# **Supplementary Material**

#### **Individual V1 surfaces**

### [Figure 1 about here.]

Table S1 shows the surface area computed from the reconstruction of the entire cortex for each hemisphere as well as the surface area of V1. The mean and standard deviation of total cortical surface areas agree with previous reports (Henery and Mayhew, 1989; Barta and Dazzan, 2003), as do the estimates for V1 surface area (Stensaas et al., 1974; Andrews et al., 1997).

Consistent with the results of previous studies, the surface area of V1 varies by a factor of more than two among individuals. The V1 atlas presented here exhibits very little variability, suggesting that this surface-based registration method compensates for surface area differences. To investigate this the relative expansion ratio of V1 was computed as the ratio of the surface area of V1 on the spherical surface representation after registration to the pre-registration surface area. The resulting relative expansion ratios for each hemisphere are shown in the rightmost column of Table S1.

#### [Table 1 about here.]

Comparison the deviation of the individual V1 surface area from the mean with the relative expansion ratios observed here reveals a clear trend for smaller V1 to expand and larger V1 to contract during registration. This was confirmed by a regression analysis of the deviation and expansion ratios ( $r^2 = 0.45$ , p < 0.001).

## Atlas generalization

To determine the amount of variability in the location of V1 when individuals are registered to a template that was not generated from the same subject pool, the surfaces reconstructed from the whole-brain scans of the *ex vivo* hemispheres were registered to an existing template generated from 40 healthy, living human

hemispheres (Fischl et al., 1999). The location of V1 for each individual was mapped in the template space, and the quality of alignment was taken to represent the degree of generalization of the atlas.

Spatial probability maps for V1 following surface registration of each *ex vivo* surface to the *in vivo* template are shown in Figure S2. The spread of V1 is very similar to that observed when registering to the template generated from *ex vivo* subjects. The similarity measures confirmed a comparable level of variability for each hemisphere, with a kernel size of 5.25 mm, a percent blurring of 43.5%, a Jaccard coefficient of 0.48, and a percent overlap of 68.3% for the left hemispheres and a kernel size of 6.5 mm, a percent blurring of 56.9%, a Jaccard coefficient of 0.34, and a percent overlap of 52.9% for the right hemispheres.

### [Figure 2 about here.]

Despite slightly increased variability, the atlas still exhibits low prediction error when registered to subjects not used to create the registration template. Interestingly, the best V1 alignment was observed at a quite high value of  $\lambda_d = 10.0$  for both the left and right hemispheres. The value of  $\lambda_A$  was similar to that found in the optimal parameters value search: 1.0 for the left hemispheres and 0.4 for the right hemispheres.

# V1 probabilistic atlas with the commonly used parameter values

A probabilistic atlas of V1 was built using the commonly employed parameter values of  $\lambda_d=0.1$  and  $\lambda_A=0.2$  instead of the optimal parameters determined here. The resulting V1 atlas is shown in Figure S3 and appears qualitatively to be of lower alignment quality than the atlas presented here, which is in agreement with the lower computed alignment quality measures.

[Figure 3 about here.]

### Calcarine sulcus probabilistic atlas

A probabilistic atlas of the calcarine sulcus was built using the same surface registrations used to build the V1 atlas. Figure S4 shows the resulting calcarine atlas resulting from registration to the template generated

from the ex vivo hemispheres.

### [Figure 4 about here.]

## Effect of neurological disease

It is well known that cortical gray matter atrophy is associated with both AD (Frisoni, 1996; De Leon et al., 1997; Jack et al., 1997) and HD (Vonsattel and DiFiglia, 1998; Halliday et al., 1998). However, due to the difficulty of measuring cortical area boundaries previous MRI studies of both diseases have focused on local atrophy, either addressing cortical thinning (Dickerson et al., 2001; Rosas et al., 2002, 2005) or a local measure of gray matter density (Thompson et al., 1998; Janke et al., 2001; Thompson et al., 2003).

The subjects from which the ten right hemispheres were obtained included five individuals that had no history of neurological disease, one individual diagnosed with AD, three individuals diagnosed with HD, and one individual for which no information regarding history of neurological disease was available. Of the left hemisphere group five individuals had no history of neurological disease, one individual was diagnosed with AD, two individuals were diagnosed with HD, and for two individuals no information regarding history of neurological disease was available. To determine if neurological disease had an effect on V1 alignment, the similarity measures were computed for each of the 252 possible groups of five individuals. The results were then sorted in descending order of alignment quality for each measure, and the rank of the group of all diseased individuals (including the individual(s) for which disease state was unknown) was noted.

Table S2 shows the similarity measure values for the all-diseased group compared to the mean value over all possible groupings of five individuals for both hemispheres separately. For the right hemispheres, the all diseased group exhibits V1 alignment quality near the low end of the range for each similarity measure. The all diseased group shows particularly low alignment quality for percent overlap. This suggests that V1 alignment quality would be greater than that reported here if all the subjects for which the right hemispheres were obtained had no history of neurological disease. However, the all diseased group for the left hemispheres exhibited no effect of disease, with the rank of the all-diseased group near the middle of the range for each similarity measure. This may explain the overall greater V1 alignment quality observed for the left hemispheres.

#### [Table 2 about here.]

Here, no consistent effect of disease on V1 alignment quality was observed. For the left hemispheres, disease state had no effect on alignment quality, while alignment quality was worse for right hemispheres with a history of disease. It is important to note that even if disease has a strong effect on area location, the results presented here would actually *underestimate* the variability in the normal population. Because the conclusions of the present study indicate low variability in the location of V1, including diseased individuals in the subject pool will not create a spurious result. However, we do not have sufficient data to make statements regarding the effect of AD or HD on the alignment quality of V1 overall.

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