

Litteraturgennemgang for perioden april 2015 – juni 2015

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Humane studier ved Afd. for Vækst og Reproduktion, Rigshospitalet

Søgning er udført på PubMed og dækker perioden 10. marts 2015 - 30. juni 2015

Følgende søgeprofil er benyttet:

Bisphenol A
Phthalat*
Paraben*
(perfluor* OR polyfluor*)
Triclocarban
Triclosan
(Flame retardant)
tributyltin
endocrine disrupters

kombineret med nedenstående tekst:

AND expos* AND (human OR men OR women OR child* OR adult* OR adolescen* OR infan*)

Limits: title/abstract, English language

Udvalgte publikationer

DDT Exposure in Utero and Breast Cancer

J Clin Endocrinol Metab. 2015 Jun 16;jc20151841. [Epub ahead of print]

Cohn BA, La Merrill M, Krigbaum NY, Yeh G, Park JS, Zimmermann L, Cirillo PM

CONTEXT: Currently no direct evidence links in utero dichlorodiphenyltrichloroethane (DDT) exposure to human breast cancer. However, in utero exposure to another xenoestrogen, diethylstilbestrol, predicts an increased breast cancer risk. If this finding extends to DDT, it could have far-reaching consequences. Many women were heavily exposed in utero during widespread DDT use in the 1960s. They are now reaching the age of heightened breast cancer risk. DDT exposure persists and use continues in Africa and Asia without clear knowledge of the consequences for the next generation.

HYPOTHESIS: In utero exposure to DDT is associated with an increased risk of breast cancer.

DESIGN: This was a case-control study nested in a prospective 54-year follow-up of 9300 daughters in the Child Health and Development Studies pregnancy cohort (n = 118 breast cancer cases, diagnosed by age 52 y and 354 controls matched on birth year).

SETTING AND PARTICIPANTS: Kaiser Foundation Health Plan members who received obstetric care in Alameda County, California, from 1959 to 1967, and their adult daughters participated in the study.

MAIN OUTCOME MEASURE: Daughters' breast cancer diagnosed by age 52 years as of 2012 was measured.

RESULTS: Maternal o,p'-DDT predicted daughters' breast cancer (odds ratio fourth quartile vs first = 3.7, 95% confidence interval 1.5-9.0). Mothers' lipids, weight, race, age, and breast cancer history did not explain the findings.

CONCLUSIONS: This prospective human study links measured DDT exposure in utero to risk of breast cancer. Experimental studies are essential to confirm results and discover causal mechanisms. Findings support classification of DDT as an endocrine disruptor, a predictor of breast cancer, and a marker of high risk.

Transdermal Uptake of Diethyl Phthalate and Di(n-butyl) Phthalate Directly from Air: Experimental Verification

Weschler CJ, Bekö G, Koch HM, Salthammer T, Schripp T, Toftum J, Clausen G.

Environ Health Perspect. 2015 Apr 7. [Epub ahead of print]

BACKGROUND: Fundamental considerations indicate that, for certain phthalate esters, dermal absorption from air is an uptake pathway that is comparable to or larger than inhalation. Yet this pathway has not been experimentally evaluated and has been largely overlooked when assessing uptake of phthalate esters.

OBJECTIVES: This study investigated transdermal uptake, directly from air, of diethyl phthalate (DEP) and di(n-butyl) phthalate (DnBP) in humans.

METHODS: In a series of experiments, six human participants were exposed for six hours in a chamber containing deliberately elevated air concentrations of DEP and DnBP. The participants either wore a hood and breathed air with phthalate concentrations substantially below those in the chamber or did not wear a hood and breathed chamber air. All urinations were collected from initiation of exposure until 54 hours later. Metabolites of DEP and DnBP were measured in these samples and extrapolated to parent phthalate intakes, corrected for background and hood air exposures.

RESULTS: For DEP the median dermal uptake directly from air was 4.0 $\mu\text{g}/(\mu\text{g}/\text{m}^3)$ in air compared with an inhalation intake of 3.8 $\mu\text{g}/(\mu\text{g}/\text{m}^3)$ in air. For DnBP the median dermal uptake from air was 3.1 $\mu\text{g}/(\mu\text{g}/\text{m}^3)$ in air compared with an inhalation intake of 3.9 $\mu\text{g}/(\mu\text{g}/\text{m}^3)$ in air.

CONCLUSIONS: This study shows that dermal uptake directly from air can be a meaningful exposure pathway for DEP and DnBP. For other semivolatile organic compounds (SVOCs) whose molecular weight and Kow are in the appropriate range, direct absorption from air is also anticipated to be significant.

Relationship between urinary concentrations of di(2-ethylhexyl) phthalate (DEHP) metabolites and reproductive hormones in polyvinyl chloride production workers

Fong JP, Lee FJ, Lu IS, Uang SN, Lee CC.

Occup Environ Med. 2015 May;72(5):346-53.

OBJECTIVES: We investigated the relationship between urinary metabolites of di(2-ethylhexyl) phthalate (DEHP) and reproductive hormones in workers of polyvinyl chloride (PVC) production plants. After exposure, most of the DEHP is rapidly metabolised to mono(2-ethylhexyl) phthalate (MEHP), mono(2-ethyl-5-hydroxyhexyl) phthalate (MEHHP) and mono(2-ethyl-5-oxohexyl) phthalate (MEOHP), which may be associated with reproductive hormone interruption and testicular toxicity. Some studies report that urinary concentrations of phthalate metabolites for plastics workers are significantly higher than for the general population. However, little is known about the disruption of reproductive hormones for DEHP exposure workers.

METHODS: This cross-sectional study of 82 male workers measured the biomarkers for their reproductive hormones and their exposure to DEHP. Relationships between urinary concentrations of DEHP metabolites were estimated using multivariate linear regression and quartile analysis models.

RESULTS: The geometric means of urinary creatinine-adjusted ($\mu\text{g/g-Cre}$) concentrations of MEHP, MEOHP and MEHHP during the post-shift period were 23.9, 66.9 and 84.6, respectively. In multiple regression models adjusted for potential confounders, there were significant positive associations between urinary concentrations of DEHP metabolites and estradiol (E2) ($p < 0.01$), and in the ratio of E2 to testosterone ($p < 0.05$). Moreover, quartile analysis showed significant positive relationships between the total urinary concentration of DEHP metabolites and E2 (ptrend=0.024), and in the ratio of E2 to testosterone (ptrend=0.031).

CONCLUSIONS: Relationships between reproductive hormones and the total urinary concentration of DEHP metabolites in male PVC production workers were significantly positive. This indicated that aromatase activity had increased in male workers exposed to DEHP, which is consistent with animal studies.

Prenatal and postnatal exposure to phthalate esters and asthma: a 9-year follow-up study of a Taiwanese birth cohort.

Ku HY, Su PH, Wen HJ, Sun HL, Wang CJ, Chen HY, Jaakkola JJ, Wang SL; TMICS Group.

PLoS One. 2015 Apr 13;10(4):e0123309. doi: 10.1371/journal.pone.0123309. eCollection 2015.

Previous studies have shown that phthalate exposure in childhood is associated with the development of respiratory problems. However, few studies have assessed the relative impact of prenatal and postnatal exposure to phthalates on the development of asthma later in childhood. Therefore, we assessed the impact of prenatal and postnatal phthalate exposure on the development of asthma and wheezing using a Taiwanese birth cohort. A total of 430 pregnant women were recruited, and 171 (39.8%) of them had their children followed when they were aged 2, 5, and 8 years. The International Study of Asthma and Allergies in Childhood questionnaire was used to assess asthma and wheezing symptoms and serum total immunoglobulin E levels were measured at 8 years of age. Urine samples were obtained from 136 women during their third trimester of pregnancy, 99 children at 2 years of age, and 110 children at 5 years. Four common phthalate monoester metabolites in maternal and children's urine were measured using liquid chromatography-electrospray ionization-tandem mass spectrometry. Maternal urinary mono-benzyl phthalate [MBzP] concentrations were associated with an increased occurrence of wheezing in boys at 8 years of age (odds ratio [OR] = 4.95 (95% CI 1.08-22.63)), for upper quintile compared to the others) after controlling for parental allergies and family members' smoking status. Urinary mono-2-ethylhexyl

phthalate [MEHP] levels over the quintile at 2-year-old were associated with increased asthma occurrence (adjusted OR = 6.14 (1.17-32.13)) in boys. Similarly, the sum of di-2-ethyl-hexyl phthalate [DEHP] metabolites at 5 years was associated with asthma in boys (adjusted OR = 4.36 (1.01-18.86)). Urinary MEHP in maternal and 5-year-old children urine were significantly associated with increased IgE in allergic children at 8 years. Prenatal and postnatal exposure to phthalate was associated with the occurrence of asthma in children, particularly for boys.

Bruttoliste

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In vitro studier ved DTU Fødevarerinstitutionen

Søgt i Pubmed med følgende kriterier:

"Endocrine disrupt* AND in vitro*" samt "Endocrine disrupt* AND expose* AND in vitro*",

"Paraben* AND in vitro*,"perfluor* OR polyfluor* AND in vitro*" og "Phthalat* AND in vitro*".

Publiceret fra i perioden 2015/03/31 to 2015/12/31 (April 2015 og fremefter)

Efter at have fjernet genganger fra forrige litteraturopdateringslister gav litteratursøgningen, med de angivne søgekriterier, tilsammen en liste med i alt 48 artikler. Artiklerne er blevet fordelt i 5 grupper: "Perfluorinated and Polyfluorinated compounds", "Parabens", "Plastic derivatives (BPA, Phthalates and others)", "Pesticides/Fungicides/Insecticides/Biocides" og "Various EDCs, Mixtures and Other endpoints".

Udvalgte publikationer

2 artikler er blevet udvalgt til nærmere beskrivelse baseret på, at de beskriver resultater der bidrager til ny eller yderligere viden om grupper af hormonforstyrrende stoffer, samt kilder til eksponering for disse.

Den første artikel omhandler et in vitro studie med det formål, at undersøge syv udvalgte perfluorerede alkylerede stoffer (PFAS) for deres potentiale til at forårsage oxidative stress (kræftfremkaldende potentiale).

Den anden artikel omhandler et studie, der har haft til formål at undersøge tilstedeværelsen af hormonforstyrrende stoffer i plastikbideringe til babyer og om disse bideringe kan udgøre en mulig eksponeringskilde for spædbørn til hormonforstyrrende stoffer.

[Perfluoroalkylated substances \(PFAS\) affect oxidative stress biomarkers in vitro.](#)

Wielsøe M, Long M, Ghisari M, Bonefeld-Jørgensen EC.

Abstract

Perfluoroalkylated substances (PFAS) have been widely used since 1950s and humans are exposed through food, drinking water, consumer products, dust, etc. The long-chained PFAS are persistent in the environment and accumulate in wildlife and humans. They are suspected carcinogens and a potential mode of action is through generation of oxidative stress. Seven long-chained PFAS found in human serum were investigated for the potential to generate reactive oxygen species (ROS), induce DNA damage and disturb the total antioxidant capacity (TAC). The tested PFAS were perfluorohexane sulfonate (PFHxS), perfluorooctane sulfonic acid (PFOS), perfluorooctanoic acid (PFOA), perfluorononanoate (PFNA), perfluorodecanoate (PFDA), perfluoroundecanoate (PFUnA), and perfluorododecanoate (PFDoA). Using the human hepatoma cell line (HepG2) and an exposure time of 24h we found that all three endpoints were affected by one or more of the compounds. PFHxS, PFOA, PFOS and PFNA showed a dose dependent increase in DNA damage in the concentration range from 2×10^{-7} to 2×10^{-5} M determined by the comet assay. Except for PFDoA, all the other PFAS increased ROS generation significantly. For PFHxS and PFUnA the observed ROS increases were dose-dependent. Cells exposed to PFOA were found to have a significant lower TAC compared with the solvent control, whereas a non-significant trend in TAC decrease was observed for PFOS

and PFDoA and an increase tendency for PFHxS, PFNA and PFUnA. Our results indicate a possible genotoxic and cytotoxic potential of the PFAS in human liver cells.

[Effect-directed identification of endocrine disruptors in plastic baby teethers.](#)

Berger E, Potouridis T, Haeger A, Püttmann W, Wagner M.

Abstract

Concerns have been raised regarding the human health effects of endocrine disrupting chemicals (EDCs), many of which are associated with and leaching from plastics. As infants are particularly vulnerable to EDCs, we have investigated whether plastic teethers for babies represent a relevant source of exposure. Applying effect-directed analysis, we use bioassays to screen teethers, toys used to soothe a baby's teething ache, for endocrine activity and chemical analysis to identify the causative compounds. We detected significant endocrine activity in two of 10 plastic teethers. Those samples leached estrogenic and/or antiandrogenic activity as detected in the Yeast Estrogen Screen and Yeast Antiandrogen Screen. After sample fractionation, gas chromatography-mass spectrometry non-target screening revealed that methyl-, ethyl- and propylparaben were responsible for the observed estrogenic and antiandrogenic activity in one product. The second product is likely to contain at least six different antiandrogenic compounds that remain so far unidentified. This study demonstrates that plastic teethers can be a source of infant exposure to well-established and unknown EDCs. Because of their limited value to the product, but potential toxicity, manufacturers should critically revisit the use of parabens in plastic teethers and further toys. Moreover, plastic teethers might leach EDCs that escape routine analysis and, thus, toxicological evaluation. The resulting uncertainty in product safety poses a problem to consumers, producers and regulators that remain to be resolved.

Bruttolisten (in vitro)

Perfluorinated and Polyfluorinated compounds

1. [Perfluoroalkylated substances \(PFAS\) affect oxidative stress biomarkers in vitro.](#)

Wielsøe M, Long M, Ghisari M, Bonfeld-Jørgensen EC. Chemosphere. 2015 Jun;129:239-45. doi: 10.1016/j.chemosphere.2014.10.014. Epub 2014 Nov 12.

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Midgett K, Peden-Adams MM, Gilkeson GS, Kamen DL. J Appl Toxicol. 2015 May;35(5):459-65. doi: 10.1002/jat.3037. Epub 2014 Jul 23.

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Plastic derivatives” (BPA, Phthalates and others)

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Pesticides/Fungicides/Insecticides/Biocides

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15. [In silico and biological analysis of anti-androgen activity of the brominated flame retardants ATE, BATE and DPTE in zebrafish.](#)

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16. [Screening breeding sites of the common toad \(Bufo bufo\) in England and Wales for evidence of endocrine disrupting activity.](#)

Pickford DB, Jones A, Velez-Pelez A, Iguchi T, Mitsui N, Tooi O. *Ecotoxicol Environ Saf*. 2015 Jul;117:7-19. doi: 10.1016/j.ecoenv.2015.03.006. Epub 2015 Mar 25.

17. [Intestinal pregnane x receptor links xenobiotic exposure and hypercholesterolemia.](#)

Sui Y, Helsley RN, Park SH, Song X, Liu Z, Zhou C. *Mol Endocrinol*. 2015 May;29(5):765-76. doi: 10.1210/me.2014-1355. Epub 2015 Mar 26.

18. [The adverse effects of aldrin and dieldrin on both myometrial contractions and the secretory functions of bovine ovaries and uterus in vitro.](#)

Wrobel MH, Grzeszczyk M, Mlynarczuk J, Kotwica J. *Toxicol Appl Pharmacol*. 2015 May 15;285(1):23-31. doi: 10.1016/j.taap.2015.03.005. Epub 2015 Mar 11.

19. [Investigation on the toxic potential of Tribulus terrestris in vitro.](#)

Abudayyak M, Jannuzzi AT, Özhan G, Alpertunga B. *Pharm Biol*. 2015 Apr;53(4):469-76. doi: 10.3109/13880209.2014.924019. Epub 2014 Dec 4.

20. [Endocrine disruptors: New players in the pathophysiology of type 2 diabetes?](#)

Chevalier N, Fénelon P. *Diabetes Metab*. 2015 Apr;41(2):107-115. doi: 10.1016/j.diabet.2014.09.005. Epub 2014 Nov 20. Review.

21. [Targeted tumor theranostics using folate-conjugated and camptothecin-loaded acoustic nanodroplets in a mouse xenograft model.](#)

Chen WT, Kang ST, Lin JL, Wang CH, Chen RC, Yeh CK. *Biomaterials*. 2015 Jun;53:699-708. doi: 10.1016/j.biomaterials.2015.02.122. Epub 2015 Mar 25.

22. [Controlling oxygen release from hollow microparticles for prolonged cell survival under hypoxic environment.](#)

Lee HY, Kim HW, Lee JH, Oh SH. *Biomaterials*. 2015 Jun;53:583-91. doi: 10.1016/j.biomaterials.2015.02.117. Epub 2015 Mar 23.

23. [Thrombolytic efficacy and enzymatic activity of rt-PA-loaded echogenic liposomes.](#)

Bader KB, Bouchoux G, Peng T, Klegerman ME, McPherson DD, Holland CK. *J Thromb Thrombolysis*. 2015 Apr 2. [Epub ahead of print]

24. [Modulating the Anticancer Activity of Ruthenium\(II\)-Arene Complexes.](#)

Clavel CM, Păunescu E, Nowak-Sliwinska P, Griffioen AW, Scopelliti R, Dyson PJ. *J Med Chem*. 2015 Apr 23;58(8):3356-65. doi: 10.1021/jm501655t. Epub 2015 Apr 7.

25. [Revealing signal from noisy \(19\) F MR images by chemical shift artifact correction.](#)

Meissner M, Reiser M, Hugger T, Hennig J, von Elverfeldt D, Leupold J. *Magn Reson Med*. 2015 Jun;73(6):2225-33. doi: 10.1002/mrm.25370. Epub 2014 Jul 17.

26. [Disruption of endocrine function in H295R cell *in vitro* and in zebrafish *in vivo* by naphthenic acids.](#)

Wang J, Cao X, Sun J, Huang Y, Tang X. *J Hazard Mater*. 2015 Jun 4;299:1-9. doi: 10.1016/j.jhazmat.2015.06.004. [Epub ahead of print]

27. [Understanding the Molecular Basis for Differences in Responses of Fish Estrogen Receptor Subtypes to Environmental Estrogens.](#)

Tohyama S, Miyagawa S, Lange A, Ogino Y, Mizutani T, Tatarazako N, Katsu Y, Ihara M, Tanaka H, Ishibashi H, Kobayashi T, Tyler CR, Iguchi T. *Environ Sci Technol*. 2015 Jun 16;49(12):7439-47. doi: 10.1021/acs.est.5b00704. Epub 2015 Jun 2.

28. [*In vitro* and *in vivo* studies of the endocrine disrupting potency of cadmium in roach \(*Rutilus rutilus*\) liver.](#)

Gerbron M, Geraudie P, Xuereb B, Marie S, Minier C. *Mar Pollut Bull*. 2015 Jun 30;95(2):582-9. doi: 10.1016/j.marpolbul.2015.03.043. Epub 2015 May 27.

29. [Disruption of Type 2 Iodothyronine Deiodinase Activity in Cultured Human Glial Cells by Polybrominated Diphenyl Ethers.](#)

Roberts SC, Bianco AC, Stapleton HM. *Chem Res Toxicol*. 2015 Jun 15;28(6):1265-74. doi: 10.1021/acs.chemrestox.5b00072. Epub 2015 Jun 2

In vivo studier ved DTU Fødevareinstituttet

Søgning er udført på PubMed og dækker perioden April 2015- ultimo Juni 2015

Følgende søgeprofil er benyttet i PubMed: ((endocrine disrupt*) AND (rat OR mice OR mammal*)) OR ((endocrine disrupt*) AND (in vivo*)) OR ((endocrine disrupt*) AND (Paraben*)) OR ((endocrine disrupt*) AND (Phthalat*)) OR ((Endocrine disrupt* AND (antiandrogen)) OR ((endocrine disrupt*) AND (behaviour OR behavior*)) OR ((Endocrine disrupt*) AND (Bisphenol A or BPA) OR ((PFAS* OR Perfluor*) AND (endocrine disrupt*) AND risk assessment

Efter at have fjernet gengangere fra dem vi havde med på den forrige litteraturopdateringsliste samt *in vitro*, human eller SDU relevante artikler, gav litteratursøgningen en liste med i alt 76 artikler (Bruttolisten).

Disse er efter Miljøstyrelsens ønske blevet fordelt i grupper efter stofnavne: "Plastic derivatives" (BPA, Phthalates and others), Perflourinated and Polyflourinated compounds, "Pesticides/fungicides", "Endocrine disruption and risk assessment" og Various EDCs, Mixtures and Other endpoints".

Udvalgte publikationer

To artikler er blevet udvalgt til nærmere beskrivelse (abstrakt og konklusion) og 2 andre blot til abstract. Disse artikler er valgt fordi vi mener de bidrager til ny viden om hormonforstyrrende stoffer og her er der særligt fokus på brystvævsmetoder (Stanko et al. 2015) og Non-monoton dosis respons (Lagarde et al. 2015) samt debat om kriterier for hormonforstyrrende stoffer (hhv. Zoeller et al. 2014 og Autrup et al. 2015). **Rigtig God læselyst.**

Ud fra bruttolisten (se længere nede i dokumentet) er udvalgt følgende 2 artikler til engelsk abstrakt og dansk resume og kommentarer samt 2 artikler til engelsk abstrakt.

[Application of Sholl analysis to quantify changes in growth and development in rat mammary gland whole mounts.](#)

Stanko JP, Easterling MR, Fenton SE.

Reprod Toxicol. 2015 Jul;54:129-35. doi: 10.1016/j.reprotox.2014.11.004. Epub 2014 Nov 15.

Abstract

Studies that utilize the rodent mammary gland (MG) as an endpoint for assessing the developmental toxicity of chemical exposures typically employ either basic dimensional measurements or developmental scoring of morphological characteristics as a means to quantify MG development. There are numerous means by which to report these developmental changes, leading to inconsistent translation across laboratories. The Sholl analysis is a method historically used for quantifying neuronal dendritic patterns. The present study describes the use of the Sholl analysis to quantify MG branching characteristics. Using this method, we were able to detect significant differences in branching density in MG of peripubertal female Sprague Dawley rats that had been exposed to vehicle or a potent estrogen. These data suggest the Sholl analysis can be an effective tool for quantitatively measuring an important characteristic of MG development and for examining associations between MG growth and density and adverse effects in the breast.

[Non-monotonic dose-response relationships and endocrine disruptors: a qualitative method of assessment.](#)

Lagarde F, Beausoleil C, Belcher SM, Belzunces LP, Emond C, Guerbet M, Rousselle C. Environ Health. 2015 Feb 11;14:13. doi: 10.1186/1476-069X-14-13.

Abstract:

Experimental studies investigating the effects of endocrine disruptors frequently identify potential unconventional dose-response relationships called non-monotonic dose-response (NMDR) relationships. Standardized approaches for investigating NMDR relationships in a risk assessment context are missing. The aim of this work was to develop criteria for assessing the strength of NMDR relationships. A literature search was conducted to identify published studies that report NMDR relationships with endocrine disruptors. Fifty-one experimental studies that investigated various effects associated with endocrine disruption elicited by many substances were selected. Scoring criteria were applied by adaptation of an approach previously used for identification of hormesis-type dose-response relationships. Out of the 148 NMDR relationships analyzed, 82 were categorized with this method as having a "moderate" to "high" level of plausibility for various effects. Numerous modes of action described in the literature can explain such phenomena. NMDR can arise from numerous molecular mechanisms such as opposing effects induced by multiple receptors differing by their affinity, receptor desensitization, negative feedback with increasing dose, or dose-dependent metabolism modulation. A stepwise decision tree was developed as a tool to standardize the analysis of NMDR relationships observed in the literature with the final aim to use these results in a Risk Assessment purpose. This decision tree was finally applied to studies focused on the effects of bisphenol A.

[A path forward in the debate over health impacts of endocrine disrupting chemicals.](#)

Zoeller RT, Bergman Å, Becher G, Bjerregaard P, Bornman R, Brandt I, Iguchi T, Jobling S, Kidd KA, Kortenkamp A, Skakkebaek NE, Toppari J, Vandenberg LN. Environ Health. 2014 Dec 22;14:118. doi: 10.1186/1476-069X-13-118.

Abstract

Several recent publications reflect debate on the issue of "endocrine disrupting chemicals" (EDCs), indicating that two seemingly mutually exclusive perspectives are being articulated separately and independently. Considering this, a group of scientists with expertise in basic science, medicine and risk assessment reviewed the various aspects of the debate to identify the most significant areas of dispute and to propose a path forward. We identified four areas of debate. The first is about the definitions for terms such as "endocrine disrupting chemical", "adverse effects", and "endocrine system". The second is focused on elements of hormone action including "potency", "endpoints", "timing", "dose" and "thresholds". The third addresses the information needed to establish sufficient evidence of harm. Finally, the fourth focuses on the need to develop and the characteristics of transparent, systematic methods to review the EDC literature. Herein we identify areas of general consensus and propose resolutions for these four areas that would allow the field to move beyond the current and, in our opinion, ineffective debate.

Principles of Pharmacology and Toxicology Also Govern Effects of Chemicals on the Endocrine System.

Autrup H, Barile FA, Blaauboer BJ, Degen GH, Dekant W, Dietrich D, Domingo JL, Gori GB, Greim H, Hengstler JG, Kacew S, Marquardt H, Pelkonen O, Savolainen K, Vermeulen NP. Toxicol Sci. 2015 Jul;146(1):11-5. doi: 10.1093/toxsci/kfv082. Epub 2015 May 30.

Abstract

The present debate on chemicals with Hormonal activity, often termed 'endocrine disruptors', is highly controversial and includes challenges of the present paradigms used in toxicology and in hazard identification and risk characterization. In our opinion, chemicals with hormonal activity can be subjected to the well-evaluated health risk characterization approach used for many years including adverse outcome pathways. Many of the points arguing for a specific approach for risk characterization of chemicals with hormonal activity are based on highly speculative conclusions. These conclusions are not well supported when evaluating the available information.

Bruttolisten (in vivo)

Plastic derivatives (BPA, Phthalates and others)

BPA (and alternatives)

1. [Bisphenol A affects placental layers morphology and angiogenesis during early pregnancy phase in mice.](#)
Tait S, Tassinari R, Maranghi F, Mantovani A.
J Appl Toxicol. 2015 Jun 9. doi: 10.1002/jat.3176. [Epub ahead of print]
2. [Bisphenol A exposure inhibits germ cell nest breakdown by reducing apoptosis in cultured neonatal mouse ovaries.](#)
Zhou C, Wang W, Peretz J, Flaws JA.
Reprod Toxicol. 2015 Jun 4. pii: S0890-6238(15)00103-3. doi: 10.1016/j.reprotox.2015.05.012. [Epub ahead of print]
3. [Early programming of uterine tissue by bisphenol A: Critical evaluation of evidence from animal exposure studies.](#)
Suvorov A, Waxman DJ.
Reprod Toxicol. 2015 May 28. pii: S0890-6238(15)00076-3. doi: 10.1016/j.reprotox.2015.05.008. [Epub ahead of print] Review.
4. [Physiological response of cardiac tissue to Bisphenol A: alterations in ventricular pressure and contractility.](#)
Posnack NG, Brooks D, Chandra A, Jaimes R 3rd, Sarvazyan N, Kay MW.
Am J Physiol Heart Circ Physiol. 2015 May 15:ajpheart.00272.2015. doi: 10.1152/ajpheart.00272.2015. [Epub ahead of print]
5. [Long-term oral exposure to bisphenol A induces glucose intolerance and insulin resistance.](#)
Moon MK, Jeong IK, Jung Oh T, Ahn HY, Kim HH, Park YJ, Jang HC, Park KS.
J Endocrinol. 2015 Jul;226(1):35-42. doi: 10.1530/JOE-14-0714. Epub 2015 May 13.
6. [Correction: An Investigation of the Endocrine-Disruptive Effects of Bisphenol A in Human and Rat Fetal Testes.](#)
PLOS ONE Staff.
PLoS One. 2015 May 4;10(5):e0128051. doi: 10.1371/journal.pone.0128051. eCollection 2015.
7. [Neurochemical impact of bisphenol A in the hippocampus and cortex of adult male albino rats.](#)
Khadrawy YA, Noor NA, Mourad IM, Ezz HS.
Toxicol Ind Health. 2015 Apr 22. pii: 0748233715579803. [Epub ahead of print]
8. [Perinatal exposure to low-dose of bisphenol A causes anxiety-like alteration in adrenal axis regulation and behaviors of rat offspring: a potential role for metabotropic glutamate 2/3 receptors.](#)
Zhou R, Chen F, Feng X, Zhou L, Li Y, Chen L.
J Psychiatr Res. 2015 May;64:121-9. doi: 10.1016/j.jpsychires.2015.02.018. Epub 2015 Mar 11.
9. [Developmental bisphenol A \(BPA\) exposure leads to sex-specific modification of hepatic gene expression and epigenome at birth that may exacerbate high-fat diet-induced hepatic steatosis.](#)
Strakovsky RS, Wang H, Engeseth NJ, Flaws JA, Helferich WG, Pan YX, Lezmi S.

Toxicol Appl Pharmacol. 2015 Apr 15;284(2):101-12. doi: 10.1016/j.taap.2015.02.021. Epub 2015 Mar 5.

10. [Prenatal Bisphenol A exposure delays the development of the male rat mammary gland.](#)

Kass L, Durando M, Altamirano GA, Manfroni-Ghibaudo GE, Luque EH, Muñoz-de-Toro M. Reprod Toxicol. 2015 Jul;54:37-46. doi: 10.1016/j.reprotox.2014.02.001. Epub 2014 Feb 22.

11. [Low circulating levels of bisphenol-A induce cognitive deficits and loss of asymmetric spine synapses in dorsolateral prefrontal cortex and hippocampus of adult male monkeys.](#)

Elsworth JD, Jentsch JD, Groman SM, Roth RH, Redmond ED Jr, Leranath C. J Comp Neurol. 2015 Jun 1;523(8):1248-57. doi: 10.1002/cne.23735. Epub 2015 Feb 17.

12. [Neurochemical impact of bisphenol A in the hippocampus and cortex of adult male albino rats.](#)

Khadrawy YA, Noor NA, Mourad IM, Ezz HS. Toxicol Ind Health. 2015 Apr 22. pii: 0748233715579803. [Epub ahead of print]

13. [Bisphenol-A treatment during pregnancy in mice: a new window of susceptibility for the development of diabetes in mothers later in life.](#)

Alonso-Magdalena P, García-Arévalo M, Quesada I, Nadal Á. Endocrinology. 2015 May;156(5):1659-70. doi: 10.1210/en.2014-1952. Epub 2015 Apr 1.

14. [Bisphenol-A induces expression of HOXC6, an estrogen-regulated homeobox-containing gene associated with breast cancer.](#)

Hussain I, Bhan A, Ansari KI, Deb P, Bobzean SA, Perrotti LI, Mandal SS. Biochim Biophys Acta. 2015 Jun;1849(6):697-708. doi: 10.1016/j.bbagr.2015.02.003. Epub 2015 Feb 25.

15. [Bisphenol A and nonylphenol have the potential to stimulate the migration of ovarian cancer cells by inducing epithelial-mesenchymal transition via an estrogen receptor dependent pathway.](#)

Kim YS, Hwang KA, Hyun SH, Nam KH, Lee CK, Choi KC. Chem Res Toxicol. 2015 Apr 20;28(4):662-71. doi: 10.1021/tx500443p. Epub 2015 Mar 3.

16. [Estrogens in the wrong place at the wrong time: Fetal BPA exposure and mammary cancer.](#)

Paulose T, Speroni L, Sonnenschein C, Soto AM. Reprod Toxicol. 2015 Jul;54:58-65. doi: 10.1016/j.reprotox.2014.09.012. Epub 2014 Sep 30.

17. [Bisphenol A environmental exposure and the detrimental effects on human metabolic health: is it necessary to revise the risk assessment in vulnerable population?](#)

Valentino R, D'Esposito V, Ariemma F, Cimmino I, Beguinot F, Formisano P. J Endocrinol Invest. 2015 Jun 24. [Epub ahead of print]

18. [Weak activity of UDP-glucuronosyltransferase toward Bisphenol analogs in mouse perinatal development.](#)

Yabusaki R, Iwano H, Tsuahima S, Koike N, Ohtani N, Tanemura K, Inoue H, Yokota H. J Vet Med Sci. 2015 Jun 15. [Epub ahead of print]

Phthalates and others

1. [Phthalate metabolites and bisphenol-A in association with circulating angiogenic biomarkers across pregnancy.](#)

Ferguson KK, McElrath TF, Cantonwine DE, Mukherjee B, Meeker JD.

Placenta. 2015 Jun;36(6):699-703. doi: 10.1016/j.placenta.2015.04.002. Epub 2015 Apr 14.

2. [Influence of phthalates on glucose homeostasis and atherosclerosis in hyperlipidemic mice.](#)

Zhou W, Chen MH, Shi W.

BMC Endocr Disord. 2015 Apr 2;15:13. doi: 10.1186/s12902-015-0015-4.

3. [Effects of uterine and lactational exposure to di-\(2-ethylhexyl\) phthalate on spatial memory and NMDA receptor of hippocampus in mice.](#)

Dai Y, Yang Y, Xu X, Hu Y.

Horm Behav. 2015 May;71:41-8. doi: 10.1016/j.yhbeh.2015.03.008. Epub 2015 Apr 11.

4. [Di\(2-ethylhexyl\) phthalate inhibits antral follicle growth, induces atresia, and inhibits steroid hormone production in cultured mouse antral follicles.](#)

Hannon PR, Brannick KE, Wang W, Gupta RK, Flaws JA.

Toxicol Appl Pharmacol. 2015 Apr 1;284(1):42-53. doi: 10.1016/j.taap.2015.02.010. Epub 2015 Feb 18.

5. [A monograph on the remediation of hazardous phthalates.](#)

Benjamin S, Pradeep S, Sarath Josh M, Kumar S, Masai E.

J Hazard Mater. 2015 May 6;298:58-72. doi: 10.1016/j.jhazmat.2015.05.004. [Epub ahead of print] Review.

6. [Lactational exposure of phthalate causes long-term disruption in testicular architecture by altering tight junctional and apoptotic protein expression in Sertoli cells of first filial generation pubertal Wistar rats.](#)

Sekaran S, Balaganapathy P, Parsanathan R, Elangovan S, Gunashekar J, Bhat FA, Jagadeesan A.

Hum Exp Toxicol. 2015 Jun;34(6):575-90. doi: 10.1177/0960327114555926. Epub 2014 Oct 28.

7. [Impact of low molecular weight phthalates in inducing reproductive malfunctions in male mice: Special emphasis on Sertoli cell functions.](#)

Kumar N, Srivastava S, Roy P.

Gen Comp Endocrinol. 2015 May 1;215:36-50. doi: 10.1016/j.ygcen.2014.09.012. Epub 2014 Sep 28.

8. [Perinatal exposure to di-\(2-ethylhexyl\) phthalate affects anxiety- and depression-like behaviors in mice.](#)

Xu X, Yang Y, Wang R, Wang Y, Ruan Q, Lu Y.

Chemosphere. 2015 Apr;124:22-31. doi: 10.1016/j.chemosphere.2014.10.056. Epub 2014 Nov 28.

9. [The Mechanism of Environmental Endocrine Disruptors \(DEHP\) Induces Epigenetic Transgenerational Inheritance of Cryptorchidism.](#)

Chen J, Wu S, Wen S, Shen L, Peng J, Yan C, Cao X, Zhou Y, Long C, Lin T, He D, Hua Y, Wei G.

PLoS One. 2015 Jun 2;10(6):e0126403. doi: 10.1371/journal.pone.0126403. eCollection 2015.

10. [DEHP exposure impairs mouse oocyte cyst breakdown and primordial follicle assembly through estrogen receptor-dependent and independent mechanisms.](#)

Mu X, Liao X, Chen X, Li Y, Wang M, Shen C, Zhang X, Wang Y, Liu X, He J.

J Hazard Mater. 2015 Jun 1;298:232-240. doi: 10.1016/j.jhazmat.2015.05.052. [Epub ahead of print]

11. [Cyclohexane-1,2-dicarboxylic acid diisononyl ester and metabolite effects on rat epididymal stromal vascular fraction differentiation of adipose tissue.](#)

Campoli E, Duong TB, Deschamps F, Papadopoulos V.

Environ Res. 2015 Apr 7;140:145-156. doi: 10.1016/j.envres.2015.03.036. [Epub ahead of print]

Perflourinated and Polyflourinated compounds

1. [Perfluorinated Alkyl Substances: Emerging Insights Into Health Risks.](#)

Grandjean P, Clapp R.

New Solut. 2015 Jun 17. pii: 1048291115590506. [Epub ahead of print]

2. [Perfluorononanoic acid disturbed the metabolism of lipid in the liver of streptozotocin-induced diabetic rats.](#)

Fang X, Gao G, Zhang X, Wang H.

Toxicol Mech Methods. 2015 Jun 9:1-6. [Epub ahead of print]

3. [Prenatal exposure to the contaminant perfluorooctane sulfonate elevates lipid peroxidation during mouse fetal development but not in the pregnant dam.](#)

Lee YY, Wong CK, Oger C, Durand T, Galano JM, Lee JC.

Free Radic Res. 2015 Apr 24:1-11. [Epub ahead of print]

4. [Hepatic Mitochondrial Alteration in CD-1 Mice Associated with Prenatal Exposures to Low Doses of Perfluorooctanoic Acid \(PFOA\).](#)

Quist EM, Filgo AJ, Cummings CA, Kissling GE, Hoenerhoff MJ, Fenton SE.

Toxicol Pathol. 2015 Jun;43(4):546-57. doi: 10.1177/0192623314551841. Epub 2014 Oct 16.

5. [Programming of metabolic effects in C57BL/6JxFVB mice by in utero and lactational exposure to perfluorooctanoic acid.](#)

van Esterik JC, Sales LB, Dollé ME, Håkansson H, Herlin M, Legler J, van der Ven LT.

Arch Toxicol. 2015 Apr 1. [Epub ahead of print]

Parabens

1. [A case study on quantitative in vitro to in vivo extrapolation for environmental esters: Methyl-, propyl- and butylparaben.](#)

Campbell JL, Yoon M, Clewell HJ.

Toxicology. 2015 Jun 5;332:67-76. doi: 10.1016/j.tox.2015.03.010. Epub 2015 Mar 31.

Pesticides/Fungicides/Insecticides

1. [Molecular Mechanisms of Amitraz Mammalian Toxicity: A Comprehensive Review of Existing Data.](#)

Del Pino J, Moyano-Cires PV, Anadon MJ, Díaz MJ, Lobo M, Capo MA, Frejo MT.

Chem Res Toxicol. 2015 Jun 15;28(6):1073-94. doi: 10.1021/tx500534x. Epub 2015 May 26.

2. [Peri-pubertal administration of 3-nitro-1,2,4-triazol-5-one \(NTO\) affects reproductive organ development in male but not female Sprague Dawley rats.](#)

Lent EM, Crouse LC, Wallace SM, Carroll EE.

Reprod Toxicol. 2015 May 9. pii: S0890-6238(15)00068-4. doi: 10.1016/j.reprotox.2015.04.013. [Epub ahead of print]

3. [Oral exposure of mice to carbendazim induces hepatic lipid metabolism disorder and gut microbiota dysbiosis.](#)

Jin Y, Zeng Z, Wu Y, Zhang S, Fu Z.
Toxicol Sci. 2015 Jun 11. pii: kfv115. [Epub ahead of print]

4. [A Comparison of ToxCast Test Results with In Vivo and Other In Vitro Endpoints for Neuro, Endocrine, and Developmental Toxicities: A Case Study Using Endosulfan and Methidathion.](#)

Silva M, Pham N, Lewis C, Iyer S, Kwok E, Solomon G, Zeise L.
Birth Defects Res B Dev Reprod Toxicol. 2015 May 27. doi: 10.1002/bdrb.21140. [Epub ahead of print]

5. [In Vitro, Ex Vivo, and In Vivo Determination of Thyroid Hormone Modulating Activity of Benzothiazoles.](#)

Hornung MW, Kosian PA, Haselman JT, Korte JJ, Challis K, Macherla C, Nevalainen E, Degitz SJ.
Toxicol Sci. 2015 May 7. pii: kfv090. [Epub ahead of print]

6. [Dietary exposure in utero and during lactation to a mixture of genistein and an anti-androgen fungicide in a rat mammary carcinogenesis model.](#)

Phrakonkham P, Brouland JP, Saad Hel S, Bergès R, Pimpie C, Pocard M, Canivenc-Lavier MC, Perrot-Applanat M.
Reprod Toxicol. 2015 Jul;54:101-9. doi: 10.1016/j.reprotox.2014.05.016. Epub 2014 Jun 8.

7. [Enantioselective disruption of the endocrine system by Cis-Bifenthrin in the male mice.](#)

Jin Y, Wang J, Pan X, Miao W, Lin X, Wang L, Fu Z.
Environ Toxicol. 2015 Jul;30(7):746-54. doi: 10.1002/tox.21954. Epub 2014 Jan 21.

8. [Peri-pubertal administration of 3-nitro-1,2,4-triazol-5-one \(NTO\) affects reproductive organ development in male but not female Sprague Dawley rats.](#)

Lent EM, Crouse LC, Wallace SM, Carroll EE.
Reprod Toxicol. 2015 May 9. pii: S0890-6238(15)00068-4. doi: 10.1016/j.reprotox.2015.04.013. [Epub ahead of print]

EDC and risk assessment

1. [A path forward in the debate over health impacts of endocrine disrupting chemicals.](#)

Zoeller RT, Bergman Å, Becher G, Bjerregaard P, Bornman R, Brandt I, Iguchi T, Jobling S, Kidd KA, Kortenkamp A, Skakkebaek NE, Toppari J, Vandenberg LN.
Environ Health. 2014 Dec 22;14:118. doi: 10.1186/1476-069X-13-118. (abstract)

2. [Principles of Pharmacology and Toxicology Also Govern Effects of Chemicals on the Endocrine System.](#)

Autrup H, Barile FA, Blaauboer BJ, Degen GH, Dekant W, Dietrich D, Domingo JL, Gori GB, Greim H, Hengstler JG, Kacew S, Marquardt H, Pelkonen O, Savolainen K, Vermeulen NP.
Toxicol Sci. 2015 Jul;146(1):11-5. doi: 10.1093/toxsci/kfv082. Epub 2015 May 30. (abstract)

3. [Non-monotonic dose-response relationships and endocrine disruptors: a qualitative method of assessment.](#)

Lagarde F, Beausoleil C, Belcher SM, Belzunces LP, Emond C, Guerbet M, Rousselle C.
Environ Health. 2015 Feb 11;14:13. doi: 10.1186/1476-069X-14-13.(valgt)

4. [Assessing dose-response relationships for endocrine disrupting chemicals \(EDCs\): a focus on non-monotonicity.](#)

Zoeller RT, Vandenberg LN.

Environ Health. 2015 May 15;14:42. doi: 10.1186/s12940-015-0029-4.

5. [An approach to the identification and regulation of endocrine disrupting pesticides.](#)

Ewence A, Brescia S, Johnson I, Rumsby PC.

Food Chem Toxicol. 2015 Apr;78:214-20. doi: 10.1016/j.fct.2015.01.011. Epub 2015 Feb 7. Review.

6. [Why endocrine disrupting chemicals \(EDCs\) challenge traditional risk assessment and how to respond.](#)

Futran Fuhrman V, Tal A, Arnon S.

J Hazard Mater. 2015 Apr 9;286:589-611. doi: 10.1016/j.jhazmat.2014.12.012. Epub 2014 Dec 12. Review.

Various EDCs, Mixtures and Other endpoints

1. [Application of Sholl analysis to quantify changes in growth and development in rat mammary gland whole mounts.](#)

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Wildlife studier ved Biologisk Institut, Syddansk Universitet (SDU)

Søgningen er udført på Web of Knowledge (all databases) og dækker perioden 21/3 – 24/6 2015.

Søgeprofilen kombinerer: "Endocrine disrupt*" and Fish*

Amphibia*
Bird* OR Avia*
Invertebrat*
Mollus*
Gastropod*
Insect*
Crustacea*
Echinoderm*
Ursus
Reptil* OR Alligator
Whal* OR seal* OR dolphin*

Fra bruttolisten (længere nede i dokumentet) er udvalgt tre artikler til medtagelse af abstract og yderligere kommentarer.

Kriterierne for udvælgelsen af publikationer til kommentering er, at de bidrager til ny viden omkring effekter af og virkningsmekanismer for hormonforstyrrende stoffer i 'wildlife' og/eller at de repræsenterer vigtig viden, som vurderes at have særlig interesse for Miljøstyrelsen bl.a. i forbindelse med styrelsens fokus på udvikling af testmetoder. Desuden kommenteres artikler, der omhandler 'nye' stoffer og miljøfaktorer, der har vist sig hormonforstyrrende; specielt hvis disse har relevans for danske forhold. Endelig medtages efter Miljøstyrelsens ønske artikler omhandlende parabener.

Udvalgte artikler

Artikel 1: Dicamba affects sex steroid hormone level and mRNA expression of related genes in adult rare minnow (*Gobiocypris rarus*) at environmentally relevant concentrations.

Zhu, L.; Li, W.; Zha, J.; and Wang, Z. 2015. *Environmental Toxicology* 30, 693-703.

Abstract: Dicamba is a benzoic acid herbicide that has been detected in surface and ground water. The herbicide has been shown to have cytogenic and DNA damaging effects and to cause organ damage in mammals; however, little is known about the endocrine disrupting effects of dicamba in fish. In this study, histological changes, plasma vitellogenin (VTG) and sex hormone levels, and mRNA expression of sex steroid hormone-related genes were determined in adult rare minnow exposed to environmentally relevant concentrations of dicamba (0, 0.05, 0.5, 5, and 50 µg/L) for 40 days. The results showed inhibition of spermatogenesis in male testes and ovarian degeneration in females. Plasma 17β-estradiol (E2) levels were significantly increased in both genders, and plasma VTG levels were significantly increased in males ($p < 0.05$). These results indicate that sex hormone homeostasis and normal reproduction of fish could be affected by dicamba. Moreover, the mRNA levels of vtg were significantly upregulated in the livers and gonads of both male and female rare minnows ($p < 0.05$). The downregulation of cytochrome P450c19a (cyp19a) and steroidogenic acute regulatory protein (star) mRNA levels, and the upregulation of cytochrome P450c17 (cyp17)

mRNA levels were observed in the livers and ovaries ($p < 0.05$). The results of the mRNA analysis suggest that changes in steroid hormone-related gene expression could serve as a regulatory mechanism to maintain sex hormone homeostasis. Overall, dicamba exposure could result in histological lesions, plasma VTG increases, changes in sex hormone levels, and alterations of hormone-related gene expression. Therefore, dicamba should be considered to be a potential endocrine disruptor.

Artikel 2: Emerging wastewater contaminant metformin causes intersex and reduced fecundity in fish.

Niemuth, N. J. and Klaper, R. D. 2015. *Chemosphere* 135, 38-45.

Abstract: The occurrence of intersex fish, where male reproductive tissues show evidence of feminization, have been found in freshwater systems around the world, indicating the potential for significant endocrine disruption across species in the ecosystem. Estrogens from birth control medications in wastewater treatment plant effluent have been cited as the likely cause, but research has shown that endocrine disruption is not solely predictable based on hormone receptor interactions. Many other non-hormone pharmaceuticals are found in effluent at concentrations orders of magnitude higher than estrogens, yet there is little data indicating the impacts of these other medications. The widely prescribed anti-diabetic metformin is among the most abundant of pharmaceuticals found in effluent and is structurally dissimilar from hormones. However, we show here that exposing fathead minnows (*Pimephales promelas*) to a concentration of metformin found in wastewater effluent causes the development of intersex gonads in males, reduced size of treated male fish, and reduction in fecundity for treated pairs. Our results demonstrate that metformin acts as an endocrine disruptor at environmentally relevant concentrations.

Artikel 3: Long-term effects of Bisphenol AF (BPAF) on hormonal balance and genes of hypothalamus-pituitary-gonad axis and liver of zebrafish (*Danio rerio*), and the impact on offspring. Shi, J.; Jiao, Z.; Zheng, S.; Li, M.; Zhang, J.; Feng, Y.; Yin, J.; and Shao, B. 2015. *Chemosphere* 128, 252-257.

Abstract: Bisphenol AF (BPAF) is one of the analogues of bisphenol A (BPA) and is widely used as a raw material in the plastics industry. The potential toxicity to fish from exposure to BPAF in the aquatic environment is largely unknown. In this study, zebrafish (*Danio rerio*) were exposed to BPAF at 5, 25 and 125 $\mu\text{g/L}$, from 4 hour-post-fertilization (hpf) to 120 day-post-fertilization (dpf), representing the period from embryo to adult. The levels of plasma hormones were measured and the expression of selected representative genes along the hypothalamus-pituitary-gonad (HPG) axis and liver were examined. The concentration of 17β -estradiol (E2) was significantly increased in male and female fish and a significant decrease of testosterone (T) was observed in male fish. The mRNA expression of genes along the HPG axis and in liver tissues in F0 generation fish demonstrated that the steroid hormonal balances of zebrafish were modulated through the alteration of steroidogenesis. The significant decrease of egg fertilization among offspring indicates the possibility of sperm deterioration of parent following exposure to BPAF. The higher occurrence of malformation and lower survival rate in the offspring from the exposure group suggested a possibility of maternal transfer of BPAF, which could be responsible for the increased prevalence of

adverse health signs in the offspring. The hatching delay in 5 lg L₋₁ BPAF indicated that parental exposure to environmentally relevant concentration of BPAF would result in delayed hatching of the offspring. A potential consequence of adverse effects in the offspring by BPAF deserves further investigation.

Bruttoliste

Alkylphenoler

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Barber, L. B.; Loyo-Rosales, J. E.; Rice, C. P.; Minarik, T. A.; and Oskouie, A. K. 2015. *Science of the Total Environment* 517, 195-206.

Interferences of an environmental pollutant with estrogen-like action in the male reproductive system of the terrestrial vertebrate *Podarcis sicula*.

Verderame, M. and Limatola, E. 2015. *General and Comparative Endocrinology* 213, 9-15.

Estrogenic environmental contaminants alter the mRNA abundance profiles of genes involved in gonadal differentiation of the American bullfrog.

Wolff, S. E.; Veldhoen, N.; Helbing, C. C.; Ramirez, C. A.; Malpas, J. M.; and Propper, C. R. 2015. *Science of the Total Environment* 521, 380-387.

Bisphenoler

Effects of the environmental estrogenic contaminants bisphenol A and 17 alpha-ethinyl estradiol on sexual development and adult behaviors in aquatic wildlife species.

Bhandari, R. K.; Deem, S. L.; Holliday, D. K.; Jandegian, C. M.; Kassotis, C. D.; Nagel, S. C.; Tillitt, D. E.; Saal, F. S. V.; and Rosenfeld, C. S. 2015. *General and Comparative Endocrinology* 214, 195-219.

Bisphenol A accumulation in eggs disrupts the endocrine regulation of growth in rainbow trout larvae.

Birceanu, O.; Servos, M. R.; and Vijayan, M. M. 2015. *Aquatic Toxicology* 161, 51-60.

Developmental exposure to bisphenol A (BPA) alters sexual differentiation in painted turtles (*Chrysemys picta*).

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Adverse effects of bisphenol A on water louse (*Asellus aquaticus*).

Plahuta, M.; Tisler, T.; Pintar, A.; and Toman, M. J. 2015. *Ecotoxicology and Environmental Safety* 117, 81-88.

Endogenous and exogenous estrogens during embryonic development affect timing of hatch and growth in the American alligator (*Alligator mississippiensis*).

Cruze, L.; Roark, A. M.; Rolland, G.; Younas, M.; Stacy, N.; and Guillette, L. J. 2015. *Comparative Biochemistry and Physiology B-Biochemistry & Molecular Biology* 184, 10-18.

Long-term effects of Bisphenol AF (BPAF) on hormonal balance and genes of hypothalamus-pituitary-gonad axis and liver of zebrafish (*Danio rerio*), and the impact on offspring.

Shi, J.; Jiao, Z.; Zheng, S.; Li, M.; Zhang, J.; Feng, Y.; Yin, J.; and Shao, B. 2015. *Chemosphere* 128, 252-257.

Phthalater

Environmental occurrence and biota concentration of phthalate esters in Epe and Lagos Lagoons, Nigeria.

Adeogun, A. O.; Ibor, O. R.; Omogbemi, E. D.; Chukwuka, A. V.; Adegbola, R. A.; Adewuyi, G. A.; and Arukwe, A. 2015. *Marine environmental research* 108, 24-32.

Long-term exposures to di-n-butyl phthalate inhibit body growth and impair gonad development in juvenile Murray rainbowfish (*Melanotaenia fluviatilis*).

Bhatia, H.; Kumar, A.; Chapman, J. C.; and McLaughlin, M. J. 2015. *Journal of Applied Toxicology* 35, 806-816.

Effects of Diisodecyl Phthalate on PPAR:RXR-Dependent Gene Expression Pathways in Sea Bream Hepatocytes.

Cocci, P.; Mosconi, G.; Arukwe, A.; Mozzicafreddo, M.; Angeletti, M.; Aretusi, G.; and Palermo, F. A. 2015. *Chemical Research in Toxicology* 28, 935-947.

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Guo, Y.; Yang, Y.; Gao, Y.; Wang, X.; and Zhou, B. 2015. *Environmental pollution (Barking, Essex : 1987)* 203, 130-136.

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Net, S.; Sempere, R.; Delmont, A.; Paluselli, A.; and Ouddane, B. 2015. *Environmental Science & Technology* 49, 4019-4035.

Flammehæmmere

Bioaccumulation and biomagnification of classical flame retardants, related halogenated natural compounds and alternative flame retardants in three delphinids from Southern European waters.

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Identification of a group of brominated flame retardants as novel androgen receptor antagonists and potential neuronal and endocrine disrupters.

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Perfluorerede forbindelser

PCB og dioxin

Hydroxylated polychlorinated biphenyls decrease circulating steroids in female polar bears (*Ursus maritimus*).

Gustayson, L.; Ciesielski, T. M.; Bytingsvik, J.; Styrishave, B.; Hansen, M.; Lie, E.; Aars, J.; and Jenssen, B. M. 2015. *Environmental Research* 138, 191-201.

Polychlorinated biphenyl exposure and corticosterone levels in seven polar seabird species.

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Contaminants reduce male contribution to reproduction at the population scale.

Hillis, J. J.; Garvey, J. E.; and Lydy, M. J. 2015. *Ecosphere* 6.

Developmental Exposure to Aroclor 1254 Alters Migratory Behavior in Juvenile European Starlings (*Sturnus vulgaris*).

Flahr, L. M.; Michel, N. L.; Zahara, A. R.; Jones, P. D.; and Morrissey, C. A. 2015. *Environmental Science & Technology* 49, 6274-6283.

Comparison of the in vitro effects of TCDD, PCB 126 and PCB 153 on thyroid-restricted gene expression and thyroid hormone secretion by the chicken thyroid gland.

Katarzynska, D.; Hrabia, A.; Kowalik, K.; and Sechman, A. 2015. *Environmental Toxicology and Pharmacology* 39, 496-503.

TBT

Disruptions in aromatase expression in the brain, reproductive behavior, and secondary sexual characteristics in male guppies (*Poecilia reticulata*) induced by tributyltin.

Tian, H.; Wu, P.; Wang, W.; and Ru, S. 2015. *Aquatic Toxicology* 162, 117-125.

Pesticider

Dicamba affects sex steroid hormone level and mRNA expression of related genes in adult rare minnow (*Gobiocypris rarus*) at environmentally relevant concentrations.

Zhu, L.; Li, W.; Zha, J.; and Wang, Z. 2015. Environmental Toxicology 30, 693-703.

Morphological alterations in the freshwater rotifer *Brachionus calyciflorus* Pallas 1766 (Rotifera: Monogononta) caused by vinclozolin chronic exposure.

Alvarado-Flores, J.; Rico-Martinez, R.; Adabache-Ortiz, A.; and Silva-Briano, M. 2015. *Ecotoxicology* 24, 915-925.

Adverse effects of thiram-treated seed ingestion on the reproductive performance and the offspring immune function of the red-legged partridge.

Lopez-Antia, A.; Ortiz-Santaliestra, M. E.; Garcia-de Blas, E.; Camarero, P. R.; Mougeot, F.; and Mateo, R. 2015. *Environmental Toxicology and Chemistry* 34, 1320-1329.

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Pandey, S. P. and Mohanty, B. 2015. *Chemosphere* 122, 227-234.

Endosulfan affects GnRH cells in sexually differentiated juveniles of the perciform *Cichlasoma dimerus*.

Piazza, Y.; Pandolfi, M.; Da Cuna, R.; Genovese, G.; and Lo Nostro, F. 2015. *Ecotoxicology and Environmental Safety* 116, 150-159.

Transcriptional profiling of the soil invertebrate *Folsomia candida* in pentachlorophenol-contaminated soil.

Qiao, M.; Wang, G. P.; Zhang, C.; Roelofs, D.; van Straalen, N. M.; and Zhu, Y. G. 2015. *Environmental Toxicology and Chemistry* 34, 1362-1368.

Atrazine and its degradates have little effect on the corticosteroid stress response in the zebrafish.

Van der Kraak, G.; Matsumoto, J.; Kim, M.; and Hosmer, A. J. 2015. *Comparative Biochemistry and Physiology C-Toxicology & Pharmacology* 170, 1-7.

Influence of triazine herbicide exposure on guppies (*poecilia sphenops*) aromatase activities, altered sex steroid concentration and vitellogenin induction.

Vasanth, S.; Arul, G.; Karthikeyeni, S.; Kumar, T.; Vignesh, V.; Manimegalai, M.; Bupesh, G.; Thirumurugan, R.; and Subramanian, P. 2015. *Indian Journal of Pharmaceutical Sciences* 77, 156-162.

The Herbicide Linuron Inhibits Cholesterol Biosynthesis and Induces Cellular Stress Responses in Brown Trout.

Webster, T. M.; Perry, M. H.; and Santos, E. M. 2015. *Environmental Science & Technology* 49, 3110-3118.

Effect of trifloxystrobin on hatching, survival, and gene expression of endocrine biomarkers in early life stages of medaka (*Oryzias latipes*).

Zhu, L.; Wang, H.; Liu, H.; and Li, W. 2015. *Environmental Toxicology* 30, 648-655.

Tungmetaller

Oxidative stress in songbirds exposed to dietary methylmercury.

Henry, K. A.; Cristol, D. A.; Varian-Ramos, C. W.; and Bradley, E. L. 2015. *Ecotoxicology* 24, 520-526.

Lægemidler og syntetiske hormoner

Emerging wastewater contaminant metformin causes intersex and reduced fecundity in fish. Niemuth, N. J. and Klaper, R. D. 2015. *Chemosphere* 135, 38-45.

Occurrence, bioaccumulation and risk assessment of lipophilic pharmaceutically active compounds in the downstream rivers of sewage treatment plants.

Liu, J.; Lu, G.; Xie, Z.; Zhang, Z.; Li, S.; and Yan, Z. 2015. *Science of the Total Environment* 511, 54-62.

Pharmaceuticals and personal care products: A critical review of the impacts on fish reproduction. Overturf, M. D.; Anderson, J. C.; Pandelides, Z.; Beyger, L.; and Holdway, D. A. 2015. *Critical reviews in toxicology* 45, 469-491.

Responses to various exposure durations of levonorgestrel during early-life stages of fathead minnows (*Pimephales promelas*).

Overturf, M. D. and Huggett, D. B. 2015. *Aquatic Toxicology* 161, 33-40.

Nortestosterone-derived synthetic progestogens do not activate the progestogen receptor of Murray-Darling rainbowfish (*Melanotaenia fluviatilis*) but are potent agonists of androgen receptors alpha and beta.

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Recovery of a Wild Fish Population from Whole-Lake Additions of a Synthetic Estrogen.

Blanchfield, P. J.; Kidd, K. A.; Docker, M. F.; Palace, V. P.; Park, B. J.; and Postma, L. D. 2015. *Environmental Science & Technology* 49, 3136-3144.

Timescale effects of 17 alpha-ethinylestradiol on behavioral consistency in male threespine stickleback.

Dzieweczynski, T. and Forrette, L. 2015. *Acta Ethologica* 18, 137-144.

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17 alpha-Ethinylestradiol can disrupt hemoglobin catabolism in amphibians.

Garmshausen, J.; Kloas, W.; and Hoffmann, F. 2015. *Comparative Biochemistry and Physiology C-Toxicology & Pharmacology* 171, 34-40.

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