



Hitting The Wall

How fast can we go?

By Joe Robinson

In our modern world athletes just keep getting better, humans are running or swimming or cycling faster, jumping higher and throwing further and world records are being smashed at almost every athletics event. Can this constant improvement continue forever? In this article I will explore the maximum human potential, mainly in the men's one hundred metres event, and ask the biggest question in athletics, when will athletes hit the wall?



First we must look at the possibilities of a man. The current world record belongs to Jamaica's Usain Bolt with a time of 9.58 seconds (10.4 ms⁻¹) so it is clearly possible that a man could run one hundred metres in ten seconds. However, can a man run the one hundred metres in one second (100 ms⁻¹)? No, it just isn't possible for a man to travel that quickly on his own. What about five seconds (20 ms⁻¹)? Again, this is extremely fast and not within human boundaries. It becomes much more difficult to predict when we reach nine seconds (11.1 ms⁻¹). Any aspiring athlete or coach will say "yes, we can keep pushing the boundaries, potentially down to nine seconds." Science tells us no.

It is certain that the current record can still be broken, sports analysts have analysed Usain Bolt and can even find flaws even in this seemingly perfect running machine. Firstly he is slow out of the blocks, secondly his finishing

technique is poor and over-arrogant, thirdly he is only 23 years old, 4 years younger than the recognised runners' peak and there are many questions over his training and diet. However, if you give this problem to biologists and mathematicians they have predicted the human limit. Independent research by a biology professor, Mark Denny, from Stanford University and research by a group of scientists from the Institute for Biomedical Research and Sports Epidemiology (IRMES) in Paris has predicted the human limit by analysing thousands of statistics. By looking at the biology of men along with technique and statistics combined with the biology, technique and statistics of racehorses and greyhounds they have calculated that the 100m wall will be hit at 9.48 seconds (10.5ms⁻¹). Not only this but they have predicted this will happen in 2019.

Their theory is that human evolution, diet and training are now as good as they can be and our improvement curve will now level

out. Humans as a species are getting taller but the scientists have figured out that the current biomechanics suit performance as well as they ever will.

They have also analysed the other athletics events to reveal that events such as long jump, pole vault and 10,000m have hit their limits whereas 100m breaststroke, the marathon and 100m backstroke will not be broken for the last time until at least 2080. This does not just affect the careers of athletes but it could have an affect on television, spectators love to see records broken and if this stops happening then interest in athletics may decline.

The same thing happened with racehorses and greyhounds but their boundaries were extended with one simple solution, selective breeding, every elite racehorse or greyhound is the carefully planned offspring of elite parents. In human athletics the elite are results of coincidence, the genes just click for athletics by chance but if

we are ever to overcome our limit, athletes must be selectively bred. This obviously raises an ethical problem and is an entirely different matter. Enjoy seeing world records while you can and from 2019 we will always wonder if the combination of mathematics and biology can be proved wrong.

Did You Know...?

A flea can jump 350 times its body length. This is the equivalent of a human being able to jump the entire length of a football pitch.

A cockroach can live for up to nine days without its head before it dies of starvation.

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Micro RNA

What Really Goes On Inside Cells?

By Tom Irons

Most of us have heard of DNA as being the 'building blocks of life' and the 'molecules of inheritance'. However, what is sometimes not mentioned is that, in humans, DNA would be completely useless if it wasn't for the mediator molecule of RNA. It isn't DNA which controls the rate of biological reactions or that connects muscle to bones, but proteins. And how is DNA turned into proteins? This, the 'central dogma of molecular biology' as it is sometimes called, can be summed up simply as DNA makes RNA (in a process called transcription) and RNA makes protein from amino acids (in a process called translation).

Each gene codes for a specific protein – so each gene can be described as a discrete section of DNA whose sequence of organic bases (the 'rungs' on the double helix ladder) is translated into the correct sequence of amino acids to make a specific protein. This might appear relatively simple however things start to get messy when it is revealed that 98% of human DNA is defunct – only around 2% is actually translated into proteins, which control the characteristics of the individual. These non-expressed pieces of 'junk DNA' are known as introns, where as the minority that is expressed as proteins are known as exons.

It took time to realise what the function of some of this junk DNA was, and this discovery came from Victor Ambrose of the University of Massachusetts in 1993 who was studying worms. He was working on a species of worm known as *Caenorhabditis elegans* which is just about the perfect scientific specimen. It takes three days from egg to adult hood and reproduces in another three days, laying hundreds of eggs. It is transparent and tiny – it only has around 950 cells as an adult so its entire life can be observed under the microscope, making it a perfect specimen for observing the effect of mutagens (something that causes a genetic mutation such as x-rays).

He had noticed a grotesque mutation called a 'bag of worms' in which the worm is unable to lay any eggs (it is hermaphrodite i.e.



it contains male and female sex organs so can self-fertilise). Rather, the eggs hatch inside it and consume the parent from the inside. It was eventually (after 13 years) found that the gene which mutated, called *lin-4*, and was responsible for the bag of worms was tiny. At only 22 nucleotides (individual molecules of DNA containing one organic base) long, it was way too small to code for a protein – a typical gene coding for a protein should be 100 times longer. Whilst it is small, it has a massive effect – it causes the bag of worms.

It took until 2001, after further investigation, for the term 'micro RNA' to be coined and their massive function to be understood in more detail. It is now thought that 90% of so called 'junk DNA' is actually transcribed into miRNA (post-transcriptional modification of 'pri-miRNA' by the use of enzymes gives the final miRNA molecule). But how do these minute RNA molecules control the chemistry of cells? An answer can be found by going back to the example with the bag of worms.

Further research by Ambrose and his team showed that a mutation in another gene in the *elegans* called *lin-14* effectively cancelled out the mutation in the *lin-4* gene and there was no nasty end product. This suggested that there was some sort of connection between them, which was broken when one of the mutated but fixed when either none or both of them had mutated. Some of his colleagues then discovered that *lin-14* was not another piece of miRNA, but a standard piece of messenger RNA – the molecule that conveys genetic information from the DNA to a sequence of amino acids during protein synthesis. It was found that, under normal conditions, the sequence of base

pairs on the *lin-4* gene was complementary to some of the *lin-14* gene i.e. it had exactly the correct sequence of organic bases to pair with that on *lin-14* (there are only two possible base pairs – AT and CG).

The conclusion was that, when *lin-4* and *lin-14* were unmutated, or both had the same mutation, the piece of miRNA could couple to the piece of mRNA along a small part of its length, to effectively 'switch off' the production of that protein. In the bag of worms scenario, the *lin-4* was mutated but the *lin-14* was not, so the miRNA could not couple to the mRNA as it was not complementary, resulting in that protein being made and the offspring eating their way out of the parent. To show that this was not a one off, the research team found another example of miRNA in *C. elegans* where a mutation in a short, 21 nucleotide-long gene, resulted in incorrect miRNA being produced and the worm to explode during the larval stage of its life. It is the state of an miRNA molecule, on or off, which determines how it affects other genes and eventually the characteristics of the organism.

There is evidence that these miRNA molecules are related to a huge number of human diseases, most notably a whole host of different types of cancer and heart disease. It has been found by Todd Golub, research scientist at the Dana-Farber Cancer Institute, that in a large number of cancers, miRNAs seem to be less active than they are in healthy tissue, however which miRNA molecules it is depends upon the type of cancer. For example, research has shown that a type of leukaemia called chronic lymphocytic leukaemia is caused by the deletion of two genes that code for micro RNA, not protein. This mechanism could be used to yield effective chemical treatments for cancer, by supplying normal miRNA molecules to reduce the activity of tumours.

Studies into the heart muscle protein myosin (the most studied muscle protein) found that there was micro RNA coded for in one of the introns of the gene, which is important in controlling functions of the

Life Before DNA

Into The World Of RNA

By Ajantb Varathanathan

During the days of the early Earth, the Earth was constantly being bombarded by asteroids. These asteroids and comets where rich with volatile substances, that were essential for the existence of organic materials. The warm waters in which these organic materials thrived came to be known as the primordial soup. This primordial soup provided building blocks for the RNA world.

RNA and DNA are incredibly similar. Though there are a few major differences. RNA has only one strand compared to DNA's double helix. There are a few other small differences, but they are incredibly closely related. Like DNA, RNA also carries genetic information.

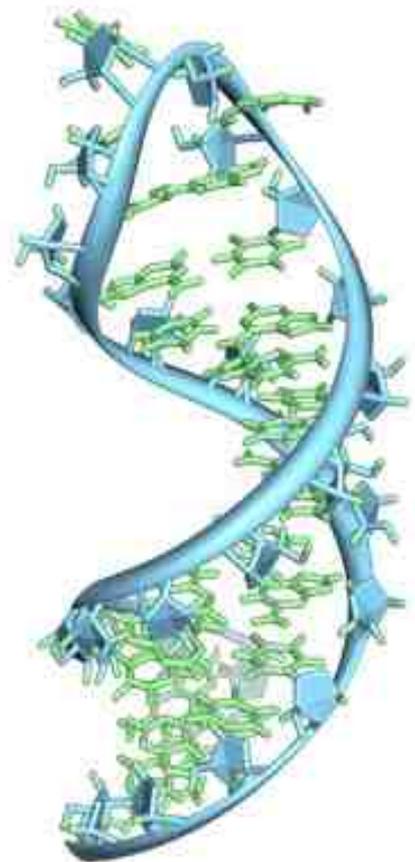
At first RNA consisted of only short nucleic acids that had properties that allowed it to accelerate reactions. RNA had the ability to code information, but could only go as far as primitive membrane bound systems. Although it was only a soup of interacting chemicals, it dominated the

world for millions of years, a period known as the RNA world. There were many different types of Ribozymes (RNA molecules that catalyse a chemical reaction) around, some catalysing more useful reactions than others. There would have been rivalry between the different Ribozymes, with the less effective Ribozymes being outcompeted. One type of Ribozymes managed to self replicate. This allowed reproduction and also natural selection. Soon, the Ribozymes became more and more complex, and started to become more bacteria like. As the complexity of these Ribozymes increased, a hardier molecule was needed. Enzymes allowed the RNA to become more robust and stable and formed DNA. Soon RNA was to lose its role as the dominant data storage medium, to be replaced by DNA. Even today RNA carries out a major function within our cells. Cellular life today uses ribosome to produce enzymes, which in itself is based on RNA. Different types of RNA, such as mRNA and tRNA still perform important processes today.

The RNA world is far from dead; RNA viruses still vastly outnumber cellular life. The RNA world is only theoretical. Its existence

was so far back that it can not be proven, but only experimentally demonstrated. Experiments being carried out today are suggesting that TNA may have emerged before RNA. TNA contains properties that were more probable on the early Earth. This simpler form of storing genetic information may have emerged first. This may mean that RNA arose from TNA.

One way or another DNA came to dominate, and has allowed the existence of very complex life to occur, possibly upon the building blocks of the RNA world.



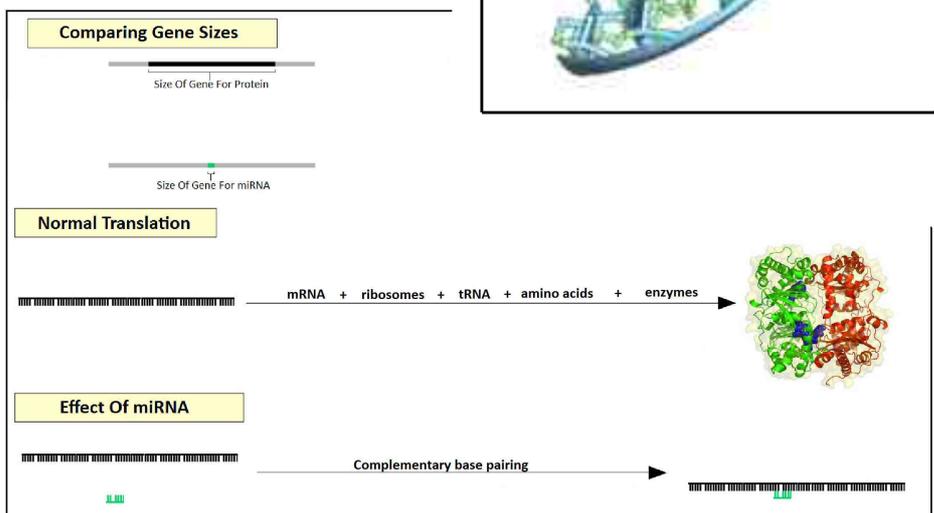
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muscle such as the ability of the heart to respond to injury. Genetically engineered mice have been created that are effectively immune to heart failure by removing an miRNA gene from their genome.

These molecules appear to have a powerful influence over many aspects of life and the chemistry of cells, having the ability to inhibit the synthesis of a particular protein. There are a large number of potential uses for miRNA technology, particularly in the fields of new treatments for any disease which has a gene based element (that includes cancer because cancer is caused by the genetic information in cells – a somatic mutation when cells divide can cause the formation of a tumour). Also, miRNA technology could potentially in the future be used to improve the efficiency of muscles and organs, or guard against diseases such as heart disease.

Did You Know...?

If all the genetic information stored in the nucleus of one human body cell was written out , it would fill around 10'000 volumes of the Encyclopaedia Britannica.



Extinction: is it 'If' or 'When'?

By James Wickenden

Although the extinction of the human race may not be a concept you regularly think about, it has always been a possible threat to our existence.

There are two main theories about how extinction can occur. Firstly there is the competitive exclusion theory, favoured by Darwin. This states that two species with the same ecological requirements cannot coexist for a long period in one region as one species is either better at using the resources it needs or has a larger initial population. An example of this can be found when the North American and South American continents came together, 3.5 million years ago. In this period the South American fauna became almost entirely extinct, replaced by a North American similar species. Human introduction of new species has also caused species to become extinct.

The other common theory is environmental, usually climatic, change. This theory takes extinction as a chance occurrence, brought about by bad luck and not inferior capabilities (the species was in the wrong place at the wrong time). These unfortunate occurrences include increased volcanism, changes in sea level and shifts in the tectonic plates these all can trigger climate change or loss of habitat.

However, sudden and catastrophic change would have to occur for even one species to be wiped out; the environmental change would have to be unprecedented in the species history and sudden enough so there is no time for natural selection to avoid extinction. Such catastrophic events include asteroid and comet impacts. However, life can still survive these disasters: the main characteristics needed in these situations include: a large geographical distribution, large population and the ability to adapt to a wide

range of habitats.

All this been said and the questions remain: Do humans have the right characteristics to adapt to major climate change? And are we well enough placed to fend off a new species? The human race will have to adapt fast, before it is too late, to stop global warming. Failing that, humans will have to adapt to a new lifestyle with higher sea levels and temperatures. Can we pay the price if we fail to act on global warming?



Dead as a dodo? Can humans survive a possible major extinction event?

Bringing Back Extinct Species

By Oliver Blagg

Cloning in biological terms is when bacteria, sea plants, protocista, fungi and other organisms create a clone of themselves. This means they are genetically identical. Similarly speaking, they have the same DNA (Deoxyribonucleic acid). Some multi-cellular organisms have both the necessary gametes to create a new organism but single cell organisms divide in half.

The synthetic way of cloning is making a realistic womb or using current species similar to the extinct species to let the clone grow in its womb. All the information we have available from the animal is the sample DNA; but it is enough. A few unexpected breakthroughs have been made in the last twenty years but they are now classed as useful. It could now be possible, in principle, to create test tube replicas of dinosaurs and other extinct animals.

The first successful cloning was on "dolly" the sheep. After many failures, there came one success. Dolly was a ewe born on July 5th 1996. She died in 2003. The clone aged

quicker as scientists believe, she had a lack of telomeres. Telomeres are the caps on the end of the chromosomes which protect the chromosome from degradation.



Can we bring back the woolly mammoth?

For a long time now, it has been the dream of some scientists to have the dinosaurs and woolly mammoth walk the earth again but now, this could become a reality. There are still serious doubts about bringing extinct animals back to life. For example, growing a dinosaur embryo in a host animal would be very difficult. The host may not be able to support the embryo.

It would also be difficult for scientists to make a synthetic environment necessary for the embryo to grow in. For example, a living host cell is required with all the right conditions for the dinosaur DNA to be im-

planted. In addition, full length dinosaur DNA may well be needed and at present only small fragments are available from fossil sources.

In the event that this difficult and expensive technology was available, dinosaurs may not be the best species to go for; it would cost money to maintain, feed and control them. A better use might be to recreate the primitive bugs and plants that made oxygen out of CO₂ when the world began. If Scientists went after the dinosaur option it could make global warming even worse with all the plants that large herbivores eat.

Other extinct species died for a reason. That reason may still be here and bringing them back to life may make them endure suffering again. For example, the heat of the modern world would kill the woolly mammoths.

In conclusion, at this stage I would like to see cloning applied to the bugs and plants that shaped early life on earth by providing oxygen from CO₂.

Identifying Individuals

By Gregory Brooks

For thousands of years, fingerprints have been known to be unique for every person. This has helped to solve crimes and make new security and recognition systems (like the fingerprint system in the canteen).

Fingerprints are unique for everyone – not even identical twins have the same fingerprints. Many criminals have tried to change or remove their fingerprints but if you burn, grind off or try to remove your fingerprints, they will always grow back looking the same.

It is not just humans that have fingerprints. Monkeys have them too. They look similar to a human's but they are slightly smaller.

There are other ways in which animals are unique. An orca's dorsal fin is different for every whale. It is marked with small cuts and scratches. On many furry animals, the

patterns of colour on their fur are unique too. There are many reasons for unique patterns and markings on animals.

The other reason for these differences is that they are caused by the DNA of an organism. This unique DNA determines the markings and growth of the organism. The whales' genes determine the white marks on the pictures above.

Even if an animal was cloned, with the same genes as another animal, they still might look different; depending on the environment they were brought up in. A plant that is grown in a place with little light will try and grow as tall as possible to reach light. A clone of that plant in a well-lit room will not need to grow as high. Two cloned animals may have the same or similar markings, but if one had been injured before, a scar would change their pattern.

If you look at two people's faces, they will be similar to their parents', the people they have inherited their genes from. However they will not look exactly the same because they have been brought up in a different environment as their parents. The same goes for any type of animal or plant.

These differences between individuals of the same species are useful to zookeepers and park rangers for identifying their animals, but also useful to the general public. Imagine trying to tell people apart when they all have the same face!



Spot the difference!

Medicine In The Media

HIV's 'silent face' could provide the answers to a new vaccine

By Oliver Curwen

A camouflage mechanism that has enabled HIV to remain the hardest disease to combat has recently been recreated by scientists– which means that a vaccine for the virus is more likely to become available.

HIV binds itself to a T-Cell, a type of white blood cell, and it mutates rapidly, making it very difficult to overpower and kill. Where the HIV membrane mutates, only one part of it stays the same – the so-called 'Silent Face'. This is made a prime target for vaccines, but until now it has not been created in a lab.

A synthetic form of the 'Silent Face' could be injected into patients to teach the body how to recognise and neutralize the real thing. In most patients the antigen, or 'silent face', of the virus is not discovered, because of its complex glucose structure. However, one patient was discovered to have developed an anti-body for the GP-120 (the 'silent face') which is called 2G12.



HIV-1 infecting a white blood cell.

"We're turning the virus' shield into its Achilles' Heel" says Professor Ben Davis, the leading scientist behind the research, taking place in Oxford University.

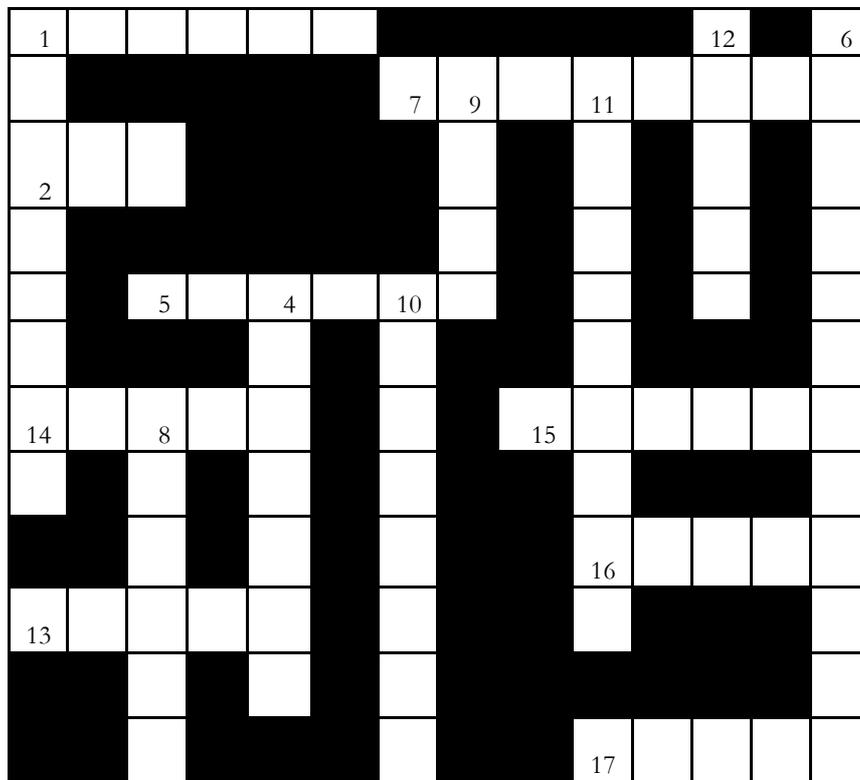
Different forms of the vaccine are now being tested on 30 rabbits at Oxford. "As soon as it [one rabbit] starts showing neutralizing properties, we hope within two years we could see it [the vaccine] being trialled in people."

Did You Know...?

There are more bacterial cells inside a human body than there are actual body cells.

The average human sheds takes 28 days to shed their skin. This means that the skin is shed around 1000 times during a lifetime!

That's Life



Down:

- 1. A complex carbohydrate beginning with 'D'. 8
- 4. The name for sex cells, beginning with 'G'. 7
- 6. A food group beginning with 'c'. 12
- 8. A ball of cells that eventually grows into a baby 6
- 9. A habitat for aquatic animals and freshwater fish. 4
- 10. A cap on the end of the chromosomes that gets smaller until there is none left and then the cell dies (beginning with 't') 8
- 11. A jelly like substance in all cells where all of the chemical reactions take place. 9
- 12. A type of animal that can dislocate its jaw on purpose and it uses its muscles and coils to move itself along. 5

Across:

- 1. Charles '...!', the author of 'The Origin of Species'. 6
- 2. The three axes on a 3D graph. 3
- 5. The cell created when the sperm and the egg cell fuse. 6
- 7. The pad that attaches the embryo to the uterus lining beginning with 'p' 8
- 13. A species that has four legs, and is ridden for the following competitions: dressage, show jumping, cross country. 5
- 14. The gamete that a male organism produces during intercourse. 5
- 15. When an organism has more chromosomes than it is meant to have. 6
- 16. The process that bats use to navigate. 5
- 17. A type of carbohydrate that helps you digest. 5

Editorial

Change has come to 'Life' magazine, like the passing of the seasons. (no Mr Davis hasn't received his A levels yet!) Life is under new management, namely Tom Irons, Joe Robinson and Nick Pepper. We hope you find the magazine as scientifically scintillating as previous editions but a bit more readable as well. A big thank you to everyone who has written in and (begrudgingly) attended our lunchtime meetings.

Hope you enjoy reading,

The editorial team

Keep sending in your articles to Mr. Davis at pdavis@suttonlea.org