

extra −**synaptic ion currents in fish retinal ganglion cells Deactivation, recovery from inactivation, and modulation of**

A.T. Ishida

doi: 10.1098/rstb.2000.0665 Phil. Trans. R. Soc. Lond. B 2000 **355**, 1191-1194

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Deactivation, recovery from inactivation, **and modulation, recovery from inactivation,
and modulation of extra-synaptic ion currents** vation, recovery from inactivati
ulation of extra-synaptic ion cu
in fish retinal ganglion cells

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ion of Neurobiology, Physiology and Behavior, University of California, Davis, CA 95616-8519, USA (atishida@ucdavis.edu
As is shown magnificently by Heron Island's reef, the visual environment of many fishes includes vario As is shown magnificently by Heron Island's reef, the visual environment of many fishes includes various
light intensities, hues and shapes that can change on large and small scales in space and time. Several
articles in t As is shown magnificently by Heron Island's reef, the visual environment of many fishes includes various
light intensities, hues and shapes that can change on large and small scales in space and time. Several
articles in t light intensities, hues and shapes that can change on large and small scales in space and time. Several articles in this issue address why fishes are sensitive to some of these properties, and how fishes and other aquatic articles in this issue address why fishes are sensitive to some of these properties, and how fishes and other
aquatic species have acquired or fostered these sensitivities. This article discusses contributions of extra-
sy aquatic species have acquired or fostered these sensitivities. This artic
synaptic ion currents, in a specific population of neurons, to the det
appearance of certain visual stimuli and the disappearance of others. synaptic ion currents, in a specific population of neurons, to the detection of ambient light levels, the appearance of certain visual stimuli and the disappearance of others.
 Keywords: retinal ganglion cells; spikes; v

1. INTRODUCTION

1. INTRODUCTION
In fishes, as in other vertebrates, visually driven changes
in the synantic output of retinal photoreceptors and inter-I. INTRODUCTION
In fishes, as in other vertebrates, visually driven changes
in the synaptic output of retinal photoreceptors and inter-
neurons increase or decrease the probability that retinal In fishes, as in other vertebrates, visually driven changes
in the synaptic output of retinal photoreceptors and inter-
neurons increase or decrease the probability that retinal
ganglion cells—the retina's output neurons—p in the synaptic output of retinal photoreceptors and inter-
neurons increase or decrease the probability that retinal
ganglion cells—the retina's output neurons—produce
action potentials. The production and control of thes neurons increase or decrease the probability that retinal
ganglion cells—the retina's output neurons—produce
action potentials. The production and control of these
action potentials, also known as 'spikes', are of interest ganglion cells—the retina's output neurons—produce
action potentials. The production and control of these
action potentials, also known as 'spikes', are of interest, action potentials. The production and control of these
action potentials, also known as 'spikes', are of interest,
because ganglion cells use them to encode different kinds
of visual information, and because ganglion cells action potentials, also known as 'spikes', are of interest,
because ganglion cells use them to encode different kinds
of visual information, and because ganglion cells must do
so efficiently (see Levick 1986) because ganglion cells use them
of visual information, and becaus
so efficiently (see Levick 1986).
Inder what conditions do gan of visual information, and because ganglion cells must do
so efficiently (see Levick 1986).
Under what conditions do ganglion cells spike, and at a

cellular level, what electrophysiological properties facili-Under what conditions do ganglion cells spike, and at a
cellular level, what electrophysiological properties facili-
tate spiking in these cells? Some ganglion cells spike at
rates related to luminance, many ganglion cells cellular level, what electrophysiological properties facilitate spiking in these cells? Some ganglion cells spike at rates related to luminance, many ganglion cells spike even
in darkness, and certain ganglion cells respon tate spiking in these cells? Some ganglion cells spike at rates related to luminance, many ganglion cells spike even
in darkness, and certain ganglion cells respond to spatio-
temporal differences in specific attributes of rates related to luminance, many ganglion cells spike even
in darkness, and certain ganglion cells respond to spatio-
temporal differences in specific attributes of light within
the visual field. The latter are dependent (in darkness, and certain ganglion cells respond to spatio-
temporal differences in specific attributes of light within
the visual field. The latter are dependent (at least more so
than the former) on integration of excitat temporal differences in specific attributes of light within
the visual field. The latter are dependent (at least more so
than the former) on integration of excitatory and inhibi-
tory synaptic inputs, as this enables gangl the visual field. The latter are dependent (at least more so than the former) on integration of excitatory and inhibitory synaptic inputs, as this enables ganglion cells to than the former) on integration of excitatory and inhibitory synaptic inputs, as this enables ganglion cells to extract novel and useful information from signals formed at different positions across the distal retina and t tory synaptic inputs, as this enables ganglion cells to
extract novel and useful information from signals formed
at different positions across the distal retina, and to
combine information from presumantic cells that respo extract novel and useful information from signals formed
at different positions across the distal retina, and to
combine information from presynaptic cells that respond
differently to changes in stimulus properties (see Ba at different positions across the distal retina, and to larger and faster with light intensity (e.g. Diamond &
combine information from presynaptic cells that respond Copenhagen 1995). Both of these changes in synaptic
dif combine information from presynaptic cells that respond
differently to changes in stimulus properties (see Barlow
1959; Sakai & Naka 1995). Voltage-gated, extra-synaptic
ion currents (i.e. ion fluxes through transmembrane differently to changes in stimulus properties (see Barlow
1959; Sakai & Naka 1995). Voltage-gated, extra-synaptic
ion currents (i.e. ion fluxes through transmembrane pores
the opening and closing probabilities of which are 1959; Sakai & Naka 1995). Voltage-gated, extra-synaptic
ion currents (i.e. ion fluxes through transmembrane pores
the opening and closing probabilities of which are
controlled by the membrane potential of ganglion cells ion currents (i.e. ion fluxes through transmembrane pores
the opening and closing probabilities of which are
controlled by the membrane potential of ganglion cells
themselves as opposed to being necessarily controlled by the opening and closing probabilities of which are
controlled by the membrane potential of ganglion cells
themselves, as opposed to being necessarily controlled by
neurotransmitters released at synapses) enable ganglion controlled by the membrane potential of ganglion cells one finds that the rate of ganglion cell spiking increases
themselves, as opposed to being necessarily controlled by with light intensity. One might, therefore, ask ho themselves, as opposed to being necessarily controlled by neurotransmitters released at synapses) enable ganglion
cells to produce action potentials. These currents can
contribute to the spike output of ganglion cells in several
wave To begin with differences in the voltage sensi cells to produce action potentials. These currents can
contribute to the spike output of ganglion cells in several
ways. To begin with, differences in the voltage sensitivity,
kinetics availability and distribution of inwa contribute to the spike output of ganglion cells in several
ways. To begin with, differences in the voltage sensitivity,
kinetics, availability and distribution of inward and
outward currents (for the most part, cation inf ways. To begin with, differences in the voltage sensitivity, depolarizing current produce 'sustained' (i.e. continuous) kinetics, availability and distribution of inward and firing of spikes in retinal ganglion cells, and kinetics, availability and distribution of inward and
outward currents (for the most part, cation influxes and
effluxes, respectively) will help determine how readily a
cell will spike when it is initially depolarized and outward currents (for the most part, cation influxes and
effluxes, respectively) will help determine how readily a
cell will spike when it is initially depolarized, and when it
could spike during repeated or sustained depo effluxes, respectively) will help determine how readily a
cell will spike when it is initially depolarized, and when it
could spike during repeated or sustained depolarizations.

Since (i) these spikes occur at rates that are maintained Since (i) these spikes occur at rates that are maintained
under certain conditions, (ii) these rates do not increase
without limits, and (iii) these spikes occur at rates that Since (i) these spikes occur at rates that are maintained
under certain conditions, (ii) these rates do not increase
without limits, and (iii) these spikes occur at rates that
fade under certain conditions the spike-genera under certain conditions, (ii) these rates do not increase
without limits, and (iii) these spikes occur at rates that
fade under certain conditions, the spike-generating
mechanisms of ganglion cells also seem geared to sha without limits, and (iii) these spikes occur at rates that
fade under certain conditions, the spike-generating
mechanisms of ganglion cells also seem geared to shape fade under certain conditions, the spike-generating
mechanisms of ganglion cells also seem geared to shape
the dependence of spiking on stimulus intensity, to low-
nass filter some types of light-evoked responses, and to mechanisms of ganglion cells also seem geared to shape
the dependence of spiking on stimulus intensity, to low-
pass filter some types of light-evoked responses, and to
high-pass filter others. A brief summary of extra-syn the dependence of spiking on stimulus intensity, to low-
pass filter some types of light-evoked responses, and to
high-pass filter others. A brief summary of extra-synaptic
current properties that might contribute to these pass filter some types of light-evoked responses, and to high-pass filter others. A brief summary of extra-synaptic current properties that might contribute to these aspects high-pass filter others. A brief summary of extra-synaptic
current properties that might contribute to these aspects
of spiking in ganglion cells, others that probably do not,
and related questions that would be of interes current properties that might contribute to these aspects
of spiking in ganglion cells, others that probably do not,
and related questions that would be of interest to resolve,
follows follows.

2. LINEARIZATION OF INTENSITY-FREQUENCY RELATIONSHIPS

ELATION OF INTENSITE-FREQUENCE

RELATIONSHIPS

Bipolar cells deliver (or direct) excitatory synaptic

out to retinal ganglion cells. If this input is triggered by EXTRONSHIPS

Bipolar cells deliver (or direct) excitatory synaptic

input to retinal ganglion cells. If this input is triggered by

illuminating photorecentors with sten flashes of light one Bipolar cells deliver (or direct) excitatory synaptic
input to retinal ganglion cells. If this input is triggered by
illuminating photoreceptors with step flashes of light, one
finds that the current activated in ganglion input to retinal ganglion cells. If this input is triggered by illuminating photoreceptors with step flashes of light, one finds that the current activated in ganglion cells by illuminating photoreceptors with step flashes of light, one
finds that the current activated in ganglion cells by
neurotransmitter released from bipolar cells becomes
larger and faster with light intensity (e.g. Diamond & finds that the current activated in ganglion cells by
neurotransmitter released from bipolar cells becomes
larger and faster with light intensity (e.g. Diamond &
Conenhagen 1995). Both of these changes in synaptic neurotransmitter released from bipolar cells becomes
larger and faster with light intensity (e.g. Diamond &
Copenhagen 1995). Both of these changes in synaptic
input will depolarize ganglion cells to spike threshold larger and faster with light intensity (e.g. Diamond $\&$ input will depolarize ganglion cells to spike threshold input will depolarize ganglion cells to spike threshold
faster, and in turn, the time between flash onset and the
first ganglion cell spike will tend to become shorter. If the
same lights are presented for tens of millisec faster, and in turn, the time between flash onset and the first ganglion cell spike will tend to become shorter. If the same lights are presented for tens of milliseconds or more, one finds that the rate of ganglion cell s first ganglion cell spike will tend to become shorter. If the same lights are presented for tens of milliseconds or more, one finds that the rate of ganglion cell spiking increases with light intensity. One might therefore same lights are presented for tens of milliseconds or more,
one finds that the rate of ganglion cell spiking increases
with light intensity. One might, therefore, ask how gang-
lion cells respond to various amounts of depo one finds that the rate of ganglion cell spiking increases
with light intensity. One might, therefore, ask how gang-
lion cells respond to various amounts of depolarizing
current current. It has long been known that exogenous injections of
It has long been known that exogenous injections of
polarizing current produce 'sustained' (i.e. continuous)

current.
It has long been known that exogenous injections of
depolarizing current produce 'sustained' (i.e. continuous)
firing of spikes in retinal ganglion cells, and that the It has long been known that exogenous injections of
depolarizing current produce 'sustained' (i.e. continuous)
firing of spikes in retinal ganglion cells, and that the
frequency of these spikes increases linearly with the depolarizing current produce 'sustained' (i.e. continuous) firing of spikes in retinal ganglion cells, and that the frequency of these spikes increases linearly with the intensity of the depolarizing current even at low stimulus intensities (Baylor $\&$ Esttinlace 1979). This lin frequency of these spikes increases linearly with the intensity of the depolarizing current even at low stimulus intensities (Baylor & Fettiplace 1979). This linearity differs from the convex (and at low stimulus intensiti sity of the depolarizing current even at low stimulus
intensities (Baylor & Fettiplace 1979). This linearity
differs from the convex (and at low stimulus intensities,

discontinuous) relationship calculated from the voltage-
gated Na⁺ and K⁺ currents described by Hodgkin & are
Huxley (1952) This implies that retinal ganglion cells are discontinuous) relationship calculated from the voltagegated Na⁺ and K⁺ currents described by Hodgkin &
Huxley (1952). This implies that retinal ganglion cells are
equipped with different or additional types of voltage gated Na⁺ and K⁺ currents described by Hodgkin &
Huxley (1952). This implies that retinal ganglion cells are
equipped with different or additional types of voltage-
gated current Huxley (1952). T
equipped with
gated current.
Patch-clamp uipped with different or additional types of voltage-
ted current.
Patch-clamp studies have shown the latter to include
 n^{2+} currents. Ca^{2+} -activated K^+ currents, transient K^+

gated current.

Patch-clamp studies have shown the latter to include
 Ca^{2+} currents, Ca^{2+} -activated K^+ currents, transient K^+

currents and inwardly rectifying cation current (Ishida Patch-clamp studies have shown the latter to include Ca^{2+} currents, Ca^{2+} -activated K^+ currents, transient K^+ currents and inwardly rectifying cation current (Ishida 1995–1998). Some of these currents influence Ca^{2+} currents, Ca^{2+} -activated K^+ currents, transient K^+
currents and inwardly rectifying cation current (Ishida
1995, 1998). Some of these currents influence the oper-
ating range and spike frequency of gangli currents and inwardly rectifying cation current (Ishida
1995, 1998). Some of these currents influence the oper-
ating range and spike frequency of ganglion cells. For
example combinations of inward and outward currents ating range and spike frequency of ganglion cells. For example, combinations of inward and outward currents that are activated by sustained depolarizations limit the example, combinations of inward and outward currents
that are activated by sustained depolarizations limit the
range of membrane potentials at which retinal ganglion
cells dwell between spikes, and thus expand the range of that are activated by sustained depolarizations limit the range of membrane potentials at which retinal ganglion cells dwell between spikes, and thus expand the range of light intensities these cells can respond to (Diamo range of membrane potentials at which retinal ganglion
cells dwell between spikes, and thus expand the range of
light intensities these cells can respond to (Diamond &
Conenhagen 1995) Whether different ganglion cells use cells dwell between spikes, and thus expand the range of
light intensities these cells can respond to (Diamond &
Copenhagen 1995). Whether different ganglion cells use
the same subtypes of current to 'clamp' the interspike light intensities these cells can respond to (Diamond & Copenhagen 1995). Whether different ganglion cells use
the same subtypes of current to 'clamp' the interspike
voltage to these levels and what biophysical properties Copenhagen 1995). Whether different ganglion cells use
the same subtypes of current to 'clamp' the interspike
voltage to these levels, and what biophysical properties
enable these currents to serve this function remain to the same subtypes of current to 'clamp' the interspike
voltage to these levels, and what biophysical properties
enable these currents to serve this function, remain to be
measured measured. able these currents to serve this function, remain to be

easured. g

Modelling studies have inferred that current flow

tween a retinal ganglion cell's soma and dendrites and

Modelling studies have inferred that current flow
between a retinal ganglion cell's soma and dendrites, and
the charging and discharging of dendritic membrane
canacitance, belps linearize the denendence of spike between a retinal ganglion cell's soma and dendrites, and
the charging and discharging of dendritic membrane
capacitance, helps linearize the dependence of spike
frequency on the intensity of current injections into the charging and discharging of dendritic membrane
capacitance, helps linearize the dependence of spike
frequency on the intensity of current injections into
somata (Fohlmeister $\&$ Miller 1997) Those studies capacitance, helps linearize the dependence of spike
frequency on the intensity of current injections into
somata (Fohlmeister & Miller 1997). Those studies frequency on the intensity of current injections into
somata (Fohlmeister & Miller 1997). Those studies
showed that calculated current–frequency relationships
resemble measured ones if resting membrane conductance somata (Fohlmeister & Miller 1997). Those studies
showed that calculated current–frequency relationships
resemble measured ones if resting membrane conductance
is negligibly small, or very small, and constant, and if showed that calculated current–frequency relationships
resemble measured ones if resting membrane conductance
is negligibly small, or very small and constant, and if
voltage-gated conductances are also small at membrane resemble measured ones if resting membrane conductance
is negligibly small, or very small and constant, and if
voltage-gated conductances are also small at membrane is negligibly small, or very small and constant, and if
voltage-gated conductances are also small at membrane
potentials below spike threshold. While whole-cell
conductances have been generally found to be small in voltage-gated conductances are also small at membrane
potentials below spike threshold. While whole-cell
conductances have been generally found to be small in
ganglion cells at and near resting potential (see Isbida potentials below spike threshold. While whole-cell
conductances have been generally found to be small in
ganglion cells at and near resting potential (see Ishida
1995–1998), a resting permeability to Cl^- ions has conductances have been generally found to be small in ganglion cells at and near resting potential (see Ishida 1995, 1998), a resting permeability to Cl⁻ ions has recently been found to be modulated by a zinc-dependent 1995, 1998), a resting permeability to Cl^- ions has 1995, 1998), a resting permeability to Cl^- ions has
recently been found to be modulated by a zinc-dependent
protein kinase C activity in fish retinal ganglion cells
(Tabata & Ishida 1999) The presence of this conductance recently been found to be modulated by a zinc-dependent
protein kinase C activity in fish retinal ganglion cells
(Tabata & Ishida 1999). The presence of this conductance
in different compartments of ganglion cells, and the protein kinase C activity in fish retinal ganglion cells
(Tabata & Ishida 1999). The presence of this conductance
in different compartments of ganglion cells, and the
extent that changes in this conductance affect spiking (Tabata & Ishida 1999). The presence of this conductance
in different compartments of ganglion cells, and the
extent that changes in this conductance affect spiking,
remain to be examined in different compartment
extent that changes in t
remain to be examined.
Toxins that block Ca^{2+} tent that changes in this conductance affect spiking,
main to be examined.
Toxins that block Ca^{2+} -activated K^+ currents increase
ike firing rate in retinal ganglion cells (Foblmeister &

Toxins that block Ca^{2+} -activated K^+ currents increase
spike firing rate in retinal ganglion cells (Fohlmeister &
Miller 1997; Wang *et al.* 1998). However, spike firing can
be slowed by several agents that reduce vo spike firing rate in retinal ganglion cells (Fohlmeister & Miller 1997; Wang *et al.* 1998). However, spike firing can
be slowed by several agents that reduce voltage-gated
 Ca^{2+} currents including D, and Ga^{AA} Miller 1997; Wang *et al.* 1998). However, spike firing can
be slowed by several agents that reduce voltage-gated
 Ca^{2+} currents, including D_1 and $GABA_B$ receptor
agonists Co^{2+} Cd^{2+} and the *Conus* toxins ω -M be slowed by several agents that reduce voltage-gated Ca^{2+} currents, including D_1 and $GABA_B$ receptor agonists, Co^{2+} , Cd^{2+} and the *Conus* toxins ω -MVIIC and ω -GVIA (Liu & Lasater 1994; Zhang *et al* 1997; Ca^{2+} currents, including D_1 and $GABA_B$ receptor agonists, Co^{2+} , Cd^{2+} and the *Conus* toxins ω -MVIIC and ω -GVIA (Liu & Lasater 1994; Zhang *et al.* 1997; Rothe *et* al , 1999). These results are not easily agonists, Co^{2+} , Cd^{2+} and the *Conus* toxins ω -MVIIC and ω -GVIA (Liu & Lasater 1994; Zhang *et al.* 1997; Rothe *et al.* 1999). These results are not easily reconciled at this ω -GVIA (Liu & Lasater 1994; Zhang *et al.* 1997; Rothe *et al.* 1999). These results are not easily reconciled at this time, in part because the cytoplasmic Ca^{2+} levels during the recordings cited here, and the Ca^{2 al. 1999). These results are not easily reconciled at this
time, in part because the cytoplasmic Ca^{2+} levels during
the recordings cited here, and the Ca^{2+} -sensitivity of K^+
currents in retinal ganglion cells, ar time, in part because the cytoplasmic Ca^{2+} levels during
the recordings cited here, and the Ca^{2+} -sensitivity of K^+
currents in retinal ganglion cells, are not known in detail. **absolute the currents in retinal ganglion cells, are not known in detail.**
3. LOW-PASS FILTERING

Retinal ganglion cells can fire action potentials as **Retinal ganglion cells can fire action potentials as

rapidly as** 250 s^{-1} **, particularly during transient flurries of

spikes. Sustained firing rates have generally been found** Retinal ganglion cells can fire action potentials as
rapidly as $250 s^{-1}$, particularly during transient flurries of
spikes. Sustained firing rates have generally been found
not to exceed $100 s^{-1}$. Several voltage-gated rapidly as $250 s^{-1}$, particularly during transient flurries of spikes. Sustained firing rates have generally been found not to exceed $100 s^{-1}$. Several voltage-gated current properties could limit the maximum spike freq spikes. Sustained firing rates have generally been found
not to exceed $100 s^{-1}$. Several voltage-gated current
properties could limit the maximum spike frequencies in
these cells not to exceed 100 s^{-1} . Several voltage-gated current properties could limit the maximum spike frequencies in these cells. operties could limit the maximum spike frequencies in

see cells.

One is the amount of Na⁺ current that can be activated

or single spikes. We have found that single spikes reduce

these cells.

One is the amount of Na^+ current that can be activated

after single spikes. We have found that single spikes reduce

the amount of Na^+ current that can be activated in One is the amount of Na^+ current that can be activated
after single spikes. We have found that single spikes reduce
the amount of Na^+ current that can be activated in the amount of Na^+ current that can be activated in *Phil. Trans. R. Soc. Lond.* B (2000)

ganglion cells by at least 75% (Hidaka & Ishida 1998), ganglion cells by at least 75% (Hidaka & Ishida 1998),
and that most of the Na⁺ current that can be activated
recovers from this 'inactivation' along an exponential ganglion cells by at least 75% (Hidaka & Ishida 1998),
and that most of the Na⁺ current that can be activated
recovers from this 'inactivation' along an exponential
time-course ($\tau = 3 \text{ ms}$). To a first approximation t recovers from this 'inactivation' along an exponential time-course ($\tau = 3$ ms). To a first approximation, this fast recovers from this 'inactivation' along an exponential
time-course ($\tau = 3$ ms). To a first approximation, this fast
rate of recovery from inactivation should not prevent
ganglion cells from generating spikes as often as time-course ($\tau = 3$ ms). To a first approximation, this fast
rate of recovery from inactivation should not prevent
ganglion cells from generating spikes as often as 100 Hz.
We found similar rates of recovery in all of rate of recovery from inactivation should not prevent
ganglion cells from generating spikes as often as 100 Hz .
We found similar rates of recovery in all of the cells we
recorded from suggesting that the Na^+ cur ganglion cells from generating spikes as often as 100 Hz .
We found similar rates of recovery in all of the cells we
recorded from, suggesting that the Na^+ current in all
retinal ganglion cells can support repeat We found similar rates of recovery in all of the cells we
recorded from, suggesting that the $Na⁺$ current in all
retinal ganglion cells can support repeated firing of
spikes and that it can do so at similar maximum r recorded from, suggesting that the Na^+ current in a
retinal ganglion cells can support repeated firing ϵ
spikes, and that it can do so at similar maximum rates.
We have also found that a fraction of the Na^+ currenc spikes, and that it can do so at similar maximum rates.

between a retinal ganglion cell's soma and dendrites, and We have also found that a fraction of the $Na⁺$ current in the same ganglion cells recovers more slowly from We have also found that a fraction of the Na⁺ current
in the same ganglion cells recovers more slowly from
inactivation (Hidaka & Ishida 1998). We do not yet know
if multiple spikes chappe the probability that Na⁺ in the same ganglion cells recovers more slowly from
inactivation (Hidaka & Ishida 1998). We do not yet know
if multiple spikes change the probability that $Na⁺$
channels recover more slowly from inactivation. In othe inactivation (Hidaka & Ishida 1998). We do not yet know
if multiple spikes change the probability that Na^+
channels recover more slowly from inactivation. In other
studies Na^+ current amplitudes have been measured aft if multiple spikes change the probability that $Na⁺$ channels recover more slowly from inactivation. In other studies, $Na⁺$ current amplitudes have been measured after terminating large-amplitude step-wise depola channels recover more slowly from inactivation. In other studies, $Na⁺$ current amplitudes have been measured after terminating large-amplitude, step-wise depolarizations of studies, Na^+ current amplitudes have been measured after varying duration. One study reported that $Na⁺$ current terminating large-amplitude, step-wise depolarizations of
varying duration. One study reported that Na^+ current
recovers rapidly from inactivation in some cat retinal
ganglion cells and slowly in others (Kaneda & Kaneko varying duration. One study reported that Na^+ current
recovers rapidly from inactivation in some cat retinal
ganglion cells, and slowly in others (Kaneda & Kaneko
1991) Because Na^+ channel isoforms differ in this resp recovers rapidly from inactivation in some cat retinal ganglion cells, and slowly in others (Kaneda & Kaneko 1991). Because Na⁺ channel isoforms differ in this respect $(e \, \sigma \quad \text{Smith} \quad & \text{Goldin 1998})$ one might infer that (e.g. Smith & Goldin 1998), one might infer that 1991). Because Na⁺ channel isoforms differ in this respect (e.g. Smith & Goldin 1998), one might infer that functionally different ganglion cells are equipped with structurally different Na⁺ channels. This possibility (e.g. Smith & Goldin 1998), one might infer that
functionally different ganglion cells are equipped with
structurally different Na^+ channels. This possibility is
raised independently by the presence of mRNA for functionally different ganglion cells are equipped with
structurally different Na^+ channels. This possibility is
raised independently by the presence of mRNA for
multiple Na^+ channel types in ganglion cells (Fiell *et* structurally different Na^+ channels. This possibility is
raised independently by the presence of mRNA for
multiple Na^+ channel types in ganglion cells (Fjell *et al.*
1997). However, the recovery of Na^+ current from f mRNA for
lls (Fjell *et al.*
current from
lult rat retinal multiple Na^+ channel types in ganglion cells (Fjell *et al.* 1997). However, the recovery of Na^+ current from inactivation was found to be rapid in all adult rat retinal ganglion cells and it was not exclusively slow 1997). However, the recovery of Na^+ current from inactivation was found to be rapid in all adult rat retinal ganglion cells, and it was not exclusively slow in any case (Wang *et al.* 1997). Our results agree with those inactivation was found to be rapid in all adult rat retinal ganglion cells, and it was not exclusively slow in any case (Wang *et al.* 1997). Our results agree with those obtained ganglion cells, and it was not exclusively slow in any case
(Wang *et al.* 1997). Our results agree with those obtained
from this latter preparation, but we do not exclude the
possibility that slow recovery from inactivati (Wang *et al.* 1997). Our results agree with those obtained from this latter preparation, but we do not exclude the possibility that slow recovery from inactivation shapes ganglion cell light responses from this latter preparation,
possibility that slow recovery
ganglion cell light responses.
In many cells multiple s possibility that slow recovery from inactivation shapes
ganglion cell light responses.

remain to be examined.

Toxins that block Ca^{2+} -activated K^+ currents increase potentials that favour recovery from inactivation.

spike firing rate in retinal ganglion cells (Fohlmeister & However, fast spike firing ganglion cell lig
In many ce
outward K^+ cu
potentials that In many cells, multiple spike firing is fostered by outward K^+ currents that begin to activate at membrane potentials that are more positive than those that activate In many cells, multiple spike firing is fostered by
outward K^+ currents that begin to activate at membrane
potentials that are more positive than those that activate
inward currents. Because these 'high-threshold' outw inward currents. Because these `high-threshold' outward potentials that are more positive than those that activate
inward currents. Because these 'high-threshold' outward
currents accelerate spike repolarization, these reduce the
time that N_a^+ channels dwell at membrane pot inward currents. Because these 'high-threshold' outward
currents accelerate spike repolarization, these reduce the
time that Na^+ channels dwell at membrane potentials
that inactivate them, and also returns them to membr currents accelerate spike repolarization, these reduce the
time that Na^+ channels dwell at membrane potentials
that inactivate them, and also returns them to membrane
potentials that favour recovery from inactivation time that Na^+ channels dwell at membrane potentials that inactivate them, and also returns them to membrane potentials that favour recovery from inactivation. that inactivate them, and also returns them to membrane
potentials that favour recovery from inactivation.
However, fast spike firing would also require rapid
deactivation of outward currents to minimize the potentials that favour recovery from inactivation.
However, fast spike firing would also require rapid
deactivation of outward currents, to minimize the
amplitude and duration of the hyperpolarization However, fast spike firing would also require rapid
deactivation of outward currents, to minimize the
amplitude and duration of the hyperpolarization
produced by the K^+ current and to maximize the net deactivation of outward currents, to minimize the amplitude and duration of the hyperpolarization produced by the K^+ current, and to maximize the net influx of positive charge due to inward currents K^+ amplitude and duration of the hyperpolarization
produced by the K^+ current, and to maximize the net
influx of positive charge due to inward currents. K^+
current properties that foster repetitive spiking have been produced by the K^+ current, and to maximize the net
influx of positive charge due to inward currents. K^+
current properties that foster repetitive spiking have been
investigated by modelling (Foblmeister & Miller 19 influx of positive charge due to inward currents. K^+
current properties that foster repetitive spiking have been
investigated by modelling (Fohlmeister & Miller 1997),
and it will be of interest to compare the kinetics current properties that foster repetitive spiking have been
investigated by modelling (Fohlmeister & Miller 1997),
and it will be of interest to compare the kinetics and
voltage sensitivities of these against measured valu and it will be of interest to compare the kinetics and

In some neurons, a transient outward K^+ current voltage sensitivities of these against measured values.
In some neurons, a transient outward K^+ current
reduces excitability because it is 'low threshold', i.e.
because it begins to activate at more negative membrane In some neurons, a transient outward K^+ current
reduces excitability because it is 'low threshold', i.e.
because it begins to activate at more negative membrane
potentials than N_a^+ currents do This effect requires t reduces excitability because it is 'low threshold', i.e.
because it begins to activate at more negative membrane
potentials than Na^+ currents do. This effect requires that
the outward current be 'primed' for activation because it begins to activate at more negative membrane
potentials than Na^+ currents do. This effect requires that
the outward current be 'primed' for activation (i.e. allowed to recover from inactivation) at suitably negative the outward current be 'primed' for activation (i.e.
allowed to recover from inactivation) at suitably negative
membrane potentials. If this happened after each spike of
a sequence of spikes, then this type of current woul allowed to recover from inactivation) at suitably negative
membrane potentials. If this happened after each spike of
a sequence of spikes, then this type of current would
reduce spike firing rate, and would also tend to li membrane potentials. If this happened after each spike of
a sequence of spikes, then this type of current would
reduce spike firing rate, and would also tend to linearize
the intensity frequency relationship alluded to ab a sequence of spikes, then this type of current would
reduce spike firing rate, and would also tend to linearize
the intensity–frequency relationship alluded to above (\S 2) reduce spike firing rate, and would also tend to linearize
the intensity–frequency relationship alluded to above (§ 2)
(Connor *et al.* 1977). However, this mechanism may not
onerate in retinal ganglion cells, because the the intensity–frequency relationship alluded to above $(\S 2)$
(Connor *et al.* 1977). However, this mechanism may not
operate in retinal ganglion cells, because the activation
threshold of the transient outward current ha (Connor *et al.* 1977). However, this mechanism may not operate in retinal ganglion cells, because the activation threshold of the transient outward current has generally

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not been found to be more negative than that of the Na⁺ current in these cells (see Ishida 1995).

4. HIGH-PASS FILTERING

Retinal ganglion cells spike transiently in response to various light stimuli. Some of these stimuli depolarize ganglion cells, so that the spike burst occurs at the various light stimuli. Some of these stimuli depolarize
ganglion cells, so that the spike burst occurs at the
appearance of these stimuli. Other stimuli hyperpolarize
ganglion cells, and the spike burst occurs at the ganglion cells, so that the spike burst occurs at the
appearance of these stimuli. Other stimuli hyperpolarize
ganglion cells, and the spike burst occurs at the
disappearance of these stimuli. Do these light responses appearance of these stimuli. Other stimuli hyperpolarize
ganglion cells, and the spike burst occurs at the
disappearance of these stimuli. Do these light responses
reflect transient synaptic inputs transient responses to ganglion cells, and the spike burst occurs at the
disappearance of these stimuli. Do these light responses
reflect transient synaptic inputs, transient responses to
that input excitation abbreviated by inhibition or transi disappearance of these stimuli. Do these light responses
reflect transient synaptic inputs, transient responses to
that input, excitation abbreviated by inhibition, or transi-
ence in the spiking mechanism of ganglion cell reflect transient synaptic inputs, transient responses to
that input, excitation abbreviated by inhibition, or transi-
ence in the spiking mechanism of ganglion cells? Two
observations imply that under certain conditions a that input, excitation abbreviated by inhibition, or transience in the spiking mechanism of ganglion cells? Two observations imply that, under certain conditions and to some extent, ganglion cells introduce transience into ence in the spiking mechanism of ganglion cells? Two observations imply that, under certain conditions and to
some extent, ganglion cells introduce transience into their
own spike volleys. First, more than one configuration of
light and dark can elicit transient spike firing some extent, ganglion cells introduce transience into their
own spike volleys. First, more than one configuration of
light and dark can elicit transient spike firing, and thus
spike bursts do not seem to require the activa own spike volleys. First, more than one configuration of
light and dark can elicit transient spike firing, and thus
spike bursts do not seem to require the activation of
particular synaptic pathways. Second a transient flu light and dark can elicit transient spike firing, and thus
spike bursts do not seem to require the activation of
particular synaptic pathways. Second, a transient flurry
of spikes can be recorded in ganglion cells after th spike bursts do not seem to require the activation of particular synaptic pathways. Second, a transient flurry
of spikes can be recorded in ganglion cells after the termi-
nation of exogenous injections of hyperpolarizing particular synaptic pathways. Second, a transient flurry of spikes can be recorded in ganglion cells after the termination of exogenous injections of hyperpolarizing current (see Tabata & Ishida 1996). of spikes can be recorded in g
nation of exogenous injectior
(see Tabata & Ishida 1996).
What voltage-gated curre tion of exogenous injections of hyperpolarizing current
e Tabata & Ishida 1996).
What voltage-gated current properties might render
nglion cell responses transient? One might be the rate

(see Tabata & Ishida 1996).
What voltage-gated current properties might render
ganglion cell responses transient? One might be the rate
at which N_a^+ current recovers from inactivation at the What voltage-
ganglion cell resp
at which Na^+ cu
membrane potent What voltage-gated current properties might render
ganglion cell responses transient? One might be the rate
at which Na^+ current recovers from inactivation at the ganglion cell responses transient? One might be the rate
at which $Na⁺$ current recovers from inactivation at the
membrane potential that ganglion cells repolarize to after
a spike (or a spike burst). This rate could at which Na⁺ current recovers from inactivation at the
membrane potential that ganglion cells repolarize to after
a spike (or a spike burst). This rate could be finite but so
slow that spikes would occur just after the o membrane potential that ganglion cells repolarize to after
a spike (or a spike burst). This rate could be finite but so
slow that spikes would occur just after the onset of a
depolarizing stimulus, but would not re-occur f a spike (or a spike burst). This rate could be finite but so slow that spikes would occur just after the onset of a depolarizing stimulus, but would not re-occur for the slow that spikes would occur just after the onset of a
depolarizing stimulus, but would not re-occur for the
remainder of a brief stimulus presentation (Kaneda & ef
Kaneko 1991) Alternatively this rate could be so slow depolarizing stimulus, but would not re-occur for the
remainder of a brief stimulus presentation (Kaneda & e
Kaneko 1991). Alternatively, this rate could be so slow
that ganglion cells would spike after the onset of a depo remainder of a brief stimulus presentation (Kaneda & Kaneko 1991). Alternatively, this rate could be so slow
that ganglion cells would spike after the onset of a depo-
larizing stimulus, but could not spike again regardles Kaneko 1991). Alternatively, this rate could be so slow that ganglion cells would spike after the onset of a depolarizing stimulus, but could not spike again regardless of the duration of a maintained stimulus, as proposed that ganglion cells would spike after the onset of a depolarizing stimulus, but could not spike again regardless of
the duration of a maintained stimulus, as proposed for
transient retinal amacrine cells (Barnes & Werblin larizing stimulus, but could not spike again regardless of
the duration of a maintained stimulus, as proposed for
transient retinal amacrine cells (Barnes & Werblin 1986).
However, these would not explain the observation t transient retinal amacrine cells (Barnes & Werblin 1986).
However, these would not explain the observation that transient retinal amacrine cells (Barnes & Werblin 1986).
However, these would not explain the observation that
exogenous current injections elicit sustained spiking in
ganglion cells even if light elicits transient spikin However, these would not explain the observation that exogenous current injections elicit sustained spiking in the ganglion cells even if light elicits transient spiking in the same cells (Baylor & Fettiplace 1979; see al exogenous current injections elicit sustained spiking in cu
ganglion cells even if light elicits transient spiking in the
same cells (Baylor & Fettiplace 1979; see also Diamond & of
Conenhagen 1995: Wang et al. 1997) ganglion cells even if light elicits transient spiking in the same cells (Baylor & Fettiplace 1979; see also Diamond & Copenhagen 1995; Wang *et al.* 1997). me cells (Baylor & Fettiplace 1979; see also Diamond &
ppenhagen 1995; Wang *et al.* 1997).
A second current proposed as the basis for transient
iking during depolarizing light stimuli is the slowly

Copenhagen 1995; Wang *et al.* 1997).
A second current proposed as the basis for transient
spiking during depolarizing light stimuli is the slowly
inactivating K^+ current termed 'L' (Inkasiewicz & A second current proposed as the basis for transient se

spiking during depolarizing light stimuli is the slowly sp

inactivating K^+ current termed ${}^{t}I_{B}$ (Lukasiewicz & T)

Werblin 1988) This possibility is attrac spiking during depolarizing light stimuli is the slowly
inactivating K^+ current termed ${}^{t}I_B^{\prime}$ (Lukasiewicz &
Werblin 1988). This possibility is attractive particularly inactivating K^+ current termed I_B ' (Lukasiewicz & These include the recovery of Na^+ and T-type Ca^{2+}
Werblin 1988). This possibility is attractive particularly currents from inactivation, and the recruitment of Werblin 1988). This possibility is attractive particularly
because this current has been found in fish, amphibian
and mammalian retinal ganglion cells (see Ishida 1995;
Tabata & Ishida 1999). This possibility has been diff because this current has been found in fish, amphibian
and mammalian retinal ganglion cells (see Ishida 1995;
Tabata & Ishida 1999). This possibility has been diffi-
cult to test, however, because a pharmacological agent and mammalian retinal ganglion cells (see Ishida 1995;
Tabata & Ishida 1999). This possibility has been diffi-
cult to test, however, because a pharmacological agent
that can block this current selectively has yet to be Tabata & Ishida 1999). This possibility has been difficult to test, however, because a pharmacological agent that can block this current selectively has yet to be identified. It is also difficult to incorporate measured cult to test, however, because a pharmacological agent
that can block this current selectively has yet to be
identified. It is also difficult to incorporate measured
values of L, into spike calculations, because it is not that can block this current selectively has yet to be identified. It is also difficult to incorporate measured values of I_B into spike calculations, because it is not known whether the apparent amplitude and kinetics of identified. It is also difficult to incorporate measured
values of I_B into spike calculations, because it is not
known whether the apparent amplitude and kinetics of
 I_A are altered by the compounds used to isolate thi values of I_B into spike calculations, because it is not
known whether the apparent amplitude and kinetics of
 I_B are altered by the compounds used to isolate this
current (tetraethylammonium and 4-aminonyridine; see known whether the apparent amplitude and kinetics of I_B are altered by the compounds used to isolate this current (tetraethylammonium and 4-aminopyridine; see Isbida 1995) I_B are altered by the compounds used to isolate this current (tetraethylammonium and 4-aminopyridine; see Ishida 1995). rrent (tetraethylammonium and 4-aminopyridine; see
iida 1995).
Two other voltage-gated ion currents could transiently
gment retinal ganglion cell excitability at the termina-

Ishida 1995).
Two other voltage-gated ion currents could transiently
augment retinal ganglion cell excitability at the termina-
tion of hyperpolarizing light stimuli: T-type Ca^{2+} Two other voltage-gated ion currents could transiently
augment retinal ganglion cell excitability at the termina-
tion of hyperpolarizing light stimuli: T-type Ca^{2+}
current (T²) and the invardly rectifying mixed cati augment retinal ganglion cell excitability at the termination of hyperpolarizing light stimuli: T-type Ca^{2+} current ('I_T') and the inwardly rectifying, mixed cation current known as 'I.' (see Isbida 1998). L-would re tion of hyperpolarizing light stimuli: T-type Ca^{2+}
current (' I_T ') and the inwardly rectifying, mixed cation
current known as ' I_h ' (see Ishida 1998). I_T would recover
from inactivation while a ganglion cell was hy current (' I_T ') and the inwardly rectifying, mixed cation
current known as I_h ' (see Ishida 1998). I_T would recover
from inactivation while a ganglion cell was hyper-
polarized by light, activate upon termination of t current known as I_h (see Ishida 1998). I_T would recover
from inactivation while a ganglion cell was hyper-
polarized by light, activate upon termination of the

hyperpolarization, then inactivate unless the cell was
hyperpolarized again (Bindokas & Ishida 1996) I. hyperpolarization, then inactivate unless the cell was
hyperpolarized again (Bindokas & Ishida 1996). I_h
would be activated by an initial hyperpolarization and hyperpolarization, then inactivate unless the cell was
hyperpolarized again (Bindokas & Ishida 1996). I_h
would be activated by an initial hyperpolarization, and
would continue to flow after termination of the hyperhyperpolarized again (Bindokas & Ishida 1996). I_h
would be activated by an initial hyperpolarization, and
would continue to flow after termination of the hyper-
polarization until it deactivated (Tabata & Ishida 1996) would be activated by an initial hyperpolarization, and
would continue to flow after termination of the hyper-
polarization, until it deactivated (Tabata & Ishida 1996).
Both of these currents would hasten the rate at whic would continue to flow after termination of the hyper-
polarization, until it deactivated (Tabata & Ishida 1996).
Both of these currents would hasten the rate at which
ganglion cells reach spike threshold (thus shortening polarization, until it deactivated (Tabata & Ishida 1996).
Both of these currents would hasten the rate at which
ganglion cells reach spike threshold (thus shortening
spike latency) and briefly boost spike frequency Becaus Both of these currents would hasten the rate at which
ganglion cells reach spike threshold (thus shortening
spike latency) and briefly boost spike frequency. Because
the voltage sensitivities and current densities of Land ganglion cells reach spike threshold (thus shortening
spike latency) and briefly boost spike frequency. Because
the voltage sensitivities and current densities of I_T and I_h
both seem appropriate for producing spike bu spike latency) and briefly boost spike frequency. Because
the voltage sensitivities and current densities of I_T and I_h
both seem appropriate for producing spike bursts, what
advantage would be gained by having both cu the voltage sensitivities and current densities of I_T and I_h both seem appropriate for producing spike bursts, what advantage would be gained by having both currents both seem appropriate for producing spike bursts, what
advantage would be gained by having both currents
unless these were recruited by different stimuli?
Salamander and turtle retinal ganglion cells suggest that advantage would be gained by having both currents
unless these were recruited by different stimuli?
Salamander and turtle retinal ganglion cells suggest that
there is no particular advantage (or that there are other unless these were recruited by different stimuli?
Salamander and turtle retinal ganglion cells suggest that
there is no particular advantage (or that there are other
ways to generate suitable responses) as these cells have Salamander and turtle retinal ganglion cells suggest that
there is no particular advantage (or that there are other
ways to generate suitable responses), as these cells have there is no particular advantage (or that there are other
ways to generate suitable responses), as these cells have
measurable amounts of I_T and no detectable amounts of
I. Most fish ganglion cells have both however and I_h . Most fish ganglion cells have both, however, and rat ganglion cells appear to as well (see Ishida 1998). Is it ays to generate suitable responses), as these cells have
easurable amounts of I_T and no detectable amounts of
. Most fish ganglion cells have both, however, and rat
unglion cells appear to as well (see Ishida 1998) Is i measurable amounts of I_T and no detectable amounts of I_h . Most fish ganglion cells have both, however, and rat ganglion cells appear to as well (see Ishida 1998). Is it possible that I_T and I, drive spike bursts aft I_h . Most fish ganglion cells have both, however, and rat ganglion cells appear to as well (see Ishida 1998). Is it possible that I_T and I_h drive spike bursts after short and long hyperpolarizing light stimuli respec ganglion cells appear to as well (see Ishida 1998). Is it
possible that I_T and I_h drive spike bursts after short and
long hyperpolarizing light stimuli, respectively? This
might occur- if I_T recovered from inactivat possible that I_T and I_h drive spike bursts after short and
long hyperpolarizing light stimuli, respectively? This
might occur, if I_T recovered from inactivation as
ranidly as does N_a^+ current after brief denolari long hyperpolarizing light stimuli, respectively? This
might occur, if I_T recovered from inactivation as
rapidly as does Na^+ current after brief depolarizations
(Hidaka & Ishida 1998) because L, activates slowly might occur, if I_T recovered from inactivation as
rapidly as does Na^+ current after brief depolarizations
(Hidaka & Ishida 1998), because I_h activates slowly
(Tabata & Ishida 1996), However, in some neurons rapidly as does Na⁺ current after brief depolarizations
(Hidaka & Ishida 1998), because I_h activates slowly
(Tabata & Ishida 1996). However, in some neurons
(see Huguenard 1996). L_{as} bas been found to recover (Hidaka & Ishida 1998), because I_h activates slowly

(Tabata & Ishida 1996). However, in some neurons

(see Huguenard 1996), I_T has been found to recover

from inactivation about as slowly as L activates in (Tabata & Ishida 1996). However, in some neurons (see Huguenard 1996), I_T has been found to recover from inactivation about as slowly as I_h activates in (see Huguenard 1996), I_T has been found to recover
from inactivation about as slowly as I_h activates in
retinal ganglion cells. This might also be true for
retinal ganglion cells if the rate at which their T-type from inactivation about as slowly as I_h activates in
retinal ganglion cells. This might also be true for
retinal ganglion cells if the rate at which their T-type
 C_2^{2+} channels recovered from inactivation became slo retinal ganglion cells. This might also be true for
retinal ganglion cells if the rate at which their T-type
 Ca^{2+} channels recovered from inactivation became slower
with longer conditioning denolarizations (not unlike retinal ganglion cells if the rate at which their T-type Ca^{2+} channels recovered from inactivation became slower with longer conditioning depolarizations (not unlike the Ca^{2+} channels recovered from inactivation became slower
with longer conditioning depolarizations (not unlike the
effect, alluded to above (§ 3), of prolonged depolarizations
on Na^+ channels) with longer conditi
effect, alluded to ab
on $\mathrm{Na^+}$ channels).

5. CONCLUSION

Like many other central neurons, retinal ganglion cells are equipped with several different extra-synaptic ion Like many other central neurons, retinal ganglion cells
are equipped with several different extra-synaptic ion
currents. These include voltage-gated Na⁺ current, three
types of voltage-gated Ca^{2+} current perhaps thre are equipped with several different extra-synaptic ion
currents. These include voltage-gated Na^+ current, three
types of voltage-gated Ca^{2+} current, perhaps three types
of K^+ current J. a resting cation permeabili currents. These include voltage-gated Na⁺ current, three
types of voltage-gated Ca^{2+} current, perhaps three types
of K^+ current, I_h , a resting cation permeability, and a
resting Cl^- permeability (Ishida 1995–19 types of voltage-gated Ca^{2+} current, perhaps three types of K^+ current, I_h , a resting cation permeability, and a resting Cl^- permeability (Ishida 1995, 1998). Voltageof K^+ current, I_h , a resting cation permeability, and a resting Cl⁻ permeability (Ishida 1995, 1998). Voltagesensitive current properties that are likely to contribute to spike frequency and timing have been summar resting Cl⁻ permeability (Ishida 1995, 1998). Voltage-
sensitive current properties that are likely to contribute to
spike frequency and timing have been summarized here.
These include the recovery of Na^{+} and T-type sensitive current properties that are likely to contribute to
spike frequency and timing have been summarized here.
These include the recovery of Na^+ and T-type Ca^{2+}
currents from inactivation, and the recruitment of spike frequency and timing have been summarized here. These include the recovery of Na^+ and T-type Ca^{2+}
currents from inactivation, and the recruitment of I_h .
Also summarized are some current properties that do not
annear to contribute in either event. These include t currents from inactivation, and the recruitment of I_h .
Also summarized are some current properties that do not
appear to contribute in either event. These include the
complete loss of N_a ⁺ current at the membrane pot Also summarized are some current properties that do not
appear to contribute in either event. These include the
complete loss of $Na⁺$ current at the membrane potentials
that ganglion cells din to between spikes, and complete loss of $Na⁺$ current at the membrane potentials that ganglion cells dip to between spikes, and the recruitcomplete loss of Na^+ current at the membrane potentials
that ganglion cells dip to between spikes, and the recruit-
ment of a low-threshold K^+ current. Lastly, some current
properties whose contributions to spiking r that ganglion cells dip to between spikes, and the recruit-
ment of a low-threshold K^+ current. Lastly, some current
properties whose contributions to spiking remain to be
studied in more detail have been summarized. T ment of a low-threshold K^+ current. Lastly, some current
properties whose contributions to spiking remain to be
studied in more detail have been summarized. These
include the rate of K^+ current deactivation the poss properties whose contributions to spiking remain to be studied in more detail have been summarized. These include the rate of K^+ current deactivation, the possibility that N_a^+ current inactivation accumulates, the r studied in more detail have been summarized. These
include the rate of K^+ current deactivation, the possibility
that Na^+ current inactivation accumulates, the rate at
which T_{t} type Ca^{2+} current recovers from include the rate of K^+ current deactivation, the possibility
that Na^+ current inactivation accumulates, the rate at
which T-type Ca^{2+} current recovers from inactivation,
and the magnitude of the resting Cl^- condu that $Na⁺$ current inactivation accumulates, the rate at which T-type $Ca²⁺$ current recovers from inactivation, and the magnitude of the resting $Cl⁻$ conductance. The functional importance of these propertie which T-type Ca^{2+} current recovers from inactivation,
and the magnitude of the resting Cl^- conductance. The
functional importance of these properties will have to be
weighed against the contributions of synantic input and the magnitude of the resting Cl^- conductance. The functional importance of these properties will have to be weighed against the contributions of synaptic inputs and the electrical properties of other retinal neurons. functional importance of these properties will have to be weighed against the contributions of synaptic inputs and the electrical properties of other retinal neurons. It will also be of interest to compare the contribution weighed against the contributions of synaptic inputs and the electrical properties of other retinal neurons. It will
also be of interest to compare the contribution of these
voltage-gated ion currents to membrane potential
changes before and after they have been modulated by also be of interest to compare the contribution of these
voltage-gated ion currents to membrane potential
changes before and after they have been modulated by
messengers messengers.

The fish retinal ganglion cell recordings discussed here were The fish retinal ganglion cell recordings discussed here were
supported by National Institutes of Health grant EY08120. I
thank Vytas Bindokas Toshihide Tabata. Soh Hidaka. Angela The fish retinal ganglion cell recordings discussed here were
supported by National Institutes of Health grant EY08120. I
thank Vytas Bindokas, Toshihide Tabata, Soh Hidaka, Angela
Pignatelli. Cecilia Fernandez-Vaquero. Ka supported by National Institutes of Health grant EY08120. I
thank Vytas Bindokas, Toshihide Tabata, Soh Hidaka, Angela
Pignatelli, Cecilia Fernandez-Vaquero, Katharine Munckton,
Ming-Hsing Cheng and Gloria Partida for cont thank Vytas Bindokas, Toshihide Tabata, Soh Hidaka, Angela
Pignatelli, Cecilia Fernandez-Vaquero, Katharine Munckton,
Ming-Hsing Cheng and Gloria Partida, for contributing to
these measurements. Also, I thank Shaun Collin Pignatelli, Cecilia Fernandez-Vaquero, Katharine Munckton, Ming-Hsing Cheng and Gloria Partida, for contributing to these measurements. Also, I thank Shaun Collin and Justin Marshall for a splendid meeting at Heron Island. Ming-Hsing Cheng and Gloria Partida, for contributing to

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