tant pooled distributions possess the properties listed above. The results of the most natural noise model are illustrated in Figure 3b. Under this model, each long-range horizontal connection, ideally designated to connect cells of orientation difference $\Delta\theta$, is shifted to connect cells of orientation difference $\Delta\theta + \epsilon_\sigma$, where ϵ_σ is a wrapped gaussian (i.e., normally distributed and wrapped on $\mathcal{S}^1$) random variable with zero mean and variance σ (see the appendix for details). As the figure shows, it takes an overwhelming amount of noise (s.d.$\geq$ 35 degrees) to transform the collinear distribution to one that resembles the measured data in terms of spread and peak height, but the nonmonotonic behavior of the variance is never reproduced. (For space considerations, we omit the results of other connection-based noise models, or the noisy distributions based on the association field model, all of which were even less reminiscent of the measured physiological data.)

A second possible source for the inconsistencies between the predicted and measured distributions may be the extracellular injection protocol commonly in use by physiologists to trace long-range horizontal connections (e.g., Gilbert & Wiesel, 1989; Malach et al., 1993; Kisvárday et al., 1994, 1997; Bosking et al., 1997; Schmidt et al., 1997; Sincich & Blasdel, 2001). Due to the site-selection procedure used, cells stained by these injections are likely to have similar orientation preferences (e.g., Bosking et al., 1997, p. 2113, or Schmidt et al., 1997, p. 1084). However, their orientation tuning may nevertheless be different, sometimes significantly (note such a cell in Bosking et al., 1997, Fig. 4B). Consequently, the distribution of presynaptic terminals (boutons) traced from the injection site may incorporate an artificial, random spread relative to the single orientation typically assumed at the injection site. Preliminary evidence from a recently developed single-cell protocol (Buzás et al., 1998) suggests that leakage in the injection site cannot bridge the gap between the predicted collinear distribution and those measured anatomically. However, we also examined this possibility computationally by modeling the leakage in the injection site as a wrapped gaussian random variable of predefined variance.[2] The base distributions (collinear or association field) of the computational cells selected by this process were then summed up and normalized, and the resultant (random) distribution was attributed to the original cell representing the injection site. Repeating this process many times yielded a collection of (different) distributions, for which we calculated an average and variance (see the appendix for details). The results are illustrated in Figure 3c. Similar to random variations at the level of individual connections, here too it takes an overwhelming amount of noise (s.d.$\geq$ 35 degrees) to transform the colinear distribution to one that resembles the measured data in terms of spread and peak height, but the nonmonotonic behavior of the variance is never reproduced.

[2] A wrapped gaussian model was particularly suitable here due to the injection site selection protocol typically used in the extracellular injection protocol; see the appendix.