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# Is there a support vector machine hiding in the dentate gyrus?

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**Abstract**

The dentate gyrus has physiological and related behavioral properties suggesting that it implements functions within the hippocampus partially associated with sensory pattern recognition. A top-down dentate gyrus model is defined in terms of an idealized support vector machine pattern recognizer constructed from spiking neurons. The resulting construction offers parallels with dentate gyrus morphology and offers explanation of some of its unique properties, in particular, the mossy fiber pathway and its connection with CA3 pyramidal cells. Derived learning rules suggest properties of the mossy fibers that might be tested experimentally.


*Keywords:* Hippocampus; Dentate gyrus; Mossy fiber; Support vector machine; Pattern recognition

## 1. Introduction

Theories of the hippocampus have assigned a variety of roles to the dentate gyrus. Associative memory models of the hippocampus typically assign the dentate gyrus a role in creating orthogonal input patterns for CA3 so that associative memory saturation is avoided [6,10]. Lesion studies suggest that the dentate gyrus has a role in pattern recognition for spatial learning [4].

Explanations as to how the dentate gyrus might create orthogonal patterns for input to CA3 vary, but a common observation is that the number of granule cells in the dentate gyrus is much larger than the comparable layer of entorhinal cortex cells, allowing the dentate gyrus to generate a sparse representation of its input. Put in other terms, the output of the dentate gyrus has higher dimensionality than its input. This increase in

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dimensionality is a clue that the formalism of support vector machines (SVMs) may offer some explanation as to the underlying transformation performed in the dentate gyrus. The relative sparseness of connectivity between the dentate gyrus and CA3 is an additional clue suggesting applicability of a SVM model.

SVMs have been more completely described elsewhere [7] but are summarized here for completeness. Let $\langle w,x\rangle$ be the normal vector dot product of vectors $w$ and $x$ in Euclidean space. Consider the following class of linear decision functions:

$$f(x) = \mathrm{sgn}(\langle w,x\rangle + b) \in \{-1,1\}, \quad w \in \Re^N, \ b \in \Re. \tag{1}$$

Let the training vectors $x_1, x_2, \ldots,$ be vector-valued inputs and let $y_i$ be the corresponding desired decision function value for the $x_i$. An optimal decision function is found by solving the optimization problem

$$\text{maximize } W(\alpha) = \sum_i \alpha_i - \frac{1}{2}\sum_{i,j} y_i\alpha_i y_j\alpha_j\langle x_i,x_j\rangle,$$

$$\text{subject to } \sum_i y_i\alpha_i = 0, \ 0 \leqslant \alpha_i \leqslant C \text{ for some constant } C. \tag{2}$$

The value $C$ allows tolerance for when the patterns are not linearly separable. When $\alpha_i = 0$, the associated $x_i$ does not enter into the decision rule. Those $x_i$ for which $\alpha_i > 0$ are termed the support vectors.

SVMs generalize to non-linear decision functions by embedding the input data in a higher-dimensional space termed "feature space". Let $F$ represent feature space and let $\Phi : \Re^N \to F$ be the map of the input data into feature space. Mercer's theorem states that if a function $k$ is a positive definite kernel, there is an embedding of $F$ in a higher-dimensional space with dot products defined as

$$\langle \Phi(x_i), \Phi(x_j)\rangle = k(x_i,x_j). \tag{3}$$

The function $k$ is termed a Mercer kernel. Consider the case where $k$ is a function of the dot product $\langle x_1,x_2\rangle$, which with some abuse of notation we write as $k(x_1,x_2) = k(\langle x_1,x_2\rangle)$. In this case, a sufficient condition for $k$ to form a Mercer kernel is that the Taylor series expansion of $k$ as a function of $\langle x_1,x_2\rangle$ should have no negative coefficients [8]. This condition is also a necessary condition if the dot product is defined in an infinite-dimensional space.

An important consideration for SVMs is that most of the $\alpha_i$ become identically zero and thus do not need to be included in the final decision rule. Support vectors lie on the boundary of decision surface and are sparse with respect to the set of training vectors.

## 2. Constructing the SVM

### 2.1. Scope of the model

In describing an existence proof construction of an SVM from spiking neurons, it is convenient to use terms such as "perforant path", "granule cell" and "CA3 pyramidal"

metaphorically. The process is to first derive a model from mathematical constraints and then compare the result with known physiological properties of the hippocampus.

### 2.2. Coding of the perforant path input

Perforant path encoding is assumed to be in the form of a population burst. Let $x_1, x_2, \ldots$, be the various perforant path input spiking patterns as vectors where $x_i \in \{0,1\}^N$. For our purposes, each pattern is unique, that is, $i \neq j \Rightarrow x_i \neq x_j$. The index $i$ in this case does not imply temporal sequence.

### 2.3. Granule cell responses

To provide a response that can be solved analytically, the granule cell is assumed to have a linear $I$–$V$ response [2]. The synaptic response of cell $j$ to input pattern $x_i$ is determined by the dot product $\langle w_j, x_i \rangle$ where $w_j$ is the vector of synaptic weights. To meet the requirements of the SVM formulation, for a given granule cell, $w_j$ is fixed equal to a unique input pattern selected from among the training vectors.

Cell membrane potential follows the dynamic equation

$$C_{\mathrm{m}} \frac{\mathrm{d}V}{\mathrm{d}t} = g_{\mathrm{GC}}(V_{\mathrm{R}} - V) + X h_{\mathrm{GC}}(t), \tag{4}$$

where $V$ is the membrane potential, $C_{\mathrm{m}}$ is the membrane capacitance, $V_{\mathrm{R}}$ is the channel reversal potential, $g_{\mathrm{GC}}$ is the effective conductance of channels in the granule cell, $X = \langle w_j, x_i \rangle$ is the effective stimulation of the granule cell, and $h_{\mathrm{GC}}(t)$ is the time course of the current from a single synapse.

Let $\lambda = g_{\mathrm{GC}}/C_{\mathrm{m}}$ and let $V_0$ be the initial membrane voltage. We assume without loss of generality that the inputs are presented at time $t = 0$. Eq. (4) can then be solved explicitly:

$$V(t) = V_{\mathrm{R}} - \mathrm{e}^{-\lambda t}(V_{\mathrm{R}} - V_0) + \frac{\mathrm{e}^{-\lambda t}}{C_{\mathrm{m}}} X \int_0^t \mathrm{e}^{\lambda s} h_{\mathrm{GC}}(s)\, \mathrm{d}s. \tag{5}$$

Let $t_{\mathrm{f}}$ be the time to firing as determined by the time at which $V(t_{\mathrm{f}}) = V_{\mathrm{th}}$ where $V_{\mathrm{th}}$ is the firing threshold. Because $h(t)$ is assumed to be short, we further assume that $h_{\mathrm{GC}}(t)$ is negligible for all $t$ greater than some value less than $t_{\mathrm{f}}$, or more formally, that there is a $T_{\mathrm{H}} < t_{\mathrm{f}}$ such that $h_{\mathrm{GC}}(t) = 0$ for all $t > T_{\mathrm{H}}$.

Under this assumption, we can rewrite Eq. (5) as

$$V(t) = \mathrm{e}^{-\lambda t}(X\beta + V_0 - V_{\mathrm{R}}) + V_{\mathrm{R}}, \quad \beta = \frac{1}{C_{\mathrm{m}}} \int_0^{\infty} \mathrm{e}^{\lambda s} h_{\mathrm{GC}}(s)\, \mathrm{d}s, \quad t > T_{\mathrm{H}}. \tag{6}$$

From this we then solve for the time of firing as a change in firing time relative to some fixed time $T_{\mathrm{F}}$ where $t_{\mathrm{f}} = T_{\mathrm{F}}$ when $X = 0$.

$$\delta(X) = T_{\mathrm{F}} - t_{\mathrm{f}} = -\frac{1}{\lambda} \ln\left(1 - \frac{X\beta}{V_{\mathrm{R}} - V_0}\right), \tag{7}$$

where $\delta(X)$ is the change in firing time and under the condition that $V_{\mathrm{R}} > V_{\mathrm{th}} > V_0$.

To check for the dot-product Mercer kernel property, we examine the Taylor series expansion of $\delta(X)$ noting that

$$-\ln(1-x) = x + \frac{x^2}{2} + \frac{x^3}{3} + \frac{x^4}{4} + \cdots, \quad -1 < x < 1. \tag{8}$$

The Taylor series of $\delta(X)$ has all positive coefficients and $\delta(\langle w,x\rangle)$ is a Mercer kernel of the input data vector $x$ and the synaptic weight vector $w$.

### 2.4. CA3 pyramidal cell responses

For analytical purposes, a simple model of pyramidal cell response is also used. As before, we assume the existence of one combined ion channel conductance and reversal potential. The membrane potential is modeled by the following dynamic behavior:

$$C_{\mathrm{m}}\frac{\mathrm{d}V}{\mathrm{d}t} = g_{\mathrm{PC}}(V_{\mathrm{R}} - V) + \sum_i w_i h_{\mathrm{MF}}(t - t_i), \tag{11}$$

where $V$ is the pyramidal soma membrane potential, $C_{\mathrm{m}}$ is the membrane capacitance, $g_{\mathrm{PC}}$ is the net conductance of channels in the pyramidal cell, $V_{\mathrm{R}}$ is the reversal potential, $w_1, w_2, w_3$, etc. are synaptic weights for the different mossy fiber connections, $h_{\mathrm{MF}}$ is the time response of a mossy fiber connection in terms of the current delivered to the soma, and $t_1, t_2, t_3$, etc. are the times at which action potentials arrive at the various mossy fiber terminals.

We model the mossy fiber terminal as an exponential decay after the initial rise interval

$$h_{\mathrm{MF}}(t) = h_{\mathrm{N}} \exp(-\gamma(t - T_{\mathrm{N}})), \quad t \geqslant T_{\mathrm{N}}, \tag{12}$$

where $T_{\mathrm{N}}$ is a time after which the decay is exponential, $h_{\mathrm{N}}$ is a constant chosen to scale the current appropriately, and $\gamma$ the decay rate. We also have $h_{\mathrm{MF}}(t) = 0$ for $t < 0$.

Unlike the granule cell, we cannot solve explicitly for the firing time. However, we can assess whether the pyramidal cell would fire within a time interval by examining the voltage at a fixed time sufficiently long after all mossy fiber inputs would have arrived and compare that with a fixed threshold. Let $T_{\mathrm{B}}$ be the time at which we make our measurement, that is, we compare $V(T_{\mathrm{B}})$ with the threshold voltage $V_{\mathrm{th}}$, and let

$$J_i = \frac{\exp(-\rho T_{\mathrm{B}})}{C_{\mathrm{m}}} \int_0^{T_{\mathrm{B}}} \exp(\rho s) h_{\mathrm{MF}}(s - t_i)\,\mathrm{d}s,$$

$$\delta_i(x) = T_{\mathrm{F}} - t_i, \quad T_{\mathrm{F}} < T_{\mathrm{B}} - T_{\mathrm{N}}, \quad \rho = g_{\mathrm{PC}}/C_{\mathrm{m}}, \tag{13}$$

where $T_{\mathrm{F}}$ is the nominal time at which granule cells fire. An explicit solution for $V(t)$ can be found giving a decision function of the form

$$f(x) = \mathrm{sgn}\left(\sum_i w_i J_i - b\right), \quad b = V_{\mathrm{th}} - \exp(-\rho T_{\mathrm{B}})(V_0 - V_{\mathrm{R}}) - V_{\mathrm{R}}. \tag{14}$$

If the function $J_i$ is a Mercer kernel with respect to inputs afferent to the associated granule cell, then Eq. (14) is in the form of a decision function of a SVM with $w_i = y_i\alpha_i$.

Using the assumed form of $h_{\mathrm{MF}}$ and the granule cell response model, we have

$$J_i = \frac{1}{\rho - \gamma}\left(P\exp(-\gamma\delta_i) - Q\exp(-\rho\delta_i)\right) + R\exp(-\rho\delta_i), \tag{15}$$

where $P$, $Q$, and $R$ are constants:

$$P = C_{\mathrm{M}}^{-1}h_{\mathrm{N}}\exp(-\gamma T_{\mathrm{B}} + \gamma T_{\mathrm{F}} + \gamma T_{\mathrm{N}}),$$

$$Q = C_{\mathrm{M}}^{-1}h_{\mathrm{N}}\exp(-\rho T_{\mathrm{B}} + \rho T_{\mathrm{F}} + \rho T_{\mathrm{N}}),$$

$$R = C_{\mathrm{M}}^{-1}\exp(-\rho T_{\mathrm{B}} + \rho T_{\mathrm{F}})\int_0^{T_{\mathrm{N}}}\exp(\rho s)h(s)\,\mathrm{d}s. \tag{16}$$

We can now substitute the granule cell-derived values from Eq. (7). To determine under what conditions the result is a Mercer kernel, we examine the following individual exponential forms:

$$\exp(-\rho\delta_i) = (1 - aX_i)^{\rho/\lambda},$$

$$\exp(-\gamma\delta_i) = (1 - aX_i)^{\gamma/\lambda}, \tag{17}$$

where $a = \beta/(V_{\mathrm{R}} - V_0)$ and $V_{\mathrm{R}}$ and $V_0$ are taken from the granule cell model in Eq. (7).

Verification that $1-(1-aX)^r$ forms a dot-product Mercer kernel in $X$ for $0 < r < 1$ and $a > 0$ follows from expansion of the Taylor series. Note that because of our assumptions regarding $\beta$, we must have $aX < 1$. A dot-product Mercer kernel does not result when $a > 0$ and $r > 1$.

There are various conditions under which the $J_i$ could be Mercer kernels of the corresponding $X_i$:

(a) If $\lambda > \gamma > \rho$ and $\rho$ is sufficiently small that the series associated with $\gamma$ dominates in each Taylor series coefficient. An obvious way to achieve this is to set $\rho = 0$ suppressing leak currents. More realistically, if $\rho > 0$, possible values of $T_{\mathrm{N}}$ are bounded and close to zero.
(b) If $\lambda > \rho > \gamma$ and the series associated with $\rho$ dominates with non-negative coefficients.
(c) If $\gamma = \rho$ and $\rho$ is small, then $J_i$ is approximately a linear function of $\delta_i$.

Of these possibilities, $\gamma > \rho$ appears most consistent with pyramidal cell physiology and mossy fiber responses. Approximate values appear to be $\gamma = 0.1/\mathrm{ms}$ [3] and $\rho = 0.03/\mathrm{ms}$ [9]. Note that the value of $\lambda$ is increased by synaptic conductances, including inhibition, and is thus may be difficult to estimate from existing electrophysiological data. If $\gamma/\lambda = 1/2$ and the granule cells have normalized synaptic weights, the resulting kernel is equivalent to a Euclidean distance metric.

The signs of mossy fiber synaptic weights have not been considered thus far. While the Lagrangian multipliers for the optimization problem posed in Eq. (2) cannot be negative, the quantity $y_i\alpha_i$ must necessarily be negative for some $i$. Synaptic weights for support vectors can be made positive, that is, excitatory, through the following algebraic transformation of the decision function.

$$w_i = y_i\alpha_i + C \geqslant 0, \quad k_I(x) = C\sum_i k(x,x_i),$$

$$f(x) = \operatorname{sgn}\left(\sum_i w_i k(x,x_i) - k_I(x) - b\right). \tag{18}$$

A physiological correlate to Eq. (18) would be the requirement for feedforward inhibition of the CA3 pyramidal cell stimulated by granule cell firing. In addition, the time course of the inhibition should approximate that of $h_{\mathrm{MF}}$.

### 2.5. Learning rules

Learning corresponds with solving the optimization problem posed in Eq. (2). Platt [5] has formulated a sequential minimization algorithm (SMO), which makes optimal pairwise adjustments to the Lagrangian multipliers, or in our terms, mossy fiber synaptic weights. Given the two multiplier values $\alpha_1$ and $\alpha_2$, SMO makes adjustments of the form

$$\alpha_2^{\mathrm{new}} = \max\left(L, \min\left(H, \alpha_2^{\mathrm{old}} + \frac{y_2(E_2 - E_1)}{\eta}\right)\right),$$

$$\alpha_1^{\mathrm{new}} = \alpha_1^{\mathrm{old}} + y_1 y_2(\alpha_2^{\mathrm{old}} - \alpha_2^{\mathrm{new}}), \tag{19}$$

where

$$y_1 = y_2 \Rightarrow L = \max(0, \alpha_2^{\mathrm{old}} + \alpha_1^{\mathrm{old}} - C), \quad H = \min(C, \alpha_2^{\mathrm{old}} + \alpha_1^{\mathrm{old}})$$

$$y_1 \neq y_2 \Rightarrow L = \max(0, \alpha_2^{\mathrm{old}} - \alpha_1^{\mathrm{old}}), \quad H = \min(C, C + \alpha_2^{\mathrm{old}} - \alpha_1^{\mathrm{old}})$$

$$\eta = 2k(x_1,x_2) - k(x_1,x_1) - k(x_2,x_2), \quad E_i = f^{\mathrm{old}}(x_i) - y_i.$$

SMO also provides for adjusting the value of the bias term $b$ in the decision rule whenever one of the $\alpha_i$ is not at an extreme value.

It is doubtful that SMO can be exactly implemented within the constraints of neurological plausibility, even in an artificially defined network, but some degree of approximation may be possible at the risk of arriving at a suboptimal decision rule. In any case, the adjustment of weights is a form of competitive learning in that increasing one weight involves decreasing others to satisfy the constraint in Eq. (2).

If the maximal possible response by a granule cell, $k(x_i,x_i)$, is normalized to a constant $k_{\max}$, the value of $\eta$ can be determined from $k(x_1,x_2)$ alone and if individual component values of the vector encoded by the perforant path have known statistical properties, for example independence, then an estimate of $k(x_1,x_2)$ can be made based on the difference in granule cell firing times. In almost all cases, $\eta < 0$ and at least

the direction of change in synaptic weights can be determined even if the value of $\eta$ cannot, suggesting a learning rule based on relative order of granule cell firing in which MF terminals of earlier firing granule cells are facilitated at the expense of those of later firing granule cells.

Estimating a value for $E_i$ involves knowing the desired outcome of the decision function. This could be accomplished in a number of ways, but in particular through the self-organizing characteristics of CA3 itself. This stands in contrast to the alternative assumption that the dentate gyrus, in essence, establishes the initial organization of CA3 via "detonator" cells [6]. Any supervisory stimulus for MF terminal learning rules would need to be reflected in synaptic inputs separate from the mossy fiber connections. Modulation of MF terminal plasticity by perforant path activity [12] and CA3 associative connections [1] is consistent with the type of learning proposed here.

## 3. What the SVM construction implies in terms of hippocampus physiology

### 3.1. Dentate gyrus

Implementing the SVM construction requires that the dentate gyrus have a set of precisely timed mechanisms. One plausible cycle of events is: (1) a brief inhibition is used to set the granule cell to a known membrane potential, (2) the perforant path synaptic response is integrated in the granule cell, (3) fixed excitatory stimulation is provided to the granule cell such that small input could potentially result in firing in a time-dependent manner, and (4) at time $T_F$ in the cycle, squelching inhibition is applied to prevent late firing. A refinement of step 4 is to use lateral inhibition to suppress further firing once other granule cells have already fired and thus already determined the dominant support vectors. Such a refinement would also reduce the need for precise timing in the mossy fiber pathway. If the inhibition in step 1 is sufficiently punctate, then step 3 can occur earlier or be ongoing, reducing the requirements for precise timing of step 3 stimulation.

Because each granule cell implements a single support vector, learning patterns that require new support vectors would require either the use of previously dormant granule cells or else granule cell neurogenesis. Adding new support vectors via neurogenesis suggests that new granule cells should exhibit relatively greater synaptic plasticity than older cells, which is consistent with experimental findings [11]. Having a consistent $k_{max}$ would imply a consistent number of synaptic connections between a granule cell and the perforant pathway.

### 3.2. Mossy fiber pathway

A key requirement of the mossy fiber pathway is that it accurately preserves the relative timing of firing events in the granule cells. This implies that routing of mossy fibers afferent to a given CA3 pyramidal cell follows parallel paths. Outside this parallel routing, granule cells would ideally be arranged in a pattern to ensure consistency of

axon lengths. To avoid negative synaptic weight values for mossy fiber terminals, mossy fibers must also stimulate inhibitory interneurons that ultimately connect with CA3 pyramidal cells following a path parallel to the mossy fibers themselves.

### 3.3. Mossy fiber terminals

Mossy fiber terminals at CA3 pyramidal cells would necessarily be complex structures. The efficiency of neurotransmitter release must be very high since relatively few terminals would be active at any one time. Rise time in the synaptic current should initially be rapid ($T_N$ near 0) with a slower decay time. In addition, the mossy fiber terminals would have to implement complex learning rules. There is nothing in the construction to differentiate presynaptic and postsynaptic effects, but the existence of a complex presynaptic organelle is consistent with the assumptions made here.

A requirement in implementing a learning rule of the form described is that the mossy fiber terminals exchange information among themselves. This argues for a physically compact arrangement and the use of a variety of extra-cellular messengers. Neurotransmitter spillover could offer one source of exchanged information, but this alone may be insufficient to implement the learning rule needed. Use of perforant path and associational connections as input to the learning rule implies a relationship between change in postsynaptic membrane potentials (or some other retrograde messenger) and MF terminal plasticity. A possibility suggested by physiology but not considered in the construction here is that neurotransmitter release time constants may be adapted under the influence of a learning rule.

### 3.4. CA3 pyramidal cells

In general, it is hard to predict which functions would be associated with pyramidal cells and which with the mossy fiber terminals. Retrograde messengers generated in the pyramidal cell would be a reasonable assumption, but no definite messengers are identified. The pyramidal cell is a plausible site for maintaining a consistent number of glutamate receptor sites associated with mossy fiber inputs and thus maintaining a constant sum of total excitatory weight values.

Suppression of pyramidal cell leak currents, at least during the interval during which mossy fiber signals are presented, would enhance the quality of the generated kernel function. Otherwise, the accuracy of the decision function is compromised by leakage during the period over which inputs are integrated. The assumption of a fixed time window for pyramidal cell spiking would imply the existence of periodic inhibition to reset the state of the pyramidal cell as might be expected if gamma cycle inhibition is present.

## 4. Conclusion

An outline for creating a support vector machine pattern recognizer using spiking neurons has been defined, providing an existence proof that support vector machines

are at least not inconsistent with the constraints of neural physiology. Correspondences between the structures needed for a support vector recognizer and the novel properties of dentate gyrus and mossy fiber systems provide a rationale for these properties and suggest that there may be some merit to a functional role of the dentate gyrus as a component of a non-linear pattern recognition system. The complex MF terminal learning rules suggested here stand in contrast with an assumption of relative non-plasticity. Experimental tests should allow these possibilities to be discriminated empirically.

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