

Zoion



SEPTEMBER 2020 | ISSUE 03

PREVENTION OF
ATHEROSCLEROSIS BY
HDL ENHANCEMENT

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KKTU GOVT. COLLEGE, PULLUT, KODUNGALLUR

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PRASAD N K

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Dear friends,

Welcome to the third issue of our monthly newsletter "Zoion". Zoion is a humble initiative to set the budding minds free, allowing them to roam in the realm of knowledge, imagination and experience to create a world of meaningful words. I am sure that this project has created a Launchpad for our students' creative urge to blossom naturally. We are proud and exuberant to introduce this issue to you, which gives special attention to articles related to Alzheimer's disease.

Alzheimer's disease is a type of dementia that affects memory, thinking and behavior. Symptoms eventually grow severe enough to interfere with daily tasks. As per the statistics revealed by Alzheimer's and related disorders Society of India, currently, around 5.3 million Alzheimer's patients are present in India and there is an annual increase in the number of patients by 10.11%. Although existing modes of treatments cannot stop Alzheimer's from progressing, they can temporarily slow the worsening of dementia symptoms and improve the quality of life for those with Alzheimer's and their caregivers. Today, there is a worldwide effort underway to find better ways to treat the disease, delay its onset, and prevent it from developing.

The month of September is considered as World Alzheimer's month. The main article of this issue is an endeavor to raise awareness of how this disease influences the daily lives of people affected and challenge the stigma that surrounds it.

We hope that the positive attitude, hard work, sustained effort and innovative ideas exhibited by our young people will surely be useful to the readers. We thank all the contributors, teaching and non-teaching faculty, students and Head of the institution for their supports.

Digital Magazine issued by

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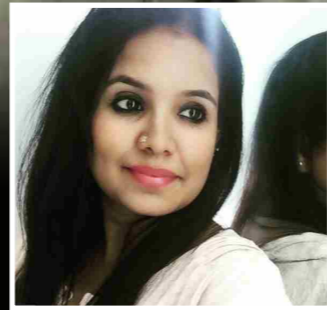
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ALZHEIMER'S DISEASE: PRIORITY OVERLOOKED?

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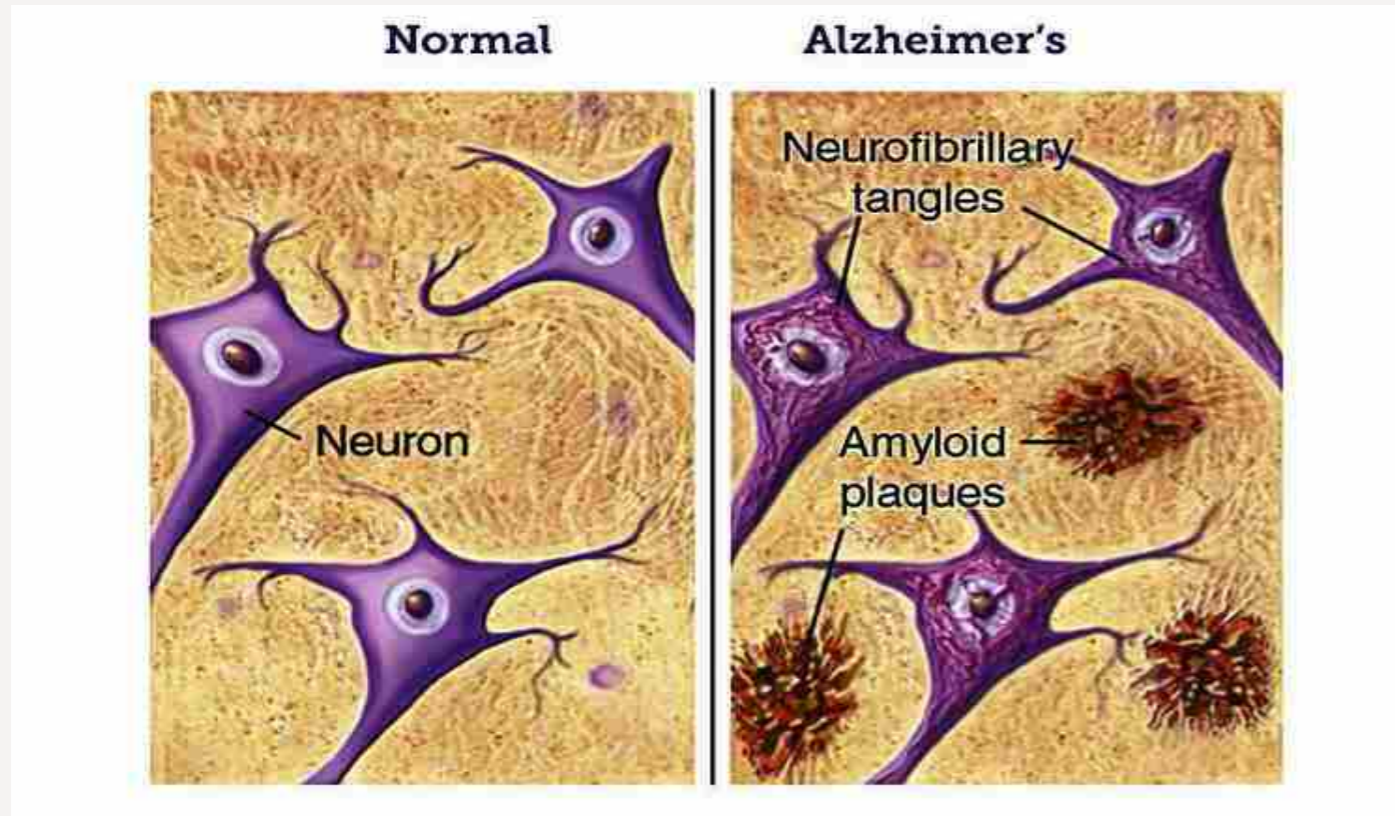
Alzheimer's is a degenerative disease of the brain and is the most common form of dementia. This is a progressive condition wherein the brain cells and cell connections die gradually, destroying one's memory and other crucial mental functions. It is characterised by progressive deterioration of intellect, including loss of memory, thinking, learning, orientation, language, comprehension and judgment. Early symptoms of Alzheimer's include short term memory loss where the individual finds it difficult to remember recent events. But for them, memories of older events remain intact. This progresses across time and manifests as other symptoms like difficulty in finding words while talking, getting lost in familiar places, losing track of time, difficulty in decision making, managing finances and performing multistep tasks. Gradual mood and behavioural changes like irritability, sadness, anxiety, mood swings, withdrawal, aggression, change in sleep habits, disinhibited behaviours and lack

of control over bladder/ bowel also set in. The patient can also show symptoms like aimless wandering, shouting etc. as the disease progresses. The impact of the disease could be so harsh that patients are even unable to carry out everyday chores.

Up to 70% of the risk of AD is genetic. Some cases of early-onset Alzheimer's disease are caused by gene mutations that can be passed from parent to child. This results in what is known as early-onset familial Alzheimer disease (FAD). Researchers have found that this form of the disorder can re-

sult from mutations in the APP, PSEN1, or PSEN2 genes. When any of these genes is altered, large amounts of a toxic protein fragment called amyloid-beta peptide are produced in the brain. This peptide can build up in the brain to form clumps called amyloid plaques, which are characteristic of Alzheimer's disease. A build-up of toxic amyloid-beta peptide and amyloid plaques may lead to the death of nerve cells and the progressive signs and symptoms of this disorder. Other cases of early-onset Alzheimer's disease may be associated with changes

in different genes, some of which have not been identified. The causes of late-onset Alzheimer's disease are less clear. The late-onset form does not run in families, although clusters of cases have been reported in some families. This disorder is probably related to variations in one or more genes in combination with lifestyle and environmental factors. A gene called APOE has been studied extensively as a risk factor for the disease. In particular, a variant of this gene called the e4 allele seems to increase an individual's risk for developing late-onset



Alzheimer's disease. History of head injuries, psychiatric illnesses like depression and lifestyle diseases like hypertension can also increase the risk of AD. There's no known cure for Alzheimer's disease. However, certain medications can manage symptoms, slow down the disease progression and alleviate suffering, for both the patient and the caregivers. Behavioural problems associated with this condition can be controlled efficiently with psychiatric medications. A good lifestyle and awareness can help in early detection, prevention and effective management of the condition.

Globally over 47 million people are living with dementia. According to WHO, the number of people living with demen-

tia doubles every 20 years. The rate of growth in the burden is expected to be highest in developing countries like India. According to the Dementia India Report 2010 by the Alzheimer's and Related Disorders Society of India (ARDSI), there were around 3.7 million Indians with dementia in 2010 with the number projected to rise to 7.6 million by 2030. Usually seen in people above 65 years of age, estimates say that there are about 2 Lakh people in Kerala who suffer from Alzheimer's Disease (AD). Alzheimer's disease imposes both economic and non-economic costs beyond that on the patient as family members still provide the bulk of the caregiving. The ARDSI 2010 report estimated the total societal costs from dementia to

be 147 billion INR in 2010 with a projected threefold increase by 2030. In addition to the direct cost of treatment e.g. cost of medication and physicians, the bulk of these economic costs stem from informal care through the loss of wages and income through absenteeism from work or withdrawal from labour force by family members. Long-term care also imposes a psychological toll on the caregiver.

The awareness about dementia is poor among common people and also general practitioners. In the Indian social scenario, forgetfulness in the elderly is often recognized as a normal variation of aging. When it is recognized, it is often in advanced stages. Poor awareness leads to poor recognition, resulting in delayed diagnosis



and sometimes catastrophic situations may arise. There is little help from health service sectors which do not provide the needed information and support for carers and family members. There is an urgent need to increase awareness about dementia in general, and about the early symptoms of Alzheimer's disease in particular. Family members and primary care physicians are best placed to recognize these early symptoms and hence, a national awareness campaign targeted towards them is likely to have the most effect. Print media and electronic media have started to raise awareness among the general public in India. ARDSI is taking part in this process through its different chapters nationally.

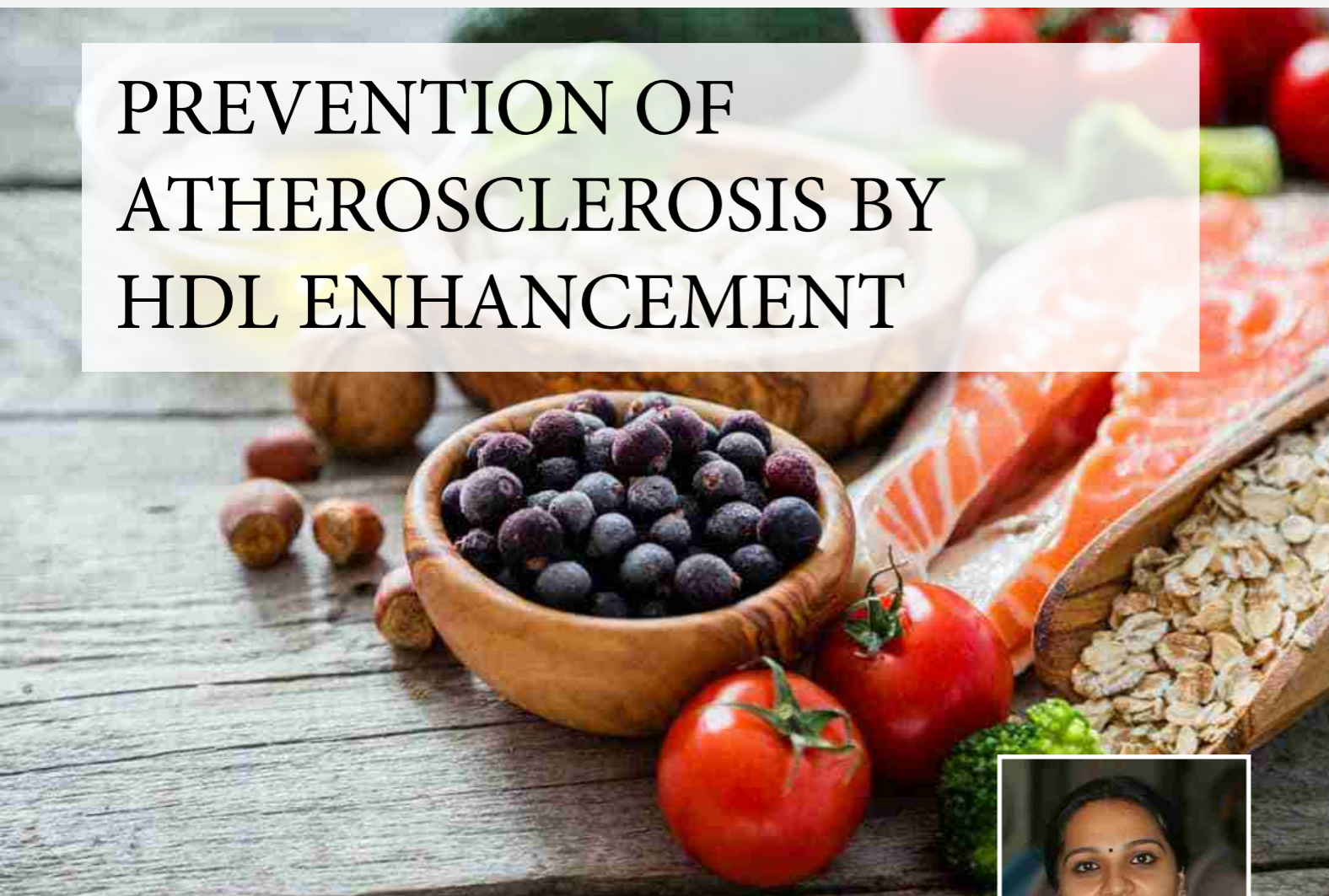
The National Program for Health Care of the Elderly under the Ministry of Health and

Family Welfare aims to bridge some of the gaps in the landscape of geriatric care in India through the setting up of Regional Geriatric Care Centres. However, a focus on dementia and Alzheimer's disease is missing. Currently, this gap is being filled by NGOs such as ARDSI who provide certification courses on dementia care. With an increasingly aging population, investments made in training healthcare professionals are likely to pay for itself as demand for these services is only going to increase. If India is to stay ahead of the curve and prepare for the needs of an aging population, a clearly defined public health strategy with a significant focus on research into degenerative diseases and investment in the training of healthcare personnel is much needed.

The government of Kerala

had introduced a project called 'Smritipadham'. Launched by the Department of Social Justice along with the Social Security Mission in association with ARDSI, it is an initiative on dementia where probably for the first time in any state, the government is roping in an NGO. It was started in 2015. Following it, one modal centre which provides round-the-clock care for patients has been started at Edavanakkad. Also, a dementia daycare centre was started in Guruvayur. The government has plans to start such model centres in all districts. However, solid plans are yet to be made. In Kerala, ARDSI has six chapters working for dementia and its various forms. It also has care homes in Trivandrum, Ernakulam, Kumbalangi, Kunnamkulam, Tripunithra and Kozhikode.

PREVENTION OF ATHEROSCLEROSIS BY HDL ENHANCEMENT



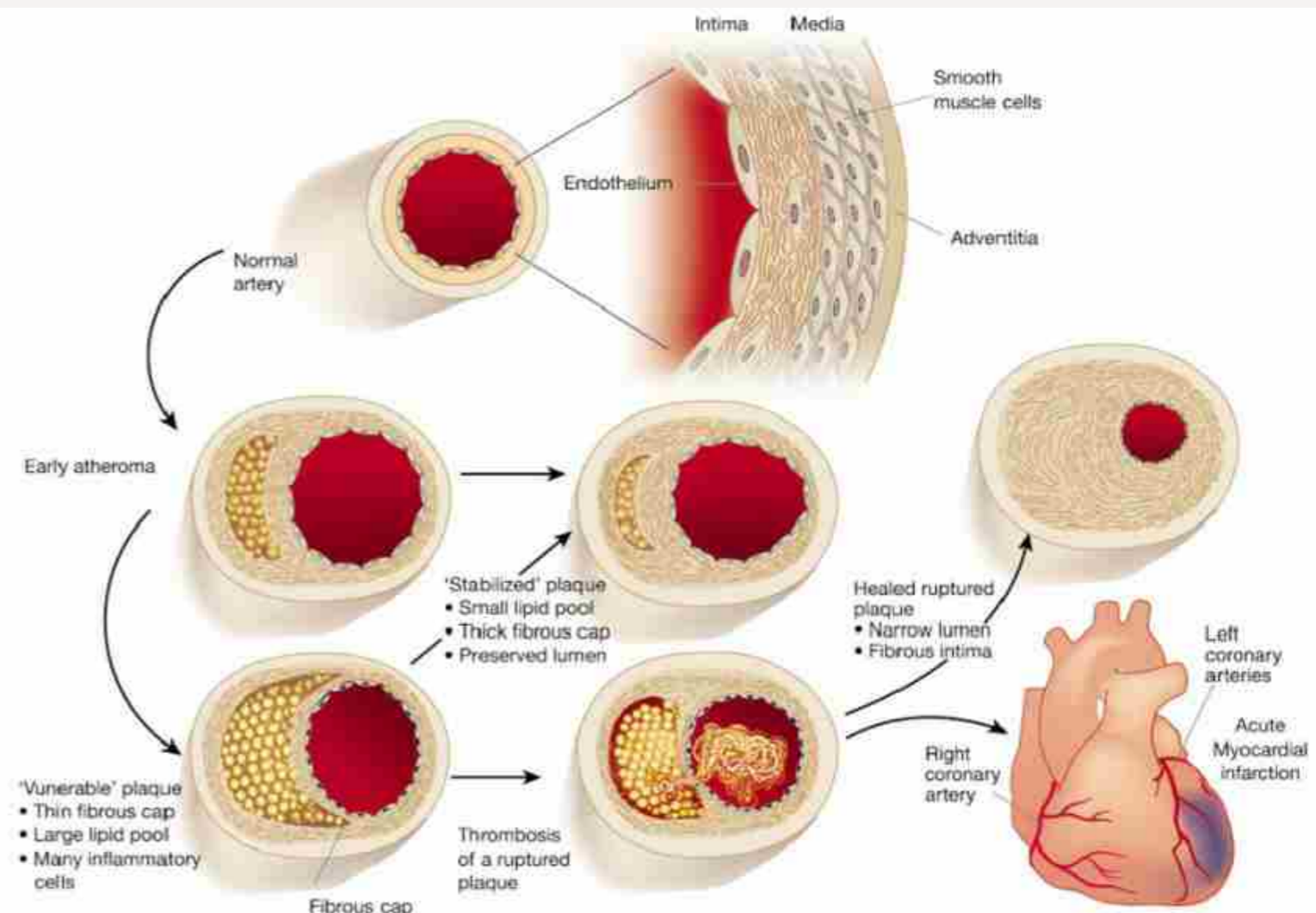
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Atherosclerosis and its complications are considered as one of the major cause of death due to cardiac disease. As atherosclerosis is a chronic disease requiring life time preventive therapy, the drugs used should be safe and inexpensive. Most of treatment options now available are oriented towards decreasing the LDL levels by exercise, diet and drugs. Long term therapies with

lipid lowering drugs are shown to have side effects like muscle pain and neuropathy. Studies have shown that increased HDL has an inverse relation with atherosclerosis. Various methods to increase HDL and the mechanism by which HDL reduces atherosclerosis are interesting area of research which requires further investigation. One of the reasons for the atheroprotective effect of HDL is its important

role in reverse cholesterol transport, the pathway by which the cholesterol is transported from the peripheral tissues to liver for metabolism and excretion. This article gives a brief review about reverse cholesterol transport pathway and new approaches targeted towards increasing the HDL and reverse cholesterol transport as a treatment modality for atherosclerosis.

Cardiovascular Disease



is the most common cause of morbidity and mortality all over the world. Approximately 50% death due to cardiovascular disease is attributed to atherosclerosis, a degenerative disease of arteries due to progressive lipid accumulation.

It is a multifactorial disease involving the interplay of genetic and environmental factors. Atherosclerosis is a slow, complex disease which may start in childhood and as people age, it progress. Foam cell formation is the key event in atherosclerosis and formation of atherosclerotic plaque is called atherogenesis.

The plaque then progress by smooth muscle cell migration and some times calcification. As the artery walls thicken, the pathway for blood narrows and decrease or block blood flow to important organs such as brain, heart and other tissues. The final event is the breakage of lesion that causes intravascular thrombosis leading to sudden cardiovascular or cerebrovascular events.

Several risk factors are identified in the patho-physiology of atherosclerosis. Hyperlipidemia represented by elevated level of triglycerides and low

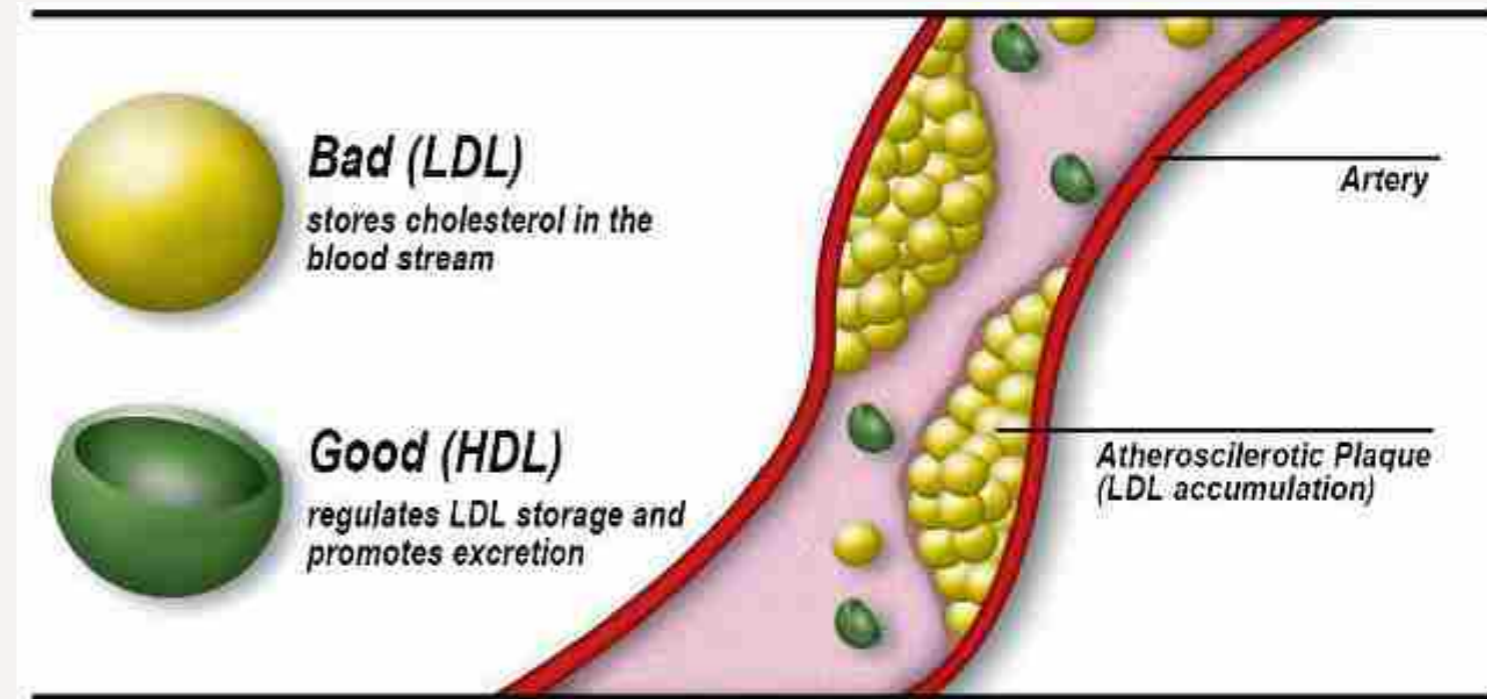
density lipoprotein cholesterol (LDL), are important determinant in atherosclerosis. LDL is the major cholesterol carrying lipoprotein to peripheral tissues. High LDL in blood is an independent risk factor. According to oxidation hypothesis, oxidized LDL can cause endothelial injury, migration of monocytes to sub endothelium, production of pro-inflammatory cytokines and enhance macrophage foam cell formation. On the other hand HDL which carries cholesterol from extra hepatic tissue and transport to liver for metabolism is considered to resist atherogen-



esis. High blood pressure exerts force against the artery walls. Over time, this extra pressure can damage the arteries, making endothelial wall more vulnerable to the injury and inflammation and also causes hypertrophy and proliferation of vascular smooth muscle cells. A higher blood pressure can induce the vulnerable plaque to rupture leading to cerebro and cardiovascular events. Smoking is yet another independent risk factor. Heavy smokers have increased free radicals in the circulation which can oxidize LDL and cause direct injury to endothelium causing permeability changes. Free radical can also enhance the production of proinflammatory cytokines by immune cells. Smoking is reported to

raise LDL cholesterol levels and blood pressure while lower a person's high density lipoprotein cholesterol. Diabetes and Insulin resistance have direct influence on the development of atherosclerotic plaque. High glucose can induce oxidative stress in the endothelial cell layer and enhance expression of adhesion molecules on the surface. Increased glucose can cause increased triglycerides in the body which eventually lead to high LDL. In type-2 diabetes, dyslipidemia characterized by elevated triglycerides and lower HDL is reported. Obesity is manifested by metabolic syndrome and is linked to diabetes and atherosclerosis. Obese persons have an increased risk of developing high blood

pressure, tend to have higher levels of cholesterol as a result of eating a high-fat diet and have an increased risk of developing diabetes. Lack of physical activity results in high blood pressure and obesity thereby worsen other risk factors for atherosclerosis. Age is also considered as a risk factor in atherosclerosis. However, it has been identified that atherosclerosis starts even at the early stages. As the body ages the elasticity of blood vessels decreases as well as the body antioxidant defence. Blood vessels become stiffer and less flexible increasing the chances of endothelial injury. Heavy Alcohol consumption can induce fatty liver as well as increase production of free radicals. This can lead to, hyper



lipidemia, hypertension and endothelial dysfunction predisposing atherogenesis (Muriel, 2009). Heavy drinking can also damage the heart muscles. However there are reports that lower consumption of alcohol may reduce heart diseases Apart from all these, family history of early heart disease is reported due to difference in gene expression. Accordingly familial lipid abnormalities have been identified.

Atherosclerosis is considered to be chronic inflammatory disease that results from interaction between oxidized low-density lipoprotein, activated endothelial cells, monocyte-derived macrophages, T Cells, and the arterial wall. An increase in plasma LDL leads

to an increase in adherence of circulating monocytes and increased rate of entry of LDL to arterial subendothelial space. Activated endothelial cells express adhesion molecules such as ICAM, VCAM which attract and recruit blood monocytes to the vessel wall. LDL undergoes oxidative modification by pro-oxidants like lipoxygenases and myeloperoxidase. The monocytes which differentiate into macrophages ingest oxLDL via scavenger receptors forming foam cells, and thereby promoting plaque formation.

Reverse Cholesterol Transport (RCT) is a pathway that transports cholesterol from extra hepatic cells and tissues to the liver and intestine for excretion. By reducing accumulation of

cholesterol in the wall of arteries, RCT prevent development of atherosclerosis. The concept of RCT was first proposed by Glomset in 1968. This concept represents the most widely accepted mechanism underlying the HDL hypothesis which proposes that pharmacological intervention to raise HDL will reduce cardiovascular risk.

The first step in reverse cholesterol transport is the efflux of cholesterol from macrophages. The cholesterol efflux is mediated by ABCA1, ABCG1, SR-B1 or by passive diffusion. ABCA1 transfers free cholesterol to nascent HDL containing Apo A1, where as ABCG1 and SR-B1 transfer free cholesterol to mature HDL. Within the mature HDL the free cholesterol



transferred is esterified by an enzyme Lecithin Cholesterol Acyltransferase (LCAT) to form cholesteryl ester (CE). Cholesteryl Esters are transported to liver by direct or indirect pathway. In direct pathway the HDL with CE gets attached to SR-B1 receptor. In indirect pathway the HDL transfers CE in exchange with phospholipid to apo-B containing lipoproteins like VLDL and LDL with subsequent uptake in the liver via the low density lipoprotein receptor (LDLR). The exchange of CE and phospholipid is mediated by Cholesteryl Ester Transfer Protein (CETP) and Phospho-

lipid Transfer Protein (PLTP). The CE taken up by the liver will be acted upon by the enzyme hepatic lipase and the cholesterol will be metabolized and excreted through bile or faeces. Primary prevention of atherosclerosis mainly involves life style changes like Cessation of cigarette smoking, Exercise, Healthy eating habits, Weight loss, Control of hypertension, Lowering total cholesterol and LDL while increasing HDL. Secondary prevention involves use of medications like Aspirin (anti-platelet agent), Statins, Niacin and beta blockers (to control cholesterol), and

surgical interventions like coronary artery bypass surgery, Angioplasty.

Lifestyle changes, such as eating a healthy diet, reducing stress and regular exercise, are often the first line of defense in treating atherosclerosis. It can help to prevent or slow the progression of atherosclerosis.

Smoking or use of tobacco damages the arteries. Quitting is the best way to halt the progression of atherosclerosis and reduce the risk of complications.

Regular exercise can condition the muscles to use oxygen more efficiently. Physical activity can also improve circulation and

promote development of new blood vessels. Exercise helps lower blood pressure and reduces risk of diabetes. Aim to exercise at least 30 minutes most days of the week. If you can't fit it all into one session, try breaking it up into 10-minute intervals. Stairs can be used instead of the elevator, walk around the block during the lunch hour, or do some sit-ups or pushups while watching television. All these will reduce the chance of atherosclerosis.

Stress Management: Practice healthy techniques for managing stress, such as muscle relaxation and deep breathing. Taking a small nap, other hobbies like listening to music, playing with pets all will help

to reduce the stress

HDL Enhancement: HDLs oppose atherosclerosis directly, by removing cholesterol from foam cells, by inhibiting the oxidation of LDLs with help of HDL associated enzyme Paraoxonase, by limiting the inflammatory processes that underlie atherosclerosis by exhibiting antiinflammatory property. HDLs also have antithrombotic properties and prevent formation of thrombus or clot. Nicotinic acid (niacin) is the most effective HDL-raising drug currently available, which increases HDL by 15% to 35%. Fiber rich materials like Bananas, guava, pomegranate, mangoes apples, and pears, Whole grains, including bran, cereals,

oats, Oils (Fats): Unsaturated fats like: olive oil, soybean oil etc., Omega-3 fatty acids: found in fishes like salmon, herring, and mackerel., Nuts: Brazil nuts, almonds, pistachios, peanuts etc. are also enhances HDL level.

LDL and HDL bears opposing relation with atherosclerosis LDL promotes atherosclerosis whereas HDL is atheroprotective. One of the important thing which we have to keep in mind is Atherosclerosis is a preventable and treatable condition and its progression can be prevented by making appropriate changes in our life style and in some situations medications or surgical interventions may be required.



GENETIC COUNSELLING; TOWARDS A BETTER FUTURE!

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“Genetic counselling is the process of helping people understand and adapt to the medical, psychological and familial implications of genetic contributions to disease.”

Genetic counselling emerged as an offshoot of Eugenics concept of Carl Rogers. The term “genetic counselling” was first used by the scientist Sheldon Reed in 1947. The Heredity Clinic was the first genetic counselling service centre established in 1940 at the University of Mich-

igan, USA. Genetic counselling is about empowering patients to make decisions that are right for them. This is especially true in prenatal settings. Genetic counselling service may be useful at all stages of development, for instance babies undergoing screening, teenagers being tested for Thalassaemia genes or assessing the genetic predisposition of adults as they enter mid-life to accommodate for lifestyle changes. With the mapping of the human genome project in 2001 the role of genetic coun-

selling has increased further both in scope and importance.

Genetic counsellors:

Genetic counsellors are highly skilled healthcare professionals, with a sound knowledge of medical genetics and genomics. They will also have advanced training in counselling and communication skills. Genetic counsellors help people understand genetic information. They pay particular attention to



the therapeutic relationship they have with patients - allowing space to explore the emotional, ethical and family issues raised by genetic information. The three key aspects of their work are empathy, unconditional positive regard and congruence (being genuine). Developing these skills remains important in genetic counselling training.

To put in simple terms, a genetic counsellor engages in a one to one rapport with the ‘patients’, approaches with empathy, helps them to unveil the anxieties or speculations they have regarding their next generation, their siblings, or sometimes themselves. Unfolding family history plays a pivotal role in genetic counselling and it remains a major hurdle to many genetic counsellors; as the clients may not be in a position to reveal their family history to a stranger(!). It is also equally probable that they may conceal some facts that might be crucial in designing the modus operandi of the counsellor. Taking a family history can also function as a psychosocial tool. It provides an opportunity for a genetic counsellor to listen to a patient’s family narrative, to understand their experience of a condition, to begin to appreciate

some of the family dynamics and to gain some understanding of a patient’s own anxieties and agendas.

The family tree is a good way of finding out about someone’s family history. Correct information is key when making a risk assessment. A family tree, though, is more than simply an information-gathering tool. Drawing out a family tree can help patients tell their stories. This can be helpful in understanding how a family functions. This is really important when thinking about how they might communicate information. A well annotated family tree allows counsellors to make a genetic risk assessment (i.e. what are the chances that a couple could have a child affected with a genetic disorder?)

Changing approaches:

Initially the genetic counsellors were mainly concerned with chalking out the probabilities a couple have with respect to genetic disorders, and communicating the same to their clients. Pre - natal diagnostic tests like Amniocentesis and Chorionic Villi sampling (CVS) were routinely prescribed for a genetic counselling session.

For CVS the sample is taken from the placenta and can be carried out from about 11 weeks of pregnancy. Amniocentesis involves taking a sample of the amniotic fluid that sits around the baby, and is performed a bit later, from about 15 weeks of pregnancy. In developed countries, the genetic counsellors work with their colleagues, such as midwives and obstetricians, who will organise and perform these tests. A new technology is also available changing prenatal testing called non-invasive prenatal testing (NIPT). A certain amount of foetal DNA circulates in maternal blood. It is now possible to isolate this DNA from the pregnant woman’s blood for genetic testing.

Genetic counsellors now work in many capacities besides the traditional setting of the hospital. They work in the fields of education, administration, policy-making, as well as for biotechnology companies as representatives. Many also work shoulder-to-shoulder with scientists and with medical doctors in interpreting test results. Amidst the technological revolutions, compassion and deep respect for patient autonomy will remain at the heart of genetic counselling.

E

ELEPHANT

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Elephant, (family Elephantidae), largest living land animal, characterized by its long trunk (elongated upper lip and nose), columnar legs, and huge head with temporal glands and wide, flat ears. Elephants are grayish to brown, and their body hair is sparse and coarse. They are often found in savannas, grasslands, and forests but occupy a wide range of habitats, including deserts,

swamps, and in tropical and subtropical regions of Africa and Asia.

Elephants are placed within the suborder Elephantoida, in the order Proboscidea. The first identifiable ancestors of today's elephants were small animals that lived 50–70 million years ago and stood about 2 ft (0.75 m) tall. The suborder Elephantoida originated in North Africa long

before that region became extensively desertified, and from there elephants spread to every continent except Australia and Antarctica. The group once included three families, several genera, and hundreds of species. Mammoths and mastodons also belonged to the sub-order Elephantoida, but these species became extinct about 10, 000 years ago. About 400, 000 years ago Asian elephants inhabited

a much wider range than they do today, including Africa. This species now survives only in southern Asia, from India to Sumatra and Borneo. There are two main types of elephants: the African elephant and the Asian elephant.

There are two subspecies of the African elephant: the savanna (or bush) elephant (*Loxodonta africana*) and the forest elephant (*Loxodonta cyclotis*). However, forest elephants may be a distinct species of an elephant instead of a subspecies. There are three subspecies of Asian elephant: the Indian elephant (*Elephas maximus indicus*), the Sri Lankan elephant

(*Elephas maximus maximus*), and the Sumatran elephant (*Elephas maximus sumatranus*). Another possible subspecies is *Elephas maximus borneensis* (Borneo pygmy elephant). According to the World Wildlife Fund, the Borneo pygmy elephant is genetically different from other Asian elephants as determined by DNA evidence.

African elephant - African elephants are bigger than Indian elephants. The African elephant has wrinkly gray skin, a swayed back, and two tips at the end of its trunk that it can use like fingers to pick stuff up. Both males and females have tusks.

It has larger ears, too.

Indian elephant - The Indian, or Asian elephants are smaller than the African elephants. Their ears are smaller. They have more of a humped back and only one fingerlike tip at the end of their trunk. Also, their skin tends to be less wrinkly than the African elephant.

Asian and African elephants can be distinguished by the shape of their backs, the Asian having a convex gently sloping back and the African a concave or saddle-shaped one. Male elephants are much larger than females. Skin texture varies from the tough, thick, wrinkled, folds on



the back and forehead, to the soft, thinner, pliable skin of the breast, ears, belly, and underside of the trunk. The tough skin bears a few, scattered, bristly hairs, while the thinner skin on the trunk, chin, ear rims, eyelids, knees, wrists, and the tip of the tail has somewhat thicker hair. Daily skincare includes showers, dusting with sand, and full-bodied mud -packs which are later rubbed off against a tree or boulder, removing dead skin as well. These activities help to keep the skin moist, supple, protected from the sun and insects, and also aid in keeping the animal cool.

Elephants produce two types of vocalization by modifying the size of the nostrils as air is passed through the trunk. Low sounds are the growl, rolling growl, snort, and roar; high sounds are the trump, trumpet, pulsated trumpet, trumpet phrase, bark, gruff cry, and cry. Rumbling sounds, initially thought to be caused by intestinal activity are now known to be produced by the voice box (larynx) and are considered to be similar to purring in cats.

Elephants live in small family groups led by old females (cows). Where food is plentiful, the groups join together. Most males (bulls) live in bachelor herds apart from the cows. Males and females both possess two

glands that open between the eye and ear. Elephants of all ages and sexes secrete a fluid called temporin out of this orifice. Males, however, enter a “musth period,” during which they secrete a fluid differing in viscosity from the fluid secreted when they are not in musth. Serum testosterone during musth is higher than in a non-musth elephant, and the animal’s behaviour is erratic; they are uncontrollable (musth is Hindi for “intoxicated”), sometimes even by their handlers (mahouts). Musth is the time for establishing reproductive hierarchy, which can differ from the usual social hierarchy in that a male in musth outranks non-musth males. In the wild, males are usually at their prime physical state during musth and ordinarily do most of the breeding. Elephants can assess the reproductive status of one another by using their keen sense of smell. Inside the skull, elephants possess from seven to nine nasal turbinates with specialized sensitive tissues for olfaction. (Humans have only three turbinates; dogs have five.) When a female is in estrus, or when a male is in musth, an elephant apparently can detect airborne hormones. Once “collected,” the information is then passed to the Jacobson’s organ, located on the roof of the

mouth. This organ conveys the molecules to the brain for analysis. Hormones are also sniffed directly from urine and feces. Gestation is the longest of any mammal (18–22 months). The newborn elephant is about a meter (3.3 feet) tall and weighs about 100 kg (220 pounds). It suckles by using the mouth, not the trunk, at mammary glands located in the chest region. Weaning is a long process and sometimes continues until the mother can no longer tolerate the pokes of her offspring’s emerging tusks. After weaning, many hours of each day are spent eating. Elephants reach sexual maturity early in their second decade of life. African elephants become sexually mature at age 10–12, whereas Asian elephants become sexually mature about age 14. It is during that period that males leave their natal herd (herd of origin) to live either singly or in small herds with other males. Females, in contrast, remain with their natal herd for their whole lives. Despite living apart, adult male and female elephants form short-lived mating or feeding associations with one another.

Because of their high intelligence level, elephants can adapt to and modify habitat, while their wide range of food choices permits habitation of a

diverse range of ecosystems, including forests, woodland, savanna, grassy forests and sparsely vegetated desert. Elephants need massive quantities of food, perhaps 300-350 lb a day, although proportional to their body weight elephants eat less than mice. The diet of elephants includes roots, bark, grass, leaves, berries, seedpods and other fruits. They may drink up to 50 gallons of

water a day, and after drinking their fill, will splash themselves with water and mud, wash their young, and sometimes just frolic, tossing and squirting water about while their young splash, play, and roll in the mud.

Elephants teach and learn by behavioural examples and talk with vocalized sounds. Each

elephant troop has its home range, but territorial fights are rare even though ranges often overlap. Most African elephants are now restricted to the protection of national parks. The average lifespan of elephants is about 65 years of age or more.



F

FLAMINGO

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Flamingo, (order Phoenicopteriformes), any of six species of tall, pink wading birds with thick down-turned bills. Flamingos have slender legs, long, graceful necks, large wings, and short tails. They range from about 90 to 150 cm (3 to 5 feet) tall. The name “flamingo” comes from the Portuguese and Spanish word flamenco, which means

“flame-colored.” The genus name *Phoenicopterus* comes from the Greek word phoinikopteros, which means “blood red-feathered.”

There are six flamingo species American flamingo (*Phoenicopterus ruber*), Andean flamingo (*Phoenicopterus andinus*), Chilean flamingo (*Phoenicopterus chilensis*), Greater flamingo (*Phoenicopterus roseus*) Lesser

flamingo (*Phoenicopterus minor*), Puna(James’) flamingo (*Phoenicopterus jamesi*) There are six different species of flamingo found around the world. The different flamingo species are the greater flamingo which is the most widespread species of flamingo found in Africa, Southern Europe and Southern Asia. The lesser flamingo is the

most numerous species of flamingo and is found in Africa and Northern India The greater flamingo is the largest bird, ranging from 3.5 to 5 feet tall and weighing between 4.4 and 8.8 pounds. The lesser flamingo is the smallest bird, with a height of 2.6 to 3 feet and weight of 2.6 to 6 pounds.

Flamingos prefer shallow aquatic habitats, including tidal flats, lagoons, lakes, swamps, and Islands . flamingo is a large colourful bird found both in South America and Africa. The flamingo is also found in the

warmer areas of southern Europe and western Asia. In India, this bird is commonly seen in Kutch, Gujarat, and Mumbai. It also reaches Kerala after flying thousands of km during the months of October-January. The wetlands of Enamavu and nearby areas of Pavaratty in Thrissur district have taken a different hue with the arrival of greater flamingos in droves to the region. These ravishing birds have been spotted at the wetland near Ponnammutha bridge on the Venkitangu-Kannoth road. As observed over the years, in Ker-

ala greater flamingos could be spotted at Thattekkad Bird Sanctuary, near Kothamangalam; parts of Kannur; Kozhikode beach as well as in a few areas of Thrissur and Alappuzha.

Flamingos have long legs, large curved bills, and plumage in shades ranging from white or gray to pink or orange. Members of some species may have black bills and some black feathers. The legs of adult flamingos are longer than the flamingo’s body measuring between 80 to 125 cm depending on the species. Neck is long and sinuous. A flamingo



has 19 elongated cervical (neck) vertebrae allowing for maximum movement and twisting. The eyes are located on either side of the head. Flamingo chicks have gray eyes for approximately the first year of life. Adult flamingos have yellow eyes . An adult flamingo's bill is black , pinkish or cream coloured. Colouration varies according to species. A flamingo's large, fleshy tongue is covered with bristle-like projections that help filter water and food particles through the lamellae. Adult feathers have a small , delicate, accessory feather arising from

the main feather at the point where the quill merges into the shaft of the feather. This is called an aftershaft.

Flamingos are omnivores that feed on blue-green algae, brine shrimp, insect, crustaceans, and mollusks. They stir up mud with their feet and dip their bills upside-down in the water to filter food. The pigment molecules in their food (carotenoids) give flamingos their pink to reddish color. Flamingos that feed primarily on blue-green algae are darker than those that get the pigment second-hand

from crustaceans. Flamingos that don't get carotenoids from their diet may be perfectly healthy, but are gray or white.

Groups of flamingos can gather into one big group called a colony, and they do everything together. They eat at the same time and sleep at the same time. They also mate around the same time. Before flamingos pick a mate, the colony even does a special dance together! Colony life helps the birds establish nesting sites , avoid predators and find food efficiently. Although flamingos only nest once a year,



flamingo colonies are known to breed at any time of the year. A flamingo reaches sexual maturity (which means the flamingo is able to breed) when the flamingo is between 3 and 6 years old. Flamingos build their nests out of mud, stones and feathers and do so about 6 weeks before they lay their eggs. Flamingos tend to lay just one egg that hatches after a 30 day incubation period. Both the mother flamingo and the father flamingo are known to help to raise the flamingo .

In order to fly, flamingos need to run a few paces to gather speed. This speed is not related to the ground but rather to the air, so they usually take off facing into the wind. In flight, flamingos are quite distinctive, with their long necks stretched out in front

and the equally long legs trailing behind. Their outstretched wings showcase the pretty black and red (or pink) coloration that, with slight variations, is shared by all flamingo species. When flying, flamingos flap their wings fairly rapidly and almost continuously. And, as with most other flamingo activities, they usually fly together in large flocks. The flamingos follow each other closely, using a variety of formations that help them take advantage of the wind patterns. Flamingos have a famous habit of standing on one leg. Scientists aren't certain, but they believe that flamingos can save more energy standing on one leg than on two. Their long and lanky legs have a special feature where they are able to "lock" their leg into place so it requires

zero effort to stand .Flamingos at rest, standing on one leg. This posture may be used by the birds to conserve body heat.

Flamingos are highly susceptible to water pollution and lead poisoning. Reproductive success decreases when the birds are disturbed by tourists, low-flying aircraft, and predators. Other threats include climate change, water level changes, and diseases. Adults and eggs of some species are killed or collected for food or pets. Human hunters, wild dogs and crocodiles are the main predators of the flamingo, along with eagles that prey upon the flamingo eggs and vulnerable flamingo chicks.

ALZHEIMER'S DISEASE

NANDHU KRISHNA,
IInd Year B. Sc. Zoology.

Alzheimer's disease is a neurological disorder in which the death of brain cells causes memory loss and cognitive decline. This disease was first described in 1906 by a German neuropathologist Alois Alzheimer. This is thought to be caused by the abnormal build-up of proteins in and around brain cells. One of the proteins involved is called amyloid, deposits of which form plaques around brain cells.

Though the cause of initiation is not fully known, scientists now know that it begins many years before the appearance of symptoms. As brain cells are affected there's also a decrease in chemical messenger (called a neurotransmitter) involved in sending messages or signals between brain cells. Signs and symptoms

- Occasional misplacing of items
- Minor short-term memory loss

- Failure to recollect exact details
- Forgetting names of family members or friends
- Arousal of confusion in situations outside the familiar

Memory Loss



- Great difficulty remembering recently learned information
 - Deepening confusion in many circumstances
 - Problems in speaking
 - Repeats the same conversations
 - More abusive, anxious, or paranoid
- There is no cure for Alzheimer's

disease. However, several therapeutic agents can be used to slow disease progression or to alleviate symptoms. In roughly 50 percent of patients, the progression of amnesic MCI can be delayed for about one year by drugs called acetylcholinesterase inhibitors. These drugs which include galantamine, donepezil, and rivastigmine, work by slowing the breakdown of acetylcholine.

Symptoms of Alzheimer's disease can be reduced in some patients by the drug memantine which decreases abnormal brain activity by blocking the binding of glutamine to certain receptors in the brain. This drug can improve cognition and enable patients to become more engaged in daily activities.

As the exact causes of Alzheimer's disease are still unknown there's no certain way to prevent the condition. But a healthy lifestyle can help reduce the risk.

MEDI
TALK



ASIAN ELEPHANTS: THREATS AND CONSERVATION

Dr. SURESH K GOVIND,
Assistant Professor (Zoology), Christ College, Irinjalakuda.



There are four elephant reserves in Kerala and they are Wayanad, Nilambur, Anamudi and Periyar. Major threats faced by the Asian elephant (*Elephas maximus*) are crop damage, human-casualties,

and estimate the crop damage by wild elephants and to identify its causative factors. Human-elephant conflict was recorded in 21 areas in the district (Figure 1). The highest damage was reported from Pattikkad Forest Range (Rs.

immediate fringe areas of forest is the cause for increased human-elephant conflict. Higher palatability and nutritive value are the reasons for preferring the cultivated crops by wild elephants. At present, solar-electric fence and trench are

the remedial measures for preventing elephants from entering the human habitations. Introduction of short term remedial measures are quite

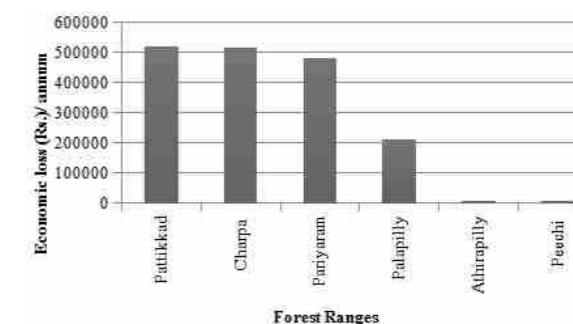
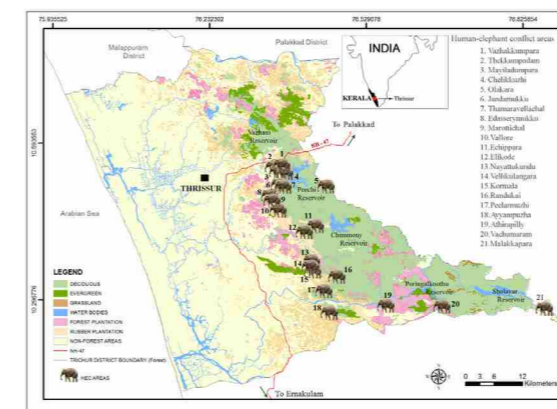


Figure 2. Economic loss due to Asian elephants in different Forest Ranges (Thirissur District, Kerala)

household damage and poaching for ivory. The shortage of food and water within the forest limits, agricultural expansion, monoculture cultivation, timber and fuel wood extraction, livestock grazing and fire are the main reasons for human-elephant conflicts. Crop damage by wild Asian elephants is a serious problem faced by the farmers in the fringe areas of forest in Kerala. A study was carried out in Thirissur District (10° 46' to 10° 7' N & 75° 57' to 76° 55' E), Kerala, India from April 2009 to March 2013, to assess

5,17,451.4/- per annum) and the mean economic loss was estimated as Rs. 17,35,625/- per annum (Figure 2). Plantain (*Musa paradisiaca*) (74.11%) was the main food of elephants. It was cultivated in between the young rubber plants (*Hevea brasiliensis*) (below four years) near the forest boundary. While consuming plantains, the unpalatable rubber plants were also destroyed. As the market price of rubber increased, farmers were expanding the cultivation of rubber, followed by plantains. The cultivation of plantain in the

relevant, especially in the areas where crop damage by elephant is only once in a year. In Africa, several innovative methods to deter elephants from cultivated areas were experimented and the sensitivity of elephants towards chilli was identified. Efficacy of chilli-tobacco rope against the crop raiding elephants in India was evaluated and it was significantly better in low-rainfall seasons than medium and high-rainfall regimes. Beehive fence was also proved as an effective control measure to mitigate conflicts.

GUEST OF KKTm



GOPIKA RAJ, IInd Year B. Sc. Zoology.

The Asian Paradise Flycatchers (*Terpsiphone paradisi*) - also known as the Common Paradise Flycatchers - occur naturally in Asia, where they have an extremely large range.

Asian Paradise-flycatchers occur naturally from Turkestan east to Manchuria, and south to India and Sri Lanka and west to the Malay Archipelago where they inhabit the islands of Sumba and Alor (Lesser Sundas). In Singapore, these birds are now regionally extinct. Vagrants have

been reported in Korea and the Maldives, where they are rare. Migratory populations travel south to spend the winters in tropical Asia. Both migratory and resident populations exist in southern India and Sri Lanka. They inhabit thick forests and well-wooded areas.

The adult Asian Paradise Flycatcher measures about 7.5 - 8.7 inches (19 - 22 cm) in length, not including the long tail, which can grow to 9.4 inches (24 cm) in length, and the male's two central, elongated tail

streamers up to 12 inches (30 cm). Their wings are between 3.4 - 3.6 inches (86 - 92 mm) long. Their legs are relatively short, and they tend to sit very upright when perched, similar to shrikes. Head of Asian paradise flycatcher is glossy black with a glossy black crown and crest. They have short legs and sit very upright whilst perched prominently. The bill is round and sturdy and bluish black in color. Eyes are black, and the eye rings are bluish black.

Males occur in two phases



(morphs) - one with a rufous (chestnut) upper plumage (rufous phase or morph) and another with a predominantly white plumage (white phase). The latter doesn't occur in all races. Intermediates of both forms also exist. Some birds also molted from rufous into white plumage .

- Rufous-Morph: The wings and the tail are mostly rufous colored, and the plumage below is white. The crown and crest is glossy black. mostly rufous with some white in the wings or tail

- White-Morphs: The body and wings are white. Long-tailed white-plumaged birds have dark shaft streaks (absent in rufous

birds). The edges of the wings and tail feathers are sometimes black.

The eye rings are blue. The long, narrow tail is rounded at the tip, except for the two streamers that droop. The black bill is round and sturdy.

Female are rufous on the back with a greyish throat and underparts, and they have short tail with rufous wings and a black head. Females have greyish throat and underpart, the eyes are black with bright bluish rings around the eyes.

Asian Paradise Flycatchers mostly feed on insects captured in the air. They usually hunt in the understory of densely can-

opied trees. The Asian Paradise Flycatcher mostly breed from May to July. These birds are monogamous and both share the duties of constructing the nest, incubating the eggs, and feeding and protecting the young. A clutch usually consists of 3 - 4 eggs, which are incubated for about 21 - 23 days. The cup nest is made out of twigs bound together with spider webs and is usually situated on the end of a low tree branch. Asian Paradise Flycatchers are quite noisy and their most common calls are described as sharp zweet's.

Asian Paradise Flycatcher frequently visit our college during the month of October- November.

Calendar

SEPTEMBER 2020



Japan Dolphin Day

September 1



Amazon Rainforest Day

September 5



International Vulture Awareness Day

First Saturday of September



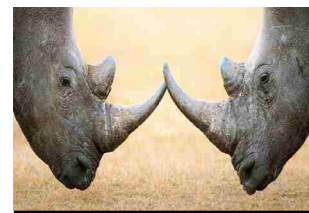
International Day for the Preservation of the Ozone Layer

September 16



World Water Monitoring Day

September 18



World Rhino Day

September 22



World Gorilla Day

September 24



World Cassowary Day

September 26

FAREWELL



SHIHABUDEEN A. S

Since you are bidding farewell to us, please know that your legacy would forever remain deep in our hearts. Thank you for being a great teacher, friend and supervisor.

Welcome



Dr. Seema Menon

Hearty welcome to Dr. Seema Menon to Department of Zoology.

W	S
O	E
R	A
D	R
	C
	H

There are 20 words to find in the grid below...

Z	G	A	A	W	B	T	K	P	S	B	A	G	G	N
N	C	A	A	Q	S	E	I	G	A	Y	J	N	N	A
G	D	V	P	U	E	I	A	R	C	K	O	F	I	K
P	E	Z	N	M	A	N	R	C	H	F	R	M	H	X
S	A	A	Z	V	W	I	E	Y	H	X	I	H	S	Y
C	M	C	N	A	E	C	A	T	S	U	R	C	I	T
I	I	A	I	R	E	N	I	H	P	L	O	D	F	N
K	K	T	R	F	D	G	E	T	I	C	C	B	V	A
E	R	E	N	D	I	L	O	R	C	X	O	G	O	I
S	E	A	F	A	T	C	Q	H	U	R	R	G	T	D
F	P	E	H	R	L	T	U	E	N	E	A	H	P	N
V	Y	M	U	S	W	T	Q	W	B	U	L	V	H	I
N	M	T	A	O	L	F	A	E	F	A	L	G	A	E
Y	H	Z	K	U	W	G	C	X	O	A	B	N	B	F
B	M	L	E	Z	L	I	S	L	A	N	D	S	O	G

ALGAE
BEACH
FISHING
ISLANDS
TIDE

ARCTIC
CORAL
FLOAT
PACIFIC
TSUNAMI



ATLANTIC
CRUSTACEAN
ICEBERG
SEAWEED
TURTLE

BARRIER REEF
DOLPHIN
INDIAN
SHARK
WAVES