JOURNAL OF
HYGIENE SCIENCES
Committed to the advancement of Clinical & Industrial Disinfection & Microbiology
VOLUME - XIII ISSUE - V DEC 2020 - JAN 2021

Editorial

Contents

'Mini Review' section - The elementary identification of medically important genera of the family Enterobacteriaceae based on few basic biochemical tests was discussed in previous issue. In this article, elementary identification of Enterobacteriaceae based on genomic level/ molecular level techniques available is elucidated.
Few of the commercially available identification systems offered are serotyping and molecular typing techniques. Laboratories should follow manufacturer's instructions and rapid tests and kits should be validated and be shown to be fit for purpose prior to use.

Current Trends section - Sterilization, as a specific discipline, has been with us for approximately 120 years, since the invention of the steam autoclave by Charles Chamberland in 1879. Sterilization processes cannot be considered in isolation; rather, they are inextricably related to the product to be sterilized. There is no single sterilization process that is suitable for all medical products. The diversity in sterilization processes and of operating systems within each process has arisen as a consequence of the efforts made to optimize medical sterilization and to meet the differing needs imposed by the vast range of products to be sterilized.

In Profile Scientist – **Suniti Solomon** was an Indian physician and microbiologist who pioneered AIDS research and prevention in India after having diagnosed the first Indian AIDS cases among the Chennai sex workers in 1986 along with her student Nirmala Sellappan . She founded the Y R Gaitonde Centre for AIDS Research and Education in Chennai. The Indian government conferred the National Women Bio-scientist Award on her. On 25 January 2017, Government of India announced "Padma Shri" award for her contribution towards Medicine.

Bug of the month – **"*Mycobacterium ulcerans*** "The organism belongs to the family of bacteria that causes tuberculosis and leprosy. However, *M. ulcerans* is an environmental bacterium and the mode of transmission to humans remains unknown. *Mycobacterium ulcerans* grows at temperatures between 29–33 °C and a low 2.5% oxygen concentration to grow. The organism produces a unique toxin – mycolactone – which causes tissue damage and inhibits the immune response.

Did You Know? Laxatives are a type of medication used to treat constipation by loosening stool or encouraging bowel movements. If you try to use laxatives for weight loss, you may well see the number on the scale go down. But this apparent drop is deceiving because it's actually water weight that you're losing. The weight loss is temporary and is not actually changing your body fat composition.

Best Practices - Health care workers who use or may be exposed to needles are at increased risk of needlestick injury. Such injuries can lead to serious or fatal infections with bloodborne pathogens. These injuries can be avoided by eliminating the unnecessary use of needles, using devices with safety features, and promoting education and safe work practices for handling needles and related systems. These measures should be part of a comprehensive program to prevent the transmission of bloodborne pathogens.

Ease your mind with the light humour in our Relaxed Mood section…….

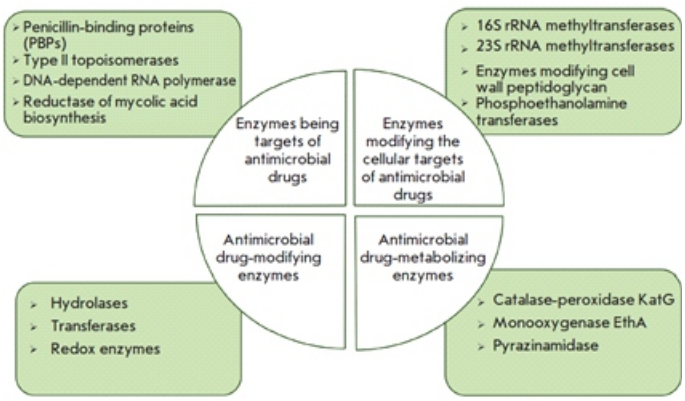
So go on, enjoy reading & don't forget to give us your valuable inputs & feedback.

Bacterial Enzymes and Antimicrobial Resistance II

Emergence of antibiotic resistant pathogenic bacteria poses a serious public health challenge worldwide. In the last sections of this review, origins of resistance determinants in clinical strains and potential mechanisms for their mobilization are discussed. The role of bacterial enzymes in resistance development is rather versatile and involves several key mechanisms.

This review presents data on the functional features of the main classes and groups of the bacterial enzymes involved in the implementation of the mechanisms of bacterial resistance to AMDs, one of which was already described in last review (i.e. **I.** BACTERIAL ENZYMES AS THE TARGETS OF AMDs).

II. BACTERIAL ENZYMES MODIFYING THE CELL TARGETS OF AMDs



- **rRNA methyltransferases**

Bacterial ribosomes act as targets for many AMDs. The small 30S subunit consists of 16S rRNA and 21 proteins. Aminoglycosides bind to the 30S subunit to yield hydrogen bonds with the nitrogenous bases of several nucleotides of 16S rRNA, which prevents proper binding of aminoacyl-tRNA to the anticodon and leads to protein synthesis errors and subsequent cell death (*Fig. 2A*). Some aminoglycosides can directly inhibit the initiation or block the elongation of the polypeptide chain.

One of the mechanisms of resistance to aminoglycosides is methylation of the A-site of 16S rRNA by bacterial 16S rRNA methyltransferases that results in a loss of the ability to bind to the ribosome by antibiotics.

Macrolides, ketolides, lincosamides, and streptogramin B (MKLS group according to the name of its components) are targeted at the large 50S subunit of the ribosome containing 5S and 23S rRNA and 33 ribosomal proteins. Despite the differences in their structure, these antibiotics have a common binding site with the 50S subunit in close proximity to the peptidyl transferase center. Meanwhile, they close the ribosomal tunnel, the structural element located in the large ribosomal subunit. This interaction results in dissociation of peptidyl-tRNA from the ribosome, which leads to translocation disruption and termination of protein synthesis (Fig. 2B).

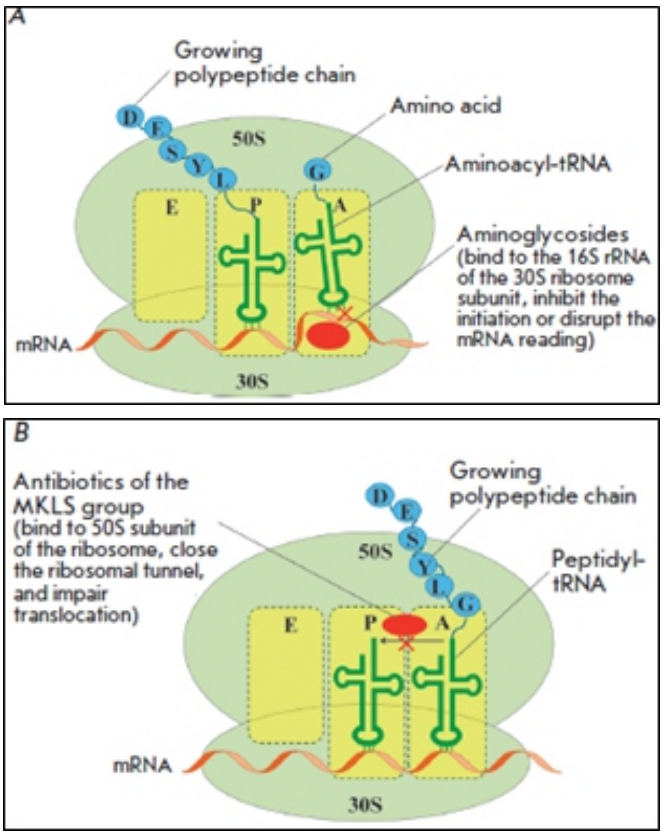


FIG NO. 2: Binding of aminoglycosides (A) and antibiotics of the MKLS group (B) to the ribosome and their effect on protein synthesis.

One of the mechanisms of resistance to MKLS drugs is the production of 23S rRNA methyltransferases, which catalyze the post-transcriptional modification of 23S rRNA that consists in methylation of A2058 located in the site of antibiotic binding to the ribosome. Thirty-nine genes encoding 23S rRNA methyltransferase have been described, mainly in Gram-positive microorganisms.

- **Enzymes involved in the modification of peptidoglycan in the bacterial cell wall**

Resistance of Gram-positive bacteria to glycopeptide antibiotics (Vancomycin and Teicoplanin) is caused by the production of enzymes (dihydrogenase, serine racemase, ligase) catalyzing peptidoglycan modification. These antibiotics are high-molecular-weight compounds consisting of glycosylated cyclic or polycyclic peptides. They form a complex with *D*-Ala–*D*-Ala peptidoglycan terminal dipeptide, which is stable thanks to the formation of five hydrogen bonds. Furthermore, these antibiotics prevent the transglycosylation and transpeptidation reactions in the cell membrane. Resistance to them is caused by substitution of the last amino acid residue *D*-Ala of peptidoglycan for *D*-Lac or *D*-Ser, which reduces the affinity of the terminal dipeptide for the antibiotic (by three orders of magnitude for *D*-Ala-*D*-Lac and by two orders of magnitude for *D*-Ala-*D*-Ser). Nine operons responsible for the resistance of enterococci to glycopeptide antibiotics have been detected. The determinants of resistance to

glycopeptide antibiotics often localize in plasmids but can also be found in the chromosome.

- **Phosphoethanolamine transferases**

Polymyxins (colistin) are targeted at the lipopolysaccharides of the outer membrane of Gram-negative bacteria. The main constituent of these AMDs is the positively charged cyclic polypeptide, whose mechanism of action is similar to that of cationic detergents. Interaction between polymyxin molecules and the negatively charged phosphate groups of lipopolysaccharides neutralizes the membrane charge and changes membrane permeability for the intra- and extracellular components. The main mechanism of resistance to polymyxins is associated with closure of the channel of antibiotic penetration into the cell. This channel is closed via the modification of lipid A (the component of lipopolysaccharides) with phosphoethanolamine, which is catalyzed by phosphoethanol amine transferase (*Fig. 3*). The gene encoding this enzyme has chromosomal localization. The *mcr-1* gene has recently been detected on plasmids. The development of this type of resistance is associated with mutations in phosphoethanolamine transferase genes.

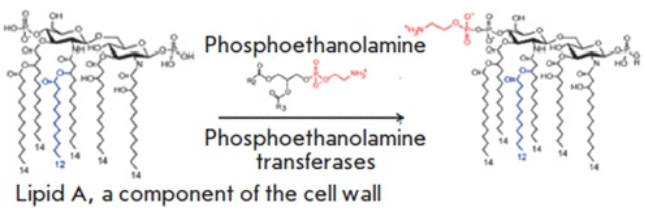


FIG NO. 3: Scheme of modification of lipid A, a component of lipopolysaccharides of the outer cell membrane, by phosphoethanolamine transferase

III. BACTERIAL ENZYMES MODIFYING AMDS

Destruction or modification of the antibiotic structure is one of the most common mechanisms of resistance involving enzymes. Depending on the type of reactions they catalyze, the enzymes involved in this resistance mechanism are subdivided into hydrolases, transferases, and oxidoreductases (*Fig. 4*).

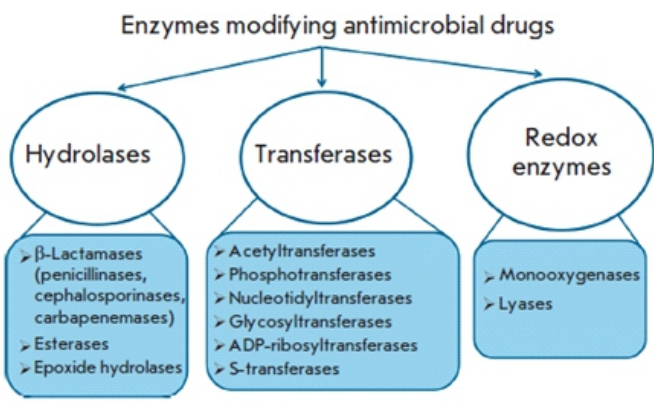


FIG NO. 4: The main classes of enzymes modifying antimicrobial drugs

- **Hydrolases**

β-Lactamase and macrolide esterases destroying β-lactams and macrolides, respectively, are the most common enzymes catalyzing antibiotic hydrolysis. The same mechanism is responsible for the resistance to Fosfomycin and chloramphenicol.

β-Lactamases

β-Lactamases hydrolyze the amide bond in the β-lactam ring, the common structural element of all β-lactam antibiotics (penicillins, cephalosporins, carbapenems, and monobactams). They form an enzyme superfamily that currently consists of more than 2,000 members. According to the homology of amino acid sequences, β-lactamases are subdivided into four molecular classes. The enzymes of classes A, C, and D are serine hydrolases, the enzymes of class B are metalloenzymes.

Serine β-lactamases have structural elements similar to those of the C-domain of PBPs, which indicates that they are evolutionarily related. The evolution of β-lactamase develops via two main mechanisms: the emergence of new mutations in the genes of known enzymes and the emergence of enzymes with a new structure. The high mutation rate of β-lactamases and the localization of their genes on mobile genetic elements contribute to the rapid spread of resistant bacteria, which poses a global threat. Bacteria simultaneously carrying up to eight β-lactamase genes have been detected.

Class A β-lactamases (CTX-M, TEM, SHV, and KPC lactamases) are the most common ones. Mutational variability is a feature of TEM and SHV β-lactamases. The key mutations in the active site increase the enzyme volume and make it capable of hydrolyzing the bulk molecules of cephalosporins of the second-to-fourth generations.

In clinical practice, combinations of β-lactams with clavulanic acid, sulbactam, and tazobactam (which contain a β-lactam ring, form a more stable acyl-enzyme complex and have a low deacylation rate) are intensively used to inhibit class A enzymes. The newest inhibitors that are structurally similar to β-lactams but contain no β-lactam ring include diazabicyclooctanes.

Transferases

Transferases modifying AMD molecules by covalently binding various chemical groups represent a large superfamily of enzymes. Their main groups, differing in terms of substrate specificity, type of modification are given below.

1. Aminoglycoside-modifying enzymes

Enzymatic modification of aminoglycoside antibiotics is the most common resistance mechanism that is implemented by aminoglycoside-modifying enzymes (AMEs). Several hundred different AME are known; almost each enzyme is represented by several isoenzymes that possess unique substrate specificity and modify aminoglycosides at certain positions. AME genes localize in mobile genetic elements; that is why they rapidly spread.

2. Enzymes modifying chloramphenicol and its analogues

Production of chloramphenicol acetyltransferases (CATs) is the main mechanism of bacterial resistance to chloramphenicol. These enzymes catalyze the addition of the acetyl group of acetyl-CoA to the 3-hydroxyl group of chloramphenicol or its synthetic analogues (thiamphenicol, azidamphenicol), thereby preventing the binding of the antibiotic molecule to ribosomes. CATs do not inactivate fluorophenicol, since the 3-hydroxyl group in its molecule is replaced with a fluorine atom. CATs of different types have extremely low homology of amino acid sequences, which does not exceed 10%. The *cat* genes can be located on chromosomes but are more typically localized on plasmids as components of transposons in association with genes encoding resistance to other AMDs. Expression of the *cat* genes is induced by chloramphenicol.

3. Enzymes modifying MKLS antibiotics

Macrolide phosphotransferases (MPHs) are enzymes that modify the structure of macrolides by adding a phosphate group to the 2'-OH group. The phosphate group is donated by nucleoside triphosphates, most typically by GTP. Seven different enzymes of this group have been described so far. MPHA preferably catalyzes the phosphorylation of 14- and 15-membered macrolides, while MPHB modifies 14- and 16-membered macrolides. The genes encoding MPH are located on mobile genetic elements containing other genes encoding resistance to macrolides and other antibiotic classes. Expression of the genes coding for macrolide phosphotransferases can be either inducible (*mphA*) or constitutive (*mphB*).

Macrolide glycosyltransferases are enzymes that inactivate macrolides by glycosylating the 2'-OH group of the macrolide ring. They use UDP glucose as a cofactor.

Streptogramin acetyltransferases inactivate only streptogramins A by acetylation of an unbound hydroxyl group; their mechanism of action is similar to that of CAT. The genes encoding these enzymes have been identified in a number of Gram-positive pathogens, including Staphylococci and Enterococci.

4. Fosfomycin-modifying enzymes

FosA, FosB, and FosX epoxidases, as well as FomA and FomB kinases, are metalloenzymes that inactivate phosphomycin. Epoxidases open the epoxy group of Fosfomycin (the oxirane ring) by adding various substrates. FosA is glutathione-S-transferase that uses Mn2+ and K+ metal ions as cofactors, besides glutathione. Bacillithiol or *L*-Cys acts as a source of the thiol group in FosB; additionally, these enzymes use Mg2+ as a cofactor. The FosX enzyme is a Mn2+-dependent hydrolase. Most of the genes encoding these enzymes localize on the plasmid, although FosA in *P. aeruginosa* and FosB in *S. aureus* are encoded by chromosomal genes.

FomA and FomB kinases add one or two phosphate groups to the Fosfomycin molecule, using ATP and Mg2+ ions as cofactors. These enzymes are isolated from fosfomycin producer *S. wedriensis*.

5. Rifamycin-modifying enzymes

Several groups of enzymes inactivate rifamycins by modifying the hydroxyl group, the key group involved in the binding of an antibiotic molecule to the β-subunit of RNA polymerase. NAD+-dependent enzymes belonging to the Arr group catalyze ADP-ribosylation, RPH kinases catalyze phosphorylation, and glycosyltransferases catalyze glycosylation.

IV. Enzymes of metabolic processes modifying AMD in the prodrug form

Antibiotics can also be modified by the enzymes that protect cells against toxic molecules. In most cases, prodrug forms of AMDs are modified to the active forms.

Isoniazid is activated by KatG catalase-peroxidase, giving rise to free radicals of isonicotinic acid, which block the enzymes involved in the synthesis of mycolic acids. Resistance is caused by mutations in the *katG* gene, which are most often localized in codon 315 and cause conformational changes in the isoniazid-binding pocket.

Structural analogues of isoniazid, ethionamide and prothionamide, are activated by NADPH-dependent FAD-containing monooxygenase encoded by the *ethA* gene. The oxidized forms of ethionamide and prothionamide in a complex with NAD+ inhibit the enzymes responsible for the synthesis of mycolic acids (primarily InhA), similar to the case of isoniazid. Expression of the *ethA* gene is regulated by the transcriptional repressor EthR. Resistance is caused by mutations in the *ethA* and *ethR* genes.

CONCLUSIONS

The question regarding the origin of the bacterial enzymes responsible for resistance development during evolution remains controversial. The genes encoding these enzymes are located on chromosomes and mobile elements. The enzymes encoded by chromosomal genes protect microorganisms producing antibiotics against modification of their potential targets. Resistance occurs when the genes coding for these enzymes are transferred to other bacteria.

Another group of enzymes encoded by chromosomal genes has evolved from enzymes belonging to superfamilies with isolation of subgroups with altered substrate specificity. Enzymes that perform vital functions and are responsible for the biosynthesis of cell wall polysaccharides, proteins, nucleic acids, and metabolites serve as targets for antibiotics. Modification of the active sites of target enzymes contributed to their ability to use antibiotics as substrates. The presence of proto-resistance genes causing the evolutionary relationship between β-lactamases and PBP, kinases and acetyltransferases, with aminoglycoside-modifying enzymes, has been established.

Many enzymes have originated from bacterial pro-enzymes that used to have other functions. Mutations in the genes encoding enzymes emerged due to exogenous and endogenous factors (in particular, antibiotics and products of their metabolism). These mutations changed the structure, catalytic properties, and substrate specificity of these products. The multiplicity of mutations indicates that both the key and accompanying amino acid residues undergo mutations. The key amino acid residues are important for catalytic processes, while changes in the accompanying residues compensate for structural changes and function as allosteric sites of activity regulation.

The multidirectionality of the processes is a feature typical of bacterial resistance. Combination of several resistance mechanisms in a single cell (e.g., modification of structural cellular elements, changes in the expression level of proteins, including porins, and activation of efflux systems) complicates the development of methods for suppressing resistance. The scientific concept of combining objects related to the most important biological processes into certain groups has emerged in recent years. Thus, the concept of "microbiome" as a combination of microorganisms of a certain species and humans appeared. Non-pathogenic microorganisms, and soil bacteria in particular, represent a huge reservoir and source of resistance genes. Their wide distribution among microorganisms is associated with localization on plasmids and other mobile genetic elements and a high rate of exchange and transmission between bacterial cells, including pathogenic strains.

The combination of the genes responsible for the resistance of pathogenic clinical strains and non-pathogenic bacteria in the environment and microbiota is known as the "resistome." Its important feature is that the genome of a single bacterium carries several resistance genes that ensure multiresistance. Bacterial cells can rapidly reproduce, change their gene structure, and undergo selection; so, they have developed new mechanisms ensuring cell survival. Enzymes with various functions play the most important role in these processes. The term "enzystome" can be used to refer to the enzyme-based defense system that has developed throughout the long-term evolution of bacteria.

MATERNAL MORTALITY: UNDERLYING FACTORS

The extent of maternal mortality is an indicator of disparity and inequity in access to appropriate health care and nutrition services throughout a lifetime, and particularly during pregnancy and childbirth. Every day, 1500 women die from pregnancy- or childbirth-related complications. In 2005, there were an estimated 536,000 maternal deaths worldwide. Most of these deaths occurred in developing countries, and most were avoidable. Extensive efforts have been made since 1987 to describe the extent and etiology of maternal mortality. Maternal deaths take place for two reasons: a direct obstetric death which is caused by complication that develops directly as a result of pregnancy, delivery or the postpartum period; an indirect obstetric death which is due to existing medical conditions that are made worse by delivery or pregnancy.

India is one of the few countries in the world where women and men have nearly the same life expectancy at birth. The fact that the typical female advantage in life expectancy is not seen in India suggests there are systematic problems with women's health. Indian women have high mortality rates, particularly during childhood and in their reproductive years due to various social and economic issues related to health and society in general. The health of Indian women is intrinsically linked to their status in society. Research on women's status has found that the contributions Indian women make to families often are overlooked, and instead they are viewed as economic burdens. There is a strong son preference in India, as sons are expected to care for parents as they age. This son preference, along with high dowry costs for daughters, sometimes results in the mistreatment of daughters.

Further, Indian women have low levels of both education and formal labor force participation. They typically have little autonomy, living under the control of first their fathers, then their husbands, and finally their sons. All of these factors exert a negative impact on the health status of Indian women.

Poor health has repercussions not only for women but also their families. Women in poor health are more likely to give birth to low weight infants. They also are less likely to be able to provide food and adequate care for their children. Finally, a woman's health affects the household economic well-being, as a woman in poor health will be less productive in the labor force.

Many social traditions like child marriage have lead to women becoming more and more prone to health related problems. Marriage and child bearing at a very early age results in the already weak female child to becoming more weak and stressed with the burden of carrying not only for her infant but also her extended family. Pregnancy and abortions are related to many complications that may not seem so obvious in the beginning but eventually show effect.

Causes of Maternal Mortality

Women die from a wide range of complications in pregnancy, childbirth or the postpartum period. Most of these complications develop because of their pregnant status and some because pregnancy aggravated an existing disease. The four major killers are: severe bleeding (mostly bleeding postpartum), infections (also mostly soon after delivery), hypertensive disorders in pregnancy (eclampsia) and obstructed labour. Globally, about 80% of maternal deaths are due to these causes. Among the indirect causes (20%) of maternal death are diseases that complicate pregnancy or are aggravated by pregnancy, such as malaria, anaemia and HIV. Women also die because of poor health at conception and a lack of adequate care needed for the healthy outcome of the pregnancy for themselves and their babies. As stated by the 2005 WHO report following are the main reasons of maternal mortality:

- Severe bleeding/hemorrhage (25%)
- Infections (15%)
- Unsafe abortions (13%)
- Eclampsia (12%)
- Obstructed labour (8%)
- Unsafe abortions (13%)
- Other direct causes (8%)
- Indirect causes (20%)

Infection is estimated to be the second highest cause of underreported maternal death. Obstetric infection accounts for more than 12% of maternal deaths. Despite the widespread application of standard aseptic techniques during vaginal birth, cesarean birth, and/or termination of pregnancy, post-pregnancy infections remain a significant source of maternal morbidity and mortality. Infection occurs most frequently in women who have caesarean births, and following spontaneous or elective termination of pregnancy.

Maternal mortality is a small but persistent aspect of induced abortion. Causes of maternal death that arise specifically from abortions include hemorrhage (ante partum or post partum), eclampsia, pre-eclampsia, obstructed and prolonged labor, infection, complications of abortion, disorders related to high blood pressure, anemia, ectopic pregnancy, and cardiomyopathy. The identification of medical and demographic risk factors may have significant implications creating initiatives aimed at decreasing the public health burden associated with maternal mortality.

Pregnancy related causes of maternal deaths

Maternal mortality and morbidity are two health concerns that are related to high levels of fertility. The high levels of maternal mortality are especially distressing because the majority of these deaths could be prevented if women had adequate health services. There are several underlying factors that need to be looked at intrinsically in order to understand the existing problems.

Few pregnant women receive prenatal care

Prenatal care and health care is very important for the health of the woman as well as her fetus. Essential and differential testing can indicate if there are any deficiencies that the woman is suffering from and thus after a differential diagnosis adequate treatment and health care can be provided in order to cause minimal or no birth and congenital defects that may be a direct or indirect cause of deficiency. Thus it is necessary that women should be

adequately educated to ensure healthy pregnancies, sound knowledge of using beneficial health care and thus ensure safe childbirths.

Majority of births in India take place at home

Place of birth and type of assistance during birth have an impact on maternal health and mortality. Births that take place in non-hygienic conditions or births that are not attended by trained medical personnel are more likely to have negative outcomes for both the mother and the child. While health care is important, there are several other factors that influence maternal mortality and health. Medical research shows that early age at first birth and high numbers of total pregnancies take their toll on a woman's health.

Anemia

Anemia (referred to as low levels of red blood cells or alternatively low levels of hemoglobin, which is the molecule that carries oxygen, or may also be low levels of iron in blood) accounts for one in five maternal deaths, is related to an easily treated problem.

Anemia is one of the major causes of maternal mortality in India. It is noted painfully that after years of independence India leads iron deficiency anemia cases in the world and more than 90% of Indian women, adolescent girls and children are anemic. Everyone is aware of the fact that anemia results in physical weakness, mental shortcomings, low intelligence and increased vulnerability to a number of diseases and causes adverse pregnancy outcomes and even death of expectant mother. Anemic mothers also bear anemic children. In none of the states were services for anemia included as a component of antenatal care. Data from Rapid Household Survey indicated that even iron folic acid consumption is still very low. Only 22.3% of pregnant women consume Iron and Folic Acid supplementation for 90 days and the percentage is less than 10% among the non-educated women compared to 50% among the well-educated. Also the disparity between rural and urban areas is significant (18% and 34.5% respectively).

Anemia can be treated relatively simply and inexpensively with iron tablets. Severe anemia accounts for 20 percent of all maternal deaths in India and can also increase the chance of dying from a hemorrhage during labor.

Unhygienic conditions or practices

Unhygienic conditions and several practices that are carried out during gestation and consecutively during deliveries can endanger the life of the mother. Unhygienic conditions which is often a consequence of poor monitoring during delivery or of untreated sexually transmitted diseases (STDs), accounts for some 15% of maternal deaths. Infections can be effectively prevented by careful attention to clean delivery and by detection and management of STDs during pregnancy. Systematic postpartum care will ensure rapid detection of infection and its management by appropriate antibiotics.

Sepsis

Sepsis, a very severe infection – is one of the most frequent cause of maternal death. It can be eliminated if aseptic techniques are respected and if early signs of infection are recognized and treated in a timely manner. Another major cause of maternal deaths, due to infections may arise from unsafe abortions, anaemia and improper care during pregnancy. Women who do not eat nutritious food during pregnancies are susceptible to infection. In rural India this is one of the commonest causes of maternal deaths.

Toxemia

Toxemia another cause of maternal mortality, also may be a result of unhygienic conditions and malpractices, however, refers to the presence of toxin in the human system, which can not only be life threatening, but in certain instances be fatal and hence even claim a woman's life.

Other causes include

Eclampsia:

There are various other causes of maternal mortality. Eclampsia is one of them, which is a fallout of pregnancy-induced hypertension. This usually happens due to improper antenatal care. Hypertension during the course of pregnancy can ultimately culminate in convulsions. Eclampsia if not treated with care in time may lead to the death of the mother.

Hemorrhage:

Another reason of maternal death is Hemorrhage. This may once again be caused by poor antenatal care, anemia during pregnancies or during operative deliveries.

Obstructed or prolonged labor

This occurs when the fetus does not deliver in the anticipated time. This may be due to the wrong position of the fetus, if it is a too large a baby or if the pelvis of the mother is narrow. In urban India, obstructed labor is generally not among the primary causes of maternal deaths anymore but in rural India, due to lack of interest in institutional delivery it is still a cause of maternal deaths.

Intermediate causes

They include the low social status of women, lack of awareness and knowledge at the household level, inadequate resources to seek care, and poor access to quality health care. Other causes are untimely diagnosis and treatment, poor skills and training of care providers, and prolonged waiting time at the facility due to lack of trained personnel, equipment and blood. The other prominent dark chapters of our society are the early age of marriage and child bearing, child spacing, family size and fertility patterns, literacy, socio-economic status and social customs and beliefs.

Reproductive factors

The risk of a woman dying in pregnancy and childbirth depends on the number of pregnancies she has in her lifetime. The higher the number of pregnancies the greater the lifetime risk of pregnancy related death. Maternal mortality rates are also higher among very young women, those aged 35 years and older and those with four or more children.

Socio–economic and cultural factors

The ability of women to command resources and make independent decisions about their fertility, their health and health care also has an impact on maternal mortality. Where women are

afforded a low status in society their health needs are often neglected, and existing health facilities may not be accessed by the women in need. Additionally lack of education and understanding around health related issues can contribute to delays in seeking care when it is needed or to the inappropriate management of life threatening pregnancy complications.

- Woman's age: The optimal child bearing age is from 20 to 30 years. There is a gradual increase in the risk of maternal mortality < 20 years and >30 years. Pregnancies outside the age mentioned can be complicated and may have deleterious effects on the child as well as the mother.
- Parity: Parity means the number of children. The higher the parity, the higher will be the chances of maternal mortality during or after partum.
- Birth interval: There is an increased risk of maternal mortality with short birth intervals. When the interval between pregnancies and child birth is not adequate, there are also chances that the child may be weak and more prone to infections.
- Poor socioeconomic status: This would mean that there is no proper maternal nutrition, this too can be dangerous for the mother as well as her unborn child.
- Bad cultural practices and beliefs.
- Nutritional status, for instance malnutrition.
- Environmental factors like poor sanitary conditions: May make the mother sick and hence the fetus may also get affected in certain cases.
- Lack of maternity services.
- Shortage of manpower in the health sector.
- Poor communication and transport facilities: In cases of emergencies, communication and transportation facilities are indispensible.

Time to death for most common obstetric emergencies:

Cause of Death	Time to death
Postpartum hemorrhage	2 hrs
Antepartum hemorrhage	12 hrs
Ruptured uterus	1 day
Eclampsia (severe hypertensive disorder of pregnancy)	2 days
Obstructed labor	3 days
Infection	6 days

Antenatal and intra partum care must contain the following features:

- Early registration of pregnancy (12 - 16 weeks);
- Minimum three Ante-Natal Check-ups;
- Screening all pregnant women for major health, nutritional and obstetric problems;
- Identification of women with health problems/complications, providing prompt and effective treatment including referral wherever required;
- Universal coverage of all pregnant women with tetanus toxoid (TT) immunization and malaria prophylaxis;
- Delivery in a very clean environment;
- Institutional delivery for women with bad obstetric history and high risk factors;
- Training of traditional birth attendants and female health care workers;
- Promotion of family planning;
- Prevention of complications such as eclampsia, malpresentations and ruptured uterus;
- Screening for anemia and providing Iron-Folic Acid (IFA) tablets to prevent anemia;
- Advice on food, nutrition and rest;
- Promotion of institutional delivery / Safe deliveries by trained personnel etc.

The main problem areas of antenatal check ups lie herewith:

- Inadequate coverage; lack of trained health personnel in antenatal screening, risk identification and referral services;
- Over crowding in PHCs/hospitals;
- Lack of Emergency Obstetric services etc.

One of the major goals of Government of India's Department of Health and Family Welfare is to reduce maternal mortality and morbidity. The focus has shifted from individualized interventions to attention to the reproductive health care, which includes skilled attendance at birth, operationalizing Referral Units and 24 hours delivery services at Primary Health Centers.

Role of the Government

The challenge for the government however is to help direct and improve privately provided services through appropriate regulatory arrangements and by encouraging an expansion of their scope to include promotion and prevention, in addition to curative care.

The link between pregnancy-related care and maternal mortality is well established. National programmes and plans have already stressed on the need for universal screening of pregnant women and operationalising essential and emergency obstetric care. Focused antenatal care, birth preparedness and complication readiness, skilled attendance at birth, and access to emergency obstetric care are factors that can help reduce maternal mortality.

The mind boggling high maternal mortality rate in India can be reduced by following the strategies enumerated below:

- Effective initiative from the government is required in terms of proper allocation of resources to all the health institutions specially Primary Health Centers. Even more important is to ensure that the funds actually reach the users whenever it is needed.
- Early registration of antenatal cases and effective health education of couples to make them understand the importance of antenatal check ups, hospital deliveries and small family norms.
- Local dais / birth attendants and female health workers should be imparted periodic training to update themselves with improved techniques and be incorporated as an integral part of health care system. The importance of observing proper aseptic measures while conducting deliveries should be emphasized to them.
- Prevention and early treatment of infection, ante partum and postpartum hemorrhage.
- Wide spread availability / supply of Iron – Folic acid tablets and nutritious food to the poor and remotest corners of the country.

- Treatment of illnesses like diabetes, tuberculosis and malaria during pregnancy should be ensured.
- Construction of better roads and transport facilities is required especially in the rural areas and urban slums to make the health care facilities more available and accessible to people in need.
- Providing facilities for hospital deliveries for high risk cases like severe anaemia, hypertension, diabetes and heart disease.

Women lying in the high risk groups should be given adequate care and proper treatment to prevent fatalities.

Treatment must be immediate and sustained with oxytocic drugs and plasma expanders; the means of referral to an equipped facility must be available to women with hemorrhage. Risk factors for obstructed labor include very young age, height below 145 cms, previous prolonged labor or stillbirth, and previous cesarean, abnormal presentation, or labor progression. Delivery for these women must be in a facility offering trained doctors and well-equipped operating rooms. Prevention of infection is possible with pre-sterilized delivery kits, antibiotics in kits or within facilities, cleanliness of hands and delivery areas, and maternal tetanus immunization. Identification of edema in pregnancy would prevent eclampsia. Abortion complications could be prevented with safe and early practices and women's control over fertility.

Though there are a number of factors that play a key role in maternal mortality, in developing countries infections also attribute to deaths, these infections can be prevented by using basic measures such as the following:

- Deliveries should be handled at proper health care facilities under the guidance of professional obstetrics and trained medical staff and also all the emergency equipment that may be required in cases of emergencies.
- All the devices that are used during deliveries or abortions have to be sterile, so that there is no transmission of infectious particles to the mother and or the fetus.
- The hands of all the attendants and the place where the delivery is undertaken have to be essentially monitored and maintained under stringent aseptic conditions, again, in order to minimize infections both for the mother and the child.
- Also vaccines that are to be administered during gestation have to be taken, to prevent infections like tetanus at bay which can lead to death of mothers pre and post-partum.

Disinfection

Disinfection and sterilization are essential for ensuring that medical and surgical instruments do not transmit infectious pathogens to patients. Several antiseptic agents are available for hand hygiene, skin antisepsis, surface and instrument disinfection. The antiseptic used should be able to perform adequate antisepsis & disinfection. Traditional antiseptics score low in this point due to resistance development, low bioburden tolerance and cytotoxicity. They also should be non-irritating and non-staining. It should also not be malodourous. The traditional alcohol-containing products and iodophors (povidone-iodine) are the most commonly used agents. None of these antiseptics are completely safe and effective.

Alcohols

- Lacks a sustained residual effect on the skin.
- Irritant and toxic to tissue cells, therefore, are unsuitable for application on mucous membranes.

Iodophors (povidone-iodine)

- Have a minimal residual effect.
- Get deactivated in the presence of organic matter and body fluids.
- Not recommended in neonates, particularly pre-term infants.
- Not recommended for thyroid patients.
- May be toxic to tissues.
- Allergy/hypersensitivity is possible.

Modern disinfectants such as Polyhexamethylene biguanidine (PHMB) is an excellent choice. PHMB a polymeric bioguanidine, is a broad spectrum cationic surface active antimicrobial agent. It is also one of the multipurpose antimicrobial agents that can be used for skin, surface and instrument disinfection.

Benefits of PHMB:

Chemically stable & non volatile

- PHMB has very low surface activity, having a surface tension essentially identical to water, & consequently can be readily water rinsed from surfaces & do not have residual streaks or tackiness.
- Odorless, non foaming, clear & colorless.
- Easily handled & applied.
- Effective & stable over a wide pH range (4-10).

Unique biguanide chemistry

- Novel non specific mode of action.
- No known evidence of development of organism resistance.

Broad spectrum of activity

- High activity against Pseudomonas, MRSA, VRE, food borne pathogenic organism, viruses & so on.
- Retains activity in presence of organic matter.

Safe antiseptic

- Not cytotoxic to human cells.
- No skin sensitization/irritation.
- Can be used for neonates and thyroid patients.

Mode of action of PHMB

1. Rapid action towards the bacterial surface.
2. Binding to a receptive site on the surface.
3. Overcoming bacterial defense mechanism.
4. Attraction towards the cytoplasmic membrane.
5. Leakage of low molecular weight cytoplasmic components and inhibition of membrane bound enzymes.
6. Extensive disruption of cytoplasmic membranes and leakage of macromolecular components.
7. Precipitation of cell contents and cell death.

Advantages of PHMB over the classically used disinfectants such as povidone iodine

1. Unlike povidone iodine, is not inactivated in the presence of organic matter.
2. Unlike povidone iodine, PHMB is not cytotoxic.
3. Unlike povidone iodine, PHMB can be used for thyroid patients.
4. PHMB is resistance free.
5. PHMB is not affected by sunlight, water, temperature and pH fluctuations. This stability allows PHMB as a better antimicrobial agent.
6. PHMB has Low acute toxicity via dermal & oral route.
7. Low skin & eye irritation potential at in-use concentration.
8. Low toxicity following long term exposure.
9. Not teratogenic & shows no reproductive effects when studied over two generations.
10. Non genotoxic in range of studies.
11. Not considered carcinogenic in humans.

Interventions and Solutions to reduce Maternal Mortality

The persistence of a high maternal mortality rate (MMR) despite half a century of efforts to bring it down indicates that somehow India has not been able to establish appropriate maternal health services especially in the rural areas. An improved, accountable health care system at primary level is essential for decreasing maternal mortality to the desired level. For the same, the following has to be implemented:

1) Make the antenatal, intranatal and postnatal services accessible to women;
2) Ensure delivery by skilled attendant nurses or doctors;
3) Improve hygienic conditions;
4) Provide better parental care.

In conclusion it can be said that, a maternal death is often not only the result of technical incompetence or negligence, but is also caused by ineffective health system and limited knowledge, social attitudes, poor health and midwife practices by the family and community itself.

The health of mother is directly related to her child's health; and without due attention to the causes behind high maternal mortality ratios, we are simply ignoring an important determinant of the health of our nation. In doing so, maybe we are running the risk of damaging our chances for an all-encompassing prosperity in future.

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https://www.who.int/

Dr. Harriette Chick

She was raised in a time where women's participation in the sciences was generally frowned upon. Nonetheless she took it in her stride, and in 1894 enrolled as a science student at University College London. After graduation, she stayed on to gain a doctorate in bacteriology, working part of the time in Vienna and in Munich where she became familiar with spoken German.

In 1905, she became the first woman to obtain a position at the Lister Institute of Preventive Medicine in London. This was the only medical research institute in Britain at that time and had been established in 1891 as a private charity.

In her role of Assistant she worked directly with Charles Martin in his laboratory. Her first assignment was to develop better methods for measuring the relative biological potency of different disinfectants, about which there had been considerable confusion. Next her research involved investigating whether "inactivation" of microorganisms could be explained in terms of the denaturation of their proteins. She then worked with Martin on a project related to his studies on the recent outbreaks of bubonic plague in India.

With the outbreak of World War I in August 1914, the whole staff volunteered its services to the Army Medical Department. In 1916, Martin was in the Middle East working at a military hospital and saw cases of both scurvy and beriberi that he believed resulted from the restricted range of foodstuffs in the army rations. The following research concluded that for the prevention of beriberi, good additions to army rations would be whole-grain cereals, pulses, dried eggs, and yeast, but not milk or cheese. For the prevention of scurvy, where fresh vegetables and fruits were not available, they recommended fresh meat and freshly germinated pulses. She also was the first to discover that the highest content of thiamine was present in the branny layers of rice grain, and that its processing was responsible for diminishing the vitamin content of rice and subsequently the diet, leading to beriberi.

She is also best remembered for demonstrating the roles of cod liver oil and sunlight in preventing rickets (Vitamin D deficiency associated) in the Vienna program.

She held many positions of responsibility over the years. In 1918 she was elected to the Physiological Society. She served as Secretary of the Accessory Food Factors Committee of the Medical Research Council from 1918–1945. She was appointed CBE in 1932 and subsequently DBE in 1949. She also served as secretary of the League of Nations health section committee on the physiological bases of nutrition from 1934 to 1937. She was also a founding member of the Nutrition Society.

Jokes

Teacher: "Kids, what does the chicken give you?"
Student: "Meat!"
Teacher: "Very good! Now what does the pig give you?"
Student: "Bacon!"
Teacher: "Great! And what does the fat cow give you?"
Student: "Homework!"

♦♦♦

A 3 years old boy sits near a pregnant woman.
Boy: Why do you look so fat?
Pregnant woman: I have a baby inside me.
Boy: Is it a good baby?
Pregnant woman: Yes, it is a very good baby.
Boy: Then why did you eat it?

♦♦♦

A Police Officer Jumps Into His Squad Car And Calls The Station.
"I Have An Interesting Case Here," He Says.
"A Woman Shot Her Husband For Stepping On The Floor She Just Mopped."
"Have You Arrested Her?" Asks The Sergeant.
"No, Not Yet. The Floor's Still Wet."

♦♦♦

Teacher: Can Anyone Give Me An Example Of Coincidence?
Sunny: Sir, My Mother And Father Got Married On The Same Day Same Time.
Teacher: How Old Is Ur Father.
Sunny: As Old As I Am.
Teacher: How Is It Possible?
Sunny: He Became Father Only After I Was Born.

♦♦♦

An elderly couple are in church. The wife leans over and whispers to her husband, "I just let out a long, silent fart. What should I do?" The husband replies, "First off, replace the batteries in your hearing aid!"

♦♦♦

A: I Have The Perfect Son.
B: Does He Smoke?
A: No, He Doesn't.
B: Does He Drink Whiskey?
A: No, He Doesn't.
B: Does He Ever Come Home Late?
A: No, He Doesn't.
B: I Guess You Really Do Have The Perfect Son. How Old Is He?
A: He Will Be Six Months Old Next Wednesday.

♦♦♦

Morganella morganii could be an important Intensive Care Unit pathogen

Critically ill patients admitted in Intensive Care Units (ICU) are predisposed to many nosocomial infections due to underlying illnesses, various invasive procedures and prolonged hospital stay. The spread and treatment of multidrug resistant organisms, such as Methicillin Resistant *Staphylococcus aureus* (MRSA), Vancomycin Resistant Enterococci (VRE), Extended Spectrum β Lactamase (ESBL) producing organisms of family Enterobacteriaceae and Metallo β Lactamase (MBL) producing *Pseudomonas aeruginosa*, can be a very challenging task. One such pathogen, which is often ignored but is of clinical significance is, *Morganella morganii*.

M. morganii belongs to the tribe Proteeae of family Enterobacteriaceae. Despite its wide distribution, it was considered as an uncommon cause of infections in human beings. However, it was suggested that *M. morganii* may become an important opportunistic nosocomial pathogen in the future by William *et al* way back in 1983 when bacteremia cases due to the organism had been recorded in a cardiac surgery unit. Since then, there have been various reports of this organism causing urinary tract infections, skin and soft tissue infections, meningitis and bacteremia often with fatal consequences. In India, sporadic cases due to infection with *M. morganii* have been reported from time to time. A case was reported from our own center, in which *M. morganii* was isolated from a diabetes mellitus patient with septic arthritis. Another study from India has reported *M. morganii* as an important uropathogen, especially amongst indoor patients.

Over a period of 6 months (January 2009-June 2009), in a mini cluster outbreak of its kind, we isolated *M. morganii* 15 times from the urine samples of 10 of ICU patients (repeated isolation four times from one patient and two times from another two patients). The male:female ratio of these 10 patients was 9:1. One patient was above 80 years of age while maximum (six patients) belonged to 40-80 years with three patients less than 40 years. The clinical diagnoses were as varied as ileal perforation, cord compression, C3-C4 fixation, occipito-parietal concussion, fracture pelvis, coronary artery disease, pulmonary fat embolism with respiratory distress, etc. However, all the patients had urinary catheters inserted and had undergone one or the other invasive procedure (four patients had undergone recent surgery and seven patients had central venous catheters inserted and were on total parenteral nutrition). Three patients presented with polymicrobial urinary infection (two with *Candida* species and one with *Klebsiella pneumoniae* along with *M. morganii*). Three patients died ultimately due to causes other than infection with *M. morganii*. No case of bacteremia due to *M. morganii* was reported. All the strains isolated had same resistogram; they were resistant to augmentin, gentamicin, amikacin, nalidixic acid, norfloxacin, cefoperazone/sulbactam, piperacillin/tazobactam, and imipenem. Only one strain was sensitive to cefoperazone/sulbactam and imipenem. All the patients were administered combination drugs along with imipenem and they responded clinically with the subsequent urine cultures showing no growth of *M. morganii*.

Although any infective site can serve as the source of bacteremia, the urinary tract accounts for the majority of cases. Another study on bacteremia patients has reported urinary tract (37%) and biliary tract (22%) infections to be the major portals of entry for *Morganella* in the blood stream, suggesting the need for antibiotic coverage for *M. morganii* in debilitated patients. Further, this problem is compounded by the fact that virtually all *Morganella* species are capable of producing inducible chromosomal Amp C β lactamases rendering them resistant to action of primary and extended spectrum penicillins and cephalosporins. Admission of patients to the ICU or in such compromised situation warrants that organisms like *M. morganii* with proven pathogenicity could be potentially dangerous and should not be overlooked. Such infections rather need to be vigorously treated, as inappropriate antibiotic therapy combined with intrinsic resistance of *M. morganii* itself becomes an independent risk factor in high risk patients.

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Older woman with Type-II Diabetes use less oxygenated blood in their brain

A University of Houston researcher is reporting that the brains of older women with Type 2 diabetes do not use as much oxygenated blood as those who don't have the disease. The research is the first to point to changes in blood use in the brain as the primary reason for diabetes-related deficits in motor function. It also furthers the understanding of sensory and motor symptoms as a precursor to developing dementia and Alzheimer's diseases, both of which are linked to diabetes.

"It's a pretty significant finding. Typically, when someone presents with a sensory or motor issue along with Type 2 diabetes mellitus, the assumption is that it's the result of peripheral nerve damage in the hands and feet," said Stacey Gorniak, associate professor in the UH Department of Health and Human Performance and director of the Center for Neuromotor and Biomechanics Research. Gorniak published her findings in the journal Neurophotonics.

Until now there has been no assumption that something is going on with respect to brain function that is affecting sensory and motor functions in persons living with Type 2 diabetes.

"Emerging evidence has suggested that factors outside of nerve damage due to Type 2 diabetes mellitus, such as impaired cortical blood use, contribute significantly to both sensory and motor deficits in people with diabetes," reports Gorniak.

Nearly 24% of the 40 million people in the United States over the age of 60 live with Type 2 diabetes. Problems with hands, fingers and feet are common side effects of the disease and can lead to a loss of independent living and decline in quality of life.

Gorniak's testing method is unique. Rather than using a typical MRI to monitor the use of oxygenated blood, she opted to use a technique called functional near infrared spectroscopy (fNIRS). The fNIRS is a method that delivers infrared light into the scalp to measure use of both oxygenated and unoxygenated blood use by the brain. This technique differs from MRI as MRI cannot measure oxygenated blood use. The fNIRS method can be used on persons who cannot have an MRI.

She tested a group of 42 post-menopausal women, over 60, half of whom had Type 2 diabetes, and asked them to perform various exercises with their hands. She chose this group because they are generally at the highest risk for diabetes, heart disease and dementia.

"Our work demonstrates that motor changes in people with diabetes occur independent of sensory impairment and that these changes are unrelated to disease duration and severity. Our data point towards other factors such as changes in muscle and reduced function of the cortex as underlying mechanisms for problems in sensory and motor functions," Gorniak reports.

Her findings, she said, opens research possibilities for other groups of people with the disease, in hopes of finding a way to therapeutically avoid the negative health effects of diabetes.

“We need to see what this looks like in a larger population, including men, and then we can start developing treatments or different ways we could potentially stop these negative impacts of Type 2 diabetes," said Gorniak.

PROPER DIET – HEALTHY FOOD INTAKES

"Health is Wealth" is the common phrase that we have heard people using and have ourselves used so very often that we may never have enough of it.

Well then, the point here would be that we have to act in the direction so as to keep ourselves as healthy as possible. Having meat, fish, vegetables, fruits, pulses, oils is not enough, there is a need to know the proper time and manner in which we should consume them. Improper consumption of all the healthy stuff would do no good, on the contrary just add bulk to the intake and further strain the digestive system.

In a bid to make our diet more healthier, we neglect the underlying point to 'think before we act.'

There are plenty of foods that help us have a healthy sustenance; Importance of such foods is mentioned in the table below:

Food	Aids in	Nutrients present
Walnut	Lowering cholesterol, combating cancer, boosting memory, protection against heart diseases	Omega 3 fatty acids, manganese, copper, tryptophan, etc.
Figs	Weight loss, lowering cholesterol, controlling palpitation	Dietary fiber, potassium, manganese, etc.
Fish	Protection against heart disease, boosting memory, fighting obesity, support to the immune system	Tryptophan, vitamins B3, B6, B12, D, omega 3 fatty acids, phosphorus, manganese, protein, etc.
Prawn	Fighting knee and joint pains, improving mood, reducing depression	Tryptophan, omega 3 fatty acids, protein, vitamins B3 and D, minerals, etc.
Olives	Protection of the heart, promotion of weight loss, battling diabetes, fighting body pains	Iron, vitamin E, dietary fiber, copper, etc.
Peanut	Promoting a healthy heart, reducing blood pressure, reducing body weight	Manganese, tryptophan, vitamin B3, folate, copper and protein
Oats	Lowering blood cholesterol, stabilizing blood sugar, prevention of hair fall	Minerals, tryptophan, vitamin B1, dietary fiber, protein, etc.
Apricots	Controlling blood pressure, protecting eyes, slowing aging	
Drumstick	Reducing blood pressure, controlling diabetes, curing asthma and TB	Protein, minerals, vitamins, etc.
Coriander	Improving respiratory health, intestinal health, lowering LDL, increasing HDL, improves digestion	Dietary fiber, minerals, etc.
Cucumber	Improving digestion, facial treatments	Vitamins A, C, minerals, dietary fiber, tryptophan, magnesium, folate, potassium, etc.
Carrot	Lowering cholesterol, fighting obesity, combating diabetes, protecting liver	Minerals, vitamins, dietary fiber, folate, etc.
Soyabean	Staying lean, lowering blood pressure, stabilizing blood sugar, fighting diabetes	Minerals, vitamins, proteins, dietary fiber, omega 3 fatty acids, copper, etc.
Pomegranate	Blood thinning, combating cancer, dental protection, protecting arteries	Minerals, vitamin B complex, vitamin C, etc.
Basil	Protection against chronic disease, enhancing memory, blood purification	Minerals, vitamins, dietary fiber, etc.

The type of lifestyle we have greatly influences the ideal food intake for us, there are some trivial facts about certain foods that we should know:

For instance a certain food contains 50 calories, but the body burns 120 calories to actually digest this food. Examples of such foods include walnuts, figs, soyabean, olives, fish, peanuts, ginger, carrot, cucumber, garlic, papaya, spinach, amla, asparagus, apples, beets, berries, broccoli, cabbage, cauliflower, celery, pineapple, lettuce, onion, lemon, turnip, grapefruit and orange are among the foods that are advisable to folks looking out for a healthy weight loss regime.

However if you want to gain some weight, it is best to eat less of the above and eat other foods that have high calorie levels.

Vitamins and Minerals and the appropriate intake

Vitamin	Daily Dose	Used for
A (Beta carotene)	10,000 IU	An antioxidant used for skin, eyes, teeth and bones.
B complex		All B vitamins are taken as a group, hence, B complex.
B1 (Thiamine)	50 mg	For nerveous system, body growth and body metabolism.
B2 (Riboflavin)	50 mg	Improves in the formation of red blood cells and antibodies and for metabolism.
B3 (Niacin)	100 mg	Improves in maintaining good skin and digestive system.
B5 (Pantothenic acid)	100 mcg	Helps with stress, improves in the release of energy from fats and carbohydrates.
B6 (Pyridoxine)	50 mcg	Helps balance sodium & phosphorus. Improves in formation of antibodies.
B12 (Cyanocobalamin)	200 mcg	Improves in formation of bloodcells, helps metabolism and nervous system.
Biotin	200 mcg	Improves in utilization of other vitamins.
Choline	100 mg	Helps in nerve transmission, liver and gall bladder functions.
Dietary fiber	20 – 30 gms	Lowering cholesterol, preventin cancer, constipation, helping weight loss.
Folic Acid	400 mcg	Improves the brain function and for normal cell division.
Inositol	150 mg	Is a necessary component for hair growth.
C (Ascorbic Acid)	2,000 mg	An antioxidant, heals wounds, tissue, bone repair, helps resist infection.
D	400 IU	Required for the body to absorb calcium & phosphorus. Helps nervous system.
E	500 IU	Prevents cancer and cardiovascular disease. An antioxidant helps blood clotting.
K	100 mcg	Necessary for normal blood clotting.
Bioflavinoids	400 mcg	Helps strengthen capillaries and improves in the absorption of vitamin C.
Coenzyme Q10	25 mg	Improves in the effectiveness of the immune system.

Mineral	Daily Dose	Used for
Calcium	1,500 mg	For bones & teeth, nervous system & muscle action.
Chromium	100 mcg	Increases effectiveness of insulin, used in metabolism.
Copper	2 mg	Formation of blood cells, works with vitamin C in healing process.
Iodine	130 mcg	Helps regulate metabolism.
Iron	18 mg	Used in the production of blood, works in the immune system.
Magnesium	400 mg	Acts as a catalyst in utilization of carbohydrates, fat, protein & other minerals.
Manganese	3 mg	For skeletal development & sex hormone production.
Molybdenum	25 mcg	Helps iron transport from liver, promotes normal cell function.
Potassium	200 mg	Necessary for heart muscle function, kidneys & nervous system.
Selenium	200 mcg	Works with vitamin E to promote antibodies. Keeps tissue and artery elasticity.
Tryptophan	3 grams	An amino acid that brings feelings of calm, relaxation, confident and sleepiness.
Zinc	25 mg	Improves in healing process, used by prostrate gland & immune system.

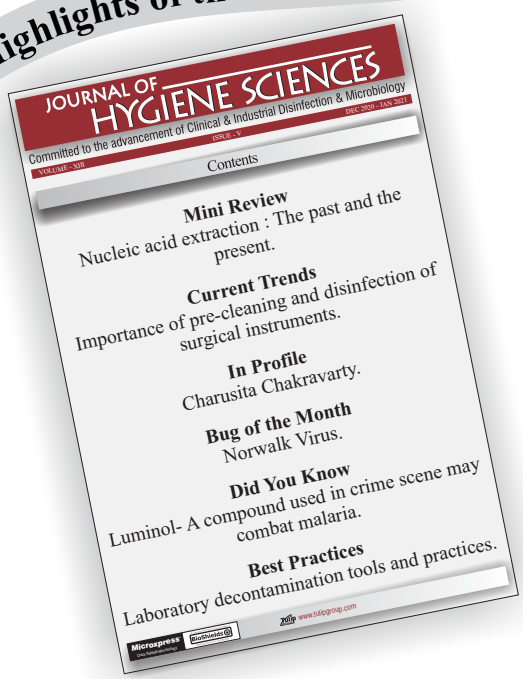
Tips for Health

- ✓ The best time to eat fruits and vegetables is ideally when we are hungry, since there are plenty of digestive juices that can digest and or segregate the plant fiber adequately, in this manner we ensure that we are reaping all the benefits of eating fiber and consuming all the vitamins and minerals. When we have high fiber foods such as fruits and vegetables on a full stomach, the fiber will just add bulk to the consumed food.
- ✓ Water should be had at a time when we are hungry and not immediately before and after meals, since just prior to meals, water dilutes the acid which will increase the time taken by the digestive system to breakdown the food into simpler molecules, and water should not be consumed immediately after meals since food gets mixed with the water and the digestive system gets strained in such a digestion process, therefore it is ideal to drink water at least half an hour before and / or half an hour after meals.
- ✓ As for protein consumption, its best to be had at night since tissue repair occurs during the night and carbohydrates are best had in the morning.
- ✓ Spice any gravy with less oil but more of ginger, onion, garlic, cinnamon and coriander. This helps since oils are rich in saturated fats, which are responsible for different health problems whereas ingredients like ginger, garlic, onion, cinnamon, coriander help in overcoming heart associated problems, diabetes, menstrual problems, body pains, arthritis, acidity and digestive problems.
- ✓ Breathing heavily, especially while climbing helps in removing the residual levels of CO_2 from the lungs and filing the lungs with more oxygen, which helps in certain breathing disorders. Taking a brisk walk will help in clearing the respiratory tract. However, it is not advisable to take a walk immediately after meals.
- ✓ It is good to exercise regularly, but even an exercise needs to be done appropriately, preferably on an apparently empty stomach, since exercising immediately after meals puts a strain on the digestive system. Therefore exercising is most beneficial in the morning before breakfast or in the evening, and best to be avoided during conditions like menstruation.

All the food items in the diet are essential and in a proper proportion give us the vigor and vitality to carry on with all our regular chores with good health.

References: https://www.who.int/

Highlights of the coming issue



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Published by D.G. Tripathi, Edited by Akshaya Tendulkar for and on behalf of Tulip Diagnostics (P) Ltd., Tulip House, Dr. Antonio Do Rego Bagh, Alto Santacruz, Bambolim Complex, Post Office Goa- 403 202, India. Fax: 0832 2458544, **Website: www.tulipgroup.com.**