

Nutrient-dependent / Pheromone-controlled thermodynamics and thermoregulation

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Abstract: Natural selection for nutrients results in their metabolism to pheromones that control reproduction in species from microbes to man. In some species, sex differences in pheromones enable sexual selection. Using what is known about the molecular mechanisms common to species from microbes to man, an argument can be made from biological facts that extends to non-random nutrient-dependent pheromone-controlled adaptive evolution. This biological-based argument can be compared to arguments that might be made to support a cosmological / mathematical argument for random mutations theory.

Introduction: The epigenetic effects of nutrients on intracellular signaling and stochastic gene expression appear to enable adaptive evolution of tightly controlled organism-level thermoregulation in mammals. Nutrient-dependent single amino acid substitutions and de novo protein biosynthesis exemplify the involvement of the *seemingly* futile thermodynamic control of intracellular and intermolecular interactions in microbes that result in stochastic gene expression.

Thermodynamically “futile” cycles of RNA transcription and degradation (Yap & Makeyev, 2013) may also be responsible for changes in pheromone production that enable accelerated changes in nutrient-dependent adaptive evolution controlled by the microRNA/messenger RNA (miRNA/mRNA) balance (see for review Meunier et al., 2013). Environmental cues, like those that signal the availability of glucose, appear to cause changes in the miRNA/mRNA balance that enable gene expression during developmental transitions required for successful nutrient-dependent reproduction in species from microbes (Park et al., 2010) to man (Jobe, McQuate, & Zhao, 2012).

What is known about species from microbes to man extends the common molecular mechanisms of thermodynamics and thermoregulation across the continuum of adaptive evolution. This literature review links the epigenetic effects of the olfactory/pheromonal sensory environment on thermodynamics and on thermoregulation to selection for phenotypic expression in a human population.

Part 1: Thermodynamically-controlled thermoregulation of reproduction

Lies, damned lies, and statistics

Statistical arguments led many people to believe in a theory of runaway sexual selection for mutations (see for review Wright, 1930). That belief is most compatible with a gradualist version of Mendelian genetics in which accumulated mutations somehow result in natural selection for observed phenotypes. Theories associated with statistics and selection for observed phenotypes should already have since been discarded by most biologists. Facts have shown that “Reproductive isolation evidently can arise with little or no morphological differentiation (Dobzhansky, 1972, p. 665).”

It is now even clearer than it was more than 8 decades ago that ecological diversification and beak morphology in finches is due to positive natural selection for nutrient-dependent amino acid changes. These changes incorporate the molecular mechanisms of AT→GC-biased gene conversion, amino acid substitutions, de novo protein biosynthesis, and expression of the insulin-like growth factor 2 receptor (Rands et al., 2013).

Common sense and biological facts support the conclusion that beak morphology adaptively evolves via molecular mechanisms that link the nutritional value of seeds to the availability of different seed types. Statistical analyses that suggest random mutations caused differences in beak morphology to be *somehow* selected represent a scientifically unsubstantiated theory that fails to address the requirements for pleiotropy and epistasis.

In another recent report that challenges the scientifically unsubstantiated theory of runaway selection for mutations and the adaptive evolution of the head crest in pigeons, researchers reported that derived traits in domesticated birds evolve in stages: 1) color variation, 2) plumage variation, 3) structural variation, and 4) behavioral differences. One gene is responsible for the head crest in all species, which means mutations that alter the head crest are not selected (Shapiro et al., 2013). The pervasive selection for mutations assumption was made with no evidence that either natural selection or sexual selection can result in behavioral differences that enable mutations to be selected. If mutations theory continues to be propagated, Darwinian Theory seems doomed to suffer from a lack of critical examination in the context of how natural selection occurs and what is selected. Blind acceptance of theory already has led to ignorance of biological facts.

Molecular mechanisms vs. statistics

We now know that new species arise from relatively sudden changes in the supply of nutrients. The problem that remains with the concept of Darwinian Natural selection for phenotype is that statistical arguments first eliminate consideration of genes with large effects (Carter, 1969). That approach has led many evolutionary theorists and philosophers to ignore existing pleiotropy, which is especially evident in microbial species, and to also ignore the epigenetic tweaking of immense gene networks by nutrients in all species. The epigenetic effects of nutrients are clearly required for individual survival, and nutrients metabolize to species-specific pheromones. The problem with mutations theory extends to a denial of species-wide pheromone-controlled reproduction, which is required for epistasis and survival of species.

Simply put, statistical analyses are used to deny nutrient-dependent / pheromone-controlled adaptive evolution (Kohl, 2013). By default, statistical analyses also tend to 1) eliminate climate change, 2) eliminate the thermodynamic complexity of molecular bonds, and 3) eliminate the complex systems biology of thermoregulation and adaptively evolved biodiversity. Because statistical analyses of cause and effect start by eliminating biological facts from calculations and equations, some researchers have viewed adaptive evolution only in the context of a mathematical “garbage-in, garbage out” theory -- with no model of biologically based adaptive

evolution. For comparison, physics is one starting point from which to model the role of thermodynamics, thermoregulation, and epigenetic effects on the evolution of biodiversity. Although I do not have the background in physics that is required, others who do may help build from here using their expertise. Here may be an acceptable starting point.

From Physics to Physiology

The vibrational force of random molecular motion is manifested as heat, which may pull two strands of DNA apart. This makes adaptive evolution via de novo protein biosynthesis impossible. Too much heat negates the epigenetic effects of nutrients on expression of receptor proteins. The heat can literally “cook” the proteins that make up the receptors, which are required for life because they enable nutrients to enter cells. Thus, the physical distance from the sun and entropy associated with thermodynamics determines, in part, one requirement for life on Earth, its thermodynamic control.

At physiological temperature ranges, two DNA single strands align with each other. As two complementary strands get longer, the additive nature of hydrogen bond (H-bond) energies allows their total number to surpass the randomness of vibrational forces that generate too much heat (Brunstein, 2013). Environmental conditions may be ecologically achieved through nutrient uptake that appears to enable the temporary reversal of entropy. Bonds form in RNA, which facilitates H-bond formation between correct pairs in DNA.

Once ecological conditions are achieved that enable the temporary reversal of entropy and the formation of H-bonds between base pairs, cells are formed via the complex interactions among different proteins. These interactions are fueled by nutrient metabolism that generates heat, which makes the creation of different proteins again fit the constraints of the 2nd Law of Thermodynamics.

The formation of cells by proteins incorporates the epigenetic “landscape” of the sensory environment into the thermodynamically controlled physical “landscape” of chromatin remodeling in the genome. Nutrients continue to fuel the thermodynamics of cellular metabolism. The need for sugars as “fuel” is manifested in unicellular organisms and in every cell of every tissue in every multicellular organism.

The pathway that is essential for glucose uptake has been detailed, which exemplifies the transition from physics to biology in the basic construct of DNA (Wong, 2013). This is also consistent with what is now described as an RNA world (Hoeppner, Gardner, & Poole, 2012).

Microbes:

In bacteria, the presence of a sufficient number of potentially useful novel nutrient molecules causes changes in protein folding in the cell wall. The changes enable a receptor-mediated “molecular handshake,” which allows the required ligand-to-receptor binding so that the nutrient can enter the cell (Blount, Barrick, Davidson, & Lenski, 2012).

Microbial pleiotropy and epistasis link the thermodynamics of intracellular and intermolecular mechanisms of protein biosynthesis to nutrient-dependent ligand-receptor binding. Adaptive evolution of bacteria appears to occur via a glucose-dependent amino acid substitution, which is associated with receptor-mediated thermodynamically-regulated antibiotic resistance (Rodriguez-Verdugo, Gaut, & Tenaillon, 2013).

Controlled ecological niche construction and social niche construction enable adaptive evolution in other microbial species (McFall-Ngai et al., 2013). The successful nutrient-dependent adaptive evolution of bacteria via thermodynamic control of their apparently limitless pan-genome enables ecological niche construction that links the metabolism of nutrients to organism-level and to colony-level thermoregulation via pheromone-controlled social niche construction.

Part 2: An epigenetic continuum of nutrient-dependent pheromone-controlled thermoregulation

Nematodes

Differences in the behavior of nematodes are determined by nutrient-dependent rewiring of their primitive nervous system (Bumbarger, Riebesell, Rödelsperger, & Sommer, 2013). Species incompatibilities in nematodes are associated with cysteine-to-alanine substitutions (Wilson et al., 2011), which can be expected to alter thermoregulation via the same molecular mechanisms of microbes.

Insects

In flies, ecological and social niche construction can be linked to molecular-level cause and effect at the cellular and organismal level via nutrient-dependent changes in mitochondrial tRNA and a nuclear-encoded tRNA synthetase. The enzyme enables attachment of an appropriate amino acid, which facilitates the reaction required for efficient and accurate protein synthesis (Meiklejohn et al., 2013). In wasps, manipulation of the genetics of evolved species-specific pheromones characterized the change in a pre-existing signaling molecule triggered by a glucose-dependent (Yadav, Joshi, & Gurjar, 1987) stereochemical inversion (Niehuis et al., 2013). In the moth, substitution of a critical amino acid, is sufficient to create a new pheromone blend (Lassance et al., 2013).

The honeybee is currently an accepted model organism of nutrient-dependent pheromone-controlled adaptive evolution of the brain and behavior that is consistent with what is known about neurogenic niche construction in nematodes (Bumbarger, Riebesell, Rödelsperger, & Sommer, 2013). What the queen eats determines her pheromone production and every interaction among the members of the colony. Her pheromone production differentially effects the development of neurogenetically predisposed brain development and the socio-cognitive niche construction manifested in the worker bees (see for review Kohl, 2012).

Mammals

Current concepts now limit attempts to explain selection for nutrient-dependent changes in coat color and kinked tails in mice via mutations theory, since mutations theory does not address pleiotropy and epistasis (see for review Feinberg & Irizarry, 2010). Until recently, the association of the nutrient choline in humans and its metabolism to trimethylamine odor in different species of mice was the best example of how a change in diet becomes associated with the presence of mammalian conspecifics whose androgen estrogen ratio-associated odor distinguishes them sexually, and also as nutrient-dependent physically fit mates (Stensmyr & Maderspacher, 2013). The mouse model makes it clearer that glucose uptake changes cellular thermodynamic equilibrium and differential pathway regulation that results in adaptively evolved fitness in species from microbes (Kondrashov, 2012) to mammals. Species-specific health and reproductive fitness is associated with nutrient-dependent amino acid substitutions and with pheromone-controlled reproduction. Disease is associated with mutations exemplified in cancer where perturbations of the glucose-dependent thermodynamic/thermoregulatory equilibrium are equally clear (Locasale, 2012).

Humans

Two additional published works link 1) the thermodynamics of the RNA world; 2) nutrient-dependent regulation of H-bond formation in DNA; and 3) organism-level thermoregulation in a human population. A change in one base pair, appears to result in the thermodynamically controlled substitution of the amino acid alanine for the amino acid valine (Grossman et al., 2013). The alanine substitution for valine does not appear to be under any selection pressure in mice. The cause and effect relationship was established in mice by comparing the effects of the alanine, which is under selection pressure in humans, via its substitution for valine in mice (Kamberov et al., 2013).

These two reports (Grossman et al., 2013; Kamberov et al., 2013) tell a new short story of adaptive evolution. The story begins with what was probably a nutrient-dependent variant allele that arose in central China approximately 30,000 years ago. The effect of the allele is adaptive and it is manifested in the context of an affect on sweat, skin, hair, and teeth. In other mammals, like the mouse, the affect on sweat, skin, hair, and teeth is due to an epigenetic effect of nutrients on hormones responsible for the tweaking of immense gene networks that metabolize nutrients to pheromones. The pheromones control the nutrient-dependent hormone-dependent organization and activation of reproductive sexual behavior in mammals such as mice and humans, but also in invertebrates (Kohl, submitted). That means the adaptive evolution of the human population, which is detailed in these two reports, is also likely to be nutrient-dependent and pheromone-controlled, since there is no other model for that.

Mathematical models vs biological facts

Random mutations that somehow cause one or more amino acid substitutions are not likely to simultaneously cause adaptive evolution from the bottom up via the thermodynamics of chromatin remodeling and control of adaptive evolution from the top down via organism-level thermoregulation. However the nutrient-dependent substitution of alanine for valine (Grossman et al., 2013; Kamberov et al., 2013) appears to result in species-specific organism-level changes in skin, glands, and hair, through pheromone-controlled reproduction.

Increased apocrine gland secretions associated with the amino acid substitution in mice links smaller human breasts capable of more milk production and more pheromone production to sex differences in imprinting on the species-specific odor of the mother and classical conditioning of associated hormone-organized and hormone-activated behaviors throughout life. For example, sex differences in preferences for breast size are probably classically conditioned via associations with the unconscious affect of odor from human female breasts that differentially alters hormone-dependent neurogenesis from birth. Fos protein expression from pubertally born neurons that are integrated into region-specific areas of the brain activated by sociosexual behavior in the Syrian hamster (Mohr & Sisk, 2013), more strongly suggests that the role of nutrient-dependent pheromone-controlled regulation of gonadotropin releasing hormone (GnRH) pulse frequency and amplitude is the biological core of mammalian reproductive sexual behavior (Kohl, 2013).

Increased numbers of hormone-regulated eccrine sweat glands in mice (Grossman et al., 2013; Kamberov et al., 2013) have similar thermoregulatory effects that help to control the core temperature of the human body. At the same time the increased secretion of watery sweat serves to alter pheromone production which results from the microbial metabolism of nutrient-dependent substances in apocrine and eccrine sweat. The effect of the alanine substitution on increased hair thickness links it to trapped pheromones and a visual signal of sexually dimorphic nutrient-dependent reproductive fitness in cultures where females have longer head hair than males. It may be that in all cultures the appearance of scalp hair is an indicator of the androgen estrogen ratio, especially in women. That does not necessarily mean the alanine substitution is responsible for pheromone-controlled reproduction in this human population. What do we know for sure?

Epistasis

Pheromone-controlled reproduction is required for fixation of the nutrient-dependent substitution. In contrast to what has been theoretically stated, one mutation or variant cannot alter the life cycle transitions of one species (e.g., from birth to puberty in the Syrian hamster), let alone enable adaptive evolution via species diversification. Mutations have effects but an affect on behavior is required to link a mutation to adaptive evolution. What is missing from mutations theory is information on how a difference in behavior is naturally selected that links the mutation to phenotype and to adaptive evolution.

For contrast, there is now evidence for thermodynamically driven adaptive evolution in microbes that extends to the adaptive evolution of thermoregulation at the organism level in species from microbes to man. In what at first may have appeared to be an explanation of an adaptive mutation, one of the two concurrently published articles shows that control of adaptive evolution from the bottom up is amino acid-dependent (Kamberov et al., 2013). The other article shows that the metabolism of nutrients like sugars and amino acids to pheromones is probably what controls adaptive evolution from the top down (Grossman et al., 2013).

The epigenetic tweaking of a genetically predisposed nutrient-dependent variation (i.e., the amino acid substitution) enables changes in immense gene networks via interactive changes in

intracellular signaling and stochastic gene expression. Those changes are required to get from the sensory environment to thermodynamically controlled de novo protein synthesis. In humans, the effect is presumably on the thermodynamics of intranuclear interactions and miRNA-controlled Toll-like receptor-mediated immunity (Fabbri et al., 2012). This links the thermodynamics of microbial antibiotic resistance (Rodriguez-Verdugo, Gaut, & Tenaillon, 2013) to fitness-related human immunity that involves natural selection for nutrients and sexual selection for pheromones.

From microbes to man

A new allele does not simply arise. The ability to express a genetically predisposed variation exists in the genome of the cell in species from microbes to man. The sensory environment causes thermodynamically regulated intracellular changes that result in de novo protein synthesis required for the allele to arise. It seems likely that nutrients, like glucose, fuel the changes in the miRNA/mRNA balance and provide the energy required for chromatin remodeling via alternative splicing that result in de novo protein synthesis.

The requirement remains to link the allele to behavior and back to genes. In other species described above, the link from nutrients to differences in pheromone production and control of reproduction is a single amino acid substitution. In humans, the effect of the amino acid substitution is best described in the context of animal models that incorporate “genes of large effect.” The result is exemplified in the honeybee model organism, which links the epigenetic effects of nutrients to brain development and behavior associated with the epigenetic effects of pheromones that link the behavior back to genes of large effect.

Conclusion

Genes of large effect were removed from consideration in the statistical analyses used to support the theory of runaway selection that supposedly was due to the accumulation of random mutations over extremely long time periods required for species to evolve from other species. In my model of Nutrient-dependent / Pheromone-controlled Adaptive Evolution (Kohl, 2013), the genes of large effect link the epigenetic effects of nutrients and pheromones to non-random adaptive evolution. Nutrients cause changes in cells that allow receptors to develop; they epigenetically tweak immense gene networks in superorganisms that solve problems through the exchange and the selective cancellation and modification of signals. For example, miRNAs that regulate gene expression appear to be key regulators of the nutrient-dependent thermodynamically controlled cellular microenvironment because they bind as ligands to receptors of the Toll-like receptor (TLR) family in immune cells.

Theoretically, that is also how miRNAs that regulate gene expression might trigger nutrient-dependent TLR-mediated prometastatic inflammatory response that ultimately leads from controlled intracellular metabolism to tumor growth and metastasis (Fabbri et al., 2012). This is one way that nutrient stress and social stress could lead to immune system dysfunction and possibly to cancer. Clearly, it is a way that deserves to be thoroughly explored.

Issues for additional consideration

Statistical analyses and mutations theory may have led to misconceptions about adaptive evolution compared to the role of mutations that cause cancer. Statistical analyses may also have caused mathematicians and physicists to abandon hope that biology would link them to their cosmic origins. The molecular biology common to all species suggests the link to our origins has always been food and pheromones.

Perhaps others should now compare biological facts to theory. Phenotypic characteristics of cancer are unlikely to be easily observed. However, it now appears that dogs can be trained to respond to odors that are linked to different types of nutrient-dependent cancers. A breath test for gastric cancer is also being developed (Xu et al., 2013)

In the context of perturbations in the miRNA/mRNA balance that result in mutations and damaged cells, it is also noteworthy that ionizing radiation through environmental, occupational, or a nuclear reactor accident might cause changes in miRNAs that may be used to determine the extent of post-exposure damage in tissues like blood (Jacob et al., 2013). This suggests malarial parasites might have altered the miRNA/mRNA balance of the human genome and caused the production of characteristic cell types and associated anemia. If sickle-cell anemia actually results from the parasite's effect on the thermodynamics of protein folding, which are manifested in thermoregulatory defects at the cellular level (e.g., fewer functional red blood cells) in one form of debilitating anemia, it might mean that other forms of anemia should be addressed in the context of a model for adaptive evolution, not mutations. Similarly, erythrocytosis (i.e., too many red blood cells) may not be due to a mutation, but caused by a perturbed miRNA/mRNA balance.

There is a choice that can now be made between random mutations theory and biological facts. The choice is to ignore the miRNA regulated typical differentiation of cell types and the atypical differentiation of cell types in cancer, and other disease processes. Those who have never before considered the fact that the epigenetic effects of social stress seem to precisely parallel the epigenetic effect of nutrient stress associated with cancer might want to more thoroughly examine nutrient-dependent pheromone-controlled adaptive evolution in the context of a model (Kohl, 2013). Alterations in hypothalamic-pituitary-gonadal and hypothalamic-pituitary-adrenal axis regulation are central to these stressors and their effects. Those alterations also are nutrient-dependent and pheromone-controlled. Obesity not only appears to increase the odds of certain types of cancers, but changes hormone-dependent pheromone production that might suggest to a dog or to a conspecific that you have altered the course of your nutrient-dependent pheromone-controlled adaptive evolution.

Will information overload prevent this article's completion?

In an accurate 5-step representation of the 4 stages of ecological, social, neurogenic, and socio-cognitive niche construction, Wu & Zhang (in press) link 1) transcription 2) translation into a novel protein 3) slight interactions with other proteins 4) minor functions of a new gene that increase the fitness of an organism and 5) the significance of new gene function to positive selection. In their Fig. 2., this explains how "The de novo gene would therefore quickly become essential in the special environmental condition."

The special environmental conditions are nutrient-dependent and pheromone-controlled. For example, they attest to the fact that adaptive evolution of *S. cerevisiae* has required frequent nutrient-dependent transitions between nutrient-poor and nutrient-rich environments. Nutrient rich environments also appear to have enabled the transition from asexual to alpha-mating pheromone-dependent sexual reproduction in this species (see Diamond, Binstock, & Kohl, 1996). The only misrepresentation in Wu & Zhang (in press) may be the concept of starvation-induced additional mutations.

Nutrient-poor conditions force organisms to attempt to acquire novel nutrients via de novo biosynthesis of receptor proteins, which is not due to mutations. The ability to produce new receptors is nutrient-dependent and must already exist in cells or novel nutrients could not be acquired. The receptors are sometimes simply described as olfactory receptors (Santoro & Dulac, 2012) that respond to the olfactory/pheromonal environment with the genetically predisposed thermodynamically-regulated ability to create additional de novo proteins from whatever nutrient source is available to enable their attempt to avoid starvation and adaptively evolve.

Thus, the 5-step representation of cause and effect in Wu & Zhang (in press), supports a model of nutrient-dependent / pheromone-controlled adaptive evolution with only one misrepresentation about the involvement of mutations. That misrepresentation may be due to concision; they did not attempt to detail the molecular mechanisms that they clearly recognize when they allude to the importance of the miRNA/mRNA balance as might be expected in published works that incorporate what is currently known about the molecular mechanisms. They write: “For example, (Meunier et al., 2012) found that de novo origination and duplication mechanisms contributed to similar degrees to the origin of miRNA genes during the evolution of mammals.” Beginning with the epigenetic effects of nutrients and pheromones on the miRNA/mRNA balance (Kohl, 2013), might help others to better detail cause and effect in the context of how the epigenetic landscape becomes the physical landscape of DNA (see for review Monahan & Lomvardas, 2012).

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