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## Frontoparietal Activation Distinguishes Face and Space from Artifact Concepts

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### Abstract

Empirical and theoretical studies suggest that human knowledge is partly based on innate concepts that are experience-independent. We can, therefore, consider concepts underlying our knowledge as being broadly divided into inherited and acquired ones. Using fMRI, we studied the brain reaction in 20 subjects to violation of face, space (inherited), and artifact (acquired) concepts by presenting them with deformed faces, impossible figures (i.e., impossible chairs), and deformed planes, respectively, as well as their normal counterparts. Violation of the inherited concepts of face and space led to significant activation in frontoparietal cortex, whereas artifacts did not, thus distinguishing neurologically between the two categories. Participants were further exposed to these deformities daily for 1 month to test the supposition that inherited concepts are not modifiable, hence that prolonged exposure would not change the brain circuits that are engaged when viewing them. Consistent with this supposition, our results showed no significant change in activation for both categories, suggesting that such concepts are stable at the neural level at least within a time frame of 1 month. Finally, we investigated the regions of the brain that are critical for object representation. Our results show distinct and overlapping areas in the ventral visual cortex for all three categories, with faces activating the ventral visual cortex inferiorly, especially centered on right fusiform gyrus, and chairs and planes activating more diffuse regions, overlapping with the superior part of face region and mainly located in middle occipital cortex and parietal areas.

### INTRODUCTION

The brain organizes sensory inputs and acquires knowledge through the application of concepts,<sup>1</sup> which can broadly be divided into two categories, inherited and acquired (Zeki, 2009). The core knowledge theory of the origins of knowledge argues that infants begin life with innate knowledge (Spelke, 1994). Immanuel Kant believed that concepts of time and space are inherited (the so-called a priori) and that all experiences are read into them, the former to distinguish simultaneity from succession and the latter for contiguity from separation of self from nonself (Kant, 1781). Among innate concepts, we include categories such as faces, which humans are able to recognize at the very earliest stages of life. Many studies have shown that newborns of ages 0.5 hr to 2 months attend to face-like patterns (Sugita, 2008; Easterbrook, Kisilevsky, Muir, & Laplante, 1999; Simion, Valenza, Umiltà, & Dalla, 1998; Valenza, Simion, Cassia, & Umiltà, 1996; Johnson, Dziurawiec, Ellis, & Morton, 1991; Kleiner, 1987; Maurer & Barrera, 1981), possibly because of a hard-wired

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<sup>1</sup>The term “concept” in this context means “representation” or, more precisely, “neural representation.” In particular, we suppose that the patterns of neural activation recorded during scanning in response to faces and objects constitute the basis of representations of faces and objects.

and inherited preference for certain geometrical configurations (Simion, Leo, Turati, Valenza, & Barba, 2007; Simion, Cassia, Turati, & Valenza, 2001). This leads us to suppose that faces fall into a very special category of objects, whose recognition is facilitated by inherited brain mechanisms.<sup>2</sup> Such a category is to be distinguished from those, like planes or cars, whose recognition depends upon postnatal experience and which we consider as falling into the category of acquired concepts. It seemed plausible that we could distinguish the neural mechanisms engaged in the recognition of these two categories of objects differentially: Those engaged in facial recognition would activate areas associated with reaction to novelty in addition to the areas associated with their perception; they would be robust enough to resist modifiability by repeated exposure of the individual to abnormal facial configurations. The recognition of object categories, by contrast, would be modifiable through repeated exposure to abnormal versions, because the abnormal versions would themselves now become part of the concept. At the same time, we thought it interesting to introduce another category, that of impossible objects. Modification of the relationship between the various constituents of a chair to create an “impossible” chair will violate spatial relationships, making it interesting to learn whether neural mechanisms engaged in their perception are also resistant to change through re-petitive exposure. The introduction of such impossible figures into our study carried the additional advantage of acting as a control, because deformed faces might generate substantial emotional responses, whereas deformity of space is relatively neutral.

To pursue our aims, we used pictures of faces and air-planes and modified them by deforming them. Faces were deformed into two types, with and without facial configuration preserved. Planes were modified in the same way, with and without configuration preserved. The lower level of deformity in both cases preserved the essential configuration, the face being recognized as a disfigured one and the planes in a state that would be interpreted as fulfilling their function of flying, whereas the higher levels of deformity presented radical departures from what a face or plane would look like. We used impossible figures as the stimuli for deformity of spatial configurations. We conducted a longitudinal fMRI study to investigate the effects of a 1-month exposure to violation of these concepts. We hypothesized that brain regions associated with prediction error, such as DLPFC, will be activated (Fletcher et al., 2001) and that this reaction will not be attenuated for inherited concepts (deformed faces and impossible spatial relationships) even after prolonged exposure, although it might be modifiable for acquired concepts.

## METHODS

### Participants

Twenty-one healthy participants (10 men, 19 right-handed; mean age = 29.9 years,  $SD = 8.48$  years) were recruited through advertisements. All gave written informed consent, and the study was approved by the Joint Research Ethics Committee of the National Hospital for Neurology and Neurosurgery and the Institute of Neurology. Participants reported no history of psychiatric or neurological disorders and no current use of any psychoactive medications. To avoid the other-race effect, the phenomenon that we recognize faces of our own race better than faces of other races (O’Toole, Deffenbacher, Valentin, & Abdi, 1994), we only recruited white volunteers to correspond to the face database we used in the study.

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<sup>2</sup>The ontogeny of face processing is a matter of active debate. Here, we assume the existence of a rudimentary ability to detect faces at birth on the basis of the findings of human and primate infant studies. This ability develops and acquires experience-dependent components. We focused on the experience-independent aspect of face processing to contrast with artifact category perception. However, we acknowledge other possibilities such as generic expertise to explain face processing (Gauthier & Nelson, 2001).

## Activation Paradigm

**Before Training Session**—We used a mixed block/event-related fMRI paradigm, consisting of 12 blocks of stimuli, each representing one of three categories of objects, namely faces, planes, and chairs. For both the face and plane blocks, there were four types of stimuli: those showing slightly deformed, highly deformed, and normal faces or planes (DF1, DF2, NF, DP1, DP2, and NP, respectively) and stimuli that consisted of a fixation cross (fix) only; there were 18 events in each block. The distinction between high and low “deformity” depended on whether the overall configuration was preserved (Figure 1). For the chair blocks, there were only three types of events: impossible chairs (IC), normal chairs (NC), and fixation. There were 10 events in each block. With all three categories, the order of the blocks was counterbalanced, and the order of the events within a block was randomized. Each event was presented for 5 sec, and the interval between them was jittered with a mean of 500 msec, ranging from 0 to 1000 msec. There were 184 events in total, and all were different (apart from fixation). Scanning lasted around 17 min. The paradigm was generated using Cogent 2000 and Cogent Graphics ([www.vislab.ucl.ac.uk/Cogent2000](http://www.vislab.ucl.ac.uk/Cogent2000), [www.vislab.ucl.ac.uk/CogentGraphics](http://www.vislab.ucl.ac.uk/CogentGraphics)). The faces were derived from the XM2VTS database ([www.ee.surrey.ac.uk/CVSSP/xm2vtsdb/](http://www.ee.surrey.ac.uk/CVSSP/xm2vtsdb/)), and the planes and chairs were from the Internet. Some images were modified to constitute deformed figures using Adobe Photoshop CS2. During scanning, participants were asked to rate how strange the images were by using a four-button response box.

**After Training Session**—The activation paradigm used to scan subjects after the training session was identical to that used before training. However, different faces, planes, and chairs were used, although the degree of deformation remained consistent; the rationale being that the learning effect we were interested in was the capacity to adapt to a modified concept rather than to a specific picture.

## Training

Participants were asked to sign in to our study Web site daily to view four images, each presented for 15 sec, and rate how strange these images appeared to them. The training images were the same as those presented in the first scanning session. Training started from the day after the first scan and finished the day before the second scan. Their login profiles were monitored throughout this period, and a reminder was sent when they failed to sign in for a few days.

## Image Acquisition

MRIs were acquired using a 3-T scanner equipped with a standard transmit–receive head coil (Magnetom Allegra, Siemens Medical). An EPI sequence was applied for functional scans, measuring BOLD signals (TR = 2.88 sec, TE = 30 msec, matrix size =  $64 \times 72$ , slice thickness = 2 mm, gap between slices = 1 mm, field of view =  $192 \times 192$  mm<sup>2</sup>). Each brain image was acquired in a descending sequence comprising 48 oblique axial slices covering the whole brain. MRI signal losses in the OFC and amygdala were reduced by applying a z shim gradient moment and slice tilt (Weiskopf, Hutton, Josephs, & Deichmann, 2006). Only one fMRI session was run during each visit (one before training and one after training), and 370 whole-brain volumes were acquired. Anatomical images were acquired using a modified driven equilibrium Fourier transform sequence (Deichmann, Schwarzbauer, & Turner, 2004) in the sagittal plane to obtain a high-resolution structural image (176 slices per volume; isotropic resolution =  $1 \times 1 \times 1$  mm<sup>3</sup>, TR = 7.92 msec, TE = 2.4 msec). Field maps were also acquired with the Siemens standard gradient-echo field map sequence for correcting geometric distortion of EPI images (Hutton et al., 2002).

## fMRI Analysis

fMRI data were analyzed using SPM5 ([www.fil.ion.ucl.ac.uk/spm](http://www.fil.ion.ucl.ac.uk/spm)). Images were realigned with the first volume (after discarding the first six dummy volumes) and unwarped using field maps. The motion-corrected images were then coregistered to the individual's anatomical image and normalized to a standard EPI template on the basis of the Montreal Neurological Institute (MNI) template image. The images were resampled to  $2 \times 2 \times 2 \text{ mm}^3$  voxels and spatially smoothed with an 8-mm full-width-at-half-maximum Gaussian kernel.

To identify fMRI signal changes related to the activation, a general linear model with a design matrix coding the on-sets of each event type was regressed onto the time series at each voxel. Separate first-level analyses were conducted on fMRI data obtained before and after training sessions for each subject. Head movement parameters calculated from the realignment preprocessing step were included as regressors of no interest. The design matrix was convolved with the default SPM5 canonical hemodynamic response function and estimated using classical restricted maximum likelihood. The statistical maps were generated from a linear contrast of interests including (1) NF versus fix, (2) NP versus fix, (3) NC versus fix, (4) DF1 versus NF, (5) DF2 versus NF, (6) DP1 versus NP, (7) DP2 versus NP, (8) IC versus NC. Contrast images for these effects for each subject were entered into a random effects (second-level) analysis.

We conducted a conjunction analysis to identify areas that were commonly activated during before and after training sessions in each category. One-sample *t* tests were performed to identify differential activation between the viewing of deformities and their normal counterparts. When comparing distorted and normal conditions, any activation since the perception of different physical properties between categories would presumably be factored out. To identify the differences in activation before and after training and the differences between categories, as well as their interactions, we performed four sets of  $2 \times 2$  repeated measures ANOVAs comprising a main effect of Category (with two levels for each set: (1) DF1 and DP1, (2) DF2 and DP2, (3) DF1 and IC, and (4) DF1 and DF2), a main effect of Time (with two levels, before and after training), and a Category  $\times$  Time interaction.

Significant BOLD responses are reported at a voxel-level threshold of  $p < .05$ , whole-brain corrected for family-wise error. We additionally tested a priori hypothesized ROIs without family-wise error correction (Brodmann's area 9 and 46 defined by the WFU PickAtlas Toolbox). We also performed subsidiary ROI analyses in the face and object processing areas of the visual cortex to explore training-related and deformation effects in these areas. These results are reported at a voxel-level threshold of  $p < .001$ , uncorrected. Extent threshold was at least five voxels. All the statistical tests included age as a covariate of no interest in the general linear models.

## RESULTS

### Participants

Twenty of 21 participants completed the training and were rescanned (10 men, 18 right-handed; mean age = 30.3 years,  $SD = 8.50$  years). To accommodate the schedules of the scanner and the participants, the training period ranged from 26 to 35 days (mean = 29.4 days,  $SD = 1.9$  days). Days that participants forgot to undergo training are referred to as missing days and ranged from 0 to 13 days (mean = 4.9 days,  $SD = 3.8$  days). Thus, the percentage of completed training days within the training period ranged from 55.2% to 100% (mean = 83.3%,  $SD = 12.9\%$ ). The scanning sequence of the second scan for one participant was accidentally run without using the setting for minimizing the artifacts in the OFC and amygdala regions. However, exclusion or inclusion of this participant did not change the results.

## fMRI Data

**Normal Faces, Planes, and Chairs**—We first studied the BOLD response profile for normal face, plane, and chair perception by contrasting each of these categories with the baseline fixation task using conjunction analysis of both before and after training. In line with previous evidence (Kanwisher, McDermott, & Chun, 1997; Haxby et al., 1994; Sergent, Ohta, & MacDonald, 1992), increased activation in response to NF versus fix was found in inferior occipital and fusiform gyrus. Enhanced neural activity in the middle occipital and inferior parietal cortices was found for NP versus fixation and for NC versus fixation (Table 1 and Figure 2). We wanted to learn if deformed faces and planes and ICs also activate face and object processing areas as identified by their normal counterparts. We compared the contrasts of deformities versus fixation with the contrasts of normal counterparts versus fixation in each category using a conjunction analysis of both before and after training. We found that both deformed faces and ICs led to greater activation than their normal counterparts in the face and chair processing areas, respectively, but found no difference for the plane category. These findings suggest a distinction in neural activation between violations of inherited and acquired concepts.

**Activation by Deformed Faces and Planes and Impossible Chairs: Before Training**—We next studied the BOLD response profile for viewing deformed faces and planes and ICs by contrasting each of these deformities with their normal counterparts. Increased activation with DF1 versus NF was found in the inferior and superior parietal cortices, middle occipital cortex, and superior frontal cortex. Enhanced neural activity in the inferior parietal cortex appeared in the contrast of IC versus NC (Table 2 and Figure 3). Because of the strong activation in parietal areas with DF1 and IC, we tested for activation in this area in other comparisons with a less stringent threshold ( $p < .001$ , uncorrected). The findings showed parietal activations for all other comparisons, but they were significantly less than those for DF1 and IC. For the ROI analysis of BA 9/46 ( $p < .001$ , uncorrected), the activation was only found with DF1 (MNI coordinates:  $[-36, 6, 40]$ ,  $t_{18} = 6.22$ ;  $[54, 10, 36]$ ,  $t_{18} = 4.96$ ;  $[56, 32, 18]$ ,  $t_{18} = 4.31$ ) and IC ( $[54, 22, 28]$ ,  $t_{18} = 5.92$ ;  $[-42, 38, 24]$ ,  $t_{18} = 4.99$ ). We found no significant activation in all other contrasts (i.e., DF2, DP1, or DP2 versus normal counterparts) under this threshold.

**Activation by Deformed Faces and Planes and Impossible Chairs: After Training**—Increased activation with DF1 versus NF was found in the angular gyrus and inferior parietal cortex (Table 2 and Figure 3). Parietal activations were also found in other contrasts but significantly less than that for DF1. BA 9/46 activation was only found in the DF1 ( $[-46, 6, 40]$ ,  $t_{18} = 7.05$ ;  $[52, 32, 20]$ ,  $t_{18} = 5.79$  and  $[-46, 36, 14]$ ,  $t_{18} = 5.38$ ) and IC ( $[-54, 12, 30]$ ,  $t_{18} = 4.79$ ;  $[-42, 32, 18]$ ,  $t_{18} = 3.95$ ). There was no significant activation in all other contrasts (i.e., DF2, DP1, or DP2 versus normal counterparts) under this threshold.

No brain area showed significantly increased activation with normal compared with abnormal forms, either for the whole brain analysis or the ROI analysis in BA 46.

**Main Effect of Categories**—We studied the BOLD responses to violation of face, plane, and chair concepts, collapsing before and after training data. There was increased activation for DF1 compared with DP1 in the parietal, frontal, and temporal cortices. There was increased activation for DF2 compared with DP2 in the inferior parietal cortex, for DF1 compared with IC in the inferior and superior parietal cortices, and for DF1 compared with DF2 in the inferior parietal cortex, SMA, and precentral gyrus. BA 9/46 activation was only found in the comparisons of DF1 > DP1 ( $[50, 32, 18]$ ,  $t_{18} = 5.47$ ;  $[-42, 42, 10]$ ,  $t_{18} = 4.01$ ;  $[-52, 8, 38]$ ,  $t_{18} = 5.13$ ;  $[52, 12, 36]$ ,  $t_{18} = 4.5$ ), DF1 > DF2 ( $[-48, 6, 40]$ ,  $t_{18} = 5.04$ ;  $[48, 12,$

30],  $t_{18} = 4.46$ ; [54, 32, 20],  $t_{18} = 4.56$ ; [-42, 32, 18],  $t_{18} = 4.09$ ), and DF1 > IC ([-50, 8, 38],  $t_{18} = 4.25$ ) (Table 3 and Figure 3).

**Main Effect of Time**—This analysis allows us to define differential BOLD responses between before and after training sessions, collapsing across categories. There was no significant difference in the whole brain and BA 9/46 ROI analyses. We wondered if the percentage of completed training, ranging from 55.2% to 100%, could confound the results. A correlational analysis on the relation between the amount of training (i.e., percentage of completed training) and differential BOLD responses between before and after training (i.e., subtraction of two) did not yield any positive result either in whole brain or BA 9/46 ROI analyses. Although the training did not change the frontoparietal activation related to novelty reaction in response to deformities, we wanted to learn whether training changes the activation in the face and object processing areas as identified above. We found that the activation in response to the deformed faces and objects within the face and object regions, respectively, did not differ between before and after training sessions; however, more distributed areas around face and object areas were recruited in response to the deformed faces and objects, respectively, before than after training, which might imply that other object representations were recruited to resolve the perceived deformities before training.

**Category by Time Interactions**—One of our primary interests was to learn whether there are any brain areas showing training-related changes that are category specific, which we did through analysis of category by time interactions. There was no significant interaction effect in the whole brain and in all ROI analyses.

## DISCUSSION

We have shown that, although viewing normal faces or objects activates the ventral visual cortex, the viewing of abnormal faces and ICs, but not of abnormal planes, significantly activates, in addition, the posterior parietal cortex and BA 9/46. Thus, right from the start, the results of our imaging experiments differentiated between two categories of stimuli: deformed faces and impossible spatial relationships (chairs) on the one hand and deformed planes on the other. A further distinction was between the strength of deformity and the activation, especially in frontal cortex (BA 9/46) and parietal cortex. Thus, although a slight facial deformity and impossible spatial relationships elicited significant activation in frontoparietal cortex, severe facial deformity or severely deformed planes did not, possibly because such deformities strayed into an extreme territory, which bore only a superficial resemblance to their normal counterparts. Moreover, exposure to abnormal figures for a period of 1 month did not reduce the activity in these areas and, if anything, enhanced it.

We chose faces as an example of inherited concepts, because many studies show that face perception has a special status compared with objects in general (Sugita, 2008). We contrasted this with another category of objects—planes—whose recognition depends upon the formation of a postnatal concept and which can, therefore, be modified with the accretion of experience. By this reasoning, humans should be able to incorporate into their concept of plane any new design with which they had not been familiar. Such incorporation will be much more difficult, if not impossible, with inherited concepts; hence, repeated exposure to departures from normal faces cannot be easily incorporated into the concept of faces. As a control to these two extremes, we used impossible objects. This carried with it the promise of giving us insights into whether there is also an inherited concept of (possible) spatial relationships. This would be so if subjects failed to adapt to repeated exposure to impossible spatial relationships. In fact, it would seem that mildly deformed air-planes, which can still be considered as flying objects (with which our subjects would have been familiar and would, therefore, have acquired a concept of), do not activate frontoparietal

cortex significantly. It is not surprising, therefore, that extensive training on the viewing of these objects did not change the cortical activity elicited by viewing them. On the other hand, extensive training with mildly deformed faces did not diminish the activity in frontoparietal cortex, thus suggesting that such activity, which we interpret as registering a departure from what an inherited concept of a face should correspond to visually, is resistant to modification by training, much as we supposed.

### **Violation of Inherited, but not Acquired, Concepts Activates Frontoparietal Areas**

DLPFC is sensitive to unpredictable stimulation, and its reaction to such stimulation decreases when tasks become well learned (Fletcher et al., 2001; Rainer & Miller, 2000; Raichle et al., 1994). DLPFC activity is associated not only with unpredictable occurrences but also with conflict resolution. Novel events can also be regarded as unpredictable ones, a mismatch between expectation and experience. The neural mechanisms for detecting novelty have been studied extensively in “oddball” (unusual) paradigms (Sutton, Braren, Zubin, & John, 1965). A distributed network is thought to be involved in novelty detection, including frontal and parietal cortex (Ranganath & Rainer, 2003). Parietal cortex, especially its posterior part, around the intraparietal sulcus, has been implicated in various functions including spatial representation, the guidance of action, and attention (Culham & Kanwisher, 2001). This region has been usually studied in the context of spatial attention, but recent evidence suggests that it may also be involved in abstract cognitive processing of nonspatial stimuli (Freedman & Assad, 2009; Lehky & Sereno, 2007; Wojciulik & Kanwisher, 1999; Sereno & Maunsell, 1998). Therefore, it is plausible to suppose that frontoparietal activation indicates that violation of inherited concepts is more salient than violation of artifact concepts; indeed, in our study, it is possible that the slightly deformed planes did not constitute a violation at all but could be incorporated into the category of planes, because they could be construed as fulfilling their function of flying. Its activation may reflect, therefore, more the recruitment of attentional resources to initiate learning than the processing of spatial information (Corbetta, Patel, & Shulman, 2008). This is especially so because we maintained similar scales of spatial modification between categories. Consistent with this, a series of monkey studies demonstrated that temporal, prefrontal, and parietal cortical areas play significant and complementary roles in visual categorization. Neurons in inferior temporal cortex were activated with visual analysis of features (Sigala & Logothetis, 2002) with area V5 showing motion direction selectivity, whereas PFC and posterior parietal cortex, connected to temporal cortex and V5, were found to be involved in more generalized and abstract encoding of category membership, an encoding that is influenced by experience (Freedman & Assad, 2006; Freedman, Riesenhuber, Poggio, & Miller, 2003). Our results, which show an involvement of temporo-occipital activation for normal object representation and of frontoparietal areas when the concepts are violated, are consistent with these results.

### **Neural Stability of Inherited Concepts**

That the activation pattern did not change after training indicates that a 1-month exposure to the deformities of faces and impossible spatial relationships was not sufficient to make participants fully adapt to modified concepts for these categories, suggesting that the concepts behind these visual stimuli are stable, although we do not know whether longer exposure would have resulted in adaptation. This result distinguishes our findings from observations of repetition suppression, when repetition of stimuli leads to experience-related attenuation of neural activity (Li, Miller, & Desimone, 1993) possibly because of perceptual fatigue, sharpening representation of stimuli or their faster processing, which might involve temporary changes in ion currents or neurotransmitter concentration or even long-term changes such as protein synthesis in synapses (Grill-Spector, Henson, & Martin, 2006). One explanation for the lack of learning effects or repetition suppression is that we used a

different set of stimuli after training although the degree of deformation remained consistent. As described above (Methods) the rationale for this is that the learning effect we were interested in was the capacity to adapt to a modified concept rather than to a specific picture. Negative findings of learning effects contradicted our prediction partially, because we had hypothesized that artifact concepts are modifiable and that the cortical activation we observed in response to abnormal configurations of this category would attenuate after training, in both parietal cortex and area BA 9/46. However, we did not observe this attenuation, and in fact, we did not obtain significant parietal and BA 9/46 activation with deformed planes even before training. This may be one of the reasons for the absence of reduced activation after training, because there was not enough activation in these areas initially. Therefore, we did not have any evidence to conclude whether acquired concepts are stable. Finally, although we observed some learning effects in the visual cortex, these changes in activation were outside the identified face and object areas (see Results: Main effect of time). Our conclusion is, therefore, that prolonged training does not alter the pattern of activation elicited by stimuli for which the brain has an inherited concept.

### Facial Configuration Has a Special Role

Newborns (median age = 9 min) show a preferential tracking for a moving schematic face compared with scrambled faces or a blank head outline (Goren, Sarty, & Wu, 1975). This suggests that faces are represented as a collection of preferred geometrical arrangements for infants (e.g., more elements in the upper part and with curved contour) (Simion et al., 2001, 2007). Theoretical accounts argue that this rudimentary face concept or template at birth is a likely product of adaptive evolution and is initially mapped sub-cortically (Johnson, 2005; Simion et al., 1998; Johnson et al., 1991), later developing more sophisticated face processing abilities with the engagement of cortical areas such as fusiform gyrus (Johnson, 2005; Johnson et al., 1991). Therefore, it seems that only a part of the face processing system is equipped to process faces at birth and that this system is refined by experiences later in life. This may explain our finding showing an interesting difference in frontoparietal activation between DF1 and DF2. If face preference at birth relies on a specific configuration, preserved only in DF1 but completely violated in DF2, then greater frontoparietal activation is expected with DF1 than DF2. This may be because the inherited mechanisms for face recognition are engaged only when exposed to specific facial configurations and not to highly fragmented facial features.

### Neural Basis of Impossible Objects

Cognitive scientists use an expectancy violation looking method to investigate whether infants are able to discriminate impossible objects from others. Results show that 4-month-old infants look longer at impossible cubes (Shuwairi, 2009; Shuwairi, Albert, & Johnson, 2007), indicating that the concept of space emerges early in life. Our results suggest that the (possibly inherited) concept of space is one of possible spatial relationships comparable with the experience of viewing Escher or Magritte paintings, which are always surprising, even in spite of familiarity with them. Our findings also showed significant frontoparietal activation in viewing impossible objects. A previous PET study showed that inferior temporal activation was associated with possible, but not impossible, objects, although participants made decisions on plausibility of spatial relationship of objects (Schacter et al., 1995). We, thus, amplify the Kantian doctrine of the a priori of space by suggesting that it relates only to normal spatial relationships. We extend this to say that what constitutes an inherited a priori in face recognition relates to normal spatial relationships between the constituents of a face.

## Distributed View of Object Representation

On the basis of clinical studies and fMRI studies of healthy individuals, two hypotheses have been proposed to account for object recognition. The distributed view suggests that object discrimination depends on distributed but overlapping representations in the brain (Haxby et al., 2001), whereas the modular view argues that different categories of objects are represented in segregated and specialized areas (Spiridon & Kanwisher, 2002). Our conjunction analyses produced by faces, planes, and chairs showed that they activated overlapping parts of the ventral visual cortex but that each category maintained its own territory within the overlapping zones (Figure 2), thus demonstrating an overlapping and segregated system for object representation.

## Acknowledgments

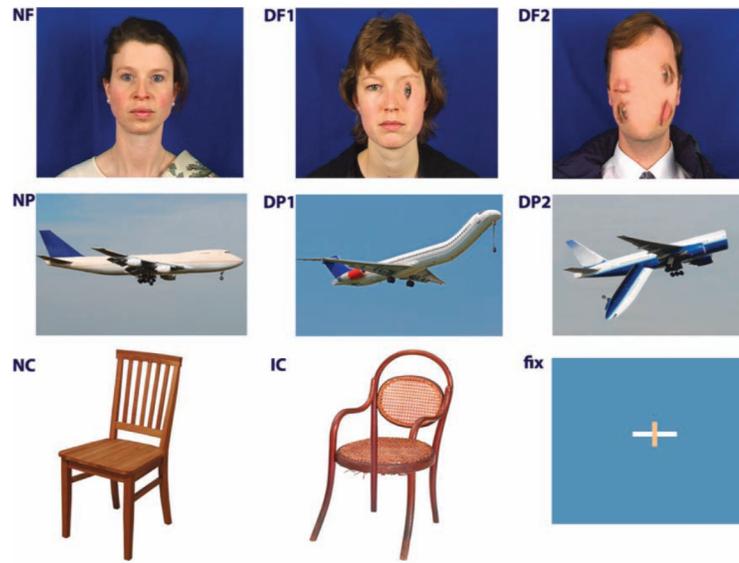
The work was supported by the Wellcome Trust, London, UK. We thank Chris Frith for commenting on the manuscript.

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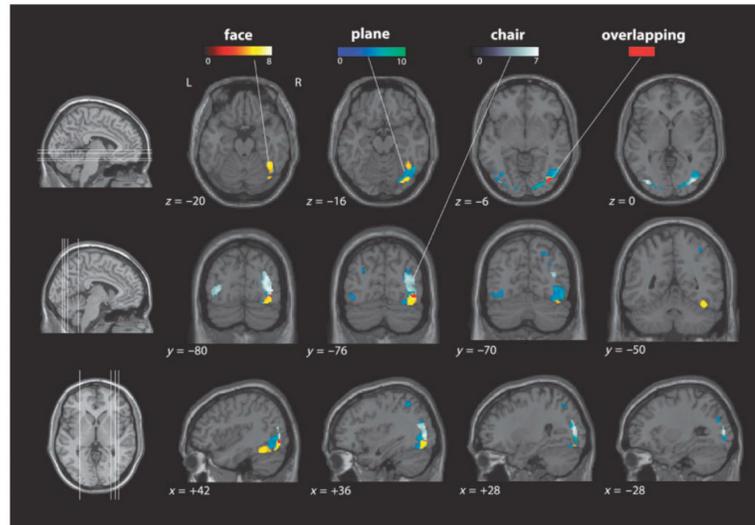
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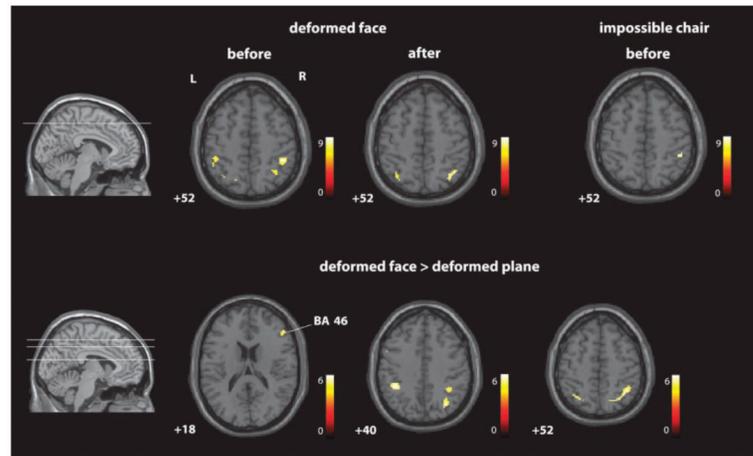


**Figure 1.**  
An example set of stimuli for each condition.



**Figure 2.**

Activation maps in three planes with normal stimuli. The location of each slice is shown on the left. Conjunction analyses of before and after training showed regions activated before and after training for each category. Activations are reported at voxel-level corrected family-wise for multiple comparisons over the whole-brain volume. Color bars represent statistic values of activations. Numbers denote the z coordinate of each axial slice.



**Figure 3.** Activation with deformities. (top) DF1 > NF before and after training; IC > NC before training. (bottom) Differential activation between categories in deformed conditions. The maps show the main effects of category derived from a  $2 \times 2$  ANOVA comparing BOLD responses between DF1 and DP1. DF1 demonstrated greater activation in parietal and frontal areas compared with DP1. Conventions as in Figure 2.

**Table 1**  
 Activations with Each Category (Conjunction of Both before and after Training)

	<i>Regions</i>	<i>MNI Coordinates</i>			<i>BA</i>	<i>t</i>	<i>Cluster Size</i>
		<i>x</i>	<i>y</i>	<i>z</i>			
<i>Conjunction of before and after Training</i>							
NF > fix	inferior occipital cortex	40	-76	-8	19	8.25	173
	fusiform gyrus	40	-52	-20	37	6.79	159
NP > fix	middle occipital cortex	28	-86	0	18	10.13	1319
	middle occipital cortex	-28	-86	2	18	7.14	89
	inferior occipital cortex	-34	-68	-4	19	6.41	121
	angular gyrus	36	-54	52	40	6.41	113
	middle occipital cortex	-16	-92	-2	18	6.30	28
NC > fix	middle occipital cortex	-28	-76	28	19	6.17	17
	inferior parietal cortex	40	-44	46	40	5.72	8
	middle occipital cortex	30	-82	12	18	7.98	425
	middle occipital cortex	-32	-82	4	18	7.63	150
	inferior parietal cortex	34	-56	54	7	5.80	5

BA = Brodmann's area.

**Table 2**  
Differential BOLD Responses between the Deformities with Their Normal Counterparts

	Regions	MNI Coordinates						df	Cluster Size
		x	y	z	BA	t			
<i>Before Training</i>									
DF1 > NF	inferior parietal cortex	44	-40	52	40	9.15	18	99	
	superior parietal cortex	-18	-68	50	7	8.78	18	22	
	inferior parietal cortex	-40	-36	42	40	8.60	18	156	
	middle occipital cortex	-26	-72	34	19	7.92	18	6	
	superior frontal cortex	24	10	56	8	7.36	18	7	
	superior parietal cortex	-34	-62	50	7	7.79	18	5	
DF2 > NF	none								
DPI > NP	none								
DP2 > NP	none								
IC > NC	inferior parietal cortex	46	-40	52	40	8.65	18	12	
<i>After Training</i>									
DF1 > NF	angular gyrus	34	-66	54	7	9.16	18	50	
	inferior parietal cortex	-46	-40	40	40	9.00	18	80	
	inferior parietal cortex	-30	-66	46	7	8.62	18	38	
DF2 > NF	none								
DPI > NP	none								
DP2 > NP	none								
IC > NC	none								

Table 3

Differential BOLD Responses between Object Categories of Deformity

<i>Main Effect of Category</i>	<i>Regions</i>	<i>MNI Coordinates</i>			<i>t</i>	<i>Cluster Size</i>	
		<i>x</i>	<i>y</i>	<i>z</i>			
DF1 > DPI	superior occipital cortex	30	-70	42	7	6.33	272
	inferior parietal cortex	-40	-42	40	40	6.28	92
	inferior temporal cortex	56	-48	-12	20	6.21	44
	superior parietal cortex	-28	-66	50	7	6.02	72
	middle frontal cortex	30	2	60	6	5.72	56
	inferior frontal cortex	50	34	18	45	5.58	25
DF2 > DP2	inferior parietal cortex	38	-46	40	40	5.46	31
	middle occipital cortex	34	-80	16	19	5.20	6
	inferior parietal cortex	38	-52	56	40	5.40	10
DF1 > DF2	SMA	2	22	46	39	5.43	39
	precentral gyrus	-48	8	40	7	5.19	7
DF1 > IC	inferior parietal cortex	-42	-40	40	5	5.22	5
	inferior parietal cortex	-42	-40	40	40	5.69	36
	superior parietal cortex	-32	-64	50	7	5.44	7

BA = Brodmann's area.