SYNAPTIC DEPRESSION AND CORTICAL GAIN CONTROL

By

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Abbott *et al.* (1) modeled short-term synaptic depression to explain experimental results concerning synaptic transmission between layers 4 and 2/3 in rat visual cortex. Thomson (2) wrote "this is a welcome refinement of more traditional models". This model has actually been used since 1968 (3-5) to analyse all the properties described in (1). Here other, untested, properties and predictions are noted concerning how synaptic depression may influence cortical dynamics. Given spike rate R, our gain control variable A obeys:

$$dA/dt = b(G-A)-AR. \qquad (1)$$

In equation (1), gain A decreases by mass action at rate AR, and accumulates to G via term b(G-A). As in Figure 1A from reference (1), gain A depresses during repetitive stimulation, and emulates steady-state depression in their Figure 1B. A similar law was derived to fit data on posttetanic potentiation of a neuromuscular junction in the rat (6). In addition, the steady-state conductance obeys:

$$S=AR, \qquad (2)$$

which increases as in their Figure 1B. We have analysed the model's transient response to sudden input increments (e.g., 7-10), its uniformizing property at large spiking rates (e.g., 11), and its Weber-Fechner property (e.g., 12, 13). Abbot (14) agrees that "The model you described is indeed the continuous firing rate form of the model that we used".

Why might many brain processes use synaptic gain control? First, note that this model is the simplest transduction law that generates unbiased signals using a chemical transmitter (12, 13): The simplest unbiased input S to a postsynaptic site is proportional to the presynaptic output R:

$$S=GR, \qquad (3)$$

where G is the gain. Let S represent release or inactivation of chemical transmitter. By equation (3), chemical G is released by mass action rate S=GR. How is G replenished or reactivated? In (3), G replenishes instantly. In vivo, it happens at a finite rate. Interpret gain A as the instantaneous amount of the chemical, and G is its target value. Then transmitter inactivates at rate S=AR and is restored towards G at rage b(G-A), as in equations (1) and (2). Synaptic depression thus tries to maintain unbiased transduction, but falls behind if input rate changes faster than recover rate. Slow recovery rates have valuable properties, which have been used in neural models of vision, audition, learning, recognition, reinforcement, attention, movement, circadian rhythms, and mental disorders.

In visual cortex positive feedback between cortical layers builds groupings of scenes (15, 16). Such feedback could maintain cortical activity long after its inputs shut off, thereby smearing visual groupings when scenes change. By depressing synapses between layers 4 and 2/3, the cortex can rapidly shut off previously active groupings to process the next scene with reduced bias (17, 18). Model cortical circuits involve opponent interactions between ON and OFF cells. Depressing synapses enable the offset of inputs at ON cells

to cause activity rebounds at OFF cells which, in turn, inhibit the previous grouping. Such rebounds have been visualized in visual cortex using optical imaging (19). Now that depressing cortical synapses have been isolated, neuroscientists can search for these and other predicted cortical circuit properties.

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