Technical Report: Parameters and Evidence for a Neurorobotic Implementation of Newborn Free Looking Behavior

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Abstract

This Technical Report presents the background and implementation details of a neurorobotic model used for testing the first version of a model of free looking behavior, as discussed in (Veale, 2013). The work dates to early 2012; an updated model with additional results and experiments will appear in subsequent publications.

0.1 Introduction

This model attempts to reproduce fixation and eye movement behavior to static black-and-white geometric stimuli. Because of the controlled nature of the stimuli, the visual sensors of the model contain only those sensors necessary to detect the stimuli type that the agent will encounter. They are luminance-based, and the organization of the visual system is such that the only relevant visual features with specifically tuned neurons are oriented lines at 0, 45, 90, 135 degrees.

The overall conceptualization of the model is as follows. The first and most superficial layers of neurons (Cone, Bipolar, and Ganglion Cells) detect local differences in luminance in the raw visual image. These local differences are represented by the activations of these neurons in visuotopically arranged sheets. By selectively sampling certain patterns of the "deepest" retinal layers (the ganglion cells), neurons in a further visuotopically organized region (V1) gain responsive properties to line stimuli of specific orientations and luminance differences (black line on white background, white on black), but only when the stimuli fall under the corresponding topologically defined region of the visual field. Neurons in the different orientation and luminance-tuned visuotopic V1 "maps" compete within themselves via further collections of inhibitory interneurons that are connected in a local fashion, so that an in-place salience map arises via these horizontal interactions. A further region (dSC) of bursting neurons then receives topological input from all these V1 maps' visuotopically equivalent positions, and thus the excitatory afferent input represents an overall picture of the distribution of salient regions for all feature maps. This overall salience map feed into a further region of neurons (PPRF). When one of the dSC neurons burst sufficiently to elicit a PPRF

neuron to pass threshold, a saccade is coded to the corresponding topological region of the visual field. Inhibition in the form of Gaussian noise is injected into dSC neurons to simulate immature and noisy top-down input from the SNr. This constant inhibition prevents saccades from happening constantly, since there is always *some* input from the visual field. Finally, another pathway of inhibition biases the more eccentric portions of the SC map depending on ocular position, such that e.g. if the "eye" is already pointing to the right side it is less likely to make a saccade to the right.

The overall resulting behavior of this should be to make eye movements around a visual stimulus placed in front of the robot. And, based on the tuning of feature-responsive neurons in V1 and the in-place salience maps, the amount of time spent on regions of the visual field should be positively related to the amount of oriented lines there. The combination of these factors with the (random) inhibitory bias of SNr-dSC connections should, with the correct parameterization, produce the types of distributions seen in free looking studies. This happens because afferent excitation to a target area is higher when there are interesting features there. But, chance must have it that the region is at a low inhibition for the neuron to successfully burst and initiate a saccade. Saccades will be made to uninteresting regions rarely because of chance low inhibition at these locations. However, the high probability of initiating saccades to high-feature areas will cause these chance fixations to be very short.

Figure 1 is a visualization of the circuit tested. Each neural region in the model has a more in-depth review of what is known regarding its maturity and function in the next sections. Specifics of the implementation in the neuro-robotic model are also given alongside the neural justification. These include justifications for decisions to include or exclude neural regions and projections, especially where there has been debate in the literature or where recent evidence usurps previous assumptions.

1 Neural Background and Model Impementation Details

1.1 The Retina

The first part of the model is the retinal portions. This includes all parts between the raw photoreceptors (in the form of pixels from the camera image) and the retinal ganglion cells, which transmit the signal out of the eye via the optic nerve.

The basic function of the retina is to provide neurons sensitive to salient differences across the visual field. This would normally include neurons tuned to other features, such as motion or color differences. By encoding only differences (instead of the raw values), the amount of information to be transmitted is lessened. The eye is well-studied in a variety of species, and its elegant mechanisms are for the most part understood (Masland, 2001). The model implements the basic center/surround and on/off functionality of the retina. It is made up of a 2-dimensional sheet of cone photoreceptors, and then two 2-dimensional sheets of bipolar cells (which respond to "on" and "off" respectively), and then two 2-dimensional sheets of retinal ganglion cells (on-center/off-surround and vise versa) which sample the bipolar cells in a center-surround fashion.

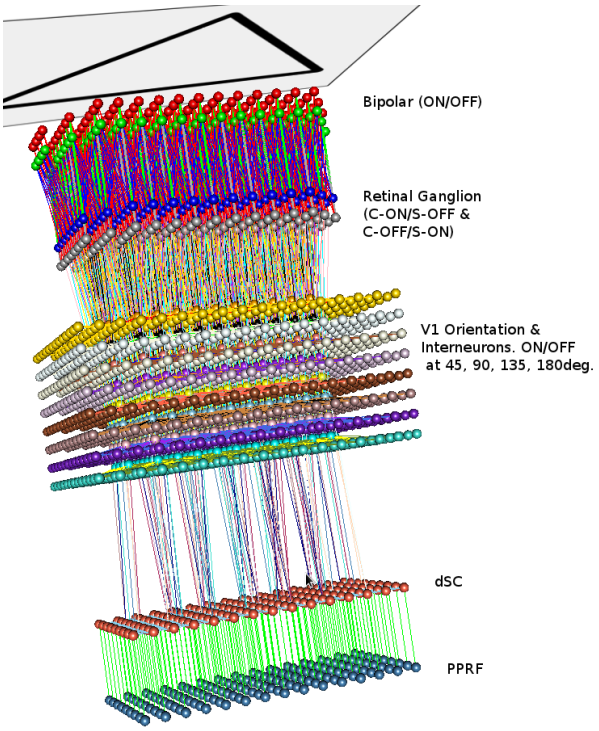

Figure 1: Visualization the neural model described in this paper. Synapses are subsampled. Proceeding from visual input (top) to brainstem (bottom), neural areas are labelled in the order they are presented in the text.

The cone photoreceptor cells approximate glutamate release, the neurotransmitter released by real cone photoreceptor cells based on the energy and number of photons being absorbed. The amount of glutamate released is based on the luminance value of the low-pass filtered pixel in the video image that corresponds to that receptor. The output of this layer is thus the level of glutamate being released for each topological location in the video frame. As in the eye, the glutamate level is inversely proportional to the luminance, such that the most glutamate is being released in complete darkness when no photons are entering the receptor, and the least glutamate is being released in very bright light.

Two sheets of cells, the on-bipolar and off-bipolar cells, receive these glutamate levels as input. The glutamate excites the off-bipolar cells, and inhibits the on-bipolar cells (in a real eye, this is accomplished by expressing different glutamate receptors). This causes the on-bipolar cells to spike more when light falls over the photoreceptors feeding into them, and the off-bipolar cells to spike less in the same conditions. Conversely, off-bipolars fire strongly when there is no light, whereas on-bipolars' firing is attenuated in these circumstances.

Finally, the retinal ganglion cells sample the on- and off-bipolar cells to produce center-surround responses. A set of on-center/off-surround ganglion cells sample from on-bipolar cells in a central region, and from off-bipolar cells in a region surrounding that. This results in the on-center/off-surround ganglion cells responding most vigorously (with high firing rates) when there is a light dot that falls directly under the center bipolar cells receptive field in a dark background. The response diminishes as there is more light in the background (surround), and as the central light spot gets darker. The off-center/on-surround ganglion cells would have the exact opposite response to the same stimulus.

The maturational state of the newborn retina has been characterized by Yuodelis and Hendrickson (1986) and Abramov et al. (1982) in human and by La Vail, Rapaport, and Rakic (1991) in monkey. While peripheral zones have adult-like receptor-morphologies, the macular (foveal) region is still under development, and seems to have the spatial resolution of peripheral regions. Ganglion and horizontal cells are generated even before the underlying cone receptors, and bipolar and amacrine cells are at least partially mature at birth (La Vail et al., 1991). For additional review of the function of these various cell types and connectivities in the primate retina, the reader is referred to Masland (2001).

Based on the known maturational state and function of various ganglion cell types in the (adult) primate retina, the retinal portion of the model comprises a cone-receptor layer which projects into bipolar cells (ON and OFF variety) retinotopically, and then into ganglion cells in a center-surround fashion (Diller et al., 2004; Croner & Kaplan, 1995; Jacoby, Stafford, Kouyama, & Marshak, 1996; Lee, Kremers, & Yeh, 1998). Cone cells respond to light by phasically modulating their tonic release of glutamate proportional to the frequency of the absorbed photon, maximally decreasing their response for the optimal frequency for their photo-pigment. The bipolar cells onto which the cone photo-receptors synapse manifest two different types of glutamate receptors depending on their type, one hyperpolarizes (deactivates) the cell membrane in response to glutamate (this is the ON bipolar cell), the other depolarizes the membrane (the OFF type). Ganglion cells receive input from one or more bipolar cells, which

grant their responsive properties.

Since modelling the dynamics of the retina is not the primary goal of this model, the overall function of achieving centre-surround receptive fields in the ganglion layer is implemented by the following means: bipolar cells receive "diffuse" input from many cone receptors of all types via a lowpass separable filter applied to the pixels of the input image. This achieves the decreased spatial resolution believed to be present in the newborn retina, and also mimicks the diffuse nature of bipolar dendrites in the periphery, which synapse with cone types non-specifically. Ganglion cells then receive input from a single centre bipolar cell (ON or OFF), and from a surround of the opposite bipolar type. This differs from the mechanism in the retina, in which horizontal cells probably work to hyperpolarize the cone receptors themselves based on the activity of adjacent cone cells. However, this method simplifies modelling and results in the same type of signal at the level of retinal ganglion cells.

Bipolar cells are 2-d layers of neurons (16×12) overlaid against the image at integer intervals (producing one bipolar cell roughly every 10 pixels in horizontal and vertical dimensions for a 160×120 pixel image). To mimick the rounded shape of the visual field, only cells within a radius$= 7.9$ of the center of the layer were generated. Bipolar cells are modelled as leaky-integrate-and-fire (LIF) neurons, with parameters ($I_{bg} = 15.5$, $\tau_m = 10.0$, $V_{thresh} = 15.0$, $V_{reset} = 14.5$, $t_{refract} = 2.0$) for OFF bipolars, and ($I_{bg} = 14.5$, $\tau_m = 10.0$, $V_{thresh} = 15.0$, $V_{reset} = 14.5$, $t_{refract} = 2.0$) for ON bipolars. Current was injected based on the value of the low-pass-filtered pixel (filtered with the linear separable filter $[1, 8, 28, 56, 70, 56, 28, 8, 1]$) lying under the bipolar cell, normalized to the size of the image with 0.4 of margin on each side to allow the LPF input to never be on an edge. The intensity value of the pixel (sum of all red, green, blue channels) was normalized to the interval $[0, 1]$ and injected as I_{inj} into the bipolar cell (this value was set negative for OFF-types). Thus, the update of the bipolar cell LIF neurons was:

$$\frac{dV_m}{dt} = \frac{-V_m + (I_{bg} + I_{inj})}{\tau_m} \tag{1}$$

and when $V_m > V_{thresh}$, V_m was reset to V_{reset} and the neuron entered a refractory period $t_{refract}$, during which no dynamics of the cell were updated.

The different parameterizations of ON versus OFF bipolar cells are such that OFF bipolar cells will fire constantly while there was no stimulation under their receptive fields, and be suppressed when there was stimulation, whereas ON-type cells would be the opposite – quiescent if there was no impinging light, but active if there was.

Ganglion cells were likewise modelled as 2-d layers of LIF neurons. In the case of ganglion cells, parameters were uniformly ($I_{bg} = 13.5$, $\tau_m = 30.0$, $V_{thresh} = 15.0$, $V_{reset} = 13.5$, $t_{refract} = 3.0$) for both ON-centre/OFF-surround and OFF-centre/ON-surround types. Ganglion cells were generated at integer-spacings underneath the bipolar cells, and only cells within a radius$= 7.5$ from the center of the layer were generated. Static synapses connect each ganglion cell to its corresponding bipolar cell centre (in ON or OFF bipolar layer), ($w = 10.0$, $t_{delay} = 2.0$). Thus, a presynaptic firing would result in a spike that the postsynaptic neuron would feel after 2 milliseconds. The spike hit would cause an increase in the (excitatory) postsynaptic conductance g_E of weight w.

Postsynaptic conductances decay exponentially, i.e.

$$\frac{dg_E}{dt} = \frac{-g_E}{\tau_{g_E}} + I \tag{2}$$

where $\tau_{g_E} = 3$ ms for the ganglion cells. The membrane resistance of the neuraons was assumed to be of the appropriate scale ($1M\Omega$) such that conversion between g_E and I_{inj} was not necessary, i.e. $I_{inj} = g_E + g_I$. Ganglion cells receive their surround-afferents from the bipolar layer of the opposite type, with all cells within a radius= 1.9 (except the centre neuron) providing afferent synapses. The weight w of each surrounding synapse was linearly normalized by the total number of surrounding afferents.

1.2 Lateral Geniculate Nucleus (LGN)

The LGN was not explicitly modelled in the network, since receptive field properties of its neurons are essentially identical to the ganglion cells from which they receive input. Some evidence has shown inhibitory interactions in the LGN, and modulations of activity via feedback. Since none of these was explicitly modelled, the LGN was abridged. However, the LGN is mature at birth in non-human primates (Rakic, 1977) and in humans (Hitchcock & Hickey, 1980). Thus, it is capable of relaying signals to the primary visual cortex (V1). All layers (magnocellular and parvocellular) seem to be at least half developed at birth, and by reference to animal studies, to be both accepting synapses via the optic tract and projecting axons to V1 (Hickey, 1977).

1.3 Primary Visual Cortex (V1)

Primary visual cortex (V1) is the next step in the geniculo-striate pathway. V1 is known to contain a retinotopic representation of the visual field. Only cells in layers 5/6 are mature at birth (Bourne, Warner, & Rosa, 2005), and receive LGN afferents (Meissirel, Wikler, Chalupa, & Rakic, 1997). Cells in these layers project to subcortical structures (thalamus, including pulvinar, and reciprocate LGN afferents), and to superior colliculus (Schiller, Malpeli, & Schein, 1979; Bender, 1983; Ungerleider, Galkin, & Mishkin, 1983; Shipp, 2003; Benevento, 1976; Gutierrez & Cusick, 1997; Rezak & Benevento, 1979; Trojanowski, 1977). There is also a sparse projection to the caudate nucleus (CD) from V1 documented (Kemp & Powell, 1970), and it arises from L5/6.

The lack of mature cells in more superficial layers (L2/3) of V1 which contain the cortico-cortical projection cells in adults, or the L4 cells that feed to those, implies that the cortico-cortical feedforward network (e.g. to higher visual areas such as V2-V5, or parietal/frontal cortices) is not mature and thus any activity in those areas, if it exists, cannot be directly mediated by the geniculo-striate pathway.

Finlay, Schiller, and Volman (1976) antidromically stimulated V1 cells from superior colliculus (next subsection) to determine their response properties. The cells that were antidromically activated resided in L5/6, and had several broadly-tuned receptive field properties, including orientation and directional motion tuning (this is in contrast to the receptive fields of SC cells that they project to, which have no such selectivities). V1-SC projection cells have large receptive fields ($> 0.3^o$ and often $> 1.0^o$) and

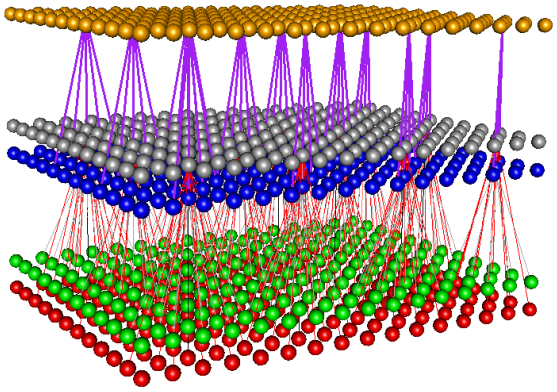

Figure 2: Example connectivity of several V1 orientation-sensitive cells (the uppermost layer) of the same map. In this case, they are 45^o angle sensitive cells – the selective sampling is most visible in the rightmost cells in the image. Also shown are the center-surround connections of the ganglion cells (middle) to bipolars (bottom). Synapses are subsampled for visualization purposes.

respond to either bright orientations on dark backgrounds or dark orientations on light backgrounds.

In light of these properties, and in combination with research showing horizontal connectivity in deep V1 layers at birth (Burkhalter, Bernardo, & Charles, 1993), which might possibly implement an implicit salience map (Z. Li, 2002), V1 was implemented as follows: 4 layers for each of the broadly tuned orientations at $45^o, 90^o, 135^o, 180^o$, each of which has one layer for light lines on dark background, and one for dark on light background, for a total of eight excitatory layers that are retinotopically organized. For an example of the selective connectivity that gives rise to these responsive properties, cf. Figure 2.

In addition to the orientation-sensitive neurons, each of the layers has its own separate set of inhibitory interneurons which receive excitatory input (as e.g. axon collaterals) only from distal neurons in their own V1 layer, and send inhibitory output only to neurons that are in their immediate vincinity in thrie own layer. In this way there are eight parallel layers that respond to different features of stimuli, and which compete spatially within their own feature type.

Each of the eight orientation-responsive layers is implemented as 2-d sets (16×12) of Izhikevich neurons arranged at integer points. Only neurons that were within a radius= 7.5 of the center of the layer and which were greater than 1.5 distance separated from the top or bottom of the layer were generated, to take into account the shape of the visual field that is reflected in retinal cells. The orientation neurons were parameterized ($a = 0.65,\ b = 0.23,\ c = -65.0,\ d = 2.0$). The dynamics of the membrane

potential for these neurons was:

$$\frac{dV_m}{dt} = 0.04V_m^2 + 5V_m + 140 - W_m + I_{syn} \tag{3}$$

$$\frac{dW_m}{dt} = a(b \cdot V_m - W_m) \tag{4}$$

and when $V_m > 30.0$, V_m is set to c, and W_m to $W_m + d$.

Afferent connections from ganglion/LGN cells were determined by selectively connecting to ganglion cells within a radius beneath the V1 cell that are arranged in a certain orientation pattern. Figure 2 demonstrates examples of this. Ganglion cells were connected if they lay within a radius= 1.8 (for diagnal cells) or radius= 1.1 (for horizontal/vertical cells) around the V1 cell, and if their relative horizontal and vertical offsets from the V1 cell were such that they were retinotopically at the right orientation.

Ganglion-V1 synapses were static synapses with parameters $w = 0.065,\ t_{delay} = 2.0$. w was distributed based on the number of presynaptic neurons, i.e. with 4 presynaptic neurons being sampled the weight of each synapse was $\frac{w}{4}$.

Post-synaptic conductances contributing to I_{syn} were modelled using realistic receptor conductance dynamics, including *AMPA*, *NMDA*, $GABA_A$ and $GABA_B$-mediated conductances. A presynaptic spike at time t would cause the post-synaptic neuron to feel a change in its conductances at time $t + t_{delay}$. In the case of a glutamateric (excitatory) presynaptic neuron, g_{AMPA} and g_{NMDA} would increase by w, assuming equal proportions of AMPA and NMDA receptors. These conductances decay exponentially, as in the equation for g_E above, with time constants $\tau_{AMPA} = 5.0,\ \tau_{NMDA} = 150.0$. The current felt on account of synaptic input, I_{syn}, is calculated based on instantaneous conductances in the following way:

$$\begin{aligned} I_{syn} = \quad & g_{AMPA}(V_m - 0) + \\ & g_{NMDA}\frac{[(V_m + 80)/60]^2}{1 + [(V_m + 80)/60]^2}(V_m - 0) + \\ & g_{GABA_A}(V_m + 70) + \\ & g_{GABA_B}(V_m + 90) \end{aligned}$$

The -70 mV and -90 mV terms correspond to the reversal potentials of $GABA_A$ and $GABA_B$ receptors, respectively. *NMDA* receptors are voltage-gated (with a J-shaped voltage-current relationship), and along with *AMPA* receptors, have a 0 mV reversal potential (Izhikevich, Gally, & Edelman, 2004).

Inhibitory interneurons in the V1 layers were likewise Izhikevich neurons with conductance-based post-synaptic responses. Model parameters for these neurons were $a = 0.02,\ b = 0.2,\ c = -50.0,\ d = 2.0$, producing chattering neurons. Afferents from V1 orientation neurons were static synapses with parameters $w = 0.03,\ t_{delay} = 1.0$, and an inhibitory interneuron was connected to every V1 orientation neuron of its corresponding orientation map that fell within a radius= 2.5. Efferent synapses (inhibitory synapses onto V1 orientation neurons) were $w = -0.001,\ t_{delay} = 1.0ms$, and were connected to all neurons of the corresponding orientation map that fell outside of the radius. GABA conductances had time constants $\tau_{GABA_A} = 6.0,\ \tau_{GABA_B} = 150.0$.

Overall, the inhibitory interneurons focus activity into only those V1 neurons receiving the strongest signals via lateral inhibition, implicitly implementating a type of winner-take-all (WTA) mechanism. This was tested by artificially removing the inhibitory connections, and observing that V1 activity quickly saturates in their absence.

1.4 Superior Colliculus (SC)

V1 projects retinotopically into the ipsilateral superior colliculus (SC), which is a laminated structure in the midbrain wherein the superficial layers (sSC) have primarily visual response properties and receive direct input from the retina (Wallace & Stein, 2001; P. May, 2006; Schiller et al., 1979). The deeper layers (dSC) have multimodal and motor-related responses which has been established to drive eye movements (Glimcher & Sparks, 1993, 1992; Isa & Sparks, 2006; Munoz, 2002; Ibbotson & Dreher, 2005). SC's retinal afferents/intrinsic circuitry are mature at birth in humans (Qu et al., 2006), as are its response properties (Wallace & Stein, 2001).

SC lesions produce massive deficits in eye movement control even in adult primates, with even greater deficits predicted in infants, who do not have access to extracolliculuar pathways (such as frontal eye fields) to drive the brainstem (Albano, Mishkin, Westbrook, & Wurtz, 1982; Schiller et al., 1979).

Extensive research documents the contribution of dSC to gaze allocation, including maintenance of ongoing fixations and the breaking of fixations to produce an eye movement (Munoz, 2002; Munoz, Dorris, Par, & Everling, 2000; Munoz & Istvan, 1998; Munoz & Wurtz, 1995a, 1995b, 1993b, 1993a, 1992; Munoz & Fecteau, 2002; Ibbotson & Dreher, 2005). dSC neurons seem to mediate fixation via the tonic firing of "fixation" cells, present in the rostral dSC (which has visual receptive fields around the fovea). Jumps in excitation in "saccade" cells in more caudal SC (containing representations of peripheral visual field) seem to interrupt fixation and cause an eye movement to that location. These regions seem to mutually inhibit one another, and to mutually excite their analogue in contralateral SC.

Thus, the activity markers of both ongoing fixations and of saccades are both understood in SC. Additionally, the mechanism by which the target of an eye movement is specified is known to be a combination of exitation and release from inhibition (Glimcher & Sparks, 1993, 1992). However, initiation of eye movements seems to be reliant on disinhibition, and it is this mechanism that is not well understood. In trained monkeys, it has been shown that the eye movement initiation disinhibition signal is via suppression of substantia nigra pars reticulata (SNr) neurons that provide tonic inhibition to dSC (Hikosaka & Wurtz, 1985, 1983c, 1983b, 1983a, 1983d; Jiang, Stein, & McHaffie, 2003), and furthermore that this suppression is mediated by striatal (in particular caudate nucleus – CD) neurons (Hikosaka, Sakamoto, & Usui, 1989a, 1989b; Hikosaka, Takikawa, & Kawagoe, 2000). The descending nigral connections are known to be functionally in place and mature at birth, at least in feline (Gabriele, Smoot, Jiang, Stein, & McHaffie, 2006).

However, it is also noted that these signals disappear during spontaneous saccades, i.e. they are probably a product of the conditioning and not of the baseline visual scanning mechanism that we are interested in. Other inhibition is known to arrive in dSC via the ventral zona incerta (ZIv), a portion of the thalamus which has known visual

responses and saccade-related pauses, and might receive visual input from deep layers of V1 (P. May, 2006; T. P. Ma, Hu, Anavi, & Rafols, 1992; Mitrofanis, 2005; Power, Leamey, & Mitrofanis, 2001; Kim, Gregory, & Hall, 1992; Romanowski, Mitchell, & Crossman, 1985; T. Ma, 1996; P. J. May, Sun, & Hall, 1997). Other portions of the central thalamus (IML) also have saccade-related neurons that are active even during spontaneous saccades and which have visual responses during trained task performance (Schlag-Rey & Schlag, 1984; Schlag & Schlag-Rey, 1984). However, the timing of their activity makes it unlikely that they are responsible for the signal to the SC.

Other possibilities for initiation signals in spontaneous saccades include not disinhibition, but a mechanism mediated by acetylcholine (ACh), perhaps delivered via the reticular formation. ACh has been shown to modulate the membrane potentials of dSC neurons in such a way that they become more likely to burst in relation to their interlaminar input from sSC neurons; this pathway is normally blocked (Kobayashi & Isa, 2002; Aizawa, Kobayashi, Yamamoto, & Isa, 1999). There is also converging evidence for complex intrinsic circuitry in dSC, including inhibitory interneurons providing distal inhibition, and local excitatory connections. dSC neurons fire spontaneously and in synchrony with one another when released from tonic GABA inhibition and when Mg^{2+} levels are artificially increased (to highlight the role of NMDA receptors in the bursting activity) (Isa & Yoshida, 2009; Isa & Sparks, 2006; Isa, 2002; Phongphanphanee et al., 2011). Thus there also seem to be some intrinsic mechanisms at play in producing the behavior observed in dSC.

In light of uncertainty regarding the details of extrinsic afferents of dSC, the simplest implementation is pursued. dSC is implemented as a 2-d array of Izhikevich neurons that receive retinotopic, converging input from all V1 orientation maps. The neurons are parameterized to demonstrate the robust bursting behavior observed in dSC tecto-reticular neurons that initiate eye-movements, and this is done via biophysically accurate mechanisms: recruitment of $NMDA$-mediated conductances and primarily expression of $GABA_A$ receptors, which mediate inhibition ($GABA_A$ conductances decay relatively quickly). In addition, to take into account the role of disinhibition from GABA, Gaussian-distributed levels of GABA are felt continuously across the dSC (this is in line with Johnson (1995)'s hypothesis regarding the role of SNr in newborns). In addition, modulation of the SC inhibition based on orbital position of the eye is implemented to mimick known centering-bias of eye-movements. Because of the known inability of sSC to drive dSC except under special conditions which do not obtain in the experimental setup, sSC was abridged from the model. This decision may need to be reexamined in the future as the role of the reticular formation in normal free-viewing is understood.

dSC was imlemented as a 2-dimensional 15×11 array of Izhikevich neurons with realistic receptor-conductances (AMPA, NMDA, etc.). Neurons were parameterized as chattering neurons with parameters $a = 0.05,\ b = 0.2,\ c = -50.0,\ d = 2.0$. Additionally, to account for the fact that inhibition from SNr afferents is primarily via expression of $GABA_A$ receptors, $GABA_B$ receptors were parameterized the same as $GABA_A$ receptors with a fast time constant of $\tau_{GABA_B} = 6.0$ ms. Neurons were placed in a 2-d array at integer spacings, but offset 0.5 from V1 neurons (to place them in the space between V1 neurons). They received converging excitatory projections from V1 neurons from all eight parallel layers, within a retinotopic radius$= 1.1$. This is in line with the phys-

iological data showing larger visual receptive fields of SC neurons, which combine many V1 afferents to form their receptive properties. These excitatory synapses had parameters $w = 0.3$, $t_{delay} = 1.0$. dSC neurons are connected to surrounding dSC neurons via static synapses within a radius= 1.9 with parameters $w = 0.00005, t_{delay} = 1.0$ and

1.5 Brainstem (PPRF)

The eye muscles are driven by a plant in the brainstem which converts from place-based codes to rate-based codes for driving each of the muscles arranged around the eyes (Scudder, Kaneko, & Fuchs, 2002; Fuchs, Kaneko, & Scudder, 1985; Gandhi, Barton, & Sparks, 2008; Bergeron & Guitton, 2002; Buttner-Ennever & Horn, 1997; Girard & Berthoz, 2005; Horn, 2006; Schall, 1995; Sparks & Hu, 2006; Waitzman, Pathmanathan, Presnell, Ayers, & DePalma, 2002; Waitzman, Silakov, DePalma-Bowles, & Ayers, 2000). It is known to close downstream of the superior colliculus (Kato, Grantyn, Dalezios, & Moschovakis, 2006). There is evidence of feedback mechanisms, perhaps via the cerebellum (Guitton, Bergeron, Choi, & Matsuo, 2003; Soetedjo, Kaneko, & Fuchs, 2002). For the sake of simplicity, we do not explicitly model the circuit that implements this plant, on account of it being known to close downstream of the SC, and because of the uncertainty regarding its actual implementation in the literature. Importantly, the neural circuitry has been shown to be at least anatomically present before birth (Shupert & Fuchs, 1988).

Properties of some of the neurons (particularly the Omni-Pause Neurons (OPNs) and Burst Neurons (BNs)) are relevant because they share properties with and are closely connected to the superior colliculus layers which we do model (Everling, Par, Dorris, & Munoz, 1998; Ibbotson & Dreher, 2005; Wurtz & Albano, 1980; P. May, 2006). These PPRF neurons are modelled as a 2-d layer of LIF neurons with fast membrane time constants, which must be driven by dSC bursts in order to elicit an eye movement.

1.6 PPRF Implementation Parameters

PPRF was implemented as an 15×11 array of LIF neurons with basic (exctiatory/inhibitory) conductances. These neurons had a fast membrane time constant of $\tau_m = 15.0$ ms, and received excitatory synapses from dSC neurons within a radius= 0.1 (the neuron right above it), of parameters $w = 4.8$, $t_{delay} = 1.0$. The spiking of any of these neurons was sufficient to elicit eye movements.

1.7 Occular Eccentricity

dSC neurons additionally felt inhibitory conductances from four canonical neurons that responded with activations linearly related to the eccentricity of the eye in that direction. Thus, when the eye was already at an angle to the right, neurons on the right side of the dSC were inhibited more, lowering the probability of a saccade to an even more eccentric position. This inhibition fell off linearly until it was zero at the centre. The raw angle value in radians of the orbital position was multiplied by a constant

weight$= -2.0$, and then added to the inhibitory conductances of dSC neurons at every time step (1.0 ms). The portion of this added to each neuron depended on its eccentricity in that direction. For example, the "UP"-coding neuron connected to the dSC neurons coding the upper-half of the visual field. The most eccentric neurons (coding the very top portion of the visual field) received the full inhibitory weight, and neurons felt less of this weight as eccentricity decreased to midline. Evidence for this being a linear function, it continuing to midline, it being mediated by inhibitory means, etc., are not based on anatomical literature, but rather on the known behavioral tendencies (centering bias) of infants. The anatomical basis is as yet unknown: neurons coding eye position are known to be present in motor cortex in the parietal lobe in adults, though the maturational state and connectivity of this region in newborns is uncertain. There is evidence that neurons coding eye position are present in SC (Campos, Cherian, & Segraves, 2006), which is known to be maturationally functional at birth. The source of these signals, however, are unknown (they might be the result of e.g. afferents from motor cortex, and thus might not exist in infants). Thalamic neurons are also known to sometimes respond selectively based on eye position. At any rate, because of such underspecification in the literature, the canonical approach described above is taken.

1.8 SNr Afferents

SNr afferents are modelled as Gaussian-distributed inhibitory ligands fed into each dSC neuron at every time-step, and re-sampled every 33 ms. This noise has mean -1.0 and standard deviation of 1.1, and is sampled separately for every neuron (realistically, a more topologically-distributed function might have been better).

1.9 Robotic Gaze Control

The firing of any PPRF neuron was sufficient to elicit eye movements. If more than one fired in a single time-step, a winner was randomly chosen from among these. The retinotopic position of the winning PPRF neuron was interpolated onto the size of the camera image, and the position from midline in horizontal and vertical directions was linearly interpolated into the necessary horizontal and vertical angles needed to center that retinotopic location. The robot controller for the pitch and yaw angles of the head was sent a signal to make this movement, which was performed in an open-loop. All neurons in the model were reset to their default values (there is evidence of different effects in animals, e.g. bursts of excitation in V1 at fixation onset, inhibition of LGN via pretectal and tectal neurons that sense global motion indicating a saccade is being made, etc.) In the future these might be experimented with. However, in the current studies, since there is no feedback from the controller at these time-scales, and the camera frames are not fast enough to realistically differentiate movement at this rate from any other rate, the time to complete the movement was estimated empirically to be 100 ms, neurons were reset, and the network did not update for 100 ms.

1.10 Abridgement of Higher Level Cortical Areas

The direct connection from retina to SC is not modelled nor is the retinal or tectal projection to inferior and lateral pulvinar. These areas are not modelled even though they are known to be mature and visually responsive, on account of converging research showing that they are not active in the conditions tested – though they may be active in other conditions and may need to be accounted for in future projects. Higher cortices including frontal and parietal cortices are not modelled, nor are higher visual areas. The basal ganglia are not modelled explicitly, though their affect on SC is. Portions of the temporal lobe are known to be mature, but they are not modelled explicitly since there is no clear pathway for visual or endogenous signals to reach them. Section 2 addresses this issue and tries to reconcile it with the observation that lesions of this area can have effects on looking even at birth.

Temporal lobe may indirectly receive input via e.g. subcortical circuits. At this point it is relevant to note that frontal areas and higher visual areas are known not to be maturationally in advance of primary striate, and in particular, their superficial layers (which are the target of subcortical projections from e.g. the pulvinar thalamus (Kostovic & Rakic, 1984; Gutierrez & Cusick, 1997)) will not be mature (Mrzljak, Uylings, Kostovic, & Van Eden, 1988; Mrzljak, Uylings, Van Eden, & Juds, 1990; Mrzljak, Uylings, & Kostovic, 1992). On account of this, a large portion of the brain, including most of the occipital lobe and frontal lobes, is likely not responsible for observed behavior in human newborns, and only gradually exerts control over the first 6 months postnatal. However, portions of the temporal lobe, especially inferior temporal lobe, and limbic cortex, do seem to mature quite early, though the level and type of input is uncertain since they do not receive projections from V1, and in light of the conclusions regarding the immaturity of other cortical areas around the time of birth. There is evidence of some subcortical projections to these areas, however (Yeterian & Pandya, 1988).

1.11 Neural Simulation Methods

LIF neurons were simulated exactly using the analytical solution

$$V_m(t) = C_1 V_m(t-dt) + C_2(I_{syn} + I_{bg}) \tag{5}$$

where constant $C_1 = e^{-dt/\tau_m}$, $C_2 = 1 - C_1$. Simulation $dt = 1.0$ ms. Base-PSR Izhikevich neurons (i.e. where the post-synaptic response is equal to the linear combination of excitatory and inhibitory post-synaptic currents) were simulated via the forward-Euler method, $dt = 0.5$ ms. For numerical stability, conductance-PSR Izhikevich neurons were simulated using the hybrid method (Izhikevich, 2010) at $dt = 0.5$ ms. Thus, the update of V_m was via the equation:

$$V_m(t) = \frac{V_m(t-dt) + dt \cdot \left(0.04V_m^2 + 5V_m + 140 + g(t-dt)E(t-dt)\right)}{1 + dt \cdot g(t)} \tag{6}$$

where the term $g(t)$ is the net conductance from all receptor-types:

$$g(t) = g_{AMPA}(t) + g'_{NMDA}(t) + g_{GABAa}(t) + g_{GABAb}(t) \tag{7}$$

note that the *NMDA* term is voltage-gated, i.e. $g'_{NMDA}(t) = g_{NMDA}(t)\frac{[(V_m+80)/60]^2}{1+[(V_m+80)/60]^2}$, instead of just the raw condutance.

$E(t)$ is the total (average) reversal potential, $i \in AMPA,\ NMDA,\ GABAa,\ GABAb$ (again, *NMDA* is the voltage-gated term, not the raw conductance):

$$E(t) = \frac{\sum(g_i(t) \cdot E_i)}{g(t)} \tag{8}$$

In our case, $E_{GABAa} = -70$ mV, $E_{GABAb} = -90$ mV, and $E_{NMDA}, E_{AMPA} = 0$ mV.

Simulation was performed via the nsim3 spiking network simulation program, developed internally for simulation of spiking neural networks controlling robots. The network integration, video visualization, and video frame capture were performed in separate execution threads run from a laptop computer connected to the robot. The simulator is based on previous publications including PCSim (Maass, Natschlager, & Markram, 2002) and a simulator by Richert, Nageswaran, Dutt, and Krichmar (2011), and methods for accurate simulation of spiking networks (Morrison, Aertsen, & Diesmann, 2007).

2 Ideas for Habituation in the Visuo-Motor Circuit

This section describes the relation of the visuo-motor circuit presented above in section 1 to learning behavior such as unimodal and multimodal habituation. Even assuming that free looking behavior is fully understood, there is still a lot to be understood regarding how learning centers of the immature infant brain interact with incoming visual information to recognize familiar stimuli and how they influence behavior. In particular, what aspects of individual fixation behavior do they influence to generate the overall patterns that are measured in looking experiments? Do they influence choice of target, length of looking, speed of processing of the visual information, or some sort of general arousal which in turns mediates several of these factors simultaneously? Or do the properties of individual fixations influence some other aspects that are not listed here?

The section begins with an overview of the neural background of habituation, citing converging evidence that portions of the (inferior) temporal lobe are responsible for familiarity and habituation even in very young infants. It presents a sketch of how these familiarity responses could be built into the visuo-motor model presented above, using only circuits known to be mature soon after birth involving the visual thalamus (pulvinar nucleus). It then addresses the second problem, which is how the response of these learning areas finds its way back to the gaze-control centers, and subsequently influences behavior in the form of modifying looking behavior.

2.1 Temporal Lobe

The temporal lobe, including limbic cortices (such as Entorhinal (EC), Perirhinal (PR), and Parahippocampal (PH) cortices) and inferotemporal visual areas (area TE and TEO in non-human primates) have been strongly related to learning and in particular habituation behaviors in the non-human primate. A large number of lesion studies have

converged to show that visual habituation (as measured via novelty preferences) is abolished in different ways when these areas are selectively damaged. In the hippocampal region, an early study followed by several more recent follow-ups showed that simultaneous lesion of EC, hippocampus, and possibly the amygdaloid complex in very young infant primates resulted in an abolishment of novelty preference (Bachevalier, Brickson, & Hagger, 1993; Bachevalier & Vargha-Khadem, 2005; Pascalis & Bachevalier, 1999; Bachevalier, 2001; Alvarado & Bachevalier, 2000), but did not affect the normal looking behavior. More specific lesions of only the hippocampus proper (Zeamer, Heuer, & Bachevalier, 2010) did not cause similar deficits. This implies that, at least in very immature primates, the hippocampus does not mediate habituation or novelty preferences, but that some other structure around the amygdaloid complex or entorhinal cortex does. Inferotemporal cortex (IT – including e.g. area TE of the non-human primate) has been likewise shown to be involved in visual habituation (in adults) based on lesion studies (Alvarado & Bachevalier, 2000; Nakamura & Kubota, 1996; Buffalo et al., 1999; Nemanic, Alvarado, & Bachevalier, 2004; Bachevalier, Brickson, Hagger, & Mishkin, 1990).

In adults, the responsive properties of neurons recorded from IT and limbic cortices point to their role in recognition memory and habituation/novelty detection (Suzuki, Miller, & Desimone, 1997; L. Li & Miller, 1993; E. K. Miller, Li, & Desimone, 1991; E. Miller, Gochin, & Gross, 1991; Wan, Aggleton, & Brown, 1999).

The developmental trajectory of the hippocampal region supports the lesion data; in humans and non-human primates the hippocampus proper (Amon's horn) develops quite slowly with respect to the surrounding cortex (EC, etc.). This surrounding cortex is known to be at least partially mature at birth (Seress, Abraham, Tornoczeky, & Koszytolanyi, 2001; Seress, 2001). Inferotemporal cortex likewise develops and demonstrates visual activity quite early (Rodman, 1994). Behavioral studies in the context of cortical ablations demonstrate that at least *something* is happening in this region which has an effect on visual behavior, and that this is related to the familiarity status of stimuli instead of to e.g. only direction of eye gaze. The amygdala (AMYG), a subcortical structure, is highly interconnected with these limbic regions (Kajiwara, Takashima, Mimura, Witter, & Iijima, 2003; Suzuki, 1996; Iwai, Yukie, Suyama, & Shirakawa, 1987; Herzog, 1976), and also has visual responses (Sanghera, Rolls, & Roper-Hall, 1979), which habituate to visual stimuli (Wilson, 1993). It remains to be determined what exactly the role of this amygdaloid system is in visual habituation and biasing eye movements.

The inferior temporal cortex and rhinal cortices accept afferents from pretty much the entire brain, in every sensory modality (Hoesen & Pandya, 1975; Streitfeld, 1980; Suzuki, 1996; Nakamura & Kubota, 1996). However, the primary cortical inputs to the visual areas (e.g. TE) are from adjacent occipital lobe areas such as V4, which is associated with complex visual feature processing. Of course, in newborns V4 is not likely to be active given its delayed maturation and lack of cortico-cortical afferentiation from earlier visual regions arising from the primary striate cortex. Thus, since we know that these regions are active in supporting habituation at birth, the problem is to determine the identity and nature of the signals that reach these circuits in newborns, and then to determine how the resulting activity influences looking behavior.

IT could mediate familiarity-biased looking in one of two ways. It could simply

be a necessary relay for a signal representing the familiarity of the visual stimulus, to influence looking. Or, it could be that IT is the area that produces this familiarity signal (or some combination of the two). Since no familiarity modulations of visual responses have been documented in any areas of the visuo-motor circuit mature in newborns, one must conclude the latter: that the IT/limbic cortices are producing the familiarity signal, and somehow influencing looking behavior by way of it.

Since the normal pathway to TE via TEO, V4, etc. is not an option, we look to alternative visual pathways via the thalamus. In particular, the pulvinar nucleus is a strong candidate on account of the fact that it is mature at birth (Ogren, 1982; Ogren & Rakic, 1981), that portions of it (inferior and lateral – Pi and Pl) contains visual responses retinotopically (Cowey, Stoerig, & Bannister, 1994; Bender & Baizer, 1984; Bundesen, Habekost, & Kyllingsbaek, 2005; Bender, 1982, 1981; Luppino, Matelli, Carey, Fitzpatrick, & Diamond, 1988; OBrien, Abel, & Olavarria, 2001; Petersen, Robinson, & Keys, 1985; Petersen & Robinson, 1987; Grieve, Acua, & Cudeiro, 2000; Williams, Azzopardi, & Cowey, 1995) which are derived from V1 afferents (Bender, 1983), and which include feature-responses such as orientation (Bender, 1982; Petersen et al., 1985). A different portion of it (dorsomedial – Pdm) also reciprocally connects with temporal cortex from birth, though the status of intrinsic connections between Pi/Pl and Pdm are not known. Lesion of pulvinar in adult monkeys produces abnormal scanning of visual arrays, though a loss of the ability to habituate or orient to a novel colored stimulus was not observed. However, saccades in the lesioned subjects were oddly delayed and the primates seemed to fix on the new stimulus and not be able to look away (Ungerleider et al., 1983). Transient subcortical connections to both TE and TEO have also been documented in infant monkeys, and a direct connection between TE and SC even seems to exist in very immature specimens, which disappears as they age (Webster, Bachevalier, & Ungerleider, 1995).

This converging evidence paints a nice picture regarding how visual feature information can reach the temporal lobe in the absence of the feedforward cortico-cortical pathway normally provided by way of the occipital lobe. Not only does the pulvinar receive retinotopic, feature-tuned afferents from the deep layers of V1, it also is known to maintain feature properties, and to project directly to IT in infants. In line with patterns of thalamic projections, this projection should synapse onto the superficial layers of IT (L2/3). IT and limbic regions would both uniquely respond to, and gradually learn to respond less to, stimulus configurations as they were experienced more times.

This could be modelled as random inputs into a large recurrent circuit modelling the IT. IT neurons would thus become responsive to only certain stimuli, based on their happenstance connectivity to various feature-responsive neurons at various retinotopic locations, e.g. one would be responsive to a horizontally oriented line at 2^o left of the centre of the fovea and a 45^o oriented line slightly above that. Whereas another might be responsive to the opposite configuration. This selectivity should be sufficient to uniquely specify most arbitrary stimuli to which infants are capable of habituating. Plastic recurrent connections would sustain the association of similarly active neurons representing the same foveal stimulus, and these would be strengthened alongside greater experience with the stimulus. A further recurrent circuit (e.g. the EC) could even have neurons which sample these higher-level features, to construct even more invariant representations of stimuli, and in the same way as in TE, recurrent plastic

connections with neurons that also sample auditory neurons would build amodal categories. In order for these to be produced, amodal synchrony between the stimuli would be necessary, thus explaining the observed synchrony requirement. Note that the mechanisms described above are simply more realistic implementations of the principle embodied in the model described in Veale, Schermerhorn, and Scheutz (2011).

2.2 Mechanisms for Biasing Looking

The next step is to determine what factors combine to produce the measured looking times in habituation studies. In paired-comparison paradigm studies, do infants 1) show similar fixation lengths to both novel and familiar stimuli (once fixated), but modulate the probability of fixating either one in the first place? Or, are they 2) equally likely to fixate a stimulus, but then either fixate for longer or shorter periods based on the familiarity status of the stimulus?[1] These are empirical questions, and they have not, to the author's knowledge, been examined. However, the resulting answers can be used to infer properties of neural areas and connectivity responsible for the behavior. For example, if there is a bias for infants to look towards familiar or novel stimuli differently in the first place, this implies that there is a function which is able to recognize stimuli in the periphery (and thus modulate the probability of looking at them). The neural circuitry for this would necessarily be complex. This is in contrast to the method used in this paper and the previous model of visual habituation. This method involved modulating peripheral vision salience based *only* on visual features, and then having recognition to come into play only once a stimulus is foveated (modulating the amount of time spent looking at it, for example). A hybrid of the two would involve linearly separable recognition of only visual features of stimuli, which could be biased in the periphery (there is evidence of such separability of features impacting looking time in infants by Cohen, Gelber, and Lazar (1971)). This hybrid method would not work for stimuli that were defined by complex contours, however. As is always the case, more empirical research is needed to decide between the myriad possible explanations.

For simplicity's sake, the current discussion will move forward under the simpler assumption above: that looking time is modulated only by foveal stimulation (relative to peripheral stimulation), and that the peripheral visual field is in principle unmodulated by the familiarity status of its denizens.

The task now becomes the characterization of neural substrates that are mature in newborns and which can perform this modulation based on IT activity from foveal stimulation. Based on tangential evidence relating baseline arousal state to habituation performance in infants (Gardner, Karmel, & Flory, 2003; Geva, Gardner, & Karmel, 1999; Gardner & Karmel, 1995; Karmel, Gardner, & Magnano, 1991; Gardner & Karmel, 1984; Gardner & Turkewitz, 1982), and knowledge of the extensive connectivity of the reticular formation to limbic cortices and subcortical structures (Doty, 1995), and in particular evidence regarding the role of acetylcholine (ACh) in arousal and even in STDP learning (Hasselmo, 2006), we move forward with the hypothesis that reticular-cholinergic-mediated arousal is the mechanism of modulating the probability

[1] This could be tested by using two salience-matched stimuli, familiarizing the subject to one of them, and then using e.g. a forced-choice looking paradigm.

of breaking fixation. Furthermore, the level of this ACh-mediated arousal, and thus the level of modulation, is modulated in a feedback loop by the very limbic and inferotemporal structures that mediate the production of stimulus-responsive representations as described above. This arousal could also be influenced by non-familiarity effects, such as stimulus salience or complexity, or by innate or learned stimulus valence, via other or identical neural pathways. Indeed, this is precisely the mechanism modelled in the simple visuo-motor system described in Section 1, wherein the level of foveal activity modulates the mean/variance of SNr inhibition into dSC – resulting in longer looks on average to more complex foveal stimuli.

There are two possible ways that this arousal-mediated mechanism could influence overt looking. One, it could modulate the probability of dSC firing based on excitatory mechanisms, such as recruitment of nicotinic receptors in dSC via ACh, thus ungating the normally locked sSC afferentation and increasing the probability of bursting in tectoreticular projection neurons (Aizawa et al., 1999; Kobayashi & Isa, 2002; Isa, 2002). Alternatively, it could modulate the probability of dSC firing via disinhibitory mechanisms, since the basal ganglia (CD) are known to receive strong reticular afferents, and also to recieve direct projections from IT areas such as TE; CD is known to have an effect on eye movements by way of SNr (cf. Section 1.4). This latter pathway could actually be implemented without changes in arousal, but simply by way of the direct connection from TE to CD, which shows visual responses and habituation of those visual responses (Hikosaka et al., 1989b). However, the previous observation that (at least via the striatal-nigro-tectal pathway) these are not at play during spontaneous saccades, but only during learned/trained saccade tasks, implies that this is not the pathway at work in newborn infants. The possibility cannot be ignored that other pathways are responsible, for instance CD acting as simply a relay to SNc, RF, etc., which in turn modulate arousal and then influence looking via the excitatory mechanism described above. Further understanding regarding how the arousal mechanism is influenced by CD versus IT activity will need to be examined for an informed decision to be made regarding that portion of the model – in the meantime, the two are not sufficiently at odds to preempt implementation or significant functioning of the model.

Thus, it is possible to paint a very plausible and neurodevelopmentally sound picture of how the visuo-motor system implemented and tested in (Veale, 2013) can be extended to account for both unimodal visual and multimodal habituation. The principles for producing phenomena such as the synchrony constraint, and gaze constraints are presented and tested in the previous work of Veale et al. (2011), thus lending credibility to the hypothesis. However, the proposed neuro-robotic model extends previous results in several important ways. It extends previous neuro-robotic models by providing more realistic visuo-motor circuits, and also a method by which familiarity status of complex stimuli can be learned, and then bias overt looking times to match human and primate data. Being a neuro-robotic model, it extends previous computational models of infant habituation (Sirois & Mareschal, 2004, 2002; Shoner & Thelen, 2006; O'Reilly & Rudy, 2001, 2000) by implementing the gritty details of sensory-interface (in both auditory and visual modalities) with the real world, as biologically realistic neural circuits which lend themselves to direct comparison with empirical data from real organisms.

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