GENETIC INFORMATION AND McCANN'S DUAL FACTOR PARADIGM FOR DEVELOPMENT AND VARIATION*

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Abstract

McCann (1991) has developed a model for embryogenesis and other aspects of differentiation that subordinates developmental genetics to a quality he defines as "cellular intelligence." We debate this paradigm in light of currently understood genetic principles and functions, information theory, and the Creationist concept of origins.

We contend that "cellular intelligence" is a non-entity, that cells are neither creative nor "intelligent" entities, but are informed entities. The dichotomy between intelligence and information is defined in terms of organizational hierarchy and antecedents.

Since information is not spontaneously generated, the information guiding development and differentiation is hereditary, being archetypical in origin and genomic in location. The ultimate source of that genetic information is Creative intelligence.

Cytodifferentiation is primarily a function of gene regulation and information transduction, according to a variety of tangible constitutive and inducible mechanisms and cellular components. The latter include homeotic genes, gene promoter, repressor, and enhancer elements, receptors, protein kinases, the cytoskeleton, and the dynamic three-dimensional tertiary structure of DNA itself.

The limitations McCann would place on "gene action" respective of phenotypic variation and development are untenable.

Intelligence and Information

We have read with interest the *CRSQ* article "Is More Than Gene Action Required to Account for Variation?" by Dr. Lester J. McCann (1991). Answering affirmatively, McCann posits that (p. 151) ". . . any aggregative construction requires an *intelligence* input" and concludes that "*Cellular* intelligence works in a copartnering arrangement with gene action" (emphasis ours). McCann's rationale for his "dual factor paradigm" is his view that genes alone play a decidedly limited role especially in embryogenesis and cytodifferentiation, requiring a superposable factor he identified as "cellular intelligence." We find his thesis seriously flawed.

Throughout his discourse on "cellular intelligence," McCann (1991) is first of all confusing *intelligence*— which he defines, we think aberrantly, as (p. 151) "the ability to select, control and direct energy"— with *information*— the quality which in biological systems limits the number of realized outcomes from the total number of putatively possible events (Lwoff, 1962). Following Lwoff (1962), information respective of probability and outcome would be expressed as follows:

If $I_0 = 0$ (when no information is available), $I_1 \neq 0$ (when information is gained), P_0 is the probability for possible outcomes, $P_1 = 1$ (when, as a result of specifying information, a single outcome is selected), then:

$$I = -k \ln P \quad \text{or} \quad \Delta I = -k \ln (P_1/P_0) = I_1 - I_0.$$

Distinguishing information from intelligence is not merely a matter of semantics. It is one of hierarchial organization. In the context of information theory and according to a Creationist model of origins, specifically where a developing organism would be concerned, intelligence would be the *source*, or *cause*, of the information guiding the system. Four additional, correlative principles apply:

(1) Following Schrodinger (1967) and Eccles (1979), neither awareness nor intelligence per se is an emergent property of matter.

(2) Information is neither spontaneously generated nor spontaneously increased (Yockey, 1981).

(3) In biological systems, the processing of information involves its encoding within and by macromolecules in a retrievable form. The archival codifer is DNA. Other informational molecules include the RNA's transcribed from DNA and proteins translated from RNA.

(4) Retrieval then becomes a function of gene regulation and information transduction by a variety of constitutive and inducible mechanisms. These are in themselves genetically specified.

Energy and Order

Where McCann's (1991) focus on energy would be concerned, the selective, incremental, and coupled utilization of energy in biological systems is prescribed by genetic information, as it is translated into enzymes, their cohorts, and their structural and spacial organization in the cell (for detailed examples, see Nakao and Packer, 1973, and Vogel et al., 1967). Energy then becomes a factor in biological ordering, according to the relationship (at equilibrium):

$$\Delta S \geq \Delta Q/T,$$

where ΔS is the change in entropy, ΔQ is the "heat"— or energy—flow into or out of a system, and T is the absolute temperature (see Thaxton et al., 1986).

The significance of entropy lies in its close reciprocal relationship to the degree of order (nonrandomness) characteristic of the system. Thus, following Thaxton et al. (1986):

$$S = k \ln \Omega,$$

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where Ω corresponds to the number of ways the energy and mass in a system may be arranged; k is Boltzman's constant. As a function of the statistical probability,

$$S = k \ln P.$$

We note the correspondence between entropy and information by comparing the two equations:

$$S = k \ln P \text{ and } I = -k \ln P.$$

In general, as the outcome becomes predictable by the informational component, or determinant (I_1), the greater the predictability, the lower the entropy. For biological systems only an exceedingly small, often singular, number of arrangements of any given set of components, as compared to the total number of possible arrangements, works.* At that point, organisms are, statistically, highly improbable events. Thus, the existence, development and maintenance of an organism depends on information. For the organism to be a repeatable event, the information guiding its development must be hereditary. Therefore, the ultralow entropy characteristic of embryogenesis and cytodifferentiation is a function of *genetic* information, as we review below. Accordingly, one can predict, with a high degree of confidence, the outcome, under normal conditions, of the development of, e.g., a fertilized frog egg, including the fate of one of its embryo's myoblasts (it will become a muscle cell), the position of its micro- and macroanatomical elements, and so on. Meanwhile, energy, per se, because of genetically informed, hence determined, pathways of metabolism, is being channeled and coupled to predictable events of biosynthesis, etc. While the availability of energy alone will satisfy the thermodynamic equations for endergonic processes, it is insufficient, absent informationally directed transduction, to reduce entropy or maintain a low entropy state respective of biological systems. Indeed, under such circumstances, energy alone becomes a potentially disruptive factor (see Morris, 1984, pp. 207-208). Energy per se does not generate the information on which biological systems are predicated, though energy is, of course, required mechanistically for the synthesis of informational molecules (nucleic acids, proteins, etc.) whose structures (ergo information content) are templated by *pre-existing* information.

Thus, ". . . the ability [of cells] to select, control, and direct energy" (McCann, 1991, p. 151), where that becomes a factor in development, is a manifestation of information which has its origin in Creative intelligence, i.e., that which brought the system into being in the first place. Kaufmann (1991, p. 66) has remarked that "The essence of intelligence is . . . the ability to select and direct processes." However, the context here is that of causation, and the "Logos" to which Kaufmann refers is correctly attributed to the Divine Designer. McCann (1991) would otherwise attribute to cells a remarkable degree of agency. In reality, cells themselves are neither creative nor "intelligent" entities, but are *informed* entities. Where their "selective and directive abilities" are concerned, cells can only execute the options that are already programmed, unless one evokes the evolutionist's hypothetical "progressive (adaptive) mutation." This conjecture has been largely defeated, however, by the mathematical algorithms of Schutzenberger (1967), Yockey (1981), and others. The quandary of "evolutionary genetics" otherwise has been cogently reviewed by Ouweneel (1977).

*Editor's Note: See Williams, E. L. 1971. Resistance of living systems to the second law of thermodynamics. *CRSQ* 8:123 or Williams, E. L. 1981. Resistance of living organisms to the second law of thermodynamics in Williams, E. L. Editor. Thermodynamics and the development of order. Creation Research Society Books. Kansas City, MO, p. 104 for a discussion of the small number of microstates possible for living systems.

Genetic Information in Development

McCann (1991, p. 152) contends that ". . . cellular involvement during embryonic development and other vital processes give evidence of being governed by non-genetic influences . . ." which McCann attributes to "cellular intelligence," However, the particular events of embryonic development McCann (1991) reviews are demonstrably governed by genetic information. Note that genetic mechanisms would include those involved in replication of the archival code, those governing its transcription, translation, and *the functions of gene products.* The latter, notably those functioning as receptors and gene regulators, close the apparent gap between cytoplasmic factors, environmental influences, and the role of structural genes per se in growth and development, when it is realized that these epigenetic/environmental principles are operating within a molecular-level cybernetic framework set by direct gene control. Their effects are not autonomous from the genome, but are genetically prescribed. For a particularly cogent discussion of nonhereditary variation and its relationship to genomic processes, see Marsh (1991). Reviews of various cytoplasmic phenomena impinging on development are found in Malacinski (1990). See also Berridge (1985) regarding cell surface receptors, their translation of external signals into internal signals, and some of the consequent molecular aspects of intracellular information flow.

Despite McCann's (1991) inferences to the contrary, by the mid-1960's it was already clear that gene action is continuous throughout embryogenesis (see review by Gross, 1967), and essential to normal development and morphogenesis. While McCann (1991) would reference the observation (Barth, 1964) that experimentally enucleated amphibian eggs (merogones) can, under certain circumstances, be stimulated to cleave and carry out a limited kind of early development, this phenomenon is readily explainable (Gross, 1967) by the presence in these merogones of genetically derived (transcribed) morphogenic RNA, released into the cytoplasm prior to removal of the nucleus (or, as in other experiments, blockage of transcription by actinomycin D). These gene products are conserved and utilized as messengers for the direction of protein synthesis, in particular of proteins involved with cleavage. However, such "embryos ex merogones," on reaching a stage morphologically comparable to a blastula, cease development and eventually disintegrate. In any event, what the data, collectively, indicate is a direct role of gene action in early development, not a lack of it. As Gross (1967) emphasizes (see also Stein, et al., 1991), genes, some being transcribed very early on, act directly and indispensibly throughout the course of normal development.

A Transition in Developmental Biology

Analagous to First Law-accommodated processing of energy within cells, the principle of transduction applies to their processing and utilization of information. Among the information transducers to which we refer, whose structure ergo function is directly determined by genes, are (besides m-, t-, and r- RNA's and enzymes, per se) DNA binding proteins, receptors, and the myriad elements of the cytoskeleton, all of which play decisive roles in cellular development and differentiation.

It is lamentable that the literature sources McCann (1991) draws upon in developing his thesis are, for the most part, limited to texts published no more recently than the 1960's. This period, it may be recalled, was one of transition for developmental biology, where traditional embryology was only beginning to be integrated with molecular biology and interpreted accordingly. For a particularly incisive discussion of the classic "boundary dispute" between geneticists and embryologists, see Gilbert (1991). Many questions posed then concerning the role of genes in development are no longer enigmas; but see Malacinski (1990) for some possible, at least arguable, current exceptions to the ubiquity of genotype in *all* aspects of development. In any event, the limitations McCann himself (1991) identifies for gene action and the products thereof are specious. While McCann (1991) finds some support from Dubois (1962) for his idea that embryonic development is governed to a substantial degree by nongenetic functions, Dubois' remarks about the "modest tasks" of genes (quoted by McCann, 1991, p. 152) must be considered in the context of the "modest" state of knowledge respective of genes and development at that time—1962! Indeed, there has been considerable progress during the last 20 years toward an understanding of the genetic basis and intermolecular linkages for the developmental processes McCann (1991) discusses. We doubt that Dubois would disagree.

For example, while McCann (1991, p. 152) cites Alston (1967, pp. 189-190) as finding ". . . no explanation of the regulation of cell movement [respective of embryonic morphogenesis] . . . by *presently* known intracellular mechanisms of information transfer" (our emphasis), we daresay that neither Alston nor any other knowlegeable developmental biologist would make that statement today (e.g., for reviews of the protein fibronectin, a gene product, its role in determining embryonic cell migrations, and how it informs the mechanics of the cytoskeleton respective of directed movements, see Alberts et al., 1989, Hynes, 1981, and Ruoslahti, 1988). Where McCann (1991) can find no genetic basis for what he calls (p. 151) the ". . . vital crafting process . . . in which cells . . . position themselves . . . in the process of which embryonic details are fashioned" (positional information?) we reference Stein et al. (1991), who discuss the genetics involved in establishing axial polarity (anterior/posterior, dorsal/ventral) in *Drosophila* embryos. Attributing the events McCann (1991) discusses to "nongenetic influences" requires disregard or ignorance of the clearly defined genetic principles discovered during the last two decades, principles that apply to embryonic cell movements and every other one of the developmental phenomena McCann (1991) cites. These would include the now well known existence and functions of, for example, homeotic genes, developmental control regulators and receptors, cell surface recognition and adhesion principles, etc., topics reviewed by Alberts et al. (1989), Berridge (1985), DeRobertis et al. (1990) Edelman (1984, 1989), Gehring (1985), Holliday (1989), McKnight (1991), Ouweneel (1975, 1977), Ptashne (1989), Rutishauser and Goridis (1986), Stein et al. (1991), and Takeichi (1988), among others.

Gene Activity

In his statement (McCann, 1991, p. 152) ". . . we have to assume that the genes in terms of function are in the protein-supply business *exclusively"* (emphasis ours), McCann overlooks the *nucleic acid* products of certain genes that do not encode peptide structure but directly regulate gene expression—we reference as one example antisense RNA molecules (Weintraub, 1990); these bind to complementarily structured mRNA molecules and thereby selectively *inhibit* translation. In the remarkable statement that follows—(p. 152) "(genes) are not involved in initiating constructions"—McCann (1991) would apparently discount the role of catalytic and regulatory proteins (gene products) in development, or fail to see the genetic linkage between such proteins and the process, and limit "genes" to but one category—structural genes—according to the classic, but now inadequate, "one gene, one peptide" notion of gene function. However, it is now known that such genes contain, besides the protein encoding element, regulatory elements, the latter controlling transcription of the encoding sequences (McKnight, 1991), ergo initiating constructions. Moreover, in addition to structural genes, there are also regulatory genes per se that exercise control over whole repertoires of other gene sequences (Beardsley, 1991) activating or repressing them as appropriate—again, initiating constructions! While it is obvious how activating a gene would initiate construction, there are cases as well where "turning off" a gene does likewise. An example would be the production (genetically prescribed) of a juvenile hormone that arrests silkworm metamorphosis (Williams, 1963). When juvenile hormone production is stopped (a genetically prescribed event), there is de-repression of genetic information for construction of the pupa and its transformation to the adult moth.

Paradoxically, the "totipotency" and "fate flexibility" (our characterizations) of early stage embryonic cells, to which McCann (1991) alludes (his references, p. 152, to those "experimentally scattered," those "faced with an unchartable problem," "tailored-to-the-situation responses," etc.), rest on the fact that in these cells the genes responsible for specialized "fate determining" proteins are repressed. Thus, for these "totipotent" (or at least pluripotent) cells, the greatest amount of genetic information is latent. What is expressed, predominantly, in these cells, are the genes from which products dedicated to cellular replication are derived. Later, as specific cell lines and cell types are differentiated, the genetics for replication tend to be downregulated as genes for the line characteristics are expressed. Thus, replication of "committed" neurons, hematocytes, muscle, connective tissue, and special-

ized epithelial elements, etc. is atypical, except where a population of "stem cells," such as found in bone marrow, the stratum germinativum of the epidermis, crypt cells of the intestinal mucosa, etc., is maintained. The genetic principle manifested in organogenesis is emphasized by the reverse process of regeneration, which involves "de-differentiation" of the "adult" cells, i.e., a genetically triggered reversion to the multiplicative "embryonic" type (Hay, 1966). A similar phenomenon is tumorigenesis.

Only a fraction of the amount of DNA in plant and animal cells is ever required, in terms of essential gene products, for building the organism (see Alberts et al., 1989, pp. 485-486). This "extra" quantity of DNA is not accounted for just by polyploidy or gene duplication otherwise. Some consider much of it the "junk" that might be expected from the stochastic process of evolution (see specific references below). However, we note that the informational content of DNA includes more than its encoding sequences (for RNA's and proteins) per se. There is a nucleotide sequence-specified, tertiary structural quality that affects recognition and reactivity respective of various mechanisms for gene regulation, significant of which are those of an allosteric nature (Felsenfeld, 1985). This, we believe, is the primary significance of the large amount of DNA present in a given organism's genome that is not transcribed/translated into gene products. Allegations to the contrary notwithstanding (Orgel and Crick, 1980; and see discussion by Augros and Stanciu, 1987, pp. 180-182, and Britten and Davidson, 1971), we do not believe that this DNA is merely an evolutionary vestige. While some of it is represented by pseudogenes (see Alberts et al., 1989, p. 602)—putatively genes rendered inoperable by mutation—the greatest amount is neither superfluous nor useless otherwise, but functional, we believe (and see Felsenfeld, 1985) to the dynamic tertiary structure of the genome, which in turn is significant to its genetic regulation. Indeed, even "pseudogenes" may have functions of their own, if distinct from those of their sibling counterpart, or "conventional" structural genes (McCarrey and Riggs, 1986). Then there is the phenomenon of introns—noncoding intragene nucleotide sequences—which may underlie a mechanism for diversifying the protein products of a single gene (Anderson, 1991; Andreadis et al., 1987). McCann (1991) does not address this subject (of non-encoding DNA), but it is germane to how DNA (ergo the genome) is involved in "initiating constructions."

Proteins and Developmental Regulation

Even where structural genes per se are concerned, McCann (1991) grievously restricts the initiative role of proteins in development by stating (p. 152):

> . . . there would seem to be no way the mere availability of inanimate, gene-produced proteins can assure the carefully timed furnishing of the proper kinds of directed energy and thereby bring about a specific energy-demanding process of assembly.

But the protein kinases (Rosen and Krebs, 1981) do just that; these enzymes (ergo "gene produced proteins") employ ATP (directed energy?) to phosphorylate other proteins and thereby regulate their biological activity. Among the roles of protein kinases, and the protein phosphatases which catalyze the reverse reaction, in development and differentiation are the assembly/disassembly of cytoskeletal ultrastructure (Hunter, 1984; Murphy et al., 1983; Rappaport et al., 1975; Rosen and Krebs, 1981), timing and execution of the cell cycle (Murray and Kirschner, 1991), epidermal growth factor-mediated cell proliferation (Hunter, 1984) and, respective of pathobiological development, oncogene-inflicted neoplasia (Hunter, 1984). For an appreciation of the myriad functions otherwise of protein kinases in metabolism, biosynthesis, regulation of gene expression, etc., see Rosen and Krebs (1981). These enzymes are themselves regulated through receptor (protein)-mediated fluxes of "second messengers" (cyclic nucleotides, Ca^{++}) acting on regulatory proteins (e.g., the inhibitory subunit of the CAMP-dependent protein kinases, the calmodulin group of proteins respective of the Ca^{++} dependent protein kinases) and the cyclin group of proteins (Berridge, 1985; Murray and Kirschner, 1991; Marx, 1991; Rosen and Krebs, 1981).

Then there is the phenomenon of post-translational enzymatically mediated modification of nascent peptide structure that includes (additional to the aforementioned phosphorylation of proteins) selective proteolysis, hydroxylation and glycosylation, respective of activation, self-assembly mechanisms and the like. As an example, we cite the steps involved in the construction of collagen fibers (Burgeson, 1988; Olsen, 1981). These biochemical processing functions further evidence the significance of protein availability to processes of macro-assembly and have a genetic basis in that gene products (proteins) are acting on or being acted upon by other gene products (proteins), events that are directed, energetic, and temporal.

Moreover, gene expression itself (i.e., transcription and all that follows therefrom) is controlled to a great extent by proteins that bind to DNA as gene activators/repressors (McKnight, 1991; Ptashne, 1989), or enzymatically modify DNA structure/gene activity per the DNA/cytosine methylases/demethylases (Holliday, 1989) ergo contributing to the ". . . timed (etc.) . . . process of assembly" (McCann, 1991, p. 152).

Environmental Influences

McCann's (1991) reference to the seasonal, adaptively significant dimorphism of *Nemoria arizonaria* caterpillars is misapplied to his thesis of non-genetic direction of development, when it is considered that the features described (Green, 1989) can be accounted for by, albeit environmentally-impacted, selective events of genomic translation. The relative concentration of tannin in the diet of the spring vis-a-vis summer caterpillars appears to be the environmental trigger to phenotypes that remarkably mimic the oak catkins or twigs, respectively, on which they crawl; the indicated mechanism is that receptors responsive to tannin levels influence the levels of circulating hormones which in turn mediate genetic regulation during development (Green, 1989). This then is but another example (see Marsh, 1991) of the interplay between environment and genotype underlying phenotypic variation and the bidirectional circuitry of information transduction.

The *Nemoria* phenomenon, and others McCann (1991) points to—the differentiation of queen and

worker bees, and insect metamorphosis generally—involve genetic regulatory mechanisms that Jacob and Monod (1961) originally elucidated for prokaryotes, later applied to eukaryotes (Jacob and Monod, 1963; and see review by Ptashne, 1989), to wit: regulatory protein-mediated selective expression of genes promulgates the phenotypic response triggered by differential feeding, or diet, humidity, light, temperature, etc. This includes, for example, the elaboration of "adaptive" enzymes and morphogenic peptide structures. That gene regulatory mechanisms in response to exogenous as well as endogenous mediators (Williams, 1963) may differ in their details for eukaryotes (vs. bacteria) does not diminish the significance of such regulation and its locus for development and differentiation generally, and it is now clear that a variety of inductive mechanisms, acting at the gene level, are operable in the development and differentiation of multicellular organisms. Otherwise, noting that the dimorphism exhibited by *Nemoria* caterpillars in their natural environment is consistent generation after generation, McCann's (1991) interpretation of the phenomenon as gene-independent strikes us as unsettlingly Lamarckian. As noted by Marsh (1991, p. 54), "The phenotype . . . of any organism is necessarily the result of the interaction of a genotype . . . with an environment . . ."

Genetic Programming of Phenotype

Where insect development in general and McCann's (1991) references to it in particular be concerned, it was already clear by the 1960's (Williams, 1963) that the programming of cells respective of metamorphic differentiation has to do with sequential gene action—as Williams (1963, p. 258) stated: ". . . the 'taping' of the individual cells for a subsequent 'playback' . . . a process that . . . predicts with utmost precision the chemical engineering of larva, pupa and adult." Further knowledge of the genetic determination of insect embryogenesis was the discovery of homeotic genes (reviewed by Gehring, 1985, and Ouweneel, 1975), which also play the decisive role in "masterminding" pattern formation for many other kinds of animals (Gehring, 1985; DeRobertis et al., 1990), as well as fungi and plants (Rennie, 1991). As important as homeotic genes are in development, however, we note parenthetically that an argument for their role in evolution, after Goldschmidt (1952), has been defeated in the insightful review of Ouweneel (1975).

McCann (1991) finds it enigmatic that the vascular elements of plants (xylem and phloem) are sorted out from a population of genetically identical cells (cambium). But it has been known for some time how this and plant histogenesis generally is accomplished—through differential expression of genes in the common genome, involving in part selective responses to a variety of growth factors (reviewed by Clowes and Juniper, 1968). Indeed, McClintock (1956; 1961), by identifying the principle of genetic regulatory circuitry in plants (maize), brilliantly anticipated the model independently derived for microorganisms in the Jacob and Monod (1961) review.

Therefore, as early as 1963, Grobstein could remark that (p. 3) ". . . the dilemma of differentiation, . . . *man-made rather than cell-made,* has never been as sharp or as fearsome as sometimes . . . portrayed" (emphasis ours). Today, it is not at all bemusing that while the somatic cells of an organism contain the same genome that this genome is differentially expressed among these cells at the same as well as different stages of development; that is, identical genotypes can generate different phenotypes. This is precisely what genetic control mechanisms and their bearing on cytodifferentiation are all about; and genes specify these control elements.

According to McCann (1991, p. 151), ". . . cells by their own effectiveness position themselves in strategic patterns in the process of which embryonic details are fashioned." He subsequently refers (p. 152) to a "painstaking self-positioning on the part of the embryo cells" and (p. 152) "Purposeful deftness on the part of embryo cells." McCann's anthropomorphic inference is that these cells are somehow electively managing their own affairs apart from the otherwise integrated genetic determinants of the process for embryonic pattern formation—but see, e.g., DeRobertis et al. (1990), Edelman (1984, 1989), Rutishauser and Goridis (1986), Stein et al. (1991), and Takeichi (1988) for evidence to the contrary. At this point, McCann (1991) errs glaringly in viewing the development of a cell (zygote) and its progeny into an anatomically definitive organism as an isolated event of agency. In particular, the ability of cells to respond to extracellular signals respective of embryonic processes, the genome to cytoplasmic feedback, clearly has a genetically informed basis. This, in part, is what proteins (gene products) serving as receptors (information transducers) and, frequently, informational ligands, are all about (Berridge, 1985; Edelman, 1989; Stein et al., 1991). It is to be emphasized that while the outcome of these functions is selective, it is by no means elective.

Intelligent Origin

In our opinion, a model for development and differentiation that requires the presence and active participation of unique yet arcane forces not explainable in terms of developmental genetics is today no more productive, at least scientifically, than the archaic concept of vitalism. While many details for any given system remain to be elucidated, the operative principles are no longer enigmatic. Genes beget phenes. This is not to reduce the phenomenon of embryogenesis to a wholly materialistic or mechanistic conception. Genetically scripted embryogenesis is a marvelous example of pre-existing design, purpose and specification, wholly consistent with a creationist model. McCann's (1991) paradigm is not only inaccurately derived, it is superfluous to the anti-Evolution, pro-Creation argument.

We would agree with the thrust of McCann's (1991) observation that stasis, vs. evolutionary change, is favored (though not necessarily compelled) by the fact that random changes in genetic information tend to be unproductive (as for "neutral" mutations) or counterproductive (as for lethal or debilitating mutations). It is not, however, a perturbation of an "*intelligence* - centered" parameter (McCann, 1991, p. 153, our emphasis) that would be required to bring about change, either constructive or destructive. *Cellular* intelligence, in any event, is a non-entity. Organic intelligence is an *organismic* parameter, and one, cogently

argued by Augros and Stanciu (1987), limited, according to its best definition, to human organisms. [Augros and Stanciu (1987, Chap. 3) differentiate other aspects of behavior and consciousness from intelligence, per se; see also Morris, 1984, p. 369 and pp. 405-413].

What is reflected at the cellular level of the developmental differentiative process is a hierarchy of information, the ultimate source of which is an *external* intelligence, the same that prescribed the kinds and properties of life in the first place. It is, in fact, a "living intelligence" (see McCann, 1991, p. 153), but one we would identify, first and last, in the context of Revelation 1:8. McCann would unnecessarily place the burden of intelligence on the cell, where we (and see Kaufmann, 1991) would attribute it otherwise to the Creator and His formative endeavors—specifically in designing and placing information and the mechanisms for information transfer and reception within the cells of the organisms He made. Having at that point been assembled and energized in the archetypes, the process is now sustained through conservation (Morris, 1984, pp. 89-92). Now that the cellular *information* is in place, represented by the totality of an organism's genome, and transmitted from one generation to the next by reproduction of kind, development becomes a matter of executing a sequence of commands through a multifaceted network of information transducing pathways, which ultimately direct and determine the appropriate cellular responses. Cells are not in the business of autonomously making decisions concerning what they should be when they grow to maturity! Indeed, to paraphrase Lwoff (1962) when there is a capricious departure from the preordained order, the observed result is pathology.

We respectfully disagree with McCann (1991, p. 153) that "*there remains the question* of how the modality for the intelligent governance of living systems could have been acquired" (our emphasis). There is no question. We reference Genesis 1:1-31, and Exodus 20:11.

Conclusion

Variation of phenotype, which includes the morphogenic differentiation of cells during embryonic development, remains altogether explicable in terms of genetic information, as that is viewed in its *totality,* and information transduction. It is the latter principle, in particular, that integrates the genomic "code" per se with extragenomic influences, where applicable, on development. The genome is both initiative and reactive. The limitations McCann (1991) would place on "gene action" respective of differentiation and phenotypic variation are specious. We note that "gene action" would include the "action" of gene products; as their structures are prescribed by genetic information, so, ultimately, are their functions and interrelationships. The superintendent quality of "cellular intelligence" he proposes is neither demonstrable, necessary, nor justified as an explanation of cytodifferentiation and the developmental process. While McCann (1991, p. 151) would decry the genetic explanation as "well-worn orthodoxy," it remains well-developed and based on empirical evaluation.

Since information is not generated spontaneously, the source of genetic information — implanted in the archetypes at the time of their fiat creation and now maintained through conservation and heredity — is Creative intelligence. Cells per se are not "intelligent" entities but are *informed* entities, marvelously made in the process.

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References

CRSQ—Creation Research Society Quarterly.

Alberts, B., D. Bray, J. Lewis, M. Raff, K. Roberts, and J. Watson. 1989. Molecular biology of the cell. Garland. New York.

Alston, R. 1967. Cellular continuity and development. Scott, Foresman. Glenview, IL.

Anderson, K. 1991. New trends in the molecular basis for variation. *CRSQ* 28:50-51.

Andreadis, A., M. Gallego, and B. Nadal-Ginard. 1987. Generation of protein isoform diversity by alternative splicing: mechanistic and biological implications. *Annual Review of Cell Biology* 3:207-242.

Augros, R. and G. Stanciu. 1987. The new biology. Shambhala. Boston, MA.

Barth, L. 1964. Development-selected types. Addison-Wesley. Reading, MA.

Beardsley, T. 1991. Smart genes. *Scientific American* 265(2):87-95.

Berridge, M. 1985. The molecular basis of communication within the cell. *Scientific American* 253(4):142-151.

Britten, R. and E. Davidson. 1971. Repetitive and nonrepetitive DNA sequences and a speculation on the origins of evolutionary origins. *Quarterly Review of Biology* 46(2):111-133.

Burgeson, R. 1988. New collagens, new concepts. *Annual Review of Cell Biology* 4:551-577.

Clowes, F. and B. Juniper. 1968. Plant cells. Blackwell. Oxford, England.

DeRobertis, E., G. Oliver, and C. Wright. 1990. Homeobox genes and the vertebrate body plan. *Scientific American* 263(1):46-52.

Dubois, R. 1962. The torch of life. Simon and Schuster. New York.

Eccles, J. 1970. Facing reality. Springer-Verlag. New York.

Edelman, G. 1984. Cell-adhesion molecules: a molecular basis for animal form. *Scientific American* 250(4):118-129.

_____ 1989. Topobiology. *Scientific American* 260(5):76-88.

Felsenfeld, G. 1985. DNA. *Scientific American* 253(4);68-87.

Gilbert, S. 1991. Cytoplasmic action in development. *Quarterly Review* of *Biology* 66(3):309-316.

Gehring, W. 1985. The molecular basis of development. *Scientific American* 253(4);152B-163.

Goldschmidt, R. 1952. Homeotic mutants and evolution. *Acta Biotheoretica* 10(1):87-104.

Green, E. 1989. A diet-induced develonmental polymorphism in a caterpillar. *Science* 243(4891):643-646.

Grobstein, C. 1963. Cytodifferentiation and macromolecular synthesis. In: Locke, M. (editor). Cytodifferentiation and macromolecular synthesis. Academic Press. New York. pp. 1-14.

Gross, J. 1967. RNA metabolism in embryogenesis. In: Malt, R. (editor). Macromolecular synthesis and growth. Little, Brown. Boston. pp. 185-235.

Hay, E. 1966. Regeneration. Holt, Rinehart, Winston. New York.

Holliday, R. 1989. A different kind of inheritance. *Scientific American* 260(6):60-73.

Hunter, T. 1984. The proteins of oncogenes. *Scientific American* 251(2):70-79.

Hynes, R. 1981. Structural relationships between fibronectin and cytoskeletal networks. In: Poste, G. and G. Nicolson, (editors). Cytoskeletal elements and plasma membrane organization. Elsevier. Amsterdam. pp. 100-137.

Jacob, F. and J. Monod. 1961. Genetic regulatory mechanisms in the synthesis of proteins. *Journal of Molecular Biology* 3(1):318-356.

_____. 1963. Genetic repressons, allosteric inhibition and cellular differentiation. In: Locke, M. (editor). Cytodifferentiation and macromolecular synthesis. Academic Press. New York. pp. 30-64.

Kaufmann, D. 1991. Functional proteins: chaos or logos. *CRSQ* 28: 64-66.

Lwoff, A. 1962. Biological order. M.I.T. Press. Cambridge, MA.

Malacinski, G. (editor). 1999. Cytoplasmic organization systems. McGraw-Hill. New York.
Marsh, F. 1991. Biological variation. *CRSQ* 28:54-59.
Marx. J. 1991. The cell cycle: spinning farther afield. *Science* 252: 1490-1492.
McCann, L. 1991. Is more than gene action required to account for variation? *CRSQ* 27:151-153.
McCarrey, J. and A. Riggs. 1986. Determinator-inhibitor pairs as a mechanism for threshold setting in development: a possible function for pseudogenes. *Proceeding* of *the National Academy of Sciences USA* 83:679-683.
McClintock, B. 1956. Controlling elements and the gene. *Cold Spring Harbor Symposia on Quantitative Biology* 21:197-216.
_____ 1961. Some parallels between control systems in maize and in bacteria. *American Naturalist* 95:265-277.
McKnight, S. 1989. Molecular zippers in gene regulation. *Scientific American* 264(4):54-64.
Morris, H. 1984. The biblical basis for modern science. Baker. Grand Rapids, MI.
Murphy, R., M. Askey, P. Dillon, W. Gerthoffer, and K. Kamm. 1983. The role of myosin light chain phosphorylation in regulation of the cross-bridge cycle. *Federation Proceedings* 42(1):51-56.
Murray, A. and M. Kirschner. 1991. What controls the cell cycle. *Scientific American* 264(3):56-63.
Nakao, M. and L. Packer (editors). 1973. Organization of energy-transducing membranes.. University Park Press. Baltimore, MD.
Olsen, B. 1981. Collagen biosynthesis. In: Hay, E. (editor). Cell biology of extracellular matrix. Plenum. New York. pp. 139-177.
Orgel, L. and F. Crick. 1980. Selfish DNA: the ultimate parasite. *Nature* 284:604-607.
Ouweneel, W. 1975. Homeotic mutants and evolution. *CRSQ* 12: 141-154.
_____. 1977. Genetics and creation studies. *CRSQ* 14:26-34.
Ptashne, M. 1989. How gene activators work. *Scientific American* 260(1):41-47.
Rappaport, L., J. Leterrier, and J. Nunez. 1975. Protein-kinase activity and in vitro phosphorylation and polymerization of purified tubulin. *Annals* of *the New York Academy of Sciences* 253:611-629.
Rennie, J. 1991. Homeobox harvest. *Scientific American* 264(6):24.
Rosen, O. and E. Krebs (editors). 1981. Protein phosphorylation. CSH. Cold Spring Harbor, NY.
Ruoslahti, E. 1988. Fibronectin and its receptors. *Annual Review of Biochemistry* 57:375-414.
Rutishauser, V. and C. Goridis. 1986. N-CAM: the molecule and its genetics. *Trends in Genetics* 2(1):72-76.
Schrodinger, E. 1967. What is life? Cambridge University Press. Cambridge, U.K.
Schutzenberger, M. 1967. Algorithms and the neo-Darwinian theory of evolution. In: Moorhead, P. and M. Kaplan (editors). Mathematical challenges to the neo-Darwinian interpretation of evolution. The Wistar Institute Symposium Monograph No. 5. Wistar Institute Press. Philadelphia. pp. 73-80; 121.
Stein, D., S. Roth, E. Vogelsang, and Nüsslein-Volhard. 1991. The polarity of the dorsoventral axis in the *Drosophila* embryo is defined by an extracellular signal. *Cell* 65:725-735.
Takeichi, M. 1988. The cadherins: cell-cell adhesion molecules controlling animal morphogenesis. *Development* 102:639-655.
Thaxton, C., W. Bradley, and R. Olsen. 1986. The mystery of life's origin. Philosophical Library. New York.
Vogel, H., J. Lampen, and V. Bryson (editors). 1967. Organizational biosynthesis. Academic Press. New York.
Weintraub, H. 1999. Antisense RNA and DNA. *Scientific American* 262(1):40-46.
Williams, C. 1963. Differentiation and morphogenesis in insects. In: Allen, J. (editor). The nature of biological diversity. McGraw-Hill. New York. pp. 243-266.
Yockey, H. 1981. Self-organization origin of life scenarios and information theory. *Journal of Theoretical Biology* 91(1):13-31.

DR. McCANN'S RESPONSE

Beginning in a very broad way, evidence of living systems performing meaningful work is apparent in the many constructions we see around us. Not all of these are of human origin. Non-human products include such items as bird nests, snail shells, spider webbings, and the larval encasements of caddis flies.

No kinds of aggregative constructions are found on those celestial bodies that we have inspected thus far. It indicates that any work-demanding assembly process is always associated with the presence of life. This observation correlates with the ability of living systems to control and direct energy, without which constructions are impossible.

However, we have a troublesome problem here relative to living systems themselves. Lumsden, Anders and Pattera, and mechanists generally, contend that living systems in their assembly patterns are different. These authors believe that living systems are put together by a non-living source, the genes. This is where Lumsden et al. and McCann most sharply differ. McCann maintains that the construction of any non-living assembly and any living system itself *both* require the involvement of *living* agencies having the ability to beget *work* of a constructive nature.

If genes could qualify as such an agency it would be fine. However, genes give every evidence of being inanimate and incapable of autonomous performance. The inert and insentient nature of genes is suggested by the way in which the nucleus reacts when removed from a cell. The nucleus, with its genes, promptly withers. The remaining cell, however, persists. Moreover, with nuclear genes absent such cells are capable of ordered divisions.

Along this same line, the human red blood cell aborts its nucleus prior to entering the blood stream where, minus its genes, it functions as a metabolizing, process-controlling, living unit. All of this suggests that genes are no more than dependent devices that need the presence of a living directorship.

If genes do not serve as the control center of the cell, what is it that does? McCann maintains that cellular activities are ultimately under the control of a living agency which he calls "cellular intelligence." This idea is vigorously rejected by Lumsden et al. Presumably it is because such a view conflicts with a materialistic and mechanistic view of cellular activities which they feel ought to prevail.

Call this controlling agency "cellular intelligence" or any other suitable expression. The intelligence connection seems appropriate, however, for if and when philosophers provide an overall definition of intelligence, surely it will include as one of its main ingredients the ability of intelligence to control and direct energy.

A view of the cell as being a kind of robotic engine made up of a meshwork of regularized, mechanistic arrangements in which a computer-like nucleus initiates and coordinates actions (even after the nucleus is no longer there) falls short. It is a concept that provides no means for solving the kinds of non-chartable demands which we know living systems are able to handle. And too, it introduces uncertainty relative to any latitude in individual decision-making when applied broadly.

Hampering inquiry, it seems manifest, is any insistence that to be acceptable a concept must have materialistic and mechanistic embodiments.

This is the reason, no doubt, why none other than Niels Bohr himself from his matchless experience with physical forces had this advice: If Biology wished to move to a higher level of understanding it would have to employ new concepts and new approaches that did not require mechanistic and reductionist interpretations

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THE DARWIN CONVERSION STORY: AN UPDATE

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Abstract

Since the discussions of Rusch (1975), Rusch and Klotz (1988) and Herbert (1990), new information on the Darwin conversion story has come to light. The earliest version of the story has been located in the Watchman-Examiner and some further biographical information on a possible candidate for Lady Hope is presented.

Introduction

Probably no other individual in modern times has had a greater influence in turning people away from the biblical account of creation, the scriptural view of mankind, and the authority of the Bible than Charles Darwin. His *Origin of Species* (1859) and *Descent of Man* (1871) were seen by many of his contemporaries as giving scientific credibility to the idea that all life developed by purely naturalistic processes. As a result Darwin provided considerable impetus for several atheistic ideologies which have dominated our troubled twentieth century.

In view of this, most people are surprised to hear that Charles Darwin allegedly became a believer in Christ near the end of his life. This story—in which we should rejoice, if true—has circulated in numerous tracts and magazine articles since 1915 (Rusch, 1975; Rusch and Klotz, 1988; and Herbert, 1990). It narrates an interview with Darwin, in the fall of the last year of his life, by a "consecrated English, woman" identified only as "Lady Hope." According to her account, she was allowed to visit with Darwin, and found him reading the New Testament epistle to the Hebrews, which he called "the Royal Book." When she asked him about Creation, he became very distressed and said that people had taken his unformed ideas and made a religion out of them. Darwin then invited her to speak to some of his servants, tenants and neighbors in his garden summerhouse on "Christ Jesus, and His salvation," and promised that he would sing along with them from his open bedroom window.

As Rusch (1975), Rusch and Klotz (1988), Herbert (1990) and others have pointed out, this account does not square well with other information we have about Darwin. His correspondence in the winter and spring following this alleged incident give no indication that Darwin's agnosticism had changed or that his belief in evolution had lessened. When Darwin's daughter, Henrietta Litchfield, heard this story, she denied that Lady Hope had visited Darwin, that Darwin's estate had a summerhouse, or that the "servants or villagers ever sang hymns to him." She claimed the story was invented in America (Rusch and Klotz, 1988, pp. 20-21, quoting from *The Christian*, 23 Feb. 1922).

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Some confusion exists in the various tracts as to whether this story was first reported by Lady Hope in Northfield, England or Northfield, Massachusetts. The date of her report is given as August 15, 1915. Rusch's attempt to find this report in the issues of the *Watchman-Examiner* available to him in the midwest was unsuccessful (Rusch and Klotz, 1988, p. 3).

Some New Findings

Intrigued by this story and spending my summers in the Washington, D.C. area, I decided to try to locate the article in the Library of Congress with its extensive resources. Assuming the *Watchman-Examiner* was some sort of periodical and using the date of August 15, 1915 as a starting point, the search was successful. The *Watchman-Examiner* was a national Baptist newspaper issued weekly from Boston and New York since 1819, with some variation in name over its history. The Library of Congress has nearly a complete run of the paper. The article turned up on the first issue following the above date (Hope, 1915) and is reprinted for your convenience:

This article was preceded by a four-page report on the 1915 Northfield Conference, a summer Bible conference held on the grounds of the Northfield Seminary, a girls' school in Northfield, Massachusetts founded years before by Dwight L. Moody. The conference that year ran from July 30 to August 15, and Lady Hope gave this testimony at one of the morning prayer services, the date not specified. The particular issue of this paper was stamped as received by the Library of Congress on August 19, so the account was in print no more than a few days or weeks after she gave it orally. Thus, so far as we know, the story was first circulated in the United States some 33 years after Darwin's death. Since this is also long after Darwin's wife Emma died in 1896, the suggestion that she started the story is unfounded.

Who was this Lady Hope? The *Watchman-Examiner* gives us no more information than has circulated in the tracts. As reported by Rusch and Klotz (1988) and Herbert (1990), a former editor of *Burke's Peerage*, L. G. Pine, was asked this question also. He could come up with only one "Lady Hope" who would have been grown in 1881 and still alive in 1915, a woman he names Elizabeth Reid Stapleton-Cotton, mentioned in