**A bilateral model of congenital prosopagnosia – connectivity between FFA and ATL**

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**Introduction**

Prosopagnosia is a condition, in which face perception or face recognition is affected (=”face blindness”). We differentiate between *acquired prosopagnosia* – caused by a lesion in a cerebral region related to face perception (e.g. OFA, FFA, ATL) – and *congenital prosopagnosia* (CP) – a quite heterogeneous disability affecting primarily identity recognition with no macroscopic structural lesions. There are two different opinions about the nature of CP. The *pathological view* sees clear differences between CPs and healthy subjects, whereas the *normative view* sees CP individuals as “just extremely bad” in the ability of face recognition. Consequently, CP individuals’ face recognition abilities are supposed to reflect the lower tail of a normal-like distribution of this ability in the population. In the following, we work with the *normative view of CP*.

We asked whether alterations of connectivity between face regions are associated with CP. Recent studies (Avidan et al., 2014) have shown that there is reduced brain activation related to face perception in anterior temporal lobe face area (ATL) in CP. As part of the extended face perception system (Haxby et al., 2000), ATL is connected to the core system most likely via fusiform face area (FFA). The reduced activation of ATL in CP may therefore be associated with disrupted connectivity between those regions. We hypothesized, that a reduction of ATL activation in CP (subjects with low face recognition scores) may result from a lack of face specific information transfer from FFA to ATL.

**Methods**

We recruited 49 subjects. Individual face recognition ability was quantified using Cambridge Face Memory Test (CFMT, Carrow et al., 2016). Participants with less than 58% accuracy were classified as CPs (8 out of 49 subjects). Each of the 49 subjects underwent a face localizer paradigm in which we presented blocks of faces with different emotions and houses as a control condition. We identified brain regions related to face perception on a single-subject level for bilateral FFA & ATL. We then extracted the time series and constructed a 4-region dynamic causal model (DCM, Friston et al., 2003) for each subject. Regressor *faces* entered the model via bilateral FFA (C-matrix). Each FFA and ATL shared structurally reciprocal connections and interhemispheric reciprocal connections between homotopic brain regions (A-matrix). Furthermore, *faces* modulated all available structural connections (B-matrix).

To estimate a group model, we used Parametric Empirical Bayes (PEB, Friston et al., 2015). We included each subjects model, and inter-subjects effects such as [1 - CFMT score] (as a proxy for prosopagnosia), and alexithymia score, autism score, gender and age as covariates of no interest. By means of the PEB model, we will present A, B and C matrices, as well as the effect of prosopagnosia and gender.

**Results**

Context-independent connection (A-matrix) became mostly positive in forward direction (FFA to ATL) and negative in backward direction (ATL to FFA), reciprocally positive between bilateral ATL, and reciprocally negative between bilateral FFA. Face perception excited the system positively via bilateral FFA (C-matrix). Furthermore, face perception increased the negative (inhibitory) connections from lFFA to rFFA, rATL to rFFA, lATL to lFFA, and flipped the sign at the connection from lATL to rATL to negative.

PEB revealed just one significant effect of CP onto the model. That was, the inhibitory – *face* specific - backward connection from lATL to lFFA was slightly weakened by CP (parameter estimate of +0.021). In contrast, gender had a very strong effect on the homologue connection in the right hemisphere, but with an effect of 50 times larger (parameter estimate of +1.18).

**Conclusion and Limitations**

We saw a weak effect of CP on our bilateral model of face perception. However, this effect was factor 50 smaller than the effect of gender onto the homologue connection of the right hemisphere. Therefore we did not find support for our hypothesis, that CP or the individual ability of face recognition may be associated with alterations in connectivity between FFA and ATL.

However, our study has several limitations: first of all, we recruited unobtrusive subjects of which only around 20% were CP. Reliable diagnoses of prosopagnosia should also be supported by another questionnaire and another face recognition test. Furthermore, individual ATL was difficult to localize and shows high inter-subject variability. Therefore we may have used heterogeneous ATL subregions for different participants.

**References**

Avidan, Galia, Behrmann (2014). Impairment of the face processing network in congenital prosopagnosia.

Haxby, Hoffman, Gobbini (2000). The distributed human neural system for face perception.

Carrow, Dalrymple, Barton (2016). Prosopagnosia: current perspectives.

Friston, Harrison, Penny (2003). Dynamic Causal Modelling.

Friston, Zeidman, Litvak (2015). Empirical Bayes for DCM. A Group Inversion Scheme.