Additional Figures | Cortical areas interact through a communication subspace

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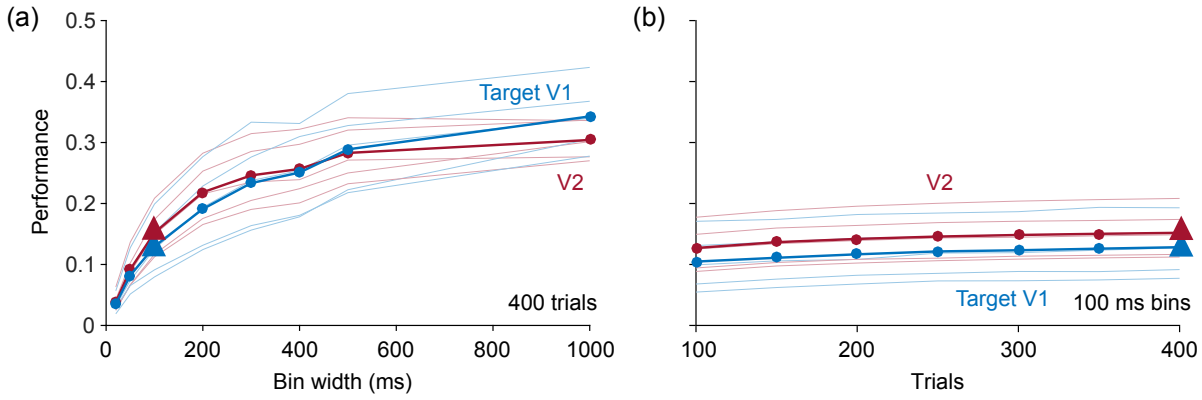
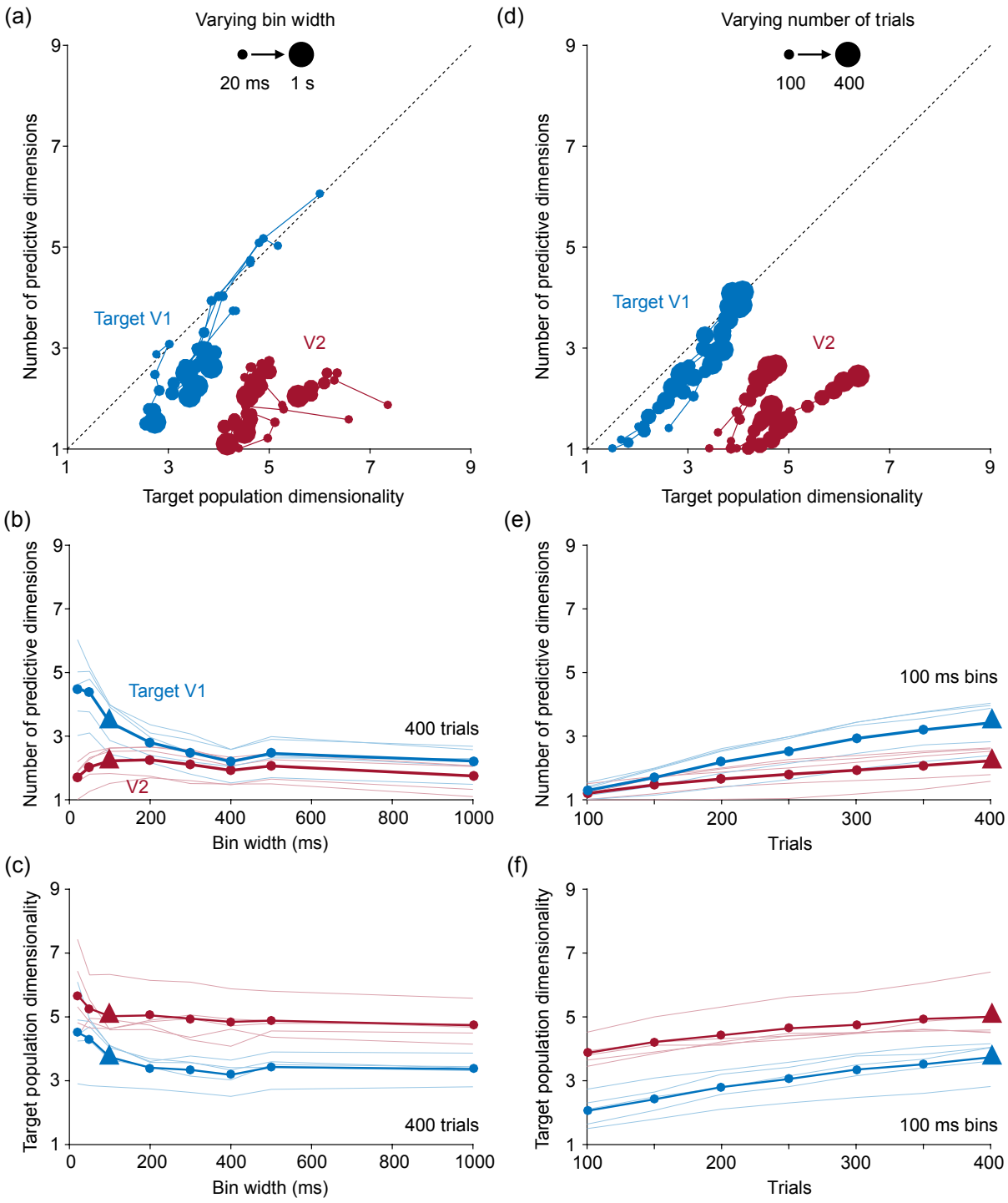


Additional Figure 1 | **Similarity of predictive performance for target V1 and V2 is robust to bin size and number of trials.** The results in the main text use a 100 ms bin width for counting spikes and 400 trials per data set. Here we assess how predictive performance of target V1 and V2 varies with the bin width and number of trials. **(a)** Using all 400 trials, we found that predictive performance increased with bin width (solid line shows the average across all data sets; faded lines show the average for each recording session; triangle indicates the 100 ms bin width used in the main text). For each bin width explored, the predictive performance for the target V1 and V2 populations remained similar to each other. **(b)** For a fixed bin width of 100 ms, we found that the predictive performance for the target V1 and V2 populations was largely independent of the amount of data used (triangle indicates the 400 trials used in the main text). Thus, the similarity in predictive performance for the target V1 and V2 populations was robust to the number of trials used to fit the regression model.


(a)
Varying bin width
20 ms
1 s
Number of predictive dimensions
Target population dimensionality
Target V1
V2
(d)
Varying number of trials
100
400
(b)
400 trials
Bin width (ms)
(e)
100 ms bins
Trials
(c)
Target population dimensionality
(f)

Additional Figure 2 | The V1-V2 communication subspace was evident across a wide range of time bin widths, but was difficult to detect with limited numbers of trials. To test whether our finding of a V1-V2 communication subspace depended on the choice of time bin width, we repeated the analyses presented in the main text for different bin widths and 400 trials per data set (left column). **(a)** We found that the V1-V2 communication subspace was present over a wide range of time bin sizes. The number of V2 predictive dimensions was smaller than the dimensionality of the V2 population responses for all bin widths (each dot corresponds to the average dimensionality for one session with the dot size indicating the bin size). In contrast, for the target V1 populations, the number of predictive dimensions roughly matched the dimensionality of the target population. **(b)** The difference in the number of estimated predictive dimensions for the target V1 and V2 populations was evident for all bin widths, but less evident for larger bins (solid lines show the average across all data sets; faded lines show the average for each recording session). Note that increasing the bin width reduces the amount of data available for model fitting; as we show below, it is more difficult to identify predictive dimensions with small amounts of data. Thus, results for larger bin widths should not be interpreted as indicating a timescale-dependence of the communication subspace. **(c)** The estimated target population dimensionalities are consistent across a wide range of bin widths: V2 population activity was always higher dimensional than the target V1 activity.

To determine how the estimated dimensionalities depend on the amount of recorded data, we repeated the analyses after subsampling a fraction of the recorded trials (using a fixed bin width of 100 ms). **(d)** For different numbers of trials, the number of V2 predictive dimensions was consistently smaller than the dimensionalities of the V2 populations. For the target V1 population, the number of estimated predictive dimensions roughly matched the dimensionality of the target population. Thus, the findings for the full data set were also evident when subsets of data were used in the analysis. **(e)** However, we found that using less data for model fitting had a significant impact on the number of estimated predictive dimensions for both target populations. When we used 100 trials per data set we could only identify, on average, a single predictive dimension for each the target V1 and V2 populations. As we increased the amount of data, the difference between the number of estimated predictive dimensions for the target V1 and V2 populations increased (as do the number of estimated predictive dimensions for each target population). This trend suggests that had we been able to record more trials, we would have detected an even larger difference between the number of V1 and V2 predictive dimensions. **(f)** The dimensionalities of the target populations increased with the number of trials, suggesting that the true dimensionality of the target populations in likely higher than what we could identify with 400 trials (see also Williamson et al., 2016).