

GROUP SELECTION AND NEURAL ONTOGENETIC CHANGE: IS BRAIN DEVELOPMENT DARWINIAN?

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The brains we possess today are vastly different in connectivity and function from the brains we possessed at birth. The most striking difference is the brain's highly complex, specific, yet surprisingly plastic organization. Each of the many billions of neurons in the brain is capable of making and receiving thousands of connections. As an organism develops, these neural connections become refined and organized into layers, anatomical structures, and functional columns. If arranged in the right way, groups or populations of these neural connections can achieve remarkable functional specificity and plasticity, even without the substantial growth of new neurons (neurogenesis) or new connections (synaptogenesis) in the mature adult (Nicholls, Martin, Wallace, & Fuchs, 2001).

How can this ontogenetic change be explained? Edelman, author of *Neural Darwinism*, argues that we can use a group selection theory to understand how neural connections are formed in large populations. We need this theory to be a selection theory because it is unlikely that genes or the environment could "instruct" each neuron on how to connect and whom to connect with. We need this theory to be a selection theory at the group level because it is unlikely that selection is occurring at the level of a single synapse, and because we want to understand the origins of high-level function such as perceptual specificity, which is the product of the connectivity of many neurons (Crick, 1989; Edelman, 1987). However, because little neurogenesis and synaptogenesis occur in the adult brain, ontogenetic change is likely restricted to synaptic modification of existing neurons (Edelman, 1987; Hull, Langman, & Glenn, 2001). Thus, according to a group selection picture, the brain has solved the immense cognitive problem of perceiving and interacting with a variable environment through the selection of only those groups of neural connections that are functionally adaptive through the Hebbian mechanism of activity-dependent synaptic strengthening and weakening, a mechanism that bears intriguing similarity to Darwinian natural selection.

This paper will evaluate selectionist theories of neural ontogeny, particularly group selection theories, in light of what is currently known about the neurobiology of perception. I will start by examining the facts that motivate Edelman and others to embrace a selection theory of neural ontogeny. I will then discuss whether neural ontogenetic change can indeed be described by a general, non-genetic account of natural selection. I will then explore whether or not the particular breed of selection we find in the brain can be described by a multi-level or group selection theory.

Finally, I will examine whether or not selectionist theories can explain the changes in neural connectivity that result in functional specificity and plasticity within an organism's lifetime, since these changes are what motivate our evaluation in the first place. If they can, then brain development involves more than just experience-driven transformation. If neural ontogenetic change is truly Darwinian, "brains evolve and adapt like large ecosystems" and the process of neural ontogeny may be truly creative, capable of generating novel kinds of connections and perhaps even novel cognitive capacities (Adams, 1998, Johnson, 1996).

I will argue that, as currently understood, the process of neural ontogenetic change lacks vital ingredients and structural attributes required for the process to bear more than superficial similarity to Darwinian cumulative natural selection. Instead, neural ontogenetic changes may more accurately be explained as the product of a hybrid of big-bang³⁶ selection and transformation. I will then argue that although group organization of neural connections is important, and although our selection-transformation hybrid process can account for the functional specificity and plasticity of these groups, selection is not operating at multiple levels. I conclude by sketching a theory of neural ontogeny that I believe is supported by the facts as we know them and speculate about the consequences of a selection-transformation hybrid process for cognition.

Selecting selectionist theories

Many facts about the brain and its development seem to call for a selectionist theory of neural ontogeny. The loss of some connections from an initial set during the course of development requires at least selection in the sense of a "picking out" of certain connections. If one denies selection even in this weak sense, then the connections must be guided by the instruction of some other entity. Edelman thus contrasts selection with instruction, based on his Nobel prize winning work on immunology, which I will briefly sketch. I will then describe the phenomena of functional specificity and plasticity, two important features of neural ontogeny that any theory must explain. I will show how a selectionist theory can explain these phenomena far better than a purely instructional account by applying the theories to a perceptual case, since the neurobiology of vision is well-studied. This explanatory power, combined with success in extending selection to the case of immunology, has encouraged theorists like Edelman to extend a selectionist theory to describe neural change.

According to Edelman, instructional theories are allied to the "notion of an empty animal driven by categories from the outside" (1987). In the case of the immune system, an instructional theory of

antibodies conceives of all antibodies floating around in the bloodstream as possessing the same amino acid sequence, with each antibody folding up in a different way according to its interaction with a unique antigen. The antigen thus "instructs" the antibody in how to fold up. Instead, what actually occurs is selection-driven change. The immune system instead produces a great many antibodies each possessing a different amino acid sequence, each folding up into a unique structure. Antigen-antibody interactions are selective. Only those antibodies that fit the shape of a given antigen can interact with that antigen, and only these antibodies are increased by clonal replication.

Thus, the immune response is not solely the product of genetic or environmental instruction. Though genetic differences create the initial pool of variant antibodies, genes are not required to anticipate which antibodies will fit which antigens in the environment and to guide the replication of those antibodies that will later become relevant. Neither does an environmental instruction account fit the case of immunology. Interaction with a varied environment of antigens is not what produces the variation we find among the antibodies. We have thus established a selection process in a non-genetic system. Edelman's ambition will be to extend this account to the nervous system as well.

A theory of neural ontogeny must help us understand how the remarkable functional specificity we find in the brain originates. Cognitively, for example, it is believed that different areas of the brain are responsible for producing language and processing the meaning of language, and for assessing how dangerous and how rewarding a situation will be (Gazzaniga, Ivry & Mangun, 1998). Examining perception, we find even more incredible specificity. There are neurons that respond specifically to certain orientations of eye gaze, biological versus mechanical motion, subtle differences in line orientation, and even input from the right eye versus the left eye in particular locations in the visual field (Gazzaniga et al, 1998; Goldstein, 2002). To illustrate how functional specificity is achieved, I have sketched a very simplified neural circuit for a direction selective neuron in Figure 1 (from the circuit in Goldstein, 2002). The group of neurons are connected in such a way that neuron E fires action potentials specifically to leftward moving stimuli.

This specificity is very real and difficult to explain without a selectionist theory. If selection is not what determines the connections between neurons, then something else must be "instructing" each neuron on how to connect and whom to connect to. Moreover, it must do this for each synapse in each group of synapses that performs a specific function. While some very general genetic and neurochemical environmental instruction has been demonstrated, it is extremely unlikely that each connection is specified by genes or instructed by the neurochemical

³⁶ The term "big-bang selection" is borrowed from Hull, et al, 2001

environment (Nicholls et al, 2001). Imagine what would happen without perfect instruction. Examine leftward selective group G2 in Figure 1. Only the connection between C and E has been altered. Just changing this connection to an inhibitory rather than excitatory connection results in very weak activity in neuron E, which means that the group's signal in response to a stimulus differs very little if at all from that expected by random noise.

Thus, even slightly imperfect instruction resulting in the change of a single connection in this very simple circuit results in two inescapable and negative consequences. First, it is difficult to see how we can justify calling the neurons in question a group since the group's signal (output from neuron E) is indistinguishable from random noise in the surrounding milieu. There is therefore no product, signal, activity, or function that might confer upon the connected neurons a group identity. Second, any organism with the misfortune of having this pattern of neural connectivity will have a hard time detecting leftward moving stimuli in their environment, whether food or predator. The sheer volume and fidelity of instruction required therefore renders an instruction account highly unlikely.

Selection can account for the persistence and amplification of functionally specific groups of neural connections versus more inefficient connections through the mechanism known as Hebb's law (Adams, 1998). Hebb's law states that if the firing of pre- and post-synaptic neurons is correlated, the connection between them will be strengthened. Synaptically, this strengthening is realized by the presentation of more or different neurotransmitters in the presynaptic terminal and more or different receptors in the postsynaptic terminal as well as certain morphological changes. If this firing correlation is absent, however, the connection between them will be weakened (Nicholls et al, 2001). Imagine that groups G1 and G2 are present in the visual cortex of the same organism and that each of these groups of connections, along with many other groups, was formed fairly randomly. Examine their fates at Times 1 and 2. At Time 1, G1 very clearly signals the presence of a leftward moving stimuli. As the organism interacts with the environment, each leftward moving stimuli causes a well-correlated firing between each pair of neurons and a group output of strong and specific signal. With repetition, the group of connections as a whole is strengthened by Time 2. The particular arrangement of connections in G3, however, is very bad at detecting leftward moving stimuli. Very little correlated group firing will result and the connections between them will become weakened and perhaps, completely nonfunctional by Time 2. Thus, it can be said that selection explains which groups of neural connections persisted, without prespecification or instruction by genes or the environment.

A theory of neural ontogeny must also help us understand the

mechanism responsible for the remarkable functional plasticity we find in the brain. Plasticity refers to the flexibility or modifiability of neural connections that allow an organism to adjust to changes in her physiology, experience, or environment (Bownds, 1999). fMRI has revealed functional plasticity in humans in response to injury and expertise. In humans who are not born blind, but become blind through injury, areas of cortex that formerly supported visual perception now start to "light up" during auditory tasks. A parallel process occurs for those who become deaf through injury. In addition, the area of somatosensory cortex devoted to representing the left hand is expanded in string players proportional to their levels of expertise (Gazzaniga et al, 1998). Direct electrophysiological recordings of the neurons in kittens exposed to abnormal environments also exemplify the limits of plasticity. For example, kittens reared with one eye shut lose the ability to recognize cues requiring the use of two eyes together to perceive depth. Kittens reared in dark rooms illuminated by a strobe light (therefore exposed only to visual "snapshots") became unable to detect movement. In both cases, it was shown that neurons and neural groups did not build the functional specificity that is developed upon normal exposure to the environment (Goldstein, 2002).

These are dramatic examples, but illustrate that functional plasticity and reorganization in the brain do occur and need to be explained. If connections are instructed by genes, we must demand a lot of the genetic code. It must specify a whole range of contingency plans for connection modification. There must be a code for how to adjust connections in the face of a whole host of unpredictable injuries not to mention the idiosyncratic patterns of experience produced by expertise. And if these connections are indeed prespecified by genes, then why do kittens raised in abnormal and temporary environments exhibit such lasting and specific functional losses? Unless one is willing to postulate that the kittens selected for experiment are genetically different from their normally reared and visually normal counterparts, a genetic prespecification account would predict recovery. In other words, once the abnormally reared kittens are exposed to a normal environment, genetics should step in and guide the alteration of their connections to induce normal function. Environmental instruction, on the other hand, while predicting loss, also predicts subsequent recovery. Kittens should be able to regain their lost perceptual abilities once they are exposed to a normal environment since that environment should be able to instruct the wiring of functional connections, just as it does in kittens reared from the start in a normal environment.

Selection can more accurately account for this striking profile of functional variation and plasticity. Let's first examine the case in which neurons that previously responded to input from one modality switch to another modality after injury resulting in blindness or deafness. What

process could result in this outcome? Selection may allow for this kind of functional plasticity by causing the selective weakening of groups of visually important connections that are no longer used and the selective amplification of pre-existing groups of auditory connections that become more relevant. This is analogous to the connections in G1, our direction-selective group (Figure 1), becoming nonfunctional and other hidden connections relevant for auditory processing becoming selectively strengthened (not shown). A selection process can also account for the paradoxical negative plasticity of the kittens in our example. If, during previous selection events, key neural connections fall into disuse, atrophy, and then disappear, no number of future selective events will be able to recreate those connections. Thus, failure to regain lost perceptual abilities makes sense on the selection account, but is mysterious if just genetics or just the environment serves as instructor.

Those holding a selectionist view claim that the brain has solved the immense cognitive problem of representing a variable environment through the selection of only those groups of neural connections that are functionally adaptive. The flexibility selection offers in resculpting functional groups of neural connections makes sense on an ontogenetic timescale. Each organism will encounter a slightly different environment and would benefit from adjustments that can occur within their own lifetimes. This would require of genetic instruction an inordinate amount of fidelity and predictive power. Environmental instruction, on the other hand, should be capable of more plasticity than we find.

A general account of selection

I hope to have shown above that selection in some sense is causing neural ontogenetic change. However, there are different kinds of selection and these differences are important. We should therefore find out what kind of selection most resembles the kind of selection that occurs during neural ontogenetic change, especially since we wish to evaluate the claim that the selection that occurs is Darwinian. First, I will contrast the two major theories of change, one of them selectional and the other transformational. We will see what makes these two processes of change different from one another and to see what is at stake when arguing that neural ontogeny involves one, the other, or a hybrid of both. I will then give a general account of selection that calls for three ingredients. If these ingredients are supplied in a certain way, we may describe the process as one of cumulative selection. If these ingredients are supplied in a different way, we describe the process as an instance of big-bang selection. If not all the ingredients are supplied, we have instead simple one-shot selection. I will show that Edelman's theory does not distinguish between these types of selection. What he sees as selection in the brain is actually a process of

simple one-shot selection followed by transformation, whose stages are cycled according to the structure of big-bang selection. Such an account presents a radically different picture of ontogenetic change than Edelman's account, which would have us see no consequence in equating transformational change with the change produced by truly Darwinian cumulative selection.

To understand what is special about selection, it is useful to contrast it with another theory, the transformational account (see Figure 2). Transformation and variational selection are two different processes that describe how populations can change. Transformational theories are associated with the early 19th century thinker Lamarck and describe changes in the population that result from changes in individuals within the population. Imagine a population of people in a room who all eat lots of French fries and clog their arteries. Thus the population has changed, increasing its overall incidence of heart attacks because each individual in the population has a higher risk of getting a heart attack. Variation describes a different process of change. By the variational account, our French-fry-filled room contains different varieties of people, for example, those who have are heart-attack-prone and those not-at-risk. A change in the population level of heart attack risk could then be brought about by changing the frequency of each variation, for example, if all the heart-attack-prone in the room became cardiac-jingoists and kicked out the not-at-risk. This would create an increase in the frequency of the heart-attack-prone type and a decrease in the not-at-risk type and result in an overall change in the population without change to any given individual, but by the selection of certain types of individuals. Thus selection is a variational, not transformational explanation of how change occurs in a population.

Though gene-based biological evolution is the paradigm of selectionist theories of change, here we need an account of selection that is general enough to include non-gene-based systems, but that still captures all the essential features of the process. The three ingredients required for selection according to Darwinian theory are: (A) variation in fitness in a population (B) inheritability of variation and (C) differential reproduction (Hull et al, 2001; Levins & Lewontin, 1985; Sober & Wilson, 1998). These three ingredients, when combined and repeated cyclically ABCABCABC, embody the process of cumulative selection we see in gene-based biological evolution, also called "natural selection." Individuals within a population vary in fitness and these variations are heritable and lead to differences in number of offspring produced. This cycle repeats itself as future generations experience further variation due to imperfect inheritance and mutation events and produce their own offspring. If variations did not affect fitness, differences among members in a population could not be "seen" by the selective forces in the environment. Without heritability, initial variation would have no consequence or effect on future variation. Without differential reproduction, any change in the system is simply a result of the combined transformation of each individual in the system. Without iteration, systems could not accumulate changes that may eventually result in complexity and

...contin, 1985; Hull et al, 2001; Godfrey-Smith, 2003). Big-bang selection requires all the same ingredients, but does not include the iteration of cumulative selection, instead requiring only “one round of mutation and selection, followed by continuous selection” (Hull et al, 2001). This is the kind of selection Edelman observed in the immune system. Almost all antibody variants are generated once in an initial “big bang” early in ontogeny, and each exposure to an antigen serves as a successive selection event that results in differential reproduction. Thus, all three ingredients are present, but are related to each other in a different way. Ingredient (A), variation, occurs early in ontogeny, but ingredients (B) and (C), inheritance and reproduction, cycle continuously as exposure to different antigens causes the clonal replication of relevant antibodies. This type of ABCBCBC process is not capable of generating complexity or novelty and may eventually run through its initial pool of variation, but does result in changes in the frequency of types in a population over time.

Simple one-shot selection describes the processes that involve selection in the most general sense. It requires only two ingredients, (1) variation of some kind and (2) differential extinction. To illustrate this kind of selection, imagine finding a box of Popsicles of several different flavors sitting in the noonday sun. You prefer cherry flavored Popsicles, so you move only the cherry flavored Popsicles from the box out in the sun to your freezer. All the remaining, non-cherry flavored Popsicles melt away. This is simple one-shot selection in the absence of reproduction or inheritance and is often what is meant when using “selection” in everyday language. Some types “survive” and other types “go extinct” and that’s the whole story (Godfrey-Smith, 2003). One-shot selection is selection in the most obvious and weakest sense. Like big-bang selection, it is a process without a source of replenishable variation, thus incapable of generating complexity or novelty. Unlike big-bang selection, it can create changes in frequency only through differential extinction since there is no reproduction.

The kind of selection that occurs in the nervous system does not match any of these kinds of selection exactly. We have ingredient (A) in the variation in neural connectivity that results in functional differences (and fitness differences, by extension) due to Hebbian mechanisms that alter connectivity strength. However, it is widely believed that no functionally significant neurogenesis or synaptogenesis occurs in a mature brain (Crick, 1989; Edelman, 1987; Hull et al, 2001; Nicholls et al, 2001)³⁷. If neural selection is to match either cumulative or big-bang selection, how is it to account for ingredients (B) and (C), inheritance and reproduction? Edelman writes that the three essential features shared by all selection theories are:

...variable repetitions of elements whose sources of variation are causally unrelated to subsequent events of selection or recognition, opportunities for encounter with an independently changing environment permitting the selection of one or more favored variants, and, finally, a means of differential reproduction or amplification with heredity of the selected variants in a population.

— Edelman, 1987

This quote shows us precisely where he parts ways with the general account of natural selection laid out earlier—he does indeed recognize the three ingredients shared by cumulative and big-bang selection, but allows *amplification* to substitute for reproduction. In equating amplification and reproduction, Edelman is allowing what is essentially transformational change to stand in for variational change.

This substitution of amplification for reproduction violates cumulative and big-bang selection strictly understood and is not fully in line with simple one-shot selection either. Iteration through successive rounds of reproduction is what provides the process of cumulative selection with its capacity to generate complexity and novelty. Big-bang selection also involves the differential reproduction of selected variants. The amplification substitution sits uneasily in a theory of simple one-shot selection as well, since it involves a change over and above simple differences in rates of survival and extinction.

The analogy between amplification and reproduction can only go so far. If we accept that little functionally significant neurogenesis or synaptogenesis occurs in a mature adult, the amplification Edelman mentions must refer to the changes in synaptic strength. Changes in synaptic strength, in other words, changes in the strength of inter neural connections, refers to an increase in correlated firing. This increase is achieved by the elaboration of a synapse during LTP such that (1) the pre-synaptic terminal releases more neurotransmitter or a different profile of neurotransmitter or (2) the post-synaptic terminal presents more receptors or less conservative receptors to increase the response of the post-synaptic neuron or (3) both (Adams, 1998; Nicholls et al, 2001). Notice, however, that the change described by amplification is created by the production of more or different neurotransmitters or receptors, not the reproduction of more synapses. This is production, not reproduction. It is analogous to reproduction in that the increase in synaptic strength may be equivalent to the increase that would result if the synapse had indeed replicated. However, one would be hard pressed to distinguish between virtual reproduction and simple transformation (Levins & Lewontin, 1985).

The kind of selection that occurs in the nervous system most resembles a hybrid of big-bang selection and transformation. Like big-bang selection and one-shot selection and unlike cumulative selection, all

³⁷ Although a neurobiologist will decide whether and when we should lay to rest the possibility that there may be important neuro- or synapto-genesis in the mature brain.

or almost all of the variation arises early in ontogeny. However, changes in the population over time are not the result of differential reproduction, as in big-bang or cumulative selection; nor are they the result only of differences in survival and extinction as in one-shot selection. Instead, they are the result of differential survival/extinction combined with differential amplification or strengthening of certain connections, which I argue is a transformational process. Thus, the timing of variation matches that of big-bang selection, followed by a process that includes differential survival/extinction and the transformational change of surviving connections.

Multi-level selection

Given this hybrid picture of selection and facts about the brain and its development, I will now explore whether neural ontogeny can be described by a multi-level selection theory in the Sober and Wilson sense that selection is pulling in different directions at the individual and group levels. I will examine the case of a simple perceptual circuit and conclude that, at least in this case, what is occurring are two different selection events, and not selection at two different levels. One can therefore view the circuit as either an individual or a group of connections, interchangeably.

In chapter three of *Unto Others*, Sober and Wilson (1998) offer a three-step procedure for discovering selection processes that operate at multiple levels.

Step 1: Determine what would evolve if group selection were the only evolutionary force.

Step 2: Determine what would evolve if individual selection were the only evolutionary force.

Step 3: Examine the basic ingredients of natural selection at each level.

For the purposes of this analysis, I will define groups as collections of neural connections (G1-3 in the appendix) that function to transmit signals distinguishable from random noise in the system and individuals as each neuron in the group on its own. Starting with Step 1, if group selection were the only evolutionary force, traits that maximize the fitness of the group will be the traits that are selected. Therefore, group selection favors the right kinds of connections arranged in just the right way, resulting in a consistent pattern of firing to detect, for example, leftward movement. The stronger the connections between the members of the group, the better the group is at performing its function – transmitting a group signal strong enough to be heard over random noise in the system; neurons that do not meet this criterion hardly constitute a group at all, as they are indistinguishable from

the surrounding milieu. Moving on to Step 2, if individual selection were the only evolutionary force, those traits that maximize the fitness of the individual will be selected. Individual neurons will therefore want to strike a balance between stronger connectivity, which lead to an increase in correlated interneural firing, and non-maximal connectivity, since each time an individual neuron fires, it puts itself at metabolic risk. The copious amount of metabolic resources required for a neuron to process and send along a signal means that if a region is short of resources at any timepoint, those neurons that are firing the most will sustain damage or death while quieter neurons will be left intact, free to enjoy the extra resources no longer needed by their overzealous neighbors. If a synapse is too quiet, causing a significant decrease in the correlation of interneural firing, by Hebb's law the connection is in danger of ceasing to exist – and a completely unconnected neuron dies (Nicholls et al, 2001).

Thus, it would appear that selection may be operating at multiple levels here. We have components that can be analyzed as individuals and as groups. In addition, selection seems to favor different traits at different levels. By Step 1, we should see strong connections and as many connections as required functionally if group level selection were the only evolutionary force. By Step 2, if individual level selection instead were the only force, we should see only a minimal number of connections between neurons and connections of non-maximal strength. To see how this would play out, let's look at our leftward detection circuit (G1, Figure 1). Selection at the group level will favor the strengthening of all functional connections (A->C, C->D, C->E, B->D, D->E). Selection at the individual level, however, would predict that neuron C, which must do twice the signaling work compared to any other neuron, will either die or downgrade the strength or number of its connections.

Step 3, however, directs us to examine the basic ingredients of natural, or cumulative selection, including heritability of phenotypic differences. By our previous analysis, we know that we will not find all of those ingredients in their standard form. How can it be that Steps 1 and 2 lead us to believe that we will find selection operating at multiple levels while Step 3 dissuades us from that idea?

A closer look at our neural circuit reveals that we can reconceptualize our case as including two selection events or tests and not selection at two different levels. The first selection event is a test of viability, the second is a test of functionality. The group, and by extension, each of its individual components, must pass both tests. During each test or selection event, what is being evaluated is the group. In our leftward detection circuit example, it is obvious that it is the group

that is evaluated during the test of functionality selection event because only those groups that perform the function of leftward detection are selected. To get to this test event, however, the group as a whole must also pass the test of viability; the group as a whole must survive over time. In the test of viability, it is true that neuron C is at the most metabolic risk, but if it or any other neuron dies, the whole group suffers. A group whose members do not all pass the test of viability will fail the test of functionality, and vice versa. Thus, our initial observations are predicted by two different selection events or tests. The test of functionality favors strong connections and the test of viability favors minimal connections of non-maximal strength. It is the group as a whole that must achieve a balance between the requirements for both selection events.

That selection is occurring not on multiple levels, but in multiple events, lets us view the neural circuit in our example as either an individual or a group of neural connections, just as we may view a sexual, multicellular organism as an individual or a group of organs, cells, or genes. To highlight this reversibility of perspective, I will use the example of a peacock (Sober, conversation, 2003). Building a huge tail helps the peacock during mating selection events, but harms it during viability selection events. A large, colorful tail will attract females, increasing the peacock's reproductive success, but will also attract predators and make escaping from predators harder, thus decreasing the peacock's viability. During both events, we can view the peacock as an individual organism or a collection of genes, one of which determines how large of a tail gets built. Similarly, a strong and well-sculpted network of connectivity between a group of neurons will give the group a better chance at transmitting a group signal, thus helping the group pass the test of functionality, while also placing the group at increasing metabolic risk with increasing functionality or firing, thus decreasing the group's long-term viability. During both events, we can interchangeably view the interconnected neurons as an individual neural circuit or as a group of individual neurons.

A theory of neural ontogeny

I will attempt here to sketch a preliminary description of neural ontogeny that I believe is supported by the facts as we know them and then speculate about the consequences of a selection-transformation hybrid process of neural change in thinking about the possibilities and limitations of cognitive change.

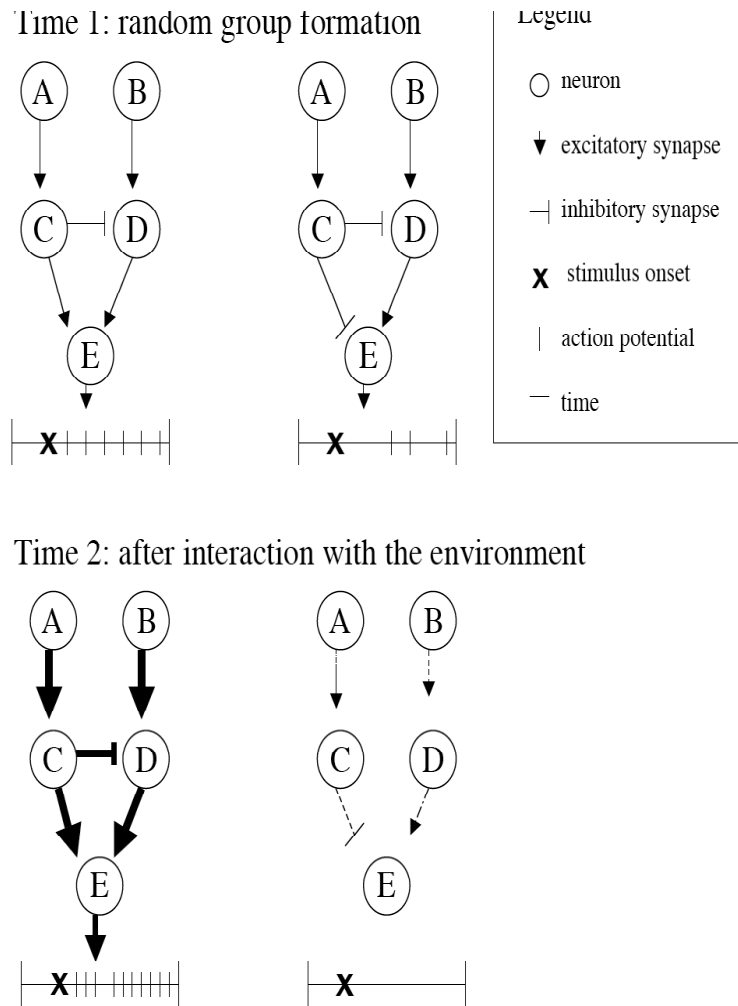
Neural ontogenetic change involves non-cumulative non-Darwinian selection. The brain has solved the immense cognitive problem of representing a variable environment by the differential survival and amplification—not reproduction—of those groups of neural connections that are functionally adaptive. This process of one-shot selection and

transformation is then cycled over and over within an initial pool of dwindling variation in accordance with what can be described as a 'big-bang' structure. Contrary to earlier intuitive assumptions, selection is not operating at both the individual and group levels that competitively pull in different directions. Instead, we find that selection operates through multiple tests of survival and functionality at a single level, allowing us to interchangeably view the brain as consisting of groups of neurons forming neural or functionally-defined individual neural circuits balancing the same set of shared costs and benefits.

What are the consequences of this special process of neural ontogenetic change? Just as the big-bang structure of antigen selection illuminates our understanding of the limitations and possibilities of ontogenetic change within the immune system, so too might our examination of the processes that drive change in the brain illuminate our understanding of the limitations and possibilities of cognitive change.

If we accept the idea that brain development is not truly Darwinian, must we also give up the idea that brains evolve like populations do, with novelty of form and function arising just as in a population, but within an individual organism's lifetime? If we do view neural ontogenetic change as primarily a process of transformation, the subsequent picture of neural and cognitive change is not necessarily devoid of the potential for change. Perhaps initial variation is so massive, for example, that the raw material for selection and transformation will not run out during the course of an organism's lifetime. One can also imagine that a source of renewable variation, mutation, or change might be an undesirable destabilizing force. As Hull et al (2001) state, "selection processes are also incredibly wasteful and inefficient. They can generate complexity and novelty primarily because they are so wasteful and inefficient." An early period of selection followed by transformation may provide an organism with the best of both worlds. The individual would be able to benefit from environment-driven novelty within her lifetime, while able to then cement that change through the more stable and efficient transformational process of synaptic amplification. Such a picture confers a special role to early experience in shaping the brain, since early experience drives selection during a time of peak variation. As for origins of this idiosyncratic method of ontogenetic change in the nervous system, I hope that the sketch laid out in this paper may help guide those intrepid enough to tackle this even deeper mystery.

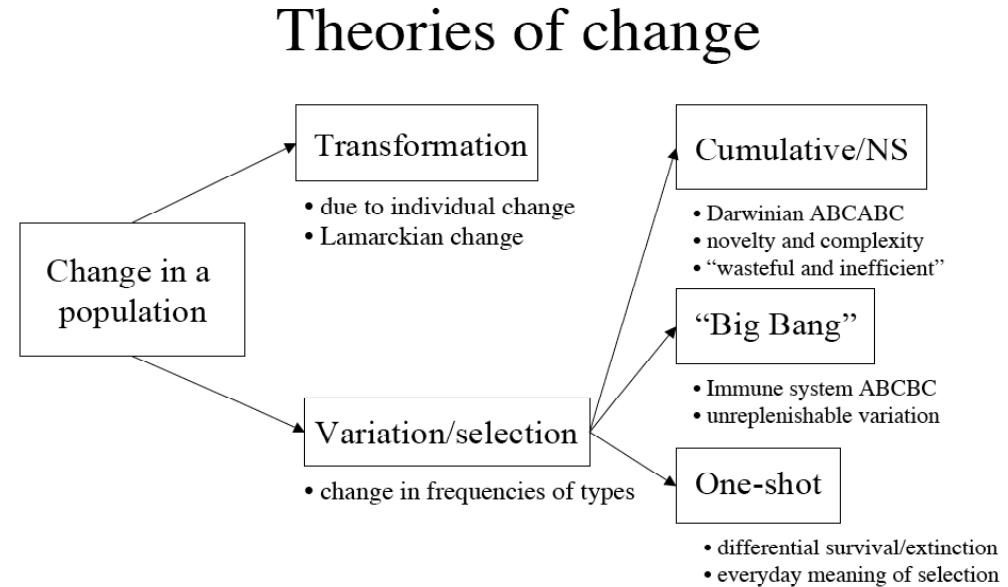
Figure 1:



G1: As a stimulus moves to the right, it first stimulates receptor neuron A, which excites C, which inhibits D. Meanwhile, the stimulus continues moving rightward and excites B, which excites D. However, D is already inhibited by C, and E will not fire any action potentials. If a stimulus moves left, however, it stimulates B first, which excites D, which excites E. As the stimulus moves left, it excites A, which excites C, which is too late to inhibit D, which still excites E. The combined excitation from C and D causes E to fire.

G2: As a stimulus moves left, it stimulates B first, which excites D, which excites E. As the stimulus moves left, it then excites A, which excites C, which is too late to inhibit neuron D, but which inhibits E. E does not fire, and thus, no correlation in firing between D and E, a weakened connection between them, and eventually, throughout the entire group.

Figure 2:



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AN INTERVIEW

AN INTERVIEW WITH MARTHA NUSSBAUM

Fall 2004

University of Chicago

Each year The Dualist includes an interview with a modern philosopher chosen by the staff. This year, Martha Nussbaum graciously agreed to answer questions posed by The Dualist and the Stanford Philosophy Department. Professor Nussbaum's wide-ranging interests have included ancient theories of ethics, the idea of fiction as a form of moral education, and the reconception of global justice in terms of basic capabilities. Her most recent book is Hiding From Humanity: Disgust, Shame, and the Law (Princeton: Princeton UP, 2004), and her new book, Frontiers of Justice: Disability, Nationality, Species Membership, will be published in fall 2005 by Harvard University Press.

David Hills:

Your insistence on the multiplicity, incommensurability, and frequent incompatibility of the things we humans properly and urgently value, and your appeal to literary fiction as the place where the resulting moral complexities are most honestly and comprehensively faced – both these things connect you to a stream of modern and mostly British ethical thinking originating (I suppose) in Anscombe's "Modern Moral Philosophy" and including such diverse figures as Isaiah Berlin, Iris Murdoch, Stuart Hampshire, and Bernard Williams. Yet this stream of thought is noticeably more hostile than you yourself are to efforts to construct general theories of the good or the right, rooted in general accounts of the capacities and vulnerabilities that go to make up our shared human nature. It views welfare economics and general theories of justice as dangerously utopian. Indeed it often agrees with Machiavelli and Hobbes that the most we can sensibly ask of the state is that it prevent interpersonal conflict from taking certain especially disastrous forms: war, famine, economic depression, genocide, and the like. How do you view the overall shape of your debts to and departures from this modern tradition of ethical antitheory (if I can call it that)?

Martha Nussbaum:

Because I believe that it is very important to distinguish ethical from political deliberation, let me address these two separately, ethics first. In my view, there is no single tradition leading to the revival of interest in virtue ethics. There are some thinkers for whom the main motivation to return to a neo-Aristotelian type of ethical thought is fundamentally anti-Utilitarian. These thinkers insist on the heterogeneity and incommensurability of values, the need for careful consideration of complexity and context in good decision-making, and the intrinsic worth of virtuous action. They are also likely to insist that we may rationally deliberate not only about means to ends, but about the constituents of the end itself. One aspect of Aristotle to which they thus attach great importance is his account of