ZOOLOGICA

DEPARTMENT OF ZOOLOGY

BARASAT GOVERNMENT COLLEGE

ZOOLOGICA

Department of Zoology

BARASAT GOVERNMENT COLLEGE

ANNUAL MAGAZINE 2021-2022

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Prologue

Greetings! It is time to publish the next issue of our departmental magazine. Due to prevailing covid situation this year also the magazine will be a digital version. We have made tremendous strides to bring this edition, filled with inspiration and information as intended. The publication of the departmental magazine ignites immense pleasure and satisfaction in me to witness the monumental pace gained in the short period of its existence. Each issue of the "ZOOLOGICA" is actually a milestone that marks our growth, infolds our imagination and presents a beautiful mosaic of activities and creative talent of our beloved students, highlighting their success and achievements. The editorial board have enjoyed making this magazine a vehicle for the students and staff members to express their innermost thoughts on the subject. It is actually a lovely experience to see these enthusiastic and budding writers voicing their feelings through articles. From this issue a new section is added that reflects the year round performance of the students beyond the classroom. These activities are not possible without motivation by the teachers. A students academic achievement list is also incorporated in this issue that reflects the overall progression of the department. Many thanks are due to the senior leadership of the department for their faith in me for the publication of the same. The editorial board and UG & PG students (Alumni & Present) have brought the magazine alive with their immense support and rich contribution and we now proudly present it for your reading pleasure. Happy reading & viewing!

Dr. Sumana Saha

Head, Post Graduate Department of Zoology

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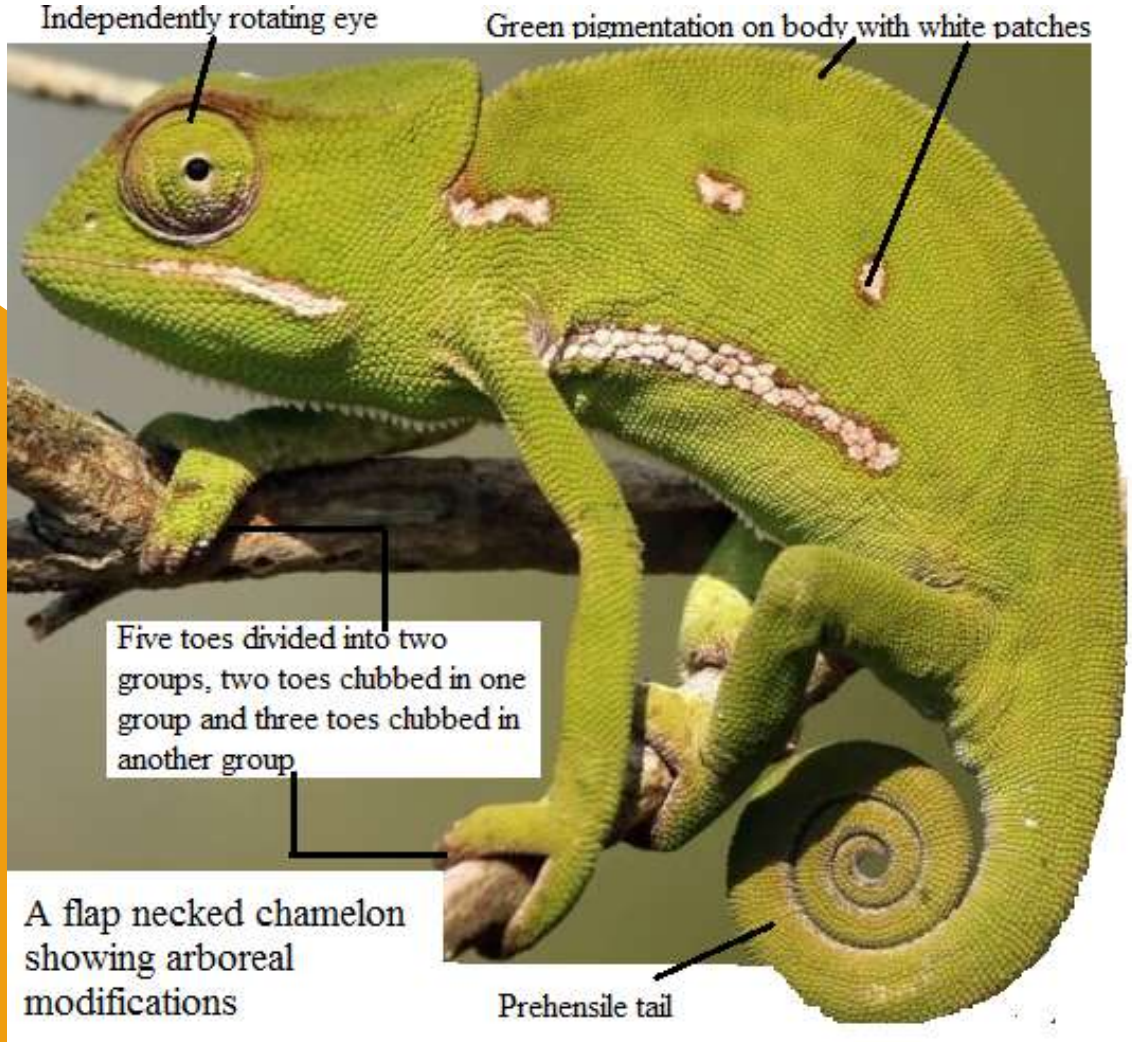
Survival Tricks – Amazing Facts of Chameleons

Dr. Anilava Kaviraj, Professor (Retired) of Zoology, University of Kalyani, Ex-PGBOS Member, Dept. of Zoology, Barasat Govt. College

Chameleon is one of the most common specimens used for spot identification in Zoology honours course, throughout India. It represents the class "Reptilia", which according to the latest records of IUCN is the third most diverse class among vertebrates with 10038 odd species, standing only after fish with 32900 plus species and birds with 10425 plus species. Apart from being used as general zoology specimen, Chameleon is also used as ecological specimen with niche specialization. Most of the specimens preserved in the laboratory, apart from showing external reptilian features in their scales, exhibit extreme morphological specialization for tree habitats like prehensile tail and limbs modified like birds (five toes divided into two groups, two toes clubbed in one group and three toes clubbed in another group).



A flap necked chamelon showing arboreal modifications

The sticky erectile tongue, which is specialized for catching insects, exhibits its predatory nature and carnivorous feeding habit. Chameleons do not move very fast. Rather they sit in one place and throw their long tongue, capture the insect prey on the sticky tip of the tongue, draw it back and crash the prey with its powerful jaw before swallowing. To locate prey items, as well as its predators (mostly birds and snakes) chameleon uses its specially designed eyes, which are spectacular in animal world. The eye balls are placed on retracting muscles, which can rotate eye up to 180 degrees, giving full view of the side, anterior and posterior part of the body. More fascinating is the fact that it can rotate its left and right eye independently in different direction enabling itself to have almost 360 degree view around its environment. When chameleons search for prey they move their left and right eye independently. It provides monocular vision separately in left and right eye. Once the prey item is located, the two eyes synchronize the vision from monocular to binocular vision and make an accurate target to catch the prey.

Chameleons are more popular for their ability to change body colour. Colour change is possible due to pigments released by the chromatophore cells, which are present in three layers beneath the skin. The outermost layer of the skin is transparent. Below this layer there are xanthophore and erythrophore cells with yellow and red pigments respectively. Below this layer there are iridophores containing blue pigments and the deepest layer contains melanophores with brown pigments. The pigments are contained in a sac within the chromatophore cells and are released when the sac is expanded following signal received through central nervous system. When the sacs in erythropores are expanded the red pigments are released, while sacs in other chromatophores remain contracted. By varying the activity of the chromatophores, chameleon can produce a wide variety of colours in their body. The pattern of colour change varies with species. The most common colour produced by a number of species are shades of green and brown. Though the mechanism of colour change is poorly understood, it is almost certain that chameleons produce different colours specifically for two purposes: (i) to adjust body temperature and (ii) to communicate with other members in a population, particularly by the males to attract females. Ligon et al. (2013) observed that in veiled chameleons *Chamaeleo calyptratus* males with brighter stripe coloration are better competitors and males with brighter head coloration win fights among males to mate with females. Studies on panther chameleon, *Furcifer pardalis* also indicate that males possess exceptionally bright colour and the ability to rapidly change the colour when encountering competitors to win a female (Teyssier et al. 2014).

Unfortunately, due to fascinating behaviours, particularly the ability to change body colour and diurnal activity, chameleons have been made pet animals by some rich men. Cost of chameleon in international market varies between 10-200 US dollars. This practice is unlawful as per regulations of CITES (Convention on International Trade in Endangered Species of Wild Fauna and Flora). On the request of the Nomenclature Specialist of the CITES Animals Committee and the German Federal Agency for Nature Conservation (BfN), Frank Glaw a famous German herpetologist and a chameleon expert working at the Bavarian State Collection of Zoology at Munich evaluated the species diversity of the chameleons and published a taxonomic checklist of chameleons in 2015 (Glaw, 2015). As per this paper there are 202 species plus 23 subspecies in 12 genera of chameleons. Interestingly, it was observed that in many species there is a tendency of miniaturization. While Parson's chameleon *Calumma parsonii* is the largest chameleon measuring (snout to vent) about 68 cm (almost like a cat), *Brookesia nana*, often termed as nano chameleon measures only 16 mm (0.55 inch) for male and 19 mm (0.75 inch) for females. For many years *Brookesia micra* was the smallest

chameleon on earth; now its place is replaced by *B. nana* (Glaw et al. 2021). The evolutionary significance behind the miniaturization is not properly known. But it is thought that miniaturization is an evolutionary compromise to shift niche from tree to forest floor. The small chameleons live among litters in forest floor, and thus lack a prehensile tail and have the poor ability to change body colour. Chameleons live in the rain forest and deserts of Africa, Madagascar island being home to almost two third of all chameleon species. A few species are also available in Southern Europe, Middle East, Southern India and Srilanka. Loss of habitat and fragmentation of habitat due to encroachment of forests by expansion of agriculture and cities are greatest threats to diversity of chameleons. 34% of chameleon species are now included under different threatened categories (CR, EN, VU) and 18% of them are included under Near Threatened category (IUCN 2019). The Chapman's Pigmy Chameleon *Rhampholeon chapmanorum* was long thought to be extinct due to habitat pressure. Recently it was relocated in the low elevation rainforest of Malawi hills of southern Malawi, a country in South-Eastern Africa. 80% of the forest in this country has been destroyed since last 25 years. Since Chapman's Pigmy Chameleon is endemic to Malawi, the species is facing extreme threat of extinction with fragmented habitat, decline in population density and impaired gene flow (Tolley et al. 2020). Not only for the Pigmy Chapman's Chameleon, but for all forest dwelling species of chameleon, there is an urgent need to prevent destruction of forest and restoration of their habitats.

References

1. Glaw F. 2015. Taxonomic checklist of chameleons (Squamata, Chamaeleonidae). Vertebrate Zoology, 65(2): 167-247
2. Glaw, F., Köhler, J., Hawlitschek, O., Ratsoavina, F.M., Rakotoarison, A., Scherz, M.D., Vences, M. 2021. Extreme miniaturization of a new amniote vertebrate and insights into the evolution of genital size in chameleons. Scientific Reports 11:2522. https://doi.org/10.1038/s41598-020-80955-1 IUCN (2019) The IUCN Red List of Threatened Species. iucnredlist.org
3. Ligon RA, McGraw KJ. 2013 Chameleons communicate with complex colour changes during contests: different body regions convey different information. Biol Lett 9:20130892. http://dx.doi.org/10.1098/rsbl.2013.0892
4. Teyssier, J., Saenko, S., van der Marel, D. et al. Photonic crystals cause active colour change in chameleons. Nat Commun 6, 6368 (2015). https://doi.org/10.1038/ncomms7368
5. Tolley, K., TilburymC., Da Silva, J., Brown G., Chapeta, Y., Anderson, C. 2021. Clinging to survival: Critically Endangered Chapman's pygmy chameleon *Rhampholeon chapmanorum* persists in shrinking forest patches. Oryx, 1-6. DOI:10.1017/S0030605320000952

Are Noble Prizes Really 'Noble'

Dr. Ivy Kundu, Assistant Professor, PG Dept. of Zoology,
Barasat Govt. College

Introduction

Noble Prize are awarded every year in the memory of Swedish industrialist Alfred Nobel in the fields of physics, chemistry, physiology or medicine, literature and peace. The award is presented by Royal Swedish Academy of Sciences for inventions or discovery. Although highly acknowledged the Noble prize has always been surrounded by criticism and controversies since its inception. This article focuses on life and work of some renowned scientists who have made significant contribution in their respective fields of research which has lead to breakthrough discoveries in the field of Medicine or Physiology. However their work was not acknowledged by the Swedish Academy of Sciences on various occasions and they were deprived of the recognition they truly deserved. Nevertheless their research still remains priceless till date and would remain so forever.

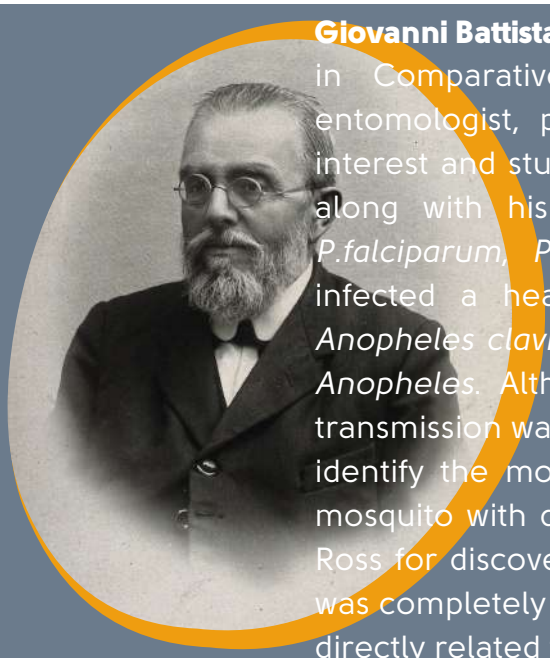
Shibasaburo Kitasato (1853–1931)

Kitasato one of the renowned bacteriologist was born at Ogunigo, Southern Japan in 1853 where he received his early education. Later he joined Medical School in Komamoto and Government Medical School at Tokyo and finally received his medical degree. In 1885 he travelled to Germany and worked under Robert Koch and was successful in obtaining pure culture of tetanus bacillus as well as *Clostridium chauvoei*, the causative agent of blackleg disease in cattle. Kitasato along with von Behring developed serum therapy for the treatment of patients infected with diptheria and tetanus and jointly published their research work. Thus Behring and Kitasato were the first researchers to use the passive immunization method in the fight and combating the spread of infectious diseases. The Nobel Prize in Physiology or Medicine in 1901 was however awarded solely to Emil Adolf von Behring while Kitasato contribution was completely obliterated. Kitasato returned to Japan in 1892 and founded the Institute for Infectious Diseases from which he resigned and later established the Kitasato Institute in 1914. In 1917 Kitasato became the first dean of the school of medicine of Keio University in Tokyo. In 1931 Kitasato succumbed to death due to cerebral hemorrhage.

Kitasato in Germany (Photo courtesy of the Kitasato Institute)

Kitasato with his mentor Robert Koch in Japan

Giovanni Battista Grassi (1854–1925)



Giovanni Battista Grassi, born in Italy on March 27, 1854 worked as Professor in Comparative Anatomy at University Regia, Rome. A renowned entomologist, physician as well as zoologist, in 1888 Grassi turned his interest and studied malaria cycles in various species of birds. Later on he along with his associates had described the complete life cycles of *P.falciparum*, *P.vivax* and *P.malariae*. In 1898 Grassi had successfully infected a healthy volunteer by exposing him to mosquito bites of *Anopheles claviger* and had described the entire cycle of *Plasmodium* in *Anopheles*. Although Ronald Ross, was the first to report that malaria transmission was caused by mosquito bites in 1897, he however had failed to identify the mosquito species correctly which he had described as 'grey mosquito with dappled wings'. In 1902 Noble prize was awarded to Ronald Ross for discovery oflife cycle malarial parasite. The contribution of Grassi was completely ignored by the Noble committee although his findings were directly related to uncover the mystery of transmission of malarial parasites.

Rosalind Franklin (1920–1958)

Rosalind Franklin, a chemist with doctorate degree from Cambridge University in 1945, worked as research associate in John Randall's laboratory at King's College, London. She had photographed the DNA's helical structure by X-ray crystallography which had laid the basis for deciphering the double helical structure of DNA. The photographed plates was shown by Maurice Wilkins to James Watson. Later Watson and Crick published in Nature journal the double helical structure of DNA titled "Molecular Structure of Nucleic acids-A Structure for Deoxyribose Nucleic Acid" in 1953 based on Franklin's unpublished crystallography data commonly known as "Photo 51". In 1962 James Watson, Francis Crick, and Maurice Wilkins were awarded the prize for their contribution in elucidating the double helical structure of DNA.Later in 1953 Franklin joined Crystallography Laboratory at Birkbeck College, London were she worked exhaustively on structures of virus particularly Tobacco Mosiac virus (TMV). Franklin who died in 1958 due to ovarian cancer was denied of the true recognition she deserved. Later Rosalind Franklin University of Medicine and Science was established in North Chicago to honor the scientist.

Rosalind Franklin

Diary of
Rosalind Franklin

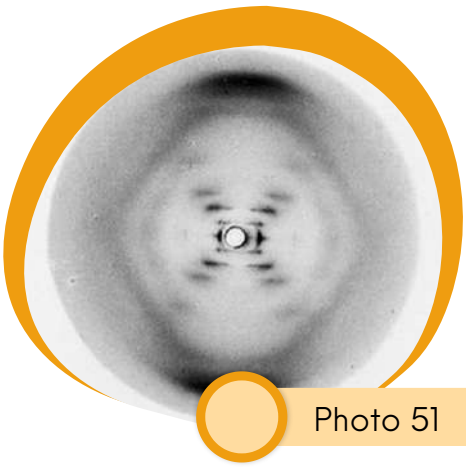


Photo 51

Esther Miriam Zimmer Lederberg (1922–2006)

Esther Miriam Zimmer Lederberg, American microbiologist. known her discovery of lambda phage, a virus that infects *E. coli* bacteria which later was used extensively for studying bacterial genetics and genetic recombination. Shehad published it as first report in Microbial Genetics Bulletin in 1951. Along with the discovery of lambda phage she is also known for discovering replica plating technique long with Joshua Lederberg which had played a pioneer role in microbial genetics. Joshua Lederberg, her husband along with George W.Beadle (1903–1989) and Edward Lawrie Tatum (1909–1975) in 1958 were awarded the Nobel Prize for Physiology or Medicine for discoveries on how bacteria can exchange DNA and create a new strain. Although Joshua and Esther had worked together and her discoveries were completely overshadowed.

Charles Herbert Best (1899 –1978)

In 1921 **Charles Herbert Best** with bachelor's degree in physiology and biochemistry at the University of Toronto was hired as a research assistant to J. J. R. Macleod, his former teacher who assigned him to Frederick Banting. Banting and Best began studies on diabetes through an experimental combination of duct ligation and pancreatectomies on dogs. After a series of trials they were able to isolate a chemical from pancreas extracts that enhanced the lives of dogs made diabetic by removal of the pancreas. On January 23, 1922, 14-year-old Leonard Thompson became the first person to receive an insulin injection as treatment for diabetes which improved his condition considerably. Later Macleod invited James Bertram Collip (1892–1965), a biochemist in the department of Physiology at the University of Toronto, to help Banting and Best with purifying their extract which he succeeded in 1922. In

Charles Herbert Best

1923, Banting and Macleodwere awarded the Nobel Prize in medicine although Charles Best, being a graduate student, was not included. Banting recognized Best's contribution by sharing the award money while Macleod shared his amount with Collip. Later insulin had become widely available for mass production by collaboration with Eli Lilly and Company laboratories in Indianapolis and abroad.


THE TORONTO DAILY STAR

TORONTO DOCTORS ON TRACK OF DIABETES CURE

RAILROAD UNIONS IN U.S. MAY JOIN IN MINERS' STRIKE

DIABETES SUFFERERS GIVEN MESSAGE OF HOPE

"DRY" LAWS HERE CAUSE HARDSHIP IN OLD FRANCE

RESERVES OF COAL ARE HIGH IN CITY, PRICE TO GO DOWN

CIVIL WAR STAGE NOT YET REACHED BUT DANGER GREAT

Report of discovery of Insulin in Toronto Daily in 1922

Robert C. Gallo (1937-Till Date)

Dr. R.C Gallo recognized internationally for his co-discovery of HIV as the cause of AIDSwas born in Waterbury, Connecticut on March 23, 193. His pioneering works lead to the discovery of retroviruses human leukemia virus - HTLV causing leukemias and lymphomas as well discovered interleukin-2 (IL-2). The development of the HIV blood test, helped healthcare workers to screen for the AIDS virus for the first time, as well as simultaneously protecting patients receiving blood transfusion. In 1986, his group also discovered the a new human herpes virus (HHV-6), which was known to cause an infantile disease Roseola. Gallo and his team identified a retrovirus which they called HTLV-III, which was later identified as HIV-I and concluded the virus as the causative agent of AIDS in 1984. However, on May 20, 1983, the French virologist Luc Montagnier and his team at the Pasteur Institute in Paris, had published their work identifying a retrovirus they called Lymphadenopathy Associated Virus (LAV), from a patient suffering from AIDS. After publication of Gallo papers a controversy followed that Gallo had acquired samples from Montagnier lab which was later cleared by NIH. The U.S. and French governments decided to settle the dispute out of court and share the royalties from the discoveries. In 2008, Luc Montagnier and Françoise Barré-Sinoussi were awarded the Nobel Prize for the isolation and characterization of HIV-1 and Gallo work was consigned to oblivion.

Conclusion

Although this article focuses on the work of few scientists, there are many more who were also not commemorated for their work by the Noble Prize committee. Nevertheless their contribution in science would remain immortal and insignificant of the fallacies of prizes and awards. Controversies surrounding the Noble prize lies in the policy of the committee of felicitating individuals (at the most 3) instead of the team involved. As most works are collaborative hence many scientists remains unacknowledged. Moreover the requirement of scientists to be alive at the time of award is absurd and should be reconsidered. Amongst all the controversies surrounding the award, Noble prize still retains its value in recognizing original works and discoveries. Thus advances should be made to increase the pursuit in science and building an environment for celebrating science in its true glory.

References

1. Abbadessa G et al. (2009).Unsung hero Robert C. Gallo. Science ;323: 206-207
2. Best C H and Scott DA (1923). The Preparation of Insulin. J. Biol. Chem; 57: 709–723
3. Brenda M (2003). "The Double Helix and the ´Wronged Heroine´." Nature ; 421: 407–408
4. Capanna E (2006). Battista Grassi: a zoologist for malaria. Contrib Sci; 3:187–195
5. Ferrell R V (2018)."Esther Miriam Zimmer Lederberg: Pioneer in Microbial Genetics". In Whitaker, Rachel J.; Barton, Hazel A. (eds.). Women in Microbiology. American Society for Microbiology. 305–315
6. Hernandez V (2019). "Photograph 51, by Rosalind Franklin (1952)". Embryo Project Encyclopedia 12-30. ISSN: 1940-5030
7. Kantha, S. S (1991). A centennial review; the 1890 tetanus antitoxin paper of von Behring and Kitasato and the related developments. Keio J Med; 40: 35–39
8. Mehlhorn H. (2015). Kitasato, Shibasaburo (1853–1931). In: Mehlhorn H. (eds) Encyclopedia of Parasitology. Springer, Berlin, Heidelberg
9. Rosenfeld L (2002). Insulin: discovery and controversy. Clin Chem ; 48(12): 2270–2288

Return to Ranthambhore

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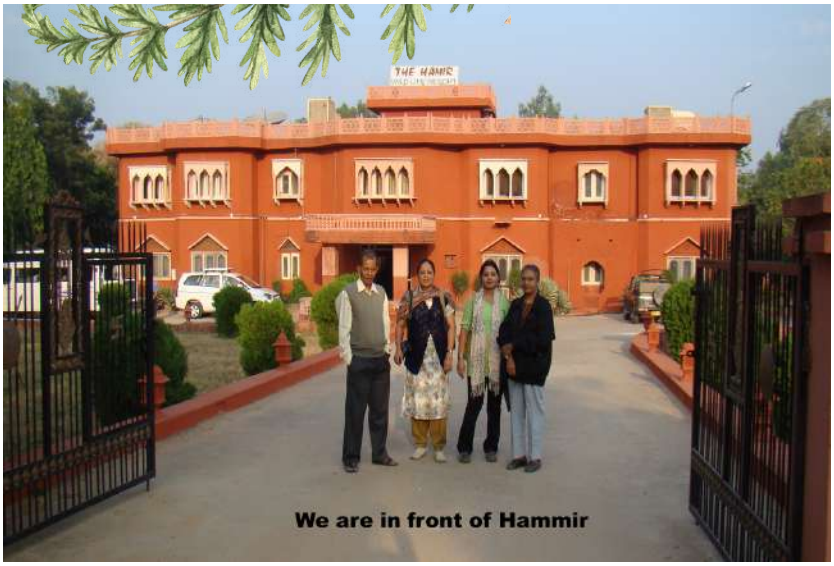
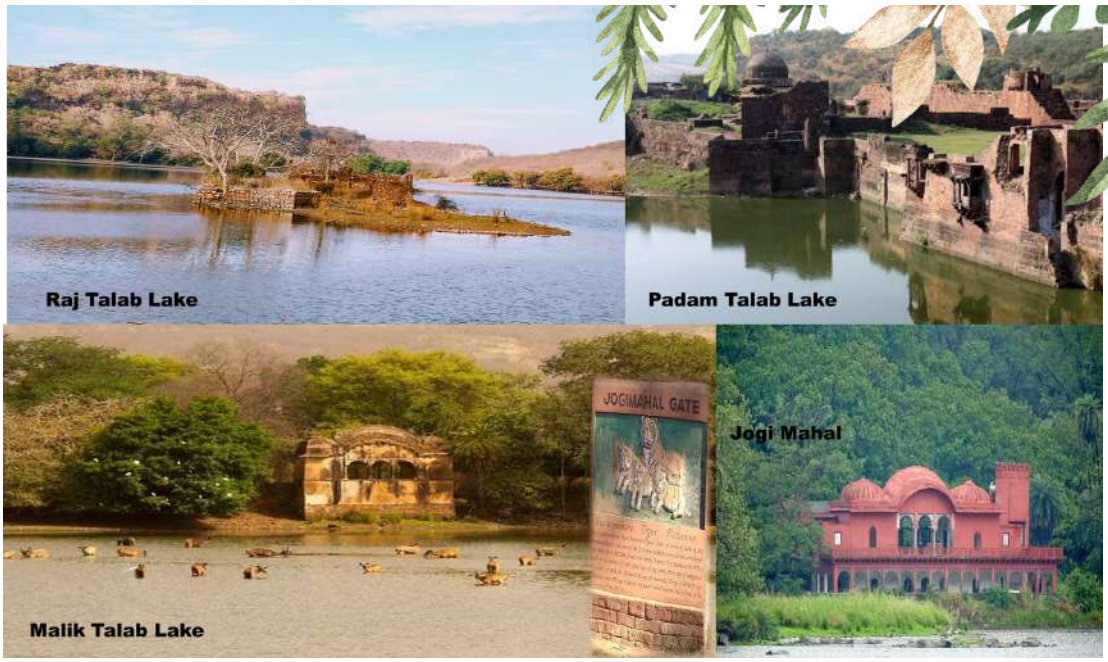
Though it was a long and tiring day of travel from Bharatpur to Sawai Madhopur our tour operator, Mr. Sengupta on behalf of 'Hindusthan Traveltour' finally accommodated us to wonderful luxurious 'Hammir Wildlife Resort'. The resort is 9 kms away from the main town, towards the park. It is a perfect blend of historical architecture of Rajasthan, luxury and wilderness. We took few minutes to refresh. Meanwhile, the two canters were ready to transport us in two groups to paradise. This was my first visit to Ranthambhore, a paradise of wildlife.

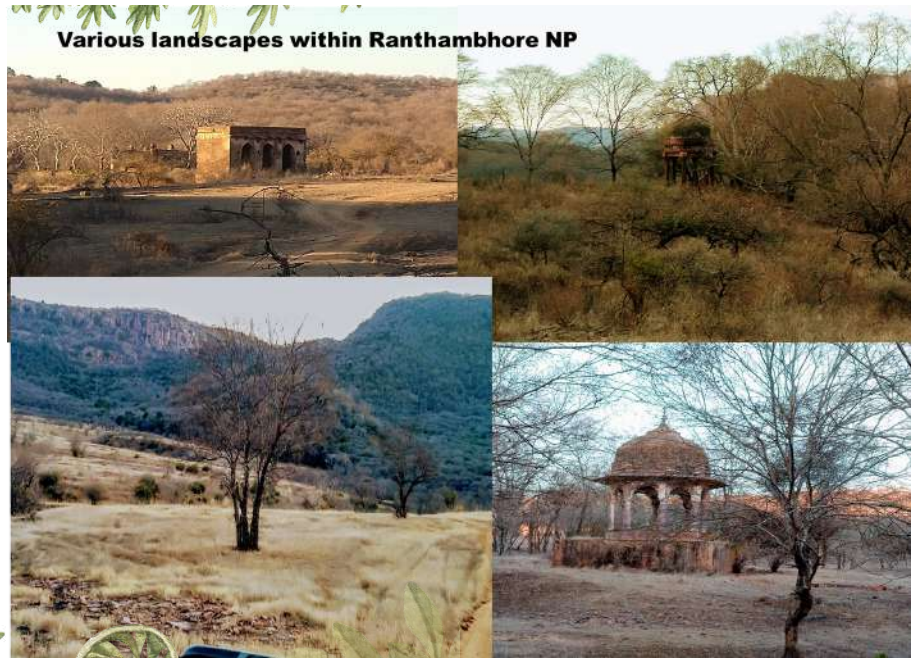


We are in front of Hammir

Ranthambhore Tiger Reserve lies on the junction of Aravali and Vindhyas, so the topography is highly undulating. The scenery changes dramatically from gentle and steep slopes of the

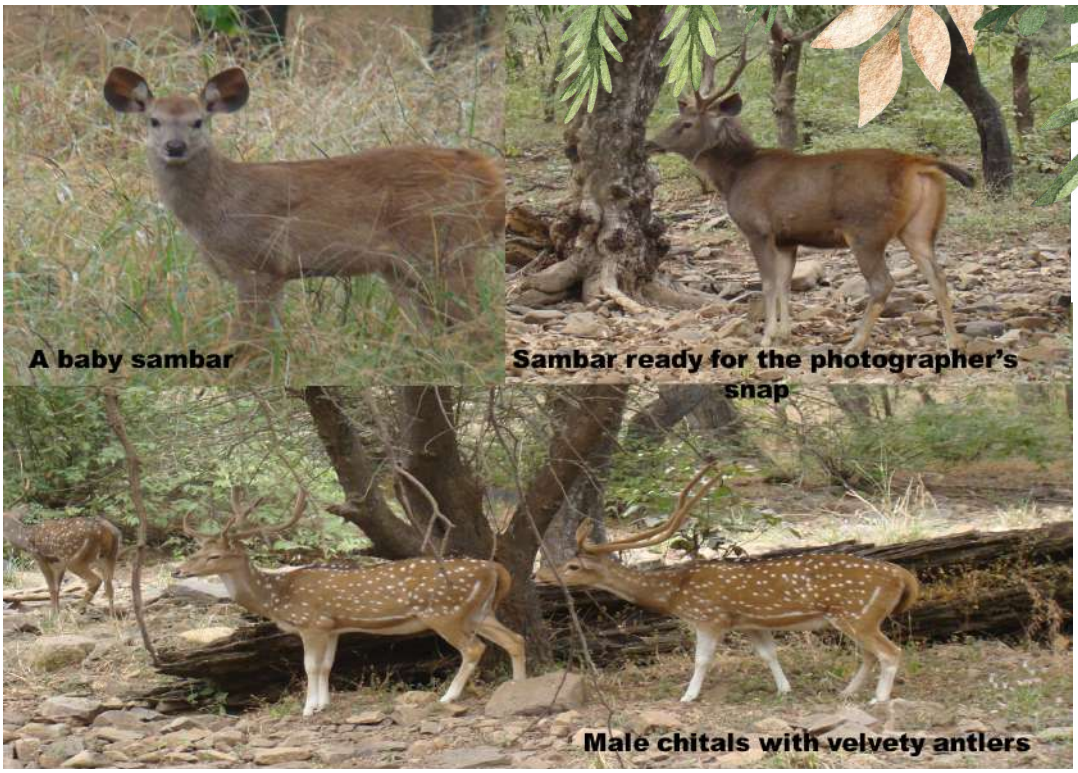
Vindhyas and sharp and conical hills of the Aravalies. A thousand year old fort also blends amicably with the background. Pure sands of Dhok interspersed with grasslands at the plateaus, meadows in valleys and luxuriant foliage around the canals make the jungle. Three big lakes namely Padam Talab, Malik Talab and Raj Talab are similar turquoises studded in the vast forest. Sight of 31 species of birds of 30 genera under 24 families in just 2 days initiated a steady state of emotion within us.





The wind bite through us even in the afternoon sun as we zipped towards the majestic gateway into the park. A caped langur greeted us with its welcome sitting on the park gate. The ramparts of Ranthambhore fort soon came into view. The thrill of entering the forest was splendid. Our sharp-eyed and knowledgeable naturist took us through the paces of Ranthambhore's astonishing afternoon moods and heavy wilderness terrain. First we saw a pair of jackals leisurely crossing the road behind our cantor. Rose ringed and plum headed parakeets, rufous treepies and red-vented bulbuls noisily ushered us in, while the silvery coats of the langurs glisten as they were playing or sitting around with thoughtful looks, hands on knees.

The gentle chital lifted their heads at the canter's sound, the males with velvety antlers. Sambars' graze and a regal stag were ready for the photographers. Nilgai galloped off at our approach, clumsily graceful and a mongoose with a black-tipped tail scurried across the path.



We stopped to look at a family of spotted owlets –two little heads popping out of a hole, another on a branch of the same tree. Their half-open widened eyes and lovely yellow irises gazed at us. That evening, after watching a handsome female wild boar with a shiny pink snout along with its kid for a while, we heard alarm calls. There were a group of jeeps and another canter at the place, and our king tiger was there, lying in the grass, but she refused to let us see her in all her glory though she did raise her head from time to time, wave her ears and tail and lick her paws. She lied there, almost flat on the grass, her beautiful colour blending marvelously with the surroundings. It was time to leave and our canter, first to drive off, unluckily for us, because after that she got up and crossed the path for all the others to see.

That evening we returned to Hammir with great excitement. There can be no greater joy than seeing a tiger in the wild. Standing out in stark contrast to the desert landscape of Rajasthan, Ranthambhore National Park offers an unprecedented, magical escape. The tiger is king here but this reserve is a mix of everything - wildlife, culture, history, religion and heart-pounding excitement. That evening at Hammir we were entertained with a cultural programme by Rajasthani musicians and dancers along with camp fire organized by our students.


Cultural programme performing by Rajasthani musicians and dancers

Our next morning ride was more eventful. Few hours of torrential rain in the midnight did transform the forest winter landscape into too chilly but an emerald paradise. We experienced the owlets again, this time two in one hole, one in another. Then two collard scope owls, sitting side by side, turned their "horned" heads to peer at us. Later, we came to one of the three lakes within park, Padam Talab, which attracts a number of migratory birds including ducks and geese. In the water, we did spot common moorhen, spot billed duck, Indian pond heron, river tern, great thick knee, black tailed godwit, gadwall and the ubiquitous red-wattled lapewing. Crocodiles lazed on the banks of the jheel, some floating just below the surface of the water and checkered keelback – water snake taking sun bath lifting their heads above on the rocks. An oriental ibis dipped for fish, while a darter stretched its wings, and a black-winged stilt stalks in water. As we drove around the Lakarda area, we stopped to watch a white-throated kingfisher on a branch near a rocky outcrop which leads down to a patch of water.


Crocodile lazed on the jheel bank

Darter stretched its wings

Black-winged stilt stalking in water

checkered keelback taking sun bath lifting their heads above on the rocks

That afternoon I was in a canter with lot of foreigners. We went to the Sultanpur area. Here one gets to experience the forest at its best, driving down tree lined paths which open into scrub, grassland, rocky landscape-peace and quiet. We saw less animals and birds in this park, just a few of the usual langur, chital, sambar, nilgai, peacock and the delicate chinkara with their curly antlers. Near the water were two woolly necked stork surveying the jheel and pond heron perched on a sambar. That afternoon we climbed upto the fort, getting spectacular views of the park at each turn. Looking over the ramparts we saw a horde of crocs sunning themselves on the little island in Padam Talab. Langurs eyed our bags, hoping to get a bite to eat. We spent sometime at Jogi Mahal with its magnificent banyan tree. A raptor settled on a tall stump of a leafless deciduous tree and after a lot of argument we concluded it's a crested serpent eagle. The sun was getting low in the sky, just then a series of langur and chital alarm calls begun, the peacocks also joining it in. Our driver turned around again, and set off at a frantic pace towards the sound.

Magnificent banyan tree

A crested serpent eagle settled on a tall stump of a leafless tree

A treepie took piece of biscuits from our hands

We found a large group of chital scattered on both sides of the road, a lots of langurs on the walls of the ramparts of the fort, frozen and looking terrified and suddenly a group of wild boar galloped away in panic. At that time we noticed nothing, but we soon returned to the spot and there we saw what we're all been longing for – a leopard on a mountain. We watched her preen herself with high resolution binocular.

A leopard on a mountain

We got a breathtaking twenty minutes of her before she decided she's had enough. In high spirits we proceeded on the drive. We stopped at a checkpoint where treepies and jungle babblers abound. The treepies took piece of biscuits from our hands. We drove on, drinking in the beauty for the last time before we say "Goodbye Ranthambhore, we shall return".

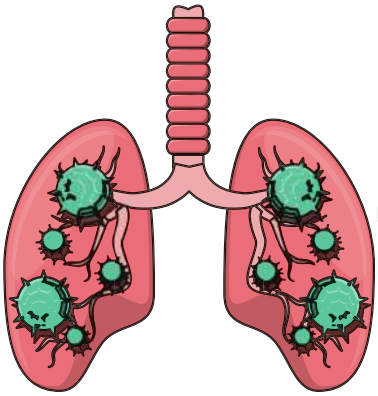
Indian Grey Hornbill (*Ocyceros birostris*) (Scopoli, 1786)

Dr. Jayati Ghosh, Associate Professor, PG Dept. of Zoology, Barasat Govt. College

Indian grey hornbill is endemic species in Indian subcontinent found mainly on the plains up to about 2000 feet. It is common in India, Pakistan, Bangladesh and Nepal. They are widely distributed in foothills of Himalaya and south Indian peninsula. These birds were sighted and photographed by author in Satkosia wildlife sanctuary during February, 2021. They have a peculiar large, horn-like casques over the bill.

Their dorsal body surface is covered with grey hairs and the abdominal side is light coloured. The adult is with red eyes, black and yellow beak. The tail is long with black base. They are fruit-loving birds. They are mostly found in pairs or in small groups. They also feed on insects and snails. But they have special preference for fig plants. These photographs were taken in early morning when they move high up in the tree canopy.

The nesting period of these birds is February to June. Plenty of food is available for chicks to grow during this period. Nesting is done in tree hollows on tall trees. The female remains inside the nest and the entrance is sealed by the female using her excreta and mud pellets provided by male partner. Only a narrow slit remains open from which male feeds the female. When chicks come out of eggs and grow sufficiently, female bird breaks open the nest.



Autophagy: A Novel Treatment Strategy for Host - Directed Therapies Against Tuberculosis

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Autophagy is an intracellular catabolic process that helps maintain homeostasis or removal of invading bacteria through the process of lysosomal degeneration and acts as an independent cell defence against intracellular microbes including Mycobacterium. Tuberculosis (TB), the world's leading health threat, is mainly caused by the major etiologic agent *Mycobacterium tuberculosis* (Mtb) infecting about one third of the world's population with about 10% having active, active TB throughout life to them. The current recommended treatment for long-term TB, used by WHO, includes the administration of first-line antibiotics isoniazid (INH), rifampicin (RIF), pyrazinamide (PZA), and ethambutol for 2 months. INH and RIF 4 months.

But this anti-TB treatment has many drawbacks such as long-term treatment, drug toxicity and the potential risk of developing drug-resistant strains if patients do not comply. As Mtb can improve resistance [Multidrug resistance (MDR); Extensive drug resistance (XDR)] very quickly with each drug, standard TB treatment [targeted treatment, short-term therapy (DOTs)] with a combination of these four drugs was introduced in the 1980s. Since then, DOTs have been shown to be effective in achieving microbiological treatment in patients with drug-resistant TB.

Depressive conditions, including HIV infection, diabetes etc. make treatment difficult for TB and increase the mortality rate of patients. Therefore, advanced TB treatment strategies are much needed. Increasing numbers of MDR-TB and XDR-TB underscore the need to develop alternative or complementary therapies that could speed up and improve TB treatment through adherence. Host-directed therapy (HDT) is a promising and emerging concept in the treatment of TB, where automatic control of Mtb and continuous disease prevention provides care-based treatment options because autophagy can be medically modified with smaller molecules and incorporate antibiotics to achieve better TB control. However, current knowledge and clinical evidence are not enough to use HDT molecules as a stand-alone, without adjunct antibiotics, a treatment modality for any form of TB in humans.

Autophagy is important in controlling the range of immune responses, including defences against Mtb. Autophagy is caused by a number of depressive symptoms including starvation, hypoxia, damage to the intracellular organelles, and minor ailments. The combination of antimicrobial and anti-inflammatory actions of autophagy prevents active disease in animal models. In population statistics, the genetic links between autophagy, inflammatory bowel disease, and the risk of tuberculosis provide additional support for these integrated autophagy roles. The autophagy process begins

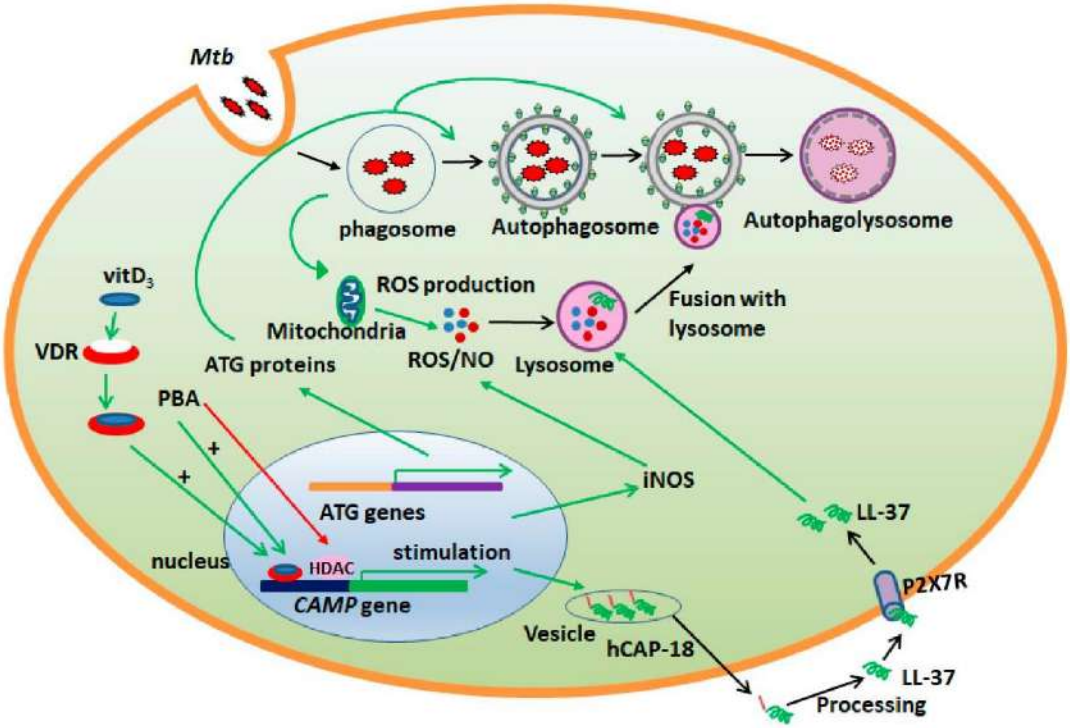
with the formation of the autophagosome, which is a double-membraned vesicle that contains cytoplasmic substances or phagocytic bacteria. These autophagosomes do not degrade until they interact with the lysosomes, forming the autophagolysosome, making it easier to degrade their contents. Xenophagy is a type of autophagy that describes the process of bringing intracellular bacteria to lysosomes through autophagic processes. It has been reported that cell cargo protein ubiquitin detects excess Mtb proteins and activates the xenophagy management process to control the intracellular growth of Mtb. The autophagy is primarily controlled by the mammalian target of rapamycin (mTOR) complex 1 and adenosine monophosphate-activated protein kinase (AMPK).

Activated macrophages are able to kill Mtb, which includes autophagy. However, it is known that harmful forms of Mtb can prevent autophagosome and lysosome synthesis and subsequent acidification of autophagolysosomal compartments by releasing antacid, 1-tuberculosinyladenosine (1-TbAd). This is an important step in the killing of Mtb by the autophagy process, thus, avoiding the immune response to survive within macrophages. The improved Mtb Eis protein (intracellular survival) prevents autophagy activation in macrophages and cell death in the form of living oxygen (ROS). In addition, 6 kDa early secretory antigen target (ESAT6), ESAT-6 secretion system-1 (ESX-1) - mediated secretion protein-mediated, plays an important role in suppressing later stage autophagy in human dendritic cells.

Tuberculosis is often associated with the immune-deviation of the host. Ineffective or incorrect Treatment leads to increased morbidity associated with Mtb, in particular, MDR and XDR Mtb infections pain, before starting effective treatment according to drug-resistant profiles. Since very few are young Strong anti-TB drugs are on the way soon, targeted treatment of the host (HDT) an attractive additional way to restore or improve host immunity. The goal can be to expand disinfection, while minimizing inflammatory tissue damage and common anti-TB drugs. HDT includes non-microbicidal agents and, instead, these host moderating agents immune, anti-Mtb and may further enhance or share anti-TB activity drugs. These new methods are called adjunctive therapy. Different clinical studies demonstrate that host-oriented therapies work using novel techniques the ability to reduce the duration of treatment, reduce infection, and improve outcomes in MDR-TB. Most HDTs focus on the most well-maintained, exhausting host display modes the risk of developing resistance.

As a strategy to prevent antagonism, intracellular Mtb selects autophagy to survive inside macrophages, preventing infected macrophages from entering apoptosis. Cytosolic Mtb DNA is identified by cyclic GMP-AMP synthase (cGAS) and gene-dependent interferon (STING) gene-mediated pathways, leading to direct bacterial uptake, which directs viruses to autophagy. These two methods have been shown to activate Type IFN and promote inflammation, which plays a key role in determining the clinical outcome of Mtb infection. The STING-based cytosolic pathway and autophagic receptors sequestosome 1 (SQSTM1) / p62 and the nucleotid protein nucleus 52 kDa (NDP52) play an important role in eliminating Mtb deficiency. The autophagy process is hindered by the activation of the mTOR complex, which focuses on the possible

treatment of Mtb. Since autophagy has emerged as an important protective mechanism to limit the growth of Mtb in host cells, it is appropriate to develop targeted home-based treatment against tuberculosis, which focuses on the functioning of autophagy. We have shown that 4-phenylbutyrate (PBA) and / or vitamin D3 (vitD3), overcome Mtb-induced inhibition of autophagy in human macrophages by a protective peptide that binds LL-37.



Host directed therapy (HDT) and macrophage immune defence against Mycobacterium tuberculosis (Mtb)

Host directed therapy (HDT) and macrophage immunity against *Mycobacterium tuberculosis* (Mtb). Macrophages are the natural host of Mtb. Mtb prevents the formation of autophagolysosome by blocking the association of autophagosome with lysosome, preventing autophagolysosome acidification, which induces the intracellular survival of Mtb. HDT compounds (vitamin D3 and phenylbutyrate) can activate the host of autophagy, which leads to autophagolysosome formation and controls Mtb growth. The production of active oxygen species (ROS) increases over Mtb infection. Treatment of Vitamin D3 (vitD3) and phenylbutyrate (PBA) triggers the production of the anti-bacterial peptide LL-37 by VDR (vitamin D receptor) or inhibition of histone deacetylase, respectively, in the genetic form of cathelicidin antimicrobial peptide (CAMP) (encoding). -18 / LL-37) promoter. The development of LL-37, inducible nitric oxide synthase (NOS) and ATG proteins in macrophages triggers the autophagy process and contributes to the killing of Mtb. ATG, related to autophagy; HDAC, histone deacetylase; NO, nitric oxide; P2X7R, purinergic receptor P2X7. The green arrows indicate the stimulus of the process and the red arrow indicates the inhibition of the process.

Table: HDT Related Compounds & Their Host Target Pathways Related to Mtb-Control

HDT Compound	Host Target	Mechanism Of Action	Effect	References
Rapamycin	Autophagy	Inhibition of mTOR	Promotion of autophagy	Corcelle et al.
Enbrel	Granuloma	TNF-α neutralization	Disrupts granuloma and reduces lung pathology	Bourigault et al.
Vitamin D3	Autophagy via cathelicidin	Stimulation of vitamin D receptor to induce cathelicidin expression; upregulation the expression of Atg5 and Beclin-1	Immunodulation and direct antimicrobial activity	Yuk et al.
Bevacizumab	Granuloma	Neutralizes VGEF	Normalize the vascular structure, decreases the hypoxia, and facilitates the entry of drug in the granuloma	Oehlers et al., Datta et al.
Metformin	Autophagy	Reduction phosphorylation of mTOR and p70S6K	Induction of mitochondrial ROS, phagolysosome fusion and increase MTB-infected cell apoptosis	Singhal et al.
Valproic acid	Autophagy	Reduction phosphorylation of mTOR and PI3-Kinase	Inhibition TBK-1, reduction TNF-mediated tissue damage	Wang et al.
Aspirin	Eicosanoids	Enhance the LXA4 production	Activates vitamin D-mediated anti-mycobacterial activities	Tobin et al.
Doxycycline, SB-3CT	Ribosomal binding	Inhibits the expression of MMPs	Reduces the bacterial load in the lung	Walker et al., Majeed et al.

A major limitation of this review is the limited information obtained from in vitro studies showing that using autophagy is beneficial in preventing the survival of Mtb in host cells. Additionally, there may be unintended consequences for modifying autophagy-modulating small molecules or agents. Studies using mouse models of autophagy genes have provided valuable information to understand how autophagy works against mycobacterial infections. There is evidence to suggest beneficial effects of GTPase IRGM on cell-autonomous protective immunity against mycobacterial infection in murine cells and human cells. However, there is clear evidence of the diversity of certain species. For example, a large axis of autophagy and immune defences including vitamin D and cathelicidin do not work in rat models. Future research on autophagy-targeted anti-mycobacterial response in vivo authorized the development of new anti-TB drugs and evaluation of non-targeted outcomes as well as specific similarities and differences.

Conclusion

M. tb is an intracellular pathogen that alters the ability of the host phagocytic cells to clear an infection. Exploitation of the natural immune response should contribute to the killing of M. tb inside the cell. Autophagy activation by various compounds may represent a promising treatment strategy against TB infection which includes drug-resistant strains. Key mediators of autophagy, which include the signature of vitamin D receptor and AMP-activated protein kinase pathway, are critical in identifying compounds that can be used as HDT. Understanding the mechanisms and key players involved in compiling antibacterial autophagy will provide new advances in anti-TB treatment through autophagy-targeting targeting. Therefore, the identification of novel compounds and the pharmacological target (in the immune system) target that can enhance and facilitate an effective immune response to help eradicate TB bacilli is an attractive approach.

References

1. Gina, P., Davids, M., Dheda, K. (2019). Manipulation of autophagy for host-directed tuberculosis therapy. African Journal of Thoracic and Critical Care Medicine. 25 (2): 49-54.
2. Paik, S., Kim, J. K., Chung, C., Jo, E. K. (2018). Autophagy: A new strategy for host-directed therapy of tuberculosis.Virulence. 10: 448-459.
3. Ahmed, S., Raqib, R., Guðmundsson, G. H., Bergman, P., Agerberth, B., Rekha, R.S. (2020). Host-Directed Therapy as a Novel Treatment Strategy to Overcome Tuberculosis: Targeting Immune Modulation. MDPI. 9(21).
4. Kolloli, A., Subbian, S. (2017). Host-Directed Therapeutic Strategies for Tuberculosis. National Library of Medicine. 4: 171.

Curious Cases Behind 'Photo 51' & Beyond

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'Science for me, gives a partial explanation for life. In so far as it goes, it I based on fact, experience and experiment' – this widely known famous quote was stated by an unsung hero, often less prioritized, supreme genius yet too much underrated ' The dark lady of DNA' (by Barbara Maddox), a British chemist; Rosalind Franklin.

It was evident very early when she first stepped in the King's College of London on 1951 at the age of 31. Born in 1920; A passionate, dedicated, calm and composed yet rebellious, extremely brilliant with inventory thought depicts Rosalind Franklin the best. She was an enigma at that time. She started schooling previously at St. Pauls' and completed her study from Cambridge University in Chemistry till 1938, worked in **CURA** (**Coal Utilization Research Association**) till 1947 and learned X-ray crystallography from **Mareel Mathieu**.

Her times in King's College London was the most significant chapter of her life where she worked upon one of the ground breaking discovery of all time not only in science but also for humanity that is to reveal the double helix structure of DNA which was still under the mystery. She faced many facets of challenges of being woman and also being a Jew but she was determined and disciplined on her work. She used to work intensely in the lab mostly on the X-ray crystallography to reveal the actual structure of DNA double helix. She along with her assistant **Gosling** continued to excel in their work. The guide of Franklin was **Maurice Wilkins** who was envious on his research scholar, never gave the credit and also used to torture mentally to Franklin and unethically wanted to reveal all of her work to his close friend **JD Watson** who was also working on the same purpose in another place names as **Cavendish Laboratory** along with his senior **Francois Crick**, they were trying to reveal the DNA structure in theoretically and were perplexed about the position of **H-bond** in double helix

structure. Rosalind Franklin was spending her time and as a true perfectionist she used to take the near perfect conclusive evident on the X-ray crystallographic image of DNA double helix for 50 times and took 100 hours to make famous 'photo 51'; the most perfect image was the 51st one. That's why the number 51 is very significant in Franklin's life, as a full timeUnfortunately her tremendous hard work had been unethically robbed by Wilkins and helped Watson and Crick to solve the puzzle of DNA double helix model. Later in life Watson became a good friend of Franklin but at that time Watson also betrayed Franklin. **Watson, Crick and Wilkins awarded Nobel Prize on 1962 for the discovery of DNA double helix model and the ill fortunate Franklin died in ovarian cancer on 1958 at the age of 38 only. She was fallen ill during her work according to Gosling.**

So, this was the epitome of a tragic story of science, excellent researcher, and amazing personality of Franklin. She got her credit after the death when whole scenario came in front, during her time she never got any recognition about her work. Now she is called as '**mother of DNA double helix model**'. Very few people knows that she was very keen to study in the field of virology also, she described the 3D structure of **TMV** (**tomato mosaic virus**), depicted the structures of **coal and graphite**. At the time of her death, she was working on the molecular structure of viruses with her colleague **Aaron Klug**, who received a Nobel Prize for the work in 1982.

Confrontation when cornered was Rosalind's tactic. The alternative- passive acquiescence in something she knew to be wrong – was intolerable, totally contradictory to her faith in the provable truth of science.

References

1. Shampo, Marc A. (2000). "The Millennium and Medicine: The 10 Most Influential Persons". Mayo Clinic Proceedings. 75 (1): 119–121.
2. Anachkova, Boyka; Djeliova, V.; Russev, G. (2005). "Nuclear matrix support of DNA replication". Journal of Cellular Biochemistry. 96 (5): 951–961.
3. Rutherford, Adam (25 April 2013). "DNA double helix: discovery that led to 60 years of biological revolution". The Guardian. Retrieved 17 December 2017.
4. Klug, A. (1974). "Rosalind Franklin and the double helix". Nature. 248 (5451): 787–788.
5. Watson, James D. (1968). The Double Helix: A Personal Account of the Discovery of the Structure of DNA. New York: Atheneum.

Cerebral Malaria: Most Dangerous Neurological Manifestation of Severe Malaria

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Malaria is a life-threatening mosquito-borne disease caused by a protozoan parasite called *Plasmodium*. The term malaria originates from Medieval Italian: *mala aria* – bad all. There are five species of *Plasmodium* responsible for malaria in man; *Plasmodium vivax* (*P. vivax*), *Plasmodium falciparum* (*P. falciparum*), *Plasmodium malariae* (*P. malariae*), *Plasmodium ovale* (*P. ovale*) and *Plasmodium knowlesi* (*P. knowlesi*). Malaria is one of the oldest human diseases. In human, *Plasmodium falciparum* is responsible for severe disease.

In severe *P. falciparum* infection, cerebral malaria is the most common complication. The majority of burden of disease occurs in Sub-Saharan Africa. The disease is transmitted to man by bite of a female mosquito. The mosquito introduces the parasites into the human's blood through its saliva and after the entry into blood, the parasite passes to the liver where they mature and reproduce. The basic defects found to be clogging of the cerebral microcirculation by the parasitized red cells. As a result parasitic RBC adhere to the endothelium of capillaries. This results in the sequestration of the parasites in the deeper blood vessels. This adhesion leads to the decrease in blood flow and cause blood damage and coma during cerebral malaria. Cerebral malaria (CM) is characterized by a range of acute neurological manifestations including a diffuse encephalopathy, changes in levels of consciousness, deep coma and seizure preceding death.

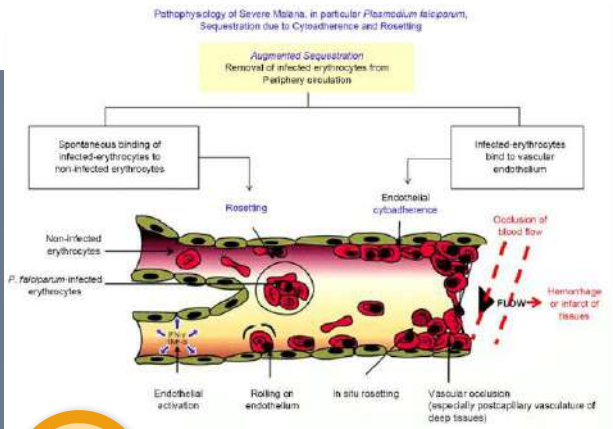
Definition of Cerebral Malaria

The term "cerebral malaria" has been used in medical literature to describe any disturbances in Central Nervous System (CNS). Cerebral malaria is defined as a deep level of unconsciousness (inability to localise a painful stimulus) in the presence of *P. falciparum* asexual parasitaemia. In adults, coma is required for more than 6 hours but in case of children it is reduced to 1 hours. In fatal cases, the cerebral malaria is diagnosed by cerebral capillaries and venules packed with PRBCs. In clinical practices, any impairment of consciousness and cerebral dysfunction is an indication for treatment and intensive care management.

Features of Cerebral Malaria

- **Sequestration**

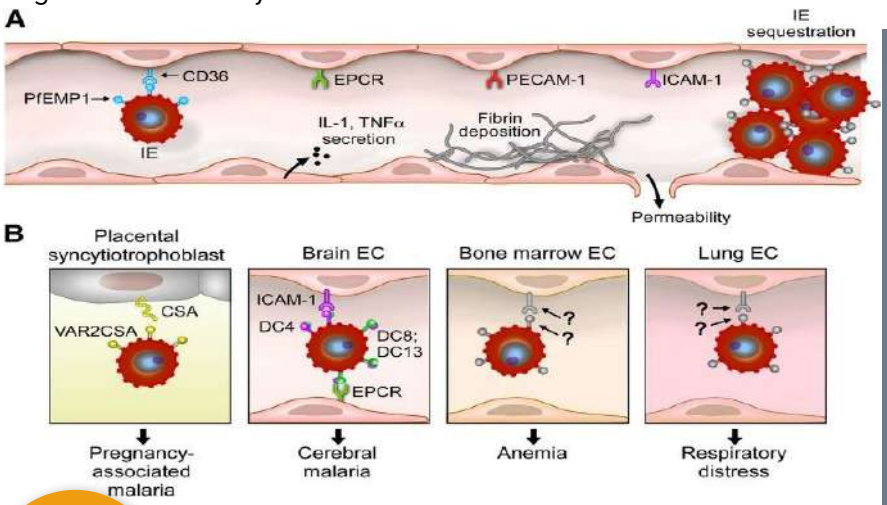
It is a process by which red blood cells containing mature parasites adhere to microvasculature and disappear from the circulation. As a result, in a peripheral blood slide, mature parasites are detected. The sequestration of red cells containing mature forms of the parasite in the microvasculature is thought to cause the major complications of *falciparum* malaria, particularly cerebral malaria. The sequestration of PRBCs allows optimal parasite growth and prevents the PRBCs from being destroyed by the spleen. By autopsy studies it is shown that sequestration is not only distributed throughout the body and is greatest in the brain- particularly in the white matter, but also present in the eye, heart, eye, kidneys, intestines, and adipose tissue.



Sequestration due to cytoadherence of infected erythrocytes

• Cytoadherence

Several hypotheses associated with the binding of PRBCs in the microvasculature have been proposed and reviewed, such as (i) changes of the RBC and PRBCs rigidity, (ii) pro-inflammatory induction of the adhesionreceptor expression, (iii) binding of PRBCs to specific adhesion receptors on endothelial cells, (iv) endothelial activation, and (v) malaria toxins, with various levels of evidence to support them. Sequestration is actually a specific interaction between PRBCs and the vascular endothelium (cytoadherence). The adhesion of the PRBCs reduces the microvascular blood flow, which may explain organ and tissue dysfunction such as coma.



PfEMP1-endothelial receptor interactions mediate microvascular sequestration of *P. falciparum* IEs (Infected Erythrocytes)

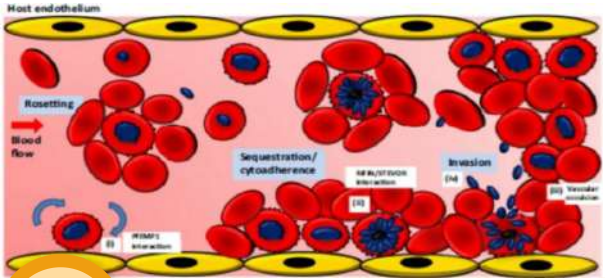
This figure shows a typical micro vessel found in a variety of organs and tissues in patients with malaria.

The parasite shown (**A**) on the left is expressing a PfEMP1 variant on the IE (infected erythrocytes) surface that binds CD36 on ECs (endothelial cells). In addition to CD36, EPCR, PECAM-1, and ICAM-1 are also expressed by ECs. IE binding to these receptors is encoded by specific PfEMP1 domain cassettes (DCs): DC8 and DC13 bind EPCR, DC5 binds PECAM-1, and DC4 binds ICAM-1. Thus, the endothelium cells are activated by developing parasites and downstream events such as secretion of proinflammatory cytokines, deposition of fibrin, and loss of barrier integrity. As a results, microvascular inflammation, obstruction, and perivascular leakage occurs.

IE sequestration in four different organs (**B**); Pregnancy-associated malaria is an organ-specific syndrome initiated by the expression of the PfEMP1 variant VAR2CSA (DC2), which mediates IE binding to placental CSA (chondroitin sulphate A) expressed by syncytio-trophoblasts. In other organs (lung) these same DC8and DC13-expressing IEs may contribute to disease but other receptor–DC-binding pairs are proposed to cause organ-specific clinical syndromes (respiratory distress). Like CM, respiratory distress and anaemia are organ-specific malaria syndromes that may occur alone or in combination with CM.

• Rosetting

Binding of two or more parasitic RBCs to an infected RBC is called rosetting. It occurs during the middle of asexual life cycle. Rosetting is very much associated with cerebral malaria and cytoadherence. Rosetting encourages adherence and the adherence of NPRBCs (Non-parasitized RBC) to PRBCs (rosetting) and PRBCs (Parasitized) to PRBCs (agglutination), have also been implicated in the pathogenesis of cerebral malaria. Rosetting may be inhibited by drugs like artemisinin and quinine.



Rosette formation between uninfected and infected RBC

References

1. Air WC, Mosnier LO, Fairhurst RM. *Plasmodium falciparum* picks (on) EPCR. Blood. 2014; 123(2): 163-167
2. Bansal D, Herbert F, Lim P, Deshpande P, Becavin C, et al. IgG autoantibody to Brain Beta Tubulin III associated with cytokine cluster-II discriminate cerebral malaria in Central India. PLoS ONE. 2009; 4(12): e8245
3. Oluwayemi IO. Cerebral malaria. Malar Chemoth Cont Elimination. 2014; 3(1)
4. Yam XY, Niang M, Madnani KG, Preiser PR. Three resetting in *Plasmodium falciparum*. Trends in Parasitol. 2017; 12

Chytridiomycosis : Amphibian Chytrid Fungus Disease

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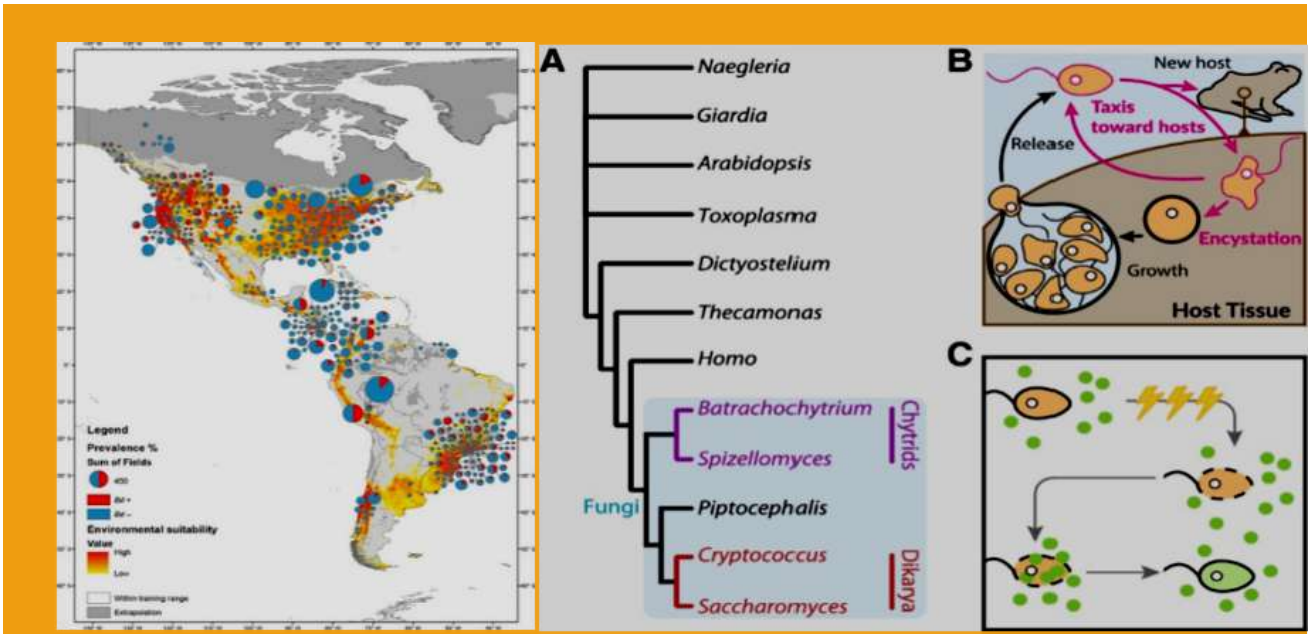
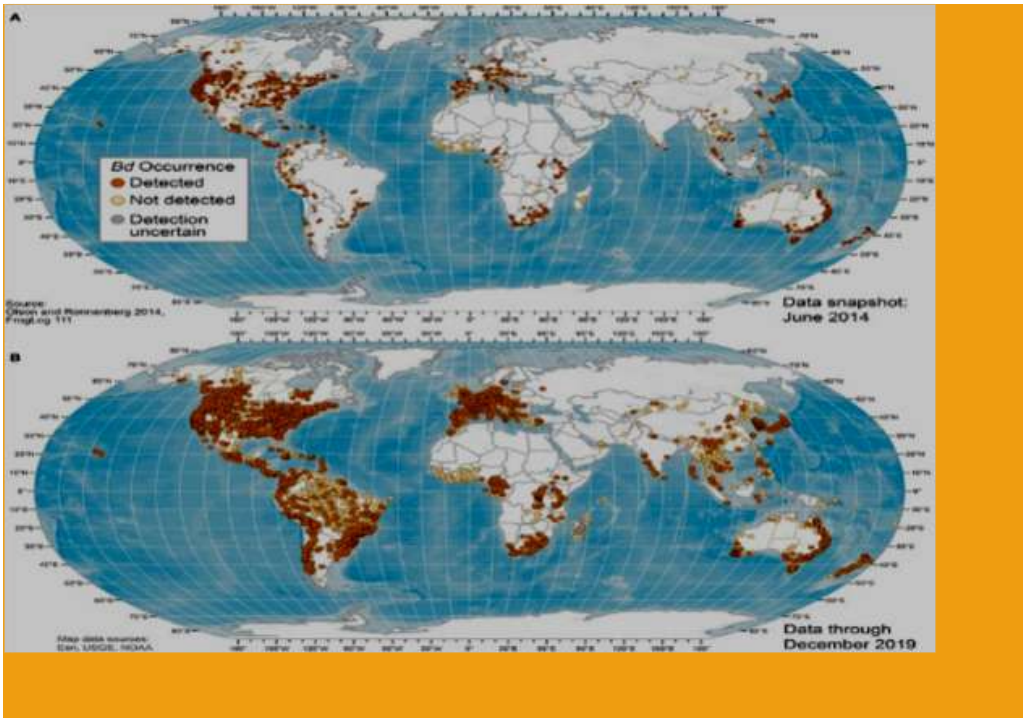
Introduction

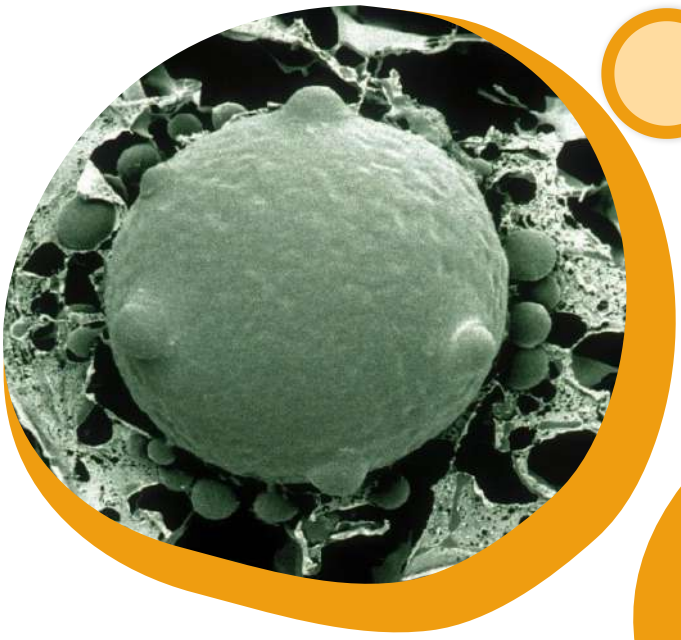
A non–hyphal zoosporic Chytrid fungus *Batrachochytrium dendrobatidis* and *Batrachochytrium salamandrivorans* is responsible to cause this infectious disease in amphibians. Worldwide, it has been linked to declination or extinction of amphibian populations mostly in parts of North America, Central America, East Africa Eastern Australia & so on. This fungus is capable of causing sporadic deaths in some amphibian population and 100 % mortality in others. Various clinical signs are seen in populations affected with this disease.





History

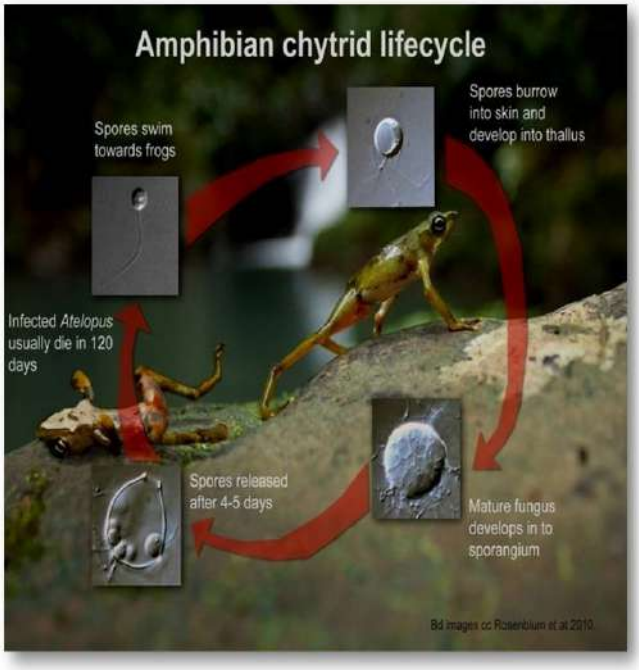
The disease in its epizootic form was first discovered in 1993 in dead and dying frogs of Queensland, Australia. Among frogs, the oldest documented occurrence of *Batrachochytrium* is from a specimen of a Titicaca water frog collected in 1863 and among salamanders the oldest was a Japanese giant salamander collected in 1902. Both are linked to mortality events.

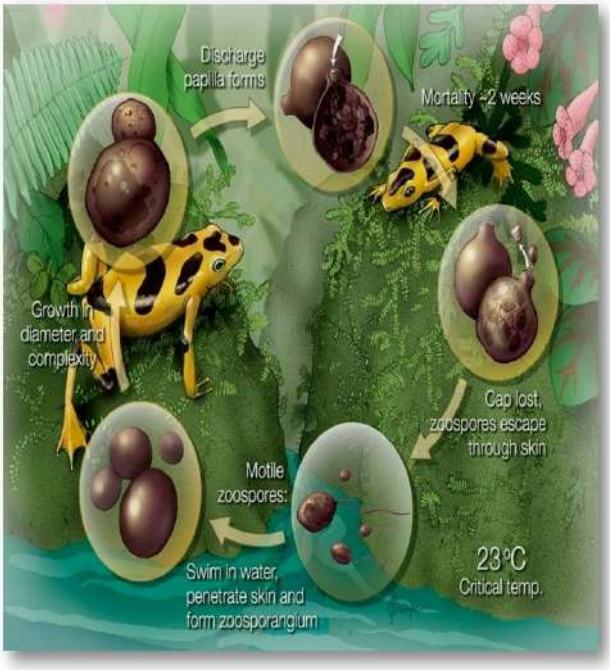


Microscopic view of Chytrid Fungus

Disease Transmission

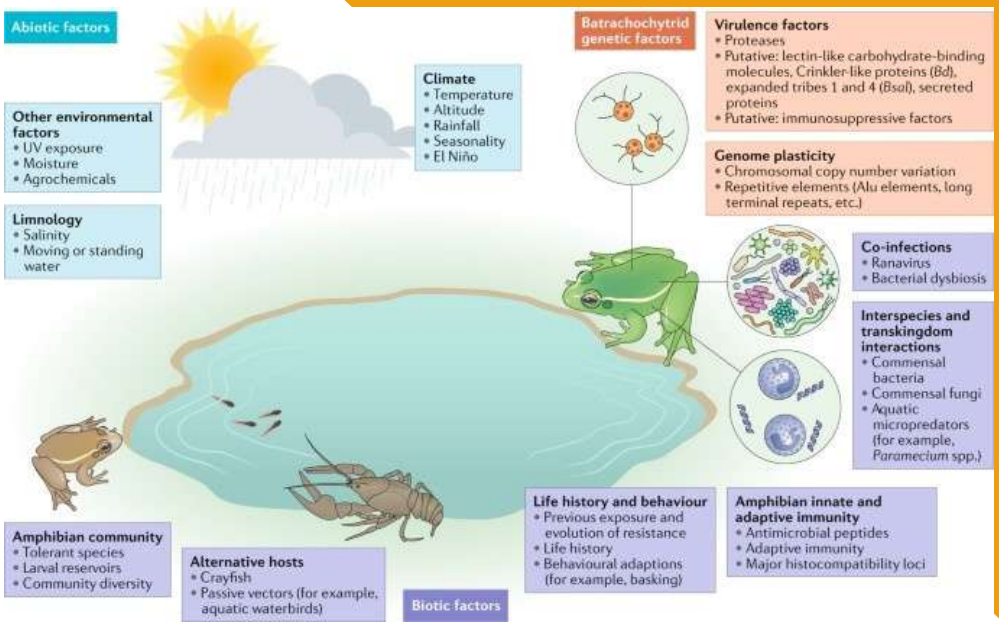
B. dendrobatidis, a waterborne pathogen disperses zoospores into the environment and this zoospores use their flagella for locomotion through water system, until they reach a new host and enter cutaneously. The fungus zoospores can survive within a temperature range of 4-25 degree Celsius and pH of 6-7. The exact mechanism is still unknown behind, how this fungus transmit from one host to the next.





Effect of Climate in Infective Proliferation

A study suggested that changing global temperatures may be responsible for increased proliferation of Chytridiomycosis. Chytrid fungus has a preferred temperature range between 17 -25 degree Celsius. It dies at temperature at and above 30 degree Celsius.

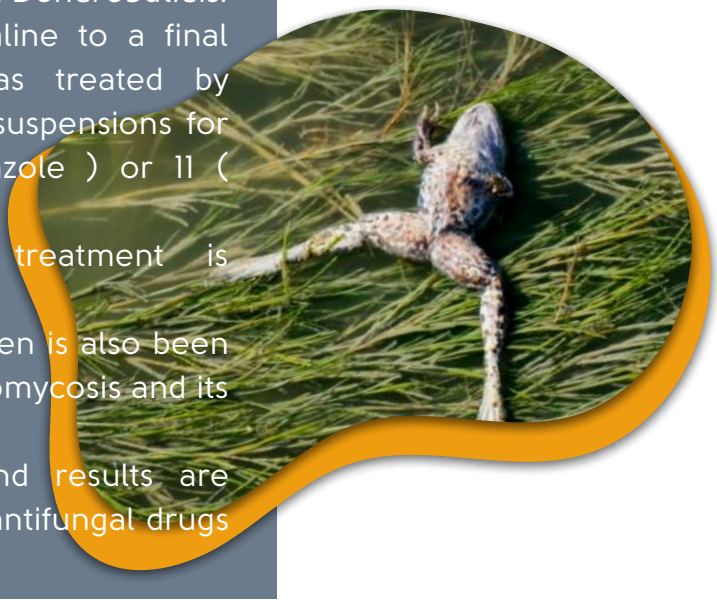


Clinical Symptoms

- Lethargic individual , slow movements.
- Excessive skin shedding in most of the affected species.
- Thickening of skin that promptly leads to death as they cannot intake proper nutrients, cannot breathe properly or release toxins.
- Reddning of skin, convulsions, loss of righting reflux.
- In tadpoles, discoloration of mouth, abnormal feeding behaviour is seen.

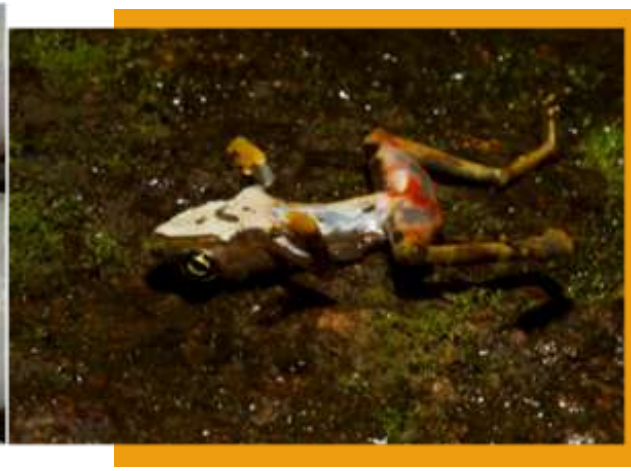
Treatment

- Use of antifungals (Itraconazole, Amphotericin B, Chloramphenicol) and heat-induced therapy has been suggested as a treatment.
- Bioaugmentation is considered as a possible treatment against chytridiomycosis. The amphibian host along with the environment can be augmented with probiotic bacteria that express antifungal metabolites that can fight *B. Dendrobatidis*.
- A 48% solution of TMS was diluted with saline to a final concentration of 0.1%. Infected frogs was treated by immersion in one of the three solutions or suspensions for five minutes per day for either 8(miconazole) or 11 (Itraconazole and TMS) consecutive days.
- Currently, most widely used anti-Bd treatment is Itraconazole.
- Chemicals such as Formalin or Malachite green is also been used to treat individuals infected with chytridiomycosis and its results are seen to be fruitful.
- Chloramphenicol is also used topically and results are curable. However, the potential risk of using antifungal drugs on individuals are high.



Amphibian Immunity

- Amphibians surviving chytrid epidemic tends to carry higher levels of bacterium *J. lividum*.
- This bacterium produces antifungal compounds like Violacein, Indole-3-carboxaldehyde, that inhibits chytrid growth even at low concentrations.



Evolutionary Resistance

- Hints of emerging evolutionary resistence in a vast population of frog species were reported, from ecological studies of an epizootically endangered stream-breeding frog *Mixophyes fleayi* (subtropical Australia).
- Rebound of frog species in Panama after decline are not associated with pathogen attenuation, but rather a host factor, whether an evolved genetic resistance to the fungus infection or an acquired trait is yet to be identified.

Effect on Amphibian Population

The effect of this deadly disease, on amphibian population is surely not what we expect to occur. It has been a threat to living community of amphibians, especially salamanders, toads, frogs, etc. Majority of these population has faced a cursed due to this disease and the outcome is, drastic diminishing population among amphibian community. Though many of the species has acquired to defend against it, but it is not noteworthy.
Thus on a whole, we can say, Chytridiomycosis is A Dead Threat To Amphibians Worldwide.

Weblinks

1. https://amphibiaweb.org/chytrid/chytridiomycosis.html
2. https://www.zsl.org/science/news/scientists-discover-method-to-eliminate-killer-fungus-in-amphibians
3. https://psmag.com/environment/a-deadly-fungal-disease-is-devastating-amphibian-populations
4. https://www.newscientist.com/article/2198070-nearly-100-species-of-frogs-toads-and-salamanders-wiped-out-by-fungus/
5. https://www.zsl/org/science/wildlife-health/amphibian-chytridiomycosis
6. https://theconservation.com/killer-frog-fungus-could-actually-help-amphibians-survive-disease-38073

Prion: The Transmissible Zoonotic Disease & Their Biochemical Mechanism

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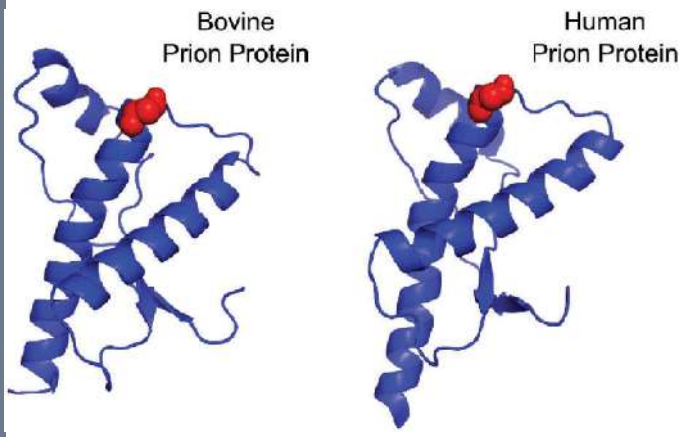
The word Prion, which is also known as Proteinaceous infectious particles responsible for several neurodegenerative diseases enlighten in mammals, including Creutzfeldt-Jakob disease (CJD) in humans. Prion proteins also touch on to the fledgling heretical hypothesis that the contagious particles purpose those diseases consists only of protein, with no nucleic acid genome. Middle of the 1960s, Tikvah Alper and others tell of that nucleic acid was dubious to be a material of the infectious agent that causes scrapie disease. In 1967, J. S. Griffith speculated that the scrapie agent strength be a protein capable of 'self-replication' without nucleic acid. In the University of California, a Neurologist Stanley B. Prusiner, first successfully purify the infectious agent and to show that it consists mostly glycoprotein proteins which attached in a sugar group. He invents the term prion in 1982 for the new pathogen consisting solely of protein, responsible for neurodegenerative diseases called Transmissible Spongiform Encephalopathies (TSE). The misfolded proteins prion has the capacity to change their misfolded shape onto common variants of the same protein. Prions form uncommon total of proteins called amyloids, which accumulate in infected tissue and are associated with tissue damage and cell death. Amyloids are also accountable for several other neurodegenerative diseases such as Alzheimer's disease and Parkinson's disease. Prion totality is unchanging, and this structural stability means that prions are resistant to denaturation by chemical and physical agents: they cannot be destroyed by ordinary disinfection or cooking. This makes scrapping and containment of these particles exacting.



The prions are purely limpid of sialoglycoprotein called prion protein (PrP). They restrain no nucleic acid. In the fit people and animals, the prion proteins are found all over the body. The PrP (infectious) has a various structure. It is resistant to proteases, the enzyme in the body that can normally break down proteins. The quintessential form of protein is called PrPc. The infections form of prion protein is called PrPsc (the c mention to cellular or common PrP while the sc refers to scrapie, a prion disease occurring in sheep). PrPc is an ordinary protein found in the membrane of cells. In human, it consists of 209 amino acids and has 1 disulfide bond. The molecular mass of this protein 35-36 KDa and it mainly composed of alpha-helical structure. The contagious isoform of PrP known as PrPSc, is able to convert normal PrPc proteins into contagious isoform by changing their conformation, or shape, this in turn, alters the way, the prion interconnect. The 3 dimension structure of PrPSc is not known. It composed of a higher proportion of B-sheet structure. Different types of prion protein are also found. They are:-

PrP^{C}

In the membranes of cells, the normal prion protein PrP are^{C}also found. It also includes various blood components of which platelets constitute the largest reservoir in humans." It incorporates 209 amino acids, 1 disulfide bond. The molecular mass of protein is 35–36 kDa. Different topological forms subsist in which one cell surface form anchored via glycolipid and two transmembrane forms. The normal protein is not sediment able; meaning that it cannot be separated by centrifuging techniques. Function of this protein is a complex issue that continues to be investigated. The normal PrP^{C} prion protein binds copper ions which has high affinity.

PrP^{res}

Protease-resistant PrP^{Sc} -namely protein (PrP^{res}) is the name stated to any isoform of PrP^{C} which is fabric altered and converted into a misfolded proteinase K-resistant form in vitro. To model conversion of PrP^{C} to PrP^{Sc} in vitro, Saborio rapidly converted PrP^{C} into a PrP^{res} by a procedure involving cyclic amplification of protein misfolding. The expression "PrP^{res}" has been used to discriminate between PrP^{Sc}, which is isolated from infectious tissue and associated with the transmissible spongiform encephalopathy emblematic.

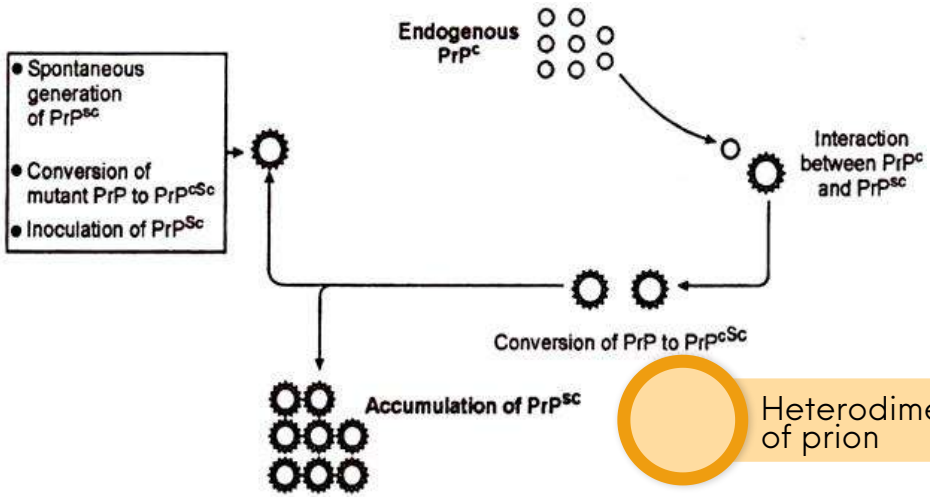
PrP^{Sc}

The contagious isoform of PrP, known as PrP^{Sc}, or plainly the prion, is able to convert normal PrP^{C}proteins into the infectious isoform by changing their fashion, or shape; this, in turn, alters the way the proteins interconnect. PrP^{Sc} always genesis prion disease. The 3D structure of infectious PrP^{Sc} prion protein is not known. It consists of higher proportion of β-sheet structure. The contagious isoforms of prion protein aggregate to form highly structured amyloid fibers, which accumulate to form plaques. In the each fiber end, the performance as a template onto which free protein molecules may attach and growing the fiber. However, sparse cross-species conveyance is also possible.

Augmentation of Prion

The proteinaceous infectious particles prion is a type of protein that can cause disease in animals and humans. It is trigger normally healthy proteins in the brain to fold non-typically. Prions grow by impart a misfold protein state. In the healthy organism, prion enters. Properly folded prion proteins into disease associated prion form. The prion acts as a decoration to guide misfolding of more proteins into prion form. These freshly formed prions can then go on to transmute more proteins themselves, these triggers a chain of reaction that produces a large amount of the prion forms.

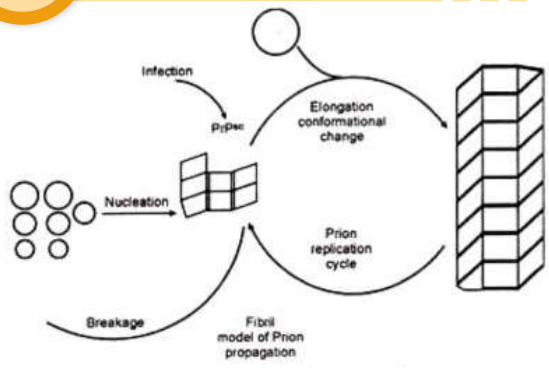
The replication of prion protein explained by the help of heterodimeric model. Single PrPsc molecule holds together to a single PrPc molecule and catalyzes its conversion into PrPsc. The two PrPsc molecules then transpire alone and can go on to convert more PrPc. The various others model takes for vouchsafe that PrPsc exists only as fibrils. This fibril ends bind PrPc. It is converting into PrPsc.



Heterodimeric model of prion

PrP^{Sc} which is infectious exists only as fibrils. These fibril ends bind PrP^{C} and transform it into PrP^{Sc}. If this were all, then the group of prions would swell linearly, forming ever longer fibrils. During prion diseases, both exponential growths of PrP^{Sc} and of the quantity of spreading particles is observed. This can be explicating by taking into account fibril breakage. A mathematical suspension for the exponential growth rate resulting from the composite of fibril growth and fibril breakage has been found. The steadily fill out rate turn on mostly on the square root of the PrP^{C} concentration. The incubation period is persevering by the exponential growth rate, and in vivo data on prion diseases in transgenic mice match this prediction.

Fibril model of prion



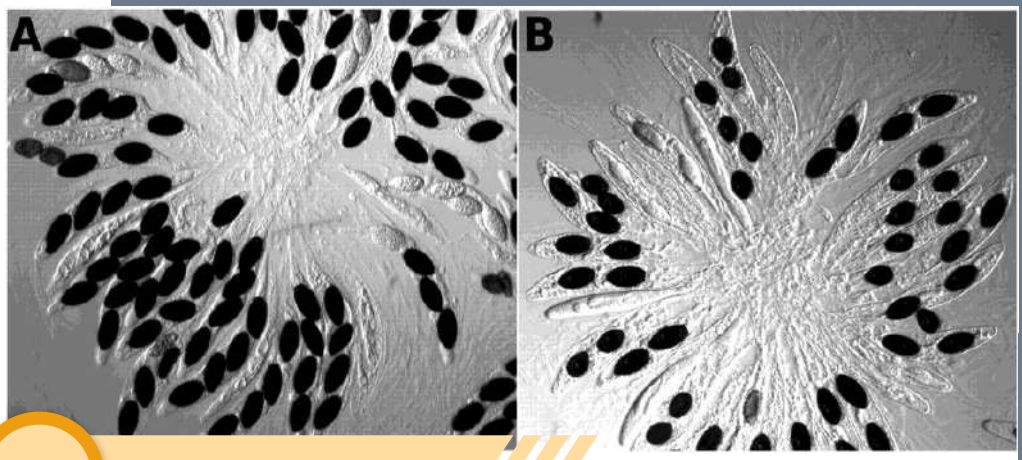
Mechanism of Transmission

Prion diseases are a group of fatal and infectious neurodegenerative diseases intuition humans and diverse animal species. Contagious prion diseases were first noticeable more than 70 years ago. Occasional transmission of scrapie disease occur in sheep.

1. It causes neurodegenerative disease by aggregating extracellularly within the central nervous system to form plaques known as amyloids, which disrupt the normal tissue assembly. This respite is signalizing by cavity in the tissue with resultant spongy architectonics due to the vacuole origination in the neurons.
2. Numerous different mammalian species can be affected by prion diseases, as the prion protein (PrP) is very similar in all mammals. Anticipated to small differences in PrP between different species it is unusual for a prion disease to transmit from one species to another.
3. The human prion disease alternative Creutzfeldt–Jakob disease, however, is conviction to be caused by a prion that typically infects cattle, causing bovine spongiform encephalopathy and is transmitted through infected meat. It has been concede that prion diseases can emerge in three different ways: acquired, familial, or sporadic.
4. One motif, the "Protein X" hypothesis, is that an as-yet obnoxious cellular protein (Protein X) sanction the turning of PrP^{C} to PrP^{Sc} by bringing a molecule of each of the two together into a complex. The top-tier procedure of infection in animals is through ingestion. It is traction that prions may be defrayed in the environment through the abide of dead animals and via urine, saliva, and other body fluids. They may then dawdle in the soil by permanent to clay and other minerals.

Fungal Prion

Fungal prion is a prion protein that adulterate host of fungus. Fungal prions could be proteinopathies kindred to the protein deposition diseases found in humans, but it was further put forward that these prions might be adaptive and confers a benefit to the hostess. Fungal prions are naturally occurring proteins that can switch between multiple, structurally well-defined conformations, no less than one of which is self-propagating and transmissible to other prions. Proteins, which form prion, have been spot in fungi, generally in the yeast *S. cerevisiae*. In these fungal prions proteins, some cases even confer a selectable advantage to the organism. A prion lable [Het-s] has been chronicle in *Podospora anserina*, a coprophilic filamentous fungus whose natural habitat is herbivore dung. Fungal spores put down with the plant material end up in the dung, germinate, and form mycelia that compete for the lean and short-lived resources. Venereal spores are forcibly sent out from the dung. It may attach to surrounding vegetation with their appendages. The [Het-s] prion protein, present in most all fungi out come in a somatic allorecognition process called heterokaryon incompatibility

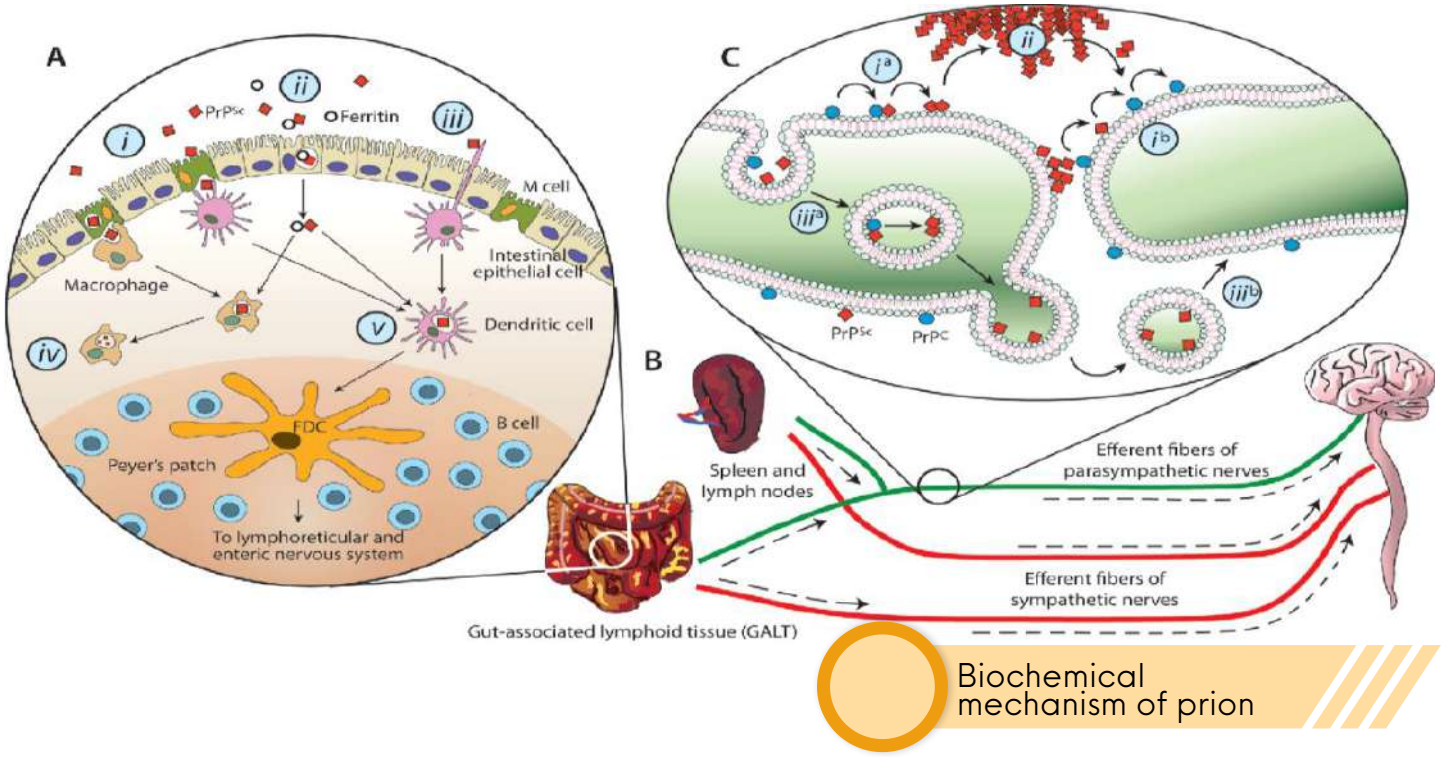


Fungal prion

Biochemical Mechanism of Prion

The reach of mammalian prions, intrinsically connected to conformational transformation of PrP^{C} to protease-resistant PrP^{Sc} , was originally described by a heterodimeric refolding mechanism. The thermodynamically less stable form of prion protein is PrP^{C}rather than PrP^{Sc}. The spontaneous conversion is kinetically determinate.

- Primary conceit of the TSE agent from the intestinal lumen has been raised to ensue through a number of alternative mechanisms, including M cell transcytosis (i), ferritin-dependent trancytosis by intestinal epithelial cells (ii), or via direct taking by dendritic cells (iii). While phagocytic cells such as macrophages arrive to demote PrP^{Sc} (iv), dendritic cells may give the TSE agent to follicular dendritic cells (FDCs) where early collection of PrP^{Sc}occurs (v).
- After manifestation of the TSE agent in lymphoid tissue such as the GALT and spleen, offensive of the nervous system is believed to proceed through peripheral nerves. The transport of the TSE agent is happen along two exclusive pathways. The sympathetic and parasympathetic efferent fibers nerves to the CNS.
- In neuronal processes, the elongation and retrograde transport of PrP^{Sc} prion may take places by step-wise interactions toward the cell surface (i^{a}, i^{b}), via the extracellular deposits (ii) and another one is by vesicle-mediated mechanisms (iii^{a}, iii^{b}).



Biochemical mechanism of prion

Role of Prion in Neurodegenerative Disease

Prion diseases are death dealing neurodegenerative diseases that can be spontaneous, genetic, or infection-related. Voluntary come about prion diseases are in the age-related in nature. Prions are proteinaceous irresistible particles, containing host-encoded prion protein (PrP).

- In prion diseases, biological prion protein (PrPC) becomes misfolded and thereby racks up and aggregates. PrPC plays a modulation role in long-term memory origination.
- The attendance of amyloid fibrils in patients with degenerative diseases has been well documented. These amyloid fibrils are seen as the outcome of pathogenic proteins that self-propagate and form highly stable, non-functional assemblage. Specifically, establishment of TDP-43, an RNA-binding protein, has been found in ALS/MND patients, and mutations in the genes computing for these proteins have been identified in familial cases of ALS/MND. These mutations taken up the misfolding of the proteins into a prion-like resemblance.
- The misfolded form of TDP-43 forms cytoplasmic placing in afflicted neurons, and is found consume in the nucleus. In addition to ALS/MND and FTLD-U, TDP-43 pathology is a trait of many cases of Alzheimer's disease, Parkinson's disease and Huntington's disease. The misfolding of TDP-43 is really directed by its prion-like domain. This domain is inherently biddable to misfolding, while pathological mutations in TDP-43 have been found to hike this propensity to misfold, explaining the presence of these mutations in familial cases of ALS/MND. As in yeast, the prion-like domain of TDP-43 has been unveil to be both necessary and enough for protein misfolding and aggregation.

Different Type of Prion Diseases

Prion diseases construct several situations.

Disease	Host	Mechanism
Kuru	Human	Cannibalism
Sporadic Creutzfeldt-Jakob disease (sCJD)	Human	Spontaneous PrP^{C} to PrP^{Sc} conversion or somatic mutation
Iatrogenic CJD	Human	Infection from prion-containing material, e.g., dura mater, cadaveric-derived growth hormone, blood transfusion
Genetic CJD	Human	Mutations in the PrP gene
Variant CJD (vCJD)	Human	Infection from bovine spongiform encephalopathy (BSE)
Gerstmann-Sträussler-Scheinker (GSS)	Human	Mutations in the PrP gene
Fatal familial insomnia (FFI)	Human	D178N mutation in the PrP gene, with M129 polymorphism
Sporadic fatal insomnia (or sCJD, MM2 thalamic type)	Human	Spontaneous PrP^{C} to PrP^{Sc} conversion or somatic mutation
Variable proteinase-sensitive proteinopathy (VPSPr)	Human	Spontaneous PrP^{C} to PrP^{Sc} conversion or somatic mutation
Scrapie	Sheep	Infection in susceptible sheep
BSE	Cattle	Infection from contaminated food
Transmissible mink encephalopathy (TME)	Mink	Infection from sheep or cattle in food
Chronic wasting disease (CWD)	Mule, deer, elk	Fecal/oral/aerosol routes of infection from other affected cervids; arose spontaneously or possibly from a scrapie source
Feline spongiform encephalopathy	Cats	Infection from BSE-contaminated food
Exotic ungulate encephalopathy	Nyala, oryx, kudu	Infection from BSE-contaminated food

Symptoms of Prion Diseases

Prion diseases have too much long incubation periods, frequently on the order of many years. When symptoms exhibit, they progressively derange, sometimes rapidly. Common symptoms of prion disease include:-

- Struggling with thoughtful, memory, and judgment occurs.
- Personality converts such as apathy, agitation, and depression.
- Confusion or perplexity occurs.
- Involuntary muscle spasms (myoclonus) occur.
- Loss of coordination (ataxia) occur.
- Trouble sleeping (insomnia) occur.
- Difficult or slurred speech occurs.
- Impaired vision or blindness occurs.

Diagnosed of Prion Diseases

Being prion diseases can present similar symptoms to other neurodegenerative disorders; they can be ticklish to diagnose. The only way to diagnose prion disease is through a brain biopsy performed after death. The tests they may use include:-

- **Magnetic resonance imaging (MRI)**
  An MRI can fashion a exhaustive image of your brain acting as providers envision changes in brain structure that are linked with prion disease.
- **Cerebrospinal fluid (CSF) testing**
  CSF can be collected and tested for markers kindred with neurodegeneration. In 2015, a test was come about Trusted Source to specifically ferret out markers of human prion disease.
- **Electroencephalography (EEG)**
  This test note electrical activity in your brain.

How Is Prion Diseases Treated?

- **Medications**
  Some pharmaceutical can be prescribed to help treat symptoms.
  Examples include:
  - decrease psychological symptoms with antidepressants or sedatives.
  - providing pain repose using opiate cure.
  - discovering muscle spasms with drugs like sodium valproate and clonazepam.
- **Assistance**
  During advanced stages, many people need help for taking care of themselves as well as performing daily activities.
- **Providing hydration and nutrients**
  IV fluids or a feeding tube may be required in the advanced stages.

Scientists carry on with to work to find an effective treatment for prion diseases. Some of the potential therapies that are living investigated include use of anti-prion antibodies and "anti-prions Trusted Source" that obstruct replication of abnormal PrP.

Prevention of Prion Diseases

Several estimates have been taken to fend off the transmission of acquired prion diseases. Following are the few preventive steps to be taken:

- Setting tight regulations on bring in cattle from countries where BSE occurs.
- Prohibiting the parts of the cow such as the brain and spinal cord from entity used in food for humans or animals.
- Precluding those with a history of or risk for exposure to prion disease from give blood or other tissues.
- Utilize robust sterilization measures on medical instrument that has come into contact with the nervous tissue of someone with suspected prion disease.

Weblinks

1. https://www.scientificamerican.com/
2. https://www.ncbi.nim.nih.gov/
3. https://courses.lumenlearning.com/
4. https://microbiologysociety.org/
5. https://www.biologydiscussion.com/

Cephalopods – The Marine Invertebrates With Extraordinary Cognitive Sophistication

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Whenever the discussion about intelligence and behavioral complexity is raised, humans are denoted as the epitome of most sophisticated cognitive capabilities. However, extensive research from the aspect of experimental psychology has revealed that, irrespective of taxonomic status, several non-human animals- for e.g. mammals like rodents and primates, other vertebrate species like birds, even invertebrate insects like honeybees do also express some of the very skilled and intricate behavioral patterns to survive in the wild which expose certain new insights regarding the evolution of cognition in humans. Cephalopods, an exclusively marine class of mollusks, are unique among other marine invertebrates to exhibit an extraordinary adaptation of cognitive abilities with impressive memory, learning skills that are enhanced and evolved along with their social behavior and with the development of a highly centralized nervous system.

Brief Biological Background

Cephalopods incorporate two distantly related subclasses namely Nautiloidea and Coleoidea or Dibranchiata. Nautiloids emerged during Paleozoic era and exist with several shell shapes and forms. The Coleoids include decapod mollusks (order Decapodiformes- cuttlefishes and squids) and octopods (order Octopodiformes- Octopus and Argonauts) and they lack a rigid outer shell.

Nautiloids (Nautiloidea)

Octopoda (Coleoidea)

Social Behaviors of Cephalopods Manifest Cognitive Intelligence

Cephalopods engage themselves in different forms of social communications. However, the degree and forms of these behaviors vary among different species.

Body patterning is a process of changing color and pattern of body surface that is displayed by these invertebrates during courtship (intra-specific interaction), during camouflage from predators, for signaling the enemies or while hypnotizing preys (inter-specific interaction). This coloration is mediated through organs under the skin called chromatophores that contain pigment sacs(pigments like red, yellow, brown or black) and other structurally reflecting cells that are iridophores and leucophores as well as certain sections of skin called papillae (that can change the texture or outline of body under pressure). With the periphery of the pigment sacs in chromatophores, dozens of radial muscles are attached which are innervated directly by the brain. Contraction and relaxation of these muscles result in selective expansion and retraction of distinct group of chromatophore pigment sacs. Thus the animal is able to show body patterns like stripes, spots or bands within milliseconds based on the different visual sensory inputs collected and analyzed from the surrounding environment. This process accounts for a high encephalization quotient and highly organized brain that indicate the presence of cognitive intelligence in these animals.

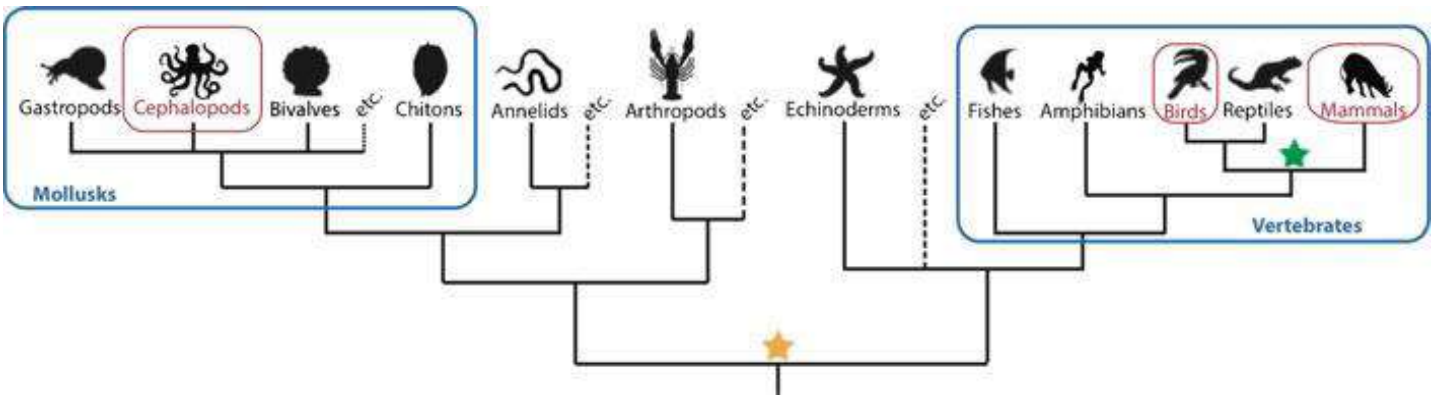
Bodypatterning in cuttlefish

School formation is another complex social interaction, observed in oceanic, coastal as well as in certain coral reef squids. Both male and female squids, juveniles as well as adults do participate in large or small structured schools ranging from 10-100 individuals. This schooling behavior demands complex cognitive skill and can be an important tool for young squids to acquire different information by following or observing the adults within the schools.

School formation in squids

Factors Contributing in the Cognitive Evolution of Cephalopods

Cephalopods are distributed across the oceans of the world and they have a highly active and predatory life style. Their sophisticated, complex, centralized nervous system with relatively larger brain size can control flexible behavioral patterns that can even be compared with some of the vertebrates' behaviors. (Certain fishes, birds and mammals).



Phylogenetic tree showing evolutionary relationship between cephalopods, birds and mammals. Stars refer to the MRCA or most recent common ancestors. The yellow star is between cephalopods and vertebrates (~600 million years ago), the green star between birds and mammals. (~300 million years ago).

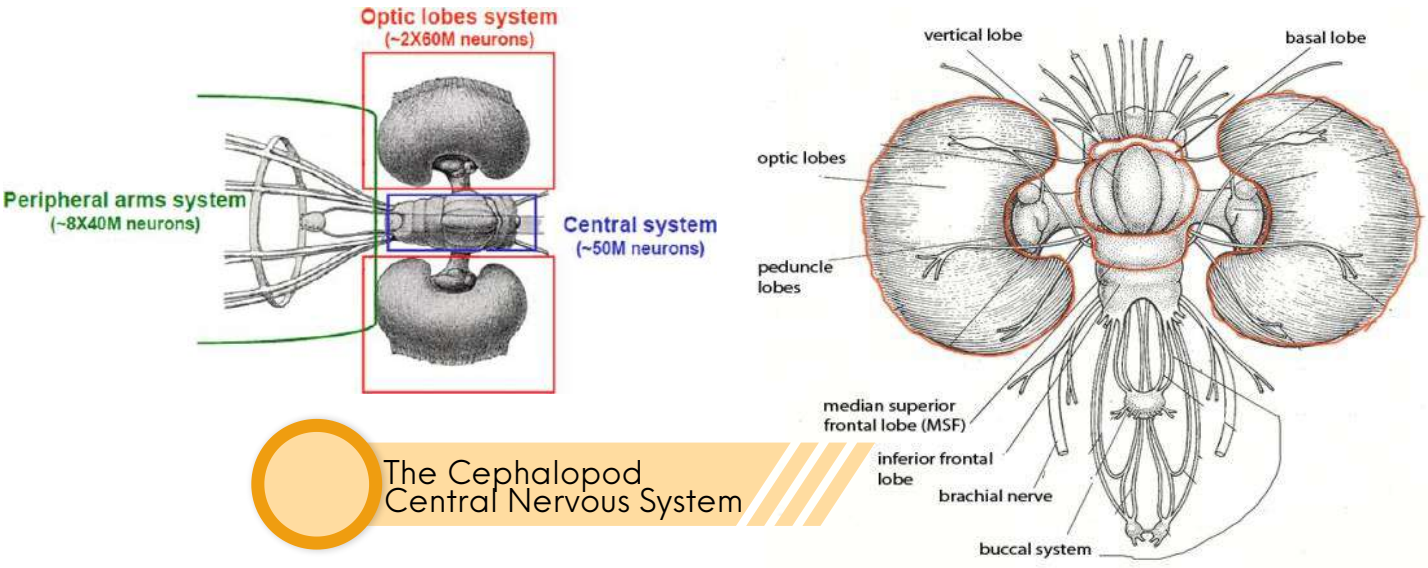
- **Theories Regarding Evolution**

There are two major selection pressures that are thought to be the driving factors of the cognitive evolution in cephalopods that can rival with that of the birds and mammals.

One of the most long standing theories is sometimes denoted as 'Packard scenario. It describes competition with the bony fishes as a selective force for the cognitive development. Along with the acquisition of swim bladder and efficient eyes, bony fishes became formidable predators in the ocean. To compete with these vertebrates, nautiloids migrated to a deeper habitat whereas the coleoids developed cognitive complexity and a much bigger size. **Another important contributor** is the loss of or the internalization of the nautiloid shell. Loss of these structures in coleoids pressurized these invertebrates to develop better cognitive skills to remain aware and protected in an unpredictable wild environment. Apart from these factors, development of a robust vision enabled the sophistication of pathways required for the processing of the visual information eventually leading to cognitive evolution.

Role of Complex Central Nervous System in Enhancement of Learning & Memory in Cephalopods

Cephalopod brain shows 3 types of designs: The Nautilus brain, the decapod (decapodiformes) brain and the octopod (octopodiformes) brain and in that order, they are characterized by the increase in centralization. The cephalopod nervous system is divided into a central and a peripheral part, the central part includes the brain proper and the optic lobe. The large peripheral part includes the nervous system of the body and of the arms.



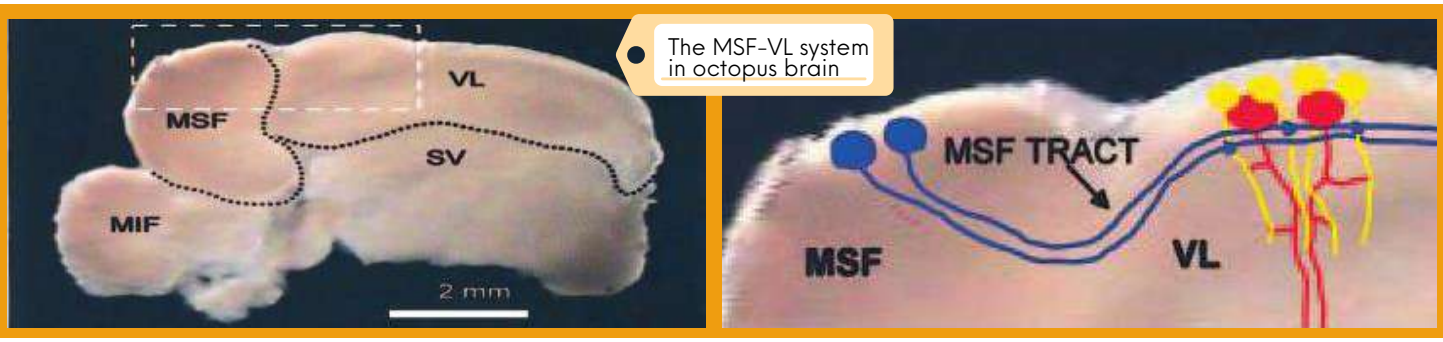
The Cephalopod Central Nervous System

- **Different sections of the cephalopod CNS are allocated for different cognitive behaviors:**

The vertical, subvertical, subpedunculate and precommissural lobes, the superior frontal, the inferior frontal, sub-frontal and also certain parts of posterior buccal lobes together with optic lobes- these all different parts of brain are involved in analysis of visual inputs, various types of learning and in short-term and long-term memory. In cuttlefishes, (for eg- *Sepia pharaonis*) the optic lobes specifically are the centers of visual discriminative learning and help in the expression of the chromatic components of the chromatophores required for camouflage colorations. The cortex of the optic lobes processes visual information where as the medulla region acts as the motor command centre for dynamic body patterning.

Role of Octopus Brain in Cognitive Behavior

Octopuses are octopod cephalopods. Unlike squids and cuttlefishes, octopuses are asocial, solitary hunters. Apart from behaviors like habituation and sensitization, they also show several forms of learning skills and memory. They do exhibit associative learning including visual and tactile discriminative capabilities, spatial learning that is retention of spatial information and exploratory behavior. Certain lobes in the supraesophageal mass in octopus brain are involved in the learning, memory behavior. In the MSF-VL system, the MSF or median supra frontal lobe integrate the sensory information and the MSF neurons reach the VL or vertical lobe via MSF tract. Each of the 1.8 million MSF axons form synapse with as many as 25 million amacrine neurons in the VL .The physiological connectivity and architecture of this MSF-VL system show similarity with mammalian hippocampus. The VL or vertical lobe in particular is required for observational short-term learning and long term memory performances. Similar organization between MIF (median inferior frontal lobe) and sub frontal lobe plays role in tactile learning.



The MSF-VL system in octopus brain

Conclusion

Despite being phylogenetically disparate species, cephalopod brain has many analogies with vertebrate brain. A comparative analysis of both similarities and dissimilarities between them can help in better understanding of several basic morphological, physiological as well as pathological principles of brain. It may also allow us to understand more well about how these distantly related animal groups converged and adapted certain very similar, advanced and flexible cognitive architecture in response to selection pressure of highly variable and competitive environment. A new report based on recent scientific studies published by the London School of Economics and Political Science regarded cephalopod mollusks as sentient invertebrate animals, which means they are capable of feeling emotions, pain, distress or harm in a much broader range. Presence of sentience is an extraordinary complex cognitive capability for any invertebrate. It also makes them a significant animal group as future animal welfare laws may include these animals to protect them from painful unethical practices.

References

1. Hochner, B.,Shomrat, T., Fiorito, G. (2006,June), The Octopus: A Model for a Comparative Analysis of the Evolution of Learning and Memory Mechanisms, The Biological Bulletin,210:308-317.
2. Young, J. Z. (1974), The Central Nervous System of Loligo I The Optic Lobe, Phil.Trans.R.Soc.Land, B267:263-302.
3. Mathger, L. M., Deuton, E.J.,Marshall, N.J., Hanlon, R.T. (2009), Mechanisms and Behavioral Functions of Structural Coloration in Cephalopods, J.R. Soc.Interface,6: S149-S163.
4. Joseph, J.V. (2013), Cephalopod Cognition in an Evolutionary Context: Implications For Ethology, Biosemiotics.

Weblinks

1. https://www.researchgate.net/figure/Octopus-nervous-system_fig2_249320328.
2. https://brofacts.weebly.com/biology-page.html
3. https://www.colorado.edu/chbe/2019/02/28/underwater-creature-inspire-shape-shifting-color-changingmaterials

Regeneration of Adipocytes From Myofibroblasts Leads to Scarless Wound Recovery

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The mammalian wound can choose either successful recovery with scar formation or chronic inflammation due to nonsuccessful recovery. Healed scarred tissue neither function nor look the same as the nearby normal tissue. Extensive scars from burning can lead to loss of function in the tissue. A wound's niche is composed of various cells, cytokines, growth factors, excessive collagen, high lactate, and low oxygen.

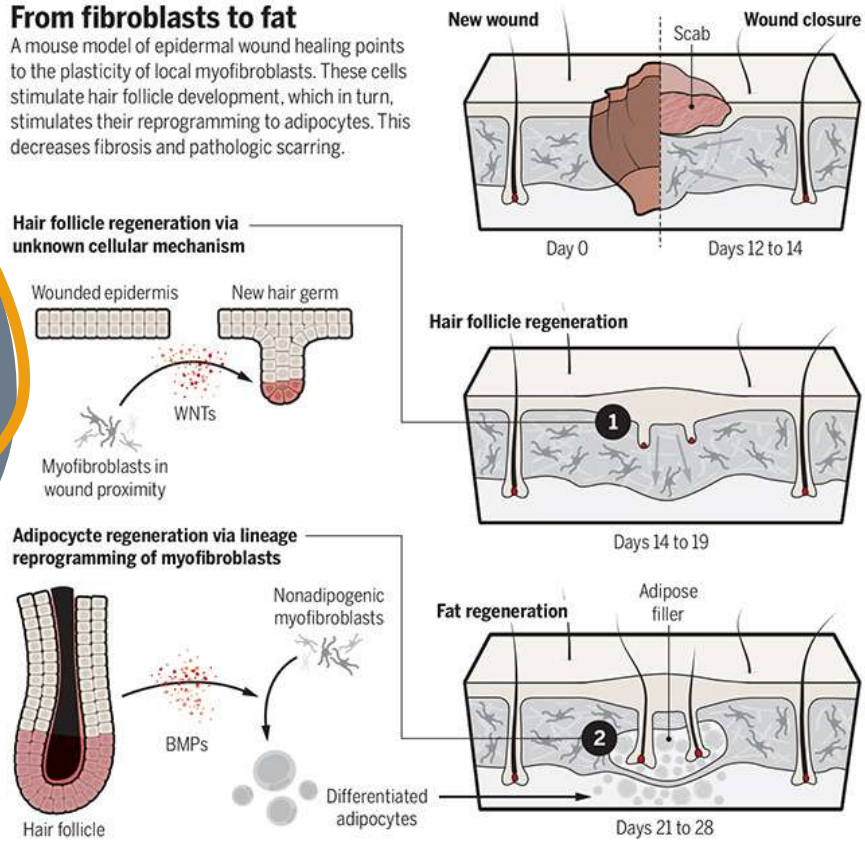
Wound healing occurs via three successive phases that are inflammatory, proliferative, and remodeling. At first, the inflammatory phase prevents blood loss and infection and clears out debris. Then, the proliferative phase helps in the proliferation and migration of keratinocytes to the wound to reseal the epithelium. Finally, during the remodeling phase, fibroblasts, adipocytes, and extracellular matrix form the scar by filling the wound. A scar is differentiated from normal tissue by colour, texture, lack of hair follicle; cutaneous fat; sweat; and sebaceous glands.

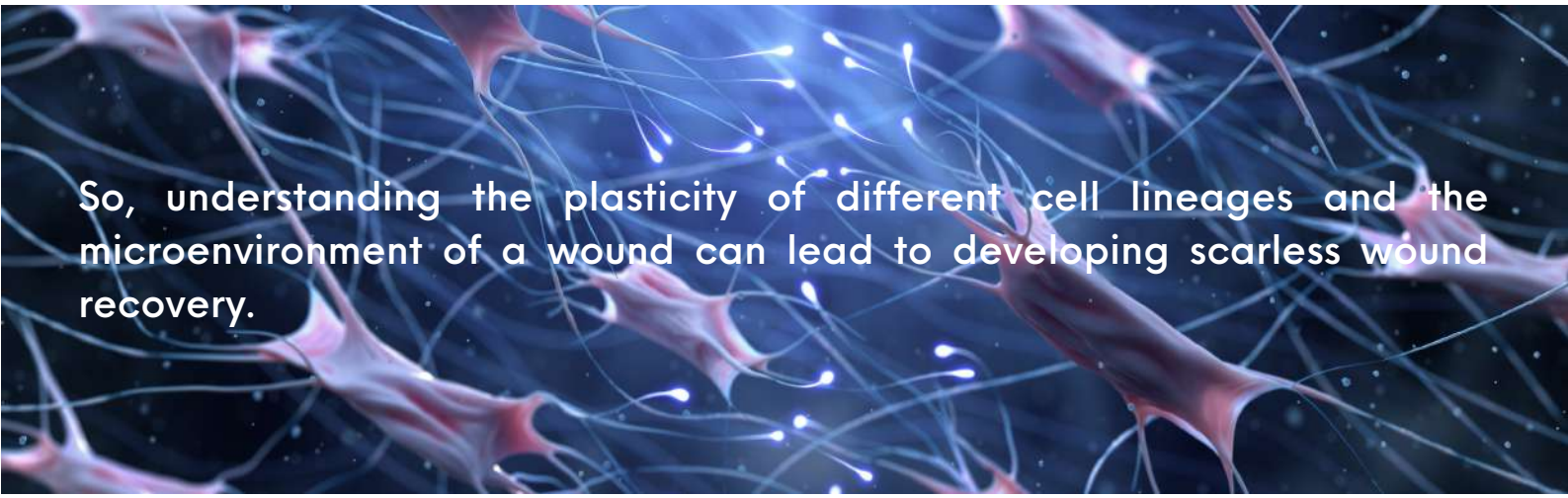
Researches in mouse model showed that fibroblast growth factor (FGF) and Wingless (WNT) signaling pathways can regenerate hair follicles in the wound. Myofibroblasts have the principal role in scar formation. Hair follicles can convert the fate of the myofibroblasts to adipocytes via a signaling pathway under the control of Bone Morphogenetic Protein (BMP). Consequently, FGF, WNT, and BMP can stimulate hair follicles regeneration followed by converting myofibroblast to adipocytes which in turn give rise to scarless wound healing. Overexpression of Wnt ligand causes an increase in regenerated hair follicles. Conversely, inhibition of Wnt signaling overrules folliculogenesis. Adipocytes never form in the hairless part but develop near new hair follicles. Newly developed adipocytes are indifferentiable from normal cutaneous fat cells regarding density, size, and depth from the skin surface.

Cultured dermal cells that are isolated from wounds, with regenerated hair follicles differentiated into BODIPY-positive (green) adipocytes. But cultured dermal cells from wounds lacking hair follicles developed no adipocytes.

Regenerated hair follicles start to develop around 14 years of post-wounding and reepithelization. Adipocytes start to develop around 23 days close to the hair follicles. They increase by number and size following the next few days. These adipocytes are metabolically active and physiologically mature as they express fat tissue specific hormones resistin and adiponectin.

Researches also showed that isolated myofibroblasts from keloids patients if treated with BMPs, can be converted into adipocytes. It is a great discovery to treat keloids because they recur even after surgical removal and they never stop growing.





References

1. Chan, C. K., & Longaker, M. T. (2017). Fibroblasts become fat to reduce scarring. Science, 355(6326), 693-694.
2. Ito, M., Yang, Z., Andl, T., Cui, C., Kim, N., Millar, S. E., & Cotsarelis, G. (2007). Wnt-dependent de novo hair follicle regeneration in adult mouse skin after wounding. Nature, 447(7142), 316-320.
3. Plikus, M. V., Guerrero-Juarez, C. F., Ito, M., Li, Y. R., Dedhia, P. H., Zheng, Y., Shao, M., Gay, D. L., Ramos, R., Hsi, T., Oh, J. W., Wang, X., Ramirez, A., Konopelski, S. E., Elzein, A., Wang, A., Supapannachart, R. J., Lee, H., Lim, C. H., Nace, A., Guo, A., Treffeisen, E., Andl, T., Ramirez, R. N., Murad, R., Offermanns, S., Metzger, D., Chambon, P., Widgerow, A. D., Tuan, T., Mortazavi, A., Gupta, R. K., Hamilton, B. A., Millar, S. E., Seale, P., Pear, W. S., Lazar, M. A., & Cotsarelis, G. (2017). Regeneration of fat cells from myofibroblasts during wound healing. Science, 355(6326), 748-752.

The Re-Emerging Threat of Yellow Fever & WHO's Position

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Yellow fever is a serious, potentially deadly flu-like disease spread through *Aedes aegypti* mosquitoes, which also transmit dengue and Zika viruses. Yellow fever is a haemorrhagic condition which prompts a high fever, red pigmented spot in the skin called petechiae and cell deaths occur in the liver and kidneys. If more number of liver cells die, liver will damage. It promotes jaundice and hence its called yellow fever. As by the World Health Organization (WHO), The estimated number of severe cases in both continents is 84,000-1,70,000, with approximately 29,000-60,000 Deaths per year in which 90% occurring in Africa.

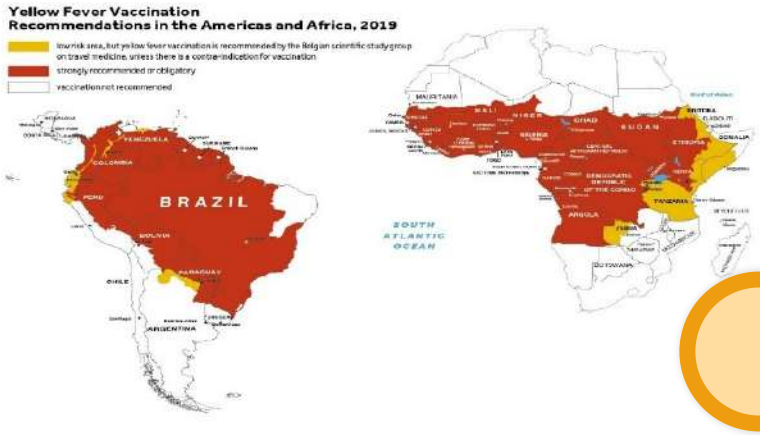
Current Scenario

After 2009 in December 2015, A yellow fever outbreak started in Luanda and Angola. This outbreak was the largest reported in Angola during the most recent 30 years and three countries in Americas have confirmed yellow fever outbreak Bolivia, Brazil and Peru.

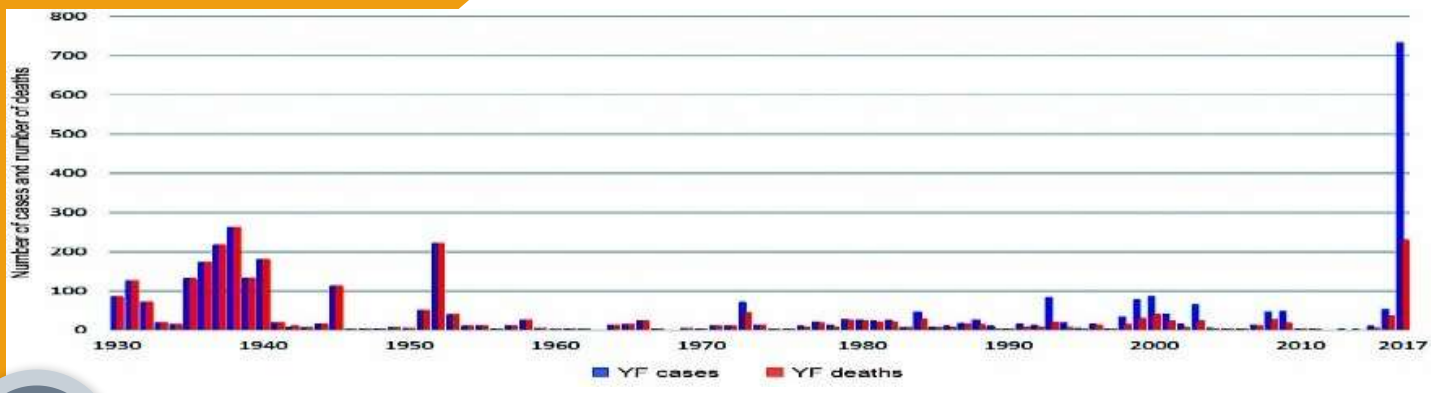
In Brazil, a re-emergence of the yellow fever virus infection contamination has been reported starting around 2014. The expansion of the historical area of yellow fever transmission to areas previously considered not at danger led to two waves of transmission, one during the 2016-2017 seasonal period, with 778 confirmed human cases including 262 deaths, and one during the 2017-2018 seasonal period, with 1,376 confirmed human cases including 483 deaths. Thus, beginning around 2020, Brazil has changed their prescribed regions for yellow fever vaccination to incorporate the entire country. In during the 2020-2021 seasonal period, between July 2020 and June 2021, a total of 527 suspected human cases of yellow fever were reported, of which 9 (1.7%) were confirmed, 13 (2.5%) remain under investigation, and 500 (94.9%) were discarded.

In Peru, between epidemiological week (EW) 1 and EW 49 of 2021, a sum of 18 cases of Yellow fever were reported, of which 10 were confirmed and 8 probable cases remain under investigation. The number of probable cases reported in 2021 as of EW 49 is greater than the number of probable cases reported every year during the beyond four years (2017-2020).

In Venezuela, between EW 39 and EW 49 of 2021, a sum of 11 confirmed human cases of Yellow fever were reported in the state of Monagas, all laboratory confirmed. Of the aggregate, 5 were asymptomatic and 6 developed signs and symptoms.



Yellow Fever vaccination recommendation in the Americas & Africa in the year 2019



Yellow fever: Human cases & case fatalities in Brazil 1930-2017

Probability of Yellow Fever Virus Transmission in the Asia-Pacific Region

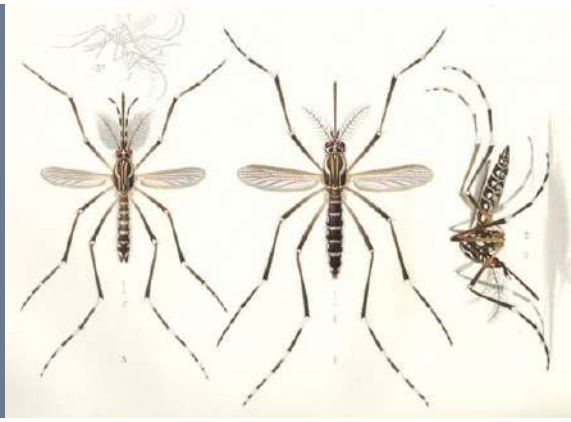
Africa receives a enormous number of Chinese workers Who are usually unvaccinated against YFV, increasing the risk of importing YF in Asia. In 2016, the return from Angola of 11 yellow fever infected workers to China posed the threat of a YF epidemic in Asia never before seen. Increasing volumes of trade and travels among China and Africa increase the risk of disease Introductions. Yellow fever virus (YFV), endemic to Africa and South America, has up to this point remained absent in Asia. The reasons explaining this absence (e.g., transmission barrier resulting from Low compatibility between mosquito and virus genotypes, limited duration and low viraemia in humans, absence of a sylvatic cycle, competition with well-established flaviviruses as Dengue and Japanese encephalitis viruses) are still poorly Explored, making the possibility of an epidemic unpredictable. The combination of repeated introductions of viraemic explorers and immunologically naive local population in an environment suitable to transmission accentuates the risk of YF emergence in Asia. Although the vector competence for YFV of mosquitoes in Africa, South America, and Caribbean regions, has been investigated, only limited information for Asian-Pacific mosquitoes could be found to measure the potential risk of YFV transmission in this region. Investigating the vector competence for YFV of mosquitoes in the Asia-Pacific region is essential to assess the possible danger of YFV transmission in a region where YF outbreaks have never been reported.

Etiological Agent

The yellow fever virus is an RNA virus of the prototypic member of the genus Flavivirus and the family Flaviviridae. The YF virus particle is small, icosahedral, and enveloped.

Vector

Aedes aegypti mosquitoes are the principal vectors for the human transmission of yellow fever, which was also the case for dengue fever. Several other Aedes and Haemagogus mosquito species are also relevant for the transmission of yellow fever, but *Aedes aegypti* is foremost important because of its adapted ecology to the human domestic environment.

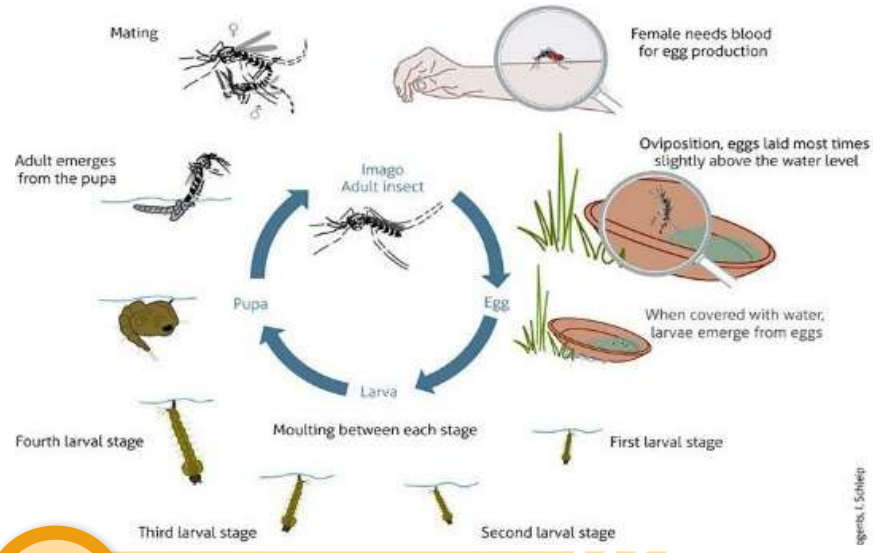


Characteristics

The dark-colored yellow fever mosquito is about 3-4 mm by its length. The mosquito can be recognised by a marking in the form of a lyre on the dorsal side of the thorax, and striking white and black patterns on the legs.

Life Cycle

This mosquito does not lay its eggs either in the water or on the surface of the water, as most other mosquito species do. Instead, *Aedes aegypti* lays its eggs above the water line on the interior wall of the vessel containing the water so that when the water vessel is refilled, from the water line at which the mosquito laid its eggs to the lip of the vessel, the eggs will have enough time to complete their developmental cycle to adulthood before evaporation depletes the water source. A truly incredible evolutionary adaptation by *Aedes aegypt*, they completed their life cycle around 8-10 days.



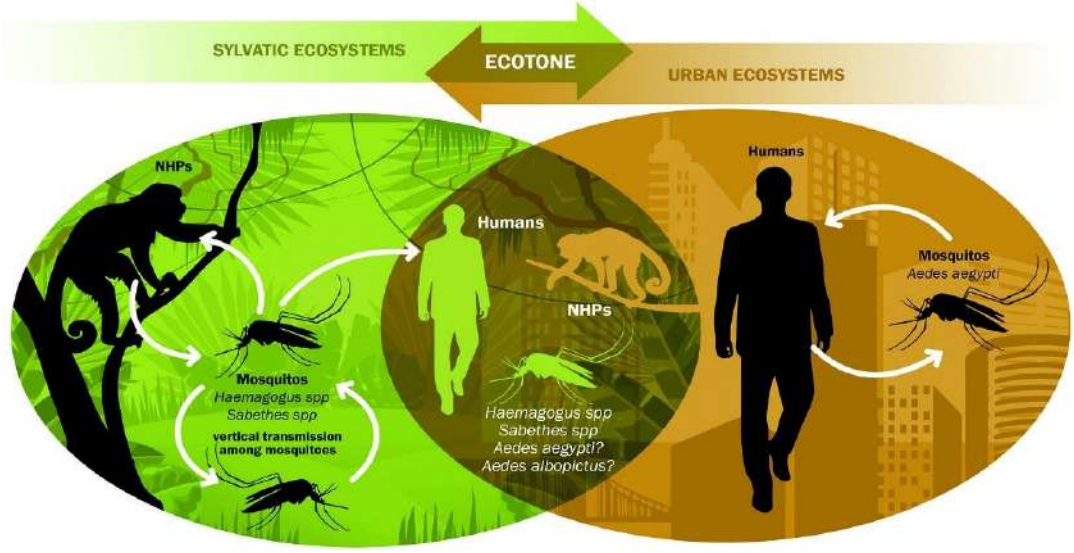
Life cycle of *Aedes* mosquito

How It Spread, Transmission Cycle

In **urban cycle** the principal way of transmission to humans through an infected mosquito at the time of blood sucking. *Aedes aegypti*, and these mosquitoes acquire the virus by feeding on other YF infected humans.

The **sylvatic**, or **jungle cycle** involves transmission of the virus through non-human primates (like monkey) in the wild. Humans can then become infected by these mosquitoes when they visiting or working in the jungle. In this case vectors involved *Aedes africanus* in Africa or mosquitoes of the genus *Haemagogus* and *Sabethes* in South America.

An **intermediate cycle** exists where semi-domestic mosquitoes of the Aedes species act as vectors, and both human and non-human primates serve as reservoirs for the disease. As the Aedes spp. Involved normally obtain blood meals from both humans and other primates (monkeys). It is essentially an ecotonal cycle, wherein the geography of transmission is determined by landscapes of transition from one habitat to another habitat.



To Protect Yourself From the Yellow Fever Mosquito

To fight these mosquitoes, a combination of several mosquito measures is the most encouraging methodology:

For an eco-accommodating persistent control without insecticides, mosquito traps such as BG-Mosquitaire, BG-GAT, or BG-Mosquitaire CO2 can help to reduce the local mosquito population on a long-term. With the BG-Home for indoors you can complement the framework. But further measures are also important like the elimination of breeding sites.

With These Some Mosquito Control Measures You Can Support Reducing Mosquito Bites

- Covering rain barrels – standing water for example in rain barrels, flower pots, water cans are ideal breeding sites for mosquitoes; therefore, they should be covered or regularly emptied. Also you can treat them with the biological mosquito control agent BTI, which kills mosquito larvae.
- Mosquito suction traps – catch mosquitoes, and reduce the population.
- Mosquito screens on doors and windows, and mosquito nets (bed nets) – prevent the invasion of mosquitoes in your house, and safeguard your sleeping area.
- Long, light, and baggy dress – mosquitoes avoid landing on bright areas. Certain manufacturers offer specially treated apparel that can prevent bites.
- If applicable electric evaporators in rooms – just for short-term use and in regions with mosquitoes that transmit diseases, because their active components are insecticides (pyrethroids) that can cause irritations to sensitive persons or infants.

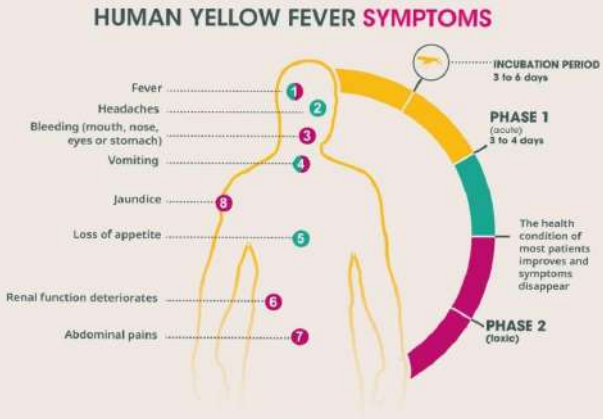
Pathogenesis

An contaminated female mosquito inoculates approximately 1000 to 100,000 virus particles intradermally during blood sucking. Virus replication begins at the site of inoculation, probably in dendritic cells in the epidermis, and spreads through lymphatic channels to provincial lymph nodes. Lymphoid cells, particularly monocyte-macrophages and enormous histiocytes, appear to be the preferred cell types for primary replication. The virus arrives at different organs via the lymph and then the bloodstream, seeding other tissues. Major amounts of virus are produced in the liver, lymph nodes, and spleen and are released into the blood. During the viraemic phase (between days three to six), contamination may be transmitted to blood-feeding mosquitoes.

Symptoms

Mild cases cause fever, headache, nausea and vomiting. Serious condition may cause fatal heart, liver and kidney conditions.

People may also experience: Pain areas: in the abdomen or muscles. Whole body: chills, fatigue, fever, or loss of appetite. Gastrointestinal: nausea or vomiting. Also common: bleeding, delirium, headache, and yellow skin and eyes.



Treatment

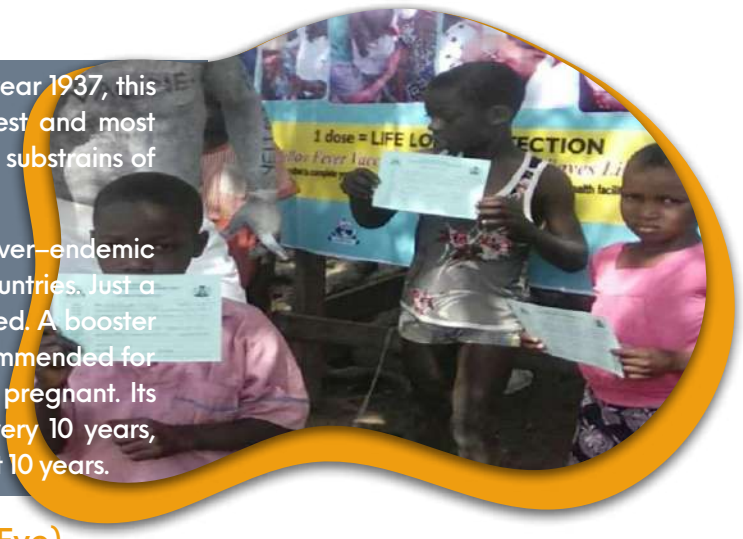
There is no particular treatment for YF infection and consequently supportive care is critical. Primary efforts focus on managing symptoms and limiting complications.

Prevention Methods

- **Vaccine**

The 17D vaccine was developed by Theiler and Smith in the year 1937, this 17D vaccine strain is generally regarded as one of the safest and most effective live-attenuated viral vaccines ever developed. Two substrains of 17D (17D-204 and 17DD) are used as vaccines now a days.

This vaccine is indicated for use in travelers going to yellow fever–endemic areas and may be required for entry into yellow fever risk countries. Just a single dose of subcutaneously administered vaccine is required. A booster dose after a 10-year interval is rarely recommended but recommended for those who received a first dose while HIV infected or while pregnant. Its depends upon on the destination, they may be required every 10 years, although the vast majority of persons retain immunity well past 10 years.



- **Eliminate Yellow Fever Epidemics Strategy (Eye)**

The Eliminate Yellow Fever Epidemics (EYE) Strategy Steered by WHO, UNICEF, and Gavi, the Vaccine Alliance, EYE supports 40 nations and involves more than 50 partners. It is a plan to control Yellow fever, with strategies to be carried out from 2017 to 2026 to expand yellow fever vaccine to prevent global spread and contain outbreak rapidly.

Future Perspectives

There are numerous unanswered questions remaining about YF disease and its agent, YFV, while the threat of urban outbreaks and endemic zone expansion continues to increase. A significant gap in our knowledge of YF and YFV is in management and treatment of patients with this disease or with serious vaccine-associated adverse events. Treatment of YF by supportive care is virtually ineffective, and even admission to the ICU does not seem to improve the prognosis or change the death rate. There is a urgent requirement for the development of specific antiviral drugs and improved rapid diagnostic tests for this and other flaviviral diseases. Finally, the 17D vaccine, on which disease control completely rests, has been associated recently with fatal adverse events. Consequently, upgrade the vaccine's safety will be required. To resolve these issues, a greatly improved understanding of complex interactions between the virus and host cell factors that control replication, as well as innate and adaptive immune responses, will be required.

References

1. Brazilian Ministry of Health, YF Epidemiological reports 1930-2018.
2. Cruz A.C.R, Noll C.A. et.al. 2010. Yellow Fever Virus in *Haemagogus leucocelaenus* and *Aedes serratus* Mosquitoes, Southern Brazil, 2008; Vol. 16, No. 12; 10.3201/eid1612.100608.
3. Drumond B, Prado A, ThoisyB, et.al. Neighbor danger: Yellow fever virus epizootics in urban and urban-rural transition areas of Minas Gerais state, during 2017-2018 Yellow fever outbreaks in Brazil. 2020; 14(10): e0008658. https://doi.org/10.1371/journal. Pntd.0008658
4. Gardner L, Ryman D, et.al. Yellow Fever: A Reemerging Threat. 2010; 30(1): 237–260. Doi:10.1016/j.cll.2010.01.001
5. Litvoc N, Terra C, et.al. Yellow fever. 2018; 64(2):106-113.
6. Mucci F, Tubaki M, Cunha S, et.al. Possible non sylvatic transmission of yellow fever between non human primates in São Paulo city, Brazil, 2017–2018. (2020) ;10:15751: | https://doi.org/10.1038/s41598-020-72794-x

Zebrafish as a Model for Tuberculosis: No Lungs Required?

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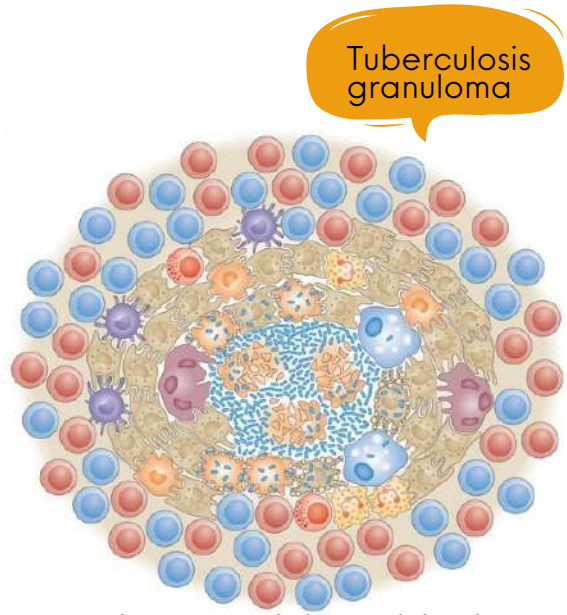
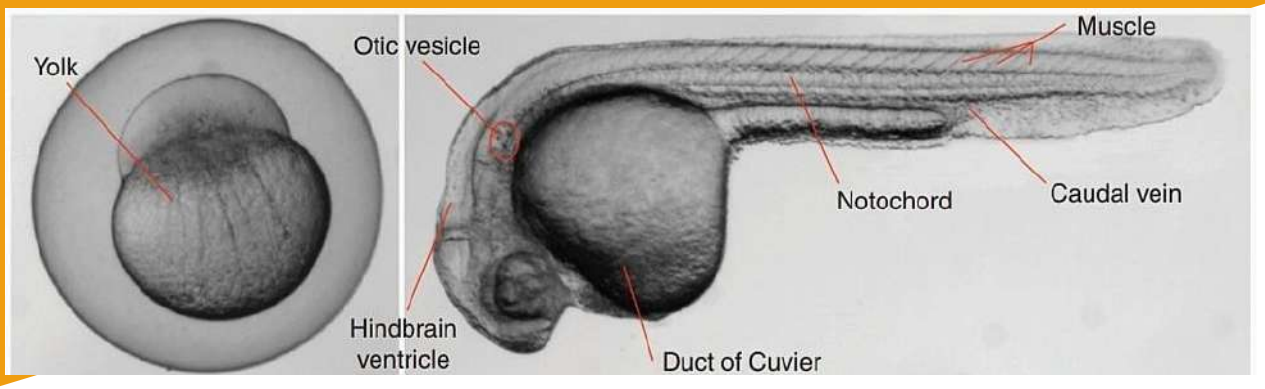
As we know that Tuberculosis (TB) is a global health emergency and to one-third of the world's population is infected with *Mycobacterium tuberculosis*. The pathogen carries on with killing 1.5 million people yearly. One of the major reasons for the poor progress in TB research has been a lack of good animal models to study the latency, dormancy, and reactivation of the disease. But the Zebrafish has newly emerged as a useful alternative to more traditional models for instance non-human primates, mice, rabbits and guinea pigs for studying the complex pathophysiology of a infection due to mycobacteria. How this is achievable is what we are going to know from this article. What is Tuberculosis? Tuberculosis is mostly a disease of the lungs, and what do you think of when you think of a TB patient! You think of someone who's thin, very thin, emaciated & in fact this disease has been called consumption over the ages. People also tend to have fevers, loss appetite and they often cough up blood. If you look at the x-ray of their chest you would see that the lungs is ravanged by TB. Also you can see a big cavity in there that is teaming up with bacteria that they're coughing up in that sputum. How is this transmitted? It's transmitted from person to person. The bacteria spew out of an infected patient and land in the lungs of a normal individuals who happens to be next to this person. The person coughs it up , and it lands in the lungs of the individual next to them and then it gets into these cells that are called macrophages, and then tricks the macrophage into taking it in, and forms big aggregates that we call tubercles then it breaks out of the tubercles to get out again and subsequently become contagious. TB is completely dependent on causing, producing active diseases in the host in order to transmit. So it's sort of an obligate pathogen(requires a host to fulfil their life cycle). But what happens next is that they induce these macrophages to form structures called granulomas and these granulomas can become quite elaborate. At first they comprise just the macrophages but then many other immune cells come in and they can form of a fairly complex structure. And furthermore to note is that the macrophages within this granuloma experience a specialized differentiation termed as epitheloid alteration where they produce interlocked projections like the fingers of folded hands and appears as a very compact form. There are many animal models that are used to study TB. The oldest ones are the rabbits and guinea pigs which were used at the time when they discovered TB. They used these animals to pass the bacterium from one animal to another to show that it was also associated with TB the most commonly used model is the mouse because mice have wonderful array of immunological and genetic array tools. By then we know that this bacterium also infects fish and it was first identified to do so in the Philadelphia Aquarium, where in 1926 fish were dying of some mysterious disease very similar to human Tuberculosis. And when they tried to culture these fish to see what bacterium they had, why where they dying? they couldn't culture any thing but when they looked at the fish by histology they could see these classic red snapper bacteria that



looked very much like TB and then Aronson had the bright idea to culture at a low temperature that was commensurate with the low body temperature of the fish and then he was able to culture the *Mycobacterium marinum*. And since then we've had *M. marinum* sequenced at the Sanger centre and it turns out to be the closest genetic relative of the human TB bacterium. It proves that *M. marinum* also causes infections in Zebrafish. Zebrafish are a pet develop organism of developmental biologist and are a natural host to *Mycobacterium marinum*. The general properties of M. marinum are: 1. *M. marinum* is a non-tuberculous mycobacterium first isolated from tubercles acquired at necropsy of dead saltwater fish in an aquarium in Philadelphia in 1926. 2. These are non-motile, non spore-forming, gram-positive bacterium. 3. They are the causative agent of a tuberculosis like disease in cold blooded animals. In humans, when the injured skin is exposed to an aqueous environment contaminated with *M. marinum*, leads to infection called fish tank granulomas. The use of Zebrafish as a model organism was pioneered by George Streisinger at the University of Oregon, U.S.A. in 1970. He is regarded as the founding father of the zebrafish as a model organism in biological investigation. Why zebrafish is considered to be an animal model? These are Fast model, small animal, provides ease of breeding and ease of genetic manipulation. It's transparency and availability of transgenic zebrafish lines make real-time imaging possible. In addition, adaptive and innate immunity can be studied one at a time These tiny fishes also enables screening possible for (i) mycobacterial virulence factors; (ii) host factors; (iii) therapeutic compounds, like antibiotic. The available evidence strongly indicates that the gastrointestinal tract is the port of entryto study mycobacterial pathogenesis in vivo, zebrafish are infected with M. marinum over non-identical inoculation routes. In Adult zebrafish: Intraperitoneal or intramuscular injection. Whereas in Embryo: Injection into the caudal vein, local inoculation routes and yolk injection at the one- to four-cell stage- for early infections. During the course of infection with M. marinum the granulomas in the zebrafish embryos develop within a few days consisting of infected, uninfected macrophages and recruited neutrophils. Zebrafish are transparent during their longish larval phase. They have a cavity called the hind brain ventricle and some bacterium were put in there very quickly macrophages came and sort of chase after the bacterium. Eventually, these macrophage gets it and then leads to an affected macrophage. A few days later it just mosey along; the bacterium grows in the macrophage and it's a permissive macrophage for the bacterium. Within a few days granuloma form, a new uninfected macrophage comes and enters the structure.



Routes of infection. Systemic infection is achieved by injection into the caudalvein or inoculation via the duct of Cuvier in embryos (Benard et al. 2012). Local injection routes are the hindbrain ventricle, muscle, notochord (Alibaudet al. 2011), or optic vesicle. Yolk injection can be applied at the one- to four-cell stage (Benard et al. 2012).

The important insights into tuberculosis gained from the zebrafish are:

- **The granuloma is dynamic in nature-**

Virulent Mycobacteria leads to granuloma formation and actively recruit phagocytes to the granuloma. Leaving of the infected macrophages from a granuloma seeds new infectious foci.

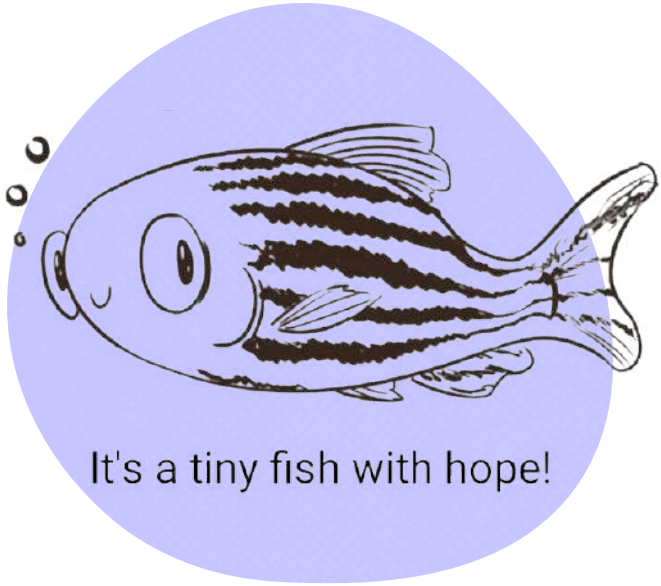
- **Mechanism of antibiotic tolerance-**

Both *Mycobacterium tuberculosis* and *Mycobacterium marinum* develop phenotypic antibiotic resistance in response to the intra-macrophage environment. This antibiotic tolerance is mediated by bacterial efflux pumps, which can be prohibited by Verapamil.

- **The Inflammation must be balanced-**

The LTA4H locus regulates the TNF gene through balanced production of pro- and anti-inflammatory eicosanoids. TNF inhibit intracell.

Therefore, the conclusion is that the Zebrafish has enabled us to piece together a cohesive impression of the innate aspects of the innate interactions between mycobacteria and host, taking us through all of the steps of pathogenesis, from gaining entry into the host via phagocytes, working around phagocytes to grow intracellularly, and finally lysing out of them to grow extracellularly so as to promote transmission. A total of 2560 papers are obtained when "zebrafish and cancer" only keywords are used whereas searching the "tuberculosis and zebrafish" keywords, only 208 different articles are obtained since the year 2000 in the Web of Science (WoS) database. This huge gap between the two numbers is attributed to the fear of using a marine model lacking the main infected organ in pulmonary TB infection. Throughout this article, zebrafish model is more and more and more proving itself to be a competent model for Tuberculosis research is reflected by the increasing trend of zebrafish applications in this field over the years. So are lungs really needed to find the key to eradicate TB?



References

1. Clay, H., Volkman, H. E. and Ramakrishnan, L. (2008). Tumor necrosis factor signalling mediates resistance to mycobacteria by inhibiting bacterial growth and macrophage death. Immunity 29, 283-294.
2. Cosma, C. L., Humbert,O. and Ramakrishnan, L. (2004). Superinfecting mycobacteria hometo established tuberculous granulomas. Nat. Immunol. 5, 828-835.
3. Davis, J. M. and Ramakrishnan, L. (2009). The role of the granuloma in expansionand dissemination of early tuberculous infection. Cell 136, 37-49.
4. Flynn, J. L. (2006).Lessons from experimental Mycobacterium tuberculosis infections. Microbes Infect. 8, 1179-1188.
5. Puissegur, M. P., Botanch, C., Duteyrat, J. L., Delsol, G., Caratero, C. and Altare, F. (2004).An in vitro dual model of mycobacterial granulomas to investigate the molecular interactions between mycobacteria and human host cells. Cell.Microbiol. 6, 423-433.
6. Ramakrishnan, L. (2012). Revisiting the role of the granuloma in tuberculosis. Nat. Rev. Immunol. 12, 352-366.

A Review on Biological Warfare: From Historical Past to Present

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Introduction

Every country, mainly the developing countries, of our modern world is under threat because of bioterrorism and biological warfare using miscellaneous types of biological agents. The objective of this review is to collect information both from the past and also in recent time.

Biological warfare is also known as the germ warfare, using various microbes as the biological agents to cause disease to human beings, animals and plants. It is considered that the biological weapons are more powerful ad effective on the target. It is done by either by an individual or by a group or by any political, religious or criminal motivation. The system of biological weapon composed of 4 vital components – payload (the biological agents), munitions (containers to keep the payloads), delivery system (missiles, artillery shells, aircraft) and dispersal mechanism (an explosive force or the spray device).

Munitions

Payloads

Delivery mechanism

Aircrafts

Historical Aspects

From the last two century, microbiology becomes to emerge and now it reaches to it's one of the maximum peak. During this time the biotechnology and the biochemistry plays a dominant role in the human welfare. But these advantages of the science become the cause of adverse effect by the help of many heinous people as well as many countries who become the constant support of those terrorist groups. This support and those planes are not very new for our world. Many evident showed that from the approximately 600 BC, this type of odious ruse was taken to ensure their rule against neighbouring areas, countries.

The events can be classified into three groups according to the time or the era –

- Before World War-I
- During World War
- After World War-II

- **Before World War-I:**

In 1346, during the siege of Caffa, a seaport of Ukraine was attacked by the Tartar force, a dangerous vagabond group. Geneose force, the controller of that seaport, used a biological weapon, the perilous plague, for protecting their seaport. But another dirty idea came from the Tartar force, as expected, that they distributed their diseased corpses to the Geneose force as well as into the city, and the pandemic of the Black Death flooded throughout the Europe to all over the world. Gabriel de Mussis, a notary of the north Genoa, claimed mainly two important points about the incident happened in the Seize of Caffa, that he first one was the epidemic of plague took place by contaminating city by the infected soldiers as well as the peoples and the second point was that the plague affected refugees along with the rats from the Constantinople, Genoa, Venice and other areas were shipped to another place and the vice-versa which led to the starting of the second wave of the plague epidemic. Even it was said about the epidemiology of the plague with very complicated format but we can understand simply that one biological attack was sufficient to start a epidemic or even pandemic as we can observe in the Seize of Caffa. During the 14th and the 15th centuries the Europe was witnessed the tragedy of the plague and more than 25 millions of people were died but at that time and after that time many war was happened where disease or the poisons were used as the weapons.

Another example of misleading construction of vision arose in the British force and caused another dangerous pandemic, smallpox. During the French-Indian War in 1754 to 1767, the commander general of the British force, Sir Jeffrey Amherst, provided the smallpox infected utensils, clothes etc. to the Indian people, since an epidemic of smallpox had been spread out in the European and North American countries. As a result for the use of contaminated blankets or clothes, that viral disease became a major curse for the native Indian population.

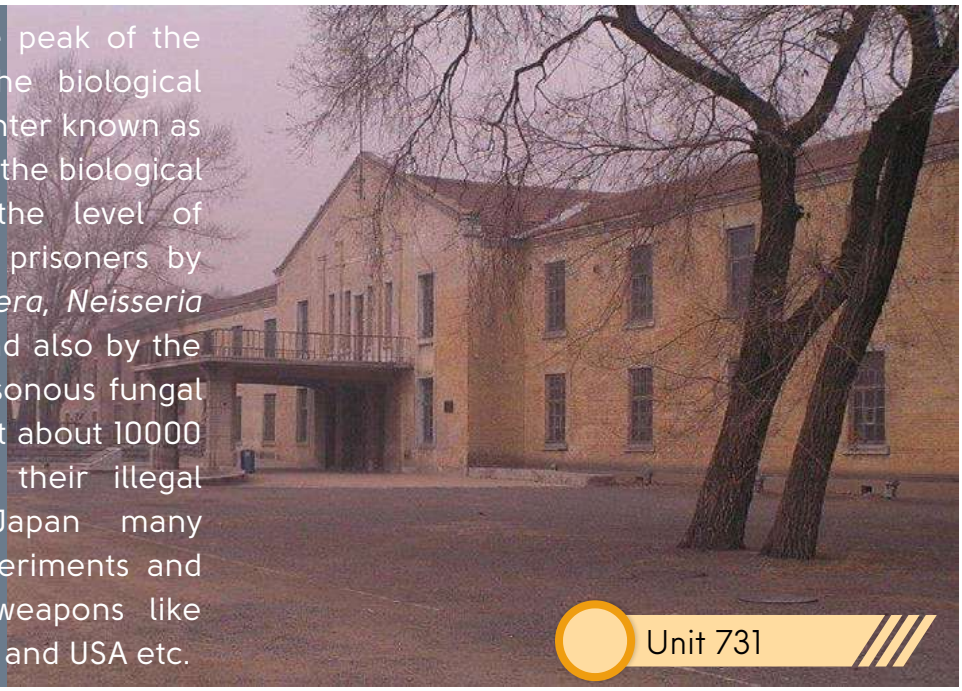
- **During World War:**

During the 19th century the starting of the golden era of microbiology became the cause of adopting the advance methodology in the bioterrorism. In this time the bio-weapons were not the crude infected materials, but they were able to isolate many bacteria and virus in their laboratory and simply inoculated into the target.

During the World War –I, Germany was accused for spreading the anthrax and glanders producing bacteria by inoculating those into the horse and cattle of the USA and its nearby

countries' ship. Not only that they also infected many Romanian, Russian sheep. Many evident proved that Germany spread vital disease like cholera and plague in the Italy and Russia respectively. But in 1924, The League of Nations fully supported the Germany and commenced that they were unable to find any strong evidence against Germany. But the whole world was shaken due to the horrible effects of the biological weapons, as their own population was being affected by their brutality, and aimed to put limitations for those types of weapons. On June 17, 1925 a protocol had been announced and signed by around 108 nations. It stated that," Protocol for to prohibition of the use of war of asphyxiating, poisonous or other gases and of the bacteriological methods of warfare", and this was named as Geneva Protocol.

During the World War –II the peak of the Japan was high for using the biological weapons. They developed a center known as 'Unit 731' for researching about the biological agents. They almost cross the level of brutality as they infected the prisoners by *Bacillus anthraces*, *Vibrio cholera*, *Neisseria meningitides*, *Yersinia pestis* and also by the terodotoxin, an extremely poisonous fungal toxins, for their research and at about 10000 prisoners were died due to their illegal experiments. Beside the Japan many countries continued their experiments and research on the biological weapons like Soviet Union, Germany, Britain, and USA etc.



Unit 731

- **After World War-II:**

The experiments of brutality with the biological agents were continued after the World War-II and still now. USA was accused to use of the biological weapons during the Korean War (1950-1953) by North Korea, Soviet Union, China and also many more disease were out broken at that time as written in many newspaper. After that event USA avoided the collaboration with the Unit 731 scientist and soon after that they planned a program to establish a new defensive protection against any terrorist attack by developing proper vaccination, treatment and therapy of their own army members. This program was a step out to become ready and then jumped ahead towards the target areas as well as countries without any massive destruction of their own troops. Approximately from 1942, but mainly after 1951, at the Fort Detrick in USA a hollow metallic sphere was constructed with a million liter volume, known as "Eight Ball", where many pathogens were exploded within it into aerosol condition. After that many volunteers were exposed inside the ball and were infected them with such pathogens. This heinous procedure was fulfilled with the aim for observing the vaccination, prophylaxis etc. In New York, San Francisco and many other cities some organisms like *Aspergillus fumigates*, *Bacillus subtilis* and *Serratia maecescens* were released over a large geographic area for studying the solar irradiation and climate condition on the viability of the organisms. After that a severe outbreak of urinary tract infection raised among the common people by *Serratia maecescens* and they admitted to the Stanford University Hospital.

Eight Ball

Table – I: Different Events Occurred After World War –II

Year	Incidents
1346	Tartar force hurled the cadavers of the plague victims to the Geneose controlled seaport
1710	The body of plague victims were used during the war between Russia and Sweden
1763	British army provided smallpox infected blankets to the native Indians
1914 - 1918	German used anthrax and glanders to infect the USA and Romanian sheep and cattle of various ships
1942 – 1945	Japan established the "unit 731" and continued the deadliest experiments upon the prisoners of the China, Mongolia, Soviet Union and America etc.
1941	1700 Japanese troops were died due to hazards of their own weapons
1949	600 Japanese prisoner were killed by Soviet Military due to only the experiments of the bio-weapons
1942 – 1969	USA produced the "eight ball" where the volunteers were deliberately infected inside the ball to determine the effectiveness of the bio-weapons and the vaccines
1964	Viral encephalitis along with death were reported from the Fort Detrick eight ball experiments
1951 – 1954	Serratia maecescens and the other agents were distributed over a large population and many people were diagnosed by urinary tract infection
1957	According to European Press the Great Britain used some bio-weapons against Oman
1961	USA infected Hong Kong city by cholera agents
1964	US military used bio-weapon against peasants of Columbia and Bolivia
1969	Egypt used cholera agent against Iraq
1984	Salad bars of a restaurant were infected by the Salmonella typhimurium in USA
1993	Anthrax spore were distributed into Japan in a ritual

Biological Agents

According to the World Health Organization (WHO), the the biological agents can be defined as an agent which is able to produce adverse effect through multiplication within the host cells or the host body causing death for accomplishing the motivation of war against animal, human, plants etc. the agents would be virus, bacteria, protozoa, any proteinous substances derived by plants which had potent cytotoxic, neurotoxic, cardiotoxic, myotoxic properties. The attackers or the terrorists always select such an agent or biological agents which fulfill the following criteria-

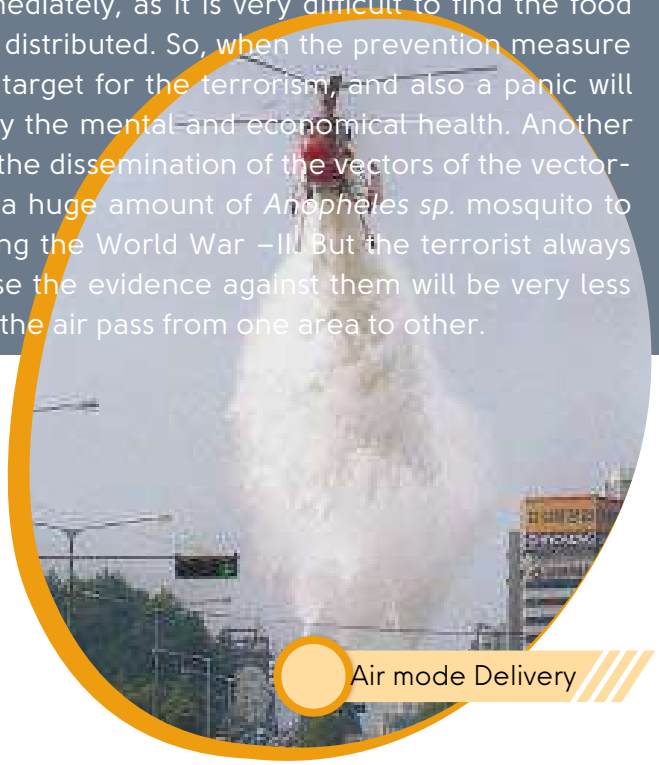
- It has to be new in morphology or in cellular content to identify,
- It has to be severely virulent,
- The disease has not been endemic in the target area,
- It must be difficult to diagnose simply,
- No vaccine should be made up against it and the treatment must be complicated

As per rules of CDC the category A agents are those that can be easily transmitted within community, having high mortality rate and will be enough to create trauma in common people and also required a high effective control measure for fighting against it. Category B agents are belonging to those group which have relatively low mortality rate but the diagnostic criterions are under development. Finally the last category C has those agents which are newly emerged in the world and have high mortality rate and very little knowledge about the diagnosis, treatment and the control measure [CDC].

Mode of Delivery

The bio-agents could be delivered by the air medium through the ventilation and air conditioning machine or system which is a very popular mode of delivery by the terrorists. As earlier about 100 kg anthrax spores was distributed over the Washington, DC, which led to about 1.3-3 millions deaths as we can say the weapon was act as the hydrogen bomb. The another route of delivery could be the food and water, as the terrorist can contaminated the drinking water or the useful water resources like pond, lake, well etc. and also contaminate the foods. The contaminated food can be the cause of death of millions people, because the food cycle is not easy to break immediately, as it is very difficult to find the food material which is infected and also where those foods are distributed. So, when the prevention measure would be taken many common people will die the main target for the terrorism, and also a panic will appear among the people which would enough to destroy the mental and economical health. Another effective way of spreading the biological agents would be the dissemination of the vectors of the vector-borne disease. As an earlier example, Japan was spread a huge amount of *Anopheles sp.* mosquito to China, which led to a malaria epidemic in the China during the World War –II. But the terrorist always accepted the air medium for disease transmission because the evidence against them will be very less than the others and the transmission rate will very high as the air pass from one area to other.

Delivery by Food & water



Air mode Delivery

Target

The target for the bioterrorism can be classified into two main streams like one is direct which the biological is and the other one is the indirect which is either political or economical. So the biological attack can be the cause of two type of epidemic – the epidemic of the disease by the causative agent and the epidemic or even the pandemic of fear and panic because of this disease. The panic is very strong weapon to destroy the economic balance as well as the mental balance of a country as that country or the affected area loss their tourism, export and the investments. The vital aim of the bioterrorism to proliferate the fear, anxiety, uncertainty, depression and finally the mistrust for the government and these are enough to collapse the commerce and the tourism. The second advantage for the bioterrorism is the physical disease which is caused to finish the man-power of an affected area. A small scale biological attack on the "soft" target, like airport, railway stations, food productive factories etc., is enough to destroy the total economical and the social dimensions. Some examples can clear our vision; the airline industry lost about $10 billion in 2003 due to SARS epidemic. In case of US anthrax attack in 2001, the estimated cost for sanitizing the containment part of the Hart Senate building in Washington DC was $26.2 billion per 100000 affected persons. The Western countries unlike the Eastern they produce food at a large scale in centralized food industry. That information may be very interesting for the bioterrorists as only one attack at those factories can lead to destroy the thousands of people who depend on it.

Impacts on Physical & Mental Health

Biological warfare is very scary for common people and it has ability to break down the total physical and mental health along with the economy. First, the terrorist use the weapons which are invisible for the naked eye, like the pathogens and the poisons. So it is impossible to determine that the individual is exposed or not. For this reason, the people are unable to say if they injured or not.

Second, the biological agents are contagious which spread person to person contact. This fact create a situation of fear helplessness when the family, friends, lover, neighbors may be the main source of disease. At that time the safe health and social support is needed but not available. We already know that every biological warfare agents have a feature to distress the mind which is enough to destroy the mental health and the social safe measures like isolation, social distancing, quarantine as well as the separation of children from their parents create a depression, anxiety among the local people.

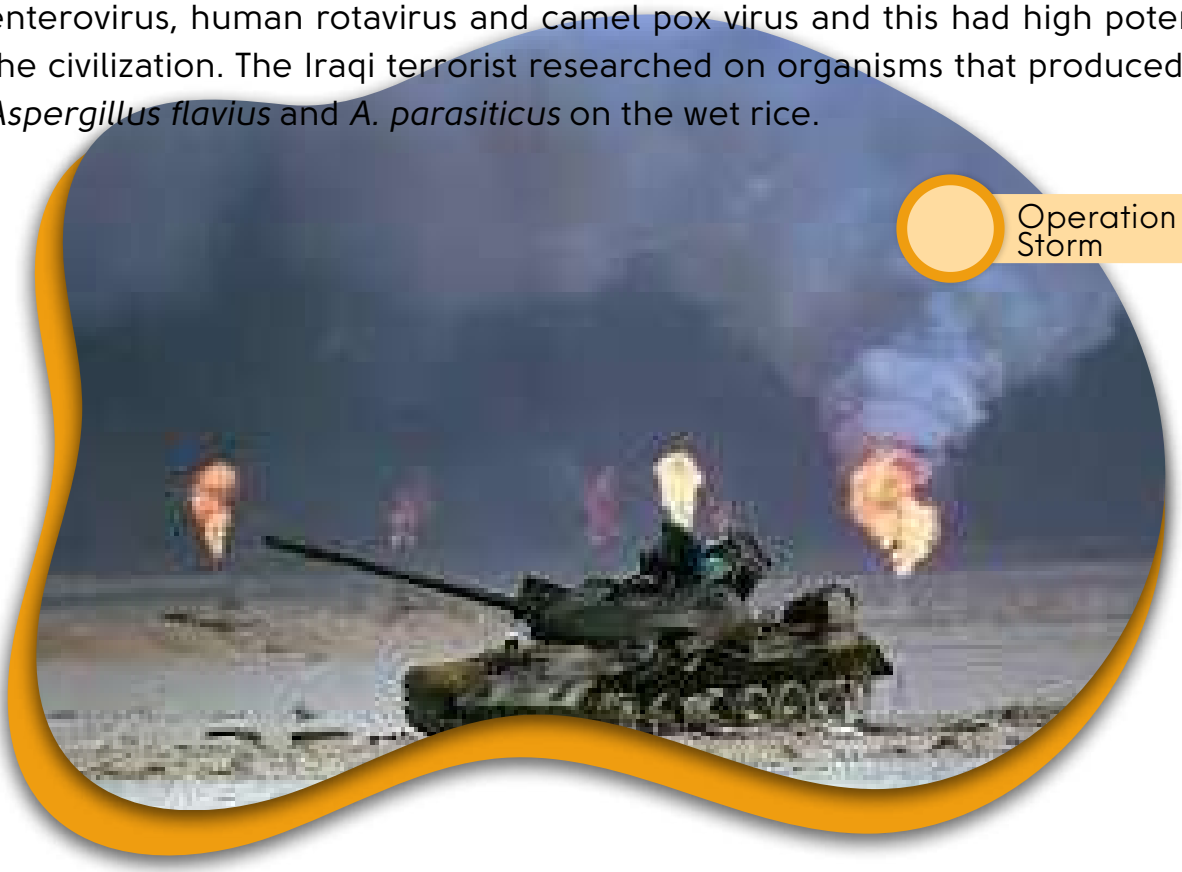
Third, biological agents enhances the fear because many pathogens are newly borne and the pathogenesis, clinical features, symptoms, treatments are rarely known and the health professionals have the lack of knowledge about who is at risk, what health concerns might be taken and how to fight against it.

Fourth, the signs of autonomic arousal may be misattributed in many persons who act as the evidence of the infection or the contamination, involves various symptoms like muscle tension, palpitation, hyperventilation, vomiting, sweating, tremors and a sense of foreboding. For this, the physically healthy but frightened person can show like autonomic arousal which leads to deal with them as an affected one and may overwhelm the heath service.

Fifth, persons have to wear musk, gloves, and clothes which can protect them from contagious disease, but but this make the people more distress because of wearing those for long tome which produce claustrophobia, breathing problems. These effects are more common in the doctors, nurse, and health workers as they wear those kits for several hours to several days constantly.

Role of Iraq in Bio-War Preparation – An Example of Brutality

The scientist had discovered many potent biological agents, planned to use by Iraq in the Operation Desert Storm, like 5 bacteria, 1 fungus, 5 virus and four toxins along with those they also developed two bacterial strains for stimulant purpose (non-pathogenic bacteria) like *Bacillus thuringiensis* and *Bacillus subtilis*. Iraq continued their experiments and production of these agents on several plants and laboratories like Salman Park, Al Hakam Single cell Protein Production Plant, Al Manal, Muthana State Establishment etc. First, amounts of some anthrax spore were imported from USA and France to the Salman Park, where the media, storage, knowledge about the pathogenecity were recovered. Then the Al Hakam supplied a huge amount of anthrax in 1989 approximately 8000 L of solution. As same as anthrax, *Clostridium perfringens* were imported from USA and then studied and produced at a large scale in the Al Hakam in 1990 and they produced nearly 340 L solution containing the C. perfringens. Another agent was a fungus, wheat cover smut, which was disclosed by the scientist in 1985 that it would be fatal for the young wheat plants. In 1988 a large production of the wheat plants took place in Mosul town and infected rapidly by this fungus and harvested those carefully then transferred to the Agriculture and Water Resource Research Center, Fudaliyah for storing and making weapons. In Al Manal 5 viruses were recognized as bio-weapons like Congo- Crimean hemorrhagic virus, Yellow fever virus, enterovirus, human rotavirus and camel pox virus and this had high potency to destroy the civilization. The Iraqi terrorist researched on organisms that produced Aflatoxin, like *Aspergillus flavius* and *A. parasiticus* on the wet rice.



Operation Desert Storm

This toxin was produced in the Salmon Park in 1989 and approximately 2000 L of solution was manufactured for the war. By Clostridium botulinum Iraqis were able to produce about 20000 L solution of botulinum toxin at Al Hakam and Al Manal plant. In 1989 *Ricinus communis*, the caster bean plants were cultivated widely which produces ricin and about 10 L of ricin solution was produced at Salman Park and used as payload in artillery shells. The fungus, *Fusarium oxysporium* and *F. granarium* were grown in the dump, supplemented rice, have ability to produce a mycotoxin called tricothecene. This fungus was cultivated inthe several center and their toxins were extracted by using the organic solvents (This tricithecene caused "yellow rain" around the H'mong and other native people of Indochina peninsula). These all type of microbes and toxins were manufactured at Al Hakam, Al Manal and Salman Park and then transferred to the Muthana State Establishment to prepare the payloads by using those materials. For proper dispersions of those materials Iraqis accepted many modern techniques. They installed Italian made pesticide dispersal system with sprayer nozzles, generating aerosols of 1-5 m and appropriate holding tank in many aircraft and vehicles. In 1990, they were able to modify a MIG -21 fighter plane to equip a 2200 L belly tank which was taken from Mirage- F1 fighter plane, and a sprayer mechanism. The United Nation Security Council Resolution 687 in April 1991 ordered their personnel to destroy all of biological agents of Iraq, after accepting the cease-fire. Then all stored biological agents were treated with formaldehyde and KMnO4 and finally those mixtures were poured on a bare land in the Al Hakam campus. All munitions were crushed and burned entirely into pits and finally the remains were sealed and simply thrown into the Tigris River.

Conclusion

The only aim for this article is to raise awareness to every people about the devastating nature of the bio agents as well as the mentality of the human being. After knowing the whole history of all attacks we can understand that we will be helpless if it will be attacked, and cannot do so much that our population can survive. So to stop this heinous disaster we should change our mentality and thus we can tend to stop bio-terrorisms.

References

1. Eitzen EM Jr, Tarafuji ET. (1997). Historical Overview on Biological Warfare. Walter Reed Army Medical Center, 415-423
2. Riedel S. (2004). Biological Warfare and Bioterrorism: A Historical Review. Baylor University, Medical Center Proceeding; 17: 400-406
3. Wheelis M. (2002). Biological Warfare at the 1346 Seize of Caffa. Emerg Infect Dis; 8: 971-975
4. Christopher GW, Cieslak TJ, Pavlin JA, Eitzen EM. 919970. Biological Warfare: A Historical Perspective. JAMA; 278: 412-417

Weblinks

1. https://www.google.com/searchq=unit+731&rlz=1C1CHBD_enIN968IN968&sxsrf=AOaemvINqSBT6_6tHGFXVNhMLinr3Fgeg:1635429805274&source=lnms&tbm=isch&sa=X&ved=2ahUKEwivkeTkou3zAhVZfisKHT_HAO0Q_AUoAXoECAEQAw&biw=962&bih=697&dpr=1#imgrc=jW8PGFK_86QibM
2. https://www.google.com/searchq=operation+deseart+storm&tbm=isch&ved=2ahUKEwj8lenBpe3zAhViMHIKHELEDMgQIABBDMggIABCABBCxAzIFCAAQgAQ6BwgjEO8DECc6CECdQusEFWNydB2DxoQdoBnAAeAOAAYsgiAHIjwGSARMwLjEuNi4xLjMuMS4xLjMuMS4ymAEAoAEBqgELZ3dzLXdpei1pbWewAQrAAQE&sclient=img&ei=ibB6YbytDLgyAPBuLCwDw&bih=697&biw=962&rlz=1C1CHBD_enIN968IN968#imgrc=AUFUKMvW-pFlLM

Prediabetes & Clinical Prevention Using Metformin

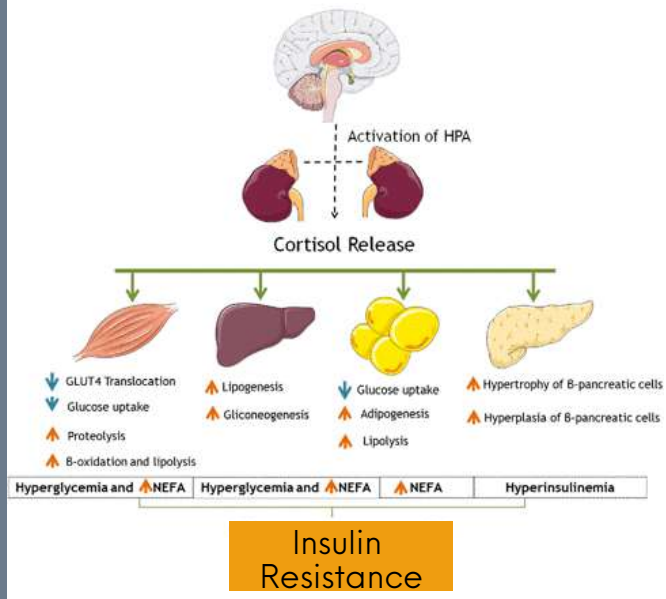
Rumi Ghosh, Ex Student (2019-21), PG Dept. of Zoology, Barasat Govt. College

Stress, hypertension, high risk of cardiac arrest, weight gain, anxiety, fatty liver, high blood pressure, high glucose levels within blood among almost 14-28 years old! ---------Are you pre-diabetic?------- A question asked for young generation in all over the world.

It's known well that excess glucose level in blood and decrease in secretion of insulin are stamp to develop diabetes in human being. Even 100 years ago, there were most probably 2-3 cases of diabetes patients per megacity, but now a days it has spread as epidemic disease in world's each population density.

Prediabetes

Global problem in young generation is declared as a form of prediabetes which introduces itself ashigh glucose level in blood than normal level but not enough to diagnose type 2 diabetes. Many Adults and children with risk of prediabetes are more likely to develop type 2 diabetes. Prediabetes may be called borderline diabetes .Almost 10-23% of people with borderline diabetes may develop type 2 diabetes within 5 years and the condition may happens in people with some insulin resistance or it may be that pancreatic beta cells aren't making enough insulin to maintain blood glucose in range in the body.sometimes Prediabetes conditions are described as impaired glucose tolerance (IGT) or sometime impaired fasting glucose (IFG).

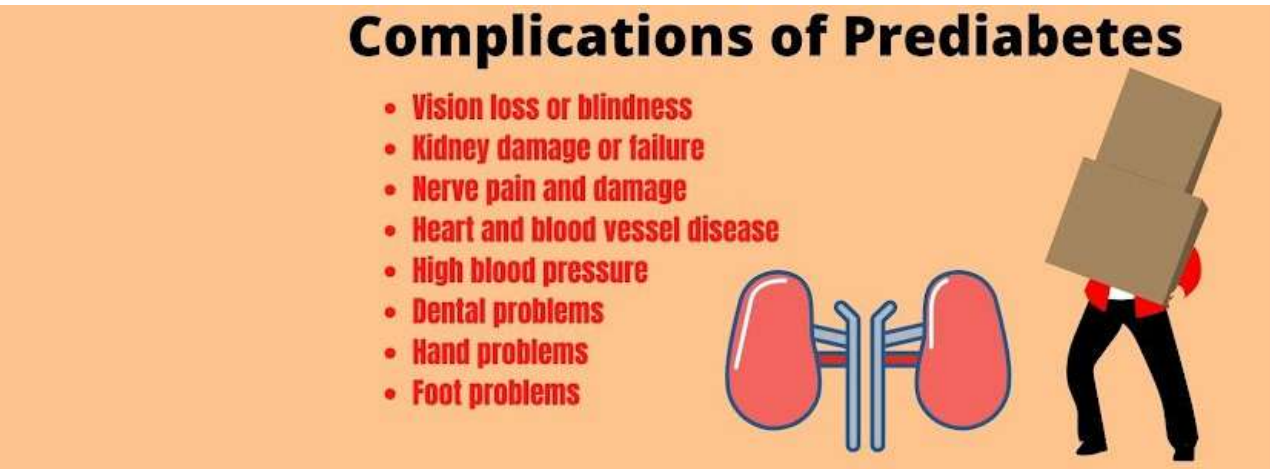


Busy Irregular Life-Style Causes Stress & It's Harmful Affects

Stress appears important consideration for pre-diabetes risk. Stress development stimulate the hypothalamic-pituitary-adrenal (HPA) axis producing cortisol, which induces hepatic resistance, decreased insulin secretion. Chronic overexpress causes HPA axis dysregulation and it is strongly implicated in pre-diabetes development.

Symptoms & Several Complications

Prediabetes has more or less initial symptoms of developing diabetes, including frequent urination, increased thirst, blurry vision, hypertension, weight gain, nausea, always hungry, sexual abnormality. Though prediabetes patients have no serious symptoms but it represents some common syndrome like diabetes initially among young generation. Several complication may arise and its absolutely cause various health deformities.

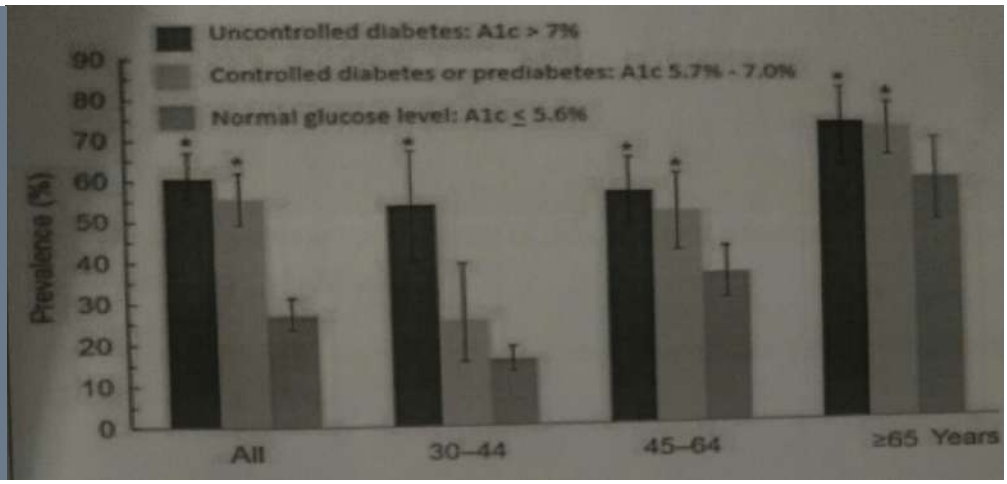


Pathogenesis of Glucose Tolerance

Pre diabetes precede type 2 diabetes development and also associated with increased plasma insulin concentration. This occurs as compensatory response by pancreatic beta cells for diminished sensitivity of target tissue (Insulin resistance).

Prevalence

The International Diabetes Federation (IDF) estimates total no of diabetes subjects to be 40.9 million in India and this may rise to 69.9 million by the year December 2025. According to national urban diabetes survey, diabetes prevalence was 12.33% and of pre-diabetes was 11.57% in India 2019 with more female numbers compared to male.

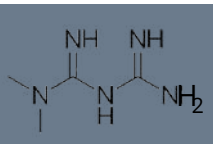


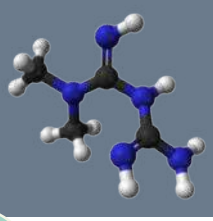
Metformin - Clinical Trials for Significant Reduction of Risk Development of Type-2 Diabetes

Metformin With Chemical Composition

Candelilla wax, cellulose acetate, hypromellose, Magnesium stratagem, polyethylene glucose, polysorbate 80, Titanium dioxide.

GLUMETZA (metformin hydrochloride) tablet may be prescribed in type-2 diabetes management as oral antihyperglycemic drug.





Metformin hydrochloride ($C_4H_{11}N_5 \cdot HCl$) is white or off-white crystalline compound. It is freely soluble in water and is also insoluble in acetone, ether, or chloroform.

Metformin hydroxychloride 500

Metformin decreases hepatic glucose production, intestinal absorption of glucose, and improves insulin sensitivity by increasing peripheral glucose uptake, utilization etc. Metformin act as a current first-line pharmacological treatment for type 2 diabetes (T2D) and it is an orally administered drug that improves insulin sensitivity, decreases the insulin resistance which is prevalent in NIDDM.

Metformin act with both AMP-activated protein kinase (AMPK)-dependent and AMPK-independent mechanisms, and it inhibits mitochondrial glycerophosphate dehydrogenase, a mechanism that involves the lysosome. Metformin improves glycaemia by the actions on the liver with AMPK activation.

Molecular Mechanisms for Metformin-Associated Ampk Activation

Mitochondrial inhibitions can explain metformin's ability for the cellular energy sensor AMP-activated protein kinase (AMPK) activation. Once activated increasing AMP: ATP and ADP: ATP ratios (indicative of cellular energy balance being compromised), AMPK restore energy homeostasis by catabolic pathways generating ATP switching, while switching off cellular processes consuming ATP. Here AMPK is involved in metformin action which was attractive.

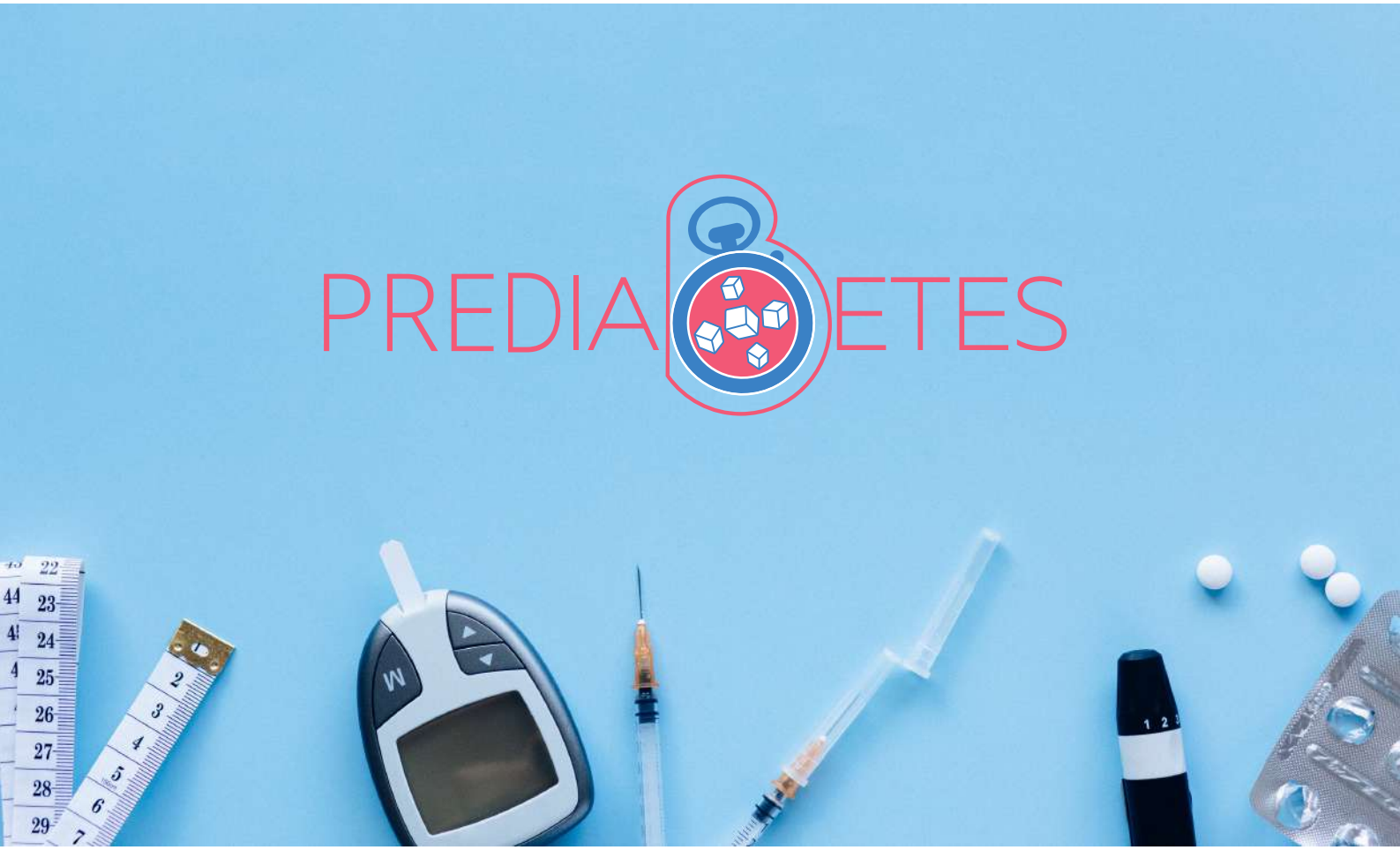
Metformin - Ampk-Dependent, Independent Effects on Hepatic Gluconeogenesis

5-aminoimidazole-4-carboxamide-ribonucleoside(AICAR), is the first pharmacological activator of AMPK which is taken up into cells and then phosphorylated to the nucleotide 5-amino-4-imidazolecarboxamide riboside 5′-monophosphate (ZMP), that becomes involved in mimicry of AMP effects regulating AMPK system. AICAR down-regulated expression of the gluconeogenic enzymes PEPCK and glucose-6-phosphatasesupported the idea that AMPK activation might be responsible for Metformin ability to inhibit hepatic glucose production. ZMP modulates other AMP-sensitive enzymes like fructose-1,6-bisphosphatase(key enzyme of gluconeogenesis)that is inhibited allosterically by AMP and ZMP and thus, AMP have AMPK-independent effect, and perform lower cAMP and reduction of gluconeogenic enzymes expression. In AMP dependent mechanism, AMPK activators reduce glucagon-induced cAMP levels and on the other hand, induce direct AMPK-mediated phosphorylation of the cAMP-specific 3′,5′-cyclic phosphodiesterase 4B (PDE4B), for triggering cAMP breakdown.

Physiologically, metformin acts on the liver directly or indirectly for lowering glucose production, and also on the gut to increase glucose utilisation . It also increases GLP-1 a altering the microbiome. At the molecular level, metformin inhibits the mitochondrial respiratory chain in the liver, which leads to AMPK activation, enhances insulin sensitivity and lowers cAMP. At last it reduces gluconeogenic enzymes. Metformin plays a role on AMPK-independent effects on the liver including inhibition of fructose-1,6-bisphosphatase by AMP.

Conclusion

Now a days, we know more or less about diabetes but no clear idea on pre-diabetes in maximum people, so to improve learning about it , many researcher recently are continuing research for giving information about prediabetic condition. Adults, young generation in a large scale, suffer from pre-diabetic condition with high blood pressure, increase heart- attack, high glucose in blood, premature death and other health hazards, being affected with pre-diabetes (40% population in USA and 29% population in India) because of busy irregular life style, lack of healthy food intakes, negligence in physical exercise and others. So to prevent this global health problem, each people should take care of health with good habits, yoga, relaxation etc. As a clinical treatment, oral dose of Metformin may be applied in human to prevent high risk of developing diabetes because of property of safety and easy availability in market.



Weblinks

1. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5552828/
2. https://www.fixdiabetes.in/2020/12/what-is-prediabetes-Causes-Symptoms-Diagnosis-Complications-Treatment%20.html?m=1
3. https://www.webmd.com/diabetes/what-is-prediabetes
4. https://www.cdc.gov/diabetes/basics/prediabetes.html
5. https://my.clevelandclinic.org/health/diseases/21498-prediabetes

Anti-carcinogenic Effect of Green Tea

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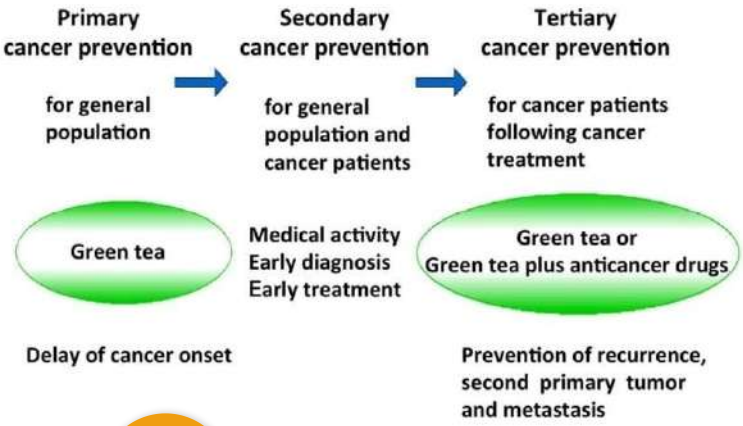
Introduction

Tea is second most important healthful beverage that people from all over the world drink since more than thousands of years. When it comes to green tea it's been one of the best aromatic beverages that more than million of people from all over the world it consume everyday. Green tea is produced from dried leaves of *Camellia sinensis*. Green tea first originates in china but now grown in other countries of Asia too. Green tea provide anti-carcinogenic property due to the presence of catechins and polyphenol out of which the important is the **Epigallocatechin gallate** (**EGCG**). So in this report we are able to investigate how green tea is able to reduce the risk of cancer in our human body. There are various types of health benefits of consuming green tea-

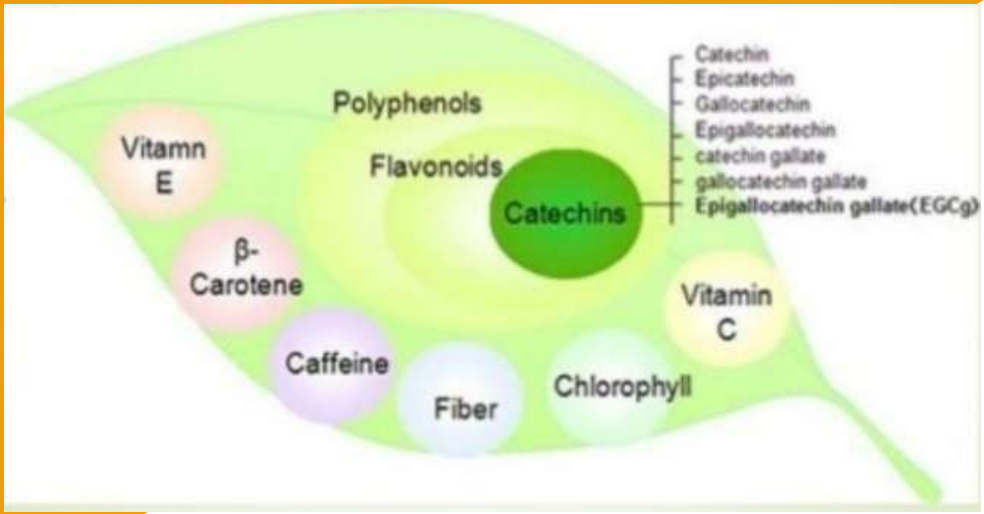
- Anti-inflammatory
- Cardiovascular diseases prevention
- Anti-arthritic
- Antibacterial
- Anti-angiogenic
- Antiviral
- Anti oxidative
- Neuroprotective
- Cholesterol lowering effects



Primary, secondary & tertiary cancer prevention in humans

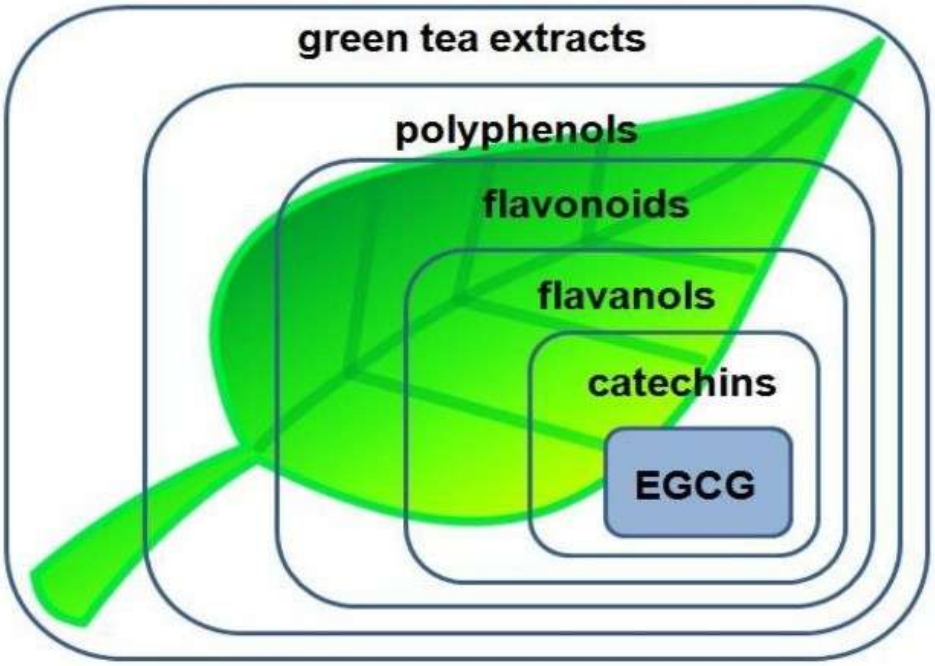
Cancer may regard as a group of disease characterized by an abnormal growth of cells, also can invade and spread to others part of the body. Cancer can affect different parts of our body and accordingly they are named like lung cancer-related cancer of lung, oral cancer, breast cancer, uterine cancer etc. Cancer can be caused by various agents which are categorized into three classes-physical (UV rays), chemical(carcinogens like nitrosamines in tobacco) and biological (infections, heredity). The eventually death of affected patient if the tumor has progressed beyond the stage when it can be successfully removed and the treatment of cancer is very costly and not available anywhere.

Chemical Composition of Green Tea



Antioxidants	For Example	Dry Weight	Functions
Lipids	Linoleic acid	2-6%	Provide energy
Carbohydrates	Glucose, cellulose	5-7%	Prevents high blood sugar
Vitamins	B,C,E	<1%	Youthfulness healthy skin
Pigments	Chlorophyll, Carotenoids	<1%	Deodorizing effect, nighttime vision
Amino acids	Glycine, Valine	1-4%	Relaxation effect
Alkaloids	Caffeine, Theaphylline	3-4%	Boost immune system
Minerals	Fe, Mg, Cr, Ca, Ni, K, P, F	5%	Biological regulators

- **Xanthic Bases**
  Caffeine and theophylline
- **Proteins**
  15-20% dry weight
- **Sterols**
  Stigmasterol
- **Volatile compounds**
  Aldehydes, esters, lactones and hydrocarbons etc.
- **Water**
  99.9 g
- **Phytochemicals**
  - Caffeines
  - Polyphenols



1. Epigallocatechin gallate (EGCG)
2. Epicatechin gallate (ECG)
3. Gallocatechin gallate (GCG)
4. Catechin gallate (CG)
5. Flavonoids (Flavonoids have antioxidant, anti-carcinogen, anti-inflammatory, anti-radiating biochemical effects.)
6. Myricetin
7. Quercetin
8. Kaempferol

Discussion

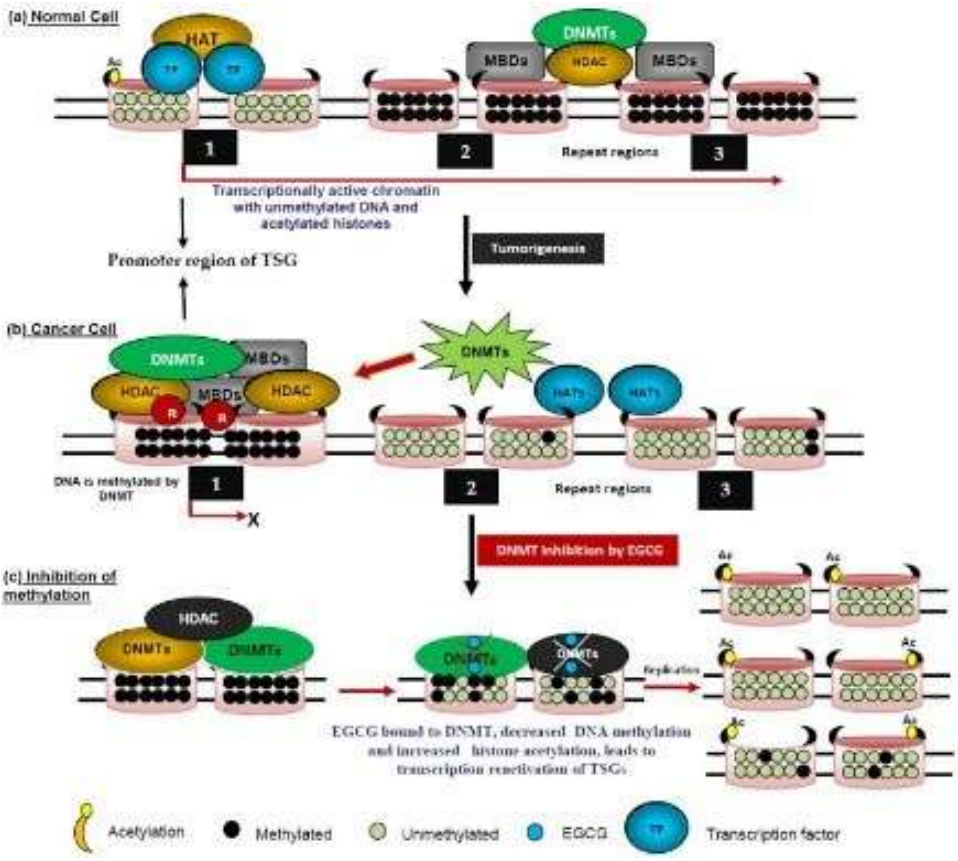
We would discuss about how the components that are present in green tea helps in the prevention or treatment of cancer by different mechanisms :

- **Inhibition of proteasome activity in the cancer cells**

Proteasomes are protein complexes and also a multi catalytic enzyme that helps in degrade which unneeded or damaged protein. The ubiquitin-proteasome pathway (UPP) is a system the that plays highly selective proteolytic a crucial a role in regulation degradation of regulatory proteins that are involved in important cellular processes such as cell cycle control, cell proliferation, apoptosis transduction etc. Ubiquitin proteasome pathway system plays an important role in cancer development. So, the inhibition of this ubiquitin proteasome system is needed in order to prevent the proliferation and growth of tumor cells and that is what EGCG a present in green polyphenol also tea do. It inhibits the catalytic activity of proteasome which results in the arrest of and the growth of tumor cells in G_1 phase of cell cycle and management cancer through the modulation of different cell signaling pathways. Also induces apoptosis of the cell. Thus in this away it prevent cancer.

- **Protection of DNA from methylation**

DNA Methylation is a biological process by which the addition of methyl groups to the DNA molecules and this process can change the activity of DNA segment without changing the sequence. DNA methylation causes suppression of gene transcription in the segment of gene promoter. Hypermethylation in the promoter region of DNA repair related gene which causing cancer. EGCG interferes with the activity of DNA methyltransferase enzyme that results in decreased methylation frequency of the promoter regions of the gene that which causes the tumor suppressor related gene and DNA repair to active again and hence the tumor formation which leads to cancer is prevented. Hypomethylation results in transcriptionally activates the oncogenes that causing metastasis overall of this, which leads to oncogenesis and thus cancer.

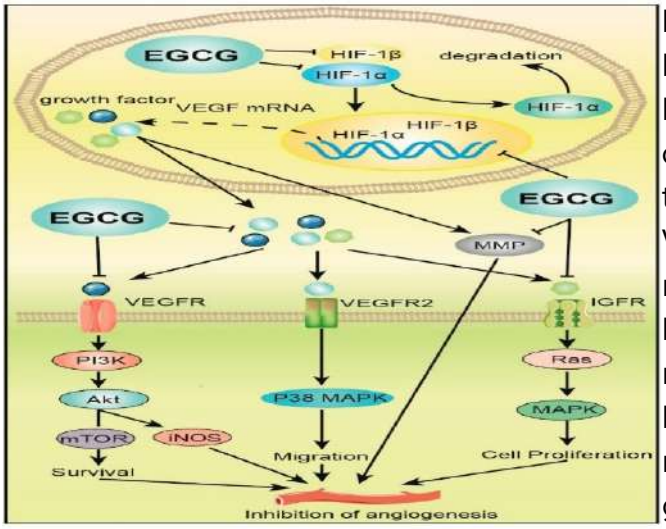


- **Inhibition of urokinase - plasminogen activator (ups)**

Urokinase is a specific serine protease present in blood and extracellular matrix of different tissues in human which catalyzes the conversation of plasminogen to plasmin within the extravascular components. Plasminogen is a broad spectrum protease which and plays a crucial role in cancer invasion and metastasis dissemination by allowing the malignant cells to invade tumor site locally and causally involved in metastasis. Urokinase also regulates chemotaxis and also act as a thrombolytic enzyme which convert plasminogen to plasmin which promotes dissolution of blood clot. In cancer cells there is a increase in the level of the expression of urokinase results in degradation of basement membrane and also extracellular matrix, which helps in tumor cell migration. The EGCG inhibits the activity of urokinase which prevent from cancer.

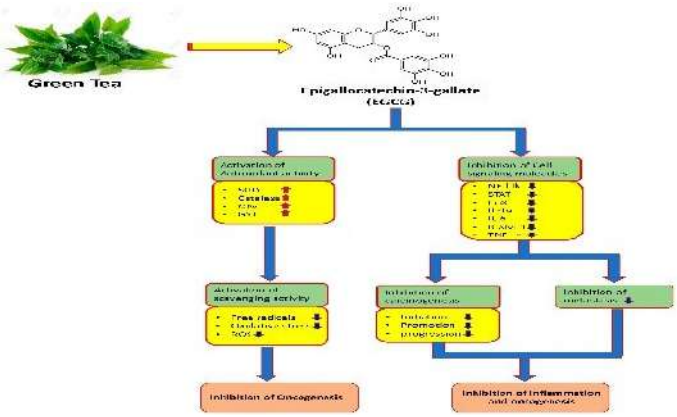
- **Inhibition of angiogenesis**

Angiogenesis is the physiological process through which new blood vessels formed from the pre-existing ones. The tumor cells require optimum amounts of and nutrient to grow and for this they need blood supply. For their metastasis tumor cells need blood vessels. So, They induce angiogenesis by secreting vascular endothelial growth factor (VEGF) and basic fibroblast growth factor (BEGF) and other angiogenic activator which results in formation of blood vessel.EGCG present in the green tea which blocks the induction of VEGF and BEGF and no blood vessels are formed, for this reason tumor cells are not growing and induce apoptosis of tumor cells. Angiogenesis leads to the secretion of VEGF which activate endothelial cells to produce metalloproteinases (MMPs) that degrades basement membranes and cancer cells can migrate through it and comes with the contact of blood and thus spread. Since no VEGF is secreted no angiogenesis and hence no cancer cells will grow.

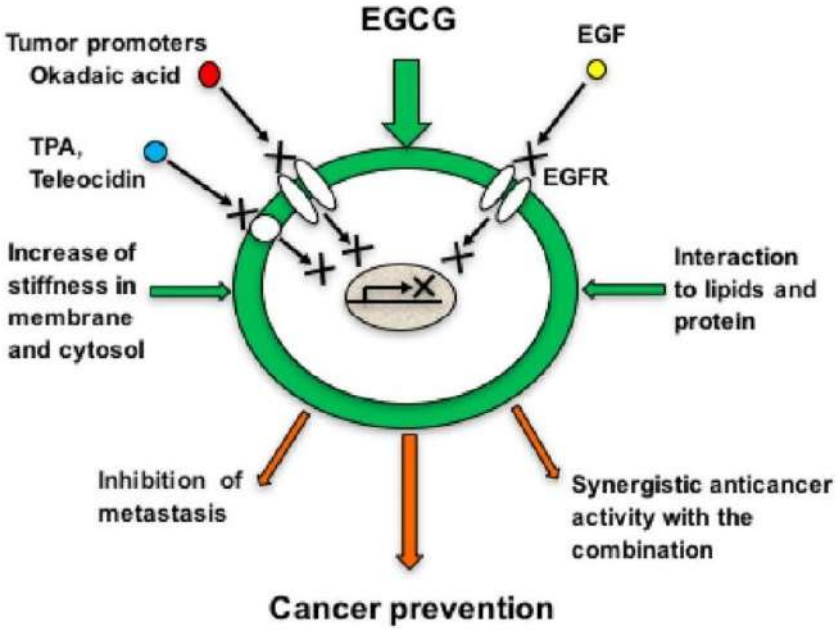


- **Inhibition of oncogene expression**

Oncogenes are the gene that causes cancer in tumor cells when it expressed at a high level. The most active anticancer component of green tea is EGCG inhibit the expression of oncogenes by binding to a protein and preventing it from functioning and inhibit oncogenic signaling in tumor cells.

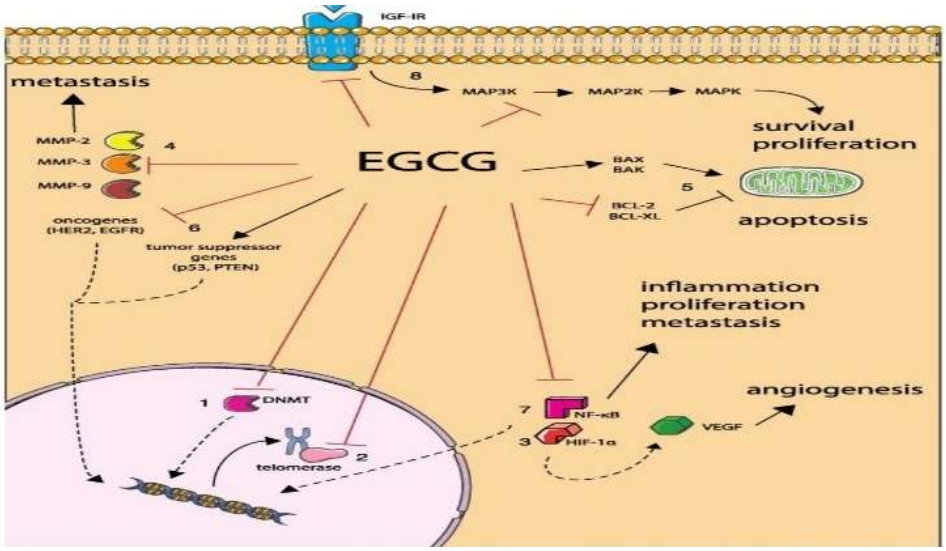


- **Inhibition of epidermal growth factor re (EGFR) mediated pathways**



Epidermal growth factor (EGF) stimulates cell growth, proliferation and differentiation protein by binding to its receptor in a normal cell. But in carcinoma cells it is over expressed which causes the cancer cells to grow and divide rapidly. The polyphenol EGCG of green tea inhibit the autophosphorylation of EGFR in cancer cells which inhibit the activation of EGFR, HER 2 and multiple downstream signaling pathways that stops cell growth and proliferation.

- **Induction of apoptosis**



Apoptosis is a form of programmed cell death in occurs in the multicellular organisms. The Bcl-2 family proteins are anti-apoptotic proteins which prevent cell survival and inhibits apoptosis. In cancer cells there is an increase in the expression of anti apoptotic genes which activate anti-apoptotic proteins of Bcl- 2 family which ultimately results in the loss of apoptotic property cells. The ECGC of green tea binds with the BH3 domain of the anti-apoptotic Bcl-2 family protein that inhibits the function of protein. Thus the balance in lost and the pro-apoptotic proteins increase and apoptosis process started.

- **Inhibition of matrix metalloproteinases (MMPs)**

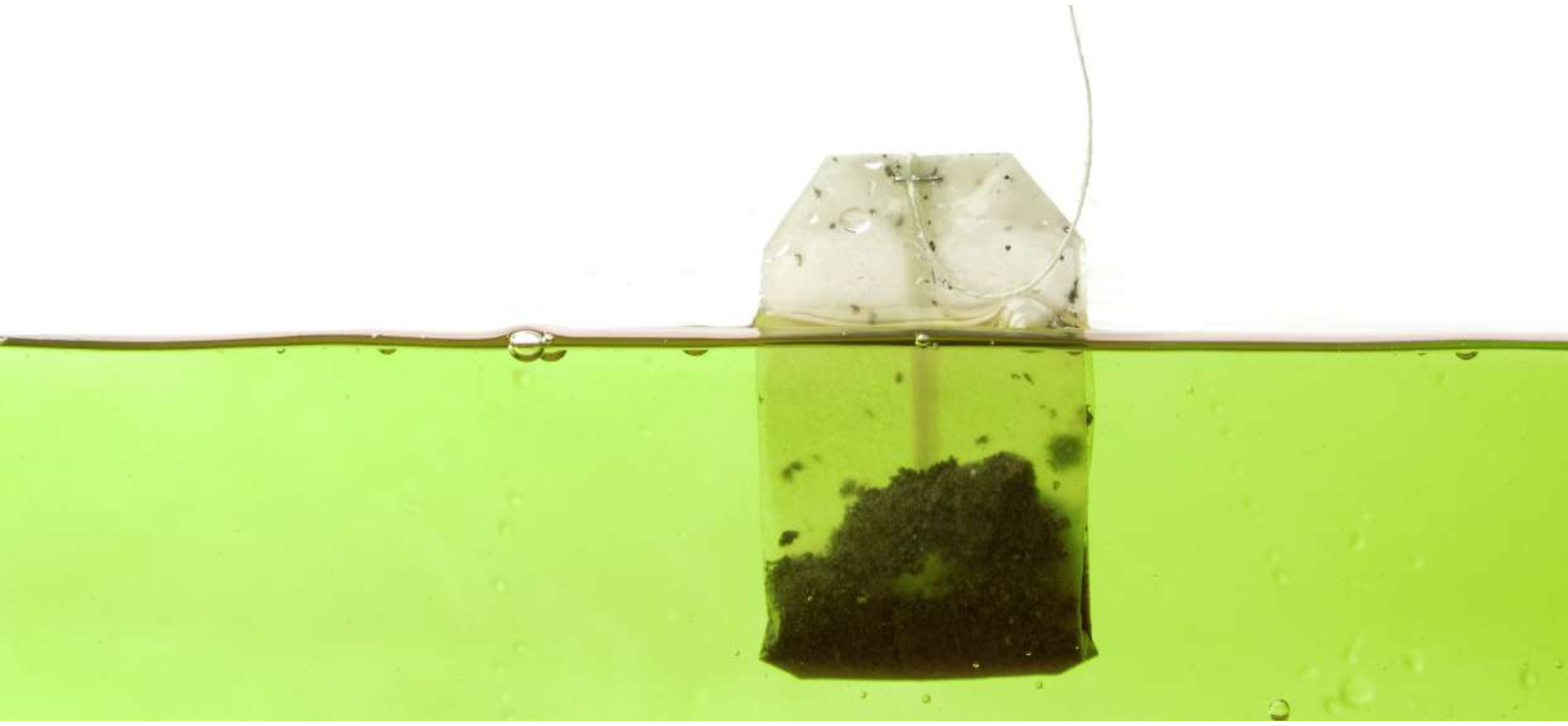
Matrix Metalloproteinases (MMPs) are a family of protein that degrade all types of extracellular matrix proteins. Over expression of MMP genes take place in carcinoma cells. The EGCG of green tea inhibit the normal function of MMPs and there is no degradation of basement membrane and so no migration of tumor cells as no angiogenesis, also unable to inactive cytokine which inhibit tumorigenesis and unable to the degradation of pro-apoptotic and anti-apoptotic molecules which helps in apoptosis. All of these causes to failure in the development of cancer.

Conclusion

The natural anti-carcinogenic property of green tea is mainly due to a phytochemical, a polyphenol named Epigallocatechin gallate (EGCG) that is present in green tea in adequate amount has a lot of healthy benefits. This polyphenol helps in prevention and treating the cancer. It is also used in the treatment of cancer in combination with some chemical that are its structural analogues and also with other chemicals. This polyphenol compound has natural anticarcinogen properties which prevent cancer by slight modifications in many processes that occurs in our normal body cells but are altered in the tumor cells. Somewhere, this compound prevent the tumor cells from migration by different mechanisms, somewhere it prevent the tumor cell form getting adequate supply for growth and proliferation, and somewhere it helps in the

Molecular targets of green tea

programmed death (apoptosis) of the tumor cell — a property that is lost form the cancer cells. This compound basically prevent the prevent or cut off thefactors that are required for the growth, proliferation , modification and transport of cancer cells. The overall effect of which leads to prevention of carcinoma. Though green tea use as medicinal purposes but the anticarcinogen effect is of prime importance as it's a natural property resent in it as all other artificial modes of treatment of cancer is high costly and not available everywhere. As we all know that prevention is better than cure, so drinking green tea daily can reduce risk of cancer by many type folds and also the person leads healthy and happy life but consumption of green tea in excess can cause some health problems so make sure that to take proper amount is necessary for healthy beneficial feedback.



References

1. Fujiki H, Watanabe T, Sueoka E, Rawangkan A, Suganuma M. Cancer Prevention with Green Tea and Its Principal Constituent, EGCG: from Early Investigations to Current Focus on Human Cancer Stem Cells. Mol Cells. 2018 Feb 28;41(2):73-82. doi: 10.14348/molcells.2018.2227. Epub 2018 Jan 31. PMID: 29429153; PMCID: PMC5824026.
2. Miyata Y, Shida Y, Hakariya T, Sakai H. Anti-Cancer Effects of Green Tea Polyphenols Against Prostate Cancer. Molecules. 2019 Jan 7;24(1):193. doi: 10.3390/molecules24010193. PMID: 30621039; PMCID: PMC6337309.
3. Pervin M, Unno K, Takagaki A, Isemura M, Nakamura Y. Function of Green Tea Catechins in the Brain: Epigallocatechin Gallate and its Metabolites. Int J Mol Sci. 2019 Jul 25;20(15):3630. Doi: 10.3390/ijms20153630. PMID: 31349535; PMCID: PMC6696481.
4. Shirakami Y, Shimizu M. Possible Mechanisms of Green Tea and Its Constituents against Cancer. Molecules. 2018 Sep 7;23(9):2284. doi: 10.3390/molecules23092284. PMID: 30205425; PMCID: PMC6225266.
5. Yang CS, Wang H. Cancer Preventive Activities of Tea Catechins. Molecules. 2016 Dec 9;21(12):1679. Doi: 10.3390/molecules21121679. PMID: 27941682; PMCID: PMC6273642.

The World From the Eyes of the Snakes

Deboleena Bandyopadhyay, (2020-22), PG Dept. of Zoology, Barasat Govt. College

During Daytime

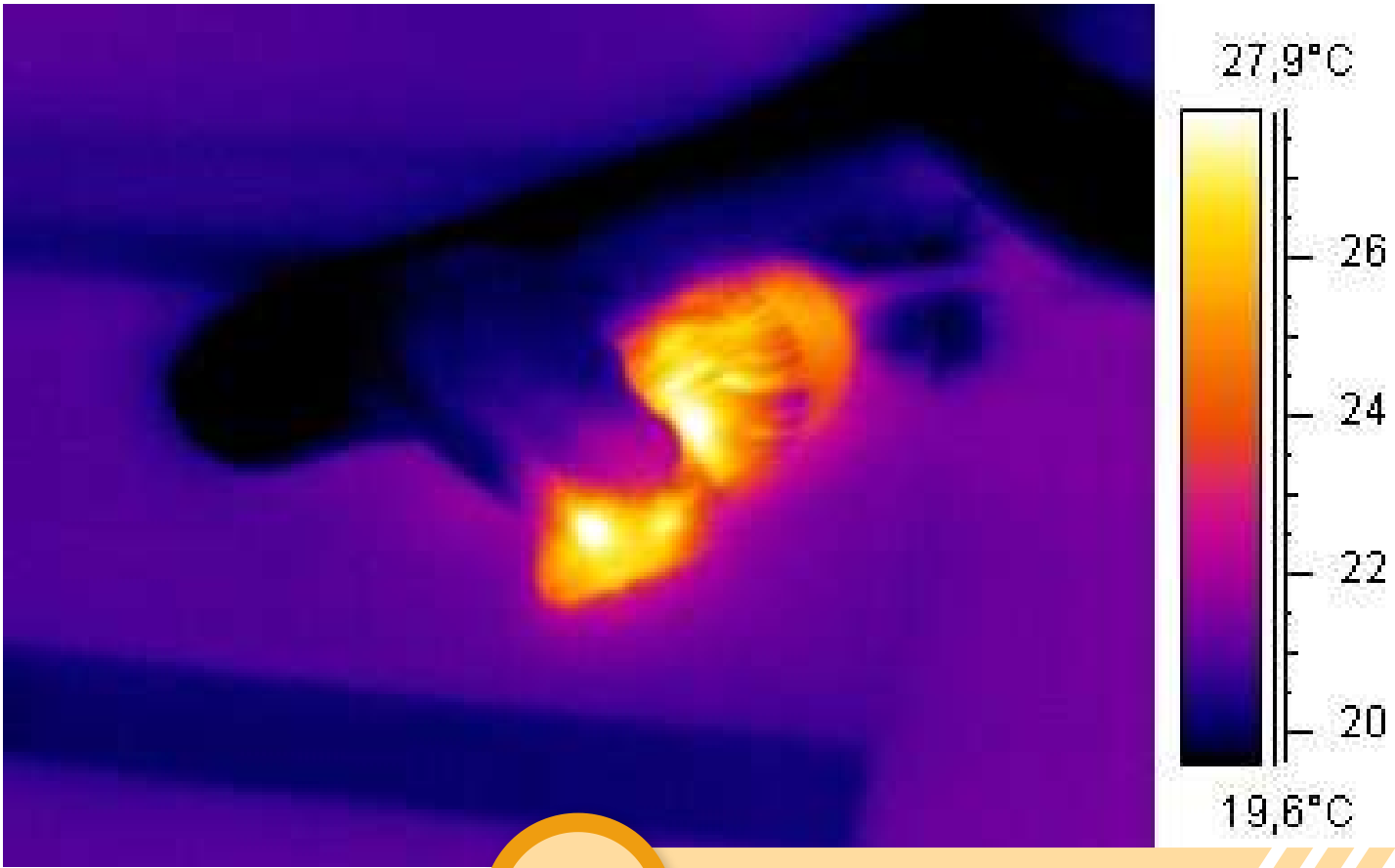
The snakes those are diurnals like – *Chrysopelea ornate* (Golden tree snake); *Ahaetulla nausta* (Vine snake) have a special feature of eyes to accommodate during the daytime i.e. the lens has inbuilt UV-filters just like a pair of high quality sunglasses & this lens filters out most of the lens & enables the snakes to see more sharper images.

Due to this alternation in their lens that allows the filtering of UV-lights & blue lights also makes the lens appear yellow, this is the reason why some snakes eye appear to have yellow.

During Nighttime

This kind of accommodation is for the nocturnal snakes i.e. those hunt at night. They do not have any such kind of UV-filters rather they do not need it at night. If will present in nocturnal snakes it would be rather a problem because in dark its already hard to get light in the eyes.

This night vision is made appropriate by the help of another specialized organ known as Pit organ. Example: *Agkistrodon contortrix* (Copperhead), *Boa constrictor*.



How snakes see by infrared vision by sensing temperature change

This pit organs actually creates infrared vision pattern for the snakes.

After comparing the pupil shapes, activity patterns, hunting time & styles and also their phylogeny of numerous snake species researchers have demonstrated that –

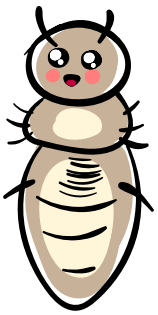
Pupils Type/Shape	Behaviour	Examples
Elliptical/vertical	Ambush hunters, better vision for moving in planes, usually nocturnal	Vipers, pit vipers such as – Copperhead (*Agkistrodon* sp.); Rattlesnake (*Crotalus* sp.)
Round	Actively foraging, usually diurnals	Family- Elapidae includes Cobras (*Naja* sp.); Mambas (*Dendroaspis* sp.)

Vertical eye of a viper snake

Round pupil of a cobra

References

1. Bullock, T. & Diecke, F. 1956. Properties of an infra-red receptor. J. Physiol. 134, 47–87.
2. Fang, J. 2010. Snake infrared detection unravelled. Nature, https://doi.org/10.1038/news.2010.122.
3. Goris, R. C. Infrared Organs of Snakes: An Integral Part of Vision. J. Herp. 45 : 2–14.
4. Hartline, P. H., Kass, L. & Loop, M. S. 1978. Merging of modalities in the optic tectum: infrared and visual integration in rattlesnakes. Science 199, 1225–1229.
5. Krochmal, A. R. & Bakken, G. S. 2003. Thermoregulation is the pits: use of thermal radiation for retreat site selection by rattlesnakes. J. Exp. Biol. 206 : 2539–2545.



Bark Lice: Tree Cattle of Mother Nature

Arghya Kamal Das, (2020-22), PG Dept. of Zoology, Barasat Govt. College

On a pleasant afternoon, I was roaming in the garden of my house and enjoying nature. Suddenly a red carpet on the trunk of a royal palm tree drew my attention. I became very curious about it and moved cautiously to get a detailed look. Despite my presence, they show no sign to fly. I got sufficient time to click some pictures of nature's wonder. From the pictures and study of morphology, I learned that the red carpet was a herd of Bark Lice belonging to the order Psocoptera.

A herd of Bark Lice on the trunk of a Royal Palm tree

Adult winged Bark Lice accompanying nymphs

Bark Lice can be identified by a prominent head, thread-like antennae, narrow neck, two pairs of wings in adults (Forewing larger than hindwing), and tarsi two or three-segmented. They are generally found in moist environments like leaf litter, under the bark, and beneath the stones. They are mainly dependent on algae, fungi, lichens and other plant products. Their size can be up to 10mm and grow wings during the adult stage. Some of the species of the order Psocoptera are gregarious.

The order Psocoptera consists of 2500 species under 230 genera belonging to 31 families worldwide. The Indian Zoogeographical region consists of 90 species under 40 genera and 16 families.

- Table: State-wise distribution and endemic species of Himalayan Psocoptera

S. No.	States	Family	Genera	Species	Endemic to India	Endemic to Himalaya
1.	Jammu and Kashmir	4	5	7	5	5
2.	Himachal Pradesh	0	0	0	0	0
3.	Uttarakhand	7	7	8	2	2
4.	Sikkim	0	0	0	0	0
5.	West Bengal	9	17	21	8	4
6.	Arunachal Pradesh	4	4	4	1	1

Data collected by Zoological Survey of India (Gurusamy et al., 2018)

Bark Lice are regarded as the most primitive living hemipteroids because they show the least modification from primitive mandibulate mouthparts. By observing the similarities in the structure of mouthparts (mainly hypopharynx), many entomologists believe in a close phylogenetic relationship between Bark Lice and Parasitic Lice.

Bark Lice moving in herd

A fascinating behaviour is exhibited by Bark Lice. When disturbed, they move in a mass like a herd of cattle or flock of sheep. They can be temporarily scattered, but they rejoin as a herd after some time. Because of this enchanting behaviour, they are called '**Tree cattle**' or '**Bark cattle**'.

The term 'lice' can mislead us as they are neither louse-like nor parasitic. A gardener should be happy by seeing the insects as they are beneficial. They are mainly scavengers as they consume an overabundant accumulation of algae, fungi, and dead bark. They do not damage a tree in any aspect (either by eating leaves or boring into bark). Moreover, they help to clean the tree bark and remove undesirable inhabitants.

Tree Cattle: scavengers of mother nature

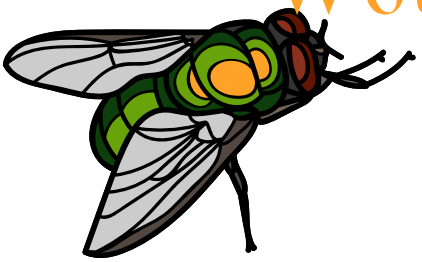
To keep mother nature tidy and clean we should preserve Bark Lice and should take no control measures against them.

Reference

1. Gurusamy, Ramesh & Babu, Rajappa & Subramanian, K.A. (2018). PSOCOPTERA: FAUNAL DIVERSITY OF INDIAN HIMALAYA.

Weblinks

1. http://indpaedia.com/ind/index.php/Psocoptera:_India
2. https://aggie-horticulture.tamu.edu/galveston/beneficials/beneficial-41_barklice2_Cerastipscus_sp.htm
3. https://projects.ncsu.edu/cals/course/ent425/library/compendium/psocoptera.html



Wound Feeding Behaviour Of *Cochliomyia hominivorax* (Diptera: Calliphoridae)

Ritom Mondal, (2020-22), PG Dept. of Zoology, Barasat Govt. College

Late in the afternoon, I was sitting in my backyard, exhausted by the heat of the day. When I was observing the wound on my knee suddenly a glossy bluish fly appeared around it. The fly was probably looking for an opportunity to reach the woozy wound spot, as I didn't make it happen. However, the fly may be starving and made its way to the wound. I simply allowed the fly to feed and watched until the end. The fly quickly fed upon the exudate, sucking up the whole fluid. Then I searched literatures for the incident and came across a fly named Cochliomyia hominivorax. It is a wound-feeding fly that lay eggs on the skin of vertebrates. The wound mass consisting of dead cells and tissue fluids are natural food of such fly. The problem is that it is a myasis-causing species and is harmful. As a preventive measure, I applied an antiseptic to the wound following a wash.

The screwworm fly feeding upon the exudate of human skin. [© R. Mondal].

The screwworm or *Cochliomyia hominivorax* are large greenish flies that lay shingle like layers of white eggs along the borders of open wounds (such as docking and castration sites), unclean skin, or abrasions. Within 24 hours, the eggs hatch. Larvae are obligate parasites of living tissue, and the cycle continues because the growing wound continues to attract the next generation of flies. Larvae eventually pupate in hot conditions, and hatch in three weeks. Malodor, enormous amounts of brown exudate, and necrosis are all symptoms of parasitized tissue developing large cavities. Individual animals or herds may be affected. Dressings and larvicidal treatments are used in the treatment. The host will succumb to subsequent infections and fluid loss if no action is taken. Current control methods include ivermectin injections under the skin and sterile male fly release programmes.

Reference

1. Donald B. Thomas, Robert L. Mangan, Oviposition and Wound-Visiting Behavior of the Screwworm Fly, *Cochliomyia hominivorax* (Diptera: Calliphoridae), Annals of the Entomological Society of America, Volume 82, Issue 4, 1 July 1989, Pages 526–534.
2. Margaret L. Delano, ..Wendy J. Underwood, in Laboratory Animal Medicine (Second Edition), 2002.

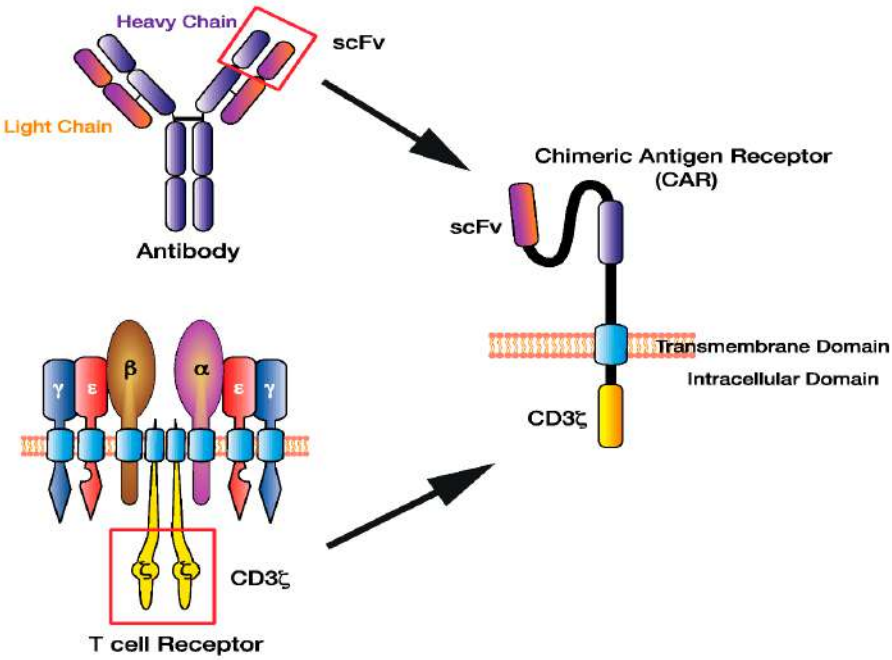
Car-T Cells: Bioengineered T Cells Acting as "Living Drug" in Cancer

Dhrubajyoti Bairagya, Ex Student (2017-20), UG Dept. of Zoology, Barasat Govt. College

Immunotherapy, the "fifth pillar" of cancer treatment is a type of therapy that boosts the body's natural defenses to fight cancer. It uses cells and molecules made by the body or in a laboratory in a semisynthetic way to improve how your immune system works to find and destroy cancer cells. **Chimeric antigen receptor-expressing T** (CAR-T) cells are examples of adoptive cellular immunotherapies (ACIs), where the engineered T cells, either autologous or allogenic, is transferred into the body where they will launch their effector function against the cancer cells.**CARs are the engineered hybrid molecule having antigen-binding property of monoclonal antibodies with the effector function of T cells and have several advantages over conventional T cells**.

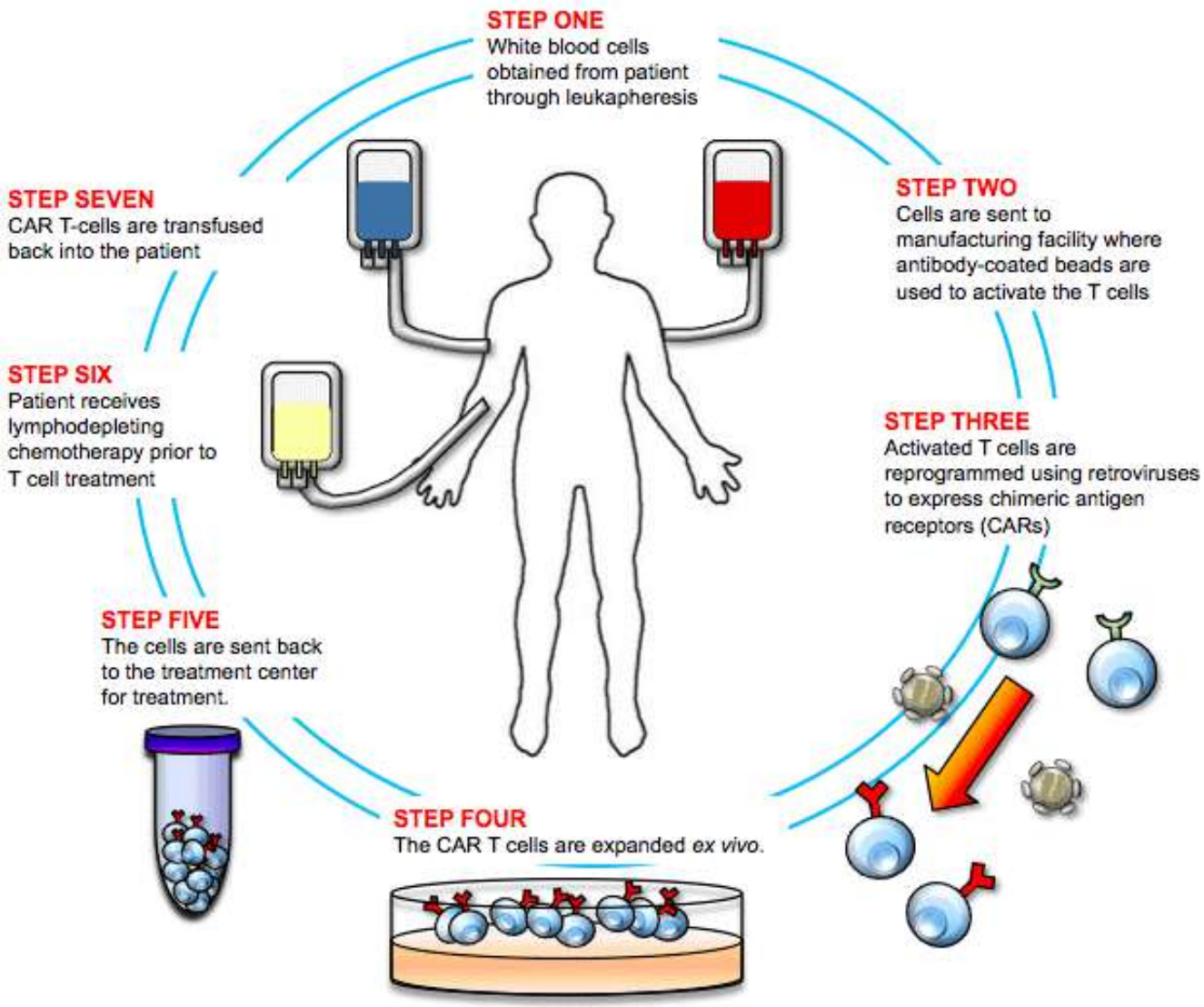
CAR T cells can be considered as "giving patients a living drug," explained Renier J. Brentjens, M.D., Ph.D., of Memorial Sloan Kettering Cancer Center in New York, an early leading person in the CAR T-cell field.

In Greek mythology, a chimera is a creature having the head of a lion, the body of a goat, and the tail of a snake. Similarly, chimeric immune receptors were first synthesized in the mid-1980s and initially consisted of the variable (antigen binding) regions of a monoclonal antibody and the constant regions of the T-cell receptor (TCR) α and β chains. In 1993, Eshhar et al. brought some modification to use an ectodomain derived from a single chain variable fragment (scFv), constructed from the antigen binding regions of both heavy and light chains of a monoclonal antibody, a transmembrane domain, and an endodomain with a signaling domain derived from CD3-ζ of T Cell Receptor (TCR) Complex.



The outline structure of a CAR, highlighting the major components of the extracellular domain, the transmembrane domain and the intracellular domain (endodomain).

Therapeutic Procedure



Summary of CAR T-Cell Therapy workflow: A patient's T cells are collected and harvested through leukapheresis, followed by T-cell activation on antibody-coated beads (act as artificial dendritic cells). The activated T cells are then transduced with a gamma-retrovirus or lentivirus encoding the chimeric antigen receptor (CAR). These T cells are known as CAR T cells, which then cultured, expanded and subjected to strict quality control testing prior to cryopreservation for transport of cells to the treatment facility. Before CAR T cell infusion, the patient receives chemotherapy for the depletion of native lymphocytes that can decrease efficacy of the infused cells.

Anti-CD3/CD28 antibody or anti-CD3/CD28 coated plates are in application to activate T cells after leukapheresis. It is to be noted that, anti-CD28 is needed to avoid T cell anergy. Anti-CD3/CD28 beads stimulated greater CD4+T cells growth rather than soluble anti-CD3, but both stimulated similar CD8 expansion. When anti-CD137 antibodies applied with anti-CD3/CD28 beads results in a significant increase in the expansion capacity for CD8 cytotoxic T cells.
Activated T cells are transduced to express the chimeric antigen receptor (CAR) with Gamma retrovirus or Lentivirus. However, active researches are going on with CRISPR-Cas9 technology to insert the CAR gene in T cell accurately resulting in optimum efficacy. Gene editing technology is also used to shut down the inhibitory molecules (like CTLA-4, PD-1) on T cell

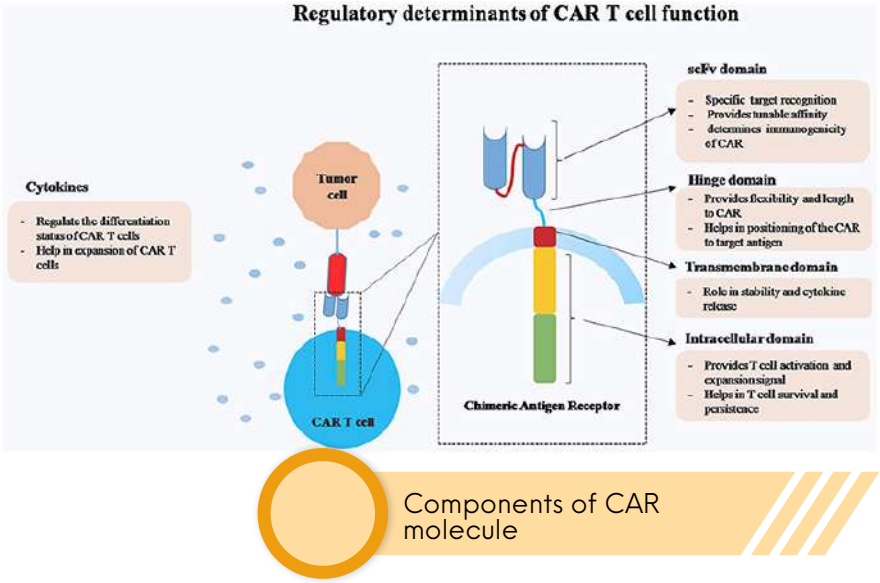
surface and also to shut off the T Cell Receptor (TCR) to rule out the possibility of graft-versus-host disease (GVHD).
In cases, patients exposed to variety of chemotherapeutic agent or immune exhaustion – quite often in any cancers, autologous T cells do provide less contribution in CAR-T cell therapy. To do away with this allogenic T cells are considered to generate a pool of CAR-T cells which further can be grouped and designed to target a set of tumor associated antigens (TAA).

CD4 & CD8 Car T Cells

It is generally thought that the efficacy of adoptive cell therapy is most often due to CD8 T cells, and infusion of CD8 CAR-T cells alone targeted against CD19 was sufficient for long-term eradication of B-cell malignancies. However, we cannot overlook the role of CD4 CAR-T cells. It is observed that in most of the clinical trials, patients receiving CAR T cells having a random composition of CD4 and CD8 cells, shows a significant variation in the efficacy. Clinical reports suggested a 1:1 ratio of CD4:CD8 CAR T cells provide a superior antitumor reactivity in vivo, indicating the synergistic antitumor effects of the two subsets.

Structure

CARs do have a very unique and precise design with four major components: 1. Antigen-binding domain, 2. Hinge and transmembrane domain and 4. Intracellular signaling domain. Each of these components has their respective, unique and particular function and optimal molecular design of the CAR can be achieved through many variations of the constituent protein domains.



Components of CAR molecule

Antigen Recognition & Binding Domains

The antigen-binding domain is the extracellular portion of the CAR that recognizes the target antigen (TAA) and encodes the specificity of CAR-expressing T cells accordingly. The unique structure of antigen-binding domains of CARs have been composed of the variable heavy (VH) and variable light (VL) chains of monoclonal antibodies, connected by a flexible linker to form a single-chain variable fragment (scFv). This scFv is binding component. This binding is MHC (Major Histocompatibility Complex) Class I and II independent unlike traditional or natural binding of TCR (T Cell Receptor). This MHC-independency is advantageous for targeting any cancerous antigens expressed on cancerous cells. Nevertheless, it gives us an opportunity to target non-protein components of antigens. It can also be targeted against the immunosuppressive secreted molecules for example, TGF-β, in case of solid tumors. However, active researches are going on to select a variety of target antigens, and linking more than one scFv, within the single CAR molecule with different antigen specificity to get rid of "Antigen Escape" which is a real challenge.

Hinge & Transmembrane Domains

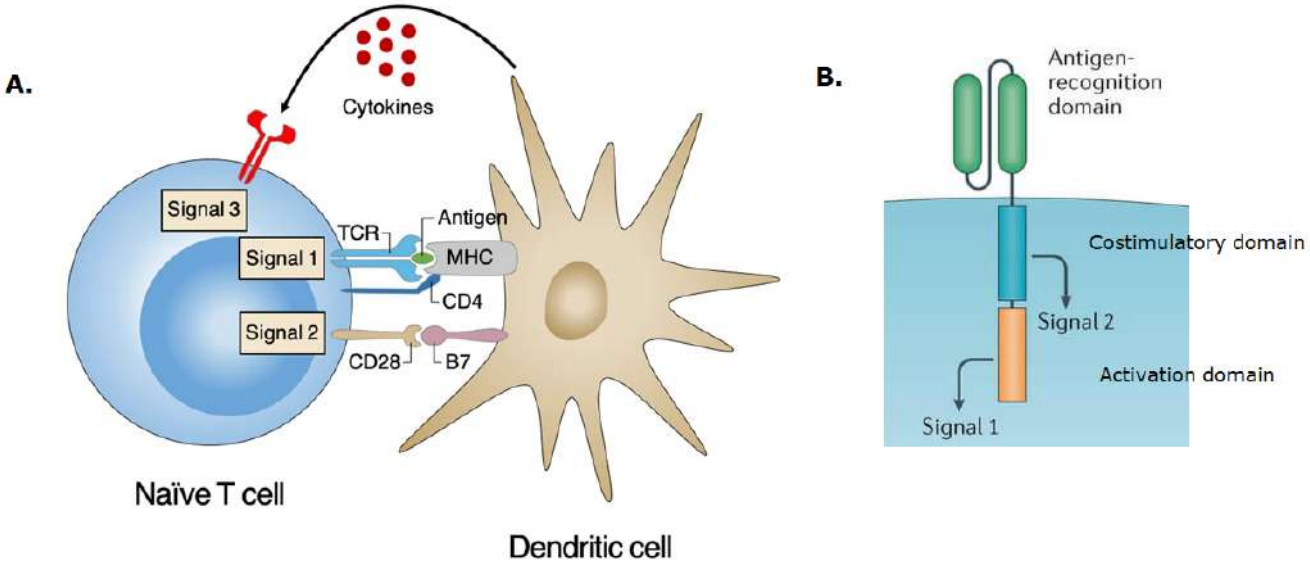
The hinge and transmembrane domains of CAR molecules makes a structural and functional connection with the extracellular antigen-binding domain to the intracellular signaling domains. The hinge portion enables flexibility to overcome steric hindrance and optimum length for accessing the target antigen. It is quite surprising to note that the length and composition of the hinge can affect antigen binding and signaling through the CAR.

The transmembrane domain anchors the CAR molecule in the T cell membrane and provides the required stability and function.

Intracellular Signaling Domains

The intracellular signaling domain, basically comprises an activation domain and one or more co-stimulatory domains (Together known as Endo-domain).

Upon binding with target antigens the binding signal is conveyed through the hinge and transmembrane domain and reaches the endo-domain. The endo-domain generate 2 basic signal required for T cell or rather CAR-T cell activation the SIGNAL 1 through activation domain and SIGNAL 2 through costimulatory domain. Researchers are now very enthusiastic about adding a portion in this endo-domain just to have the SIGNAL 3, that is the cytokine signaling to enhance the cellular immunity.



A. Conventional T cell activation
B. CAR-T cell activation. New generation CAR-T cells having domains inserted for the purpose of SIGNAL 3 (Not shown in this figure)

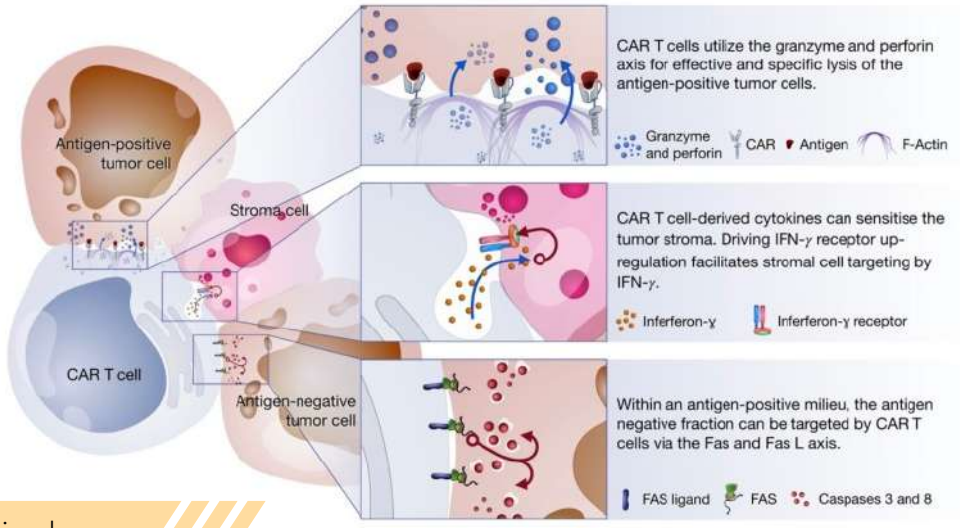
The CARs activate CAR T cells via CD3ζ-derived immunoreceptor tyrosine-based activation motifs (ITAMS), the main role player of T-cell activation. It is observed that, signaling mediated by these activation motifs alone is insufficient to generate an appropriate T cell responses and results in limited in vivo T cell persistence and activity; a co-stimulatory signal is also play a significant role for optimal T cell function, metabolism and persistence. The most thoroughly studied and investigated co-stimulatory domains are derived from CD28 or 4-1BB (CD137), and CAR T cell products utilizing either of these domains are FDA approved. CARs containing co-stimulatory domains along with activation domains produce IL-2, key to proliferate upon repeated antigen exposure. Again, it is the signal from costimulatory molecules which leads to the generation of memory CAR-T cells.

Extensive researches are going on in designing this endo-domain part leading to the production of many "GENERATIONS" of CAR-T cells.

Basically, what T cell does against MHC restricted peptide antigens with TCR Complex, Costimulatory and other cell adhesion molecules; CAR-T cells are designed to do it with just a CAR molecule in an MHC independent way against both protein and non-protein antigens.

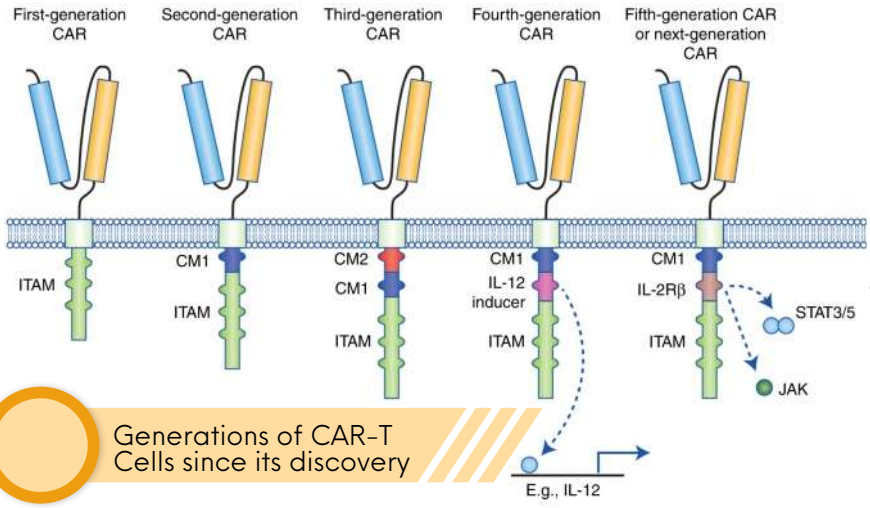
Effector Function of Car-T Cells

Upon engagement of scFv with target antigen, CAR T cells form a non-classical immune synapse (IS), required for their effector function. CAR-T cells then perform their anti-tumoral effects through the perforin and granzyme axis, the Fas and Fas ligand axis, and last but not the least with the release of cytokines to sensitize the tumor stroma.



Axes of tumor killing by CAR-T Cells.

Evolution of Car-T Cell Devellopment



Generations of CAR-T Cells since its discovery

First generation CARs contained only ITAM motifs in the intracellular domain. In Second-generation there is an inclusion of a single co-stimulatory domain, and in third-generation it contains two consecutive co-stimulatory domains. The fourth generation of CAR construction was based on second-generation CARs, containing 1–3 ITAMs paired with a chemokine (e.g. IL-12) inducer. These CAR-T cells are also known as T cell redirected for universal cytokine-mediated killing (TRUCKs). A fifth, or 'next generation' of CAR-T cells is having a current research interest and is being explored; these are also based on the second generation of CARs, but their uniqueness is a truncated cytoplasmic IL-2 receptor β-chain domain which is designed to activate the JAK-STAT pathway leading to better cell proliferation, prevents terminal differentiation, and shows better persistence.

The first-generation CARs were not yet clinically effective. Currently only second and third generation CAR-T cells has got clinical application. The fourth and fifth generations is still under active research.

Approved Car-T Cell Therapy

Most successful CAR-T cell therapy is approved for hematological malignancies particularly B-cell malignancy targeting CD19 as antigen. Presently, a few CAR-T therapies have been approved by the United States Food and Drug Administration (US FDA). Tisagenlecleucel (Kymriah) was approved by US FDA for the treatment of acute lymphoblastic leukemia (ALL) and large B-cell lymphomas. Axicabtagene ciloleucel (Yescarta) was approved by the US FDA for treatment of certain large B-cell lymphomas.

For solid tumors this therapy is facing some obstacles which are the part of active research.

Challenges

Though the idea of CAR-T cells enlightening us with a positive hope in immunotherapeutic approach but there are some notable challenges.

- ### Cytokine Release Syndrome (CRS)

CRS is the most common adverse reaction in CAR-T cell treatment. After infusion of CAR-T cells, a systemic inflammatory reaction caused by a rapid elevation of cytokines such as IL-1 and IL-6 was observed in a mouse model. CRS usually occurs within 2 days after CAR-T cell infusion, and get worsen within 1–2 weeks after CAR-T cell infusion. The pathophysiological process of CRS is not only due to activated CAR-T cells but also due to the active participation of monocytes, macrophages, and dendritic cells participate in synthesizing and release of cytokines, which is the major cause of the clinical symptoms.

Tocilizumab, an anti–IL-6 receptor monoclonal antibody, is used to prevent the adverse effect of CRS.

- ### On-Target–Off-Tumor Toxicity

The ideal and most desired target antigen is tumor-specific and expressed only on the surface of cancer cells. It is quite unfortunate that most of the antigens expressed in tumors are not specific to tumors. Most CAR-T cells target tumor-associated antigens (TAA), but this often leads to the possibility of mistargeting. As long as the target antigen is not 100% tumor-specific, there will be an off-target effect, which is the main source of CAR-T cell side effects.

Active investigation and targeting tumor specific antigen is the only way to get rid of this problem.

- ### Neurotoxicity

The incidence of neurotoxicity associated with CAR-T cells is approximately 40%. The symptoms include decreased consciousness, confusion, seizures, and brain edema. Mild clinical manifestations regress by itself but if get severe then it requires clinical management.

- ### Tumor Escape

If the tumor is not completely eradicated, surviving cells may lose the antigen being targeted by the CAR and the tumor may recur. One way of minimizing this problem is to introduce two CARs, specific for two tumor antigens, into T cells and transfer these cells into patients. Trials using this approach are ongoing.

- **Solid Tumors**

Major obstacle faced with this CAR-T cell therapy is in case of solid tumors. T cells entering the solid tumors may stop working due exhaustion induced by the immunosuppressive cells and molecules present in the tumor mass. The antigens of hematological tumors are often specific and not expressed in other normal tissues, while the antigens of solid tumors are generally expressed in small amounts in many other locations in the body, such as the heart, lung, and liver, resulting in mistargeted effects after treatment.

Last but not the least, the cost of CAR-T cell therapy itself is a big challenge. Treatment is excluded in a significant number of people who cannot afford the cost of this therapy. Novartis's CAR-T cell therapy, Kymriah, costs an average of ~$510,963, and Yescarta costs an average of approximately $402,647. With development of the production strategy and the implementation of a series of national medical policies, it is expected that an increasing number of patients will be benefitted.

In-spite of all challenges, CAR-T cell present us with a scope of taking the immunotherapeutic approach of cancer therapy to a full proof stage and modern researches in this area are enlightening us with new possibilities!

References

1. Design and Development of Therapies using Chimeric Antigen Receptor-Expressing T cells. Gianpietro Dotti, Stephen Gottschalk, Barbara Savoldo, and Malcolm K Brenner. Immunol Rev. 2014 Jan; 257(1): 10.1111/imr.12131.
2. Teaching an old dog new tricks: next-generation CAR T cells. Nicholas Tokarew, Justyna Ogonek, Stefan Endres, Michael von Bergwelt-Baildon & Sebastian Kobold. British Journal of Cancer volume 120, pages26–37 (2019)
3. Killing Mechanisms of Chimeric Antigen Receptor (CAR) T Cells. Mohamed-Reda Benmebarek,† Clara Helke Karches,† Bruno Loureiro Cadilha, Stefanie Lesch, Stefan Endres, and Sebastian Kobold* Published online 2019 Mar 14. doi: 10.3390/ijms20061283
4. Engineered T Cell Therapy for Cancer in the Clinic. Lijun Zhao and Yu J. Cao*. Front. Immunol., 11 October 2019 | https://doi.org/10.3389/fimmu.2019.02250

Weblinks

1. https://www.cancer.gov/about-cancer/treatment/research/car-t-cells
2. https://cellculturedish.com/fda-approves-first-car-t-cell-therapy-the-evolution-of-car-t-cell-therapy/

Image Sources

1. https://www.mdpi.com/1422-0067/21/1/204
2. https://cellculturedish.com/fda-approves-first-car-t-cell-therapy-the-evolution-of-car-t-cell-therapy/
3. https://www.frontiersin.org/articles/10.3389/fimmu.2018.03180/full
4. https://www.nature.com/articles/s12276-020-00486-7
5. https://www.cancer.gov/about-cancer/treatment/research/car-t-cells
6. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6470706/
7. https://www.nature.com/articles/s41416-018-0325-1

Technology to Monitor the Ecosystem

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Introduction

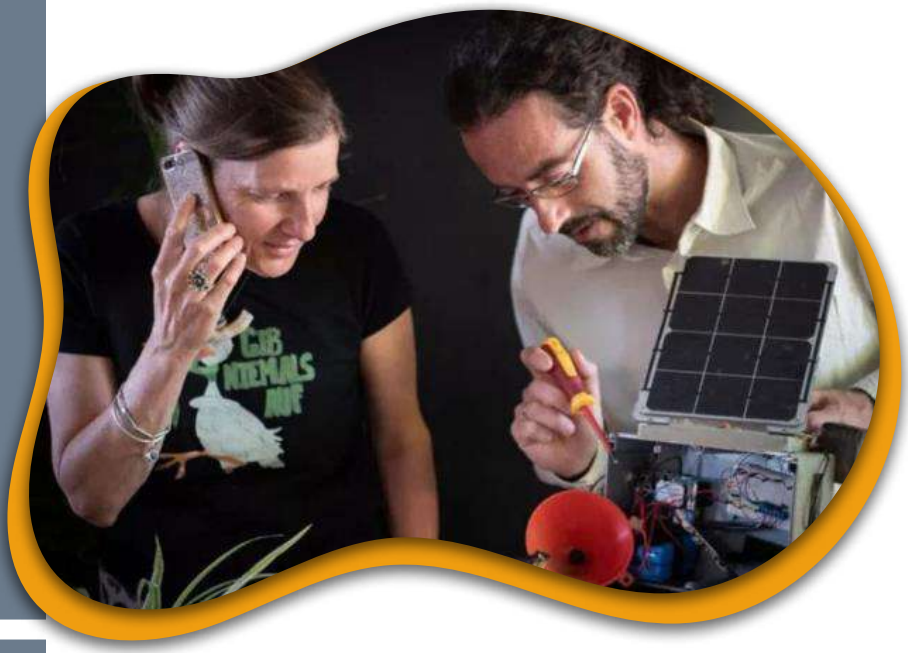
Technology is the new and comprehensive host of the nature of life. Every once in a while, a new technology, incl. an old problem to turn into a innovation. Such an outstanding invention which has made science, mainly zoological science to take giant lap towards progress are FROG PHONE and GPS. It not only helped the scientists in their survey but also securing their time and manpower.

1. What Is Frog Phone

The frog phone is the world's first solar powered remote survey device,that can be installed at any frog pond which recives a 3G or 4G cellular network.

Developers

The device has been developed at the University of New South Wales (UNSW) and the University of Canberra in collaboration with the Australian Capital Teritory (ACT) and Region Frogwatch program and the Australian National University.



How It Works

- The Frog Phone utilize 3G/4G cellular mobile data. It does not utilize direct satalite technology.
- Whenever ecologists need reading then they call to this frog phone.
- The frog phone accepts incoming calls independently after three seconds.
- These three seconds time allow to activate the temperature sensors pressure sensors, wind speed measuring sensors.
- Within three seconds it measures the temperature, pressure, wind-speed and the battery voltage. It also records crystal clear voice of the frogs which live there.
- All readings then get automatically texted to caller's phone.
- Relays environmental data to the observer via text messages which conducting real time acoustic surveys over the phone.

Benefits

- Normally researchers go to field in the night to collect data. It may disturb the habitat, also riskly, and costly too. But now the data can be collected directly without visiting the field.
- Its use will also minimize potential negative impacts of human presence at survey sites.
- This supports clear sound quality and minimal background noise, allowing users to identify the calls of different frog species.
- Its current microphone can detect calling frogs from a 100-150m radius.
- Allows scientists to call up a frog survey site and monitar them in the wild.
- The frog phone will help to drastically reduce the costs and risks involved in remote or high intensity surveys .
- The system has a large battery capacity coupled to a powerful solar panel.

Limitations

- Works only on 3G/4G technology, not directly from satellite signals.
- Needs sunlight and many devices to work and regular maintainance is needed.

2. Gps Wildlife Tracking

GPS Wildlife Tracking is a process whereby biologists, scientific researchers or conservation agencies can remotely observe relatively fine-scale movement or migratory patterns in a free ranging wild animal using the Global Positioning System and optional environmental sensors or automated data retrieval technologies such as Argos satellite uplink, mobile data telephony or GRPS and a range of analytical software tools.

Data Retrieval

- **Argos**

GPS tracking devices have been linked to an Argos Platform Transmitter Terminal (PTT) enabling them to transmit data via the Argos System, a scientific satellite system which has been in use since 1978. Users can download their data directly from Argos via telnet and process the raw data to extract their transmitted information.

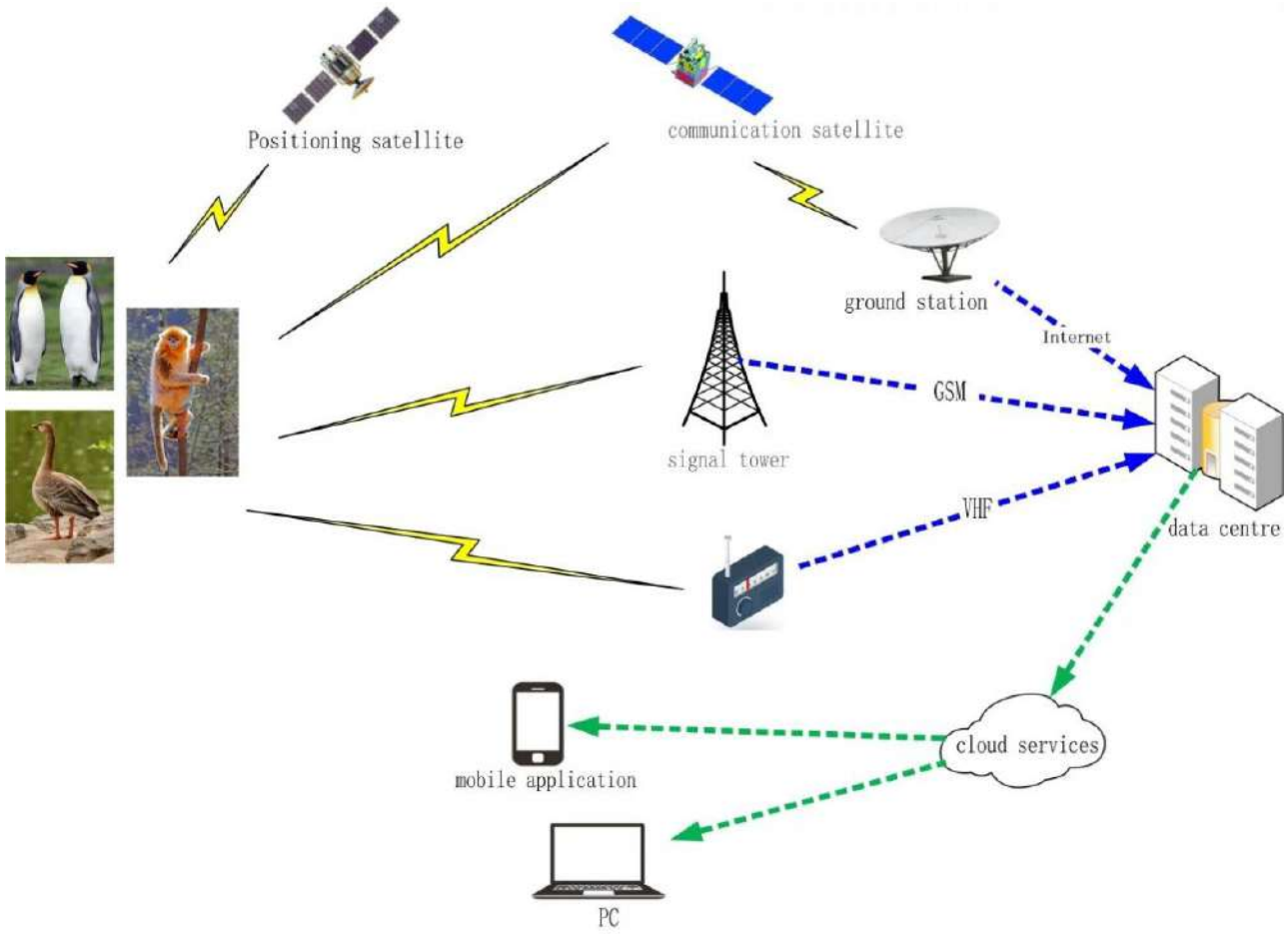
Where satellite uplink fails due to antenna damage, it may be possible to intercept the underpower transmission locally using a satellite uplink receiver.

- **GSM**

GPS location data can be transmitted via the GSM mobile/cell phone network, using SMS message or internet protocols over a GRPS session. The EPASTO GPS is dedicated to follow and locate cow.

- **UHF/VHF**

GPS data may be transmitted via short-range radio signals and decoded using a custom receiver.



Attachment

- **Collar Attachment**

Collar attachment is the primary attachment technique where the subject has a suitable body type and behaviour. Tracking collars would normally be used on the animal's neck (assuming the head has a larger circumference than the neck) but also on a limb, perhaps around an ankle. Suitable animals for neck attachment would include primates, large cats, some bears etc. Limb attachment would work well in animals such as kiwi, where the foot is much larger than the ankle.

- **Harness Attachment**

Harness attachment may used in situations where collar attachment is not suitable, such as animals whose neck diameter may exceed that of the head. Examples of this type of animal may include pigs, tasmanian devils etc. Large, long necked, birds such as the graylag goose may also need to be fitted with a harness to prevent removal of the tag by the subject.

- **Direct Attachment-**

Direct attachmentis used on animals where a collar cannot be used on animals where a collar cannot be used, such as birds, reptiles and marine mammals.

Benefits

Animal tracking data helps us understand how individuals and populations move within local areas, migrate across oceans and continents and evolve across generations. This information is being used to address environmental challenges such as climate and land use change, biodiversity loss, invasive species, wildlife tracking and the spread of infectious disease.

Complications

- **Effect on Animals**

A study was done with mantled howler monkeys to see if GPS ball and chain collars had any effect on the monkeys behavior. The study involved observing a group of collared and uncollared female howler monkeys. There was no major difference in the collared and uncollared behavior but when the study was over it was discovered that the monkeys had injuries. The collars had caused damages to the necks of the monkeys, one had small scratches and some swelling while four other monkeys had deep cuts from the collar. Two of the monkeys with the lacerations had their tissue healing over the collar.

References

1. Encyclopedia of General Science, Arihant
2. PT365
3. Environmental Technology & Innovation-Journals.
4. Technology And Innovation Management

Weblinks

1. www.wikipedia.com
2. www.thehindu.com
3. elsevier.com
4. https://indianexpress.com

Genetically Modified Organisms

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Introduction

A genetically modified organism (GMO) are living organisms whose genetic material has been artificially manipulated in lab using genetic engineering techniques.

GMO is what constitutes genetic engineering varies, with the most common organism altered in a way that does not occur naturally by mating or natural recombination.

A wide variety of organisms have been genetically modified like, animals, plants and microorganisms, genes have been transferred within the same species and even across kingdom.

History Of GMO

In 1973, Herbert Boyer and Stanley Cohen developed the first Genetically modified organism by cloning engineered DNA molecules in foreign cells (Zhang et al., 2016). The first Genetically Modified Crop, a tobacco plant, was reported in 1983 (Lemaux,2008). An antibiotic gene was transmitted in tobacco plant with the help of *Agrobacterium* (Bevan et al.,1983). The first genetically modified food, flavrsavr tomato, was marketed in 1994 (Bruening and Lyons, 2000).

Example of GMO

1. Transgenic Animals

- **GIFT**

Genetically improved farmed tilapia has an important role to play in increasing aquaculture production in developing countries. It is the most important product of the world first traditional selective breeding procedure for tropical fish as it grows more rapidly than indigenous tilapia strains.

Beside that it is an affordable source of proteins, vitamins, minerals, and essential fatty acids that are necessary for maintaining good human health.

- **Cloned Sheep**

In 1936 British scientists created the first cloned sheep, named Dolly, by transferring the nucleus from an adult cell into an unfertilized premature egg whose nucleus had been removed by a process called nuclear transfer. Sadly Dolly died of a lung diseases at the age of six.

Example of GManimals, are GMO salmon that contains a gene from the chinook salmon that makes it grow faster, super muscly pigs etc.

In medical research transgenic animals are used to identify the functions of specific factors in complex homeostatic system through over or under expression of a modified gene (the inserted Transgene).

2. Transgenic Microorganisms

GM bacteria were the first organisms to be modified in the laboratory, due to their simple genetics. These organisms are now used for several purpose and are particularly important in producing large amount of pure human proteins for use in medicine.

- **Human Insulin**

Recombinant human insulin has been produced predominantly using *Escherichia coli*. The human insulin producing gene insert into the plasmid. Researchers return the plasmid to the bacteria and put the 'Recombinant bacteria' in large fermentation tanks there, the recombinant bacteria use the gene to begin producing human insulin.

- GMM has been utilized to deliver numerous molecules for example, enzymes, organic acids and biofuels.
- In modern days genetically modified microorganisms (GMM) has been widely used in various industries like dairy, chemical pharmaceutical, biotech and agrochemical. In the western countries most of the foodstuffs such as bread, wine, cheese, butter, yogurts, fermented meats are produced by the action of GMM.

3.Transgenic Plants

Transgenic plants are the ones, whose DNA is modified using genetic engineering. The main advantages of transgenic plants include larger yield, resistance to diseases and pests and capable of growing under stressful conditions while their main disadvantages include allargic reaction emergence of super pests and loss of biodiversity.

- **BT Cotton**

That is resistant to a notorious insect pest (which contain cry gene). *Bacillus thuringiensis* is a bacterium that is pathogenic for a number of insect pests. Its lethal effect is mediated by a protein toxin it produces. Through recombinant DNA methods, the toxin gene can be introduced directly into the genome of the plant, where it is expressed and provides protection against insects pests of the plant.

- TVM Resistant Tobacco plants are produced by introducing viral coat proteins. Other viral resistant transgenic plants are -potato virus, resistant plants, RSV resistant rice etc.
- According to the manufacturers of these GM crops using these seeds will yield a number of benefits including increased yields and decreased costs. They push GM crops as a second 'Green Revolution' in a world with billion of hungry mouth to feed.

References

1. ESSENTIAL BIOLOGY, SANTRA, CLASS 12, NANDI;MIDYA;SANTRA

Weblinks

1. www.wikipedia.com
2. www.nongmoproject.org
3. www.britanica.com
4. www.nature.com

Butterfly Drinks Turtle's Tears

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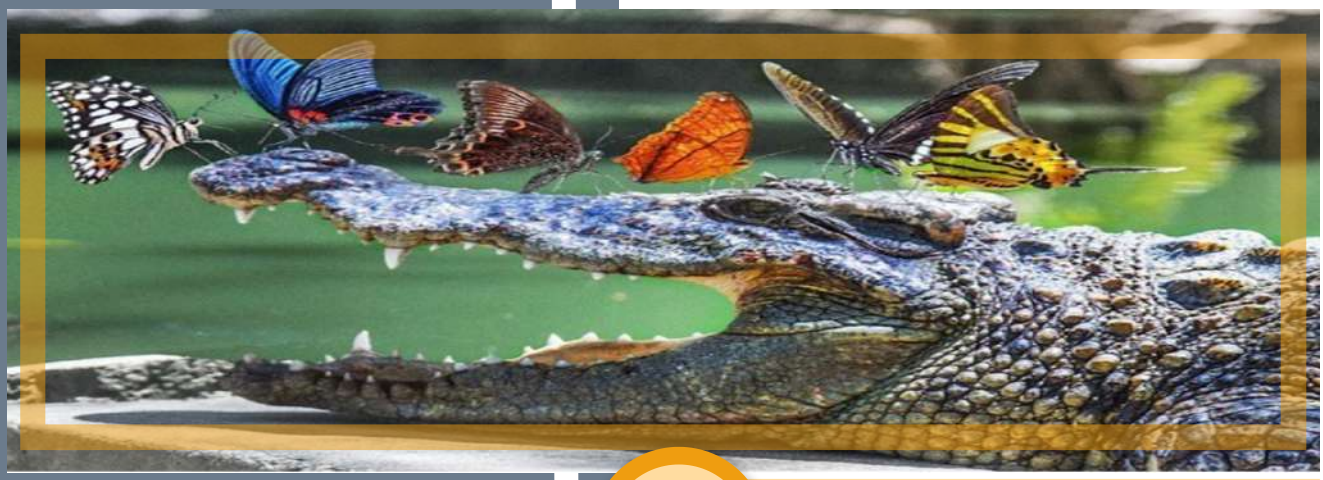
There are many phenomena in our nature. Among them an interesting phenomena is turtle's tears. The saline water turtle's have huge volume of salt (sodium chloride) in their body entering through their salty watermedium. So they are used to decrease the high volume of salt from their body by secreting sodium chloride through their eye. There are gland located in the corner of each eye, called lachrymal gland, to remove excess salt. As the reptilian's kidneys are unable to excrete large volume of salt via urine, through this way they can maintain the salt level of their body.

There are many types of minerals and sodium chloride in turtle's tears. Minerals are very essential for all living organisms. The turtle's tear helps the ovaries in female butterfly for maturation. So only the male butterflies who collect the turtle's tears and transfer to female ovaries during copulation.

Male butterflies are collecting turtle's tears from eye



Male butterflies are also collecting excess salt from saline water's crocodiles

The Most Unique Mating Tactics of Different Animals

Basusri Dandapat, 4th Semester, (2020-23), UG Dept. of Zoology, Barasat Govt. College

All living organisms want to keep behind on earth some of their part still alive when they don't exist. It can be done in different ways. The easiest way to do so is by transferring genes from one generation to generation. One of the main motives of all living beings as proved by the great history is to carry on life in the universe. To perform this huge responsibility, we get to see an amazing and unusual tactics adopted by the animal kingdom. Let's gain some knowledge about some of the out of the way mating tactics of different animals.

Bowerbird

Bowerbirds show a beautiful skill to attract their mates. They build extravagant structures called bower with twigs. They don't stop there, they decorate them with shiny objects, flower petals and other attracting objects. The floor of the bower is made of stones, shells etc. They present their interesting engineer skills to impress the female.

Praying Mantis

For praying mantis mating can be very dangerous leading to loss of life as they are eaten by the females. They are attracted to the females by pheromones. The praying mantis may not be able to mate before the female eats them up but some of them manage are able to escape. During mating season more than half of the female diet are made by the males. Females that eat males often lay more eggs.

Pufferfish

Pufferfish presents a unique way to attract females. They don't just sit wait for the female to arrive rather they spend days to create symmetrical patterns and structures in sand. These can reach up to 2m in diameter. If the female is impressed with the creative patterns made by the male, she lays her egg in the center of the circular structure.

Great Sage Grouse

Male sage grouse come in groups called lex to attract the females. Each spring they perform strutting display. They also produce popping noise by the feathers and whistles to win the females. They take in air and inflate their air sacs in their chests and the tail is fanned opened to make them look attractive. Often the one male dominating the whole group is chosen by the female.

Rainbow Peacock Spider

Rainbow spiders are very small creatures but they use bright colors on their body to woo their female partners and attract them. They also display great jumping skills. They use optical illusions to facilitate themselves in mating. The bright colors are produced by the scales present on their body that spilt the light into different colors giving beautiful array of colors, one of the nature's beauties.

Sometimes we get astonished by learning the brilliant and amazing way our nature is build. Every part of it fits so well possessing beautiful breathtaking features. We must always appreciate them and try our best to keep them safe.

References

1. "How peacock spiders make rainbow on their backsides" by Jason Daley, January 5, 2018.

Weblinks

1. https://inteng-storage.s3.amazonaws.com/images/DECEMBER/sizes/Peacock_Spider_md.jpg
2. https://bloximages.newyork1.vip.townnews.com/victoriaadvocate.com/content/tncms/assets/v3/editorial/7/35/735c93aa-f218-11ea-9749-d7d6b23f79d4/5f57f4c7c97f8.image.jpg?resize=1200%2C900
3. https://ucanr.edu/blogs/bugsquad/blogfiles/8765.jpg
4. https://i.pinimg.com/originals/88/de/2e/88de2eb62323185d4ddb365cbf363ac1.jpg
5. https://ewscripps.brightspotcdn.com/dims4/default/7ccbc96/2147483647/strip/true/crop/640x360+0+60/resize/1280x720!/quality/90/?url=http:%2F%2Fmedia.thedenverchannel.com%2Fphoto%2F2015%2F05%2F29%2Fgreater-sage-grouse_1432898474139_18971826_ver1.0_640_480.jpg

World Cancer Day

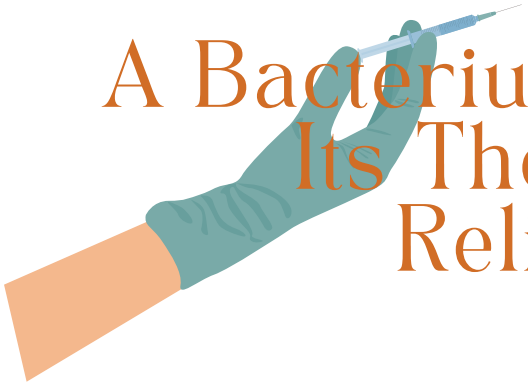
An Awareness Initiative by Dept. of Zoology, Barasat Govt. College

World Cancer Day is the one singular initiative under which the entire world can unite together in the fight against the global cancer epidemic. It takes place every year on 4 February.

World Cancer Day aims to prevent millions of deaths each year by raising awareness and education about cancer, and pressing governments and individuals across the world to take action against the disease.

World Cancer Day 2022 is observed on **February 4** which is on Friday, the main goal of World Cancer Day 2022 is to educate and encourage people about cancer disease prevention, early detection, and treatment.

Our Students of Department of Zoology, aimed to boost up the level of knowledge and of awareness about cancer by submitting short articles to know more about Cancer and awareness on February 4, 2022.



A Bacterium Toxin Botulinum and Its Therapeutic Effect as Pain Reliefer in Cancer Pateints

Arpita Samanta, (2020-22), PG Dept. of Zoology, Barasat Govt. College

We recently familiar to a term Botox Therapy, which is very much famous in film industries. It has not only a cosmetical site but also a therapeutic site. There are so many injections under Botox i.e. botulinum toxin, botulinum neurotoxin, abobotulinumtoxin A, rimabotulinumtoxin B incobotulinumtoxin A etc.

Botox is botulinum toxin, derived from a bacterium named *Clostridium botulinum*, which is a nonsurgical injection, used in cosmetic treatment but in therapeutic focuses Botox can be used in relieving pain. This type of injections are basically sub cutaneous or intramuscular type. In case of cancer patients, mainly patients with breast cancer (20-60%) and patient with neck and brain cancer (30%) experience chronic pain at the particular site of radiation or surgery (2). Normally this type of post-radiational or post-surgical pains are treated with many topical medicines containing hyaluronic acid, calendula officinalis, trolamine etc. but all these remedies give them a temporary relief.

Botulinum toxin or Botox can be used as pain relief treatment in cancer patients, who complete their chemotherapy. Botox can blocks signals in neuromuscular junction by preventing muscles from contraction and also introduction of BoNT-A can open a tumor vascular bed by inhibiting neurogenic tone in tumor cell line (1). BoNT can inhibit the release of acetylcholine at neuromuscular junction, which is the main hero behind the pain relief strategy due to muscle spasms. In case of neuropathic pain, the usage of botulinum neurotoxin inhibits the release of neurotransmitter both at sensory and peripheral level. Peripheral injection like botulinum toxinA is used close to peripheral nerve endings, which has an effect in reduction of calcitonin protein release from trigeminal ganglion. It has been also found in an experiment that BoNT can slow the growth and mitotic activity of cancer cell line and induces apoptosis.

There are 2 prominent advantages of using botulinum toxin are:

- Side effects of usage of this toxin (BoNT-A & BoNT-B) is very lower and safer in comparison to side effects of other potent analgesic agents (opoids).
- The duration of action of Botox injection at the site of radiation or surgery, lasts for almost 3-6months.

Many works about the methodology of BoNT functions are going on but from so many experiments it is clear that there is a neurological effect in reducing pain. So this botulinum toxin can use to get relief from pain after taking radiation in cancer patients.

References

1. Kitamura Y., Matsuka Y., Spigelman I., Ishihara Y., Yamamoto Y., Sonoyama W., Kamioka H., Yamashiro T., Kuboki T., Oguma K. botulinum toxin type a(150 kDa) decreases exaggerated neurotransmitter release from trigeminal ganglion neurons and relieves neuropathy behaviors induced by infraorbital nerve constriction. Neuroscience. 2009; 159: 1422-1429. Doi: 10.1016/j.neuroscience.2009.01.066.
2. Schreiber K.L., Kehlet H., Belfer I., Edwards R.R., Predicting, preventing and managing persistent pain after breast cancer surgery: The importance of psychosocial factors. Pain Manag. 2014; 4: 445-459. Doi: 10.2217/pmt.14.33.

Awareness of Cancer

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Cancer is very familiar term to us. In cell biology It is a type of cell transformation, where abnormal proliferation of cells occur, so a cell loses its ability to control the cell division. Our body is continuously managing the cell division, regulation process. But when the essential genes do not work properly, then the regulation breaks down. The uncontrolled growth of cells forms tumours. Thus the nature of the cell become cancerous, it spreads throughout the tissues via circulatory system,it is called METASTASIS.

The reason behind cancer is genetic because, In genetic alternation cases, the cancerous, defective gene flows into one generation to another. Thus it arises in the DNA of somatic cells and causes the genetic changes to produce malignant tumours, affecting the good, healthy tissues of the body.

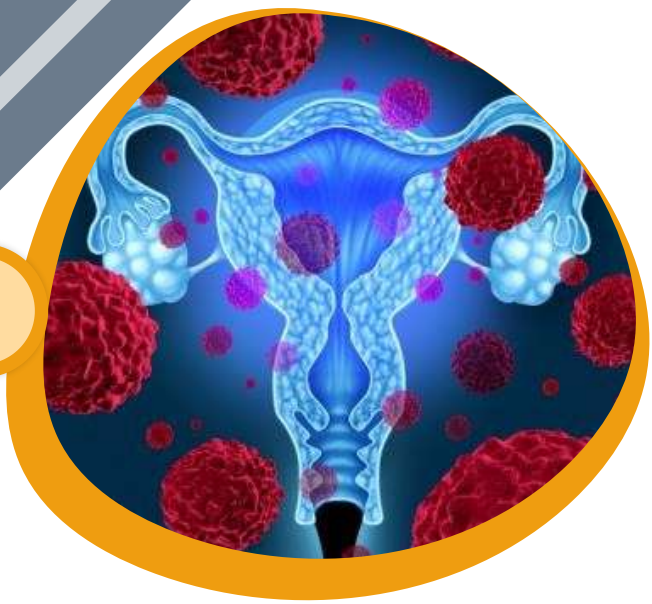
Depending upon the growth pattern, tumours is of two types: the benign tumour and the malignant tumour. Benign tumours are not very dangerous, it actually indicates the primary symptoms of cancer, like, common skin warts. The malignant tumours are serious one because it is capable to invade or spread within the body, as result, it causes the cancer. The gene related to cancer is called as ONCOGENE.

Generally it affects all over the body, it causes the cancer of breast, ovary, most common in women. Although, there are cancer of lung, brain, colon, kidney, prostate gland, bladder, blood etc.

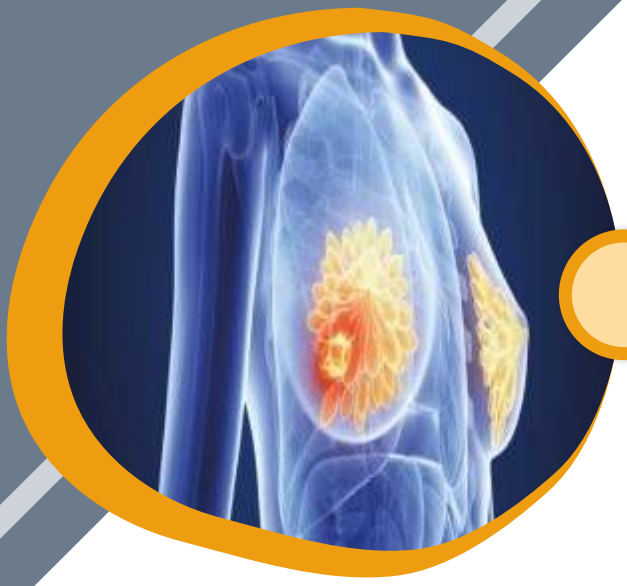
Treatments are of various types. Depending upon the stage of cancer, the medication and treatment is different. If a patient feels discomfort or any abnormality in their body, they should immediately appoint with doctor. In case of first stage, it can be curable. But for last stage, it is very difficult to save the life. In most of the cases, the diagnosis shows the last stage, that's why the death rate is very high.

As conclusion, the most important thing is the treatment, it should be within reach, so all kind of people can do their treatment. If we change in lifestyle and food habit, it can improve our health.

Lung Cancer



Ovarian Cancer



Breast Cancer

Brain Cancer

References

1. Zoology for Degree students, Author- Dr. V.K. Agarwal, 2018, 1st edition.

Image Sources

1.pinterest.com

World Wildlife Day

An Awareness Initiative by Dept. Of Zoology, Barasat Govt. College

WORLD WILDLIFE DAY
3 March

The animals and plants that live in the wild have an intrinsic value and contribute to the ecological, genetic, social, economic, scientific, educational, cultural, recreational and aesthetic aspects of human well-being and to sustainable development.

World Wildlife Day is an opportunity to celebrate the many beautiful and varied forms of wild fauna and flora and to raise awareness of the multitude of benefits that their conservation provides to people. At the same time, the Day reminds us of the urgent need to step up the fight against wildlife crime and human-induced reduction of species, which have wide-ranging economic, environmental and social impacts.

To celebrate World Wildlife Day, launched on March 3 by the United Nations in 1973 to help raise awareness about how people benefit from "fauna and flora," we asked our Knights and our faculty to share some pictures from their field work.

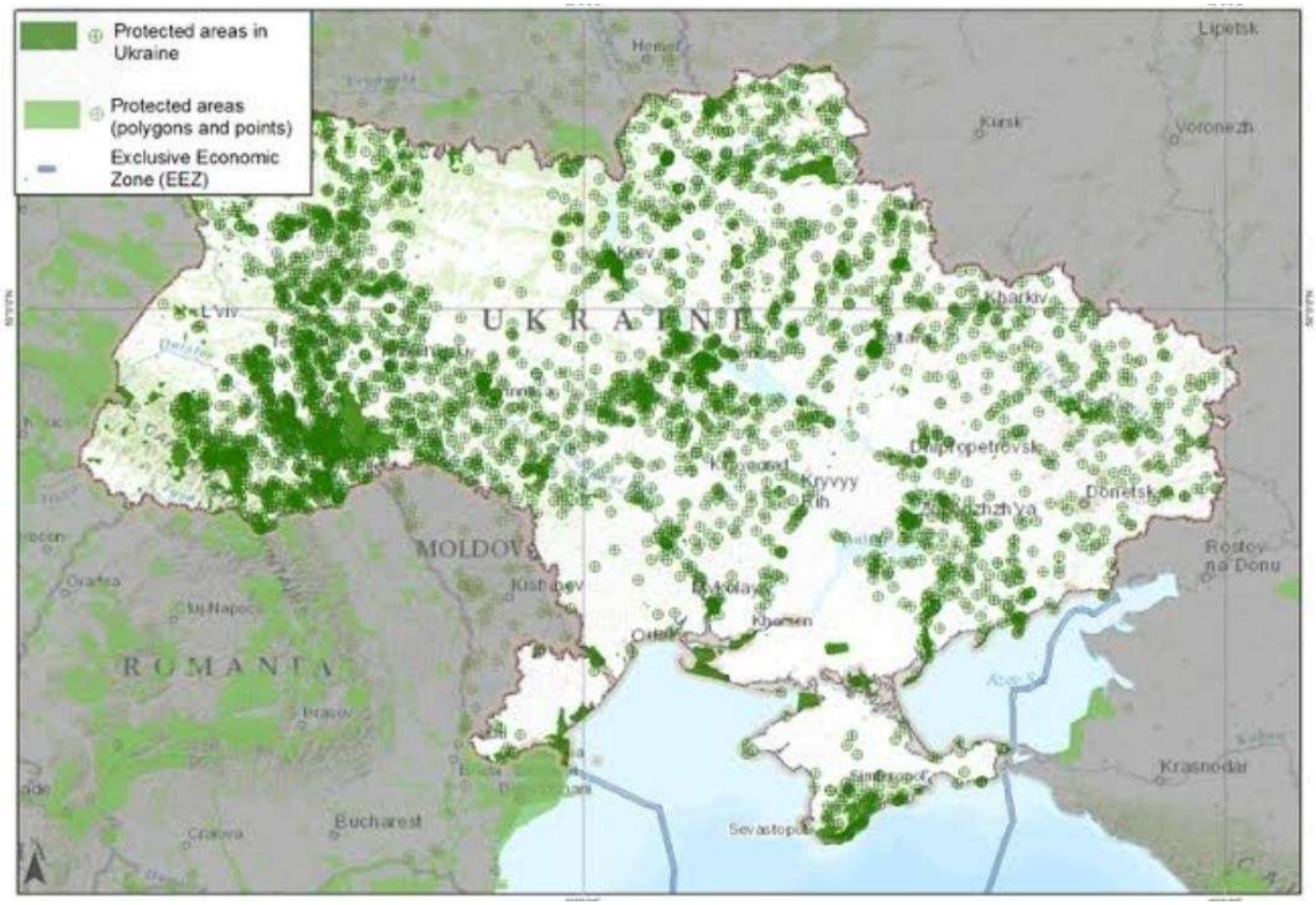
In 2022, the theme was 'Recovering key species for ecosystem restoration. 'The theme for 2022 seeks to raise awareness of the critically endangered species of flora and fauna in our ecosystems, with a view to generating and implementing workable solutions to conserve them.

Our Students of Department of Zoology, aimed to boost up the level of knowledge and of awareness about wild life by submitting short articles to know more about wild life and awareness on March 3, 2022.

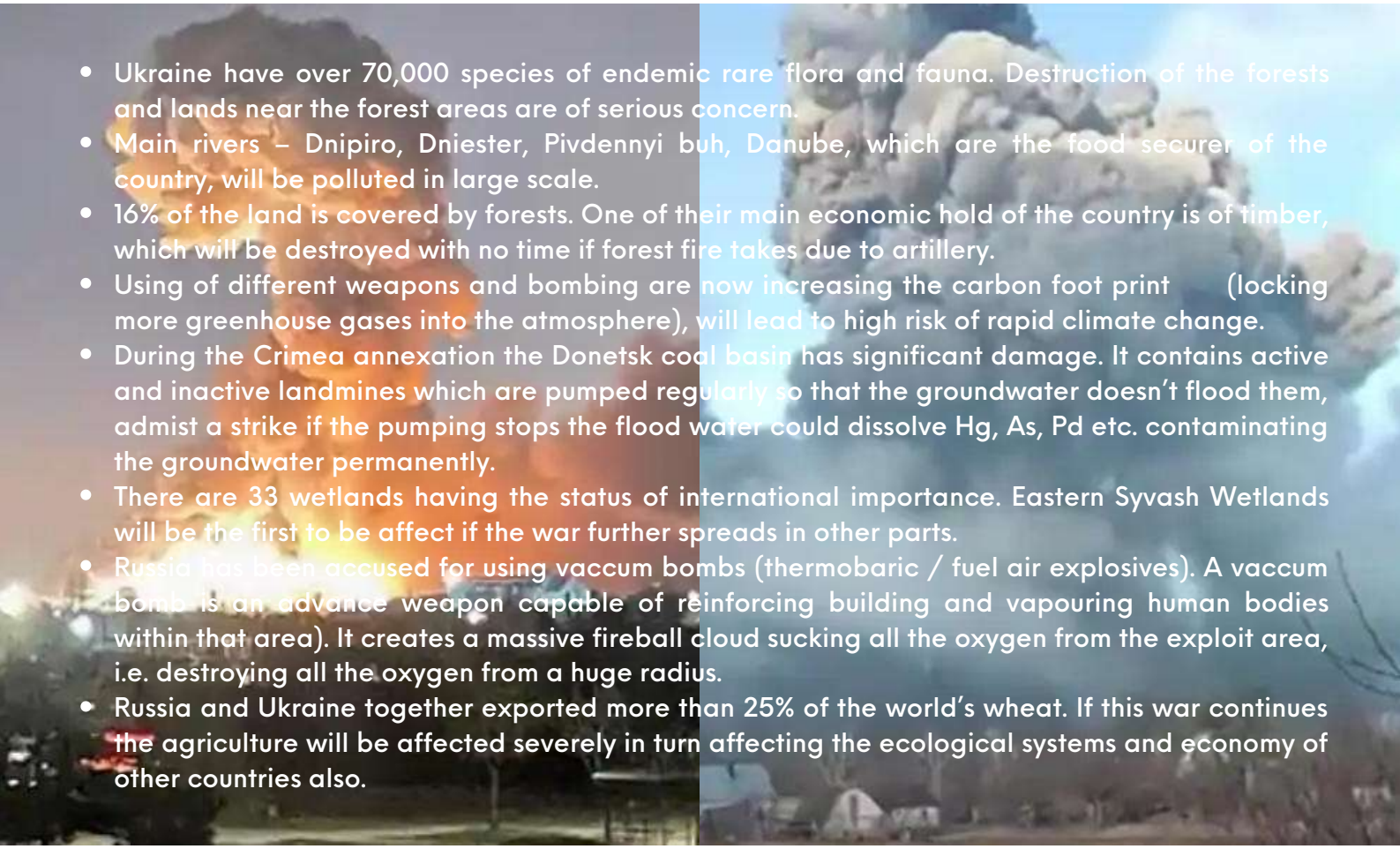
Impact of Russia-Ukraine Conflict on Ukraine's Biodiversity, Wildlife & Ecology

Deboleena Bandyopadhyay, (2020-22), PG Dept. of Zoology, Barasat Govt. College

Biodiversity of any country plays a pivotal role on its economic and social life. Despite having only less than 6% of Europe's land, Ukraine contributes 35% of its diversity. The recent war situation between Russia and Ukraine surely damages both country's economic structure but in case of Ukraine as we can see the damage is high and also in respect of ecology. So, obviously damage on Ukraine ecosystem is not only their national or local concern but it should be a global concern. The **Chernobyl disaster** on 26 April, 1986 was rated seven, the maximum severity—on the International Nuclear Event Scale where 134 staffs and fireman immediately admitted to hospital due to acute radiation syndrome out of them 28 died on the next month and 14 suspected radiation-induced cancer deaths followed within the next 10 years. Also during the time of Crimea annexation the Donetsk coal basin of Ukraine already faced significant damages. Now on 2022 continuous attack of Russian forces on the land of Ukraine not only started destroying cities, buildings and taking lives but also raising the risk of ecosystem disbalance in many paths.



Let us see what can be the probable impact, if this situation lasts long or further more severity rises on this geopolitical issue :



- Ukraine have over 70,000 species of endemic rare flora and fauna. Destruction of the forests and lands near the forest areas are of serious concern.
- Main rivers – Dnipiro, Dniester, Pivdennyi buh, Danube, which are the food securer of the country, will be polluted in large scale.
- 16% of the land is covered by forests. One of their main economic hold of the country is of timber, which will be destroyed with no time if forest fire takes due to artillery.
- Using of different weapons and bombing are now increasing the carbon foot print (locking more greenhouse gases into the atmosphere), will lead to high risk of rapid climate change.
- During the Crimea annexation the Donetsk coal basin has significant damage. It contains active and inactive landmines which are pumped regularly so that the groundwater doesn't flood them, admist a strike if the pumping stops the flood water could dissolve Hg, As, Pd etc. contaminating the groundwater permanently.
- There are 33 wetlands having the status of international importance. Eastern Syvash Wetlands will be the first to be affect if the war further spreads in other parts.
- Russia has been accused for using vaccum bombs (thermobaric / fuel air explosives). A vaccum bomb is an advance weapon capable of reinforcing building and vapouring human bodies within that area). It creates a massive fireball cloud sucking all the oxygen from the exploit area, i.e. destroying all the oxygen from a huge radius.
- Russia and Ukraine together exported more than 25% of the world's wheat. If this war continues the agriculture will be affected severely in turn affecting the ecological systems and economy of other countries also.

We, the humans are also an important part of these ecosystem. So, destroying human lives along with wildlife and nature's resources will effects the ecology for decades to come.

References

1. The Hindu, 3 March, 2022.
2. Fifth national report of Ukraine to the convention on biological diversity.
3. Ukraine Biodiversity Analysis (June 2017 This report was produced for review by the United States Agency for International Development. It was prepared by ECODIT LLC for the Ukraine Biodiversity Analysis (Assessment) Task Order No. AID-121-17-00002.)
4. Down to earth, 27 April, 2016; 25 February, 2022.
5. Green matters, 24 February, 2022.
6. The Weather Channel (The Times of India), 22 February, 2022.

World Wildlife Day

Basusri Dandapat, 4th Semester, (2020-23), UG Dept. of Zoology, Barasat Govt. College

World wildlife day is celebrated on 3rd March to acknowledge the international day for the adaptation of the CITES (Convention on International Trade in Endangered Species of Wild Fauna and Flora). This day recognizes the raised awareness worldwide about the flora and fauna.

The General Assembly talked put forward the pure and natural value of the wildlife. It also brightly lighten up the various ecological, genetic, cultural, social and studies related help they provided us. Most importantly they have a crucial role in human wellbeing. On this day we all the part of this amazing world get a chance to cherish more the world's biodiversity. United Nations also mentioned that " Though World wildlife Day is an annual celebration, wild conservation is an issue that needs attention and action every day."

Our world is residence of a great number of plants, trees, animals and flowers. It includes an amazing spectrum of diversity of different forms of lives. If we try to look at the margin of differences it includes it goes something like this which comprises of the largest flower of the world *Rafflesia arnoldii* which is locate in the rainforest of Indonesia to the smallest and simplest flower *Wolffia sp.* Belonging to the Lemnaceae family.

Our history is an example of huge display of impressive diversity of animal kingdom. Here the animal kingdom makes up 0.4% of global biomass. There are 1.05 million insects; 11000 birds, 11000 reptile species and mammal species are 6000 in number. The beauty of the majestic white tiger of India and also the colourful Mandarin fish make us blaffled, while the wise tactics adapted by the angelfishes in the marine environment and small little ants make us think about the creation of the almighty.

We must happily and responsibly response and take care to our environment to save our wildlife, to safe our mother earth.

The Crisis of Our Glorious Wild Life

Anushka Ghosh, 4th Semester, (2020-23), UG Dept. of Zoology, Barasat Govt. College

Wild life is the another name of life. It creates our environment and makes earth beautiful. There are many creations in wild life. The first life-forms appeared nearly 4 billion years ago. From a single eukaryotic cell, it has become an outstanding and mainly diversified group in earth. There are various habit-habitat, behaviours, life sustaining mechanisms are found in different group of animals. Members of kingdom animalia has adopted to various environments, from the bottom of the ocean to the highest mountain peaks.

Wildlife is very essential for so many of the important things in our lives. It is the main source that provides us natural systems on which we are dependent. Like, balanced climate, healthy water and food, medicines and many more. But day by day, our wildlife is in crisis for our own deeds. That is why our natural ecosystem loses its balance. Each year, **3rd March** is celebrated as **WORLD WILD LIFE DAY**, the day of signature of the Convention on International Trade in Endangered Species of Wild Fauna and Flora (CITES) in 1973.

Large mammals, small mammals, upland birds, waterfowl, wetland birds are categorized as wildlife. Due to the rapid growth of human population, the habitat, the life is in risk, to expand the industry, humans are continuously destroying their natural environment , taking over their beauty for industrial uses. Due to scarcity of food, habitat, some of specific species of both flora and fauna cannot survive on earth. The most common species which are considered as endangered are, **GIANT PANDA** (*Ailuropoda melanoleuca*), **ROYAL BENGAL TIGER** (*Panthera tigris*), **BLUE WHALE** (*Balaenoptera musculus*) **ASIAN ELEPHANT** (*Elephas maximus*), **GORILLA** (*Gorilla beringei* and *Gorilla gorilla*), **WHOOPING CRANE** (*Grus americana*), **SEA OTTUR** (*Enhyra lutris*), **SNOW LEOPARD** (*Panthera uncia*), **ORANGUTAN** (*Pongo pygmaeus*) etc.

Many national and international organizations like, **WORLD WILDLIFE FUND**, **Conservation International**, the **Wildlife Conservation Society**, are working for rescue the crisis of wildlife. They are working with the government to establish and protect public lands, like national parks and wildlife refuges. They are helping write legislation, such as the Endangered Species Act (ESA) of 1973 in the United States, to protect various species. They are working with law enforcement to prosecute wildlife crimes, like wildlife trafficking and illegal hunting (poaching). Also they are promoting biodiversity to support the growing human population while preserving existing species and habitats.

For our own requirement, we are rapidly breaking the web of life, which protects us and works like a natural barrier which gives a shelter against the catastrophy. Not only the earth, the total universe is in risk. As they are losing their shelter and food, they are entering in locality of human, that is why both wild and human populations are in danger. We have no right to destroy this beautiful universe, because they have evolved before us. We should protect and care for earth.

References

1. The Britannica Illustrated Science Library 2009 - (Malestrom) (BISL -07)

Weblinks

1. https://www.hindustantimes.com/lifestyle/travel/travel-to-save-the-wildlife-101646397373203.html
2. https://explore.panda.org/wildlife
3. https://www.nationalgeographic.org/encyclopedia/wildlife-conservation/
4. https://www.britannica.com/list/10-of-the-most-famous-endangered-species

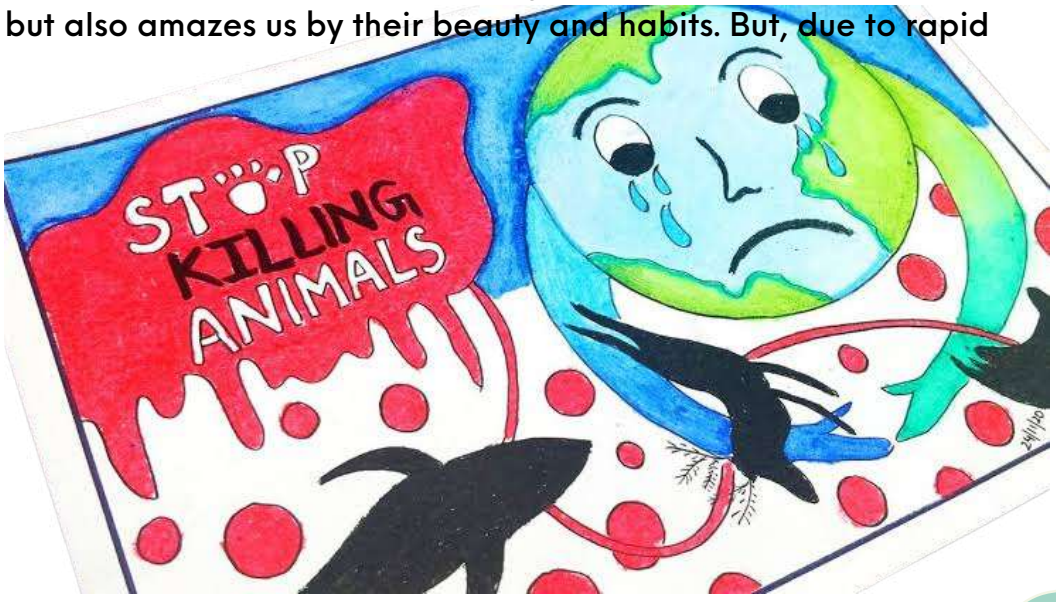
Image Sources

1. pinterest.com

Wildlife Protection & It's Importance to Humans & Impact of War on Wildlife

Praggaparamita Ray, 6th Semester, (2019-22), UG Dept. of Zoology, Barasat Govt. College

Wildlife, a term which is widely used to those animals living in forests and all the organisms dwelling in the forests. It is found in all ecosystems throughout the world, in plains, grasslands, rainforests, deserts, etc. even in rural and developed urban areas, all of which have their own distinct forms of wildlife. Wild animals are an integral part of our biodiversity. They are also one of the beautiful creations given to us by Mother Earth. They not only make us scared but also amazes us by their beauty and habits. But, due to rapid population growth, war, urbanisation and increase in food production has lead to decrease in forests as well as the wildlife. Today, on World Wildlife Day, we will discuss about the importance of wildlife to humans as well as it's protection.



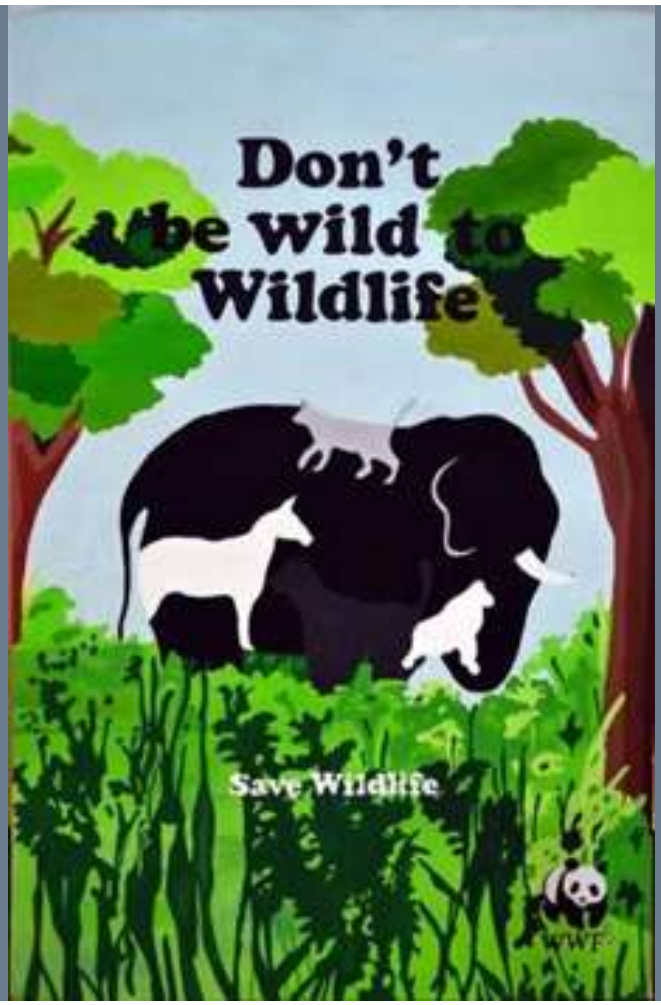
Wildlife Protection & It's Importance to Humans

The practice of conserving wildlife to prevent it from getting extinct or exploited is known as Wildlife conservation.

Wildlife conservation is very important as it helps us to restore, protect and enhance the natural ecosystems of the earth. Humans exploit wildlife and exercised poaching for their personal benefits. For this, many species have gone extinct or, are at the verge of extinction. Many birds and animals like vulture and lion, tigers, respectively are endangered now due to them being hunted for recreational purposes (and vultures getting extinct because of a drug named Diclofenac, which is used in cattle). We, humans don't have the power to re-create wildlife once it is destroyed, but we do have the power to preserve them not only for us, but also for our Mother Earth. Wildlife provides us with a lot of valuable things like food medicines, etc. There are still many animals and plants which are yet to be discovered for their various usages. Wildlife helps us in a lot of ways, which is quite indirectly, like, if forests are destroyed, the carnivorous animals inhabiting the forests have a high possibility of invading the human habitats, thereby killing them. Killing carnivorous animals will cause an increase in herbivorous animals which feeds on herbs and shrubs in the forests, thereby decreasing plants which, in turn, affects the vegetation of the forests and they come to our crop fields and eat our food crops. Wildlife can inspire people to lead a sustainable lifestyle. They can invoke feelings of sympathy and compassion which causes people to be conscious of damaging effects of their lifestyles. Wildlife are also important for their scenic beauty along with plants. There are several ways through which wildlife can be conserved by humans.

These are,

- Developing consciousness among people about wildlife, educating them about wildlife, it's importance to humans, like the way education about STDs, safe sex and population control are given.
- Educating people about extinct, vulnerable, endangered species through social media like youtube, twitter, facebook, whatsapp, instagram and through advertisements with a famous film celebrity preaching about importance of conservation and protection of wildlife. Films on wildlife can also help people to educate about wildlife.
- Encouraging people to protect animals and birds by educating them about the importance of particular birds and animals.
- Severe punishments for people who try to hunt endangered animals and birds.



Impact of War on Wildlife

Wars are the worst ways to destroy lives. Not only humans, but also wildlife are affected and destroyed by war. This is probably a topic, which is less discussed as how war affects wildlife because of political tensions which arose between Ukraine and Russia leading to war for past 7-8 days and people are more concerned about the people staying in Ukraine (especially the foreign nationals studying MBBS in Ukraine and how to return them to their countries safely) and other political matters concerning Russia, Ukraine and countries supporting them for war. Here are list of species which are at risk in Ukraine (and also endangered):-

Desmana moschata

Spalax arenarius

Falco cherrug

Isophya zubowskii

There are many more animals which probably got extinct in Ukraine due to war or may be at the verge of extinction. So, war in Ukraine is not only killing humans, but also killing the flora and the fauna of the country. So, on world wildlife day, we should all take an oath to protect wildlife and also to pray no more war happens because if there is war, there will always be loss of habitat, vegetation and wildlife.

Weblinks

1. https://therevelator.org/endangered-species-ukraine/
2. https://www.wikiwand.com/en/Wildlife_of_Ukraine

World Down Syndrome Day

An Awareness Initiative by Dept. of Zoology, Barasat Govt. College

World Down Syndrome Day (WDSD) is celebrated annually on the 21st of March and is a global awareness day that has been officially observed by the United Nations since 2012.

The date for WDSD is set on the 21st day of the 3rd month as a representation of the extra copy of the triplication (trisomy) of the 21st chromosome that people with Down syndrome are born with.

On this day, people with Down syndrome and those who live and work with them throughout the world organize and participate in activities and events to raise public awareness and create a single global voice advocating for the rights, inclusion and well-being of people with Down syndrome.

Our Students of Department of Zoology, aimed to boost up the level of knowledge and of awareness about World Down Syndrome Day (WDSD) by submitting short articles to know more about Down Syndrome.

Genetics Behind Down Syndrome

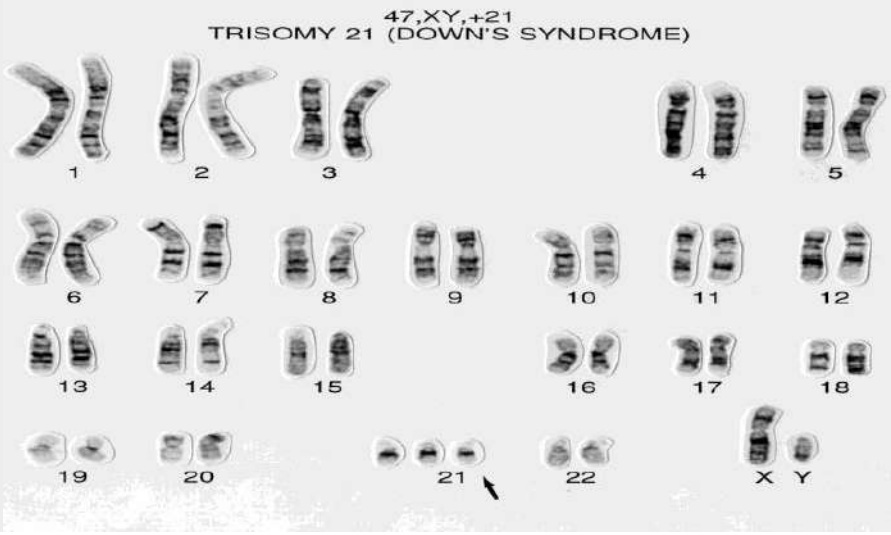
Anushka Ghosh, 4th Semester, (2020-23), UG Dept. of Zoology,
Barasat Govt. College

Down syndrome is also referred as **TRISOMY 21**. It is a genetic disorder, that can't be cured. Specifically disorder of **chromosome 21**, due to development of extra genetic material. People with down syndrome generally looks like **MONGOLIANS**, so also reffered as **MONGOLISM** or **MONGOLOID IDIOCY**, includes flattened face, Mongolian types of Epicanthal fold on eyelid, short heighted, small ears, small neck and many more abnormalities.

The followings are the causes of down syndrome:

Trisomy 21

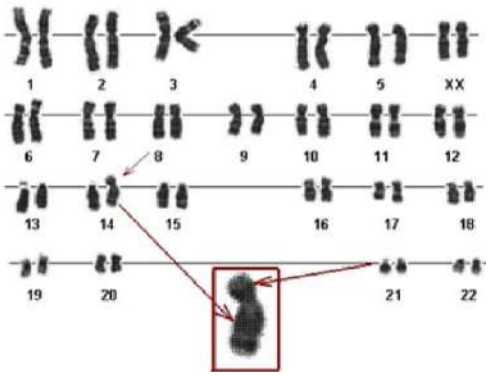
The main reason behind down syndrome is the trisomy (2n+1) of chromosome 21, that is why, there is an extra chromosome 21 occurs along with normal diploid (2n) chromosome. It means there are three copies of chromosome 21 instead of normal two. People with down syndrome having 47 chromosomes in their cells instead of 46. The karyotype of down syndrome is 46, XX+21 or 46, XY+21. About 95 percent of the time, the reason is trisomy.



To create the trisomy condition of chromosome 21, the **NON-DISJUNCTION** of chromosome 21 occurs during the development of normal gamet with normal chromosome. The **NON-DISJUNCTION** occurs during **MEIOSIS I** or **MEIOSIS II** cell division, so, the one gamet contain two chromosome 21 and another gamet contain no chromosome 21.

Now the gamet with abnormal and exceptional two chromosome 21 fertilizes with the gamet of normal chromosome 21, resulting three copies of chromosome 21, this is called as TRISOMY of chromosome 21. The result is the birth of baby with down syndrome.

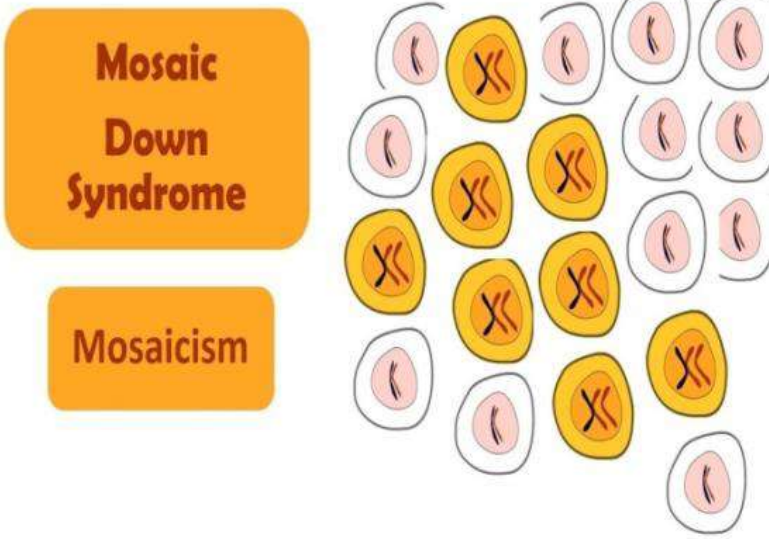
Robertsonian Translocation



In this rare case, the chromosome 21 attaches with chromosome 14 through translocation. Now during the development of gamet, one 14-21 chromosome and one free chromosome 21 are unitedly included into single gamet. Due to the fertilization of this abnormal gamet with normal one, three copies of chromosome 21 will occur in that zygote and from that zygote, the down baby will born. This type of down syndrome often occurs in family, that is why it is known as FAMILIAL DOWN SYNDROME. About 4 percent of the cases are this type.

Mosaic Down Syndrome

In this form of down syndrome, a person has only some cells with an extra copy of chromosome 21. This mosaic of normal and abnormal cells is caused by abnormal cell division after fertilization. Mosaicism is the rare and least common form of down syndrome and accounts for only about 1 percent of all cases of down syndrome.



Weblinks

1. https://en.wikipedia.org/wiki/Down_syndrome

Image Sources

1. cdc.gov
2. en.wikipedia.org

How to Treat With Down Syndrome Patient

Tista Das, 4th Semester, (2020-23), UG Dept. of Zoology, Barasat Govt. College

Down syndrome is a genetic disorder causing developmental and intellectual delays. People with Down syndrome are in a great risk for a number of health problems. We can identify such patients with many symptoms like flattened face, short neck, almond shaped eyes that slant up, small hands and feet, tiny white spots on the Irish of the eye etc. Down syndrome cannot be cured. Early treatment programmes like speech, physical, occupational, educational therapy and mental support is the essential ways that help them to lead a happy, productive life.

- **Physical Therapy** includes activities and exercises that help to build up skills, increase muscle strength. A physical therapist might help a child to establish an efficient walking pattern.
- **Speech Language Therapy** can help children with Down syndrome improve their communication skills and use language effectively before they can speak. A speech therapist can help children to learn many other ways of communication such as sign languages and pictures etc.
- **Occupational Therapy** helps find ways to maintain daily tasks and conditions to match a person's need and ability. This type of therapy teaches self dependable skills such as eating, getting dressed, writing and using a computer. At the high school level, an occupational therapist could help teenagers to identify jobs, careers, or skills that match their personalities.
- **Emotional & Behavioral Therapies** work to find useful responses to both pleasant and unpleasent behaviors.

Weblinks

1. https://www.nichd.nih.gov/health/topics/down/conditioninfo/treatments

Image Sources

1. pinterest.com

Down Syndrome: Population & Age

Meghna Das, 4th Semester, (2020-23), UG Dept. of Zoology, Barasat Govt. College

World Population for Down Syndrome

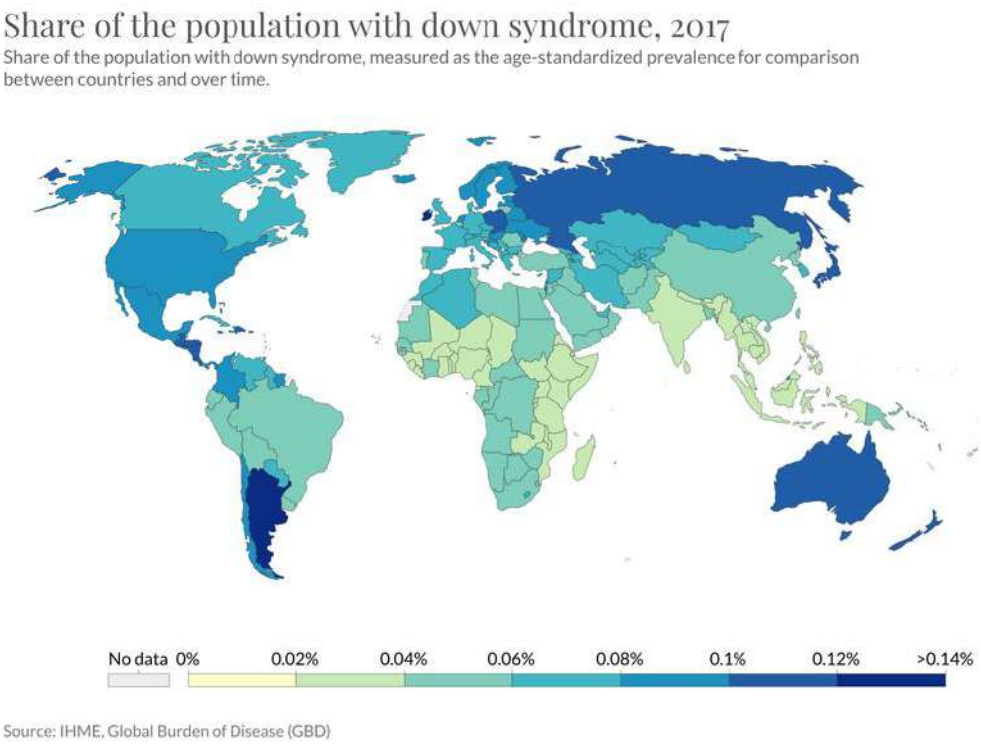
Down syndrome is said to be occurred when there is an extra copy of 21st chromosome during pregnancy. This leads to some distinctive signs and symptoms such as recognizable facial features in addition to developmental & intellectual difficulties. Though Down syndrome is the most commonly occurring genetic disorder, the way the condition presents itself in each person will differ.

The estimated occurrence of Down syndrome is between 1 in 1,000 to 1 in 1,100 babies worldwide, according to the World Health Organization. Down syndrome is the most common chromosomal disorder in USA. About 6,000 babies are born with Down syndrome each year.

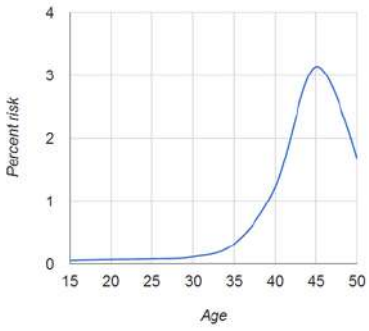
Between 1979 and 2003, the number of babies with Down syndrome has increased by 30%. Older mothers are more likely to have a baby affected by Down syndrome than younger mothers. In other words, the occurrence of Down syndrome increases as the mother's age increases. For estimation of the occurrence of Down syndrome, the number of pregnancies affected by Down syndrome is compared to the total number of live births.

West Bengal:

in the Genetics Department of West Bengal there 85 cases which are diagnosed as Down syndrome. Ramakrishna Mission Seva Pratishthan, Kolkata, India has taken control of 30 individual. More than 90% of Down syndrome patients have flat faces, low life span, and abnormal eye problems. 56.5% patients of such conditions suffer from Congenital Heart Diseases and 41.2% of them suffer from jaundice at birth. These factors are significantly low by the help of Health Drive.

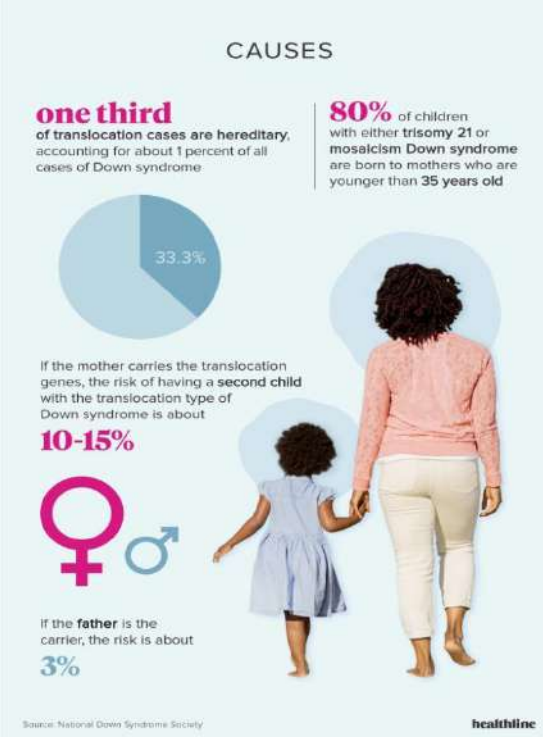


Maternal Age Factor in Down Syndrome



Almost 80% of the newborns are born with trisomy 21 or mosaicism Down syndrome to the younger mothers below the age of 35. Younger mothers tend to have babies more frequently so the number of babies with Down syndrome increases subsequently. However mothers of age more than 35 have the possibility to have a baby with this condition is naturally more.

According to the National Down Syndrome Society, a 35 year old mom has a chance of having a child with Down syndrome is 1 in 350. This chance gradually increases to 1in 100 at the age of 40 an approximately 1 in 30 by the age of 45 and above. About one-third of total translocation cases are hereditary accounting for about 1% of all the cases of Down syndrome. Both parents can be carriers of the translocation genes without showing any symptoms of Down syndrome in their body. Mothers who have a child with Down syndrome have an increased chance of having another baby with same condition. When a woman has a child with Down syndrome, than the risk of having another child with Down syndrome is about 1 in 100 up to the age of 40.



The risk of having a second child with the translocation gene of down syndrome is about 10 to 15% if the mother carries the genes. But is about 3% if the father carries the gene.

Weblinks

1. https://www.researchgate.net
2. https://journaliijcar.org
3. https://www.cdc.gov
4. https://www.healthline.com

Just One Extra Chromosome – Makes One Extraordinary Child

Basusri Dandapat, 4th Semester, (2020-23), UG Dept. of Zoology, Barasat Govt. College

All the living beings are are made of cells- basic functional and structural unit of life. Each cell contains several organelles among which the most important one is the NUCLEUS. We all know the nucleus contains the genetic material in the form of genes which are codes for replicating the cell and to carryout functions of a cell properly. These are responsible for our inherited traits and are arranged in structures called chromosomes. Human cell contains 23 pairs of chromosomes or 46 chromosomes.

Down syndrome is a consequence of genetic variation. The full or partial extra copy of chromosome 21 in an individual results in down syndrome. This extra amount of genetic material varies the developmental process and it is associated with certain traits.

Discovery

British physician, John Langdon Down in 1866 first described Down syndrome and called it “Mongolism”. Afterwards it came to known as Down syndrome after him. The term came to use during the early 1970s. In 1959 French Pediatrician/Geneticist Professor Jerome Lejeune discovered that individuals with Down syndrome have an extra chromosome. Very soon chromosome studies were developed to know about the proper diagnosis of this syndrome.

Symptoms

The symptoms include intellectual disability and characteristic facial profile. Early symptoms are:

- Loss of interest in being sociable, conversing or expressing thoughts and declined ability to pay attention.
- Sadness, fearfulness or anxiety, irritability, uncooperativeness or aggression.
- Restlessness or sleep disturbances. Unable to coordinate.
- Seizures occurring in adulthood.

Characteristic Traits

The characteristic traits of this syndrome are:

- Small sized skull
- Upward slant of eyes and epicanthic folds
- Small nose with the flat nasal bridge
- Mouth has a narrow short palate with small teeth and furrowed protruding tongue
- Single crease on the hand (simian crease) at birt
- Delayed development and behavioural problems & Cognitive disability

Other complications like heart defects, vision problems, gut problems etc. are also associated.

The only prevention can be prenatal diagnosis. The families with members suffering from this syndrome must seek consultation from mental health professionals. The child or adult must be treated with care and love.

Weblinks

1. https://i1.wp.com/sport4you.net/wp-content/uploads/2021/07/boy-in-green-shirt-with-downs-syndrome-1.jpg?w=675&ssl=1
2. https://www.ptckids.com/wp-content/uploads/2019/02/down_syndrome.jpg

Students' Academic Achievement & Progression

Batch	Name of Student	Academic Achievement
2012-14 PG student	Priya Prasad	Senior Research Fellow, pursuing PhD in ZSI
2012-14 PG student	Shubhajit Saha	Joined as Assistant Professor in Sundarban Hazi Desarat College, South 24 Parganas
2013-15 PG student	Samriddhi Sen	Qualified NET LS in 2019; GATE XL 2021 (AIR 2067), pursuing PhD in Sambalpur University, Orissa
2014-16 PG student	Deb Shankha Goswami	Joined as JRF in Wild life institute of India, Dehradun in 2018; Qualified GATE XL 2021 (AIR 2460)
2014-16 PG student	Anirban Chakraborty	Qualified NET UGC JRF, pursuing PhD in Dept. of Zoology, University of Calcutta
2014-16 PG student	Himadri Nath	Qualified NET JRF, CSIR-UGC and admitted in PhD program in IICB, Kolkata
2014-16 PG student	Shahina Parveen	Assistant Teacher; Qualified 22nd SET, 2020; Awarded DBT BET JRF Category – I 2021
2015-17 PG student	Jayita Ghosh	Assistant Primary Teacher, Indian Girl Primary School in 2020
2015-17 PG student	Antara Sarkar	Qualified NET UGC CSIR JRF (RANK 137); Joined as JRF in University of Calcutta
2015-17 PG student	Keyai Pramanick	Employed as Assistant Grade III Technical, Food Corporation of India on 8.10.2020 posted at Birbhum
2016-18 PG student	Sonali Ghatak	Qualified SET, 2020, Joined as SACT-II in Behala College, 2020; Presently pursuing PhD. in Dept. of Zoology, WBSU

Batch	Name of Student	Academic Achievement
2016-18 PG student	Amartya Paul	Joined as JRF (Zoological Survey of India) in 2021, Qualified RET for enrollment of PhD in 2022
2016-18 PG student	Ankur Banerjee	Qualified NET UGC JRF (RANK 74) 2019; Joined as JRF, Dept. of Zoology, WBSU in 2019
2016-18 PG student	Pritam Mandal	Qualified NET LS on 4.02.2021; Qualified GATE XL 2021 (AIR 4676); Joined as PhD Scholar in Dept. of Zoology, WBSU in 2021
2016-18 PG student	Sayanty Dasgupta	Qualified GATE XL in 2019, Qualified UGC NET LS on 4.02.2021
2016-18 PG student	Tanusri Das	Assistant Teacher in Holy Christ School, Kolkata from 2018-2021; Qualified GATE XL in 2020 (AIR 5998); Joined as JRF, Zoological Survey of India on 16.06.2021
2017-19 PG student	Sayan Dutta	Employed as Assistant Grade III Technical, Food Corporation of India, posted at Siliguri
2017-19 PG student	Koustav Bhattacharjee	Joined as Assistant Teacher, RKM Vidyapith, Deoghar, Jharkhand in 2019
2017-19 PG student	Subhra Prakash Mandal	VBD Technical Supervisor, Basirhat Health District
2017-19 PG student	Subhradip Pandit	Qualified UGC CSIR JRF NET on 4.02.2021, Joined as Project Assistant, Dept. of Zoology, Panchanan Barnma University, Coach Beher
2017-19 PG student	Priya Chatterjee	Worked as Project Assistant in CIFRI, Barrackpur, Kolkata
2017-19 PG student	Susraba Chatterjee	Qualified GATE XL 2021 (AIR 3749); UGC CSIR NET on 4.02.2021 (AIR UGC 75); JRF in School of Tropical Medicine in Glyco Immunology Laboratory

Batch	Name of Student	Academic Achievement
2017-19 PG student	Pradipta Kumar Ghosh	JRF in Zoological Survey of India, Kolkata; Qualified UGC NET LS 2020-21
2017-19 PG student	Shami Akhtar	Enrolled for PhD in the Department of Physiology, Calcutta University
2017-19 PG student	Nabajit Mondal	Qualified GATE XL 2020 (AIR 6794)
2017-19 PG student	Arghya Biswas	Qualified GATE XL 2021 (AIR 2150)
2017-19 PG student	Shrubawati Sarkar	QualifiedGATE XL 2022 (AIR 2810)
2017-19 PG student	Mayuri Dutta	Qualified Central Teacher Eligibility Test, 2022
2016-19 UG student	Apluta Majumder	Selected for M.Sc. in Vellore Institute of Technology in Applied Microbiology, 2019; Joined Syngene in 2021
2016-19 UG student	Ayan Mandal	Selected for M.Sc. in Banaras Hindu University, 2019; Admitted to PhD program , IISER, Mohali
2016-19 UG student	Banhishikha Datta	Selected for M.Sc. in Punjab University, Joined in Bio MedCAS 2021-2022, Van Andel Institute Graduate School PhD program in Molecular & Cellular biology
2018-20 PG student	Koustuv Chakraborty	QualifiedGATE XL 2022 (AIR 4562)
2018-20 PG student	Jibandeep Ghosh	Qualified 23rd SET 2022
2018-20 PG student	Soumyadip Mukherjee	Qualified JRF(NET)-UGC, 17.03.2022 (RANK-187); Joined as JRF, Dept. of Zoology, WBSU

Batch	Name of Student	Academic Achievement
2018-20 PG student	Avirup Ghosh	Qualified JRF (NET)-UGC, 17.03.2022 (RANK-109)
2018-20 PG student	Samim Hossain	Qualified LS (NET)-UGC, 17.03.2022 (RANK-20)
2018-20 PG student	Arup Das	Joined Department of Post, Gramin Dak Sevak on 20.7.2020
2018-20 PG student	Manashi Ash	Joined as GDS packer, Dept. of Post, India on 06.03.2020
2017-20 UG student	Rebanta Roy	QualifiedGATE XL 2022 (AIR 503); Qualified23rd SET
2019-21 PG student	Suman Kalyan Dinda	Qualified GATE XL 2021 (AIR 2783); Awarded BET (DBT-JRF) Category -I, 02.06.2022
2019-21 PG student	Ankita Mondal	Qualified GATE XL 2022 (AIR 3944)

PHOTOGRAPHY

EXPLORE *Nature*

Biodiversity is the greatest treasure we have...
Its diminishment is to be prevented at all cost

DR. SOMADITYA DEY
ASSISTANT PROFESSOR, BARASAT GOVT. COLLEGE

Camouflaged owl
(Collared scops owl)
(*Otus lettia*)

Brilliant & lusty
(White tailed kite)
(*Elanus leucurus*)

Free to fly
(Indian roller)
(*Coracias benghalensis*)

Ankur Banerjee, Ex Student (2016-18), PG Dept. of Zoology, Barasat Govt. College

Noisy Neighbours

Serene Sunset at Navegaon Lake

Into the wilds of Tadoba & Andhari (National Park in Maharashtra)

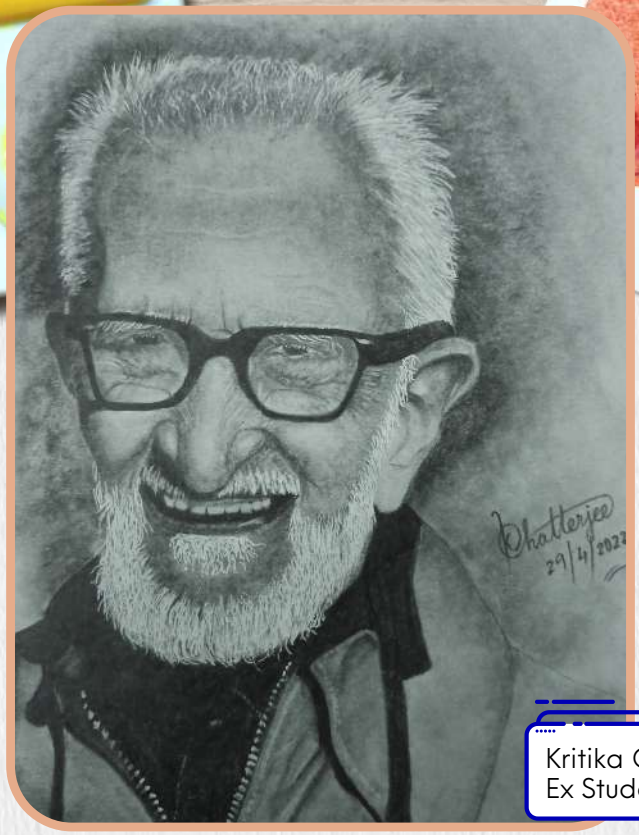
Shami Akhter, Ex Student (2017-19), PG Dept. of Zoology, Barasat Govt. College

Last year on October I have witnessed the most indelible incident of my life. It was in Tadoba National Park, Madhya Pradesh. after 3 days of waiting with bated breath I saw the Mother tiger named "Choti Madhu".

Her entry was like as if a queen entering in her kingdom: that doesn't end here, there was a severed head of Sambar deer in her mouth. It was a breath taking moment for me which will be everlasting.

Pritam Chakraborty, 4th Semester (2020-22), PG Dept. of Zoology, Barasat Govt. College

PAINTINGS

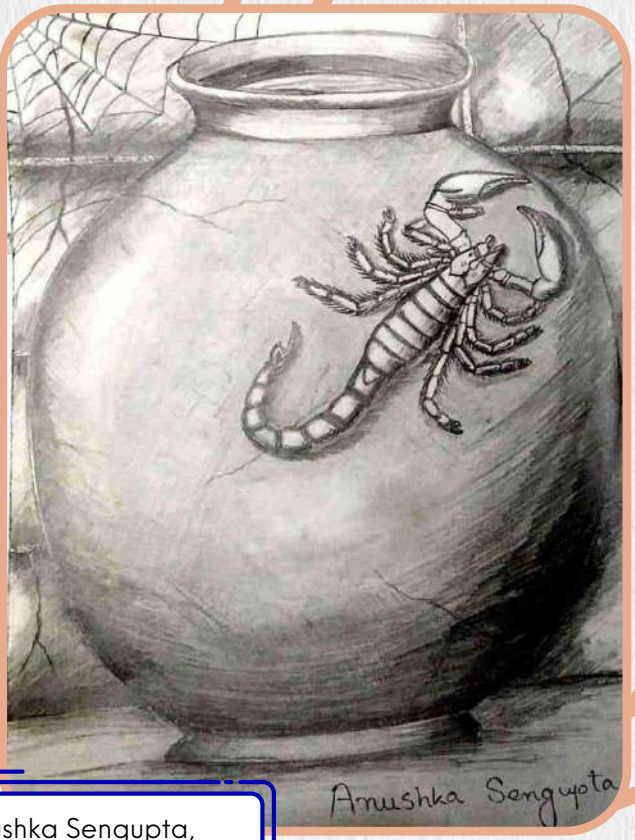


Kritika Chatterjee,
Ex Student, PG 2018-20

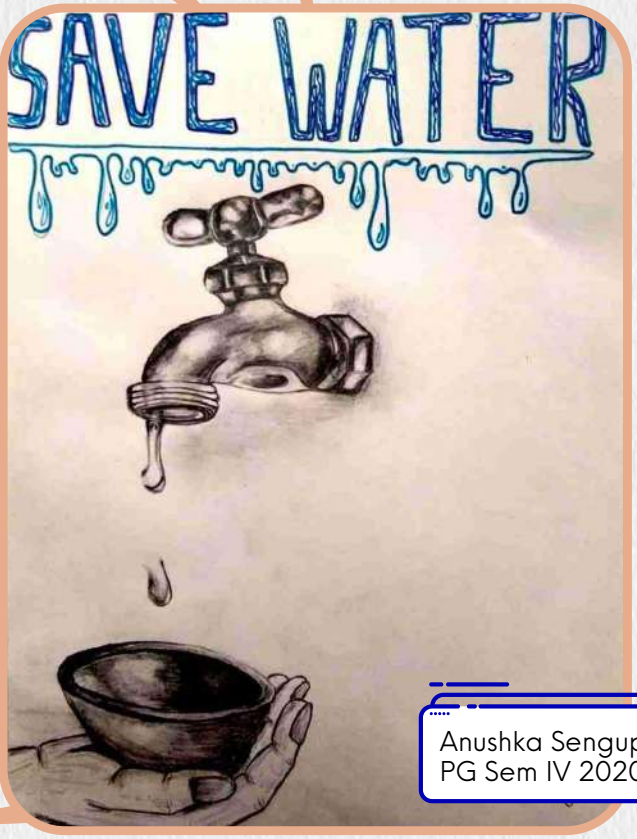
Michael Changjit Pal,
Ex Student, PG 2019-21

Michael Changjit Pal,
Ex Student, PG 2019-21

Paulami Ghosh,
Ex Student, PG 2019-21



Anushka Sengupta,
PG Sem IV 2020-22



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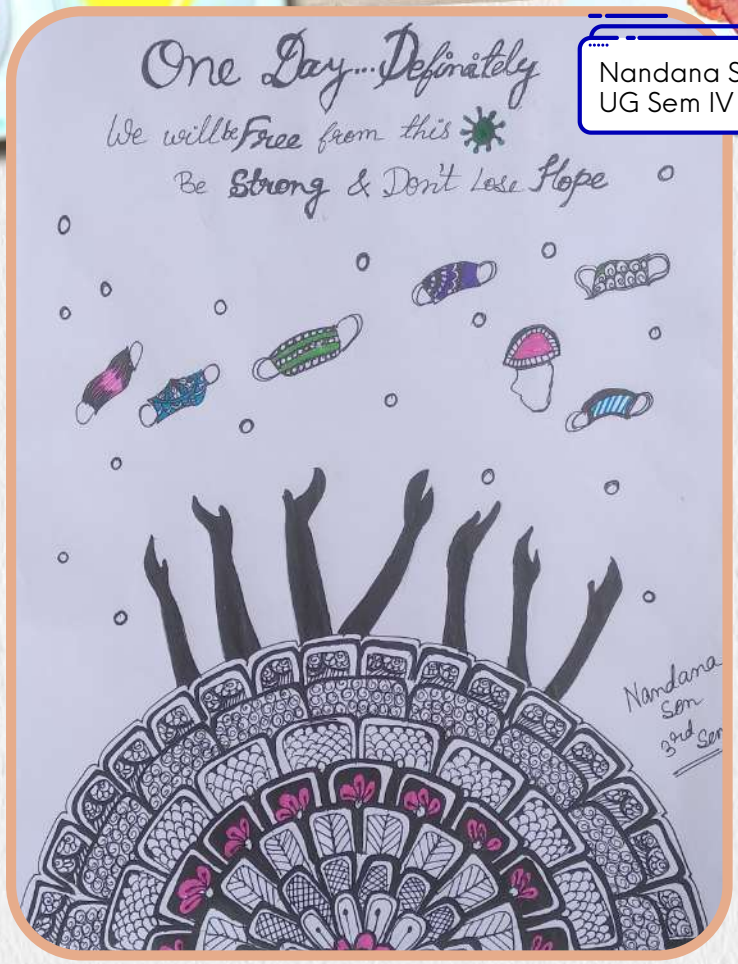
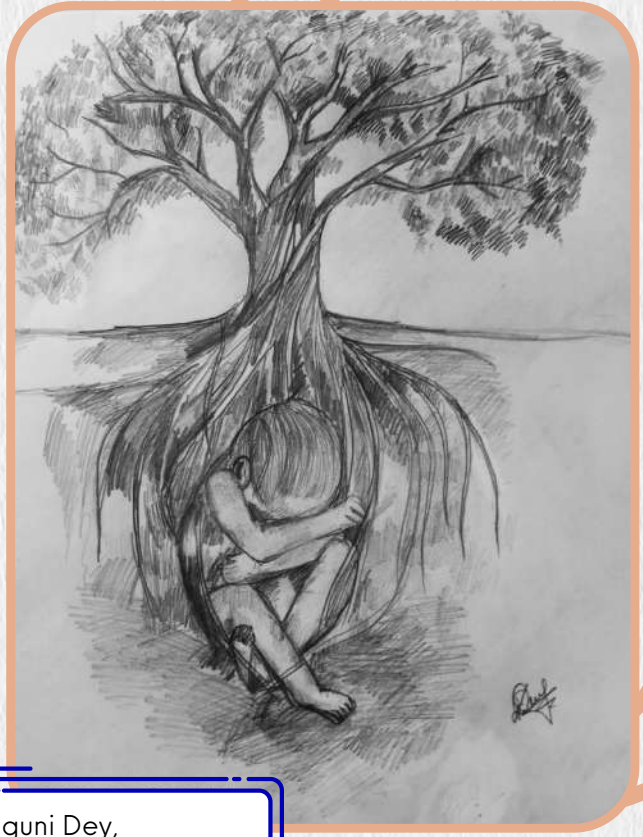
PG Sem IV 2020-22

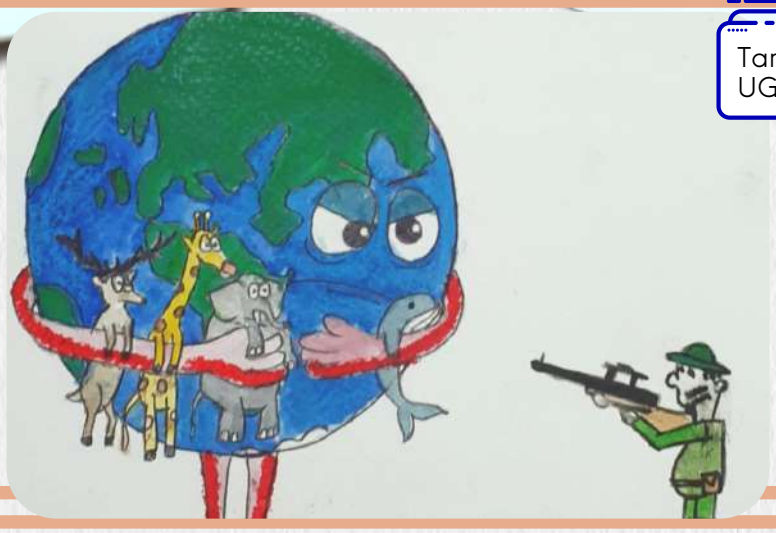


Nandana Sen,
UG Sem IV 2020-23



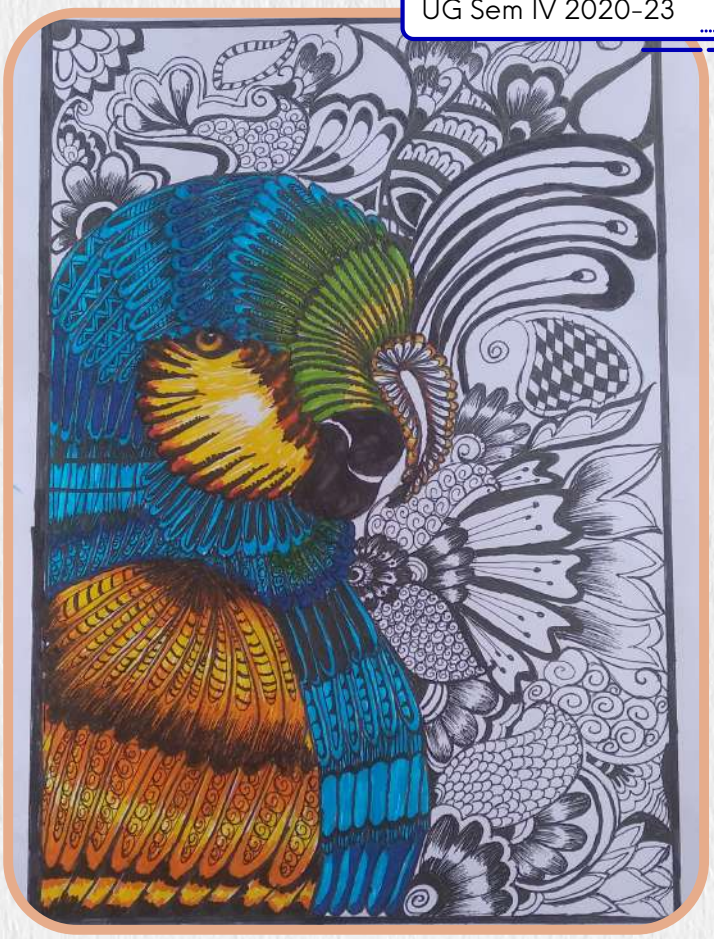
Falguni Dey,
UG Sem IV 2020-23

Anushka Ghosh,
UG Sem IV 2020-23



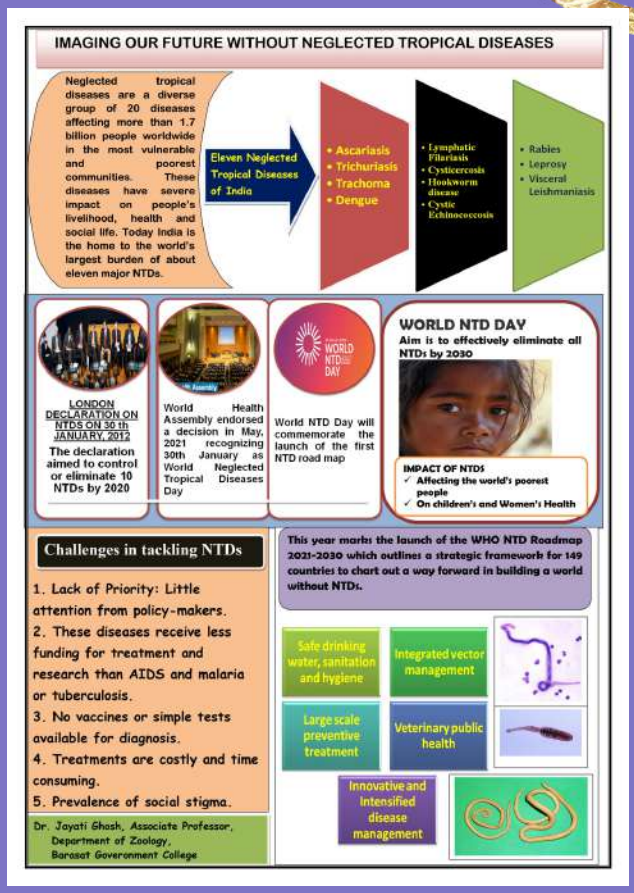
Tanisha Nowrin,
UG Sem II 2021-24

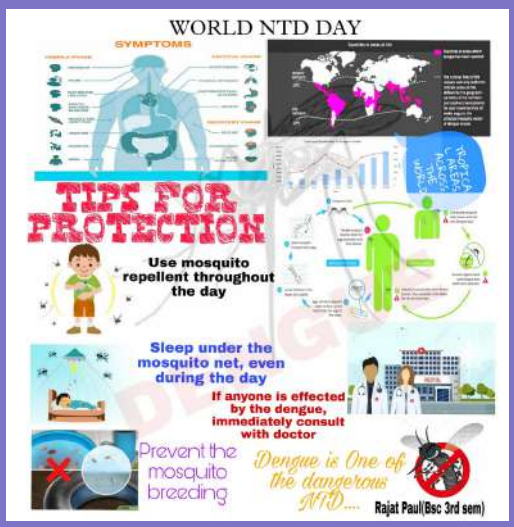
Nandana Sen,
UG Sem IV 2020-23

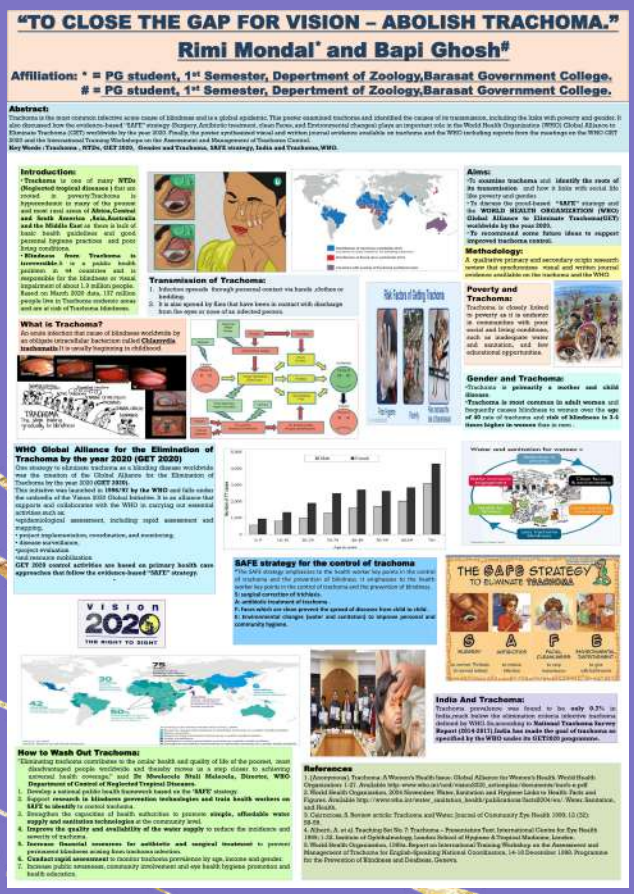


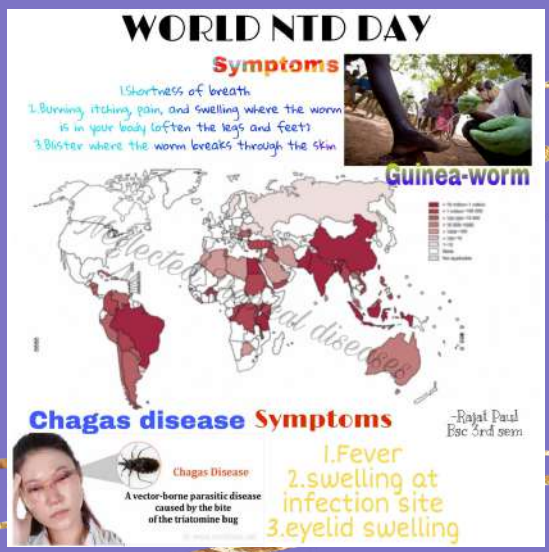
Nandana Sen,
UG Sem IV 2020-23

WORLD NTD DAY POSTERS

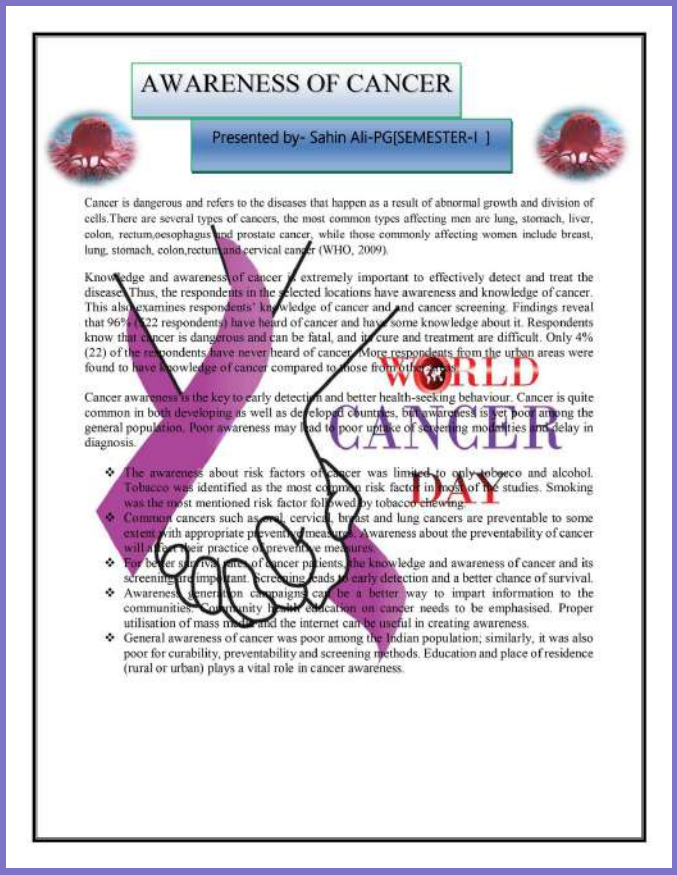








WORLD CANCER DAY POSTERS



AWARENESS OF CANCER

Presented by- Sahin Ali-PG[SEMESTER-I]

Cancer is dangerous and refers to the diseases that happen as a result of abnormal growth and division of cells.There are several types of cancers, the most common types affecting men are lung, stomach, liver, colon, rectum,oesophagus and prostate cancer, while those commonly affecting women include breast, lung, stomach, colon,rectum and cervical cancer (WHO, 2009).

Knowledge and awareness of cancer is extremely important to effectively detect and treat the disease. Thus, the respondents in the selected locations have awareness and knowledge of cancer. This also examines respondents' knowledge of cancer and and cancer screening. Findings reveal that 96% (522 respondents) have heard of cancer and have some knowledge about it. Respondents know that cancer is dangerous and can be fatal, and its cure and treatment are difficult. Only 4% (22) of the respondents have never heard of cancer. More respondents from the urban areas were found to have knowledge of cancer compared to those from rural areas.

Cancer awareness is the key to early detection and better health-seeking behaviour. Cancer is quite common in both developing as well as developed countries, but awareness is yet poor among the general population. Poor awareness may lead to poor uptake of screening modalities and delay in diagnosis.

- The awareness about risk factors of cancer was limited to only tobacco and alcohol. Tobacco was identified as the most common risk factor in most of the studies. Smoking was the most mentioned risk factor followed by tobacco chewing.
- Common cancers such as oral, cervical, breast and lung cancers are preventable to some extent with appropriate preventive measures. Awareness about the preventability of cancer will aid in their practice of preventive measures.
- For better survival rates of cancer patients, the knowledge and awareness of cancer and its screening are important. Screening leads to early detection and a better chance of survival.
- Awareness generation campaigns can be a better way to impart information to the communities. Community health education on cancer needs to be emphasised. Proper utilisation of mass media and the internet can be useful in creating awareness.
- General awareness of cancer was poor among the Indian population; similarly, it was also poor for curability, preventability and screening methods. Education and place of residence (rural or urban) plays a vital role in cancer awareness.

AWARENESS OF CANCER IN ACCORDANCE WITH WORLD CANCER DAY.

ANTARA SINGHA RAY(PG, 1'st Semester, Department of Zoology)

Cancer is a disease in which body's cells grow uncontrollably and spread to the other parts of the body.**4th february** is known as world cancer day. So, let's know some do's and don'ts about cancer.

Do's of cancer :

- Exercise often: Exercise habit is directly related to cancer. Several researcher claimed that exercise can reduce the chance of having cancer by lowering calories and stress which are the main reason to cancer. So, do 30 minutes exercise for at least 3 times a week.
- Clear your mind: clearing mind will lower your stress level and will also improve the body's immune system. A healthy mind can lower the chance of having cancer. Practice regular meditation to clear your mind.
- Eat fruits and vegetables: vegetables are not only high in antioxidant and fiber ,but also contains dietary cancer chemo preventive such as carotene, lycopene etc. so, eat about 500grams fruits and vegetables per day.

Have adequate and appropriate nutrition: consume appropriate proportion of each 5 food group per meal. Avoid grilled, deep fried or pan fried food that are brunt .Avoid red meat and fermented foods also.

Don'ts of cancer:

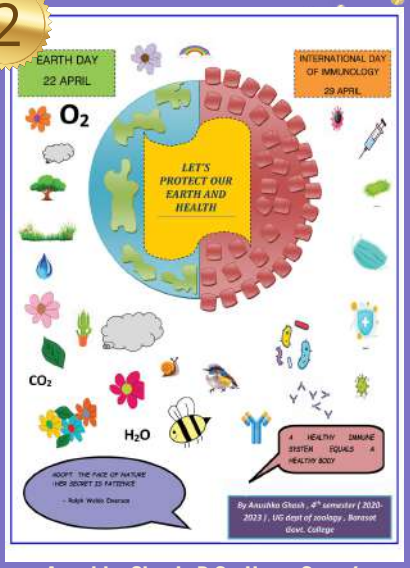
- Don't smoke: smoking is one of the main causes of lung cancer, larynx cancer ,liver cancer even ovaries cancer .smoke from cigarette contains over 4,000 types of chemicals,60 of which are carcinogenic. So, avoid smoking.
- Do not drink often: Drinkers are prone to liver cancer, breast cancer, esophageal cancer. It is recommended not to drink.
- Don't expose to intense sunlight: sunlight contains uv rays , which is the main cause for skin cancer. uvB is believed to be the main cancer developing agent. Stay clear from intense sunlight, use long sleeves, eye glasses and a hat to cover sunlight.
- Do not engage illicit sex: women who have many sexual partners are more prone to ovarian cancer. So, do not change sexual partner often. Wear appropriate protection while having sex.

WORLD EARTH DAY & INTERNATIONAL IMMUNOLOGY DAY POSTER COMPETITION

1

Rajdip Chandra Dafader, B.Sc. Hons. Sem-2

2

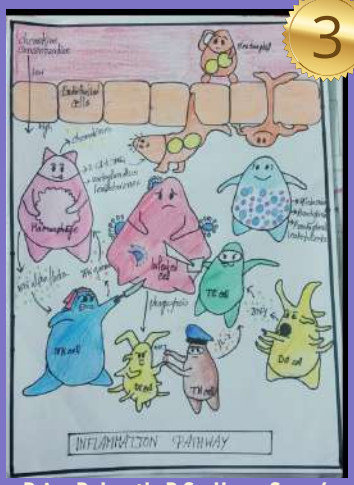


Anushka Ghosh, B.Sc. Hons. Sem-4

2

Basusri Dandapat, B.Sc. Hons. Sem-4

3



Priya Debnath, B.Sc. Hons. Sem-4

3

Rimpa Byapari, B.Sc. Hons. Sem-4

SPECIAL MENTION

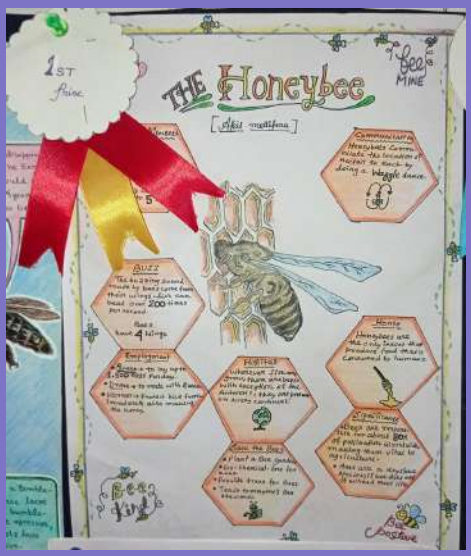
Mahesh Paik B.Sc. Hons. Sem-6

Nandana Sen, B.Sc. Hons. Sem-4

Tanisha Nowrin, B.Sc. Hons. Sem-2

INAUGURATION OF DEPARTMENTAL WALL MAGAZINE, ZOOLOGY, TO CELEBRATE WORLD EARTH DAY & INTERNATIONAL IMMUNOLOGY DAY, 2022

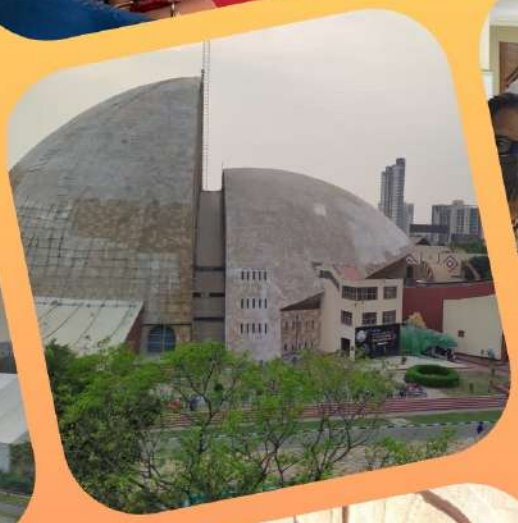
POSTER PRESENTATION ON THE EVE OF WORLD BEE DAY (M.Sc. SEM II) & WORLD ENVIRONMENT DAY & WORLD BIODIVERSITY DAY (UG SEM II, IV & VI)



1st and 2nd winner of World Bee Day Poster

ONE DAY TRIP


PROTEROZOIC

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Bengal 700105, India
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Kolkata, West Bengal, India
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Bengal 700105, India
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Kolkata, West Bengal, India
SCIENCE EXPLORATION HALL, SCIENCE CITY, Mirani
East Topsia, Topsia, Kolkata, West Bengal 700105, Ind
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VISIT TO SCIENCE CITY (UG)

Barasat Govt. College, Zoology had organized a One Day Science City Education Tour for the B.Sc. Hons. 2nd Semester and 4th Semester students. The purpose of visit is to promote and enhance students understanding of the culture of science and technology to supplement science education given in colleges and to organize various out-of-college educational activities to foster a spirit of scientific enquiry and creativity among the students. The trip to science city was a wonderful experience with full of knowledge of Science, Mathematics, Physics, and particularly Biological and much more, making science simple and interesting.

A visit to science exploration hall, Space Odyssey, Dynamotion hall, etc. was very exciting for students and really helps them understanding basic sciences. A special show on digital Panorama, being the first of its kind in the world, depicts the important milestones during the last 6 million years of human evolution and 'Evolution of Life – A Dark Ride' comprises 56 robotic animal models divided into seven sections to showcase the milestone events of evolution of life and representative life forms of respective eras. Science on a Sphere has provided an overview of the Earth's environmental process through a large visualisation system.

It was just a Complete and Knowledgeable trip. In this visit, around 50 students visited Science City along with six faculty members of our departments.

DROSOPHILA & LEISHMANIA LABORATORY VISIT WITH MSc SEM2 STUDENTS AT IISER-K

Indian Institute of Science Education and Research Kolkata (IISER-K or IISER Kolkata) is an autonomous public university in science and education field located in Mohanpur near the town of Kalyani in Nadia district, West Bengal, India. Barasat Govt. College, Zoology had organized a One Day Science City Education Tour for the M.Sc. 2nd Semester students along with three Faculty members (Dr. Enamul Haque, Dr. Ivy Kundu & Dr. Somaditya Dey) to visit the research laboratories related to *Drosophila* developmental biology and genetics (PI: Mohit Prasad, Professor, Department of Biological Sciences). They have got the opportunities to hear a lecture from Dr. Prasad and to study the handling of *Drosophila*, identification of mutants and their genetic crosses.

LABORATORY VISIT WITH MSc SEM4 SPECIAL PAPER (PARASITOLOGY & IMMUNOLOGY) STUDENTS AT DEPT OF CLINICAL IMMUNOLOGY & RHEUMATOLOGY, SSKM & IPGMER HOSPITAL

The M.Sc. students of Sem IV (No.-18; Special paper- Parasitology and Immunology have visited the research laboratories (Dept. of Clinical Immunology & Rheumatology, IPGMER & SSKM Hospital, Kolkata) related to immunology to partially fulfil the CBCS syllabus accompanied by two of our Faculty Members (Dr. Srikanta Guria and Dr. Somaditya Dey). In this regard, the purpose of the visit was studying the common laboratory protocols, instruments, and practices. This program allowed the students to acquire knowledge and hands on training on _8-color multiplex flow cytometry, DNA/RNA isolation, RT-PCR, Immunofluorescence, and ELISA etc. This visit has enriched our students about the current status of research in this field.

LABORATORY VISIT WITH UG STUDENTS (ESCORTED BY DR. IVY KUNDU & DR. SOMADITYA DEY) – DR. SUJAY GHOSH, CYTOGENETICS & DROSOPHILA LABORATORY, CALCUTTA UNIVERSITY

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GPS Map Camera
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UNIVERSITY OF CALCUTTA
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Kolkata, WB, India
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>

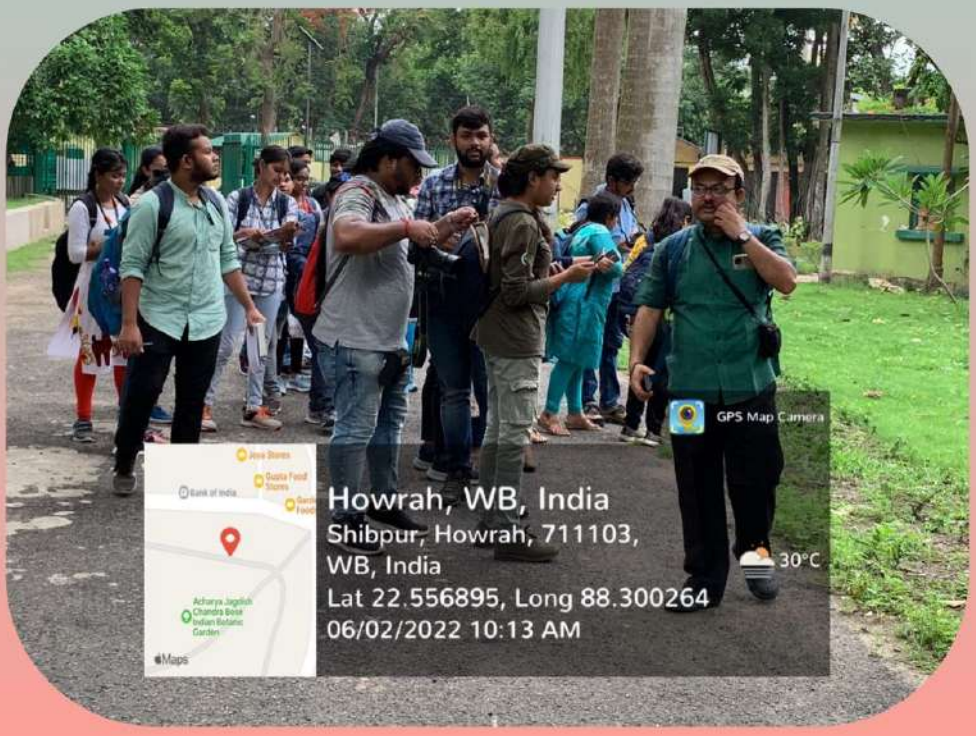
LABORATORY VISIT PROGRAMME WITH UG STUDENTS (ESCORTED BY DR. SRIKANTA GURIA & DR. SOMADITYA DEY) – INSTITUTE OF GENETIC ENGINEERING, BADU, MADYMGRAM

>

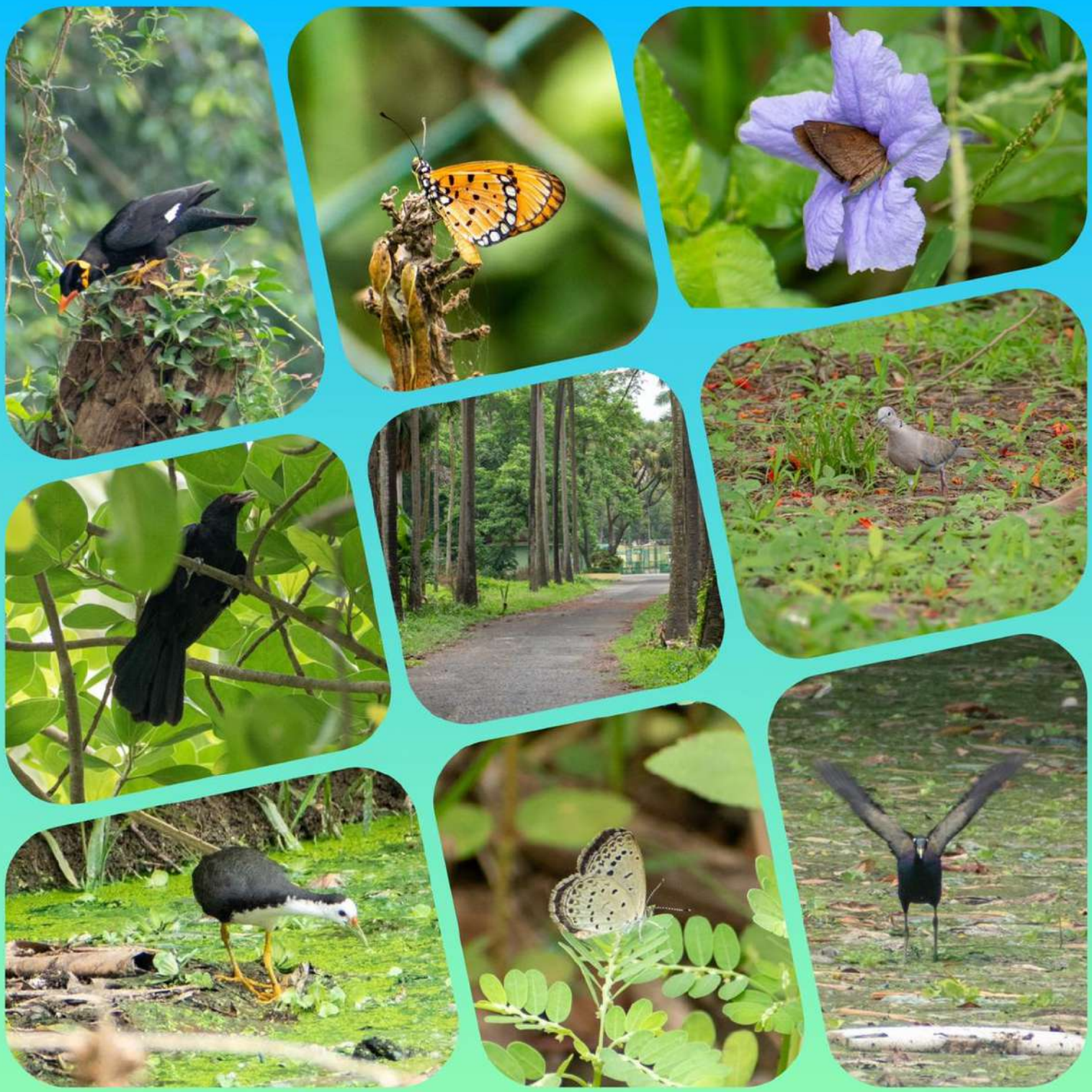
FIELD TRIP TO CHINTAMONI KAR WILDLIFE SANCTUARY

M.Sc. students of Sem IV (No. - 11; Special paper- Ecology & Environmental Biology have visited Chintamoni Kar Wildlife Sanctuary, Ramchandrapur, near Narendrapur, Kolkata, for one day field trip cum nature trail related to partially fulfilment of the CBCS syllabus accompanied by three of our Faculty Members (Dr. Sumana Saha, Dr. Jayati Ghosh & Dr. Srikanta Guria). By celebrating International Biodiversity Day students have experienced various flora & fauna in their natural habitat & also received hands on training of various techniques for assessing biodiversity in the protected areas.

BIRD WATCHING AT ACHARYA JAGADISH CHANDRA BOSE INDIAN BOTANIC GARDEN WITH BSc & MSc SEM4 STUDENTS – CELEBRATING "WORLD BIODIVERSITY DAY" (DEPT OF ZOOLOGY, BGC)




आचार्य जगदीश चन्द्र बोस भारतीय वनस्पति उद्यान
ACHARYA JAGADISH CHANDRA BOSE INDIAN BOTANIC GARDEN
HOWRAH - 711103




ACHARYA JAGADISH CHANDRA BOSE INDIAN BOTANIC GARDEN
GPS Map Camera
Howrah, WB, India
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Maps


GPS Map Camera
Howrah, WB, India
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GPS Map Camera
Howrah, WB, India
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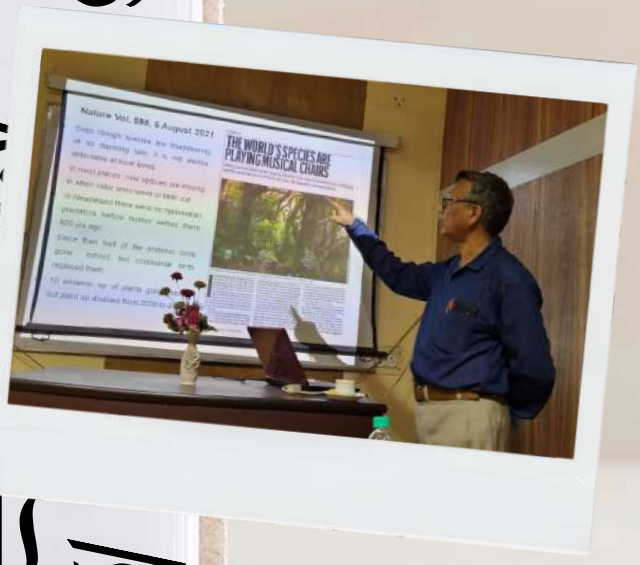
INTERNATIONAL DAY FOR BIOLOGICAL DIVERSITY
BIRD WATCHING
ORGANIZED BY:
PG DEPARTMENT OF ZOOLOGY
BARASAT GOVERNMENT COLLEGE
SUPPORTED BY:
IQAC, BGC
ACHARYA JAGADISH CHANDRA BOSE INDIAN BOTANIC GARDEN
JUNE 2, 2022
9 AM
CONTACT: 8013247086
WWW.BGCAC.IN
Dr. SUBHENDU MAJUMDAR
Assistant Professor in Zoology, Shibpur Dinobundhoo Institution, Shibpur, Howrah
Field Ornithologist
ANTARA SARKAR & SOUVIK BARIK,
DEPT. OF ZOOLOGY, CU
CHIEF PATRON
DR. SAMAR CHATTOPADHYAY, WBSES,
PRINCIPAL, BGC
ACCOMPANYING FACULTIES
DR. SOMADITYA DEY, WBES
ASSISTANT PROFESSOR, DEPT. OF ZOOLOGY, BGC
SMT. INDRANI BANERJEE
SACT II, DEPT. OF ZOOLOGY, BGC

DEPARTMENTAL SEMINAR ON THE OCCASION OF BHASHA DIBASH



Biodiversity Graph for the Next Generation

আগামী প্রজন্মের জন্য জীববৈচিত্র্যের রেখচিত্র

Dr. Anilava Kaviraj, Professor (Retd.), Dept. of Zoology, University of Kalyani

Prof. Edward Osborne Wilson, who died on 26th December, 2021 at the age of 92, is considered as the 'Darwin of the 21st Century' for his contribution in biodiversity research and involvement in biodiversity awareness campaign. No talk on biodiversity is complete without a reference to Prof. Wilson. He presented the 'Island Biogeography Theory' in 1967, which is now taught in Post Graduate course in Zoology throughout the world. Besides writing many books on biodiversity, he edited the first book on 'Biodiversity' in 1988, which played a pivotal role in the 'Earth Summit'- the United Nations Conference on Environment and Development (UNCED) held in Rio de Janeiro in 1992 to start the "Convention on Biodiversity", the first and the only forum at government level to control biodiversity issues.

What is biodiversity?

Biodiversity can be revealed in several ways such as (1) richness of species, (2) genetic diversity of each species, (3) diversity of the habitats of species and (4) the function of each species in the ecosystem. As per IUCN, 18 to 19 lakhs of species have so far been identified, of which approximately 10 lakhs species are only insects, while other invertebrates, vertebrates and plants approximately constitute 3 lakhs, 66 thousands and 3 lakhs species respectively. We do not know how many species remain unidentified. Using a method known as "taxonomic scaling" Mora et al. (2011) predicted a total number of about 87 lakhs of species on this earth including 22 lakhs marine animals.

Extinction

All animal and plant species undergo a background rate of extinction, which range from 0.1 to 1 extinction per million species per year (**0.1-1 E/MSY**). For mammals this rate is considered as **2 E/MSY**. Due to expansion of human civilization and subsequent destruction of natural habitats of plants and animals by man, the extinction rate has increased to 100 to 2000 times of the background rate. Even under most conservative measure, approximately 900 species of animals became extinct during the last 500 years. As per Intergovermental Science Policy Platform on Biodiversity and Ecosystem (IPBES), a non-govt. organization sponsored by UNEP and IUCN, about 10 lakhs of species are facing the risk of extinction. This prediction is based on species number calculated by Mora et al. (2011) and IUCN calculated rate of extinction for a taxon. The most vulnerable species are Cycas among plants and corals and amphibians among animals. The last two species are victims of global warming. The large bodied animals and specialized species (restricted to specific niche) are more vulnerable than small bodied animals and generalized species.

Are we heading for 6th Mass Extinction?

So far this planet has seen five mass extinctions, when 50-95% of species had been extinct. The last one occurred 65 million years ago. Every time new species evolved and the planet again had become full of species. But due to rapid expansion of cities, industries, roads and agricultural lands since last 500 years, large areas of habitats for plants and animals have been irreparably lost, thereby making evolution of new species extremely difficult. Many authors including Prof. Wilson predicted that unless strong conservation measures are taken we may soon achieve sixth mass extinction. Let us hope for better awareness and conservation programme so that we can slower the extinction rate and save this planet from sixth mass extinction.

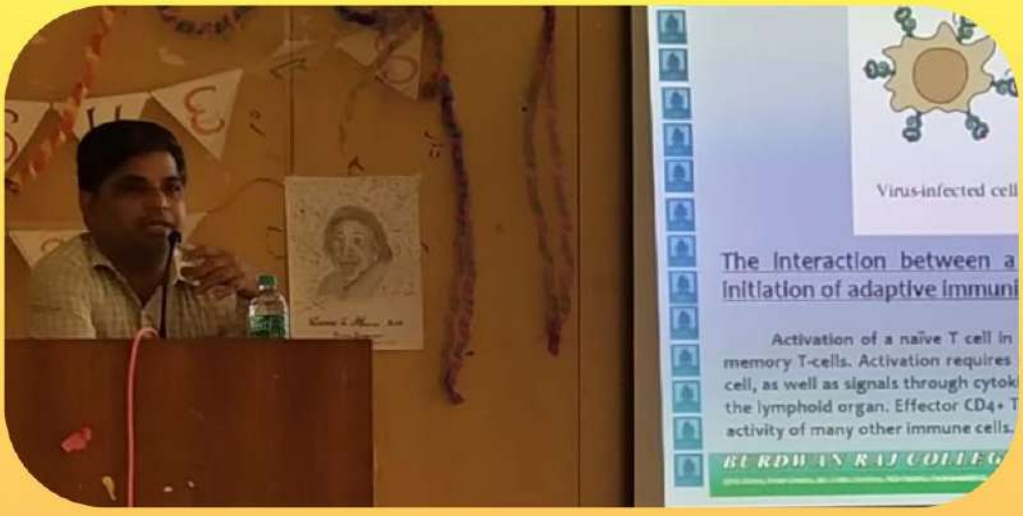
References

1. Mora et. al (2011). How Many Species Are There on Earth and in the Ocean?
2. PLoS BIOLOGY 9 (8): e1001127
3. IPBES.(2019). The global assessment report on Biodiversity and Ecosystem Services - Summary for Policymakers.

WEBINAR ORGANISED BY THE DEPARTMENT

WEBINAR

ACADEMIC SEMINAR, DEPARTMENT OF ZOOLOGY, BGC, TO CELEBRATE INTERNATIONAL IMMUNOLOGY DAY 2022



Speaker - Dr. Syamdas Bandyopadhyay, Assistant Professor, Dept of Zoology, Burdwan Raj College

International Webinar

International Webinar on "Comprehensive Current Approaches to Oncogenetics & Pollination Ecology" has been arranged by our department on September 27, 2021. The two young and energetic speakers were Dr. Sayan Chakraborty, who is now serving as a Research Assistant Professor & NMRC Young Investigator, Institute of Molecular & Cell Biology, A-STAR, Singapore and Dr. Priyadarshini Chakrabarti Basu, now serving as Assistant Professor, Pollinator Health & Apiculture, Department of Biochemistry, Molecular Biology, Entomology & Plant Pathology, Mississippi State University, USA.

100 Years of Insulin Discovery

The department has also arranged a national webinar on "Celebrating 100 years of insulin discovery: advances and opportunities in diabetes research" on November 24th, 2021. The speakers of the said webinar were Dr. Anirban Sinha, Assistant Professor, Department of Endocrinology, Medical College, Kolkata (Topic: Recent Advances In Insulin And Delivery Technology In Diabetes) and Dr. Partha Chakrabarti, Principal Scientist, Cell Biology & Physiology CSIR-IICB, Kolkata (Topic : Insulin And Indian Diabetes : Ideas And Experiments).

Neglected Tropical Disease Day 2022

PG Dept. of Zoology has celebrated World NTD day. Students of UG and PG program has submitted posters, graphical artworks, and drawings to arrange an online awareness initiative on "Neglected Tropical Disease Day" (30/01/2022). They have also submitted brief talks or group discussions (5-6 minutes each). The themes of the presentations are vectors, modes of transmissions, impacts, implications, control, prevention, elimination and eradication of these diseases. A webinar has also been arranged by our department featuring the lecture of Dr. Madhumita Manna, WBSES, Additional Director of Public Instruction (Administration) Education Directorate, Department of Higher Education, Govt. of West Bengal, India on the theme 'Beat NTDs. For Good. For All. Together, We Can Combat NTDs' on 29th January,2022, 7 PM (IST). The title of her speech was "Whole genome sequencing and multi locus sequence typing of the clinical isolates of Indian Kala-azar and Parakala-azar dermal Leishmaniasis patients".

International Immunology Day 2022

An academic Seminar (Offline) has been arranged by Department of Zoology, BGC to celebrate International Immunology Day 2022 (APRIL 30, 2022). Speaker of the said seminar was Dr. Syamdas Bandyopadhyay, Assistant Professor, Dept of Zoology, Burdwan Raj College. The seminar was a one & half hour long program being curated by PG Dept of Zoology, Barasat Govt. College, with an audience of about 100 participants (approx.), made up of undergraduate & postgraduate students, and faculties of our college. His discussion on "The Saga of CD1d", was a great addition to our event.

For all the seminars and webinars, departmental faculties perform their duties as organizing secretaries, Convenors and/or members on rotational basis under the patronship of Dr. Samar Chattopadhyay, Principal, Barasat Govt College. The programs were supported by IQAC, BGC. The webinars were live streamed at college YouTube channel and the numbers of participants were approximately 500+ for the webinars and 100+ for the offline seminars, comprising of UG and PG students, faculties, researchers of several institutions in India. The programs were documented on college website and certificates were given to the participants.

AWARDS

Anish Malakar, 2nd Semester, (2021-24), UG Dept. of Zoology

Winner & Runners Up at Indoor & Outdoor Games

3rd Position at Instant Speech Competition

Raya Datta, 1st Semester, (2021-24), UG Dept. of Zoology

OUR COLLEGE TEAM STOOD 3RD IN THE QUIZ ON WORLD ENVIRONMENT DAY CELEBRATION, ORGANIZED BY WB POLLUTION CONTROL BOARD AT SCIENCE CITY AUDITORIUM (05/06/22)

পশ্চিমবঙ্গ দূষণ নিয়ন্ত্রণ পর্ষদ
পরিবেশ দপ্তর, পশ্চিমবঙ্গ সরকার

শংসাপত্র

বিশ্ব পরিবেশ দিবস উদ্‌যাপন উপলক্ষ্যে কলকাতার সায়েন্স সিটি অডিটোরিয়ামে পরিবেশ বিষয়ক প্রশ্নোত্তর প্রতিযোগিতায় তৃতীয় স্থান অর্জন করার জন্য ধারাবাত গভঃ কলেজ কে

আজ ৫ই জুন, ২০২২ তারিখে এই শংসাপত্র প্রদান করা হল।

ডঃ রাজেশ কুমার, আইপিএস
সদস্য সচিব

ডঃ কল্যাণ রুদ্র
চেয়ারম্যান

FRESHER'S WELCOME 2022 (UG)

FAREWELL CEREMONY OF SRI ASUTOSH DAS, LABORATORY ASSISTANT, ZOOLOGY DEPT.

শতবর্ষ পরে

শ্রী আশুতোষ দাস, প্রাক্তন ল্যাবরেটরি সহায়ক,
প্রাণীবিদ্যা বিভাগ, বারাসাত সরকারি মহাবিদ্যালয়

আজি হতে শতবর্ষ পরে
কে তুমি ডাকিতেছ আবার চেনা নাম ধরে
আজই হতে শতবর্ষ পরে
২০২১কে বিদায় জানিয়ে ২০২২কে আহ্বান করেছি সবাই মিলে
সহস্র বাজির রামধনুতে।মিলায়েছে সবাই।
আজি হতে শতবর্ষ পরে
হয়তো বা কেউ চিনবেনা মোরে
ছবিতে পড়বে ধুলো মুছবেনা কেউ
ভেবেও পারবেনা তুমি অঙ্ক কি
করে মিলাই ?
আজি হতে শতবর্ষ পরে
নতুন প্রজন্মের হাত ধরে
আসবে অজানা গ্রহ থেকে
অসম্ভব সব ইলেকট্রনিক যন্ত্র
নতুন মানবের উদ্ভাবিত মন্ত্র
থাকবে যারা স্মৃতি নিয়ে
কোনো মতেই মেলাতে পারবেনা
ফেলে যাওয়া শতবর্ষের অংশ
আজি হতে শতবর্ষ পরে।
থাকিবে না আর খনিজ পদার্থ,
পানীয় জল, চাষের জমি
বিকল্পে ছেয়ে যাবে সারা পৃথিবী
মানব সভ্যতা এসে দাড়াবে
এক ধ্বংসের সম্মুখীন।
প্রখর সূর্য তাপ হারায়ে
হইবে মলীন।

FAREWELL CEREMONY OF SRI AJOY KUMAR BISWAS, LABORATORY ATTENDANT, ZOOLOGY DEPT.

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