Training Module 5
Children's Environmental Health
Public Health and the Environment
World Health Organization

November 2011

<<NOTE TO USER: Please add details of the date, time, place and sponsorship of the meeting for which you are using this presentation in the space indicated.>>

<<NOTE TO USER: This is a large set of slides from which the presenter should select the most relevant ones to use in a specific presentation. These slides cover many facets of the issue. Present only those slides that apply most directly to the local situation in the region. You should replace the case studies as well as the figures with those relevant to your area and your audience>>

<<NOTE TO USER: This module presents several examples of risk factors that affect reproductive health. You can find more detailed information in other modules of the training package that deal with specific risk factors, such as lead, mercury, pesticides, persistent organic pollutants, endocrine disruptors, occupational exposures; or disease outcomes, such as developmental origins of disease, reproductive effects, neurodevelopmental effects, immune effects, respiratory effects, and others.>>

<<NOTE TO USER: For more information on reproductive health, please visit the website of the Department of Reproductive Health and Research at WHO: www.who.int/reproductivehealth/en/>>
LEARNING OBJECTIVES

To describe case studies involving toxicant exposure and the resulting effect on male reproductive health

❖ Pesticides: dichlorodiphenyltrichloroethane (DDT) and dichlorodiphenyldichloroethylene (DDE)
❖ Polychlorinated biphenyls (PCBs)
❖ Bisphenol A (BPA)

This presentation will describe specific case studies related to male reproductive health and the environment.

<<NOTE TO USER: This module will present case studies of specific exposure scenarios. Thus, if you would like more background information regarding male environmental reproductive health, please reference Module 1: Reproductive Environmental Health and Module 4: Male Environmental Reproductive Health.>>

Refs:

Reproductive health involves all of the reproductive processes, functions and systems at all stages of human life. This definition implies that people are able to have a satisfying and safe sex life and that they have the capability to reproduce and the freedom to decide if, when and how often to do so.

Male reproductive disorders affect the health status and overall quality of life of a man. Male reproductive disorders may develop during various life phases. Alterations in proper reproductive functioning may be the result of various occurrences and experiences throughout fetal development, childhood, adolescence, or adulthood.

While much is known about the male reproductive system, its development, and many causes of specific disorders, the research pertaining to the mechanisms of action for certain pathologies is still largely unknown. However, exposure to environmental contaminants has been proposed in recent years to potentially contribute to male reproductive disorders. Research has been focused on exposures that occur during critical periods of development. This is still an emerging field of research that demands greater scientific investigation.

Refs:

Image: WHO
Reproductive health and the environment focuses on exposures to environmental contaminants during critical periods of human development. These periods are directly related to reproductive health throughout the life course, including the period before conception, at conception, fertility, pregnancy, child and adolescent development, and adult health.

Environmental toxicants may potentially induce effects in human reproductive processes, specifically, for male reproductive health. However, the extent of this hypothesis must be supported through greater levels of research.

Currently, health care providers are growing increasingly aware of the potential for environmental factors to influence male reproductive health status.

Refs:
In the last 50 years the incidence of infertility, testicular and prostate cancers and associated maladies has increased significantly. Infertility now affects 15-20% of couples as opposed to 7-8% fifty years ago. Average sperm counts among adult men have decreased by 50% since 1938, with a decline of 2% every year from 1973. This decline in male reproductive health has been linked to an increased presence in the environment of man-made chemical contaminants in the form of pesticides and plastics. Rapid and unplanned industrialization caused large amounts of these synthetic compounds and their by-products to be released in the environment (air, soil, water and food). Studies have shown that occupational exposure to pesticides caused neonatal deaths, congenital defects, testicular dysfunction and male infertility. Despite vehement opposition from the plastics industry, plastics and plasticizers have been proven to cause trans-generational reproductive abnormalities and infertility. Many of these chemicals found in our environment and households have estrogenic properties “xenoestrogens” and are toxic because they affect the endocrine system “endocrine disruptors”. Endocrine disruptors have been shown to disturb the pro-oxidant/anti-oxidant system of the cells, thereby leading to generation of oxygen free radical and reactive oxygen species (ROS). If not suitably metabolized, free radicals are potentially harmful because they destabilize electrolytic balance within cells. Trace amounts of ROS are beneficial to spermatozoa and aid their capacitation, hyperactivation and acrosome reaction. But increased ROS have been shown to cause the peroxidation of polyunsaturated fatty acids (PUFA). Mammalian spermatozoa, being rich in PUFA, are more susceptible to ROS attack, and are vulnerable targets of ROS-producing polychlorinated biphenyls.

Image: WHO
The first case study that will be presented will be about the pesticide dichlorodiphenyltrichloroethane (DDT) and its effect on the reproductive health of men in South Africa.

Note: Spraying indoor walls with synthetic pesticides such as DDT, malathion or pyrethroids is an effective method to interrupt transmission and protect communities from malaria. Experts are concerned however, that incorrect application and management of DDT can have a damaging effect on the environment and health. There is also concern over increasing mosquito resistance to the pesticide. Efforts are under way to combat malaria with an incremental reduction of reliance on DDT, by supporting sustainable alternatives. These include chemical and non-chemical methods ranging from increasing distribution of insecticide-treated nets, eliminating potential mosquito breeding sites, environmental engineering, securing homes with mesh screens and deploying mosquito repellent trees and the introduction of fish that eat mosquito larvae.

Ref:
SPERM QUALITY AMONG MEN IN SOUTH AFRICA

- Men in a South African town were found to have decreased sperm quality by WHO parameters
  1. Marked by lower motility
  2. Decreased ejaculate volume
  3. High incidence of abnormal sperm cells (teratospermia)
  4. Low levels of sperm cells in semen (oligospermia)

- One similarity between the men: All lived in endemic malaria areas that were annually sprayed with the pesticide dichlorodiphenyltrichloroethane (DDT).

Ref:
The pesticide DDT [1,1,1-trichloro-2,2-bis(p-chlorophenyl)ethane or dichlorodiphenyltrichloroethane] is one of the twelve persistent organic pollutants under negotiation at the Stockholm Convention to restrict or ban their use due to their toxicity to human health and to the environment. DDT was widely used to control malaria by killing the mosquito that carries the parasite during the last half of the 20th century. However, several countries banned its use in the 1970s and 1980s because of the possibility of long-term adverse effects on human health and the environment. Several studies have proven that within the body, DDT exhibits estrogenic activity and its metabolite, DDE (dichlorodiphenyldichloroethylene), is anti-androgen. Thus, both DDT and its breakdown product have the potential to alter hormonal signaling in the human body.

**DDT: dichlorodiphenyltrichloroethane**

**DDE: dichlorodiphenyldichloroethylene**

**Refs:**

**Image:**
DDT. Available at commons.wikimedia.org/wiki/File:DDT.svg - accessed 5 August 2010. This image is public domain.
In specific regions where DDT has been found in high concentrations, there has been evidence of demasculinisation and reproductive health effects in certain male wildlife species. For example, in the state of Florida in the United States extremely high levels of DDT pollution were associated with androgynous alligators that exhibited changes in reproductive organ morphology and decreased semen quality. DDT is a potent estrogenic element and its breakdown molecule, DDE acts as an anti-androgen and may also inhibit testosterone action. It has been hypothesized that exposure to DDT may result in a variety of adverse male reproductive health endpoints. However, there are limited human studies to support this hypothesis.

**DDT**: dichlorodiphenyltrichloroethane  
**DDE**: dichlorodiphenyldichloroethylene

**Refs:**  
Both DDT and DDE are considered to be hormonally active, with DDT having estrogenic activity via binding and activation of the estrogen receptor and DDE being antiandrogenic. The physiological consequence of this hormonally active action is direct impairment of Sertoli cell function. The primary role of these cells is to support spermatogenesis. Therefore, the abnormal sperm function and structure observed may result from the affected Sertoli cells and their inability to normal spermatogenesis.

DDE could be associated with low-normal sperm motility and impaired sperm morphology, and abnormal sperm condensation. This is because anti-androgenic effects on spermatogenesis are more pronounced in the later stages of spermatogenesis or during the spermiogenic phase in which spermatids transform into spermatozoa. Any effect on this stage of development may become evident in impaired sperm motility and morphology, as seen in this group of South African males.

**DDT: dichlorodiphenyltrichloroethane**

**DDE: Dichlorodiphenyldichloroethylene**

**Refs:**

**Image: WHO**
The observed male reproductive health endpoints were subtle among men in the South African town. Any effects of DDT on sperm quality and function could have far-reaching implications.

**DDT**: dichlorodiphenyltrichloroethane  
**DDE**: dichlorodiphenyldichloroethylene

**Refs:**  

*Image: WHO*
The second case study that will be presented will be about PCBs (polychlorinated biphenyls) exposure in Taiwan.
THE YUCHENG INCIDENT IN TAIWAN

- In 1979, 2,000 individuals in central Taiwan began experiencing unusual symptoms of chloracne, peripheral neuropathy, and headache.
- Later tests disclosed further adverse health consequences, such as thyroid goiter, reproductive problems, and cognitive outcomes in their descendents.
- Symptomatic individuals consumed same type of rice oil in the region.
- Affected persons were named Yucheng or Oil-disease victims.

Refs:


Yucheng (oil-disease) victims were Taiwanese people exposed to polychlorinated biphenyls (PCBs) and their heat-degradation products, mainly polychlorinated dibenzofurans (PCDFs), from the ingestion of contaminated rice oil in 1978–1979. Serial studies in Yucheng offspring born between 1978 and 1992 are summarized. Children of the exposed women were born with retarded growth, with dysmorphic physical findings, and, during development, with delayed cognitive development, increased otitis media, and more behavioral problems than unexposed children. Recently, examination of the reproductive system has suggested that prenatal exposure exerts late effects on semen parameters in young men after puberty. Results of the investigation in Yucheng children will provide important information about the human health effects and toxicology of PCB/PCDF exposure. Prenatal exposure to these environmental chemicals causes the fetus to be sensitive to the toxic effects of persistent organic pollutants.

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Refs:

PCBs: polychlorinated biphenyls

Refs:
Polychlorinated biphenyls (PCBs) are synthetic chlorinated hydrocarbon compounds that consist of two benzene rings linked by a single carbon bond. PCBs have been produced since 1929. They have been used in plasticizers, inks, adhesives, flame retardants, pesticide extenders, and paints. Many countries have restricted or banned the production of PCBs.

PCBs adsorb strongly to soil and sediment and are persistent in the environment. They can accumulate in the food chain. Humans are commonly exposed to PCBs either by ingestion of contaminated food or water, or inhalation of contaminated air.

PCBs have been shown to cross the placenta, can be excreted in breast milk, and can accumulate in a fetus/infant.

PCBs: polychlorinated biphenyls

Refs:


Recent studies indicate that consumption of PCB-contaminated fish can cause disturbances in reproductive parameters and cause neurobehavioral and developmental deficits in newborns and older children. Prenatal exposure to PCBs from the mother’s body burden, rather than exposure through human milk, is believed to account for the developmental effects of these compounds (AAP 1999). In rhesus monkeys, exposure to PCBs is associated with alterations in the menstrual cycle, decreases in fertility, increases in spontaneous abortion, and a reduced number of conceptions (ATSDR and EPA 1998). Some of these effects have also been reported in human populations. In a study of 626 married couples in Michigan, the relative risk of conception failure (defined as an inability to conceive after 12 months) increased in men but not in women with increasing consumption of PCB-contaminated fish.

In studies on humans exposed to PCBs, it has been documented that males exposed to the compounds in utero showed adverse reproductive health effects. For example, males exposed to PCBs in utero demonstrated reduced penile length, reduced sperm motility, changes in sperm morphology, and abnormal testosterone levels. Other health effects include: lower birth weight, smaller head circumference, shorter gestational age, neuromuscular immaturity, impaired autonomic function, increased number of abnormally weak reflexes, reduced memory capacity, lower IQ scores, and attention deficit.

**PCBs:** polychlorinated biphenyls

**Refs:**
PCBs: POTENTIAL MECHANISM OF ACTION

- Polychlorinated biphenyls (PCBs) demonstrate wide range of mechanisms of action depending on chemical construction of molecule
- May be estrogenic or anti-estrogenic or anti-androgenic
  - Mixtures of PCBs may contain unknown and inconsistent quantities of both types
- Estrogenic PCBs may increase sex hormone production
  - Gonadotrophin-releasing hormone; luteinizing hormone
- May activate aryl hydrocarbon receptor receptor
  - Leads to changes in gene expression and signal transduction

PCBs: polychlorinated biphenyls

Refs:
Case Studies of Male Reproductive Environmental Health

POPULATIONS AT INCREASED RISK FOR PCBs EXPOSURE

1. Recreational and subsistence fishers

2. Individuals living near incinerators, other polychlorinated biphenyls (PCBs) disposal facilities

3. Workers in regions where PCBs are still manufactured or used and workers involved in cleanup of PCBs

PCBs: polychlorinated biphenyls

Refs:

Image: WHO
The third case study that will be presented will be about bisphenol A (BPA) exposures in China.
BPA: bisphenol A

Ref:
• Li D. Occupational exposure to bisphenol-A (BPA) and the risk of Self-Reported Male Sexual Dysfunction. Human Reproduction. 2009, 0:1–9.

BACKGROUND: Animal studies have suggested that bisphenol-A (BPA) is a potential human endocrine disrupter; but evidence from human studies is needed. METHODS: We conducted an occupational cohort study to examine the effect of occupational exposure to BPA on the risk of male sexual dysfunction. Current workers from BPA-exposed and control factories were recruited. The exposed workers were exposed to very high BPA levels in their workplace. Male sexual function was ascertained through in-person interviews using a standard male sexual function inventory. RESULTS: BPA-exposed workers had consistently higher risk of male sexual dysfunction across all domains of male sexual function than the unexposed workers. After controlling for matching variables and potential confounders, exposed workers had a significantly increased risk of reduced sexual desire (odds ratios (OR) = 3.9, 95% confidence interval: 1.8–8.6), erectile difficulty (OR = 4.5, 95% CI 2.1–9.8), ejaculation difficulty (OR = 7.1, 95% CI 2.9–17.6), and reduced satisfaction with sex life (OR = 3.9, 95% CI 2.3–6.6). A dose–response relationship was observed with an increasing level of cumulative BPA exposure associated with a higher risk of sexual dysfunction. Furthermore, compared with the unexposed workers, BPA-exposed workers reported significantly higher frequencies of reduced sexual function within 1 year of employment in the BPA-exposed factories. CONCLUSIONS: Our findings provide the first evidence that exposure to BPA in the workplace could have an adverse effect on male sexual dysfunction.

Image: Halshka Graczyk. Copyright permission granted.
Researchers compared the rates of sexual dysfunction in two groups of workers in China: men who worked at factories that produce bisphenol A or epoxy and compared their sexual health to workers not exposed to abnormally high levels of bisphenol A. Epoxy resin is used in the lining of canned foods and is another potential source of bisphenol A. A positive association was observed between a worker's exposure to bisphenol A and his likelihood of experiencing sexual dysfunction. It is important to note that the dysfunction was apparent even in workers who had worked in a bisphenol A factory for one year or less.

Men who worked in the bisphenol A and epoxy-resin factories were exposed to levels about 50 times higher than the average levels of human exposure.

BPA: bisphenol A

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Bisphenol A (BPA) is an industrial chemical that is used as an ingredient for the production of polycarbonate plastics and epoxy resins. It can be found in many consumer goods around the world, including baby bottles, plastic containers, and metal-based food and beverage cans. Polycarbonate is a hard clear plastic that is identified by the “other plastics” category for recycling, designated by a triangle with the number 7. Bisphenol A also may be added to other kinds of plastics in many consumer goods.

**BPA: bisphenol A**

*Refs:*

It is believed that most human populations throughout the world experience continuous exposure to bisphenol A. This is directly related to the widespread presence of bisphenol A within plastic consumer good that are used on a daily basis. Bisphenol A can leach from plastics and resins into the food and beverages that humans consume. Therefore, humans can be directly exposed to this chemical compound via ingestion.

The graph on the slide demonstrates the level of bisphenol A found in foods and beverages in the United States. However, due to globalized commercial networks, bisphenol A exposure pattern may be similar for other nations.

**BPA: bisphenol A**

Ref:

Animal studies show that bisphenol A directly alters the male reproductive system. These studies have determined that bisphenol A exhibits both estrogenic and antiandrogenic activity in the body. By directly working on estrogen and androgen receptors in the male reproductive system, bisphenol A is believed to alter male sex hormone levels, and the development and proper functioning of the male reproductive organs, including the testes, epididymus, sperm and seminal vesicles. In addition, animal studies have also demonstrated that bisphenol A may affect male sexual behavior, such as erectile function, probably through changes in male sex hormone levels.

BPA: bisphenol A

Refs:
Recent animal studies have documented that even very low doses of bisphenol A exhibited significant weight gain in male offspring, advanced sexual maturation and an altered sexual differentiation process. In addition, prenatal exposure to low dose bisphenol A increased ano-genital distance and prostatic size in the newborn male, caused malformation in the urethra, as well as decreased epididymal weight. Adult animals exposed to low doses of bisphenol A exhibited decreased sperm quality and quantity, and enlarged prostates. Fetal exposure to low doses of bisphenol A induced a decrease in uterine weight associated with increased DNA synthesis within the endometrial glandular epithelium and an endometrial expression of both estrogen receptors and progesterone receptors.

BPA: bisphenol A

Ref:
Maffini MV et al. Endocrine disruptors and reproductive health: The case of bisphenol A. Molecular and Cellular Endocrinology. 2006, 25:179-186. There is some evidence to suggest that these effects were mediated through increased expression of androgen receptor in the prostate stroma and BPA induced disruption of cell differentiation in the peritubular stroma. A recent study also revealed that BPA induced an overall increase in prostate duct volume due to an increase in the proliferation of basal epithelial cells. The authors postulated that the reported findings may impact on fertility, and may have led to changes in the age of male reproductive maturity, and to the onset of disease later.
The precautionary principle is a response to uncertainty, given a risk to health or the environment. It involves pre-emptive acting to avoid serious or irreversible potential harm despite lack of scientific certainty as to the likelihood, magnitude, or causation of that harm. These measures are strictly precautionary.

**BPA: bisphenol A**

*Ref:
THE PRECAUTIONARY PRINCIPLE

First, there is a great need for more scientific research to fill the gaps in our knowledge.
Second, while waiting for a more complete understanding we must find ways to make decisions that are based on the best available evidence, while acknowledging the uncertainties that remain. Thus, there is no contradiction between pursuing scientific progress and taking precautionary action.


The concepts of precaution and prevention have always been at the heart of public health practice. Public health is inherently about identifying and avoiding risks to the health of populations, as well as about identifying and implementing protective measures. In the past, public health interventions focused on removing hazards that had already been identified and “proven” (even if the etiological mechanisms were not well understood). As “modern” potential risk factors become more complex and farreaching, the precautionary principle addresses uncertain risks and seeks to shift the ways in which science informs policy from a strategy of “reaction” to a strategy of “precaution.” Together with related approaches such as health impact assessment, precaution provides a useful means of guiding public health decisions under conditions of uncertainty, in a manner that appropriately addresses the issues of power, ownership, equity and dignity. The precautionary principle encourages policy-makers and public health professionals to consider, in their approach to public health, how to account for growing complexity and uncertainty. Substantial evidence supports the conclusion that contemporary environmental health risks result from complex interactions among genetic, nutritional, environmental and socioeconomic factors. The precautionary principle can be used to encourage research, innovation and cross-disciplinary problem-solving in the face of these complex risks. It serves as a guide for considering the effects of human activities and provides a framework for protecting humans, other species and lifesustaining ecological systems now and in the future. The precautionary principle is occasionally portrayed as contradicting the tenets of sound science and as being inconsistent with the norms of “evidence-based” decision-making. These criticisms might be based on effective misuse of the precautionary principle, but it is nevertheless important to clarify the role of environmental science in policy-making. Many pressing environmental crises share a fundamental characteristic: they appear to arise from disruptions of natural systems or cycles, the behaviour of which is only partially understood. Two conclusions may be drawn. First, there is a great need for more scientific research to fill the gaps in our knowledge. Second, while waiting for a more complete understanding we must find ways to make decisions that are based on the best available evidence, while acknowledging the uncertainties that remain. Thus, there is no contradiction between pursuing scientific progress and taking precautionary action. Indeed, applying precaution demands more rigorous science in order to characterize complex risks, clarify gaps in knowledge and identify early warnings and unintended consequences of actions. It also means using science not only for the diagnosis of environmental hazards but to identify, develop and assess safer alternatives to potentially harmful activities. Countries whose economies are in transition have special environment and health problems. The consequences of past pollution, economic hardship, poor or even deteriorating public health and the demands of rapid political, social and economic change pose additional problems for decision-makers. In such countries economic priorities may outweigh the need to protecting health. The precautionary principle is thus very important here, because it can inform decisions under the great uncertainty that prevails, can help build public confidence, can raise research and innovation capacities, can ensure that mistakes made in the past in industrialized countries are not repeated, and can help shift burdens from the public institutions to those creating the risks. There is no single recipe for applying precaution. Applying precaution should encourage decision makers to use the broadest possible range of information, including stakeholders’ views, and to examine alternative courses of action. Flexibility in applying precaution is critically important, since each decision is different – with different types of risk, evidence, uncertainty, affected communities, availability of alternatives, and technical and financial resources. Consistency thus comes from using the same precautionary framework and process in each case. What is considered an “acceptable risk” or sufficient evidence to act is a function not only of the level of risk and the strength of evidence and uncertainty, but also of the magnitude, reversibility and distribution of the risk, the availability of opportunities to prevent risk, the public’s risk aversion, society’s culture and values, and the pros and cons of alternative options. These preventive precautionary actions ultimately aim at continuously reducing and if possible removing exposures to potentially harmful substances, activities and other conditions. If progress is to be made in this direction, one should encourage the replacement of dangerous substances and activities with less dangerous substances or technologies where suitable alternatives are available, reconsider production processes, products and human activities so as to minimize significant adverse effects on health and the environment, for example through the use of integrated pest management strategies, land use planning and cleaner production, establish public health goals for protecting the health of humans and ecosystems (such as for reducing blood lead levels or improving fisheries); provide information and education to the public to promote empowerment and accountability; integrate precautionary considerations into the research agenda to facilitate rapid interventions to prevent damage to health; and minimize, so far as possible, unintended adverse consequences that may be caused by precautionary actions. Notes from WHO/EURO. The precautionary principle: protecting public health, the environment and the future of our children. Martuzzi M, Ficurseri u eds. WHO, Europe, 2004.
Bisphenol A (BPA) is an industrial chemical that is widely used in the production of polycarbonate (PC) plastics (used in food contact materials, such as baby bottles and food containers) and epoxy resins (used as protective linings for canned foods and beverages and as a coating on metal lids for glass jars and bottles). These uses result in consumer exposure to bisphenol A via the diet.

Although a large number of studies on the toxicity and hormonal activity of bisphenol A in laboratory animals have been published, there have been considerable discrepancies in outcome among these studies with respect to both the nature of the effects observed as well as the levels at which they occur. This has led to controversy within the scientific community about the safety of bisphenol A, as well as considerable media attention.

In light of uncertainties about the possibility of adverse human health effects at low doses of bisphenol A, especially on reproduction, the nervous system and behavioural development, and considering the relatively higher exposure of very young children compared with adults, the Food and Agriculture Organization of the United Nations (FAO) and the World Health Organization (WHO) jointly organized an Expert Meeting in late 2010 to assess the safety of bisphenol A. The Expert Meeting concluded that:

- For many end-points, points of departure are much higher than human exposure. Hence, there is no health concern for these end-points.
- Studies on developmental and reproductive toxicity in which conventional end-points were evaluated have shown effects only at high doses, if at all.
- However, some emerging new end-points (sex-specific neurodevelopment, anxiety, preneoplastic changes in mammary glands and prostate in rats, impaired sperm parameters) in a few studies show associations at lower levels.
- The points of departure for these low-dose effects are close to the estimated human exposure, so there would be potential for concern if their toxicological significance were to be confirmed.
- However, it is difficult to interpret these findings, taking into account all available kinetic data and current understanding of classical estrogenic activity. However, new studies indicate that bisphenol A may also act through other mechanisms.
- There is considerable uncertainty regarding the validity and relevance of these observations. While it would be premature to conclude that these evaluations provide a realistic estimate of the human health risk, given the uncertainties, these findings should drive the direction of future research with the objective of reducing this uncertainty.

**BPA: bisphenol A**

**Ref:**
Case Studies of Male Reproductive Environmental Health

EXAMPLES OF PREVENTION MEASURES IN THE US

❖ Avoid plastic containers numbered 3, 6, or 7 especially for baby bottles and infant feeding cups
❖ Avoid heating plastics

With your food, use 4, 5, 1 and 2. All the rest aren’t good for you.

Safer choices for foods and beverages

Avoid

PETE HDPE LDPE PP

This is specifically important for consumer products intended for infants (baby bottles, teethers, toys, etc.)

Ref:


Case Studies of Male Reproductive Environmental Health

POINTS FOR DISCUSSION

<<NOTE TO USER: Add points for discussion according to the needs of your audience.>>
ACKNOWLEDGEMENTS

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