Temporal dynamics of subliminal and supraliminal emotional face perception in individuals with autistic traits

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BACKGROUND

The dual route model of face processing[1] & implications for autism

Conscious face processing is important for identity encoding and sensitive to high-spatial frequencies. It relies on **cortical face-processing areas** such as the fusiform gyrus and superior temporal sulcus. Faces can also be perceived with non-conscious processes, automatic modes of functioning outside conscious awareness. Non-conscious face processing involves rapid, automatic operations and is driven by coarse, socially salient features. It is oriented to low-spatial frequencies and mediated by **subcortical regions.** The subcortical route consists of the amygdala, pulvinar and superior colliculus and operates outside sensory awareness. It is crucial in eye-contact detection and navigation of emotional attention. Functional (hypoactivation during fearful face processing)[2] and structural (volumetric differences)[3] disruptions of the subcortical face processing system have been reported in populations with autism, but its temporal dynamics remain poorly understood.

ERP components indexing conscious emotional processing

N2 and **P3** components reflect conscious processes.

- N2 marks changes in detectability of the stimulus; earliest conscious awareness
- P3 reflects higher-level, conscious cognitive processes

ERP components indexing Non-conscious emotional processing

- Recent electrophysiological and magnetoencephalographic findings provide evidence for early latency neural system separable from higher cortical regions[4]
- Early latency responses to faces (**P1, N170**) are related to early stages of face processing and feed into higher cortical brain regions

Non-conscious processes in the general population with autistic traits

- Autistic traits are highly heritable and continuously distributed across the general population[5]
- Variation in autistic traits in neurotypical populations is linked to functioning of brain structures associated with social cognition
- Common genetic variants observed in autism are also documented in typically developing individuals with high level of autistic traits

Aims of the present study

1. To explore temporal dynamics of neural processing of unconscious affect
   - Unconscious emotional processing will be revealed *by shorter latencies of early components (P1, N170)* during subliminally presented fearful expressions
   - Conscious emotional processes will be reflected in *enhanced amplitudes of N2 and P3 components* during supraliminal trials only
2. To capture the neural underpinnings of subtle autistic-like impairments in the neurotypical population with mild autistic characteristics
   - Individuals with high autistic traits (High AQ) will show reduced neural sensitivity reflected in the form of *longer N170 latencies* to subliminal emotion cues when compared to individuals with low level of autistic traits (Low AQ)

METHOD

Participants

- 28 participants (M = 23.18 years, SD = 2.21;17 females; all right-handed)

Behavioral Measures

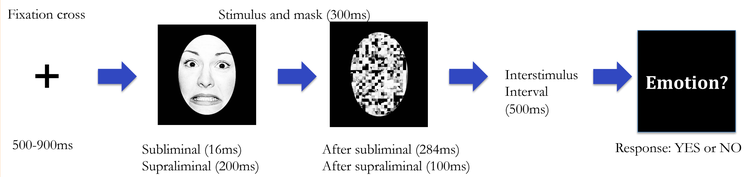
- Autism Quotient
- Empathizing Quotient
- Systemizing Quotient

ERP Data Acquisition & Processing

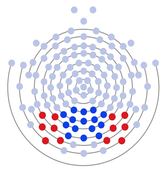
- ERP recorded continuously at 250 Hz using EGI 128 electrode Geodesic Hydrocel Net
- Peak amplitude and latency extracted from averaged data across 4 windows:

  P1: 90-150 ms N170: 140-200 ms
  N2: 300-390 ms P3: 300-390 ms

Design (as per Pegna, Landis, & Khateb, 2008)[6]: 360 trials (6 x 60 for each category; SUBLIMINAL: neutral, fearful, sad; SUPRALIMINAL: neutral, fearful, sad)



Montage



Sensor clusters used to quantify P1, N170, N2 (red) and P3 (blue).

RESULTS

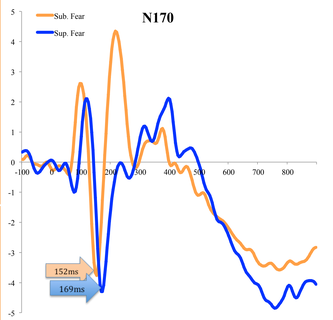


Figure 1: N170 latencies for subliminal and supraliminal fear processing.

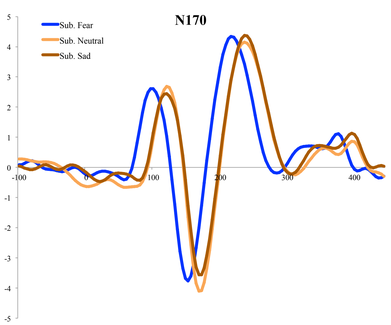


Figure 2: N170 amplitudes for processing of subliminal fear, neutral and sad expressions.

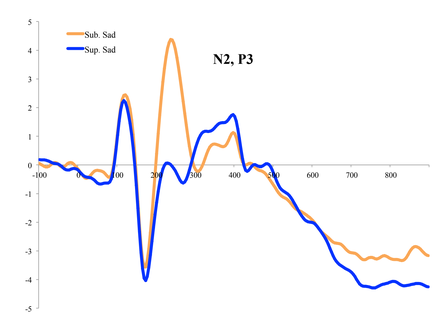


Figure 3: N2 and P3 amplitudes and latencies for subliminal and supraliminal sad expressions.

Temporal dynamics of unconscious emotional face perception

Separate univariate 2x3x2 repeated measures ANOVA with Condition (Sub/Supra), Expression (Fear/Neutral/Sad), Hemisphere (L/R) as within-subject factors and AQ as the between-subject factor were performed for each ERP component. Paired t-tests were conducted to further investigate main effects.

Behavioral results

Groups determined by AQ median split: Low AQ group: M = 11.19, SD = 3.08; High AQ group: M = 21.92, SD = 4.09

P1 and N170 latencies

- Subliminally presented stimuli elicited faster P1 ($F(1, 27) = 24.26$, $p < .01$) and N170 latencies ($F(1, 27) = 71.57$, $p < .01$) than supraliminal stimuli.
- The N170 effect was driven by subliminally presented fearful expressions that were processed significantly faster than other subliminal neutral ($t(27) = 13.92$, $p < .01$), sad faces ($t(27) = 15.74$, $p < .01$) and supraliminal fearful stimuli ($t(27) = -16.22$, $p < .01$).
- This effect was unique to subliminal presentation, as supraliminal fear was not processed significantly faster than sad and neutral faces.
- There was no between-group effect on N170 latencies.

P1 and N170 amplitudes

- P1: main effect of Condition ($F(1, 27) = 6.9$, $p < .01$). Subliminal emotions elicited greater amplitudes than supraliminal emotions.
- P1: Emotion*AQ interaction ($F(1,27) = 3.62$, $p = .034$). Compared to Low AQ group, High AQ showed greater amplitudes to sad than to neutral emotions. ($t(11) = -2.28$, $p = .043$).
- N170: main effect of Emotion ($F(2, 27) = 3.37$, $p = .042$) driven by sad expressions ($t(27) = -2.4$, $p = .023$).
- There was no between-group effect on N170 amplitudes.

N2 and P3 latencies

- N2: Condition*Emotion interaction ($F(1, 27) = 23.8$, $p < .01$). Supraliminal expressions were processed faster than subliminal expressions.

N2 and P3 amplitudes

- N2: Condition*Emotion interaction ($F(1, 27) = 28.53$, $p < .01$). There was a significant negative amplitude enhancement across sad ($t(27) = -5.18$, $p < .01$) and neutral supraliminal trials ($t(27) = -4$, $p < .01$).

RESULTS

N2 and P3 amplitudes and latencies

- N2 Amplitude: Condition*Hemisphere*AQ interaction ($F(1, 27) = 4.4$, $p = .046$). Compared to Low AQ group, High AQ group showed greater amplitudes in the right hemisphere during subliminal presentations. By contrast, Low AQ group showed greater amplitudes in the right hemisphere during supraliminal presentations.
- P3 Amplitude: Greater amplitudes for supraliminal compared to subliminal conditions ($t(27) = -2.73$, $p < .01$; $t(27) = -4$, $p <. 01$, $t(27) = -5.18$, $p < .01$).
- P3 Amplitude : Condition*Hemisphere*AQ interaction ($F(4, 27) = 3.86$, $p = .032$).
- P3 Latency: Condition*Emotion*Hemi*AQ interaction ($F(2, 27) = 7.84$, $p = .001$).

Figure 4

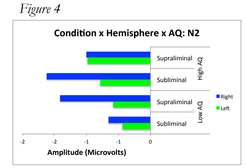
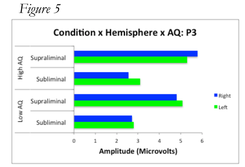


Figure 5



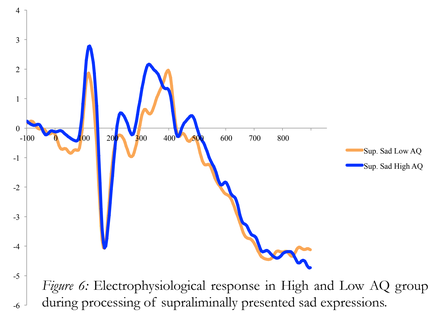


Figure 6: Electrophysiological response in High and Low AQ group during processing of supraliminally presented sad expressions.

CONCLUSION

Temporal dynamics of conscious and unconscious emotional face perception

- This study reveals distinct patterns of neural response for conscious and unconscious emotional face processing. The N170 was modulated by subliminally presented emotional expressions.
- Faster P1 and N170 latencies were evident only during subliminal fear perception, consistent with the existence of a rapid subcortical route. Latency of the N170 reflects unconscious processes.
- Enhanced amplitudes of the N2 and P3 may mark conscious awareness, consistent with previous findings.

Unconscious face perception and autistic traits

- Individuals with high autistic traits did not significantly differ during conscious or unconscious face perception from individuals with low autistic traits.
- However, individuals with high autistic traits consistently trended towards greater P1, N2 and P3 amplitudes compared to individuals with low autistic traits. Data collection in progress may reveal different brain activation reflecting reliance on more effortful, conscious processing associated with higher levels of autistic traits in the absence of clinical impairment.

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