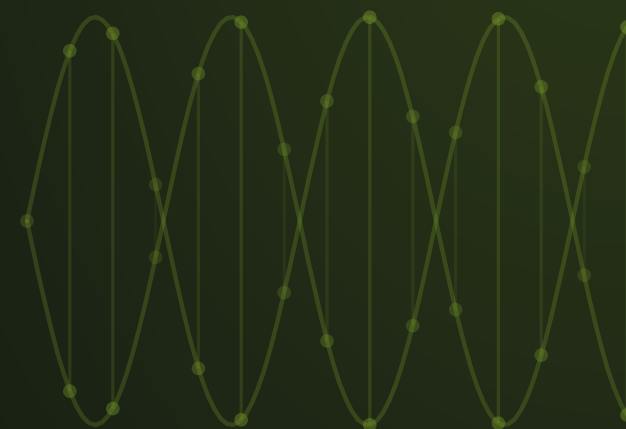


The Real Genetics Behind Spider-Man



Biology

Physics

Mathematics

Age 13+

Intermediate

Spider-Man is back in cinemas, so here is the honest version: what each of those abilities would actually require, and what the published research says about the three that turn out to be real — in people, right now.

Free to read, use and adapt. No account, nothing to buy, nothing collected from anyone.

Full lesson, figures and updates: <https://www.mygenelog.com/updates/the-genetics-of-spider-man>

The new film is in cinemas, which makes this a good week to ask a question the story never has to answer: what would each of those abilities actually require? Not whether it is possible — it obviously is not — but which part of the biology would have to be different, and whether anything like it exists in people.

Three of the four turn out to be real. One is ruled out by arithmetic that has nothing to do with genetics at all.

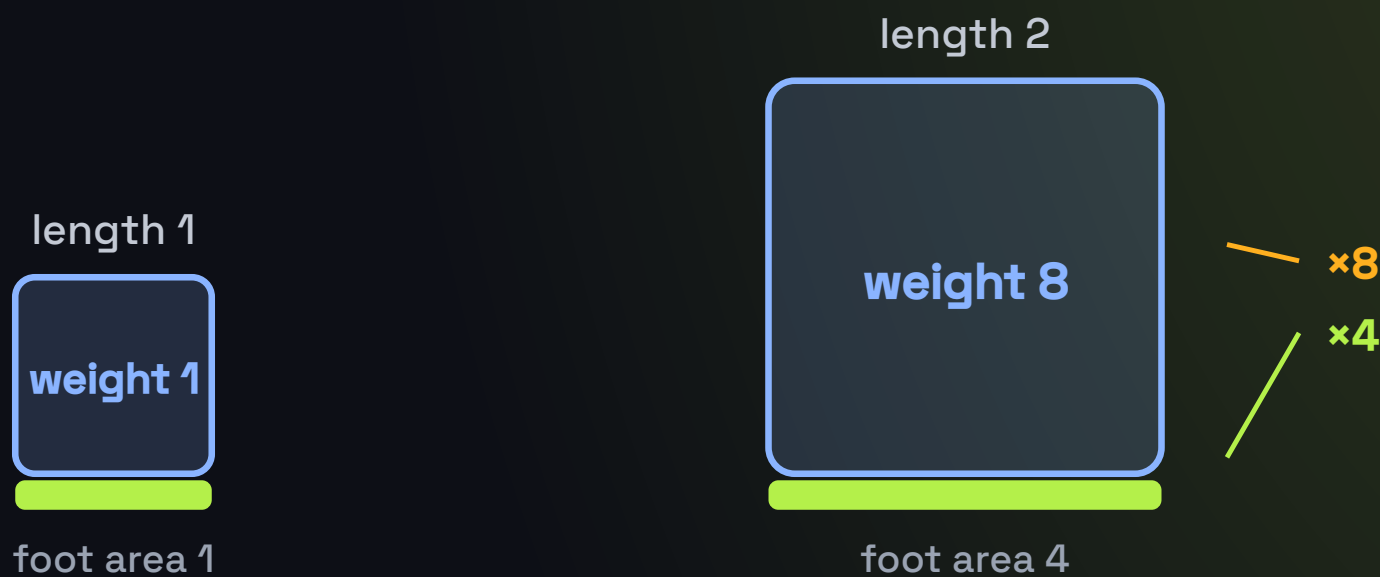
The wall-crawling is the impossible one, and not for the reason you think

The problem is not that humans lack a climbing gene. It is that adhesion is a surface, and a body is a volume.

Double an animal's length and its weight goes up eightfold, while the area of its feet goes up only fourfold. Anything that hangs on by surface contact has to devote a steadily larger share of its body to that surface as it gets bigger. A 2016 study in PNAS measured exactly this across 225 climbing species, from mites to geckos, spanning more than seven orders of magnitude in weight. Relative adhesive pad area rises roughly 200-fold across that range. The gecko is not the largest climber by coincidence; it is roughly where the strategy stops being possible.

A spider manages it easily because a spider is tiny. Scaling a spider to human weight does not scale up the trick — it destroys it. This is the rare case where the honest answer is not "we don't have that gene" but "no gene would help".

eight times the weight, four times the grip — each patch carries double



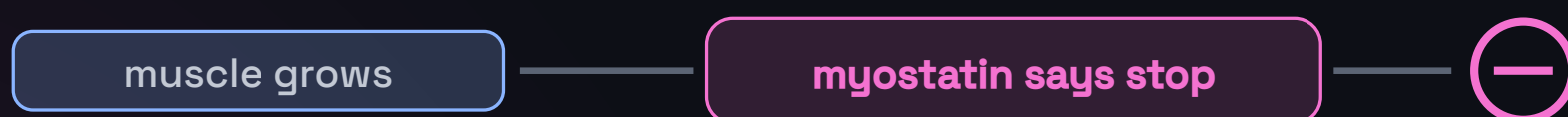
This is geometry, not biology. The gecko is not the largest climber by coincidence; it is roughly where the strategy stops working. A 2016 PNAS study measured this across 225 species.

The strength is real, and it is a missing brake

Muscle growth is not left to run freely. A protein called myostatin, made by the gene *MSTN*, actively limits it — a governor rather than an accelerator. Cattle breeds famous for their muscling carry loss-of-function changes in it, and so, it turns out, do some people.

In 2004 the New England Journal of Medicine described a child with loss-of-function changes in both copies of *MSTN*, with gross muscle hypertrophy apparent from birth. Not the result of an accident in a laboratory: an inherited absence of the thing that normally says stop.

usual



MSTN not working



Something removed, not something added. One gene's job is to limit muscle growth, and the 2004 NEJM report describes one child with neither copy working. One case is not a population, and a missing limiter is not a super-power.

Two cautions, both important. This is a single published case, and one case is not a population. And a missing brake is not a super-power in the comic sense — what it

produces is unusual muscle mass, not the ability to stop a train.

The everyday version of this question is ACTN3 rs1815739, the so-called sprinter gene, which really does affect fast-twitch muscle fibre and really is the most oversold result in consumer genetics. It is on this site with what the evidence supports and what it does not.

The spider bite leads somewhere real: the channels that decide pain

Here is the part the film gets accidentally right. Spider venom is not a random poison. It is a precision toolkit aimed at voltage-gated sodium channels — the proteins that decide whether a nerve fires at all. That is why a bite hurts, and it is why spider venom has become one of the most studied sources of new painkillers.

The channel most of that work targets is Nav1.7, made by the gene SCN9A. And the reason it is targeted is a genetic finding, not a chemical one. In 2006, Nature reported three consanguineous families in northern Pakistan in which children could not feel pain at all — no injury, no burn, nothing — and mapped it to loss of function in SCN9A. Everything else about their nervous systems was intact. Take one channel away and pain stops existing, while touch, temperature and thought carry on.

The reverse is also known: gain-of-function changes in the same gene cause conditions in which people burn with pain from ordinary warmth.

So a spider bite genuinely does reach into human pain genetics — just not the way the story tells it. A 2024 paper in Advanced Science works through the current problem with turning spider-venom peptides into medicines, which is selectivity: hitting Nav1.7 without hitting the channels that run the heart and the muscles is the whole difficulty, and it has not been solved yet.



The selectivity problem. A molecule shaped to fit one tends to fit its relatives, and the relatives include the one running the heartbeat. That makes it a safety problem rather than a potency one, and it is not solved.

The radiation is the part that is exactly backwards

Radiation does not write instructions. It breaks them.

What ionising radiation does to DNA is snap strands — including double-strand breaks, the kind that are hard to repair correctly. Cells carry elaborate machinery to find those breaks and rejoin them, and the reason that machinery is elaborate is that the alternative is a mistake in the repair. That is a mechanism for cancer, not for climbing.

The genetics that follows from this is real and has nothing heroic about it: inherited faults in the repair machinery itself. They are why some families carry markedly higher cancer risk, and why radiation dose is treated with the care it is.

A radioactive spider is, biologically, an unusually specific way of damaging someone.

Why the honest version is the better story

Strip the film out and what is left is not nothing. It is a gene whose whole job is to limit muscle, and people who inherited it switched off. It is one channel out of many whose absence deletes an entire category of human experience. It is a size limit written into the physics of sticking to things, which no amount of biology can argue with.

None of it is a reason to have your genome tested this week. All of it is on public record, with the papers, and most of it is stranger than the version with the costume.

Using this in a classroom

Free to use and adapt in any school, college or university. Nothing to buy, no account, and we collect nothing from anyone who uses it.

- Level: about age 14 through first-year undergraduate.
- Time: 30–45 minutes as a discussion, or set the article as reading and use the questions in the following lesson.
- You need: nothing. No equipment, no genetic testing, no personal data.
- It covers: the square–cube law and why scaling arguments beat genetic ones sometimes; loss of function versus gain of function; why one published case is not a population; what a drug target is; and what ionising radiation actually does to DNA.

Warm up

1. Before reading: which of the four abilities — wall-crawling, strength, the bite, the radiation — would you guess is the least possible? Write it down. Compare after.
2. The article says the wall-crawling problem is not a missing gene. What is it instead, in one sentence of your own?
3. Myostatin is described as a brake rather than an accelerator. What is the difference, and why does it matter for what breaking the gene does?

Core

1. Double an animal's length: weight goes up eightfold, foot area only fourfold. Work out what happens at ten times the length. Then say why the gecko is roughly where the strategy stops.
2. The myostatin finding is one child. List three things you cannot conclude from a single case, and one thing you can.
3. People with loss of function in SCN9A feel no pain, while touch, temperature and thought are unaffected. What does that tell you about how the nervous system is organised that a general painkiller would not?
4. Spider venom targets sodium channels, and Nav1.7 is a painkiller target because of a genetic finding. Explain the route from "some families feel no pain" to "a company is trying to make a drug" — each step, in order.
5. The article says the selectivity problem is unsolved: you must hit Nav1.7 without hitting the channels that run the heart. Why is that hard, given how similar those proteins are?

To stretch

1. "Radiation does not write instructions, it breaks them." Using that, explain why the origin story is not merely unlikely but backwards — and name the real genetics it points at.
2. Losing pain entirely turns out to be dangerous rather than convenient. Say why, then decide what a good painkiller should actually do differently from what SCN9A loss does.
3. Three of these four claims are supported by published work and one is refused on physics.

Which kind of argument is harder to overturn, and why? Use the article's own examples.

4. Find the four PMIDs at the end of the article and look one up. Does the abstract say what the article says it says? Report any difference — that is the exercise.

Notes for whoever is teaching — where these questions are going

4. At ten times the length, weight is 1,000x and area 100x, so the load per unit of sticking surface is ten times worse. The measured version is in the paper: relative pad area rises about 200-fold across the 225 species studied. The point students should reach is that this is geometry, so no amount of biology negotiates with it.

5. Cannot conclude: how common it is, whether it is harmless, whether the effect size generalises, whether there were other contributing factors. Can conclude: that losing myostatin function in a human is compatible with life and produces muscle hypertrophy — which is exactly what the animal work predicted, and that agreement is why one case was worth publishing.

6. That pain is a separate channel rather than an intensity of touch. A general anaesthetic removes everything; this removes one modality and leaves the rest. That separability is what makes a selective drug conceivable at all.

7. Observation in families → map to a gene → identify the protein it makes → confirm the protein's role → look for something that blocks that protein → find spider venom already does. Students often skip the confirmation step; that is the one worth naming.

8. There are nine closely related human sodium channels built from the same plan. A molecule shaped to fit one tends to fit its relatives, and the relatives include the ones running the heartbeat, which is why this is a safety problem and not a potency problem.

10. People who cannot feel pain injure themselves without noticing — fractures, burns, joint damage, and a shortened life. So the goal is not "no pain" but "less of the pain that has stopped being useful", which is a much harder specification to write.

12. The intended answer is that the abstracts do support the article. The real lesson is that they checked, and that checking is available to them: all four are indexed and the abstracts are free to read.

Nothing here needs anyone's genetic data, and no part of this lesson involves testing a student or asking about their family. It runs entirely on published research and a film.

Sources: adhesive pad scaling — Labonte et al., PNAS 2016, PMID 26787862. Myostatin — Schuelke et al., NEJM 2004, PMID 15215484. SCN9A and congenital insensitivity to

pain — Cox et al., Nature 2006, PMID 17167479. Spider-venom peptides as Nav1.7 inhibitors — Luo et al., Advanced Science 2024, PMID 39248322. This post is educational and is not medical advice.