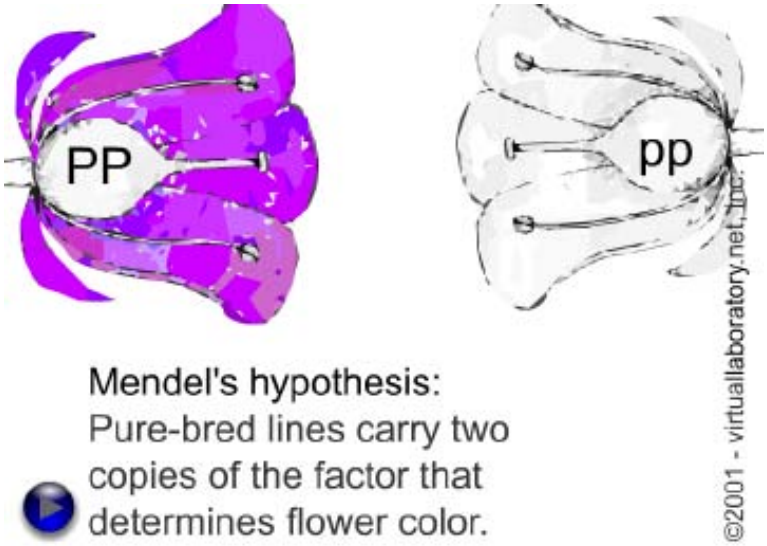
Mendel's Factors & Muller's mutations – page 1of 9

As you almost certainly already know (and can review here), in the 1800's Mendel deduced the existence of genetic factors (which we now know as genes).

These factors are passed unaltered from parent to offspring - but importantly, not all factors held by a parent are transmitted (note on simplifications).

Each parent has two copies of each gene, but one and only one copy is transmitted to any particular offspring. Which copy is transmitted is random (stochastic).



As you answer these questions, remember, there is rarely a single correct answer.

What did Mendel know about the physical nature of his factors?

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One of Mendel's major discoveries was that genes can exist in different forms, now known as **alleles**. Within a particular organism, there are two alleles of each gene; these alleles can be the same or different.

While there are at most two different alleles within a single organism, there can be many different alleles within a population. Typically, the most common alleles are known as "wild type".

An important question is, where did these various alleles come from? One hint came from the observation that new alleles can arise spontaneously - these new alleles are known (initially) as mutations.

Herman J. Muller won the Nobel prize in 1946 for his work showing that mutations (new alleles) could be generated by exposing organisms to radiation, specifically X-rays.

As you answer these questions, remember, there is rarely a single correct answer.

What does the ability of X-rays to generate mutations tell you about the physical nature of genes and gene products?

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Let us assume that each gene encodes a gene product (we are not concerned about what a gene product is or what it does). We can now consider all of the logically possible effects of a mutation. Compared to the original, or "wild type" gene product ("***wt***"), the mutant gene product ("***mut***") could be

- more active
- less active
- totally inactive or absent, or
- have a new activity

There are two more possibilities. The first arises because there are two copies of each gene in an organism – it is therefore possible that the mutated (***mut***) gene product interferes with the activity of the wild type mutant (***wt***) gene product. We will call this

- antagonistic

Finally, there could be mutations that produce no visible effect, we will call these

- neutral

As you can imagine, neutral mutations are difficult to identify or study.

As you answer this question, remember, there is rarely a single correct answer.

When a mutation in a gene occurs, the mutated gene product is most likely to be (compared to the original gene product)...

- ☐ neutral
- ☐ more active
- ☐ less active
- ☐ inactive or absent
- ☐ have a new activity
- ☐ interfere with the activity of the wild type gene product

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Please explain the logic of your answer.

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Now here is Muller's trick - a way of evaluating the activity of a gene without knowing what that activity is!

In the fruit fly *Drosophila melanogaster*, the organism that Muller worked with, it is possible to make deletions and duplications of genes. We can analyze their effects based on some (overly simplistic) assumptions:

- the deletion of a gene (which we note as Δ) leads to the complete absence of the gene product.
- the duplication of a gene (which we note as 2x) leads to twice the gene activity.

As you answer this question, remember, there is rarely a single correct answer.

If you know your molecular biology, you know that there are cases where these assumptions are not strictly true. Can you describe such situations.

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We will use deletions and duplication to compare the activity of the original version of the gene (***wt***) with the mutant version (***mut***). *Drosophila* is diploid, so there are (normally) two copies of each gene. We indicate our situation as

$\frac{wt}{wt}$ produces the original phenotype

$\frac{mut}{wt}$ produces the mutant phenotype, if ***mut*** is dominant

$\frac{mut}{mut}$ produces the mutant phenotype, if ***mut*** is recessive

For the moment, let us assume that ***mut*** is recessive for the trait we are looking at (remember, recessive and dominant are not absolutes; a mutation/allele can be recessive for one trait and dominant for another).

Now let us ask, is the phenotype of

$\frac{mut}{\Delta}$ the same, more, or less severe (extreme) than the phenotype of $\frac{mut}{mut}$?

If ***mut*/Δ** and ***mut*/*mut*** individuals have the same phenotype, Muller concluded that ***mut*** does not encode a functional gene product; either no product is produced or the product produced has no activity - he called such a mutation/allele **amorphic** (no activity).

If the phenotype of ***mut*/*mut*** is less severe than that of ***mut*/Δ**, Muller concluded that ***mut*** produced a gene product with the same, but less than normal activity - he called that a **hypomorphic** (low activity) allele.

As you answer these questions, remember, there is rarely a single correct answer.

In molecular terms, how might a mutation reduce but not eliminate or change the activity of a gene product?

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To extend our analysis, let us compare the "strength" of the trait expressed in organisms with the following genotypes. Remember we have been assuming that ***mut*** is recessive; we find that the phenotype of

mut	is more severe	***wt***
mut	than that of	**Δ**

If ***mut*** is dominant (with respect to the trait we are considering) then,

mut	is more severe	***mut***	which is more	***mut***
2x *wt*	than that of	***wt***	severe than	**Δ**

Muller concluded that ***mut*** produced either more of, or a more active version of the gene product than did the ***wt*** version of the gene. He termed such an allele **hypermorphic** (more function).

As you answer these questions, remember, there is rarely a single correct answer.

In molecular terms, how could a mutation increase, but not change the activity of a gene product?

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At this point, we have found ways to identify (using deletions and duplications of the gene under study), whether a mutation abolishes gene function, reduces it, or enhances it. But are there other possible effects of a mutation?

Muller found some.
Consider the following is the case. The trait is dominant and the phenotype of

$\frac{mut}{wt}$ is more severe than that of $\frac{mut}{2x\ wt}$

Muller called mutations that behaved in this way **antimorphic**, that is, the mutated gene product antagonized the function of the wild type gene product.

A related behavior can be observed with a recessive trait, in this case

$\frac{mut}{mut}$ is less severe than that of $\frac{2x\ mut}{wt}$

but this requires that we have a duplication of the mutant allele.

As you answer these questions, remember, there is rarely a single correct answer.

In molecular terms, how could a mutation produce an antimorphic gene product?

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Muller identified one final class of mutation, their behavior was described by the following relationship; the phenotype of

$\frac{mut}{mut}$ is the same or worse than $\frac{mut}{\Delta}$ but different from $\frac{wt}{wt}$

Remember, we are assuming that ***mut*** is recessive!
If ***mut*** is dominant then the phenotype of

$\frac{mut}{\Delta}$ is the same as $\frac{mut}{wt}$ which is the same as $\frac{mut}{2x\ wt}$

Muller called such mutations **neomorphic**; he assumed that the mutated gene product had a new function or activity, a function/activity that the original gene product did not have.

As you answer these questions, remember, there is rarely a single correct answer.

In molecular terms, how could a mutation produce a neomorphic gene product?

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At this point we have exhausted the universe of possible types of mutations. For any particular trait, influenced by a mutation, we can place the mutation in one of Muller's groups, or in the group with no effect, which we could call neutral or "normomorphic".

That said, remember we are talking about the mutation's effect on a single trait. The situation gets more complex when we recognize that many traits are influenced by multiple genes, and many genes influence multiple traits, but we will worry about that elsewhere.

As you answer these questions, remember, there is rarely a single correct answer.

How do mutations generate novel structures and behaviors?

Why might the random nature of mutations lead some people to reject biological evolution?

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