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HAEMOPHILIA: A BLEEDING DISORDER

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Abstract

Haemophilia, a blood clotting disorder, is an inherited disorder in which one of the proteins needed to form blood clots is missing or reduced which affects the blood's ability to clot. There are three different types of Haemophilia, A, B, and C. Haemophilia A and B are the best known types of haemophilia than haemophilia C. Haemophilia is a genetic disorder i.e. it is the result of a change in genes that was either inherited or occurred during development in the womb. Haemophilia affects people from all racial and ethnic groups.

Introduction

Normally when a people get an injury or cut his/her body naturally protect itself. Clotting of the blood takes place at the cut site. A clot helps to stop bleeding after a cut or injury. In the first step of clotting the platelets of the blood plug the cut and then along with proteins form fibers. These fibers make the clot stronger and the bleeding become stops. These proteins which are helpful in making clotting fibers are known as clotting factors. There are 12 clotting factors I to XII in the human.

Haemophilia is a disease that weakens the body's ability to make blood clots, causing people to bleed for a longer period of time than normal. Sometimes it is so dangerous, become fatal resulting into death of the person.

Haemophilia is sometimes referred to as 'the royal disease' because it affected royal families across Europe in the 19th and 20th centuries. Queen Victoria, who ruled England between 1837-1901, is believed to have been a carrier of Haemophilia B or factor IX deficiency. She passed the trait on to three of her nine children. Her son Leopold died of a haemorrhage after a fall when he was 30. Two of her daughters, who married into other royal families across Europe, are believed to have been carriers who passed on the faulty gene to their children (1). Later on it was found that this blood disease affecting the royal families of Great Britain, Germany, Russia and Spain also.

Haemophilia

A haemophilic person when injured does not bleed harder or faster than a person without haemophilia, but he bleeds longer. Small cuts or surface bruises are usually not a problem, but more traumatic injuries e.g., injury during accident may result in serious problem called "bleeding episodes". Bleeding can occur anywhere in the body, such as into the joints, muscles or digestive tract. There are different levels of haemophilia. Some people have mild disease, some moderate and some more severe depending upon the level of clotting factors in plasma.

- People with mild haemophilia have 5% up to 50% of the normal clotting factor in their blood. In many cases, mild haemophilia is not diagnosed until an injury, surgery or tooth extraction results in prolonged bleeding.
- People with moderate haemophilia have 1% up to 5% of the normal clotting factor in their blood. They tend to have bleeding episodes after injuries and some without obvious cause. These are called spontaneous bleeding episodes.
- People with severe haemophilia have <1% of the normal clotting factor in their blood. They have bleeding following an injury and may have frequent spontaneous bleeding episodes, often onto their joints and muscles.

Types of Haemophilia

Haemophilia is a congenital bleeding disorder caused by deficiency of clotting factors or proteins. There are three different types of Haemophilia A, B, C related to deficiencies of three different clotting factors; factor VIII, factor IX and factor XI respectively. A person with haemophilia will lack only one factor, either factor VIII or IX or factor XI. The discovery of multiple forms of haemophilia was first made in 1944 (2).

Haemophilia A: Haemophilia A or classic haemophilia caused by deficiency in factor VIII (F VIII). The F VIII protein is required for propagation of the intrinsic coagulation pathway (3). It is most common of the inherited bleeding disorder and occurs one in 5000 male births. (Ref.4,5). Patients with severe haemophilia have an absence of circulating plasma F VIII activity, resulting in spontaneous bleeding affecting joints and soft tissues. Without treatment, recurrent bleeding results in the development of chronic arthropathy (i.e., bleeding in joints at knee, elbow and ankle) and early mortality (6).

Haemophilia B: Haemophilia B is another bleeding disorder associated with a deficiency of clotting factor IX. It is caused by a mutation of the factor IX gene leading to deficiency of factor IX (F IX). It is the second most common form of haemophilia occurs in about one in 25000 male births. It is also called Christmas disease, named after Stephen Christmas, an 8 year old boy, the first patient with this disease in 1952. The first report of this identification was published in British Medical Journal in 1952(7,8). It causes spontaneous bleeding into joints, which can lead to joint deformity and arthritis at an early age (6).

Haemophilia C:Haemophilia C also known as Rosenthal Syndrome is caused by low levels of factor XI (F XI). Factor XI (FXI) deficiency was first described in 1953 (2). It is also associated with bleeding but haemophilia C differs from haemophilia A and B in cause and bleeding tendency. Haemophilia C is a mild form of disease, generally occurs in one of every 100,000 people. It is also known as Plasma Thromboplastin Antecedant (PTA) deficiency (4). People with this rare type of haemophilia often do not experience spontaneous bleeding. It lacks joint bleeding and infrequent need for treatment. Bleeding typically occurs after trauma or surgery and is commonly seen in sites that have a high fibrinolytic rate, such as the genitourinary tract (10). Incidence of haemophilia C is increase among people with Noonan syndrome (10).

Genetics of Haemophilia

The gene of haemophilia is a recessive character. Haemophilia is an inherited disease that shows an X-linked pattern, which means the faulty gene is located on the X chromosome. Everyone inherits two sex chromosomes, X and Y, from his or her parents. A female inherits one X chromosome from her mother and one X chromosome from her father (XX). A male inherits one X chromosome from his mother and Y chromosome from his father (XY). Males only have one copy of the X chromosomes and so a single mutation is enough to cause haemophilia. Females have two X chromosomes and the mutation is needed in both copies of the gene to cause haemophilia. A female with one mutated X chromosome and one healthy X chromosome is known as carrier. She may experience haemophilia symptoms but not a severe form of the disease. As a carrier, she can pass the gene on her children. Because it is an X-chromosome –linked condition, males are more affected and therefore more frequently diagnosed.

Haemophilia A (HA) and B (HB) are an X-linked bleeding disorder caused by heterogeneous mutations in the factor VIII gene (F 8) and factor IX gene (F9) respectively. F 8 gene maps to the distal end of the long arm of X-chromosome (Xq 28) and spans 186 kb of genomic DNA (11, 12). The factor IX gene (F9) is located at chromosome X (Xq 27) and 34 kb in size (13). A mutation of the F8 gene or F9 gene on the X chromosome causes haemophilia A or B. The mutation means that not enough of protein clotting factor VIII or factor IX is made (1). The factor XI deficiency, a cause of haemophilia C (HC) is inherited as an autosomal recessive pattern. The gene for factor XI is located on chromosome 4, an autosomal chromosome (10). Thus both parents must carry the gene to pass it on to their children. The haemophilia C affects both genders equally.

Diagnosis and Treatment of Haemophilia

Pedigree analysis and clotting factors VIII levels were previously used to diagnose carriership for haemophilia (12). In the early 1980's, it became possible to ascertain the carrier status by means of DNA analysis, which has evolved from haplotyping to mutation analysis offering certainty about the carrier status (14). During the last three decades, genetic counselling, carrier testing, and prenatal diagnosis of haemophilia have become an integrated part of the comprehensive care for haemophilia (12, 15).

In general, small cuts and scrapes are treated with regular first aid i.e. after cleaning the cut a band-aid is applied with pressure. Deep cuts or internal bleeding require more complex treatment. In such cases a factor replacement treatment, prophylaxis is recommended to prevent bleeding episodes before they happen. The Medical and Scientific Advisory Council of National haemophilia Foundation recommends prophylaxis as optimal therapy for children with severe Haemophilia A and B.

Haemophilia provides an attractive target for gene therapy studies, due to the monogenetic nature of this disorder (16 -19). Gene therapy for haemophilia is justified because it is a chronic disease. This therapy is required a very regular factor infusion that may involve fatal risks (18). Several strategies have been proposed for gene therapy for haemophilia. These strategies are based on both in vivo and ex vivo approaches. All successful, pre-clinical and clinical studies to date have utilized recombinant adeno-associated viral (AAV) vectors for factor VIII or IX hepatocyte transduction.

Conclusion

The landscape of available treatments for persons with haemophilia is rapidly evolving. Patients with more serious cases of haemophilia must get regular shots of clotting replacement therapy to prevent bleeding episodes. Novel replacement therapies are undergoing clinical trials in parallel with gene replacement/modification strategies. Availability of these high-cost therapeutics will require evolution of both clinical and financial healthcare services to allow equitable personalization of care for persons with haemophilia.

- 1. Peyvandi, F., Jayandharan, G., Chandy, M., Srivastava, A., Nakaya, S.M., Hohnson, M.J., Thompson, A.R., Goodeve, A., Garagiola, I., Laverotano, S., Menegatti, M., Palla, R., Spreafico, M., Tagliabue, L., Asselta, R., Diga, S., Mannucci, P.M. (2006): Genetic diagnosis of haemophilia and other inherited bledding disorders. Haemophilia, 12(3), pp.82-89.
- 2. Trevor L. Adams, Gregory J. Latham, Michael J. Eisses, M.A. Bender, Charles M. Haberkern.(2019) Essentials of Hematology In: A Practice of Anesthesia for Infants and Children (Sixth Edition) chapter 10, pp. 217-239.e8 https://doi.org/10.1016/B978-0-323-42974-0.00010-0
- 3. Renault, N.K., Dyack, S., Dobson, M.J., Costa, T., Lam, W.L., Greer, W.L. (2007): Heritable skewed X-chromosome inactivation leads to haemophilia A expression in heterozygous females. Eur J Hum Genet, 15 (6), pp. 628-637.
- 4. Hedner, U., Ginsburg, D.L., Jeanne, M., High, K.A. (2000) Congenital haemorrhagic Disorders: new insights into the pathophysiology and treatment of Haemophilia. Hematology, pp.241-265.
- 5. Ng,H.J., Lee, L.H.: (2009): Haemophilia in 21st century Singapore. Ann Acad Med Singapore, 38 (4), pp. 378-379.
- 6. Batty,P., Lillicarp, D. (2019): Advances and challenges for haemophilia gene therapy. Hum Mol. Genetics. 28(R1), pp. 95-101. https://doi.org/10.1093/hmg/ddz157.
- 7. Biggs, R., Douglas, A.S., Mac Farlane, R.G., Dacie, J V., Pitney W.R., Merskey, C., O'Brien J.R. (1952): Christmas Disease: a condition previously mistaken for haemophilia. Br Med J 2 (4799), pp. 1378-1382. Doi: 10.11.1136/bmj.2.4799.1378.
- 8. Giangrande, P(2005): Haemophilia B: Chistmas Disease. Expert Opin Pharmacother, 6(9), pp. 1517-1524.
- 9. Thomas C., Abshire, M.D. (2009). Factor Xi deficiency. In: Transfusion Medicine and Hemostasis.
- 10. Lynne G. Maxwell, , Thomas J. Mancuso, Victor C. Baum, Aaron L. Zuckerberg, Philip G. Morgan, Etsuro K.Motoyama, Peter J.Davis, Kevin J.Sullivan (2011): Systemic Disorders. In: Smith's Anesthesia for Infants and Children (Eighth Edition), chapter 36, pp. 1098-1182. https://doi.org/10.1016/B978-0-323-06612-9.00036-5
- 11. Gitschier, J W W., Goralka, T.M., Wion, K.L., Chen, E.Y., Eaton, D.H., Vehar, G.A., Capon, D.J., Lawn, R.M. (1984): Characterization of human factor VIII gene. Nature, 312, pp. 326-330.
- 12. Husain, N.: (2009): Carrier analysis for haemophilia A: ideal versus acceptable. Expert Rev Mol Diagn, 9(3), pp.203-207.
- 13. Jayandharan, R., Srivastava, A. (2011): Haemophilia: Genetics, Diagnosis and Treatment. Journal of Genetic Syndrome and Gene Therapy, Gene Therapy ISSN: 2157-7412. DOI: 10.4172/2157-7412.S1-005.
- 14. Tantaway, A.A.G. (2010): Molecular genetics of Haemophilia A: Clinical perspectives. Egyptian journal of Medical Human Genetics, 11(2), pp.105-114.
- 15. Street, A.M., Ljung, R., Lavery, S.A. (2008): Management of carriers and babies with haemophilia. Haemophilia, 14 (Suppl. 3), pp. 18,1-187
- 16. Batty, P., Lillicarp, D. (2019): Advances and challenges for haemophilia gene therapy. Human Molecular Genetics, 28 (R1), pp. 95-101, https://doi.org/10.1093/hmg/ddz157.
- 17. Batorova, A. High, K.A., Gringeri, A. (2010): Special lectures on haemophilia management. Haemophilia, 16 (suppl.5), pp. 22-28.
- 18. Liras, A., Olmedillas, S. (2009): Gene Therapy for haemophilia---yes, but... with non-viral factors? Haemophilia, 15(3); 811-816.

Editors: Hemlata Pant, A.R.Siddiqui, Neetu Mishra, Manoj Kumar Singh

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TOXICITY, MECHANISM AND HEALTH EFFECTS OF CADMIUM (Cd)

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Introduction

Cadmium was first exposed by F. Strohmeier in the year 1817 and is a toxic element in the atmosphere ranging from 0.1 to 0.5 ppm. In an environment, the sources of cadmium could be sulfide ores of copper, copper, chloride or with lead Compounds of cadmium (oxide, sulfate, chloride) remain constant and do not undergo a substantial transformation in the environment. Cadmium may possibly disperse worldwide to different areas and set down into the environment and persist for a long time

Cadmium is present in the atmosphere at a very low concentration but generally discharged into the environment through anthropogenic activities such as industrial uses, burning of fossil fuels and through wastewater and solid wastes Cadmium is moreover released from the mining and refineries Phosphate fertilizers have Cd on different concentrations Resulting, increased use of fertilizers, cadmium quantity become higher in the earth and further absorbed by green plants Cadmium is extensively used in industries and frequently in the production of nickel-cadmium batteries Also, Cd is applicable in colouring agents, stabilizers, electroplating, nonferrous alloys of lead, tin and copper. Besides, cadmium is further used in innovative equipment with huge potential in developing image for medical use to cure cancer and conveyance of drugs. Nevertheless, Cd carries high risk through direct transfer to cellular as well as to biological components (Rzigalinski, B.A. et al., 2009, EPA, 2010).

The main route of cadmium exposure

There are many routes by which human are exposed to Cd such as dietary consumption, inhalation, polluted water as well as smoking Equally, dermal absorption of Cd is limited Depending on the particle size, inhaled or digested Cd enters in blood circulation. Several factors increase the Cd absorption in gastrointestinal tract like deficiencies of essential nutrients in nutrition. Consequently, Cd absorbed at a higher rate in females with less amount of iron stores

Cadmium is quickly absorbed by green plants and subsequently transported to all parts for example, to bacco accumulates high amount of Cd in leaves which, in turn, gets accumulated in smokers (Pan *et al.*, 2010). Each cigarette holds roughly 1-2 μ g of Cd and 10% of which is breathed in and 50% is absorbed in the blood plasma Like to bacco, there are many food items which are plant derivatives hold greater amounts of Cd

Cd travels from green vegetation to livestock and finally reaching to humans eating animal products, mainly organ meats Cadmium also enters in aquatic ecosystems generally *via* human actions and accumulates on each trophic level which many types of seafood contain high Cd concentrations

Absorption and distribution of cadmium

In the intestinal wall, there are multiple transporters which are essential to absorb necessary metals Divalent Metal Transporter 1 (DMT1) which is present on intestinal membrane and passage required metal is one such transporter. DMT1 can bind other divalent cations also like Cd and other heavy metals but leads deficit of vital metal besides increment of Cd with other toxic metals absorption. There is a reduction of DMT1 transporters in mice feeding on iron deficit food Similarly, it is also evident that administration of Cd to mice accumulates in the liver as well as in kidney accordingly as per given concentration.

Cd and related metals induce the Metallothionein (MT) gene expression primarily in the liver and kidney to facilitate tissue retention of heavy metals Consequently, metals remain stored in liver and kidney first, but after excess exposure, these heavy metals

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store in several other organs and tissues also It's prerequisite for stopping complete toxicity and protective cellular methodology to increase survival

Safety levels of cadmium and its exposure during pregnancy

Since earlier findings recommend substantial health threat related to previous safe levels of Cd consumption. Thus, in 2010, JECFA restructured level for tolerable Cd monthly consumption to 25 μ g/kg body weights, which relates to weekly Cd consumption of 5.8 μ g/kg body weight In spite of this, the European Food Safety Authority (EFSA) suggested a weekly consumption of Cd at 2.5 μ g/kg body weight

Exposure to cadmium associated with chronic diseases

Epidemiological studies on humans give proof of changed kidney and liver function reduction of fertility and accumulation of Cadmium in follicular fluid Furthermore, plasma Cd level of premenopausal women was related positively with higher testosterone in addition inversely related to estradiol level A positive interaction of blood plus urinary Cd through dietary intake or smoking and higher frequency of atherosclerosis higher systolic blood pressure higher fasting glucose and type-2 diabetes and kidney disease have also been described previously.

Similarly, accumulation of Cd in tissues has been described in animal studies, for example, six-month exposure of CdCl₂ resulted in Cd accumulation in liver and kidney of mice Likewise, the raised value of Cd has been described in kidney, spleen, liver, muscle, testes and ovaries of mice after administration of CdCl₂Earlier research finding also revealed an interaction between plasma Cd and oocyte declining in super ovulated mice. Administration of Cd to female rats for the weeks results in its substantial bioaccumulation in the uterus and placenta with increase DMT1 in the placenta, proving Cd passage to developing fetus Adverse Cd toxicity on respiratory tract already identified in rodents

Cadmium exposure to mother as a possible reason for chronic diseases

Exposure to Cd has health consequences for offspring exposed maternally Blum *et al.*, 2014Maternal nutrient absorption through the intestine is augmented at the time of pregnancy because of nutritional requirements to the developing embryos. Furthermore, the increasing demand for iron and some other nutrients which are essential in pregnancy regulates metal transporters, such as DMT1 Consequently, during pregnancy women can absorb more Cd and other metals through such type of transporters Thus Cd exposure during gestation hinders the placental transmission of nutrients which are essential and can influence growth and metabolism of developing embryos Maternal plus cord blood Cd extent is contrariwise allied by means of litter birth weight A similar type of connotation is documented between birth weight and maternal smoking throughout the gestation Extensive studies to determine the toxic effects of parental Cd experience on healthiness of progenies earlier to puberty revealed important influences such as, higher leptin value neural tube defects behavioural abnormality reduction in IQ scores adiposity change with bodyweight and more risk of upper pulmonary diseases

Reduction in birth weight further described in animal models exposed daily with changing doses of CdCl₂ throughout the gestation or at the time of third trimester Furthermore, maternal Cd exposure can cause fetal diminishing with growth restraint through the increased oxidative stress and free radicals production (Wang, Z. *et al.*, 2012). Lipid peroxidation in the embryonic stage was also increased after subcutaneous administration of CdCl₂ from gestational day 7 to 9 Likewise, 24 h after exposure of CdCl₂ to mice on the first gestation day also noticed in the fetus

Maternal plus fetal Cd exposure may lead to several physiological constraints in neonates and the adult. Such developmental abnormal programming effects can be intervened through fetal exposure to glucocorticoids Administration of CdCl₂ to rats up to day 19 of gestation brought preeclampsia. Such abnormalities were associated with Cd stimulated placental and fetal corticosterone production that supported preeclamptic placentas formation Production of glucocorticoid was achieved through the more expression of 21-hydroxylase (CYP21) plus 11β-hydroxylase (CYP11B1) enzymes and both are necessary for glucocorticoid production Ionic structure of Cd and Calcium (Ca) are of similar types suggesting that Cd could mimic Ca on the steroidogenic routes and bring the synthesis of corticosteroid through the occurrence of Cd in maternal blood Similarly, exposure of Cd to pregnant rats during complete gestation reduces litter weight and enhanced maternal and fetal corticosterone level deprived of any noticeable accumulation of heavy metal in tissues of the fetus

Cadmium effects on glucose homeostasis

Exposure to Cd in initial developing stages is excessive probable alarm as high sensitivity of the developing embryo to chemical toxicants and effects on glucose homeostasis with insulin resistance. Exposure of neonatal rats to different doses of CdCl₂

diminished glucose homeostasis plus glycogen in the liver through gluconeogenesis in addition to reticent insulin discharge by the pancreas It is also evident that a functional diminishing in pancreatic function after Cd exposure leads to the reduction of insulin levels Such type of link revealed in the smelter, exposed to Cd and had lesser serum insulin Incubation of human islet cells through $CdCl_2$ consistent to levels noted in normal pancreas produced islet addition which further diminished β -cell activities without harming cell viability or increasing the oxidative stress Similarly, subcutaneous Cd exposure to rodents gives rise to Cd deposition in pancreatic tissues, diminished glucose tolerance with reduction of insulin expression

The Cd brought a decline of glucose tolerance is associated with deterioration in glucose transporter type-4 (GLUT4) plus decreased insulin inductive glucose uptake by adipocytes Additionally, adult male rats treated with Cd showed a smaller amount of insulin receptors in the adipose tissue

Cadmium effects on obesity and metabolism

Disturbance of metabolism leads to several disorders such as high blood pressure, abnormal glucose homeostasis and higher adiposity causing hypertension, obesity and diabetes That condition is known as Metabolic Syndrome (MS) which further augmented by a high-calorie diet and sedentary lifestyle Available data proves that exposure to toxic metals is an important factor for MS and obesity. For instance, higher-frequency of MS has been detected in Korean population with increased Cd level in the blood Concentrations of Cd in Maternal blood also affect indices of metabolic functions in fetus, like fetal leptin and adiponectin (Ashley-Martin, J. *et al.*, 2015). Higher Cd concentrations in the majority of women at childbearing age support metabolic complications in progeny through maternal Cd exposure.

Exposure to Cd can affect adiposity through disturbing cholesterol metabolism as well as fatty acid metabolism. Oral administration of Cadmium Sulfate (CdSO₄) triggered increasing fatty acid, overall cholesterol, triglycerides and albumin and decrease the concentration of high-density lipoprotein cholesterol Similarly, Cd-induced changes in the lipid metabolism have been detected after administration of CdCl₃ to rats in drinking water for 6 months

Exposures to Cd in adults modify the expression and production of adipokine factors which are associated with insulin resistance, obesity and diabetes. Exposure to CdCl₂ in adult rats for 7 days reduced leptin concentration in serum as well as reduces leptin mRNA expression in white adipose tissue. Similarly when cultured adipose-tissue derived stromal cells treated with CdCl₂ at varying doses, increased triglyceride in culture medium with transcript reduction on behalf of fatty acid generation plus lipids degradation such as acetyl-CoA Carboxylase Alpha (ACACA), Fatty Acid Synthase (FASN) and Peroxisome Proliferator-activated Receptor Gamma (PPARγ) (Kawakami, T. *et al.*, 2013). Findings suggest that Cd treatment disturbs adipocyte metabolism associated with MS with obesity, type-2 diabetes and insulin resistance Transcript for genes responsible for adipocyte differentiation and hypertrophy such as paternally expressed gene 1/mesoderm specific transcript (PEG1/MEST) in addition to peroxisome receptor gamma 2 (PPARγ2) reduced after Cd toxicity as already confirmedFurthermore, treating 3T3-L1 preadipocyte cell lines with CdCl₂ inhibited expression of CCAAT/enhancer-binding protein alpha (C/EBPα) and PPARγ associated with adipogenesis

Cadmium effects on the male reproductive system

Recently, the effects of heavy metal on male fertility have gained interest Similarly, animal research confirmed Cd toxicity on male reproductive system like decrease in sperm concentration; enhance apoptosis with abnormal biosynthesis of steroids

Administration of Cd to adult Wistar rats reduced the testes weights, and testosterone, Follicle-stimulating Hormone (FSH) and Luteinizing Hormone (LH). Transportation of cytoplasmic cholesterol to mitochondria, required for steroid synthesis is regulated by steroidogenic acute regulatory protein (StAR) Mitochondrial cholesterol transportation is controlled by LHwhich increases the concentrations of intracellular cyclic adenosine monophosphate (cAMP) through the activation of adenylate cyclase enzyme Definitely, Leydig cells cultivation by way of Cd reduced progesterone plus intracellular cAMP (Zhang, Q. *et al.*, 2011). Cadmium treatment also shows a reduction of 3β -hydroxysteroid dehydrogenase (3β -HSD), testicular steroidogenic enzymes and 17β -hydroxysteroid dehydrogenase (17β -HSD) activities

Cd brought reduction in testosterone further associated with oxidative stress Wistar rats administered a single subcutaneous injection of CdCl₂showed markers of apoptosis in testis Cadmium-induced genotoxicity and cytotoxicity in cultured Leydig cells of rat showed a reduction of cell viability through increasing the amount of malondialdehyde and glutathione peroxidase activity as a result higher numbers of single-strand DNA breaks occurs Besides this, exposure to Cd may be triggered apoptosis in gonadal tissues resulting male sterility. Survival of germ cell is controlled *via* a glycoprotein clusterin, produced by Sertoli cells inflow into gonadal cells is a sign of cell death. Male rat exposed to varying dose of Cd for about 8 weeks through drinking water shows a dose-dependent

reduction in sperm motility at puberty Furthermore, sperm from dissecting epididymis showed changed transcript majority for Ca channel responsible for Ca and Cd entry.

Blood Testis Barrier (BTB) is responsible for splitting seminiferous epithelium into basal and apical parts Sertoli cells are necessary to regulate germ cell development Thus, by disturbing the interaction of BTB from adhesion of Sertoli-germ cell, reverse effects on spermatogenesis with infertility arises Zhang, X. et al., 2014). Nectin-2 protein required developing the BTB and Sertoli-spermatid adhesion and it is evident that Nectin-2 knockdown carries infertility Exposure to CdCl₂ to *in vitro* cultured mouse Sertoli cell lines inhibited expression of Nectin-2 at transcriptional and post-transcriptional levels Administration of Cd also affects BTB interactions target those proteins which are necessary for its maintenance, like claudin-11

Cultured human fetal testis when treated with $CdCl_2$, there is decrease number of germ cell without any harm to the production of testosterone Maternal exposure to $CdCl_2$ to pregnant mice from gestational day 13 to 17 initiated a reduction in fetal testosterone and StAR, P450scc, $P45017\alpha$ and 17β -HSD expression is down regulated

Cadmium effects on female reproductive system

Available information suggests that Cd shows toxicity on the female reproductive structure and disturb the growth of follicles with alteration of steroidogenesis Leoni, G. *et al.*, 2002). This has been detected earlier in human as well as in animal models The considerably high amount of Cd is found in follicles of smokers Further it is evident that Cd has suppressive effects on growth and maturation of occytes Pisa, J. *et al.*, 1990) plus reduction of occytes number in female rats after CdCl, treatment

Oocyte-cumulus complexes obtained from the antral porcine follicle showed an FSH-induced cumulus developmental suppression as a result of incubation with Cd There is a decrease of progesterone production when Cd-treated oocyte-cumulus complexes incubated with FSH that suggests an interaction between suppressive effects of Cd on the cumulus growth Cultured rat ovarian follicles exposed with CdCl₂ on the second day of culture, resulted in a decrease of survivability and morphological abnormalities of follicle Additionally, exposure of cultured rat ovarian follicles by equal amount of Cd enhanced the oocytes percentage arrested at the germinal vesicle stage

Research already confirmed more probability for chronic diseases in humans after Cd exposure Ovaries lack developed follicles in the insulin-resistant female rats treated by Cd

Cadmium effects on behaviour

Available data recommends that exposure to Cd can lead behavioural abnormalities Haider, S. *et al.*, 2015; Longer exposure to Cd raised up anxiety in rats Study of brain regions engaged in the controlling of depressed behaviour, like cerebral cortex, cerebellum and hypothalamus confirmed reduction of acetylcholinesterase (AChE) plus Na⁺-K⁺-ATPase activity after administration of CdAChE and Na⁺-K⁺-ATPase are important enzymes controlling nerve impulse transmission and their disturbance carries memory discrepancies and depression One hour after the CdCl₂ administration to adult rats at a different dose, enhanced anxiety and depression

Longer Cd and lead (Pb) exposure through drinking water to pregnant rats at the time of pregnancy and lactation enhance depressed type behaviour of offspring These behavioural abnormalities were associated with insufficiencies of dopaminergic and serotonergic systems of the hippocampus Developmental toxicity of prenatal exposure to Cd at the time of lactation has also been studied in animal models

Conclusion

The rapid increase of chronic diseases in adults poses a significant long term threat to the health and wellbeing of humans. However, the causes of chronic diseases are complex, but evidence indicates the *in utero* environment can determine the susceptibility of offspring to adult chronic diseases. For the last decade, much emphasis has been put on the effect of nutritional insults whereas less attention on *in utero* developmental programming effects of exposure to environmental contaminants such as the heavy metals like Cadmium (Cd). Cadmium is widely used in various industries with limited understanding of their developmental programming effects. Several research findings indicate depressed behaviour in the male offspring while there was a reduction in glucose tolerance in both male and female offspring after the exposure to Cd in female mice during her periconception period. Male offspring of Cd treated female mice showed molecular as well as endocrine markers for insulin resistance and depressed behaviour, such as higher concentrations of insulin and leptin in serum, higher glucose level after administration of insulin, mRNA percentage increase for those genes which are directly allied to hepatic glucose, lipid homeostasis besides mRNA percentage reduction in the abdominal tissue of mice. Similarly, there is a decreased mRNA percentage of neurotrophin as well as expressed protein concentration associated with depressed type behaviour like BDNF, which was considered as a molecular marker for depression.

- 1. Abdalla, F.H., R. Schmatz, A.M. Cardoso, F.B. Carvalho, J. Baldissarelli, J.S. de Oliveira, M.M. Rosa, M.A. Goncalves Nunes, M.A. Rubin, I.B. da Cruz, F. Barbisan, V.L. Dressler, L.B. Pereira, M.R. Schetinger, V.M. Morsch, J.F. Goncalves, and C.M. Mazzanti, Quercetin protects the impairment of memory and anxiogenic-like behavior in rats exposed to cadmium: Possible involvement of the acetylcholinesterase and Na(+),K(+)-ATPase activities. Physiol Behav, 2014. 135: p. 152-67.
- 2. Al-Faiyz, Y.S., M.M. El-Garawany, F.N. Assubaie, and M.A. Al-Eed, Impact of phosphate fertilizer on cadmium accumulation in soil and vegetable crops. Bull Environ Contam Toxicol, 2007. 78(5): p. 358-62.
- 3. Ali, I., A. Engstrom, M. Vahter, S. Skerfving, T. Lundh, J. Lidfeldt, G. Samsioe, K. Halldin & A. Akesson, Associations between cadmium exposure and circulating levels of sex hormones in postmenopausal women. Environ Res, 2014. 134: p. 265-9.
- 4. Andersson, H., E. Lindqvist, and L. Olson, Downregulation of brain-derived neurotrophic factor mRNA in adult rat brain after acute administration of methylmercury. Mol Chem Neuropathol, 1997. 31(3): p. 225-33.
- 5. Angenard, G., V. Muczynski, H. Coffigny, C. Pairault, C. Duquenne, R. Frydman, R. Habert, V. Rouiller-Fabre, and G. Livera, Cadmium increases human fetal germ cell apoptosis. Environ Health Perspect, 2010. 118(3): p. 331-7.
- 6. Aschner, M., T. Syversen, D.O. Souza, and J.B. Rocha, Metallothioneins: mercury species-specific induction and their potential role in attenuating neurotoxicity. Exp Biol Med (Maywood), 2006. 231(9): p. 1468-73.
- 7. Ashley-Martin, J., L. Dodds, T.E. Arbuckle, A.S. Ettinger, G.D. Shapiro, M. Fisher, S. Taback, M.F. Bouchard, P. Monnier, R. Dallaire, and W.D. Fraser, Maternal blood metal levels and fetal markers of metabolic function. Environ Res, 2015. 136: p. 27-34
- 8. Ashley-Martin, J., L. Dodds, T.E. Arbuckle, A.S. Ettinger, G.D. Shapiro, M. Fisher, S. Taback, M.F. Bouchard, P. Monnier, R. Dallaire, and W.D. Fraser, Maternal blood metal levels and fetal markers of metabolic function. Environ Res, 2015. 136: p. 27-34
- 9. Belani, M., N. Purohit, P. Pillai, and S. Gupta, Modulation of steroidogenic pathway in rat granulosa cells with subclinical Cd exposure and insulin resistance: an impact on female fertility. Biomed Res Int, 2014. 2014: p. 460251.
- 10. Benoff, S., K. Auborn, J.L. Marmar, and I.R. Hurley, Link between low-dose environmentally relevant cadmium exposures and asthenozoospermia in a rat model. Fertil Steril, 2008. 89(2 Suppl): p. e73-9.
- 11. Benoff, S., K. Auborn, J.L. Marmar, and I.R. Hurley, Link between low-dose environmentally relevant cadmium exposures and asthenozoospermia in a rat model. Fertil Steril, 2008. 89(2 Suppl): p. e73-9.
- 12. Benoff, S., K. Auborn, J.L. Marmar, and I.R. Hurley, Link between low-dose environmentally relevant cadmium exposures and asthenozoospermia in a rat model. Fertil Steril, 2008. 89(2 Suppl): p. e73-9.
- 13. Bernhoft, R.A., Cadmium toxicity and treatment. Scientific World Journal, 2013. 2013: p. 394652.
- 14. Blum, J.L., L.K. Rosenblum, G. Grunig, M.B. Beasley, J.Q. Xiong, and J.T. Zelikoff, Short-term inhalation of cadmium oxide nanoparticles alters pulmonary dynamics associated with lung injury, inflammation, and repair in a mouse model. Inhal Toxicol, 2014. 26(1): p. 48-58.
- 15. Chen, M., R. Tang, G. Fu, B. Xu, P. Zhu, S. Qiao, X. Chen, Y. Qin, C. Lu, B. Hang, Y. Xia, and X. Wang, Association of exposure to phenols and idiopathic male infertility. J Hazard Mater, 2013. 250-251: p. 115-21.
- 16. Cheng, C.Y. and D.D. Mruk, The blood-testis barrier and its implications for male contraception. Pharmacol Rev, 2012. 64(1): p. 16-64.
- 17. Delvaux, I., J. Van Cauwenberghe, E. Den Hond, G. Schoeters, E. Govarts, V. Nelen, W. Baeyens, N. Van Larebeke, and I. Sioen, Prenatal exposure to environmental contaminants and body composition at age 7-9 years. Environ Res, 2014. 132: p. 24-32.
- 18. Edwards, S.E., P. Maxson, M.L. Miranda, and R.C. Fry, Cadmium levels in a North Carolina cohort: Identifying risk factors for elevated levels during pregnancy. J Expo Sci Environ Epidemiol, 2014.
- 19. EFSA, Cadmium dietary exposure in the European population, in Scientific Report of EFSA2012, European Food Safety Authority: EFSA Journal.
- 20. EFSA, Cadmium dietary exposure in the European population, in Scientific Report of EFSA2012, European Food Safety Authority: EFSA Journal.

- 21. El Muayed, M., M.R. Raja, X. Zhang, K.W. MacRenaris, S. Bhatt, X. Chen, M. Urbanek, T.V. O'Halloran, and W.L. Lowe, Jr., Accumulation of cadmium in insulin-producing beta cells. Islets, 2012. 4(6): p. 405-16.
- 22. El Muayed, M., M.R. Raja, X. Zhang, K.W. MacRenaris, S. Bhatt, X. Chen, M. Urbanek, T.V. O'Halloran, and W.L. Lowe, Jr., Accumulation of cadmium in insulin-producing beta cells. Islets, 2012. 4(6): p. 405-16.
- 23. Environmental Protection Agency (EPA). Bisphenol A (BPA) Action Plan Summary. 2010 June 2, 2015 (cited 2015 July 24); Available from: http://www.epa.gov/oppt/existingchemicals/pubs/actionplans/bpa.html.
- 24. Fagerberg, B., L. Barregard, G. Sallsten, N. Forsgard, G. Ostling, M. Persson, Y. Borne, G. Engstrom, and B. Hedblad, Cadmium exposure and atherosclerotic carotid plaques-- results from the Malmo diet and Cancer study. Environ Res, 2015. 136: p. 67-74.
- 25. Faroon, O., A. Ashizawa, S. Wright, P. Tucker, K. Jenkins, L. Ingerman, and C. Rudisill, Toxicological Profile for Cadmium. 2012.
- 26. Faroon, O., A. Ashizawa, S. Wright, P. Tucker, K. Jenkins, L. Ingerman, and C. Rudisill, Toxicological Profile for Cadmium2012, Atlanta GA.
- 27. Faroon, O., A. Ashizawa, S. Wright, P. Tucker, K. Jenkins, L. Ingerman, and C. Rudisill, Toxicological Profile for Cadmium2012, Atlanta GA.
- 28. Faroon, O., A. Ashizawa, S. Wright, P. Tucker, K. Jenkins, L. Ingerman, and C. Rudisill, Toxicological Profile for Cadmium2012, Atlanta GA.
- 29. Fernandez, E.L., A.L. Gustafson, M. Andersson, B. Hellman, and L. Dencker, Cadmium-induced changes in apoptotic gene expression levels and DNA damage in mouse embryos are blocked by zinc. Toxicol Sci, 2003. 76(1): p. 162-70.
- 30. Fickova, M., V. Eybl, D. Kotyzova, V. Mickova, S. Mostbok, and J. Brtko, Long lasting cadmium intake is associated with reduction of insulin receptors in rat adipocytes. Biometals, 2003. 16(4): p. 561-6.
- 31. Goncalves, J.F., F.T. Nicoloso, P. da Costa, J.G. Farias, F.B. Carvalho, M.M. da Rosa, J.M. Gutierres, F.H. Abdalla, J.S. Pereira, G.R. Dias, N.B. Barbosa, V.L. Dressler, M.A. Rubin, V.M. Morsch, and M.R. Schetinger, Behavior and brain enzymatic changes after long-term intoxication with cadmium salt or contaminated potatoes. Food Chem Toxicol, 2012. 50(10): p. 3709-18
- 32. Goncalves, J.F., F.T. Nicoloso, P. da Costa, J.G. Farias, F.B. Carvalho, M.M. da Rosa, J.M. Gutierres, F.H. Abdalla, J.S. Pereira, G.R. Dias, N.B. Barbosa, V.L. Dressler, M.A. Rubin, V.M. Morsch, and M.R. Schetinger, Behavior and brain enzymatic changes after long-term intoxication with cadmium salt or contaminated potatoes. Food Chem Toxicol, 2012. 50(10): p. 3709-18.
- 33. Goncalves, J.F., F.T. Nicoloso, P. da Costa, J.G. Farias, F.B. Carvalho, M.M. da Rosa, J.M. Gutierres, F.H. Abdalla, J.S. Pereira, G.R. Dias, N.B. Barbosa, V.L. Dressler, M.A. Rubin, V.M. Morsch, and M.R. Schetinger, Behavior and brain enzymatic changes after long-term intoxication with cadmium salt or contaminated potatoes. Food Chem Toxicol, 2012. 50(10): p. 3709-18.
- 34. Grant, C.A., Influence of Phosphate Fertilizer on Cadmium in Agricultural Soils and Crops. Pedologist, 2011: p. 143-155.
- 35. Haider, S., L. Anis, Z. Batool, I. Sajid, F. Naqvi, S. Khaliq, and S. Ahmed, Short term cadmium administration dose dependently elicits immediate biochemical, neurochemical and neurobehavioral dysfunction in male rats. Metab Brain Dis, 2015. 30(1): p. 83-92.
- 36. Han, J.C., S.Y. Park, B.G. Hah, G.H. Choi, Y.K. Kim, T.H. Kwon, E.K. Kim, M. Lachaal, C.Y. Jung, and W. Lee, Cadmium induces impaired glucose tolerance in rat by down-regulating GLUT4 expression in adipocytes. Arch Biochem Biophys, 2003. 413(2): 213-20.
- 37. Huang, P.L., A comprehensive definition for metabolic syndrome. Dis Model Mech, 2009. 2(5-6): p. 231-7.
- 38. Ige, S.F. and R.E. Akhigbe, Common onion (Allium cepa) extract reverses cadmium-induced organ toxicity and dyslipidaemia via redox alteration in rats. Pathophysiology, 2013. 20(4): p. 269-74.
- 39. Jeon, J.Y., K.H. Ha, and D.J. Kim, New risk factors for obesity and diabetes: Environmental chemicals. J Diabetes Investig, 2015. 6(2): p. 109-11.
- 40. Ji, Y.L., H. Wang, P. Liu, X.F. Zhao, Y. Zhang, Q. Wang, H. Zhang, C. Zhang, Z.H. Duan, C. Meng, and D.X. Xu, Effects of

- maternal cadmium exposure during late pregnant period on testicular steroidogenesis in male offspring. Toxicol Lett, 2011. 205(1): p. 69-78.
- 41. Ji, Y.L., H. Wang, P. Liu, X.F. Zhao, Y. Zhang, Q. Wang, H. Zhang, C. Zhang, Z.H. Duan, C. Meng, and D.X. Xu, Effects of maternal cadmium exposure during late pregnant period on testicular steroidogenesis in male offspring. Toxicol Lett, 2011. 205(1): p. 69-78.
- 42. Jin, L., J. Liu, B. Ye, and A. Ren, Concentrations of selected heavy metals in maternal blood and associated factors in rural areas in Shanxi Province, China. Environ Int, 2014. 66: p. 157-64.
- 43. Kaisman-Elbaz, T., I. Sekler, D. Fishman, N. Karol, M. Forberg, N. Kahn, M. Hershfinkel, and W.F. Silverman, Cell death induced by zinc and cadmium is mediated by clusterin in cultured mouse seminiferous tubules. J Cell Physiol, 2009. 220(1): p. 222-9.
- 44. Kaisman-Elbaz, T., I. Sekler, D. Fishman, N. Karol, M. Forberg, N. Kahn, M. Hershfinkel, and W.F. Silverman, Cell death induced by zinc and cadmium is mediated by clusterin in cultured mouse seminiferous tubules. J Cell Physiol, 2009. 220(1): p. 222-9.
- 45. Kanter, M., M. Yoruk, A. Koc, I. Meral, and T. Karaca, Effects of cadmium exposure on morphological aspects of pancreas, weights of fetus and placenta in streptozotocin-induced diabetic pregnant rats. Biol Trace Elem Res, 2003. 93(1-3): p. 189-200.
- 46. Kawakami, T., H. Sugimoto, R. Furuichi, Y. Kadota, M. Inoue, K. Setsu, S. Suzuki, and M. Sato, Cadmium reduces adipocyte size and expression levels of adiponectin and Peg1/Mest in adipose tissue. Toxicology, 2010. 267(1-3): p. 20-6.
- 47. Kawakami, T., K. Nishiyama, Y. Kadota, M. Sato, M. Inoue, and S. Suzuki, Cadmium modulates adipocyte functions in metallothionein-null mice. Toxicol Appl Pharmacol, 2013. 272(3): p. 625-36.
- 48. Kawakami, T., K. Nishiyama, Y. Kadota, M. Sato, M. Inoue, and S. Suzuki, Cadmium modulates adipocyte functions in metallothionein-null mice. Toxicol Appl Pharmacol, 2013. 272(3): p. 625-36.
- 49. Kawakami, T., K. Nishiyama, Y. Kadota, M. Sato, M. Inoue, and S. Suzuki, Cadmium modulates adipocyte functions in metallothionein-null mice. Toxicol Appl Pharmacol, 2013. 272(3): p. 625-36.
- 50. Kayaalti, Z., D.K. Akyuzlu, and T. Soylemezoglu, Evaluation of the effect of divalent metal transporter 1 gene polymorphism on blood iron, lead and cadmium levels. Environ Res, 2014. 137C: p. 8-13.
- 51. Kim, N.H., Y.Y. Hyun, K.B. Lee, Y. Chang, S. Rhu, K.H. Oh, and C. Ahn, Environmental heavy metal exposure and chronic kidney disease in the general population. J Korean Med Sci, 2015. 30(3): p. 272-7.
- 52. Kippler, M., F. Tofail, J.D. Hamadani, R.M. Gardner, S.M. Grantham-McGregor, M. Bottai, and M. Vahter, Early-life cadmium exposure and child development in 5-year-old girls and boys: a cohort study in rural Bangladesh. Environ Health Perspect, 2012. 120(10): p. 1462-8.
- 53. Klaassen, C.D., J. Liu, and B.A. Diwan, Metallothionein protection of cadmium toxicity. Toxicol Appl Pharmacol, 2009. 238(3): p. 215-20.
- 54. Kumar, P. and A. Singh, Cadmium toxicity in fish: An overview. GERF Bulletin of Biosciences, 2010. 1(1): p. 41-47.
- 55. Kuriwaki, J., M. Nishijo, R. Honda, K. Tawara, H. Nakagawa, E. Hori, and H. Nishijo, Effects of cadmium exposure during pregnancy on trace elements in fetal rat liver and kidney. Toxicol Lett, 2005. 156(3): p. 369-76.
- 56. Leazer, T.M., Y. Liu, and C.D. Klaassen, Cadmium absorption and its relationship to divalent metal transporter-1 in the pregnant rat. Toxicol Appl Pharmacol, 2002. 185(1): p. 18-24.
- 57. Lee, B.K. and Y. Kim, Blood cadmium, mercury, and lead and metabolic syndrome in South Korea: 2005-2010 Korean National Health and Nutrition Examination Survey. Am J Ind Med, 2013. 56(6): p. 682-92.
- 58. Lee, E.J., J.Y. Moon, and B.S. Yoo, Cadmium inhibits the differentiation of 3T3-L1 preadipocyte through the C/EBPalpha and PPARgamma pathways. Drug Chem Toxicol, 2012. 35(2): p. 225-31.
- 59. Lei, L.J., L. Chen, T.Y. Jin, M. Nordberg, and X.L. Chang, Estimation of benchmark dose for pancreatic damage in cadmium-exposed smelters. Toxicol Sci, 2007. 97(1): p. 189-95.
- 60. Lei, L.J., L. Chen, T.Y. Jin, M. Nordberg, and X.L. Chang, Estimation of benchmark dose for pancreatic damage in cadmium-exposed smelters. Toxicol Sci, 2007. 97(1): p. 189-95.
- 61. Leoni, G., L. Bogliolo, G. Deiana, F. Berlinguer, I. Rosati, P.P. Pintus, S. Ledda, and S. Naitana, Influence of cadmium exposure

- on in vitro ovine gamete dysfunction. Reprod Toxicol, 2002. 16(4): p. 371-77.
- 62. Leret, M.L., J.A. Millan, and M.T. Antonio, Perinatal exposure to lead and cadmium affects anxiety-like behaviour. Toxicology, 2003. 186(1-2): p. 125-30.
- 63. Leret, M.L., J.A. Millan, and M.T. Antonio, Perinatal exposure to lead and cadmium affects anxiety-like behaviour. Toxicology, 2003. 186(1-2): p. 125-30.
- 64. Lin, C.M., P. Doyle, D. Wang, Y.H. Hwang, and P.C. Chen, Does prenatal cadmium exposure affect fetal and child growth? Occup Environ Med, 2011. 68(9): p. 641-6.
- 65. Liu, Y., J. Liu, S.M. Habeebu, M.P. Waalkes, and C.D. Klaassen, Metallothionein-I/II null mice are sensitive to chronic oral cadmium-induced nephrotoxicity. Toxicol Sci, 2000. 57(1): p. 167-76.
- 66. Liu, Y., J. Liu, S.M. Habeebu, M.P. Waalkes, and C.D. Klaassen, Metallothionein-I/II null mice are sensitive to chronic oral cadmium-induced nephrotoxicity. Toxicol Sci, 2000. 57(1): p. 167-76.
- 67. Loganathan, P., K. Louie, J. Lee, M.J. Hedley, A.H.C. Roberts, and R.D. Longhurst, A model to predict kidney and liver cadmium concentrations in grazing animals. New Zealand Journal of Agricultural Research, 2010. 42(4): p. 423-432.
- 68. Massanyi, P., L. Bardos, K. Oppel, S. Hluchy, J. Kovacik, G. Csicsai, and R. Toman, Distribution of cadmium in selected organs of mice: effects of cadmium on organ contents of retinoids and beta-carotene. Acta Physiol Hung, 1999. 86(2): p. 99-104.
- 69. Menai, M., B. Heude, R. Slama, A. Forhan, J. Sahuquillo, M.A. Charles, and C. Yazbeck, Association between maternal blood cadmium during pregnancy and birth weight and the risk of fetal growth restriction: the EDEN mother-child cohort study. Reprod Toxicol, 2012. 34(4): p. 622-7.
- 70. Merali, Z. and R.L. Singhal, Diabetogenic effects of chronic oral cadmium adminstration to neonatal rats. Br J Pharmacol, 1980. 69(1): p. 151-7.
- 71. Millard, K.N., D.M. Frazer, S.J. Wilkins, and G.J. Anderson, Changes in the expression of intestinal iron transport and hepatic regulatory molecules explain the enhanced iron absorption associated with pregnancy in the rat. Gut, 2004. 53(5): p. 655-60.
- 72. Mlynarcikova, A., S. Scsukova, S. Vrsanska, E. Nagyova, M. Fickova, and J. Kolena, Inhibitory effect of cadmium and tobacco alkaloids on expansion of porcine oocyte-cumulus complexes. Cent Eur J Public Health, 2004. 12 Suppl: p. S62-4.
- 73. Myong, J.P., H.R. Kim, T.W. Jang, H.E. Lee, and J.W. Koo, Association between blood cadmium levels and 10-year coronary heart disease risk in the general Korean population: the Korean National Health and Nutrition Examination Survey 2008-2010. PLoS One, 2014. 9(11): p. e111909.
- 74. Nakamura, Y., K. Ohba, and H. Ohta, Participation of metal transporters in cadmium transport from mother rat to fetus. J Toxicol Sci, 2012. 37(5): p. 1035-44.
- 75. Nath, R., R. Prasad, V.K. Palinal, and R.K. Chopra, Molecular basis of cadmium toxicity. Prog Food Nutr Sci, 1984. 8(1-2): p. 109-63.
- 76. Nordberg, G.F., Historical perspectives on cadmium toxicology. Toxicol Appl Pharmacol, 2009. 238(3): p. 192-200.
- 77. Olmedo, P., A. Pla, A.F. Hernandez, F. Barbier, L. Ayouni, and F. Gil, Determination of toxic elements (mercury, cadmium, lead, tin and arsenic) in fish and shellfish samples. Risk assessment for the consumers. Environ Int, 2013. 59: p. 63-72.
- 78. Pan, J., J.A. Plant, N. Voulvoulis, C.J. Oates, and C. Ihlenfeld, Cadmium levels in Europe: implications for human health. Environ Geochem Health, 2010. 32(1): p. 1-12.
- 79. Panchal, S.K., H. Poudyal, A. Iyer, R. Nazer, A. Alam, V. Diwan, K. Kauter, C. Sernia, F. Campbell, L. Ward, G. Gobe, A. Fenning, and L. Brown, High-carbohydrate high-fat diet-induced metabolic syndrome and cardiovascular remodeling in rats. J Cardiovasc Pharmacol, 2011. 57(1): p. 51-64.
- 80. Pandya, C., P. Pillai, L.P. Nampoothiri, N. Bhatt, and S. Gupta, Effect of lead and cadmium co-exposure on testicular steroid metabolism and antioxidant system of adult male rats. Andrologia, 2012. 44 Suppl 1: p. 813-22.
- 81. Paniagua-Castro, N., G. Escalona-Cardoso, and G. Chamorro-Cevallos, Glycine reduces cadmium-induced teratogenic damage in mice. Reprod Toxicol, 2007. 23(1): p. 92-7.
- 82. Park, J.D., N.J. Cherrington, and C.D. Klaassen, Intestinal absorption of cadmium is associated with divalent metal transporter 1 in rats. Toxicol Sci, 2002. 68(2): p. 288-94.
- 83. Piasek, M. and M. Gomzi, (Environmental tobacco smoke and children: assessment of exposure and health effects). Lijec

- Vjesn, 2004. 126(11-12): p. 325-30.
- 84. Pisa, J., J. Cibulka, and M. Ptacek, Effect of subcutaneous application of a single cadmium dose on oocyte maturation in vitro. Physiol Bohemoslov, 1990. 39(2): p. 185-90.
- 85. Pollack, A.Z., S.L. Mumford, P. Mendola, N.J. Perkins, Y. Rotman, J. Wactawski-Wende, and E.F. Schisterman, Kidney biomarkers associated with blood lead, mercury, and cadmium in premenopausal women: a prospective cohort study. J Toxicol Environ Health A, 2015. 78(2): p. 119-31.
- 86. Rogalska, J., M.M. Brzoska, A. Roszczenko, and J. Moniuszko-Jakoniuk, Enhanced zinc consumption prevents cadmium-induced alterations in lipid metabolism in male rats. Chem Biol Interact, 2009. 177(2): p. 142-52.
- 87. Rogers, J.M. and M.L. Mole, Critical periods of sensitivity to the developmental toxicity of inhaled methanol in the CD-1 mouse. Teratology, 1997. 55(6): p. 364-72.
- 88. Ronco, A.M., E. Llaguno, M.J. Epunan, and M.N. Llanos, Effect of cadmium on cortisol production and 11 beta-hydroxysteroid dehydrogenase 2 expression by cultured human choriocarcinoma cells (JEG-3). Toxicol In Vitro, 2010. 24(6): p. 1532-7.
- 89. Rzigalinski, B.A. and J.S. Strobl, Cadmium-containing nanoparticles: perspectives on pharmacology and toxicology of quantum dots. Toxicol Appl Pharmacol, 2009. 238(3): p. 280-8.
- 90. Sadik, N.A., Effects of diallyl sulfide and zinc on testicular steroidogenesis in cadmium-treated male rats. J Biochem Mol Toxicol, 2008. 22(5): p. 345-53.
- 91. Sadik, N.A., Effects of diallyl sulfide and zinc on testicular steroidogenesis in cadmium-treated male rats. J Biochem Mol Toxicol, 2008. 22(5): p. 345-53.
- 92. Satarug, S., S.H. Garrett, M.A. Sens, and D.A. Sens, Cadmium, environmental exposure, and health outcomes. Environ Health Perspect, 2010. 118(2): p. 182-90.
- 93. Satarug, S., S.H. Garrett, M.A. Sens, and D.A. Sens, Cadmium, environmental exposure, and health outcomes. Environ Health Perspect, 2010. 118(2): p. 182-90.
- 94. Satarug, S., S.H. Garrett, M.A. Sens, and D.A. Sens, Cadmium, environmental exposure, and health outcomes. Environ Health Perspect, 2010. 118(2): p. 182-90.
- 95. Schwartz, G.G., D. Il'yasova, and A. Ivanova, Urinary cadmium, impaired fasting glucose, and diabetes in the NHANES III. Diabetes Care, 2003. 26(2): p. 468-70.
- 96. Schwartz, G.G., D. Il'yasova, and A. Ivanova, Urinary cadmium, impaired fasting glucose, and diabetes in the NHANES III. Diabetes Care, 2003. 26(2): p. 468-70.
- 97. Sioen, I., E. Den Hond, V. Nelen, E. Van de Mieroop, K. Croes, N. Van Larebeke, T.S. Nawrot, and G. Schoeters, Prenatal exposure to environmental contaminants and behavioural problems at age 7-8 years. Environ Int, 2013. 59: p. 225-31.
- 98. Stocco, D.M., StAR protein and the regulation of steroid hormone biosynthesis. Annu Rev Physiol, 2001. 63: p. 193-213.
- 99. Sun, H., W. Chen, D. Wang, Y. Jin, X. Chen, and Y. Xu, The effects of prenatal exposure to low-level cadmium, lead and selenium on birth outcomes. Chemosphere, 2014. 108: p. 33-9.
- 100. Tchounwou, P.B., C.G. Yedjou, A.K. Patlolla, and D.J. Sutton, Heavy metal toxicity and the environment. EXS, 2012. 101: p. 133-64.
- 101. Thompson, J. and J. Bannigan, Cadmium: toxic effects on the reproductive system and the embryo. Reprod Toxicol, 2008. 25(3): p. 304-15.
- 102. Thompson, J. and J. Bannigan, Cadmium: toxic effects on the reproductive system and the embryo. Reprod Toxicol, 2008. 25(3): p. 304-15.
- 103. Thompson, J. and J. Bannigan, Cadmium: toxic effects on the reproductive system and the embryo. Reprod Toxicol, 2008. 25(3): p. 304-15.
- 104. Vrsanska, S., E. Nagyova, A. Mlynarcikova, M. Fickova, and J. Kolena, Components of cigarette smoke inhibit expansion of oocyte-cumulus complexes from porcine follicles. Physiol Res, 2003. 52(3): p. 383-7.
- 105. Wan, X., J. Zhu, Y. Zhu, X. Ma, Y. Zheng, F. Wang, Z. Liu, and T. Zhang, Rat ovarian follicle bioassay reveals adverse effects of cadmium chloride (CdCl2) exposure on follicle development and oocyte maturation. Toxicol Ind Health, 2010. 26(9): p. 609-18.

- 106. Wan, X., J. Zhu, Y. Zhu, X. Ma, Y. Zheng, F. Wang, Z. Liu, and T. Zhang, Rat ovarian follicle bioassay reveals adverse effects of cadmium chloride (CdCl2) exposure on follicle development and oocyte maturation. Toxicol Ind Health, 2010. 26(9): p. 609-18.
- 107. Wan, X., J. Zhu, Y. Zhu, X. Ma, Y. Zheng, F. Wang, Z. Liu, and T. Zhang, Rat ovarian follicle bioassay reveals adverse effects of cadmium chloride (CdCl2) exposure on follicle development and oocyte maturation. Toxicol Ind Health, 2010. 26(9): p. 609-18.
- 108. Wang, F., Q. Zhang, X. Zhang, S. Luo, D. Ye, Y. Guo, S. Chen, and Y. Huang, Preeclampsia induced by cadmium in rats is related to abnormal local glucocorticoid synthesis in placenta. Reprod Biol Endocrinol, 2014. 12: p. 77.
- 109. Wang, F., Q. Zhang, X. Zhang, S. Luo, D. Ye, Y. Guo, S. Chen, and Y. Huang, Preeclampsia induced by cadmium in rats is related to abnormal local glucocorticoid synthesis in placenta. Reprod Biol Endocrinol, 2014. 12: p. 77.
- 110. Wang, F., Q. Zhang, X. Zhang, S. Luo, D. Ye, Y. Guo, S. Chen, and Y. Huang, Preeclampsia induced by cadmium in rats is related to abnormal local glucocorticoid synthesis in placenta. Reprod Biol Endocrinol, 2014. 12: p. 77.
- 111. Wang, F., Q. Zhang, X. Zhang, S. Luo, D. Ye, Y. Guo, S. Chen, and Y. Huang, Preeclampsia induced by cadmium in rats is related to abnormal local glucocorticoid synthesis in placenta. Reprod Biol Endocrinol, 2014. 12: p. 77.
- 112. Wang, Z., H. Wang, Z.M. Xu, Y.L. Ji, Y.H. Chen, Z.H. Zhang, C. Zhang, X.H. Meng, M. Zhao, and D.X. Xu, Cadmium-induced teratogenicity: association with ROS-mediated endoplasmic reticulum stress in placenta. Toxicol Appl Pharmacol, 2012. 259(2): p. 236-47.
- 113. Warner, M., P. Mocarelli, P. Brambilla, A. Wesselink, S. Samuels, S. Signorini, and B. Eskenazi, Diabetes, metabolic syndrome, and obesity in relation to serum dioxin concentrations: the Seveso women's health study. Environ Health Perspect, 2013. 121(8): 906-11.
- 114. WHO, Evaluation of Certain Food Additives and Contaminants. Seventy-third Report of the Joint FOA/WHO Expert Committee on Food Additives., in WHO Technical Report Series 2011, World Health Organization (WHO).
- 115. Wu, S.M., P.J. Tsai, M.Y. Chou, and W.D. Wang, Effects of maternal cadmium exposure on female reproductive functions, gamete quality, and offspring development in zebrafish (Danio rerio). Arch Environ Contam Toxicol, 2013. 65(3): p. 521-36.
- 116. Yang, J.M., M. Arnush, Q.Y. Chen, X.D. Wu, B. Pang, and X.Z. Jiang, Cadmium-induced damage to primary cultures of rat Leydig cells. Reprod Toxicol, 2003. 17(5): p. 553-60.
- 117. Yang, J.M., M. Arnush, Q.Y. Chen, X.D. Wu, B. Pang, and X.Z. Jiang, Cadmium-induced damage to primary cultures of rat Leydig cells. Reprod Toxicol, 2003. 17(5): p. 553-60.
- 118. Yang, Q., J. Hao, M. Chen, and G. Li, Dermatopontin is a novel regulator of the CdCl2-induced decrease in claudin-11 expression. Toxicol In Vitro, 2014. 28(6): p. 1158-64.
- 119. Zenzes, M.T., S. Krishnan, B. Krishnan, H. Zhang, and R.F. Casper, Cadmium accumulation in follicular fluid of women in in vitro fertilization-embryo transfer is higher in smokers. Fertil Steril, 1995. 64(3): p. 599-603.
- 120. Zenzes, M.T., S. Krishnan, B. Krishnan, H. Zhang, and R.F. Casper, Cadmium accumulation in follicular fluid of women in in vitro fertilization-embryo transfer is higher in smokers. Fertil Steril, 1995. 64(3): p. 599-603.
- 121. Zhang, Q., P. Zou, H. Zhan, M. Zhang, L. Zhang, R.S. Ge, and Y. Huang, Dihydrolipoamide dehydrogenase and cAMP are associated with cadmium-mediated Leydig cell damage. Toxicol Lett, 2011. 205(2): p. 183-9.
- 122. Zhang, Q., P. Zou, H. Zhan, M. Zhang, L. Zhang, R.S. Ge, and Y. Huang, Dihydrolipoamide dehydrogenase and cAMP are associated with cadmium-mediated Leydig cell damage. Toxicol Lett, 2011. 205(2): p. 183-9.
- 123. Zhang, X. and W.Y. Lui, Dysregulation of nectin-2 in the testicular cells: an explanation of cadmium-induced male infertility. Biochim Biophys Acta, 2014. 1839(9): p. 873-84.
- 124. Zhang, X. and W.Y. Lui, Dysregulation of nectin-2 in the testicular cells: an explanation of cadmium-induced male infertility. Biochim Biophys Acta, 2014. 1839(9): p. 873-84.
- 125. Zhou, T., G. Zhou, W. Song, N. Eguchi, W. Lu, E. Lundin, T. Jin, and G. Nordberg, Cadmium-induced apoptosis and changes in expression of p53, c-jun and MT-I genes in testes and ventral prostate of rats. Toxicology, 1999. 142(1): p. 1-13.

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NUTRITION AND WEIGHT CONTROL DIET

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Nutrition

At health after 50 you will learn the basics of good nutrition and how much of each nutrient you should be consuming. The focus will be on the nutrients that play the most important roles in preventing and managing chronic diseases fats, fibre and specific minerals and vitamins.

Weight control: In theory, weight control is a simple matter of balancing energy intake (the calories supplied by food) with energy output (the calories expended by physical activity, the digestion of food, and the functioning of your body).

To lose weight, you need to expend more energy than you take in. In practice, however, the task is not that simple.

While the basic principle of energy balance remains true, several mechanisms- genetic, metabolic, and environmental can affect how much you eat and how your body uses and stores energy. Even if the genetic and metabolic components of weight regulation are mostly beyond control, environmental factors are controllable and make a significant impact. By manipulating these factors to your advantage, you can successfully lose weight and keep it off.

What it takes to lose weight?

To lose weight, you have to eat fewer calories than your body uses for energy. Calories are found in foods that you eat and show the amount or energy in those foods. Some foods have more calories than others. For example, foods that are high in fat and sugar are also high in calories. If you eat more calories than your body uses for energy, the extra calories will be stored as extra body fat. A pound of fat is about 3,500 calories. To lose 1 pound of fat in a week, you have to eat 3,500 fewer calories (that is 500 fewer calories a day), or you have to "burn off" a just by being more active. Talk to your family doctor before you begin any type of exercise program. Your doctor can help you determine what kind of exercise programme is right for you.

The best way to lose weight and keep it off is to eat fewer calories and be active. If you cut 250 calories from your diet each day and exercise enough to burn off 250 calories, that adds up to 500 fewer calories in one day. If you do this for 7 days, you can lose 1 pound of fat in a week. Many experts believe you should not try to lose more than 2 pound per week losing more than 2 pounds in a week usually means that you are losing weight and lean muscle mass instead of losing excess fat. If you do this, you will have less energy and you will most likely gain the weight back.

How often should I eat?

Most people can eat 3 regular meals and 1 snack every day. The 3 meals should be about the same size and should be low in fat. Some people benefit more if they eat 5 to 6 smaller meals, your body stays satisfied and you are not as hungry throughout the day. Try to make half of your plate fruits and vegetables eat more whole grains, and eat lean meats instead of processed meats (hot dogs,) Eat breakfast and don't skip meals while skipping meals may help you lose weight in the beginning, it fails in the long run, skipping meals may make you feel too hungry late in the day, causing you to overeat at your next meal.

What is so bad about foods high in fat?

Foods high in fat are usually high in calories, which could lead to unwanted weight gain. Consuming too much saturated and trans fats may increase LDL cholesterol ("bad" cholesterol) level, and increase your risk of heart disease. You can decrease your

saturated fats by increasing the amount of fruits and vegetables you eat, avoiding fried foods, eating lean meats, and using less butter or margarine. It is important to remember that some fats can be beneficial to your overall health, "Good" fat. Such as polyunsaturated and monounsaturated fats, are found in fish, nuts, and low or nonfat dairy products.

What are empty calories?

Some foods are referred to as "empty calories" because they add lots of calories to, your diet without providing much nutritional benefit. An example is sugar sweetened drinks, such energy drinks, sweetened or flavored milk, and sweetened tea. These drinks can add lots of sugar and calories to your diet,.

But staying hydrated is important for good health. To reduce the calories and sugar in your diet, drink more water, zero calorie flavored water, non fat or reduced fat milk. Talk with your family doctor or a dietitian if you have questions about your diet or healthy eating for your family.

Weight loss and body composition

The body is made up of different components (water, muscle, fat bone, nerve, tissue, tendons, and so on), and each has a different density. From a functional standpoint, tissues are grouped together into those that are mainly fat (fat mass), which is mainly anhydrous (has little water associated with it), and those that have little fat (fat-free mass), which is hydrous is also commonly referred to as lean mass, although this is viewed by many to be an inaccurate description because the fat-free mass includes a Great deal of water (greater than 65 percent). Because techniques for estimating body composition are widely available, bone mass is now also included as a third commonly assessed component of body composition. The components of body composition are referred to as fat mass (the body tissue that is mainly fat) and fat-free mass (the body tissue that is mainly free of fat, including muscle and bone).

Fat mass is composed of essential fat and storage fat. The essential fat is a required component of the brain, nerves, bone marrow, heart tissue, and cell walls that we cannot live without. Approximately 12 to 15 percent of total body weight in adult females is essential fat, the majority of which is associated with reproductive function and includes the additional fat associated with breast tissue. Because males do not have this reproductive function, their essential fat levels are considerably lower. Storage fat on the other hand is and energy reserve that builds up in adipose tissue underneath the skin (subcutaneous fat) and around the organs (inter abdominal fat). It is common for healthy men and women to have a storage fat level that contributes 11 to 15 percent to total body weight. Combining the essential fat and storage fat components, normal body fat percentage for males is approximately 15 percent (3 percent essential; 12 percent storage), while normal body fat percentage for females is 26 percent (15 percent essential; 11 percent storage).

Women with extremely low body fat percentage are at risk of developing reproductive system difficulties, commonly manifested as irregular menstrual periods. Oligomenorrhea and amenorrhea are associated with increased fracture risk and low estrogen production, which increases the risk of osteoporosis (a bone disease associated with low bone density). It appears that a body fat percentage of 17 to 22 percent is needed to maintain a normal menstrual cycle in most women. Women who develop an excessively low body fat percentage typically exercise excessively for the amount of energy they consume, or they have an eating disorder. The female athlete triad, a condition prevalent in many female athletes, includes the interrelated presence of an eating disorder; amenorrhea; and low bone density, osteopenia, or osteoporosis.

Fat free mass is mainly water and protein but also includes small levels Osteoporosis of minerals and stored carbohydrate Amenorrhea (glycogen). The main constituents of fat-free mass include skeletal muscle, the heart, and other organs. Although total body weight is approximately 60 percent water, the water content of the fat-free mass is 70 percent. This can be compared with the water content of the fat mass which is below 10 percent. 3 Athletes typically have a higher fat-free mass and a lower fat mass than do nonathletes.

Weight

The measurement of weight (pounds) or mass (kilograms) by itself does not discriminate between fat mass and fat-free mass so is not a measure of body composition. Therefore, the statement "My weight is increasing, so I must be getting fat is common but not necessarily correct. It is possible for an athlete to increase fat-free mass (i.e. muscle) without increasing fat mass.

The result would be ad increase in weight but not an increase in fat weight. It is possible for an athlete to increase fat-free mass (i.e. muscle) without increasing fat mass.

The result would be an increase in weight but not an increase in fat weight experience changes in fat or fat0free mass. This could be either desirable or undersirable depending on which element is increasing. All athletes, regardless of sport, find it desirable to achieve a high strength to weight ratio, which is associated with a relative increase in the ratio of fat-free mass to fat mass.

This can be achieved by maintaining the fat-free mass while fat mass is decreased (lower total weight); increasing the fat-free mass while fat mass is maintained (higher total weight); increasing the fat-free mass while fat mass decreased (lower total weight); or increasing the fat-free mass (higher total weight). As you can see, monitoring a change in weight alone is an inadequate means of understanding what really matters; the components of weight change.

Although tracking weight is an appropriate measure for understanding the athlete's energy balance. It does nothing to explain whether the components of the weight are changing in a desirable direction. It is for this reason that body composition evaluation should be standard component of the athlete assessment protocol.

Ideal Weight

There are several common means of predicting ideal body weight, but they should all be considered of limited value when used with athletes. The most common formulas for predicting ideal body weight.

Devine Formula

Male: Ideal body weight (in kilograms)=

50 kilograms + 2.3 kilograms per inch over 5 feet

Female: Ideal body weight (in kilograms)=

45.5 kilograms + 2.3 kilograms per inch over 5 feet

Robinson Formula

Men: Ideal body weight (in kilograms)=

52 kilograms + 1.9 kilograms per inch over 5 feet Women: Ideal body weight (in kilograms)= 49 kilograms + 1.7 kilograms per inch over 5 feet

Miller Formula

Men: Ideal body weight (in kilograms)=

56.2 kilograms + 1.41 kilograms per inch over 5 feet

Women: Ideal body weight (in kilograms)=

53.1 kilograms +1.36 kilograms per inch over 5 feet

Body Mass Index

Body mass index (BMI) is a useful tool for categorizing be weight. However, it is not likely to be useful for athletes because athletes more weight muscle than do non athletes, which increase the weight to height ratio. BMI considers weight in relation to height using one of the following formula:

BMI = weight in kilograms divided by height in meters squared

BMI = kg/m

BMI = weight in pounds divided by weight in inches squared multiplied by 703

BMI = ([lxb]x703)

BMI Classification

Classification	BMI	
Underweight	<18.5	
Normal	18.5 to 24.9	
Overweight	25.0 to 29.9	
Obese	≥ 30	

Weight Issues

There is no question that total body weight is an important issue for athletes because it influences how easily they can perform their skills. A study assessing the relationships between body composition and fundamental movement skill among children and adolescents found that unhealthy weight gain reduced movement sill. However, looking at weight by itself may provide athletes with a misleading picture of what is good or bad about their body composition.

In a number of sports, athletes will increase the time or intensity of a training regimen to improve performance, but then they inappropriately use changes in weight as a marker of success or failure. Imagine a football player who comes to training camp at a weight much higher than the coach is accustomed to seeing in this player. It may well be that the football player worked hard during the off-season to increase muscle mass and the increase in weight is a result of more muscle. Wouldn't the coach be wrong in telling that player that he has to lose weight? Gymnasts often reach their competitive peak during adolescence, a time when fast growth is the normal biological expectation.

Despite this, gymnasts and other athletes are often weighed weekly or more often to make certain they are maintaining their weight. Shouldn't all the training they've doing increase their muscle mass and therefore their weight? Shouldn't they be growing and thus increasing their weight? These are examples of how weight is often used arbitrarily and incorrectly. Tracking the constituents of weight makes much more valuable information on the nature of body changes that are occurring.

The principle of energy thermodynamics is always with us. Consumption of more calories than the lady burns leads to a weight gain; consumption of fewer calories than the lady burns leads to a weight loss; and consumption of exactly the same number of calories that the body burns leads to weight stability. But making a change in body weight is not as straight forward as the principle of energy thermodynamics may make it appear to be. The most common belief is that low calorie dieting is an effective but unpleasant means of weight and fat loss. It seems logical that a 25 percent reduction in energy make will lead to a 25 percent reduction in weight.

The reality, however, is that energy expenditure after weight loss is less than would be expected by the amount of weight that was lost for. This means that the adjustment in energy expenditure to inadequate intake is greater that the mathematical expectation and leads to a return to the original weight, even with a lower energy intake (i.e. the loss you eat, the less you can eat to maintain weight). A close look at the reason for this lower metabolic rate is clear. With an inadequate calorie intake, the body catabolizes the metabolic (lean) mass so it can survive on less energy. Logic also suggested that a 25 percent increase in weight.

In fact, although weight gain does occur, it doesn't appear to increase as much as the increase in energy intake suggests it should, but it's close. When people are purposefully overfed to gain weight, the amount of weight gain is proportionate to the amount of overfeeding 5-8. These studies strongly suggest that we have homeostatic mechanisms during periods of energy deficit that help us maintain our weight. This may be a "survival of the species" mechanism that helps humans survive periods of famine. We also appear able to store energy effectively (as fat) during periods of excess. This may be another survival mechanism that enables us to store energy when we are lucky enough to have excess food available. Since major energy surpluses and deficits appear to activate homeostatic mechanisms, a possible means of making a desired change in weight and body composition is to avoid major energy balance shifts. Exercise should be at the core of any desired body composition change (i.e. an increase in lean mass and a decrease in fat mass, coupled with a small decrease in weight). But such a change surplus created are never too large during the day. Energy surpluses and deficits are represented, respectively by variation above and below the perfect energy balance line (zero).

In the figure, when the line moves above zero, the athlete has consumed more energy than was expended. When the line moves below zero, the athlete has expended more energy than was consumed. Eating pattern 1 represents an athlete's eating small meals frequently; there are no energy surpluses or deficits that exceed 400 calories. Eating pattern 2 represents infrequent eating, with excess calories (high surplus energy peaks) consumed at each meal. Eating pattern 3 represents an athlete who spends the majority of the day in an energy deficit state from not eating enough when the energy is needed, a condition that stimulates the breakdown of muscle tissue for energy. At the end of the day, a very large meal brings the athlete into energy balance, but much of this meal will be stored as fat. Within any given day, energy balance is important for both performance and body composition.

Weight is the best indicator of the adequacy of caloric intake, and body composition helps determine if the calories are being consumed in the proper amounts and at the correct intervals. Since the standard three-meal-a-day schedule forces athletes to consume a large amount of energy at each meal to obtain the necessary energy, staying in energy balance is easier on a six meal pattern. Frequent consumption of small meals to maintain a steady energy flow can be an important strategy in making the desired changes.

Overweight and Obesity in children and Adolescents

In contrast to colloquial usage, where obesity and overweight generally refer to culturally undesirable body size ("being fat"), these terms represent specific conditions with unique criteria in the medical and scientific literature. While obesity is a condition of excess body fat (adiposity). Which is associated with adverse health states and risk for future disease, the medical definition obesity in children and adolescents is not as straight forward as for adults. At present, there is no universally accepted definition that distinguishes children with normal or healthy weight from those whose level of adiposity is unhealthy. While the presence of obesity in

some children and adolescents is obvious with simple observation, it is difficult to determine when a child who is not obviously overweight faces health risks from adiposity. In the absence of a clear, health based definition of obesity, children are instead categorized as "overweight" and "obese" based on how they compare with a normative sample of children of the same age and sex.

Body Mass Index (BMI) is the most common measure used to define overweight and obesity in children adolescents, and adults. BMI is a height adjusted weight measure that is calculated from measured weight (in kg) and height (in meters) as kilograms divided by meters squared (kg/m²). Clinicians compare a child's BMI to that of other children of the same age and sex to determine a percentile score base on published norms, such as those developed by the Centers for Disease Control and Prevention in the United States.

Because BMI naturally changes with ate, percentile scores based on age and sex specific norms are based to determine overweight and obesity and monitor growth and development in children and adolescents. Over time, changes in percentile scores can show clearly when a developing child has become fatter slimmer. Thus, while BMI might increase in an overweight growing child, a decrease in percentile score would indicate a positive outcome, as their growth in height outstripped their weight gain the BMI base terms that denote different levels of excess weight in children and adolescents and compares them to terms in adults.

We've provided adult terminology and classification both for context to help the reader interpret BMI values reported in children and adolescents and because the categorizations may be valid for older adolescents who have achieved their adult height. Also provide comparisons between various height (inches or centimeters) and weight (pounds and kilograms) measures and absolute BMI. BMI percentiles, and BMI standard deviation scores (SDS).

The Expert Committee (A committee covened by the American Medical Association (AMA) and co-funded in collaboration with the Department of Health and Human Services Health Resources and Services Administration (HRSA) and the CDC recommends using the term "overweight" to refer to children with BMI in the 85th to 94th percentiles for their age and sex. They recommended the term "Obese" to refer to children with BMI at or above the 95th percentile for their age and sex or with a BMI at or above 30, which is the adult standard for defining obesity.

These definitions were originally derived from population norms rather than health states, and research continues to focus on clarifying the health risks associated with various definitions of overweight and obesity in children, adolescents, and adults. These and other definitions can be found in the glossary.

Although it is not a direct measure of adiposity BMI for age percentile measures in boys and girls correlate reasonably well with percentile rankings of directly measured percept body bat (correlations generally between 0.78 to 0.88). obesity (primarily defined as BMI >95th percentile) has also been correlated with childhood health consequences and with risk factors for obesity related morbidity in adults.

Since BMI is an imperfect measure of body fat, however, categorizing children and adolescents as above based on BMI definitions can be problematic. Recent data from the Bogalusa Heart Study found that 35 percent of children aged 5 to 17 years with BMI > 95th did not have excess body fat. At or above the 99th percentile, however, almost all (94 percent) had excess adiposity. Those with the highest BMI percentiles (>99th) were also much more likely to have two or more cardiovascular risk factors (59 percent) compared with those in the broader group at or above the 95th percentile (39 percent). Noting these differences experts have recently proposed distinguishing the "severely obese" defined by the 99th percentile as those in particular need of clinical evaluation and treatment.

An absolute BMI level can indicate very different weight states in children and adolescents of different ages. A BMI of 20 would categories and 8 year old as obese, but would categorize a 16 year old as normal weight. Absolute BMI levels may be more informative for clinical and research outcomes that percentile in children and adolescents particularly those above the 95th percentile, where there can be a broad range of actual BMIs (and therefore weight).

Above the 99^{th} percentile, BMI measures can overlap with BMI levels use to define obesity in adults (30 kg/m^2). Thus, experts recommend that obesity in children and adolescents be defined as BMI $> 95^{th}$ percentile or BMI $> 30 \text{ kg/m}^2$, whichever is lower.

Since no measure is ideal for every age, many youth obesity researchers report multiple measures, including BMI. BMI percentiles, BMI standard deviation scores (SDS also known as z scores), or an older measure, "percent overweight"

Prevalence of Children and Adolescents Obesity

Between the early 1970s and 2003 to 2004, the prevalence of obesity (defined as age and sex specific BMI" 95th percentile) increased three to six fold, depending on age, sex and ethnicity. In 2003 to 2004, the prevalence of obesity among 6 to 19 year old

children and adolescents was approximately 16 to 18 percent.

When children and adolescents who are overweight (defined as age and sex specific BMI in the 85th to 94th percentile) are also included, this prevalence increase to almost one in three children and adolescents identified as overweight or obese (31 to 33 percent). Looking at the youth with the most severe levels of obesity 3 to 6 percent of boys aged 13 to 17 years are at or above the 99th percentile. For girls, the comparable figure is 1 to 3 percent.

The prevalence of obesity varies somewhat with age. Children aged 6 to 11 years have the highest prevalence of obesity (18.8 percent), compared with younger children (13.9 percent) and adolescents (17.4 percent) according to data from the 2003 to 2004 National health Nutritional Evaluation Survey (NHANES). Males have slightly higher prevalence of obesity for all age categories. Childhood obesity is increasing all around the world, not just in the United States. A meta analysis calculated that the annualized change in prevalence of obesity in school children in the United States from 1971 until 200 was approximately 0.4 percentage points per year. Twenty-three North American, Eastern European, Western European, and Asian countries reporting comparable data also showed increases in childhood obesity with annualized changes ranging from less that 0.1 percentage points (in Finland and the Netherlands) to over 0.7 percentage points (Singapore and East Germany). The estimated prevalence of overweight (including obesity) in children and adolescents in the America as a whole is 27.7 percent. Europe has the next highest estimate at 25.5 percent, then Eastern Mediterranean countries (23.5 percent) followed by countries in the West Pacific (12.0 percent) and South East Asia (10.6 percent). Prevalence of overweight and obesity are low in African nations (1.6 percent).

Weight Gain Diet and Nutrition

It you're naturally thin, then a proper weight gain diet becomes absolutely crucial in your endeavor to gain healthy lean muscle mass, probably far more so than your weight training programe. The purpose of this article is to outline the fundamentals of a good weight gain diet and then we'll look at specifically what and when you could eat over a sample day.

First up, is something you've almost certainly heard before....

Consume More Calories

The only way to gain weight whether it's fat or muscle, is to consume more energy than you expend.

There is no escaping this basic law of human anatomy regardless of how many explanations you hear to the contrary. Admittedly some of us have faster metabolisms than others but that simply means that those who do need to eat even more again.

The basis of any weight gain diet should contain nutritious, high calorie foods. If you find to hard to put on weight then the greatest challenge you face is to consume enough energy without feeling full all the time. Don't worry it can be done quite easily!

So how many calories should you consume? Well, there's probably a separate formula for everyone who asks the question. Some base it solely on your weight and age, other take lean mass into account and the most complicated have you recounting every bit of activity during a typical day. There's a short article at the bottom of his page that has some formulas for calculating your calorie needs. It also briefly explains basal metabolism and why it's important.

To sum up, calorie counting isn't much fun and this is not something you have to do long term. Once you establish a quantity of food and energy that maintains your ideal weight, you will know instinctively how much to eat each day.

Protein

The issue of how much protein we should consume incites fierce debate between Nutritionists, Bodybuilders and Sports Scientists alike. We'll leave the debate for another article dedicated to the protein issue. Just know that Federal Drug Administration (FDA) recommends 50g of protein per day for the average male adult and the World Health Organization (WHO) recommends 56g.

The Recommended Daily Allowance (RDA) for protein intake is 0.8 g per kg or 0.36g per lb of bodyweight. A 140lb person would need to consume about 51g to meet their RDA. Sports Scientists concede that athletes and bodybuilders need more than this and conservatively recommend up to 1.5g per kg or 0.7g per lb of bodyweight.

If you talk to the vast majority of bodybuilders they will advocate a much higher intake than this. And they have some convincing arguments. In fact although in the minority at the moment the anecdotal evidence from bodybuilders is being backed up by some credible research. According to many lifters, coaches and some sports nutritionists an ideal weight gain diet should contain up to 2g per kg or 0.9g per lb of protein. This might seem like a lot but don't forget you are consuming more calories than the general population and those calories have to come from somewhere. Is it unhealthier than they come purely from carbohydrates or just from fat? It's probably best it comes form all three.

So why is protein important?

From a weight gain perspective protein is made up of amino acids. There are 20 in total and 8 essential amino acids must come from food. Weight training increases the demand for amino acids and will break existing muscle down if it does not get enough from a weight fain diet. Without adequate protein, and more specifically, amino acids muscle gain is unachievable.

Good sources of protein include fresh and canned fish, lean cuts or red meat, chicken, turkey, low fat milk and yogurt, low fat cottage cheese, egg whites, soy products and whey protein powder.

Carbohydrate

Any weight gain diet worth its salt will contain plenty of unrefined carbohydrates. Just because you're increasing your protein intake does not mean you should omit or even limit your carbohydrate intake.

Carbohydrate, which is converted into glucose and glycogen in the body, is the only macronutrient that can supply your body with an immediate source of energy – essential for any type of training. Good sources of carbohydrate for a weight gain diet include whole meal bread, potatoes, brown rice, pasta, couscous, fresh and tinned fruit and dried fruit.

Fat

Certain dietary fats are crucial to your well being and your ability to gain weigh. One gram of fat contains or 1g of carbohydrate. A tablespoon of Flaxseed Oil contains as many calories as a banana for example so it makes sense to incorporate good fats into your weight gain diet. What is good fat?

Without going into too much detail about how fat is subdivided, the fats you want to consume are monounsaturated fats found in olive oil and avocados, and polyunsaturated fats found in oily fish, flax, sunflower, safflower and cod liver oil and some raw nuts. Essential fatty Acids (EFA's) are what the health care professionals love to talk about. And with good reason EFA's also known as Omega-3 and Omega-6 fatty acids are found in polyunsaturated fats, particularly oily fish. As well as having a numerous health benefits they also play an important role in muscle building. In short, a weight gain diet containing fish like mackerel, tuna and salmon (to name a few) or supplemented with a product like Flaxseed oil will not only help you build muscle but will keep you alive longer too.

Meal Frequency

Finally, forget about eating 3 large meals a day with a few snacks. The best approach to an effective weight gain diet is to eat 5 or 6 small meals a day. Separate them by 3 hours so your stomach as time to digest each meal fully. If your goal is to consume 33ooKcals a day I would eat 3 larger meals of about 700kcal and 3 smaller meals of about 400kcals. You will find an example in one of the articles below.

One last point before we wrap up. Eat a variety of fruits and vegetables. They are the richest source of vitamins and minerals (particularly antioxidants) and have both health and weight gain implications. The elements above have the most influence of your level and rate of weight and muscle gain. There are other important factors we haven't touched on such as vitamins and minerals, fibre, water, alcohol and cholesterol all very important to your health. Now that you have a good grounding of what an effective weight gain diet should incorporate you can us this article as a starting point for reaching your weight goals.

Opportunities For Nutritionists And Dieticians

The healthcare industry in India is worth Rs. 61,000 crores (excluding the pharmaceutical industry). It is expected to touch dizzy height with the entry of private hospitals, healthcare centers health management organizations and health insurance companies. Large scale I vestments to the tune of Rs 2,000 crores in the next four years will open up numerous job opportunities including that of dieticians or nutritionists.

Dieticians/ Nutritionists are the professionals recognized as experts on food and nutrition. They are an authority on diet and the application of the principles of nutrition. They plan nutrition programmes and supervise the preparation and serving of meals. They are often responsible for promoting sound eating habits through education and research. They are concerned with food and health in its widest sense and their work is preventive and therapeutic. Dieticians must know about food production and processing, social, economic and psychological factors that influence food choice the digestion, absorption and metabolism of food, its effect on nutritional well being; how to treat disease and prevent nutrition related problems.

Dieticians manage food service systems for institutions such as hospitals and school, promote sound eating habits through education and conduct research. Major areas for practice include clinical, community, management and consultant dietetics. Clinical dieticians provide nutritional services for patients in institutions such as hospitals and nursing care facilities. They also confer with

doctors and other healthcare professionals in order to coordinate medical and nutritional needs. Some clinical dieticians specialize in the management of overweight patients or the car of critical ill or renal (kidney) and diabetic patients. In addition, clinical dieticians in nursing care facilities small hospitals or correctional facilities may manage the food service department.

Community dieticians counsel individuals and groups on nutritional practices designed to prevent disease and promote health. Working in places such as public health clinics, home health agencies and health maintenance organizations, community dieticians evaluate individual needs, develop nutritional care plans, and instruct individual and their families. Dieticians working in home health agencies provide instruction on grocery shopping and food preparation to the elderly, individuals with special needs, and children.

Increased public interest in nutrition has led to hob opportunities in food manufacturing advertising and marketing. In these areas dieticians analyze foods, prepare literature for distribution or report on issues such as the nutritional content of recipes, dietary fiber, or vitamin supplements. Management dieticians oversee large scale meal planning and preparation in healthcare facilities, company cafeterias, prisons and schools. They hire train and direct other dieticians and food service workers; budget for and purchase food, equipment and supplies enforce sanitary and safety regulations; and prepare records and reports.

Consultant dieticians work under contract with healthcare facilities or in their own private practice. They perform nutrition screenings for their clients and offer advice on diet related concerns such as weight loss or cholesterol reduction. Some work for wellness programs sports teams supermarkets and other nutrition related businesses. They may consult with food service managers providing expertise in sanitation, safety procedure menu development, budgeting and planning. A home science course right after plus two or equivalent is a direct training route. Sometimes, there is the provision of home science as a subject during plus two. Admission in to the 3 or 4 year Home Science course is open to those with Plus Two (Science/ Home Science). A three year B.Sc.(Bachelor of Home Science) course could offer Food, Nutrition and Dietetics as a specialization.

The 2 year Master's degree programme in Home Science also offers Food & Nutrition as a specialization. Postgraduate diploma courses in Nutrition & Dietetics are offered to science (Microbiology/ Biochemistry/allied) graduates and home science graduate. Hotel and catering technology degree may also lead to a postgraduate diploma in dietetics. Medical graduate may pursue post graduation programmes in nutrition science.

Food Science & Technology/ Nutrition can be offered in a B.Sc. course or as a Bachelor's degree in Applied Science as in Delhi University. Food Technology course of 4 year duration after Plus two in Science is an option for those who are keen on going in depth into the technical research aspects of food, food processing and technology. The ultimate making of a nutritionist/ dietician is the culmination of education training personal skills, interests and updating of knowledge. Continuing education in the form of seminars and symposiums on nutrition and food are helpful.

Dieticians and nutritionist work in different settings such as hospitals, nursing home, school, old age homes, catering services in railways, airlines, hotels etc. they may work with local authorities in helping to develop and implement for policies or work as faculty in centers of higher learning. They could be involved in research or join industry (food processing) catering organizations, and other industrial set ups. A consultants dieticians and nutritionists may work in quality control or freelance for sports nutrition, media and fashion/ beauty industry. Some may eventually specialize in the management of overweight patients care of the critically ill, diabetic patients, or patients with renal failure. Dieticians may work in health and fitness clubs, gyms, private weight loss companies, health spas and convalescent centers. They may be attached to sports teams, individual athletes, sports medicine clinics in a consulting role. Experienced dieticians may move up the hierarchy in the dietetics department of a health facility of with other employer or become self employed. Private consulting is usually lucrative.

- 1. Ballentine R: Diet and Nutrition: A Holistic Approach, Himalyan Press, Delhi 1989.
- 2. Burkitt D.P.: Western Diseases: Their Dietary Prevention and Reversibility, Humana press, 1994.
- 3. Chmelynski, Canal and Capsione: Opportunities in Food Services Careers Lincoln Wood, VGM career Horizons 2000.
- 4. Pattenrson, Charless: Eternal Trreblinka: Our Treatment of Animals and the Holo court, Newyork: Lanten Books 2002. https://www.acefitness.org
- 5. , 2015 https://www.hindawi.com
- 6. > job 2019

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CHEMICAL PESTICIDES USE IN AGRICULTURE : CAUSE HAZARDOUS EFFECT ON HUMAN HEALTH AND ENVIRONMENT

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Abstract

The term pesticide covers wide range of compounds including insecticides, fungicides, herbicides, rodenticides, molluscicides, nematicides, plant growth regulators etc. Pesticides are toxic; they are also potentially hazardous to humans, animals, other organisms, and the environment. Hazard depends on the toxicity of the pesticide and the amount of exposure to the pesticide. The toxicity of pesticide is measure of its capacity to cause injury and dosages of the active ingredient (a.i.) of products. The harmful effects occur from a single exposure by any route. The four routes of exposure are dermal (skin), inhalation (lungs), oral (mouth) and the eyes. The amount of concentration toxicant a.i. required to kill 50 percent of the animals in test population. This measure is usually called LD_{50} (lethal dose 50) and LC_{50} values are are recorded in milligrams of pesticide per kilogram of body weight (mg/kg) of the test animal. Pesticides that are classified as highly toxic on the basis of either oral, dermal, or inhalation toxicity must have the signal words DANGER and POISON printed in red with a skull and crossbones symbol prominently displayed on the package label and the acute oral LD_{50} is 50 mg/kg. Pesticide products considered moderately toxic must have the signal word WARNING and the acute oral LD_{50} ranges from 50 to 500 mg/kg. Pesticide products classified as relatively slightly toxic to have the signal word CAUTION on the pesticide label and acute oral LD_{50} values in this group are greater than 500 mg/kg. The basic safety issues related to pesticides exposure, adverse effects on human health, environment and on non-target organisms as well as their reliability and accuracy in adverse effects.

Introduction

Chemical pesticides compounds are insecticides, fungicides, herbicides, rodenticides, molluscicides, nematicides, plant growth regulators etc. Pesticides eliminate insects, weeds, fungi, bacteria on crops as spread disease. Organochlorine (OC) insecticides, used against controlling diseases as malaria and typhus, it's restricted after the 1960s in most of the countries. Poisoning from pesticides is a global public health problem and deaths worldwide every year. Pesticides are used extensively in agricultural. These chemicals are believed to cause many disorders in humans and wildlife. Most common disease is cancer as associated with pesticides and also involved in the pathogenesis of Parkinson's and Alzheimer's diseases as well as various disorders of the respiratory and reproductive. Persistent of pesticides because they do not biodegrade and not easily to removed from the environment. One of the greatest risks to environment and health comes from chemical pesticides.

Pesticides are widely used in the agricultural production to prevent yield losses by pests and improve quality to consumers (Cooper and Dobson 2007). Pesticides improve the nutritional value of food and sometimes its safety (Narayanasamy 2006). There are many kinds of benefits to pesticides, these benefits often unnoticed by the public (Cooper and Dobson 2007; Damalas 2009). Pesticides can be considered as an economic, labor-saving and efficient tool of pest management in the agricultural production. Extensive use, pesticides a serious concern about health risks arising from the exposure to the farmers when applying pesticides in fields and residues on food and in drinking water for the general population (Wilson and Tisdell 2001; Maroni *et al.*, 2006; Soares and Porto 2009). These activities have caused accidental poisonings, major health risks to farmers and degrade the environment. In developing countries, farmers face great risks of exposure due to the use of toxic chemicals that are restricted in other countries as application techniques, poorly maintained, spraying, inadequate storage practices and reuse containers for food and water storage

(Ibitayo 2006; Asogwa and Dongo 2009). Exposure to pesticides cause health hazard, especially in the agricultural working environment. Most pesticides are high degree of toxicity and kill certain organisms and thus create risk. Pesticide raised serious concerns in potential effects on human health, impacts on wildlife and sensitive ecosystems (Power 2010). Pesticide applications are also kill beneficial species of natural enemies of pests and increase the chances of development of pest resistance to pesticides. Users have poor knowledge about risks to the pesticides, correct application and necessary precautions (Yassin *et al.*, 2002; Damalas *et al.*, 2006). Pesticides have been developed with reasonable and minimal risk to human health and the environment. Pesticides exposure during pesticide use, consumer exposure to pesticide residues as found in fresh fruit, vegetables and drinking water and the environment viz; water and air contamination, toxic effects on non-target organisms are fully justified (Damalas 2009; Mariyono 2008).

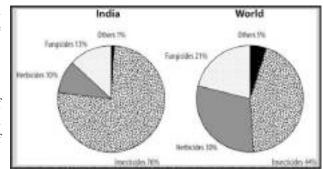
Pesticide registration is a scientifically, legal, and administrative process, the use of a pesticide its potential effect on human health and the environment (Monaco *et al.*, 2002). The registration is an important step in the management of pesticides are permitted to be used to exercise labelling, packaging and advertising of pesticides, thus ensuring that the best users as well as the environment protected (WHO 2010). The registration process is restricted to the only use their does not encourage on human health or on the environment. Effects in any non-target species explain into ecosystem unbalance and food-web disruption that ultimately may affect human health and edible species. Human exposure to pesticides in the case of agricultural workers in open fields and greenhouses, workers in the pesticide industry and house pests (Wilson and Tisdell 2001; Maroni *et al.*, 2006; Soares and Porto 2009;

Martínez *et al.*, 2009). Exposure of the general population to pesticides mainly through eating food and drinking water contaminated with pesticides, whereas exposure to pesticides living close to a workplace (Davis *et al.*, 1992; Jaga and Dharmani 2003).

Groups of Chemical Pesticides and their effects

Synthetic insecticides such as organophosphate (OP) in the 1960s, carbamates in 1970s and pyrethroids in 1980s and introduction of herbicides and fungicides in the 1970s–1980s to the pest management in agriculture crop production. India is the second largest manufacturer of pesticides in Asia after China and ranks twelfth globally (Mathur, 1999)

Indian economy is dependent on food grain production; the



Consumption of pesticides

use of machinery. Pesticides are essential part of the reducing losses by manage the weeds, diseases and insect pests. **Fungicides** toxicity to humans is generally irritating to the skin and eyes. Inhalation of spray mist causes throat irritation, sneezing, and coughing. Mostly cases of human fungicide poisoning have been consumption by the seed grains. **Herbicides** are strong acids, amines, esters, and phenols. Inhalation of spray mist may cause coughing and a burning sensation in the nasal passages and chest. Prolonged inhalation sometimes causes dizziness. Ingestion will usually cause vomiting, a burning sensation in the stomach, diarrhea, and muscle twitching. **Insecticides** cause the greatest number of pesticide poisonings. The most poisoning exposure to the organophosphate and carbamate insecticides. Organophosphate insecticides such as chlorpyrifos, diazinon, dimethoate, disulfoton, malathion, methyl parathion and ethyl parathion. The carbamate compounds are carbaryl, carbofuran, methomyl, and oxamyl. Organophosphates and carbamates inhibit the enzyme cholinesterase, causing a disruption of the nervous system. All life forms with cholinesterase in their nervous system, such as insects, fish, birds, humans, and other mammals, can be poisoned by these chemicals.

Organophosphates & Carbamates are like nerve gas they attack the brain and nervous system, interfering with nerve signal transmission. Symptoms include headaches, nausea, dizziness, vomiting, chest pain, diarrhea, muscle pain and confusion. In severe poisoning incidents, symptoms can include convulsions, difficulty breathing, involuntary urination, coma and death. Acute poisoning of the nervous system by these pesticides affects hundreds of thousands of people around the world each year. Organchlorine are banned pesticides (including DDT) although several organochlorine pesticides are still in use. Although these pesticides are generally persist in the environment and tend to accumulate in tissue as they pass up the food chain, they are extremely hazardous. Organochlorine pesticide residues and breakdown products are found in human breast milk worldwide, and also in soil and plant and animal tissue from the middle of the Pacific Ocean to the Arctic Circle. Pyrethroids are similar to the natural pyrethrins as produced by chrysanthemum flowers and are in increasingly wide use. In fact, pyrethroids are a synthetic copy of a natural poison they are an excitatory nerve poison and known carcinogen. They are also highly toxic to insects, fish and birds, even in very small doses. While

natural pyrethrum breaks down in as little as twelve hours, the synthetic forms have been engineered to be more stable, and persist in the environment. **Fumigants** are like methyl bromide and metam sodium can severely injure any tissue they touch. Effects from even minor exposures can include burning and itching of the eyes and skin, respiratory tract irritation as well as coughing and nose bleeds. Fumigants can severely injure the lungs.

As per Gazette of India extraordinary notification 14th May, 2020, following listed pesticides prohibited for import, manufacture, sale, transport, distribution and use in agriculture.

Sl. No.	Pesticides	Toxic effects
1	Acephate	There are reports of Endocrine Disruption concerns in public domain. It is an organophosphate compound, toxic to honey-bees.
2	Atrazine	Its endocrine disruption potential in public domain and toxic to aquatic organism including fish.
3	Benfuracarb	Presence of carcinogenic impurities, highly toxic via in-halatory exposure, reprotoxic effects observed in rat and rabbit. The metabolites of the products have proven to be more toxic.
4	Butachlor	The product is toxic to aquatic organism including fish.
5	Captan	The product is toxic to aquatic organism including fish.
6	Carbendazim	Carbendazim is active component of Thiophanate methyl and Benomyl. Benomyl has already been banned for use in the country. Also in view of the risk associated to pregnant women and presence of toxic impurities. Carbendazim in high risk category and its resistance is common in many fungal species.
7	Carbofuran	It is also toxic to honey bees, aquatic organisms and birds. It is also toxic to honey bees, aquatic organisms and birds.
8	Chlorpyriphos	The product is an organophosphate and is neurotoxic and has been banned for Household. Health hazards to children and infants, product is organophosphate and neurotoxic. There are reports on genotoxicity and health hazards. Only for use in desert locust.
9	2,4-D	Concentration of dioxin content, as it is carcinogenic.
10	Deltamethrin	The product is toxic to honey bees. Only except for use in desert locust and public health.
11	Dicofol	Contamination of DDT and its metabolites is a concern. Product is highly toxic to aquatic organisms including fish
12	Dimethoate	Dimethoate is an organophosphorus compound and is highly toxic. An organophosphorus compound and its metabolites are highly toxic. There are reports on genotoxicity concern.
13	Dinocap	The product is toxic to aquatic organisms including fish.
14	Diuron	The product is toxic to aquatic organisms including fish.
15	Malathion	Product is eco toxic. Only except for use in desert locust and public health.
16	Mancozeb	Product is toxic to aquatic organisms including fish and has Ethylene Thio Urea concerns.
17	Methomyl	It is extremely toxic. It is toxic to honey bees, silkworms, birds, and aquatic organisms
18	Monocrotophos	Monocrotophos is extremely toxic,, It is toxic to honey bees and aquatic organisms.
19	Oxyfluorfen	Alteration in blood parameters causes' anemia, h emolytic consequences and in liver. Possible human carcinogen It is toxic to aquatic organisms including fish and is possible human carcinogen.
20	Pendimethalin	It is highly toxic to aquatic organisms including fish.
21	Quinalphos	high acute mammalian toxicity and is an organophosphorus compound and also is highly toxic to aquatic organisms., It is highly toxic to aquatic organisms including fish.
22	Sulfosulfuron	The product is resistant against the target weed. Resistance was observ ed against target weed Phalaris minor in Punjab and Haryana.
23	Thiodicarb	High mammalian toxicity It is toxic to honey bees and aquatic organisms. Concern is methomyl metabolite which is banned.
24	Thiophanat emethyl	Carbendazim is an active component of Thiophanate methyl and Benomyl. Benomyl has already been banned for use in the country and Fungicide Resistance. It is toxic to earthworm.
25	Thiram	It is toxic to aquatic organisms including fish.
26	Zineb	Product is toxic to aquatic organisms including fish.
27	Ziram	It is toxic to aquatic organisms including fish.

Effect on Human Health

Most of the pesticides kill pests directly on contact but systemic pesticides work differently by poisoning the pollen and nectar of flowers can kill pollinators like butterflies and bees. Pesticides are toxic to humans, animals, the children are also susceptible to the toxic of pesticides which are recorded higher incidence of childhood as leukemia, brain cancer, and congenital disabilities. The Farmers and their families using the chemical pesticides in Agricultural practices regularly cause toxicity in their bodies. The pesticides can enter the body through skin, eyes, mouth and nose and cause fatigue, skin irritations, nausea, vomiting, breathing issues, brain disorders, blood disorders, liver & kidney damage. When chemical pesticides are spread across larger areas, they are carried on the wind, residues of produce, animals, run off into open water, contaminating public water supply as well as fish and seafood.

Impact of pesticide on general population as pesticide residues found on food and drinking water consists of a potential threat to human health (Magkos et al., 2006). The oral LD50 is usually lower than the dermal LD50 since pesticides can enter the bloodstream more easily through the stomach than through the skin (Nesheim et al., 2008). Highly toxic pesticide becomes more toxic when is formulated as emulsifiable concentrate because it includes very toxic organic solvents (Surgan et al., 2010, Vasilakoglou and Eleftherohorinos 1997). Pesticides are classified as there evidence of carcinogenicity in humans. The toxicity characterization (WHO and Pesticide Action Network) as 276 marketed active substances in Europe indicate that 32 out of the 76 fungicides, 25 out of the 87 herbicides and 24 out of the 66 insecticides are related to health effect (carcinogenic, endocrine disruptor, reproductive and developmental toxicity, acute toxicity) (Karabelas et al., 2009). In 51 and eight pesticides (fungicides, herbicides, and insecticides) are characterized as carcinogenic according to databases, 22 pesticides are characterized as reproductive and developmental toxicity (Pesticide Action Network) and 28 pesticides acute toxicity (WHO). Mainly toxicological studies on experimental animals (rats, dogs, and rabbits) and in some cases from epidemiological studies (health effects of long-time human exposure to low concentrations of pesticides) associated with high uncertainty in the estimation of the relevant human exposure pattern.

Pesticides increased production of food and fibre and manage vector-borne diseases, the resulted in serious health implications to man and environment. The evidence that some chemicals pesticides possible risk to humans and unwanted side effects to the environment (Forget, 1993; Igbedioh, 1991; Jeyaratnam, 1981). The serious health effects on peoples of developing countries (WHO, 1990). The world-wide deaths of millions people due to chronic diseases by pesticides poisoning per year (Environews Forum, 1999). The high risk exposed to pesticides to workers, formulators, sprayers, mixers, loaders and agricultural farm workers. Chemicals pesticides are elicit their adverse effects by antagonising natural hormones in the body long-term, low-dose exposure is increasingly linked to human health effects such as immune suppression, hormone disruption, diminished intelligence, reproductive abnormalities and cancer (Brouwer et al., 1999; Hurley et al., 1998).

Dust and liquid formulations of various pesticides in unorganised industrial sector a high occurrence of generalised symptoms (headache, nausea, vomiting, fatigue, irritation of skin and eyes) besides psychological, neurological, cardiorespiratory and gastrointestinal symptoms (Gupta et al., 1984). In the Vietnam War, United States military forces sprayed of herbicide on approximately 3.6 million acres of Vietnamese and Laotian land to remove from forest cover area destroy crops, and clear vegetation from the US bases. Herbicide formulations used mixtures of the phenoxy herbicides 2,4-dichlorophenoxyacetic acid (2,4-D) and 2,4,5-trichlorophenoxyacetic acid (2,4,5-T). Americans armed forces in Vietnam during the Vietnam War. There was evidence on cancer risk of Vietnam veterans, workers occupationally exposed to herbicides of the Vietnamese population (Frumkin, 2003).

Contamination of food material monitored of pesticide residues in plant origin products by the European Union since 1996. In 1996, seven pesticides of two groups were analysed in apples, tomatoes, lettuce, strawberries and grapes. The highest value found in the maneb group in lettuce which corresponded to a mancozeb residue of 118 mg/kg. In 1997, 13 pesticides were assessed in five commodities (mandarins, pears, bananas, beans, and potatoes). In 1998, four commodities (oranges, peaches, carrots, spinach) were analysed for 20 pesticides. In India the first reported of poisoning due to pesticides from Kerala in 1958, whereas over 100 people died by consuming wheat flour contaminated with parathion (Karunakaran, 1958). In a multi-centric study to pesticide residues in selected food commodities and collected from different states of the country as DDT residues were found in about 82% of the bovine milk collected from 12 states. The average DDT and BHC consumed by an adult were 19.24 mg/day and 77.15 mg/day respectively (Kashyap et al., 1994).

Effect on the Environment

Pesticides and their possible negative effects on human health and adverse effects on the environment (Mariyono 2008). Pesticides linked with harmful effects on non-target micro-organisms, water contamination from pesticides, air pollution from volatile pesticides, injury on non-target plants from herbicide, injury to rotational crops from herbicide residues, crop injury due to high

application rates, unfavourable environmental pesticide application (Eleftherohorinos 2008). The harmful effects of pesticides on the environment, interactions between the physic-chemical properties of the pesticide, soil adsorption and soil persistence, soil factors plant species, and climatic variation (Eleftherohorinos 2008). Soil factors and weather conditions have long been recognised as the most important factors of the pesticide in the environment and pesticide activity, selectivity cause adverse effects on the environment (Monaco et al., 2002). The agricultural soil is the primary recipient of pesticides, water bodies that are adjacent to agricultural areas are usually the recipient for pesticide residues (Pereira et al., 2009) assess the risks of pesticides for water contamination, soil organisms (earthworms), bees, air emissions, bio-accumulation and human health (Reus et al. 2002; Bockstaller et al. 2009).

Pesticides can contaminate soil, water, grass and other vegetation. In addition to killing insects and weeds, pesticides can be toxic to birds, fish, beneficial insects, and non-target plants. Insecticides are generally the most toxic class of pesticides, but herbicides can also risks to non-target organisms. Contamination of water by pesticides is widespread through runoff from treated plants and soil. More than 90 percent of water and fish samples contained by several pesticides (Kole et al; 2001). Pesticides were found in all samples from major rivers (Bortleson and Davis, –19871995). Groundwater pollution to pesticides is a worldwide problem. During one survey in India, drinking water samples from various hand pumps and wells around Bhopal were contaminated with Organo Chlorine pesticides (Kole and Bagchi, 1995). Pesticides are determined by some parameters, such as water solubility, soil-sorption, water coefficient, and half-life in soil. Pesticides grouped into hydrophobic, persistent, and bio-accumulable pesticides that are strongly bound to soil. Pesticides such as organochlorine DDT, endosulfan, endrin, heptachlor, lindane are now banned in agriculture but their residues are still present. Polar pesticides are represented mainly by herbicides; they include in carbamates, fungicides and some organophosphorus insecticide. They moved from soil by runoff and leaching, causing problem for the supply of drinking water to the population. The organic matter adsorption the pesticides. The capacity of the soil to hold positively charged ions in form pesticides. Soil pH is also of some importance. Adsorption increases with decreasing soil pH for ionizable pesticides (2,4-D, and atrazine) (Andreu and Pico', 2004).

Overuse of chemical fertilizers and pesticides have effects on beneficial soil microorganisms to decline. Indiscriminate use of chemicals might work for a few years, but there aren't enough beneficial soil organisms to hold onto the nutrients" (Savonen, 1997). Landscape herbicides disrupt process of soil bacteria that transform ammonia into nitrite (Pell et al., 1998); glyphosate reduces the growth and activity of free-living nitrogen-fixing bacteria in soil (Santos and Flores, 1995) and 2,4-D reduces nitrogen fixation by the bacteria that live on the roots of bean plants (Arias and Fabra, 1993; Fabra et al., 1997), reduces the growth and activity of nitrogen-fixing blue-green algae (Singh and Singh, 1989; Tözüm-Çalgan and Sivaci-Güner, 1993), and inhibits the transformation of ammonia into nitrates by soil bacteria (Frankenberger et al., 1991, Martens and Bremner, 1993). Mycorrhizal fungi fungi also be damaged by herbicides in the soil (Kelley and South, 1978).

Pesticide sprays can directly hit non-target vegetation, volatilize from the treated area can contaminate air, soil, and non-target plants. Phenoxy herbicides, including 2,4-D, can injure nearby trees and shrubs (Dreistadt et al., 1994). Exposures to the herbicide glyphosate severely reduce seed quality (Locke et al., 1995). It can also increase the susceptibility of certain plants to disease (Brammall and Higgins, 1998). Some insecticides and fungicides can also damage plants (Dreistadt et al., 1994). Pesticides are found in contaminants in soil, air, water in urban area. They can harm non-target plants, animals, beneficial soil microorganisms, insects, fish, birds etc. The Ganges river basin is heavily polluted by fertilizers, pesticides, and industrial and domestic effluents (Mohan, 1989). The herbicide oxadiazon is also toxic to bees, which are pollinators (Washington State Department of Transportation, 1993).

Reduce the harmful effect

To reduce the exposure to entry routes such as dermal, inhalation, oral and eyes and protect your health against pesticides. Many toxic chemicals through the respiratory system is the quickest and most direct route of entry into the circulatory system when pesticide powders, dusts, gases, vapors and spray droplets. After handling with pesticides, wash your hands and face thoroughly with soap and water before eating, drinking, or smoking. Use of natural remedies for pests' management and come across organic and local food products, visit to local farmer's markets. Several produce items that are contains the highest levels of pesticides such as fruit like cherries, apples, peaches, pears and grapes and vegetables such as celery, spinach, and sweet bell peppers. The people have increasingly pesticide use and about their impacts on human health and environmental quality (Damalas 2009). These increased be troubled from reduced in the agricultural and industrial production as well as the authority's regulations at protecting both the environment and human health. The probability of reducing the environmental risk associated with the pesticide use is very low because the producers believe that lowering risk either decreased output or increased input resulting by the substitution for the pesticide inputs (Paul *et al.*, 2002). Reducing the risks associated with the use of pesticides will impose costs on the agricultural

community, which in turn has implications for agricultural commodity prices. This has been confirmed by the cost-function-based production model (Paul *et al.* 2002). The costs are directly associated with effective pesticides in agricultural output and pesticide quality associated with increased cost.

The agricultural scientists started to develop alternative crop management systems to minimize the harmful effects of farming to the environment and to human health. The Integrated Crop Management includes guidelines to be used by the farmer for production of safe agricultural products with simultaneous respect to the environment (Frangenberg, 2000; Baker *et al.*, 2002; Tsakiris *et al.*, 2004; Nwilene *et al.*, 2008). Implementation of good agricultural practices, the safety and hygiene of workers, the safety of the products, the full traceability of the measurements, and specific actions for the preservation of the environment (Chandler et al., 2008). In the pest management use of complementary methods such as crop resistance against insects and fungi, biological control, and other cultural or physical measures to reduce the economic injury level and minimise pesticide on other components of the agroecosystem (Kogan 1998; Way and Van Emden 2000). Integrated Crop Management allows pesticide use only through an Integrated Pest Management (IPM) program (Mariyono 2008, Nwilene *et al.*, 2008; Chandler *et al.*, 2008). Pesticides that are selected for use in IPM, biologically effective, eco-friendly, environmentally compatible, economically viable (Palacios 2010). The introduction of IPM system would contribute a significant reduction of the pesticide impact on human health and the environment without affecting crop productivity. (Burger et al., 2008;, Mariyono 2008; Nwilene *et al.*, 2008). New agrochemicals with novel modes of action are safety and reality (Bhattacharyya *et al.*, 2009). More effective pesticides application is the well-established technologies in agriculture that will play important role in the agribusiness and rapid emergence of novel biotechnological. (Neumann 1997; Smith *et al.*, 2008).

Conclusions

Pesticides are widely used and considered as quick, easy, inexpensive solution in agricultural crop production to prevent the pests, diseases, weeds to reduce yield losses and maintain high product quality. Pesticides are developed through the regulation processes with reasonable and minimal impact on human health and the environment. All pesticides have the potential to be harmful to humans, other living organisms and the environment. The health risks resulting from exposure and residues in food and drinking water. Pesticides occurs primarily through eating food and drinking water contaminated with pesticide residues. The adverse effects on the environment *viz*; water, soil and air contamination from leaching, runoff, and spray drift, as well as the injurious effects on wildlife, fish, bird and plants and non-target organisms depend on the toxicity of the pesticide as pesticide persists in the environment. The development of new pesticides use in cropping systems for minimize to exposure on human, residues in food and drinking water and reduction of the adverse effects on human health, environment and agricultural production. The risks to human health due to pesticides are variables on age, race, socio-economic status, diet, state of health affect exposure to pesticides. The prevent health effects in adverse environment and support to sustainable crop production and development economics.

- 1. Andreu V, Pico' Y. Determination of pesticides and their degradation products in soil: critical review and comparison of methods. Trends Anal Chemistry. 2004; 23(10–11):772–789.
- 2. Arias RN, Fabra PA. Effects of 2,4-dichlorophenoxyacetic acid on Rhizobium sp. growth and characterization of its transport. Toxicol Lett. 1993; 68:267–273.
- 3. Asogwa EU, Dongo LN. Problems associated with pesticide usage and application in Nigerian cocoa production: A review. Afr. J. Agr. Res. 2009; 4:675–683.
- 4. Baker BP, Benbrook CM, Groth E, III, Lutz Benbrook K. Pesticide residues in conventional, integrated pest management (IPM)-grown and organic foods: insights from US data sets. Food Addit. Contam. A. 2002; 19:427–446.
- 5. Berny P. Pesticides and the intoxication of wild animals. J. Vet. Pharmacol. Ther. 2007; 30:93–100.
- 6. Bhattacharyya A, Barik SR, Ganguly P. New pesticide molecules, formulation technology and uses: Present status and future challenges. J. Plant Prot. Sci. 2009;1:9–15
- 7. Bockstaller C, Guichard L, Keichinger O, Girardin P, Galan MB, Gaillard G. Comparison of methods to assess the sustainability of agricultural systems. A review. Agron. Sustain. Dev. 2009; 29:223–235.
- 8. Bortleson G, Davis D. 1987–1995. U.S. Geological Survey & Washington State Department of Ecology. Pesticides in selected small streams in the Puget Sound Basin; pp. 1–4.
- 9. Brammall RA, Higgins VJ. The effect of glyphosate on resistance of tomato to Fusarium crown and root rot disease and on the formation of host structural defensive barriers. Can J Bot. 1988;66:1547–1555.

- 10. Brouwer A, Longnecker MP, Birnbaum LS, Cogliano J, Kostyniak P, Moore J, Schantz S, Winneke G. Characterization of potential endocrine related health effects at lowdose levels of exposure to PCBs. Environ Health Perspect. 1999;107:639.
- 11. Chandler D, Davidson G, Grant WP, Greaves J, Tatchell GM. Microbial biopesticides for integrated crop management: An assessment of environmental and regulatory sustainability. Trends Food Sci. Technol. 2008; 19:275–283.
- 12. Commission of the European Communities. Council Directive 91/414/EEC of 15 July 1991 Concerning the Placing of Plant Protection Products on the Market. Commission of the European Communities; Brussels, Belgium: 1991. Official Journal L 230.
- 13. Cooper J, Dobson H. The benefits of pesticides to mankind and the environment. Crop Prot. 2007; 26:1337–1348.
- 14. Damalas CA. Understanding benefits and risks of pesticide use. Sci. Res. Essays. 2009; 4:945–949.
- 15. Davis JR, Brownson RC, Garcia R. Family pesticide use in the home, garden, orchard, and yard. Arch. Environ. Contam. Tox. 1992; 22:260–266.
- 16. Damalas CA, Theodorou MG, Georgiou EB. Attitudes towards pesticide labelling among Greek tobacco farmers. Int. J. Pest Manage. 2006; 52:269–274.
- 17. Dreistadt SH, Clark JK, Flint ML. An integrated pest management guide. University of California Division of Agriculture and Natural Resources; 1994. Pests of landscape trees and shrubs. Publication 3359.
- 18. Eleftherohorinos IG. Weed Science: Weeds, Herbicides, Environment, and Methods for Weed Management. AgroTypos; Athens, Greece: 2008.
- 19. Environews Forum. Killer environment. Environ Health Perspect. 1999; 107:A62.
- 20. Fabra A, Duffard R, Evangelista DDA. Toxicity of 2,4-dichlorophenoxyacetic acid in pure culture. Bull Environ Contam Toxicol. 1997;59:645–652.
- 21. Frankenberger WT, Tabatabai MA, Jr, Tabatabai MA. Factors affecting L-asparaginase activity in soils. Biol. Fert. Soils. 1991; 11:1, 5.
- 22. Forget G. Balancing the need for pesticides with the risk to human health. In: Forget G, Goodman T, de Villiers A, editors. Impact of Pesticide Use on Health in Developing Countries. 1993. IDRC, Ottawa: 2.
- 23. Frumkin H. Agent Orange and Cancer: An Overview for Clinicians. CA Cancer J Clin. 2003;53:245.
- 24. Frangenberg A. Integrated Crop Management as fundamental basis for sustainable production. Pflanzenschutz-Nachrichten Bayer. 2000; 53:131–153.
- 25. Gupta SK, Jani JP, Saiyed HN, Kashyap SK. Health hazards in pesticide formulators exposed to a combination of pesticides. Indian J Med Res. 1984; 79:666.
- 26. Hurley PM, Hill RN, Whiting RJ. Mode of carcinogenic action of pesticides inducing thyroid follicular cell tumours in rodents. Environ Health Perspect. 1998; 106:437.
- 27. Ibitayo OO. Egyptian rural farmers' attitudes and behaviors regarding agricultural pesticides: Implications for pesticide risk communication. Risk Anal. 2006;26:989–995
- 28. Igbedioh SO. Effects of agricultural pesticides on humans, animals and higher plants in developing countries. Arch Environ Health. 1991; 46:218.
- 29. Jaga K, Dharmani C. Sources of exposure to and public health implications of organophosphate pesticides. Pan. Am. J. Public Health. 2003; 14:171–185.
- 30. Jeyaratnam J. Health problems of pesticide usage in the third world. B M J. 1985; 42:505.
- 31. Kashyap R, Iyer LR and Singh MM. "Evaluation of Daily Dietary Intake of Dichlorodiphenyltrichloroethane (DDT) and Benzene Hexachloride (BHC) in India," Archives of Environmental Health. 1994; 49: 63-66
- 32. Karabelas AJ, Plakas KV, Solomou ES, Drossou V, Sarigiannis DA. Impact of European legislation on marketed pesticides—A view from the standpoint of health impact assessment studies. Environ. Int. 2009; 35:1096–1107.
- 33. Karunakaran C.O. The Kerala food poisoning. J Indian Med Assoc. 1958; 31:204.
- 34. Kelley WD, South DB. Weed Sci. Soc. America Meeting. Auburn, Alabama: Auburn University; 1978. In vitro effects of selected herbicides on growth and mycorrhizal fungi; p. 38.

- 35. KEMI . Interpretation in Sweden of the Impact of the "Cut-off" Criteria Adopted in the Common Position of the Council Concerning the Regulation of Placing Plant Protection Products on the Market (Document No 11119/08) Swedish Chemicals Agency; Sundbyberg, Sweden: 2008. p. 14.
- 36. Kole RK, Bagchi MM. Pesticide residues in the aquatic environment and their possible ecological hazards. J Inland Fish Soc India. 1995; 27(2):79–89.
- 37. Kole RK, Banerjee H, Bhattacharyya A. Monitoring of market fish samples for Endosulfan and Hexachlorocyclohexane residues in and around Calcutta. Bull Environ Contam Toxicol. 2001; 67:554–559.
- 38. Kogan M. Integrated pest management: Historical perspectives and contemporary development. Ann. Rev. Entomol. 1998; 43:243–270.
- 39. Locke D, Landivar JA, Moseley D. The effects of rate and timing of glyphosate applications of defoliation efficiency, regrowth inhibition, lint yield, fiber quality and seed quality; Proc. Beltwide Cotton Conf., National Cotton Council of America; 1995. pp. 1088–1090.
- 40. Mathur SC. Future of Indian pesticides industry in next millennium. Pesticide Information .1999: 24(4): 9–23.
- 41. Magkos F, Arvaniti F, Zampelas A. Organic Food: Buying more safety or just peace of mind? A critical review of the literature. Crit. Rev. Food Sci. Nutr. 2006;46:23–55.
- 42. Martens DA, Bremner JM. Influence of herbicides on transformations of urea nitrogen in soil. J Environ Sci Health B. 1993; 28:377–395.
- 43. Martínez-Valenzuela C, Gómez-Arroyo S, Villalobos-Pietrini R, Waliszewski S, Calderón-Segura ME, Félix-Gastélum R, Álvarez-Torres A. Genotoxic biomonitoring of agricultural workers exposed to pesticides in the north of Sinaloa State, Mexico. Environ. Int. 2009;35:1155–1159
- 44. Mariyono J. Direct and indirect impacts of integrated pest management on pesticide use: A case of rice agriculture in Java, Indonesia. Pest Manag. Sci. 2008; 64:1069–1073.
- 45. Maroni M, Fanetti AC, Metruccio F. Risk assessment and management of occupational exposure to pesticides in agriculture. Med. Lav. 2006; 97:430–437.
- 46. Mohan RSL. Conservation and management of the Ganges river dolphin, *Platanista gangetica*, in India. In: Perrin WF, Brownell RL Jr, Kaiya Z, Jiankang L, editors. Proceedings, Workshop on Biology and Conservation of the Platanistoid Dolphins; 1989. pp. 64–69. Wuhan, China, October 28–30, 1986.
- 47. Monaco JT, Weller SC, Ashton FM. Herbicide registration and environmental impact. In: Monaco TJ, Weller SC, Ashton FM, editors. Weed Science: Principles and Practices. 4th ed. John Wiley & Sons; New York, NY, USA: 2002.
- 48. Narayanasamy P. Postharvest Pathogens and Disease Management. John Wiley & Sons; New York, NY, USA: 2006.
- 49. Nesheim ON, Fishel FM, Mossler M. Toxicity of Pesticides. Pesticide Information Office, Food Science and Human Nutrition Department, Florida Cooperative Extension Service, Institute of Food and Agricultural Sciences, University of Florida; Gainesville, FL, USA: 2008. p. 7.
- 50. Neumann R. Chemical crop protection research and development in Europe. Develop. Crop Sci. 1997; 25:49–55.
- 51. Nwilene FE, Nwanze KF, Youdeowei A. Impact of integrated pest management on food and horticultural crops in Africa. Entomol. Exp. Appl. 2008; 128:355–363.
- 52. Paul CJM, Ball VE, Felthoven RG, Grube A, Nehring RF. Effective costs and chemical use in United States agricultural production: using the environment as a 'free' input. Am. J. Agr. Econ. 2002; 84:902–915.
- 53. Palacios Xutuc CN. Manual to Train Trainers on Safe and Correct Use of Plant Protection Products and Integrated Pest Management (IPM) CropLife Latin America; Guatemala City, Guatemala: 2010.
- 54. Pell M, Stenberg B, Torstensson L. Potential denitrification and nitrification tests for evaluation of pesticide effects in soil. Ambio. 1998; 27:24–28.
- 55. Pereira JL, Antunes SC, Castro BB, Marques CR, Gonçalves AMM, Gonçalves F, Pereira R. Toxicity evaluation of three pesticides on non-target aquatic and soil organisms: Commercial formulation *versus* active ingredient. Ecotoxicology. 2009; 18:455–463.
- 56. Power AG. Ecosystem services and agriculture: Tradeoffs and synergies. Phil. Trans. R. Soc. B. 2010; 365:2959–2971.

- 57. Reus J, Leendertse P, Bockstaller C, Fomsgaard I, Gutsche V, Lewis K, Nilsson C, Pussemier L, Trevisan M, van der Werf H, et al. Comparison and evaluation of eight pesticide environmental risk indicators developed in Europe and recommendations for future use. Agr. Ecosyst. Environ. 2002;90:177–187
- 58. Santos A, Flores M. Effects of glyphosate on nitrogen fixation of free-living heterotrophic bacteria. Lett Appl Microbiol. 1995;20:349–352.
- 59. Savonen C. Soil microorganisms object of new OSU service. Good Fruit Grower. 1997. http://www.goodfruit.com/archive/1995/6other.html.
- 60. Singh JB, Singh S. Effect of 2,4-dichlorophenoxyacetic acid and maleic hydrazide on growth of bluegreen algae (cyanobacteria) Anabaena doliolum and Anacystis nidulans. Sci. Cult. 1989; 55:459–460.
- 61. Smith K, Evans DA, El-Hiti GA. Role of modern chemistry in sustainable arable crop protection. Phil. Trans. R. Soc. B. 2008;363:623–637.
- 62. Soares WL, Porto MFD. Estimating the social cost of pesticide use: An assessment from acute poisoning in Brazil. Ecol. Econ. 2009;68:2721–2728.
- 63. Surgan M, Condon M, Cox C. Pesticide risk indicators: Unidentified inert ingredients compromise their integrity and utility. Environ. Manag. 2010;45:834–841.
- 64. Tözüm-Çalgan SRD, Sivaci-Güner S. Effects of 2,4-D and methylparathion on growth and nitrogen fixation in cyanobacterium, Gloeocapsa. Intern J Environ Stud. 1993; 23:307–311.
- 65. Tsakiris IN, Danis TG, Stratis IA, Nikitovic N, Dialyna IA, Alegakis AK, Tsatsakis AM. Monitoring of pesticide residues in fresh peaches produced under conventional and integrated crop management cultivation. Food Addit. Contam. A. 2004;21:670–677
- 66. Vasilakoglou IB, Eleftherohorinos IG. Activity, adsorption, mobility, efficacy, and field persistence of alachlor as affected by formulation. Weed Sci. 1997;45:560–565
- Washington State Department of Transportation. Draft roadside vegetation management environmental impact statement, appendix B. 1993:B2–10.
- 68. Way MJ, Van Emden HF. Integrated pest management in practice—pathways towards successful application. Crop Prot. 2000;19:81–103
- 69. WHO. Geneva: World Health Organization; 1990. Public Health Impact of Pesticides Used in Agriculture; p. 88.
- 70. WHO. International Code of Conduct on the Distribution and Use of Pesticides: Guidelines for the Registration of Pesticides. World Health Organization; Rome, Italy: 2010.
- 71. Wilson C, Tisdell C. Why farmers continue to use pesticides despite environmental, health and sustainability costs. Ecol. Econ. 2001; 39:449–462.
- 72. Yassin MM, Abu Mourad TA, Safi JM. Knowledge, attitude, practice, and toxicity symptoms associated with pesticide use among farm workers in the Gaza Strip. Occup. Environ. Med. 2002;59:387–393.

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LASODA: A POTENTIAL FRUIT WITH IMMENSE PHARMACOLOGICAL PROPERTIES

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Abstract

Minerals play a vital role in the regulation of various metabolic activities in a human body. Wild fruits are an important and cheaper source of minerals, required for growth and development of the body. Cordia dichotoma (Lasoda) is an unexploited fruit with immense nutritional potential. Fruits are rich in various macro and micro minerals. Immature fruits of Cordia dichotoma possess high calcium, magnesium, sodium, iron and zinc, while mature fruits of Cordia dichotoma were rich in phosphorus. Mature fruits were also good in potassium and copper content. Herbal drugs play an important role in healthcare programs in the treatment of various diseases including depression disorders. In India Ayurveda, a traditional medicinal system, assigns much importance to the pharmacological aspects of many plants. Cordia dichotoma is used as herbal drug to treat various diseases. This herb has various medicinal effects like astringent, anthelmintic, diuretic, demulcent and expectorant. Thus, their consumption can lead to a healthy life free from deficiency disorders, cardiovascular diseases and cancer risks. The fruit and the bark of the tree can be highly beneficial for control of respiratory tract infections, anthelmintic, and cough.

Introduction

Medicinal plants play an important role in Indian culture since Rig Veda (5600 BC) where in 67 medicinal plants were recorded. Out of 250, 000 higher plants, more than 80, 000 plants have medicinal values and India occupies a unique position among the world's 12biodiversity centers. The use of natural substances, particularly plants, to control diseases is a centuries old practice that has led to the discovery of more than half of all modern pharmaceuticals. A growing worldwide interest in the use of phytopharmaceutical as complementary or alternative medicine either to prevent or ameliorate many diseases has been noted in recent years. [1] Lisoda is a common medicinal plant in southern India and its botanical name is Cordia dichotoma. Apart from Bhokar, the plant is also known as Chhota lasora and Gondi, Guslasah, Lasora and Rassala, in Hindi. The people speaking Marathi know the plant in the names of Bhokar, Chokri, Sherti, Shelu and Shelvant. While the Tamil speaking people name the plant as Kalvirusu, Naruvili, Vidi, Viriyan or Viruvu; It is named as Botuka, Chinna, Inki, Iriki, Nakheri and Nakkeru, in Telugu. [2] Lasoda is a tree growing to a height of 10-15 m and is seen all over the plains of India. The bark of the stem and trunk is brown. The leaf of the tree looks slimy and smooth. The fruits are whitish in color and bloom during the spring. The fruits are oval, sweet in taste and contain a solitary seed.



Lasoda: Cordia dichotoma

The plant parts like fruits, leaves, stem bark, seeds and roots of most species of plants of the genus Cordia, has long been used in traditional medicine for cicatrizant, astringent, anti-inflammatory, anthelmintic, antimalarial, diuretic, febrifuge, appetite suppressant, cough suppressant and to treat urinary infections, lung diseases and leprosy.

Nutritional content

The whole plant of *C.dichotoma* is edible and is used as food. Immature fruits are pickled and are also used as vegetables [3]. Mature fruits of lasoda are very nutritious as they provide 65 calories, contains 74-82.5g water, 1.0g fat, being rich in carbohydrates (12.2/100g) of edible portion, Phosphorus (60mg/100g) of pulp, 1.8-2.0g protein, calcium (40mg/100g) of pulp, total ash (2.13%), vitamins and minerals. The polysaccharide gum (97%) obtained from the plant used for various pharmaceutical purposes.

In Ayurveda, leaves and stems bark is used for dyspepsia, fever, diarrhoea and lprosy. The bark is moistened and applied to boils and tumours to hasten ripening. Bark is used as an anti dyspeptics and as febrifuge. Powdered bark is used for mouth ulcers. Infusion of bark used as a gargle. The bark juice mixed with coconut milk is used to relieve colicky pains. Chromium present in the fruit has therapeutic value in diabetes. The highly mucilaginous fruits are used for cough and ailments of the chest, uterus and urethra. In large quantities it may be used as a laxative. A fruit also contains some anti-nutritional factors such as phytic acid (355mg), phytate phosphorus (100g) and oxalic acid (250mg) per 100g [6]. In India, it is traditionally used for ulcerative colitis, ulcers and colic pain. In Bengal, fresh fruit is used as a laxative and pectoral. In Java, it's fruit is used for gonorrhea. In Punjab and Kashmir, its dried fruit is used as an expectation.

Usability in Various aspects

- 1. **Food:** The immature fruits are pickled and are also used as a vegetable Fodder: The leaves yield good fodder and are lopped for this purpose. The seed kernel of *C.dichotoma* contains a high proportion of fatty oils and proteins [46 and 31%, respectively) which has potential as cattle feed.
- 2. **Fuel:** The tree is used as a fuel wood. Timber: The wood is used to make agricultural implements.
- **3. Insecticides:** fruit extract of *C. dichotoma* suppresses larval hatching of Meloidogyne incognita.
- **4. Pharmaceutical uses:** seeds of the species are anti-inflammatory, two compounds alpha-amyrin and 5- di rhamnoside have been isolated. The bark is medicinal and several chemicals have been identified; Allantoin, beta-sitosterol and 3',5-dihydroxy-4'-methoxy flavanone-7-O- alpha-L-rhamnopyranoside isolated from it. The seed kernel has also many medicinal properties.
- **Services:** It is a quick growing fruit tree, performing well under semi-arid conditions and suitable for planting along boundary and farm roads.
- **Miscellaneous:** In the Philippines, rope is made from the phloem of the tree usually for cord and matting, the fruit, the white gelatinous substance is used as glue. Fishes are cooked wrapped in leaves. The leaves substitute for wrappers.

Pharmacological Properties

Antimicrobial activity: Extract of *Cordia dichotoma* also shows moderate activity against bacterial, fungal and yeast species. Antibacterial activity of methanol and butanol extracts of the bark was carried out against two gram negative bacteria (Escherichia coli, and Pseudomonas aeruginosa) and two Gram positive bacteria (St. pyogenes and Staphylococcus aureus). The antifungal activity of the extracts was carried out against three common pathogenic fungi (Aspergillus niger, A.clavatus, and Candida albicans). The extracts showed remarkable inhibition of zone of bacterial growth and fungal growth and the results obtained were comparable with that of standards drugs against the organisms tested. The activity of extracts increased linearly with increase in concentration of extract (mg/ml). The results showed the antibacterial and antifungal activity against the organisms tested.

Antiulcer activity: The volume of gastric secretion, free acidity, total acidity and ulcer index is reduced by Lasoda.[4] The fruit extract of Cordia dichotoma Forst shows antiulcer activity. Extractions of Cordia dichotoma fruits were carried out using ethanol. Plant extract has phenolic content and anti- oxidant activity. It possesses hepatoprotective activity.

Anti-Inflammatory activity: The dry powdered seeds were found to contain alkaloids, glycosides, saponins, tannins and carbohydrates. Thus it is found that ethanol extract and aqueous fraction of this plant possesses acute antiinflammatory activity .[5]

Antidiabetic activity: Anti hyperglycemic effects of Cordia dichotoma Forst in glucose induced hyperglycemia.

Anthelmintic: The anthelmintic activity of ethanolic and aqueous extracts of C. dichotoma on Eudrilus eugeniae earthworms. Both extracts showed concentration dependent paralysis and death of worms, with the aqueous extract showing more significant activity. [6]

Pharmaceutical industrial uses

- (a) **Tablet binder:** The fruit of cordia dichotoma is highly sticky in nature. This property is used for binding tablets. In future Cordia gum could compete favorably with gelatin as binder in tablet formulations. fruit of cordia dichotoma is highly sticky in nature this property is used for binding of tablets. In future Cordia gum could compete favorably with gelatin as binder in tablet formulations.
- **(b) Emulsifier:** The Cordia gum is a good option as biodegradable, cheap, economic and easily available emulsifier in the list of pharmaceutical excipient.[7-8]

Processing for value - added products: Tender fruits can not be retained for long periods at room temperature as they turn yellow and become unsuitable for consumption. They require cleaning, grading and blanching for consumption proposes. The fruits are blanched in a solution containing NaCl (1percent)+ sugar (1percent) at 100 oC for eight to ten minutes followed by sudden cooling in tap water and dipping in 0.2 percent NaCl solution. The destoning of fruits is also required. The destined pulpy halves of the fruits are either sundried or put in mechanical driers. The dried pieces are packed in polyethylene bags and can be used for cooking after rehydration.

Conclusion

The use of natural substances, particularly plants, to control diseases is a centuries old practice that has led to the discovery of more than half of all modern pharmaceuticals. Various texts in literature and research, studies in modern science describe the useful properties of Cordia dichotoma as anthelmintic, antimalarial, diuretic etc which shows usefulness and importance of this plant. However, only a few work has been done on this plant and there is a large scope of investigation for researchers to explore its potential in the field of medicinal research and pharmaceutical sciences.

- 1. Anjana K. Patel, Nimish Pathak, Hardik Trivedi, Mahendra Gavania, Mihir Patel, Nitin Panchal C.U.Shah College of Pharmacy & Research, Wadhwan.Phytopharmacological Properties of Cordia Dichotoma As A Potential Medicinal Tree: AN OVERVIEW, International journal of institutional pharmacy and life sciences,
- 2. http://www.indianetzone.com/38/indian_cherry_bahuvaraka_plants.htm.
- 3. Hussain N, Kakoti BB. Review on ethnobotany and psychopharmacology of Cordia dichotoma. J Drug Deliv Ther. 2013;3(1):110–113. [Google Scholar]
- 4. Wassel G, El-Menshaw B, Saud A, Meharuna G and El-Merzabani M. Screening of selected plants for Pyrrolizidine alkaloids and antitumor activity. Pharmazine 1987; 42:709.
- 5. Anjana K Patel, Nimish pathak, Hardik Trivedi, Mahendra G, Mihir P, Nitin P. Phytopharmacological properties of Cordia dichotoma as a Potential Medicinal Tree: an overview. Int. J. of Institutional Pharmacy and Life Sciences 2011;1(1).
- 6. Maisale AB, Attimarad SL. Anthelmintic Activity of Fruit Pulp of Cordia dichotoma. Int Journal Res App Pharm 2010; 1 (2): 597-600.
- 7. Nazim Hussain, Kakoti BB. Review on ethnobotany and phytopharmacology of cordia dichotoma, J Drug Deli & Ther 2013; 3(1): 110-113.
- 8. Yoganarasimhan SN. Medicinal plants of India, Banglore: Interline publishing pvt. Ltd. 2000; 138-140

Three Major dimensions of life: Environment, Agriculture and Health

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Jyoti Verma, Sandeep Kushwaha, Shishu Pal Singh and Piyush Raman Pandey

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ROLE OF HEALTH, HYGIENE AND SANITATION IN MANAGEMENT OF PANDEMICS

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Abstract

Novel Corona Virus Disease-19 (Covid-19) has been declared on March 11, 2020 by World Health Organization as a global pandemic considering the following points:-

- Corona Virus was reported 5 months back from the date of declaration from WUHAN the city of China and has spread
 speedily throughout the world in geographic and climatic areas. It has also spread in urban, rural, desert, hilly and coastal
 areas.
- 2. The disease is deadly and infects only human beings reported so far. There is no effect of any antibiotic and other medicine in management of disease. Symptoms reported so far concerned to this disease are related to respiratory tract of human. The disease is contaminable when vector comes in contact with another healthy person with possible source of transmission of nasal sneezing, cough, contact of body etc.

The precautions suggested by various health organizations to terminate the possibilities of transmission include:-

- (1) Social distancing
- (2) Avoid as much possible following the norms which include health, hygiene and sanitation practices.

Health, hygiene and sanitation plays key role in management of pandemic. The present communication deals with health, hygiene and sanitation practices to keep person, family, society and nation free from such unknown deadly pandemic at least till discovery of its vaccine by the medical science. However Health, hygiene and nutrition are the key pillars which can overcome such emergency.

Introduction

Corona Virus is a kind of viruses that infects our respiratory tract. The name "corona virus" is derived from Latin word meaning "crown". The virus is simple a dead particle outside its host. It specifically enters human body i.e. human being is its vector where it behaves like living object which multiplies its RNA strand at the expense of material and energy of the host. The first host of Covid-19 is claimed to be a lady of WUHAN the city of China. Such units are called zero patients. How it spread from the zero patient to others is a matter of debate? People say that basically it was found to be present in flying mammals Bats. Such reports are still to be looked extensively and intensively. The viral infection has so speedily spread in humans that it could not be managed so far by any therapy. Its infection has feared all sections of human populace. The update figure of rate of infection, casualty and multiple speed of infection are self explanatory.

Globally statistics of Covid-19 Patients date 27/5/2020

Total Affected-559 M

Recovered-229M

Death-350K

Health is a state of physical, mental and social well being in which diseases and infirmity is present. Health of an individual is determined by above three aspects.

As physical health is defines the condition of individual body taking into consideration the absence of diseases. It is affected by lifestyle, diet, level of physical activity and behavior. During Covid-19 every individual has to work from home and so the normal

routine is disturbed leading to various physical health issues. For healthy living during this crisis holistic approach has to be made to overcome the crisis. It includes:-

- Set a modified life style change in routine
- Changing food habits
- Get enough sleep
- Exercise/Yoga

During Covid-19 mental health is a subject of concern to all as it enhances the issues like fear, worry due to mental stress. Everyone is facing with new challenges of no social gathering, staying in isolation, work from home, unemployment, being away from friends and colleagues.

All these have affected individual social health. It ultimately affected the mental strata of the individual as Man is a social animal. To overcome such stresses it is recommended to follow modified schedule to which may includes- music, self management, try new hobbies or any creative works etc.

Social activities have to be performed by the individual to keep himself mentally and physically active, increases life expectancy, enhanced immunity, lower risks of BP, depression, cardiovascular diseases, Alzheimer etc.

Hygiene is the groups of practices that can perform to improve cleanliness and helps to maintain good health. It includes hand washing, face washing and bathing with soap and water so as to stop the spread of germs responsible for illness. Simple hygiene practices should be made as compulsory habit by everyone because it keeps away from all sorts of health disorders specially contaminated and spreaded by microbes. Following precautions should be kept in mind-

- · Wash hand frequently with soap and water for at least 20-30 seconds.
- · Avoid touching unnecessary parts of body.
- · Cover your mouth and nose with elbow while sneezing and coughing.
- · Cleaning and disinfecting surfaces in your home regularly.
- · Precautions should be kept in mind during laundry at home like Do not wave your clothes, Use soaps or detergents and water, Strictly avoid using used clothes by anyone else.
- · Cover your face with mask

In Environment human beings occupies a key position because human are supposed to the highest evolved creatures. Besides they have capacity to interact with the resources available to them and make use as they deserve. This has generated many vital environmental problems. Man faces large number of a biotic and biotic components some of them are serious thereto to life a person can easily counter such threat in case his/her inbuilt potential i.e. immune system to strong enough to counteract such threats.

Nutrition is vital to health and ultimately supports inbuilt immunity to certain extent.

Immune nutrition can be defined as modulation of either the activity of immune system or modulation of the consequences of activation of the immune system by nutrients or specific food items feel in amount above those normally encountered in the diet.

Nutrition is almost important to maintain the physiological functions of life.

Nutrition is a vital biological activity which makes nutrients taken by an individual to build up maintain and energy transformation process in human system via digestion, assimilation, and absorption.

Certain Immune Boosters nutrients and their dietary sources:

Nutrients Dietary SourcesVitamin CPapaya, Brooccolli, Capsicum, Potatoes, Strawberries, Lemon, Orange Vitamin B₆Potatoes, Spinach, Banana, Chicken, Eggs, Lentils, Brewers YeastVitamin DSalmon, Tuna, Egg Yolk, Cheese, Butter Meat, Mushroom, Cod Liver Oil Vitamin ESunflower seeds, Peanuts, Avocado, Olive Oil, Eggs, Shrimps, Sweet PotatoesMagnesiumWhole Grains, Spinach, Nuts, Wallnut, Peanuts, Almonds, Whole Wheat Breads ZincGreen Peas, White Beans, Corn, Oatmeal, Eggs Oysters, Chicken, Whole Wheat BreadsCopperLentils, Chickpea, Kidney Beans, Hazel Nuts, Walnuts, Apricot, Almonds, Chicken Oysters Selenium Soybean, Whole Wheat Bread, White Beans, Nuts, Cereals, Milk Products, Salmon, Sardines, Cod, TunaArginine Peanut, Oatmeal, Soybean, Eggs, Shrimps, Lamb Chicken, Wheat Germs GlutamineCheddar Cheese, Milk, Egg, Cabbage, Spinach, Papaya, Brussels, Sprouts, Fish, Chicken, BreastLysineLentils, Wheat Germ, Soybean, Parmesans Cheese, Tuna, Shrimps, Chicken Breast to maintain healthy life and counteract threatening sources such as viral, bacterial, fungal and a biotic components a person must take in to the account the following points-

1. **Balanced diet**

- · Protein such foods
- · Probiotics should be included
- Diet should be light, 3-4 tunics a day with variety and pleasant as per taste of an individual.
- · Special supplements as prescribed by Ayush like Turmeric, Milk, Kadha, Kesari Jeevan
- · Through non-conventional foods articles (Ginger, Tulsi, Cinnamon, Cardamom, Honey, Basil, Garlic
- Water is an essential nutrient as it regulates its temperature and maintains other bodily functions.

Sanitation practices are essential because its keeps an individual healthy and keep away from endemic, epidemic, pandemic and spreadable, non-spreadable diseases.

Sanitation practices includes treatment and disposal of sewage and drinking water, civic facilities such as public toilets, its general hygiene upkeeps, supply of drinking water, availability of proper dustbins for disposable waste products etc.

One of the most dangerous elements which are being added every moment by human activity is plastic. Most of them are not recycled and has a irreplaceable effect on terrestrial, aquatic and aerial environment.

Conclusion

Health, hygiene and sanitation protect us against life damaging components like microbial activity including virus, bacteria, Protozoan's and chemicals. It is therefore suggested to keep ourselves follow all the activities to keep away biotic and a biotic agents. It will also maintain the ecological equilibrium of nature.

- 1. B. Sri Lakshmi (2018) Food Science, New age international publication, New Delhi, PP: 1-3
- 2. B. Sri Lakshmi (2018) Dietetics, New age international publication, New Delhi, PP: 2-6
- 3. Grimble, RF, (2008) Basic in clinical nutrition: Immunonutrition-Nutrient which influences immunity: Effect and mechanism of action, *The European journal of clinical nutrition and metabolism* **4**, 10-13

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IMPORTANCE OF FRUITS AND VEGETABLES IN HUMAN NUTRITION

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Abstract

Fruits and vegetables are considered protective foods as their consumption prevents a number of diseases, such as cancer, diabetes, neurodegenerative diseases, and heart and brain vascular diseases. Protective properties of these fruits and vegetables result from the presence of low-molecular antioxidants that protect the cells and their structures against oxidative damage. Besides this fruits are rich source of vitamins, minerals and dietary fibre are low in calories. Functional foods, containingfruit and vegetable juices or extracts, are an important part of the healthy lifestyle, which includes a balanced diet and physical activity. Nutrients in fruits and vegetables, such as dietary fibre, vitamins, minerals, and phytochemicals, including polyphenols, all provide support for the biological plausibility that fruits and vegetables play a role in health. Nutritional composition of fruits and vegetables has been discussed in the present chapter

Nutrients in the study of nutrients in food, how the body uses them, and the relationship between diet, health, and disease. Nutrients are Substances obtained from food that provide nourishment. Nutrients required by human body are divided into macronutrients and micronutrients. Macronutrients are nutrients that people need in relatively large quantities. Proteins, carbohydrates, fats are macronutrients. Micronutrients are essential in small amounts. They include vitamins and minerals. Right balance of nutrients in human diet is essential, unbalanced diet poses risk of developing certain health conditions. Macronutrients provide energy while micronutrients are protective in function. Horticultural crops are among the main components of a healthy diet. The constituents obtained by the human body from fruits and vegetables include water, carbohydrates, fats, proteins, fibre, minerals, organic acids, pigments, vitamins and antioxidants, among others. Fruits and vegetables, especially, are a good source of fibre, selected minerals, vitamins and antioxidants. Most fruits and vegetables are available almost year-round in a wide variety and they not only taste good, but they also have favourable attributes of texture, color, flavor and ease of use. They are relatively low in calories and fat (avocado and olives being the exceptions), they have no cholesterol, they are rich in carbohydrates and fibre, they contain vitamin C and carotene, and some are a good source of vitamin B 6. Fruits and vegetables are relatively low in sodium and high in potassium. Ascorbic acid in fruits and vegetables enhances the bioavailability of iron in the diet. Because of all these characteristics, fruits and vegetables have a unique role in a healthy diet. Indian council of medical research has recommended consumption of 400 gms of vegetables and 320 gms of fruits per capita per day.

Nutritional composition of fruits and vegetables:

1. Water:

Water is most abundant single component of fruits and vegetables, which may account for up to 90% of the total mass. Moisture plays an important role in fruits and vegetables because many of the nutrients exist in soluble state in them. Most of the fruits and vegetables contain 70-80% moisture while some vegetables like leafy vegetables and melons contain almost 92-95% moisture. The tubers crops like cassava, yam and corms contain less moisture (around 50%) and are more starchy. Water is substrate of biological reactions or acts as the matrix or vehicle in which these reactions take place, regulates the temperature and pH in the human body and maintains cell and tissue integrity.

2. Carbohydrates:

After water, carbohydrates are the main components of fruits and vegetables and represent more than 90 % of their dry matter. Carbohydrates occur in fruits in the form of sugars and fibres. Sugars are classified into three groups *viz* Monosaccharides (glucose, fructose, arabinose, xylose), Oligosaccharides (sucrose, maltose, raffinose, stachyose) and Polysaccharides (starch, cellulose, hemicellulose). RDA for carbohydrates is 130 g/ day, except in cases of pregnancy (175 g/day) and lactation (210 g/day). In fruits and vegetables carbohydrates contribute mainly for its calorific value. These are responsible for sweetness. Other functions include storage of energy reserves and the make-up of much of the structural framework of cells. Simple carbohydrates, which are also the immediate products of photosynthesis, are important components of sensorial quality attributes. Many tropical and sub-tropical fruits contain highest level of sugars. Glucose and fructose are the major sugars in all fruits and often present in similar level, while sucrose is only present in about 2/3rd of the produce. It helps in imparting colour, flavour, appearance and texture to the fruits. Flavour is fundamentally the balance between sugar and acids ratios. Sugar is the primary substrate for respiration and energy. The glycaemic index (GI) of fruits and vegetables varies from 22 (cherries) -97(parsnip), Potato and sweet potato - 55 – 60, Bread-70

3. Organic acids

Organic acids provide imparts taste and flavour. There are two types of acids, namely aliphatic (straight chain) and aromatic acids. The most abundant acids in fruits and vegetables are citric and malic (both aliphatic) acids. However, large amounts of tartaric acid occur in grapes. Malic acid is the major component in oranges and apples. The acid content of fruits and vegetables generally decreases during maturation. Aromatic organic acids occur in several fruits and vegetables, but in very low concentrations. Benzoic acid occurs in cranberries, quinic acid in bananas and chlorogenic acid in potatoes. Organic acids plays important role in Photosynthesis and respiration, Synthesis of phenolic compounds, lipids and volatiles aroma.

4. Proteins

Fruits and vegetables are not an important source of proteins. Though some vegetables like brassica group contains 3-5% of proteins and legumes (5g), majority of fruits and vegetables contain not more than 1-2%. These proteins are mainly present as enzymes. Fruits are low in proteins, but tree nuts are a good source of high-quality proteins. The protein content of fresh fruits or vegetables is calculated by multiplying the total nitrogen content by a factor of 6.25. This calculation uses the fact that protein is comprised of about 16% nitrogen, and the assumption that all nitrogen present is protein.

5. Lipids and fatty acids

Lipids are not more than 1% in majority of fruits and vegetables except some like avocado (20%) and olive (15%). In most of them it is present as protective cuticle layer on surface. However, nuts contain considerable amount of fats. Generally low fat levels seen in fruits and vegetables make it healthier foods to combat heart related diseases and disorders like hyperlipidaemia. Many of the physical and chemical properties of lipids are due to the fatty acids present in their structure. Fatty acids are aliphatic monocarboxylic acids that may be saturated or unsaturated to varying degrees. Saturated fatty acids do not contain any double bonds along the chain. Monounsaturated fatty acids have a single double bond.

6. Dietary fibre

Dietary fibre consists of non-digestible carbohydrates and lignin that are intrinsic and intact in plants . Dietary fibre includes very diverse macromolecules exhibiting a large variety of physico-chemical properties. The main components included as fibre are cellulose, hemicelluloses, pectins, lignin, resistant starch and non-digestible oligosaccharides. Cellulose is a cell wall polymer of β -1,4-linked glucose . In fruits and vegetables, the cell wall constitutes 1% to 2% of the fresh weight, and cellulose could be as much as 33% of that amount. In general, with the exception of avocado in which the whole cell wall seems to be degrade. Cell wall polymers soluble in alkalis are classified as hemicelluloses or cross-linking . Within the primary cell wall, hemicellulose levels are usually around 30% . The most common hemicellulose polymer in dicotyledonous species is known as xyloglucan, composed as cellulose of a backbone of β -1,4-linked glucose, but with lateral chains of the pentose xylose (α -1,6 linked). Xylans are hemicellulosic compounds more abundant in monocotyledonous species, having a backbone of β -1,4-linked xylose which could be decorated with side chains of arabinose and/or glucuronic acid. Other hemicellulosic compounds usually less abundant include glucomannans, galactomannans and galactoglucomannans. Fruit tissues are particularly rich in pectins, which can account for up to 40% of the total cell wall polysaccharides. Pectins are also a diverse group of polymers rich in galacturonic acid. The most abundant pectic polysaccharide in the cell wall is homogalacturonan, a homopolymer of α -1, 4-linked galacturonic acid residues, with variable degrees of methyl esterification at C6. The degree of polymerization and the proportion of methyl esters affect the solubility of pectins. Pectins are deposited in

the cell walls, with a high degree of esterification, and methyl ester usually decreases during ripening. Another modification commonly observed in several fruits during ripening is a reduction in pectin polymer size. The extent of pectin depolymerization is variable, ranging from fruits such as avocado showing a dramatic downshift in polyuronide size to products in which these changes are negligible, such as pepper or some berries. Rhamnogalacturonan I (RG I) and rhamnogalacturonan II (RG II) are pectic polysaccharides which are also present in the plant cell wall. Pectins, which are used in the commercial manufacture of jams and jellies, are extracted from certain fruits and vegetables such as citrus, apples and beets. Lignin is one of the most abundant biopolymers in nature. It is an aromatic heteropolymer formed by the association of three hydroxycinnamyl alcohol derivatives (p-coumaryl, coniferyl and sinapyl alcohols). Lignin is a highly resistant polymer present in secondary cell walls, and is associated with fibres and xylem vessels. In the case of fruits and vegetables, lignin content is relatively low. Non-digestible oligosaccharides (NDOs) Oligosaccharides are low molecular weight carbohydrates intermediate in nature between simple sugars and polysaccharides. Dietary fibre present in fruits and vegetables results in modulation of function of the intestinal tract. Meals rich in fibre promote satiety earlier, and are usually relatively low in calories compared to meals rich in other food types (Marlett et al., 2002). Recommendations for adult dietary fibre intake generally fall in the range of 20 to 35 grams per day. Fruits and vegetables are considered very good sources of fibre . Fibre content of fruits and vegetables is usually in the range of 1% to 3%. Nuts and dried fruits have higher levels of fibre than fruits and vegetables. The main modifications during storage of most fruits and vegetables occur because of changes in the solubility and molecular size of the cell wall constituents due to the action of several proteins.

7. Vitamins

Vitamins are organic molecules required in trace amounts for normal development, which cannot be synthesized in sufficient quantity by the organism and must be obtained from the diet. The term "vitamin" derives from the words "vital amine" because the first vitamin discovered (thiamine) contained an amino group. Generally fruits and vegetables are rich in vitamins but their quantity is varied among them. Fat-soluble vitamins A, D, E and K and water-soluble vitamins C and B complex are found in fruits and vegetables. These are needed for growth, normal function of the body. The vitamins present in fruits and vegetables make an important contribution to human nutrition, as they have specific functions in normal body performance.

Vitamin A

Carotenoids are liposoluble pigments responsible for the yellow, orange and red color of several fruits and vegetables. Vitamin A plays an important role in vision, cell division and differentiation, bone development and reproduction. Deficiency causes night blindness and keratomalacia . The average daily requirement for vitamin A for an adult is estimated at 5000 international units (1 IU $_{\rm 0.3}~\mu$ g retinol or 0.6 μ g β -carotene). Among this group there are, basically, two different classes: carotenes containing C and H (e.g. α -carotene, β -carotene, lycopene, etc.), and oxygenated derivatives known as xantophylls, such as lutein, violaxanthin and zeaxanthin. Carotenoids in plants have functions related to radiation interception, mainly in the blue–green region of the spectrum, which may be transferred to the photosynthetic centres. Moreover, these pigments protect the photosynthetic structures from excessive energy. They are usually present in low concentrations and their levels are highly variable among species. Vegetables that can supply useful amounts of carotene include carrots, pumpkins and squashes. Compared to vegetables, fruits are generally not as good a source of carotenoids, although there are a few notable exceptions such as apricot, mango, citrus , papaya and watermelon . Tomatoes and peppers also contain high levels of carotenoids.

Vitamin B complex

Thiamine is required in the human body for the metabolism of carbohydrates. A daily intake of 1–2 mg is generally considered as necessary for a normal adult. Legumes are especially rich in thiamine. Compared with ascorbic acid, thiamine is relatively stable at cooking temperatures, especially in a slightly acidic solution. However, losses of 25% to 40% may occur during cooking. The average human requirement for riboflavin is estimated to be 1–2 mg per day. Green vegetables such as beans, beets, peppers and spinach are particularly rich in riboflavin. Starchy vegetables and fruits are relatively poor sources of riboflavin. Niacin, also known as nicotinic acid, is a precursor to NADH, NAD, NAD _ and NADP, which play essential roles in living organisms. A daily intake of 10 mg to 15 mg niacin is recommended. There is evidence that niacin can be synthesized in the body from tryptophan. Almonds are a rich source, but no fruits or vegetables can be singled out as being rich in niacin except perhaps, cape gooseberry and avocado. Niacin is relatively stable. Vitamin B 6 (pyridoxal phosphate) is a cofactor in many transamination, decarboxylation and deamination. Common symptoms of vitamin B 6 defi ciency include dermatitis around the eyes, elbows and mouth, along with soreness of the mouth and a red tongue. It can also lead to dizziness, vomiting, weight loss and severe nervous disturbances. Vitamin B 6 is present in appreciable amounts in beans, cabbage, cauliflower, spinach, sweet potatoes, grapes, prunes, avocados and bananas. It is

fairly heat stable. Pantothenic acid can be obtained from fresh, canned or frozen fruits and vegetables containing this vitamin if they are included in the diet. Pantothenic acid occurs widely in peas, beans, nuts, broccoli, mushrooms, potatoes and sweet potatoes. Symptoms of pantothenic acid deficiency in the diet include fatigue, headaches, sleep disturbance, tingling of hands and feet and lack of antibody production. Biotin is stable during cooking, processing and storage of fresh, canned and frozen fruits and vegetables. Deficiency leads to depression, sleeplessness and muscle pains. It is synthesized in the intestinal tract. Folic acid is essential for reproduction and normal growth. The vitamin is present in fruits, spinach, cabbage and other green vegetables. Lack of folic acid in the diet can cause a red tongue, diarrhea and anemia. Choline is heat-stable and occurs in dried legumes and vegetables. Choline deficiency in humans has never been reported. Vitamin B 12 does not occur in fruits and vegetables. Because vitamins of the B group are water-soluble, leaching losses occur during cooking.

Vitamin C

Ascorbic acid (AsA) and its first oxidation product dehydroascorbic acid (which can be reduced in the human body) might be considered as vitamin C. The recommended dietary allowance of vitamin C for men is 75 mg daily, while the recommended dietary allowance for young women is higher, at 90 mg daily. Ascorbic acid is required for collagen synthesis, bone and teeth calcification, Helps in absorption of dietary iron, Builds resistance to infection, aids in the prevention and treatment of the common cold and gives strength to blood vessels. Deficiency results into Scurvy i.e. soft and bleeding gums. Fruits, vegetables and juices are the main dietary sources of vitamin C. Vitamin C is present in fresh fruits and vegetables, as well as in fruit juices. Fruits, particularly tropical species, and leafy vegetables are rich in ascorbic acid. Rosehip, jujube and guava have very high levels of ascorbic acid. Other good sources of As A include persimmon, strawberry, kiwifruit, peppers, and citrus fruit, and spinach, broccoli and cabbage among vegetables. A main environmental factor determining the level of ascorbic acid is radiation interception. In general, the greater the amount of sunlight received during growth, the higher the ascorbic acid content. The retention of AsA is also markedly affected by storage and processing. Potatoes lose up to 75% to 80% of the original levels over nine months of storage. In most cases, other fruit and vegetable AsA levels decline during storage, because the losses are accelerated by storage at high temperatures. Bruising and mechanical damage greatly increase the rate of ascorbic acid loss. Ascorbic acid is highly susceptible to oxidation, either directly or through the enzyme ascorbate oxidase catalyzing the oxidation of AsA to dehydroascorbic acid, with the concomitant reduction of molecular oxygen to water. Ascorbic acid can even be oxidized during eating, while food is being chewed. However, it is important to consider that the first breakdown product of AsA, dehydroascorbic acid, still has vitamin C activity and all activity is lost if oxidation proceeds beyond this stage. When vegetables are cooked before eating, high losses of vitamin C can occur. For instance, starchy vegetables may lose between 40% and 80% of their vitamin C during cooking, because of leaching and oxidation. Loss of vitamin C can be reduced by steaming or by placing the vegetables directly into boiling water. Freezing reduces vitamin C slightly, but at the end of long-term frozen storage (12 months), a significant decrease (33% to 55%) in vitamin C can occur.

Vitamin E

Vitamin E includes tocopherols and tocotrienols. Possesses antioxidant property, reduces risk of degenerative diseases and prevents oxidation of lipids and maintains cell integrity. Deficiency causes enhanced fragility of red blood cells and increased urinary excretion of creatine indicating muscle damage. In general, vitamin E levels are more abundant in oily seeds, olives, nuts, peanuts, avocados and almonds. Even though the levels of tocopherol in broccoli and leafy vegetables are lower than in fat-rich products, they are good sources compared to other fruits and vegetables. Vitamin E is highly susceptible to oxidation during storage and processing.

Vitamins D and K

Vitamin D is a group of fat-soluble compounds. The main forms of vitamin D are ergocalciferol and cholecalciferol. It is essential for healthy bones and helps in calcium absorption in body. Deficiency results into Rickets (in children) and osteomalacia (in adults)- degeneration of bones . It occurs only in trace amounts in fruits and vegetables. Vitamin K is essential for blood coagulation, but dietary deficiency is uncommon. The recommended daily intake is $120~\mu$ g. It occurs abundantly in lettuce, spinach, cauliflower and cabbage.

8. Antioxidants

Imbalance in the production of reactive oxygen species (ROS) leading to negative cellular alterations is known as oxidative damage, which is caused by several molecules. The process of oxidation in the human body damages cell membranes and other structures, including cellular proteins, lipids and DNA. When oxygen is metabolised, it creates unstable molecules called 'free radicals', which steal electrons from other molecules, causing damage to DNA and other cells. The body can cope with some free

Table: 1 - Major fruits and vegetables as source of vitamins		
Vitamin A	mango, papaya, leafy vegetables, radish tops, carrots etc.	
Thiamine (B1)	bael, pomegranate, jamum, fresh peas & beans, cabbage etc.	
Riboflavin (B2)	Banana, litchi, papaya, radish top, pineapple, cowpeaetc.	
Niacin(B3)	Banana, strawberry, peach, cherry, green vegetables etc.	
Vitamin C	Anola, Guava, Citrus Fruits, Cashew Apple, Leafy vegetables, Green Chilli, Drumstick etc.	
Vitamin D	Cabbage, carrot	
Pyridoxine (B6)	Vegetables	
Folic acid (B9)	Fresh Green Leafy Vegetables, Okra, Cluster beans etc.	

radicals and needs them to function effectively. However, the damage caused by an overload of free radicals over time may become irreversible and lead to certain diseases, including heart disease, liver disease and some cancers (such as oral, oesophageal, stomach and bowel cancers). Antioxidants the damage caused by free radicals by neutralising them. They are most abundant in fruits and vegetables. Main antioxidants in fruits are Ascorbic acid, Carotenoids (Carotenes and xanthophylls), Vitamin E (Tocopherols and Tocotrienols) and Phenolics . Phenolics include Phenolic acids (benzoic acid and cinnamic acids) and Flavonoids (Flavonols, Flavones, Isoflavones, Flavanols, Flavanones, Anthocyanidins and proanthocyanidins). Besides this some sulphur antioxidants are also present in fruits and vegetables. Antioxidants in fruits are affected by various genetic and environmental factors. Genetic factors include species and variety. Environmental factors include preharvest factors (Radiation and stress during development), harvest factors (maturity and handling) and postharvest factors (storage, postharvest treatments and processing).

Some Bioactive Compounds or Phytochemicals are substances present in fruits and vegetables at low levels that may have a role in health maintenance in humans and have antioxidant properties.

9. Minerals

Minerals are normally classified as macro and micronutrients, based on the relative concentration of each nutrient when those concentrations are adequate for normal tissue function. Macronutrients include potassium (K), calcium (Ca), magnesium (Mg), nitrogen (N), and phosphorus (P), and their concentrations in plant tissues range from 1000 to 15 000 μ g per gram of dry weight. In contrast, the concentrations of micronutrients usually found in plant tissues are 100- to 10 000-fold lower than those of macronutrients. Mineral micronutrients considered essential in human nutrition include manganese (Mn), copper (Cu), iron (Fe), zinc (Zn), cobalt (Co), sodium (Na), chlorine (Cl), iodine (I), fl uorine (F), sulfur (S), and selenium (Se). Macronutrients can also be classifi ed into those that maintain their identity as ions within plant tissues (e.g. K, Ca 2 and Mg 2), and those that are assimilated into organic compounds (e.g. N and P). Fruits and vegetables are not usually recognized as primary sources of minerals. In general, vegetables are a richer source of minerals than fruits, but both vegetables and fruits are considered "nutrient-dense foods" in that they provide substantial amounts of micronutrients, such as minerals and vitamins, but relatively few calories (Ariel et al., 2009). Minerals have both direct and indirect effects on human health. The direct effects of minerals focus on the consequences of their consumption on human nutrition, while the indirect effects refer to their incidence in fruit and vegetable quality and subsequent consumer acceptance. From a direct nutrition standpoint, potassium has the biggest presence in both fruits and vegetables, but nitrogen and calcium show major impacts on horticultural crop quality. Potassium is the most abundant individual mineral element in fruits and vegetables. It plays a role in a myriad of cellular and whole plant functions: it serves as an osmoticum for cellular growth and stomatal function, balancing the charges of anions, activating almost 60 plant enzymes and participating in numerous metabolic processes, including protein synthesis, oxidative metabolism and photosynthesis. In fruits and vegetables, potassium occurs mainly in combination with various organic acids. Examples of potassium-rich fruits and vegetables include bananas and plantains, leafy green vegetables, many dried fruits, oranges and orange juice, cantaloupes and honeydew melons, tomatoes and root vegetables. Horticultural crops are considered a secondary source of calcium. Dark green leafy cabbage family vegetables and turnip greens are good calcium sources and most green leafy vegetables are potential calcium sources because of their absorbable calcium content (Jodral-Segado et al., 2003; Titchenal and Dobbs, 2007). Magnesium is important in protein synthesis, release of energy from muscle storage and body

temperature regulation. It is critical for proper heart function and plays a role in bone formation, as previously described. Magnesium activates over 100 enzymes Generally, magnesium levels are significantly higher in vegetables than in fruits, but nuts are good sources of this nutrient. Dry fruits and legumes are the food groups that rank higher in magnesium content (Jodral-Segado et al., 2003). Inorganic phosphate is essential for skeletal mineralization and for multiple cellular functions, including glycolysis, gluconeogenesis, DNA synthesis, RNA synthesis, cellular protein phosphorylation, phospholipid synthesis and intracellular regulatory roles (DiMeglio et al., 2000). Among tree fruits, nuts are natural sources of phosphorus. Nitrogen is an essential component of nucleic acids, cofactors and other metabolites. Green vegetables have high nitrogen content. Sulfur is an essential nutrient required for growth, primarily used to synthesize cysteine and methionine, white cabbage, broccoli, cauliflowers are rich source of sulphur. Manganese is a key component of enzyme systems, including oxygen-handling enzymes. Among horticultural crops, spinach is a good source of manganese. Copper, a redox active metal, plays an important role in the oxidative defense system. Vegetables are rich source of copper. Iron is required in numerous essential proteins, such as the heme-containing proteins, electron transport chain and microsomal electron transport proteins, and iron-sulfur proteins and enzymes such as ribonucleotide reductase, prolyl hydroxylase phenylalanine hydroxylase, tyrosine hydroxylase and aconitase (Arredondo and Nuñez, 2005). Almonds, pistachio nuts, walnuts, pecans, etc., are very good sources of iron. Different vegetables (e.g. parsley, broccoli, kale, turnip greens and collards) and legumes (e.g. green peas and beans) are also considered good sources of iron. Zinc is a pervasive microelement that plays a catalytic or a structural role in more than 200 enzymes. Fruits are poor in zinc, but pecans and walnuts are good sources of this essential mineral. Parsley is also a good source of zinc. Sodium is a systemic ion. It is important in electrolyte balance and essential in coregulating ATP with potassium. In addition, it has an important role in the regulation of blood pressure. Olives and spinach are horticultural sources of sodium. In general, fruits are poor in sodium, and are recommended for low-sodium dietary patterns. Mineral content of fruits and vegetables is effected by species and the cultivar, preharvest and Postharvest practices.

References

- 1. Ariel R. Vicente, George A. Manganaris, Gabriel O. Sozzi and Carlos H. Crisosto 2009. Nutritional Quality of Fruits and Vegetables. *From Postharvest Handling: A Systems Approach*, Second Edition ISBN: 978-0-12-374112-7 Edited by Wojciech J. Florkowski, Robert L. Shewfelt, Bernhard Brueckner and Stanley E. Prussia pp. 58-93
- 2. Arredondo, M., Núñez, M.T. (2005). Iron and copper metabolism. Mol. Aspects Med., 26, 313–327.
- 3. Dimeglio , L.A. , White , K.E. , Econs , M.J. (2000) . Disorders of phosphate metabolism . Endocrinology and Metabolism Clinics of North America , 29 , 591 609
- 4. Jodral-Segado , A.M. , Navarro-Alarcón , M. , López-G de la Serrana , H. , López Martínez , M.C. (2003) . Magnesium and calcium contents in foods from SE Spain: Infl uencing factors and estimation of daily dietary intakes . Sci. Total Environ. , 312,47–58.
- 5. Marlett JA, McBurney MI, Slavin JL (2002). Position of the American dietetic association: health implications of dietary fibre. *J Am Diet Assoc* **102**, 993–1000.
- 6. Titchenal, C.A., Dobbs, J. (2007). A system to assess the quality of food sources of calcium. J. Food Compos. Anal., 20, 717–724.

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SYNTHESIS AND CHARACTERIZATION OF CROSS-LINKED GRAFT COPOLYMER BASED ON CARRAGEENAN AND ACRYLAMIDE USING REDOX SYSTEM

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Abstract

In this research article, we have synthesizedcrosslinked graft copolymer via free radical graft copolymerization of acrylamide (AAm) onto carrageenan polymeric backbone and crosslinking occurred by using methylenebis acylamide (MBA) crosslinker. The polymerization reaction was carried out by potassium bromate and thiourea redox system in an aqueous and in nitrogen atmosphere. Infrared spectroscopy were carried out to confirm the formation of cross-linked graft copolymer between carrageenan, acrylamide and methylene bisacrylamide. Swelling ratio of the synthesized sample has also been determined.

Introduction

The growth of polymer science has led to the development of new materials in direct competition with natural materials, many of which have been in use since earliest times. This has caused researchers to look more critically at both natural and synthetic macromolecules in order to learn more about their underlying structures and their relation to the properties exhibited by the macromolecules. In this regard, chemical modifications have been devised to impart certain desirable properties of both natural and synthetic macromolecules, and their applications have become an integral part of such chemical modifications such as replacement transparency, good adhesion, easy handling, oxygen permeability, control of drug dosage. Various chemical modifications (e.g., change of functionality, oxidative degradation, inter- and intra-molecular gelatin, graft copolymerization), have been practiced to add improved properties to the base polymers. However, among all these methods, modification of polymers via graft copolymerization has been the subject of much interest and has made paramount contribution toward improved industrial and biomedical applications. Well-defined graft copolymers are most frequently prepared by either a "grafting through" or a "grafting from" controlled polymerization process. However the development of "click" chemistry has led to a third approach based on site specific "grafting to" chemistry [1-10]. The biomaterial is generally used to recognize materials for biomedical application. Hydrogels have been largely used in medicine, pharmacy, and life science. Their morphology and physic-chemical properties make them suitable for several applications in particular as drug controlled release system and tissue engineering. Several polysaccharides have been utilized as materials for hydrogel production. Their chemical structure can be easily modified to introduce new biological properties. We have chosen carrageenan as biomaterial and modified its degradable property with the help of vinyl monomer and make it useful in the form of hydrogel using MBA crosslinker.

> Characteristics features of Carrageenan

Carrageenan is natural polymer obtained from certain species of the red seaweed, class *Rhodophycea*. *The main* sources for carrageenan are the *Chondrus Crispus*, *Eucheuma Cottonii* and *Eucheuma Spinosum* species. Commercial carrageenan are available as stable sodium, potassium, and calcium salts or, most generally, as a mixture of these. Carrageenan can be produced via a variety of process/techniques; alcohol extraction, potassium chloride gel press or extracted with various alkali.

Carrageenan has unique properties, which cannot be replaced by other food grade, safe and non-toxic materials. Carrageenan are far more widely used than agar as emulsifiers/stabilizers in numerous foods, especially milk based products. There are mainly three

types of Carrageenan Kappa, Iota and lambda carrageenan and they are differ in gelling and milk reactivity.

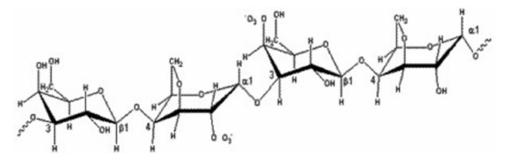


Fig. - 1: Structure of Carrageenan

It is generally considered a high-molecular-weight linear polysaccharide. It comprises repeating galactose units and 3, 6-anhydrogalactose (3,6-AG), sulfated and non-sulfated, joined by alternating α (1-)-and β (1-4)-glycosidic linkages. Carrageenan consists of alternating 3-linked- β -D-galactopyranose and 4-linked- α -D-galactopyranose units.

Characteristics features of N-N methylene bisacrylamide (MBAAm)

 N_7 -Methylenebisacrylamide (MBA) is a cross-linking agent used during the formation of polymers such as polyacrylamide. Its molecular formula is $C_7H_{10}N_2O_2$. Bisacrylamide is used in biochemistry as it is one of the compounds of the polyacrylamide gel . Bisacrylamide polymerizes with acrylamide and is capable of creating cross-links between polyacrylamide chains, thus creating a network of polyacrylamide rather than unconnected linear chains of polyacrylamide.

$$H_2C$$
 N
 N
 N
 CH_2

Fig. - 2: Structure of MBA

MBA is an ideal raw material for a wide varieties of applications. It is especially suited to the preparation of cross linked polymers used in:

Adhesives ,Cosmetic thickener, Biological, pharmaceutical and personal home care products, Catalysts, Chromatographic
materials, Coagulating and flocculating agents, Coatings, Construction materials, Ion exchange resins(anionic, cationic and
ampholytic), Paper application, Polyacrylamide gels for electrophoresis, Soil grouting systems, Superabsorbent
polyacrylate resins, Textile thickeners

Reactivity of MBAAm

The normal vinyl addition polymerization is possible with monomers, but it should be noted that cross linking properties are acquired in the first place by the existence of double bonds in the molecules, rather than by a secondary reaction. MBA is widely used for the ease of controlling its cross linking properties in different polymers formulations. Copolymerization of acrylamide with MBA produces a polymer gel with a nearly uniform distribution of pendant methylenebisacryamide groups. The pendant double bonds have the same reactivity as the first double bond of the monomer except for the restriction of mobility.

> Characteristics feature of Acrylamide

Acrylamide is also called **2-propenamide, ethylenecarboxamide,** or **acrylic amide**, a white, odourless, crystalline substance belonging to the family of organic compounds; its molecular formula is C_3H_5NO . Acrylamide is produced as a result of industrial processes and is generated in certain foods as a result of cooking at high temperatures. Because acrylamide is neurotoxic and is listed as a probablecarcinogen (cancer-causing agent) in humans, its presence in many processed foods has been a source of public health concern.

Application of acrylamide

Manufactured acrylamide is incorporated into grout and soil-stabilizer products that are used to prevent or plug leaks in dams, tunnels, and other structures. It also forms the basis for the generation of polyacrylamide, which has a variety of applications, including wastewater treatment and gelelectrophoresis for laboratory research.

In food, acrylamide forms during frying, baking, or roasting. These forms of heating initiate the Millard reaction, in which reducing sugars (simple monosaccharide capable of carrying out reduction reactions) present in carbohydrate-rich foods react with amino acids to produce acrylamide. Asparagineappears to be the primary amino acid involved in the generation of acrylamide via the Maillard reaction. Acrylamide has been detected in raw foods such as olives. Research has suggested that various treatment processes, as well as the release of acrylamide from the breakdown of polyacrylamide-containing herbicides, may be reasons for the contamination of raw plant-based foods.

Characteristics feature of Polyacrylamide

Polyacrylamide (IUPAC poly (2-propenamide) or poly(1-carbamoylethylene)) is a polymer (-CH2CHCONH2-) formed from acrylamide units. It can be synthesized as a simple linear-chain structure or cross-linked, typically using N,N'-methylenebisacrylamide. Polyacrylamide is not toxic. However, unpolymerized acrylamide, which is a neurotoxin, can be present in very small amounts in the polymerized acrylamide, therefore it is recommended to handle it with caution. In the cross-linked form, the possibility of the monomer being present is reduced even further. It is highly water-absorbent, forming a soft gel when hydrated, used in such applications as polyacrylamide gel electrophoresis and in manufacturing soft contact lenses. In the straight-chain form, it is also used as a thickener and suspending agent. More recently, it has been used as a subdermal filler for aesthetic facial surgery.

Polyacrylamide is often used in molecular biology applications as a medium for electrophoresis of proteins and nucleic acids in a technique known as PAGE.

Stability

In dilute aqueous solution, such as is commonly used for Enhanced Oil Recovery applications, polyacrylamide polymers are susceptible to chemical, thermal, and mechanical degradation. Chemical degradation occurs when the labile amine moiety hydrolyzes at elevated temperature or pH, resulting in the evolution of ammonia and a remaining carboxyl group. Thus, the degree of anionicity of the molecule increases. Thermal degradation of the vinyl backbone can occur through several possible radical mechanisms, including the autooxidation of small amounts of iron and reactions between oxygen and residual impurities from polymerization at elevated temperature. Mechanical degradation can also be an issue at the high shear rates experienced in the near-wellbore region. However, cross-linked variants of polyacrylamide have shown greater resistance to all of these methods of degradation, and have proved much more stable.

Chemicals Used

Carrageenan is used as polymeric backbone for graft copolymerization of acrylamide monomer using MBA as crosslinker. All these chemicals were purchased from Sigma Aldrich Chemicals, USA and used as such without further purification. The Potassium Bromate and Thiourea are used as redox pair, both are purchased from Himedia chemicals. The sulphuric acid was used for maintaining the reaction media acidic. Methanol was used for precipitation of graft copolymer and are purchased from Himedia chemicals.

Method: Procedure for graft copolymerization

For each experiment desired amount of carrageenan has been added to 50 ml of triple distilled water in a 100 ml narrow mouth conical flask. A calculated amount of acrylamide (ACM), thiourea and sulphuric acid solutions has been added to the carrageenan solution and mix well after that initiate the reaction with the addition of calculated amount of Potassium Bromate. The reaction mixture has been kept in thermostat at 45°C. The reaction turned slight turbid after completing the polymerization, which was first cooled and the product was precipitated by pouring the reaction mixture in methanol or Acetone. The precipitate has been filtered, washed twice – trice times with distilled water to dissolve unreacted chemicals after that dried and weighed. The above reaction has been repeated in the presence of small amount of crosslinker methylenebisacrylamide (MBA).

Determination of grafting ratio and rate of grafting

The graft copolymer has been characterized according to Fanta's definition by calculating the % grating ratio.

$$Grafting \ ratio \ (\%G) \ = \ \frac{Grafted \ polymer}{Weight \ of \ substrate} \times 100$$

The rate of grafting has been calculated according to following formula, which was studied by a number of authors.

Rate of grafting (Rg) =
$$\frac{1000 \times W}{V \times t \times M}$$

Where:

W is the weight of grafted polymer; V is the volume of the reaction mixture, t is the time of the reaction in second and M is the molecular weight of monomer

Mechanism

The bromate-thiourea system is an efficient initiator for the aqueous polymerization of vinyl monomers. Nayak and colleagues studied the graft copolymerization of methyl methacrylate onto silk fibre using a bromate-thiourea redox system and Kunj Behari groups have also used the same in different graft copolymerization reactions. In general, the graft yield increases with increasing bromate ion concentration upto certain range of bromate/thiourea and thereafter it decreases.

Potassium Bromate Initiation:

It is well known that the potassium bromate is good oxidizing agent, which readily gives free radical when coupled with various reducing agents (reductants). Neither potassium bromate nor thiourea when used alone initiates polymerization. On the other hand, thiourea coupled with bromate ion can initiate vinyl polymerization. The following mechanism is suggested for grafting of acrylamide onto carrageenan and cross-linked the acrylamide chains with methylene bisacrylamide.

Formation of Radical

It was noticed that in acidic medium thiourea forms protonated thiourea which reacts with bromate ion and generate isothiocarbamido radicals R^{\bullet}

$$S = C \xrightarrow{NH_2} \xrightarrow{H^+} HS - C \xrightarrow{NH_2} HS = C \xrightarrow{NH_2$$

Initiation

$$CgOH + R^{\bullet} \longrightarrow CgO^{\bullet} + RH$$

$$M_{1} + R^{\bullet} \longrightarrow RM_{1}^{\bullet}$$
 [Where $R = R_{1}S^{\bullet}$, $CgOH = Carrageenan and M = Monomer]$

The interaction of amidinosulphenyl radical with Carrageenan produces macromolecule radical CgO and with the monomer produces RM and both these secondary radical start the graft copolymerization of acrylamide onto carrageenan in the presence of crosslinker.

Propagation

Where, C is the crosslinker

The monomer molecule M_1 and croslinker C have been react with carrageenan macroradical and propagate the growing croslinked grafted chains

Termination

Termination of growing grafted chain and growing homopolymer chain have been occurred through coupling of different growing chains. There are following possibilities to couple the growing grafted chains and gives different size of grafted crosslinked chains.

$$(\operatorname{CgOM}_{1}\operatorname{C}\operatorname{M}_{1}^{\bullet})n + (\operatorname{CgOM}_{1}\operatorname{C}\operatorname{M}_{1}^{\bullet})n \longrightarrow \operatorname{Graft\ copolymer\ chain\ 1}$$

$$(\operatorname{CgOM}_{1}\operatorname{C}\operatorname{M}_{1}^{\bullet})m + (\operatorname{CgOM}_{1}\operatorname{C}\operatorname{M}_{1}^{\bullet})m \longrightarrow \operatorname{Graft\ copolymer\ chain\ 2}$$

$$(\operatorname{CgOM}_{1}\operatorname{C}\operatorname{M}_{1}^{\bullet})n + (\operatorname{CgOM}_{1}\operatorname{C}\operatorname{M}_{1}^{\bullet})m \longrightarrow \operatorname{Graft\ copolymer\ chain\ 3}$$

$$(\operatorname{CgOM}_{1}\operatorname{C}\operatorname{M}_{1}^{\bullet})n \text{ or } (\operatorname{CgOM}_{1}\operatorname{C}\operatorname{M}_{1}^{\bullet})m \longrightarrow \operatorname{Graft\ copolymer\ chain\ 1}$$

$$(\operatorname{RM}_{1}^{\bullet})n + (\operatorname{RM}_{1}^{\bullet})n \longrightarrow \operatorname{Homopolymer\ chain\ 2}$$

$$(\operatorname{RM}_{1}^{\bullet})n + (\operatorname{RM}_{1}^{\bullet})m \longrightarrow \operatorname{Homopolymer\ chain\ 2}$$

$$(\operatorname{RM}_{1}^{\bullet})n + (\operatorname{RM}_{1}^{\bullet})m \longrightarrow \operatorname{Homopolymer\ chain\ 3}$$

Characterization

Evidence of grafting: FTIR Analysis

The FTIR spectra of carrageenan and crosslinked graftcopolymer of Carrageenan with MBA have been recorded by using a JASCO FTIR -5300 model of spectrophotometer. The FTIR spectrum of carrageenan and crosslinked graft copolymer of Carrageenan with MBA have been represented by figure 3 and figure 4 respectively. The FTIR spectral analysis have been utilized as characterization technique to prove the grafting. On comparing the FTIR spectra of carrageenan and crosslinked graft copolymer of Carrageenan with MBA, we have found some additional peaks in the spectrum of crosslinked graft copolymer which are not present in the spectrum of Carrageenan. The following additional peaks/bands have been observed in spectrum of crosslinked graft copolymer.

- The peak at 1666 cm⁻¹ (Amide I band) is appeared which attributed to C=O stretching vibration of Amide group of AAm and MBAAm.
- A very small peaks is appeared at 1570 cm⁻¹ due to C-N-H bending vibration of polyacrylamide (Amide II)
- The peak occurred at 1449 cm⁻¹ is due C-N stretching vibration of amide.

The presence of these peaks/bands confirms the grafting of acrylamide on to carrageenan and crosslinked with MBAAM.Disappearance of peaks related to O-H bending vibration occurred at 1418 cm⁻¹ from the spectrum shown by fig. 4 indicates that grafting occurred at OH site of the carrageenan.

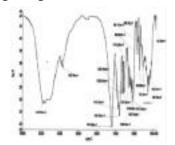


Fig 3:-FTIR spectrum of carrageenan

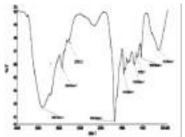


Fig 4:-FTIR spectrum of grafted carrageenan

Result and Discussion

I. Effect of temperature on grafting

The graft copolymerization of AAm on to carrageenan using bromate/thiourea redox system in aqueous medium has been carried out at different temperature, which ranges from 40° C to 60° C. The results have been summarized in table 1. The grafting ratio has been increased up to 55° C after that decreased. The increase in grafting ratio up to cited range of temperature might be attributed to the fact that with increasing the temperature rate of production of primary free radicals increase causing an increase in %G due to the fast diffusion of AAm to carrageenan. However beyond the optimum value further increase in temperature could results the decomposition of HBrO₃into Br₂, H₂O, O₂ since O₂ act as scavengerof free radicalsthereby decreasing the concentration of free radical hence decreases %G. The rate of grafting is also increased with increasing the temperature of reaction because rate of reaction is increased with increasing the temperature of the reaction.

Table 1: Effect of temperature on grafting

 $[BrO_3^-] = 1 \times 10^{-2} \text{ mol dm}^{-3} [Thiourea] = 2.5 \times 10^{-3} \text{ mol dm}^{-3}$

 $[ACM] = 2x \cdot 10^{-2} \text{ mol dm}^{-3} [H^{+}] = 5.0 \times 10^{-3} \text{ mol dm}^{-3}$

 $[CgOH] = 1.0 \times 10^{-2} \text{ g dm}^{-3} [MBAAm] = 0.3 \times 10^{-3} \text{ g dm}^{-3}$

Time = 150 min.

S. No.	Temperature (°C)	% G	Rg
1	40	80	4.3
2	45	128	32.7
3	50	140	35.8
4	55	180	46.0
5	60	170	38.0

II. Effect of crosslinker on grafting

The graft copolymerization of acrylamide has been tested for better yield by crosslinking the polyacrylamide chain with methylene bisacrylamide crosslinker and to get the crosslinked graftcopolymer. The amount of MBAAm has been increased from 1.0 x 10^4 g dm⁻³ to 5.0 x 10^4 g dm⁻³. It is observed that grafting ratio has been increased from 180 to 530 by using the MBAAm in the same reaction condition as in the above reaction. It has been seen that %G increased with increasing the amount of MBAAm from 1.0 x 10^4 g dm⁻³ to 3.5 x 10^4 g dm⁻³ after that decreased, it may be due to formation of MBAAm bridge between polyacrylamide chains and it is decreased due to saturation of all pendent polyacrylamide chain or may be crowdness of MBAAm that makes homopolymer of MBAAm.

Swelling Studies

The dry crosslinked graft copolymer were immersed in excess of water and allowed to swell for 24 hour to obtain an equilibrium-swelling at room temperature. The completely swollen crosslinked graft copolymer were removed from water and excess water on the sample has been soaked with the help of tissue paper and weighed accurately. The equilibrium-swelling ratio (S_q) have been calculated as per eqn. no. (1).

$$S_{eq} = \frac{\text{wt of swell polymer - wt of dry polymer}}{\text{wt of dry polymer}} \times 100 \qquad \dots (1)$$

The results of swelling behavior of crosslinked graft copolymer have been given in table 2. The equilibrium-swelling ratio (S_{eq}) values of these crosslinked graft copolymer in triple increases the crosslinker content increases due to increase of network and the equilibrium swelling capacity of the hydrogel decreased after certain amount of crosslinker. It is obvious that the higher crosslinker content makes the gel network denser, which restricts the water penetration into the hydrogel network and, therefore, lowers their swelling capacity.

Table 2: Effect of crosslinker on grafting

 $[BrO_3^-] = 1 \times 10^{-2} \text{ mol dm}^{-3}; [Thiourea] = 2.5 \times 10^{-3} \text{ mol dm}^{-3}$

 $[ACM] = 2 \times 10^{-2} \text{ mol dm}^{-3}; [H^{+}] = 5.0 \times 10^{-3} \text{ mol dm}^{-3};$

 $[CgOH] = 1.0 \times 10^{-2} \text{ g dm}^{-3};$ Temp. = 55°C;

Time = 150 min.

S. No.	[MBAAm] x 10 ³ g dm ⁻³	% G	Seq %
1	0.10	260	550
2	0.20	380	600
3	0.30	450	760
4	0.35	530	480
5	0.40	320	300
5	0.45	180	200
6	0.50	160	180

Conclusion

It is concluded that bromate/thiourea is a good redox system for synthesizing the acrylamide and carrageenan based on crosslinked graft copolymer than others because it gives better yield than bromate/glycolic acid and bromate/ascorbic acid. Further, it is concluded that grafting and rate of grafting is increased with increasing the temperature. The swelling capacity is increased with increasing the %G and decreased with increasing the amount of MBAAm.

References

- Kolb, H. C.; Finn, M. G.; Sharpless, K. B. *Angewandte Chemie, International Edition***2001**, *40*, 2004-2021.
- Hong, S. C.; Jia, S.; Teodorescu, M.; Kowalewski, T.; Matyjaszewski, K.; Gottfried, A. C.; Brookhart, M. *Journal of Polymer Science, Part A: Polymer Chemistry* **2002**, *40*, 2736-2749.
- 3 Kaneyoshi, H.; Inoue, Y.; Matyjaszewski, K. *Macromolecules* **2005**, *38*, 5425-5435.
- 4 Neugebauer, D.; Zhang, Y.; Pakula, T.; Sheiko, S. S.; Matyjaszewski, K. Macromolecules 2003, 36, 6746-6755.
- 5 Shinoda, H.; Matyjaszewski, K.; Okrasa, L.; Mierzwa, M.; Pakula, T. Macromolecules 2003, 36, 4772-4778.
- 6 Shinoda, H.; Matyjaszewski, K. Macromolecules 2001, 34, 6243-6248.
- 7 Matyjaszewski, K.; Beers, K. L.; Kern, A.; Gaynor, S. G. J. Polym. Sci., Part A: Polym. Chem. 1998, 36, 823-830.
- 8 Lutz, J.-F.; Jahed, N.; Matyjaszewski, K. Journal of Polymer Science, Part A: Polymer Chemistry 2004, 42, 1939-1952.
- 9 Neugebauer, D.; Zhang, Y.; Pakula, T. Journal of Polymer Science, Part A: Polymer Chemistry 2006, 44, 1347-1356.
- 10 Inoue, Y.; Matsugi, T.; Kashiwa, N.; Matyjaszewski, K. Macromolecules 2004, 37, 3651-3658.

Three Major dimensions of life: Environment, Agriculture and Health

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MECHANISM OF ACTION AND CHEMISTRY OF SANITIZERS AND DISINFECTANT

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Introduction

The world is facing a pandemic in the form of the COVID-19 outbreak. This pandemic came into light on 31st December 2019 when China informed the World Health Organization about spreading cases of disease like pneumonia griping Wuhan city in Hubei Province. Later on this disease started spreading to more provinces of China and to the rest of the world. The WHO has already declared it a pandemic. The virus has been named **SARS-CoV-2** and the disease is now called **COVID-19**. The SARS means – severe acute respiratory syndrome and COVID means Coronavirus disease. It was first reported in 2019 so it is named as COVID-19. It is genetically similar to the bat Coronavirus. It is **zoonotic** in origin i.e. the disease is caused by germs that spread between animals and people. This virus is very contagious and has rapidly spread throughout the world. Corona virus has a crown like appearance having positive stranded RNA. It is an enveloped virus with spikes like glycoproteins. It belongs to the Corona-Viridae family, the sub family is Ortho corona virus and order is Nidovirales. To win the battle against coronavirus, disinfectants and sanitizatiors are playing very important roles.

Alcoholic sanitizers

According to world Health Organization (WHO), hand sanitizers containing alcohol are capable to inactivate coronavirus disease and i.e. COVID-19. There is no antiviral drug or vaccine which can prevent the spread of severe acute respiratory syndrome coronavirus 2 (SARS –CoV-2), the virus that causes COVID-19. WHO recommends two alcohol based formulations to kill coronavirus.

Formulation 1

It contains 80% (vol/vol) ethanol, 1.45% (vol/vol) glycerin and 0.125% (vol/vol) hydrogen peroxide.

Formulation 2

The composition of this sanitizer is 75% isopropanol (vol/vol), 1.45% glycerin (vol/vol) and 0.125% hydrogen peroxide. **Prof. Stephanie Pfander** of Germany showed that both these formulations inactivate Corona virus in 30 seconds.

Mechanism of action of alcohol on coronavirus

Generally viruses contain genetic material. It may be DNA or RNA. Both DNA and RNA may be single stranded or double stranded. These DNA and RNA may be positively sensed or negatively sensed. If it is directly translatable into protein if it were RNA then this DNA is known as positive sense DNA. On the other hand, complementary strand of messenger RNA is negative sense RNA. Apart from the genetic material, viruses also contain a protein coat which is known as **capsid**. Viruses may contain a lipid envelop (non enveloped virus). The most important fact about viruses is that they can replicate only inside a host only. Outside a host, they are not living entities. The life cycle of a virus consists of following steps- Attachment to the host, penetration, biosynthesis, maturation and then lysis. Scientists are focusing on altering the structure and function of the viral envelope derived from the host lipid envelopes, the protein capsid, which surrounds the genetic material and genetic material itself. Scientists believe that alcohols have strong virucidal

activity by disrupting lipid envelopes. Ethanol is more effective than propanol. Concentrated ethanol is highly effective against clinically relevant enveloped viruses. It has also been observed that mixture of ethanol and acid shows high efficiency against viruses that are more resistant to ethanol alone (Kampf, G.2018)

The World Health Organization suggests alcohol based hand sanitizers not only for coronavirus but also for hepatitis C virus, Zika Virus, Murine norovirus and bovine viral diarrhea virus. The **Quantitative suspension test** showed the complete inactivation of corona virus using alcohol based hand sanitizers. Scientists also performed different tests in which virus is applied to the fingertips, then applied alcohol based sanitizers and determined the number of recoverable viral particles. They observed significant reduction in number specifically, corona virus infected patients when used four different alcohol based hand sanitizers, all the hand sanitizers were able to inactivate the virus below the limit of detection

Non alcohol based hand sanitizer

The alcohol free sanitizers contain chemicals other than alcohol. These chemicals have antiseptic properties. Therefore they show antimicrobial activities. The major advantages of these sanitizers are that they are non flammable, required only in very low concentrations and safe to use specially among children. Their mechanism of action depends upon their functional groups. Some of the examples are

Quaternary ammonium compounds

As the name indicates quaternary ammonium compound contains a central nitrogen atom connected to four alkyl groups. eg. Benzalkonium chloride benzethonium chloride or cetyl peridium chloride. The main target of the quaternary ammonium compound is the lipid envelope of the virus. Lipid bilayer contains phospholipids having negatively charged phosphate moiety. The cationic 'N' adsorbed on the anionic phosphate group. As its concentration increases the fluidity of the lipid envelope reduces and there is creation of hydrophilic gaps. Side by side alkyl chains disrupt the physical and biochemical properties of membranes. This results in solubilization of the lipid bilayer and protein function is interrupted. It also causes conformational change in the genetic material.

Chlorhexidine

0.05 Chlorhexidine is effective against the novel coronavirus. It is as effective as 70% ethyl alcohol. This study was done in Hong Kong University(Chin.AWH,2020).

It acts on the cytoplasmic membrane, disrupts it& precipitates the cell- contents. Sometimes 0.12% chlorhexidine – gluconate solution shows antiviral activity against enveloped viruses such as coronavirus. It is not advisable to use chlorhexidine with soaps and hand creams because chlorhexidine is cationic in nature and soaps, hand creams contain anionic emulsifying agents so precipitation take place which reduces its activity.

Iodine-Iodophors

Iodine is antiseptic in nature but it causes skin irritation so it is replaced by iodophors. Iodophors actually consist of either iodine, iodide or triiodide. In addition it also contains polyvinyl pyrrolidone which is a high molecular weight polymer. The main function of this polymer is to increase the solubility of iodine, improving the sustained release of iodine and decreasing the skin irritation. Scientists have reported that providone- iodine (5-10%) formulations help to mitigate the perioperative spread of (COVID-19) in patient decolonization.

Triclosan

At high concentration, triclosan disrupts the bacterial membrane leading it to death. Actively it is harmful to those bacterial enzymes which form cell walls & membranes. The activity of triclosan is pH based.

Role of glycerol in hand sanitizers

Glycerol or glycerin is added to hand sanitizers in order to protect the hand skin from dryness. It also prevents dermatitis which is caused by repeated use of sanitizers. Glycerol acts as a humectant that maintains the skin moisture. It is safe and relatively cheap.

Role of H₂O₂ in hand sanitizers

It is mainly used to eliminate viable bacterial spores. Its low concentration is recommended because it is corrosive in nature

Use of water

Proper water should be used in making hand sanitizers formulations. Distilled and sterile water is preferred. Ordinary tap water can be boiled & cooled, then it may be used but it should be free from visible particles.

Storage of alcohol based hand sanitizers

Alcohol is highly flammable. The flash point of ethanol (80% V/V) is 17. 5°C and that of isopropyl alcohol (75% V/V) is 19°C. Therefore these sanitizers should be stored in cool rooms or air-conditioned rooms. These should not be kept near open flame or smoke.

Side effects of hand sanitizers

Government has recommended the frequent use of hand sanitizers to combat the COVID-19 pandemic. Washing hands with soap and water can also be prevent from this novel Coronavirus but this is not always possible especially when people are travelling in car or bus doing shopping in the market, attending meetings, in lifts etc. In such situations sanitizers are only alternative. Excessive use may cause side effects which are not healthy but unpleasant also. Some of them are as follows-

- 1. **Disruption of microbiomes** -Sanitizers are known for killing bacteria and viruses to prevent us from different diseases but they can affect the body's microbiomes in a negative way also. Many bacteria are beneficial for our body. These sanitizers may kill them also causing a havoc in healthy bacterial community. The solution of this problem is that one should use sanitizers only when it is not possible to wash hands with soap and water.
- 2. Development of antibiotic resistance bacteria-It has been observed that exposure to hand sanitizers may create the bacteria which are antibiotic resistant meaning that such bacteria can not be killed by antibiotics as they develop tolerance against drugs. Alcohol based sanitizers as well as non alcohol based hand sanitizer contain triclosan contribute to making bacteria resistant to antibiotics.
- 3. Endocrine disruptors- Sanitizer are scented. Sanitizers containing phthalates which are synthetic fragrances. To increase product's shelf life, parabanes are added. Both these compounds are endocrine disruptors and mimic hormones and could alter their functions.
- **4. Dryness of hands** Frequent use of sanitizers faces the problem of incredibly dry hands which is due to alcohol. This problem can be avoided using moisturizer to hydrate the skin.
- 5. Alcohol poisoning- Inhaling sanitizers everyday may cause alcohol poisoning especially in children.
- **6.** Chemicals and sanitizers are dangerous together People working in chemistry labs or dealing with chemicals should avoid using sanitizers because the combination of liquid gel and chemicals can be really harmful for the body as it help in absorption of toxic chemicals in the cells if the hand are not cleaned properly before using sanitizers.
- 7. **Risk of eczema-** Increased contact with sanitizers may increase the risk of eczema which is also known as hand dermatitis. The symptoms of eczema are redness, cracks, dryness and blisters, in acute cases causing itching or pain so it is important to moisturize the skin after every use. Moisturizers generally contain mineral oil or a petrolatum which prevent hand dermatitis. After hand washing with soap and water, moisturizer can be used until hands become completely dry i.e. alcohol should be evaporated. It may take 15-30 seconds.

Nanoparticle based hand sanitizers Department of Biotechnology (DBT) and Department of Science and Technology(DST), India funded a project which is Pune based startup Weinnovate Biosolution(Nature India, April 2020). This Indian start-up prepared a non alcohol colloidal silver solution which contains nanoparticles of silver. This solution is very effective and can be used to disinfect hands. It can also be used for the surfaces where COVID 19 may survive from hours to days . These people are preparing 200 liters every day to meet the demand of hand sanitization. The market price is Rs. 150 for 200 ml bottle. It is a non inflammable liquid.

Silver nanoparticles inhibit synthesis of viral RNA molecules. The nano particles can therefore, stem viral budding. The size of nanoparticles is less than 100 nm. This size matches to the size of COVID-19 virus and plethora of functionalities such as targeting and drug delivery that can be tailored (Nkaura, S. et al, 2019)

Sodium hypochlorite as disinfectant

It is used as coronavirus disinfectant. Its chemical formula is NaOCl. It is also used for disinfection of water, removal of odor, bleaching and for surface purification.

Mechanism of action of sodium hypochlorite

When water is added to sodium hypochlorite, two compounds are formed, one is hypochlorous acid (HOCl) and other is sodium hydroxide (NaOH). Because of formation of NaOH, pH of the solution increases. The reaction is as follows—

$$NaOCl + H_2O \rightarrow HOCl + NaOH$$

 $HOCl \rightarrow HCl + [O]$

Hypochlorous acid disintegrate in hydrochloric acid [HCl] and oxygen [O]. Oxygen atom is a strong oxidizing agent and very effective in killing viruses, bacteria and fungi.

In dilute solutions sodium hypochlorite contains Na and OCI. The following species are present in solution of NaOCI:

$$\begin{split} NaOCl &\rightarrow Na^{^{+}} + OCl^{^{-}} \\ OCl^{^{-}} + H_2O &\rightarrow HOCl + OH^{^{-}} \\ HOCl(aq) + Cl^{^{-}} + H^{^{+}} \rightarrow H_2O + Cl_2(aq) \\ Cl_2(aq) + Cl^{^{-}} \rightarrow Cl_3^{^{-}} \\ Cl_2(aq) \rightarrow Cl_2(gas) \end{split}$$

It releases chlorine which is also a disinfectant. The WHO recommends 2-10% concentration sof bleach solution (sodium hypochlorite) to disinfect the hard surfaces from the novel coronavirus. This solution is not only applicable to coronavirus but also prevent from flu, other viruses and bacteria. In Uttar Pradesh the migrants coming from different states are sprayed with sodium hypochlorite solution.

Characteristics of sodium hypochlorite

Anhydrous sodium hypochlorite is explosive in presence of air and CO2 but its pentahydrate is stable and non explosive. Pentahydrate is crystalline and formula is NaOCl.5H2O although sometimes it is also expressed as 2NaOCl.10H2O. It is greenish yellow in nature and rapidly decomposes at room temperature. However only 1% decomposition is observed at 70°C after 360 days. Potassium hypochlorite can also be used but sodium hypochlorite is cheaper than potassium hypochlorite.

As disinfectant Sodium hypochlorite solution is widely used in healthcare facilities because it shows broad spectrum antimicrobial activities. The 0.5% solution of hypochlorite is known as 'Strong chlorine solution' and used to inactivate C. difficile and HPV. For washing hand weak chlorine solution is used which is 0.05% solution of sodium or calcium hypochlorite.

Dakin Solution- Daking solution is nothing but a dilute solution of sodium hypochlorite. During world war I, English chemist **Henry Dokin** and French surgeon **Alexis Carrel** prepared the sodium hypochlorite solution and used it to clean and irrigate wounds (Sabbatani,S.2017). This solution was known as the **Carrel Dakin** Solution but nowadays it is known as **Dakin solution.** To preserve and save limbs Carrel and Dakin used this solution to irrigate wounds after surgical debridements. They saved many lives by this treatment.

It acts as a germicidal and bacteriostatic topical agent. It is very effective against a broad spectrum of aerobic and anaerobic bacteria as well as viruses, fungi and spores. It shows bactericidal activity against Enterococcus, Streptococcus aureus, Streptococcus epidermidis, Escherichia coli, Klebsiella pneumonia, Enterobacter local, Serratia marcescens, Proteus mirabilis and Pseudomonas aeruginosa(Cardile, A.P. et al, 2014).

It is antiseptic in nature also.

Uses of sodium hypochlorite

- 1. To purify water of wells 2% solution is used for shock chlorination.
- 2. 3-6 % solution of sodium hypochlorite is used in fish processing, mushroom production, clothes laundering, Hog, Beef and Poultry productions, root canal treatment in dentistry, household disinfectant, hospital disinfectant.
- 3. Wastewater treatment, water treatment and for disinfection of swimming pools, 12-16% sodium hypochlorite solution is used.
- 4. It is commonly used as a bleaching agent in the textile industry, in detergents, paper & pulp factories. For the refining of petroleum products, it is widely used in the petrochemical industry.
- 5. Sanitary equipment used it in large quantities.
- 6. It also disinfects glass and ceramics.

- 7. The house-hold bleach which is actually a 3-8% sodium hypochlorite solution. Sometimes it contains 0.01-0.05% sodium hydroxide which prevents it from decomposition. It is also used in laundering clothes. It is applicable as surface cleaner.
- 8. It is used for cleaning purposes. It has disinfectant properties. It removes stains of molds. Fluoresces is a disease which develops stains on teeth. It is helpful in removing dental stains.

Precautions

- 1. Ammonia should not be mixed with sodium hypochlorite solution in any case. It produces choking.
- 2. Eyes and skin should not come in contact with strong NaOCl solution.
- 3. It should not be swallowed.

Stability of sodium hypochlorite

As a disinfectant, the stability of sodium hypochlorite at 4° for 2 years can be maintained. But after 2 years and at 24° , the concentration of chlorine decreases and becomes less than 50% than original. A more rapid deterioration occurred in a solution containing approximately 100,000 ppm chlorine than those containing 50,000 or 100,000 ppm chlorine. When the pH is 5-6 decomposition rate is rapid. That is why NaOH is added to increase the pH so reduce decomposition.

Formulations of sodium hypochlorite

It is sold in the market by different names. The Trade Names are: Antiformin, BK Liquid, Chlorax, Dakin's Solution, Hypochlorite, Milton Chloros, Jovella water, Piochlor, Purix etc.

Preparation of 1% sodium hypochlorite solution

1% Sodium hypochlorite is used to disinfect the metallic surfaces e.g. security locks, keys, machine, table tops, chair handles, handrails, handles, laptops, elevator call buttons etc. One should dip a cloth in 1% sodium hypochlorite solutions and wipe the sweepings.

Product: Sodium hypochlorite-liquid bleach **Preparation**: 1 part bleach to 2.5 parts of water

Product: Sodium hypochlorite liquid

Preparation: 1 part of liquid to 4 parts water.

The government of India issued a guideline for disinfection of public places with 1 % sodium hypochlorite solution for preventing corona virus. Finally we can say hand sanitizers are not as good as soaps.

References

- 1. Cardile, A.p.; Sanchez, C.J.; Hardy, S.K;Romano, D.R.; Huntgen B.J.; wenke, j.C., Murray, C.K.; Akers, K.S. (2014) Dakin solution alters macrophages viability and function. J. Surg. Res., 192(2), 692-9.
- 2. Chin, A.W.H., Hu, JTS., Perera, MRA., Hul, K.P.Y., Yen, H.L., Char, M.C.W, peiris, M. and Poon, L.L.M (2 April 2020) Stability of SARS-CoV-2 in different environmental conditions, Lancet Microbel(1) published online.
- 3. Kampf, G.(2018) Efficiency of ethanol against viruses in hand disinfection. J. Hosp Infect. 98:331-338
- 4. Nkaura, S. et al(2019) synthesis and application of silver nanoparticles. (AgNPs) for the prevention of infection in healthcare workers, Int. J. Mol. Sci. 20. 3620
- 5. Nature India, springer, research highlights, published on line 6 April 2020
- 6. Sabbatani, S. and Fiorino, S. (2017) the treatment of wounds during world war I, Infexz. Med. 25(2):184-192

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IMPORTANCE OF SUPPLEMENTARY FOOD FOR INFANTS

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Abstract

Supplementary foods are defined as the foods that are provided along with breast milk. In the past such foods were often called weaning foods. Supplementary food also improved the health of older children because adequate amount of suitable nutrients are available in these food. The present study designed to investigate the impact of supplementary food on infants between age of 0-12 months. This study is an effort to described weaning practices and effect on infants, further more to find out the reason responsible to the health and growth of normal infants. 200 beneficiaries of different families were selected. Under the study mothers were interviewed in the selected areas of the Prayagraj City through questionnaire method. The finding of the present study demonstrates that 50.5% mothers were started first semi-solid food at the recommended time.

Introduction

Supplementary feeding, also known as weaning mixed feeding or introduction of solid food should begin for infants by six months of age but not before seventeen weeks. Breast milk or infant formula should continue during the complementary feeding period with among gradually reduced as the verity of food increase. As all infants needs are different, health care, professional have to be aware of key nutrients and foods needed at the same time as monitoring growth and understanding the needs of parents and the resources available to them(1), Introduction of solid, semi solid or soft food is very important to the health and well being of a child. According to the new world health organization (WHO) indicators the timeliness is assessed by whether infants aged 6 to 8 months received solid, semi solid or soft food, irrespective of being breast feed or not (2), Complementary feeding occurs when children receive foods to complement breast milk or infant formula. Ideally, it begins at six month of age and continues to 24 months or beyond, reflecting the world health organizations recommendation for exclusive and continued breast feeding. Around the age of six months, an infants need for energy and nutrients starts to exceed what is provided by breast milk and complementary foods are necessary to meet those needs. An infant of this age is also developmentally ready for others food. If complementary foods are not introduced around the age of six months or if they are given inappropriately an infant's growth may falter (3). Appropriate breast feeding and supplementary feeding practices are fundamental to children's nutrition, health and survival during the first two years of life. The world health origination recommends exclusive breast feeding until six months of age and continued breast feeding for at least two years, along with the timely introduction of adequate amounts of complementary foods of suitable nutritional and complementary feeding of children up to two years of age (4), National family health survey (NFHS-3) for Karnataka State, India showed that 72.5% of children aged 6 to 9 months were receiving complementary feed and breast milk also(5), delayed weaning can lead to serious health complications for the infants because after six months of age, breast milk alone is not sufficient both in quantity and quality to meet the nutritional requirements of the child specially for energy and micronutrients. Breast milk is notably insufficient in iron, zink and vitamin A requirement of the nursing baby (6). United Nation Children Emergency Fund (UNICEF), founder of baby friendly initiative complementary feeding should start after the six month of life. At six months the first solid food should be iron-fortified cereals, pap with soyabean power, vegetable, egg, meat (7). According to NFHS-4 Tamilnadu, at age 6 to 8 months a little over two third (68%) of children receive breast milk and complementary food also (8). There is vital importance of supplementary feeding for infants for their health and proper nutrition. Supplementary food are additional sources of essential nutrients like protein, iron, energy, vitamin A and C. These are usually deficient from the diet which a baby is given, The foods with which a baby begins his journey to

varied tastes are pulses, cereals, juices, fruits and soups, especially of leafy vegetables (9).

Material and Methods

The proposed study was conducted on mother to be aware of feeding practices and reduction of malnutrition and improve health and development through infant feeding practices. Our main purpose of this study infants and young child feeding is established as a social norm in all communities, so the study was conducted on 200 mothers of infants 0 aged to 12 months in Prayagraj City U.P. The targeted sample size of 200 subject was achieved all mothers aged between 20 to 40 years with infants aged 0-12 months. Subject were explained orally about the study and verbal data collection and educational counseling was done by single observer. A pretested questionnaire was used, questionnaire includes various demographic and socioeconomic factors like age, religion, regarding initiation and duration of breast feeding, exclusive breast feeding, pre-lacteal feeding, supplementary feeding etc.

Results

As per the survey total 200 infants in the age group of 0 to 12 months were participated in the study out of them one hundred five (52.5%) were girls and ninety five (47.5%) were boys. It was noticed that the maximum awareness in the age group of 30 to 35 years. In the present study 50.5% mother were started first semi-solid food at the age of six to nine months and seven percent start between 3 to 6 months and 42.5% mothers not given to her infants till now because its behind some causes due to under age of six months.

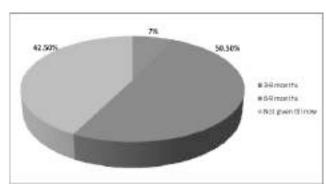


Fig. - 1: Distribution of infants according to when gave first semi solid foods

The distribution of infants according to given first semi-solid food 26.5% were given market preparation and 16.5% infants were given semolina porridge, sago etc. and 5% were given fruits.

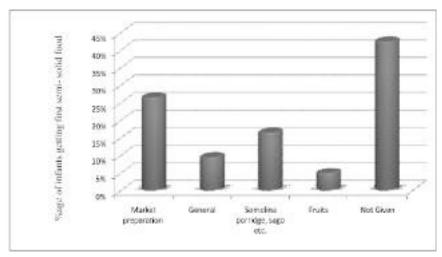
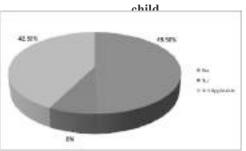


Fig - 2: Distribution of infants according to given first semi solid foods.

Distribution of infants according to any special ceremony for starting general semi-solid or solid foods that 49.5% babies were celebrated special ceremony for starting general semi-solid or solid food and 8% were not celebrated any ceremony and 42.5% were not applicable for that special ceremony

Fig. - 3: Distribution of infants according to any special ceremony for starting general semi-solid or solid foods to the



Discussion

In the present study maximum mothers 50.5% had started first semi-solid food at the recommended time and 7% mothers had started semi-solid food at earlier time. The most common region given for the earlier time that mother was back to work or illness. In developing countries, the age of which breast fed infants were first given complementary foods is of public health importance because of the risk of diarrhea disease from contaminated weaning foods, and the potential risk of growth fathering if foods are inappropriately delayed. In the present study 7% of infant were weaned prematurely. Premature weaning is also reported in other studies according to WHO/UNICEF breast feeding should also be prolonged, up to 2 years it is usually observed that significant emphasis is placed by the mothers on the beginning of the weaning period, but there are no added Physiological, Psychological, Economic or nutritional advantages of early weaning. The majority 26.5% mothers had initiated weaning with market preparation, 16.5% mothers had started weaning semolina, sago etc. In the previous study shown that the supplementary foods given by most of the mothers were commercial infant food like carelac, nutranul and home made preparations like Kheer, Khichri, dalia, halwa, semolina kheer, mushed fruits and vegetables. In the hills if a lactating mother get back her first period the weaning (Annaprashan) ceremony is proponed and the mother in-law cooks some thing sweet, feeds the baby one tea spoon first, than only they allow the mother to feed the baby. In the present study 49.5% infants gave semi-solid or solid food with celebrate the special ceremony and 8% were not celebrate. Although centuries old tradition, modern science has established the fact that child's digestive system is capable of processing solid food when they were approximately six months old.

Conclusion

Supplementary feeding program can have a greater impact on child growth if supplement foods are provided when children are two years old or younger. Result from the studies in the systematic review were mixed but generally showed that children grow more if they received supplementary food earlier in life the period between six months and 24 months is a time critical window for supplementary. Supplementary feeding does however, also improve the growth of older children. The first two years of life represent an especially challenging period for children's nutrition and health because their relatively high metabolic rates and rapid rates off growth during this period impose proportionately greater nutrient requirements. The aim of present study is the best interest of infants and their mothers to ensure the best possible start their life as the foundation for full filament of the every baby right to survival, growth and development, protection and participation without any hindrance.

References

- 1. Cow brough Kathy complementary feeding for infants 6 to 12 months. J FAM Health Care .2010;20(1):20-3 PMID: 20397553
- 2. World Health Organization, complementary feeding
- 3. World Health Organization. Infant and young child feeding.2010 http://www.who.int/mediacentre/facts heets/fs342/en/ Accessed November 17,2013
- 4. Kenneth H. Brown breast feeding and complementary feeding of children up to 2 years of age department of nutrition and program in International and community nutrition, University of California, Davis. CA.USA
- 5. Nutrition in India National Family Health Survey (NFHS-3) India 2005-6
- Ahmad Z, Kyi DW and Lsa AR. Breast feeding and weaning practices in Rural Communities of Kelantan. MAL J Nutr 2. 1996, 148-154
- 7. WHO/NUICF/BFI 2000 The UNICEF U.K. Baby Friendly Initiative: ten steps.
- 8. National family Health Survey (NFHS-4) 2015-16
- 9. My Child Health Supplementary Feeding for Infants.

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VALUE ADDED PRODUCTS FROM KARONDA FRUIT

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Abstract

Karonda (*Carissa carandus* L.) is a fruit of dry areas and flourishes well on lands with high temperatures. It is a widely used medicinal plant grown by tribal throughout India and popular in various indigenous system of medicine like Ayurveda, Unani and Homoeopathy. Fruits are sour and astringent in taste, and are rich source of iron and vitamin C. Its fruits have antiscorbutic properties and arevery useful in the cure of anaemia. The storage life of karonda fruit is very short due to its high moisture content. Ripe fruits are sub-acidic to sweet in taste with a peculiar aroma and can be used in the preparation of various value added products. Mature fruit contains high amount of pectin and, therefore, besides being used for making pickle, it can be exploited for making jam, jelly, squash, syrup and chutney *etc* which are of great demand in the international market. Processing of karonda into a variety of products with extended shelf life provides opportunity to consumers all over the country to enjoy them throughout the year.

Keywords: Karonda, processing, Value addition

Introduction

Karonda (Carissa carandas L.) popularly known as 'Christ's thorn' is an underutilized minor fruit crop of India. Karonda is an indigenous fruit of India and belongs to the family Apocynaceae. The fruit is very hardy, evergreen bush growing well even on marginal and inferior land where most other fruits either fail to grow or give poor performance. It is found in Africa, Australia and Tropical Asia, particularly in Western Peninsula and dry tracts of India, Sri Lanka and Malaysia mainly in wild form. In India, it was cultivated by the Europeans in kitchen garden to get fruit for jelly preparation (Gupta, 2019). Fruits are harvested at both mature and ripe stage depending on their use. The unripe fruit is sour, astringent, bitter, thermogenic, constipating, aphrodisiac, appetizer, antipyretic, hyperdipsia, diarrhoea, anorexia and intermittent fevers. The fruits are rich in protein (1.12-2.25%), vitamin C (1.6-17.9 mg/100g) and minerals especially iron (39.1mg/100g), calcium (21 mg/100g) and phosphorus (38 mg/100g) (Kumar and Singh, 1993). The ripe fruit is sweet in taste with peculiar aroma, dark brown to purple in colour and it has cooling effect, good appetizer and antiscorbutic. It is also useful in burning sensation, skin diseases and scabies. The fruits may be eaten as a dessert when ripe or used in the preparation of fruit products such as squash, candies, jelly and chutney (Shaheel et al., 2015). The fresh karondafruits are generally not consumed as it is due to highly acidic and astringent properties; therefore karonda is not a popular table fruit. But, it has got great potential in processed forms. The fruits may be eaten as a dessert when ripe or used in the preparation of fruit products such as candies, jelly, squash and chutney. Moreover, the storage life of karonda is very short because of its soft flesh and high moisture content. The unripe fruit harvested at maturity can be stored for 5 to 7 days at room temperature, but at ripe stage, it can be stored only up to two days. Although, recently value added commercial preparations are made for domestic use and for export by food processing companies, the plant has largely remained an underutilized species (Manivasagan et al., 2006).

Medicinal importance of Karonda

The karonda fruit is an astringent, antiscorbutic and as a remedy for biliousness and useful for cure of anaemia. In traditional medicine, the fruit is used to improve female libido and to remove worms from the intestinal tract. The fruits have anti-microbial and antifungal properties and its juice used to clean old wounds which have become infected. The fruit have an analgesic action as well as an anti-inflammatory one. The juice can be applied to the skin to relieve any skin problems. Traditionally, karonda has been used to

treat anorexia and insanity. A leaf decoction of karonda is used against fever, diarrhoea, and earache. The roots serve as a stomachic, vermifuge, remedy for itches and insect repellent.

Composition

Table: 1 - Nutritive value of karonda fruits

Nutrients	Nutritional value (100 g)		
	Fresh	Dried	
Energy (Calorie)	42	364	
Moisture (%)	91	18.2	
Protein (%)	1.1	2.3	
Carbohydrate (%)	2.9	67.1	
Fat (%)	2.9	9.6	
Mineral (%)	-	2.8	
Calcium (mg)	2.1	160	
Phosphorus (mg)	28	60	
Iron (mg)	-	39	
VitaminC (mg)	200-500	1	

Source: (Tripathi et al., 2014)

Karonda and its uses

Karonda is a good appetizer. Karonda is used mainly used for making pickle, for making jelly, jam, squash, syrup and chutney. Ripe fruits exude white latex when severed from the branch. The fruits have astringent properties and used for tanning and dying. The ripe fruit emits a gummy latex when it is cooked, but yields a rich red juice which clears when it is cooled, so this is used as a refreshing cooling drink in hot weather. It is also sometimes substituted for apples to make an apple tart, with cloves and sugar to flavour the fruit. Usually the fruit is pickled before it gets ripened. Ripe karonda fruit contains high amount of pectin therefore it is also used in making jelly, jam, squash, syrup, tarts and chutney. The sweeter types may be eaten raw out-off hand but the more acid ones are best stewed with plenty of sugar(Maheshwariet al., 2012)

Value addition of Karonda fruit

Fruits are very important for nutritional security and have high potential for value addition. Among all the commercial fruits, Karonda (*Carissa carandus* L.) is one of those fruits, though it has less commercial value but has potential to support more livelihoods once value-added. Karonda fruits can be processed into number of quality products viz., jam, jelly, candy, preserve, chutney, powder, squash, crush, syrup, pickle, powder, syrup, flavoured ice cream, flavoured milk.

Karonda jam

Jam is a product made by boiling fruit pulp with sufficient quantity of sugar to a reasonably thick consistency, firm enough to hold the fruit tissues in position (Srivastava and Kumar,2006). The studies were based on variations of sugar and to find out the best treatment for maximum storage period. The experiment comprised of 5 levels of addition of sugari.e. 850, 950, 1050, 1150 and 1250 g were added to 1.0 kg of fruit pulp, respectively and data obtained was analysed by completely randomized design. Results obtained from a study showed that 1000 g pulp + 1150 g sugar possessed an ideal value of total soluble solids (TSS), pH, acidity, moisture, ascorbic acid, iron and overall acceptability at 0, 20, 40 and 80 days of storage. These seven parameters showed that the quality of karonda jam obtained by incorporating 1150 g of sugar was of good texture and quality (Wani et al., 2013).

Ripe firm fruits \rightarrow Washing \rightarrow Peeling \rightarrow Pulping (Remove seed and core) \rightarrow Addition of sugar and acid \rightarrow Boiling (with continuous stirring) \rightarrow Judging of end point by further cooking up to 105° C (or) 68% TSS (or) by sheet test \rightarrow Filling hot into sterilized bottles \rightarrow cooling \rightarrow Sterilized bottles \rightarrow cooling \rightarrow Sterilized bottles \rightarrow cooling \rightarrow Storage (at ambient temperature).

Fig.: 1 - Flow sheet for preparation of jam (Srivastava and Kumar, 2006)

Karonda jelly

Jelly is a semi solid product prepared by boiling a clear, strained solution of pectin containing fruit extract, free from pulp, after the addition of sugar and acid. A perfect jelly should be transparent, well set but not too stiff, and should have the original flavour of the fruit(Srivastava and Kumar, 2006). Chaudhary *et al.* (2007) reported that the physico-chemical changes in karonda (*Carissa carandas*L.) jelly during storage were found to be organoleptically acceptable for a period of 4-5 months. After that, the jelly started changing to brownish colour at ambient temperature. The total soluble solid, acidity, total sugar, reducing sugar, browning, flavour, texture, taste and after taste increased with storage period while ascorbic acid, non-reducing sugar, colour and appearance and overall acceptability decreased at ambient temperature.

Selection of fruit \rightarrow Washing \rightarrow Deseed the fruit and cut it into thin pieces \rightarrow Boiling with equal quantity water for about 20-30 min \rightarrow Addition of citric acid during boiling(2 g/kgof fruit) \rightarrow Addition of citric acid during boiling \rightarrow Straining of extract \rightarrow Pectin test(For addition of sugar) \rightarrow Straining of extract \rightarrow Addition of sugar and boiling of extract \rightarrow Addition of desired quantity of pectin \rightarrow Judging of end-point(Sheet test) \rightarrow Removal of scum or foam \rightarrow Addition of remaining citric acid \rightarrow Filling hot into clean sterilized glass jars \rightarrow Waxing \rightarrow Cooling \rightarrow Capping or lidding \rightarrow Storage at ambient temperature

Fig.: 2 - Flow sheet for preparation of jelly (Singh, 2010)

Karonda candy

Fruit candies are becoming more and more popular because of high acceptability, minimum volume, higher nutritive value and longer storage life. Any fruit/vegetable impregnated with cane sugar (or) glucose syrup and subsequently drained free of syrup and dried is known as candied fruit/vegetable (Srivastava and Kumar, 2006). The osmotic dehydration of karonda was studied with two concentrations of sugar *viz.*, 60 and 70° Brix syrup with three different durations of dipping times *viz.*, 6, 18 and 24 hours. After osmosisof the karonda slices in sugar solutions these were laid on the hot air oven for dehydration. After osmotic dehydration, the products were packed in high density polyethylene bags and stored in ambient temperature for a period of 4 months. The microbiological changes and a taste panel evaluated the organoleptic quality of the products during the storage period. Minimum microbial count (2.00) was recorded for osmosis in 70° Brix syrup for 24 hours. The product of 70° Brix syrup for 18 hwhen stored 4 months at room temperature secured highest score (81.97) in organoleptic evaluation (Suhasini *et al.*, 2015). Gupta *et al.* (2009) reported that effect of storage on chemical parameters of karonda candy prepared by various methods was found to be organoleptically acceptable for five months. The result revealed that the TSS content showed an increasing trend, however decreasing trend was observed in ascorbic acid, acidity and moisture content with the advancement of storage period.

Selection of hard ripe fruits→ Washing→ Slice the fruit into two equal parts→ Remove seeds→ Salt solution preparation →Osmotic treatment→ Draining→ Hot-air oven drying→ Packaging→ Storage

Fig.: 3 - Flow sheet for preparation of Karonda candy (Suhasini et al., 2015)

Karonda Preserve

A mature fruit/vegetable (or) its pieces impregnated with heavy sugar syrup till it becomes tender and transparent is known as preserve (Srivastava and Kumar, 2006). Karonda fruits were osmotically preserve and packed in three different packaging materials *viz.*, polyethylene, aluminium laminated and shrink packages. The osmotically preserved fruits were stored under refrigerated conditions for six months and analysed for quality and sensory attributes at an interval of two months. Aluminium laminated packaging proved to be the best among the all packaging in maintaining superior quality up to six months of storage as indicated by higher mean titratable acidity (5.78%), reducing sugars (10.11%), total sugars (37.71%), ascorbic acid (4.77mg/100g) and lower moisture content (11.14%). All the sensory parameters including colour, taste, texture, flavour and overall acceptability declined significantly during storage period of six months (Singh and Sexana, 2019).

ature fruits \rightarrow Washing \rightarrow Preparation of fruit for sugar treatment \rightarrow Keeping fruit and sugar in alternate layers (1.0 kg Fruit:1 kg Sugar) (or) steeping fruit in syrup of 40% TSS for a day \rightarrow Removal of fruit \rightarrow Increasing consistency of syrup to 60% TSS by boiling \rightarrow Steeping of fruit for a day \rightarrow Repeating the process and raising strength of syrup by 5% TSS to 70% TSS alternate days \rightarrow Steeping in 70% TSS for a week \rightarrow Preserve \rightarrow Draining \rightarrow Filling in jar or container \rightarrow Covering fruit with freshly prepared sugar syrup of 68% TSS \rightarrow Sealing (airtight) \rightarrow Storage.

Fig. : 4 - Flow sheet for preparation of preserve (Srivastava and Kumar, 2006)

Karonda Chutney

Karonda chutney is generally hot, sweet, smooth spicy, mellow flavoured and appetizing. Sometimes raising and dry fruits are also added to increase its taste and nutritional value. Good quality chutney should be palatable and appetizing. They improve the digestion and are good appetizer. The method of preparation of chutney is similar to that of jam except that spices, vinegar and salt are added. The ingredients used for preparation of karonda ingredients are pulp 200 g,sugar110g, ginger 10g,onion 10g,black pepper 2g,cardamom 2g, cumin seed 2.5g,salt 9g,garlic 5g, vinegar 20ml, red chilly 2.5g, cinnamon 2g and aniseed 2.5g.

Mature fruit \rightarrow Washing \rightarrow Pee ling \rightarrow Pulping Cooking with little water to make the pulp soft \rightarrow Mixing with sugar and salt and leaving for an hour \rightarrow Keep all the ingredients (except vinegar) in cloth bag, tied loosely \rightarrow Putting in mixture and cooking on low flame \rightarrow Pressing spices bag occasionally while cooking \rightarrow Cooking to consistency of jam (upto 105° C) with occasionally stirring \rightarrow Removal of spice bag after squeezing \rightarrow Addition of vinegar \rightarrow Cooking for 2-5 min \rightarrow Filling hot into sterilized bottles \rightarrow Sealing (airtight) \rightarrow Storage at ambient temperature.

Fig.: 5 - Flow sheet for preparation of Karonda chutney (Sharma, 2011)

Karonda powder

Saxena*et al.* (2016)studied that the three cultivars of karonda (*Carissa carandus* L.), *viz.*, *Pant Suvarna*, *Pant Manohar* and *Pant Sudarshan* were picked at 40, 55 and 70 days after fruit set and used for the preparation of fruit powder. The powder was prepared by two methods of drying, *i.e.*, Sun and cabinet drying. The maximum yield (21.7%) of powder was obtained in the sun-dried samples of cultivar *Pant Sudarshan*. Highest ascorbic acid content (30.45 mg/100 g) was found in the cabinet dried samples of 70 day old fruits of cv. *Pant Sudarshan*. Phosphorus (0.447%), potassium (18.73%), iron (0.365 mg/100 g), copper (0.012 mg/100 g), and manganese (0.193 mg/100 g) contents were higher in the powder prepared from the fruits of cv. *Pant Suvarna*. Among the drying methods, cabinet drying resulted in better retention of nutrients and less non-enzymatic browning. In general, organoleptic score in respect of colour, texture and overall acceptability was more in cabinetdried powder of karonda fruits picked 70 days after fruit set.

Firm fruits — Washing — Fruits were bleached in 15% brine solution containing 500ppm KMS for 24 h — Fruits were blanched for 2 min — Cut into two halves and seeds were removed — The pieces were spread in aluminium trays and kept in cabinet drier — Dried pieces were ground to form powder — Packaging — Storage.

Fig.: 6 - Flow sheet for preparation of Karonda powder (Saxenaet al., 2016)

Karonda beverages

Various types of beverages like ready to serve (RTS) drinks, nectar, crush, squashes can be prepared from karonda juice/pulp using the methods of Srivastava and Kumar (2006).

Karonda squash

This is a type of fruit beverage containing at least 25% fruit juice (or) pulp, 45% TSS, 1.0% acidity and 350 ppm of SO₂ (or) 600 ppm of sodium benzoate. It is diluted before serving (Srivastava and Kumar, 2006). Bajpai *et al.* 2015 studied formulation of different recipes and organoleptic quality of karonda squash. Results indicated that the recipe containing juice and sugar ratio 1:1.5 with the 0.20% acidity was found to be the best followed by recipe containing juice and sugar ratio 1:1.2 with 0.21% acidity.

Ripe Karonda fruit \rightarrow Washing \rightarrow Extraction of pulp \rightarrow Removal of seeds \rightarrow Preparation of sugar syrup \rightarrow Straining and cooling of sugar syrup \rightarrow Mixing it with karonda pulp \rightarrow Addition of preservatives \rightarrow Bottling and sealing \rightarrow Labelling and storage.

Fig.: 7 - Flow sheet for preparation of Karonda squash (Gupta et al., 2018)

Karonda crush

Divete *et al.* (2020) studied that karonda crush was prepared by using its juice with different percentage of juice level such as 25, 30, 35 and 40 per cent and maintain 55°Brix TSS and 0.8% level of citric acid in each treatments. The physico-chemical composition and sensory qualities of karonda crush were studied during 3 months of storage period to standardize optimum recipe for the preparation of karonda crush. An increasing trend in TSS, reducing and total sugars whereas decreasing trend in titratable acidity and ascorbic acid was observed during storage period of 90 days. The crush recipes *i.e.* 35 and 40% juice with 550 °Brix TSS and 0.8% acidity were found to be the best recipes for the preparation of karonda syrup with highest organoleptic score for colour, flavour and

overall acceptability.

Selection of ripe karonda fruits \rightarrow Washing of fruits \rightarrow Extraction of juice using basket press \rightarrow Straining through four fold muslin cloth \rightarrow Addition of sugar, citric acid, water and mixing it with juice to maintain 55°B TSS and 0.8% acidity \rightarrow Slight heating up to 72°C till sugar completely dissolved \rightarrow Hot filling of the product in pre-sterilized bottles \rightarrow Labelling \rightarrow Storage of the product at cool and dry place.

Fig.: 8 - Flow sheet for preparation of Karonda crushes (Divate et al., 2020)

Karonda syrup

This type of fruit beverage contains 25% fruit juice (or) pulp, 65% TSS, 1.3 to 1.5% acidity and 350 ppm of SO₂ or 600 ppm of KMS. It is diluted before serving (Srivastava and Kumar, 2006). The ripe fruits of karonda are boiled with baking soda and salt. For every cup of juicy pulp half tea spoon of baking soda is added and boiled in 1 L of water at 100° C. The mixture is then boiled down one half of the original quantity, removing the rising scum in the process and juice is again strained. For every cup, a quarter cup of sugar is added. The mixture is again boiled for 40 minutes. The cooled syrup is poured in to sterilized bottle and sealed (Arif *et al.*, 2016).

Karonda pickle

The preservation of food in common salt (or) in vinegar is known as pickling. It is one of the most ancient methods of preserving fruits and vegetables. Pickles are good appetizers and add to the palatability of a meal. They stimulate the flow of gastric juice and thus help in digestion (Srivastava and Kumar, 2006). Manivasagan *et al.* (2007) reported that two karonda types *i.e.* pink and green for making pickle and qualitative changes taking place during storage period of four months. The results showed that pH, TSS/acid ratio, ascorbic acid, non-reducing sugars, organoleptic values decreased whereas acidity, reducing sugar and browning increased during storage. The TSS and total sugars decreased in pickles prepared from pink and green type and observed that pickles prepared from pink type of karonda was of good quality as compared to sweet pickle.

Firm and mature fruits were selected, washed and wipe dried. The fruits are individually crushed lightly to create cracks. Chillies were slit vertically and cut in to pieces. For the preparation of cured karonda pickle; the crushed fruits were mixed with salt and allowed to cure for 30 days. After curing all other ingredients (green chillies-250 g, mustard oil-300 ml, salt-250 g, fennel seed-60 g, mustard seed dhal-100 g, chilly powder-10 g, kalunjiseeds-5 g) were mixed thoroughly and stored in a bottle. The contents were stirred on alternate days by shaking the bottles on alternate days of curing. (Hiregoudra, 2012). Karonda pickle is easy to prepare and ready to eat. This pickle can be stored for at least four months.

Karonda flavoured ice cream

Pulp obtained from ripe fruits can be incorporated as natural flavouring agent in ice-creams. Ice-cream with 20% karonda pulp has shown good overall acceptability (Gaikwad *et al.*, 2005).

Karonda flavoured milk

Utilization of karonda juice in the manufacture of flavoured milk was explored by Hanwate *et al.*(2005). The flavoured milk containing 10% karonda juice and 7.5% sugar recorded highest acceptability.

Conclusion

The storage life of karonda is very short because of its soft flesh and high moisture content. Moreover, the fruits are not being highly consumed because of strong acidic and astringent properties and have no or very less market value. Thus, value addition in these fruits can lead to high commercial value as well as their uses. Thus, there is an opportunity for the rural people of *kandi* areas to explore more through value addition which can be a potential source of nutrition and livelihoods.

References

- 1. Arif, M., Mehnaz K., Jawaid, T., Khalid, M., Saini, K.S., Kumar, A. and Ahmad, M. 2016. *Carissa carandas* Linn. (Karonda): An exotic minor plant fruit with immense value in nutraceutical and pharmaceutical industries. *Asian Journal of Biomedical Pharmaceuticals Sciences*, 6(58): 14-19.
- 2. Bajpai, R., Yadav, M., Mure, S. and Kushwah, R.S. 2015. Browning analysis of different karonda processed products during storage. *Plant Archives*, **15**(1): 339-342.
- 3. Chaudhary, R., Yadav, M. and Singh, D.B. 2007. Changes in physico-chemical characteristics of karonda jelly during storage period. *Plant Archives*, 7(2):885-887.

- 4. Divate, S.M., Savale, B.G., Patil, N.B. and Relekar, P.P. 2020. Study of preparation and standardization of Karonda (Carissa congesta L.) crush *The Pharma Innovation Journal*, **9**(1): 176-179.
- 5. Gaikwad, R.P., Bhambure, C.V., Kadam, R.M., Joshi, S.V. and Yadav, D.N. 2005. Incorporation of karonda (*Carissa carandas* Lam.) pulp in ice-cream In Souvenir- National Seminar on Value Added Dairy Products, held at National Dairy Research Institute, Karnal, December 21-25, 2005.
- 6. Gupta, N. 2019. Studies on preparation of blended karonda-beet root ready to serve beverage. *Indian Journal of* Horticulture, **76**(4): 735-740.
- 7. Gupta, N., Singh, D.B., Singh, V.B. and Kumar, V. 2009. Effect of storage on chemical parameters of Karonda candy prepared by various methods. *Annals of Plant and Soil* Research, **11**(2):112-114.
- 8. Gupta, N., Trilokia, M., Sood, M., Dogra, J. and Singh, J. 2018. Utilization of under-utilized fruits through value addition in *kandi* areas of Jammu region. *International Journal of Current Microbiology and Applied Sciences*, 7(5): 1965-1977.
- 9. Hanwate, B.D., Kadam, R.M., Joshi, S.V. and Yadav, D.N. 2005. Utilization of Karonda (*Carissa carandas* L.) juice in the manufacture of flavoured milk. In the Proceedings of National Seminar on Value added Dairy Products.21-22, December, 2005, NDRI, Karnal, Haryana, India.pp. 164.
- 10. Hiregoudra, V.S. 2012. Physico-chemical characteristics, value addition and shelf life of evaluation Karonda(*Carrisa carandas*). M.Sc. Food Technology, UAS, Dharwad.
- 11. Kumar, S. and Singh, I.S. 1993. Variation in quality traits of Karonda (*Carissa carandas* L.) germplasm. *South Indian Horticulture*, **41**(2): 108-110.
- 12. Maheshwari, R., Sharma, A. and Verma, D. 2012. Phyto-therapeutic Significance of Karaunda. *Bulletin of Environment, Pharmacology and Life Sciences*, **1**(12):34-36.
- 13. Manivasagan, S., Rana, G.S., Joon, M.S. and Godara, A.K. 2007. Study on qualitative changes in pickles prepared from karonda (*Carissa carandas* L.) during storage. *Haryana Journal of Horticultural Sciences*, **36**(1/2): 44-45.
- 14. Saxena, D, Misra, K.K.and Rai, R. 2016. Studies on suitability of cultivars, picking dates and drying methods for the preparation of karonda (*Carissa carandus* L.) fruit powder. *Indian Journal of Horticulture*, 73(2): 267-273.
- 15. Shaheel, S.K., Swami, D.V., Kumar, B.P. and Uma Krishna, K. 2015. Effect of blending of karonda (carissa carandas l.) juice with guava, papaya and pineapple juices on its quality and organoleptic evaluation. *Plant Archives*, **15**(1):187-192.
- 16. Sharma, 2011. Nutritional quality evaluation and value addition of dhew (*Artocarpous lakoocha*) fruits and karonda(*Carissa carandas*) fruits. M.Sc. (Department of Food Science and Nutrition), CSKHPKV, Palampur, H.P
- 17. Singh, 2010. Studies on suitability of cultivars and dates of picking of berries for preparation of imitation cherry and jelly from karonda (*Carissa carandas* L.) fruits. PhD Thesis, GBPUAT, Pantnagar -263 145, Uttarakhand.
- 18. Singh, S. and Saxena, A.K. 2019. Impact of different packages on quality and storability of osmotically preserve Karonda (*Carrisa carandus* L.) fruits under refrigerated storage conditions. *International Journal of Food Science and Nutrition*, **4**(5): 13-17.
- 19. Srivastava, R.P. and Kumar, S. 2006. Principles of fruits and vegetables. 3rd revised and enlarged edition.pp235-238.
- 20. Suhasini, L., Vanajalatha, K., Padmavathamma, A.S. and Vekateshwar, R.P. 2015. Effect of sugar as an osmotic agent on sensory evaluation and microbial count of Karonda (*Carissa carandas* L.). *Research Journal of Agricultural Sciences*, 6 (1): 170-174.
- 21. Tripathi, P.C., Karunakaran, G., Sankar, V. and Kumar, S.R. 2014. Karonda-A Potential fresh fruit of future. Technical Bulletin 7/2014, ICAR-IIHR, CHES, Kodagu, Karnatakapp 14.
- Wani, R.A., Prasad, V.M., Hakeem, S.A., Sheema, S., Angchuk, S. and Dixit, A. 2013. Shelf life of Karonda jams (Carissa carandas L.) under ambient temperature. *African Journal of Agricultural Research*, **8**(21):2447-2449.

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ALBINISM IN INDIA AND DISCRIMINATION BASED ON IT

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Introduction

The word "Albinism" comes from the Latin word albus means "white".it is a congenital (present from birth) disorder characterized by the complete absence of the pigments of the skin, eyes and hairs. This disorder generally occurs due to the absence or deficiency of Tyrosinase (tyrosine 3-monooxygenase) enzyme which is a copper containing enzyme and is involved in the production of the melanin pigments. Albinism was first discovered in humans by a scientist named Sir Archibald Edward Garrod in 1908. He observed that the skins of some people are hypersensitive to sunlight and they also faced vision problems.

Function of the Tyrosinase enzyme: this enzyme synthesizes melanin pigments from the amino acid tyrosine.

Albinism is an inherited autosomal recessive disease. But the exception is X-linked ocular albinism which is an X-linked inherited albinism.in an autosomal recessive inheritance, an individual receives genes bother from the mother and the father but the parents who carries the gene for albinism dost not show any symptoms for albinism. In contrast, X- linked recessive genes mainly affect the males.however, females can still carries and pass the gene for albinism is an extremely rare genetic disease not only in India but in the whole world. If we are talking about India, only 100, 0000-200,000 albino individuals are found. Albinism often causes white skin, light hairs and vision problems. This disease can affect anyone but its prevalence varies by region. Albinism affects the sexes evenly and both males and females are equally susceptible to this disease. Albinism being a recessive trait gets magnified if the parents are closely related.

World Albinism Day is celebrated on June 13 every year.







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Albiinos children having their normal parents

Types of albinism

Types of Albinism-there are different types of albinism based on the type of mutation.

Albinism is divided into subgroups

- 1. Oculocutaneous albinism (OCA): this type of albinism is basically occurs due to the mutation in one of the 4 genes. It is further divided into 4 types i.e. OCA type 1, OCA type 2, OCA type 3, OCA type 4.
- OCA type 1- individuals have milky white skin and hairs, blue eyes. Some individual's skin and hairs may darken with age. It is caused due to mutation in chromosome 11. Some people with this type never experience changes in skin colour but others begin to produce melanin during childhood. Their hairs may become golden to brown in colour. The iris of the eye may also change colour and lose its translucence.
- OCA type2-less severe than OCA type 1. This type of OCA type 1 is not found in India. It is caused due to mutation in chromosome 15. People have yellow or reddish hairs. Their skin may be light brown.
- OCA type 3-this type of OCA is also not found in India. It is caused due to mutation in chromosome 9.
- OCA type 4- it is similar to type 2. it is caused due to mutation in chromosome 5. It is a very rare form of albinism.
- 2. X-linked ocular albinism: this is caused by a mutation in the gene of X-chromosome. It generally affects the males. Skin, hairs and eye colours are generally normal but vision problems are found.
- Hermansky-Pudlak-Syndrome- it is also a rare form of albinism and is caused due to mutation in at least 8 genes associated
 with this syndrome. Along with having symptoms similar to oculocutaneous albinism, people may have lung and bowel
 problems also.
- Chediak-Higashi Syndrome- again a rare form of albinism and is caused due to mutation in the LYST gene. People generally have symptoms similar to oculocutaneous albinism.in addition to this; people also have problems related to immune system.

Characteristics of albinism-

- Albinism is a group of genetic condition that results in less or no production of melanin pigments.
- Most individuals having albinism are very sensitive to sun exposure. Continued photosensitivity leads to many skin diseases including skin cancer.
- All types of albinism cause problems related to development and function of the eyes, vision impairments are common among all types of albinism.
- These impairments are caused due to Disfunctioning of the optic nerves and optic muscles.
- Types of albinism are based primarily on which mutated gene caused the disorder.



Causes

1. Albinism primarily affects skin, hairs, eyes and vision. The most common cause of this genetic condition is the disturbance in the functioning of the enzyme Tyrosinase. Tyrosinase is a copper containing enzyme involved in the production of Melanin pigments (skin pigments). So, if the function of the Tyrosinase is disrupted then it directly leads to the deficiency or absence of melanin pigments. This melanin is the pigment responsible for the coloration of the skin, hairs and eyes.

Function of melanin pigment- melanin is the pigments responsible for the coloration of the skin, hairs and eyes. It protects the skin from harmful ultraviolet rays coming from the sun. Therefore, because of its absence, individuals having albinism are also hypersensitive to the sun rays which in future lead to many skin diseases (acne, rosacea, psoriasis, eczema) as well as skin cancer also. Melanin also involved in the development of the retina of the eyes and helps in optic nerve pathways from eyes to brain.

- 2. Albinism results from a mutation in genes. One out of 70 people are carries genes for albinism. These mutations disturb the functioning of the enzyme Tyrosinase. Melanin production becomes slow or completely stopped due to the mutation.
- 3. Patients with OCA1 have mutations in a gene called TYR which is responsible for producing the enzyme Tyrosinase, used by cells to convert the amino acid tyrosine into pigment molecules that color the skin, hair, eyes. While, OCA2 is caused due to mutation in OCA2 gene, which encodes the P protein (function of P protein is still not known)

Symptoms

- 1. People suffering from albinism generally have light coloured skin and hairs than rest of the family members or society.
- 2. Individuals also have vision problems which is also a very common symptom.
- 3. Effect of albinism on skin- this condition makes the skin lighter in colour.but it is also seen that in some individuals, levels of melanin pigment increase slowly over time so darkening of skin occurs as the person ages. Sunburn is also common.
 - After sun exposure, their skin also develops moles (which are usually pinkish in colour due to the reduced melanin pigments), and spots.
 - For some people with albinism, skin pigmentation never changes. For others, melanin production may increase during childhood and then slowly decrease as the person ages.
- 4. Effect of albinism on hairs-people having albinism have hairs which are usually whit to grayish in colour.
- 5. Effect of albinism on eyes and vision eyelashes and eyebrows are generally pale. Eyes in such people are generally light blue to brown in colour. It is because of low levels of melanin in the iris of eyes makes the eyes appears to be translucent. Due to the absence of this pigment, the iris is unable to block the harmful sun rays which directly lead to vision problems. Vision problems include: Amblyopia (also known as lazy eye), Myopia (near sightedness), Hypermetropia (far sightness), photophobia (eyes becomes sensitive to light) Astigmatism (blurred vision).
- 6. Children with albinism may sometimes have symptoms such as nosebleeds and chronic infections .these signs and symptoms may indicate the presence of albinism (Hermansky-Pudlak syndrome, a type of albinism) but it very rare among children of India.

Treatment

However, there is no cure for the Albinism because this disease is genetic. But some precautions and treatment is helpful.

- 1. If there are any moles or spots on skin which are increasing in no. with time, then it is advisable that such people should report to a doctor. As there is very high risk of skin cancer in people having this disease, it is suggested that people with albinism must use sunscreen lotions and creams of SPF 30 or higher.
- 2. People having albinism have vision problems. So, they are advisable to use telescopic lenses attached in the glasses and also suggested to avoid use of computer screens and laptops as much as possible. Regular eye checkups are recommended.
- 3. Albinism can be diagnosed by genetic testing and comparing the individual's coloration with the rest of the family members.
- 4. At the birth of a child, if the doctor notices a lack of pigment in the baby's skin and hair, the doctor will order an eye exam.
- 5. Albino patients are more susceptible to develop malignancy .so, early diagnosis and treatment of premalignant lesions and mole is necessary. Patients and their families are counsel about the nature of disease and about protective measures.
- 6. Lifestyle changes such as minimizing of outdoor activities during peak sunlight hours should be avoided.
- 7. Surgery can also be done to correct some vision problems but it is rarely a part of treatment .however in some cases, ophthalmologist suggests surgery on optical muscles that minimize blinking problems of eye especially.
- 8. In schools and colleges also, teachers may make some adjustments for albinos like: seat in front of the classroom, providing large-prints notes and texts.
- 9. A compete diagnosis for albino patients include:

- Physical examination
- Description of changes in pigmentation of skin, hairs, nails.
- Eye checkups
- Genetic counseling
- A test called electroretinogram, which measures waves of brain (alpha, beta, theta waves) produced when light is focused on eyes, helps in identifying the Disfunctioning of the optic nerves and optic muscles.

Jeevan Trust - an NGO in India for people with Albinism

Jeevan trust is an NGO founded by Anubhav Gupta the helps albinos and transpeople. It has a facebook group with about 400 members that brings albinos together from around different places in India and help them come out of their problems.

Anubhav Gupta along with his some friends founded Jeevan Trust in Delhi to create and aware people about albinism and helps those people who already being suffer from this.

In 2010, Anubhav Gupta started his work on a project on albinism. His NGO also approaches media, schools, colleges, societies to aware people about albinism Sameer Garg; a teacher is working with Anubhav Gupta to raise awareness about this issue. Being an albino himself, Garg helps the Anubhav in this initiative. The aim of this NGO is to help the albinos with their low vision, social, emotional, personal, professional issues, marriage issues, genetic counseling. They also shares protective measures about eye vision, self-esteem issues and many more problems.

Anubhav Gupta also nominated for this great work with Karma veer Puraskar in year 2016.

According to Sammer Garg, family members support is not enough, other people support is also necessary. In addition to this, this NGO is helping a family of 12 members living in Delhi who all have albinism.

In India, albinism is often confused and related with another disease called leucoderma (a medical condition characterized by skin colour changes). But this NGO aware the people more on this issue.





Anubhav Gupta- director of Jeevan Trust (in the left)
Family of 12 members in Delhi (in the right)



Anubhav Gupta with a person having albinism

Problems and discrimination faced by the people having albinism in our country

Individuals suffering from this disease face some social problems such as they may be considered as an outsider in schools, colleges and places as they look different from the normal people because of the lack of information and knowledge among normal people about albinism. Such people also face many social challenges like people make social distance from them and keep them isolated and also not talk to them which can lead to stress, worries. They feels low self-esteem and also lack confidence .due to these social factors, such individuals avoid social interactions and also become emotionally unstable. They may also find difficulties in completing their education, employment and in finding their life partners.

According to a study carried by National Geographic estimated in 2013, person suffering from albinism are murdered for profit also and their body parts are sold at a valuable rate to earn money because it is wrongly believed by some people that having sex with people having albinism cures AIDS. (This is a big myth)

People with albinism may face teasing and questions based on their appearance and vision. The problems and impairments due to their conditions make the life of albino persons difficult. They have trouble with the sun and have to cover their body parts with heavy and long sleeved clothes wear sunglasses always on exposure to the sunlight to protect their eyes. They even lack the company of friends and people. They are constantly abused by the public with bad names and social tags that feel them guilt. This results in social stigmatization and discrimation in societies for the albino people.

There are also some big myths on albino people like such type of humans never die as albinism is a curse from god and anyone who touches will be cursed. For centuries some people believed that if they go to a doctor with albino body parts, it makes them rich. Many albino persons feel like that they do not belong to society by the institution. So, they are deprived of healthcare facilities, education, employment because they are considered as different people. They also faced limited social and medical support because albinism is still greatly misunderstood. People do not know the exact and scientific reasons behind this genetic condition. So, they are isolated and face violence also. The term albino is often used in a negative way to demoralize such people. Many negative jokes are also made which create stress among albino people. There is another big myth that it is believed that such people bring bad luck or have some magical powers. Their safety is at risk as they are kidnapped and killed. So, they are hopeless which damage their emotional and mental balance causes anxiety, depression and other physiological problems. Women are generally blamed to have a child with albinism so people refuse to marry them.

Indian population with this disorder suffers more discrimination because of superstitious beliefs and the stigma associated with it. This is one of the reasons that albino patients report to doctors about this in very later stage which ultimately lead to complications of this disorder.

The case of albinism in India is a curious one. In our country obsessed with the fair skin we see opposite and rude reactions to people with albinism. People call them as "Suraj mukhi or Angrez" because of their pale skin.

In order to protect the rights of persons with albinism and prevent violations against their human rights, states must adopt legislation to criminalize attacks against person with albinism including harmful practices as well as trafficking of albinos. Legal reform is necessary for protecting the rights of persons with albinism. Public education and awareness raising campaigns and projects aimed at combating prejudice, superstition, misconceptions and discriminations including law officers, members of the judiciary, social workers, families and communities of persons with albinism. This genetic condition is also included in educational courses to educate students and children on the rights of persons with albinism. Also providing resources to develop activities such people and create an environment to respects those people and their rights and dignity.



Discrimination in India based on albinism

Conclusion

Albinism is a rare, genetically inherited disease from birth due to the lack of melanin pigments and is found in all regions. People having albinism have white and light skin, hairs and eyes and also they have vision problems. Their skin is too much sensitive to sunlight. Even they are very sensitive o the bright light so they have higher risk of sunburn and skin cancer on exposure to the sun.

Education and awareness are the keys to stop the murder; abuse and discrimination of the albino people. We have to learn that such humans are just like us. We all humans are the same. We should not treat them wrong just on the basis of their odd appearance. It should be ensured that public education systems are trained to provide specific needs to the people with albinism. It is our responsibility to make efforts to promote the safety of the albino people by investigating crimes against such people and reunified with such people and even with their family members. Regular examination of all albinos for early detection and treatment should be done. Patients should be educated for preventive sun protection measures and early signs of any new developing moles and spots on their body parts. Psychological and genetic counseling should be included as a part of the treatment.

Social message: Being an albino is not to be diseased; it is just a recessive gene.

References:

- 1. https://www.hindustantimes.com
- 2. https://www.thebetterindia.com
- 3. https://rarediseases.info.nih.gov/diseases/5678/albinism
- 4. Chatterjee K.Rasool F,Chaudhari A, Chatterjee G, Singh N. Basal cell carcinoma,oculocutaneous albinism and actinic keratosis in a native Indian. Indian journal of dermatology.20713; 58:377.

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SOCIAL EPIDEMIOLOGY

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Introduction

A branch of medical science that deals with the incidence, distribution and control of disease in a population is epidemiology. It deals with the sum of the factors, controlling the presence or absence of a disease or pathogen that means epidemiology is a whole study of why an epidemic spread.

Epidemiology is a science of epidemics and social epidemiology is a branch of epidemiology which tries to find the social cause of disease.

Social epidemiology deals with the relation of social conditions and population health. It is not a new thought. Several investigations were conduct in the 19th century based on this idea. However, as a branch of epidemiology social epidemiology is relatively new.

About 2400 years ago, Hippocrates known as father of modern medicine, recorded. His observations about how climate living conditions and people's occupation affected the health and various types of illness of people as he saw in his practice (Lilienfeld and Lilienfeld 1980). In his articles "On Airs, Waters and Places, he discusses the natural elements in relation to medicine and medical care. In his treaties he trying to explain that medicine include detailed observation of disease and its effects and an understanding of how health is often influenced by bodily processes and the environment. The idea that social influence health is not new. Chadwick (Flinn 1965) wrote about the insanitary conditions of the working classes and how overcrowding, damp the filth contributed to their lower life expectancy. Durkheim (1996), wrote about how social norms and conditions affect risk of suicide in the population (1). The root of social epidemiology goes back to the work of Emile Durkheim on suicide rates between Protestants and Catholics.

Social epidemiology

Social epidemiology is a branch of epidemiology that studies the distribution and determinants of health and disease in populations(2).In "Social Epidemiology," Berkman and Kawachi defined social epidemiology as "the branch of epidemiology that studies the social distribution and social determinants of states of health (3).

Social epidemiology is that branch of epidemiology concerned with the way that social structure, institution and relationships influences health. It means social epidemiology attempts to search social -cultural factor behind the disease, for example if somebody suffering from cancer it may be due to smoking, drinking or any other reason. If a person is drinking too much alcohol it may be due to fashion or enjoyment. On the other hand, it can also be due to depression or alienation. It may be that his family environment is like this. Social epidemiology explores these reasons. Epidemiology search about the cause of disease but social epidemiology tries to find out why person is drinking alcohol or why he is taking any intoxication, what type of environment or culture is responsible for this.

The major premise of social epidemiology is that each society forms its own distribution of health and disease (3).

In other words, social epidemiology assumes that distribution of advantages and disadvantages in a society directly or indirectly influenced the distribution of health and disease in the society. There are many socio-cultural factors which affect the distribution of health, illness and disease in a society, for example social class, gender, community, religion, race, ethnicity, discrimination, social network, income distribution and social policy etc. All these factors and along with these factors why and how these all affect individual and population health comes in the study scope of social epidemiology.

Man is a biological and social animal, there is an influence of family, social environment behind his behavior as well as his

every act or behavior has an effect on family and social environment. At the same time his social environment also affects his biological life. Thus, it should be clear that core of social epidemiology is "biological expression of social inequality". Examples include biological expression of poverty and of diverse type of discrimination, for example, based on race/ethnicity, gender, sexuality, social class, disability, or age. Whether these biological expressions of social inequality are interpreted as expression of innate verses imposed, or individual verses societal, characteristic in part is shaped by the very social inequalities patterning population health (4)(5).

Now we are going to discuss major approaches of Social epidemiology

These concepts are not limited to Social epidemiology alone but they are totally applicable to modern epidemiology as well.

1. Bio-Psychosocial Perspective:

Social epidemiology uses the bio-psychosocial perspective in contrast, modern epidemiology often uses the biological paradigm(6)-(8).

The biological paradigm assumes that behind all disease there are biological factors. It guides the views that a population is merely the sum of its individuals, and that the population pattern of diseases is simply a reflection of individual risk factors. Consequently, social level factors cannot be considered "real" causes of disease, if the biological paradigm is used(9)(10). In contrast, the biopsychosocial paradigm of social epidemiology assumes that the biology of organisms is determined in multilevel, interactive environments (7)(8).

It means disease are the products of mutual interaction of individual factors, biological factors, and social factors. When social factors combined with biological and individual factors, it becomes more harmful to human health.

2. Population Perspective

Population perspective is another significant concept in the field of social epidemiology. Geoffrey Rose pointed out that an individual's risk of disease cannot be isolated from the disease risk of the population to which s/he belongs (11). It means individual risk factor is related to largerpopulation. For example, we can predict that a person living in slum area is more likely to suffering from disease like malaria, dengue and chikungunya compared to someone living in higher society. This prediction is reasonable not only because any given person in slum area may have more confronted with mosquitoes but also because that area is mostly not clean and hygienic as compared to higher society because of this population distribution of mosquito in slum area is higher than civilized area. We know that life expectancy in the area where poorest people's lives is less than the area of rich people because of their life style and better treatment.

3. Multilevel Analysis

A third important concept in the field of social epidemiology is the use of new statistical approaches, such as multilevel analysis (6), to determine the effect of sociostructural factors on health (12).

Multilevel analysis allows several levels of analysis to be announced for simultaneously and more effectively than in conventional multivariate analysis (13). If guided by well-developed conceptual models clearly specifying which variables are to be studied at which level. (14).

These analysis can potentially assess whether individual's health is shaped by not only "individual" or "household" characteristics (for example, individual or household income) but also "population" or "area" characteristics; the latter may be "compositional" (for example, proportion of people living in poverty) or "contextual" (irreducible to the individual level, for example, income distribution, population density, or absence of facilities, such as super markets, libraries, or health centres)(15)(16).

4. Ecosocial Perspective

Ecosocial theory is an emerging multilevel theory of disease distribution that seeks to integrate social and biological reasoning, along with a dynamic, historical, and ecological perspective, to address population distribution of disease and social inequalities in health. This theory invites consideration of how population health is generated by social conditions necessarily engaging with biological process at every spatiotemporal scale, whether from subcellular to global, or nanoseconds to millenniums. (4)

The key contracts of ecosocial theory are: (17)

1-Embodiment, 2-Pathways to embodiment, 3-the cumulative interplay of exposure, susceptibility, and resistance 4-agency and accountability. All these constructs work together and must be understood in order to assess the impact of multiple levels of

influence on the distribution of disease in populations.

5. Social production of disease/political economy of health

Social production of disease/ political economy of health perspective focus on economic and political determinants of health and distribution of disease within and across societies, including structural barriers to people living healthy lives (4).

These theories accordingly focus on economic and political institutions and decisions that create, enforce, and perpetuate economic and social privilege and inequality are root- or 'fundamental'(18)- cause of social inequalities in health.

Conclusion

Social epidemiology is a branch of epidemiology that attempts to explain that a human being is a biological as well as social animal, so the society, culture and environment in which he/she lives has a profound effect on his/her health. It focuses particularly on the effects of social-structural factors on states of health and distribution of disease in a population.

This science suggest that a person needs a healthy and pleasant environment to be perfectly healthy. A social epidemiological view should be taken into account to promote

Population health.

References

- 1. Durkheim E (1996), Suicide, free press, New York.
- 2. Succer M. Causal thinking in the health sciences: concepts and strategies in epidemiology. New York: Oxford Press, 1973.
- 3. Berkman, L F, Kawachi I. A Historical framework for social epidemiology. In: Berkman L F, Kawachi I, eds. Social Epidemiology. New York; Oxford University Press, 2000:3-12.
- 4. Krieger N. Emerging theories for social epidemiology in the 21st century: an ecosocial perspective. *Int J Epidemiol* (in press).
- 5. Berkman L, Kawachi IKrieger N (2000) Discrimination and health. In Social epidemiology. Eds Berkman L, Kawachi I(Oxford University Press, Oxford), pp 36-75.
- 6. Pearce N. Traditional epidemiology, modern epidemiology, and public health. *Am J Public Health* 1996; 86: 678-683.
- 7. Susser M, Susser E. Choosing a future for epidemiology: I. Eras and paradigms. *Am J Public Health* 1996; 86: 668-673.
- 8. Susser M, Susser E. Choosing a future for epidemiology: II. From Black box to Chinese boxes and eco-epidemiology. *Am J Public Health* 1996; 86: 668-673.
- 9. Krieger N. Epidemiology and the web of causation: has anyone seen the spider? Soc Sci Med 1994; 39: 887-903.
- 10. Rothman K. Modern epidemiology. Boston: Little, Brown & Co., 1986.
- 11. Rose G. The strategy of preventive medicine. Oxford: Oxford University Press, 1992.
- 12. Macintyre S, Ellaway A. Ecological approaches: rediscovering the role of physical and social environment. In: Berkman LF, Kawachi I, editors. Social epidemiology. New York: Oxford University press, 2000: 332-348.
- 13. Kariya T. Japanese public education diplomaism and myth of equality in Japan's postwar history. Tokyo: Chuoukoronsya,1995
- 14. Blalock HM, Jr (1984) Contextual-effects models: theoretic and methodologic issues. *Annu Review Social* 10:353-372.
- 15. Diez-Roux AV (1998) Bringing context back into epidemiology: variables and fallacies in multilevel analysis. *Am J Public Health* 88:216-222.
- 16. Berkman I, Kawachi Macintyre S, Ellaway A (2000) Ecological approaches: rediscovering the role of the physical and social environment. In Social epidemiology. eds Berkman L, Kawachi I (Oxford University Press, Oxford), pp 332-348.
- 17. Krieger, Nancy (2011). Epidemiology and the People's Health: Theory and Context. Oxford University Press. ISBN 9780199750351.
- 18. Link BG, Phelan JC. Editorial: understanding sociodemographic differences in health -the role of fundamental social causes. *Am J Public Health* 1996;86:471-73.

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FIRST AID

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Definition: It is Define as the first and emergency treatment given to the patient who suffering from either a minor or serious accident or unexpected illness while waiting for the doctor to come is known as first Aid.

Aim of First Aid:

- To preserve life of the injured person.
- Promote recovery.
- Pain relief.
- Protect the unconscious.
- Prevent from further harm.
- Reduce the anxiety of the victim.

First Aid Kit: It is the collection of basic drug and equipment that is used to give basic medical treatment when any person is ill or injured.

Basic content of First Aid:

- Surgical mask
- Adhesive bandages.
- Plastic tweezers.
- Disposable gloves.
- Sphygmomanometer
- Stethoscope.
- Cotton balls.
- Cotton swabs.
- Iodine.
- Gauze piece.
- Dressing material.
- Adhesive tape.
- Scissors.
- Tweezers.
- Syringe.
- Torch.
- Thermometer.
- Safety pin.

- Eye pads.
- Hydrogen peroxide.
- Antiseptic solution.
- Alcohol/non alcohol antiseptic solution.

Some basic emergency drugs:

- Aspirin (for chest pain and heart attack).
- Diphenhydramine (for anaphylactic shock).
- Paracetamol, naproxen, carpal, amoxicillin, morphine ibuprofen codeine (pain killer).
- Loperamide, diphenoxylate, cholestyramine, Pepto-Bismol (for diarrhea).
- Charcoal, dextropropoxyphene (for poison treatment).
- Burn gel, antifungal cream, antihistamine, calamine lotion, Betamethasone valerate ointment.

Important of first aid:

- Providing quick medical treatment until Doctor arrives.
- It help to ensure that the right methods of administering medical assistance are provide.
- Knowledge of First aid also benefits the individuals themselves.
- It affords people with the ability to provide help during various emergency situation.

First aid treatment of shock:

Shock (Circulatory shock)

Definition:

Shock is the condition in which not enough flow of blood to the tissues of the body as a result problem caused in circulatory system is known as shock.

Sign & Symptom of Shock:

- Weakness
- Nausea.
- Dry mouth.
- Blue red vision.
- Blueness of lips.
- Paleness of skin.
- Cold & clamy skin.
- Anxiety
- Difficulty in breathing.
- Fast pulse rate.
- Sweating.
- Increased thirst
- Temporary loss of consciousness.

Emergency treatment of shock:

- Victim should be placed in highly ventilated area.
- Tight cloth should be loose.
- Patient is covered with a blanket.

- Be learn that don't give any thing orally.
- Patient should allowed to lay flat on the flour to improve blood circulation to the brain.
- If there is difficulty in breathing then the patient head & chest should be elevated.
- Carry him & hospitalized immediately.

First aid treatment of snake bite

Snake

Snake are elongated, legless, carnivorous reptiles. These are ectothermic amniotes vertebrate covered in overlapping scale, they are found in land as well as aquatic region.

Snake Bite

Snake bite is a kind of injury that is caused by the bite of snakes especially a venomous snake .a common sign of a bite from venomous snake is presence of 2 puncture wounds from the animals who have fangs

There are 3 types of families of venomous snake are:

- Elapidae: kraits, cobras & mambas
- Hydrophidae: sea snake or coral reef snakes
- Viperidae

Sign and symptom of snake bite:

- If a snake is poisonous then 2 fangs sign will appear without scratches.
- If a snake is non poisonous then 2 fangs sign will not appear only scratches are found.
- Usually there is a burning pain, redness, swelling at the site of bite area.
- Skin colour change at the site of bite area.
- Blurred vision, swelling, sweating, nausea, vomiting, tingling of the limbs.

First aid treatment:

- Victim should be place in ventilated area
- Firm pressure bandaging of the bite area.
- Don't move the injured part.
- With the help of sterile knife 1cm length and half centimeter depth incision will be made and squiceezed out poisonous blood from the site of bite area.
- When you treat the victim be sure that victim will not sleep, better to give coffee or tea.
- If there is insufficient breathing then give artificial breathing.
- Wash the wound with soap and water to remove any more poison on the skin.
- After washing the wound give intravenous or intramuscular administration of the antivenomes.
- When victim is feel better, then carry it and hospitalized.

First aid treatment of burn

Burn

Burn is a type of damage of person skin or deeper tissues which is caused by sun, heat, fire, electricity, chemicals, friction or radiation.

Types of burn:

There are six types of burn are:

- Friction burn
- Cold burn
- Thermal burn
- Electricity burn

- Radiation burn
- Chemical burn

History of burn

History of burn can help the Doctor to treat the patients who is suffering from burn and also try to recover the patient life.

The following component of patient are involve to known the history of burn are:

- Nature of burn
- Duration of burn
- Previous treatment
- Tetanus immunization
- A) Nature of burn: it is Define as the agent which are responsible for burn & environment in which burn occur.
- B) Duration of burn: it Denote the time elapsed after sustaining the burn injuries which affect the development of sign and general condition of the patient.
- C) Previous treatment: it can play very important role to treat any patient in acute case and also help the doctor to treat future line of treatment of the patient.
- D) Tetanus immunization: In the case burn tetanus immunization can help to kill the infection and provide protection against tetanus.

Symptom of burn:

- Blister
- Pain
- Peeling skin
- Red skin
- Swelling
- White or charred skin
- Itching
- Spot appear
- Numbness

Diagnosis

Nine rule of Burn

It is derived by Pulaski and Tennison in 1947 & published by Alexander burn Wallace in 1951. It is a type of tool which is used in hospital to determine and measurement of depth of burn & surface area of burn.

Table of nine rule of burn

Body part Estimated of body surface area			
	Adult	Children	
Entire left arm	9%	9%	
Entire right arm	9%	9%	
Entire head	9%	18%	
Entire chest	9%	9%	
Entire abdomen	9%	9%	
Entire back	18%	18%	
Entire left leg	18%	13.5%	
Entire right leg	18%	13.5%	
Groin	1%	1%	

First aid treatment

For minor burn:

- Hold burn skin under cool running water until the pain subside.
- Cover with sterile non adhesive bandage or clean cloth.
- Avoid breaking blister.
- Apply moisturizing lotion.
- Give over an counter pain reliever if necessary such as acetaminophen, ibuprofen or naproxen etc.
- If develop of any symptom of an infection immediately contact your doctor.

For major burn:

- Call emergency number 108 medical care.
- Cover the burn area with sterile non stick bandage.
- Separate burn toes and finger with dry sterile dressing.
- Be learn that do not soak burn in water in major burn treatment.
- Immediately contact your doctor and get start treatment of the burn.

Emergency treatment of poisoning:

Poisoning It is also known as Envenomation. The branch of medicine that deals with the detection & treatment of poison is known astoxicology.

Poisoning is the condition in which an organism becomes harmed by a toxic substance or venom of animals.

Some basic harmful Causative agent that are responsible form poisoning are:

- Nail polish remover
- detergent
- paint thinner
- pesticides
- lead, mercury, illegal drug
- venom
- spoiled food
- carbon monoxide gas

ypes of poisoning

There are type two of poisoning:

- Acute poisoning
- Chronic poisoning
 - A) **Acute poisoning:** it is Define as exposure to a poison during a short period of time is known as acute poisoning. In acute poisoning a contrast substance can destroy tissue but not absorb in the body.
 - B) **Chronic poisoning:** it is Define as exposure to a poison during a long period of time and the symptom do not occur immediately and Patient Become ill after a long period of time.

Sign or symptom of poisoning

Sign or symptom of poisoning is based on the factor of the poisoning. Some common symptom to aware of poisoning such as:

- Dizziness
- Disorientation
- Vomiting
- Shortness of breathing
- Pain

- Burning
- Headache
- Blue lips & skin
- Loss of consciousness
- Coma in severe cases
- Felling faint
- Skin rashes

Treatment of poisoning

- Call emergency number 108 medical care.
- If the patient will not breathing then immediately give artificial breathing.
- If the casualty is conscious but shows signs of burning around the mouth then send him immediately to hospital don't make vomit.
- If the patient is conscious but show no sign of burning around his mouth then make him vomit.
- For making vomiting give that patient to eat charcoal powder, 2 tablespoon of salt in cup of warm water, antidote substance of poison.
- After he has vomited give him at least a liter of water, milk, tea or coffee.
- At last give that patient confident and suggested him to never do poisoning again.

Table of Some Basic Antidote of Poison

Antidote name	Poison name	Recommend
Acetylcysteine	Acetaminophen toxicity.	20% oral solution-7 x 30ml, 20% IV Solution -4x30ml.
Atropine	Cholinesterase inhibitor toxicity.	1g.
Calcium disodium EDTA	Lead, Copper, zinc, cobalt, cadmium toxicity.	18 x 5ml, 200mg/ml
Activated charcoal powder	Use to treat Most poison.	1 tablespoon
Dimercaprol / BAL	Arsenic, lead, mercury toxicity.	6 x 3ml, 100mg/ml
Diphenhydrmine	Acute dystonia; histamine receptor blockade.	8 x1ml, 50mg/ml
Folic acid (Folvite TM)	Methanol toxicity.	6 x10ml, 5mg/ml
Naloxone (Narcan TM)	Opioid toxicity.	3 x 10ml, 1mg/ml
Vitamin B1	Wernicke-korsakoff syndrome, ethylene glycol toxicity.	5 x 2ml, 100mg/ml

First aid treatment of heart attack

Heart attack (Myocardial Infarction)

A heart attack is the condition in which flow of blood and oxygen is stop due to blockage of coronary artery and these blockage occur when fat, cholesterol and other substance are deposit on the wall of coronary artery as a result plaque formation take place and after over time these plaque can become damage and release platelet.

These platelet are responsible for clotting of blood and these clotting of blood become deposit on the wall of artery and become blockage it

Types of heart attack:

There are three types of heart attack are:

- STEMI ST Segment Elevation Myocardial Infarction.
- NSTEMI Non ST Segment Elevation Myocardial Infarction.
- Coronary spasm or unstable angina.

Symptom of heart attack:

Some common Symptom of Heart Attack are:

- Chest pain
- Coughing
- Nausea
- Vomiting
- Dizziness
- Shortness of breathing (dyspnea)
- Face seeming gray in colour
- Felling of terror that life is ending
- Felling clammy and sweating Warning sign of heart attack
- Pressure, squeezing, fullness in the chest
- Pain
- Sudden shortness of breathing
- Cold
- Sweating
- Sick
- Nausea felling

Factor causing heart attack

There are several factor that cause heart attack are:

- Age
- Tobacco
- High blood pressure
- High blood cholesterol level
- Obesity
- Diabetes
- Metabolic syndrome
- Stress

Diagnosis of heart attack:

- by Electrocardiogram (ECG)
- By Blood test
- By Chest X-ray
- By CT Scan or MRI Test
 - Treatment of heart attack:
- Call emergency number 108 medical care.

- Give aspirin as well as nitroglycerin to the patient immediately.
- if the patient under unconscious condition then immediately give C.P.R Technique (cardiopulmonary resuscitation technique) until doctor arrive.

Emergency drug of heart attack:

- Aspirin
- Thrombolytic drug (Clot buster)
- Anti platelet agent
- Morphine
- Nitroglycerin
- Beta blocker
- Statins

Surgical prevention of heart attack:

- Coronary angioplasty.
- Coronary by pass surgery.

Prevention of heart attack

- Lifestyle change
- Not smoking
- Eat healthy diet
- Get regular exercise
- Keep diabetes under control
- Maintain blood cholesterol level

Dressing

Dressing is a sterile pad applied to a wound to promote healing & protect the wound from further harm.

Types of dressing: There are several types of dressing are:

- Gauze sponge dressing
- Gauze bandage roll dressing
- Non Adherent pads dressing
- Non-Adherent wet dressing
- Foam dressing
- Hydro gel dressing
- Transparent dressing

Classification of dressing: Dressing are classified are as follows:

- Primary dressing
- Secondary dressing
- Passive dressing
- Active dressing
- Interactive dressing
 - A) Primary dressing: it is also known as contact layer which come direct contact of wound ex-Band aid
 - B) Secondary dressing:it placed over primary dressing to provide increased protection. Ex- self adhesive elastic bandage.

- C) Passive dressing: for protection, as protective dressing.
- D) Active dressing: promotes healing through the creation of moist wound environment.
- E) Interactive dressing: not only creates a moist wound environment but interact with the wound bed component to further enhance wound healing. It can reduces bacterial colonization count in wound bed & remove cellular debris.

Some basic Material use in dressing:

- Apron
- Medical gloves
- Disposable face mask
- Alcohol pads
- Cotton balls
- Cotton tipped applicators
- Gauze sponge
- Non-woven sponges
- Dressing & bandages
- Medical tape
- Ointments & medicines
- Suture removal kits
- Gauze rolls
- Medical drapes
- Scissors
- Forceps

Procedure of dressing:

- Position the patient comfortably.
- Wash hand thoroughly.
- Put on gloves mask etc as necessary.
- Open the sterile tray. Spread the sterile towel around the wound.
- Pickup the dissecting forceps & remove the dressing & put it in the paper bag. Discard the dissecting forceps in the bowl of lotion.
- Note the type & amount of drainage present.
- Ask the assistant to our small amount of cleansing solution into the bowl.
- Clean the wound from center to periphery, discarding the used swab after each stroke.
- After cleaning dry the wound with dry swab.
- Apply medication if ordered.
- Apply sterile dressings. Gauze piece 1st then cotton pads.
- Remove the gloves and discard it.
- Secure the dressing with bandage or tapes.

Aim of dressing:

- To protect the wound from mechanical injury.
- To protect the wound from microbial contamination.
- To provide or maintain moisture of the wound.

• Reduce physiological stress of patient by obscuring the wound.

References

- 1. Bhardwaj Kr Lokesh, Iglasia Kumar Vimal (2019) Health education and community pharmacy (series) 1st edition published by R. Narain publishers & distributors page No-53.
- 2. Parmar N.S. (1995) Health education and community pharmacy
- a. (book) 1st edition published by Satish Kumar Jainand produced by Varun Jain for CBS Publishers and distributors Pvt. Ltd page No-264.
- 3. https://en.m.wikipedia.org/wiki/first aid
- 4. https://en.m.wikipedia.org/wiki/Dressing (medical)
- 5. https://www.slideshare.net/mobile/sunilvish123/dressing-90435208
- 6. https://poisoncontrol.utah.edu/healthpros/antidotes-stock.php
- 7. https://en.m.wikipedia.org/wiki/Myocardial_infarction
- 8. https://en.m.wikipedia.org/wiki/burn

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ONLINE EDUCATION CHALLENGES AND STRATEGIES DURING COVID-19

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Adaptation is a law if nature. As humans we learn, we adapt and we keep moving. Due to COVID-19 outbreak the schools are closed and there is no option but to adapt and utilize online education.

Among the 1.26 billion children worldwide, India comprises over 320 million of learners. It is still uncertain when they can return to their school and colleges. The pandemic has also significantly disrupted the higher education sector. A large number of Indian students enroll in universities abroad and these countries are worst affected by the Pandemic. A decline in the demand for international higher education is expected if the situation persists in the long run.

Parents, teacher and administrators are now coming online to ensure staying at home doesn't come at the cost of students losing their studies. The world economic forum reports a surge in the use of language apps, virtual tutoring, video conferencing tools and online learning software in the last three months.

India too is witnessing an e-learning boom. The transition has been smooth for most private institutions, the public institutions are still adapting. Students especially the under privileged ones who live in places with poor connectivity do not have access to online facility. Faculty members are giving lectures through the use of recorded classes or online on Google Meet, Zoom etc. The note and assignment would be posted in their WhatsApp Class Group.

The abrupt transition to online hardly compensates for the absence of the classroom experiences. Students find it very boring. There is increased screen time for children which are now inevitable. Few parents are not comfortable with technology themselves. At times classes are disturbed due to internet issues.

Teaching institution require high speed internet and learning management systems, stable IT infrastructure and faculty members who are comfortable teaching online. Students also need high speed internet and computers or mobiles to attend these sessions or watch pre-recorded classes.

There is a substantial learning that is lost when education goes online. Teachers, who have built rapport with the children over a period, by observing students in class, just see them on computer screens. The greatest advantage of face to face teaching is eye contact with which it is easy to guage if students are following the lessons. Education is about interaction, broadening of ideas, free flowing open discussions, debates and mentoring of each student. There is an excitement present in the real classroom which is lost when education goes online.

Real classrooms are great spaces for young citizens to interact with each other across the lines of diversity, get along, have fun and engage in academic pursuits. Online education should only be a supplement.

References:

1. "Full coverage on Corona Virus" in Business Standard Online.