory processes and their relation to motor responses (actions) play a particularly important role[4]. Thus, GTS might be conceived as a condition of altered 'perception-action binding'[4] **or association**; i.e. that the propensity to form and **maintain perception-action bindings** is higher **in GTS** [4]. One influential theoretical concept addressing binding between perception and action is the theory of event coding (TEC)[5]: **It assumes that whenever a stimulus is encountered and a response is executed, stimulus-action bindings are established and stored in so called 'event files' [6]. Importantly, previously established bindings can strongly affect the execution of subsequent actions that have to be executed on the basis of slightly altered stimulus input. Therefore, the *context* established by previously or intermittently encountered stimulus-response mappings is crucial when assessing a given response. This has important consequences: Whenever a binding between a stimulus and an action is particularly strong, stimuli can effectively trigger actions[6–10]. In cases, however, where the same action (or action inhibition) has to be executed on the basis of different stimulus input, i.e. in a changed context, previously established and still existing stimulus-response associations cause problems[6,8]. This is because such established or 'preferred' stimulus-feature/action-feature bindings are only partially fulfilled[6,8]. Therefore, the strength of previously and repeatedly encountered stimulus-response bindings can affect behavioral performance in situations where these stimuli are not**

evident.

It is well-known that multisensory stimuli **effectively** trigger behavioral responses [11] indicating a strong binding between multimodal **stimuli** and actions. Given the proposed general propensity of GTS patients for stimulus-action binding [4] it is possible that binding on the basis of multisensory stimuli is particularly strong in these patients. **Of note, GTS patients have been shown to use** multi- or bi-modal sensory stimuli for behavioral control very efficiently [12]. **We hypothesize that compared to healthy controls this causes problems in GTS when the same action has to be executed using different stimuli, e.g. uni-modal stimuli. Crucially, 'actions' also encompass inhibition of motor responses [5]. In addition, also bi-modal stimuli affect response inhibition** [13–17]. **Such 'inhibitory control' is of particular interest since GTS has long been considered a disorder characterized by deficient inhibitory control, although inhibitory control dysfunction in GTS is a contentious issue [18–24]. We therefore specifically expect that GTS patients have behavioral costs over and above those in healthy controls when response inhibition is triggered by uni-modal compared to bi-modal stimuli.**

In this context, it is important to consider that multi-modal stimuli can also interfere with the ability to inhibit premature responses if different modalities carry conflicting information [15]. It is therefore unclear whether there are further differential effects between GTS patients and healthy subjects depending on whether bi-modal stimuli carry conflicting or non-conflicting information.

In the current study, we test these hypotheses in a systems neurophysiological approach using high density EEG recording and source localization analysis. We examine adolescent GTS patients and healthy subjects in a Go/Nogo task where we use uni-modal (visual) and bi-modal (audio-visual) stimuli in the Nogo condition and also manipulate the level of conflict in information to be used for response inhibition [15] (**please see method section for further details**). In the EEG, response inhibition processes are reflected by the

Nogo-N2 and Nogo-P3 event-related potential (ERP) component [25]. The Nogo-N2 ERP component is generally assumed to represent pre-motor processes including conflict monitoring and the selection of an action program, whereas the Nogo-P3 component may reflect the inhibitory process itself or an evaluation process [25–27]. Since processes related to stimulus-feature/action-feature bindings also affect neurophysiological processes in the N2 ERP time window [10], it is possible that interactive effects between "group" (GTS vs. healthy controls) and uni-modal/bi-modal information during Nogo trials are specifically reflected by modulations in the N2 time window. It is known that different codes related to 'perceptual processing' and 'response selection' are intermingled in the N2 time window [28] during response inhibition [29]. In particular, response selection codes refer to mechanisms of stimulus-response translation closely related to processes occurring in event files [5,30]. It is therefore possible that differences between GTS patients and healthy controls can only be seen after isolating stimulus-response translation codes within the neurophysiological data. This can be achieved applying residue iteration decomposition (RIDE) [29,31,32] [33,34]: RIDE provides different component clusters; i) the 'C-cluster' **reflecting** stimulus-response translation processes [31,32,35] **and ii)** the S-cluster **referring** to stimulus-related processes including perception and attention [33]. Therefore, it is likely that the C-cluster may better reflect interactive effects between "group" (GTS vs. healthy controls) and uni-modal/bi-modal information during response inhibition (Nogo) trials than standard ERP data. Because stimulus-response translation processes have been shown to be a function of the temporo-parietal junction (TPJ) [36], which has also been shown to underlie modulations in the C-cluster amplitude [32], we hypothesized that activity modulations in this region are associated with the expected group effects in the C-cluster. **At the behavioral level, the main outcome measure is the rate of false alarms, at the neurophysiological level it is the amplitudes of the ERPs or the RIDE cluster components.**

Materials and Methods

GTS patients and healthy controls

A sample of N = 39 GTS patients between 9 to 19 years participated on the study. All patients were recruited from our outpatient clinic (Faculty of Medicine, TU Dresden, Germany) and were diagnosed according to the ICD-10. Tic severity and subjective impairment was assessed using the Yale Global Tic Severity Scale (YGTSS), a clinician rated structured interview. Additional comorbidities were assessed with the M.I.N.I. KID (Mini International Neuropsychiatric Interview for Children and Adolescents). Obsessive compulsive symptoms were assessed with the CY-Bocs (Children's Yale-Brown OC Scale). Due to poor EEG data quality N=4 GTS patients were excluded from the analysis **(the age was unaffected by this)**. The N=35 GTS patients included had a mean age of 12.97 ± 2.52 (29 males) and a mean IQ of 110.58 (±14.8). Out of the N=35 patients, N=3 also had a diagnosis of attention deficit hyperactivity disorder and N=11 a diagnosis of obsessive-compulsive disorder with a mean CY-Bocs Score of 9 (± 4.84). The mean total YGTSS Score (0-100) was 38.97 (± 17.91) and the mean total-TS-Score 18.94 (± 9.62). N=11 of the N=35 patients were on medication during the testing including treatment with Tiapride (N=3), Aripiprazole (N=3), Methylphenidate (N=3) or Fluoxetine (N=2). A sample of N=39 age and gender-matched controls was recruited. The mean IQ in the control group was 112.22 (±14.33). The controls did not have psychiatric disorders as indicated by the M.I.N.I. KID and had also no history of psychiatric disorders. The study was approved by the ethics committees of the Universities of Dresden and Lübeck.

Task

Full details on the task can be found in the supplemental material. **The task setup is shown in Figure 1.**

Insert Figure 1 about here

Briefly, a visual-auditory Go/NoGo task developed by our group [14] was employed. Go trials required participants to press a response key, whenever the word 'Press' (German: 'DRÜCK') was presented on a computer screen. During NoGo trials, the word 'Stop' (German: 'STOPP') was presented on the screen and participants were asked to refrain from responding. Additional auditory stimuli were presented in some Nogo trials: In 72 NoGo trials an auditory NoGo stimulus ('STOPP') was presented, thus facilitating to withhold the response ($NoGo_{compatible}$). In another 72 NoGo trials, a competing auditory Go stimulus (spoken word 'DRÜCK') was presented creating a conflict between the auditory stimulus and the primary visual stimulus and therefore compromising response inhibition performance ($NoGo_{incompatible}$)[14]. The remaining 144 NoGo trials were not accompanied by an auditory stimulus ($NoGo_{without}$). There were 336 Go trials without any additional auditory information and 336 with compatible auditory information (i.e. the spoken word 'DRÜCK' was presented). All participants received the instruction to respond only to visual stimuli and to ignore auditory stimuli. NoGo trials were treated as false alarms (FA), if responses occurred in a 1000ms time window after stimulus presentation.

EEG recordings and analyses

Full details on the EEG analyses methods can be found in the supplemental material.

Briefly, the EEG was recorded from 60 Ag/AgCl electrodes arranged in equidistant positions (500 Hz sampling rate; electrode impedances < 5 kΩ). Data processing involved manual inspection of the data to remove technical artefacts. We used a band-pass filter (0.5 to 20 Hz, 48 db/oct) and a notch filter at 50Hz. Further data processing (i.e. ocular artifact correction, technical artifact rejection, re-referencing using current source density (CSD) transformation, baseline correction) was done as in previous studies using the paradigm [15]. After averaging

the data at the single subject level for each electrode position and experimental condition separately, ERPs were quantified peak to baseline on a single-subject level. The electrodes and time windows used for ERP data quantification were determined by visual inspection and validated using statistical methods. Residue iteration decomposition (RIDE) was performed following established procedures [31,33,35] by using the RIDE toolbox and manual available on http://cns.hkbu.edu.hk/RIDE.htm. Because the R-cluster cannot be reliably be determined for correctly inhibited NOGO trials due to the lack of a motor response [37], only the S-cluster and C-cluster was calculated. The electrodes and time windows used for S-cluster and C-cluster data quantification were determined by visual inspection and validated using statistical methods. The statistical procedures used to analyze the data are outlined in the supplemental material.

The RIDE clusters were used for source localization analyses[38], which were calculated using sLORETA (standardized low resolution brain electromagnetic tomography) [39]. The voxel-based sLORETA images for the different calculated contrasts between groups and conditions were calculated using the sLORETA-built-in voxel-wise randomization tests with 2000 permutations, based on statistical nonparametric mapping (SnPM). Voxels with significant differences ($p < .01$, corrected for multiple comparisons) between contrasted conditions were located in the MNI-brain www.unizh.ch/keyinst/NewLORETA/sLORETA/sLORETA.htm

Results

Behavioral data

Rates of correct responses, misses, and reaction times (RTs) in Go trials only revealed significant main effects "condition" (all $F > 48.72$, all $p < .001$) with higher accuracies (97.61% ± 4.32), fewer misses (2.31% ± 4.22) and faster RTs (488ms ± 90) in the $Go_{without}$ condition than in the $Go_{compatible}$ condition (accuracy: 96% ± 5.52; misses: 3.93% ± 5.45; RTs: 520ms ± 86). All other main or interaction effects were not significant (**all $F < 2.29$; $p > .1$**).

However, the false alarm rate (FAs) is the most important behavioral parameter **(i.e. the main behavioral outcome measure)**. The mixed effects ANOVA revealed a main effect "condition" ($F_{2,144}$ = 132.37, p < .001, η^2 = 0.648) with FA rates increasing from the $NoGo_{compatible}$ (14.12% ± 1.19) to the $NoGo_{without}$ (21.18% ± 1.24) and to the $NoGo_{incompatible}$ condition (28.99% ± 1.54) (p < .001). Most importantly, there was a "condition x group" interaction ($F_{2,144}$ = 6.16, p = .003, η^2 = 0.079), which is shown in Figure 1. Post-hoc independent samples *t*-tests revealed significant differences between both groups in the $NoGo_{without}$ condition (t_{72} > 2.39, p = .02) with higher FA rates in GTS patients (24.15% ± 10.62) than in the Controls (18.22% ± 10.74). The two other conditions did not differ between groups (both p > .285).

Insert **Figure 2** about here

Neurophysiological data

Standard ERPs

The standard ERP are shown in **Figure 3**.

Insert **Figure 3** about here

As hypothesized, there were no interactive effects between "group" (GTS vs. healthy controls) and uni-modal/bi-modal information during Nogo trials (all F < 1.89, p > .10) and during Go trials (all F < 1.89, p > .10). This was the case for the P1 and N1 ERP-component reflecting perceptual gating and attentional selection processes, as well as for the (Nogo)-N2 and (Nogo)-P3 ERP-component. A detailed analysis of the ERP-components can be found in the supplemental material. In line with our hypotheses, reliable effects reflecting the

interaction between "group" (GTS vs. healthy controls) and uni-modal/bi-modal information during Nogo trials were obtained after applying RIDE. These analyses are outlined below.

Residual iteration decomposition (RIDE)

The S-cluster data is shown **in Figure 4.**

Insert **Figure 4** about here

As outlined in the introduction, it is hypothesized that especially the C-cluster and not the S-cluster should reflect interactive effects between "group" (GTS vs. healthy controls) and uni-modal/bi-modal information during Nogo trials. In fact, for the S-cluster no such interaction was present in any of the examined time intervals, i.e. in the P1 and N1 time range as well as in the N2 time range (all $F < 0.66$, all $p > .5$). Detailed analyses of the S-cluster are shown in the supplemental material.

The C-cluster is shown in **Figure 5**. For the Go trials, there was a main effect "condition" ($F_{1,65} = 14.59$, $p < .001$, $\eta^2 = 0.183$) showing higher amplitudes in the $Go_{compatible}$ condition (7.10 $\mu V/m^2 \pm 1.82$) than in the $Go_{without}$ condition (2.57 $\mu V/m^2 \pm 1.89$). No other effects were detected (all $F < 2.60$, $p > .10$).

Insert **Figure 5** about here

For Nogo trials and the Nogo-N2 time window, the mixed effects ANOVA revealed a main effect "condition" ($F_{2,144} = 7.15$, $p = .001$, $\eta^2 = 0.090$). It is shown that the $NoGo_{incompatible}$ condition (6.11 $\mu V/m^2 \pm 2.25$) differed significantly from the $NoGo_{without}$ condition (0.36 $\mu V/m^2 \pm 3.71$) ($p = .003$) and the $NoGo_{compatible}$ condition (0.24 $\pm$ 2.29 $\mu V/m^2$) ($p = .002$). The latter two did not differ from each other ($p > .9$). This shows that a higher

level of conflict (i.e. $NoGo_{incompatible}$ condition) was generally related to larger C-cluster amplitudes. No main effect "group" was shown ($F_{1,72}$ = 0.37, p > .50), but an interaction "condition x group" was found ($F_{2,144}$ = 4.01, p = .021, η^2 = 0.053). **A post-hoc power analysis showed that the achieved power in this interaction is greater than 95%.** Post-hoc independent samples *t*-tests showed that GTS patients and controls differed in the $NoGo_{without}$ condition (t_{72} = 1.21, $p_{one\text{-}tailed}$ < .03). GTS patients showed positive amplitudes (4.41 $\mu V/m^2$ ± 21.65) while amplitudes were negative in the control group (-3.70 $\mu V/m^2$ ± 12.78). The sLORETA analysis shows that these modulations were associated with the right inferior parietal cortex (BA40); i.e. the TPJ. There were no group differences in the $NoGo_{compatible}$ condition (GTS: -0.36 $\mu V/m^2$ ± 22.86; controls: 0.85 $\mu V/m^2$ ± 16.22) (t_{72} < 0.30, p > .70) and in the $NoGo_{incompatible}$ condition (GTS: 6.21 $\mu V/m^2$ ± 21.15; Controls: 6.02 $\mu V/m^2$ ± 17.52) (t_{72} < -.04, p > .90). Further dependent samples *t*-tests showed that within the control group, the $NoGo_{without}$ (-3.70 ± 12.78 $\mu V/m^2$) and the $NoGo_{compatible}$ (0.85 ± 16.22 $\mu V/m^2$) condition revealed smaller C-cluster amplitudes than in the $NoGo_{incompatible}$ (6.02 ± 17.52 $\mu V/m^2$) (all t_{38} < -3.61, p < .001). However, in the GTS group, the pattern was different. There was no difference between the $NoGo_{without}$ (4.41 ± 21.65 $\mu V/m^2$) and the $NoGo_{incompatible}$ (6.21 ± 21.15 $\mu V/m^2$) condition (t < -0.70, p > .4). Only the $NoGo_{compatible}$ condition (-0.36 ± 22.86 $\mu V/m^2$) differed from the $NoGo_{incompatible}$ condition (t_{38} = -2.45, p < .02).

To summarize, the C-cluster data reflected the hypothesized interactive effects between groups and experimental conditions as found at the behavioral level. **In additional analyses, we examined whether the existence of comorbidities or medication status affected the pattern of results observed for the C-cluster. To this end, we included an addition between subject factor comorbidities ("yes or no") as well as "medication" ("yes" or "no") in the ANOVAs. The results were not affected by this as can be seen by non-significant interactions with these factors (all F < 0.57, p > .30) and the fact that the other interaction effects remained unaffected by when adding these factors.**

Discussion

In the current study, we examined how variations in the complexity of sensory information affect response inhibition processes in children and adolescents with GTS. The behavioral data (false alarm rates) show that GTS patients indeed have stronger performance differences between uni-modal and bi-modal stimuli compared to healthy controls. In particular, the rate of false alarms when inhibitory control had to be exerted using uni-modal visual stimuli was higher in GTS patients than healthy controls. In the other conditions, there were no group differences and both groups showed the same modulatory effects between conflicting and non-conflicting auditory information that were described previously [14,15]: i.e., worse response inhibition performance when auditory information was conflicting with the visual information. **Since the uni-modal NoGo condition occurred as often as the multi-modal Nogo conditions (i.e. frequency $NoGo_{without}$ = frequency $NoGo_{incompatible}$ + frequency $NoGo_{compatible}$), the observed effects cannot reflect a simple frequency effect. No modulations were present in Go trials. However, responses in Go trials were (i) highly automated and (ii) did not require a choice-response. This setting makes the execution of Go trials very easy, so that possible modulations of uni-modal/bi-modal stimuli or group differences might not become apparent due to a ceiling effect**.

The neurophysiological data show that specific cognitive-neurophysiological subprocesses are related to these effects. In line with our hypotheses, no interactive effects between group and conditions were detected in standard ERPs, **because these intermingle 'perceptual processing' codes with 'response selection' codes particularly in the N2 time window** [28,29]. Importantly, interactive effect were **not present** in the S-cluster **reflecting stimulus-related processes like perception and attention [33], but in the C-cluster.** The C-cluster has been suggested to reflect stimulus-response translation processes [31,32,35,33,40]. The C-cluster amplitude in the $NoGo_{without}$ condition significantly differed between groups. It was positive in

GTS patients and negative in healthy controls. There was no group difference in the $NoGo_{incompatible}$ and $NoGo_{compatible}$ condition. This is in line with the behavioral (false alarm) data. Importantly, the C-cluster amplitude in the $NoGo_{incompatible}$ condition was also positive and did not differ from the C-cluster amplitude in the $NoGo_{without}$ condition in GTS patients. The finding that the C-cluster amplitude was positive in the $NoGo_{incompatible}$ condition, in which a conflict between visual and auditory information was evident [14,15] and response inhibition performance was also worse in both groups, suggests that a higher C-cluster amplitude reflects difficulties to withhold a response. The higher C-cluster amplitude in the $NoGo_{without}$ condition in GTS patients **therefore very likely reflects difficulties to withhold a response. The sLORETA analysis showed that C-cluster amplitude modulations between groups in the $NoGo_{without}$ condition are associated with the inferior parietal cortex (BA40) and the TPJ. This region has already been shown to be associated with C-cluster amplitude modulations [32] and is involved in the updating of mental representations by means of sensory information to initiate appropriate actions [36] during response inhibition [41]. It therefore seems that updating processes of mental representations that are used during response selection are more difficult in GTS patients in the $NoGo_{without}$ condition. The results therefore show that GTS patients have difficulties to inform response selection mechanisms during inhibitory control particularly when only limited sensory information is available. When there is more sensory information (regardless of whether this information fosters or impedes inhibitory control) performance is similar to healthy controls. The finding that the GTS group shows a closer correspondence between the C-cluster and the behavioral data may suggest that neurophysiological processes in GTS are regulated in more restricted boundaries. In controls variations at the neurophysiological level are larger. This suggests that variation in neurophysiological processes have a stronger effect in GTS than controls.**

On a theoretical level, these results might have the following implications. According to TEC, response selection mechanisms (reflected by the C-cluster) occur in event files [5,30] [10]. **In the TEC framework**, deficits or costs in behavioral performance occur when identical action programs have to be executed on the basis of altered stimulus input [6]. This is particularly the case when the other (no longer evident) bi-modal stimulus input **putatively triggered behavioral responses very efficiently. It has been proposed that GTS might be conceived as a condition of altered 'perception-action binding' [4]; i.e. that the propensity to build strong associations/bindings between stimuli and actions is higher in GTS[4]. Bi-modal stimuli are well-known to establish strong stimulus-response associations** [11] **and the data presented here suggest that this is particularly the case in GTS** [12]. **Importantly, and according to the TEC framework, this implies that it is also more difficult to reconfigure such stimulus-response associations/bindings.** It is possible that GTS patients have difficulties to reconfigure a given event file and to bind uni-modal stimuli to the same action (i.e. the inhibition of a response). **Clearly, further research is needed to examine whether the TEC construct is suitable to explain such findings in GTS.**

To conclude, **it appears that response inhibition processes in GTS are strongly affected by the nature of sensory stimuli that need to be used to trigger this aspect of behavioral control. It seems that GTS patients have difficulties to flexibly use different sensory stimuli to trigger behavioral inhibition. It is possible that this reflects an altered (stronger)** stimulus-action binding and stimulus-action inhibition binding in GTS patients.

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Authors role contributions
1) Research project: A. Conception (V.R., C.B., A.M.), B. Organization (all authors), C. Execution (all authors);
2) Statistical Analysis: A. Design (V.P., C.B.), B. Execution (V.P., C.B.), C. Review and Critique (all authors);
3) Manuscript: A. Writing of the first draft (V.P., C.B., A.M.), B. Review and Critique (all authors)

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Figure Legends

Figure 1

Illustration of the experimental paradigm. Go trials occurred with a 66.6% frequency, Nogo trials with a 33.3% frequency. Upon presentation of the "PRESS" stimulus, a response had to be executed. Upon presentation of the "STOPP" stimulus, the response had to be inhibited. These conditions could either occur without concomitant auditory stimuli, or with an auditory stimulus. When the latter was the case, the auditory stimulus could be compatible or incompatible with the visual stimulus. The experimental setup creates a context in which trials with bi-modal stimuli were more frequent (especially in the demanding Nogo condition) than trials with uni-modal stimuli. Therefore, this context (stimulus-response binding) can interfere with performance in the Nogo$_{\text{without}}$ condition, because then the stimulus conditions partly overlap with those in trials with bi-modal stimuli.

Figure 2

Rate of false alarms in the three Nogo conditions (without auditory stimuli, with compatible auditory stimuli and with incompatible auditory stimuli) in controls (light grey triangles) and

GTS patients (dark grey squares). The mean and SEM is given. ** $p < .02$. The Nogo compatible condition and the incompatible condition did not differ between groups, which can clearly be the by closer or overlapping SEMs.

Figure 3

Event-related potentials on Go (left column) and Nogo trials (right column). The top rows shows the P1 and N1 ERP (pooled across electrode P7 and P8) including their scalp topographies. The bottom row shows the N2 and P3 ERPs at electrode Cz including the scalp topography plot of the N2. In the topography plots, red values denote positive potentials, blue values negative potentials. The different colours of the ERP traces reflect the different conditions in the GTS groups and the control group.

Figure 4

The RIDE S-cluster on Go (left column) and Nogo trials (right column). The top rows shows the S-cluster reflecting the P1 and N1 ERPs (pooled across electrode P7 and P8) including their scalp topographies. The bottom row shows the S-cluster reflecting the N2 ERP at electrode Cz including the scalp topography plot of the N2. In the topography plots, red values denote positive potentials, blue values negative potentials. The different colours of the ERP traces reflect the different conditions in the GTS groups and the control group.

Figure 5

(A) The RIDE C-cluster on Go trials is shown for electrode TP8. (B) The RIDE C-cluster on Go trials is shown for electrode TP8. The different colours of the ERP traces reflect the different conditions in the GTS groups and the control group. For a better comparability, the C-cluster in the N2 time window in the different Nogo conditions are presented separately on the right. The plot showing the interaction for the C-cluster amplitudes in the Nogo-N2 time

between the three Nogo conditions (without auditory stimuli, with compatible auditory stimuli and with incompatible auditory stimuli) in controls (light grey triangles) and GTS patients (dark grey squares) is shown at the bottom. The mean and SEM is given (** $p < .02$). The sLORETA plots show the source of the group difference in C-cluster amplitudes in the $Nogo_{without}$ condition (corrected for multiple comparisons) in BA40. (C) The topography plots for the C-cluster in the N2 time window are given for Go and Nogo trials in all experimental conditions for GTS patients and controls. Red values denote positive potentials, blue values negative potentials. For the Nogo trials the topography plots denoting the difference in C-cluster amplitudes between the groups is given.