Speciation: Goldschmidt’s Chromosomal Heresy, Once Supported by Gould and Dawkins, is Again Reinstated

Donald R. Forsdyke

Received: 29 October 2016 / Accepted: 13 December 2016 / Published online: 28 December 2016 © Konrad Lorenz Institute for Evolution and Cognition Research 2016

Abstract The view that the initiation of branching into two sympatric species may not require natural selection emerged in Victorian times (Fleeming Jenkin, George Romanes, William Bateson). In the 1980s paleontologist Stephen Jay Gould gave a theoretical underpinning of this nongenic “chromosomal” view, thus reinstating Richard Goldschmidt’s “heresy” of the 1930s. From modeling studies with computer-generated “biomorphs,” zoologist Richard Dawkins also affirmed Goldschmidt, proclaiming the “evolution of evolvability.” However, in the 1990s, while Gould and Dawkins were recanting, bioinformatic, biochemical, and cytological studies were providing a deeper underpinning. In 2001 this came under attack from leaders in the field who favored Dawkins’ genic emphasis. Now, with growing evidence for the uncoupling of speciation from adaptation, we can reinstate again Goldschmidt’s view and clarify its 19th-century roots.

Keywords Adaptation uncoupled · Biomorphs · Collective variation · Evolvability · Macroevolution · Species selection

Hypotheses on Initiation

There are two groups of hypotheses on the initiation of a branching process that can lead to new species. One group emphasizes the role of natural selection in changing gene frequencies so that species arrival is fundamentally no different than species survival. Macroevolution is merely extrapolated microevolution. It is challenged by a group that downplays the role of natural selection and posits nongenic discordance between members of a species. Macroevolution is not extrapolated microevolution. For some speciations one group may apply. For other speciations the other group may apply. Agreement is sought as to which initiation mechanisms are actually, rather than hypothetically, capable of originating species, and which are most likely to have operated in the general case (Kliman et al. 2001; Forsdyke 2004; Johannesson 2010).

Both groups of hypotheses agree that the “spark” that initiates involves a mechanism for securing reproductive isolation so that the process is not subverted by recombination between the genomes of diverging types—such recombination would tend to homogenize rather than retain differences. Both groups also agree that reproductive isolation begins with interruption of the reproductive cycle—gamete, zygote, embryo, meiotic adult gonad, gamete, etc. Being a recursive cycle, any point, be it before or after union of gametes to form a zygote, will serve to mediate the primary interruption. Most points are susceptible to genic influence. Thus, there may be discordances (incompatibilities) between paternal and maternal genes affecting gamete transfer or union (prezygotic isolation), or between paternal and maternal genes affecting somatic development or gametogenesis (postzygotic isolation). The gametogenic point is under both genic and nongenic influence. Thus, here there is the potential for either genic or nongenic discordance. Whatever the point, for successful branching evolution, two independent cycles—two species—must eventually emerge.

While such hypotheses that unify a range of apparently disparate observations are valuable, they should be testable by experimentation and/or by computer modeling. Most speciation events occurred millions of years ago and
seem beyond the range of experiment. However, organisms with short generational cycles (viruses, bacteria) show promise in this respect (Forsdyke 2014; Kassen 2014; Meyer et al. 2016). Furthermore, there are computer simulations with various artificial life models (Anderson and Harmon 2014). Indeed, it will be shown here that one computer model (Dawkins 1988), accords well with long-held nongenic hypotheses for species initiation.

**Darwin and Jenkin**

The conceptually simplest form of primary reproductive isolation is the separation of members of a population into two types by a geographical barrier. This prezygotic isolation prevents the gametes on one side of the barrier uniting with gametes on the other side. Thus, two independently breeding types arise (allopatrian speciation). If their isolation is sustained, then other prezygotic and/or postzygotic differences will accumulate so that, should the primary barrier be removed, these secondary differences will then serve as barriers to maintain the reproductive isolation. Under an isolation shield, phenotypic differences appearing in members of branching species will positively or negatively affect their reproductive success in response to natural selection pressures. They will be adaptively successful or unsuccessful.

This view corresponds closely to that of Charles Darwin (1859). However, Darwin was challenged by Fleeming Jenkin (1867), who pointed out that adaptive responses to selection pressures must be balanced. Adaptation for flight, for example, invokes counter-adaptations in body weight. Better cognition seems to require an increase in brain weight, but flight imposes a limit on brain size. Thus, one adaptation invokes counter-adaptations, so achieving an organism best balanced to "pursue" a particular evolutionary strategy.

Jenkin saw members of a biological species as enclosed within a sphere that limited their variation. They could evolve so far, but no further. To escape beyond the limits of the sphere—to increase the evolvability of the species—something more was needed. Thus, there had to be what in modern terminology can be called an "evolution of evolvability." Once the Jenkian limit was overcome, the pace of evolution might increase and then perhaps slow as a new limit was approached. Thus its rate on a geological time scale might appear "punctuated."

**Evolution of Evolvability**

Jenkin, a Scottish professor of engineering with little biological expertise, claimed to be "an impartial looker-on" who would "admit the facts, and examine the reasoning." A century later, as in silico modeling of "artificial life" became increasingly feasible (Altenberg 1994), an Oxford professor with considerable biological expertise, Richard Dawkins, began from "what started out as an educational exercise" to follow the evolution of computer-generated "biomorphs" (Dawkins 1986). The "surprising consequences" (Dawkins 1988) were consistent with Jenkin’s viewpoint, although Jenkin was not cited in this respect.

While certain computer programs encoded "embryologies" (developmental plans) that were able cumulatively, under the selection pressure of the human eye, to generate elaborate model organisms, there were found to be limits. When exploring production of a "biomorph alphabet" with his "Blind Watchmaker" computer program, Dawkins (1988, p. 216) was "astonished and, delighted" to find:

There are some shapes that certain kinds of embryology seem incapable of growing. My present *Blind Watchmaker* embryology, that is the basic nine genes plus segmentation with gradients and symmetry mutations, is, I conjecture, forever barred from breeding a respectable K, or a capital B.

Likewise, when he tried to computer-breed radially symmetric starfish, Dawkins (1996, p. 239) observed that: "Computer biomorphs can look superficially like echinoderms, but they never achieve that elusive five-way symmetry." Indeed, "The program itself would have to be rewritten for that." In other words, *something more* was needed beyond the selection he was able to impose on a biomorph evolving within the limits that were intrinsic to a given computer program. Dawkins (1988, pp. 215–216) concluded:

Huge vistas of evolutionary possibility, in real life as well as in artificial life, may be kept waiting a very long time, if not indefinitely, for a major, reforming change in embryology. … [As for] the evolution of evolvability, … certain kinds of embryology find it difficult to generate certain kinds of biomorphs; other kinds of embryology find it easy to do so. It is clear that we have here a powerful analogy for something important in real biology, a major principle of life that is illustrated by artificial life. It is less clear which of several possible principles it is!

**Gonadal Location of a Primary Isolation Mechanism**

A location for a "major principle of life" that might allow escape beyond the limits of Jenkin’s sphere was inferred by the London physician St. George Mivart (1871, p. 249):
Now the new forms must be produced by changes taking place in organisms in, after, or before their birth, either in their embryonic, or toward or in their adult, condition. … It seems probable therefore that new species may arise from some constitutional affection of parental forms—an affection mainly, if not exclusively, of their generative system. Mr. Darwin has carefully collected numerous instances to show how excessively sensitive to various influences this system is.

Likewise, Darwin’s young research associate, George Romanes, inferred a constitutional affection of the “reproductive system” where cryptic “collective variations” might accumulate in a sector of a species (see below). This theme was extended by geneticist William Bateson, who was cognizant of Michael Guyer’s elegant studies of meiotic chromosomes (Forsdyke 1999, 2001, 2010; Cock and Forsdyke 2008). A more specific localization to the chromosomes of reproductive system germ cells was later postulated by the Danish “father of yeast genetics” Øjvind Winge (Szybalski 2001).

Winge interpreted the precise pairing of homologous chromosomes at meiosis in the gonads of diploid organisms as an error-correcting mechanism that demanded close sequence complementarity between parental chromosomes (Winge 1917). Should the parental chromosomes have diverged beyond a certain limit—a point of no return—then pairing would fail and their children (hybrids), while appearing phenotypically normal, would be sterile. Their sterility was a parental phenotype. As manifest in their offspring, the parents were reproductively isolated from each other. Should they marry again, then, with appropriate mates, fertile offspring might be produced and the line could continue. The couple-specific discordance was a general property of the chromosomes themselves and the prediction was made that an experimental genome duplication to generate tetraploid hybrids would “cure” the sterility. Each parental chromosome would then be able to pair with its like at meiosis, as is now recognized (Wolfe 2015).

Chromosomes as “Reaction Systems”

At that time Goodspeed and Clausen (1917, p. 38), from studies of crosses between allied species of tobacco plants, were also pointing to a higher order of organization that lay much above that of individual Mendelian genes (“factors”):

If, for example, it is possible to obtain hybrids involving not a contrast between factors within a single system, but a contrast of systems all along the line, then it is obvious that we must consider the phenomenon on a higher plane, we must lift our point of consideration as it were from the units of the system [genes] to the systems as units in themselves.

The latter unit systems were referred to as “reaction systems.” These seemed to correspond to chromosomes or large parts of chromosomes, and the degree of sterility of offspring correlated positively with differences (potential discordances) between such systems (Goodspeed and Clausen 1917, p. 50):

When distinct reaction systems are involved, as in species crosses, the phenomena must be viewed in the light of a contrast between systems rather than between specific factor differences, and the results obtained will depend upon the degree of mutual incompatibility displayed between the specific elements of the two systems. Sterility in such cases depends upon non-specific [nongenic] incompatibility displayed between elements of the system involved, and the degree of this sterility depends upon the degree of such incompatibility rather than upon a certain number of factors concerned in the expression of such behavior.

Crowther and Bateson

With prompting from Plymouth physician C. R. Crowther, William Bateson took this further. In remarks at the Toronto meeting of the American Association for the Advancement of Science he had attacked the Darwinian notion of species arising as a mere “summation of variations” affecting the conventional phenotype (i.e., microevolution; Bateson 1922a, p. 58):

But that particular and essential bit of the theory of evolution which is concerned with the origin and nature of species [Bateson’s italics] remains utterly mysterious. We no longer feel … that the process of variation, now contemporaneously occurring, is the beginning of a work which needs merely the element of time for its completion; for even time cannot complete that which has not yet begun. The conclusion in which we were brought up, that species are a product of a summation of variations ignored the chief attribute of species first pointed out by John Ray that the product of their crosses is frequently sterile in greater or less degree. Huxley, very early in the debate pointed out this grave defect in the evidence, but before breeding researches had been made on a large scale no one felt the objection to be serious.

Crowther (1922) began by noting that, while parental chromosomes had to cooperate for development of the
zygote from embryo to adult, a far higher degree of cooperation—a high concordance—would be needed when the chromosomes paired ("conjugated") in the gonad of that adult:

Homologous chromosomes ... have to cooperate to produce the somatic cell of the hybrid, and their cooperation [for embryo development] might be expected to require a certain resemblance; but for the production of sexual cells [gametogenesis] they must do more, they must conjugate [pair]; and for conjugation it is surely reasonable to suppose that a much more intimate resemblance would be needed. We might, therefore, expect, on purely theoretical grounds, that as species and genera gradually diverged, it would be increasingly difficult to breed a hybrid between them; but that, even while a hybrid could still be produced, a fertile hybrid would be difficult or impossible, since the cells of the germ-track would fail to surmount the meiotic reduction stage when the homologous chromosomes conjugate. This is exactly what happens: the cells go to pieces in the meiotic phase.

Bateson’s disparagement of the idea that species might be “a product of a summation of variations” left Crowther (1922) “frankly puzzled,” for “the proposition is certainly not self-evident.” Surely, if the sterility of an offspring were due to a failure within that offspring of homologous chromosomes to pair, it mattered little whether the lack of complementarity responsible for that failure was produced by one large variation, or by the summation of many smaller variations. That Crowther was thinking of primary variations occurring at the chromosomal level, rather than anatomical variations of the sterile individual, was explicit:

If a sword and scabbard are bent in different directions, it will happen sooner or later that the sword cannot be inserted, and the result will be the same whether the bending be effected by a single blow, or whether it be, in Dr. Bateson’s words, “a product of a summation of variations.” Is this illustration apt? The sword and the scabbard are the homologous chromosomes. ... It seems easier to imagine sterility arising from a gradual modification, spread over a length of time, and involving many chromosomes.

Bateson (1922b) conceded that discontinuity of variation (sudden change) was not critical:

It is ... not difficult to “imagine” interspecific sterility produced by a gradual (or sudden) modification. That sterility might quite reasonably be supposed to be due to the inability of certain chromosomes to conjugate, and Mr. Crowther’s simile of the sword and the scabbard may serve to depict the sort of thing we might expect to happen.

Thus, Bateson agreed with Crowther that a fundamental form of reproductive isolation, manifest as the hybrid sterility seen when members of allied species were crossed, could be due to an incompatibility characterized cytologically as defective pairing of paternal and maternal chromosomes at meiosis. It was inferred that if we can understand what makes chromosomes incompatible, then we can understand hybrid sterility. And if we can understand hybrid sterility, we can understand an origin of species.

A Modern Interpretation

But how do chromosomes that are homologous (i.e., are alike) pair with each other? Do they pair by virtue of this likeness (like-with-like), or by virtue of some key-in-lock (sword-in-scabbard) complementarity, which implies that they are not really alike? One must be the sword and the other the scabbard.

We now appreciate that this paradox was resolved when it was discovered that hereditary information was stored and transmitted as duplex DNA, with two strands—a “Watson” strand and a “Crick” strand—that paired with each other by virtue of base complementarity. So, in Crowther’s terminology, potentially the sword strand of one chromosome can pair with the scabbard strand of the homologous chromosome (and vice versa). For this swords have to be unsheathed from their own scabbards and then each inserted into the scabbards of the other. Thus the Watson strand of one chromosome must pair with the Crick strand of the other, and vice versa. This requires that the Watson strand be displaced from pairing with the Crick strand of its own chromosome. Likewise, the Crick strand of the homologous chromosome must be displaced from pairing with the Watson strand of its own chromosome. Then cross-pairing can occur.

The pairing requires complementarity of DNA base sequences. A sporadically appearing change in an individual base could, if dominant, introduce a new phenotype, but would not greatly affect the overall complementarity between parental chromosomes. However, over time, base changes—including some affecting genes, but many not-affecting genes—could accumulate. Romanes’ “collective variation” that would build up in “a section of a species” (Romanes 1897; Forsdyke 1999), can now be interpreted as a general variation between paternal and maternal DNA sequences. When such differences between chromosomal homologs reached a critical value, meiotic pairing in an offspring’s gonads would be impaired and gametogenesis would begin to fail. This early gametogenesis barrier would
eventually yield to the developmental and transmission barriers—both of genic origins (Forsdyke 2001, 2016). To understand how this view came about, we must go back to the 1930s prior to any appreciation of the structure and role of DNA.

**Chromosomal Repatterning**

The idea of chromosomes as “reaction systems” was taken up by Richard Goldschmidt in the 1930s (Forsdyke 2003). Such “reaction systems,” through a “repatterning,” involving “systemic mutations,” might change into other “reaction systems.” Thus Goldschmidt (1940, pp. 200–206) wrote:

> The classical theory of the gene and its mutations did not leave room for any other method of evolution. Certainly a pattern change within the serial structure of the chromosome, unaccompanied by gene mutation or loss, could have no effect whatsoever upon the hereditary type and therefore could have no significance for evolution. But now pattern changes are facts of such widespread and, as it seems, typical occurrence that we must take a definite stand regarding their significance. ... The pattern changes are in themselves effective in changing the genotype without any change of individual genes. ... Point mutations have never been known to change the point-to-point attractions between the homologous chromosomes in the heterozygote. ...A repatterning of a chromosome may have exactly the same effect as an accumulation of mutations. ... The change from species to species is not a change involving more and more additional atomistic changes, but a complete change in primary pattern or reaction system into a new one, which afterwards may again produce intraspecific variation by micromutation.

Chromosomal “repatterning,” namely a change in “the arrangement of the serial chemical constituents of the chromosomes,” proceeded slowly and progressively, without necessarily producing any change in the structure or function of organisms, until a new species emerged that was reproductively isolated from the old one by virtue of the new pattern being “incompatible” with that of the old:

> A systemic mutation (or series of such) ... consists of a change of intrachromosomal pattern.... Whatever genes or gene mutations might be, they do not enter this picture at all. Only the arrangement of the serial chemical constituents of the chromosomes into a new, spatially different order; i.e., a new chromosomal pattern, is involved. The new pattern seems to emerge slowly in a series of consecutive steps....

These steps may be without a visible effect until the repatterning of the chromosome... leads to a new stable pattern, that is, a new chemical system. This may have attained a threshold of action beyond which the physiological reaction system of development, controlled by the new genetic pattern, is so basically changed that a new phenotype emerges, the new species, separated from the old one by a bridgeless gap and an incompatible intrachromosomal pattern. (Goldschmidt 1940, p. 206)

By “incompatible” Goldschmidt was here referring to differences between the chromosomes of two potential parents. These chromosomes would consequently not be able to cooperate functionally and/or to pair properly at meiosis within their child. They would be discordant.

An unlimited number of patterns is available without a single qualitative chemical change in the chromosomal material, not to speak of a further unlimited number after qualitative changes (model: addition of a new amino acid into the pattern of a protein molecule).... These pattern changes may be an accident, without any significance except for creating new conditions of genetic isolation by chromosomal incompatibility.... (Goldschmidt 1940, p. 245)

This may seem labored, but in the 1930s it was known neither what genes were chemically, nor how that chemistry might be altered when mutations occurred. However, in the 1870s Ewald Hering and Samuel Butler had laid a framework for thinking about heredity in informational terms (Forsdyke 2006). Striving to give some meaning to his concept of pattern, Goldschmidt (1940, p. 248) wrote:

> Let us compare the chromosome with its serial order to a long printed sentence made up of hundreds of letters of which only twenty-five different ones exist. In reading the sentence a misprint of one letter here and there will not change the sense of the sentence; even the misprint of a whole word (rose for sore) will hardly impress the reader. But the compositor must arrange the same set of type into a completely different sentence with a completely new meaning, and this in a great many different ways, depending upon the number of permutating letters and the complexity of the language (the latter acting as a “selection”). To elevate such a model to the level of a biological theory we have, of course, to restate it in chemical terms.

Four years before Oswald Avery showed that DNA was the form in which hereditary information was transferred through the generations, and 13 years before Watson and Crick presented a model for DNA, it was not unreasonable...
to think of chromosomal patterns in terms of amino acid, rather than nucleotide, sequences. Thus Goldschmidt (1940, p. 248) wrote:

I do not think that an actual chemical model can yet be found. But we might indicate the type of such a model which fulfills at least some, though not all, of the requirements. It is not meant as a hypothesis of chemical chromosome structure, but only as a chemical model for visualizing the actual meaning of a repatterning process .... Let us compare the chromosome to a very long chain molecule of a protein. The linear pattern of the chromosome is then the typical pattern of the different amino acid residues.

Improvements in staining technologies facilitated chromosome studies in dividing cells and lent further support to views on chromosomal incompatibilities as drivers of speciation (White 1978; King 1993). However, in the latter half of the 20th century the controversy coalesced around two figures, Dawkins (advocate of natural selection and genes) and Stephen Jay Gould (advocate of hierarchical species selection). This gained wide public attention, as is related in popular texts such as The Evolutionists (Morris 2001) and Dawkins versus Gould (Sterelny 2007). The major, albeit transient, support for Goldschmidt’s nongenic “macroevolutionary” approach to species initiation came from both the theoretical underpinnings of paleontologist Gould (1980), and the biomorph studies of zoologist Dawkins (1988).

**Gould and Higher-Level “Species Selection”**

In “Is a New and General Theory of Evolution Emerging?” Gould (1980) recapitulated Goldschmidt’s case. Arguing that “macroevolutionary trends [speciations] do not arise from the gradual, adaptive transformation of populations, but usually from a higher-order selection operating upon groups of species,” Gould distinguished “species selection”—one of various forms of group selection—from conventional natural selection that acts upon individual organisms. Species selection was achieved by chromosomal alterations in isolating mechanisms, sometimes called the theory of chromosomal speciation.” The conventional Darwinian view was that selection preceded isolation. Gould (1980, p. 124) reversed this order:

But in saltational, chromosomal speciation, reproductive isolation comes first and cannot be considered as an adaptation at all. It is a stochastic event that establishes a species by the technical definition of reproductive isolation. To be sure, the later success of this species in competition may depend upon its subsequent acquisition of adaptations; but the origin itself may be non-adaptive. We can, in fact, reverse the conventional view and argue that speciation, by forming new entities stochastically, provides raw material for selection.

Gould had cited the second volume of Romanes’ Darwin, and After Darwin (1895), but neither in his 1980 paper, nor subsequently, did Gould refer to Romanes’ masterpiece—the posthumously published third volume (Romanes 1897). Here a case identical to Gould’s had been made. In a theory of “physiological selection” Romanes (1897, pp. 52–52) had declared that “diversification of character can never be originated by natural selection.” A “morphological divergence” guided by natural selection could only be secondary. There was some “physiological peculiarity” of the reproductive system, the basis for which it was for the future to determine. Whatever its basis:

At least in a large number of cases, it was the physiological peculiarity which first of all led to the morphological divergence, by interposing the bar of sterility between two sections of a previously uniform species; and by thus isolating the two sections one from another, started each upon a subsequent independent course of divergent evolution. ... In the absence of other forms of isolation [e.g. geographical], the morphological divergences could not have taken place at all, had not the physiological peculiarity arisen.

Praising Goldschmidt for having provided a logical basis for “species selection,” Gould (1982) encouraged, and wrote a forward for, a reprinting of The Material Basis of Evolution (Goldschmidt 1940). However, Gould rejected Goldschmidt’s notion of “‘systemic mutations’ involving the entire genome.” On the other hand, Romanes (1897, p. 60) had characterized his “physiological peculiarity” as “a ‘collective variation’ affecting a number of individuals simultaneously, and therefore characterizing a whole race or strain” (i.e., a section of a species). This is consistent with our modern understanding. The Goldschmidtian abstractions can now be fleshed out in both bioinformatic and biochemical terms (Forsdyke 2001, 2003, 2016).

**Dawkins and Higher-Level “Species Selection”**

Apparently overlooked by Gould, powerful support for Goldschmidt’s viewpoint came from the computer simulations of Dawkins. Like Goldschmidt’s microevolution/macroevolution dichotomy, as a result of his biomorph simulations Dawkins (1988, p. 217) called for a distinction...
“between two kinds of mutation: ordinary changes within an existing genetic system, and change to the genetic system itself.” The former were “the standard mutations that may or may not be selected in normal evolution within a species.” The latter were “changes to genetic systems [that] must have been, at least in one sense, major changes, changes of a different order from the normal allele substitutions that go on within a genetic system.” The latter class of mutation was associated with “changes in embryology which … are … evolutionarily pregnant.” Thus:

As the ages go by, changes in embryology that increase evolutionary richness tend to be self-perpetuating. … I am talking about a kind of higher-level selection, a selection not for survivability but for evolvability. … Others have pointed out that we should speak of “species selection” only in those rare cases where a true species-level quality is being evolved. Species selection, for instance, should not be invoked to explain an evolutionary lengthening of the leg, since species don’t have legs, individuals do. It might, on the other hand, be invoked to explain the evolution of a tendency to speciate, since speciating is a thing species, but not individuals, do. It now seems to me that an embryology that is pregnant with evolutionary potential is a good candidate for a higher-level property of just the kind that we must have before we allow ourselves to speak of species or higher-level selection. (Dawkins 1988, pp. 218–219)

However, unlike Gould whose higher-level selection was due to “a stochastic event,” Dawkins would not stray from the agency of natural selection and, furthermore, did not recognize a relationship between his own work and that of Goldschmidt:

Perhaps there is a sense in which a form of natural selection favors, not just adaptively successful phenotypes, but a tendency to evolve …. I have been in the habit of disparaging the idea of “species selection” …. But selection among embryologies for the property of evolvability, it seems to me, may have the necessarily qualifications to become cumulative in evolutionarily interesting ways. After a given innovation in embryology has been selected for its evolutionary pregnancy, it provides a climate for new innovations in embryology. Obviously the idea of each new adaptation serving as the background for the evolution of subsequent adaptations is commonplace, and is the essence of the idea of cumulative selection. What I am now suggesting is that the same principle may apply to the evolution of evolvability, which, therefore, may also be cumulative. (Dawkins 1988, p. 219)

### Uncoupling of Speciation from Adaptation

Sadly, in The Structure of Evolutionary Theory, Gould (2002, pp. 1002–1003) recanted his Goldschmidtian heresy of the 1980s, while still maintaining “a hierarchical theory of selection.” Thus:

I do not, in fact or retrospect … regard this 1980 paper as among the strongest … that I have ever written …. I then read the literature on speciation as beginning to favor sympatric alternatives to allopatric orthodoxies at substantial relative frequency, and I predicted that views on this subject would change substantially, particularly towards favoring mechanisms that would be regarded as rapid even in microevolutionary time. I now believe I was wrong in this prediction.

Likewise we peruse in vain Brief Candle in the Dark (Dawkins 2015) for an expanded recognition of the implications of Dawkins’ biomorph studies. Forgetting what was once “drummed into my innermost consciousness” (Dawkins 1988), he remains a self-proclaimed “dyed-in-the-wool, radical neo-Darwinian” reiterating previous condemnations of the “utter nonsense” perpetrated by William Bateson. The full case for reinstallation of the Goldschmidtian certitudes, once so lavishly entertained and then later disavowed by these great evolutionists, is set out elsewhere (Forsdyke 2016). Here, a few quotations must suffice.

Greig (2007) found that “speciation genes do not play a major role in yeast speciation,” and proposed that “simple sequence divergence is the major cause of sterility in F1 hybrids formed between S. cerevisiae and S. paradoxus.” Commenting on this, Louis (2009) concluded that “one must be cautious in labelling gene incompatibilities as speciation genes, or at least in interpreting them as being causal in the speciation process rather than a result of divergence post-speciation.”

Venditti et al. (2010) noted that a dependence of branching on synonymous mutations, which do not change the encoded amino acid, seemed to exclude natural selection as a general initiator of species divergence and linked “speciation to rare stochastic events that cause reproductive isolation.” Thus:

Species do not so much “run in place” as simply wait for the next sufficient cause of speciation to occur. Speciation is freed from the gradual tug of natural selection; there need not be an “arms race” between the species and its environment, nor even any biotic effects. To the extent that this view is correct, the gradual genetic and other changes that normally accompany speciation may often be consequental to the event that promotes the reproductive isolation, rather than causal themselves.
Likewise, Hedges et al. (2015, p. 842), from analyses of 50,000 eukaryotic species, infer “an uncoupling of speciation from adaptation,” and conclude that “adaptive change that characterizes the phenotypic diversity of life would appear to be a separate process from speciation.” Furthermore, Bhattacharyya et al. (2013, p. E468) from mouse breeding studies point to nongenic (noncoding) sequence differences as a basis for hybrid sterility:

We propose the heterospecific pairing of homologous chromosomes as a preexisting condition of asynapsis [failure of chromosome pairing] in interspecific hybrids. The asynapsis may represent a universal mechanistic basis of F1 hybrid sterility manifest as pachytene arrest. It is tempting to speculate that a fast-evolving subset of the noncoding genomic sequence important for chromosome pairing and asynapsis may be the culprit.

As for molecular footprints of isolation mechanisms, Lawrie et al. (2013) found strong selection at synonymous sites in the fruit fly and concluded that: “The underlying biological function disrupted by these [synonymous] mutations is unknown, but it is not related to the forces generally believed to be the principal actors shaping the evolution of synonymous sites.”

That such a force might relate to speciation and DNA base composition (“1-mer” base frequencies; Forsdyke 1996) is generalizable to higher oligonucleotide compositions (“k-mer” frequencies; Forsdyke 2016). Consistent with this, Brbic et al. (2015, p. 1519) note that there is a conflict between the needs of DNA, and of the proteins it encodes, that strongly favors DNA. Genomes are dominated by oligonucleotide frequencies that can overrule the needs of protein encoding to the extent that the latter is reflected in amino acid compositions:

We find that G + C content, the most frequently used measure of genomic composition, cannot capture diversity in amino acid compositions and across ecological contexts. However, di-/trinucleotide composition in intergenic DNA predicts amino acid frequencies of proteomes to the point where very little cross-species variability remains unexplained. … A corollary is that the previously proposed adaptations of proteomes to environmental challenges … may need to be reinterpreted, while taking into account the evolutionary forces shaping DNA oligonucleotide frequencies.

Conclusion

We still do not know which species initiation mechanisms are most likely to have operated in the general case, but “nature’s experiments,” which long ago left their marks on the plenitude of nucleotide sequences that became available for bioinformatic study in the 1990s, continue to be supportive of Goldschmidt’s chromosomal viewpoint. In the 1980s, despite their different perspectives, two highly influential “public intellectuals” had also been supportive. However, having reached a degree of consensus, they later dismissed and declined to reinstate it. Discounting the historical and growing new evidence, there developed a notorious dispute between these celebrity scientists much of which, with other perhaps prejudicial views (Kliman et al. 2001; Johannesson 2010), must now be laid to rest.

Acknowledgements Queen’s University hosts my web pages where much of the early literature, including the relatively inaccessible texts of Guyer and Winge, has been made available.

References

Altenberg L (1994) The evolution of evolvability in genetic programming. In: Kinneir KE (ed) Advances in genetic programming. MIT Press, Cambridge, pp 47–74
Anderson CJR, Harmon L (2014) Ecological and mutation-order speciation in digital organisms. Am Nat 183:257–268
Bateson W (1922a) Evolutionary faith and modern doubts. Science 55:55–61
Bateson W (1922b) Interspecific sterility. Nature 110:76
Bhattacharyya T, Gregorova S, Mihola O et al (2013) Mechanistic basis of infertility of mouse intersubspecific hybrids. Proc Nat Acad Sci USA 110:E468–E477
Brbić M, Warnecke T, Kriško A, Supek F (2015) Global shifts in genome and proteome composition are very tightly coupled. Gen Biol Evol 7:1519–1532
Cock AG, Forsdyke DR (2008) “Treasure your exceptions.” The science and life of William Bateson. Springer, New York
Crowther CR (1922) Evolutionary faith and modern doubts. Nature 109:777
Darwin C (1859) The origin of species by natural selection, or the preservation of favoured races in the struggle for life. John Murray, London
Dawkins R (1986) The blind watchmaker. Longman Scientific, Harlow
Dawkins R (1988) The evolution of evolvability. In: Langton C (ed) Artificial life, SFI studies in the sciences of complexity. Addison-Wesley, Boston, pp 201–220
Dawkins R (1996) Climbing mount improbable. Norton, New York
Dawkins R (2015) Brief candle in the dark. My life in science, Harper-Collins, New York
Forsdyke DR (1996) Different biological species “broadcast” their DNAs at different (G + C)% “wavelengths.”. J Theor Biol 178:405–417
Forsdyke DR (1999) Two levels of information in DNA: Relationship of Romanes’ “intrinsic” variability of the reproductive system, and Bateson’s “residue”, to the species-dependent component of the base composition, (C + G)%. J Theor Biol 201:47–61
Forsdyke DR (2001) The origin of species, revisited. A Victorian who anticipated modern developments in Darwin’s theory. McGill-Queen’s University Press, Montreal
Forsdyke DR (2003) William Bateson, Richard Goldschmidt, and non-genic modes of speciation. J Biol Syst 11:341–350
Forsdyke DR (2004) Chromosomal speciation: a reply. J Theor Biol 230:189–196

2 Springer
Forsdyke DR (2006) Heredity as transmission of information. Butlerian intelligent design. Centaurus 48:133–148
Forsdyke DR (2010) George Romanes, William Bateson, and Darwin’s “weak point.” Notes Rec R Soc 64:139–154
Forsdyke DR (2014) Implications of HIV RNA structure for recombination, speciation, and the neutralism-selectionism controversy. Microbes Infect 16:96–103
Forsdyke DR (2016) Evolutionary bioinformatics, 3rd edn. Springer, New York
Goldschmidt R (1940) The material basis of evolution. Yale University Press, New Haven
Goodspeed TH, Clausen RE (1917) Mendelian factor differences versus reaction system contrasts in heredity. Am Nat 51:31–46, 92–101
Gould SJ (1980) Is a new and general theory of evolution emerging? Paleobiology 6:119–130
Gould SJ (1982) The uses of heresy: introduction. In: Goldschmidt RB (au) The material basis of evolution, Yale University Press, New Haven, reprint of 1940, pp xii–xlii
Gould SJ (2002) The structure of evolutionary theory. Harvard University Press, Cambridge
Greig D (2007) A screen for recessive speciation genes expressed in the gametes of F1 hybrid yeast. PLoS Genet 3:281–286
Hedges SB, Marin J, Suleski M et al (2015) Tree of life reveals clock-like speciation and diversification. Mol Biol Evol 32:835–845
Jenkin F (1867) The origin of species. North Brit Rev 46:277–318
Johannesson K (2010) Are we analyzing speciation without prejudice? Ann N Y Acad Sci 1206:143–149
Kassen R (2014) Experimental evolution and the nature of biodiversity. Roberts, Englewood
King M (1993) Species evolution. In: The role of chromosome change. Cambridge University Press, Cambridge
Kliman RM, Rogers BT, Noor MAF (2001) Differences in (G + C) content between species: a commentary on Forsdyke’s “chromosomal viewpoint” of speciation. J Theor Biol 209:131–140
Lawrie DS, Messer PW, Hershberg R, Petrov DA (2013) Strong purifying selection at synonymous sites in D. melanogaster. PLoS Genet 9:e1003527
Louis EJ (2009) Origins of reproductive isolation. Nature 457:549–550
Meyer JR, Dobias DT, Medina SJ et al (2016) Ecological speciation of bacteriophage lambda in allopatry and sympatry. Science 354:1301–1304
Mivart St GJ (1871) On the genesis of species. Appleton, New York
Morris R (2001) The evolutionists. Norton, New York
Romanes GJ (1897) Darwin, and after Darwin. Isolation and physiological selection, vol 3, Longmans, Green, London
Sterelny K (2007) Dawkins vs Gould: survival of the fittest. Icon Books, Cambridge
Szybalski W (2001) My road to Öjvind Winge, the father of yeast genetics. Genetics 158:1–6
Venditti C, Meade A, Pagel M (2010) Phylogenies reveal new interpretation of speciation and the red queen. Nature 463:349–352
White MJ (1978) Modes of speciation. Freeman, San Francisco
Winge Ö (1917) The chromosomes, their number and general importance. Compt Rend Trav Lab Carlsberg 13:131–275
Wolfe KH (2015) Origin of the yeast whole-genome duplication. PLoS Biol 13:e1002221