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Why don't elephants get cancer?A new genetic study shows they have genes with special powers to fix mutated DNA — and humans have slightly different versions of the same genes.The report, published in the journal Cell Reports, could open the door to helping people fight cancer better in the future.And the team at the University of Utah found a batch of other animal traits that shed light on human genetics.For instance, a stretch of DNA that gives bats their distinctive pointy ears can, when mutated, cause an ear deformity in people called Stahl ear. Another mutation can also cause fused fingers in people, but may have helped bats evolve their unique wings.And humans share genes that give ground squirrels their distinctive stripes. In people, mutations of those genes can cause Noonan or Leopard syndrome, marked in part by distinctive freckle-like spots.Geneticist Christopher Gregg and colleagues were not looking for anything in particular in their study.“We were working on the elephant as a positive control in our study and we discovered parts of the genome that were important for nose development,” Gregg said.“I thought that’s very funny to find in elephants, since they have these long noses.”He chatted with a colleague at Utah, Dr. Josh Schiffman, who is a pediatric oncologist and who suggested that Gregg take a different tack.“He had been studying why elephants don’t get cancer,” Gregg said.“This is a master regulator of DNA repair.”It’s a question that has bothered biologists for years. Cancer is linked with extreme body growth. Very tall people have a higher risk of some cancers, and tumors are formed by the out-of-control growth of some cells.But elephants are far less prone to cancer than humans are, even though they are much bigger and live very long lives.In 2015, Schiffman found that elephants have 20 copies of a gene called p53, well known for its ability to fix damaged DNA, which can lead to cancer. Humans have only one.Now Gregg’s study showed a possible way to discover more.“He said, ‘Chris, why don’t you check in your data to discover if there are any more secrets that explain why elephants don’t get cancer?’” Gregg said of Schiffman.Gregg’s team found a big one.It was a network of genes that all mammals, including humans, have called the Fanconi anemia pathway.“The elephants had just radically changed it,” he said.“This is a master regulator of DNA repair.” Gregg said.“People with mutations in that gene pathway have very high rates of cancer.”Elephants appear to have extra elements in their genetic complex. The team tested elephant immune cells, zapping them with radiation and looking to see which genes became active. Elephant immune cells quickly repair damage to other DNA — an important mechanism for preventing cancer.Studying these genes further in humans might lead to new ways to fight cancer, the team proposed.“They were good candidates for why the elephant has this superpower of getting very low rates of cancer,” Gregg said.Humans share much of their genetic code with other animals, from fruit flies and worms to dogs and apes. And sometimes the precise genetic function doesn’t have much to do with evolutionary similarities.For instance, many of the genes that cause diseases in humans can be found in macaque monkeys but not in chimpanzees — the nearest living relative to humans.Gregg’s team had a formula for making good comparisons among the genomes of humans and other animals, finding regions that have taken markedly different paths in certain species.“Once we recognized that the method worked, we started applying it to other animals that have really interesting and distinctive traits,” Gregg said.They did what’s called a genome wide association study — sequencing all the DNA — in a bunch of animals, including elephants, bats, orcas, squirrels, dolphins and naked mole rats.They looked for stretches of shared DNA that different species had in common with each other and with people that might provide insights into health and development.“We looked at bats, which are the only mammals adapted for flight,” Gregg said.“We discovered new parts of the human genome that bats have changed that are candidates for developing these wings.” And the ear finding was intriguing, too. “None of us knew what Stahl ear was. We had to get on Google and figure out what is Stahl ear and then it all makes sense, because it causes those funky ears,” he said.Naked moles rats live underground in big colonies and are blind, and changes in their vision genes might point researchers to how the visual system developed, Gregg said.“Once we recognized that the method worked, we started applying it to other animals that have really interesting and distinctive traits.”They also looked at dolphins and orcas — mammals that have evolved to live in water. “They can do deep dives underwater and hold their breath for very long periods of time,” Gregg said.“They must have some way of carrying more oxygen in their blood.” he hypothesized.If humans try holding their breath for too long, they can change the blood chemistry and develop blood clots.It turns out orcas and dolphins have changes in the genes involved in the production of erythropoietin, a hormone that stimulates production of red blood cells, which carry oxygen. “We are interesting in following that up,” he said.Now it will take years of hard laboratory work to see if the genes really do what the team thinks they do.“We need to do real functional studies in the lab with human cells and animal models to test these elements,” Gregg said. “Do they really prevent cancer? Do they really shape ear morphology?” Elephants’ extra anti-cancer genes Problems with p53 are linked to most adult cancers. The gene responsible for making p53 is called TP53. Humans have one copy of it (containing two versions) in every cell. It’s so important for protecting us from cancer that people born with only one functioning version of TP53 (a condition called Li-Fraumeni Syndrome) have been reported to have a lifetime cancer risk of more than 70%. Every cell in an African elephant contains 20 copies (or 40 versions) of TP53. They don’t all work the same way. But with 30 extra versions of the gene on the job, elephant cells are much more vigilant about DNA damage. They will self-destruct to prevent mutations that our cells won’t even respond to. It’s not that all, or even most, of those changes would lead to cancer. With so many chances for things to go wrong, though, elephant cells shoot first and ask questions later. Elephant TP53 seems to act that way wherever you put it. Lab studies have shown that it presses the self-destruct button when introduced to human cancer cells, too. A study last year by a team of researchers from across Europe (including the Chairman of Save the Elephants, Professor Fritz Vollrath) helped explain how. There are important differences between each copy of elephant TP53. They all have their own ways of interacting with other proteins in the cell, making it harder for cancers to disable enough of them to escape detection. By comparing exactly how these different copies of TP53 work, the researchers hope to learn more about how our own bodies fight cancer. They’ve also suggested that supporting the TP53 in our cells - mimicking elephants’ different copies of the gene - could be a new model for cancer medicine. Scientists have recently shed some light on exactly why elephants, one of the biggest animals on the planet, paradoxically experience unusually low rates of cancer. The research found these remarkable mammals carry unique genetic variants that reduce their risk of tumors, and the findings could help develop new cancer therapies for humans.Editor’s note: Readers often ask us for follow-ups on memorable stories. What has happened to this story over the years? This article was originally published in 2022 but has been re-edited and updated with new information current as of May, 2025. Enjoy!As an organism grows older, and its cells continue to replicate, the chances of cancerous mutations increase. And the bigger the organism, the more cells it has, so the more chances for mutations, and the higher risk for cancer as it ages. Right?Within individual species this observation has generally held true. From taller humans to bigger dogs, cancer risk has been found to positively correlate with body size. So in theory animals that live long lives should experience higher rates of cancer than smaller, short-lived organisms. But this hasn’t been the case, and the discrepancy has long been known as “Peto’s Paradox”, named after epidemiologist Richard Peto, who discovered per-cell carcinogenesis rates were not consistent between species. In fact, in some larger species such as whales and elephants there seemed to be very little evidence of cancer at all, despite them being massive and living long lives. In early 2020’s a team of researchers challenged the basic idea of Peto’s paradox, offering the first large-scale dataset of cancer prevalence in around 260 organisms. They found larger species do tend to have a slightly higher prevalence of cancer compared to smaller species. However, things got really interesting when they homed in on how certain large organisms evolved techniques to protect themselves from cancer.While it is generally understood that each species evolved its own unique abilities for suppressing cancer, elephants have always been of particular interest to researchers. These animals have similar lifespans to humans yet, despite their size, show little signs of cancer, even in very old age. It has been estimated as little as 5% of elephants ultimately succumb to cancer, compared to 25% of humans.A landmark study a few years ago homed in on one of the key ways these massive mammals may be avoiding cancer. Elephants seem to have 20 different copies of a tumor-suppressing gene known as p53.This gene encodes a protein, also known as p53, which serves as a crucial cell protector. This protein acts a bit like a guard whose job is to stop a cell dividing when it detects any DNA damage or mutation.When the p53 gene is not working properly damaged cells can multiply and cancerous tissue accumulates. Dysregulation of the gene is thought to play a role in more than half of all human cancers, but unlike elephants, we only have one copy of the gene.A 2022 study set out to investigate exactly how the variety of p53 genes in elephants suppress cancer.“This intricate and intriguing study demonstrates how much more there is to elephants than impressive size and how important it is that we do not only conserve but also study these signature animals in minute detail,” explained study co-author Fritz Vollrath at the time. “After all, their genetics and physiology are all driven by evolutionary history as well as today’s ecology, diet and behavior.”The activity of p53 in a cell is regulated by another gene known as MDM2 that encodes a protein that essentially inactivates the p53 protein.This p53-MDM2 pathway is fundamental to the workings of healthy cells – p53 stepping in to check the health of a cell, and MDM2 stopping p53 from triggering cell death by sending a signal to say everything is working fine. The new research found elephants have an incredible array of different p53 proteins that can increase the way around MDM2 inactivation.In humans, because we only have one copy of the p53 gene, it doesn’t take much for MDM2 to take over and allow cancerous cells to replicate. But in elephants the p53 protein takes on dozens of different molecular shapes, avoiding inactivation by MDM2, and stopping the replication of so many more cancerous cells.“This is an exciting development for our understanding of how p53 contributes to preventing cancer development,” said study co-author Robin Fähræus. “The existence of several p53 isoforms in elephants with different capacities to interact with MDM2 offers an exciting new approach to shed new light on p53’s tumor suppressor activity.”The 2022 study presented impressive new insights into the mechanisms evolved by elephants to avoid cancer. As well as demonstrating how these massive mammals deployed different p53 molecules to suppress the growth of cancerous cells, the study also pointed to potential human clinical outcomes. By highlighting dozens of novel ways p53 molecules can be activated researchers are now presented with a number of new pathways for targeted cancer therapies in humans.The next year, in 2023, a different team of researchers presented a bold new hypothesis to explain why elephants may have evolved so many iterations of the p53 gene. The question they asked was what could have prompted so many variations in the p53 gene when cancer primarily develops during old age, long after an animal has reproduced. If elephants were dying of cancer after they produced most of their offspring then there wouldn’t be a great deal of selection pressure to minimize cancer. Here, they suggested the answer lay in the animal’s testicles. In most sperm-producing mammals, the testicles drop from the body into the scrotum. This is crucial because healthy sperm needs to be kept at a temperature slightly cooler than mammalian body temps, but elephants are somewhat unique in lacking the evolutionary tactic of housing its testicles outside of the body. This means elephant sperm is frequently exposed to high body temperatures that can damage DNA.So this novel hypothesis claimed the proliferation of p53 genes in elephants was not to protect the animals from cancer but instead to protect the animals’ sperm. These genes have a general job of stopping a cell dividing when DNA damage is detected so they likely are important in maintaining the health of elephant sperm. The cancer protections are possibly a secondary benefit helping the animals further live long healthy lives.A version of this article was originally published in 2022. UNDATED (WKRC) - A new study that examined animal species with a cancer resistance gene could provide help for human cancer research.Scientists have long been intrigued by Peto’s Paradox, which suggests that large animals with long lifespans should develop cancer more frequently due to having more cells and time for mutations. However, elephants, whales and other large animals tend to have lower cancer rates than humans, according to BGR.In a 2015 studyBGR reported that researchers discovered that elephants possess 19 extra copies of the TP53 gene, a powerful cancer-stopping gene that acts as a genetic safeguard by detecting DNA damage and triggering cell death in potentially cancerous cells. This genetic advantage likely explains elephants’ low cancer rates despite their size, according to BGR.A new studyBGR reported expanded this research to nearly 300 animal species, analyzing over 16,000 necropsy records.The findings revealed that elephants are not unique in their cancer resistance. Many other species including some birds, bats and lizards, also exhibit low cancer levels, while animals like ferrets and opossums have higher rates. The study highlighted that larger body mass was linked to a slightly higher cancer risk, though not as strongly as anticipated, BGR reported.The implications for human cancer research are significant, according to BGR. While humans have only two copies of the TP53 gene, understanding how elephants and other species bolster their cancer defenses could lead to new treatments that enhance human cell resilience. BGR reported that researchers hope mimicking or boosting the effects of this gene could help develop therapies to reduce cancer incidence or slow its progression. Certain animals, like elephants, have evolved ways to be resistant to cancer. Researchers are studying these animals for clues about how to treat cancer in humans. You’d think elephants would be getting cancer left and right. They are giants of the animal kingdom and have trillions more cells than humans – cells that, in theory, could turn into cancer over their decades-long lifespans. But you’d be wrong. It’s not that they never get cancer, but less than 5% of elephants die from it, versus up to 25% of humans. “Because of their body size and how many cells they have and how long they live, they should all be developing cancer,” said pediatric oncologist Dr. Joshua Schiffman, professor of pediatrics at University of Utah and an investigator at Huntsman Cancer Institute. Researchers like Schiffman study animals that have evolved ways to “naturally resist cancer” despite their largeness and longevity, including elephants and bowhead whales. The latter can live for over two centuries. It’s not just gee-whiz science, either. By picking apart the inner workings of genes and molecules in the animal kingdom, scientists hope to unravel new ways to prevent or even treat cancer in humans. “This is where the field is moving as a whole,” Schiffman said. “If we can understand how these genomic changes are contributing to ... cancer resistance, then we’ll be able to start thinking about how do we translate this to our patients?” One likely mechanism, according to a paper published Tuesday in the journal Cell Reports, could be a “zombie” gene that, when brought to life by DNA damage, causes that cell to die off. “If [that cell] kills itself, then that damaged DNA never has the potential to eventually give rise to cancer,” said study author Vincent J. Lynch, an evolutionary biologist at the University of Chicago. The zombie gene arose from what’s known as a “pseudogene,” a mutated or inactive copy of a normal gene that can accumulate over eons of evolution. Elephants, as well as close living relatives like manatees, have many duplications of a gene known as LIF – but these copies don’t actually work like the original. In elephants, however, one copy seems to have reanimated and “evolved a new on-off switch” that responds to DNA damage, Lynch said. His study’s findings comprise one piece of a larger puzzle, he adds. “There’s probably lots of things which can contribute to augmented cancer resistance, and we found one of them in elephants,” said Lynch. “The way we normally think that evolution works is by acquiring lots and lots of genetic changes,” he said. “Each one of them contributes a small effect. “And when you add all those things together, you end up with a super cancer-resistant elephant.” “All cells get mutated all the time,” said University of Utah’s Schiffman. “My cells are mutating. Your cells are mutating. But hopefully our DNA repair genes are able to catch them. “Cancer likes to shut these genes off.” Schiffman, who was not involved in Lynch’s study, described the new report as part of a growing body of evidence of ways animals have evolved to bolster how their cells naturally fight DNA damage and DNA mutations. Schiffman’s own research includes other genes that could give elephants their exceptional ability to quash cancer before it starts. Schiffman referred to one in particular, a tumor suppressor gene called p53, as the “genetic police” for its role in stopping DNA damage from turning into cancer. Elephants have dozens of copies of the gene. Humans normally have just two copies of p53 – a less robust form than in elephants. And when that gene is mutated or inactivated, as with many of Schiffman’s patients, cancer is allowed to grow unchecked. In elephants, p53 also revs up the zombie gene, according to Lynch’s study. Though experts say more research is needed to confirm these findings and figure out exactly how the zombie gene kills cells on a molecular level. “This will not be the final story,” Schiffman said. Experts say there are many steps before findings like Lynch’s are tested or used toward human cancer therapies. One of those steps could involve inserting elephant genes into lab mice, which do not have the same resistance to cancer, according to Vera Gorbunova, a professor of biology at the University of Rochester and co-director of the Rochester Aging Research Center. She was not involved in the new study. Gorbunova also wondered what else might be going on in elephants that balances out their cells’ tendency to die under stress. Enhancing pathways like p53 in isolation could have unintended consequences, she said. For example, in earlier mouse studies, increasing the gene’s activity caused mice to age faster than normal. “It’s a very fine-tuned system,” said Gorbunova, who stressed the importance of taking a closer look at these “unusual organisms” that rarely develop cancer. It’s not just large animals, either. There are smaller cancer-resistant critters, too, including naked mole rats, microbats and grey squirrels. “I think we have to study all of these cancer-resistant animals and then choose strategies that are most easy to apply to people,” she said. That means that zoologists, veterinarians, anthropologists, mathematicians and human doctors must “come together with a common cause,” said Schiffman, whose colleagues also include elephant conservationists “to study those rare times when they do develop cancer.” “When we put on our clinician’s hat ... do we look for drugs that are affecting the same pathways?” Schiffman said. “Evolution has been at work evolving cancer-resistant organisms for ... millions of years,” Lynch said. “So why not just look to evolution to give us insight into how we might be able to do that?” In 1977, Sir Richard Peto, FRS, FAACR, postulated that larger animals with longer lifespans should develop cancer more readily than their smaller, shorter-lived companions. Since cancer is driven largely by errors in DNA replication, animals with more cells and more time to accumulate mutations should also develop more cancer, he argued. He observed, however, that this does not appear to be the case. Why, then, do most elephants, whales, and bears get less cancer than humans? The finding became known as “Peto’s paradox,” a challenge to comparative oncologists to learn more about cancer in vertebrates and understand why cancer incidence doesn’t correlate directly with mass and longevity. Researchers speculated that perhaps understanding the evolutionary processes that prevent cancer in other animals could help us learn how to prevent or treat it in humans. Work on that front has produced some intriguing findings. In 2015, for instance, researchers found that elephants have 19 extra copies of the tumor suppressor gene TP53 to help them ward off cancer. But there’s more to cancer risk than genetics, explained Zachary Compton, PhD, a postdoctoral fellow in the University of Arizona Cancer Center’s NCI T32 fellowship program and first author of a recent study in Cancer Discovery, a journal of the American Association for Cancer Research (AACR). “Evolution has all of these crazy stochastic ways it can deal with the same problem,” Compton said. “You don’t expect there to be just one way of handling cancer across the diversity of life.” In the study, Compton and colleagues analyzed cancer incidence in nearly 300 vertebrate species to identify characteristics associated with cancer prevalence in animals. In addition to finding more species with incredibly high or low cancer rates to study further, the researchers demonstrated a smidge of support for Peto’s original hypothesis and hinted at an explanation for the apparent paradox. Compton is particularly interested in the evolutionary mechanisms that regulate cancer. The constraints of natural selection suggest that cancer would be selected against during the evolutionary development of a species. “Somatic dysfunction is going to decrease the fitness of an individual,” Compton said. “If you have to pump energy into cells that are not working correctly ... you would expect natural selection to prune that.” Zachary Compton, PhD But traits like body mass, longevity, and gestation time are also subject to selection pressure, Compton explained. With all of these traits intertwined, it can be difficult to tease apart the push and pull of different selective advantages and how they may drive or suppress cancer. Such an endeavor requires a large sample size, for starters. Compton and colleagues collected 16,049 necropsy records from 292 different animal species at 99 animal care institutions in the United States and London. The records were derived from institutions bound by accreditation requirements to perform a necropsy on every animal that dies in their care, whether from known causes or simply old age. “All of these deceased animals have had a really thorough investigation by a veterinary pathologist to determine ... not only what they died from but what they died with,” Compton explained. The researchers assessed whether neoplasia (uncontrolled cell growth) was present and in what tissue, and they developed a terminology dictionary based on the neoplasia descriptions to predict whether neoplasms were benign or malignant. Across all species, each of them had a minimum of 20 necropsy records, the median neoplasia prevalence was 3.2%, and the median malignancy prevalence was 0.2%. Further, Compton and colleagues evaluated the associations between evolutionary traits—including maximum lifespan, adult body mass, basal metabolic rate, gestation length, litter size, time to sexual maturity, and growth rate—and the prevalence of neoplasia and malignancy. Larger body mass was significantly associated with a higher prevalence of neoplasia, but every tenfold increase in body mass, the risk of neoplasia increased by 2.1%. Other factors associated with malignancy and/or neoplasia included increased maximum longevity, larger litter size in mammals, and, in a subset analysis of 15 species, somatic mutation rate, or the rate at which new mutations occur in the body. Longer gestation time was significantly associated with a lower prevalence of both neoplasia (5.3% decrease in neoplasia risk per tenfold increase in months of gestation) and malignancy (5.65% decrease in malignancy risk per tenfold increase in months of gestation). Compton speculated that for a large animal to develop rapidly in the womb (or an egg), they must possess mechanisms to suspend natural growth restriction programs—mechanisms that could potentially be exploited later to form cancer. Because both longevity and gestation time are generally associated with body mass, the researchers performed additional analyses in which they normalized data based on these factors. Body mass was more strongly associated with neoplasia and malignancy prevalence when accounting for gestation time, and vice versa. Compton and colleagues also addressed the notion that animals in captivity may live longer than nature intended and thus be more susceptible to cancer. In this study, the vast majority of species typically developed tumors within their natural lifespan, and age at death only correlated with higher rates of malignancy in amphibians. “We not only controlled for natural lifespan, but we discovered that we didn’t need to control for it,” Compton said. “The rates [of neoplasia] we were seeing weren’t because animals live longer in zoos.” A component of the study that Compton found particularly exciting was the identification of animals with unusually high or low levels of neoplasia prevalence—a “leaderboard” of species for other researchers to explore. He hopes that animals particularly resistant to neoplasia or malignancy can provide clues to cancer prevention mechanisms that can be repurposed in humans. Similarly, species with abundant cancer may point to genetic, viral, or environmental causes that we can model to study analogous cancer drivers in humans. Overall, the median prevalence of neoplasia and malignancy varied among taxonomic orders, from 12% and 1%, respectively, in mammals to 1.2% and 0%, respectively, in amphibians. Still, there were outliers in each group. “It’s not like all of the large mammals have low cancer,” or a certain group of amphibians or reptiles,” Compton said. “There are representative species from all of these clades that seem to be very good at preventing cancer.” Mammals with exceptionally low levels of neoplasia included the Nubian cheet, the tammar wallaby, and several species of bats. Many species of birds appeared particularly resistant to neoplasia, including the black-footed penguin and common songbirds like finches and starlings. Lizards such as the plumed basilisk and the chameleon forest dragon, plus a handful of snakes, also had no neoplasia detected. This figure shows the relative prevalence of neoplasia (green) and malignancy (purple) across different mammalian species. Mammals silhouetted in green boxes represent species for which no neoplasia was detected in this study. On the other end of the leaderboard, ferrets and opossums had the highest risks of neoplasia—63% and 56%, respectively. Rounding out the top 10 list for neoplasia prevalence were the Dominican mountain chicken frog, jaguar, four-toed hedgehog, Asian elephant, pygmy sugar glider, milk snake, common mouse, and chimpanzee. According to federal statistics, approximately 39.3% of Americans will be diagnosed with cancer during their lifetime, but Compton warned that we may not know the exact prevalence of malignancy in humans. Most natural human deaths do not warrant autopsies, and there is currently no robust way to estimate benign neoplasia at the population level. But Compton hopes that the data from this study may help researchers better understand and model cancer development and cancer suppression mechanisms that may be translatable to humans. Then, perhaps, we can decrease our own species’ cancer incidence rate or make the disease less deadly. This study, however, was not designed to do that. “We’re not trying to change the way we live, we’re just trying to understand it better,” Compton said. “We’re trying to understand the mechanisms that are causing cancer in animals, and then we can use that information to help us prevent cancer in humans.” “There are representative species from all of these clades that seem to be very good at preventing cancer.” Mammals with exceptionally low levels of neoplasia included the Nubian cheet, the tammar wallaby, and several species of bats. Many species of birds appeared particularly resistant to neoplasia, including the black-footed penguin and common songbirds like finches and starlings. Lizards such as the plumed basilisk and the chameleon forest dragon, plus a handful of snakes, also had no neoplasia detected. 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