The Latest Science on Bisphenol A, Health and Exposure – January to July 2010

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Preface

This compilation of the most recent published scientific studies of human exposure to and health effects linked to bisphenol A was prepared by Toxics Action Center as part of our ongoing work with the Alliance for a Clean and Healthy Maine and organizing in communities around the state to protect human health from dangerous chemicals.

Bisphenol A and its impacts on human health is an expansive field of scientific research. This document provides a compilation of the studies published in scientific literature between January 2010 and July 2010 concerning exposure to and health impacts linked to BPA. This compilation is meant to be comprehensive within these narrow parameters – a snapshot of the most recent results in a vast field of research. Of the 81 scientific studies included in this compilation, 75 conclude that humans are exposed to BPA or that there are one or more adverse health impacts associated with exposure to BPA.

About Toxics Action Center

Toxics Action Center provides assistance to residents working to prevent or clean up toxic hazards in their communities. Since 1987, Toxics Action Center has helped more than 650 communities clean up hazardous waste sites, decrease industrial pollution, curb pesticide spraying, and oppose the siting of dangerous waste, energy and industrial facilities. When the government won't take action and the company denies that there is a problem, Toxics Action Center is a resource for residents concerned with toxic hazards in their communities. We provide residents with information about environmental laws, strategies for organizing, a network of activists throughout the state, and access to legal and technical experts. Toxics Action Center is funded by donations from concerned citizens and grants from private foundations. This financial support enables us to provide our services free of charge to communities facing the threats of toxic pollution. Find out more at www.toxicsaction.org.

About the Alliance for a Clean and Healthy Maine

The Alliance for a Clean and Healthy Maine is a diverse coalition of Maine-based organizations, including Toxics Action Center, engaged in a public health campaign to phase out the long-lived toxic chemicals that build up in the food web and our bodies. Through a series of strategic issue campaigns, The Alliance is targeting sources of persistent toxic chemicals to be replaced with safer alternatives. Over time, we will seek government and business commitments to phase out the entire class of persistent toxics in favor of clean production. The organizations committed to the campaign represent health-affected children, workers, doctors, public health professionals, environmentalists and impacted communities. Together we are committed to protecting human health from toxic chemical exposure and building organizational capacity to sustain environmental health improvements. Working collectively in Maine, we can set the pace nationally for policy development and best practices that end the dangerous use of persistent toxic chemicals. Find out more at www.cleanandhealthyme.org.

THE BASICS OF BISPHENOL A (BPA): THE CHEMICAL AND ITS USES

Bispenol A (BPA) is a synthetic chemical used to make polycarbonate plastics, which are commonly used in consumer products such as baby bottles, sippy cups, reusable water bottles, athletic equipment, and medical and dental equipment. BPA is also used to make the epoxy resin lining many food cans and found on the back of many point-of-sale receipts.

In 2009, global production of BPA was estimated at over 2.2 million tons.[i] The most common producers of BPA in the United States are Bayer MaterialScience, Dow Chemical Company, SABIC Innovative Plastics (formerly GE Plastics), Hexion Speciality Chemicals, and Sunoco Chemicals.[ii]

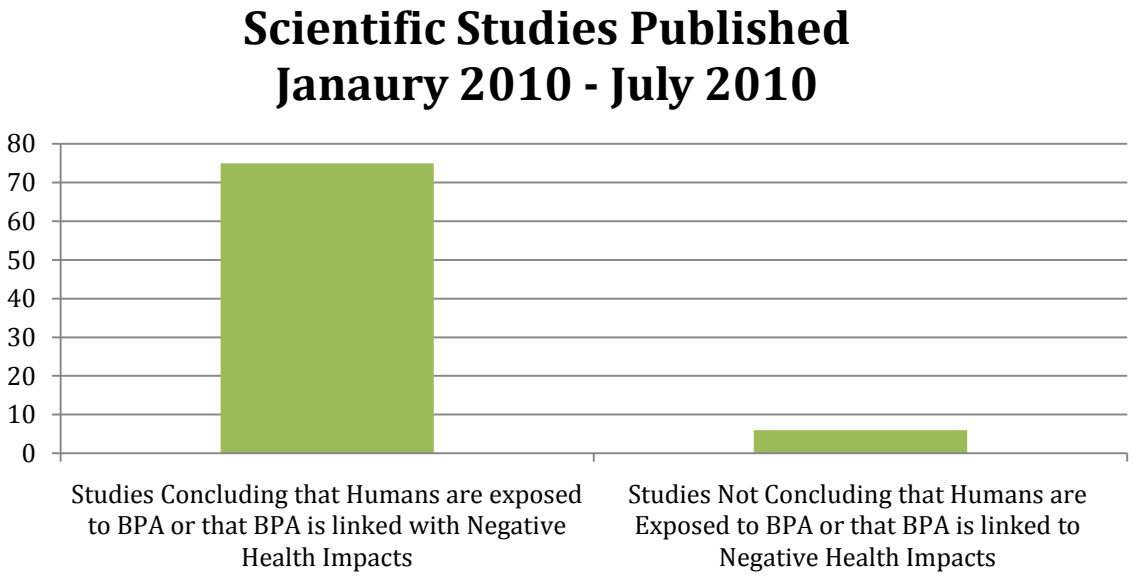
METHODOLOGY OF STUDY COMPILATION

BPA and its impacts on human health is an expansive field of scientific research. This document provides a compilation of the studies published in scientific literature between January 2010 and July 2010 concerning the exposure to and health impacts of BPA. This list is meant to be comprehensive within these narrow parameters – a snapshot of the most recent results in an extensive area of research.

We used six online search engines to compile the studies included in this document. We did two Google Scholar searches, one for "Bisphenol A" + "human" and one for "Bisphenol A" + "health effects." We used the Yale University Library to search for articles. We used Pubmed.gov, a database maintained by the National Library of Medicine at the National Institutes of Health. We used the MEDLine (Medical Literature Analysis and Retrieval System) which has online access to journals on medicine, nursing, pharmacy, dentistry, veterinary medicine, biology, biochemistry, and health care. We used the Directory of Open Access Journals which has over 2,000 journals searchable on the article level. Lastly, we searched articles from the Vermont Law School on-line library.

FINDINGS

Of the 81 scientific studies included in this compilation, 75 conclude that humans are exposed to BPA or that there are one or more adverse health impacts associated with exposure to BPA.



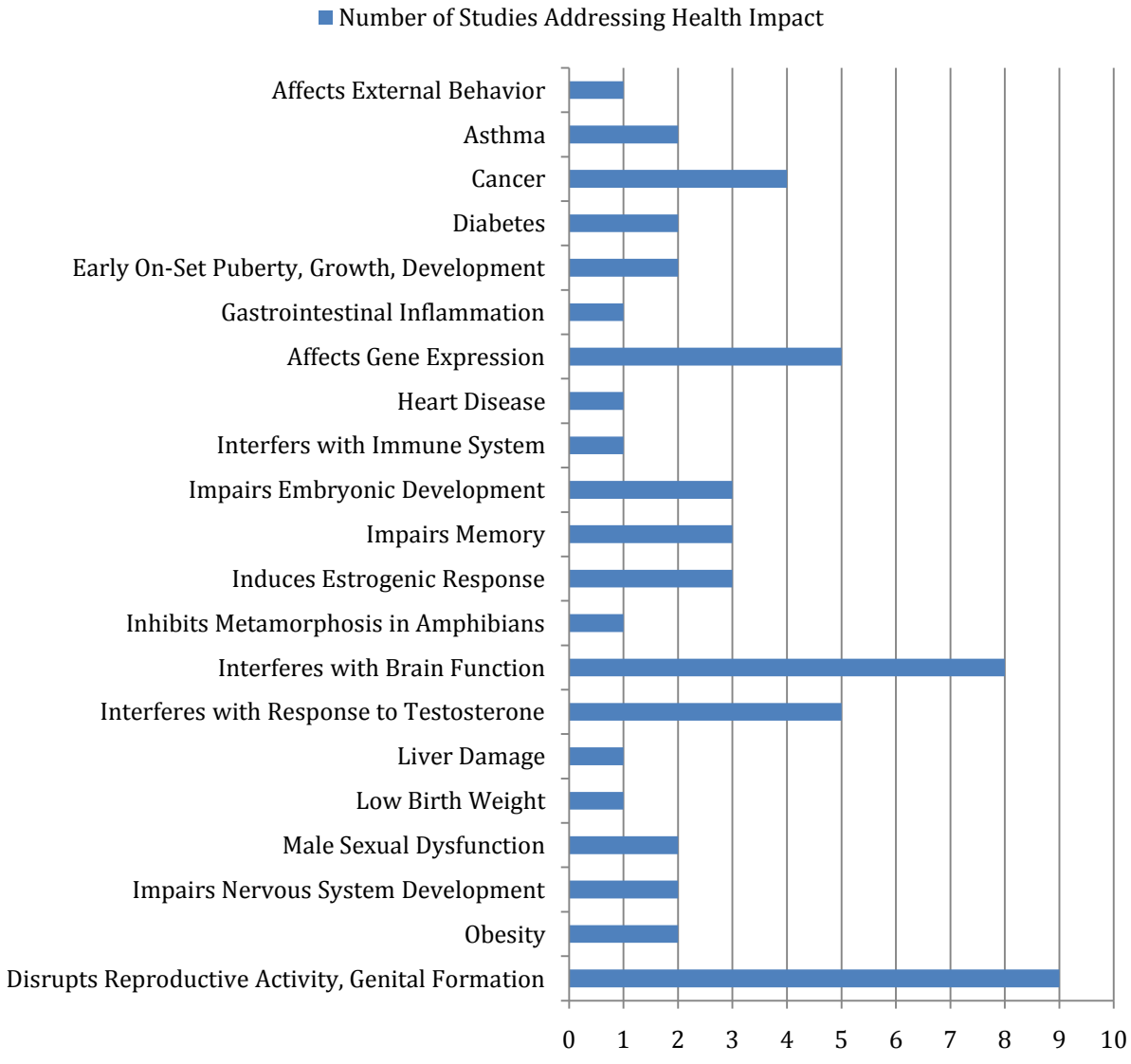
Together, those 75 studies indicate that BPA is associated with early on-set puberty, diabetes, disruptions to growth hormones and developmental programming, different types of cancer, disruptions to gene expression, changes in external behavior, memory loss, interference with response to testosterone, male sexual dysfunction, heart disease, interference with brain function, impaired reproductive activity and genital formation, impaired embryonic development, obesity, impaired nervous system development, liver damage, interference with the immune system, asthma, low birth weight, and gastrointestinal inflammation. Some of the studies also indicate that BPA is transferred from mother to fetus and from mother to infant in breast milk. Others document that BPA leaches from food storage containers into food or liquid, and that the rate of contamination increases with time and temperature. One study indicates that humans may be exposed to BPA from dental sealants in their mouths.

More than 200 scientific studies have linked BPA to known health effects.

There are six 2010 studies that did not find links between BPA and negative effects on human health, or evidence of BPA leaching from food containers into food or liquid. One concludes that while BPA seemed to cause obesity in baby mice, the obesity did not carry into adulthood. Four studies found no evidence of a specific health impact that was the focus of the study linked to exposure to BPA (changes in luteinizing hormone secretion, sexually dysmorphic behavior, early on-set puberty, fertility, anatomy, or evidence that BPA is a developmental neurotoxin). The final study concluded that BPA migrates from cans in "very low amounts." At least one of these six studies - *Developmental Neurotoxicity Study of Dietary Bisphenol A in Sprague-Dawley Rats* - was conducted by the American

Chemistry Council, Dow Chemical, Bayer Pharma, and Sunoco, Inc. These companies and organization all have financial incentives to keep BPA present in the market.

Seventy-Five 2010 Studies Conclude that Humans are Exposed to BPA or that BPA Negatively Impacts Health



FINDING: HUMANS ARE REGULARLY EXPOSED TO BPA

Because of its widespread use and previous extensive research, we already know that human exposure to BPA is ubiquitous. Most people are exposed to BPA when we eat foods or liquids that have been stored in packaging made from or containing BPA. Small children are susceptible to direct exposure to BPA when they put materials containing BPA into their mouths.[iii] In the *Fourth National Report on Human Exposure to Environmental Chemicals*, the Centers for Disease Control and Prevention (CDC) scientists measured BPA in the urine of 2,517 participants aged six years and older who took part in CDC's National Health and Nutrition Examination Survey during 2003–2004. By measuring BPA in urine, scientists can estimate the amount of BPA that has entered peoples' bodies. CDC scientists found BPA in more than 90% of the urine samples representative of the U.S. population.[iv]

FINDING: BPA IS LINKED TO SERIOUS HEALTH IMPACTS

Scientific research indicates that exposure to BPA can seriously harm human health and behavior. BPA is an endocrine disruptor that mimics estrogen in the body. The results of the research presented in this compilation build on an already clear trend. According to a 2007 38-member expert panel sponsored by the U.S. National Institute of Environmental Health Sciences, "recent trends in human diseases relate to adverse effects observed in experimental animals exposed to low doses of BPA."[v] In March 2010, The U.S. Environmental Agency declared BPA a chemical of concern. In April 2010, the President's Cancer Panel also warned the public to avoid BPA.[vi]

"The current consensus of most scientists, as well as the U.S. and international governmental agencies, is that there is sufficient evidence that BPA produces adverse [health] effects at environmentally relevant exposures."

– Maine Center for Disease Control and Preventions letter to Maine Department of Environmental Protection Commissioner David Littell. May 13, 2010.

GROWING PUBLIC CONCERN AND ACTION

We all want to live in safe and healthy homes where our children can thrive. Growing scientific evidence points to an undeniable trend: the vast majority of scientists share the consensus that human exposure to BPA is bad for our health. Public concern about the presence and effects of BPA is following suit. Vermont, Maryland, Minnesota, Connecticut, New York, Wisconsin, and Washington have all successfully banned BPA in certain consumer products, along with a number of other cities and counties.

BPA AND MAINE'S KID-SAFE PRODUCTS ACT

In April 2008 in Maine, Governor John Baldacci signed into law LD 2048, *An Act To Protect Children's Health and the Environment from Toxic Chemicals in Toys and Children's Products*. The law requires Maine to adopt a list of Chemicals of High Concern already proven to be hazardous and name Priority Chemicals for immediate action, requires manufacturers to disclose use of Priority Chemicals in their products, and authorizes the state to require use of safer alternative chemicals if they are available and affordable.

Under the law, if a prioritized chemical can harm the health of exposed children and there is a safer alternative available, the hazardous chemical should be phased out of use in consumer products. The law:

- **Covers Many Products:** "Children's products" are defined broadly to include both products intentionally marketed for children (like toys) and any other consumer product containing that may expose a child or fetus through use or disposal of the product.

- **Lists Chemicals of High Concern:** The State of Maine was required to publish a list of Chemicals of High Concern by January 1, 2010. The State published this list with over 1,700 chemicals, including BPA, in June 2009.[vii] These chemicals have all been identified as known or likely carcinogens, reproductive or developmental toxicants or endocrine disruptors; persistent, bioaccumulative and toxic (PBT); or very persistent and very bioaccumulative (vPvB); based on credible scientific evidence.

- **Identifies Priority Chemicals:** By January 1, 2011, the State is required to designate at least two chemicals of high concern (or groups of similar chemicals) as Priority Chemicals based on triggering of one or more exposure-related criteria. The State proposed BPA as the first Priority Chemical in June 2010.[viii]

- **Notifies Consumers:** Not later than six months after a Priority Chemical is named, a manufacturer or distributor must report to the State on which of their products contain the priority chemical, the number of units sold, the amount of the chemical and its purpose. The State is also granted authority to require manufacturers or distributors to submit information on the likelihood of chemical releases from the product and any biomonitoring or environmental effects data, and to prepare an alternatives assessment on the availability, cost, feasibility, performance and safety of alternatives to the priority chemical.

- **Can Require Safer Alternatives:** The State is authorized to restrict the sale of a product for specific uses if it contains a Priority Chemical, if they find that distribution of the product directly or indirectly exposes children and vulnerable populations to the priority chemical, and that one or more safer alternatives to the priority chemical are available at a comparable cost. Rulemaking and approval by the Legislature are required.

RECOMMENDATIONS

The most recent science on BPA published from January to July 2010 corroborates the already extensive literature documenting widespread human exposure to BPA and linking BPA to a variety of health impacts. It's time to act on the overwhelming scientific evidence and move aggressively to protect our children from this dangerous hormone disrupting chemical by replacing it with safer alternatives.

Ban BPA in Consumer Products Sold in Maine

The State of Maine should immediately ban BPA in all consumer products sold in Maine for which there is a safer alternative to BPA available. The best science shows that BPA is harming our children. Safer alternatives to BPA for many uses are widely available and already in use. Broad government and public support exists for immediate action on BPA. Many other states, local governments, and companies have already moved away from BPA. Maine should follow suit with a broad ban on BPA in consumer products.

Continue to Implement the Kid-Safe Products Act Thoroughly and Quickly

In May 2010, the Alliance for a Clean and Healthy Maine released a report identifying more than 40 chemicals that meet all the criteria to be named "Priority Chemicals" under the Kid-Safe Products Act and targeted for immediate action. Once a chemical is named as a Priority Chemical, manufacturers are required to disclose their use of these dangerous chemicals in products and research safer alternatives. The public has the right to know which products contain any one of these potentially dangerous chemicals, and know that manufacturers are looking for safer alternatives. Maine should act swiftly to target these 40 worst-of-the-worst chemicals for immediate action.

Reform the Federal Toxic Substance Control Act (TSCA)

With each new scientific report linking toxic chemical exposure to a serious health problem, it becomes more obvious that the national law intended to keep harmful chemicals in check — the Toxic Substances Control Act (TSCA) of 1976 — is not working. By updating TSCA, Congress can create the foundation for a sound and comprehensive chemicals policy that protects public health and the environment, while restoring the luster of safety to U.S. goods in the world market. To be effective, TSCA reform should take immediate action on the most dangerous chemicals, hold industry responsible for the safety of their chemicals and products, and use the best science to protect all people and vulnerable groups. For more information, see www.saferchemicals.org.

SCIENTIFIC STUDIES ON BPA, HEALTH, AND EXPOSURE JANUARY 2010 – JULY 2010

A Mouritsen, L. Aksglaede, K. Sorensen, S. Sloth Mogensen, H. Leffers, K.M. Main, H. Frederiksen, A.M. Anderssn, N.E. Skakkebaek, and A. Juul, Hypothesis: exposure to endocrine-disrupting chemicals may interfere with timing of puberty, March 2010, International Journal of Andrology, Volume 33, Issue 2: 346-359.

This study proposed that BPA, along with other endocrine-disrupting chemicals, contributes to the growing trend of early on-set puberty.

Alonso-Megdalena P, Vieira E., Soriano S., Menes L., Burks D., Quesada I., and Nadal A, Bisphenol – A Exposure during Pregnancy Disrupts Glucose Homeostasis in Mothers and Adult Male Offspring, Available online: May 2010, Environmental Health Perspectives, http://www.ncbi.nlm.nih.gov/pubmed/20488778.

The study suggests BPA may contribute to metabolic disorders and may also act as a risk factor for diabetes.

Angela M Betancourt, James A. Mobley, Jose Russo, and Coral A Lamartiniere, Proteomic analysis in mammary glands of rat offspring exposed in utero to bisphenol A, April 2010, Journal of Proteomics, Volume 73, Issue 6: 1241-1253.

The study aids in the understanding of how BPA may be increasing the mammary gland's susceptibility to cancer transformation.

Baowei Jiao and Christopher H.K. Cheng, Disrupting actions of bisphenol A and malachite green on growth hormone receptor gene expression and signal transduction in seabream, June 2010, Fish Physiology and Biochemistry, Volume 36, Number 2: 251-261.

This study suggests that BPA affects gene expression and growth hormone receptors in fish.

Biju Balakrishnan, M., Kimiora Henare, Eric Thorstensen, Anna Ponnampalam, Murray Mitchell, Transfer of Bisphenol A across the Human Placenta, April 2010, American Journal of Obstetrics and Gynecology, Volume 202, Issue 4: 393.

The study found that BPA at low levels transfers across the placenta to the fetus in humans.

Bitna Yi, Changsung Kim and Mihi Yang, Biological monitoring of bisphenol A with HLPC/FLD and LC/MS/MS assays, Available online: February 2010, Journal of Chromatography. http://www.ncbi.nlm.nih.gov/pubmed/20202916.

The study found infants are exposed to BPA through their mother's breast milk.

Bromer J.G, Zhou, Taylor M.B., Doherty L., Taylor H.S., Bisphenol-A exposure in utero leads to epigenetic alterations in the developmental programming of uterine estrogen response,

Available on-line: February 2010, Federation of American Societies for Experimental Biology Journal, http://www.fasebj.org/cgi/content/abstract/fj.09-140533v1.

This study identified mechanisms by which BPA administered to mice in utero altered their developmental programming.

Bruan, J.M, Yolton K., Dietrich K.N., Hornung R., Ye X., Calafat A.M., and Lanphear B.P., Prenatal bisphenol A exposure and early childhood behavior, December 2010, Environmental Health Perspectives, 117(12): 1945-1952.

A study of prenatal BPA exposure in 249 mothers and their children in Cincinnati, Ohio suggest that exposure alters externalizing behavior in 2-year-old children, especially females.

Bryce C. Ryan, Andrew K. Hotchkiss, Kevin M. Crofton, and L. Earl Gray, Jr., In Utero and Lactational Exposure to Bisphenol A, In Contrast to Ethinly Estradiol, Does Not Alter Sexually Dimorphic Behavior, Puberty, Fertility, and Anatomy of Female L.E. Rats, 2010, Toxicological Sciences, Volume 114, Number 1: 133-148.

A study of the effects of BPA on rats exposed in utero showed no evidence of sexually dismorphic behavior, early on-set puberty, fertility, and anatomy.

Cao X.L., Corriveau J., Popovic S., Migration of bishpenol A from can coatings to liquid formula during storage at room temperature, December 2010, Journal of Food Protection, 72(12): 2571-1574.

A study of BPA migration in liquid infant formula cans stored at room temperature showed levels increased by 30-110% when stored for 10 months.

Carjone Rosa Goncalves, Raqual Wigg Cunha, Daniela Marti Barros, and Pablo Elias Martinez, Effects of prenatal and postnatal exposure to a low does of Bisphenol A on behavior and memory in rats. Available online: July 2010, Environmental Toxicology and Pharmacology, http://www.sciencedirect.com/science?_ob=ArticleURL&_udi=B6T6D-50G067H-1&_user=10&_coverDate=07%2F05%2F2010&_rdoc=1&_fmt=high&_orig=search&_sort=d&_docanchor=&view=c&_searchStrId=1412316463&_rerunOrigin=scholar.google&_acct=C000050221&_version=1&_urlVersion=0&_userid=10&md5=c5a89c56f04f8c66898e46a7976732a2.

The study suggests exposure to BPA impairs both short term and long-term memory in rats.

Csaba Leranth, Klara Szigeti-Buck, Neil J. MacLusky, and Tibor Hajszan, Bisphenol A Prevents the Synaptogenic Response to Testosterone in the Brain of Adult Male Rats, January 2010, Endocrinology, Volume 149, Number 3: 988-994.

The study presents evidence that BPA interferes with the adult male rat brain's response to testosterone.

D. Li, Z. Zhou, D. Quing, Y. He, T. Wu, M. Miao, J. Wang, X. Weng, J.R. Ferber, L.J. Herrinton, Q. Zhu, E. Gao, H. Checkoway, and W. Yuan, Occupational exposure to bisphenol A and the risk of

Self-Reported Male Sexual Dysfunction, 2010, Human Reproduction, Volume 25, Number 2: 519-527.

An occupational study of males exposed to BPA at work showed evidence that exposure is tied to male sexual dysfunction.

Daichi Nakamura, Yukie Yanagiba, Zhiwen Duan, Yuki Ito, Ai Okamura, Nobuyuki Asaeda, Yoshiaki Tagawa, ChunMel Li, Kazuyoshi Taya, Shu-Yun Zhang, Hisao Naito, Doni Mikmat Ramdhan, Michihiro Kamijima, and Tamie Nakajima, Bisphenol A may cause testosterone reduction by adversely affecting both testis and pituitary systems similar to estradiol, April 2010, Toxicology Letters, Volume 194, Issues 1-2: 16-25.

Bisphenol A directly affected the Leydig cells, the pituitary gland, and resulted in decreased testosterone in male rats.

David Melzer, Neil E. Rice, Ceri Lewis, William E. Henley, and Tamara S. Golloway, Association of Urinary Bisphenol A concentration with Heart Disease: Evidence from NHANES 2003/2006. Available online: January 2010, Public Library of Science, www.plosone.org/article/info%3Adoi%2F10.1371%2Fjournal.pone.0008673.

The study concluded that higher BPA exposure in humans is consistently connected with heart disease.

De-Kun Li, ZhiJun Zhou, Maohua Miao, Yonghua He, Dandan Qing, Tongjun Wu, JinTao Wang, Xiaoping Weng, Jeannette Ferber, Lisa Herrinton, Qianxi Zhu, ErSheng Gao, and Wei Yuan, Relationship between Urine Bisphenol-A Level and Declining Male Sexual Function, Available online May 2010, Journal of Andrology, http://www.andrologyjournal.org/cgi/content/abstract/jandrol.110.010413v1.

A sample of 427 male workers showed worsening sexual function is associated with increasing urine BPA levels on a continuous scale.

Dieldrich S. Bermundez, Leon E. Gray, Jr. and Vickie S. Wilson, Modeling the Interaction of Binary and Tertiary Mixtures of Estradiol with Bisphenol A and Bisphenol AF in an In Vitro Estrogen-Mediated Transcriptional Activation Assay, May 2010, Toxicological Sciences, 116(2): 477-487.

The study found that high levels of BPA induced estrogenic response.

Donald G. Stump, Mellissa J. Beck, Ann Radovsky, Robert H. Garman, Lester L. Freshwater, Larry P. Sheets, M. Sue Marty, John M. Waechter, Jr., Stephen S. Diamond, John P. Van Miller, Ronald N. Shitsuka, Dieter Beyer, Anne H. Chappelle, and Steven G. Hentges, Developmental Neurotoxicity Study of Dietary Bisphenol A in Sprague-Dawley Rats, Published on-line February 2010, Toxicological Sciences http://toxsci.oxfordjournals.org/cgi/content/abstract/kfq025v1.

This study found no evidence that BPA is a developmental neurotoxicant in rats.

F. Grasselli, L. Baratta, L. Baioni, S. Bussolati, R. Ramoni, S. Grolli, G. Basini, Bisphenol A disrupts granulose cell function, July 2010, Domestic Animal Endocrinology, Volume 39, Issue 1: 34-39.

The study found that BPA interferes with reproductive activity by affecting the granulose cell.

Funda Yigit, and Suzan Daglioglu, Histological changes in the uterus of the hens after embryonic exposure to bisphenol A and diethylstil bestrol, Available online: April 2010, Protoplasma, http://www.ncbi.nlm.nih.gov/pubmed/20393758.

A study of BPA in chicken eggs found that administration in high doses negatively affected embryonic development.

G.H.M. Biego, A.S.S. Oga, K.D. Yao, and L.P. Kouadio, Evaluation of bisphenol A content in food from lacquered cans, 2010, Chemical Society of Ethiopia, Volume 24, No. 2.

The study found that BPA migration from cans of food increases with storage duration, temperature of the storage, and the temperature of sterilization. The study also found that such concentrations may lead to toxicological effects on the consumer.

Giuseppe Letini, Franscesco Gallo, and Lorenz Iughetti, Toxic environment and obesity pandemia: Is there a relationship?, January 2010, Italian Journal of Pediatrics, Volume 36.

The study suggests that environmental chemicals such as BPA may be associated with the obesity epidemic.

Haitao Zhu, Jicui Zheng, Xianmin Xiao, Shan Zheng, Kuiran Dong, Jiangbin Lui, and Yang Wang, Environmental endocrine disruptors promote invasion and metastasis of SK-N-SH human neuroblastoma cells, 2010, Onocology Reports, Volume 23, Number 1: 129-139.

The results suggest that BPA exposure may contribute to the incidence of neuroblastoma, a form of pediatric cancer.

Hyunseok Cho, Seung Jun Kim Hye-Won Park, Moon-Ju Ph, So Yeon Yu, Seung Yong Lee, Chansoo Park, JaeRyul Han, Jung-Hwa Oh, Seung Yong Hwang, and Seok-Joo Yoon, A relationship between miRNA and gene expression in the mouse Sertoli cell line after exposure to bisphenol A, March 2010, BioChip Journal, Volume 4, Number 1: 75-81.

The study found that BPA exposure in mice resulted in changed gene expression.

Izzotti, A, Longbardi M., Cartiglia C., D'Agostini, F. Kanitz S., and De Flora S., Pharmacological Modulation of Genome and Proteome Alterations in Mice Treated with the Endocrine Disruptor Bisphenol A. March 2010, Current Cancer Drug Targets, Volume 10, Number 2: 147-154.

Administration of BPA to mice resulted in DNA adducts (cancer-starting cells) forming in the liver and mammary tissue.

Jakub Kwintkiewicz, Yoshihiro Nishi, Toshihiko Yanase, and Linda C. Giudice, Peroxisome Proliferator-Activated Receptor-Y Mediates Bishpenol A inhibition of FSH-Stimulated IGF-1, Aromatase, and Estradiol in Human Granulosa Cells, March 2010, Environmental Health Perspectives. 118(3): 400-406.

The study supports the theory that BPA alters the ovarian follicular compartment because of its role as an endocrine disruptor.

Ji Young Kim, Eun Hee Han, Hyung Gyum Kim, Kyo Nyeo Oh, Sang Kyum Kim Kwang Yeol Lee, and Hye Gwang Jeong, Bisphenol A-induced aromatase activation is mediated by cyclooxygenase-2 Up-regulation in rat testicular Leydig cells, March 2010, Toxicology Letters, Volume 193, Issue 2: 200-208.

The results of the study suggest that BPA increases aromatase activity in rat testicular Leydig cells.

Ji Young Kim, Eun Hee Han, Hyung Gyun Kim, Kyo Nyeo Oh, Sang Kyum Kim, Kwang Yeol Lee, and Hye Gwang Jeong, Bisphenol A-induced aromatase activation is mediated by cyclooxygenase-2 up-regulation in rat testicular Leydig cells, January 2010, Toxicology Letters, Volume 193, Issue 2: 200-208.

The results suggest that BPA increases aromatase activity in rat testicular Leydig cells.

Jingjing Wang, Xiaolin Lui, Houpeng Wang, Tingting Wu, Xiaoqi Hu, Fang Qin, and Zaizhao Wang, Expression of two cytochrome P450 aromatase genes is regulated by endocrine disrupting chemicals in rare minnow *Gobiocypris rarus* juveniles, September 2010, Toxicology and Pharmacology, Volume 152, Issue 3: 313-320.

The study suggests the BPA suppresses gene expression in rare minnow Gobiocypris rarus.

Joaquim Maia, Jose Manuel Cruz, Raquel Sendon, Juana Bustos, Maria Eugenia Cirrgeda, Jose Juan Sanchez, and Perfecto Paseiro, Effect of amines in the release of bisphenol A from polycarbonate baby bottles, June 2010, Food Research International, Volume 43, Issue 5: 1283-1288.

This study examines methods by which BPA is able to migrate from polycarbonate baby bottles into food.

Jorge M. Naciff, Zubin S. Khambatta, Timothy D. Reichling, Gregory J. Carr, Jay P. Tiesman, David W. Singleton, Sohaib A. Khan, and George P. Daston, The genomic response of Ishkawa cells to bisphenol A exposure is dose-and time-dependant, April 2010, Toxicology, Volume 270, Issues 2-3: 137-149.

The study found 2,794 genes are changed by BPA in a dose and time-dependant manner.

Joyce M. Zimmerman Downs, Deanne Shuman, Sharon C. Stull, and Robert E. Ratzlaff, Bisphenol A Blood and Saliva Levels Prior to and After Dental Sealant Placement in Adults, June 2010, Journal of Dental Hygiene, Volume 84, Number 3: 145-150.

BPA was found in dental sealants and the study suggests it leaches after oral work is completed.

Karen K. Ryan, April M. Haller, Joyce E. Sorrell, Stephen C. Woods, Ronald J. Jandacek, and Randy J. Seeley, Perinatal Exposure to Bisphenol-A and the Development of Metabolic Syndrome in CD-1 Mice, Endocrinology, Volume 151, Number 6: 2603-2612.

When the hypothesis that BPA leads to obesity and glucose intolerance in adult mice was tested, this study found that weanling mice exposed to BPA during gestation and lactation were heavier and longer than the control group, but this data did not translate into differences once the mice reached adulthood.

Katsushiro Okuda, Masufumi Takiguchi, and Shin'ichi Yoshihara, In Vitro estrogenic potential of 4-methyl-2,4-bis(4-hydroxphenyl)pent-1-ene, an active metabolite of bisphenol A, in uterus of ovariectomized rat, August 2010, Toxicology Letters, Volume 197, Issue 1: 7-11.

The study suggests that in vitro exposure to BPA in rats causes estrogenic activity.

Kazushi Okada, Shoko Hashimoto and Susumu Imaoka, Biological Functions of Protein Disulfide Isomerase as a target of Phenolic Endocrine-disrupting Chemicals, 2010, Journal of Health Science, Volume 56, Number 1: 1-13.

The study found evidence that BPA acts as a neural toxicant in the rat brain.

Kembra L. Howdeshell, and Ferderick S. vom Saal, Developmental Exposure to Bisphenol A: Interaction with Endogenous Estradiol During Pregnancy in Mice, June 2010, The Society for Integrative and Comparative Biology, Volume 40, Number 3: 429-437.

The findings suggest that BPA alters the course of development in the human fetus.

Ken Okabayshi, Toshi Wantanabe, Excretion of bisphenol A into rat milk, March 2010, Toxicological Mechanisms and Methods, Volume 20, Number 3: 133-136.

A study of BPA migration from mother mice to their pups through milk indicated that BPA exposure through polycarbonate containers bio-accumulates and is likely to be transferred to pups in a more concentrated form.

Kenichiro Ishil, Shigeki Arase, Yuko Yoshio, Katsuhide Igarashi, Kenichi Alsaki, Yasuhide Hoir, Kohel Nishikawa, Norihito Soga, Hideaki Kise, Kiminobu Arima, Jun Kanno, and Yoshiki Sugimura, 373 Biological Effects of Fetal Exposure to Bisphenol A On Urogenital Sinus, March 2010, Journal of Urology, Volume 183, Issue 4, Supplement 1: 148.

The study found that exposure to BPA had many effects on the urogenital sinus during fetal development.

Kuo-Ching Wang, Ta-Liang Chen, Cheng-Hong Qin, Yung-Feng Lin, and Chen-Ho Chen, Bisphenol-A interferes with estradiol-medicated protection in osteo-chondrocytes, available on-line June 2010, Toxicology Letters, http://www.sciencedirect.com/science?_ob=ArticleURL&_udi=B6TCR-50DW3TF-1&_user=10&_coverDate=06%2F30%2F2010&_rdoc=1&_fmt=high&_orig=search&_sort=d&_docanchor=&view=c&_searchStrId=1412134067&_rerunOrigin=scholar.google&_acct=C000050221&_version=1&_urlVersion=0&_userid=10&md5=cc8de39e0c699457f794e751dba9edd7.

The study found that BPA interferes with estrogen functions in chondrocytes and may promote osteoarthritis.

Kwintkiewicz J., Nishi Y., Yanase T., and Guidice L.C., Peroxisome proliferator-activated receptor-gamma mediates bisphenol A inhibition of FSH-stimulated IGF-1, aromatase, and estradiol in human granulose cells, March 2010, Environmental Health Perspective, 118(3): 400-406.

The data indicated that BPA may alter steriodogenesis and proliferation in the ovarian follicular compartment.

Lina Xing, Yajun Xu, Yang Xiao, Lanquin Shang, Ran Liu, Xuetao Wei, Jianjun Jiang, and Weidong Hoa, Embryotoxic and teratogenic effects of the combination of bisphenol A and geinstein on in vitro cultured postimplantation rat embryos, March 2010, Toxicological Sciences, Volume 115, Number 2: 577-588.

A study of BPA and GEN showed a higher frequency of abnormal nervous system development in rat embryos.

M.A. Verner, T. Magher, and S. Haddad, High concentrations of commonly used drugs can inhibit the in vitro glucuronidation of bisphenol A and nonylphenol in rats, February 2010, Xenobiotica, Volume 40, Number 2: 83-92.

The study found that BPA detoxification is impaired by pharmaceuticals, and further stated that a health risk assessment should be undertaken for further examination.

M.F.L. Lemos, C.A.M. van Gestel, and A.M.V.M. Soares, Reproductive toxicity of the endocrine disruptors vinclozolin and bisphenol A in the terrestrial isopod *Porcellio scaber* (Latreille, 1804), February 2010, Chemosphere, Volume 78, Issue 7: 907-913.

The study suggests BPA levels in the rough woodlouse result in decreased female reproduction and may even lead to population decline.

Marina Olga Fernandez, Nadia Bouruignon, Victoria Lux-Lantos, and Carlos Libertun, Neonatal Exposure to Bisphenol and Reproductive and Endocrine Alterations Resembling the Polycystic Ovarian Syndrome in Adult Rats, Environmental Health Perspectives, Available online: April 2010
http://ehsehplp03.niehs.nih.gov/article/fetchArticle.action?articleURI=info%3Adoi%2F10.1289%2Fehp.0901257

Exposing neonatal rats to BPA resulted in an increased risk of ovarian morphology, a large number of cysts, and reduced fertility.

Masami Ishido and Junko Suzuki, Quantitative Analysis of Inhibitory Effects of Bisphenol A on Neural Stem-Cell Migration Using a Neurosphere Assay in vitro, January 2010, Journal of Health Science, Volume 56, Number 2: 175-181.

This study quantifies the neurodevelopmental toxicity of BPA and found that exposure inhibits cell migration and decreases proliferative cells in rats.

Meeker J.D., Ehrlich Toth, T.L. Wright, D.L. Calafat, A.M. Trisini, A.T. Ye, and X. Hauser, Semen Quality and Sperm DNA Damage in Relation to Urinary Bisphenol A among Men from an

Infertility Clinic, Available online: July 2010, Reproductive Toxicology, http://www.ncbi.nlm.nih.gov/pubmed/20656017.

A study of 190 men recruited at an infertility clinic showed that urinary BPA may be associated with lower quality of semen and increased DNA damage in sperm.

Meghan M. Mahoney, and Vasantha Padmanabhan, Developmental programming: Impact of fetal exposure to endocrine-disrupting chemicals on gonadotropin-releasing hormone and estrogen receptor mRNA in sheep hypothalamus, Available online: June 2010, Toxicology and Applied Pharmacology, http://www.sciencedirect.com/science?_ob=ArticleURL&_udi=B6WXH-507BHJY-1&_user=10&_coverDate=06%2F04%2F2010&_rdoc=1&_fmt=high&_orig=search&_sort=d&_docanchor=&view=c&_searchStrId=1414538085&_rerunOrigin=scholar.google&_acct=C000050221&_version=1&_urlVersion=0&_userid=10&md5=534c0433ffaf36ee63e3f44a334894a1.

The study found that sheep fetuses exposed to BPA show altered neural development and result in long-term reproductive difficulties for adult females when those fetuses mature.

Mendiola J., Jorgensen N., Andersson A.M., Calafat A.M., Ye X, Redmon J.B., Drobnis E.Z., Wang C., Sparks A., Thurson S.W., Lui F., and Swan S.H, Are Environmental Levels of Bisphenol A Associated with Reproductive Function in Fertile Men?, Available online: May 2010, Environmental Health Perspectives, http://www.ncbi.nlm.nih.gov/pubmed/20494855.

The results suggest that men exposed to low environmental levels of BPA have a modest reduction in markers of free testosterone as a result.

Michele Avissar-Whiting, Keila R. Veiga, Kristen M. Uhl, Matthew A. Maccani, Luc A. Gagne, Erika L. Moen, Carmen J. Marsit, Bisphenol A exposure leads to specific microRNA alterations in placental cells, July 2010, Reproductive Toxicology, Volume 29, Issue 4: 401-406.

The results of the study suggested that BPA alters miRNA expression in placental cells.

Miyu Nishikawa, Hidetomo Iwano, Risa Yanagisawa, Nanako Koike, Kiroki Inoue, and Hiroshi Yokota, Placental Transfer of Conjugated Bisphenol A and Subsequent Reactivation in the Rat Fetus, Available online: April 2010, Environmental Health Perspectives, http://ehp03.niehs.nih.gov/article/info%3Adoi%2F10.1289%2Fehp.0901575.

The study's results demonstrate that BPA conjugatestransfer from rat mothers to pups and the BPA becomes deconjugated (released) in the fetus due to its vulnerable drug metabolizing system.

Neelakanteswar Aluru, John F. Leatherland, and Mathilakath M. Vijayan, Bisphenol A in Oocytes leads to Growth Suppression and Altered Stress Performance in Juvenile Rainbow Trout, May 2010, Public Library of Science, Volume 5.

The study suggests that BPA present in water bio-accumulates in fish eggs and the result is delayed hatching, growth suppression, and altered stress response in juvenile trout.

P. Vinas, N. Campilla, N. Martinez-Castillo, and Hernandez-Cordoba, Comparison of two derivatization-based methods for solid-phase microextraction-gas chromatography-mass spectrometric determination of Bisphenol A, bisphenol S and bisphenol migrated from food cans, May 2010, Analytical and Bioanalytical Chemistry, Volume 397, Number 1: 115-125.

The study found that BPA migrates from cans in very low amounts.

Paloma Alonso-Magdalena, Ana Belin Ropero, Sergi Soriano, Ivan Quesada, Angel Nadal, Bisphenol-A: A new diabetogenic Factor?, Hormones, 2010, 9(2): 118-126.

A study of rodents found BPA has a profound effect on glucose metabolism, and this alteration may lead to a greater risk of developing type 2 diabetes.

Peitro G. Signorile, Enrico P. Spugnini, Luigi Mita, Pasquale Mellone, Alfeedo D'Avino, Mariangela Bianco, Nadia Diano, Lucia Cuputo, Fransceca Rea, Rosa Viceconte, Marianna Portaccio, Emanuela Viggiano, Gennaro Citro, Riccardo Pierantoni, Vincenzo Sica, Bruno Vincenzi, Damiano G. Mita, Feliciano Baldi, and Alfonso Baldi, Prenatal exposure of mice to bisphenol A elicits an endometriosis-like phenotype in female offspring, Available online March 2010, General and Comparative Endocrinology, http://www.ncbi.nlm.nih.gov/pubmed/20350546.

The study found that BPA exposure during development causes long-lasting effects on the genital system of mice.

Poimenova, E. Markaki, C. Rahiotis and E. Kitraki, Corticosterone-regulated actions in the rat brain are affected by perinatal exposure to low dose of bisphenol A, February 2010, Neuroscience, Volume 167, Issue 3: 741-749.

A University of Athens study concluded that corticosterone in the brain is sensitive to the programming effect of BPA at does below the currently acceptable daily intake level for Greek citizens.

Rachel A. Heimeier, and Yun-Bo Shi, Amphibian metamorphosis as a model for studying endocrine disruption on vertebrate development: Effect of bisphenol A on thyroid hormone action, Available online: February 2010, General and Comparative Endocrinology, http://www.ncbi.nlm.nih.gov/pubmed/20178801.

The findings suggest that BPA inhibits metamorphosis in amphibians and argues more analysis is needed to determine whether BPA has similar effects on human embryogenesis.

Robert G. Berger, Warren G. Foster, and Denys deCatanzaro, Bisphenol-A exposure during the period of blastocyst implantation alters uterine morphology and perturbs measures of estrogen and progesterone receptor expression in mice, Available online: July 2010, Reproductive Toxicology, http://www.sciencedirect.com/science?_ob=ArticleURL&_udi=B6TC0-50FGY3J-1&_user=10&_coverDate=07%2F03%2F2010&_rdoc=1&_fmt=high&_orig=search&_sort=d&_docanchor=&view=c&_searchStrId=1412317539&_rerunOrigin=scholar.google&_acct=C000050221&_version=1&_urlVersion=0&_userid=10&md5=8bf99701fa2e98a52e81df3caee7b0f5.

The results indicate that BPA exposure during gestation affects the uterus to disrupt intrauterine implantation.

Satoru Sakuma, Massahiko Nakanishi, Kazuhiro Morinaga, Mihoyo Fujitake, Shun-ichi Wada, and Yohko Fujimoto, Bisphenol A 3,4-quinone induces the conversion of xanthine dehydrogenase into oxidase in vitro, June 2010, Food and Chemical Toxicology, Volume 48, Issues 8-9: 2217-2222.

The study suggests that BPA 3,4-quinonein the liver and may lead to oxidative DNA damage there.

Severine H. Collet, Nicole Picard-Hagen, Catherine Viguie, Marlene Z. Lacroix, Pierre-Louise Toutain, and Veronique Gayard, Estrogenicity of bisphenol A: a concentration-effect relationship on luteinizing hormone secretion in a sensitive model of prepubertal lamb, Available online: June 2010, Toxicological Sciences, http://toxsci.oxfordjournals.org/cgi/content/abstract/kfq186v1.

Seven weeks of treating lamb with BPA showed no accumulation, and no evidence of alterations. The results showed the disrupting dose in lamb is 50-fold higher than the currently recommended tolerable daily intake value of humans in France.

Shingo Matsuda, Shizuko Saika, Keiko Amano, Eiji Shimizu, and Junko Sajiki, Changes in brain monoamine levels in neonatal rats exposed to bisphenol A at low doses, February 2010, Chemosphere, Volume 78, Issue 7: 894-906.

The study presents evidence that BPA, even at low doses, has an impact on the brain of neonatal rats.

Shinichi Asano, Jonathan D. Tune, and Gregory M. Dick, Bisphenol A activates Maxi-K channels in coronary smooth muscle, March 2010, British Journal of Pharmacology, Volume 10, Issue 1: 160-170.

The study found that BPA increases Maxi-K channel activity and concluded that this may represent toxicological effects.

Steven D. Holladay, Shuo Xiao, Honglu Diao, Jamie Barber, Tomas Ngy, Xiaoqin Ye, and Robert M. Gogal, Preinatal Bisphenol A Exposure in C57B6/129svj Male Mice: Potential Altered Cytokine/Chemokine Production in Adulthood, 2010, International Journal of Environmental Research and Public Health, Volume 7, Issue 7: 2845-2852.

The study suggests that exposing pregnant mice to BPA causes shifts in cytokines in male offspring.

Sung-Hyun-Nam, Young-Min Seo, and Man-Goo Kim, Bisphenol A migration from polycarbonate baby bottles with repeated use, February 2010, Chemosphere, Volume 79, Issue 9: 949-952.

The study of BPA migration from polycarbonate baby bottles showed increases based on both time and temperature.

Susan M. Rankin, and Evan M Grosjean, Effects of Bisphenol A in the ring-legged earwig, Euborellia annulipes, April 2010, Ecotoxiology, Volume 19, Number 4: 635-642.

The study found that 6 day-old earwigs exposed to BPA presented inhibited testis and ovarian growth.

Takashi Iwakura, Makiko Iwafuchi, Daisuke Muraoka, Makoto Yokosuka, Takashi Shiga, Chiho Watanabe, and Ritsuko Ohtani-Kaneko, In vitro effects of bisphenol A on developing hypothalamic neurons, June 2010, Toxicology, Volume 272, Issues 1-3: 52-58.

A study of BPA in developing hypothalamic neurons showed that BPA decreased protein levels of synapsin, a protein essential for the neurotransmission of synapses.

Terumi Midoro-Horiuti, Ruby Tiwari, Cheryl S. Watson, and Randall M. Goldblum, Maternal Bisphenol A Exposure Promotes the Development of Experimental Asthma in Mouse Pups, February 2010, Environmental Health Perspectives: 118(2): 273-277.

The study concluded that perinatal exposure to BPA enhanced the animal model of asthma.

Thit J. Morck, Gluseppina Sorda, Micoletta Bechi, Brian S. Rasmussen, Jesper B. Nielsen, Francesca Letta, Erik Rytting, Line Mathiesen, Luana Paulesu, and Lisbeth E. Knudsen, Placental transport and in vitro effects of Bisphenol A, August 2010, Reproductive Toxicology, Volume 20, Issue 1: 131-137.

The study showed that fetal exposure to BPA occurs through the placenta, and poses risks to fetal development.

Tibor Hajszan, Csaba Leranth, Bisphenol A interferes with synaptic remodeling, Frontiers in Neuroendocrinology, July 2010, Yale University School of Medicine, Science Direct. http://www.sciencedirect.com/science?_ob=ArticleURL&_udi=B6WFS-50G6W5F-2&_user=10&_coverDate=07%2F06%2F2010&_rdoc=1&_fmt=high&_orig=search&_sort=d&_docanchor=&view=c&_searchStrId=1409964843&_rerunOrigin=scholar.google&_acct=C000050221&_version=1&_urlVersion=0&_userid=10&md5=2b46fba1d48993f170a39a3187e41bce

BPA was found to negatively affect the nervous system, resulting in the loss of spine synapses in rats and non-human primates. This could result in cognitive decline, depression, and schizophrenia.

Toyoko Hiroi, Kazushi Okada, Susumu Imaoka, Mayuko Osada, and Yoshihiko Funae, Bisphenol A Binds to Protein Disulfide Isomerase and Inhibits Its Enzymatic and Hormone-Binding Activities, January 2010, Endocrinology, Volume 147, Number 6: 2773-2780.

The study reports that BPA binds to the protein PDI and this pairing is likely to influence hormonal activity in several ways.

V. Padmanabhan, H.N. Sarma, M. Savableasfahani, T.L. Steckler, and A. Velga-Lopez, Developmental reprogramming of reproductive and metabolic dysfunction in sheep: native steroids vs. environmental steroid receptor modulators, January 2010, International Journal of Andrology, Volume 33, Issue 2: 394-404.

The study found that exposing mother sheep to BPA resulted in low birth weight of offspring.

Veronica L. Bosquiazzo, Jorgelina Varayoud, Monica Munos-de-Toro, Enrique H. Luque, and Jorge G. Ramos, Effects of Neonatal Exposure to Bisphenol A on Steroid Regulation of Vascular Endothelial Cell Proliferation in the Adult Rat Uterus, January 2010, Biology of Reproduction, Volume 82, Number 1: 86-95.

The study suggests that neonatal exposure to BPA has a negative effect on female fertility.

Viorica Braniste, Aurore Jouault, Eric Gaultier, Arnaud Polizzi, Claire Buisson-Brenac, Mathilde Leveque, Pascal G. Martin, Vassillia Theodorou, Jean Fioramonti, and Eric Houdeau, Impact of oral bisphenol A at reference doses on intestinal barrier function and sex differences after perinatal exposure in rats, January 2010, Proceedings of the National Academy of Sciences of the United States of America, Volume 107, Number 1: 448-453.

The study suggests that BPA influences intestinal barrier function, gut nociception, and can result in severe inflammation in adult female rats.

Von Goetz N., Wormuth M., Scheringer M., Hungerbuhler, K., Bisphenol A: How the most relevant exposure sources contribute to total consumer exposure, March 2010, Risk Analysis, 30(3): 473-487.

The study found that the highest BPA exposure in infants came from bottles, while adults are exposed at the highest levels from canned food. The study went on to say that the median levels of BPA found in humans are below the tolerable daily intake level but higher than the levels at which health effects were found in rodents at the same concentrations.

Xiao-hong Xu, Jing Zhang, Ya-min Wang, Yin-ping Ye, and Qing-qing Luo, Perinatal exposure to bisphenol-A impairs learning-memory by concomitant down-regulation of N-methyl-D-aspartate receptors of hippocampus in male offspring mice, July 2010, Hormones and Behavior, Volume 58, Issue 2: 326-333.

The results of the study show that prenatal BPA exposure in mice affects behavior and development of spatial and avoidance memory in adulthood.

Xiao-Hong Xu, Ya-Min Wang, Jing Zhang, Qing-Qing Luo, Yin-Ping Ye, and Qin Ruan, Perinatal exposure to bisphenol-A changes N-methyl-D-asparate receptor expression in the hippocampus of male rat offspring, 2010, Environmental Toxicology and Chemistry, Volume 29, Issue 1: 176-181.

The data suggests that BPA exposure affects the brain development of male rats.

Xiaohui Lui, Ayami Matsushima, Hiroyuki Oknada, and Yasuyuki Shimohigashi, Distinction of the binding modes for human nuclear receptor ERR between bisphenol A and 4-hydroxytamoxifen, Available online: June 2010, Journal of Biochemistry, http://jb.oxfordjournals.org/cgi/content/abstract/mvq056v1.

The study found that BPA binds to human estrogen-related receptor ERR and acts as an endocrine disruptor even at low doses.

Xie X, Wng X., Xu X, Sun H, Chen X. Investigation of the interaction between endocrine disruptor Bisphenol A and human serum albumin, May 2010, Chemosphere, available on-line May 2010, U.S. National Library of Medicine National Institutes of Health, http://www.ncbi.nlm.nih.gov/pubmed/20510433

In this study, BPA was found to alter the secondary structure of the protein human serum albumin (the most abundant protein found in human blood) upon interaction.

Xu Li, Guang-Guo Ying, Hoa-Chang Su, Xiao-Bing Yang, and Li Wang, Simultaneous determination and assessment of 4-nonylphenol, bisphenol A, and triclosan in tap water, bottled water, and baby bottles, May 2010, Environmental International, Volume 36, Issue: 557-562.

The study found no negative human health effects from BPA exposure in drinking water.

Y. Nakajima, R.M. Goldblum, and T. Midoro-Horiuti, Does Response of Maternal Exposure to Bisphenol A on the Development of Experimental Asthma in Mouse Pups?, February 2010, Journal of Allergy and Clinical Immunology, Volume 125, Issue 2, Supplement 1: 127.

The study suggests that maternal exposure to BPA leads to increased development of asthma in mouse pups.

Yu-Hua Tian, Joung-Hee Baek, Seok-Yong Lee, and Choon-Gon Jang, Prenatal and postnatal exposure to bisphenol a induces anxiolytic behaviors and cognitive deficits in mice, February 2010, Synapse, Volume 64, Issue 6: 432-439.

The study found that exposing mice to BPA daily resulted in behavioral alterations and lead to changes in memory and anxiety level.

Yun-jung Yang, Shin-young Lee, Kyung-yong Kim, and Yeon-pyo Hong, Acute Testis Toxicity of Bisphenol A Diglycidyl Ether in Sprauge-Dawley Rats, March 2010, Journal of Preventative Public Health, Volume 32: 131-137.

A study of male rats exposed to single doses of BPA suggests that oral exposure affects testis development.

Endnotes

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