Common examples of 'one gene, one trait' exposed

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I taught college level human genetics and was chagrined to learn that the textbook/workbook I used for a decade until 2017[1] incorrectly claimed the older, now-refuted, view that common examples of traits were produced by a single gene was valid. A number of claimed examples were researched, finding the expression of all of them were influenced by many genes. Of these examples, 10 were selected and summarized. This finding supports the fact that to produce new traits, even seemingly simple ones, requires several genes as a set, increasing the complexity significantly and reducing the likelihood of producing these new traits by random changes in the genotype as postulated by evolution.

High school and college biology students are often taught that many basic human traits are examples of simple one-gene regulation.[2] This is reflected in common statements, such as "twenty thousand years later, another mutation allowed the hair on our heads to (unlike monkey hair or body hair) grow indefinitely long—a haircut gene".[3] The genetics workbook used at the college where I taught, besides the examples discussed below, included the shape of the face, the hair curl, eyebrow size and shape, eyelash length, tongue folding, and others for a total of 27 examples.[1] A review completed for this paper found that many traits long assumed to be a result of a single mutation, or a single gene, have turned out to be produced by numerous genes.

In studying factors that produce specific nose appearances, scientists have so far identified 14 different facial traits from analyzing about 6,000 photographs. Two genes were linked to nostril shape (GLI3 and PAX1), one (RUNX2) affected the bridge, and the DCHS2 gene was found to influence how far the nose extends outward, and the tip angle.[4] No doubt others also exist. McDonald concluded, "It is an embarrassment to the field of biology education that textbooks and lab manuals continue to perpetuate these myths" of one gene-one trait, even today.[5]

The significance for evolution of the 'one gene producing one protein or one trait' argument can be easily explained by William Paley's classic watch example. A wind-up watch has hundreds of parts, all of which function as part of an interconnected unit. If any one part in the movement mechanism is removed, the watch will not function properly or, most likely, will not function at all. The only exception may be the complete movement mechanism which is often held together by four to six screws and, if one is removed, it may still function, but the snugness in the area where the screw was removed, even if minor, may cause a slight sloppiness that could eventually cause uneven wear and less accurate time, leading to significant inaccuracies and, eventually, failure.

Likewise, if most genetic functions are controlled by several genes, a mutation, even if it improves some aspect of the organism, will likely have an adverse effect on some other part of the living system because, like the watch, all of the parts function together as a unit with many, or at least several, other genes and even gene systems.

Furthermore, the concept of what defines a single gene has now been blurred by unexpected genetic complexity, such as the fact that differential splicing of introns can produce many different proteins from one gene transcript.[6] The classic view of the genome, that protein-producing genes are distinct segments of DNA transcribed into one mRNA and in one direction, has now been overthrown. It is now recognized that multiple and overlapping genes often occupy a single strip of DNA that produces several functional mRNAs.[7] An estimated 94% of human genes generate more than one product by alternative splicing, producing proteins with sometimes dramatically different functions, despite being produced from the same gene.[8]

This fact is solace to those disappointed by the small number of genes in the human genome: humans have roughly the same number of genes as *Caenorhabditis elegans*, a small transparent worm. A homologue of the human gene Dscam (Downs Syndrome Cell Adhesion Molecule) as expressed in *Drosophila melanogaster,* produces 95 alternative spliced exons and 38,000 possible isoforms.[9] In comparison, the entire *Drosophila melanogaster* genome contains only 15,016 genes. This fact allows us a glimpse of the complex interconnectivity of the genome.

University of Delaware Genetics Professor John McDonald has documented 20 examples, including arm folding, asparagus urine, beeturia, cheek dimples, hair whorl, and toe length, that were commonly used to teach the one gene-one trait concept in high school and college genetics

classes.[10] A literature search was completed on the 10 most common examples of the one gene-one trait claim, selected according to my experience teaching college-level genetics. The review found the 10 most common examples of the one gene-one trait myth used in textbooks were controlled by numerous genes.

Some refutations of the one-gene control system

Eye colour

A common example is the genetic control of eye colour, actually the colour of the iris diaphragm which controls the amount of light allowed to enter the eye. Its function is protection.[11] For example, light-blue eyes cause visual problems because the lack of brown pigment on the iris diaphragm does not fully protect the retina against the adverse effects of excess bright light.[12] Iris colour is determined primarily by the ratio of eumelanin, which produces a dark-brown colour, and pheomelanin, which produces a reddish colour. Also important is how melanin is distributed on the iris.[13]

The common iris colour categories are blue, green, hazel, and brown. A more detailed analysis has produced at least nine hue and saturation values, which are: (i) light blue; (ii) darker blue; (iii) blue with brown peri-pupillary ring; (iv) green; (v) green with brown iris ring; (vi) peripheral green central brown; (vii) brown with some peripheral green; (viii) brown; and, (ix) dark brown.[14] This more complete category obviously requires far more genetic input than the traditional two genes, which is what genetic research has found. Furthermore, eye colour can also change, especially during early childhood.[15] The two main genes involved in colour variation are HERC2 and OCA2 genes, located next to each other on chromosome 15, but at least 10 other genes, and the complicated interactions between them, also influence eye colour.[13]

Earlobe attachment

A common genetics classroom exercise is to examine each other's earlobe type and determine how many had attached versus unattached or free earlobes. Attached earlobes blend in with the side of the head, and unattached earlobes have hanging lobes that can dangle. Usually, many more students have unattached than attached earlobes.

The Mendelian claim is this trait is due to a dominant-recessive dichotomy. In this case, the claim is that the free earlobe trait is dominant and the attached is recessive.[16] The attached-free variant is used as an example of a classic single-gene recessive trait, an explanation that implies genetic control is relatively simple.[17] In fact, earlobes do not fall into two categories, rather there are continuous variations in attachment points, from up near the ear cartilage to well below the ear.[18]

This observation caused biologists to question this oversimplified paradigm even before modern genomics. As early as 1937, one anatomist suggested that earlobe attachment may be a multi-gene trait.[19] One new earlobe genetics study analyzed DNA sequences and earlobe measurements from 74,660 people, including those of European, Latin American, and Asian ancestry. By associating DNA sequences across the genome with ear development patterns, the researchers identified 49 genomic regions related to earlobe-attachment design. They also sequenced the products of genes activated during ear development, confirming that the different genes discovered in their DNA trait study were located among many regions in the genome. The authors of the paper concluded: "These genes provide insight into the complex biology of ear development."[20]

As expected, the genetics behind these variations are complicated but not a research priority because, as far as is known, earlobe variations are unrelated to disease causation.[21] In short, it is a "myth … that earlobes can be divided into two clear categories, free and attached, and that a single gene controls the trait, with the allele for free earlobes being dominant".[22] Modern research techniques have now confirmed that "even the concept of what clearly defines a single gene is blurred by unimaginable and unexpected complexity".[23]

Hair colour

Another common example of the one-gene myth is that red hair is determined by a single recessive red allele. Most studies divide hair colour into four discrete colours, namely blond, red, brown, and black. As is true of eye colour, hair colour is also determined by the amount of eumelanin (dark-brown colour) and pheomelanin (a reddish colour) in the hair shaft. The amount of eumelanin ranges from very little, producing light-blonde hair, to relatively large amounts, producing jet black hair. In addition, people with large amounts of pheomelanin have red hair, which ranges from pale red ('strawberry blonde') to bright red and reddish-brown.[24]

Because light level variations and colour hue differences can affect viewers' perceptions of hair colour, Reed used a reflectance spectrophotometer to measure the light levels reflected by hair at different wavelengths in persons labelled as redheads.[25] He found no evidence of a clear separation of hair into two categories. Instead, intermediate colours were noted that could not easily be classified as, for example, red or non-red. The three most common amino acid

polymorphisms associated with red hair are R151C, R160W, and D294H, indicating that many more genes are involved in other hair shades.[26]

Skin colour

Human genetics studies have debunked the belief that skin colour is controlled by only a few major genes.[27] Several studies have utilized human subjects from the continent with the largest spectrum of skin colour diversity in the world, namely Africa. One study found that six major genes contributed only 30% of the total skin-colour variability,[28] and numerous other genes were responsible for the other 70% contribution. In another study, researchers found a total of 15 different genes that make major contributions to skin colour.[29]

Hitchhiker's thumb

'Hitchhiker's thumb' is the ability to bend the thumb significantly backwards to produce a large angle between the two bone segments (figure 1). The claim is that there are two kinds of thumbs—straight, where one cannot bend it backwards (dominant) and hitchhiker's thumb (recessive), a trait controlled by a single gene with two alleles. This idea was proposed by Glass and Kistler[30] in 1953, and the claim, which could have easily been evaluated in a large classroom of students, has been widely repeated by teachers since then.

At least two studies have falsified this myth. Harris and Joseph[31] used X-rays of 294 individuals to accurately measure the angle between the first and second bones of the thumb. Their analysis found a continuous distribution, and that most individuals had intermediate values, not a dichotomy as described in the myth. A similar study using a protractor held against the outside of the thumb to measure the thumb angle obtained a normal bell curve.[32] Although hitchhiker's thumb is often used to demonstrate Mendelian genetics, the extant data falsify the claim: thumbs don't fall into two discrete categories, and the trait is not controlled by a single gene.

PTC tasting

Small amounts of the compounds phenylthiocarbamide (PTC) or propylthiouracil (PTU) impregnated in paper strips are tasted by students to determine if the taste is very bitter or, for non-tasters, the only taste is the paper itself. I have used the test with hundreds of students when teaching a wide variety of science classes. The idea came from Du Pont chemist A.L. Fox, working with phenylthiocarbamide when a colleague complained about the bitter taste of the chemical dust.[33] Fox insisted it was tasteless so he asked his colleagues to taste the PTC, discovering that for some people it had a strong bitter taste, and others found it tasteless.[34]

Guo and Reed reviewed the PTC-tasting subject, citing 392 references. They concluded that how the test is administered makes a big difference in the results.[35] Early studies put PTC crystals directly on the tongue while others used solutions of PTC, or paper soaked in PTC, which is then dried.[36] When this factor is controlled, some people would be classified as tasters with one technique and non-tasters with the different technique.[37] Genetic analysis found linkage to DNA polymorphisms in 26 large families and much of the variation in PTC tasting was associated with chromosome 7 and a variation in chromosome 16, indicating several genes are involved.[38]

The common myth is the claim that only two kinds of people exist—tasters and non-tasters. The myth is that the

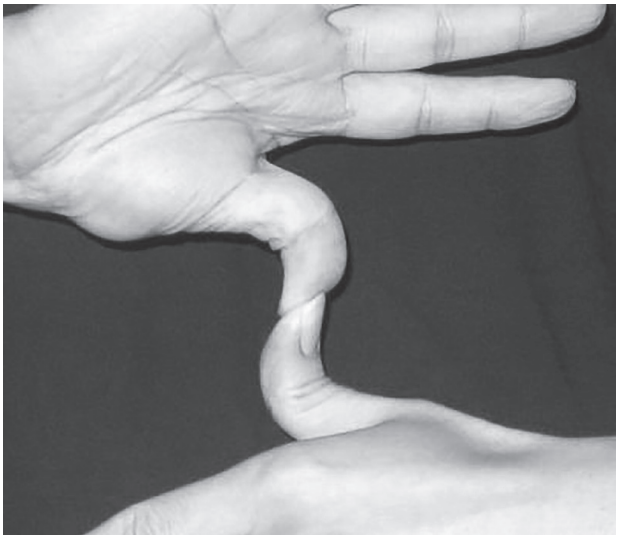

Image: Adrigon/CC BY-SA 3.0

Figure 1. A Hitchhiker's Thumb

Image: Gideon Tsang/CC BY-SA 2.0

Figure 2. Example of tongue rolling

taster trait is controlled by a single gene which is dominant over the allele for non-tasting. The fact is, after "almost 70 years, the origins of that variability, apparently due in large part to genetic factors, remains a conundrum".[39]

Tongue rolling

Some people can easily roll their tongue into a small tube (figure 2) while others can't. This is one of the most common traits that biology teachers use to demonstrate basic genetic principles that can easily be tested. The trait is attributed to *Drosophila* genetics pioneer Alfred Sturtevant's description of tongue rolling as a simple two-allele trait with the allele for rolling dominant, and non-rolling recessive.[40]

It has now been determined that the tongue-rolling skill is often not genetic, but learned. The fact that some people learn to roll their tongues after first being unable to is evidence that this trait is not the result of a simple genetic character as previously believed. Further evidence that the trait is not genetic is the finding that identical twins can have discordant tongue-rolling abilities.[41] The proportion of people who learn to roll their tongue ranges from 65 to 81%, and a slightly higher proportion of tongue rollers exists in females.[42]

The skill also exists on a continuum. Some people can only roll their tongue's edges slightly and cannot consistently be classified as rollers or non-rollers.[43] Even though numerous studies have shown that the skill is not genetic, but largely learned, tongue rolling remains a popular test for Mendelian genetics.[44] In short, the tongue-rolling myth has been debunked.

Widow's peak

Some people have a prominent V-shaped point at the centre front of their hairline called a widow's peak (figure 3), in contrast to a hairline that goes straight across. The allele for widow's peak is said to be dominant over the allele for straight hairline. Major problems with this claim include ambiguities about who has a widow's peak. Hairlines exist on a continuum requiring imprecise judgments to make this determination. One study of male medical students concluded that only 32 out of 1,039, or 3%, had a "slight but noticeable" widow's peak.[45] Another study of 360 women concluded 81% had a widow's peak.[46]

The problem of age also has to be considered. The hairline of many men and some women recedes over time, often more slowly in the middle, producing the widow's peak. It thus may be very difficult to distinguish between a receding hairline and a true widow's peak in adult men. The problem with the widow's peak example used to illustrate the basic genetics is the myth that it is controlled by one gene with two alleles, yet researchers have been unable to locate these genes. McDonald states he does not know how the myth began nor any experimental evidence that supports it in spite of a careful search of the scientific literature.[47]

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Figure 3. A prominent V-shaped point at the centre front of the hairline known as a widow's peak

Hand clasping

Most people have a strong preference for clasping their hands either with the left thumb on top or the right thumb on top. It also feels very unnatural to clasp the hands in the opposite way than one normally does. In one study roughly half of the people studied were right thumb on top and the other half were left thumb on top.[48] Since the first study on this topic was done over a century ago, no clear evidence has been found that supports the hand-clasping preference that fits the dominant-recessive myth.[49]

A review of nearly 100 publications that have surveyed hand-clasping frequencies in populations around the world found most populations had between 40 and 75% left on top and no preference for either was about 1%.[50] One hypothesis suggested left-handed persons, when folding their hands, strongly tended to put their left thumb on top

Figure 4. Young man with a cleft chin

and right-handed persons their right thumb on top.[51] Another hypothesis holds that preferences are probably chosen by each individual as a young child and reinforced over many years until the opposite thumb feels unnatural. No evidence has indicated the trait is genetic.

Cleft chin

Some people have a prominent dimple or crease in the chin's front called a cleft chin (figure 4). The claim is this trait is controlled by a single gene with two alleles, called the dominant cleft chin and recessive smooth-chin trait. The little genetic data available does not support this claim.[52] A major problem is that many chins are intermediate between cleft and smooth, and chins come in a variety of shapes, including round, dimples, vertical, and Y-shaped furrows.[53] Furthermore, a significant increase in cleft chin occurs with age; about 5% of boys 6 to 10 years old have cleft chins, while 10% of men over age 35 have a cleft chin. This change with age is also evidence against the simple genetic model. Weight gain and loss also influences the cleft chin trait.

Summary

The oversimplified evolutionary paradigm that evolutionists use to justify their worldview is not supported by human genome studies that consistently show much greater levels of complexity in producing these and other traits than once assumed. Actually, very few traits are the result of simple gene pairs, and, as more genetic information becomes available, the number of different genes that determine most features usually increases. Seemingly simple traits turn out to be very complex due to the genetic network interconnectivity of functioning in complex dynamic systems throughout the genome.

Applying the findings of this review of the one gene-one protein theory to the 2017 Gershoni and Pietrokovski study identifying 6,500 genes that produce human sexual dimorphism (and are, therefore, expressed differently in men and women), the results are not unexpected. Many of the traits which define sexual dimorphism that were long assumed to follow this one gene-one trait rule in fact do not follow it.[54] Consequently, the 20 major traits that are believed to make up human sexual dimorphism may actually be determined by several hundred genes, not 20. Another factor is genetic pleiotropy, the situation where one gene influences two or more, often many more, seemingly unrelated phenotypic traits.[55]

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