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STEM OCTOBER BIOLOGY CLUB

Creating a biology-passionate community for the students, who have that deep desire to break new grounds in the science of life. We aim to change the student's perspective on biology through multiple activities besides helping students who find it daunting to deal with biology.

ABOU

VISION

The club vision is to immerse high school students in a challenging environment that pushes their creativity and curiosity to discover research interests around them.

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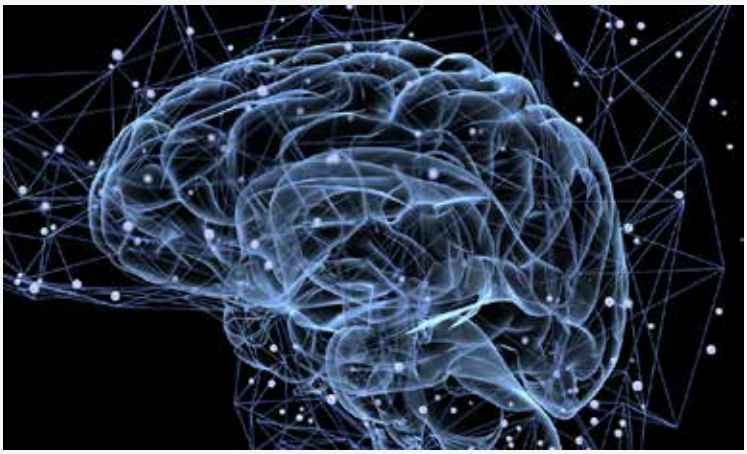

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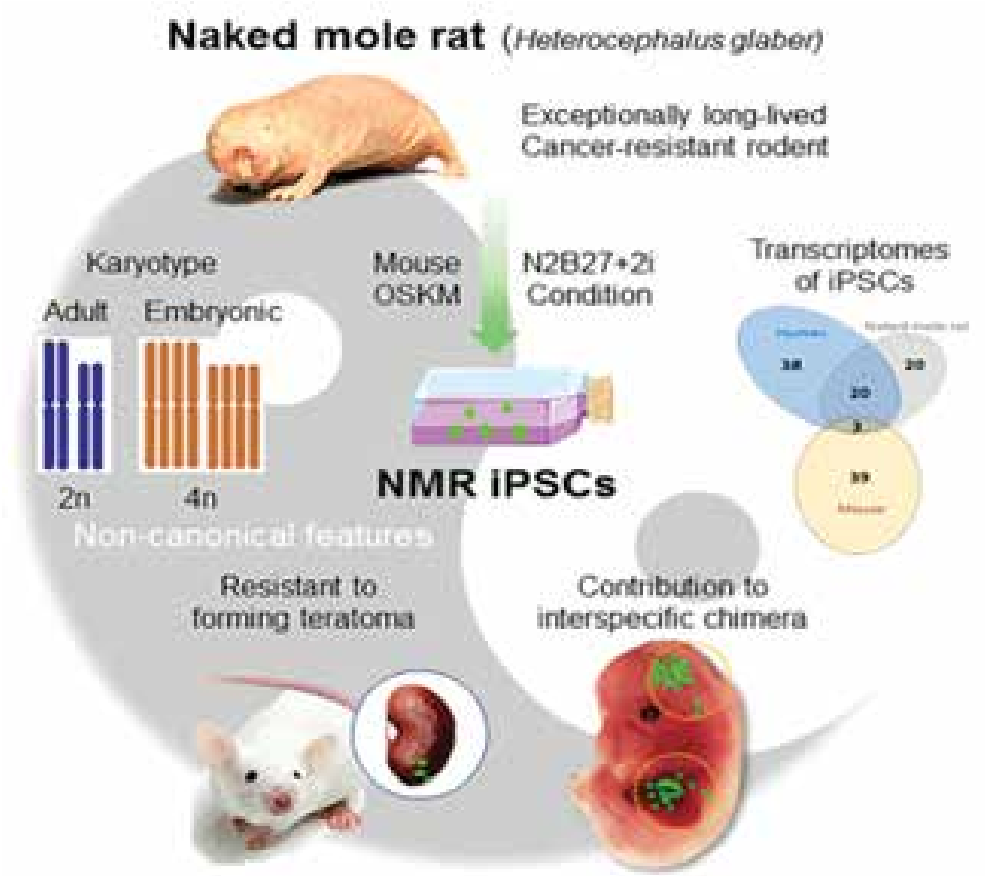
01 *Mole Rat Cancer* Resistance

Briefly, the naked mole rat is a genus of burrowing rodents in the family Bathyergidae. They have short and hairless bodies supported by wrinkled, pinkish skin. Their large heads with powerful, muscular jaws harbor two long incisor teeth, which Burke and Friedler describe as tools for digging into a sandy soil to build complex burrow systems. They have been scientifically recorded as among the longest-living rodent species, commonly characterized by low cancer rates and colonial forms of social structure. Contrary to their namesake, naked mole rats possess around 100 small hairs on their skin that give them a feel for their surrounding environment.

Naked mole rats reside underground within the grasslands and semiarid areas of East Africa, including Ethiopia, Kenya, Somalia, and Djibouti.

One of their most fascinating biological traits, however, is their remarkable resistance to getting cancer, although a few cases have been diagnosed. Their rate of cancer is at least a few times lower than other similarly sized animals and humans. Other research indicates that the secret of their not getting cancer lies in a sugar-like molecule called hyaluronan, which prevents cells from clumping together to form a tumor.



While all animals contain hyaluronan, naked mole rats have a version that is approximately five times greater than that of mice, rats, and humans.

Meanwhile, these oversizes degrade more quickly, providing an additional layer of protection. Understanding how these exceptionally strange creatures nearly escape cancer may provide valuable insight into the early stages of the disease in humans, which may consequently lead to new strategies for prevention or treatment. Until now, the stance on cancer appeared to be that naked mole rats only ever got it infrequently, for their normal cells were simply incapable of being transformed into cancer cells. But recent findings are challenging this view.

A groundbreaking study conducted by researchers at the University of Cambridge and published in Nature demonstrated that genes known to induce cancer in other rodent cells could also trigger naked mole rats. This discovery suggests that naked mole rats are protected by their microenvironment—the complex system of cells, substances, and immune responses surrounding them—rather than an intrinsic property of their cells. Dr. Walid Khaled, one of the study's senior authors from Cambridge's Department of Pharmacology, noted, "The results surprised us and completely transformed our understanding of cancer resistance in

naked mole rats."

If we can uncover what makes these animals' immune systems uniquely capable of preventing cancer, we might be able to develop similar protective strategies for humans. In addition to their cancer resistance, naked mole rats exhibit several other unusual biological features, such as being the only known cold-blooded mammal, lacking pain sensitivity to certain chemical stimuli, and surviving in extremely low-oxygen environments (hypoxia).

To further investigate their cancer resistance, researchers examined 79 distinct cell lines derived from five different tissues (intestine, kidney, pancreas, lung, and skin) from 11 naked mole rats.

Using engineered viruses, they introduced cancer-causing genes that are known to induce tumors in mice and rats. Initially, scientists believed these genes would not be capable of transforming naked mole rat cells into cancerous ones. However, to their surprise, infected cells began to multiply rapidly in lab cultures. Dr. Fazal Hadi, lead researcher of the study from the Cancer Research UK Cambridge Centre, stated, "To our surprise, the infected naked mole rat cells began to multiply and rapidly form colonies in the lab. We understood from their rapid growth that they had developed cancer."

To confirm their findings, researchers implanted these transformed cells into mice, which developed tumors within weeks. This unexpected result suggests that the naked mole rat's body prevents cancer from growing rather than its cells being inherently immune to transformation—a major shift from previous assumptions.

Going forward, scientists aim to explore the mechanisms by which naked mole rats suppress tumor growth. A key area of interest is their immune system, which may play a critical role in stopping cancer at an early stage. Immunotherapy has already revolutionized cancer treatment in humans, and uncovering the unique properties of the naked mole rat's immune defenses could inspire new therapeutic approaches.

"All our work with naked mole rats—from studying their hypoxia resistance to pain insensitivity and cancer resistance—is aimed at leveraging the extreme biology of this species to better understand how our bodies function."
- Dr. Ewan St. John Smith

Dr. Ewan St. John Smith, a senior author from Cambridge's Department of Pharmacology, emphasized, "All our work with naked mole rats—from studying their hypoxia resistance to pain insensitivity and cancer resistance—is aimed at leveraging the extreme biology of this species to better understand how our bodies function."

By studying these extraordinary rodents, researchers hope to unlock new insights that could revolutionize human cancer treatment and further our understanding of aging, immunity, and disease resistance.

02 Rhabdomyolysis: cellular response & evaluation of discovery

Rhabdomyolysis tends to be associated with some form of muscular disease due to stress and injury, which has indeed long been recognized among men and animals. Due to the release of myoglobin, creatine phosphokinase, and other intracellular constituents of the striated muscle into the blood after its disintegration, when not addressed in time, myoglobin can become toxic to the kidneys and can cause acute renal failure. The etiology of rhabdomyolysis is multifactorial, including trauma, severe muscular effort, infections, drugs, and toxins. The history of rhabdomyolysis spans millennia, and pervasive knowledge of causes, diagnosis, and treatments has changed from early chronicling of injury to skeletal muscle to the understanding of more systematically oriented backgrounds and perspectives.

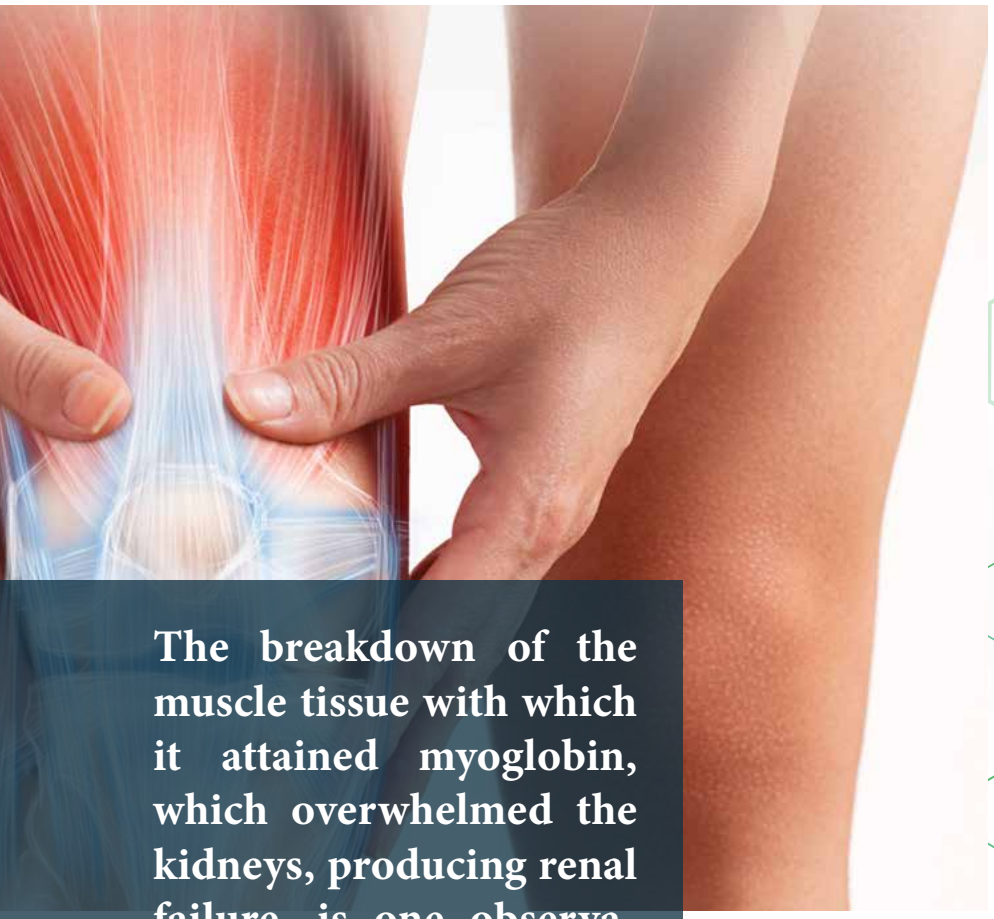
Disease Frequency: 26000 reported cases per year (U.S.)

Early records that may be construed as pertaining to rhabdomyolysis were made as far back as ancient Egyptian, Greek school, and Roman writings on post-traumatic or post-exertional injury to muscles. It is, however, not until near the beginning of the twentieth century that the term rhabdomyolysis was used. Rhabdo means striated muscle, myo means muscle, and lysis means breakdown of muscle. Despite an account being rampant, early findings do not show any acumen of systemic implication, nor does the term arise at that moment.

Conceivably the relationship of muscle injury and renal failure manifested more clearly during the early 1900s. One of the more historic events that helped define this entity was the Messina earthquake in Italy of 1908, which resulted in crush syndrome development and subsequently renal failure.

Rhabdomyolysis | Causes, Symptoms And Treatment | Figure 2

The 1940s saw a great breakthrough with British researchers Bywaters and Beall observing rhabdomyolysis in patients following traumatic crush injuries during World War II. They demonstrated that trauma-induced muscle injury led to the release of myoglobin with subsequent filtering by the kidneys and renal damage. Their work further contributed to the understanding of the pathophysiology of rhabdomyolysis by focusing attention on the role of myoglobin in kidney damage. This reformulated the clinical approach of considering rhabdomyolysis as a clinicopathological entity of wide clinical relevance, thus providing a critical issue in the history of this condition.



The breakdown of the muscle tissue with which it attained myoglobin, which overwhelmed the kidneys, producing renal failure, is one observation anchoring this relation. This laid the cornerstone of a defined relationship between muscle breakdown and kidney malfunction and also opened up a new page in the historiography of rhabdomyolysis.

Throughout the 20th century, further studies provided clarity on the mechanisms and triggers of rhabdomyolysis. The authors cited causes including trauma, infection, drug abuse, and physical exertion lingering in muscle damage. Several drugs, including alcohol, cocaine, and statins, were documented as causes leading to muscle damage and thus potential triggers of rhabdomyolysis. The studies of the 1960s and 1970s expanded the understanding of triggers for rhabdomyolysis, emphasizing the huge variety of factors contributing to muscle breakdown and kidney dysfunction

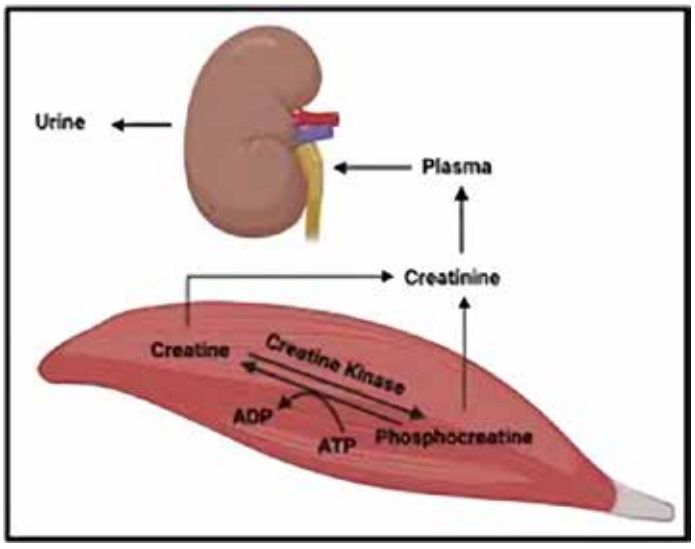
> **They found that many patients who survived the crush injury often died several days later of kidney failure**

The habit of calling the syndrome "rhabdomyolysis" began in the 1970s, with increased emphasis on diagnosis and treatment from the clinical point of view. The mechanism of nephrotoxicity had up until then been understood, which was related to myoglobin and its toxic effect after filtering by the kidneys. Myoglobin, a protein present in muscle cells, when released into the blood, can become toxic when forming complexes with heme. Such complexes cause oxidative stress in the kidneys and lead to acute renal failure. This significantly advanced the understanding of the clinical presentation of diseases, as well as the relationship between muscle injury and renal dysfunction

Development in diagnostic methods increased understanding of rhabdomyolysis in the past two decades of the 20th century. The two major diagnostic tests would be CK elevation in the serum and urine tests for myoglobin. CK is an enzyme that is released from damaged muscle and serves as a marker for rhabdomyolysis; myoglobinuria, pinkish-brown urine, is a prime indicator of muscle damage.

The advances in diagnostics permitted earlier detection, which in turn improved processing because treatment could then be initiated earlier.

Biochemical studies during the 1980s and 1990s also provided insight into the cellular mechanisms responsible for rhabdomyolysis. Oxidative stress by free radical accumulation during muscle injury was defining one of the culprits for the degeneration of muscle tissue. Such oxidative damage can cause disruption of muscle cell membrane integrity, thereby leading to the efflux of intracellular components like myoglobin. This realization has underlined the contribution of oxidative stress to rhabdomyolysis and potential therapeutic avenues to ameliorate oxidative stress.



Exertional rhabdomyolysis, a relatively well-studied injury caused by strenuous physical activity, was only widely recognized during the latter half of the 20th century. Athletes, military personnel, and other individuals engaged in vigorous activities were found to be at higher risk of developing rhabdomyolysis. The clinical picture of exertional rhabdomyolysis is typically caused by, but is not limited to, overexertion, dehydration, and extreme environmental temperatures that lead to muscle damage. It has been documented in many sports, such as marathon running, football, and weightlifting, and extended to military training or fighting. While exertion-related rhabdomyolysis can affect physically fit people, severe complications, including kidney failure, may arise if the condition is not treated urgently.

In recent years, research into the genetic and molecular mechanisms underlying rhabdomyolysis has gained momentum. Genetic mutations affecting muscle function may predispose individuals to rhabdomyolysis. For example, inherited muscle disorders like muscular dystrophy increase the risk of muscle injury, making these individuals more susceptible to the condition.

Additionally, studies have explored the role of inflammation and immune responses in the development of rhabdomyolysis, offering new insights into potential treatment strategies. Understanding the interplay between muscle injury, oxidative stress, and immune responses may lead to better-targeted therapies for rhabdomyolysis.

The treatment of rhabdomyolysis focuses primarily on addressing the underlying cause of muscle damage, such as trauma or overexertion, and preventing kidney injury. Hydration is crucial in preventing kidney failure, as it helps dilute myoglobin in the bloodstream, reducing the strain on the kidneys. In severe cases, renal replacement therapy, such as dialysis, may be required to support kidney function. Early detection and timely intervention are vital to improving patient outcomes, preventing complications, and reducing the risk of long-term kidney damage.

CAPYBARAS

A scope on evolutionary adaptations and unique behaviors

Capybaras, scientifically known as Hydrochoerus hydrochaeris, are the world's largest rodents, measuring over a meter in length and weighing up to 70 kg. They are 60 times larger than their closest relatives, Guinea Pigs, which are also adorable.

The key to such a big physique is the abundance of food in their environments (who can resist a midnight snack!) as well as the dearth of predators when capybaras' ancestors arrived in South America. Capybaras, as semiaquatic creatures, prefer to live near lakes and springs. Such water sources support lush flora, which provides food for our herbivores, the capybaras. Nonetheless, capybaras did not survive purely on food. Capybaras are more than just cute creatures, with their peculiar diets, habits, and evolutionary adaptations from both morphological and molecular viewpoints. While "capybaras" signifies grass eaters, their diets are far more complicated and adaptable.

03

When the grass dries up, capybaras eat grains, melons, reeds, and squashes. In other regions, such as Brazil, they make their way to crops and consume sugarcane and corn. Still, our large rodents lack a complicated digestive system and, like many other animals, do not digest grass properly. In such circumstances of maldigestion, you can take fiber-rich supplements or eat foods that contain good bacteria. However, capybaras tackle the problem in a more basic way: they consume their own feces! As awful as it appears, it improves vitamin absorption. This technique, known as coprophagy, allows them to digest food twice while also providing helpful bacteria for digestion. Do you still think capybaras are adorable?

Aside from coprophagy, they are both gorgeous and kind. Capybaras are herbivores and do not prey on other animals. As a result, you may see birds, monkeys, and even children riding capybaras. Capybaras still have certain predators, and their pups are vulnerable to assaults by ocelots and harpy eagles.

Caimans, Jaguars, and Anacondas are natural predators of capybaras. A capybara, on the other hand, may be seen "chilling" with a bunch of caimans if there are enough of food supplies around; aren't they lovely?

In addition to their sociability, capybaras' anatomy and other habits make them less vulnerable to predators and hunters. When a capybara feels danger, it barks or whistles to alarm the remainder of the herd. Their ears, eyes, and nose are located on the top of their heads, allowing them to plunge into the water while attentively monitoring and perceiving their environment. Their webbed feet (like ducks') also help them swim well.

However, in 1977, Richard Peto proposed Peto's Paradox, which states that the relationship between the number of somatic cells and cancer incidence is nil. Elephants, bowhead whales, and naked rat moles are examples of big creatures that seldom or never develop cancer. Only three occurrences of cancer in capybaras were recorded up till 2018.

FACT

Another remarkable fact is that they can hold their breath underwater for up to five minutes, allowing them to effortlessly avoid predators! Their varied mating seasons, which rely on habitat and partner availability, and their capacity to produce up to 8 pups per litter are also advantageous.

> **Capybaras, the world's largest rodents, are well-adapted to semi-aquatic life with several key**

So, how do giant rodents combat cancer? In 2018, the same group of researchers observed that, in addition to the development of cancer pathways in capybaras, several tumour suppression systems were visible. First, scientists discovered genes involved for telomere maintenance, such as TERF2IP (telomeric repeat-binding factor 2-interacting protein 1), which can slow cell division and hence lower cancer risk. Second, they uncovered three genes associated with tumor reversal and cancer suppression that are more elevated in capybaras than in other rodents.

The first gene, TPT1 (tumor protein, translationally controlled 1), leads to tumor reversal in capybaras by removing cancer cells' malignant character. The second gene, MAGEB5 (melanoma antigen family B5), is naturally inactive, but it is reactivated once the cell becomes neoplastic (tumorous), at which point suppression is mediated by capybara T cells. The third gene, GZMB (granzyme B), is critical in the perforin/granzyme-mediated cell-killing mechanism of cytotoxic T lymphocytes, causing apoptosis via a caspase-dependent route.

All of the genetic bases stated above reveal the marvels of capybara structure at the molecular level, as well as at the observable/morphological level.

In conclusion, capybaras are famous for their cuteness, but they also have amazing habits, morphology, and features that help them survive. The world's largest rodents are remarkable at both the visible and molecular levels. Capybaras have apparent flexibility in feeding, mating, and risk management, as well as adaptability to dive and communicate warnings via their morphological capacities (webbed feet and elevated ears, eyes, and nose). Furthermore, capybaras' herbivorous nature and the abundance of food supplies in their environments increased their friendliness toward other species, particularly caimans, who are natural predators of capybara. On the molecular level, researchers discovered that, despite their big size, capybaras had well-developed tumor reversal and cancer suppression pathways compared to other rodents. Capybaras' genetic traits include telomerase shortening via TPT1, T-cell mediated responses triggered by MAGEB5, and a particular cell-killing mechanism produced by GZMB. Finally, if temperature and food supply change, we may see further changes in capybara morphology, such as reduced hair growth, a smaller body mass, longer ears, and so forth. However, they will continue to be a fascinating example of adaptability, as well as cuteness.

04

The Woman Who Forgot How to Fear

Fear is a basic survival mechanism that is encoded into the brain. It activates the fight-or-flight response, which results in immediate reactions to dangers. So what happens when there's no fear? The case of a lady called SM, whose entire amygdala was destroyed, provided a rare insight into the neural basis of fear. This case study not only reveals the biological underpinnings of fear but also offers insight into how its absence affects human behavior and decision-making.

SM's story begins with a rare genetic disorder called Urbach-Wiethe disease, first described in 1929. This condition led to the bilateral calcification and destruction of her amygdala. The amygdala integrates sensory information and gives rise to the physiological changes, such as the faster heartbeat and heightened attention, during fear responses. With her amygdala rendered dysfunctional, SM lost all sense of fear. Unlike anxiety disorder patients who may suffer from irrational and unrealistic fear, she showed a stunning incapacity to recognize or respond appropriately to threats. In a series of experiments involving neuroscientists in the early 2000s, SM was found to have never experienced fear, no matter the fear-inducing context. Stimuli such as horror movies and haunted houses incited awe or enjoyment, but not fright. Given the opportunity to handle a variety of dangerous snakes and spiders at a pet store, SM did so willingly, all the while expressing how genuinely dangerous the animals were. More alarmingly, SM walked through a park at night, despite a prior attempted mugging in a similar location. These behaviors highlighted her detachment from fear-based decision-making.

SM was not very familiar with detecting socially relevant cues, either. For instance, she could definitely not identify an image of a frightened face. Functional imaging studies performed in the year 2010 revealed that the amygdala of SM showed no activation in the processing of fearful expressions.

This impaired her ability to experience fear and map others' feelings or detect fear on a person's face. Such findings show that diminished or heightened fear perception could contribute to insights into disorders like psychopathy or social anxiety.

Interestingly, SM's condition did not render her completely reckless. Her social interactions and expressions of happiness, sadness, and anger remained relatively normal. Nevertheless, her failure to see threats rendered her susceptible to dangerous situations, making her a victim of numerous crimes, such as assaults. Further, her lack of feelings of fright removed anticipatory anxiety inasmuch as it would help others to avoid danger. SM's brain did not enthuse fear towards her past suffering. This diverges from individuals with exaggerated fears, like PTSD.

Even though her amygdala was permanently damaged, an experiment in 2013 found a way to induce fear in SM by other means: high concentrations of carbon dioxide were administered to create a sensation of suffocation, inducing a panic attack in SM. This suggested that, while the amygdala is critical to dealing with external threats, primitive fear responses could still activate through other pathways when the body senses some kind of internal danger. The results point toward the complex nature of fear, providing evidence that multiple neural pathways regulate it. The case of SM is applicable to neuroscience, psychology, medicine, and even artificial intelligence.

Fear-processing mechanisms may offer insights into devising better treatments for anxiety disorders, PTSD, and phobias, where fear responses are increased or dysfunctional. The study of fearless individuals may also give direction for designing smart systems that could assess and respond to threats.

This case raises questions as to whether fear is an integral component of the human experience. The role of the amygdala brings to light how emotion, cognition, and survival drive instinct coalesce. The information suggests that fear is not unitary but a multidimensional process, supported by competing brain structures and responses. Such findings might trigger new research avenues into emotional resilience, risk behavior, and adaptive coping methods not driven by fear.

SM offers an excellent, though harrowing, example of how specific brain structures shape fundamental emotions. Researchers are working to better understand fear and related disorders, such as PTSD and anxiety, by studying subjects with atypical neurological diseases. Although fearlessness may seem advantageous, SM's story illustrates the critical role fear plays in survival, social functioning, and personal security. Her case continues to shape our understanding of the neurocognitive basis of emotions and highlights the delicate balance between adaptation and vulnerability in human behavior.

05 Unlocking Weight Loss The Hidden Impact of Insulin Resistance

Insulin resistance is a commonly under-recognized metabolic disease that can turn weight loss into a battle, even for those of us who zealously stick to a diet and exercise plan. While some individuals appear to find it effortless to meet their level of fitness, others will sweat in vain for the most part due to underlying metabolic reasons. To truly gain insight into how insulin resistance negatively affects weight regulation, we should first study the highly significant role that insulin plays within the body.

Insulin, a hormone produced by the pancreas, is the master regulator of blood glucose. Insulin enables cells to absorb glucose from the bloodstream and utilize it as the body's primary energy source. Glucose, derived from carbohydrate food intake and glycogen stores, fuels basic processes such as muscle contraction, brain operation, and cellular repair. The pancreas produces insulin directly into the bloodstream in order to modulate glucose homeostasis so that blood sugars are maintained in a normal concentration.

For insulin to be effective, it must bind to specialized receptors on the surface of cells, telling them to absorb glucose from the blood. But in insulin resistance, these receptors are less sensitive, thus preventing the uptake of glucose. Therefore, glucose accumulates in the blood, leading to high blood sugar levels. Impaired insulin sensitivity is also another name for this and is a predisposition to dangerous metabolic disorders like type 2 diabetes.

As a substitute, the pancreas produces and secretes extra insulin in an attempt to keep blood sugar in balance. The resulting hyperinsulinemia overproduces glucose for a brief time, holding it momentarily but taking an immense toll on the pancreas. Ultimately, the compensatory mechanism becomes impractical, inducing pancreatic fatigue and decreased insulin secretion. As production decreases, levels of blood sugar become unregulated, significantly elevating the chance for type 2 diabetes.

Aside from glucose regulation, insulin also influences fat storage and metabolism. Another one of its lesser-known effects is that it can facilitate fat storage, particularly around the abdominal region. Elevated levels of insulin encourage the body to store the surplus glucose as fat rather than to burn it as energy. As such, patients with insulin resistance are far harder to lose surplus weight compared to patients with optimal insulin sensitivity.

Even with strict dieting and physical exercise, insulin resistance can disturb the body's ability to utilize stored fat as energy. Instead of burning fat, the body remains in a condition of fat buildup, making weight loss even more difficult. It is this metabolic inefficiency that makes some individuals unable to lose stubborn fat pads despite maintaining a calorie deficit and an active lifestyle.

Both chronic inflammation and oxidative stress are implicated in insulin resistance. Inflammatory substances interfere with insulin receptor function and thus enhance insulin resistance. In addition, oxidative stress, which is a free radical-antioxidant imbalance, damages cells and exacerbates metabolic derangements. Both chronic inflammation and oxidative stress drive a vicious cycle that increases insulin resistance and distorts overall metabolic health.

There are different causes of the development of insulin resistance, including diet, age, genetics, and lifestyle. One is more susceptible to developing insulin resistance if a family member had type 2 diabetes. Natural aging has an effect that diminishes sensitivity to insulin, therefore making elderly individuals more susceptible. However, lifestyle, particularly inappropriate diet and a sedentary lifestyle, contributes the most. Overconsumption of processed foods, refined carbohydrates, and saturated fats can impair insulin action, while physical inactivity exacerbates metabolic dysregulation. Obesity is one of the strongest predictors of insulin resistance, with specific fat accumulation around the abdominal area.

The better news is that insulin resistance is reversible through deliberate lifestyle adjustments. A perfectly balanced diet that focuses on whole, nutrient-rich foods will increase insulin sensitivity.

Focusing on fiber vegetables, lean protein, healthy fat, and low glycemic index complex carbohydrate intake can regulate blood sugars and prevent insulin surges. Avoiding refined sugar, trans fat, and heavily processed foods is also critical to insulin resistance management.

Daily physical exercise is another potent weapon against insulin resistance. Aerobic exercise, such as running and cycling, as well as resistance training, such as weight lifting, both enhance the muscle uptake of glucose and make muscles more sensitive to insulin. Exercise also stimulates glucose transporters to be translocated to the cell membrane, allowing for muscles to take up glucose in a healthy way, even in insulin-resistant patients. Physical exercise also reduces visceral fat, a key causative factor of metabolic derangement.

In some cases, medication will be necessary. Physicians can write prescriptions for medications such as metformin, which increases insulin sensitivity and lowers liver glucose production. Medication, on the other hand, should be considered as a supplement to lifestyle changes rather than a replacement. Routine screening for health issues such as insulin resistance, such as fasting insulin and glucose, can identify it early and guide individualized treatment plans.

Other factors, such as stress management and sleep, influence insulin sensitivity as well. Chronic stress raises cortisol, which can cause insulin resistance. Mindfulness, relaxation techniques, and a proper amount of sleep can rebalance hormones and assist with metabolic processes. Sleep loss, in particular, has been found to contribute to impaired insulin sensitivity, so it's necessary to maintain a consistent sleeping schedule and get sufficient rest.

Conquering insulin resistance is the key to effective, long-term weight loss and prevention of chronic disease. Understanding how insulin resistance undermines glucose metabolism, promotes fat storage, and triggers inflammation enables individuals to take proactive, healthy steps to maximize their metabolic health. Optimal nutrition, exercise, stress reduction, and physician guidance can improve insulin sensitivity, promote fat loss, and reduce the risk of type 2 diabetes and other metabolic syndromes.

Resistance to insulin is a complex but reversible syndrome that dramatically affects weight reduction and well-being. While plain common sense oftentimes reduces the weight management to a simple low-calorie formula of calories in versus calories out, considerations of metabolism, such as insulin resistance, are of utmost priority. Individuals who recognize insulin resistance as an underlying barrier to weight loss and address its root causes can break free from the cycle of metabolic disruption and achieve long-term health gains. Understanding the science of insulin resistance empowers individuals to make informed choices, leading to a healthier future.

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