

# Review of: "Mutational selection: fragile sites, replicative stress, and genome evolution"

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David Haig here describes the “evolution of dominance” in “continuous Weismannian germlines.” The citing of Weismann (1892) invites attention to the history. It has long been known that mutations that confer a proliferative advantage on germ cells, apart from influencing somatic phenotype, can influence Weismannian continuity. The influence on offspring is greater with elderly male parents (paternal age effect). Although Haig does not cite Weinberg (1912), he cites Goriely and Wilkie (2012) and Arnheim and Calabrese (2016), who do.

Both groups also refer to the pioneering studies of James F. Crow. In a 2012 paper – “Upsetting the dogma: germline selection in human males” (PLoS Genet 8(2): e1002535), Crow described Weinberg’s study as “an astonishing intellectual leap.” The present paper might benefit from a clarification of the history in two respects. First, a comment attached to Crow’s paper might provide guidance:

*“We salute Wilhelm Weinberg for suggesting in 1912 that achondroplastic dwarfs would tend to be among the last-born children in a family, because there was then an increased probability that parental gamete-forming cells had accumulated the necessary pre-meiotic mutations (1). However, whether this was an ‘astonishing intellectual leap’ can be questioned. As far as my primitive German will allow, it seems that Weinberg did not cite Bateson. Yet, it is likely that Weinberg was familiar with the reduplication hypothesis of William Bateson and Reginald Punnett that postulated such mutations. Their hypothesis was presented in 1911 at the 4th International Congress of Genetics in Paris. The same year it was published in both English (2) and German journals (Mendel Festschrift; 3). From a parallel with plants, they suggested that the mutations might be localized to particular testicular segments, the ‘jackpot’ approach adopted by the Arnheim laboratory. In his 1974 Presidential Address at the 13th International Congress of Genetics, Curt Stern noted that while the reduplication hypothesis ‘could not beat the competition’ in explaining genetic linkage, nevertheless ‘it was an ingenious suggestion’ to propose that ‘Mendelian segregation ... occurs during a somatic cell division followed by differential multiplication of the different genotypes’ (4). In our 2008 biography of Bateson we drew attention to the possibility that achondroplastic dwarfism can be explained in such terms (5).”*

Second, since much of the paper concerns alleles competing for dominance, clarification of the history of hypotheses for dominance, dating back to de Vries, George Shull and Bateson, might be helpful. Reference 5 (pp. 369-370) might assist.

1. Weinberg W (1912) Zur Vererbung des Zwergwuchses. Arch. Rassen-u Gesell Biolog 9: 710-717.
2. Bateson W, Punnett RC (1911) On gametic series involving reduplication of certain terms. Journal of Genetics 1: 293-302.
3. Bateson W, Punnett RC (1911) Verhandlungen Naturforsch. Verein. Brunn 49: 324-334.
4. Stern C (1974) The domain of genetics. Genetics 78: 21-33.
5. Cock AG, Forsdyke DR (2008) Treasure You Exceptions. The Science and Life of William Bateson. New York: Springer.