# Consciousness, A Darwinian Process

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**Abstract**

We start at the level of a bacterium where an observer feature associated with foraging measurements motivates introduction of a notion of primitive awareness (a proto-consciousness) as a dualist construct. The relative simplicity of Darwinian concepts at the level of a bacterial colony then lead to development of an analytic theory of perceptual consciousness that includes colony notions of qualia, intentionality, and self. We extend this approach, first to a neuron and then to a neuronal assembly, each extension accompanied by a derived awareness construct. For each construct (dualist or derived), a mathematical model in the form of a measurable quantity called a token is developed. The pairs, token and construct permit the design of experiments that would validate the theory. Finally applications of the theory are developed, each based upon the mathematical model (the tokens) and each explaining a familiar aspect of human consciousness.

## 1. Introduction

A dualist approach is used to develop an analytic theory of perceptual consciousness. Darwinian concepts (Darwin (1859)): competition for nutrient, survival of the fittest and reproduction are used. A principle difficulty in studying human consciousness is its complexity[1]. So we start with a version of the theory for a bacterium (Margulis (2001)) and a bacterial colony (Ben Jacob et al (2004), Waters, Bassler (2005)), introducing a notion of primitive awareness (a proto-consciousness) as a dualist construct in this simpler context[2]. A construct (dualist or derived) is complimented by a mathematical model, having the form of a measurable quantity called a token. Tokens allow for specification of experiments that would validate the approach. The colony analogs of the aspects of consciousness that are developed bear only a remote resemblance to their human level counterparts that are subsequently developed, but it is just this simplification that is key.

### 1.1 Survey

In Sect. 2 we develop the bacterial case. We model the locomotion of bacteria that is based upon nutrient concentration measurement differentials (Curtis, Barnes, p. 131 (1989)), and we introduce a notion of fitness that characterizes bacterial ability at foraging for nutrient. A virtual gene that records the fitness, that is, that records progress in the competition for survival is used. An observer feature is required to accompany a physical measurement (Stapp (1999)), and we introduce a dualist construct that supplies one (Margenau (1950, 1978)). The construct motivated by those foraging measurements is interpreted as a notion of primitive awareness (a proto-consciousness). It is introduced in association with bacterial fitness, and its extension to awareness at the colony level is developed (Velmans (2000)). We show how the dualist construct coupled with Darwinian features of competition, survival of the fittest and reproduction enable development of colony level notions of qualia, of intentionality, and of self. The last informs a connection to Cartesian dualism as well as a notion of pan-psychism for living forms whose survival is informed by Darwinian principles. Along with its simplicity, it is the modest resemblance of features of colony consciousness to human level counterparts that facilitate the development.

In Sect. 3 we deal with the neuronal case. The notions of foraging for nutrient, of fitness (along with an associated dualist awareness construct), and of reproduction are extended first to a neuron and then to an assembly of neurons. Nutrient flux is replaced by neurotransmitter flux. Foraging, that is motion in space is replaced by motion in synaptic weight space. This brings with it an increase of as much as five orders of dimension magnitude, which speaks to the subtlety to be found in human consciousness. A bacteria's virtual gene is

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[1] An indication of this complexity is portrayed in the varied collection of literature on the subject, a sample of which is: Chalmers (1996), Churchland (1984), Dennett (1996), McDermott (2001), Penrose (1994), Stapp (1998), Velmans (2000)...

[2] See Sheets-Johnstone (1998) for discussion of consciousness without nervous systems.

replaced by a neuron's virtual chromosome, the latter composed of the neuron's synaptic weights. Competition and reproduction in the colony are replaced by neuronal inhibition and the opportunity for a neuron to fire. Then the features of a quale, of intentionality, and of self for the neuronal case are developed employing Darwinian aspects analogously to the colony case. Now the resemblance to human level features of consciousness is more direct.

In Sect. 4, we show that the colony's awareness vanishes with the disappearance of nutrient. This prototype application of the theory facilitates development of the following applications at the neuronal level that are relevant to human consciousness. Each application suggests an experiment (as described in the following Sect 1.2) that contributes to validation of the theory.

1. The inability to experience certain qualia in the absence of sensory input.
2. Answering the question: Why does the brain locate the perception of a quale (such as pain, taste, smell) in the peripheral nervous system? We confirm as well, the existence of a threshold for such a perception.
3. A corollary of 2: How do visual and auditory imagery come to be located 'out there' in the surrounding space?
4. How do dream images come to be located in a seemingly virtual space?
5. Hallucinations, phantom pain.
6. The threshold process involved in unconscious sensing (pheromones).

The theory informs the evolution of consciousness, such as its enhancement or deterioration as the case may be. We conclude Sect. 4 with relevant comments.

### 1.2 Tokens, experimental verification

The theory makes use of an observer quality, a primitive awareness (proto-consciousness) introduced as a dualist construct. Other constructs are derived as ramifications of the basic level of awareness (Llinas (2001)). (A derived construct is perforce not dualist.) Each construct is complemented by a measurable quantity called a token (Miranker (2000, 2006)). A token is observable but unconscious, while its corresponding construct is unobservable but conscious. Each token is specified by a specific mathematical model, a formula that expresses it in terms of measurable physical quantities. These formulas could form the basis of experimental verification of the theory at the human level. Indeed measuring that there is adequate strength in the value of a token makes for the theory's prediction of a feature of consciousness corresponding to the token's associated construct. That prediction could be compared to the experience of the subject. We do not know how to learn of experiences of non-human connectionist systems (living or machine), so we might apply this token/construct procedure to them to motivate study and speculation on the nature of such experiences.

## 2. Awareness in a Colony of Bacteria

**2.1 Bacteria: nutrient supply, measurement, colony population**

Bacteria are imbedded in a nutrient bath, whose concentration $n(x,t)$ represents information critical to bacterial survival. To process this information, bacteria make measurements to which they respond by changing location in an attempt to improve nutrient supply (Curtis, Barnes, (1989)). We introduce a dynamic fitness quality called *a,* which records the current signed performance of a bacterium in making a nutrient supply improvement (Damasio (2003)). We also introduce a virtual gene (whose name and value are both called *g*) that records fitness cumulatively (see (2.8)) and that grades the bacteria's motile behavior.

**Measurement:** To characterize motility, a bacterium is modeled as a rod-like object of length *d*. Its two ends are denoted *r* and *l*, respectively. Let $x_r, x_l$ be the locations at time *t* of the *r, l* ends, resp. of a bacterium. Measurements $n(x_r,t)$ and $n(x_l,t)$ at the ends are made by the bacterium. (To complete any measurement process, we take it that an observation of the measured values must be made. This feature, central to the development of the primitive awareness dualist construct will be described in the following Sect. 2.2.) The difference and mean of the values of these measurements are computed:

2.1) $$\Delta n(t) = n(x_r,t) - n(x_l,t),$$

2.2) $$\bar{n}(t) = \frac{n(x_r,t) + n(x_l,t)}{2}.$$

**Motion:** The change in position of a bacterium from $x(t)$ to $x(t+1)$ is described as a displacement proportional to $g\Delta n$ plus a rotation. To specify $x(t+1)$, first consider the rectilinear displacement

2.3) $$y(t+1) = x(t) + \alpha g z \Delta n,$$

where $\alpha$ is a scaling factor, and

2.4) $$z = \operatorname{sgn}(\Delta n)\frac{x_r - x_l}{d}$$

is a unit vector. Next let *R* be the rotation matrix $\begin{vmatrix} \cos 2\pi\theta & -\sin 2\pi\theta \\ \sin 2\pi\theta & \cos 2\pi\theta \end{vmatrix}$, where $\theta$ is chosen uniformly at random from the unit interval. Then $x(t+1)$ is given by

2.5) $$x(t+1) = R(y(t+1) - \bar{y}(t+1)),$$

where $\bar{y}(t+1) = \frac{1}{2}(y_r(t+1) + y_l(t+1))$. $y(t+1)$, $y_r(t+1)$, and $y_l(t+1)$ are computed from (2.3). So a bacterium jumps along its length a distance specified in (2.3) followed by a random rotation centered at $\bar{y}(t+1)$ (specified in (2.5)).

**Colony population and nutrient supply:** Consider a colony of bacteria. Let $p(x,t)$ denote the number of bacteria at position[3] $x$ at time $t$. Let $\beta$ be the quantity of nutrient consumed by a bacterium in unit time. Then

2.6) $$n(x,t+1) = n(x,t) - \beta p(x,t) + n_e(x,t),$$

where $n_e(x,t)$ is an exogenous (signed) influx of nutrient[4].

**2.2 Fitness token, dualist construct, awareness hypothesis and conventions**

With movement, the fitness of a bacterium is taken to be

2.7) $$a(t+1) = \text{sgn}\left[\frac{\bar{n}(t+1)}{\bar{n}(t)} - 1\right], \text{ with } \text{sgn}0 = 1.$$

We say that a bacterium is fit or not-fit at time $t$ if $a(t) = 1$ or $-1$. $a(t)$ is a signature-valued indicator of change in the bacterial nutrient supply as a result of motion. The process summarized by (2.7) is taken as an observation of the nutrient measurement values anticipated in Sect. 2.1 Measurement. This process, taken as a dualist construct, is hypothesized to be an awareness quality (a proto-consciousness) of nutrient supply change by the bacterium[5]. Motivation for this choice comes from the observation that the signs of $a$ correspond to alternative responses of a bacterium, to behavior favoring/disfavoring survival.

A signatory choice for fitness is not a limitation, since the value $g$ of the gene records bacterial fitness history, namely

2.8) $$g(t+1) = g(t) + a(t+1).$$

Moreover, it is in terms of $g$ that bacterial mobility is graded (see (2.3)). Note the correspondence between this observation/awareness notion and the one supplied by Hebb's law in the neural network consciousness model of Miranker (2005).

**Conventions: processes and their tokens, awareness**

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[3] Allowing more than one bacterium at a point $x$ is an inessential idealization, adapted for convenience. The idealization also offers compatibility with the neural assembly model of Sect. 3.

[4] A supply of nutrient entering within the colony is allowed for reasons of compatibility with the treatment of neuronal assemblies to follow in Sect. 3. In the colony case, one could take $n_e(x,t)$ as vanishing everywhere except on the periphery of the colony.

[5] Measurement and observation of the measured value is taken as a fundamental pairing in modern physics. This duality has entered the study of consciousness (Miranker (2001, 2002) and Stapp (1998, 1999)).

1.The dualist awareness process just described will be denoted by $\hat{a}$. $a$, the fitness value itself (specified in (2.7)), is termed a token[6] of the process $\hat{a}$.

2. Throughout, a token and its associated process will be given the same label, except that the latter will have a hat (as with the pair $(a, \hat{a})$).

3. A quality of being aware/not-aware is attributed to a process if its token is positive/negative (i.e., the awareness equals/not-equals $\varnothing$). At the bacterial level, being aware/not-aware is equivalent to being fit/not-fit.

**2.3 Population changes, causality, changes in *g* upon reproduction**

$p(x,t)$ changes due to three effects, (*i*) motion, (*ii*) reproduction, and (*iii*) competition for survival.

***i*) Motion:** From (2.5), we see that a point $x$ in a bacterium is displaced to $x(t+1)$ if $x(t+1) = R(y - \bar{y})$, the variables being relevant to that bacterium. For convenience we consider a bacterium as located at its midpoint.

***ii*) Reproduction:** Bacterial reproduction proceeds by binary fission, and the resulting daughters are assigned fitness values at random from among the four possibilities, $\{\pm 1, \pm 1\}$, except that the assignments $(1,1)/(-1,-1)$ are denied to the daughters of a not-fit/fit mother. This genetic protocol embodies a mutation effect. Of course, the fitness may change as soon as the next bacterial displacement. (A possible inheritance variation, not pursued here, is to allow $a$ to have fractional values and to randomly partition the fitness among the daughters, while maintaining sign and conserving the total fitness.)

***iii*) Competition:** There is enough nutrient at $x(t+1)$ for

2.9) $$s = \left\lfloor \frac{n(x,t+1)}{\beta} \right\rfloor$$

bacteria to survive a unit time. If there are $f$ fit bacteria at $x(t+1)$, $u = \min(s, f)$ of them survive. Naturally, bacteria that don't survive are eliminated from the colony. Why might fitness (as defined) be causal in the propensity for survival? The heuristic is that a bacterium moving from poorer to richer nutrient supply is better adapted at consuming, and reversely. We see this commonly in higher animals, including man. Hungry individuals tend to be voracious, and by comparison, well-fed individuals tend to be relaxed about consuming food.

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[6] Classical mechanics supplies an analogical example of such a pair. The pressure (a measurable physical quantity) is a token of its dualist construct, the specific force (the latter a concept specified in terms of the dualist constructs of mass, space and time).

An alternative survival protocol is to allow some of the not-fit to survive. In this case $u = f$, so that first of all, all the fit survive. Then there is adequate nutrient for $v = \max(0, s-u)$ of the not-fit to survive also. Recall that fit/not-fit bacteria are aware/not-aware. If the number of aware and/or not-aware bacteria exceed the numbers $u$ and/or $v$, resp., make a random choice for the $u$ fit and/or the $v$ not-fit survivors, resp. Variations on this procedure (for example, allowing access to nutrient to depend stochastically on fitness) as well as study of such survival issues as bacterial competition for space is left for further study.

**Population dynamics:** Combining these three effects, we have

2.10) $$p(x,t+1) = \min\left[ f, s, \sum_{x(t)} \{p(x,t)\}_{x(t+1)} \right].$$

Here the subscript $x(t+1)$ denotes, for an occupied location, that fraction of the $p(x,t)$ bacteria that are displaced from $x(t)$ to $x(t+1)$. In the case of the alternative protocol, the symbol $f$ is dropped from the bracket in (2.10).

**Causality:** If reproduction, guided by the fitness $a$, is viewed as guided alternatively by the awareness $\hat{a}$, we see that the latter may be regarded as causal.

**Updating the value of the gene, recording of fitness/awareness, Darwinism:**

With fission, the value of the gene is taken to change as follows.

2.11) $$g_{daughter} = g_{mother} + a_{daughter} + v\chi .$$

In (2.11), $v$ is a scaling factor, and $\chi$ could be taken as normally distributed random variable with zero mean. Then $v\chi$ is a mutation term. From (2.7) and (2.11), we see that the cumulative fitness/awareness competitive reproductive history is recorded in the gene.

Referring to *(ii)* reproduction, we see the tendency for fit/not-fit progeny to improve/degrade in motility. Referring to *(iii)* competition, note that the causal nature of fitness/awareness informs a *Darwinian* survival mechanism

**2.4 Sensory input: quale density, intentionality, self, mirroring**

We now indicate how our model supports concepts of a quale, intentionality, and self. These concepts are usually set in the context of human consciousness. Their analogs for the colony of bacteria will be far simpler variants, bearing in each case only the most modest resemblance to a human level counterpart. We suggest there is an advantage to this, since the complexity of the latter stymies progress in our understanding of such features of consciousness.

***a)* Hypotheses: colony awareness, a quale defined, migration, mirroring:** The cooperative, competitive development of the colony (described in Sect. 2.3) represents a colony-wide awareness of its nutrient supply. In particular, we take

2.12) $$A(x,t) = \sum_{\text{bacteria at } x} a(t)$$

as the awareness density token of the colony. Unlike $a(t)$, $A(x,t)$ is not taken with a signatory value, since we anticipate a need for gradations in awareness at the colony level. The colony awareness density construct itself is

2.13) $$\hat{A}(x,t) = \bigcup_{\text{bacteria at } x} \hat{a}(t),$$

Characterization of the $\cup$–symbol for combining awareness in (2.13) (and elsewhere) is implicit. For discussion of how awareness/consciousness agglomerates, see Miranker (2001). With the constructs $A(x,t)$ and $\hat{A}(x,t)$, the colony can be viewed as functioning as a multi-cellular organism as in quorum sensing (Waters, Bassler (2005)). We expect the colony to migrate towards its nutrient supply[7] in the sense that $p(x,t)$ will develop to approximate $n(x,t)$, up to a scale factor. We describe this as a *mirroring* of nutrient, $n(x,t)$ by population, $p(x,t)$. Then we interpret $p(x,t)$ as a physical quantity associated with the colony awareness density, $\hat{A}(x,t)$. We shall view $p(x,t)$ and $\hat{A}(x,t)$ as dual; $p(x,t)$ being an externally observable quantity, $\hat{A}(x,t)$ an externally unobservable quality. With this convention, we say that $\hat{A}(x,t)$ (awareness)/ $p(x,t)$ are sensations/sensors of nutrient that at the level of response, corresponding to human perception, we might by way of example call aroma/olfaction. That is, $\hat{A}(x,t)$ is an experiencing of its dual $p(x,t)$, in turn, of $n(x,t)$ (the latter mirrored by $p(x,t)$). These observations amount to a positing that a quale density is a pair $(p(x,t),\hat{A}(x,t))$, where $p(x,t)$ is a token of the quale and that $\hat{A}(x,t)$ is the experiencing of it. We summarize these features in the schematic (2.14), where for convenience the term density is omitted. Note that if the dualist features (the terms with hats) are denied the bacteria, they become so-called zombie bacteria.

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[7] In one dimension, the equation of motion (2.3) becomes

$$x(t+1) = x(t) + \alpha g(t)\Delta n,$$

since rotation $R$ and unit vector $z$ may be discarded. Using (2.8), we rewrite this as

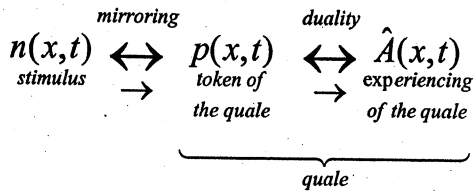
$$\Delta x = \alpha[a(t) + g(t-1)]\Delta n.$$

The mirroring follows by taking the time average, since an informal calculation gives

$$\overline{\Delta x} = \alpha a(t)\overline{\Delta n},$$

since $\overline{g} = 0$. Since the fit bacteria $(a(t) = 1)$ tend to survive, the last expression shows that their displacements and the nutrient concentration changes tend to proportionality.

2.14)




***b*) Intentionality:** Intentionality at the human level is shown by timing experiments (Libet, (2003)) to be the experiencing of unconscious decisions, the latter made prior to the experiential effect. We see the correspondent of this in our model (summarized in (2.14)), wherein the unconscious competition for survival (conducted in terms of fitness $a$) is an externally observable feature (in (2.7) via $\overline{n}$ values) that informs population changes (summarized in (2.10)), which drive the subsequent unobservable adjustment to the experiencing process, the awareness $\hat{A}(x,t)$ (see (*a*) and (2.7) *f*).

***c*) Self:** In our model,

(*1*) First there is measurement and motion where an externally observable procedure of assigning bacterial level fitness $a$ occurs (see (2.7)). This procedure does not have awareness associated with it (i.e., is unconscious).

(*2*) This is followed by reproduction where a random assignment of the fitness $a$ to progeny occurs (see (*ii*) reproduction in Sect 2.2). This fitness corresponds to an experiential process, namely the awareness $\hat{a}$ (a proto-consciousness).

(3) Finally there is a physical change of population (via a competitive survival process; see (2.9) and (2.10)), and a modification of the colony's awareness density, token and construct, $A(x,t)$ and $\hat{A}(x,t)$ (see (*b*) intentionality).

The computation described in (*1*) is a process lacking awareness (akin at the human level to an unconscious proto-thought process). Let us posit then that (*2*) is an experiencing called $\hat{a}$ (a proto-consciousness) of this thought process in (*1*); and that (*3*) is the competitive survival process that has been taken to be the mirroring and the experiencing of the quale $p(x,t)$ (see (*a*) here). In the context of a bacterial colony, these features describe what is usually called the self.

This discussion of self frames a pan-psychism for living matter organized to forage for nutrients for survival and that has the ability to reproduce. For discussion of this pan-psychism for nonliving matter see Miranker (2000, 2005).

## 2.5 Cartesian dualism, a pan-psychism for living matter

If we modify Descartes (as suggested by Libet's 2003 result) to read, "*I* (experience) *think*(ing)*, therefore I am*", we have a bacterial colony realization of the venerable Cartesian postulate (as modified) (Descartes (1637), Damasio (1994), Naur (2001)). The modified postulate applies to neural assemblies and the notion of self at the human level, as we may observe in the following Sect. 3.

## 3. Awareness in a Neural Assembly

We convey the colony of bacteria formalism of Sect. 2 over to a neural assembly. A model bacterium is replaced by a model neuron, where for convenience we employ binary valued neural activity. While neurons consume both nutrients and neural transmitter, it is the latter that is implicated directly in the information processing. Moreover neurons do not move about (forage) in physical space, but they do change their processing behavior as they change location in synaptic weight space $W$. Letting $w \in W$ denote the neuron's vector of afferent synaptic weights, a bacteria's position $x$ in physical space is replaced by a neuron's location $w$ in $W$. Neurons can become better/poorer consumers of transmitter flux depending on displacements in $W$, and better/poorer producers of this flux as well (i.e., more/less fit, as we shall see). Consuming flux is the neuron's way of measuring the information the flux represents. So a neuron's motion in $W$ is its response to information measurements. For such reasons, we selected the indicated neuronal framework as the analog to the bacterial case.

The dimension of $W$ can be very large, corresponding to the largest number of afferents of any single neuron in the assembly, a number as large as 100,000 in the human brain. This increase of five orders in dimension magnitude in passing from colony to network suggests the subtlety to be found in human consciousness.

**3.1 The assembly model, virtual chromosome, synaptic weight dynamics**

Let $N_i = N_i(w,t)$ denote the $i$-th neuron at $w$. Set

3.1) $$h_i = \begin{cases} 1, \ \textit{if } N_i(w) \textit{ fires} \\ 0, \ \textit{otherwise.} \end{cases}$$

The arguments of $h_i(w,t)$ and $N_i(w,t)$ are exhibited only for clarity.

We view neural activity (say, in terms of neural transmitter flux) as the analog of bacterial nutrient. That is, each active afferent synapse consumes a measure of nutrient (i.e., of transmitter flux), and each firing efferent, counting its axonal arborization, represents an augmentation of nutrient supply. Let $n_i(w,t)$ be the number of afferents of $N_i(w)$ receiving endogenous (of the assembly) signal, and let $n(w,t) = \sum_{i:\, \text{neurons at } w} n_i(w,t)$. Then the assembly's transmitter flux dynamics that correspond to bacterial nutrient dynamics in (2.6) are

3.2) $$n(w,t+1) = \sum_{i:\, \text{neurons at } w} h_i \kappa_i(w,t) - n(w,t) + n_e(w,t).$$

Here $\kappa_i(w,t)$ is a multiplier representing the degree of axonal arborization of $N_i(w)$, and $n_e(w,t)$ is the number of afferents of neurons at $w$ receiving exogenous (from outside the assembly) signal. The first term on the right hand

side in (3.2), representing the production of transmitter flux, is a feature not present in the colony.

The colony's motion in physical space is replaced by synaptic weight change of the assembly, that is, motion in $W$. Each synapse changes its weight, often characterized using Hebb's law.

3.3) $$w(t+1) = w(t) + \alpha g H.$$

Here the scaling factor, $\alpha$ and the value of a virtual gene, $g$ are employed in correspondence to (2.3). The Hebb function (Hebb (1946, 1949)),

3.4) $$H = H\left(v^a(t), v^e(t+1)\right)$$

determines the correlation of each synaptic input with the neuronal output. In (3.4), $v^a(t)$is the vector of afferents (voltages that are approximately proportional to the transmitter flux arriving at an afferent) of a neuron, and the scalar $v^e(t+1)$ is that neuron's efferent. In fact we shall presently replace the gene by a chromosome that arises naturally in the neuronal model, and along with that, we shall replace the synaptic weight dynamics (3.3) as well.

Take

3.5) $$a_i(w,t) = \operatorname{sgn}\left[\frac{n_i(w,t)}{n_i(w,t-1)} - 1\right], \quad \forall i,$$

for the fitness of $N_i(w)$ (compare (2.7)). That is, $N_i(w)$ is fit/not-fit at $w$ at time $t$ if it improves/degrades in ability to acquire input-flux, according to the flux dynamics in (3.2). As with bacteria where fitness depends on the both the bacterium and its environment (neighbors and nutrient supply). So fitness is not solely a quality of a neuron, depending also on the neuron's environment (connectivity arrangements and activity). As in Sect. 1, the measurable physical quantity $a_i(w,t)$ specified in (3.5) is the unconscious token of the neuronal awareness. The corresponding dualist construct, the proto-consciousness $\hat{a}(x,t)$ is the observer process of the measurement and computation represented by (3.5).

**Virtual chromosome:** The model neuron already possesses a feature that can perform the function of the virtual gene $g$. In particular, this intrinsic feature is the vector $w$ of its synaptic weights, which we shall regard as a virtual chromosome. (Each individual weight can be viewed as playing the role of a gene in that chromosome). Indeed, in order to accommodate this revision, we drop $g$ from the neuronal model and modify Hebb's law (3.3) to read

3.6) $$w_i(t+1) = w(t) + a_i(w,t) H\left(v_i^a(t), v_i^e(t+1)\right) + \nu\chi, \quad \forall i.$$

See the last paragraph of the following Sect. 3.2 for motivation of the choice (3.6). The quantities $a_i(w,t)$ and $H\left(v_i^a(t),v_i^e(t+1)\right)$ refer to $N_i(w)$. $w_i(t+1)$ is the subsequent location in $W$ of $N_i(w)$. $v\chi$ (with scaling factor $v$and random variable $\chi$) representing noise can be viewed as a stochastic mutation generating term. If $v<0$ and $\chi \geq 0$, $v\chi$ is called a forgetting factor (Haykin (1999)).

**Darwinism and genetic mutation:** Genetic mutation, the twentieth century addendum to Darwin's ideas can be adapted into the genes and chromosome of our neuronal model, for example as abrupt, perhaps random changes in synaptic weights. Study of the resulting effects is left for future work.

### 3.2 Population dynamics

Population dynamics have a more complex (stochastic) character in the neural assembly than in the colony case. The analog of $p(x,t)$ is $p(w,t)$, the number of neurons at a point $w$ in $W$ that have fired at time $t$. The three types of factors; motion, reproduction and competition of Sect. 2 drive the changes in $p(w,t)$ as well, with the modification that reproduction and competition for nutrient in the colony are replaced by inhibition and competition for the opportunity to fire in the assembly. However unlike the colony case, neurons that don't fire, while not contributing to the current value of $p(w,t)$, are not eliminated from the model. Such neurons stand by and await a further opportunity to participate. In nature a neuron that doesn't participate (fire) for too long a period may suffer apoptosis.

Population dynamics (the analog of (2.10)) are defined in terms of so-called winning neurons, namely

3.7) $p(w,t) =$ # of neurons at $w$ specified as winners at time $t$.

**Specification of winning neurons:** If at least one neuron fires, the transmitter flux per firing neuron at $w$ at time $t$ is

3.8) $$s = s(w,t) = \frac{\sum_i n_i}{\sum_i h_i}.$$

Let $w_j = w_j(t) = (w_{j1}, w_{j2}, \ldots)$ be the synaptic weight vector of $N_j(w)$. Let $s_j$ be the random variable that takes on the values

3.9) $$s_j = \sum_i^{(s)} w_{ji},$$

where the sum is over a collection of $\lfloor s \rfloor$ of the components of $w_j$ chosen from all of these components, uniformly at random. Let $\bar{s}_j$ denote the mean value of $s_j$. If

3.10) $$\bar{s}_j > \theta_j,$$

where $\theta_j$ is the firing threshold, then $N_j(w)$ is a winning neuron. Neurons that fail the test in (3.10) are losers. The intuition is that, during a sustained epoch of neuro-processing, a neuron that satisfies (3.10) is expected to fire more frequently than a neuron that does not.

**3.3 Awareness hypothesis: experiencing qualia, intentionality, self, mirroring**

As with $p(x,t)$ and $\hat{A}(w,t)$ for the colony, we take $p(w,t)$ to be a physical quantity standing in duality to an assembly fitness density $\hat{A}(w,t)$. Let (compare (2.12), (2.13)*f*)

3.11) $$A(w,t) = \sum_{i:\text{neurons at } w} a_i(w,t)$$

be the token of the derived construct $\hat{A}(w,t)$, where

3.12) $$\hat{A}(w,t) = \bigcup_{i:\text{neurons at } w} \hat{a}_i(w,t).$$

Note that

3.13) $$A(w,t) = \frac{n(w,t)}{n(w,t-1)} - 1$$

ia an alternative choice for the assembly awareness density token.

Up to a scale factor, we expect $p(w,t)$ to mirror the transmitter flux $n(w,t)$. As a physical quantity, $p(w,t)$ (say, unconscious), is measurable (see (3.7)*f*), while the derived construct $\hat{A}(w,t)$ is externally unobservable (say, a proto-consciousness density). With this convention, we say that $\hat{A}(w,t)$ (proto-conscious)/ $p(w,t)$ (unconscious) are sensations/sensors of the encoded information being processed by the neural assembly. That information is conventionally specified in terms of the assembly's collection of action potentials (the efferent $v^e$ of (3.4)), and these in turn, are each approximately proportional to a quantity of transmitter flux. We posit that $\hat{A}(w,t)$ is an experiencing of its dual $p(w,t)$. That is, $\hat{A}(w,t)$ is an experiencing of an information density encoded and being processed by the sub-assembly of neurons at $w \in W$. The pair $(p(w,t),\hat{A}(w,t))$ is the quale density corresponding in turn to a stimulus $n(w,t)$, the latter mirrored by $p(w,t)$ (see the schematic (2.14)). In this neural assembly case, the stimulus could be conveyed in whole or in part from another neuronal assembly, perhaps one corresponding to a sense organ.

The qualities of intentionality and the self as described for the colony in Sect. 1 are likewise available for the neural assembly. Since the corresponding constructs (dualist or derived, as the case may be) in the two cases are represented by the same symbols, the relevant descriptions carry over directly

## 4. Applications: Colony level, human level, evolution

The applications considered model the presence or absence of consciousness, and if present they specify its location. Since it is the sign of the token corresponding to a neuronal structure that specifies this (see the subsection on conventions in Sect. 2.2.), applications are developed through consideration of a relevant neuronal arrangement along with derivation of the mathematical expression of its associated token.

The human mind is unable to generate certain qualia in the absence of (exogenous) sensory input, for example, unable to create the smell of a rose without a rose being present. That is, sensory input is necessary for experiencing a quale. We first examine this effect in the context of the colony, using it as an application of the bacterial level construct that serves as a model of more extensive applications in the case of neuronal assemblies that follow.

### 4.1 Colony awareness vanishes with $n_e(x,t)$

To proceed, set $n_e(x,t)$ (the analog of sensory input) to zero. Then using (2.6), the bracketed term in (2.7) can be written as

4.1) $$\frac{\bar{n}(x,t)-\beta\bar{p}(x,t)}{\bar{n}(x,t)}-1=-\beta\frac{\bar{p}(x,t)}{\bar{n}(x,t)}.$$

According to our convention (see Sect. 2.2), colony awareness/perception disappears ($\hat{A}=\varnothing$), since the members of (4.1) are negative.

### 4.2 Neuronal assembly applications

We now develop, in order, the neuronal assembly applications listed at the end of Sect. 1.1 of the introduction.

#### 4.2.1 Conditions for generating qualia, a threshold effect

This application is an adaptation of the colony level application of Sect. 4.1 to the neural assembly, and so is relevant to human consciousness. Namely, sensory input is a necessary condition for the experiencing of qualia. To show this, we proceed as before, setting $n_e(w,t)=0$. Then inserting (3.2) into (3.13), we have

4.2) $$A(w,t)=\frac{\sum_{j:\text{neurons at } w} h_j\kappa_j(w,t-1)}{n(w,t-1)}-2.$$

Then define the awareness token of the entire assembly at time $t$ to be

4.3) $$\mathbf{A}(t)=\sum_w A(w,t),$$

the sum over the finite set of $w$ specified by the assembly. Correspondingly, assembly awareness is

$$\hat{\mathbf{A}}(t)=\bigcup_{w}\hat{A}(w,t). \tag{4.4}$$

The numerator in (4.2) represents all of the neural transmitter flux produced by the assembly at $t-1$. The denominator represents that flux plus exogenous flux entering the assembly at time $t-1$. Then the fraction in (4.2) is less than or equal to one, so $A(w,t)\le -1$. So we have a result analogous to the application for the colony in Sect. 4.1. Namely, in the absence of exogenous (say, sensory) input, $\hat{\mathbf{A}}(t)=\varnothing$. Moreover to experience a quale, the input must be present and the ratio $n_e(w,t-1)/n(w,t-1)$ must exceed $A(w,t)$ given in (4.2), a threshold effect.

### 4.2.2 Qualia are located in the peripheral nervous system

For this application that applies to olfaction, gustation and haptic response, we employ a three-layer compartmental model of the nervous system. Layer 0 is the sensory input layer. Layer 2 is the brain. Layer 1 represents the remainder, the peripheral nervous system and the cord. So layer 0 feeds layer 1, and layer 1 interacts reciprocally with layer 2. To reflect these three compartments, (3.2) is replaced by

$$\begin{aligned} n^{(j)}(w,t+1)= &\sum_{i:\text{neurons at } w} h_i^j\lambda_i^j(w,t)-n^{(j)}(w,t)+(2-j)n^{(0)}(w,t) \\ &+\sum_{i:\text{neurons at } w} h_i^j\kappa_i^{jk}(w,t),\quad j,k=1,2,\quad j\neq k. \end{aligned} \tag{4.5}$$

Here $n^{(0)}(w,t)$ is the sensory input from layer 0 to layer 1. $n^{(j)}(w,t)$ is the number of afferents in layer $j$, $j=1,2$ receiving input. Corresponding to layer $k$, $k=1,2$, $\kappa_i^{jk}(w,t)$ is a multiplier representing the part of the axonal arborization of the $i$-th neuron at $w$ that enervates a neuron in layer $j$, $j=1,2$, $j\neq k$. $\lambda_i^j(w,t)$ corresponds analogously to lateral enervation within layer $j$. $h_i^j$ is 1/0, if in layer $j$, $N_i(w)$ fires/not-fires.

Correspondingly, (3.13) is replaced by

$$A^{(j)}(w,t)=\frac{n^{(j)}(w,t)}{n^{(j)}(w,t-1)}-1,\quad j=1,2. \tag{4.6}$$

Replacing $t+1$ by $t$ in (4.5) and inserting the result into (4.6), the latter becomes

$$A^{(j)}(w,t)=\frac{\Phi^{jk}(w,t-1)+(2-j)n^{(0)}(w,t-1)}{n^{(j)}(w,t-1)}-2,\quad j,k=1,2,\quad j\neq k, \tag{4.7}$$

where

4.8) $$\Phi^{jk}(w,t-1) = \sum_{i:\,\text{neurons at } w} \left[ h_i^j \left( \lambda_i^j(w,t-1) + \kappa_i^{jk}(w,t-1) \right) \right].$$

The term $B = \Phi^{jk}(w,t-1)/n^{(j)}(w,t-1)$ from (4.7) is smaller than unity (for reasons analogous to those discussed in the paragraph following (4.4)), so $A^{(j)}(w,t)$ is negative unless the term $(2-j)n^{(0)}(w,t-1)/n^{(j)}(w,t-1)$ survives and dominates $B$. Survival happens only for $j=1$ (for layer 1), and dominance when $n^{(0)}(w,t)$ is sufficiently large (when there is adequate sensory input). Thus the awareness $\hat{A}(w,t) \neq \varnothing$ only in the peripheral nervous system $(j=1)$ and then only in the presence of adequately strong sensory input, a threshold effect.

### 4.2.3 Visual and auditory imagery are located out in space, a role of the environment in consciousness

This application is a corollary of the result in Sect. 4.2.2, so we proceed by identifying the three layers in model in question, not all of which are corporeal. We shall discus the case of vision only, since the changes for audition (e.g., eye replaced by inner ear) are straightforward.

Layer 0 is the light source that supplies nutrient in the form of light flux (photons). Layer 1 consists of three successive compartments. The first compartment consists of the objects seen[8]. The flux from layer 0 is processed by absorption in and reflection from the first compartment, and the resulting flux (processed light stream) is passed on to the second compartment, the eye. The eye processes the flux from the first compartment by first focusing it on the retina, which after some preliminary processing passes flux (action potentials) via the optic nerve to the LGN, the third compartment. Layer 2 is the rest of the visual cortex that interacts reciprocally with the LGN.

Referring to Sect. 4.2.2, we see that the awareness corresponding to vision occurs in layer 1 whose first compartment consists of the objects seen. As we see, vision makes use of a mix of structure and flux types. The structures are neuronal, corporeal (but not neuronal) and extra-corporeal. The nutrient flux changes from streams of photons (proceeding first to and then from layer 0 and through the front of the eye) to the customary neural transmitter. The processing of the flux is perforce disparate. This suggests that consciousness is not only a product of the neuronal and non-neuronal corpus, but of the environment as well. As we've noted, the environment's role in perception is more substantive than as a provider of input. While this last remark applies to vision and audition, it might very well apply to other senses. Certainly it seems to be relevant to the perception of heat (infrared radiation) by pit vipers, the perception of electrical waves to certain eels, and perhaps the perception of gravity to migratory birds.

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[8] The use of the environment as a working store for cognitive processing of visual information is an idea explored by Ballard, Hayhoe, Pook, & Rao, (2001).

**Model complexity:** Layer 2 represents neuronal processing of more than 20 known richly enervated reciprocally connected visual brain regions. It is not merely a principle of economy in the model (Occum) that permits this collapse of 20 into one. Indeed examining the details of (4.5)-(4.7) in Sect 4.2.2 upon which the vision arguments in the present section are made, we could accommodate these 20 such regions by appending additional layers to the model, and correspondingly, replacing $j$ and/or $k=1,2$ by $j$ and/or $k=1,...,21$ in (4.5)-(4.7). The conclusion would be the same. Namely, what was called survival in the last paragraph of Sect. 4.2.2 still occurs only for $j=1$. That is, awareness occurs only in layer one. There is no awareness in any of the newly introduced layers.

### 4.2.4 Dream imagery is located in a virtual space

As in Sect. 4.2.3, we proceed by describing the three layers in the dream imagery model. These are three different parts of the brain: 1) a source of imagery (from memory, say dream memory), 2) an intermediate structure where the awareness of the imagery is manifest, and 3) the remainder of the brain. That is we postulate that a sufficiently complex brain is able to simulate a visual sensory system (say as described in Sect. 4.2.3) endogenously. So the location of these dream images is in the intermediate structure of (2). We apparently are not able to assign a specific physical location to the intermediate structure, so we perceive the location as some unknown virtual space.

### 4.2.5 Hallucinations, in particular phantom pain

Hallucinations are a pathological phenomenon, and an explanation of them flow from our model when certain variables in it behave pathologically. We illustrate this in the case of phantom pain.

Referring to Sect. 4.2.3, to accommodate the missing limb, layer 0 and part of layer 1 are absent. Then set $n^{(0)}(w,t-1)=0$ in (4.7). Next set $j=1$ and $k=2$ in (4.7), giving

$$A^{(1)}(w,t)=\frac{\sum_{i:\text{neurons at } w}\left[h_i^1\left(\lambda_i^j(w,t-1)+\kappa_i^{12}(w,t-1)\right)\right]}{n^{(1)}(w,t-1)}-2. \tag{4.9}$$

In Sect. 4.2.2, we argued that this quantity is negative (in fact, $A^{(1)}(w,t)\le -1$). So under normative conditions, there is no perceptual awareness in layer 1 (in the part of it that is missing). That is, there is no phantom pain. Phantom pain has recently been shown to result from abnormally high levels of Nav1.3, a specific sodium channel in thalamic neurons, causing in turn, a cortical stimulation (Waxman (2005)). These events are layer 2 events in our model that we shall characterize as a pathological augmented creation of neurotransmitter flux in layer 2, this flux-stimulates layer 1, in turn. That is, these events are characterized in our model as a pathological and excessively high value of $\kappa_i^{12}$ in (4.9). If this value is large enough, $A^{(1)}(w,t)$ will be positive, and so there is a perception ($\hat{A}^{(1)}(w,t)\neq\varnothing$), of (phantom) pain in the missing part of layer 1. Compare this

outside the body perception with the location of the perception of vision described in Sect. 4.2.3.

### 4.2.6 Pheromones

This is also a corollary, one directly of (4.7). The argument following (4.7) shows that awareness of a sensory input can only occur in layer 1, but then only in the presence of adequately strong input. Adequate means that the relative input satisfies the inequality

4.10) $$\frac{n^{(0)}(w,t)}{n^{(1)}(w,t)} > 2.$$

This inequality is derived by setting $j=1$ in the term $(2-j)\frac{n^{(0)}(w,t)}{n^{(j)}(w,t)}$ in (4.7).

Then we posit that an unconscious sense (such as a response to a pheromone) corresponds to a neuronal structure that is wired up so that the inequality in (4.10) is never satisfied. This indicates that it is a limitation of the associated circuitry of layer 1 and/or its activity, which inhibits consciousness of pheromones.

### 4.3 Evolution

The argument in Sect. 4.2.6 on pheromones suggests how perceptual ability might be an evolving, alternatively a deteriorating property; appearing (strengthening even to awareness), alternatively evanescing as the relevant neural circuit/activity becomes more robust, alternatively degenerates. Referring to Sect. 4.2.3 on vision, we see that alterations in the environment on an evolutionary scale might also come into play in this evolving aspect of consciousness. An example of deterioration where sighted creatures that convert to a lightless environment evolve sightless descendents is supplied by Axtyanax fasciatus mexicanus (blind cave fish). Brain injuries furnish examples on the scale of a lifetime (Sachs (1995)).

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