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The Biophysical Properties of Spines as a Basis for their Electrical Function: a Comment on Kawato & Tsukahara (1983)

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In a theoretical study of the passive cable properties of dendritic spines Kawato & Tsukahara (1983) claim to have proved that "the dendritic spine has no significant electrical function" (from their discussion). However, Kawato & Tsukahara restrict their analysis to current inputs to spines. Since the dimensions of spines are very small, their input resistance is expected to be very large and the synaptic input to spines has to be modeled as conductance change. Under this assumption, spines show interesting (non-linear) electrical properties: i) the somatic potential induced by an excitatory synapse on a spine may depend strongly on the shape of the spine and ii) the effect of inhibition might be confined to the spine.

Kawato & Tsukahara (1983) consider the somatic potential induced by a synapse injecting current into a passive dendritic spine. They show for current inputs that under realistic assumptions of membrane parameters and dendritic spine dimensions the attenuation from the spine head to the dendritic stem below the spine can be neglected. Therefore, the authors conclude, dendritic spines do not have any interesting electrical function. However, while their specific result is correct, their general conclusion is not warranted and is in general wrong. Due to the small dimensions of the spine, their input resistance is expected to be quite high, easily surpassing 200 MΩ. Therefore, as we have specifically pointed out before (Koch & Poggio, 1983*a,b*) it is inappropriate to approximate the synaptic input as current. If one considers conductance changes as synaptic inputs, spines do show a variety of interesting non-linear properties, a point emphasized by various authors (Diamond, Gray & Yasargil, 1970; Rall, 1974, 1978; Jack, Noble & Tsien, 1974; Crick, 1982; Koch & Poggio, 1983*a,b*; Horwitz, 1983; Perkel, 1983). In particular, due to the strong dependence of the somatic *weight* of spines on their shape, they could serve as a locus for short- and long-term memory.

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In order to analyse the electrical properties of dendritic spines on the basis of passive cable theory, Kawato & Tsukahara use a mathematical technique developed by Butz & Cowan (1974) that generates a closed expression of the voltage in arbitrary dendritic structures as a function of synaptic *current* inputs. Modeling a spine by a very short cylinder with a sealed end, they derive the expression $PSP_{\text{spine}}(t)$ denoting the somatic depolarization caused by a synapse injecting current into a spine. If the same current is injected at the base of the spine, the potential $PSP_{BP}(t)$ is induced at the soma. Comparing these two expressions, Kawato & Tsukahara define the function $h(t)$ describing the attenuation effect of a spine. $PSP_{\text{spine}}(t)$ can now be represented as the convolution of $PSP_{BP}(t)$ with $h(t)$. Therefore, if one can show that $h(t)$ is very much shorter than the duration of $PSP_{BP}(t)$, $h(t)$ can be approximated by a delta function and all of the current injected into the spine reaches the dendritic stem. Using the Butz & Cowan graphical calculus and electrical circuit theory, Kawato & Tsukahara prove in a mathematical tour de force that $h(t)$ can indeed be approximated by $\delta(t)$.

Without considering the range of validity of the current input case, the authors conclude that both the attenuation and the isolation effect of the spine can be neglected and that a morphological change of the spine only leads to negligible changes in somatic potential. This conclusion is, in general, incorrect.

In our analysis of the biophysical properties of dendritic spines, we also used a technique based on basic properties of the Green function of a linear branched cable (to compute numerically the transfer function in extended dendritic trees we likewise used the Butz & Cowan algorithm). Modeling a spine by a thin and long cylindrical spine neck (of length l_N and diameter d_N) and a shorter and stubbier cylindrical spine head we first consider the linear properties of spines; i.e. the properties for *current* inputs and passive membrane. Neglecting the very small current losses through the membrane of the spine neck and for a broad range of membrane parameters, we derived an approximated expression for the spine input impedance

$$\tilde{K}_{11}(\omega) = (\tilde{K}_{22}(\omega) + R_N)/(1 + i\omega\tau_s) \tag{1}$$

where $\tilde{K}_{22}(\omega)$ is the input impedance of the dendritic stem just below the spine neck as a function of frequency ω, R_N is the ohmic resistance of the spine neck cylinder ($R_N = 4R_i l_N / \pi d_N^2$), and $\tau_s = R_N C_H$ is the time constant of the spine (R_i is the specific intracellular resistivity and C_H the total capacity of the spine head). Thus the spine input resistance equals the dendritic shaft input resistance plus the spine neck resistance filtered by a low pass filter. In the time domain the above expression is equivalent to a

convolution of $(K_{22}(t)+R_N)$ with $\tau_s^{-1}\exp^{-t/\tau_s}$. For a reasonable range of parameter values (see Koch & Poggio 1983*b*) τ_s is three to four orders of magnitude smaller than the time constant τ_m of the neuron (while τ_m has a typical value of some tens of msec τ_s lies in the μsec range). Therefore we conclude similar to Kawato & Tsukahara, that this function can be approximated by a delta function and

$$\tilde{K}_{1s}(\omega)=\tilde{K}_{2s}(\omega) \tag{2}$$

where $\tilde{K}_{1s}(\omega)$ (resp. $\tilde{K}_{2s}(\omega)$) is the transfer impedance between the spine head (resp. the dendritic stem just below the spine) and the soma (see in particular equations (7) to (10) in Koch & Poggio, 1983*b*). In other words, for a *current input* the depolarization at some location in the dendritic tree (for instance the soma) is the same, irrespectively of whether the synapse is on the spine or directly on the dendrite (Koch & Poggio, 1983*b*). This result is easy to understand without much calculation: since the membrane surface of the spine is minute ($\approx 1\ \mu m^2$), essentially no current flows through the spine membrane. Thus all the current injected in the spine head reaches the dendrite.

Synaptic inputs, however, consist of transient *conductance changes* to specific ions and cannot in general be approximated by currents. Synaptic inputs effectively open 'holes' in the membrane for ions with a reversal potential E measured with respect to the local resting potential. Restricting ourselves to steady state conductance changes g (for the full, transient case see Rall & Rinzel, 1974; Koch & Poggio, 1983*b* or Perkel, 1983), the change in somatic potential is given by

$$V_s=\frac{gK_{1s}E}{1+gK_{11}} \tag{3}$$

where K_{11} is the steady state spine input resistance. If g is small with respect to the spine input resistance, the term gK_{11} in the denominator can be neglected and the input can be approximated as current input gE. In the more general case, we make use of equation (1)

$$V_s=\frac{gK_{1s}E}{1+g(K_{22}+R_N)}. \tag{4}$$

We calculated the electrical properties of spines on the basis of measurements made on a Golgi-stained pyramidal cell, implementing the Butz & Cowan calculus in a computer program which determines the potential at location i if a transient conductance input is applied at location j. If one assumes $R_m=8000\ \Omega\ cm^2$, $C_m=1\ \mu F\ cm^{-2}$ and $R_i=70\ \Omega$ cm, the dendritic input resistance varies between 30 and 150 MΩ, depending on the location

within the apical tree. If the neck of the spine is assumed to be 1 μm long and 0·1 μm thick the neck resistance R_N is equal to 87 MΩ. Increasing the spine neck length and decreasing its diameter can increase the spine neck resistance up to 1000 MΩ. The actual size of the conductance change at a synapse is an open question, though a value between 10^{-8} and $10^{-6} S$ is not unreasonable. For the change in conductance underlying a quantal EPSP, Barrett & Crill (1974) give an estimate of $8\text{-}19.10^{-9}$ S in cat motoneurones and Turner (1984) a value of 2.10^{-9} S in hippocampal granule cells. Within this range of synaptic input amplitudes, the input cannot be considered as current but must be modeled as conductance change. Figure 1 shows the somatic depolarization induced by a synaptic conductance change of varying amplitude on a distal spine of our pyramidal cell as a function of the dimension of the spine neck. If the synaptic amplitude is above a critical value (in this case approximately given by $5 . 10^{-9} S$) the somatic weight of the spine depends strongly on its dimension. This strong dependence could be the biophysical mechanism underlying the short- and long-term storage of information (see Rall, 1974, 1978; Jack *et al.*, 1974; Crick, 1982).

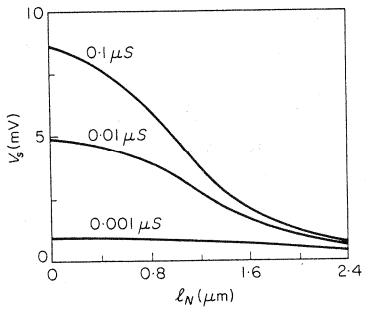

FIG. 1. The change in somatic potential for small, medium and large steady state synaptic conductance inputs on a dendritic spine of a cortical pyramidal cell with varying neck dimensions. The assumed reversal potential E is 80 mV relative to the resting potential. The spine neck dimensions were changed in such a way as to leave the total neck surface area constant and equal to 0·1 μm^2 (although this is not required). Plausible changes in neck length (from 0·8 to 1·6 μm) could alter the "weight" of the synapse by a factor of 3 (from Koch & Poggio 1983*b*).

A second interesting non-linear property of spines that depends on the input being a conductance change is the possibility of a very specific veto-like synaptic operation between excitatory and inhibitory inputs that can occur within the same spine, without inhibition affecting the ongoing electrical activity in the rest of the cell (see Fig. 7 in Koch & Poggio, 1983*b*). Inhibition would effectively only shunt excitation localized onto the same spine (for a similar suggestion see Diamond *et al.*, 1970).

REFERENCES

BARRETT, J. N. & CRILL, W. E. (1974). *J. Physiol.* **239,** 325.
BUTZ, E. G. & COWAN, J. D. (1974). *Biophys. J.* **14,** 661.
CRICK, F. (1982). *Trends Neurosci.* **5,** 44.
DIAMOND, J., GRAY, E. G. & YASARGIL, G. M. (1970). In: *Excitatory synaptic mechanisms.* (Andersen, P. & Jansen, J. eds). Oslo: Universitetsforlag.
GAGE, P. W. & MCBURNEY, R. N. (1972). *J. Physiol., Lond.* **226,** 79.
HORWITZ, B. (1983). *Soc. Neurosci. Abst.* **9,** 321.
JACK, J. J., NOBLE, D. & TSIEN, R. W. (1974). *Electric current flow in excitable cells.* Oxford: Clarendon Press.
KAWATO, M. & TSUKAHARA, N. (1983). *J. theor. Biol.* **103,** 507.
KOCH, C. & POGGIO, T. (1983*a*). *Trends Neurosci.* **6,** 80.
KOCH, C. & POGGIO, T. (1983*b*). *Proc. R. Soc. Lond., B* **218,** 455.
PERKEL, D. H. (1983). *J. Physiol., Paris* **78,** 695.
RALL, W. (1974). In: *Cellular Mechanisms subserving Changes in Neuronal Activity.* (Woody, C. D., Brown, K. A., Crow, Jr, T. J. & Knispel, J. D. eds.). Los Angeles: University of California.
RALL, W. (1978). In: *Studies in Neurophysiology.* (Porter, R. ed.). Cambridge: Cambridge University Press.
RALL, W. & RINZEL, J. (1974). *Biophys. J.* **14,** 759.
TURNER, D. A. (1984). *Biophys. J.* **46,** 85.