Vitamin A and Prevention of Blindness

by Nilratan Halder

Vitamin A deficiency in per capita daily consumption has fallen from 93 per cent of requirement in 1964 to only 38 per cent in 1987. According to a Helen Keller research paper, 88 per cent of families in the country now consume less than the estimated minimal requirement. No factor has been referred to for this abnormal fall in vitamin A intake. But this can be attributed to the population explosion and shrinkage of lands for cultivation of vegetables and fruits as against the expansion of rice cultivation by the average farmer.

FOR the 30,000 pre-school-age children who go blind each year due to vitamin A deficiency, one half of them dying within months, and those young ones in labour force constituting five per cent of the age group 5-9, and 25 per cent of the 10-14 age group, it is cold comfort that Bangladesh is a signatory of the Convention on the Rights of the Child and one of the first 22 countries to ratify the same. Poverty, illiteracy and ill health are the issues concerned here. In short, it is a reflection on the quality of life in Bangladesh society. Malnutrition may not be always related to poverty but here the nutritional deficiency is certainly a problem for the poor people. The high incidence of blindness in rural areas and still higher one in urban slums explain the close relations between these two.

The dietary deficiency of vitamin A had become acute over a period spanning more than two decades. Indeed, the problem was far less acute 25 years ago. Vitamin A deficiency in per capita daily consumption has fallen from 93 per cent of requirement in 1964 to only 38 per cent in 1987. According to a Helen Keller research paper, 88 per cent of families in the country now consume less than the estimated minimal requirement. No factor has been referred to for this abnormal fall in vitamin A intake. But this can be attributed to the population explosion and shrinkage of lands for cultivation of vegetables and fruits as against the expansion of rice cultivation by the average farmer. The increase in the output of cereals has been possible at the cost of vegetables, fruits and fish.

It is against such an alarming backdrop of food habit that the blindness of children from six months to six years of age should be analysed and the urgency of about six-monthly distribution of high-potency vitamin A capsules to children of those group considered. Blindness is a direct consequence of vitamin A deficiency and protein-energy malnutrition but many other child disease also have a subsequent bearing on this ultimate type of eye disease. On the other hand, vitamin A deficiency exposes millions of children to increased risk of illness, poor growth and early death. Over 60,000 children under six years of age directly suffer from some degrees of permanent loss of sight due to lack of vitamin A.

Several conditions combine with vitamin A deficiency to expedite the process of blindness. These are (a) weaning practices, (b) diarrhoea, (c) measles, (d) worm infestation, (e) socio-cultural habits. One of the causes of malnutrition leading to blindness is defective weaning practice. Feeding children only breast milk upto two years of age is as imperfect as adding simple rice or rice gruel. The ideal feeding practice ought to be a mixture of vegetables and fruits with their diet in addition to breast milk. This however is neglected thanks to both ignorance and poverty.

The damaging effect of diarrhoea is to the extent that the vitamin A stored in the liver is used up. More, a child falling victim to this disease cannot absorb nutrients, including vitamin A from the foods consumed. According to an IPHN/UNICEF/HKI (Institute of Public Health and Nutrition/ United Nations Children's Emergency Fund/ Helen Keller International) publication, three out of four children blinded from vitamin A deficiency had diarrhoea in the four weeks prior to losing sight.

Measles' contribution to blindness of a vitamin A-deficient child is pronounced and quick. The fever caused by measles destroys the small amount of vitamin A stored up in the liver. Lack of vitamin A causes the eyes to get dry and then the measles virus can easily attack eyes causing ulcers in them. One out of every three blind children in Bangladesh had measles at the time they were blind.

Roughly nine out of 10 children in Bangladesh have worms in their stomach, some of them being heavily infested with round worms. The worms eat the food before the children can absorb the required nutrients from the foods eaten for their body. Naturally, nutritional disorders follow and ultimately leads to blindness.

Not all socio-cultural habits are necessarily bad. The fact that nine out of 10 rural mothers breast-feed their babies upto age two is certainly a plus point. Because breast-milk is rich in vitamin A. However the bad part of this is that mothers in villages discard colostrum from breast-milk in the belief that it is harmful for babies. Contrary to their belief, colostrum is highly nutrient with vitamin A. Even sticky milk of the later part of the natal period is good for them. Similarly, vegetables and fruits do not figure in children's diets because of lack of knowledge of appropriate foods for young ones aged six months to six years. Another bad practice is to stop feeding — both food and water — children when attacked by diarrhoea. More ludicrous is the fact that some even consider green vegetables responsible for diarrhoea.

So one thing is clear that eye diseases can be prevented in most of the cases if timely actions are taken. The best preventive method would be to educate mothers about the virtue of vitamin A. But if the preventive method is not successful to stop the disease, curative measures have to be taken. In this effort too, what counts even more vitally is some sort of primary knowledge for early detection of the eye problems. Only then eye sight can be saved. Eye complications start manifesting in the following forms: night blindness, conjunctival dryness, Bitot's spots, corneal dryness, ulcer/softening of cornea and corneal scar.

Testing of children's eye vision — Photo: Vision

Night blindness is the first symptom of vitamin A deficiency. In bright sun light a child's vision may not be impaired but with the approaching darkness, he/she starts groping. He/she shows no keen interest in food and is reluctant to go out and play with other children. The poor quality tears are the first sign of conjunctival dryness which ultimately leads to xeropthalmia. The white part of the eye is no longer smooth and shiny. Named after a French doctor Bitot, Bitot's spot is a classic sign of xerophthalmia. When conjunctivia gets dry, patches form on the side of the white part. The patches appear to be like foamy bubbles from soapy water. Bitot's spots are a consequence of vitamin A deficiency. Chronically deficient in vitamin A, children develop corneal dryness of acute form. So affected corneas are destroyed leading to blindness within 24 to 48 hours. If vitamin A-deficient children are attacked by measles or diarrhoea, corneas can suffer blindness immediately. Untreated dry corneas also develop ulcer. In case the ulcer is big, the eye becomes soft and if the corneal ulcer is small, a small scar remains after recovery through proper treatment. Untreated eyes will lose sight with a large scar. A deep corneal ulcer is also curable but if treated early with vitamin A, the scar may be confined to a limited scar.

Clearly, prevention of blindness is within the reach and means of most of the people of the country. Even the poor can avoid the tragic blindness for their children if they are aware of the food values of green and leafy vegetables. Health education, nutritional in particular, is the first criterion in bringing about a qualitative change in other areas of the whole matter. Vitamin A can be supplemented through changing dietary habits, horticulture and preparation of daily dishes. The most common vegetables full of vitamin A are often ignored by common people, rather wrongly. Sometimes those are available without cultivation and at other times they become scarce. But the main problem is not availability alone. Once people start appreciating their value as vitamin A, their shortage is the natural outcome. That will call for cultivation of those items in a systematic manner.

As for the distribution of vitamin A capsule and other clinical method of eye treatment, the delivery system is far short of the requirement. Under the plan of the government is a project of Taka 37 crore to fight blindness. If this gives adequate emphasis on nutritional education, more than anything else, it may end up doing a good job. The expenditure of massive amount on prevention of blindness is more than reimbursed if the burden of a great number of blind people in society can be avoided through such a campaign.

Emerging Strategies to Combat Common Cold

by G S Mudur

FROM boiled snails in barley water to tailor-made molecules that prevent cold-causing viruses from invading cells in the body, the centuries-old fight against the common cold has come a long way.

Gaining an upper hand on these viruses is expected to significantly boost productivity in workplaces worldwide and alleviate much human discomfort characterised by sneezes, running noses, and sore throats.

While the body relies on its immune system to detect and destroy invading microorganisms, the cold viruses have been remarkably efficient in evading the immune system.

The sheer variety of viruses that cause cold is one reason for their ability to escape the immune system. The common cold is a complex infection triggered off by nearly 200 strains of viruses. Most modern strategies devised to fight the infection are focussed on a family of viruses called rhinoviruses, the major cause of colds.

Proving clinical efficacy of some of the strategies has been difficult because the mechanism of pathogenesis of the infection is little-understood. There are no animal models for common cold, the infection is short-lived, and it is difficult to detect at low levels.

The chemical armouly against the cold-causing viruses has grown over the past two decades with researchers pitting an assortment of agents to fight or prevent the infection: aqueous iodine solution, nasal tissues, interferon and in recent years hybrid molecular compounds have been tested.

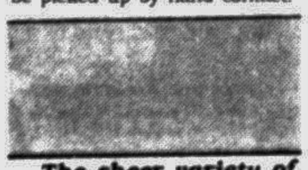
Although the first report on the efficacy of interferon against rhinoviruses emerged in the early seventies, a series of clinical trials began in the early eighties when high purified and potent genetically-engineered interferon was available.

Although the work has convincingly shown that it is effective in reducing the symptoms of experimental rhinovirous cold, the successes have been offset by local side-effects, according to Jack Gwaltney, Professor of Internal Medicine at the University of Virginia School of Medicine in the US.

Among the side effects observed were nasal dryness, stuffiness, discomfort, and appearance of blood-tinged nasal mucus.

Although the effects are reversible, daily interferon doses as a routine anti-cold therapy is not considered feasible today, say researchers.

Studies have demonstrated that cold-causing viruses are not only inhaled, but can also be picked up by hand contact.



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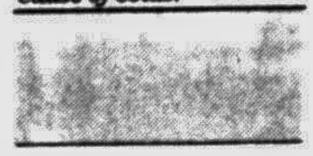

Susceptible people can unwittingly infect their nasal mucosa with virus acquired on their hands form contaminated skin or surfaces.

Experiments with volunteers have shown that aqueous iodine has a potent rhinovirucidal activity on the skin, persisting for up to two hours. An alternative technique involves the use of nasal tissues treated with virucidal agents. One study showed that a mixture of citric and malic acids and sodium lauryl sulphate has activity against many types of rhinoviruses and a few other cold-causing viruses.

University scientists working in collaboration with a drug company in the United States reported in 1986 that they had developed two structurally-related compounds, they called WIN 51711 and WIN 52084, that inhibited the replication of rhinoviruses.

Their report was the first description of an antiviral drug interaction with a virion at the atomic level. Reporting their findings in the US Journal Science, the researchers said their results raise the possibility of anti-viral drug design for rhinoviruses.

A Belgium-based company Janssen has since produced a hybrid molecule with similar antiviral activity that so for protects against the widest spectrum of rehinoviruses in human trials, according to a report in the journal Biotechnology.

Another strategy has been to neutralise the site on cells that the cold virus attaches itself to in order to gain entry.

Three years ago, scientists identified a molecule called ICAM-1 (Intracellular Adhesion Molecule-1), that they believe is the site that rhinovirus sticks to in order to enter human cells.

Some researchers believe antibodies against this receptor site could deactivate the receptor and prevent the rhinovirus from making contact with the receptor which is vital for viral entry into the cells.

According to the report in the journal Biotechnology, a research group in Princeton in the US has already shown that blocking the receptor can inhibit rhinovirus entry. In a human trial, antibodies, administered in the form of a nasal spray, delayed the onset of a cold and made its effects less severe.

A pertinent question is related to the cost-effectiveness of the new treatments if and when they are widely available.

Researchers do not rule out the possibility that the cost of preventing the infections might in some cases be more expensive than simply fighting the symptoms with presentday over-the-counter drugs.

PTI Science Service

A World View from Down Under

by Kalinga Seneviratne

TO see the world is the dream of many people, but 63-year-old Australian doctor Fred Hollows makes this wish literally come true for thousands of people in the Third World in danger of losing their sight.

For the past few years, Hollows has abandoned a lucrative private practice to fly around the world performing surgery and establishing eye clinics, in countries like Eritrea and Nepal.

Specialising in the treatment of cataracts, he has also made it a point to train eye doctors in Third World countries to preform the same type of advanced operation he is known for.

"Some countries still send teams of surgeons from the Occident, Western Europe or America to Third World countries to do surgery on the poor," says Hollows. "We are opposed to that. We are interested in developing indigenous surgeons".

Cataract blindness is caused by the eyes' lenses growing opaque and hindering vision. With surgical removal and the inserting of a plastic lens, normal vision can be restored.

Hollows says 25 million people worldwide go blind every year because of cataracts, but only about five million ever get surgery. In Nepal, which Hollow praises as having one of the best blindness prevention programmes in the Third World, about 32,000 cataract operations are needed each year.

Hollows spent 18 months training Nepali surgeon Sanduk Ruit on his technique. Ruit has since gone back to his country where he has modified the procedure.

"It is a better procedure," quips Hollows. "In Australia, we now call it the Nepalese technique."

That may be a welcome improvement, but in cash short countries like Nepal, where annual per capita income is US $ 170, even just the lenses needed for the operation may be out of reach. In Australia, each lens costs from US $100 to US $ 150.

Hollows thinks the answer lies in putting up low cost lens manufacturing plants in the developing country itself.

Here in the industrial city of Woolongong, about 100 km south of Sydney, the opthalmic surgeon is now working with a team of engineers on such a facility. Once ready the factory will be dismantled and created for transport to Eritrea in February 1993. Hollows and his team will then work on two more similar plants for installation in Nepal and Vietnam.

The plants are expected to produce lenses that would cost less than five dollars each.

Engineers Andemeskel Abraha from Eritrea and Surof Dangol of Nepal are now working with Hollows. Both are being trained to be in charge of the first ocular lens factories to be set up in their respective countries by next year.

"This modest project will transfer skills and technology to (some) of the world's poorest countries and will help to set them on the path to independence," the doctor says.

Abraha believes the Eritrea plant will not only help solve his country's problem of getting a supply of affordable lenses, but will also stimulate a whole new industry for Africa.

"After we have fulfilled the demand for lenses and doctors in our country, we will be able to export to neighbouring nations," he adds.

Hollows first made his name in medical circles with his discovery of the existence of Labrador keritopathy and trachoma among the Aborigines in the Australian Outback at a time when the diseases were still unheard of in this country.

He used to take medical teams to Aborigine enclaves in the desert and helped establish the first Aboriginal Medical Service (AMS) in Sydney 20 years ago. Today, there are 64 medical centres in Australia modelled on the AMS.

After winning the Australian of the Year award in 1991, Hollows, through the foundation named after him, raised almost one million dollars for the Eritrean project.

Cash donations made up nearly half of the amount, while the Australian government chipped in with about US $ 460,000 from its foreign aid budget. The rest of the money came from the royalties generated by the sales of Hollows's autobiography.

The foundation has been scouring the Australian business community since January for funds to build a similar plant in Nepal.

So far, the response has been encouraging, says a spokesperson for the Fred Hollows Foundation.

— IPS

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Yellow Fever Makes Comeback After 50 Years

by Megan Durnford

An outbreak of yellow fever in Kenya is being contained. It is the first to be officially reported there since 1943, and the occurrence serves as a reminder of the agility with which the virus and its mosquito carrier travel. Yellow fever, which is often fatal, is endemic in tropical regions of Africa and South America. Gemini News Service reports on the mechanism and epidemiology of this disease.

KENYA'S Ministry of Health has reported an outbreak of yellow fever in two districts of the Rift Valley Province, northwest of Nairobi. This is the first time the disease has been officially reported in Kenya since 1943. The World Health Organisation (WHO) has not yet been informed of the number of cases and dates of occurrence.

Yellow fever is a viral disease characterised by hepatitis, bleeding and kidney failure that is endemic in tropical regions of Africa and South America.

The Kenya outbreak is limited to two divisions of Baringo district — Kabernet and Tanges — and the Southern division a Elgeyo Marakwet district. The government has organised a mosquito control programme and yellow fever vaccination, launched February 9, is underway throughout the affected districts. These measures are expected to contain the outbreak.

Although the Kenyan government recommends that visitors are vaccinated for yellow fever before they travel, official policy is still not to require vaccination. Yellow fever is endemic in Kenya. That doesn't mean the disease is rampant there, but simply that the mosquitos which can transmit it are found in that country.

In 1943 only a couple of cases of yellow fever occurred in Kenya, says the WHO. In fact, only 200-4,000 cases are officially reported worldwide each year, but the disease is vastly under-reported, especially in remote areas of the developing world.

Nigeria has one of the highest rates of yellow fever in Africa. Epidemics in that country between 1986 and 1990 caused more than 150,000 cases and more than 30,000 deaths.

The yellow fever virus enters the body by a mosquito bite and travels to the lymph glands, part of the immune system, where it replicates. Then it travels via the bloodstream to the liver and kidney. The virus damages both organs as well as heart muscle.

The first symptoms of yellow fever — chills, headache, fever, muscle pain and nausea — resemble those of influenza. They can last several days, during which time the patient is a source of infection for mosquitos. Next the victim becomes intensely ill and restless, with flushed face, swollen lips and a bright red tongue. The face then darkens, the gums become swollen and bleed easily and the victim may have black vomit. The pulse rate is slow, despite high fever.

The destruction of liver cells by the virus is responsible for two of yellow fever's well-known symptoms. Jaundice, for which the disease was named, is a yellow discoloration of the skin and the white of the eyeball due to bile pigments in the blood. And liver damage is also responsible for bleeding as the production of clotting factors is decreased.

About 20 to 50 per cent of jaundiced patients die, usually 6-9 days after onset. Mortality varies greatly, depending on the viral strain, and to a certain extent on the patient's exposure to the yellow fever virus. A poor prognosis is indicated by early appearance and deepening of jaundice and severe bleeding.

Treatment is largely supportive, with an emphasis on fluid maintenance and reduction of fever. Blood transfusion is sometimes required to compensate for blood loss.

Survivors recover rapidly from the acute infection, but may experience weakness and fatigue for several weeks.

The virus replicates in the mosquito gut and when the mosquito bites again, it is regurgitated into the wound in a substance that the mosquito uses to prevent coagulation. Thus the virus can circulate from a primate host to an insect and replicate in both. Humans can also join the deadly cycle.

persons and *Aedes aegypti* travelling on slave and trading ships. During long voyages, each round of breeding mosquitos introduced new episodes of the epidemic.

Major ports on the eastern coast of the United Sates, as far north as Boston, were affected and the tropical and subtropical regions of the Americas were subjected to devastating epidemics which decimated populations and paralysed industry and trade. The last US outbreak was reported in 1905, although yellow fever virus is now commonly found throughout Central and South America. Before the European arrival, yellow fever was not a problem because indigenous people had developed immunity to the disease.

Originally, controlling mosquito populations was the only preventive measure available, as, for example the campaign against *Aedes aegypti* that made the construction of the Panama Canal possible.

Immunization is another useful weapon in the war against yellow fever. In 1937, South African microbiologist Max Theiler first introduced a yellow fever vaccine. Fourteen years later, Theiler was awarded a Nobel Prize for the 17-D vaccine, which is still used today.

This vaccine is a live, attenuated virus injected under the skin. One of the most common ways to weaken a virus is to introduce it into an unnatural host. The 17-D vaccine is produced in chicken embryo calls. After several generations, the virus replicates better in the chicken cells than in human cells.

Although it can still replicate well enough in humans to confer immunity, it can no longer cause disease.

Over the last 50 years, more than 200 million doses of yellow fever vaccine have been administered, with a remarkable record of safety and efficacy. Side-effects such as fever and headache are mild and occur in less than five per cent of vaccinations.

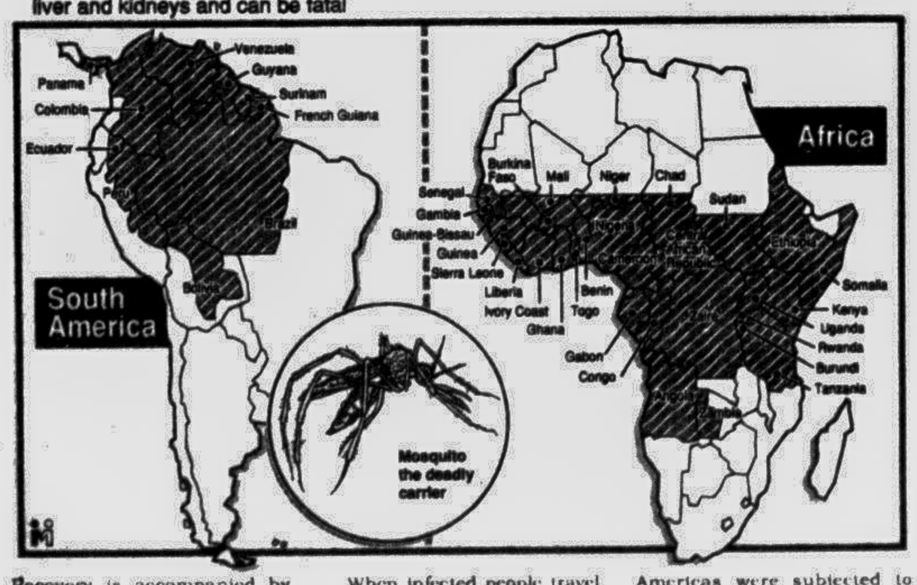
Immunization and mosquito eradication programmes are effective means of preventing yellow fever infection. However in endemic areas, human cases will continue as long as there are unimmunized people, for there is no known practical way of eliminating the yellow fever virus from the vast tropical forests of South Amerlea and Africa.

The virus's ability to adapt to a new carrier and increased mobility of human communities contribute to the spread of yellow fever into virgin populations.

— Gemini News

Yellow fever danger zones

Mosquitos spread yellow fever through Africa and South America. This disease attacks liver and kidneys and can be fatal



Recovery is accompanied by complete immunity to the disease.

In central Africa, where yellow fever is thought to have originated, wild primates such as howler, owl, spider and squirrel monkeys are infected. Several species of mosquitos, including the *Aedes africanus*, take blood meals from the monkeys to provide nutrients for developing eggs.

When infected people travel to towns and cities the yellow fever virus adapts to a new mosquito host, *Aedes aegypti* (also called yellow-fever mosquito), which breeds in barrels, cisterns and any other container holding water. This mosquito transmits yellow fever between humans.

The yellow fever virus left Africa in the 17th, 18th and 19th Centuries via infected

***MEGAN DURNFORD** is a Canadian freelance journalist specialising in medicine. She is currently completing an internship at Gemini News Service.*