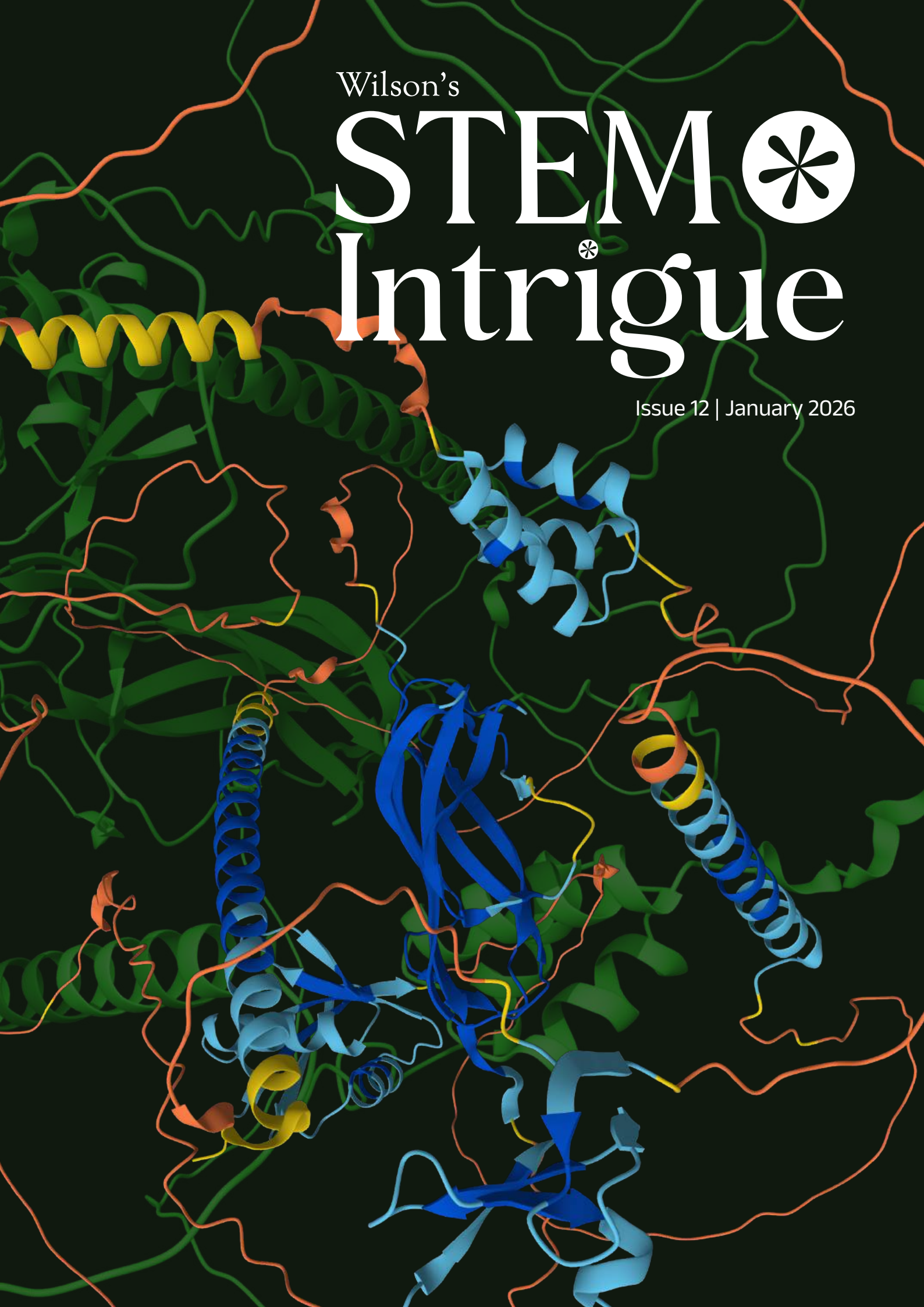

Wilson's

# STEM Intrigue

Issue 12 | January 2026

Cover design by Oscar Wong

Protein image from AlphaFold (Next to BRCA1 gene 1 protein):

Jumper, J et al. Highly accurate protein structure prediction with AlphaFold. Nature (2021)

Fleming J. et al. AlphaFold Protein Structure Database and 3D-Beacons: New Data and Capabilities. Journal of Molecular Biology, (2025)

# Editor's Foreword

The Intrigue Team is proud to present you a new issue of the Wilson's Intrigue to start of the new year, filled with incredible articles from budding scientists ranging from Y11 to Y13. Entirely independent and student-run, the magazine is made possible by the dedication of writers and editors who devote significant time to researching, refining, and making science more accessible, engaging, and inspiring for the Wilson's community. We are also deeply grateful to the staff who support the magazine's growth, and we look forward to seeing how future editorial teams continue to help Intrigue reach new heights.

Our Mission:
* Expand your knowledge
* Contribute to the Wilson's community
* Make complicated parts of science more accessible
* Popularise science and make it more interesting
* Inspire creativity through wider research

Acknowledgements:
This issue would simply not be possible without the perseverance of the writers and editors, skilfully balancing their school and super-curricular explorations. Their intrigue for STEM and enthusiasm to share their research are the fundamental pillars of the magazine. A massive thank you to all students involved for their contributions! A special thanks must go to all the teachers that have made the production and publishing of the magazine possible. If you would like to write in the thirteenth issue of the STEM magazine to indulge in researching and sharing a STEM curiosity, please email Oscar at WONGO@wilsonsschool.sutton.sch.uk or Quang at TRANQ@wilsonsschool.sutton.sch.uk for more information.

**Oscar Wong and Quang Tran**
Chief Editors 2025/6

## Thank you to all the authors and the editing team!

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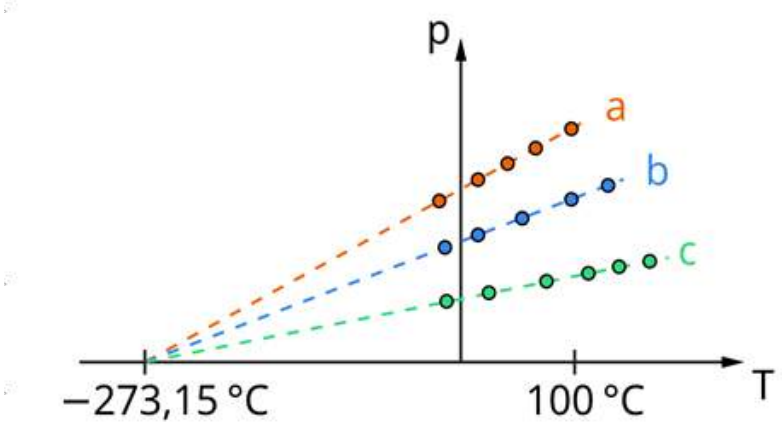
# MATHS

# Where do you draw the line?: Linear regression and correlation coefficients

Written by Egemen Oztop | Edited by Emad Rehman

There is one key skill that ties all STEM subjects together - making sense of data. A fundamental technique to help with this is plotting data points and then drawing an appropriate line of best fit - a skill you would have been initially exposed to in Year 7 science lessons. These lines not only allow us to easily visualise relationships between variables that might not be identifiable at first glance but even make predictions by extrapolating beyond the data range.

For example, by measuring the pressure of different gases at different temperatures, we can plot several lines, as shown in Figure 1. Extrapolating these lines back to where the pressure equals zero allows us to ascertain the value of absolute zero (roughly -273°C). The conditions of absolute zero are a theoretical limit and are therefore impossible to recreate in a lab - yet the value can be confidently determined thanks to these lines of best fit.

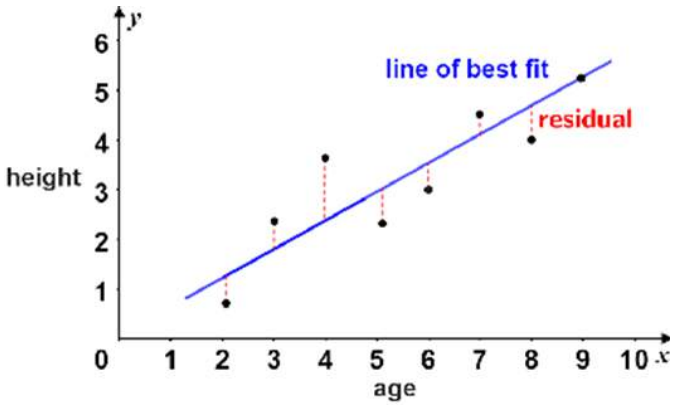



(Figure 1)

Drawing a line of best fit can sometimes feel like an art. The very name suggests that some lines are a "better" fit than others, which raises a critical question: how can we define a line of best fit mathematically? What actually makes one line the *best*?

A simple approach is to draw a line so that roughly the same number of data points lie above and below it and then estimate a reasonable gradient. However, there must be a more precise method - especially given that software such as Excel can generate a line of best fit almost instantly. The answer takes us from judging by eye to a more rigorous, algorithmic approach.

Intuitively, the goal is for the line of best fit to lie as close to the data points as possible. We can express this mathematically by looking at the vertical distance between each point and the proposed line, known as the residual (shown in red in Figure 2).




(Figure 2)

Then, it would be sensible to minimise the sum of these residuals by repeatedly proposing different lines, by slightly varying the parameters that define a straight line (that is, changing the value of $B_0$ and $B_1$ in an equation of the form $y = B_0 + B_1x$). This is the framework underlying linear regression analysis - defining the most suitable linear relationship between an independent and a dependent variable.

However, there is a significant flaw in the approach we have taken so far. If we simply sum the raw residual values, the positive and negative distances can cancel each other out. As a result, a line that is still far from the data points could end up with a total residual of zero, which is clearly an unsatisfactory outcome.

The solution? The least squares method, first developed in the early 19th century and accompanied by a rather interesting controversy. The technique was first published in 1805 by the French mathematician Legendre. A few years later, in 1809, the renowned mathematician Gauss presented the same method in his own work, claiming he had actually discovered it earlier, in 1795. Today, Gauss is often the one credited.

Notably, both Gauss and Legendre used this method to model and predict the orbits of comets far more accurately than had ever been possible before.

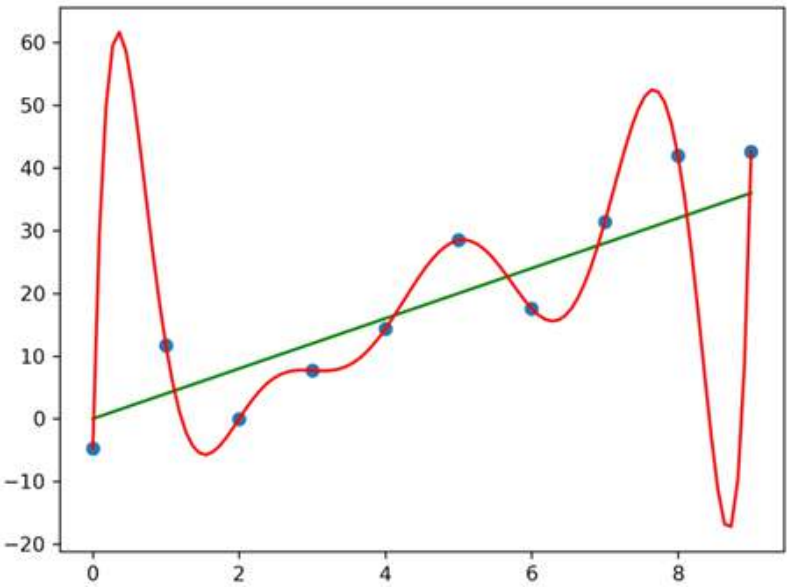
The least squares method works by minimising the sum of the squares of the residuals. Squaring the residuals removes their signs — similar to taking an absolute value — but has the added advantage of giving larger errors greater weight. This means that significant anomalies influence the result more strongly, allowing the final line of best fit to account for them more effectively. Thanks to these benefits, and despite the historical dispute surrounding its origin, linear regression now lies at the heart of modern statistics.

Of course, the assumption underpinning our reasoning so far is that there is a sensible linear relationship between our two variables. The method would fail for a clear quadratic or higher-degree relationship; thus, we can simply add more parameters, creating a polynomial of the form $y = B_0 + B_1x + B_2x^2 + B_3x^3 + \ldots + B_nx^n$. Similar to linear regression, varying these parameters by random amounts to minimise the sum of the squares of the residuals creates a new line of best fit achieved through polynomial regression.

In some cases, polynomial regression is more suitable than linear models, as it can describe higher-degree relationships more accurately. However, paradoxically, if the proposed polynomial line of best fit passes through every single data point, this could in fact be even more unreliable than a linear model. Fitting a dataset perfectly is characteristic of overfitting, as demonstrated in Figure 3. The red polynomial does fit the provided data more closely than the green line of best fit, but this comes at the cost of having misleading suggestions for values between the data points, and it is therefore unsuitable for interpolation.

To conclude, the statistical techniques we've explored in this article have evolved to form the core of machine learning. Those advanced systems behind fascinating AI models operate on the same principle of finding a 'best fit', except on a much larger scale. For example, consider large language models - predicting the next word in a sentence isn't magic, but instead can be modelled as a complex equation of the different words that precede it. So the next time you draw a line of best fit, it's worth remembering that the very same techniques that you are subconsciously applying can help us to make sense of our increasingly complex world.

**"How can we define a line of best fit mathematically? What actually makes one line the best?"**




(Figure 3)

# The maths behind AI and how it's causing the microchip revolution

Written by Soham Madnurkar | Edited by Mark Meng

AI feels like magic — being able to generate complete essays, applications, and nowadays even films. It feels as though the software has developed and advanced so rapidly that perhaps the hardware it runs on cannot keep up with this extreme rate of progress. However, new hardware is also being developed to embrace and accommodate the AI revolution, pushing the boundaries of what is possible (and practical) for us to do with AI.

Let us break down what feels like magic into its basic form: mathematical equations and silicon chips.

### What is AI?

Simply speaking, artificial intelligence is when a machine can perform tasks that typically require human intelligence. However, when I refer to AI in this article, I am referring to a specific subfield of AI called machine learning. Machine learning is when an algorithm can improve its performance at a task through experience, without being explicitly programmed to do so.

### How does machine learning work?

Machine learning is implemented through a computer programme called a model. A model comprises two parts: the model architecture and the weights. The model architecture refers to the type of programme used for the machine learning model — the process used to convert inputs into outputs. The weights refer to the data held specifically by the model, which make it specialised at what it does; they are the numbers and multipliers used within the architecture to convert inputs into outputs.

It is these weights we try to optimise through machine learning so that we can accurately predict the output given the input. Let's first take a simplistic approach to how this is done.

Each set of data is passed into a machine learning algorithm as a feature vector. Take, for example, information about a house, where x is a feature vector that represents one house:

```
x = [square footage, bedrooms, bathrooms]
e.g. x = [3000, 3, 2]
```

If the weights for this algorithm were:

```
w = [1000, 20000, 10000]
```

That would mean that for each square foot of the property, £1000 is added to the property value; for each bedroom, £20,000 is added; and for each bathroom, £10,000 is added. While this is a very simplified example, the general mechanism by which machine learning models make predictions involves converting all data into arrays and multiplying input values by the weights, which represent how much each input should contribute to the final prediction.

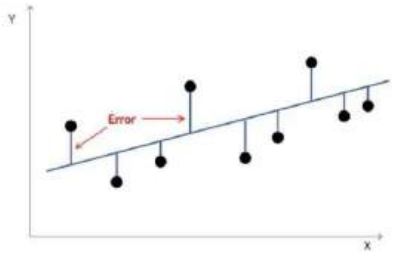
After this, a gradient descent function is used to adjust the weights to fine-tune the model (explored later), and the process is repeated on all of the data again with the new weights.

Now imagine there are millions of data items, and thus millions of feature vectors, with thousands of parameters in each feature vector — quite common in complex AI tasks. The data is no longer just a vector, but a matrix built up of all the feature vectors stacked together. Often, the weight vector also becomes a multidimensional matrix, especially when neural networks with multiple hidden layers are involved.

Hopefully, this gives you a sense of the scale of computation required in real-life problems, where an abundance of data is available, collected, and used in machine learning. Some very modern, powerful CPUs may be able to cope with the workload of performing billions of simple multiplications, but CPUs are inherently not the best devices for the task due to the massive parallelism required for speed and efficiency. I will discuss the hardware best suited to this task later in the article.

### Calculating the loss

After this step is completed, and the prediction based on each individual feature vector is calculated, the loss of each data value is compiled.




The loss refers to how far from the actual values the predicted values were, and all the losses are averaged to find the aggregate loss — how wrong the algorithm is on average. After this, each weight is individually nudged slightly in the direction that minimises aggregate loss, and the following equation is applied:

$$w_{\text{new}} = w_{\text{old}} - \eta \frac{\Delta L}{\Delta w}$$

This is known as the gradient descent optimisation formula. Here, w refers to the weights, L refers to the loss, and η (eta) refers to the learning rate. The last part of the equation — the change in loss divided by the change in weight — shows how much impact the weight would have had on the aggregate loss if it were closer to the optimum value. This gives an indication of how much we should change the weight to reach that optimum.

The old weight is incremented by the change in loss over change in weight, multiplied by the learning rate. The learning rate is a very small multiplier

used to ensure the model doesn't change itself too quickly and doesn't over-train on anomalous data.

This process of performing matrix multiplication and gradient descent is carried out many times until the model has trained itself on the data. The extremely small multipliers used to guarantee accuracy often cause this process to take much longer than expected. Hopefully, this, again, gives you an idea of the scale of computation necessary to train the model on all of this data.

## The architecture and working of semiconductor chips

A semiconductor — as the name suggests — has a level of conductivity that lies between that of metallic conductors and insulators. In conductors, electrons are completely free to move and conduct charge, while in insulating materials, electrons are bound to atoms and cannot move at all. However, in semiconducting materials, the electrons sit at a borderline between these two, allowing us to control whether or not they move by fine tuning the number of electrons in a transistor. While in a conductor a current would be transmitted at any voltage, and an insulator wouldn't allow a current to go through at any voltage, a semiconductor is fine tuned so that current can only pass through if current is above or below a certain threshold.

For this reason, silicon semiconductors are used in computers to make transistors. In a transistor, we can control whether or not there is a current due to its semiconducting properties, which can be converted into ones and zeroes so that data can be transmitted in the form that computers understand — binary.

These tiny transistors can be used to create logic gates, which can in turn work together to create separate processing units to be placed on a chip, each specialised for a different task. In the classic CPU architecture, this involves having separate memory units and only one arithmetic unit per core. The problem with this is that it is designed to perform one very complex operation at a time, with a large number of transistors connected together to form complex circuits, meaning that while complex operations can take place, only very few operations can take place at a time. Therefore, even the fastest CPUs, with perhaps a few dozen cores, simply cannot keep up with the volume of parallel computation demanded.

Understandably, this can be very problematic for the extensive matrix multiplications and other matrix operations involved in AI. A component is needed that can perform many very simple arithmetic operations efficiently and at very high speed. Conveniently, such a unit already exists.

Originally designed for processing graphics, the similarity of the matrix operations involved makes GPUs very suitable for AI operations, since they have thousands of cores and can handle parallelism extremely well — which is almost a necessity for AI processing. However, even GPUs are still limited. They draw massive amounts of power and cause memory bottlenecks, as the GPU cannot feed data to its cores fast enough.

## Solving hardware limitations

To solve these problems, many new chip architectures are being developed or have already been developed.

First of all, GPUs have very high power consumption. This is mainly due to the extremely high number of cores constantly drawing energy for their consistently heavy workload, which also increases the energy requirements for cooling. This can be seen even now, where modern data centres use millions of litres of water for cooling, and the servers themselves draw large amounts of power.

One modern solution to this is the use of neuromorphic chips. While all cores in a GPU constantly process data — even data another core may already be processing — neuromorphic chips imitate the efficiency of the brain and only trigger a neuron when there is a task to be done. This means that cores with no work to do remain idle and do not consume energy, rather than splitting a task between cores unnecessarily. This reduces the total amount of power drawn by chips and increases their energy efficiency.

GPUs also cause memory bottlenecks. This occurs when GPUs can process data faster than they can access it. GPUs do not have large enough memory to store the gigabytes of data needed to train a machine learning model, so data has to be moved to and from VRAM, which is significantly slower.

To solve this, new chips are being created that can process data in memory itself, so that data does not need to be moved into and out of memory. This drastic reduction in data movement reduces energy consumption and increases maximum processing speed, allowing more complex algorithms to be run and enabling systems to cope with heavy AI models.

Another chip designed specifically for machine learning operations is a tensor processing unit (TPU). TPUs use specialised structures known as systolic arrays, which move data through the chip in carefully timed patterns that maximise reuse and minimise energy consumption. By integrating the processing operations with memory management, TPUs are able to achieve far higher efficiency than general-purpose GPUs in large-scale machine learning.

Overall, the rapid progress in both AI algorithms and specialised hardware shows that the two evolve hand in hand. What appears complex or mysterious ultimately comes down to mathematics executed at massive scale, supported by increasingly advanced silicon.

**"NEW HARDWARE IS ALSO BEING DEVELOPED TO EMBRACE AND ACCOMMODATE THE AI REVOLUTION, PUSHING THE BOUNDARIES OF WHAT IS POSSIBLE (AND PRACTICAL) FOR US TO DO WITH AI."**

# Principles of Linear Regression, and applications

Written by Houting Li | Edited by Mark Meng

**Abstract:**

Linear regression is one of the most important machine learning methods. It has wide applications in medicine, engineering, and maths. In this article, I explain the principles and the theory background of this method. Based on a two-variable linear regression model, I derived the least square estimation for the regression model. Using house price estimation as an example, I show how to use the method for predicting the house price in 2030. Finally, I introduce the extension of the method for other studies.

## Introduction

### Background:

In many situations, we would like to predict the future outcome based on present and historical datasets. For example, we would like to predict house prices in 2030 based on the market data we have today. Mathematically, this is a regression problem. There are many statistical methods that can be used to address this kind of problem. I would like to introduce linear regression to answer this question.

As an example, we plot a set of points on a graph (Figure 1). Assume that the x-axis is the time, and each small square represents one year, and that the y-axis is the house price (£/ft²). The question is to predict the house price in 2030. Mathematically, we need to draw a line of best fit through the points (red line in Figure 2), called model fitting in machine learning literature.

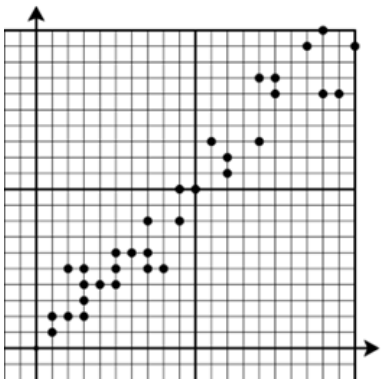


Figure 1

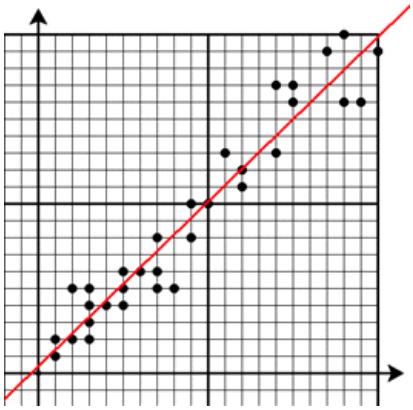


Figure 2

Now, you may ask, how did I calculate the line of best fit? Here, the best fit is the minimum distance between the dots and the line.

## Methods

To tackle this problem, we need to define what a line of best fit is. In mathematical terms, the best is the 2-norm or Euclidean norm of the distance (Dokmanic et al., 2015). By observation, it is sufficient to fit the dot into a line function. In reality, we can try different functions for the fitting, but here it is the line that results in the sum of the squared vertical distances from the points to the line being minimised. We assume and define this (red line in Figure 2) line equation as:

$$g(x) = \alpha x + \beta \quad (1)$$

where x is the independent variable (feature, in machine learning literature), and y is the function of g(x), which is the dependent variable. α is the slope, and β is the associated intercept.

To calculate the distance between the dot and the line, we define the L2 distance, which is the sum of the squares, as:

$$F(\alpha,\beta) = \sum_i (\alpha x_i + \beta - y_i)^2 \quad (2)$$

where i is the index and $x_i$, $y_i$ is the collected data at time point. F(α,β) takes the form of a quadric function, which can be a concave or convex function. To find the optimal α and β which minimise the objective or loss function (2), we usually need to use numerical methods. From calculus, we know that the **necessary but not sufficient** condition for a function to reach maximum or minimum value is the first order derivative equal to 0 (Stewart, 2016). Particularly, to find α and β (in terms of x and y) that minimises the sum of the squares, we differentiate the equation with respect to both α and β, and set both differentials equal to 0, e.g.,:

$$\frac{\partial F}{\partial \alpha} = 0, \frac{\partial F}{\partial \beta} = 0 \quad (3)$$

which leads to:

$$\begin{Bmatrix} \sum_i 2x_i(\alpha x_i + \beta - y_i) \\ 2\sum_i (\alpha x_i + \beta - y_i) \end{Bmatrix} = 0 \quad (4)$$

Now it's a matter of solving simultaneous equations. Eventually, the following for α and β is retrieved:

$$\alpha = \frac{n\sum_i x_i y_i - \sum_i x_i \sum_i y_i}{n\sum_i x_i^2 - (\sum_i x_i)^2} \quad (5)$$

$$\beta = \frac{\sum_i y_i - \alpha \sum_i x_i}{n} \quad (6)$$

where n is the number of plotted points, and ($x_i$,$y_i$) is the data(dots).

Based on equations (5), (6), and (1), we can make inference of the unseen data.

## Results

As an example, we use the Python Sci-learn toolbox to implement the method for predicting the house price. The idea is to estimate the house price in 2030, based on the historical data. In this example, the house price data in 2000, 2005, 2010, 2015, and 2020 were obtained from UK land registration. The average house prices in these years were £160000, £230000, £260000, £288000, and £362400, respectively. Below is the Python code (version 3) to implement the method.

```
import numpy as np
import matplotlib.pyplot as plt
import matplotlib.ticker as mticker
from sklearn.linear_model import
LinearRegression
```

```
# Example data
# X = house price (£), the data was
not accurate, as the source is not
reliable.
X = np.array([2000, 2005, 2010,
2015, 2020]).reshape(-1, 1)
# y = price (£)
y = np.array([160000, 230000,
260000, 288000, 362400])

# Create model
model = LinearRegression()
model.fit(X, y)

print("Slope:", model.coef_[0])
print("Intercept:", model.
intercept_)
print("Price for 2030 year:",
model.predict([[2030]])[0])
m,b    = model.coef_[0], model.
intercept_
y_pred = m * X + b

# Plot
plt.scatter(X, y,color='red')
plt.plot(X, y_pred)
plt.gca().xaxis.set_major_
locator(mticker.MultipleLocator(1))
plt.grid()
plt.xlabel("X")
plt.ylabel("y(£)")
plt.title("Simple Linear Regression
for house price in Sutton")
plt.show()
```

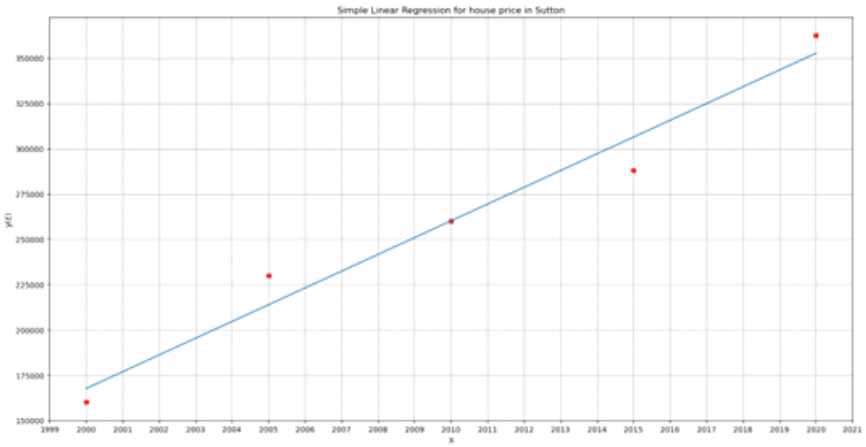



Figure 3. An example of house price estimation using linear regression. X-axis is the time (year), and y-axis is the house price (£). The dot is the sample time point with the house price, and the blue line is the final fitting results.

By running the Python code, Figure 3 was generated, and the red dot represents the house price at the specified time. The blue line is the final fitting results. From the code, we estimate that the slope (α) is 9256.0, and the intercept (β) -18344480.0. Putting 2030 into equation (1), we have the house price for 2030 year, that is, £445200.00.

## Discussion

There are many applications for these methods in artificial intelligence. For example, universities use this to predict the number of students they will admit next year, and prepare for the number of classrooms for them in advance.

Machine learning methods can be classified into two categories. One is unsupervised, the other supervised, This requires a predetermined model (i.e., a linear equation in our case) (Géron, 2022).

Linear regression is often used as a supervised machine learning method, where machines are trained to predict new, unseen data (Mehryar et al.). In general, supervised machine learning methods, including linear regression, are trained in the following steps[1a]:

- 1. Determine the type of training samples
- 2. Gather a training set
- 3. Determine the input feature representation of the objective/ loss function.
- 4. Determine the structure of the objective/loss function and corresponding algorithm.
- 5. Complete the design
- 6. Accuracy evaluation

As the house price prediction example shows, the linear regression calculations are applied entirely in step 4 (steps 1-3 are for setting up the data to be ready for calculation, and steps 5 and 6 are for testing). In the linear regression example for house prices prediction, the training data is the historical house price data from 2000 to 2020, while the testing data, which is unseen, is 2030. It can be plotted as shown in Figure 3, because it has two variables which need to be estimated.

The points (x,y) are plotted onto a graph similar to the plots in the introduction (Figure 2). The points from vectors that represent the trend in regression should scatter approximately into a straight line respective to that category, under the condition that the points resemble a straight line to a sufficient extent (or non-linear functions will be considered). Then, these points can be used to calculate a line of best fit. For any new object analysed by the fitted model (equation 1). It is up to the machine learning designer to determine whether the point is "close" to a line of best fit, and to continually refine the algorithm to minimise the distance.

However, linear regression shows its weaknesses when we try to add complexities. For example, the calculated equations in the introduction only work for one independent variable. Often, pattern recognition will require detection of multiple features. Also, in some cases, linear regression will be inferior to other regression methods, such as curves. One extension of linear regression is logistic regression, which is a supervised classification model for many studies, including cancer detection. For more information, please see other references (Montgomery et al., 2021). Also, the idea of the method has been applied to non-linear regression (Seber, 2008)

In conclusion, it is without doubt that linear regression is a suitable tool in providing information for decision-making. Linear regression methods are effective in addressing some regression and classification problems. It is a useful method for predicting unseen data and has many practical applications. However, do not expect it to work as well in certain cases without difficult and complex workarounds to the limitations of being restricted into one shape.

# Behind the Game: The Silent Mathematics Powering Every Sport

Written by Atharv Bhatt | Edited by Shyam Ashokan

Sports - instinctive, fast-paced and unpredictable. It could be a game for both the elites and the everyday person like you and I, where every movement, kick or swing seems driven purely by talent or destiny. Yet underneath lies an entire system grounded in angles, physics, probability and geometry. As Galilelo Galilei famously said, "Mathematics is the language in which God has written the universe." (1) and today we'll explore this part of the universe: the overlooked laws guiding precision, performance and decision making in sports.

Football is one of the world's most beloved sports; every minute of play can feel like hours as you watch your team play. Whether it's Ronaldo's 40-yard screamer against Arsenal or Messi's solo goal against Getafe, one thing is certain - maths is always there. When Ronaldo lined up that free kick, he observed the defender's wall and made the critical decision. Don't shoot straight at it, but rather curve the ball around it. This is the concept of angle of incidence - the ideal angle that allows the ball to evade defenders and head towards the goal. The motion that follows produces a flawless parabolic path - typical of gravity-influenced projectile motion – slamming straight into the back of the net.

Footballers constantly work with vectors too - the magnitude of the shot (how hard the ball is hit) and direction (where it's aimed). To make the ball curve, they add spin, which creates a pressure imbalanced around the ball's surface through the **Magnus Effect** (2). As the rotating ball flies through the air, one side of travels with the airflow while the other moves against it, producing different air speeds and pressures. A perfectly executed freekick brings this idea to life - not just a moment of skill but an orchestra of angles, forces and motion working in harmony to cause the ball to swerve beautifully in the air. Any football fan can appreciate the elegance of that moment – but the real sophistication lies in the hidden maths. The player estimates the curve, height and distance and even if they don't think in terms of "parabolas" and "vectors", their instincts do the maths for them – balancing force and precision to silently guide the ball from boot to net.

Cricket is often described as a game of patience and skill, but beneath its calm exterior is a world shaped by probability, statistics and data. Every decision – whether to play an aggressive shot or set a certain fielding position – is anchored in maths. A batsman constantly weighs risk and reward: is the chance of hitting a boundary worth the risk of losing a wicket? This thought process is probability theory in action. Coaches and analysts rely on tools like batting averages, strike rates and run-rate projections to assess performance and predict the next outcome. Bowlers use data too - analysing weaknesses in a batsman's game and plan their deliveries with any identified patterns (3).

Shakuntala Devi, known as the "human computer" once said (4), "Without mathematics, there's nothing you can do. Everything around you is mathematics." In cricket, that couldn't be more true. The Duckworth-Lewis method, used when rain interrupts play, is a sophisticated mathematical model based on run expectancy and remaining resources. Analysts use probability distributions, standard deviations and predictive data modelling to refine team strategies and forecast match results. What fans see as instinct or luck is often the result of patterns, percentages and pure calculation. Mathematics doesn't just support cricket - it shapes it.

Basketball on the other hand is a mix of athleticism and geometry, where every shot demonstrates the beauty of parabolic motion. When a player attempts a jump shot, they subconsciously calculate the angle, speed and release height needed for the ball to follow the perfect arc into the hoop. This arc is the parabola – the natural trajectory of any object moving under gravity. Studies show that the best shooting angle is around 45 degrees (5), but varies depending on the distance between the player and the hoop, as well as the player's height. If the angle's too steep, the ball loses momentum; too flat and it hits the rim.

The symmetry and predictability of a basketball's flight reveals just how deeply geometry is integrated into the game. Even without writing down equations like $s = ut + \frac{1}{2}(at^2)$, players naturally adjust their strength and angle to create the perfect shot. Coaches and sports scientists use motion tracking and maths-based modelling to build upon this intuition (6), analysing every move – from release angles to spin rate and entry velocity. Every successful basket is more than just muscle memory; it's mathematics in motion, where theory becomes action and numbers turn into grace and rhythm.

Maths doesn't just sit in classrooms or textbooks – it moves across football stadiums, cricket fields and basketball courts. Every curving freekick, every tactical field change and every perfect jump is no coincidence. Angles, trajectories, probability and timing form the quiet foundation beneath instinct or talent. Sport does not stand apart from mathematics, it's so deeply entwined that it is one of its most human expressions. It shows that numbers aren't cold or distant, but alive in every goal, boundary and basket – with pattern precision and purpose.

"THE LAWS OF NATURE ARE BUT THE MATHEMATICAL THOUGHTS OF GOD." – EUCLID[7]

# The Rise of Chaos Mathematics in a Predictive World

Written by Shourya Gupta | Edited by Quang Tran

When most people think about mathematics, they picture certainty and precision, a world of exact answers and predictable outcomes. But there's a fascinating branch of math that turns this idea on its head. Chaos theory shows us that even when systems follow precise rules, they can behave in ways we simply can't predict over the long term[2,4]. And the implications? They're everywhere.

The story begins in the 1960s with a meteorologist named Edward Lorenz. He was running weather simulations on an early computer when something strange happened. He rounded off one tiny number in his calculations, expecting roughly the same result. Instead, he got a completely different weather pattern[1,5]. For a system that should be deterministic, meaning the same inputs always give the same outputs, this was mind-blowing. What Lorenz had stumbled upon was sensitivity to initial conditions: the idea that minuscule changes can snowball into massive differences down the line[4]. We know it better as the butterfly effect[3,7].

The name might sound whimsical, but the math behind it is solid. Chaotic systems are typically governed by nonlinear equations, which behave very differently from the linear ones we're used to[4,8]. Linear systems are predictable—double the input, you double the output. Nonlinear systems? Not so much. They can swing wildly, create unexpected patterns, and generally refuse to play by simple rules[8]. Yet despite appearing random, these systems often have hidden structure[6].

That's where Benoît Mandelbrot comes in. His work on fractals revealed that many irregular shapes in nature, such as coastlines, mountain ranges, and cloud formations, aren't actually chaotic in the sense of being completely random[6,8]. They follow patterns that repeat at different scales. Zoom in on a coastline, and you'll see similar jaggedness whether you're looking at a hundred miles or a hundred feet. The famous Mandelbrot set isn't just beautiful to look at; it's proof that deep structure can exist within apparent chaos, just not the kind of order that traditional geometry prepared us for[6].

Why does this matter? Because chaotic behavior shows up everywhere. Weather systems, brain activity, turbulent fluids, animal populations, traffic jams, and even financial markets all display these characteristics[4,8]. This doesn't mean we can't forecast anything—it means our predictions need to be probabilistic rather than absolute[8]. Scientists aren't trying to achieve perfect foresight anymore. Instead, they're working to understand how uncertainty grows over time and where the fundamental limits of prediction lie[7].

This shift in thinking has already started changing technology. AI researchers, for instance, are incorporating chaos theory into their algorithms. Traditional neural networks can get stuck in "local minima": dead-end solutions that aren't actually optimal. By adding controlled chaotic elements, these systems can explore more possibilities and find better solutions[4]. Chaos is a tool for flexibility and adaptation here.

The finance world has also taken notice. Stock prices don't move randomly, but they certainly don't follow neat, predictable patterns either[8]. They often contain brief, recurring structures buried in apparent noise. Chaos theory doesn't promise to predict the next market crash, but it does help set realistic expectations about what can and can't be forecast[4]. It also helps economists distinguish between different types of volatility, which changes are fundamental to the system and which are just temporary noise[8].

Beyond the practical applications, chaos theory raises some deep philosophical questions. It challenges the old Enlightenment idea that the universe is perfectly knowable if we just gather enough data[7]. Predictability is a spectrum. A system can follow exact rules (deterministic) while still being impossible to predict in practice, simply because tiny uncertainties multiply too quickly[1,4].

Far from diminishing mathematics, chaos theory actually expands what math can do. It gives us tools to study turbulence, uncertainty, and emergence, not as problems to be solved but as natural phenomena worth understanding on their own terms[6,8]. Chaos mathematics doesn't celebrate disorder for its own sake. It shows us that complexity has patterns, uncertainty has shape, and even when we can't predict exact outcomes, we can still understand the rules of the game[6].

As we navigate the twenty-first century with its climate uncertainties, algorithmic systems, financial instability, and increasingly connected infrastructure, understanding complex systems matters more than ever[7]. Chaos mathematics isn't about giving up on prediction. It's about being smarter about what we can and can't know. By recognizing where certainty ends, mathematics actually becomes more powerful. It learns to map not just the paths we can predict, but also the terrain where precision breaks down and we need to think differently about knowledge itself[8].

# PHYSICS

# Implications of supersymmetry in stabilising the Higg's Mass

Written by Nicolas Nanias | Edited by Gautham Subramanian

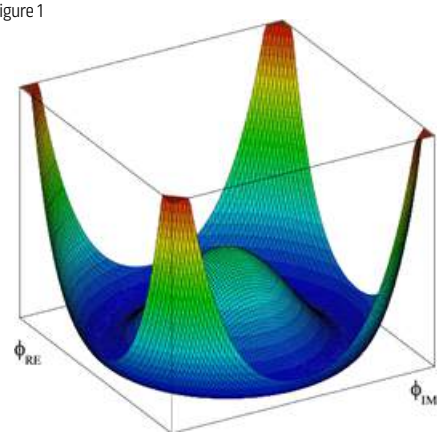
Our understanding of mass dates to the 17th century with Sir Isaac Newton in his Principia Mathematica. This was the first time in history where an explicit understanding of mass and its relation to phenomena such as gravity were first formally described. Within the next two centuries, our understanding was refined by Einstein to be synonymous with energy, and discoveries of sub-atomic matter developed the understanding to revolve around interactions of fundamental particles and fields. In 1964, Peter Higgs theorised the existence of the Higgs field, a scalar field which was responsible for assigning mass. By using the Klein Gordon equation (which can be derived by the associated field Lagrangian), we can describe how this Higgs field operates and interacts with other elementary particles.

$$(\Box + m^2)\phi = 0$$

$$\mathcal{L} = \frac{1}{2}(\partial_\mu \phi)(\partial^\mu \phi) + \mu^2\phi^2 - \lambda\phi^4$$

Assuming this derivation, we are primarily interested in the final two terms. These are responsible for describing the potential V(ϕ). Due to not being both positive, they characterise the Higgs to have a non-zero potential energy at ϕ=0. When we represent this graphically, we are able to see the characteristic Mexican hat potential of the Higgs:

Figure 1




The Higgs field is a fundamental field which permeates through all space and time. As a result, any fluctuation in the Higgs field will lead to a process known as Spontaneous Symmetry Breaking, this is when the field sets a non-zero vacuum expectation value.

Suppose we take a ball and place it on the top of a hill (representing a symmetric but unstable potential). Naturally, it will fall to the ground, breaking the symmetry of the process, gaining a lower potential energy which can be thought to be more stable. In the same sense as this, by building the characteristic Higgs field, all particles will interact with this field and be forced to take a lower, vacuum expectation energy. As a result of this, depending on the particle's coupling strength to the Higgs field, a different mass will be 'assigned' to the particle.

After the discovery of the 2012 Higgs Boson, and its mass of 125 GeV, a paradox was formed. If we were to look at the smallest possible mass of a black hole and compare them with the massive Z and W bosons, then we see a discrepancy of mass by a ratio of $10^{16}$, with no clear reason of why the Planck mass should be so large. Or why the Z and W bosons are so small in comparison. Without any new physics at play, quantum corrections should push up the Higgs mass to the Planck scale. But, as we can see in Figure 3, there is a slight peak at 125GeV, however the discovery of such a light particle seems unnatural.

$$M_{\mathrm{Pl}} = \sqrt{\frac{\hbar c}{G}} \approx 1.22 \times 10^{19}\ \mathrm{GeV}$$

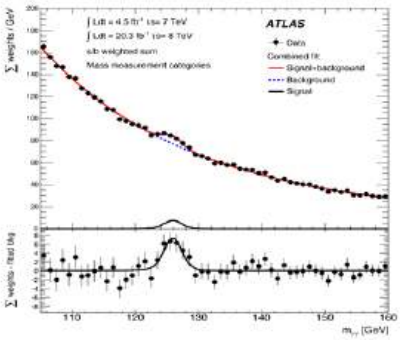



Figure 2: The 2012 discovery of the Higgs Boson by ATLAS. The significant peak at 125GeV, signals the mass.

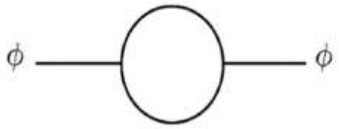



Figure 3: Quantum corrections to the Higgs mass. We can see the creation of virtual particles circling in the loop, which cause the mass to be 'dressed'.

One explanation involves the concept of quantum corrections. These arise from loop diagrams of Feynman graphs and can be calculated via perturbation theory - an idea where you manipulate the simplest interactions with the Higgs field and build up in complexity to find an approximate solution.

Now, to account for these quantum corrections, we have created a theoretical concept that the mass recorded is only a dressed mass.

$$m_H^2 = m_0^2 + \delta m_H^2$$

Where:

- $m_H$ is the physical (dressed) Higgs boson mass,
- $m_0$ is the bare mass parameter,
- $\delta m^2_H$ is the mass correction arising from quantum effects.

This brings us to the fine-tuning paradox. For us to have the outputted dressed mass to be around 125GeV, we require:

$$m_0^2 \approx -\delta m_H^2 + m_H^2$$

This represents an extraordinary fine-tuning, because there must be an extreme cancellation between the bare mass $m^2_0$ and the mass correction parameter $\delta m^2_H$, to leave a much smaller remainder on the order of (100 GeV)$^2$. Such precision appears extremely unnatural and suggests that if these constants were different even by $10^{-12}$ %, the universe would be extremely different, and life as it is may not even exist.

To address the problem of infinite contributions to the base mass arising from quantum corrections, a fundamental theorem of supersymmetry has risen. This theory assigns each particle in the standard model with a superpartner of a different spin. For example, an electron's superpartner is a boson known as the selectron.

top and stop quarks cancel each other's quantum corrections, hence reducing the net change to a negligible amount, successfully stabilising the Higgs mass at the electroweak scale without the need for fine-tuning theory!

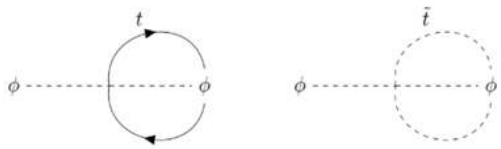



Figure 5: The Top and stop quarks cancel each other's quantum corrections.

Interestingly, this theory is also compatible with other unsolved problems, such as dark matter. Since we know particles can only decay into lighter fermions or barions, there is a potential for the Lightest Supersymmetric Partner (or LSP), to be a dark matter candidate. Crucially, the conservation of R parity - the number of fermions and barions in the decay – suggests that this LSP will remain stable. Since it is unable to decay, it will likely interact with minimal fields, sliding perfectly into our theoretical framework on dark matter.

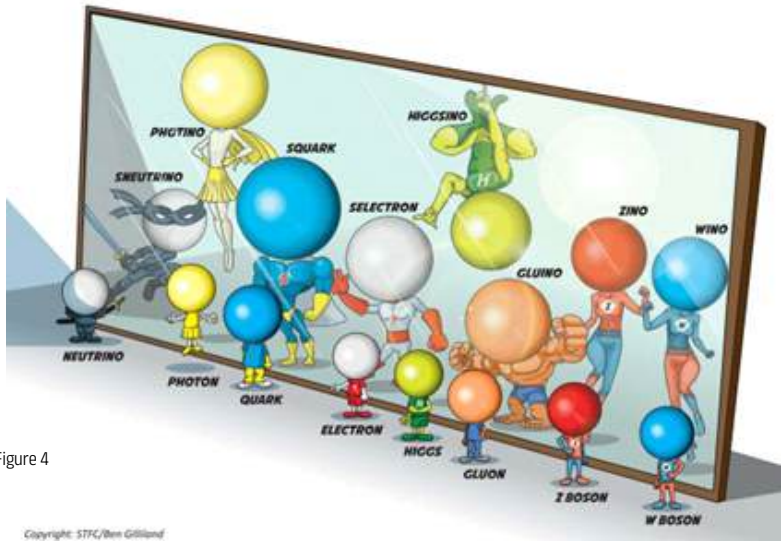



Figure 4



Supersymmetry provides a natural solution to the hierarchy problem by introducing a mechanism that cancels the large quantum corrections to the Higgs mass. In the Standard Model, particles like the top quark contribute large quadratically divergent loop corrections to the Higgs boson's squared mass. These corrections are proportional to the square of the ultraviolet cutoff scale Λ, which could be as high as the Planck scale. Essentially, by their interaction, from top quark interactions the mass of the Higgs should be on the order of the planck mass - hence, leading to an enormous contribution; supersymmetry addresses this by introducing a superpartner for every Standard Model particle. Each superpartner contributes loop corrections of the opposite sign due to their differing spin values. This cancellation can be seen in Figure 5, where the

**"Supersymmetry addresses this by introducing a superpartner for every Standard Model particle"**

# Advancing Silent Flight through Plasma Actuators

Written by Aadit Dixit | Edited by Quang Tran

The fact that we can make objects fly in the air, and that we can use this state of flight for many uses, such as travelling across the world, transporting goods, satellite imaging and so on, is an astounding feat. Yet scientists and engineers across the world are still constantly striving to improve the conditions of flight through vast research and testing of hypotheses; one way they are attempting to do so is by reducing the noise which comes along with flight. We have all surely heard the problems and complaints from residents who perhaps live near Heathrow, Gatwick or any other major airport in the UK about the noise pollution caused by constant flights in and around the area, which may cause difficulties for those who may work at home, or just want a state of peace when they are in their area. Along with this, the constant noise can also be inconsiderate to wildlife and other living creatures who are constantly exposed to the noise from flying objects; therefore, scientists have begun to look into ways to reduce noise caused by flight, and one of these ways is by using plasma actuators.

## What are Plasma Actuators?

Plasma Actuators are devices which are designed to convert electrical energy into airflow, and they do so without any moving parts, as will be explained later (Enloe et al., 2004). Most plasma actuators which are used are Dielectric Barrier Discharge (DBD) plasma actuators, and these actuators take a specific arrangement, which will also be explained later. These are being developed constantly and have undergone a significant improvement in their properties and efficiency over the past two decades.

## How do they work?

Plasma Actuators use a high-voltage alternating current (AC) to ionise the air around electrodes, and this voltage is usually in the range of a few kV – 30kV (da Silva et al., 2020). This creates a small and thin layer of rapid air which flows along the surface of the electrodes, due to the electric field – a pseudo wall-jet. When the electric field is strong, 'streamers', which are thin and highly ionised channels of air, form between the two electrodes, and they transfer charge, which creates a force on the air which is being moved from one electrode to the other. Streamers have a lifetime of around 10ns (Thomas, Kozlov and Corke, 2008). Although on most DBD Plasma Actuators, an alternating current is used, the

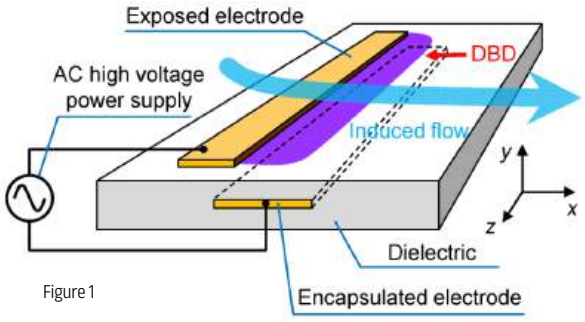



Figure 1

force on the air between the two electrodes always points in the same direction because one of the electrodes is exposed whilst the other one is covered by an insulating (dielectric) barrier, and due to this asymmetrical placement, the opposite half of the current cycle is cancelled. This has been proven in research, showing that about 97% of momentum comes from the negative half of the AC cycle, which produces an airflow in one direction (da Silva et al., 2020). This shows how airflow can be managed in aerodynamic objects, with no moving parts, and only the force of electric fields. It is also important to note that, whilst most DBD Plasma Actuators use AC, DC Actuators do exist but are just used less frequently.

## Why are Plasma Actuators used for silent flight?

These devices are very useful to reduce noise in flight, mainly since they don't contain any moving parts. This means that airflow is smoother, which reduces turbulence within systems and therefore will cut noise and make the flight quieter. High-lift devices, such as flaps and slats, are used to increase the lift of aircraft when they are at lower speeds, and plasma actuators also have utility here, as they can reduce the lift noise created by these devices. This has been tested in many studies, for example, in one Aircraft flap test, the use of DBD Plasma Actuators had a slat noise reduction of up to 3.3dB (da Silva et al., 2020). In another study, which involved a Helicopter rotor test, there was a broadband noise (general noise to surroundings) reduction of up to 8dB (Thomas et al., 2008). It is important to note that in the Helicopter rotor test, a DC DBD Plasma Actuator was used, in contrast to the AC used in the flap test, which may slightly offset the comparison, however the main factor in the difference of the two results is the scale the tests were done on, with the Helicopter rotor test done in an anechoic chamber with 15 microphones arranged around the rotor to

gather data. Both studies, in fact, did show that plasma actuators made a noticeable difference in noise, showing that plasma actuators are now very optimal for the goal of moving towards silent flight.

### What are the Challenges and Limitations?

Although Plasma Actuators seem very advanced and bulletproof in their utility, there are a few challenges that come with their use in silent flight. The main challenge is the difficulty in scaling them up to face real-world uses in aerodynamics, due to their high-voltage requirements. Since they require such a high voltage, this raises safety concerns due to more weight needed on aircraft to supply this and other issues with such high voltages running through aircraft. Additionally, scaling them up is difficult as the force caused by the high voltage, which pushes air to one side, may work in a lab or under ideal conditions, but not when in a conventional flying condition, where airflow is much heavier and more resistive. There are also additional limitations, such as the fact that plasma actuators are sensitive to humidity, as studies have shown that performance depends on humidity, which significantly affects thrust generation of air (Wilkinson et al., 2014).

### How can Plasma Actuators be integrated into the future of flight?

Although we have acknowledged that there are indeed challenges with the use of plasma actuators on a large scale, we should also realise that these are devices that are still being developed and bettered for future use in industry. However, this doesn't mean that they can't start to slowly be integrated from now onwards, as we could combine traditional designs for flaps and slats with plasma actuators for more modern designs, which could later be altered to make them even quieter and more silent. In Addition, they could also be integrated into hybrid propulsion systems, which combines two or more types of propulsion to power an aircraft (Köhler and Jeschke, 2021), by using them to perhaps reduce air resistance over wings or flaps, as they can control airflow, or perhaps to reduce noise further by using them in smaller components within aircrafts, in conjunction with traditional systems which already exist to control airflow.

Overall, these impressive devices have a lot of potential to improve aerodynamic performance in the future, and could lead to many new and exciting developments, such as stealth drones for military use, or aircraft for wildlife observation, which could be an exciting new way to go on a safari tour! The future for plasma actuators looks bright, and we should expect them to start being used far more often to make aerodynamics smoother and more efficient.

# The Martian Trailblazers: How Physics and Tech Unite on Mars

Written by Paarush Dhawan | Edited by Parth Shete

When NASA's Perseverance rover landed on Mars in February 2021, it marked the latest chapter in a long history of human ingenuity and exploration. From Sojourner to Curiosity, each rover has transformed Mars from a distant red speck into a scientifically rich world filled with geological and chemical secrets. Behind every success lies the seamless collaboration of physics and technology, combining to overcome the immense challenges of landing, operating, and analysing data on a planet many, many miles away.

The physics of landing on Mars is one of the most difficult problems in space exploration. Mars' atmosphere is only about 1% as dense as Earth's, making parachutes far less effective at slowing spacecraft during descent. To land a one-tonne rover safely, engineers had to apply Newton's laws of motion and energy conservation in intricate ways. Perseverance's landing involved a heat shield to dissipate energy through frictional heating, a supersonic parachute to slow descent, and retro-propulsive rockets to counteract gravitational acceleration. In the final seconds, the "sky crane" system lowered the rover on cables, balancing upward and downward forces so delicately that it touched the surface without raising damaging dust or tipping over[1][2].

Once on the ground, the rover's mobility depends on a precise understanding of forces, torque, and friction. Its six-wheel rocker-bogie suspension ensures stability on uneven terrain by distributing the rover's weight evenly and maintaining contact with the ground[3]. Electric motors convert energy from radioisotope thermoelectric generators (RTGs) into motion. These generators rely on the Seebeck effect - a temperature difference between a hot radioactive core and the cold Martian air, which generates a voltage. This process ensures a constant power supply even during the planet's severe dust storms, when solar energy becomes unreliable[4].

The rovers act as self-contained laboratories, using advanced instruments to study the Martian environment through the principles of light and matter interaction. Perseverance, for example, carries SHERLOC and PIXL - devices that fire lasers and X-rays at rock surfaces. When atoms in the rock absorb energy, their electrons move to higher energy levels/shells, and as they fall back, they emit light at characteristic wavelengths. This is a direct application of quantum physics and electromagnetic theory, allowing scientists to identify minerals and detect organic compounds potentially linked to ancient life[5]. Using this method, Perseverance detected carbonate and olivine minerals in the Jezero Crater (located in the northern hemisphere of Mars, near the planet's equator) - strong evidence that the region once hosted a long-standing lake, raising exciting possibilities that microbial life could have thrived there in Mars' distant past.

Even sound plays a role in Martian research. Microphones aboard Perseverance record wind gusts and mechanical noises, allowing scientists to calculate the speed of sound and understand how it varies with air density and temperature. This data reveals the unique acoustic properties of the thin carbon dioxide atmosphere and offers a simple but elegant application of wave physics in a real extraterrestrial setting[6]. In fact, by analysing the sounds of the rover's laser zaps and the gentle Martian wind, scientists discovered that high-pitched sounds travel faster than low-pitched ones on Mars - an unprecedented phenomenon caused by the planet's low pressure and carbon dioxide–rich air.

Because radio signals take up to twenty minutes to travel between Earth and Mars, rovers must also think for themselves! Perseverance's autonomous navigation relies on stereo cameras that construct 3D maps of the terrain and on-board algorithms that process these images using geometric and trigonometric principles. Machine learning models trained on previous rover data estimate slippage, calculate safe paths, and decide how to manoeuvre in real time. This autonomy represents the fusion of physics, computer science, and artificial intelligence—each contributing to motion, perception, and decision-making thousands of kilometres from human control[7].

The findings from these missions have been profound. Mars rovers have revealed dried riverbeds, sedimentary rocks, and mineral traces that indicate the planet once had liquid water[8]. These discoveries have reshaped our understanding of planetary evolution and hinted at Mars' potential to support life in the past. Perseverance is now collecting and storing samples that will one day be returned to Earth - a process requiring yet another technical and physical breakthrough: launching a rocket from Mars' thin atmosphere. Engineers must design systems capable of generating sufficient thrust with minimal fuel, using precise calculations of momentum, pressure, and aerodynamics to achieve the first off-world rocket ascent.

Future missions, such as the European Space Agency's ExoMars rover and NASA's planned Mars Sample Return, promise to push these boundaries even further[9]! By combining cutting-edge robotics, artificial intelligence, and the timeless principles of physics, these missions aim to deepen humanity's understanding of both the Red Planet and our place within the universe!

Mars rovers are much more than just mere machines; they are the embodiment of human curiosity, persistence, and intellect! Every movement, image, and signal transmitted from Mars reflects the unbreakable link between physical law and technological innovation. Together, they demonstrate how humanity's mastery of physics can turn distant dreams into reality - and how the partnership between science and engineering continues to drive exploration to new frontiers!

# From heat to horsepower: the energy recovery systems behind race cars

Written by Ayaan Camran | Edited by Quang Tran

Every time a Formula 1 car races around a circuit, you'd think it's just transferring energy away, burning fuel. After all, they just need to go as fast as possible over a set distance. Interestingly, for this to happen, energy collection is a key player – it's not all about emptying the tank. Modern F1 cars are packed with clever systems that capture power that would normally be lost and reuse it to help the car go faster and more efficiently. This process is called energy recovery, and it's one of the most interesting examples of applied physics in real life (Formula 1, 2023).

## What Is Energy Recovery?

When a car moves, its fuel is converted into kinetic energy — the energy of movement. But a lot of that energy doesn't end up doing useful work. Friction, braking, and hot exhaust gases waste much of it as heat (BBC Science Focus, 2022). Formula 1 engineers wanted to find a way to reuse some of this lost energy. By converting motion and heat into electricity, the cars could perform better without consuming more fuel. That's why every F1 car now includes a hybrid energy recovery system (Fédération Internationale de l'Automobile, 2024).

## MGU-K: Collecting Energy from Braking.

The first part of this system is the MGU-K, which stands for Motor Generator Unit – Kinetic. It's linked to the car's drivetrain — the parts that transfer power from the engine to the wheels (Formula 1, 2023). When a driver brakes, the car's kinetic energy usually turns into heat in the brake pads. However, the MGU-K operates like a generator, capturing some of that energy and converting it into electrical power. This is stored in a battery and can later be released to give the car extra acceleration. If you've ever driven or seen a hybrid road car, you'll notice that it uses a similar concept called regenerative braking. The difference is that F1's version is far more powerful and precise — it constantly adjusts in real time to suit each corner of the track (BBC Science Focus, 2022).

## MGU-H: Using Heat as Power

The other part of the system is called the MGU-H, short for Motor Generator Unit – Heat. This one captures energy from the hot exhaust gases that come out of the engine. These gases can reach over 800 °C — a huge amount of energy that would


otherwise be wasted (Fédération Internationale de l'Automobile, 2024). The MGU-H converts part of that heat into electricity. The energy it produces can either go straight to the battery or help the MGU-K drive the car's electric motor. In simple terms, it means even the exhaust helps power the car (Formula 1, 2023).

## Smart Energy Management.

The car can't store unlimited energy, so it has to be managed carefully. Complex software constantly decides when to harvest energy and when to release it (BBC Science Focus, 2022). Every time the driver brakes, the system charges; every time they accelerate, it can use that stored power. It all happens automatically, within milliseconds. The process is so well-timed that most of it goes unnoticed — yet it plays a huge part in making F1 cars incredibly efficient.

## Why It's Important

Even if you're not into racing, these systems are an amazing example of how science and engineering can make machines smarter. The same ideas are now used in everyday hybrid and electric cars. When you brake in a hybrid, you're using a small version of F1's MGU-K. Energy recovery is helping make transport cleaner and more sustainable, showing how technology developed for racing can benefit everyone (Formula 1, 2023).

# BIOLOGY

# Evolution of the AlphaFold AI

Written by Gautham Subramanian | Edited by Parth Shete

In 2024, three scientists by the names of David Baker, Demis Hassabis and John Jumper won the Nobel Prize in Chemistry: Baker for his groundbreaking work in building entirely new proteins with distinct functions; Hassabis and Jumper for their work in analysing and predicting the 3-Dimensional folding structures of over 200 million proteins from their amino acid sequences through the creation of Google DeepMind's AlphaFold 2 AI (The Nobel Prize, 2024) (Jumper et al., 2021). But just how did they do that? And why is it important?

There has been significant excitement surrounding the rise of Google DeepMind's AlphaFold AI and its potential applications in the development of cures for diseases and cancer, use in vaccine development, developing plastic-degrading enzymes (Google DeepMind, 2024), paleoproteomics (Google DeepMind, 2022), and much more. While one might assume that the success of AlphaFold can be attributed to its access to vast compute power and diverse data sets, a significant contributing factor is its efficient algorithms. Understanding the evolution of these optimised algorithms (e.g. AlphaFold 2 vs AlphaFold 3) allows us to derive solutions to complex problems in simpler ways.

The AlphaFold 2 AI is a deep neural network, which takes the amino acid sequence of a particular protein from the subject organism as input, with the aim to find out its 3-Dimensional structure.

As a first step in this process, the input amino acid sequence is used to query the Protein Data Bank to compile a matrix of similar amino acid sequences, called multiple sequence alignment or MSA (Looking Glass Universe, 2024a). The MSA contains the evolutionary table of that protein in different organisms. This MSA is then passed as input into a transformer called the Evoformer which uses a process called attention whereby, the input amino acid sequence is broken up into chunks, with each chunk being converted into vector-based representations, (Veritasium, 2025) to infer connections between the amino acids in the sequence.

Importantly, unlike AlphaFold 1, which only took the MSA as input into a deep neural network, AlphaFold 2 also takes a pair representation matrix as input into the Evoformer. This is a separate matrix whereby each amino acid is compared to every other amino acid in the order that they appear in the original sequence. Details of their relationships including distances and angles between them are recorded, whereby lighter tiles in the matrix indicate closer amino acids. Sometimes placeholder values are inputted where such data is unknown as the AI would recursively finetune these values as they pass through the Evoformer.

The Evoformer brings together the MSA and pairwise representations, continuously updating both 48 times. Within the MSA, columnar attention can help to identify which amino acids are highly conserved amongst the several sequences (Looking Glass Universe, 2024a), further helping to determine their folding shape. As an example, note that the purple arrow in Fig.2 appears in all 4 amino acid sequences. Moreover, row attention can help to find pairs of amino acids which may have co-evolved and are thus important to maintaining structure. In the second row of Fig.2, the 2nd and 5th amino acids have changed, suggesting that as one mutated the other must have also mutated for the protein to remain folded up and kept a similar shape. This data is also communicated to the pairwise representation.

Figure 2

Triangular attention (Looking Glass Universe, 2024a) within the pairwise representation is a very simple but ingenious constraint on the folding of the amino acid sequence. It works by taking any pair of amino acids in the sequence, calling them *i* and *j*, and constructing a triangle between them and a third amino acid *k*. For any triangle, it is always given that the sum of the lengths of any two sides must be greater than the length of the third side:

$a + b > c$ (Triangle Inequality)

Through consideration of the distances listed in the pair representation matrix, the AI considers the side lengths *ij*, *ik* and *jk* but also the permutations of these for symmetry. It then repeats this process with a different amino acid for *k* until it

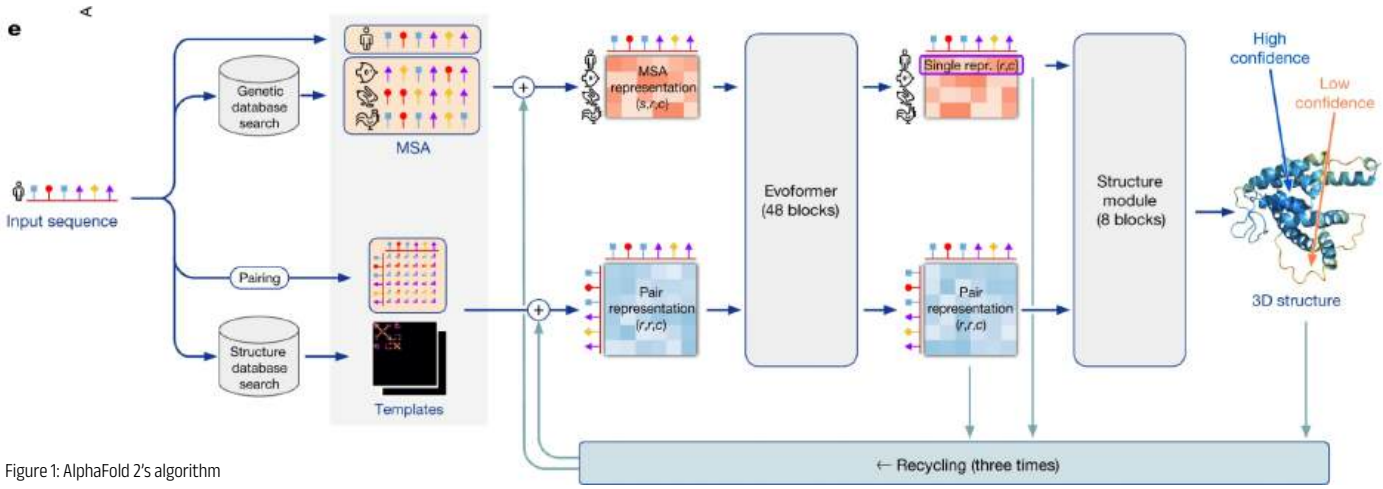


Figure 1: AlphaFold 2's algorithm

has iterated through them all. As it iterates through the Evoformer, the position of each amino acid is fine-tuned more precisely as the AI gains a better understanding of the more plausible locations for each amino acid to be placed. (Looking Glass Universe, 2024b)

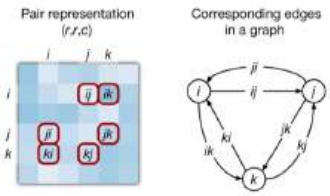


Figure 3

The updated pairwise representations and first row of the MSA (which represents the input amino acid sequence) is then inputted to the structure module. Here, each amino acid is converted into a triangular frame between the central Carbon and Nitrogen atoms. The AI then works out what combination of rotations and translations to apply to each triangle, relative to each other, to ensure they can fold into the final structure (Looking Glass Universe, 2024b). The output of the structure module is then fed back into the Evoformer at least 3 more times until the AI outputs the final protein's 3-Dimensional structure with a respective confidence rating.

With this algorithm, AlphaFold 2 was able to find the structure of almost every protein in existence and proved how AI can enable rapid progress in the field of structural biology.

Additionally, Baker's work contributed significantly to the evolution of AlphaFold 3 (Abramson et al., 2024). His use of RoseTTAFold (RF) diffusion (Watson et al., 2023) to build completely new proteins, known as synthetic proteins ushers in a new age of biology whereby proteins with a specified function can now be created. It works by inputting to the AI, the known structure of a protein and then adding some noise to the input, alongside a description of the functional role of the protein. The goal of the AI is simple: to remove the noise and return the original protein's structure through iterative improvement. Once the AI is trained in this way several hundreds of times, it can expect a seemingly random noise sample and a given functional description, then, come up with the 3-Dimensional structure of a completely new protein (Veritasium, 2025).

This concept of diffusion is incorporated as a key feature of AlphaFold 3, which is a more general form of AlphaFold 2, capable of making predictions of the structures of not only proteins but DNA, RNA, ligands and complexes (Looking Glass Universe, 2024c). Thus, rather than a sequence of amino acids, it takes as input, tokens. Each token may be an amino acid, base pairs or a combination of those - it depends on the molecule. The overall algorithm operates in a quite different way to AlphaFold 2 with particularly less emphasis given to the MSA, as some of the tokens may not have enough evolutionary history (e.g.: ligands). Instead, the pairwise representation is of greater importance, yet it gets updated in a similar way to AlphaFold 2 using triangular attention within a Pairformer instead of the Evoformer.

The structure module is replaced by a diffusion module, operating in a similar way to Baker's RF diffusion process, to predict the positions of both the tokens and atoms in the structure (Looking Glass Universe, 2024b). With these changes, AlphaFold 3 can predict the 3-Dimensional structures of all these different types of molecules with high accuracy (even better than AlphaFold 2).

The development of the AlphaFold AI has accelerated our understanding of protein folding in unimaginable ways. It enables us to tackle a range of challenging problems, not just within the world of structural biology. It has become easier, cheaper and faster to conduct scientific research, so let's embrace this AI and work towards exciting, new discoveries.

**"WITH THIS ALGORITHM, ALPHAFOLD 2 WAS ABLE TO FIND THE STRUCTURE OF ALMOST EVERY PROTEIN IN EXISTENCE"**

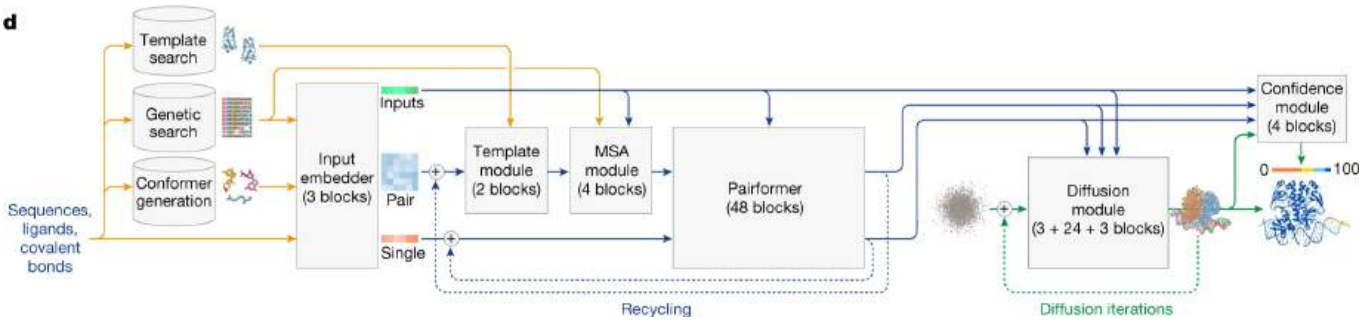


Figure 4: AlphaFold 3's algorithm:

# An In-Depth Guide to Hand and Wrist Conditions

Written by Ved Saklani | Edited by Arjun Bhatt

Although one of the most common hand conditions that runs rampant through society is osteoarthritis, which, in 2019, affected about 528 million people worldwide: an increase of 113% since 1990 (Institute for Health Metrics and Evaluation, n.d.), the most common hand and wrist condition is carpal tunnel syndrome (www.bssh.ac.uk, n.d.).

In a study of a region in southern Sweden with a population of 170,000, it was found that, of a stratified sample of 3000, sixty-six symptomatic subjects had clinically and electro-physiologically confirmed carpal tunnel syndrome (Atroshi, 1999). Furthermore, experts estimate that around 3 out of every 1,000 people in the U.S. experience carpal tunnel syndrome each year (Cleveland Clinic, 2019). For such a widespread condition, there has been extensive research done on the causes and symptoms of it. Some symptoms include an ache or pain in your fingers, hand or arm; numb hands; tingling or pins and needles; a weak thumb or difficulty gripping (NHS, 2021). It arises because of the compression of the median nerve in the wrist. Orthopaedic doctors use several techniques to make sure the diagnosis of carpal tunnel is accurate. For instance, the two point discrimination test is carried out, which involves a two-pointed pin being pressed on the fingers of the patient. The median nerve carries electrical impulses from the first three fingers in the hand, so these fingers are of most importance in this test. The doctor simply asks the patient whether they can feel the pin as two points, or just one; in which the latter response indicates numbness in the finger: a telling symptom of carpal tunnel syndrome. Other conformational tests for CTS include tapping the wrist at the location of the median nerve (Tinel's sign), and a wrist-flexion test (Phalen's test). This external stimulation from the doctor would induce a tingling sensation in the first three fingers in patients who have CTS.

Once the doctor has diagnosed CTS, there are many avenues of treatment; from simply continuing to live with the pain, to a ligament decompression surgery. These treatments are dependent on the patient's individual needs; specifically, how badly the CTS affects their quality of life. Quality of life is a concept that aims to capture the well-being of a population or individual regarding both positive and negative elements within the entirety of their existence at a specific point in time (Teoli and Bhardwaj, 2023). Therefore, a doctor assesses and informs the patient on what treatment would be suitable for them, following obtaining a patient's history. For instance, if the patient had a job which involved the extensive use of a computer and typing, then immediate relief via surgery or steroid injections would be the most reasonable treatment plan. However, if the patient was retired, and only felt minor discomfort, then they could avoid treatment altogether, or undergo less invasive treatment, such as hand exercises or wearing a splint (Mayo Clinic, 2024).

The surgery for relieving CTS is known as carpal tunnel release surgery. This entails the surgeon making a cut near the bottom of the palm, before using a hook knife (Lee et al., 2023) to divide the carpal ligament. This will widen the carpal tunnel and ease pressure on the median nerve. The cut in the skin is closed with stitches, followed by a bandage being tied around your hand. In keyhole (endoscopic) surgery, the doctor makes one or two small cuts (portals) in your skin near the carpal tunnel. They then put in a tiny camera to see inside the hand and wrist. They also put in endoscopes equipped with trocars, or blades (Noszczyk, Krzesniak and Nowak, 2014) to perform the surgery and cut the carpal ligament. This avoids placing a surgical scar on the hand (www.bupa.co.uk, n.d.). Despite the careful care of the surgeon to minimise any post-operation complications, there are still risks of surgery to treat CTS. These include bleeding, infection, injury to the median nerve or nerves that branch out from it, injuries to nearby blood vessels, a sensitive scar, the need for more surgery (Johns Hopkins Medicine, 2019). While this surgery is not a particularly risky one, the road to a full recovery is extensive, with frequent post-operation checkups, physical therapy, and the mental toughness required to stick to the recovery programme for an extended period. For adults specifically, the prospect of driving after a carpal tunnel release surgery is limited. While it is not illegal, and the DVLA requires no notice of the operation when recovery takes less than three months (www.bupa.co.uk, n.d.), the tenderness and lack of grip strength means that many adults abstain from driving until they regain confidence that their strength is back to normal. Overall, the surgery for relieving CTS is minimal risk, but recovery can be extensive, and a return to normal life (such as driving) may be limited because of the operation.

To avoid these complications, however, steroid injections can be used to reduce the pain caused by CTS, with fewer than 1 out of 1,000 people left with

nerve damage after an injection. These injections are known as corticosteroids injections. This reduces swelling in the connective tissue of the carpal tunnel and decreases the pressure on the median nerve (Information et al., 2017b). One benefit is that they are useful for short-term relief, so are sometimes used while awaiting surgery or during early CTS. However, a limitation to these steroid injections is that they are not effective in the long term, and, whilst they are effective in reducing pain in the first 2-4 weeks, it is likely that the pain will return in the long term. Constantly returning for corticosteroid injections, however, is a risky treatment plan, as continued extended injections with steroids can lead to tendon and nerve damage, exacerbating the issues of hand and wrist pain that originated. Hence, while the injections cause temporary relief from the pain, repeated use of them can lead to more harm than good, negatively impacting the patient in the long term.

The next plausible treatment is the use of a hand splint with hand exercises to relieve any discomfort on the hand in daily life. This holds the wrist in a neutral position, especially at night when the hand tends to bend more as the body shuffles in the bed unbeknownst to the patient. However, it is important for the hand to be in motion throughout the day, to keep it from becoming stiff and stop the muscles becoming weak (National Library of Medicine, 2017). This would cause the hand to be numb and unusable, contradicting the idea of using a splint for relief of pain.

However, as a doctor, there is always the looming thought of scarcity of medical resources, such as corticosteroid injections and surgical equipment. Therefore, whilst it may seem contradictory, one mode of treatment is to abstain from treatment altogether and reassess in a future appointment. Shockingly, Harvard Health cites that avoiding treatment can lead to permanent nerve damage (Harvard Health Publishing, 2017), however, as CTS is a progressive disease, the patients can always return for treatment if the pain inhibits them from having a good quality of life. This method of avoiding treatment is only used for those patients that have caught the pain early, and it does not interfere with their daily affairs in a significant way.

"Carpal tunnel syndrome is an extremely widespread condition that affects many people worldwide."

In summary, carpal tunnel syndrome is an extremely widespread condition that affects many people worldwide. It causes pain and numbness in the first three fingers in the hands, due to the compression of the median nerve by tissues in the carpal tunnel. Treatments can go from an invasive open or keyhole carpal tunnel release surgery to wearing a splint at night and physical exercises to restore strength in the nerve and tendons. As one of the most common conditions worldwide, acknowledgement of how it is caused and the various advantages and disadvantages of several avenues of treatment is important, particularly in a society where people regularly go to the internet for how to treat such a well-researched and easily treatable disease. If you feel that you are experiencing or have experienced any kind of the symptoms of CTS discussed, it is advised that you go and see your GP (Cleveland Clinic, 2019).

# Dementia - Music as a Medicine

Written by Rohan Savjani | Edited by Gautham Subramanian


Figure 1: (Tech, 2016)

Imagine being so confused that you can't perceive where you are. Imagine finding it impossible to follow seemingly simple conversations. Imagine being incapable of remembering your loved ones. Confusion, disorientation and memory loss are not imaginary symptoms but a reality for the millions of people with dementia.

Whether it's a symphony from Strauss or Stormzy's latest hit, music has a refreshing, restorative and revitalising effect. But can it have a deeper therapeutic value for people with dementia? In short, can music be a medicine?

**Dementia:**

Dementia is a syndrome - a collection of related symptoms - resulting from damage to the brain caused by multiple different diseases, such as Alzheimer's [1]. These symptoms vary according to the part of the brain that is damaged but typically include memory loss, confusion, problems with language, and changes in behaviour.

In the UK there are around one million people with dementia [2] and with an ageing population dementia is only becoming more prevalent. Alzheimer's Research UK estimates that 50% of people in the UK will be directly impacted by dementia: either developing the condition themselves; caring for someone who has it; or both [3].

**Demographics and causes:**

The incidence of dementia increases rapidly with age between the ages of 65 and 90 years, doubling approximately every 5 years [4]. There is currently no cure for dementia - it being the leading cause of death in the UK [5]. However, it is important to realise that dementia results from physical diseases that damage the brain and is not simply a normal part of ageing.

Alzheimer's disease (AD) is the most common cause of dementia in the UK [6]. With AD the abnormal accumulation of amyloid beta proteins forms plaques between neurones. Additionally, tau proteins form tangles inside them, which disrupts and eventually destroys the synapses, between neurones. During the period of protein build up, some individuals may not exhibit symptoms, while others may begin to show signs of memory decline or some cognitive impairment. This preclinical phase generally advances to a stage of further cognitive dysfunction, with the death of nerve cells, culminating in profound AD. This process typically takes several years.

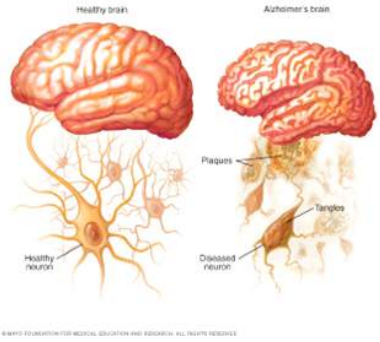



Figure 2: (Mayo Clinic 2025)

**Treatment Options:**

Pharmaceutical treatments, such as acetylcholinesterase inhibitors, which increase the levels of the neurotransmitter acetylcholine, can help alleviate cognitive impairment. However, these treatments do not alter the progression of AD. It is this lack of disease-modifying drugs that has spurred research into non-pharmacological therapies for AD, which focus on mental stimulation. Activities that stimulate the brain form new synapses and can improve brain health. This idea of rewiring the brain – increasing its neuroplasticity - is crucial as these new connections reduce the symptoms of AD.

Music therapy has received considerable attention as an alternative therapy. Studies have shown that music therapy (which includes singing, rhythmic movement and playing instruments) can

enhance several cognitive abilities in people with AD [7]. In particular, it has shown to be effective in improving memory and language capability in people with mild AD [8].

There is a threefold physiological basis for the enhancement of memory through music. Firstly, musical stimuli are strong correlated with steroid hormones [9] which in turn are linked to cerebral neuroplasticity [10]. In addition, music promotes the release of several chemical messengers, called hormones, including dopamine [11] which improves memory retention. Furthermore, music can influence the activation of immune cells, particularly microglia of the brain, which can help reduce the formation of amyloid Beta plaques [12] [13].

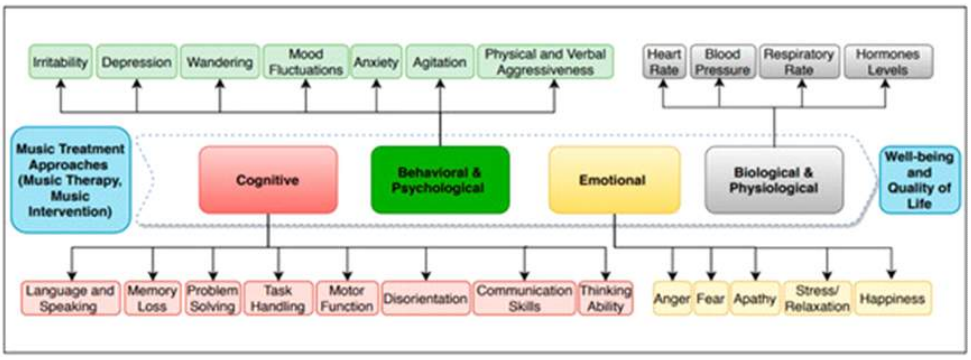



Figure 3: (Soufineyestani, Khan and Sufineyestani, 2021)

> "Music is a therapy. It is a communication far more powerful than words, far more immediate, far more efficient" [4]

**Practical Action:**

Various charities run musical therapy programmes throughout the UK. One of these is Lost Chord, which was established with a vision "to harness the transformative power of music to enhance the quality of life for those facing cognitive challenges" [14]. Their sessions are run by professionally trained musicians in a variety of settings from memory cafes, wellness centres, libraries and community events. They also bring music to care homes and hospices.

Music therapy singularly, or in conjunction with other complementary therapies, can perhaps decelerate neurodegeneration in individuals with AD. Moreover, it could also play a part in delaying the onset of symptoms in people at risk of AD, such as those with a genetic risk.

Overall, people with AD often find listening to, or playing music can reignite memories. Scientific studies have gone further and suggest that activities involving music can be a beneficial non-pharmacological treatment. Specifically, music therapy can improve neuroplasticity, activate dopamine release and help modulate cerebral inflammation through the activation of microglia.

Music, and specifically music therapy, do appear to have a role in the treatment of AD. As the world-renowned violinist Yehudi Menuhin so poignantly put it, "Music is a therapy. It is a communication far more powerful than words, far more immediate, far more efficient" [4].

# The chemical switch silently governing our lives

Written by Snehith Gannu | Edited by Arjun Bhatt

The screen glows. Your thumb flicks down. Each new scroll offers the chance of something shocking, amusing, or enraging. You do not know what you will see next, but you keep going, hunting for novelty. This is the essence of doomscrolling: an endless loop of uncertainty and surprise, where the possibility of the next "hit" keeps us hooked. But the strange thing is, this behavior is not driven by willpower, or even conscious choice. It is orchestrated by a single molecule, invisible but powerful, guiding our actions from deep inside the brain: dopamine.

## Reward Prediction Error

Dopamine is often labelled as the "pleasure molecule," but this can be misleading. It is not the feeling of joy itself, but the force that drives us towards it. More precisely, dopamine governs anticipation — it makes us want, not necessarily like it. This distinction becomes clear when we think about reward prediction error: the gap between what we expect and what we get. Imagine standing in front of a Japanese vending machine with six mysterious buttons. You pick one at random, and the outcome is exactly the drink you were hoping for — an unexpected delight. Your brain registers this pleasant surprise as a positive prediction error, and next time you will press the same button. But if, weeks later, the same button dispenses a disappointing alternative, your brain records a negative prediction error (Schultz, 2016). Now, you will adjust your choices. In both cases, dopamine signals that error, teaching you where to direct your future behavior.

The neuroscientist Wolfram Schultz demonstrated this with macaque monkeys. He implanted electrodes into their brains and placed them in an apparatus with two lights and two boxes. One light signaled that food would appear on the right, the other that it would appear on the left. At first, dopamine fired when the monkeys saw food when they opened a random box- this was expected. Over time, the monkeys learnt the rule: when the light flashed, they already knew where the food would be. However, what Schultz found after this was striking — dopamine neurons did not fire when the monkeys received the food. Instead, the burst of activity came when the light turned on. Once the monkeys had learnt the rule, the food was no longer a surprise but rather it was the light, and dopamine stopped firing on its arrival. (Schultz, Apicella and Ljungberg, 1993) This showed that dopamine is not a pleasure molecule at all, but a reaction to the unexpected — a driver of possibility and anticipation.

## Dopaminergic learning

Dopamine is powerful because it teaches us what to do. rewards produce learning. Think of Pavlov's dog: it hears a bell which prompts it to see the sausage and then salivate in response. With enough repetition, the bell alone is enough to trigger drool, because the brain has learnt to predict the sausage. That is Pavlovian learning — it happens automatically, just by being awake and exposed. (Windholz, 1997) Then there's operant conditioning, which requires action.

Thorndike's cat scrambles around a cage until it accidentally presses a latch, escapes, and eats. The food is good, so the cat repeats the behavior, refining it each time (Mcleod, 2024). In both cases, dopamine is the teacher. It signals when outcomes differ from expectations — when a prediction error has occurred — and pushes us to update our behavior accordingly.

The logic of prediction errors is simple but profound. If the reward is better than expected, dopamine spikes, and we do more of the behavior that led to it. If it is worse than expected, dopamine dips, and we avoid it. If the reward is exactly as predicted, there is no prediction error, and nothing changes. This is why mistakes, unpleasant as they feel, are essential to learning: every error forces an update from the level of dopamine received, aligning behavior with reality. Through countless micro-adjustments, dopamine makes us creatures of habit, repeating behaviors that once paid off. (Schultz, 2010)

## Familiarity

But here is the catch: once something becomes familiar, the dopamine fades. That first croissant from a bakery might be the best you have ever tasted. A week later, it is still good, but the thrill is gone. Your brain knows exactly what to expect, so there is no more prediction error, which means no dopamine. The excitement of anticipation has been replaced by routine. This is dopamine's protective mechanism — preventing us from wasting energy chasing after things that are already known. (Lieberman and long, 2019) Yet modern habits like doomscrolling exploit a loophole in this system, because novelty never truly runs out online. Each swipe offers the possibility of something new, unexpected, and therefore dopamine-releasing. It is this endless cycle of prediction errors that keeps us scrolling long past the point of satisfaction. (University of Law, 2023)

## "Satisfying molecules" vs Dopamine

Unsurprisingly, dopamine is involved in several important brain circuits, one of which is the mesolimbic pathway, better known as the dopamine desire circuit. Here, dopamine is released in the ventral tegmental area (VTA) and travels to the nucleus accumbens (*image A*), where it fuels motivation and drive. (Warren, 2020) Activation of this circuit triggers feelings of energy, enthusiasm, and hope. It feels good — not because we have achieved something, but because we believe we are about to. This is why dopamine is sometimes described as the molecule of "craving." It paints vivid pictures of a better future: whether it is

the delicious meal we are about to be served or the old friend we are about to see. Dopamine turns into imagination, filling it with possibility and promise.

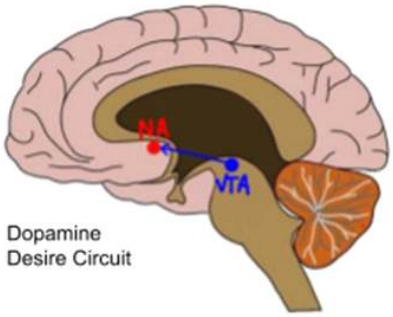



Image A

But dopamine only cares about the future. Once dinner is in your mouth, or your friend stands beside you, dopamine switches off. The sense of excitement and energy fades, leaving many people disappointed. That is because dopamine circuits do not process real-world experience — they only deal in imagined futures. To enjoy the present moment, we need a distinct set of chemicals; chemicals we can call "satisfaction molecules": oxytocin, endorphins, and endocannabinoids. These neurotransmitters are the ones that allow us to savour life as it is— the textures and aromas of a meal, the warmth of laughter with friends, the comfort of being close to someone we love. Dopamine makes us want things with intensity, but it is the satisfaction molecules that let us enjoy them. (Healthy Life Recovery, 2022)

The tension between "wanting" and "liking" is one many of us feel every day. Take buyer's remorse. We see something — an expensive car — and the desire circuit lights up. Dopamine promises that owning it will transform our lives, flooding us with joy. But once the purchase is made, reality falls short. The thrill of anticipation fades, and satisfaction does not measure up. This happens because the desire circuit is, in a sense, a salesperson: it sells us a vision of the future but has no role in delivering the enjoyment itself. True fulfillment comes only when the satisfaction chemicals take over, and often they cannot match the promise dopamine made. (Boag, 2024) Sometimes, if the purchase really is wise, satisfaction molecules can compensate for the loss of dopaminergic arousal. However, this is not always the case due to the influence pleasure received from satisfaction chemicals does not compare to dopamine's influence. Hence, other times, we fall back into the cycle — buying things that promise still more anticipation. In this way, dopamine keeps us moving forward, but it rarely allows us to rest content.

## Addiction

However, it is important to note that if what we receive surpasses our expectations and goes beyond our imagined happiness, dopamine does not switch off but is released intensely — that is a reward prediction error. (Huberman, 2023) When this happens, we crave the feeling again and can go to great lengths for it even though, logically, it is an impossible mission (we know what the reward is). Unfortunately, drugs spike dopamine to levels the brain was never made to handle. As we get less reward prediction error when repeating the same task, less dopamine is released and we need a stronger stimulus to reach the baseline level we once got with a weaker one — whether it is higher doses of drugs or another bottle of alcohol. The harsh reality is that getting less dopamine than expected feels far worse than the joy of receiving more than expected — so addicts go to great lengths to keep consuming, creating a vicious cycle where less dopamine is received but more dopamine is desired.

## Conclusion: how powerful really is dopamine?

In the end, dopamine fuels our anticipation, guiding what we chase and reinforcing what we learn through surprise. Yet the thrill of wanting is different from the joy of being — that belongs to the chemistry of satisfaction molecules that let us savor life as it unfolds.

Recognizing this balance explains why habits like doomscrolling keep us hooked on endless craving, and why genuine fulfillment arises only when we step back and appreciate what is already present.

"Dopamine is powerful because it teaches us what to do. rewards produce learning."

# Not all who wander are lost - some just innervate everything: The Vagus Nerve

Written by Shayan Azaz | Edited by Emad Rehman

## What is the Vagus Nerve?

You're about to walk into the exam hall: your heart is pounding, your hands are shaking, and your mind is racing. In short, your sympathetic nervous system, otherwise known as your '*fight or flight*' response, is in overdrive. Originally designed for life-threatening scenarios such as facing wild animals, but now largely reserved for exam halls and forgotten homework, this system is your body's response to acute stress. Later that day, however, once you are resting on the sofa, the exam having gone well enough, it is not your sympathetic nervous system in charge but rather its lesser-known counterpart - the parasympathetic nervous system, also known as the '*rest and digest*' system. This is largely controlled by your vagus nerve, sometimes referred to as the 'polymath of the parasympathetic nervous system'.

The vagus nerve coordinates many automatic bodily functions that occur when the body is at rest. These include breathing, heart rate, swallowing, digestion, appetite, and more.[1] All of these have profound impact on our physical and mental health, and so have led scientists and wellness researchers alike to explore how stimulating or supporting this "information superhighway" may help manage chronic conditions and improve mental wellbeing.

## Anatomy of the Vagus Nerve

"Vagus" is the Latin word for wandering, an appropriate name given the long, branching pathway this nerve follows through the body.[2] Exiting from the medulla oblongata, the vagus nerves travel down the neck - between the carotid artery and jugular vein - before extending to the heart, lungs, liver, spleen, stomach, intestines, and kidneys.[3] The vagus nerve consists of two thick bundles of individual neurones that originate in the brain and travel down to the left and right sides of your body.[1] Structurally, the vagus nerve is made up of approximately 80% sensory neurones, which transmit signals from the organs to the brain, and 20% motor neurones, which transmit signals from the brain to the organs. This enables bidirectional communication between the central nervous system and multiple organ systems.[4][5]

## Functions of the Vagus Nerve[5]

### Cardiovascular Functions

Heart rate is normally controlled by a part of the heart known as the sinoatrial (SA) node. The vagus nerve helps to slow the heart rate by releasing a neurotransmitter (acetylcholine), which binds to the SA node, making it harder for these cells to reach the threshold voltage required for a heartbeat to occur, thus slowing down the heart rate. Effectively, the vagus nerve acts as a brake on heart rate, and this braking effect is known clinically as vagal tone.[38] Individuals with strong vagal tone tend to show better heart-rate variability, reduced anxiety, lower risk of depression, and - as emerging research suggests - potential cognitive benefits.[3]

The vagus nerve is also central to the baroreceptor reflex, which regulates blood pressure. When blood pressure rises above its set point, arterial walls stretch. Baroreceptors detect this stretch and increase signaling to the brain via the vagus nerve, prompting a reduction in heart rate and restoring blood pressure to normal. The opposite response occurs when blood pressure is too low.[39]

### Immune Functions

When tissues are damaged, immune cells secrete pro-inflammatory cytokines. The motor neurones of the vagus nerve communicate with organs such as the spleen, liver, and gut to control and modulate this response. This helps in maintaining homeostasis and controlling inflammation. It does this by stimulating the secretion of acetylcholine (a neurotransmitter) from its motor neurones, which binds to the α7 nicotinic acetylcholine receptors (α7 nAChRs) on immune cells, inhibiting their activation, and so inhibiting pro-inflammatory cytokine release. This mechanism is known as the "cholinergic anti-inflammatory pathway".[7]

Furthermore, acetylcholine secreted by the vagus nerve has been linked with the regulation of regulatory T cells (Tregs (🦖)), a type of T-lymphocyte that helps suppress excessive immune responses. The activation of α7 nAChRs in Tregs contributes to the anti-inflammatory effects of acetylcholine, helping maintain immune homeostasis.[6]

## The Vagus Nerve in the Gut-Brain Axis

The vagus nerve is a major communication pathway between the brain and gastrointestinal tract.[29] The gut microbiome (consisting of trillions of microorganisms that live in the digestive tract) influences specific metabolites in the intestinal lumen, which subsequently activate chemosensory vagal neurones (neurones that detect and convert chemical signals into electrical signals), enabling continuous two-way communication between the gut and brain.[30] Disruptions in this signalling can contribute to psychological or neurological conditions.

For example, mice treated with JB-1, a type of bacteria that affects the gut microbiome, saw reduced levels of stress hormones, anxiety and depression related behaviour. However, these behavioural alterations were inhibited by cutting the vagus nerve below the diaphragm to reduce communication between the brain and the rest of the

body via the vagus nerve (subdiaphragmatic vagotomy, SDV), indicating that the vagus nerve is a key communication route between gut bacteria and the brain.[31]

Another study found that transplanting gut bacteria from chronically stressed mice into healthy mice induced similar depression-like behaviour; again, SDV blocked this effect.[32] Together, these findings suggest that disturbances in the gut-brain axis via the vagus nerve play a role in the development of depression and other psychiatric and neurological disorders, such as Parkinson's disease and multiple sclerosis[33][34], potentially offering a future pathway for developing new treatments for these conditions.[5]

## Treatments involving the vagus nerve[5]

### Vagotomy

Vagotomy - surgically cutting branches of the vagus nerve - has been performed since the early 20th century, mainly as a treatment for peptic ulcer disease[8], the idea being that severing the vagus nerve, which stimulates acid secretion in the stomach via the gut-brain axis, would reduce the formation of ulcers. However, following the development of alternative means of treating peptic ulcers,[9] vagotomy as a form of treatment declined significantly.

Towards the late 20th century, focus shifted to the possible use of vagotomy as a treatment for other inflammation-related diseases, although this has been problematic. It has been shown to decrease the risk of liver and intrahepatic cancer[10], lower the risk of ischemic stroke and ischemic heart disease[11][12], and significantly decrease the risk of developing diabetes in patients with upper GI diseases.[13][14] However, it carries risks such as impaired gut motility,[15] and an increased likelihood of enlarging the prostate gland (benign prostatic hyperplasia).[16]

Furthermore, research has been done to explore the link between vagotomy and neuropsychiatric diseases. For example, (truncal) vagotomy has been shown to lower the risk of Parkinson's disease, compared to the general population, in cohort studies[17][18]; however, other results were less positive, with some cohort studies showing no risk changes following truncal and selective vagotomies for dementia and multiple sclerosis.[20][21] Some population studies have even found that truncal vagotomy was linked to a small increase in the risk of neuropsychiatric disorders such as dementia and Alzheimer's disease, with a 69% increase in risk of developing schizophrenia.[19]

These inconsistent findings underscore the complexity of brain-body interactions and the need for larger, controlled studies.

### Vagal Nerve Stimulation (VNS) - Surgical/Invasive

Vagus nerve stimulation involves surgically implanting a device with electrodes around the vagus nerve in the neck to deliver electrical impulses to the nerve.[5][22] It became more common in the late 20th century as a treatment for drug-resistant epilepsy and treatment-resistant depression (TRD). It has been shown to reduce seizure frequency in epilepsy patients and improve the mood of those with TRD.[23][24]

In fact, preclinical data suggests that its benefits may arise partly from reducing neuroinflammation, as electrical stimulation triggers biochemical pathways that lower inflammatory markers.[25]

An interesting area of ongoing research is looking at VNS mimetic therapy. VNS works by stimulating the release of certain chemicals from the vagus nerve, which begins a chemical cascade that results in reduced inflammation. VNS mimetic therapy is an interesting idea that seeks to begin this chemical cascade using drug-based means, as opposed to physical, electrical stimulation of the vagus nerve, to simulate the effects of VNS on drug-resistant epilepsy, TRD and other chronic-inflammatory diseases.[26]

### Transcutaneous auricular vagus nerve stimulation (taVNS) - Non-Surgical

Traditional VNS requires surgery and carries common side effects such as hoarseness, coughing, throat discomfort, and breathlessness. Hardware complications can also arise.[5]

In contrast, taVNS uses electrodes on the outer ear to stimulate the auricular branch of the vagus nerve. It is safer, non-invasive, and more comfortable.[5] Commercial wearable devices already exist for anxiety and mood disorders,[35] and clinical studies show promising results for depression.[36]

Although research is still developing, taVNS may enable broader access to vagus-nerve-based treatments.

### Vagal Maneuvers[37]

Vagal maneuvers are physical actions, some of which you can do yourself, that result in the vagus nerve slowing down the electrical impulses of the heart, thus slowing a fast heartbeat, and calming you down. These include the diving reflex, whereby you take several deep breaths and then place your face into a container of ice water; the Valsalva maneuver, whereby you lie on your back, take a deep breath and act like you're exhaling, but with your mouth and nose closed for 10-30 seconds. Coughing and even handstands can have the same effect!

## Conclusion

The vagus nerve interacts with many organs and influences essential functions that support physical and mental health. As research advances, the vagus nerve continues to emerge as a promising target for treating neurological, psychiatric, and inflammatory conditions. So next time you're panicking in an exam, pause, take a deep breath, and know that your vagus nerve has your back (and the majority of your body!)

"THE VAGUS NERVE INTERACTS WITH MANY ORGANS AND INFLUENCES ESSENTIAL FUNCTIONS THAT SUPPORT PHYSICAL AND MENTAL HEALTH."

# Editing the Athlete: Is Gene Doping the Future of Sports?

Written by Rayyan Ali | Edited by Quang Tran

Usain Bolt set the world record for the 100m sprint back in 2009, running it in only 9.58 seconds. That record has survived more than a decade. But what if it could be broken not through relentless training and talent, but by switching off a single gene? Through targeted gene editing, an athlete could bypass natural biological limits and gain muscle power or explosive speed that no amount of coaching alone could create. Some will see it as a risk, and others will praise it as progress, but it is simply raw biological power now under human control. And as humans obtain more power, the potential for its misuse heightens too.

## Gene Doping: what it is and how it works

Gene doping is defined by the World Anti-Doping Agency (WADA) as "the non-therapeutic use of genes, genetic elements and/or cells that have the capacity to enhance athletic performance" (Unal and Ozer Unal, 2004). This contrasts with traditional doping through the use of performance-enhancing drugs, which rely on external substances (steroids, stimulants, human growth hormones, etc) that can be detected in the blood or urine. In contrast, gene doping rewrites the athlete's biology from within, making it harder, if not impossible, to trace.

Is this crossing a moral line in sport, or is it simply the next stage in human development in competition?

Before addressing this dilemma, we must first understand how it works on a biological level. At its core, gene doping is based on the same fundamentals as gene therapy, except its focus is on enhancement over treatment. The most well-known method is CRISPR technology, which is a "customisable tool that lets scientists cut and insert small pieces of DNA at precise areas along a DNA strand" (del Aguila III, 2025). It is often compared to molecular scissors because it allows us to cut and rewrite specific parts of the genome. These edits are delivered into muscle cells using modified viruses, known as viral vectors. To put it simply, vectors are "vehicles" designed to deliver modified genetic material, such as an enhanced gene, directly into a cell (ASGCT, 2024). By utilising these methods, permanent edits can be created in an athlete's genome, enhancing speed, endurance and strength forever, unlike traditional drugs.

The real impact lies in how these edits can reshape the limits of human performance. In practice, gene doping in athletics would target specific performance pillars, such as endurance, strength and speed - the bread and butter of physical fitness.

## Endurance: the body's ability to keep performing over time

Most students dread the notorious "bleep-test", a brutal measure of endurance. It requires the person to keep running until their body can no longer keep up, essentially till failure. Gene doping could change that by rewiring how long the muscles can work before fatigue.

Endurance performance depends heavily on how much oxygen the muscles can access during sustained activity. More oxygen means more aerobic respiration, allowing the muscles to keep contracting for longer without fatigue.

EPO (erythropoietin) is a hormone made in the kidneys which stimulates the bone marrow to produce more RBCs (red blood cells). An increase in RBCs results in more oxygen being carried to working muscles (due to there being more haemoglobin available). Previously, professional cyclists would inject synthetic EPO as a drug to improve endurance, but gene doping takes it one step further. It edits the body to produce more EPO permanently, increasing $VO_2$ max (maximum oxygen uptake) and improving aerobic capacity.

A second route to improve endurance is through the activation of HIF-1α, a gene that switches on when the body senses low oxygen. When scientists artificially stabilise HIF-1α, the body behaves as if it is constantly training at high altitude, causing it to produce more EPO, grow extra capillaries in muscle tissue and improve mitochondrial efficiency.

Interestingly, in 2004, researchers engineered so-called "marathon mice" by activating the PPAR-delta gene (another gene linked to endurance), which reprogrammed their muscle fibres to become more fatigue-resistant. These mice could run almost twice as far as normal mice without additional training, because their muscles were metabolically wired for endurance (NBC News, 2004). In humans, a similar modification would turn a genetic baseline for stamina into a built-in advantage before training even begins.

Together, these changes create a powerful endurance boost, not through external drugs, but by rewriting the body's internal oxygen capacity. The same bleep-test that forces most people to run till they drop would, instead, become a walk in the park for someone engineered for oxygen efficiency.

## Strength: how much force the body can produce

Muscle size and power are negatively regulated by a hormone called myostatin (produced by the MSTN gene), which acts as a biological "brake" on muscle growth (Deng et al., 2017). Under normal circumstances, myostatin prevents muscle fibres from growing beyond a certain size, keeping the body within typical physiological limits. But what if we wanted to change that?

With the flip of this literal genetic "switch", we can turn the MSTN gene off, and the muscle-growth restriction disappears. The result is pronounced muscular hypertrophy, where muscle

fibres grow faster, larger and more densely without extreme stress stimuli (such as weight training).

This is not theoretical. The removal of the myostatin gene already exists in nature, as seen in double-muscled cattle (e.g. Belgian Blues), certain highly muscular dog breeds, and a few rare humans with naturally occurring myostatin mutations, all displaying dramatic increases in muscle mass from early life. In sports, gene doping may be used to provide weightlifters with higher peak force output, removing the previous human limits through enhancement. Rather than adding muscle through training alone, it would shift the baseline biology itself, allowing athletes to start from a level of power that previous generations could only build toward.

### Speed: how quickly force can be produced

Speed, unlike strength, isn't about large muscles, rather how rapidly they can switch on. Sprinting performance depends on two biological systems operating in sync - muscle fibre type and neural signalling. Fast-twitch fibres (Type II) are responsible for explosive contraction, which is exactly what a sprinter needs. The ACTN3 gene (often nicknamed the "sprinter gene"), with the right variant, provides instructions for the alpha-actinin-3 protein, which is found in fast-twitch muscle fibres and is crucial for their development (Yang et al., 2003). Beyond fibre type, genes affecting calcium handling, such as those controlling the RYR1 and SERCA1 proteins, influence how quickly a muscle can contract and then reset for the next contraction (Zhao et al., 2015). Faster calcium cycling allows the muscles to relax and contract quicker than normal, which is where elite sprinting performance is truly won.

Nature has already produced the blueprint for peak sprint biology. A study on cheetahs found that 83% of the vastus lateralis muscles (one of the quadriceps) and 61% of the gastrocnemius muscles (calf muscles) comprised fast-twitch fibres (Williams et al., 1997), explaining their explosive acceleration over short distances. This contrasts with humans, who generally have a balanced 50/50 composition of fast and slow twitch fibres. Through different combinations, an athlete could acquire both speed and fatigue resistance, resulting in a terrifyingly fast, enhanced athlete. With gene doping to improve speed, fast bowlers will be breaking the 100mph speed barrier, basketball players will be breaking ankles more often, and Usain Bolt's 100m record won't stand for long before a modified, hyper-enhanced athlete smashes it with ease.

### But surely we could test for this?

Not so fast! Traditional doping tests work by looking for traces of banned substances in blood or urine. Gene doping completely bypasses that because there is no enhancer within the body. It instead becomes "part" of the body. To even attempt to detect a change in DNA, regulators would need to sequence the athlete's genome and analyse their gene expression in fine detail, but this would look almost identical to natural variation. Distinguishing a genetically edited sprinter from someone born with a rare ACTN3 "super-sprinter" genotype is nearly impossible.

### Ethical issues can arise

Where is the boundary between healing and enhancing? Where someone may undergo gene therapy to overcome muscular dystrophy, is it cheating for a healthy athlete to undergo genetic modification as well? The line between medical treatment and performance enhancement becomes increasingly blurred as technology becomes more available.

Furthermore, wealthy nations, federations, or even individual athletes could access elite bioengineering long before others, due to their affluence. This further heightens the inequality in sport caused by financial status, as the rich can afford biological upgrades while others are limited to training alone, widening the gap between engineered and unenhanced competitors.

Most importantly, to properly detect gene doping, regulators would need access to the athlete's genome. This crosses into bio-surveillance, which makes the line between testing and genetic intrusion unclear, because the only way to prove innocence would be to reveal deeply personal DNA information that could also disclose ancestry, disease risk, or future health traits. The question becomes not just how to enforce the rules, but whether any sporting authority should be allowed to "search" inside someone's genome at all.

Yet some argue that gene editing doesn't undermine fairness at all. Gene doping is possible, but undetectable. So what counts as a "fair" competition? Take Usain Bolt, Michael Phelps and Mo Farah for example, who are all, undeniably, genetically gifted. Is it not pure luck that they had been born with their lung capacity, limb proportions and muscle fibre composition? If Michael Jordan had been 5'9 instead of 6'6, his world-class basketball career would simply not exist in the same way. Sport and athletics already reward genetic winners. In this view, gene editing could simply equalise the advantage some have, by allowing others to access the "lucky mutation" that some had been born with. Enhancement could become a corrective, not a corruption, narrowing the gap between the genetically privileged and everyone else.

However, even this more optimistic view leads to a deeper dilemma. If enhancement becomes normalised in the name of fairness, the nature of competition begins to shift: are we still testing human ability, or the quality of the technology behind the athlete? Are we competing between individual athletes, or the laboratories behind their performance? Gene editing may equalise opportunity, but it threatens to turn sports into a contest of genetically engineered, machine-like human beings.

In the end, the future of gene doping will depend less on what science can do, and more on what we decide sports is meant to represent.

"Is this crossing a moral line in sport, or is it simply the next stage in human development in competition?"

# I (almost) died to a Chicken

The Biology behind Allergies and Anaphylaxis

Written by Shyam Ashokan | Edited by Kaivalya Pullakandam

Yes, you did read that right. As someone living with several allergies – from the normal suspects of hay fever and nuts to the rarer allergies such as chicken and even turkey – I've had more close calls than I'd like to admit. It also makes me one of roughly 6% of the UK population (around 2.4 million people) living with food allergies[1]. To most, allergies are a mild nuisance - maybe an excuse to not go outside in the spring - but these seemingly harmless reactions can escalate frighteningly quickly, quite literally leave me a breath away from death. Yet few stop to consider what's actually happening inside the body – the immunophysiology of allergies and anaphylaxis.

## What is an Allergy?

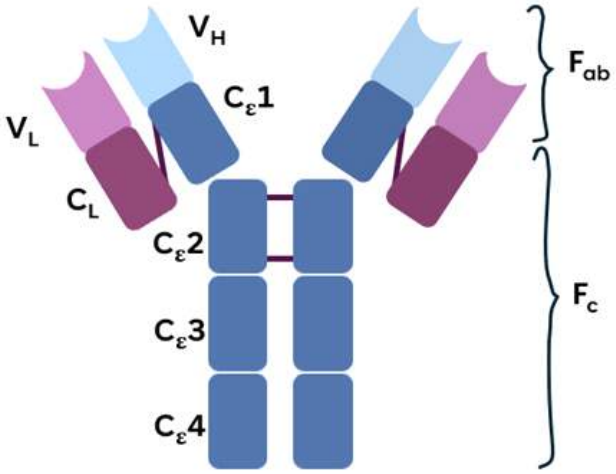
An allergy refers to a hyperactive or inappropriate immune response to a normally harmless substance[2]. These substances – termed innocuous allergens – can range from animal dander and pollen grains to proteins in foods. The body, in an exaggerated response to these allergens, misidentifies these substances and launches complete immune assault – visible to us as allergic reactions. Allergies in common parlance refer to Type I "Immediate Hypersensitivity," a group of 4 major immunological classes that differ in their relevant antibody or T-cell involvement (e.g. sarcoidosis and contact dermatitis are T-cell based Type IV reactions). Type I reactions can then be subdivided into conditions like food allergies, hay fever[3], eczema[4], asthma and anaphylaxis.

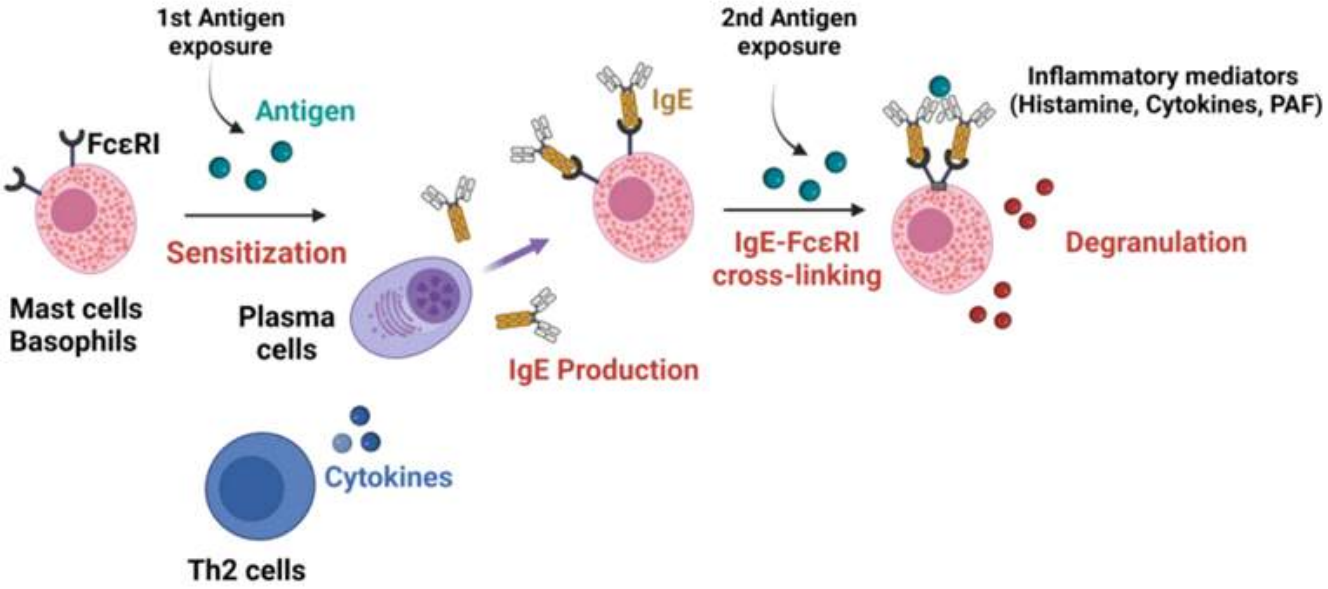
The term atopy – directly translating to "out of place" – is often interchanged with allergy but specifically refers to the genetic predisposition some people carry to develop allergic diseases[5]. Those described as being atopic own immune systems that tend to overproduce certain antibodies when exposed to innocuous substances. This inherited tendency explains why allergic conditions often arise together – particularly hay fever, eczema and asthma which form the "atopic triad"[6]. Atopic rhinitis or hay fever, atopic dermatitis or eczema and atopic asthma each represent inflammation in distinct parts of the body – the skin, nasal passages and bronchioles respectively – and can result in intrusive symptoms like itching, swelling, hives, congestion and respiratory distress. Symptoms and causes will no doubt differ between both allergies and individuals, but all arise from the same fundamental biological trigger, and to understand why we'll have to take a detour into the science of antibodies.

## Immunoglobulin E and Histamine

Antibodies are specialised immune proteins (also known as immunoglobulins or Ig) that function as molecular locks to antigens on the surface of other cell bodies and pathogens. Each antibody comprises of a Y-shaped protein, with some distinct differences depending on the class of human immunoglobulin: IgA, IgD, IgG, IgM and – notably for us – IgE[7,8]. These are made of 2 identical, longer polypeptide "heavy" chains (shown in blue) and 2 shorter "light" chains (shown in purple), joined together via disulphide bridges between residual Cysteine molecules (Cys-S-S-Cys) and dipeptide bonds. The upper regions of each peptide chain form the variable domains VL and VH which are collectively referred to as the antigen-binding fragment (Fab). The Fab , as the name suggests, varies between antibodies, its active site being complementary to specific antigens (e.g. on the surface of albumin proteins in chicken) for the binding process. The lower tail of the molecule forms the constant or crystallisable fragment (Fc), which unlike the Fab , stays consistent throughout antibodies and acts as a point of receptor-based communication to activate various immune cell mediator functions. Particularly, for IgE the tetrameric Fc gives it a particularly high affinity to bind to mast cell and basophil receptors, creating FcεRI receptor complexes[7].

Say a pollen grain were to bind to the variable region, molecular changes in the Cε4 domain of the Fc will eventually lead to the release of histamine, a chemical mediator that binds to H1 histamine receptors responsible for hyperdilation of blood vessels (leading to hypotension), increased vascular permeability (leading to inflammation) and bronchoconstriction (restricting airflow and inhibiting ventilation). Paradoxically, this is an attempt to clear out the allergen from the body by increasing blood flow and to that region and along with it T-cells to kill the pathogen. However, the body is unable to recognise allergens are completely innocuous; coupled with cytokines that stimulate inflammatory responses, this can produce severe local symptoms at the region where the allergen had entered[2]. However, when this localised response becomes systemic, it moves onto the far more dangerous condition of anaphylaxis.






## The Anaphylactic Map

Anaphylaxis is the life-threatening extreme end of Type 1 Hypersensitivity, when histamines, cytokines and prostaglandins systemically flood the body. Imagine we have our villainous chicken allergens again, likely the albumin as mentioned earlier, entering the body and are perceived as a threat by the immune system. During this initial antigen exposure, the allergen antigens bind to the mast cell receptors in a process called sensitisation, prompting immune cells to produce cytokines that proliferate new primed IgE antibodies specifically tailored towards the chicken as a chemical trap. On the re-exposure of the allergen (often less than a minute), its antigens readily bind to the Fab, cross-linking with many mast cells to form this large repeating mesh of histamines, cytokines and mast cells. This triggers a chemical cascade – and the result is explosive. The mast cells undergo degranulation and release vast quantities of histamine and cytokine granules into the surrounding tissue and bloodstream, leading to a positive feedback loop that accelerates the onset of the reaction around the body by recruiting more immune cells[9].

This then has detrimental impacts on the body, notably respiratory distress where both the lung airways and throat "close up", causing choking and inhibiting breathing. The systemic release of histamine also greatly reduces the blood pressure by the widespread vasilodilation of blood vessels, reducing the blood flow to the brain in a process known as cerebral hypoperfusion. This is when the brain tissue become oxygen deprived and this localised ischemia can cause intense dizzines, confusion or sudden unconsciousness[10]. This is particularly perilous; an unconscious individual under anaphylaxis will be unable to signal or self-administer their auto-adrenaline injector, more commonly known as an EpiPen, a device that administers a concentrated dose of adrenaline to quickly constrict blood vessels and airways to almost immediately alleviate anaphylactic symptoms[11]. Without it, anaphylaxis can become fatal, not in hours but in minutes.

## Conclusion

Every meal, every walk home past the chicken shop, every minor event that people never have to think about. For those of us living with severe allergies, the war between our antibodies and allergens is at risk of outbreak at any moment. It's frankly terrifying. Yet to understand what happens beneath the surface transforms that fear into knowledge. While almost dying to a chicken may sound absurd, it serves us a reminder that the molecular basis of our biology, however fragile or temperamental, is still one of the most fascinating systems worth understanding.

"SYMPTOMS AND CAUSES WILL NO DOUBT DIFFER BETWEEN BOTH ALLERGIES AND INDIVIDUALS, BUT ALL ARISE FROM THE SAME FUNDAMENTAL BIOLOGICAL TRIGGER"

# What Sitting All Day Does to Someone

Written by Gajan Gogulen | Edited by Quang Tran

At one point or another, most of us have been guilty of it. The "it" being looking back on our day and realising that we can't remember doing anything that wasn't sitting and watching TV or using some other form of technology. Most of the time, we tend to brush these days off as a one-off and try to make sure it doesn't happen the next day, but from experience, it's fair to say these sedentary days happen all too often (the mean daily duration of sedentary behaviour is 8.3 hours among the Korean population and 7.7 hours among the American adult population). Studies suggest, however, that a sedentary lifestyle should be avoided at all costs, as it can cause a whole host of problems.

One of these problems is a significantly increased risk of cardiovascular disease (it is estimated that approximately 35% of coronary heart disease mortality is due to physical inactivity), partly caused by an increase in blood pressure levels. Exercise means that the heart muscles are strengthened, so the heart doesn't have to work as hard to pump blood, which lowers blood pressure. However, since people leading sedentary lifestyles aren't getting as much exercise, the inverse happens, causing their blood pressure levels to rise. In addition, arteries become stiffer in people who are sedentary, meaning blood pressure levels further increase as the artery cannot expand to reduce the blood pressure. The higher blood pressure leads to the artery walls becoming damaged, leading to plaque forming on the artery wall. This plaque can rupture, leading to a blood clot, and the blood clot can break off, and if it gets stuck in the coronary artery, a heart attack would occur. In addition, studies show that waist circumference increased by 3.1 cm with a 10% increase in sedentary time. The waist:hip ratio is used to determine someone's risk of CVD, and an increase in waist:hip ratio indicates that someone is at greater risk of CVD, showing the indirect link between time spent sedentary and CVD, as being sedentary is shown to increase one's waist:hip ratio. Furthermore, sitting for long periods and being physically inactive causes LDL cholesterol (the so-called bad cholesterol) levels to rise and HDL cholesterol (the so-called good cholesterol) levels to fall. A study showed that women at medium and high physical activity levels had 6% and 9% higher HDL-cholesterol levels, respectively, as compared to sedentary women. HDL particles pick up extra cholesterol in the bloodstream and deliver it to your liver, meaning there is less cholesterol in blood vessels, which means the risk of CVD decreases. In contrast, when sedentary, HDL levels are lower, meaning less cholesterol is removed from blood vessels. This causes dyslipidaemia, which refers to abnormal levels of lipids in the bloodstream, causing blood vessels to narrow and increasing blood pressure, which, as previously explained, can lead to heart attacks. On a positive note, as little as 75 minutes a week of light physical activity could reduce cardiovascular risk by as much as 14%, based on studies, which can be explained by HDL cholesterol levels rising as a result of physical activity.

Another effect of sedentary lifestyles is the onset of musculoskeletal disorders. Osteoporosis (a condition that weakens bones) is considered more likely to occur in people living a sedentary lifestyle. A study on white women aged 65 or over found that the women who walked regularly had about a 30% lower risk of breaking a bone, and that women who stood for four hours or less each day had double the risk of fracturing a bone compared to their counterparts. Bones experience mechanical loading (the application of external forces or stress to a material or structure) when doing physical activity. More mechanical loading on the bones stimulates osteoblasts, which are bone-building cells, meaning more bone mineral content is made. However, in the case of people leading sedentary lifestyles, fewer osteoblasts are stimulated and more osteoclasts (cells which dissolve and break down old bone tissue) are stimulated, meaning people can lose bone mineral content and thus have a lower bone mineral density, increasing the risk of bone fractures. In contrast, a study on tennis players found they have significantly more bone mineral content in their dominant arm (229.0 ± 43.5 g) compared to their non-dominant arm (188.2 ± 31.9 g), showing how significant it is to have some sort of mechanical load

(by staying active) on our bones, as it can make a huge difference to our bone mineral density and bone strength as a result, and lead to us being less vulnerable to bone fractures.

An increased risk of cancer has also been linked to sedentary lifestyles. Compared with men who mostly sit during their main work or occupation, men who sit half of the time experienced a 20% lower risk of prostate cancer, and furthermore, about 25% of cancer cases globally are due to excess weight and a sedentary lifestyle, based on data from 2008. In a separate study, participants with obesity were 2.41 times more likely to develop colorectal cancer than participants with under- and normal weight, and their risk increased by 7% for each 1 kg/m$^2$ increase in BMI. Clearly, there's a strong link between being sedentary and cancer risk. Generally, people with higher BMIs (which can be caused by a sedentary lifestyle) have a larger amount of adipose tissue (another word for body fat). The adipose tissue releases pro-inflammatory cytokines, which are signalling molecules produced by the immune system that promote and heighten inflammation to fight infection. A study showed that people with a BMI of less than 30 produced 1.3 pg/ml of IL-6 (a

type of pro-inflammatory cytokine), but people with a BMI of more than 35 produced 2.2 pg/ml of IL-6. The release of pro-inflammatory cytokines isn't inherently a bad thing; the issue is that because people with higher body fat percentages have more adipose tissue, they release more pro-inflammatory cytokines, which then becomes a problem, as cytokines can cause DNA damage. If someone has more cytokines being produced in their body, it becomes more likely that DNA damage will occur. Pro-inflammatory cytokines cause DNA damage by triggering reactive oxygen species (ROS). The ROS then damage DNA, and the damaged DNA can cause cancer by leading to mutations that disrupt cell growth and division control.

On top of the physical issues caused by a sedentary lifestyle, our mental health can also be affected. A six-year cohort study on university graduates in Spain revealed that participants who were seated for over 42 hours per week were 1.3 times more likely to experience mental disorders such as stress, anxiety, and depression compared to participants who sat 10.5 hours per week. Whilst researching, I noticed that most studies would caveat their findings, saying that more research would need to be done to provide conclusive evidence linking sedentary lifestyles to mental health issues, indicating that the idea that there is a strong link between the two isn't certain (however, most of the studies were certain there was a link, just not certain it was a strong link). Partaking in physical activity triggers the release of endorphins, which are the body's own version of a painkiller and create feelings of pleasure. Endorphins are released during physical activity in response to the physical stress on the body caused by the activity, to reduce feelings of pain during exercise. In contrast, people who lead sedentary lifestyles will not be producing the same levels of endorphins, leading to fewer feelings of happiness that the endorphins cause (studies suggest 20–33% lower odds of depression for active groups).

A separate study asked people with type 2 diabetes (however, the fact that there will be a health improvement is applicable to everyone) to follow different regimens: one "Sitting" regimen and one "Sit Less" regimen. The Sitting regimen meant the participants sat for 14 hours a day, and the Sit Less regimen saw participants sit for 9.3 hours a day, with 4.7 hours spent on light walking (2.2 hours) and intermittent standing (2.5 hours). In the Sit Less regimen, the duration during which participants were hyperglycaemic was reduced (food was standardised to ensure a fair test). Hyperglycaemia occurs when the level of sugar in your blood is too high and can result in reduced cognitive function due to reduced blood flow to the brain. A study on rats with prolonged exposure to hyperglycaemia followed by a return of glucose levels to normal values showed glucose transport to the brain was inhibited by 20%. This reduction in glucose means less respiration can take place, leading to the brain not having as much energy and thus not being able to think as clearly, and therefore making more mistakes.

I hope it is evident by now that leading a sedentary lifestyle is incredibly likely to lead to health problems, so what are some of the things people who sit too much can do to try to improve this? The obvious answer of moving more (and maybe in some cases eating less) is correct, but there is also nuance to be explored. In one study, 11 women were asked to sit uninterrupted for five hours and then have their reaction times tested. Separately, they were told to sit for five hours, but have the sitting time broken up with three minutes of treadmill walking every 30 minutes. Reaction times were 210 ms quicker in the condition of having to take breaks between sitting, reflecting how breaking up time spent sitting down is a viable way of reducing the negative effects of sitting for long periods. In addition, the idea that doing very high-intensity exercise whilst spending many hours sitting is equal to doing lighter-intensity exercise with fewer hours spent sitting isn't true. A study on 1,422 people found that successful weight maintenance following weight loss was associated with avoidance of common sedentary behaviour (like watching TV) and wasn't related to self-reported moderate to vigorous physical activity or any other type of exercise. This suggests the most important factor determining health outcomes is reducing sitting time as much as possible, instead of trying to shoehorn in an exercise session to balance out extended periods of sitting.

In addition, combining exercise with taking breaks from sitting is another valuable extension that further improves health outcomes. A study on 67 Australians found that combining one hour of exercise with scheduled breaks every 30 minutes caused significant improvements in working memory and executive function compared to just doing an hour of exercise, reinforcing the idea that reducing time spent sedentary is the key factor. Furthermore, burning an equal number of calories in two different ways doesn't mean that both of these ways are equally effective. In the previously mentioned Sit Less regimen, there was a third regimen called "Exercise", where participants would do three consecutive 20-minute bouts of moderate to vigorous cycling. In terms of calories burned, the Exercise regimen and the Sit Less regimen were similar, yet the health outcomes of Sit Less proved far greater in terms of reducing the duration in which participants were hyperglycaemic, indicating that taking breaks between periods of sitting is a huge difference-maker in improving health outcomes. Another consideration is the intensity of exercise: a study showed that 35 minutes of light-intensity cycling (30% of maximum oxygen uptake, which is a measure of exercise intensity) caused 30% more brain glucose uptake compared with higher-intensity cycling (75% of maximum oxygen uptake). In one study of dementia patients in a nursing home setting, a nine-month tai chi intervention successfully preserved cognitive function relative to a control group who performed simple handicrafts. This suggests that choosing light-intensity exercise that focuses more on physical movement than providing a mental challenge is the best route for optimal health outcomes.

Finally, here is an action plan you can follow to reduce the likelihood of dealing with health issues caused by a sedentary lifestyle: aim for around 150–300 minutes of moderate-intensity physical activity per week (the WHO recommends this or more if you are able, and to try to make this exercise daily), and look to take breaks after sitting for long periods of time (around three minutes of walking after an hour of sitting seems to make a significant difference based on studies). Mentality-wise, the best way to approach fixing a previously sedentary lifestyle is to take a holistic view and try to implement healthy behaviours throughout the day instead of blocking out a couple of hours per day for being healthy and then falling back into old behaviours for the rest of the day.

I hope this helps anyone looking to become more active and trying to spend less time in their chair.

# “Trapped in your own body”

Written by Jayden Bheekha | Edited by Quang Tran

Trapped. Suffocated. Hopeless. You look around for help, but no one sees you. You try to reach out, but you can’t. It is as if you are caged, unable to cry for help. The word ‘paralysis’ makes us wonder what it would be like for us. Naturally, we feel afraid to think about things that seem so out of reach for us. However, this article is going to provide a clearer idea of what paralysis is truly like for patients and is going to explore the developments in treatments for paralysis.

It is first worth realising that someone can become paralysed even when it seems most unlikely. This reflects the story of Christopher Reeve, a former A-list actor, most well-known for his role as Superman in 4 movies. In 1995, Reeve was injured in a near-fatal horse-riding accident that left him paralysed from the neck down. He became a symbol of resilience and hope, in contrast to the dreary image of paralysis that some people may have. To fully understand the science behind paralysis and what happened in the tragic case of Christopher Reeve, we must first examine the root causes of paralysis and how it can affect one’s movement.

Paralysis is defined as the “complete or partial loss of function, especially when involving motion or sensation in a part of the body” (*Merriam-Webster Dictionary*). Paralysis is a complex subject; there is often a common underlying cause for most cases. For example, paralysis is often caused by one of the following:

- The brain is unable to relay electrical impulses to the rest of the body due to injuries to the brain.
- The brain can sense touch and other sensations, but is unable to relay electrical impulses to the rest of the body due to injuries to the spinal cord.
- The brain can neither receive electrical impulses nor relay them due to injuries to the spinal cord.

It is therefore neurological injuries that underpin most cases of paralysis.

Out of the almost 5.4 million people paralysed worldwide (*World Health Organisation*) and almost 1.5 million people living in the US with spinal cord injuries (*“Spinal Cord Injury Prevalence in the U.S. | Reeve Foundation”*), there are many different types of paralysis. One example of paralysis is monoplegia. Monoplegia is the paralysis of a single limb, most often caused by cerebral palsy. Cerebral palsy is a neurological disorder caused by damage to the brain, usually during foetal development, birth, or early infancy. It mainly affects motor control, muscle tone, coordination, and sometimes posture (*Maricle et al.*) Damage to areas of the brain such as the motor cortex, cerebellum, and basal ganglia disrupts electrical impulses from the brain to the muscles so they cannot contract or relax as they should. This could lead to monoplegia if neurons controlling a single limb are damaged. As electrical impulses along the motor neuron to that limb are weak, inconsistent or absent, the muscle cannot contract or relax and becomes paralysed (*Gage et al.*).

The reason for the paralysis being limited to only one part of the body stems from the brain. Focal brain lesions, damage to a specific region of the motor cortex or the descending corticospinal tract that controls one limb leads to the impairment of that limb only (*Chien and Barsottini*). Monoplegia can be cured, and so is a temporary form of paralysis.

Some other forms of paralysis include: diplegia, hemiplegia, paraplegia, quadriplegia, and locked – in syndrome.

Quadriplegia is paralysis of all four limbs (*Clinic*) and is the specific type of paralysis that affected the late Christopher Reeve. It was understood that after his equestrian injury, his cervical vertebrae became compressed, and so electrical impulses could not travel down the spinal cord to the muscles in his limbs. Quadriplegia also hindered his ability to breathe by himself, and so he needed to be administered to a ventilator for the rest of his life. However, his brain function remained normal – he was mentally sharp and able to communicate.

Furthermore, we frequently associate paralysis with locked-in syndrome, even though it is one of the least common types of paralysis – only about 1 in 1,000,000 people develop the disease (*“Orphanet: Diseases”*). Locked-in Syndrome is a rare neurological disorder in which a person is unable to move their body except for their eyes, which is why we often relate paralysis to being ‘trapped in your own body’. The disease is usually caused by damage to the brain stem, or specifically the pons, which connects the cerebrum with the spinal cord or cerebellum. Electrical impulses that determine voluntary movements pass through the pons, and so damage to the pons prevents these electrical impulses from reaching the muscles despite the fact that the cerebral cortex is intact.

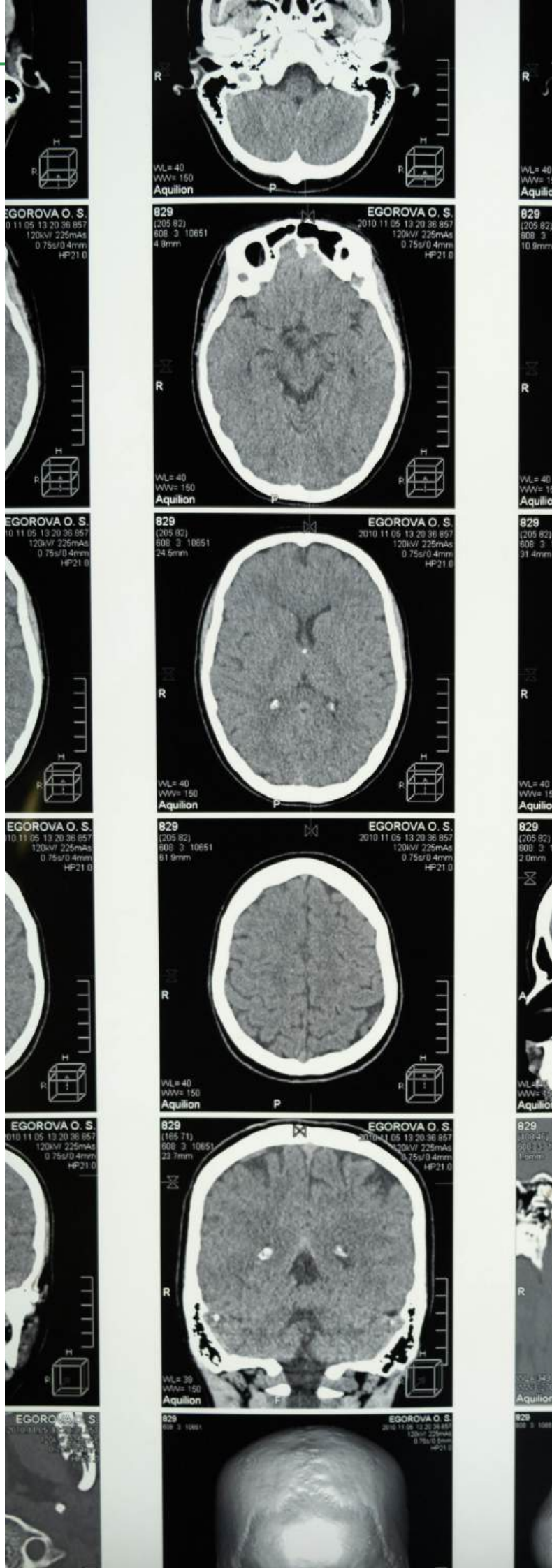
Whilst a valid cure for locked-in syndrome has not been discovered yet, many paralysis patients with other forms of paralysis can be treated. One example of a historically used treatment is steroid

use, as it reduces inflammation and prevents secondary injuries to nerves after an incident (*Zou et al.*). However, steroids are less frequently used in modern medicine due to their limited ability to improve motor function and plethora of potential side effects such as blood clotting, gastrointestinal bleeding and pneumonia (*Suberviola et al.*). More commonly used forms of treatment are physiotherapy and occupational therapy. Physiotherapy prevents muscle stiffness and allows for the strengthening of muscles in compensation for the paralysed ones. Occupational therapy helps paralysed patients perform daily tasks so that they can eventually do these by themselves without the need for intervention from occupational therapists, which would not only maximise their independence but also ensure that they remain confident in themselves.

There are also some more advanced treatments that are in the early phases of clinical trials, such as stem cell therapy and exoskeletons. Stem cells are undifferentiated cells that can become specialised cells in the body. Embryonic stem cells or induced pluripotent stem cells (iPSCs) can be transplanted to paralysed patients to differentiate into functioning neurones. Scientists believe that this could become a method of restoring pathways for electrical impulses from the central nervous system to the muscles. There are ethical considerations regarding stem cell therapy that limit its clinical use, including whether it is ethical to extract stem cells from an embryo. The progression of stem cell therapy in curing paralysis is also somewhat restricted due to its inconsistent effect in curing animals.

"Be brave, be determined. Overcome the odds."

The famous final words of Stephen Hawking accurately mirror the attitude of paralysed patients worldwide, including that of the late Christopher Reeve, who overcame the odds and lived for 9 years while being paralysed. The future is promising for the development of treatments for paralysis, fuelled by the inspirational patients who will be treated.



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