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CONTINUOUS ENVIRONMENTAL TRACKING: AN ENGINEERING FRAMEWORK TO UNDERSTAND ADAPTATION AND DIVERSIFICATION

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ABSTRACT

We offer a new framework for understanding biological adaptability based on interpreting the findings of 342 journal articles and 67 online reports related to adaptation, bioengineering, and design in view of the assumption that biological functions are most accurately explained by engineering principles. We hypothesize that organisms actively and continuously track environmental variables and respond by self-adjusting to changing environments—utilizing the engineering principles constraining how human-designed objects self-adjust to changes—which results in adaptation. We termed this hypothesis *Continuous Environmental Tracking* (CET). CET is an engineering-based, organism-focused characterization of adaptation. CET expects to find that organisms adapt via systems with elements analogous to those within human-engineered tracking systems, namely: input sensors, internal logic mechanisms to select suitable responses, and actuators to execute responses. We derived the hypothesis by reinterpreting findings and formalizing biological adaptability within a framework of engineering design, considering: (1) objectives, (2) constraints, (3) variables, and (4) the biological systems related to the previous three. The literature does identify internal mechanisms with elements analogous to engineered systems using sensors coupled to complex logic mechanisms producing highly "targeted" self-adjustments suitable to changes. Adaptive mechanisms were characterized as regulated, rapid, repeatable, and sometimes, reversible. Adaptation happened largely through regulated gene expression and not gene inheritance, per se. These observations, consistent with CET, contrast starkly with the evolutionary framework's randomness of tiny, accidental "hit-and-miss" phenotypes fractioned out to lucky survivors of deadly challenges. Evolutionists now divide over their framework's need of modification, and a trend among some seeks to infuse more engineering into biology. This disarray affords a rare, transient opportunity for engineering advocates to frame the issue. CET may fundamentally change how we perceive organisms; from passive modeling clay shaped over time by the vicissitudes of nature, to active, problem-solving creatures that continuously track environmental changes to better fit existing niches or fill new ones.

KEY WORDS

rapid adaptation, specific adaptive mechanism, tracking systems, epigenetics, sensor, engineering principles, systems biology, intelligent design, evolutionary synthesis

INTRODUCTION

The search for *the* mechanism underlying the adaptive diversification of organisms could be futile. This is for good reason. Research continues to discover wide-ranging adaptive mechanisms—from genetic to epigenetic, behavioral, and ecological—and, therefore, they resist being pigeon-holed into a single category (Laland et al. 2015; Muller 2017). Several theorists have cautioned that unless the structure of the current theory of adaptation itself adapts to accommodate these diverse mechanisms into its research programs and explanations—and refrains from shoe-horning them into old theory—then advances in the whole field of biological adaptation may be hindered (Lewontin 1983).

Subsequently, a meeting of the British Academy and the Royal Society was held on November 7-9, 2016 focused on reconciling theory with certain observations and mechanisms of adaptation, some of which are discussed herein. Commenting on why no one should be alarmed over sharp differences about interpretations of the same data, several key organizers said, "But let us also remember that no scientific, mathematical or philosophical advance occurs simply by quoting authority. In the end, evidence is what counts. Remember also the philosophical insight that evidence is *evidence* precisely because it can be so interpreted. We all work, explicitly or implicitly, from or within metaphysical assumptions. That is so whether or not we recognize it. Moreover, different assumptions dominate alternative academic fields, which can lead to differences in interpretation, and to different emphases between individuals and field on what is causally relevant" (Bateson et al. 2017, p. 1 emphasis in original)

We agree that the theory needs revision, but we find the new mechanisms incompatible with current theory. In the spirit of Bateson's counsel about interpretations, we offer a new framework for understanding adaptability that interprets observations and results in the literature in view of the assumption that biological systems and functions are most accurately explained by engineering principles. At a broad level, fresh interpretations form a new description of *what* organisms achieve when they adapt. Then data is re-interpreted to identify *where* adaptive capacity resides at the organism-environment interface. The mechanisms themselves are re-interpreted to identify *how* adaptation happens via a myriad of diverse mechanisms. Thus, this paper offers a new way to characterize the body of literature about adaptation. This approach

is a contribution toward the development of a new, comprehensive engineering-based framework for understanding biological phenomena (Snoke 2014). How different is this characterization of adaptation from the status-quo? Why select an *engineering* framework? Why now?

1. The Current Framework of Diversification and Adaptation

Tenets of the current, widely-held conceptual framework used to characterize diversification and adaptation are defined by Muller (2017):

> In a condensed form, these tenets are as follows: (i) all evolutionary explanation requires the study of populations of organisms; (ii) populations contain genetic variation that arises randomly from mutation and recombination; (iii) populations evolve by changes in gene frequency brought about by natural selection, gene flow and drift; (iv) genetic variants generate slight phenotypic effects and the resulting phenotypic variation is gradual and continuous; (v) genetic inheritance alone accounts for the transmission of selectable variation; (vi) new species arise by a prevention of gene flow between populations that evolve differently; (vii) the phenotypic differences that distinguish higher taxa result from the incremental accumulation of genetic variation; (viii) natural selection represents the only directional factor in evolution (p. 3).

Muller's equivalence of diversification and adaptation with evolution is questioned by those in creationist and Intelligent Design circles. Still, key elements accepted within selectionism include the familiar elements of natural selection, population-level thinking, Mendelian inheritance (gene-centricity), mode (accumulation of favored genetic variants), and rate (gradual and linear) (Denton 2013; Jeanson, 2017; Jeanson and Lisle 2016; Laland et al. 2015). Further, the entire framework, particularly research programs, is grounded in the assumptions that the diversification and adaptation process is random, unregulated, unguided toward any need-based outcome, and that organisms are driven by the vicissitudes of nature (Reigner 2015).

2. Discontent with the Current Understanding of Adaptation

However, a growing list of observed phenomena and mechanisms seem to be anomalous to the current understanding of adaptation (Koonin 2009) unless numerous ad hoc modifications are devised to eliminate apparent conflicts (Futuyma 2017). The principle incongruities which prompted the 2016 Royal Society meeting are recently identified mechanisms regularly characterized with non-random descriptors such as: biased and directional, rapid, predictable, and repeatable (Table 1 lists multiple examples). These findings are contrary to the classic framework of gradual diversification and adaptation resulting from *random* variation fractioned out through diverse death-driven scenarios where survival and reproduction are highly dependent on "luck" (Snyder and Ellner 2018). Accordingly, there is a growing insistence on modification of this framework (Laland et al. 2014).

Some believe the current framework is so out-of-date that it still fails to incorporate knowledge of developmental regulatory mechanisms into both theoretical population genetics and genetic accommodation theory (Laland et al. 2015; Muller 2017). While true, criticizing the current framework as merely "out-of-date" may be misidentifying the problem. This paper explores the possibility that the primary cause of the clash between recent discoveries and the current framework is that the basic tenets of the framework—and their underlying naturalistic, design-exclusive assumptions—are fundamentally incompatible with recent discoveries. Adaptive mechanisms characterized as regulated, rapid, repeatable, and predictable are anomalous precisely because those words seem to describe the purposeful outcomes of engineered systems. In areas of applied biological research, a growing trend is to incorporate engineering principles to frame biological phenomena. We believe that there are good reasons to incorporate this approach into a replacement framework for diversification and adaptation as well.

Table 1. Biological observations identified as appearing to be anomalous to the dominant conceptual framework for adaptation and differentiation (Bateson 2014; Danchin et al 2011; Endara et al 2017; Esquerre and Keogh 2016; Deem 2013; Hull et al 2017; Laland et al 2014; Laland et al 2015; Muller 2017; Reigner 2015; Shapiro 2013)

- Adaptations often appear to be "targeted" not "hit-and-miss" solutions to environmental challenges.
- Organisms can modify their developmental course to produce novel and suitable phenotypic variants.
- Organisms do track dynamic conditions and self-adjust their traits correspondingly.
- Diverse organisms repeatedly express similar morphological traits when located in similar environments.
- Adaptive mechanisms seem to be highly regulated under the control of precise cellular circuits.
- Many adaptive "mutations" increasingly looks to be controlled internally.
- The genome is increasing viewed as a read–write library of genetic functions under continuous revision and not as mostly read-only with a few rare mutations.
- The rate of adaptation can be variable and is often rapid.
- Some adaptations are known to be repeatable and reversible across taxa and time.
- Adaptive means include: genetic, epigenetic, developmental, behavioral, founder effect, and ecological.
- Transgenerational inheritance also includes epigenetic, physiological, behavioral, ecological, and cultural.

3. A Growing Tendency to Explain Biological Functions with Engineering Principles

Life itself seems to have attributes which are currently beyond the reach of scientific methods to discover, but making sense of biomolecular, physiological, or anatomical functions is not mysterious. Could it prove useful to apply engineering principles to explain how a biological function like adaptation works? There is good justification to begin doing so. Despite their vastly different substrates, living organisms are subject to the same fundamental constraints that govern all regulatory mechanisms, and they function within the same laws of nature as man-made designs (Khammash 2008). This makes it possible to study birds to gain insight into aircraft design, for example. Research demonstrates a remarkable correspondence in design, purpose, and function of

many organs and systems of organisms to similar devices produced by human engineers. Yet, knowing that organisms have functions operating by the same engineering principles as man-made things does not equate to saying that living things are only machines.

Just like human-engineered devices, organism have organs and systems which utilize the properties of natural laws like such as gravity, inertia, and momentum. Biomechanical engineers advocate the mimicry of systems found in living things and use them for inspiration in design (Socha 2012). Biological researchers already "reverse engineer" biological systems by methodically disassembling their components piece by piece to discover their operation. Further, the ever-increasing awareness that biological functions bear striking resemblance to man-made systems using sophisticated engineering has not been ignored. In 2016, an international conference dedicated to engineering biology was held at the University of Pittsburgh (http://www.pitt.edu/~pittcntr/Events/All/ Conferences/others /other_conf_2015-16/04-15-16_reengineering/reengineering.html). Its goal was to develop a new engineering paradigm in biology that emphasizes how engineering-based perspectives on biology contrast with established biological thinking. Conference organizers maintained that engineering-inspired fields such as integrative systems biology, biomedical engineering, and synthetic biology appear to have more in common with engineering approaches than with traditional biological ones. Thus, even evolutionary biologists, though rooted in design-exclusive assumptions, will face the inevitable rising tide of scientific literature from other disciplines that are using engineering principles to better explain biological functions.

4. Biblical Rationale for Explaining Biological Functions Using Engineering Principles

It would seem natural for researchers who claim to embrace the explanation that living things look designed because they are designed, to get out in front of this trend for using engineering principles to explain biological functions. For those who believe that the Bible provides insight into biological function, there is justification for approaching research from an engineering perspective. Psalms 19:1-6 and Romans 1:18-25 are key passages stating that some attributes of God are revealed in nature. Both texts emphasize that living things manifest "workmanship." Nature displays features unique to designing agency that humans would ascribe to the workmanship of artists or engineers. For instance, living things are full of systems with multiple parts working together for a purpose, which are otherwise only found in human-engineered devices. It is this clearly-seen tight correlation between the function of living things and human-engineered contrivances that strongly indicates that living things were, in fact, designed by an intellect—with engineering prowess far surpassing the best human engineers. Analysis of Romans 1:18-25 prompts a profound question related to biological research: is there a biblical (or scientific) reason to believe that any biological function will be discovered that will operate by different engineering principles than those by which human-engineered apparatuses are already operating or could be operating? Therefore, researchers open to intelligent causation for living creatures should *expect* to find an ever-increasing resemblance of biological function to sophisticated engineering.

Since creatures were commanded to "fill the earth" (Genesis 1:22, 28; 8:17; 9:1, 7) which was a highly dynamic place after Creation and the Flood, design-guided biological researchers should begin to identify the basic engineering principles governing the operation of systems which appear to self-adjust to dynamic external conditions (i.e., the *how* of biological adaptation). It is time to rethink the concepts of diversification and adaptation, and to develop a framework that explains biological function with engineering principles; one that *naturally* incorporates the numerous highly regulated mechanisms that appear to be incompatible with current evolutionary theory.

5. Formalizing a New Engineering-based Framework

To begin formalizing, rather than just conceptualizing, biological adaptability within a valid framework of engineering design, we should consider: (1) objectives, (2) constraints, (3) variables, and (4) the biological systems (corresponding to mathematical equations in engineering) related to the previous three. Incorporating several broad observations and re-interpreting them in this light will form a new description of *what* organisms achieve when they adapt (i.e., the design objective for what they are doing).

Organisms are observed to modify their own developmental course by continuously monitoring, responding to, and adjusting, their internal and external states, and these adjustments appear to play a significant role in producing novel, potentially beneficial, phenotypic variants (Bateson 2017). Further, there is a growing body of evidence that appears to indicate that recently-discovered regulated mechanisms enable organisms to actively and continuously track environmental changes. For instance, organisms appear to quickly respond to significant environmental changes—often making surprisingly rapid adaptations—and then more gradually adapt to conditions during periods with relatively little environmental change (Reigner 2015). What are organisms doing? We posit that organisms are continuously tracking environmental changes and responding with suitably self-adjusted traits to maintain homeostasis—within their lifetime and cross generationally—resulting in adaptation. How might they achieve this objective within realistic constraints?

A. Hypothesis

We observe sophisticated human engineering demonstrated in automated, autonomous robotic drones equipped with exquisite detectors, logic-centered algorithms, and locomotive performance to reliably track a target. If the application of engineering design objectives, constraints, and variables for human-created tracking systems can be applied to biological systems, then this readily offers a testable hypothesis:

Organisms actively and continuously track environmental variables and respond by self-adjusting to changing environments—utilizing the engineering principles that constrain how human-designed things adapt to changing conditions—resulting in adaptation.

This assumes that fluctuating environmental conditions are comparable to the variability of moving targets followed by tracking systems. If this assumption and the above hypothesis are true, then we expect:1) organisms should possess innate mechanisms with features that correspond to elements of human-engineered tracking systems, 2) these mechanisms should be demonstrably utilized to

actively and continuously track environmental changes, and 3) changes in an organism's traits should occur in parallel with the rate and magnitude of environmental changes.

We have termed this hypothesis *Continuous Environmental Tracking* (CET). It is the foundational assumption of a new framework for understanding diversification and adaptation. Scientific activities including interpretations of findings take place within a structure of ideas and assumptions defining a field of study. The framework we offer promotes comparing data and observations (findings) of reported biological functions to those of human-engineered entities to search for: corresponding systems and their elements, mechanisms, engineering principles fundamental to operation, and assembly processes to assist in research. Our framework widens the bi-directional conduit between engineering and biology by aiding bioengineers in their job of biomimicry and aids biologists to predict where to search for elusive system elements or steps, guided by the assumption of functional purpose(s). Reinterpreting the findings of biological studies by an "engineering approach" or "design analysis" means that they are evaluated with this comparison of biological functions to those of human-engineered entities in mind.

B. Expected features of CET biological mechanisms

What are the elements of biological systems that should correspond to man-made tracking systems? Tracking systems are generally part of larger, robust, adaptive control systems. These follow the movement of a select "target" within a specified "field of view" and elicit response per a predetermined algorithm. These "surveillance systems" use one or more sensors, coupled to a computer system, that gather and interpret incoming data about uncertain environments. (Blackman and Popoli 1999).

There are three irreducible elements common to all tracking systems: 1) sensors to detect pre-specified conditions; 2) condition-consequence logic mechanisms that process information by specifying *if* (+) condition *then* (+) consequential output response, and 3) output responses which adjust activities to effectively pursue a target (Blackman and Popoli 1999; Ioannou and Sun. 2012). (If navigation or interception at a precise location is desired, then a chronometer or circadian device to measures time is also an essential element.) If the CET hypothesis is correct, we would expect to find biological mechanisms with corresponding irreducible elements that are recognizable by the following characteristics:

(1) Sensors. The element linking the system to its environment is the sensor. Fraden, a system design specialist, highlights the role of sensors in initiating data acquisition, "a sensor does not function by itself; it is always a part of a larger system that may incorporate many other detectors, signal conditioners, signal processors, memory devices, data recorders, and actuators…A sensor is always a part of some kind of a data acquisition system…Depending on the complexity of the system, the total number of sensors may vary from as little as one (a home thermostat) to many thousands (a space shuttle)" (Fraden 2010, p.5).

Understanding three key characteristics of how sensors integrate into systems helps illuminate important details of the relationship between an entity and exposures. First, sensors are exquisitely designed to be selective by specifying the environmental conditions to which they will be sensitive and insensitive. Sensors should minimally disturb the condition being monitored so its "true value" remains. Second, a sensor must be ready to collect data by means of detecting a condition, often by "active surveillance." Third, sensors are an integral part of the system. This relationship may be difficult to see since sensors are often remotely located.

(2) Logic mechanisms. Sophisticated internal logic mechanisms are currently being designed as more than basic *if-then* types of *on-off* switches (or gates.) Engineers are patterning logic mechanisms in tracking systems after the nervous system in living organisms so that they function as artificial neural networks. Ioannou (2012) believes his approach to adaptive control systems "…will be of great interest to the neural and fuzzy logic audience who will benefit from the strong similarity that exists between adaptive systems, whose stability properties are well established, and neural networks, fuzzy logic systems where stability and convergence issues are yet to be resolved" (p. xiv). The logical programming may be extraordinarily complex and mathematically rigorous to process the array of incoming variables, especially when multiple sensors are tracking multiple moving targets (Oh et al. 2013). Advanced logical programming integrates data from multiple sensors with pre-programmed ranges to further reduce target-tracking uncertainties by filtering out useless data or "noise" and to make determinations on the validity of data prior to specifying an output response (Luo et al. 2002).

(3) Output responses. The final step in tracking is to respond to target movements. Though the response is usually a necessary consequence when specific conditions are encountered, responses can range from a simple discreet on-off action, to a continuous range produced by an algorithm utilizing input variables. This is illustrated in the multiple uses for tracking eye movements which range from medical diagnostics, refractive surgery, human-computer interfaces, and commercial marketing (Gneo et al. 2012). Responses may be integral to a tracking system itself, such as a mechanism using stepping motors to keep a solar panel tracking the sun's movement.

Implications of this hypothesis help clarify biological adaptability

A fundamental design constraint is that the capacities for a designed entity to both relate to—and adapt to—external conditions must be built entirely *into* an entity. In terms of external conditions, these are insufficient to cause changes to an internal system's function. An engineer would identify conditions pertinent to performance and may specify some (amongst a myriad of conditions) as *variables* that are either present or not. The implication of the utilization of engineering principles and causation is reflected in the second half of our engineering-based, *organism-focused* characterization which keeps the operational spotlight on the organism rather than the environment.

An engineering approach focuses on whole systems and not individual elements. Since the entire system ceases to function with the loss of *any* vital element, then, no single element is declared to be causal. Only verifiable elements are included—and no vital element is omitted—in causal chains. With this primarily descriptive approach, causal chains in organism will: 1) generally link genetic or epigenetic information through, 2) specific systems

to, 3) modified traits and then, 4) to the specific environmental conditions to which they relate.

METHOD

We reviewed 342 articles from the scientific literature and 67 online reports (not duplicating the journal articles) from four topic areas pertaining to our hypothesis: 1) mechanisms conferring adaptability in varied taxa, 2) bioengineering, systems analysis, human-engineered tracking systems, robust control networks, robotics, and logical algorithms, 3) papers urging a greater integration of engineering analysis into biology, and 4) papers calling for modification of the current framework. The main body of data relating to the validity of our hypothesis deals with the varied mechanisms of adaptation. The body of this report describes 23 examples that are a select subset most representative of different types and mechanisms for adaptation. Table 2 (found at the end of the paper) lists 22 different highly regulated, non-random mechanisms that would not be classified as environmentally fractionated heterozygosity, but instead confer phenotypic diversity through other means to enable (usually) rapid adaptation to new environments. The *Reference* section and Table 2 identify many of the major source materials, but not all those reviewed.

Findings were analyzed for correspondence of mechanisms utilized in living organisms to elements of human-engineered tracking systems. Results were also investigated to answer specific questions about adaptive mechanisms: Do published results identify a predominant mechanism for adaptation utilized by organisms? Is modification of genetic sequence the principle mechanism to express phenotypic changes? Could variations be categorized, irrespective of genetic or epigenetic causality, in discernable patterns that would give clues that organisms were tracking environmental changes? The final area investigated involved cataloging mechanisms of adaptation that could potentially lead to speciation or other diversification events.

RESULTS

A remarkable number of non-random mechanisms specifically directing variable, adaptive responses to changes in distinct environmental conditions were reported. Several of the following examples thoroughly describe the chain from exposure to a changed condition to a phenotypic response by identifying sensors, logic mechanisms, and an output. These were analogous to the elements of human-engineered tracking systems. The significance of these results as evidence for the CET framework is withheld until the *Discussion* section.

By way of overview, results can be grouped into systems-based adaptive mechanisms and phenotypic responses. Non-random phenotypic output responses could be traced to both genetic and epigenetic mechanisms. Responses encompass modifications to physiological systems involved in maintaining cellular and organismal homeostasis. External modifications ranging from color variation to the total non-development of organs, major morphotypic reformations, and alterations in behavior. Some responses happened in a single organism within minutes, while others were found to affect entire populations and persist for several generations. Other novel responses did not result directly from either genetic or epigenetic changes but were the consequence of internal processes initiated upon detection of changed conditions. Of particular note was the identification of certain mechanisms and phenotypic responses which are both predictable and reversible.

We further subcategorize variations as developmental responses (both embryonic and juvenile)—these are the primary drivers of morphology—and adult responses, which directly influence the distribution of traits in diverse niches.

1. Developmental Response to Environmental Parameters

A. Embryonic development

The development of blind cave fish, *Astyanax mexicanas*, from a population of sighted river fish is the subject of active research. A critical question was how a river fish finding itself suddenly trapped in a cave environment would respond. Rohner et al (2013) investigated the activity of a common stress-related chaperone protein HSP 90 [heat shock protein 90] which has wide-ranging activity in cells, including a molecular mechanism for buffering latent, adaptable genetic variation (if present in the genome) and expressing it in response to differing environmental conditions. The target environmental condition was cave water abiotic factors (but not the presence of light.) Caves have other distinguishing conditions besides darkness. The ability of water to conduct electricity may show up to a five-fold decrease in cave water compared to surface streams. The authors presupposed that *A. mexicanas*, would respond (by an undescribed mechanism) to fluctuations in water conductivity. They showed that fish embryos which develop in low conductivity up-regulate HSP 90 response genes, which enabled expression of innate variability in eye size ranging from slightly decreased to absent within the first generation. River fish placed in low conductivity during larval development displayed a 50% increase in eye and orbit size variation. Additional tests showed that de novo mutations did not cause these genetic variations for small eye size, and after being "unmasked" they seemed to remain expressed in offspring.

For various reptiles including some lizards, snakes, turtles, and alligators, a single clutch of eggs may all converge on the same sex (Sifuentes-Romero 2017). Their sex is not determined by heteromorphic chromosomes, but by a developmental program using data they collect about their incubating temperature during a temperature-sensitive period. All females develop at one temperature, all males at another, and a ratio of both sexes at temperatures in between (ratios are further modulated by added data on sand moisture content.) This data is used to regulate different ratios of gene products for sex-affecting hormones. The process is triggered by temperature sensors in eggs discovered by Yatsu et al. (2015) in "…the first experimental demonstration of a link between a well-described thermo-sensory mechanism, TRPV4 channel, and its potential role in regulation of TSD [temperature-dependent sex determination] in vertebrates, shedding unique new light on the elusive TSD molecular mechanism" (p. 1).

B. Juvenile development

Phenotypic plasticity refers to the expression of different combinations of traits from a single genotype as an organism responds to different environmental conditions. Plasticity is a broader description for a graded response that usually correlates to the quantity of exposure to certain conditions. The nature of

conditions is broad and includes temperature, sunlight, moisture, chemicals, nutrition, population density, etc. Polyphenism is a type of plasticity where discrete, all-or-nothing expressions of traits happens upon exposure—usually to a threshold level. The change in color of an artic fox's fur from grey-brown to white in the fall is an example of seasonal polyphenism.

The significance of phenotypic plasticity to diversification and adaptation is described by West and Packer (2002) who say that the "environmental effects on trait morphology can be substantial, outweighing both genetic effects and reproductive advantages" (p. 1339). A few illustrations highlight the importance of external-condition detectors to initiate developmental, physiological, phenotypic, or behavioral changes, and the extent to which these changes could lead to speciation and diversification.

Observable phenotypic differences that distinguish species, and certainly genera, are assumed to be the result of genetic polymorphisms. But this assumption may not be accurate. Susoy et al. (2016) report experiments which indicate that some genera-level morphotypes of a fig-associated nematode *Pristionchus* are the result of polyphenism and not genetic polymorphism. Upon colonizing the island-like microecosystem of individual figs, symbiotic nematodes of the genus *Pristionchus* expressed a polyphenism with up to five discrete adult morphotypes per species. The principle target condition in this study was found to be both fig type and fig maturation. Since juvenile development cannot be cultured outside of figs, any environmental cues detected during development associated with differing morphs cannot be identified. Yet, the five major morphotypes identified were associated with fig type, fig phase (early and late interfloral), and transit on, versus through, their specific wasp vector (*Ceratosolen* spp.). Genetic sequencing demonstrated that from a single genotype, developmental plasticity had led to discontinuous novelties whose variation exceeded level of genera in the same family. They concluded that this was a case of "macroevolutionary-scale" diversification, with some structures having no analogs in other nematodes, without genetic divergence.

Tadpoles of the tree frog *Hyla chrysoscelis* demonstrate developmental phenotypic adjustment when exposed only to aquarium tank water that had harbored dragonfly larvae of the tadpole predators *Aeshna* or *Anax*. McCollum and Leimberger (1997) document that tadpoles have exquisite capability to, "detect waterborne chemical" substances "produced by predators" (p. 616). Post-exposure, tadpoles developed a thick, muscular, bright red tail which increased their probability to escape future predation better than tadpoles isolated from predator exposure during development. Relyea (2005) followed up on the tadpole-predator study to determine whether a plastic trait expressed in one generation could be passed on to offspring which themselves experienced variable levels of the exposure during their development. He concluded that "predator-induced traits can frequently be heritable, although the magnitude of heritability can be wide ranging across environments. Moreover, the plasticity of these defenses also can be heritable" (p.864).

Multiple studies identify an exquisite detection-response linkage in some organisms. They detect the presence of predators, respond with phenotypic adjustments either during development or as adult forms, and then pass a tendency for the adjusted form on to offspring. Stabell and Lwin (1997) conducted experiments to determine elements of an underlying mechanism that might explain why the body depth and muscle mass of crucian carp, *Carassius carassius*, increases in the presence of the predator northern pike, *Esox lucius*. They demonstrated that crucian carp did not respond with growth changes after exposure to either the pike itself, nor to pike-fed Arctic char, *Salvelinus alpinus.* Morphological changes occurred only after carp were exposed to pike which had been feeding on other crucian carp, or when exposed to water containing skin tissue (prepared and homogenized) of conspecifics. They conclude that chemical substances from the skin of conspecific fish are a stimulus for induction of the phenotypical changes.

Another review paper indicates that the role of phenotypic plasticity for diversification and speciation may be going unnoticed. Pfennig et al. (2010) document cases of speciation resulting from phenotypic plasticity and conclude that "generally, phenotypic plasticity can play a largely underappreciated role in driving diversification and speciation" (p. 459). They point out that an organism's abilities to rapidly respond to changes facilitate diversification since "… alternative resource-use morphs might be particularly effective at facilitating speciation because the same conditions that promote resource polyphenism simultaneously foster speciation's three components: genetic isolation, divergence and reproductive isolation" (p. 462).

2. Adult Response to Environmental Dynamics

A. Phenotypic response

Patterson (2007) discusses how the thickness, length, and color of a male lion's mane, which may vary over the course of a single year, depends on its ability to detect at least two conditions: temperature and rainfall. West and Packer (2002) also note how the presence of these conditions, available nutrition, and a non-environmental exposure (age), play a more prominent role than genetics in determining the characteristics of mane.

The speed of adult phenotypic alteration is demonstrated in desert locusts which can change reversibly between solitarious and gregarious phases. These are so dissimilar in physiology, morphology and behavior, that they were recognized as different species until 1921. Rogers et al. (2014) shows that, when a previously discovered sensor on the hind femora is subjected to increased tactile stimulation due to forced crowding, solitarious locusts begin within one hour to exhibit the behaviors of the long-term gregarious locusts. Then by the next molt (within 4-7 hours) they completely morph into the gregarious phenotype. Miller et al. (2008) establishes how "depending on their rearing density, female desert locusts *Schistocerca gregaria* epigenetically endow their offspring with differing phenotypes…[which] affords organisms robustness against environmental fluctuation…[and is] persistent for some duration in the absence of inducing stimuli" (p. 300).

Adult forms that maintain sexual plasticity demonstrate the potential of adult phenotypic modification. In a large resident female Blue-headed Wrasse, *Thalassoma bifasciatum*, ovaries regress and testes grow within a single day if a territorial male is lost (Warner and Swearer 1991). Even though Godwin et al. (2008) assert that the environment is sending information to a female

Wrasse, they document a thoroughly internal mechanism mediated by the hypothalamo-pituitary-gonadal axis in a female detecting an absent male, and subsequent sexual transformation.

B. Epigenetic response

Some theorists speculate that hydrogen sulfide (H_2S) is essential to a natural origin of life, shapes evolutionary diversification, and contributes to mass extinctions (Olson and Straub 2015). H_2S is largely an environmental toxin introduced via natural geochemical and biological processes, or industrialization. Kelley et al. (2016) looked at the target condition of H_2S in three river drainage systems in Mexico. They found that when exposed to varying H_2S concentrations, genetic transcription within gill tissue of small live-bearing fish of the *Poecilia mexicana* species complex demonstrated, on average, 1,626 up-regulated and 1,827 down-regulated transcripts adaptively correlated to mediating H_2S flux into the fish through diffusion, regulating H_2S homeostasis, and mitigating side effects by detoxification.

To investigate one mechanism which might link ambient temperature changes to adaption, Weyrich et al. (2016) obtained five genetically heterogenous male wild guinea pigs (*Cavia aperea*) originating from Argentina and Uruguay. The environmental target condition was ambient heat. Researchers proceeded on the assumption that guinea pigs have a neurological mechanism to detect temperature changes (without identifying a specific sensor.) Males sired an F1 generation. Prior to the next mating, males were kept in cages placed on a heating plate which kept the floor at 30 °C for one cycle of spermatogenesis (60 days). F1 and F2 generations were obtained after mating with the same females. Comparison of epigenetic methylation of specific genomic regions in liver and testis between pre-and-post heat treatment found epigenetic changes in both paternal guinea pigs and F2 offspring on 13 of 19 temperature-regulating genes, and 12 additional genes involved in temperature regulation had their promoters epigenetically altered.

If an adult mouse learned to fear the scent of fox urine, such information could be useful to offspring. Dias and Ressler (2014) conditioned male lab mice (*Mus muluscus*) to fear a target condition: acetophenone (cherry blossom) odor. With each exposure, males received painful foot shocks. The sensor was an olfactory bulb developmentally controlled by the *M71* gene related to acetophenone. Offspring of males mated to naïve females had an increased number of odor-specific cells, increased size of odor-specific glomerulus in their nose, and 200% increase in response to acetophenone compared to controls. Phenotypic changes were mediated by epigenetic methylation of an unaltered *M71* sequence. Offspring conceived by artificial insemination from sperm of acetophenone-fearing fathers had similar changes.

The molecular basis of the adaptive changes in Darwin's finches on the Galapagos islands is assumed to be genetic variation fractioned out through differential survival. However, McNew et al. (2017) note that "growing evidence suggests that epigenetic mechanisms, such as DNA methylation, may also be involved in rapid adaptation to new environments" (p.1). Comparing over 1,000 birds in adjacent "rural" and "urban" populations of each of two species of ground finches (*Geospiza fortis* and *G. fuliginosa*) on Santa Cruz Island, they found significant morphological differences in beak depth, width, length, chord length, and tarsus length between urban and rural populations of *G. fortis* (but not for body mass), and no statistical changes for *G. fuliginosa*. Copy number variations between populations of either species were mostly unchanged. Phenotypic differences were associated with the dramatic DNA methylation variances discovered between urban and rural populations. They speculate that a change toward human-associated foodstuffs is the target environmental condition. Urban finches face far greater exposure. They reported no identifiable link between the exposure and epigenetic changes, which were explained as "environmentally-induced epimutations."

C. Distributive response

Drosophilids have innate and species-specific humidity preferences. Enjin et al. (2016) were the first to describe genes and neurons necessary for hygrosensation in the vinegar fly. The target environmental condition is relative humidity, which is used as a cue to navigate to different environments. *D. melanogaster* has sensors for dry, moist, and cold conditions through neuron tips in a specialized organ in the antenna. They identified the detector enabling *D. melanogaster* to track humidity changes and migrate accordingly.

Gulls of the family *Laridae* are found in both freshwater and saltwater environments. Barrnett et al. (1983) describe the de-novo membrane biogenesis of an "avian salt gland." The gland's osmotic action extracts excess sodium from plasma and excretes it through a port in the nasal beak. The target environmental condition is brackish water. Gulls possess an osmotic sensor in the cardiac vasculature. After detecting increased sodium ion concentration, neurologic and endocrine actions control cell differentiation and hypertrophy to form the gland. The organ formation is reversible, enabling gulls to migrate between freshwater and brackish estuaries.

3. Reversibility of Adaptation

If a population steadily expresses traits highly specialized for one niche, then it could head down a genetically unrecoverable one-way street. Mundy et al. (2016) observed that this circumstance could "lead to a genetic constraint on adaptation if the environment subsequently changes" (p. 1) which forces them into occupying a certain niche or dying. Their concern, within the current framework, is that "in evolutionary biology, Dollo's Law [of irreversibility] proposes that complex adaptations cannot be reacquired easily once lost" due to degeneration of developmental pathways as mutations accumulate.

Other theorists question the validity of the "irreversibility" concept. Reversals have been documented in the reappearance of teeth in certain frogs (Wiens 2011) and in the lineage of an extinct kangaroo (Couzens et al. 2016), and in beak morphology in a lineage of Hawaiian birds (Freed et al. 2016). These researchers also document, by way of historical background, the reacquisition of certain traits, including: reversals for wings in stick insects, coiling in snail shells, color vision, eggshells in boid snakes, and others. Two microbiologists (Ogbunuga and Hartl 2016) working to treat drug-resistant malaria through various paths of "reverse evolution" stated, "the lack of a coherent understanding of reverse evolution is partly due to conceptual ambiguity: the term 'reverse

evolution' is misleading, as it implies directionality in a process [Darwinian evolution] that is near-sighted and agnostic with regard to goal. This has spawned similarly dubious concepts, such as Dollo's Law, asserting that evolution is intrinsically irreversible…" (p. 2). Considering the dwindling evidence for the law-like nature of Dollo's Law, Collin and Miglietta (2008) conclude that support for this view has become untenable.

The mechanisms for recovery of ancestral states are slowly being deciphered. Evidently, the developmental pathways and genetics for idled traits are often retained. Galis, et al. (2010) found that, after loss of a structure, in many cases "…the genetic and developmental architecture to develop such structures continues to be fully present…" (p.2466). Couzens et al (2016) also reviewed how reversibility may be variably widespread among organisms, and state "…it has been argued that trait reversibility may be promoted when there is reutilization of conserved developmental pathways…[and] the reutilization of regulatory pathways and constituent genes is widespread in development and ancestral states are recoverable across a diverse spectrum of metazoan structures." (p. 568). Collin and Miglietta. (2008) also describe cases where genetic and developmental pathways of "lost" traits are reactivated. Two other aspects of these reversals make them quite remarkable. First, there are the multiple instances of *repeated* oscillation between loss-and-reversal which, second, happened over time spans purported to range from tens to hundreds of millions of years—which counterintuitively indicates that the genetic and developmental pathways remained undegraded for astonishingly long times (Couzens et al. 2016; Freed et al. 2016; Wiens 2011).

Reversion to a prior phenotype in a population would not necessarily involve a back-mutation. Hubert et al. (2016) acknowledge that "reversibility of evolution is a long studied and questioned aspect of evolutionary biology. Especially in small populations, slightly deleterious mutations may accumulate and become fixed by genetic drift" (p. 1). They report on European carp bred to be scale-free (homozygotous.) A population transported in 1912 from France to Madagascar (which had no native carp populations) colonized natural waters. By the 1950s, carp had re-grown scales. This development was inexplicable, since they could cite no studies confirming a survival value of scaled over non-scaled phenotypes. Their analysis found that these fish were still homozygous to be scale-free, but that scale growth was under polygenic control and the current fish were expressing scales by accessing another genetic route. They conclude that these "visible and striking" findings are "…evidence for a rescue of the wildtype-like scale cover…[by] polygenes from standing genetic variation…[through] other routes than reversion mutation, and suggests that natural populations can host enough capacity for adaptation on the short-term to face a sudden environmental change, even if a harmful mutation was formerly fixed" (p. 6).

DISCUSSION

1. Variation appears directed, not random

Variation perpetuates "in the classical view…[when] species experience spontaneous genetic mutations that produce various novel traits—some helpful, some detrimental. Nature then selects for those most beneficial, passing them along to subsequent generations. It's an elegant model." (Whitehead 2013, p. 1) Thus, in the classical view, genetic heterozygosity generated via random mutations, or conceivably originating as a standing assemblage, becomes fractioned into subpopulations by the struggle to survive in challenging environments. In dramatic contrast, the results from our survey of the literature (summarized in the regulated changes described above and further detailed in Table 2) provide evidence of multiple controlled mechanisms that bias or direct phenotypic variation in a population toward specific, adaptive outcomes. However, it has taken several decades to discover these alternative mechanisms; recognize and collate them into non-random, directed mechanistic categories; and then to realize their contributions toward variation, diversification, and adaptation. As Charlesworth et al. (2017) discuss, researchers are just beginning to determine the *relative importance* with regard to diversification and adaptation of these dissimilar and distinct mechanisms compared to, or perhaps contributing to, the status quo genetic fractionation explanation which they endorse. Our survey of the literature uncovered evidence that epigenetic changes are the product of systems that have sensors to detect changed conditions, that process data within logic mechanisms, and that are observed to have rapid and targeted responses. It appears that regulated, condition-sensing epigenetic mechanisms also "prime the pump" for specific adaptive variation in offspring.

For example, after Weyrich (2016) detected that paternal Wild guinea pigs and their offspring had improved long-term resilience to temperature increases, he concludes, "we demonstrated immediate and inherited paternal epigenetic response with a potential adaptation reaction that occurred in response to increased ambient temperature in a wild genetically heterogeneous mammal species, the Wild guinea pig" (p.8). That epigenetic control can be adaptive and transgenerational was corroborated by Ressler commenting on his journal-published research (see Dias and Ressler 2014): "such information transfer would be an efficient way for parents to 'inform' their offspring about the importance of specific environmental features that they are likely to encounter in their future environments" (Le Roux 2013).

In addition, Rohner et al. (2013) deduce from their results on blind cave fish, *Astyanax mexicanas*, from a population of sighted river fish that "because multiple variants can be unmasked at the same time, this system provides a mechanism to create complex traits in a single step… (p.1372) [that] would have helped potentiate a rapid response to the cave environment" (p.1375). Variants that can be "unmasked" *precede* the environmental challenge. Unmasking of variants is under tight, internal regulation so that responses occur only after organisms detect specified changes. Also, the expression of variants occurred repeatedly, within one generation, after fish were exposed during development to either an HSP90 inhibitor or to water conductivity mimicking cave conditions. These internally, self-adjusted phenotypic outputs enable this fish's adaptive response to migration into cave environments. Taken together, these results indicate that expression of variants is a necessary and predictable consequence of an internal logic-based algorithm altering embryonic development. Those fish with unmasked variants that successfully fit the specific cave conditions tend to become dominant in a new population.

Further, we found that directed phenotypic change—enabling

colonization of an "empty niche" (i.e., an environment not previously inhabited by a species)—and its potential contribution to diversification can be extensive. Susoy, et al.'s work (2016) on the fig-associated nematode *Pristionchus* found "macroevolutionary-scale diversification without genetic divergence…[so] that rapid filling of potential ecological niches is possible without diversifying selection on genotypes. This uncoupling of morphological diversification and speciation in fig-associated nematodes has resulted from a remarkable expansion of discontinuous developmental plasticity" (p. 1). The morphotypic changes exceeded what would be observed at the species level. They summarize that "…given the 'empty niche' conditions predisposing an ecosystem to the trophic diversification of its colonists…and [given] that developmental plasticity can lead to the multiplication of discontinuous novelties from a single genotype" (p.6) substantial phenotypic change can *precede* speciation.

Finally, we found that many taxa exhibit mechanisms that enable reversal to ancestral states in future generations. This ability correlates to a useful "turn back" mechanism that engineers embed in tracking systems on drones. It seems reasonable that organisms could also reverse direction so that they could escape getting trapped in a genetic dead-end. It appears this can occur through the persistence of underlying "developmental architecture" (Galis, et al. 2010) that "reanimates" genetically mothballed features. This compares to engineered "turn off-turn on" control mechanisms. The fact that genetic and developmental pathways can be deactivated but remain functionally intact for generations (giving the appearance of a "lost" trait), and then reactivated during embryonic development in future generations, is mechanistic evidence of how some populations can continuously track environmental changes even over long periods. Evidence for reversibility at phenotypic and morphotypic levels appears contrary to evolutionary and heterozygous fractionation models.

Thus, it appears that multiple mechanisms exist which direct variation toward specific adaptive responses to specific environmental changes, contrary to the classical view with its reliance on random variation. These mechanisms can reasonably be described as "tracking" the environment in the sense that they appear to *detect* environmental changes, *process* this information, and then *adjust* traits in an adaptive manner. Thus, they appear to have features correlating to all three components of man-made tracking systems (sensors, logic mechanisms, output responses), just as expected from our Continuous Environmental Tracking hypothesis.

2. Continuous Environmental Tracking (CET) is the foundation for adaptation

CET is purely a descriptive title for what creatures seem to do as they adapt. CET assumes that the most accurate way to explain the function of adaptable biological systems is with the same engineering principles that govern human-engineered tracking systems. CET, therefore, could become the basis for a new framework (i.e., framing observations, interpreting facts, and guiding research) meant to replace the current framework for understanding adaptability. This engineering-based, organism-focused characterization of organism's systems is only changing the *way* existing data are organized and interpreted. This means that other approaches are not necessarily excluded.

With CET, we assert that adaptation *primarily* occurs during embryonic and juvenile development (genotype, morphotype, phenotype), and as the result of continuous surveillance and shadowing of the environment (i.e., response to environmental parameters made possible by sensors and mechanisms), and secondarily as an adult response to environmental dynamics (phenotype, epigenotype) (Figure 1). This assertion is made based on our findings (highlighted in *Results* and detailed in Table 2) regarding the observed *internal* mechanisms of adaptive change. The current framework of random variation, randomly efficacious hit-and-miss responses, and subsequent death-driven fractionation, is not excluded as an explanation, but either demoted in importance or, more properly, seen as a contrary process. Instead, this approach views organisms as active, problem-solving entities that respond to environmental challenges instead of passive entities which are shaped by environmental challenges. CET thus implies that adaptation is fundamentally produced by *regulated gene expression* and *not gene inheritance,* per se. Adaptation at the population level then results from a combination of directed variation in individuals (resulting from CET during all life stages) and differential inheritance of those variations (i.e., from unequal distribution and reproduction) by the next generation.

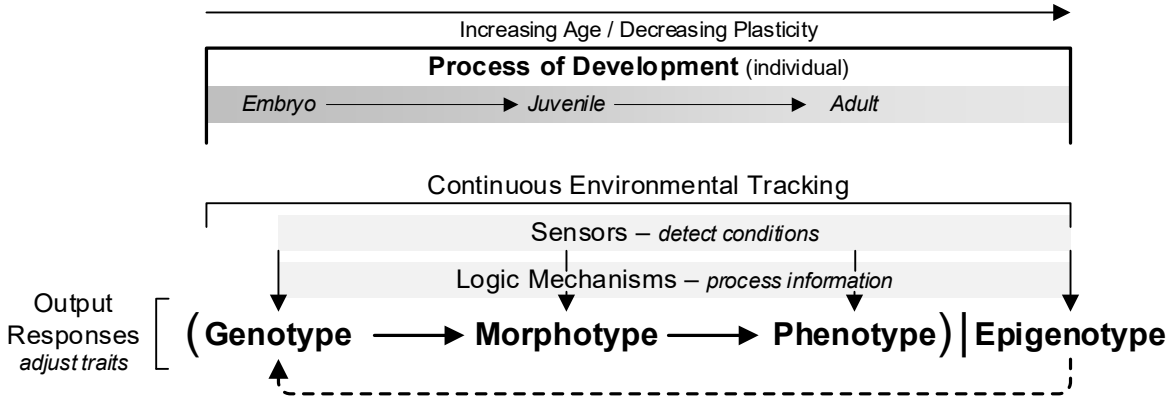

Figure 1. Continuous Environmental Tracking (CET) results in adaptation at every stage of development through the detection of changes in environmental conditions (via Sensors), processing of condition-response information (via Logic Mechanisms), and adjustment of traits (via Output Responses).

3. CET aptly describes the evidence

Theorists are still absorbing the full implications of these newly-discovered internal mechanisms of adaptation and are trying to determine if they fit within current theory. So now, in this rare time of unsettled and open-ended discourse among evolutionary scientists over theories of adaptation, it is very appropriate to frame new findings using an engineering-based, organism-focused structure into which they seem to naturally fit. We now consider additional observations within the literature which seem to point to CET, clarify what CET implies, and favorably contrast an internalistic, engineering-based framework to current theory.

A. "Regulated," "rapid," "repeatable," and "reversible" describe adaptable tracking systems

A significant observation from the literature review is that internal mechanisms of adaptation yield responses habitually characterized as "regulated," "rapid," very often "repeatable," and, surprisingly at times, even "reversible." These words fittingly describe the purposeful outcomes of robust, adaptable engineered tracking systems. Prior to the discovery of regulated phenotypic outcomes, Stephen J. Gould (1994) contrasted the random, death-driven parsing out of genetic variation he assumed was evidence supporting current theory, with the controlled, purposeful outcomes of internally-regulated systems. He said, "natural selection is a theory of 'trial and error externalism'—organisms propose via their storehouse of variation, and environments dispose of nearly all—not an efficient and human 'goal-directed internalism' (which would be fast and lovely, but nature does not know the way)" (p. 6). New findings provide evidence that "nature" does indeed "know the way."

B. Innate capacity controls responses to the environment

Consistent with Gould's recognition that the internalist and externalist approaches are contradictory, discoveries of internal systems producing what seem like targeted responses are forcing a move away from environment-driven, selectionist explanations. Researchers have started coining terms for adaptive mechanisms that descriptively sound very close to engineered, *innate* capacity such as: "stem plasticity" (Susoy et al. 2016, p. 6), "standing genetic variation" (Rohner et al. 2013, p. 1372), "natural genetic engineering" (Shapiro 2011, p. 161), "cryptic genetic variation" (Sangster et al, 2008, p. 2963), "facilitated phenotypic variation" (Gerhart and Kirschner 2007, p. 8582), "adaptively inducible canalizers" (Meiklejohn and Hartl 2002, p. 468), and "evolutionary capacitors" (Rutherford and Lindquist 1998, p. 336). All of these terms describe means to access innate self-adjusting capacity for what Waddington noted in 1942 as, "a suitable genetically controlled reactivity in the organism" which he referred to as "a set of alternative canalyzed [innate] paths" (Waddington 1942, p. 564, 565). Taken together, these results establish a growing recognition that internal systems specify if organisms can, and how they will, relate to external conditions.

C. Diverse inheritance mechanisms facilitate transgenerational environmental tracking

Most of the mechanisms reviewed here demonstrate that organisms utilize input elements (sensors) to sense environmental conditions, logic mechanisms to interpret those inputs and determine responses, and output mechanisms to implement responses. Additionally, these same elements are utilized in tandem to trans-generationally track environmental changes in two distinct steps. That is, first, the parent directly detects an environmental change, which is processed, and then an output response is sent into the milieu of the developing offspring which, second, detects it and responds with self-adjusted phenotypic outputs suitable to conditions detected by the parent. Recall the paternal mouse's detection of acetophenone—which started the process leading to epigenetic modifications in offspring modifying expression of the M71 gene, as documented in the work of Dias and Ressler (2014). A Duke University study on the tiny worm *C. elegans* observed detailed maternal-to-offspring signals about a nutrition-deprived environment (Hibshman *et al* 2016). It described "a genetic network that mediates effects of a mother's diet on the size and starvation resistance of her offspring" that worked by "signaling through [an] insulin-like receptor" which "function in the mother to transmit information about her diet to her offspring." Remarkably similar findings, most likely due to epigenetic modifications, were noted for humans as well. Children born to parents exposed to starvation during conception and gestation had an increased risk of type 2 diabetes and hyperglycemia, which indicated to researchers that in-utero epigenetic modifications predisposed them to be calorie hoarders in the face of being born into a starvation environment. The transgenerational odds of developing hyperglycemia were about 2:1 in both children *and* grandchildren, while there was about a 75% increased risk of type 2 diabetes in the children of starved parents (Li *et al* 2016).

We are not arguing that cross-generational variability of traits that could result from standing heterozygosity is not a source of adaptive phenotypes, but that this mode of inheritance should be viewed no differently than any other trait that is a product of internally regulated systems that produce a potential solution to an environmental challenge. Furthermore, the mechanisms described here signal that the penultimate role in the current framework accorded to genetics, primarily genes, to propel adaptation should be diminished. Evidence indicates that the genome may function as any other sub-system of the cell since, ultimately, it is the organism which senses and responds to external variables (Keller 2014). But it should also be noted that the traits derived during development principally under genetic/epigenetic control are an important, but not the sole, determinant of the fit of offspring to its niche. Heredity would broadly include all mechanisms facilitating offspring-parent resemblance that promote the maintenance of homeostasis or enhance the suitability of an organism to its niche, which includes: genetic, epigenetic of all types, physiological, microbial symbionts, behaviors, physical resources, parent-altered ecological niches, and population-modified cultures. These mechanisms are wide-ranging in mode of transmission, rate of effect, and manner of action.

D. Environmental tracking starts as a developmental necessity

Environmental tracking begins early in development and continues throughout the lifetime of an individual organism. An engineering-based, organism-focused framework doesn't view organisms as constructed by their parents or their environment. Organisms begin with innate capacity to self-construct, self-metabolize, self-maintain, self-repair, self-adjust, reproduce, and transfer an inheritance. Genetic and non-genetic factors transferred from parent

are viewed from a design perspective as highly influential *variables* utilized in the offspring's assorted developmental algorithms, but they do not control the entire development. External conditions do not bypass an organism's boundary and directly control the expression of its genes, but expression is a system-derived outcome with no single system element elevated to causal status. Embryonic control systems, sensors, and developmental response mechanisms map the course of development by constantly monitoring and self-adjusting to internal and external states. Recall how tadpoles of the tree frog *Hyla chrysoscelis* will rapidly morph into a form with thick muscular bright red tails when they "detect waterborne chemical" substances "produced by predators" of the dragonfly larvae in the water (McCollum and Leimberger 1997, p. 616). The detection of dragonfly larvae chemical signatures is only data. That data is a reliable conditional input to their developmental program that directs them to develop tails to better escape predation. Sensors specifically tuned for dragonfly larvae are the actual "triggers" within organisms to initiate their self-adjustment processes.

E. Environmental tracking requires the whole organism

The tracking elements of sensors, logic mechanisms, and response systems may reside very distantly from each other on the organism but nonetheless work very tightly together. Possessing a sensor is not enough. Programming internal to the organism *specifies* what constitute actual environmental "signals" or "stimuli" for itself (which explains why a myriad of other exposures are never stimuli for the sensor). This implies that adaptations result from the functioning of the whole organism, and not merely from genomic changes. Likewise, the research of Shapiro (2016) demonstrates that in one of it several roles, the genome looks like a dynamic logic-housing sub-system of the cell (or organism) supporting its role of responding to detected conditions. Thus, environmental tracking ability appears to be irreducible below the organismal level.

F. Targeted, rapid solutions solve environmental challenges

As organisms track environmental targets, their responses are directed toward solutions specifically targeted to the challenge. *Astyanax mexicanas*, progeny's reduced eye size in cave conditions is a single-step, focused response. *Mus muluscus* pups' increased glomeruli and neuronal support was specifically targeted for acetophenone. Crucian carp, *Carassius carassius*, responded precisely to gape-limited predators by rapidly morphing into a larger size. *Cavia aperea* guinea pig pups had epigenetic changes on genes specific for temperature regulation, and so forth.

Targeted responses indicate mechanisms operating in a vastly different manner from those which would produce the random results expected in the dominant framework where "further improvements were accidentally thrown up (by genetic mutation, according to modern biology) then retained in turn" (Millikan 2014, p. 63) or the hit-and-miss results that Peter Godfrey-Smith states are expected from a selectionist framework that must "…rely on a process that can be described loosely as 'trial and error'. New variations are produced in a spontaneous and unintelligent way, and a few successful variants are kept while others are discarded" (Godfrey-Smith 2010, p.29).

G. Epigenetic mechanisms facilitate rapid phenotypic "flexing"

Phenotypic flexing captures two concepts related to engineered robustness: the ability to rapidly "bend" phenotypically, but not break, to stressful conditions; and the ability to return to "baseline" if conditions revert. Accumulation of mutations within the germ line, or even variation from a standing population of heterozygous alleles, seems far too slow to solve some challenges. Internally-regulated epigenetic mechanisms variably mark specific nucleotides of DNA with different molecules which exert control over how the information in DNA is expressed, but without changing the genetic sequence.

From a design-based view, genetic stability combined with plastic variable expression confers the ability for phenotypes to rapidly "flex" to a rapid environmental change. CET would imply epigenetic changes such as rapid (within one generation) adaptations that of necessity fill an intermediate time gap between very rapid physiologic changes and slow, multi-generational genetic changes. Weyrich et al. (2016) sum up this function: "The regulation of genes and their expression is fundamental for immediate adaptation processes in the same generation. In addition, the inheritance of responses to experienced changes (adapted traits) is fundamental for long-term adaptational memory. The mechanism regulating gene expression and conferring such immediate and inherited adaptation is 'epigenetic response'" (p.1).

H. Diversification as a continuum of adaptations succeeding continuous tracking

With CET, environmental tracking should happen continuously from development through the time offspring inherit niche-suitable resemblances. Therefore, adaptive phenotypes produced by epigenetic mechanisms or phenotypic plasticity could be viewed as a *continuum* of change which would range from rapid physiologic to multi-generational. Speciation may not be a "goal," "target," or "end-product" as researchers generally understand it, but it could be thought of as simply a transient manifestation of a discreet set of characters along a continuum of adaptation.

I. CET of variable conditions correlates with observed episodic speciation rates

The CET hypothesis implies that organisms track environmental changes at whatever rate and manner that they occur. This contrasts with a fundamental tenet of the current framework: gradualism. Gradualism holds that the rate of diversification, (as seen by the magnitude of morphologic change within a taxon,) must be essentially linear in terms of the number of species over time. Stephen J. Gould explains why this belief may not consistently align with actual observations but is a core principle of selectionist theory nonetheless: "substantial change might occur as a very rare event, but *most* alteration must be insensible, even on geologic scales." (Gould 2002, p. 147, emphasis in original). CET is free from a constraint on how rates will be interpreted. Therefore, it would be expected that at times the rate of change could be described as linear, episodic, or asymptotic, and the trajectory may be either positive or negative.

Jeanson (2015) completed a unique in-depth study of mtDNA, amassing data he used to graph the cumulative total number of species for representative kinds of organisms versus time, covering

approximately the last 4000 years. His data could be interpreted as episodic speciation across diverse taxa as they track rapid and slower rates of environmental change. Though Jeanson interprets the data as a linear change, we see his plotted data as consistently resembling episodic graphs. These data seem to fit CET well since an abrupt and extensive change in conditions seems more likely to lead to the appearance of unique characteristics. Tight environmental tracking could show a tendency of traits to change rapidly at times and then, during periods of steadier environmental change, organisms would "ratchet" an ever-closer fit of their traits to the conditions—a phenomenon which was noted by Reigner (2015). Rohner et al. (2013) demonstrates that complex traits in organisms can appear in a single step as they track sudden changes in conditions. This observation fits the episodic changes we see in Jeanson's data and may explain why some species could appear without morphological intermediates.

J. CET harmonizes rapid acquisitions of similar traits by diverse organisms

A remarkable biological phenomenon occurs when two unrelated organisms express very similar traits (usually in similar environments), or the when offspring rapidly express similar new traits when relocated to remote islands with similar conditions. A tracking mechanism could explain how two or more groups of organisms arrive at a specific phenotypic "location." Thus, if two unrelated organisms are actively tracking similar environmental changes and their internal logic centers use similar algorithms to directly express suitable traits as responses, then the fact that they exhibit similar features is explained as a particular and necessary consequence of similar internal plans within independent organisms. For example, Esquerre and Keogh (2016) show that pythons and boas display strong and widespread morphological similarity when they occupy equivalent ecological niches ranging from arboreal to aquatic. They demonstrate strong coupling of similar phenotypic traits to ecological diversification. Losos (2017) documents a similar coupling of rapid, predictable phenotypic expressions in equivalent ecological niches in anolis lizards—and across many other taxa—which appears to be normal throughout the known history of some groups of organisms (Moen et al. 2015). These appear to be targeted solutions in independent groups of organisms following a specific plan which rapidly closes in on similarly suitable traits. This may be better described as "rendezvous" rather than "convergence."

CONCLUSION

Bateson et al. (2017) welcomes sharp, legitimate differences of interpretation regarding data. Hence, we offer a new framework for understanding biological adaptability that reinterprets findings in the literature in view of the assumption that biological systems and functions are most accurately explained by engineering principles.

Using an engineering approach to reinterpret data led us to an engineering-based, organism-focused characterization of adaptation. We hypothesized that organisms actively and continuously track environmental variables and respond by self-adjusting to changing environments—utilizing the engineering principles that constrain how human-designed things adapt to changing conditions—resulting in adaptation. We termed this hypothesis *Continuous Environmental Tracking* (CET).

CET expects to find that organisms adapt by using mechanisms with elements analogous to those underlying the self-adjustable property of human-engineered tracking systems. These are: input sensors, internal logic mechanisms to select suitable responses, and output actuators to execute responses. We came to our hypothesis by reinterpreting findings and formalizing biological adaptability within a framework of engineering design, considering: (1) objectives, (2) constraints, (3) variables, and (4) the biological systems related to the previous three. Reinterpreting observations of behavior suggests a new description of what organisms achieve when they adapt: i.e., the design *objective*. Organisms appear to continuously track environmental changes and self-adjust with suitable and often heritable traits, resulting in adaptation.

A basic design *constraint* is that the capacities for a designed entity to both relate to—and adapt to—external conditions must be built entirely *into* an entity. Interpreting the data to identify the location of adaptive capacity at the organism-environment interface suggested that, without exception, adaptive mechanisms reside internal to organisms; mechanisms controlling how adaptation happens appear internally regulated and integrated.

Engineers identify external conditions pertinent to performance as *variables.* They are either present or not. Using engineering principles to interpret the role of external conditions suggests that conditions are detected and their presence is recognized as input data that innate systems process. Additionally, external conditions themselves were interpreted from an engineering approach as insufficient to cause the production of adaptive traits.

To evaluate whether biological function could be framed by an engineering approach, and in order to determine if the CET hypothesis is valid, we performed an extensive literature review for study results across various taxa. We identified multiple internal mechanisms utilizing diverse sensors coupled to complex logic mechanisms that produced condition-specific output responses. Not only did organisms use elements analogous to engineered tracking systems, they were used in ways that can readily be interpreted as continuously tracking environmental changes. Biological adaptations often occurred within one generation. We found an array of phenotypic self-adjustments functioning as purposely designed "targeted solutions" to the challenges of dynamic external conditions. Adaptations frequently occurred from well-organized modifications of genetic output, often executed at points during development that significantly affect the traits at an organism's environmental interface. CET thus implies that adaptation is fundamentally produced by regulated gene expression and not gene inheritance, per se.

The underlying mechanisms enabling biological adaptations can be described as non-random. This observation is in stark contrast to the randomness characterizing the standard framework that posits tiny, accidental "hit-and-miss" phenotypic adjustments fractioned out to lucky survivors of deadly challenges. Adaptive mechanisms were characterized as regulated, rapid, repeatable, and predictable. This depiction is anomalous to selectionism's iterative stacking of fortuitous results, precisely because regulated, rapid, repeatable, and predictable describe purposeful outcomes of engineered systems. But, it is consistent with an engineering-based premise

that adaptation results from heritable programming intended to enable creatures to adapt and fill changing niches.

Continuous Environmental Tracking is the foundation of a new framework for understanding adaptation, but several key gaps in our framework need to be filled by further research. For instance, we predict further investigation will demonstrate that many variations which are assumed to be—but never documented to be—random in nature were never random at all but directed. Also, more research on sensors in needed. It is notable that only a small fraction of the papers we reviewed identified a sensor for the exposure of interest. In addition, they did not indicate that identification of the sensor and the signaling pathway would be an important area of future research. Though it would be likely that most researchers would instruct students against drawing a causal link between the association of an exposure to an outcome without providing a plausible biological mechanism to explain the outcome, most researchers were content to report that the exposure caused the response. Yet, it seems that during experimental setup they did intuitively look for something in their research subject that would relate directly to the exposure. Also requiring more research is the characterization of the reversibility of adaptation and the determination of whether it is rare or common. Additionally, analysis of whether heterozygous fractionation models can plausibly explain reversibility is lacking, and more analysis of the role and impact of genetic loading and genetic drift on adaptation in a CET-based framework is needed. In this vein, more work needs to be done to detail how a CET-based framework differs in assumptions, concepts, interpretations, and predictions from current theory.

The CET framework demonstrates that an engineering-based, organism-focused framework can contribute to science because it does suggest testable hypothesis about biological function—that are being overlooked under the current theory—and make predictions of findings In addition to illuminating regulated mechanisms producing targeted responses and system elements corresponding to human-designed tracking systems, CET provides an engineering-based explanation for epigenetic mechanisms as a way for organisms to rapidly "flex" phenotypically to sudden environmental changes and then possibly revert to "baseline." CET allows for episodic changes by tracking mechanisms that can "step up" or "step down" as needed, perhaps without producing noticeable intermediate forms.

CET expects and explains "convergence" by diverse organisms on similar traits and thus accounts for why some evolutionists now claim that evolution is "predictable." CET goes beyond simply asserting that common function is due to common design, since CET specifies the systems which are the common design and can anticipate where the common traits might appear.

CET incorporates the engineering principles that are likely essential to make correct cause-effect associations for biological functions. Engineering causality is different from philosophical, psychological, theological, or other causation. Engineering causation focuses on whole systems and not individual elements. Since the entire system ceases to function with the loss of *any* vital element, no single element is declared to be causal. Engineering causes are distinguished by clarity, objectivity, and thoroughness. Only verifiable elements are included—and no vital element is omitted—in causal chains. Research informed by engineering principles searches for *all* system elements within an organism that *must exist* between its detection of environmental exposures and its conditioned self-adjustments.

We note increasing calls to reform or replace the current framework, yet "reformers" themselves have not integrated a replacement. One impression is that the field of evolutionary biology is somewhat in disarray and that practitioners are having difficulty framing the discussion regarding how to explain these new mechanisms. We see this as an opportunity to set the agenda. We suggest that many anomalous findings are explained within the CET framework.

CET is not simply a critique of the insufficiencies of adaptationism, random mutations, or selectionism. It is a new engineering-based, organism-focused model that flows from the latest findings from molecular biology—identifying sensing systems and logic mechanisms which direct suitable responses ranging from rapid physiologic changes to multi-generational modifications. Most importantly, it fundamentally changes the way we perceive organisms; from passive modeling clay shaped over time by the vicissitudes of nature, to active, problem-solving entities that continuously track changing environmental conditions to better fit existing environmental niches or fill new ones.

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Table 2. Multiple regulated mechanisms identified to produce rapid phenotypic variation. Variations are often predictable and a targeted solution to a specifically detected environmental challenge. The role a mechanism serves to enable an organism to continuously track environmental conditions is supported by an extraction from the reference article.

Mechanism	Action	Reference	Research Entity	Results	Descriptive Extract of Function in CET Framework
1. Regulated Tandem Repeat Sequence Number	Gradual changes in the repeated number of short DNA sequences, dubbed "units," arranged in head-to-tail (TR) sequences found within a promoter which yield gradual variations in gene expression.	Fondon and Garner. 2004. Molecular origins of rapid and continuous morphological evolution. *PNAS*	Comparative genomic study of repetitive elements in developmental genes in 92 breeds of dogs.	TR expansion and contraction contributes to incremental control of: transcription, mRNA processing, protein translation, folding, stability, aggregation rates, and gross morphology. Variations in the number of repeats in the coding regions of the *Alx-4* and *Runx-2* were quantitatively associated with significant differences in limb and skull morphology.	"The high frequency and incremental effects of repeat length mutations provide molecular explanations for swift, yet topologically conservative morphological evolution...revealing evidence supporting an alternative hypothesis that length variations in tandemly repeated sequences are a major source of morphological variation... that permit rapid generation of useful alleles...abundant in the coding sequences of vertebrate genes, especially those involved in development...repeat expansions or contractions vary in a locus-specific manner and occur at rates up to 100,000 times higher than point mutations...how broadly this mode of evolutionary change is exploited in nature remains to be seen, but if the prevalence of repetitive elements within genes is any indicator, then mammals, insects, plants, and other genomes throughout the natural world may use this mechanism to achieve evolutionary agility" pgs. 18058, 18062.
		Gemayel, et al. 2010. Variable Tandem Repeats Accelerate Evolution of Coding and Regulatory Sequences. *Annual Review of Genetics*	Review article	Properties or functions influenced by tandem repeats: overlap with regulatory protein binding sites, chromatin structure, Z-DNA formation, spacing of promoter elements, RNA structure.	"That these sequences are rapidly changing among primates suggests that this mutational hot spot may also be driving rapid evolution of *MMP3* gene expression and its associated phenotypes....tandem repeats provide a simple, monogenic mechanism that allows tuning of gene expression or function. The role of variable tandem repeats in mediating variable gene expression for quantitative phenotypic changes…whereas tandem repeat variation is by definition a genetic change, it also shares certain characteristics with epigenetic changes (e.g., the high instability and complete reversibility)" p. 468, 470.

Table 2 continued on next page.

Table 2 continued.

Mechanism	Action	Reference	Research Entity	Results	Descriptive Extract of Function in CET Framework
2. Mobile Genetic Elements	The regulated movement of defined segments of DNA carrying multiple genome formatting and coding sequences including molecularly symmetric DNA transposons characterized by terminal inverted repeats at each end; Long Interspersed Nucleotide Elements (LINEs), Short Interspersed Nucleotide Elements (SINEs); and Long Terminal Repeat retrotransposons .	Jiao, et al. 2017. Improved maize reference genome with single-molecule technologies. *Nature*	Improved de novo assembly and annotation of the maize reference genome.	Characterization of the repetitive portion of the genome revealed more than 130,000 intact transposable elements, allowing us to identify transposable element lineage expansions that are unique to maize. LINEs can be a mechanism of exon mobilization in protein-coding genes. LTRs only mobilize within the genome of a single cell.	Different transposon insertions confer drought tolerance, altered flowering time, ability to grow in toxic aluminum-rich soils, and have allowed maize to spread to temperate latitudes by breaking sensitivity to the long days of the tropics. And broadly, transposable element insertions have been shown to alter gene expression in stressful conditions.
		Shapiro. 2013. How life changes itself: The Read–Write (RW) genome. *Physics of Life Reviews*	Review article		"There is now an extensive literature on the great diversity of challenges and stress factors that activate genome instability...[which] include nutritional deprivation, intercellular signaling molecules, exposure to toxic substances… and life history events such as hybridizations and infections... coupling DNA restructuring to transcription is particularly important because there is no question that cells have the ability to target transcription to particular sites in the genome as part of a biologically adaptive response to external and internal circumstances" pgs. 303, 306.
		Casacuberta and González. 2013. The impact of transposable elements in environmental adaptation. *Mol. Ecol.*.	Review article		"Overall, the examples described previously strongly suggest a role of TEs in the ability of the host to respond to changes in the environment. The evidence that only some specific TE families, and not all the TEs in the genome, are activated in response to stress and the evidence that these TEs respond to some specific stress conditions and not others, strongly suggest that activation of TEs by stress is not only a byproduct of genome deregulation" p. 1513.

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Mechanism	Action	Reference	Research Entity	Results	Descriptive Extract of Function in CET Framework
3. Modulating Cis-regulatory Control of Genes	Modulation of cis-regulatory regions changes expression of gene products without changing genetic sequence.	Cleves et al. 2014. Evolved tooth gain in sticklebacks is associated with a cis-regulatory allele of Bmp6. *PNAS*	Threespine stickleback *Gasterosteus aculeatus*	Derived benthic freshwater stickleback population have about twofold gain in ventral pharyngeal tooth number and assays of *Bmp6* in developing teeth show that cis-regulatory changes have ~1.4-fold up-regulation of *Bmp6* relative expressio compared with their ancestral marine counterparts during late stages of stickleback development indicative of late-acting cis-regulatory up-regulation of *Bmp6* expression underlies an increase in tooth number.	"*BMP* family members have been implicated in several vertebrate evolved traits: size and shape of the beak in Darwin's finches, size and shape of the jaw in cichlids, jaw and skull variation in brachycephalic dogs, and avian feather patterning... this apparent reuse of the same signaling pathway across taxa may reflect a predisposition for *Bmp* genes to be used during morphological evolution, perhaps due to having complex, modular cis-regulatory architecture to generate evolutionary variation" p. 13916.
4. Regulated Signaling Pathways	Regulated control of amplification pathways that act in concert to mediate rapid, directional ribosomal DNA copy number change.	Carmen, et al. 2015. Regulation of ribosomal DNA amplification by the TOR pathway. *PNAS*	Multiple strains of budding yeast	"Here we show that signaling pathways that sense environmental nutrients control genome change at the ribosomal DNA. This demonstrates that not all genome changes occur at random and that cells possess specific mechanisms to optimize their genome in response to the environment" p. 9674.	"Our results reveal how a signaling pathway can orchestrate specific genome changes and demonstrate that the copy number of repetitive DNA can be altered to suit environmental conditions...through two pathways that are coordinately regulated [to] be tailored to suit the current environment...[which] departs from the standard model of adaptation through random mutation followed by selection... [and] raises the fascinating possibility that copy number of other regions of the genome may also be controllable in response to environmental conditions" pgs. 9676- 9678.
5. Directed Homologous Recombination	Homologous recombination is the exchange (crossing over) or replacement (gene conversion) of a DNA region by its homologous DNA sequence from the homologous chromosome or the sister chromatid during meiosis.	Shibata, et al. 2001. Homologous genetic recombination as an intrinsic dynamic property of a DNA structure induced by RecA/Rad51-family proteins: A possible advantage of DNA over RNA as genomic material. *PNAS*	Review article.	The induction of meiotic recombination depends on several genes regulated by a complex network of cellular signaling systems, as revealed by genetic studies in both yeasts.	"The ability to induce homologous recombination in response to unfavorable environmental changes would be adaptive for each species, as it would increase genetic diversity and would help to avoid species' extinction" p. 8430.
		Shibata. 2001. Functions of homologous DNA recombination. *RIKEN Review*. See also: Ohta. 2001. Hierarchic regulation of recombination, *RIKEN Review*	Review article.		"Homologous recombination is actively induced in bacteria and simple eukaryotes when cells are subjected to conditions unfavorable for their survival, such as nutritional starvation and a high cell density. While meiotic crossing over is supposed to create genetic diversity by producing new combinations of the alleles derived from parents and the genetic diversity may help cells to adapt to such unfavorable conditions..." p. 22

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Mechanism	Action	Reference	Research Entity	Results	Descriptive Extract of Function in CET Framework
6. Directed Mutation	Regulated genetic changes and histone acetylation directed to particular loci leading to the rapid emergence of adapted clones upon detection of particular environmental conditions.	Hull and Houseley. 2017. Environmental change drives accelerated adaptation through stimulated copy number variation. *PLoS Biol (*see also: Metzgar and Wills 2000. Evidence for the Adaptive Evolution of Mutation Rates. *Cell.* Chakrabati. 2008. Mutagenic evidence for the optimal control of evolutionary dynamics. *Physical Review Letters)*	Multiple strains of budding yeast	Yeast cells exposed to copper stimulate copy number amplification of the copper resistance gene *CUP1*. "Stimulated copy number variation (CNV) provides cells with a remarkable and unexpected ability to alter their own genome in response to the environment...CNV therefore represents an unanticipated and remarkably controllable pathway facilitating organismal adaptation to new environments."	"The assertion that adaptation occurs purely through natural selection of random mutations is deeply embedded in our understanding of evolution. However, we have demonstrated that a controllable mechanism exists in yeast for increasing the mutation rate in response to at least 1 environmental stimulus and that this mechanism shows remarkable allele selectivity ... evolutionary theory asserts that adaptive mutations, which improve cellular fitness in challenging environments, occur at random and cannot be controlled by the cell...evidence for adaptation through genome-wide nonrandom mutation is substantial...regarding the function of particular loci in particular environments that is encapsulated in existing gene regulatory systems" pgs. 16-20.
7. GC-Biased Mutational Heterogenous Gene Conversion	Nonreciprocal genetic exchanges of during meiosis elevate GC biased mutations in specific regions leading to gene conversion and massive stretches of "dark DNA."	Hargreaves, et al. 2017. Genome sequence of a diabetes-prone rodent reveals a mutation hotspot around the *ParaHox* gene cluster. *PNAS*	Insulin-regulating homeobox gene *Pdx1* of the sand rat *Psammomys obesus*	An unusual genomic region of biased mutation where 7 of the top 10 highest "protein deviation index" results correspond to genes located within the GC-biased mutational hotspot which contribute to coding sequence divergence across this region.	Extremely large diversification parameter, PDI, enable tracking of environmental changes diverging greatly from the norm. "Hotspots of mutation could drive rapid evolutionary change at the molecular level, and it will be important to decipher to what extent such hotspots have constrained and influenced evolutionary adaptation across the animal kingdom" p. 7680.

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Mechanism	Action	Reference	Research Entity	Results	Descriptive Extract of Function in CET Framework
8. Mutational "Hot Spots"	Bias of point mutations at CpG sites that can occur at a rate that is an order of magnitude higher than the average for all other nucleotide sites which produces function-altering alleles.	Galen, et al. 2015. Contribution of a mutational hot spot to hemoglobin adaptation in high-altitude Andean house wrens. *PNAS*	Andean house wrens, *Troglodytes aedon*	Analysis of house wren Hb highlights the influence of a 10-fold higher rate of mutation a CpG dinucleotide to any other affinity enhancing amino acid. The genetic basis of phenotypic divergence is demonstrated by a large-effect amino acid replacement that produced a significant increase in Hb–O2 affinity for high-altitude wren populations relative to lowland conspecifics.	This site-specific, and repeatable, variation in mutation rate may exert a strong influence on the genetic basis for fine-tuned adaptive traits suitable to fill a broad and continuous range of an environmental condition.
9. Repeatable Synonymous Mutation	Highly adaptable point mutations repeated at a specific genetic locus.	Agashe, et al. 2016. Large-effect beneficial synonymous mutations mediate rapid and parallel adaptation in a bacterium. *Molecular Biological Evolution*	Key enzyme-coding gene (*fae*) of *Methylo-bacterium extorquens* AM1	Synonymous variants of (*fae*) with decreased enzyme production rapidly regained activity in multiple experiments via parallel, yet variant-specific, highly beneficial genetic changes at single points within the gene.	The resuslts demonstrates that single, repeatable, and highly beneficial synonymous mutations can allow organisms to rapidly adapt to environmental changes. (See also: Caspermeyer. 2016. When Silent Mutations Provide Evolutionary Advantages. *Molecular Biology and Evolution*)
10. Amplified Micro-Satellite Mutation Rate	Heterozygous sites mutate faster than equivalent homozygous sites resulting in increased genetic diversity.	Amos. 2016. Heterozygosity increases microsatellite mutation rate. *Biology Letters*	1163 genome sequences from 1000 genomes utilizing the presence of rare alleles	Rare alleles were more likely to be found at locus-population combinations with higher heterozygosity . "Thus, as a population expands the resulting increase in heterozygosity will drive a further increase in microsatellite mutation rate."	The mechanism facilitates rapid increases in genetic diversity. It challenges the "classical population genetic theory based on the largely untested assumption that alleles mutate independently" (given that mutation rate increases as and population size heterozygosity increases) and calls into question mutation rates and timing of lineages splits and other historical factors based on mutation rates.

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Mechanism	Action	Reference	Research Entity	Results	Descriptive Extract of Function in CET Framework
11. Hyper-Mutability	Reactive oxygen species (ROS) and SOS mechanism within bacteria trigger a state of hypermutability which produces increased genetic variability.	Kohanski, et al. 2010. Sublethal Antibiotic Treatment Leads to Multidrug Resistance via Radical-Induced Mutagenesis. *Molecular Cell*	*E. coli, S. aureus* and an *E. coli* clinical isolate	Increasing ROS levels are sensed with internal mechanisms initiating higher mutation rates with up to an eightfold rise (in the case of norfloxacin.)	Bacteria use adversity as a stimulus to adapt to almost everything. Bacteria fill niches with increasing minimal inhibitory concentrations of antibiotics due to the increased probability of mutant strains possessing traits to overcome antibiotic challenges.
12. Regulated Transcriptome Plasticity by RNA Editing	Proteome diversity amplified by post-transcriptional mechanisms that dynamically modify RNA bases on the fly via a fine-tuning process that enriches genetic information beyond the genomic blueprint.	Porath, et al. 2017. A-to-I RNA Editing in the Earliest-Diverging *Eumetazoan* Phyla. *Molecular Biology and Evolution*	*Eumetazoan* Phyla corals	At over 500,000 sites in coral genes the sequence had been altered with RNA editing.	RNA editing levels increase during spawning and in newly released gametes. RNA editing patterns in corals resembled those found in mammals. RNA editing is known to be involved in the adaptation and function of the nervous system where lightning-quick responses are required.
		Liscovitch-Brauer, et al. 2017. Trade-off between Transcriptome Plasticity and Genome Evolution in Cephalopods. *Cell*	Diverse cephalopods		"Why would the coleoids choose to alter genetic information within RNA rather than hardwire the change in DNA? There are several potential advantages to making changes within RNA. First of all, the changes are transient. Thus, an organism can choose to turn them on or off, providing phenotypic flexibility, a quality that is particularly useful for environmental acclimation...RNA-level changes can better augment genetic diversity. With DNA, an organism is limited to two alleles. With RNA, all messages need not be edited, and thus the pool of mRNAs can include edited or unedited versions at given sites. When a message contains more than one site, complexity can increase exponentially" p. 200.

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Mechanism	Action	Reference	Research Entity	Results	Descriptive Extract of Function in CET Framework
13. RNAi - Regulated Epigenetic RNA inheritance	An active, tunable process to regulate the duration of epigenetically-controlled phenotypes in subsequent generations which comprises detection of specific conditions and modulation of the persistance or termination of epigenetic effects.	Houri-Ze'evi et al. 2016. A Tunable Mechanism Determines the Duration of the Transgenerational Small RNA Inheritance in *C. elegans. Cell*	*Caenorhabditis elegans* nematodes	Exposure to dsRNA activates a feedback loop whereby gene-specific RNAi responses dictate the transgenerational duration of RNAi elicited separately in previous generations. The effect was observed to last up to 14 generations.	"Perhaps, similarly to worms, organisms with longer generation times can regulate the duration of heritable effects, using homologous 'transgenerational timer' mechanisms...long-term transmission of epigenetic responses could be adaptive also in 'higher' organisms, for which the parental environment is often very different from that of the progeny. Adaptive control over the duration of environmental responses could affect the process of evolution" p. 97.
14. Regulated Short-lived Enzymatic Clusters	RNA polymerase II (Pol II) gathers for a few, but variably regulated, seconds in clusters on genes primed for transcription just prior to mRNA's appearance, then scatters apart. Cluster duration assists in regulating the quantity of mRNA product.	Cho, et al. 2016. RNA Polymerase II cluster dynamics predict mRNA output in living cells. *eLife*	RNA polymerase II	Manipulated enzyme clusters that stayed together for longer periods of time produced correspondingly more molecules of mRNA. Clusters of Pol II likely play a central role in triggering mRNA production and controlling gene transcription.	"We think these weak and transient clusters are a fundamental way for the cell to control gene expression. If a small mutation changes the cluster's lifetime ever so slightly, that can also change the gene expression in a major way. It seems to be a very sensitive knob that the cell can tune...to express a gene in response to some environmental stimuli." [MIT news on May 25, 2016]
15. Innate Heterozygosity	Heterozygous allelic variation with random additive genetic variance predominantly by SNPs.	Van Heerwaarden and Sgro. 2014. Is adaptation to climate change really constrained in niche specialists? *Proceedings of the Royal Society B*	Australian tropical fly, *Drosophila birchii*	In five generations, one species survived 23 per cent longer in only 35 per cent humidity due to innate genetic variation	Effectively track slow and steady environmental changes where "the expression of additive genetic variance for ecologically important traits will depend on the severity of the stress experienced."

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Mechanism	Action	Reference	Research Entity	Results	Descriptive Extract of Function in CET Framework
16. Hybridization:	Genetic mixing through sexual reproduction of two different varieties (breeds, species, genera) of organisms.	Seehausen, 2013. Conditions when hybridization might predispose populations for adaptive radiation. *Journal of Evolutionary Biology*	Review article	a) Adaptive introgression: a population acquires adaptive alleles though hybridization, b) Recombination links or unlinks genes which further promote adaptation and/or speciation. "Hybrid speciation has been quite well documented, and hybridization appears to be particularly common in the most species-rich and rapidly diversifying groups of organisms" p. 279.	1) rapid diversification of organisms to fill new niches 2) restored diversity enables population to backtrack or make a "U"-turn from genetic dead-end due to loss of genetic diversity "Through enrichment in standing genetic variation, hybridization can boost heritability in adaptive traits and increase realized rates of adaptive evolution. This mechanism is instantaneously effective. It may bring some genotypes in a hybrid population instantaneously into the attraction zone of an adaptive peak that neither parental population could tap into because of lack of suitable variation" p. 279.

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Mechanism	Action	Reference	Research Entity	Results	Descriptive Extract of Function in CET Framework
17. Unreduced Gametes	Unreduced gametes are now considered the primary mechanism for polyploidization following detection of specific stressful conditions.	Mason and Pires. 2015. Unreduced gametes: meiotic mishap or evolutionary mechanism? *Trends in Genetics*	Review article	Indications for unreduced gametes in speciation are: 1) they are often observed to be stimulated by stressful environments, 2) they are thought to be the primary mechanism for polyploidization, 3) their prevalence in diverse plant and animal species which occur via wide ranging molecular and cytogenetic causes across divergent lineages of life, 4) heritable genetic variation for production exists within and between species.	"Polyploidy is prevalent across eukaryotic life, particularly in the plant, animal, and fungal lineages. It has been suggested that the rationale for this prevalence of polyploids is that it provides novel genetic and genomic variation that can allow polyploid individuals to exploit new environmental niches and outcompete their diploid progenitors... we propose that unreduced gametes are maintained across widely disparate lineages because the ability to produce unreduced gametes facilitates lineage survival by allowing polyploid speciation, particularly in response to stress" pgs. 1, 5.
		Mable. 2013. Polyploids and hybrids in changing environments: winners or losers in the struggle for adaptation? *Heredity*			"Nevertheless, there has also been much emphasis on the alternative view that polyploidisation and hybridisation can promote diversification and speciation, by creating new combinations of genotypes that could increase the adaptive potential compared to the progenitor species..." p. 95.
18. Regulated Multiple Single Copy Gene Usage	Multiple usage of single copy genes by regulated "co-option" to take on new functions while continuing in their previous function.	Martinson et al. 2017. The Evolution of Venom by Co-option of Single-Copy Genes. *Current Biology*	Parasitic Jewel Wasp venom of *Urolepis rufipes, Trichomalopsis sarcophagae, Nasonia vitripennis*, and *N. giraulti*	Regulatory regions adjacent to venom genes initiates rapid turnover leading to more than half of the venom components coming from single copy genes that had been "co-opted" without being duplicated. There were both gains and losses in each species.	Venom composition rapidly changes allowing wasps to adapt to different hosts. Closely related species can differ by up to 40 percent of their venom repertoire. "In contrast, the mechanism of expression specialization by co-option of existing genes allows for a much faster mechanism for adaptation to novel or changing environments…[and could represent] a more general but underappreciated mechanism for rapid adaptation and gene neofunctionalization" p. 2010.

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Mechanism	Action	Reference	Research Entity	Results	Descriptive Extract of Function in CET Framework
19. Taxon-omically Restricted (Orphan) Genes	Every eukaryotic genome contains 10–20% of genes without any significant sequence similarity to genes of other species; these are classified as 'orphans' or 'taxonomically-restricted genes'.	Khalturin, et al. 2009. More than just orphans: are taxonomically-restricted genes important in evolution? *Trends in Genetics*	*Hydra magnipapillata*, *H. oligactis* and transgenic *H. vulgaris*	Most antimicrobial peptide genes show no sequence similarity to genes in other species with some showing rapid response to a wide variety of bacterial and tissue 'danger' signals leading to the creation of phylum-specific novelties, in the generation of morphological diversity, and in the innate defense system.	"We propose that taxon-specific genes, in combination with rewiring of the genetic networks of conserved regulatory genes, drive morphological specification and allow organisms to adapt to constantly changing ecological conditions" p. 404.
20. Klepto-genesis	Female of one species mates with three or more different species, then, through a yet unknown genetic mechanism, disassembles genomes from sperm and selectively recombines genes in roughly equal proportions into a single genome	McElroy, et al. 2017. Genome Expression Balance in a Triploid Trihybrid Vertebrate. *Genome Biology and Evolution*	Male: *Ambystoma laterale, A. texanum, and A. tigrinum;* Female: unisexual *Ambystoma.*	Unisexual *Ambystoma* individuals can possess up to five nuclear genomes derived from up to five phylogenetically diverse *Ambystoma* species. Genes are generally equally expressed.	Hybridized genome from multiple species increases genetic diversity and confers resilience to wide-ranging changes in environmental conditions through non-reliance on a single genome.
21. Adaptive Prediction	Adaptive prediction is a capability of diverse organisms, including microbes, to sense a cue and prepare in advance to deal with a future environmental challenge.	Amardeep, et al. 2017. Adaptive Prediction Emerges Over Short Evolutionary Time Scales. *Genome Biology and Evolution.*	Yeast, *Saccharomyces cerevisiae*	Yeast subjected to repetitive, coupled exposures to a neutral chemical cue (caffeine), followed by a sublethal dose of a toxin (5-FOA) internalized a novel environmental pattern within 50–150 generations by adaptively predicting 5-FOA stress upon sensing caffeine.	"...a novel structured environment can consistently generate AP in yeast within a remarkably short timeframe...to adaptively predicting 5-FOA toxicity upon sensing caffeine…[which] permits investigation into ecological implications of AP with regard to its role in enabling adaptation of an organism to new environmental condition" p. 1621.
22. Soma to Germline Feedback	Communication between Soma and germline cells in epigenetic inheritance that is coordinated by regulatory RNAs and specific hormones.	Steele and Lloyd. 2015. Soma-to-germline feedback is implied by the extreme polymorphism at IGHV relative to MHC. *Bio Essays*	Haplotype data on the polymorphism of the Major Histocompatibility Complex	Comparisons between the magnitude of Major Histocompatibility Complex polymorphism with estimates for the human heavy chain immunoglobulin V locus suggests IGHV could be many orders of magnitude more polymorphic than the MHC.	An under-investigated mechanism to transfer memory of environmental exposure from parent(s) to offspring through gametes since, "soma-to-germline feedback is forbidden under the neo-Darwinian paradigm" p. 557.

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Mechanism	Action	Reference	Research Entity	Results	Descriptive Extract of Function in CET Framework
		Sharma. 2013. Transgenerational epigenetic inheritance: Focus on soma to germline information transfer. Progress in Biophysics and Molecular Biology			"In germline-dependent mode, memory of environmental exposure in parental generation is transmitted through gametes, leading to appearance of phenotypes in the unexposed future generations...environmental exposure may cause epigenetic modifications in the germline either directly or indirectly through primarily affecting the soma. The latter possibility is most intriguing because it contradicts the established dogma that hereditary information flows only from germline to soma, not in reverse. As such, identification of the factor(s) mediating soma to germline information transfer in transgenerational epigenetic inheritance would be pathbreaking" p. 439.