control gene. This is based on the observation that when marrow cells were tested for the presence of CA I by a single-cell immunofluorescence method (*12*), moderately high levels of fluorescence were detected in a number of unidentified cells (*13*). This finding suggests that a variant CA I may be synthesized in red cell precursor cells, but that it, or its messenger RNA, is degraded at some point during red cell maturation. It is possible that a similar mechanism is responsible for the human CA I deficiency.

The role of CA I in mature mammalian erythrocytes remains unclear. Not only may the levels of this isozyme be greatly suppressed as the result of a mutation, but CA I is also characteristically absent from peripheral red cells (although present in other tissues) of ruminants such as the ox (*14*) and the sheep (*15*) and felids such as the domestic cat (*16*). As yet, a similar reduction in the levels of CA II has not been reported in mammalian red cells. It would thus appear that CA II has a more important role than CA I in the mature red cell. It has even been suggested that CA I may not function in the hydration of CO_2 in mammalian red cells because concentrations of chloride and bicarbonate equivalent to those found in normal human red cells have been observed to greatly inhibit this reaction in vitro (*17*).

If the CA I variant reported here is found to be due to a defect in the structural gene for CA I, we propose that it be designated CA I Icaria.

It is worth noting that another rare genetic mutation, resulting in an elongated alpha globin chain and the formation of hemoglobin Icaria, has been reported in a family from the same sparsely populated Greek island (*18*).

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Calcium-Dependent Depression of a Late Outward Current in Snail Neurons

Abstract. *Neuron cell bodies of* Helix pomatia *were voltage-clamped with a 300-millisecond depolarizing test pulse (pulse II) delivered 1 second after a depolarizing conditioning pulse (pulse I). The outward current, measured 200 milliseconds after the onset of pulse II, exhibited a strong depression that was dependent on the presence of pulse I. The maximum depression of the pulse II outward current occurred when pulse I voltages lay in the range over which calcium influx is inferred to be greatest; depression of the pulse II current subsided as pulse I potentials approached the putative calcium equilibrium potential. In the presence of extracellular [ethylenebis(oxyethylenenitrilo)]tetraacetic acid (EGTA) or D600, the intensity of the pulse II current became largely independent of pulse I, approaching the values of maximal depression seen in normal Ringer solution. On the other hand, lowering the intracellular pH with extracellular carbon dioxide–carbonate buffer had no measurable effect on the outward currents. Other experiments showed that it is primarily the calcium-dependent, outward-current hump of the N-shaped late current-voltage curve that is depressed by presentation of the conditioning pulse. It was concluded that distinct from an early potassium-activating role, calcium entering during a depolarization leads, during a subsequent depolarization, to a depression of the calcium-activated potassium system that persists for many seconds.*

Depolarizations of nerve cell membranes are accompanied by a voltage- and time-dependent delayed flow of outward current carried by potassium ions. During normal nervous function this outward current plays an important role in repolarizing the membrane from a depolarized state. Potassium activation has been implicated in the repolarization of nerve, muscle, and cardiac action potentials and in the regulation of pacemaker potentials (*1–4*). In a number of membranes the delayed outward current shows a pronounced decay with time during maintained depolarization. In neurons of the snail, depression of outward current is also apparent in the second of two identical pulses separated by an interval of up to several seconds (*5–8*). Measurement of potassium activity during such experiments has shown that a major reason for such outward current depression is an actual drop in K^+ efflux during the second of two identical depolarizing pulses, although a facilitating partial inward current also appears to participate (*6, 8*).

The experiments reported here indicate that a large component of the depression of K^+ efflux depends on a prior influx of Ca^{2+}. The presence of a slow calcium system has been demonstrated by voltage clamp experiments on snail neurons for small depolarizations (*9*) and has been inferred for larger depolarizations in which the massive K^+ efflux obscures the smaller inward current (*8–11*). An inward calcium current during a depolarizing pulse is essential for activation of a component of the outward potassium current, $I_{K(Ca)}$ (*12, 13*). We present evidence here that distinct from the mediation of a calcium-coupled potassium current, there develops a depression or desensitization of the calcium-activated potassium conductance, $g_{K(Ca)}$, which lasts many seconds. This depression is causally related to a prior influx of Ca^{2+} and appears to play a role in the normal functioning of some excitable membranes.

The soma of a bursting pacemaker neuron in the right parietal ganglion of *Helix pomatia* was exposed, impaled with capillary electrodes filled with 3*M* KCl (2 to 10 megohms), and voltage-clamped, and the membrane current was measured locally from a portion of the

soma with an 80-μm patch pipette as described elsewhere (*9, 11, 14*). The isolated ganglion was bathed in a Ringer solution containing 80 m*M* NaCl, 4 m*M* KCl, 10 m*M* $CaCl_2$, 5 m*M* $MgCl_2$, 10 m*M* glucose, and 5 m*M* 4-(2-hydroxyethyl)-1-piperazineethanesulfonic acid (Hepes) (Servo, Inc., Heidelberg), at *p*H 7.8. The temperature was 21° ± 2°C. Holding potentials were at −50 or −55 mv as noted. The two pulses were separated by a 1-second interval, and the paired-pulse program was repeated every 30 seconds. Except for the experiment in Fig. 1A, all pulses were 300 msec long. Currents were measured 200 msec after pulse onset.

Depression of outward current during the test pulse (pulse II) occurred in response to a prior depolarizing pulse (pulse I) (Fig. 1). The outward current during pulse II exhibits a depression for pulse I depolarizations too small (< 20 mv) to produce a noticeable outward current on the present scale (Fig. 1A); but at these voltages a measurable net inward calcium current can be seen during current recording at higher gain (*9*). Depression of the pulse II current approached a maximum as pulse I approached 0 mv. The current remained maximally depressed up to pulse I potentials of 50 to 75 mv (Fig. 1B). In some preparations the normal bathing solution was replaced with a CO_2-saturated carbonate Ringer solution buffered to *p*H 7.1. No changes in the outward currents were seen, even though this treatment is reported to lower the intracellular *p*H (*15*).

With progressively more positive pulses the current-voltage (*I-V*) relations during pulse I generated an N-shaped curve, the most prominent feature of which is a region of negative resistance which typically extends from ~ 70 to ~ 150 mv (Fig. 1B). The N-shaped characteristic of the late *I-V* curve has been ascribed to the calcium-activated component of potassium conductance, $I_{K(Ca)}$, since outward current in this region declines as the voltage is increased toward levels commensurate with the calcium equilibrium potential, E_{Ca}, and since the N-shaped component diminishes or disappears if Ca^{2+} influx is reduced or blocked (*12, 13*). Within the range of pulse I potentials which generated the negative resistance region of the *I-V* curve, the depressing effect of the pulse on pulse II outward current became smaller. At the positive end of this range the current during pulse II approached the control value measured in the absence of pulse I (Fig. 1B). This suggests that a major component of depression of outward current during pulse II is not produced directly by depolarization of the membrane during pulse I, but is, instead, related to an influx of Ca^{2+} during pulse I (*16*).

Further evidence that outward current depression is related to prior Ca^{2+} entry is given in Fig. 1, C and D. When pulse I was presented at a fixed amplitude and an *I-V* curve was generated with pulse II, it was seen that the occurrence of pulse I was followed by a selective depression of the N-shaped calcium-dependent component (*12, 13*) of the *I-V* plot (Fig. 1C). The depression of the calcium-dependent component is again reduced if pulse I approaches E_{Ca} (Fig. 1D).

The involvement of calcium was further examined by interfering with Ca^{2+} influx. This was done by lowering the extracellular concentration of Ca^{2+} by buffering to $\leq 10^{-7}M$ with [ethylene-bis(oxyethylenenitrilo)]tetraacetic acid (EGTA) (*17*) (Fig. 2B) or by blocking a major portion of Ca^{2+} influx with D600 (*18*) (Fig. 2A). In both cases there was a marked reduction in pulse I outward current at potentials below 150 mv. This was accompanied by a significant overall flattening of the calcium-blocked pulse II current curve toward the minimum current (that is, maximal depression) seen in the control curves. The similarity between the depressed pulse II current level in the control curves and the flattened

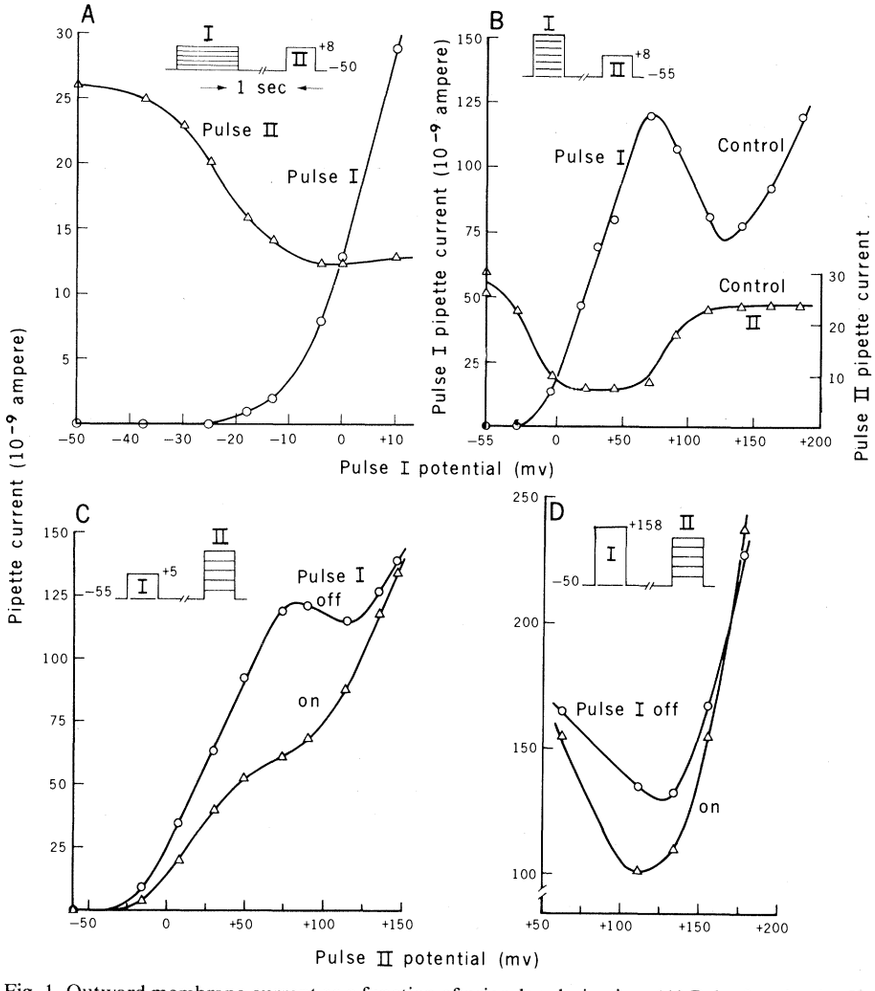

Fig. 1. Outward membrane current as a function of prior depolarization. (A) Pulse I and pulse II late currents plotted against pulse I potential. Currents were measured with a patch pipette 200 msec after onset of the respective pulses. Pulse I duration, 1 second; pulse II duration, 300 msec; holding potential, −50 mv; pulse II potential, 8 mv. The ordinate shows the feedback current required to clamp the opening of the 80-μm patch pipette at virtual ground. (B) Similar plot at lower current sensitivity from a different cell. Pulse II voltage was kept constant throughout while pulse I was varied in voltage, as shown on the abscissa. Note the different current scales for pulses I and II. (C) Pulse II current plotted against pulse II voltage with pulse I on and off as indicated. Pulse I went to 5 mv; the holding potential was −55 mv. (D) Same experiment but with pulse I amplitude selected to approach E_{Ca} in order to minimize Ca^{2+} influx during the conditioning pulse. Note the relatively small difference between pulse I on and pulse I off. (A to D) Different cells were used. Pulse intervals were 1 second throughout. Abscissas are labeled in absolute membrane potential.

pulse II current strength obtained under conditions of suppressed Ca^{2+} entry suggests that it is primarily $g_{K(Ca)}$ (*12*) that is depressed.

The residual outward current in the presence of D600 is presumed to consist primarily of the potential-activated potassium current, $I_{K(pot)}$, which is independent of Ca^{2+} influx (*12*) and is activated directly by the depolarization. Failure of the pulse II curve in Fig. 2A to flatten completely with D600 may have been due to incomplete elimination of Ca^{2+} influx by the D600.

The effectiveness of pulse I in depressing the outward current during pulse II is a function of the interval between the pulses I and II (Fig. 2C). Maximal depression of pulse II outward current did not develop with intervals of less than several hundred milliseconds between pulses. After maximal depression following an interval of the order of 1 second, depression diminished, going to one-half after about 4 seconds and becoming negligible after about 20 seconds. Except for the characteristic delay in the development of full depression, these kinetics agree qualitatively with those reported by Kostyuk *et al.* (*19*). However, they are not interpreted here as indicative of simple Hodgkin-Huxley kinetics (*20*), since the depression of the outward current is not independent of its activation.

The kinetics of the outward current during pulse II depends on the pulse interval (Fig. 2C, top). Two components can be seen: a rapid, steplike current (record *a*) seen at intervals that produce maximal depression, and a slowly developing component that is nearly absent at short intervals, and is seen at longer intervals superimposed on the rapidly rising component. The slow component has been identified as the calcium-activated, cobalt-sensitive (*12*) current $I_{K(Ca)}$ in separate experiments on cells injected with the calcium-indicator aequorin (*21*). This component becomes progressively larger during pulse II as the interval is increased (records *b* to *d*). In these cells, then, the major part of late outward current depression appears to be a reduction in the slow, calcium-activated component. The small drop in the point of inflection on the upstroke of the current trace at short intervals indicates that the fast component also undergoes some depression, but to a more limited extent in these cells than does $I_{K(Ca)}$.

The results presented here indicate that the intracellular Ca^{2+} accumulated during pulse I interferes with the activation of $I_{K(Ca)}$ during pulse II. That it is Ca^{2+} influx and not merely the pulse I depolarization is apparent from the failure of pulse I to depress the pulse II current when pulse I approaches E_{Ca}.

In experiments reported elsewhere (*12*, *22*) it was shown that pressure injection of $CaCl_2$ (and $CaCl_2$ plus EGTA) to increase cytoplasmic free Ca^{2+} causes a prolonged decrease in outward current under depolarizing voltage clamp. This effect resembles the delayed consequences of Ca^{2+} entry described here. Depressed outward current activation related to an increased intracellular Ca^{2+} concentration has also been noted in the ventral eye of *Limulus*. Illumination, which is known to result in the release of internally sequestered Ca^{2+} in the photoreceptor cell (*23*), is accompanied by a depression of the outward current recorded during slow depolarizing ramps imposed by voltage clamp (*24*). Depression of outward current in the *Limulus* eye was also seen after injection of Ca^{2+}, and after injection of Na^+, which is known to produce a rise in intracellular Ca^{2+} in these cells (*25*).

The depression of outward current reported here takes place concurrently with a calcium-mediated increase in potassium leakage conductance measured in the unclamped cell by simple injection

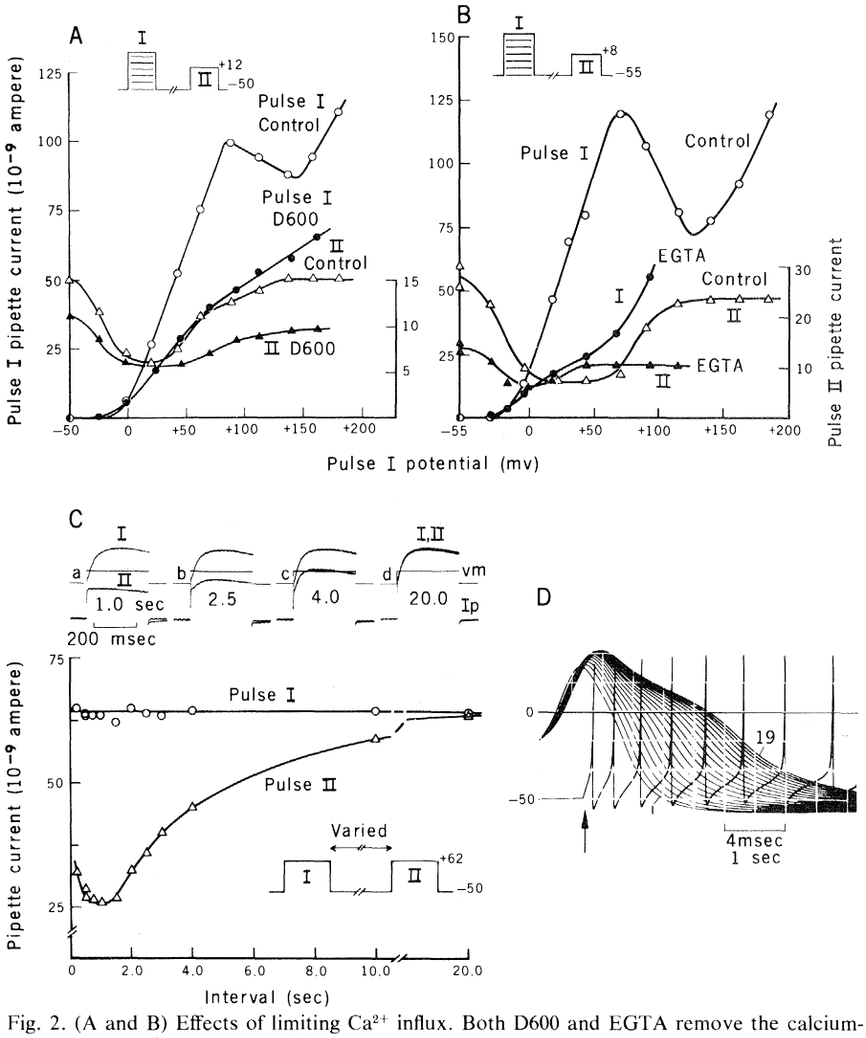

Fig. 2. (A and B) Effects of limiting Ca^{2+} influx. Both D600 and EGTA remove the calcium-activated hump from the pulse I *I-V* curves and flatten the pulse II curves toward the minimum of the pulse II control curve. The experimental protocol was as described for Fig. 1B. Pulse intervals were 1 second. (Open symbols) Control; (closed symbols) $10^{-4}M$ D600 in normal Ringer solution in (A) and $10^{-7}M$ Ca^{2+} in calcium-EGTA Ringer solution in (B). (C) Effect of pulse interval on pulse II current. At the top are representative records of superimposed pulses separated in time as indicated for each pair. The potential was stepped to 62 mv for 300 msec. Note that it is primarily the slow, calcium-activated component of the outward current trajectory (*12*) that is depressed during pulse II. The graph at the bottom shows the pipette current measured 200 msec after the start of a pulse plotted against the pulse interval. The holding potential was −50 mv; *vm*, membrane potential; *Ip*, pipette current. (D) Progressive change in upstroke, sizes of overshoot and undershoot, and repolarization kinetics during a train of impulses. Action potentials 1 and 19 are labeled in the fast sweep traces triggered by the upstroke of the action potential. The separate slow sweep shows the first eight impulses of another train. Both trains were evoked by releasing the cell (at the arrow) from the −50-mv holding potential. (A to D) Different cells were used.

of hyperpolarizing current pulses (not shown). Thus, although the accumulation of intracellular Ca^{2+} can produce an increase in resting potassium conductance (*26*), the results reported here indicate that at least one major component of potassium conductance, activated during depolarization, becomes depressed rather than activated by Ca^{2+} accumulation.

There are several possible means by which the calcium-dependent depression might occur. The simplest suggestion is that activation of $I_{K(Ca)}$ leaves it in a fatigued condition. This hypothesis seems unlikely, however, since the maximum depression takes time to develop (Fig. 2C) and since the effect is well developed after pulse I depolarizations too small to evoke a substantial outward current (Fig. 1A). The delayed depressing action of Ca^{2+} on $g_{K(Ca)}$ might also be a secondary consequence of a calcium-mediated change in intracellular *p*H. This is also unlikely because the currents and their depression remained unchanged in the presence of a CO_2-carbonate buffer, *p*H 7.1, reported to lower the intracellular *p*H of snail neurons (*15*). Another possibility is that internal calcium receptors [analogous to the acetylcholine receptors of the end-plate postsynaptic membrane (*27*)] desensitize on prolonged exposure to Ca^{2+}. The inward calcium current might initially activate $I_{K(Ca)}$, and accumulated intracellular Ca^{2+} might bind to a secondary site so as to interfere with potassium current. In any event, the calcium-dependent late depression of outward current cannot be due to a depression in Ca^{2+} influx and hence a reduced activation of $I_{K(Ca)}$, for Ca^{2+} entry has been shown to facilitate rather than depress with repetitive depolarization (*8, 28, 29*). Undoubtedly, such facilitation of a partial inward current contributes to the observed depression of net outward current. However, a true decrease in outward K^+ movement was previously shown to be correlated with depression of the pulse II net outward current (*6*).

Physiological correlates of the delayed depression of the outward current by intracellular Ca^{2+} accumulation can be seen in a train of action potentials (Fig. 2D). The increase in overshoot, the slowing of the repolarizaiton, and the positive shift in undershoot which occur during successive impulses are all consistent with a progressive reduction in activation of outward current. This reduction may be a correlate of the increment in intracellular Ca^{2+} resulting from Ca^{2+} influx known to occur during each action potential (*30, 31*). Prolongation of the action potential has been reported to occur in presynaptic branches with repetitive firing (*32*). Spike prolongation, due in part to a depression of the potassium system, may contribute to increased entry of Ca^{2+} and thus play a role in presynaptic facilitation.

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Inhibition of a Lymphocyte Membrane Enzyme by Δ^9-Tetrahydrocannabinol in vitro

Abstract. *Delta-9-tetrahydrocannabinol (Δ^9-THC) inhibited the activity of lysolecithin acyl transferase, a membrane-bound lymphocyte enzyme, at concentrations above 1.3 μM. Stimulation of acyl transferase activity by concanavalin A, an early response in lymphocyte activation, was entirely abolished in the presence of Δ^9-THC.*

Controversy exists as to the effect of Δ^9-tetrahydrocannabinol (Δ^9-THC) on the immune system. It has been reported that lymphocyte activation in vitro can be inhibited by Δ^9-THC concentrations as low as 1.6 μM (*1*) although this has been disputed (*2*). The lipophilic nature of Δ^9-THC suggests that any such action would be mediated at the level of the lymphocyte plasma membrane, and it has been shown that the early acceleration of phospholipid turnover during lymphocyte transformation can be blocked by Δ^9-THC (*3*). Other workers have suggested that later intracellular events in the transformation process, such as DNA synthesis, are the site of Δ^9-THC inhibition (*4*). It is clear from other studies (*5*) that perturbations of membrane structure by lipophilic substances may have a profound influence on intracellular events in lymphocyte transformation. We now describe the inhibition by low levels of Δ^9-THC of a membrane-bound lymphocyte enzyme that normally participates in the events of transformation. Lysolecithin acyl transferase (E.C. 2.3.1.23) catalyzes the formation of lecithin from lysolecithin and coenzyme A–activated fatty acids. As such, the enzyme has an important role in maintaining or altering membrane structure. Its level in T (thymus-dependent) lymphocytes is rapidly increased by mitogens such as concanavalin A (Con A) (*6*). We report here the complete