

Sri Lanka College of Paediatricians; Presidential Address 2004
TODAY'S ENVIRONMENT: TOMORROW'S CHILDREN
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The Sri Lanka College of Paediatricians together with its forerunner, the Sri Lanka Paediatric Association, has over the years contributed enormously towards improving infant and child survival rates of Sri Lanka. Today it is said that children are healthier than they have ever been before. However, the disease spectrum confronting children is changing rapidly. It is to this emerging situation that I wish to draw your attention.

Having stepped into the 21st century we find that the disease entities facing us are changing. It is public knowledge that asthma and cancers in children are on the rise. Literature documents a worldwide increasing incidence of bronchial asthma, autism, attention deficit hyperactivity disorder, developmental disorders, allergies, congenital abnormalities and malignancies, especially leukaemia and brain cancers^{1,2}. The present day paediatricians encounter these conditions almost daily. On closer scrutiny these conditions are mostly *chronic illnesses of multifactorial origin*. Having already combated acute infections of childhood, developed nations find themselves facing threats from new environmental hazards³. If this trend is left unabated these disorders may soon mask the successes achieved in paediatrics and child care⁴.

Many of these disorders remain without an exact aetiology. Genetic factors account for 10 to 20%. In the remainder *environmental factors* are strongly suspect. We may not know with any great certainty the exact causative factor or have a clear understanding of pathogenesis but we know enough to worry about their environmental link and outcome. It is therefore up to us to safeguard our children, and the children of tomorrow, from today's environmental threats and hazards.

In 1993 World Health Organisation, in recognition of the link between environment and health defined *environmental health* as “*human health or quality of life altered by physical, chemical, biological, social or psychosocial processes in the environment*”. This was further expanded to include “*assessment, control and prevention of environmental factors that potentially affect present or future generations adversely*”.

We all accept that environmental degradation is a powerful contributor to ill health. It is, however, not my intent to dwell on global environmental challenges such as global warming and climate change, depletion of the ozone layer, the rising sea level, unsustainable consumption patterns, urbanisation, deforestation or desertification, although all of these affect child health. Nor will I address issues of the special group of children called “environmental refugees” - children displaced by environment related catastrophes, although Sri Lanka has had her share of weather-related mishaps in recent times. I will instead address those environmental issues that paediatricians need to be aware of, be able to diagnose, treat, prevent or even eliminate, during their day to day clinical practice.

Environmental paediatrics

Environmental paediatrics is a relatively young branch of paediatrics and deals with *the diagnosis, treatment and prevention of diseases in children caused by environmental exposures*. Although still in its adolescence this field has taken rapid strides to occupy a front seat in paediatrics. It is now an essential component in both community and acute paediatrics. Today's medical students include the environment in any paediatric history they take!

The impressive intensity of activity in this field that took place over a five year time span was summarised by Landrigan of Mount Sinai School of Medicine, New York, in 2001⁵. His list includes:

- * *National and international conferences on environment and children.* The first such international meeting on the impact of the environment was held in Amsterdam in 1997. By the following year the eight leading economic nations (the G8 countries), issued declarations, making the protection of children from environmental threats a national priority.
- * *Prominence in paediatric journals.* Entire issues of widely read journals have been devoted to environmental diseases of children.
- * *Expanding literature.* As the literature mounts a handbook on Paediatric Environmental Health (“the Green Book”) was released by the American Academy of Paediatrics.

- * *Children's environmental research centres.* Research centres and parallel networks of clinically oriented paediatric environmental units have been established in several countries.
- * *Interest by international agencies* - Many international agencies including WHO, UNICEF, UNEP have taken up this theme and warnings such as “the environment is killing our children” are “resounding louder with each passing year⁶.”

History of environmental paediatrics

It was exactly a hundred years ago that environmental paediatrics, as we recognise it today, began. In 1904 an outbreak of lead poisoning among children in Queensland, Australia resulted in the first report of lead in paint⁷. Research involving children has culminated in a ban on leaded paints. In the 1920s came the biggest environmental mistake of the 20th century. The introduction of tetraethyl lead to petrol as an anti knock agent resulted in chronic lead poisoning which was rightly termed the ‘Number One’ environmental disease of childhood⁸. Until 2002 children in Sri Lanka continued to be exposed to this source of lead but recent research shows that the desired change in the blood lead levels of children has been achieved with change to unleaded petrol^{9,10}.

The brunt of the damage from ionizing radiation following atomic bombings of Hiroshima and Nagasaki was borne by children. Those exposed prenatally manifested epidemics of microcephaly. Childhood haematological malignancies followed. Another historical milestone was the London smog of 1952. It brought medical attention to air pollution. The increase in mortality was primarily among the very young and the aged. Research on air pollution, which began with walking laboratories that sampled air, has reached a high level sophistication across the globe within half a century. Our own research assessed pollutant levels in air using clip on passive filters fixed to children’s garments^{11,12}.

In 1960’s an epidemic of cerebral palsy, mental retardation and convulsions occurred in a fishing village in Japan. The cause was mercury containing waste discharged from a plastic manufacturing plant in to the Minamata Bay. Micro-organisms in the sea transformed mercury to methyl mercury which through bio – accumulation, moved up the marine food chain to reach pregnant mother who consumed contaminated fish or shellfish. The fetus is at the apex of the food chain. Exposure to methyl mercury in utero became known as “Minamata Disease” and similar outbreaks have been reported in U.S.A, Mexico Guatemala and Iraq¹³. Studies show that exposure to even low doses of methyl mercury is harmful. Pregnant women are warned against consuming shark, mackerel, tuna and other predatory fish from specified and untested waters. Mercury level in fish is regulated in some countries¹⁴. In 1972 what is termed the world’s worst industrial catastrophe again highlighted the vulnerability of children who breathe dangerous air. Children contributed enormously to mortality figures in the Bhopal tragedy. Over 15,000 cases of phocomelia in the thalidomide tragedy revealed that drugs in early pregnancy could be calamitous. In utero exposure to diethyl stilboestrol and resulting adenocarcinoma of the vagina in young women is another example¹⁵.

Man’s interference with nature results in health consequences. In Sri Lanka, deforestation and paddy cultivation resulted in non immune populations settling in Mahaweli areas and mosquito breeding in areas with “JE activity” brought in by the migratory bird, *cattle egret* caused deadly outbreaks of Japanese encephalitis. An additional vaccine to the national immunization schedule was necessary to control this newly emerged disease among children¹⁶.

The importance of environmental paediatrics

History teaches us many lessons. Important lessons learnt from the above events are:

- * *Children are the first targets* - Children are the earliest indicators of environmental hazards and have been even described as “sentinels of society”. It is noteworthy that pregnant mothers had little or no symptoms of methyl mercury poisoning. Yet their babies suffered great damage.
- * *Paediatricians are in the frontline of environmental medicine.*- With children being the first targets the need for paediatricians to be aware of environmental disease is obvious.
- * *Environmental hazards concern both community paediatricians and clinicians* It is the clinician who first detected “unusual”, or “cluster cases” which eventually was realised to, be environmental in causation. Risk assessments and community based studies, as well as protecting

- the community, are important activities in community paediatrics.
- * *Evidence of a cause and effect linkage* - The cause and effect manner of environmental hazards and disease was revealed.
 - * *Clinical manifestations were varied – either dramatic or silent* - Pathogenesis can be slow and stealthy or dramatic.
 - * *A single insult can cause permanent life long damage* - The thalidomide tragedy and Minamata disease are important examples of damage to children that were life long.
 - * *Children are affected very differently to adults* - We say that “children are not little adults”. In environmental paediatrics this difference is particularly obvious and disadvantageous to children.
 - * *The incidence of environmentally linked diseases is increasing* - Global epidemiology shows bronchial asthma to have increased twenty fold in some developed countries¹⁷. A similar trend is seen for childhood cancers.

Role of the paediatrician

Environmental paediatrics is not confined to the public health arena. Active intervention by paediatricians is necessary. To begin with the paediatrician should be an alert clinician. The paediatrician should recognise clusters of cases, and identify associations between disease and exposure. Paediatricians should be competent to make the correct diagnosis and establish linkage. An environmental history is essential in this diagnostic work up¹⁸.

It is often the paediatrician who first sounds the warning bell when a cluster or even a single case is recognised as of environmental origin. The role of raising the alarm by paediatricians is an important one when an ongoing risk, whether real or potential, is posing a threat to the health of children and the community. Community paediatricians, a sub speciality that the College has been repeatedly requesting for, would have the additional role of performing risk assessments at community level, establishing associations and detecting dose response relationships while taking steps to protect the community. Both community and acute paediatricians should provide information on environmental concerns, to parents, the public and policy makers. An advocacy role at local, national and international level is also one for paediatricians.

The clinical approach to environmental disease

Clinical evaluation in environmental paediatrics requires:

- * A high degree of suspicion.
- * Awareness of the community and environment the patient lives in.
- * A detailed environmental history.
- * Recognition of unusual or clusters of patients.
- * Investigation for an environmental cause in any patient with unexplained neuro-developmental or behaviour disorder.
- * Ability to identify known environmental diseases.
- * Detection of a link between the clinical problem and an environmental agent.
- * Recognising the timing of the insult.
- * Good record keeping.

In this clinical exercise, laboratory assistance is often unavailable. Even if this constraint makes confirmation of the offending agent difficult in the individual clinical situation a cluster of ‘cases’ will help establish the linkage. Removal to a place of safety appears to be an obvious option whereby further damage can be prevented. However the damage is often already done and irrevocable, but diagnosis will protect other children.

Taking an environmental history¹⁹

All patient encounters should include at least a brief environmental history. Detailed interrogation is needed when an environmental disease is suspected. Asthma is such an instance. An environmental history should contain the present and prior locations of both home and school, characteristics of the surroundings and neighbourhood, proximity to industries, traffic congested places and rubbish dumps and incineration of hazardous material, chemicals, radioactive material and even jewellers waste. Sanitation, source of water, hous-

ing and indoor conditions both at home and classroom, parental occupations and recreations, the child's hobbies and leisure activities, diet and drug history, home and folk remedies should not be forgotten ²⁰.

Clinical presentations

Subclinical toxicity

The situation where the child is asymptomatic but exposure has occurred is perhaps the commonest. This is particularly so with toxins such as lead that affect at very low doses. The pioneering work of Needleman on asymptomatic children is a good example of this. Our research on apparently normal children living near Borella junction provided evidence of such sub clinical toxicity in the local scene ¹⁰.

Non specific symptoms

Some environmental diseases produce only non specific symptoms and can mimic other non environmental conditions. For example, carbon monoxide poisoning causes headache, nausea, vomiting and chronic lead poisoning pallor, recurrent abdominal pain and constipation.

Neuro-developmental disease

The developing central nervous system is a site of ongoing change due to cell division until 6 months and cell migration, differentiation, myelination and apoptosis that continue beyond two years. Therefore the brain is particularly vulnerable to environmental insults. The four best known neuro-developmental toxins are lead, ethanol, polychlorinated biphenyls (PCBs) and methyl mercury. Exposure of children with resultant effects on cognitive abilities, newborns with lead detected in cord blood and the fetal alcohol syndrome have been well documented in Sri Lanka.

Bronchial asthma

This multifactorial disease with a large environmental overlay is one of the commonest conditions in paediatric practice today. Our own research has shown a statistically significant link with ambient air pollution levels of oxides of nitrogen and sulphur dioxide and acute wheezy episodes in children presenting to the Lady Ridgeway Children's Hospital Colombo ¹¹.

Haematological conditions

The rapid cell turnover makes the bone marrow another potential target to toxins. The cancer registry indicates leukaemia to account for 25% of all childhood malignancies in Sri Lanka.

Childhood cancer

Environmental toxins causing carcinogenesis is gathering public concern and over 80,000 synthetic chemicals are said to have been invented since the Second World War. Our lives are bombarded with these. From the colouring or flavouring in your toothpaste to the mosquito coil you light at night our environment is teeming with synthetic chemicals. In the USA between 1973 and 1992 childhood cancer has been increasing at the rate of 1 % each year. Leukaemia and CNS tumours are the leading conditions.

Accidental ingestion of toxins

Classically, environmental hazards strike slowly and stealthily. But accidental ingestion by poisons found in and around the house is yet a hazard faced by preschoolers. Acute poisoning by agricultural pesticides, weedicides and plant poisons found in the child's environment have been brought to the attention of the Sri Lanka College of Paediatricians by Lucas ²¹.

Auditory and non auditory effects of noise

Noise levels above the stipulated safety and comfort levels can cause several adverse effects, auditory and non auditory, apart from annoyance. Sleeplessness, irritability, poor attention span, deterioration in cognition and hypertension are documented. We examined a series of young children who were affected thus and their plea for justice was upheld by court²².

Endocrine disorders

Several endocrine and reproductive anomalies are increasing in incidence and are linked to synthetic chemicals that act as environmental oestrogens. Isolated thelarche, precocious puberty, decreased sperm counts, increased rates of hypospadias and cryptorchidism as well as increase in testicular tumours are now be-

lieved to be due to pesticides, poultry and synthetic additives in hair care products. Early onset of puberty in girls has caused the attention of the public in Sri Lanka, too, although a scientific study on this has not yet been made^{23,24}.

Reassurance seekers

Parents come to paediatricians for guidance on a variety of exposures. Hence the paediatrician has to be informed of risks of plastic feeding bottles, mosquito repellents and mosquito coils, lice shampoo, artificially ripened fruit and genetically modified food among several others.

Age of patient and timing of insult

Timing of insult determines damage. Besides dose and route, timing also needs to be considered in the clinical evaluation. The age of the patient is of vital importance. The dynamic nature of paediatrics requires this approach. The entry of environmental agents at each of the stages of embryo, newborn, infant, toddler, child, and adolescent causes different abnormalities. This developmental approach has been described in detail by Gitterman and Bearer.

Preconception exposure

The environment can cause damage even before conception. Xenobiotics are compounds foreign to living beings that have been described in follicular fluid of ova illustrating environmental harm even before life starts. Epidemiological evidence incriminates paternal exposure to various chemicals causing damage to his children. Father's employment in textile and art industries, glass - and mining industries and rubber manufacturing factories have been statistically linked with stillbirths and other neonatal abnormalities. Vehicle mechanics and welders run an increased risk of fathering a child with Wilms tumour. Poly carbonated biphenyls (PCBs) have caused harm by entering the mother through contamination of cooking oil. Mothers exposed seven years before conception have produced mentally retarded children. Further, it is documented that people living within 3 km from a hazardous waste landfill had a higher chance of having a child with a chromosomal defect. It is indeed correct to say that "Today's environment affects Tomorrow's children"¹¹.

In utero exposure

The placental barriers are permeated by alcohol and other lipophilic chemicals, small molecular toxins and minerals such as lead that are actively transported across. In this group is environmental tobacco smoke and passive smoking is as harmful as maternal smoking. Increased cotinine levels that occur are linked with several abnormalities in the newborn e.g. asphyxia, low birth weight, retarded postnatal growth, impaired lung function, congenital abnormalities and even cancer. Lead deposited in the maternal bone poisons the unborn child. This long lasting and deep reservoir is an endogenous source because 30% of the maternal skeleton becomes available for calcium needs of the fetus by bone demineralisation during pregnancy. It was possible to demonstrate the presence of lead in cord blood in Sri Lanka one year after introduction of leaded petrol. The fetal alcohol syndrome and fetal hydantoin syndrome are among a long list of such conditions. It should not be forgotten that physical agents can by-pass the placenta. Noise, electromagnetic and ionizing radiation are examples that cross to the fetus.

Preterm exposure in neonatal units

Neonatal intensive care units subject very sick preterm newborns to continuous bright lights, loss of circadian rhythmicity, sleep deprivation and high noise levels. Oxygen toxicity and the adverse effects of drugs, infections and pain are better known but since the pioneering work of Hubel and Wiesel long term effects of subjecting sensory systems of newborns are being substantiated. Hearing, cognitive abilities, and behaviour are the ill effects described.

The newborn period

The newborn period is one of extreme vulnerability. The skin surface is highly permeable and so is the gastrointestinal tract. The large skin surface area and high gastric pH worsen this risk. Transporting toxins from parental occupational sites, on clothes or body, must be borne in mind. Fortunately the newborn is only exposed to breast milk in his diet. This remains the safest milk although pollutants such as DDT and PCBs can accumulate in the mother and be secreted in breast milk. These pollutants are poorly excreted in urine, faces and expired air and breast milk is a good outlet.

Infancy and preschool age

During this stage of development the effects of developmental neuro-toxins manifest. Lead, mercury, and PCBs are in this group. So is alcohol exposure in fetal life. It should be remembered that water can contain noxious chemicals as seen when nitrates from fertiliser caused the “blue baby syndrome”. Increased mouthing (i.e. hand-to- mouth) activities and exploratory behaviour in this age group increases their risk of ingesting toxins. This can result in slow, low-dose exposure to chemicals containing toxins such as lead or acute accidental poisonings. The dangers posed by 80,000-100,000 newly invented synthetic chemicals should be borne in mind.

School age

Many unhealthy factors accost the child in the school environment. Among these are overcrowding in classrooms, high numbers of children to a functioning toilet, unsafe playgrounds, unhygienic canteen conditions, indoor pollution, poorly ventilated and ill lit classrooms, rubbish dumps and vector breeding sites in and around school premises, high noise levels and the excessive weight of the school bag. The hazards of noise pollution on children should not be forgotten. Several harmful effects occur and a situation where the courts upheld such a plea made in the name of affected children in Colombo was reported in the College journal a few years ago.

Why are children particularly at risk of environmental hazards?

The child’s physiology and maturity play a vital role. In childhood organs are undergoing differentiation and growth. Biochemical immaturity hampers detoxification and excretion is less efficient and chemical injury is hence more likely. Differences in dietary patterns together with greater gut absorption invite the ingestion of more toxins. For example, children have a much higher absorption rate of lead than adults. Differences in behaviour and activities indulged in, by mouthing when exploring the world, compound this increased vulnerability. Children are also more prone to air pollution because of relatively higher tidal volumes taking in greater quantities of air into the lungs. Young children spend more time indoors and it is now well accepted that indoor air is more polluted than outdoor air. School children are out on the roads at the most congested traffic times breathing polluted air. Further, it has been demonstrated that the child breathes in more particulates due to his proximity to ground level. In summary, children run a disproportionately higher exposure risk, have immature metabolic pathways less able to handle toxic agents, and have developing organs more vulnerable to physical and chemical insults²⁵. Children of the 21st century face an environment very different to that of 50 years ago. In its broadest context the environment consists of four fields - physical, chemical, biological and social. In each of these the hazards are several folds greater than what it was a decade ago. It is up to us, those caring for children, to express the urgent need to curb these dangers.

Are we ready to accept this challenge?

Most paediatricians are not well versed in risk assessment or management of paediatric environmental diseases. Therefore it is time that training and resources were provided for capacity building in this field. Children’s environmental health issues should be incorporated into both undergraduate and postgraduate medical curricula. Paediatricians should be able to detect diseases early, assess risks and monitor environmental factors that affect children. Raising public awareness at community and national level and educating parents are tasks ahead of us. Environmental education of parents should be tailored to local needs. Correct and up to date information should be provided to parents. Behavioural change of parents and caregivers is another necessary intervention. Modifying dietary habits, advocating precautions from exposure to chemicals and environmental tobacco smoke, reusing and recycling waste and managing limited resources such as water are further interventions that need our attention.

Strengthening inter-sectoral partnership and multi-agency efforts will be necessary to achieve these goals. Research laboratories and universities should broaden their research agendas to include children’s environmental health issues. Vulnerable groups should be highlighted, specific exposure pathways evaluated and dose-response relationships established. Research data on children is absolutely necessary because data on the “average adult” alone is inadequate if one is to stipulate safety standards for children. Children deserve special protection as they breathe more air, drink more water and eat more food, per unit body weight, exposing them to environmental hazards much more than adults. Playing an advocacy role is integral to paediatrics²⁶. It is up to us paediatricians to ensure that children are placed in the forefront of all agenda on sustainable development.

References

1. Gurney JG, Davis S, Severson RK. Trends in cancer incidence among children in the US. *Cancer* 1996; 78:532-41.
2. Eggleston PA, Buckley TJ, Breyse PN. The environment and asthma in US inner cities. *Environ Health Perspect* 1999; 107(3):439-50.
3. Zahm SH, Devesa S.S. Childhood Cancer; Overview of incidence trends and environmental carcinogens. *Environ Health Perspect* 1995; 103(6):177-84.
4. Teague WG, Bayer CWO Outdoor air pollution: Asthma and other concerns. *Paediatric Clinics of North America* 2001; 48(5):1167-8.
5. Landrigan P.J. Children's environmental health. Lessons from the past and prospects for the future. *Paediatric Clinics of North America*. 2001; 48(5):1319-30.
6. United Nations Secretariat of Convention on Biological Diversity. Available from: <http://www.biodiv.org>
7. Gibson J.L. A plea for painted railing and painted walls of rooms as the source of lead poisoning among Queensland children. *Australian Medical Gazette* 1904; 23:149-53.
8. Needleman HL, Schell A, Bellinger D, Long-term effects of exposure to low doses of lead in childhood: 11 year, follow-up report. *N Eng J Med* 1990; 322:83-8.
9. Amaratunga S, Sumanasena SP, Hubert HDM, Senanayake MP. Blood lead levels in children living near Borella junction. *Ceylon Medical Journal* 2001; 30:55-9.
10. Senanayake MP, Rodrigo MDA, Malkanthi R. Blood lead levels of children before and after introduction of unleaded petrol. *The Ceylon Medical Journal* 2004; 49(2):60-1.
11. Senanayake MP, Samarakkody RP, Sumanasena SP, Kudalugoda Arachchi J, Jasinghe SR, Hettiarachchi AP. A relational analysis of acute wheezing and air pollution. *Sri Lanka Journal of Child Health* 2001; 30:55-9.
12. Senanayake MP. The air we breathe: Is it safe for children? (C.C.de Silva Memorial Oration). *Sri Lanka Journal of Child Health*, 2004; 33(3) :64-72.
13. Bakir F, Damlogi S.F, Amin-Zaki L. Methylmercury poisoning in Iraq. *Science* 1973; 81:230-41.
14. American Academy of Paediatrics, Committee on Environmental Health: Mercury. In: Etzel RA, Balk SJ. editors, Handbook of Pediatric Environmental Health Elk Grove Village, IL, American Academy of Pediatrics, 1999; p 145.
15. Herbst AL, Scully RE. Adenocarcinoma of the vagina in adolescence: A report of 7 cases including 6 clear-cell carcinomas (so called mesonephromas). *Cancer* 1970; 25:745-57.
16. Mendis L. Microparasitism, macroparasitism and infectious disease. 1996 (Personal communication).
17. Centers for Disease Control: Surveillance for asthma - United States, 1960-1995. *MMWR Morb Mortal Wkly Report* 1998; 47:1022-8.
18. Goldman LR. What do I do when I see a child who may have an environmentally related illness? *Paediatric Clinics of North America* 2001; 48(5): 1085 -98.
19. Frank A, Balik SJ. Taking an exposure history: Case studies in environmental medicine. Washington DC, Department of Health and Human Services, Agency for Toxic Substances and Disease Registry, 1992.
20. Goldman R, Shannon M, Woolf A. Approach to the environmental health history. Association of occupational and environmental health clinics and the Agency for Toxic Substances and Disease Registry. (CD-ROM). 1999.
21. Lucas GN. Acute childhood poisoning in Sri Lanka: A hospital based prospective study (Presidential Address). *Ceylon Journal of Child Health* 1991; 20:4-12.
22. Senanayake MP. Noise from power generators: it's impact on the health of five children below two years of age. *Sri Lanka Journal of Child Health* 2002; 31:115-7.
23. Bongiovanni AM. An epidemic of premature thelarche in Puerto Rico. *J Pediatr* 1983; 103:245-6.
24. Carlsen E, Giwercman A, Keiding N. Evidence for decreasing quality of semen during past 50 years. *BMJ* 1992; 305:609
25. Faustman EM. Mechanisms underlying children's susceptibility to environmental toxicants, *Environ Health Perspect* 2000; 108(1):
26. Christoffel KK. Public Health Advocacy: Process and product. *Am J Public Health*, 2000; 90:722-6.