Center for Hormonforstyrrende Stoffer

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Indhold

Humane studier ved Afd. for Vækst og Reproduktion, Rigshospitalet

Søgning er udført på PubMed og dækker perioden 26. november 2014 - 10. marts 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
For søgetermen "endocrine disrupters" har vi fjernet alle de hits, der også fremkomved de øvrige søgninger.

De udvalgte artikler kredser om emnet fertilitet, både i form af ventetid til graviditet og i form af kønsratioen hos afkommet. Desuden er medtaget en artikel om blodtryksændringer efter BPA indtag, og om associationer mellem udsættelse for phthalater i fostertilværelsen og senere reproduktionsfunktion hos unge mænd.

Siden sidst er der desuden udkommet 4 artikler om omkostninger forbundet med endocrine disrupters (disse er highlightet i bruttolisten). Vi har ikke inkluderet kommentarer til disse studier, da vi allerede har korresponderet med Miljøstyrelsen om dem. Det er muligt at lære mere om undersøgelserne på siden http://www.healthandenvironment.org/partnership_calls/16776, hvor der ligger præsentationer af L Trasande og P Grandjean, der begge har været centrale drivkræfter i arbejdet, der ligger bag studierne.

God læselyst!

Udvalgte publikationer

Exposure to bisphenol A from drinking canned beverages increases blood pressure: randomized crossover trial
Bae S, Hong YC.
Hypertension. 2015 Feb;65(2):313-9

Bisphenol A (BPA) is a chemical used in plastic bottles and inner coating of beverage cans, and its exposure is almost ubiquitous. BPA has been associated with hypertension and decreased heart rate variability in the previous studies. The aim of the present study was to determine whether increased BPA exposure from consumption of canned beverage actually affects blood pressure and heart rate variability. We conducted a randomized crossover trial with noninstitutionalized adults, who were aged ≥60 years and recruited from a local community center. A total of 60 participants visited the study site 3 times, and they were provided the same beverage in 2 glass bottles, 2 cans, or 1 can and 1 glass bottle at a time. The sequence of the beverage was randomized. We then measured urinary BPA concentration, blood pressure, and heart rate variability 2 hours after the consumption of each beverage. The paired t test and mixed model were used to compare the differences. The urinary BPA concentration increased after consuming canned beverages by >1600% compared with that after consuming glass bottled beverages. Systolic blood pressure adjusted for daily variance increased by ≈4.5 mm Hg after consuming 2 canned beverages compared with that after consuming 2 glass bottled beverages, and the difference was statistically significant. The parameters of the heart rate variability did not show statistically significant differences.The present study demonstrated that consuming canned beverage and consequent increase of BPA exposure increase blood pressure acutely.

Maternal exposure to perfluorinated chemicals and reduced fecundity: the MIREC study.
Vélez MP, Arbuckle TE, Fraser WD
Hum Reprod. 2015 Mar;30(3):701-9

STUDY QUESTION: What is the effect of maternal exposure to perfluorooctane sulfonate (PFOS), perfluorooctanoic acid (PFOA) and perfluorohexane sulfonate (PFHxS) on female fecundity?
SUMMARY ANSWER: Increasing concentrations of PFOA or PFHxS in maternal plasma were associated with reduced fecundability and infertility.
WHAT IS KNOWN ALREADY: Perfluorinated chemicals (PFCs) are a group of synthetic compounds used in industrial production. There is a concern about the effect of PFCs on fecundity, as measured by time-to-pregnancy (TTP). Although some recent studies suggest that increasing concentrations of PFCs may decrease fecundity, divergence in the methodological approaches used to evaluate this association have prevented firm conclusions being reached.
STUDY DESIGN, SIZE, DURATION: The Maternal-Infant Research on Environmental Chemicals (MIREC) Study is a cohort study of 2,001 women recruited before 14 weeks of gestation in 10 cities across Canada between 2008 and 2011.
PARTICIPANTS/MATERIALS, SETTING, METHODS: A questionnaire was administered and medical chart data and biospecimens were collected from participants. After excluding women who withdrew, those for whom data were incomplete, those whose pregnancies followed birth control failure, and accounting for male fertility, 1743 participants remained. TTP was defined as the number of months of unprotected intercourse needed to become pregnant in the current pregnancy, as self-reported in the first trimester of pregnancy. Plasma concentrations of PFOA, PFOS and PFHxS measured in the first trimester were considered as a surrogate of preconception exposure. Fecundability odds ratios (FORs) were estimated using Cox proportional hazard models for discrete time. FOR < 1 denote a longer TTP and FORs >1 denote a shorter TTP. The odds of infertility (TTP > 12 months or infertility treatment in the index pregnancy) were estimated using logistic regression. Each chemical concentration (ng/ml) was log-transformed and divided by its SD.

MAIN RESULTS AND THE ROLE OF CHANCE: The cumulative probabilities of pregnancy at 1, 6 and 12 months were 0.42 (95% confidence interval (CI) 0.40-0.45), 0.81 (95% CI 0.79-0.83) and 0.90 (95% CI 0.89-0.92), respectively. The mean maternal age was 32.8 (SD 5.0) years. The geometric means (ng/ml) of PFOA, PFOS and PFHxS were 1.66 (95% CI 1.61-1.71), 4.59 (95% CI 4.46-4.72) and 1.01 (95% CI 0.97-1.05), respectively. After adjustment for potential confounders, PFOA and PFHxS were associated with a 11 and 9% reduction in fecundability per one SD increase (FOR = 0.89; 95% CI 0.83-0.94; P < 0.001 for PFOA and FOR = 0.91; 95% CI 0.86-0.97; P = 0.002 for PFHxS), while no significant association was observed for PFOS (FOR = 0.96; 95% CI 0.91-1.02; P = 0.17). In addition, the odds of infertility increased by 31% per one SD increase of PFOA (odds ratio (OR) = 1.31; 95% CI 1.11-1.53; P = 0.001) and by 27% per one SD increase of PFHxS (OR = 1.27; 95% CI 1.09-1.48; P = 0.003), while no significant association was observed for PFOS (OR = 1.14; 95% CI 0.98-1.34; P = 0.09).
LIMITATIONS, REASONS FOR CAUTION: Women with the highest concentrations of PFCs might have been excluded from the study if there is a causal association with infertility. The MIREC study did not assess concentrations of PFCs in males, semen quality, menstrual cycle characteristics or intercourse frequency.
WIDER IMPLICATIONS OF THE FINDINGS: Our results add to the evidence that exposure to PFOA and PFHxS, even at lower levels than previously reported, may reduce fecundability.

Perfluoroalkyl substances and time to pregnancy in couples from Greenland, Poland and Ukraine
Jørgensen KT, Specht IO, Lenters V, Bach CC, Rylander L, Jönsson BA, Lindh CH, Giwercman A, Heederik D, Toft G, Bonde JP.
Environ Health. 2014 Dec 22;13(1):116

BACKGROUND: Perfluoroalkyl substances (PFAS) are suggested to affect human fecundity through longer time to pregnancy (TTP). We studied the relationship between four abundant PFAS and TTP in pregnant women from Greenland, Poland and Ukraine representing varying PFAS exposures and pregnancy planning behaviors.
METHODS: We measured serum levels of perfluorooctanoic acid (PFOA), perfluorooctane sulfonate (PFOS), perfluorohexane sulfonic acid (PFHxS) and perfluorononanoic acid (PFNA) in 938 women from Greenland (448 women), Poland (203 women) and Ukraine (287 women). PFAS exposure was assessed on a continuous logarithm transformed scale and in country-specific tertiles. We used Cox discrete-time models and logistic regression to estimate fecundability ratios (FRs) and infertility (TTP >13 months) odds ratios (ORs), respectively, and 95% confidence intervals (CI) according to PFAS levels. Adjusted analyses of the association between PFAS and TTP were done for each study population and in a pooled sample.
RESULTS: Higher PFNA levels were associated with longer TTP in the pooled sample (log-scale FR = 0.80; 95% CI 0.69-0.94) and specifically in women from Greenland (log-scale FR = 0.72; 95% CI 0.58-0.89). ORs for infertility were also increased in the pooled sample (log-scale OR = 1.53; 95% CI 1.08-2.15) and in women from Greenland (log-scale OR = 1.97; 95% CI 1.22-3.19). However, in a sensitivity analysis of primiparous women these associations could not be replicated. Associations with PFNA were weaker for women from Poland and Ukraine. PFOS, PFOA and PFHxS were not consistently associated with TTP.
CONCLUSIONS: Findings do not provide consistent evidence that environmental exposure to PFAS is impairing female fecundity by delaying time taken to conceive.

Couples' urinary bisphenol A and phthalate metabolite concentrations and the secondary sex ratio
Bae J, Kim S, Kannan K, Buck Louis GM
Environ Res.2015 Feb;137:450-7

With limited research focusing on non-persistent chemicals as exogenous factors affecting human sex selection, this study aimed to evaluate the association of urinary bisphenol A (BPA) and phthalate metabolite concentrations with the secondary sex ratio (SSR), defined as the ratio of male to female live births. The current analysis is limited to singleton live births (n¼220, 43.9%) from the Longitudinal Investigation of Fertility and the Environment (LIFE) Study, in which couples discontinuing contraception with the intention of becoming pregnant were enrolled and followed while trying for pregnancy and through delivery for those achieving pregnancy. Using modified Poisson regression models accounting for potential confounders, we estimated the relative risks (RRs) of a male birth per standard deviation change in the log-transformed maternal, paternal, and couple urinary BPA and 14 phthalate metabolite concentrations (ng/mL) measured upon enrollment. When maternal and paternal chemical concentrations were modeled jointly, paternal BPA (RR, 0.77; 95% confidence interval [CI], 0.62–0.95) and mono-isobutyl phthalate (RR, 0.82; 95% CI, 0.67–1.00) were significantly associated with a female excess. Contrarily, maternal BPA (RR, 1.16; 95% CI, 1.03–1.31), mono-isobutyl phthalate (RR, 1.28; 95% CI, 1.06–1.54), mono-benzyl phthalate (RR, 1.31; 95% CI, 1.08–1.58), and mono-n-butyl phthalate (RR, 1.24; 95% CI, 1.01–1.51) were significantly associated with a male excess. These findings underscore varying patterns for the SSR in relation to parental exposures. Given the absence of previous investigation, these partner-specific associations of non-persistent chemicals with the SSR need future corroboration.

Prenatal phthalate exposure and reproductive function in young men
Axelsson J, Rylander L, Rignell-Hydbom A, Lindh CH, Jönsson BA, Giwercman A
Environ Res. 2015 Mar 2;138C:264-270

BACKGROUND: Prenatal exposure to phthalates is suggested to negatively impact male reproductive function, but human data are lacking.
OBJECTIVES: To study associations between prenatal exposure to diethylhexyl phthalate (DEHP) and diisononyl phthalate (DiNP), and reproductive parameters of adolescent men.
METHODS: Using linear regression models adjusted for potential confounders, we studied associations between levels of DEHP- and DiNP metabolites in maternal sera from mean 12 weeks of pregnancy, and testicular size, semen quality and reproductive hormones in 112 adolescent sons, recruited from the general population.
RESULTS: Men in the highest exposure tertile of one DiNP metabolite [mono-(carboxy-iso-octyl) phthalate], compared with men in the lowest tertile had: 4.3mL (95% CI: 0.89, 7.6mL; p<0.001) lower total testicular volume; 30% (95% CI: 3.6, 63%; p=0.02) higher levels of follicle-stimulating hormone; and 0.87mL (95% CI: 0.28, 1.5mL; p=0.004) lower semen volume. Men in the highest exposure tertile of one DEHP metabolite [mono-(2-ethyl-5-hydroxylhexyl) phthalate] had 0.70mL (95% CI: 0.090, 1.3mL; p=0.03) lower semen volume than men in the lowest exposure tertile. The levels of two DiNP metabolites [mono-(hydroxy-iso-nonyl) phthalate and mono-(oxo-iso-nonyl) phthalate] were linearly associated with luteinizing hormone (p<0.01).
CONCLUSION: Prenatal levels of some metabolites of DEHP and DiNP seemed negatively associated with reproductive function of adolescent men.

Bruttoliste

Bisphenol A

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7: Vega A, Baptissart M, Martinot E, Saru JP, Baron S, Schoonjans K, Volle DH. Hepatotoxicity induced by neonatal exposure to diethylstilbestrol is maintained throughout adulthood via the nuclear receptor SHP. Expert Opin Ther Targets. 2014 Dec;18(12):1367-76. doi: 10.1517/14728222.2014.964209. Epub 2014 Sep 29. PubMed PMID: 25263461.

8: Larsson K, Ljung Björklund K, Palm B, Wennberg M, Kaj L, Lindh CH, Jönsson BA, Berglund M. Exposure determinants of phthalates, parabens, bisphenol A and triclosan in Swedish mothers and their children. Environ Int. 2014 Dec;73:323-33.doi: 10.1016/j.envint.2014.08.014. Epub 2014 Sep 16. PubMed PMID: 25216151; PubMed Central PMCID: PMC4207945.

10. Bellanger M, Demeneix B, Grandjean P, Zoeller RT, Trasande L. Neurobehavioral Deficits, Diseases and Associated Costs of Exposure to Endocrine Disrupting Chemicals in the European Union. J Clin Endocrinol Metab. 2015 Mar 5:jc20144323. [Epub ahead of print]

11. Trasande L, Zoeller RT, Hass U, Kortenkamp A, Grandjean P, Myers JP, DiGangi J, Bellanger M, Hauser R, Legler J, Skakkebaek NE, Heindel JJ. Estimating Burden and Disease Costs of Exposure to Endocrine-Disrupting Chemicals in the European Union. J Clin Endocrinol Metab. 2015 Mar 5:jc20144324. [Epub ahead of print]

12. Hauser R, Skakkebaek NE, Hass U, Toppari J, Juul A, Andersson AM, Kortenkamp A, Heindel JJ, Trasande L. Male Reproductive Disorders, Diseases, and Costs of Exposure to Endocrine-DisruptingChemicals in the European Union. J Clin Endocrinol Metab. 2015 Mar 5:jc20144325. [Epub ahead of print]

13. Legler J, Fletcher T, Govarts E, Porta M, Blumberg B, Heindel JJ, Trasande L. Obesity, Diabetes, and Associated Costs of Exposure to Endocrine-Disrupting Chemicals in the European Union. J Clin Endocrinol Metab. 2015 Mar 5:jc20144326. [Epub ahead of print]

In vitro studier ved DTU Fødevareinstituttet

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 2014/12/30 to 2015/03/30 (Januar 2015 og fremefter)

Efter at have fjernet genganger fra forrige litteraturopdateringslister gav litteratursøgningen, med de angivne søgekriterier, tilsammen en liste med i alt 54 artikler. Artiklerne er blevet fordelt i 5 grupper:”Perflourinated and Polyflourinated 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 (fra bruttolisten) til nærmere beskrivelse baseret på, at de beskriver resultater der bidrager til ny eller yderligere viden om grupper af hormonforstyrrende stoffer.

Den første artikel omhandler *in vitro* studier med det formål, at undersøge de hormonforstyrrende egenskaber af svampetoksinet (mykotoksinet) Enniatin B.

Den anden artikel omhandler et *in vitro* studie, der har haft til formål at undersøge de hormonforstyrrende effekter af såvel kendte som "nye" fødevarekontaminanter.

An investigation of the **endocrine disrupting** potential of enniatin B using **in vitro** bioassays.
Kalayou S, Ndossi D, Frizzell C, Groseth PK, Connolly L, Sørlie M, Verhaegen S, Ropstad E.

Abstract

Evidence that some of the fungal metabolites present in food and feed may act as potential endocrine disruptors is increasing. Enniatin B (ENN B) is among the emerging Fusarium mycotoxins known to contaminate cereals. In this study, the H295R and neonatal porcine Leydig cell (LC) models, and reporter gene assays (RGAs) have been used to investigate the endocrine disrupting activity of ENN B. Aspects of cell viability, cell cycle distribution, hormone production as well as the expression of key steroidogenic genes were assessed using the H295R cell model. Cell viability and hormone production levels were determined in the LC model, while cell viability and steroid hormone nuclear receptor transcriptional activity were measured using the RGAs. ENN B (0.01-100μM) was cytotoxic in the H295R and LC models used; following 48h incubation with 100μM. Flow cytometry analysis showed that ENN B exposure (0.1-25μM) led to an increased proportion of cells in the S phase at higher ENN B doses (>10μM) while cells at G0/G1 phase were reduced. At the receptor level, ENN B (0.00156-15.6μM) did not appear to induce any specific (ant) agonistic responses in reporter gene assays (RGAs), however cell viability was affected at 15.6μM. Measurement of hormone levels in H295R cells revealed that the production of progesterone, testosterone and cortisol in exposed cells were reduced, but the level of estradiol was not significantly affected. There was a general reduction of estradiol and testosterone levels in exposed LC. Only the highest dose (100μM) used had a significant effect, suggesting the observed inhibitory effect is more likely associated with the cytotoxic effect observed at this dose. Gene transcription analysis in H295R cells showed that twelve of the sixteen genes were significantly modulated ($p<0.05$) by ENN B (10μM) compared to the control. Genes HMGR, StAR, CYP11A, 3βHSD2 and CYP17 were downregulated, whereas the expression of CYP1A1, NR0B1, MC2R, CYP21, CYP11B1, CYP11B2 and CYP19 were upregulated. The reduction of hormones and modulation of genes at the lower dose (10μM) in the H295R cells suggests that adrenal endocrine toxicity is an important potential hazard.

Endocrine disruptor activity of multiple environmental food chain contaminants.
Wielogórska E, Elliott CT, Danaher M, Connolly L.

Abstract

Industrial chemicals, antimicrobials, drugs and personal care products have been reported as global pollutants which enter the food chain. Some of them have also been classified as endocrine disruptors based on results of various studies employing a number of in vitro/vivo tests. The present study employed a mammalian reporter gene assay to assess the effects of known and emerging contaminants on estrogen nuclear receptor transactivation. Out of fifty-nine compounds assessed, estrogen receptor agonistic activity was observed for parabens (n = 3), UV filters (n = 6), phthalates (n = 4) and a metabolite, pyrethroids (n = 9) and their metabolites (n = 3). Two compounds were estrogen receptor antagonists while some of the agonists

enhanced 17β-estradiol mediated response. This study reports five new compounds (pyrethroids and their metabolites) possessing estrogen agonist activity and highlights for the first time that pyrethroid metabolites are of particular concern showing much greater estrogenic activity than their parent compounds.

Bruttoliste

Perflourinated and Polyflourinated compounds

1. Transformation Pathways of Isomeric **Perfluorooctanesulfonate** Precursors Catalyzed by the Active Species of P450 Enzymes: In Silico Investigation.

Fu Z, Wang Y, Wang Z, Xie H, Chen J.
Chem Res Toxicol. 2015 Jan 12. [Epub ahead of print]

2. Effect of **perfluorooctane** sulfonate on pluripotency and differentiation factors in mouse embryoid bodies.

Xu B, Ji X, Chen X, Yao M, Han X, Chen M, Tang W, Xia Y.
Toxicology. 2015 Feb 3;328:160-7. doi: 10.1016/j.tox.2014.12.010. Epub 2014 Dec 12.

3. The effects of **perfluorinated** chemicals on adipocyte differentiation **in vitro**.

Watkins AM, Wood CR, Lin MT, Abbott BD.
Mol Cell Endocrinol. 2015 Jan 15;400:90-101. doi: 10.1016/j.mce.2014.10.020. Epub 2014 Nov 5.

Plastic derivatives" (BPA, Phthalates and others)

1. Bisphenol A reduces fertilizing ability and motility by compromising mitochondrial function of sperm.

Singh RP, Shafeeque CM, Sharma SK, Pandey NK, Singh R, Mohan J, Kolluri G, Saxena M, Sharma B, Sastry KV, Kataria JM, Azeez PA.
Environ Toxicol Chem. 2015 Feb 27. doi: 10.1002/etc.2957. [Epub ahead of print]

2. 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 Feb 25. pii: S1874-9399(15)00051-6. doi: 10.1016/j.bbagrm.2015.02.003. [Epub ahead of print]

3. An investigation of the **endocrine-disruptive** effects of bisphenol a in human and rat fetal testes.

Maamar MB, Lesné L, Desdoits-Lethimonier C, Coiffec I, Lassurguère J, Lavoué V, Deceuninck Y, Antignac JP, Le Bizec B, Perdu E, Zalko D, Pineau C, Chevrier C, Dejucq-Rainsford N, Mazaud-Guittot S, Jégou B.
PLoS One. 2015 Feb 23;10(2):e0117226. doi: 10.1371/journal.pone.0117226. eCollection 2015.

4. Cell-Specific Biotransformation of Benzophenone-2 and Bisphenol-S in Zebrafish and Human **in Vitro** Models Used for Toxicity and Estrogenicity Screening.

Le Fol V, Aït-Aïssa S, Cabaton N, Dolo L, Grimaldi M, Balaguer P, Perdu E, Debrauwer L, Brion F, Zalko D.
Environ Sci Technol. 2015 Mar 2. [Epub ahead of print]

5. Structural bisphenol analogues differentially target steroidogenesis in murine MA-10 Leydig cells as well as the glucocorticoid receptor.

Roelofs MJ, Berg Mv, Bovee TF, Piersma AH, Duursen MB.
Toxicology. 2015 Mar 2;329:10-20. doi: 10.1016/j.tox.2015.01.003. Epub 2015 Jan 8.

6. The **In Vitro** Immune Modulatory Effect of Bisphenol A on Fish Macrophages via Estrogen Receptor α and Nuclear Factor-κB Signaling.

Yang M, Qiu W, Chen B, Chen J, Liu S, Wu M, Wang KJ.
Environ Sci Technol. 2015 Feb 3;49(3):1888-95. doi: 10.1021/es505163v. Epub 2015 Jan 21.

7. Evaluation of the toxic effects of brominated compounds (BDE-47, 99, 209, TBBPA) and bisphenol A (BPA) using a zebrafish liver cell line, ZFL.

Yang J, Chan KM.
Aquat Toxicol. 2015 Feb;159:138-47. doi: 10.1016/j.aquatox.2014.12.011. Epub 2014 Dec 18.

8. Di-(2-ethylhexyl)-**phthalate** induces oxidative stress in human endometrial stromal cells **in vitro**.

Cho YJ, Park SB, Han M.
Mol Cell Endocrinol. 2015 Mar 9. pii: S0303-7207(15)00114-8. doi: 10.1016/j.mce.2015.03.003. [Epub ahead of print]

9. Di(2-ethylhexyl) **phthalate** inhibits B cell proliferation and reduces the abundance of IgM-secreting cells in cultured immune tissues of the rainbow trout.

Martins K, Applegate B, Hagedorn B, Kennish J, Zwollo P.
Fish Shellfish Immunol. 2015 Mar 5. pii: S1050-4648(15)00096-0. doi: 10.1016/j.fsi.2015.02.037. [Epub ahead of print]

10. 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 Feb 18;284(1):42-53. doi: 10.1016/j.taap.2015.02.010. [Epub ahead of print]

11. Identification of di-2-ethylhexyl terephthalate (DEHTP) metabolites using human liver microsomes for biomonitoring applications.

Silva MJ, Samandar E, Calafat AM, Ye X.
Toxicol **In Vitro**. 2015 Feb 14;29(4):716-721. doi: 10.1016/j.tiv.2015.02.002. [Epub ahead of print]

12. The effect of mono-(2-ethylhexyl) **phthalate** on apoptosis of rat ovarian granulosa cells **in vitro**.

Li N, Liu K, Yuan H, Zhu J, Yu G, Xie J, Fu S, Guo K, Ye L.
Environ Toxicol Pharmacol. 2015 Jan 20;39(2):643-650. doi: 10.1016/j.etap.2015.01.005. [Epub ahead of print]

13. **In vitro** evaluation of the quality of blood products collected and stored in systems completely free of di(2-ethylhexyl)**phthalate**-plasticized materials.

Lagerberg JW, Gouwerok E, Vlaar R, Go M, de Korte D.
Transfusion. 2015 Mar;55(3):522-31. doi: 10.1111/trf.12870. Epub 2014 Oct 21.

Parabens

1. Paternal Urinary Concentrations of **Parabens** and Other Phenols in Relation to Reproductive Outcomes among Couples from a Fertility Clinic.

Dodge LE, Williams PL, Williams MA, Missmer SA, Toth TL, Calafat AM, Hauser R.
Environ Health Perspect. 2015 Mar 13. [Epub ahead of print]

2. Associations between urinary phenol and **paraben** concentrations and markers of oxidative stress and inflammation among pregnant women in Puerto Rico.

Watkins DJ, Ferguson KK, Anzalota Del Toro LV, Alshawabkeh AN, Cordero JF, Meeker JD.
Int J Hyg Environ Health. 2015 Mar;218(2):212-9. doi: 10.1016/j.ijheh.2014.11.001. Epub 2014 Nov 18.

Pesticides/Fungicides/Insecticides/Biocides

1. Very low-dose (femtomolar) 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) disrupts steroidogenic enzyme mRNAs and steroid secretion by human luteinizing granulosa cells.
Baldridge MG, Marks GT, Rawlins RG, Hutz RJ.
Reprod Toxicol. 2015 Feb 16;52:57-61. doi: 10.1016/j.reprotox.2015.02.003. [Epub ahead of print]

2. Effects of currently used pesticides and their mixtures on the function of thyroid hormone and aryl hydrocarbon receptor in cell culture.

Ghisari M, Long M, Tabbo A, Bonefeld-Jørgensen EC.
Toxicol Appl Pharmacol. 2015 Feb 12. pii: S0041-008X(15)00055-1. doi: 10.1016/j.taap.2015.02.004. [Epub ahead of print]

3. **In vitro** study of the binding between chlorpyrfos and sex hormones using headspace solid-phase microextraction combined with high-performance liquid chromatography: A new aspect of pesticides and breast cancer risk.

Farhadi K, Tahmasebi R, Biparva P, Maleki R.
Hum Exp Toxicol. 2015 Feb 11. pii: 0960327114559990. [Epub ahead of print]

4. Dietary exposure to the **endocrine disruptor** tolylfluanid promotes global metabolic dysfunction in male mice.

Regnier SM, Kirkley AG, Ye H, El-Hashani E, Zhang X, Neel BA, Kamau W, Thomas CC, Williams AK, Hayes ET, Massad NL, Johnson DN, Huang L, Zhang C, Sargis RM.
Endocrinology. 2015 Mar;156(3):896-910. doi: 10.1210/en.2014-1668. Epub 2014 Dec 23.

5. Diofenolan induces male offspring production through binding to the juvenile hormone receptor in Daphnia magna.

Abe R, Toyota K, Miyakawa H, Watanabe H, Oka T, Miyagawa S, Nishide H, Uchiyama I, Tollefsen KE, Iguchi T, Tatarazako N.
Aquat Toxicol. 2015 Feb;159:44-51. doi: 10.1016/j.aquatox.2014.11.015. Epub 2014 Nov 24.

Various EDCs, Mixtures and Other endpoints

1. Steroid Hormone Related Effects of Marine Persistent Organic Pollutants in Human H295R Adrenocortical Carcinoma Cells.

van den Dungen MW, Rijk JC, Kampman E, Steegenga WT, Murk AJ.
Toxicol **In Vitro**. 2015 Mar 9. pii: S0887-2333(15)00043-0. doi: 10.1016/j.tiv.2015.03.002. [Epub ahead of print]

2. Cadmium at nanomolar concentrations activates Raf-MEK-ERK1/2 MAPKs signaling via EGFR in human cancer cell lines.

Ali I, Damdimopoulou P, Stenius U, Halldin K.
Chem Biol Interact. 2015 Mar 2;231:44-52. doi: 10.1016/j.cbi.2015.02.014. [Epub ahead of print]

3. **Endocrine disruptive** effects of cadmium on steroidogenesis: Human adrenocortical carcinoma cell line NCI-H295R as a cellular model for reproductive toxicity testing.

Knazicka Z, Forgacs Z, Lukacova J, Roychoudhury S, Massanyi P, Lukac N.
J Environ Sci Health A Tox Hazard Subst Environ Eng. 2015 Mar 21;50(4):348-56. doi: 10.1080/10934529.2015.987520.

4. The ChemScreen project to design a pragmatic alternative approach to predict reproductive toxicity of chemicals.

van der Burg B, Wedebye EB, Dietrich DR, Jaworska J, Mangelsdorf I, Paune E, Schwarz M, Piersma AH, Kroese ED.
Reprod Toxicol. 2015 Feb 2. pii: S0890-6238(15)00009-X. doi: 10.1016/j.reprotox.2015.01.008. [Epub ahead of print]

5. An exposure:activity profiling method for interpreting high-throughput screening data for estrogenic activity-Proof of concept.

Becker RA, Friedman KP, Simon TW, Marty MS, Patlewicz G, Rowlands JC.
Regul Toxicol Pharmacol. 2015 Feb 3;71(3):398-408. doi: 10.1016/j.yrtph.2015.01.008. [Epub ahead of print]

6. Effects of the brominated flame retardant TBCO on fecundity and profiles of transcripts of the HPGL-axis in Japanese medaka.

Saunders DM, Podaima M, Wiseman S, Giesy JP.
Aquat Toxicol. 2015 Mar;160:180-7. doi: 10.1016/j.aquatox.2015.01.018. Epub 2015 Jan 22.

7. Integrated assessment of wastewater treatment plant effluent estrogenicity in the Upper Murray River, Australia, using the native Murray rainbowfish (Melanotaenia fluviatilis).

Vajda AM, Kumar A, Woods M, Williams M, Doan H, Tolsher P, Kookana RS, Barber LB.
Environ Toxicol Chem. 2015 Jan 23. doi: 10.1002/etc.2895. [Epub ahead of print]

8. An investigation of the **endocrine disrupting** potential of enniatin B using **in vitro** bioassays.

Kalayou S, Ndossi D, Frizzell C, Groseth PK, Connolly L, Sørlie M, Verhaegen S, Ropstad E.
Toxicol Lett. 2015 Mar 4;233(2):84-94. doi: 10.1016/j.toxlet.2015.01.014. Epub 2015 Jan 24.

9. Persistent organic pollutants and obesity: are they potential mechanisms for breast cancer promotion?

Reaves DK, Ginsburg E, Bang JJ, Fleming JM.
Endocr Relat Cancer. 2015 Apr;22(2):R69-R86. Epub 2015 Jan 26. Review.

10. Analysis of the developing neural system using an **in vitro** model by Raman spectroscopy.

Hashimoto K, Kudoh SN, Sato H.
Analyst. 2015 Jan 22. [Epub ahead of print]

11. 4-Methylbenzylidene camphor microspheres: reconstituted epidermis (Skinethic®) permeation and distribution.

Monti D, Chetoni P, Burgalassi S, Tampucci S, Centini M, Anselmi C.
Int J Cosmet Sci. 2015 Jan 6. doi: 10.1111/ics.12199. [Epub ahead of print]

12. Bioaccumulation, Biotransformation, and Toxicity of BDE-47, 6-OH-BDE-47, and 6-MeO-BDE-47 in Early Life-Stages of Zebrafish (Danio rerio).

Liu H, Tang S, Zheng X, Zhu Y, Ma Z, Liu C, Hecker M, Saunders DM, Giesy JP, Zhang X, Yu H.
Environ Sci Technol. 2015 Feb 3;49(3):1823-33. doi: 10.1021/es503833q. Epub 2015 Jan 21.

13. Whole organism responses and intersex severity in rainbow darter (Etheostoma caeruleum) following exposures to municipal wastewater in the Grand River basin, ON, Canada. Part A.

Bahamonde PA, Fuzzen ML, Bennett CJ, Tetreault GR, McMaster ME, Servos MR, Martyniuk CJ, Munkittrick KR.
Aquat Toxicol. 2015 Feb;159:290-301. doi: 10.1016/j.aquatox.2014.11.023. Epub 2014 Dec 3.

14. 6-OH-BDE-47 promotes human lung cancer cells epithelial mesenchymal transition via the AKT/Snail signal pathway.

Qu BL, Yu W, Huang YR, Cai BN, Du LH, Liu F.
Environ Toxicol Pharmacol. 2015 Jan;39(1):271-9. doi: 10.1016/j.etap.2014.11.022. Epub 2014 Dec 5.

15.Direct effects of the algal toxin, domoic acid, on ovarian function: Bovine granulosa and theca cells as an **in vitro** model.

Pizzo F, Caloni F, Schreiber NB, Schutz LF, Totty ML, Albonico M, Spicer LJ.
Ecotoxicol Environ Saf. 2015 Mar;113:314-20. doi: 10.1016/j.ecoenv.2014.12.009. Epub 2014 Dec 18.

16.Metabolism of UV-filter benzophenone-3 by rat and human liver microsomes and its effect on **endocrine-disrupting** activity.

Watanabe Y, Kojima H, Takeuchi S, Uramaru N, Sanoh S, Sugihara K, Kitamura S, Ohta S.
Toxicol Appl Pharmacol. 2015 Jan 15;282(2):119-28. doi: 10.1016/j.taap.2014.12.002. Epub 2014 Dec 17.

17. **In vitro** re-expression of the aryl hydrocarbon receptor (Ahr) in cultured Ahr-deficient mouse antral follicles partially restores the phenotype to that of cultured wild-type mouse follicles.

Ziv-Gal A, Gao L, Karman BN, Flaws JA.
Toxicol **In Vitro**. 2015 Mar;29(2):329-36. doi: 10.1016/j.tiv.2014.11.011. Epub 2014 Dec 9.

18. Effects of progesterone and norethindrone on female fathead minnow (Pimephales promelas) steroidogenesis.

Petersen LH, Hala D, Carty D, Cantu M, Martinović D, Huggett DB.
Environ Toxicol Chem. 2015 Feb;34(2):379-90. doi: 10.1002/etc.2816. Epub 2015 Jan 6.

19. **In vitro** immune toxicity of polybrominated diphenyl ethers on murine peritoneal macrophages: apoptosis and immune cell dysfunction.

Lv QY, Wan B, Guo LH, Zhao L, Yang Y.
Chemosphere. 2015 Feb;120:621-30. doi: 10.1016/j.chemosphere.2014.08.029. Epub 2014 Oct 28.

20. A mixture of the novel brominated flame retardants TBPH and TBB affects fecundity and transcript profiles of the HPGL-axis in Japanese medaka.

Saunders DM, Podaima M, Codling G, Giesy JP, Wiseman S.
Aquat Toxicol. 2015 Jan;158:14-21. doi: 10.1016/j.aquatox.2014.10.019. Epub 2014 Nov 1.

21. 17β-trenbolone, an anabolic-androgenic steroid as well as an environmental hormone, contributes to neurodegeneration.

Ma F, Liu D.
Toxicol Appl Pharmacol. 2015 Jan 1;282(1):68-76. doi: 10.1016/j.taap.2014.11.007. Epub 2014 Nov 25.

22. Screening of hormone-like activities in bottled waters available in Southern Spain using receptor-specific bioassays.

Real M, Molina-Molina JM, Jiménez-Díaz I, Arrebola JP, Sáenz JM, Fernández MF, Olea N.
Environ Int. 2015 Jan;74:125-35. doi: 10.1016/j.envint.2014.10.006. Epub 2014 Oct 27.

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

Kharlyngdoh JB, Pradhan A, Asnake S, Walstad A, Ivarsson P, Olsson PE.
Environ Int. 2015 Jan;74:60-70. doi: 10.1016/j.envint.2014.09.002. Epub 2014 Oct 20.

24. **Endocrine disruptor** activity of multiple environmental food chain contaminants.

Wielogórska E, Elliott CT, Danaher M, Connolly L.
Toxicol **In Vitro**. 2015 Feb;29(1):211-20.

25. Liposomal Aloe vera trans-emulgel drug delivery of naproxen and nimesulide: A study.

Venkataharsha P, Maheshwara E, Raju YP, Reddy VA, Rayadu BS, Karisetty B.
Int J Pharm Investig. 2015 Jan-Mar;5(1):28-34. doi: 10.4103/2230-973X.147230.

26. Effect of tea tree (Melaleuca alternifolia) oil as a natural antimicrobial agent in lipophilic formulations.

Mantil E, Daly G, Avis TJ.
Can J Microbiol. 2015 Jan;61(1):82-8. doi: 10.1139/cjm-2014-0667.

27. Magnetic nanoparticle and magnetic field assisted siRNA delivery **in vitro**.

Mykhaylyk O, Sanchez-Antequera Y, Vlaskou D, Cerda MB, Bokharaei M, Hammerschmid E, Anton M, Plank C.
Methods Mol Biol. 2015;1218:53-106. doi: 10.1007/978-1-4939-1538-5_5.

28. Potent protein tyrosine phosphatase 1B (PTP1B) inhibiting constituents from Anoectochilus chapaensis and molecular docking studies.

Cai J, Zhao L, Tao W.
Pharm Biol. 2015 Jan 22:1-5. [Epub ahead of print]

29. The crystal structure of the versatile cytochrome P450 enzyme CYP109B1 from Bacillus subtilis.

Zhang A, Zhang T, Hall EA, Hutchinson S, Cryle MJ, Wong LL, Zhou W, Bell SG.
Mol Biosyst. 2015 Mar 17;11(3):869-81. doi: 10.1039/c4mb00665h. Epub 2015 Jan 14.

30. A novel transcriptomics based **in vitro** method to compare and predict hepatotoxicity based on mode of action.

De Abrew KN, Overmann GJ, Adams RL, Tiesman JP, Dunavent J, Shan YK, Carr GJ, Daston GP, Naciff JM.
Toxicology. 2015 Feb 3;328:29-39. doi: 10.1016/j.tox.2014.11.008. Epub 2014 Dec 2.

31. **In vitro** and in vivo evaluations of a novel pulsed and controlled osmotic pump capsule.

Zhang W, Zhang L, Qu X, Zhu Z, Pan Y, Guan J, Pan W.
Drug Dev Ind Pharm. 2015 Feb;41(2):322-32. doi: 10.3109/03639045.2013.859265. Epub 2013 Dec 3.

In Vivo studier ved DTU Fødevareinstituttet

Søgning er udført på PubMed og dækker perioden midt Dec. 2014- medio Marts 2015

(September- primo December 2014)

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 toxicity) OR (Burden or cost) AND ((Endocrine disrupt*).

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 54 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”, ” Burden & Cost of exposure” og Various EDCs, Mixtures and Other endpoints”.

Udvalgte publikationer

Tre artikler er blevet udvalgt til nærmere beskrivelse (abstrakt og konklusion). Disse artikler er valgt fordi vi mener de bidrager til ny viden om hormonforstyrrende stoffer og her er der særligt fokus på xenograftstudier og kønsceller. Den første artikel er et xenograft studie med DBP og DEHP (Rodriguez-Sosa et al. 2014 December). Den anden artikel kommer fra Richard Sharpes gruppe og har til formål at undersøge effekten af phthalateksponering på humane føtale kønsceller i et xenograft studie (van den Driesche S et al. 2015). Den sidste artikel er fra Bernard Jégous gruppe. Studiet undersøger mulige direkte hormonforstyrrelser af Bisphenol A (BPA) på føtale testikler fra 2 rottestammer og mennesker (Maamar MB et al. 2015).

Ud fra bruttolisten (se længere nede i dokumentet) er udvalgt følgende 3 artikler til engelsk abstrakt og dansk resume og kommentarer.

Phthalate esters affect maturation and **function** of primate testis tissue ectopically grafted in mice.

Rodriguez-Sosa JR, Bondareva A, Tang L, Avelar GF, Coyle KM, Modelski M, Alpaugh W, Conley A, Wynne-Edwards K, França LR, Meyers S, Dobrinski I.
Mol Cell Endocrinol. 2014 Dec;398(1-2):89-100. doi: 10.1016/j.mce.2014.10.004. Epub 2014 Oct 27

Abstract
Di-n-Butyl (DBP) and Di-(2-EthylHexyl) (DEHP) phthalates can leach from daily-use products resulting in environmental exposure. In male rodents, phthalate exposure results in reproductive effects. To evaluate effects on the immature primate testis, testis fragments from 6-month-old rhesus macaques were grafted subcutaneously to immune-deficient mice, which were exposed to 0, 10, or 500 mg/kg of DBP or DEHP for 14 weeks or 28 weeks (DBP only). DBP exposure reduced the expression of key steroidogenic genes, indicating that Leydig cell function was compromised. Exposure to 500 mg/kg impaired tubule formation and germ cell differentiation and reduced numbers of spermatogonia. Exposure to 10 mg/kg did not affect development, but reduced Sertoli cell number and resulted in increased expression of inhibin B. Exposure to DEHP for 14 week also affected steroidogenic genes expression. Therefore, long-term exposure to phthalate esters affected development and function of the primate testis in a time and dosage dependent manner.

Comparative Effects of **Di(n-Butyl) Phthalate** Exposure on Fetal Germ Cell Development in the Rat and in Human Fetal Testis Xenografts.

van den Driesche S, McKinnell C, Calarrão A, Kennedy L, Hutchison GR, Hrabalkova L, Jobling MS, Macpherson S, Anderson RA, Sharpe RM, Mitchell RT.
Environ Health Perspect. 2015 Mar;123(3):223-30. doi: 10.1289/ehp.1408248. Epub 2014 Dec 16.

Abstract:

BACKGROUND:
Phthalate exposure induces germ cell effects in the fetal rat testis. Although experimental models have shown that the human fetal testis is insensitive to the steroidogenic effects of phthalates, the effects on germ cells have been less explored.

OBJECTIVES:
We sought to identify the effects of phthalate exposure on human fetal germ cells in a dynamic model and to establish whether the rat is an appropriate model for investigating such effects.

METHODS:
We used immunohistochemistry, immunofluorescence, and quantitative real-time polymerase chain reaction to examine Sertoli and germ cell markers on rat testes and human fetal testis xenografts after exposure to vehicle or di(n-butyl) phthalate (DBP). Our study included analysis of germ cell differentiation markers, proliferation markers, and cell adhesion proteins.

RESULTS:
In both rat and human fetal testes, DBP exposure induced similar germ cell effects, namely, germ cell loss (predominantly undifferentiated), induction of multinucleated gonocytes (MNGs), and aggregation of differentiated germ cells, although the latter occurred rarely in the human testes. The mechanism for germ cell aggregation and MNG induction appears to be loss of Sertoli cell-germ cell membrane adhesion, probably due to Sertoli cell microfilament redistribution.

CONCLUSIONS:
Our findings provide the first comparison of DBP effects on germ cell number, differentiation, and aggregation in human testis xenografts and in vivo in rats. We observed comparable effects on germ cells in both species, but the effects in the human were muted compared with those in the rat. Nevertheless, phthalate effects on germ cells have potential implications for the next generation, which merits further study. Our results indicate that the rat is a human-relevant model in which to explore the mechanisms for germ cell effects.

An **investigation** of the **endocrine-disruptive** effects of bisphenol a in human and rat fetal testes.
Maamar MB, Lesné L, Desdoits-Lethimonier C, Coiffec I, Lassurguère J, Lavoué V, Deceuninck Y, Antignac JP, Le Bizec B, Perdu E, Zalko D, Pineau C, Chevrier C, Dejucq-Rainsford N, Mazaud-Guittot S, Jégou B.
PLoS One. 2015 Feb 23;10(2):e0117226. doi: 10.1371/journal.pone.0117226. eCollection 2015.

Abstract

Few studies have been undertaken to assess the possible effects of bisphenol A (BPA) on the reproductive hormone balance in animals or humans with often contradictory results. We investigated possible direct endocrine disruption by BPA of the fetal testes of 2 rat strains (14.5-17.5 days post-coitum) and humans (8-12 gestational weeks) and under different culture conditions. BPA concentrations of 10-8M and 10-5M for 72h reduced testosterone production by the Sprague-Dawley fetal rat testes, while only 10-5M suppressed it in the Wistar strain. The suppressive effects at 10-5M were seen as early as 24h and 48h in both strains. BPA at 10-7-10-5M for 72h suppressed the levels of fetal rat Leydig cell insulin-like factor 3 (INSL3). BPA exposure at 10-8M, 10-7M, and 10-5M for 72h inhibited testosterone production in fetal human testes. For the lowest doses, the effects observed occurred only when no gonadotrophin was added to the culture media and were associated with a poorly preserved testicular morphology. We concluded that (i) BPA can display anti-androgenic effects both in rat and human fetal testes; (ii) it is essential to ascertain that the divergent effects of endocrine disruptors between species in vitro do not result from the culture conditions used, and/or the rodent strain selected; (iii) the optimization of each in vitro assay for a given species should be a major objective rather than the search of an hypothetical trans-species

consensual model-system, as the organization of the testis is intrinsically different between mammalian species; (iv) due to the uncertainty existing on the internal exposure of the human fetal testis to BPA, and the insufficient number of epidemiological studies on the endocrine disruptive effects of BPA, caution should be taken in the extrapolation of our present results to the human reproductive health after fetal exposure to BPA.

Bruttoliste

Plastic derivatives (BPA, Phthalates and others)

BPA (and alternatives)

1. **Bisphenol A** reduces fertilizing ability and motility by compromising mitochondrial function of sperm.
Singh RP, Shafeeque CM, Sharma SK, Pandey NK, Singh R, Mohan J, Kolluri G, Saxena M, Sharma B, Sastry KV, Kataria JM, Azeez PA.
Environ Toxicol Chem. 2015 Feb 27. doi: 10.1002/etc.2957. [Epub ahead of print]

2. An investigation of the **endocrine-disruptive** effects of **bisphenol a** in human and rat fetal testes.
Maamar MB, Lesné L, Desdoits-Lethimonier C, Coiffec I, Lassurguère J, Lavoué V, Deceuninck Y, Antignac JP, Le Bizec B, Perdu E, Zalko D, Pineau C, Chevrier C, Dejucq-Rainsford N, Mazaud-Guittot S, Jégou B. PLoS One. 2015 Feb 23;10(2):e0117226. doi: 10.1371/journal.pone.0117226. eCollection 2015.(**valgt**)

3. Environmental Health Factors and Sexually Dimorphic Differences in **Behavioral** Disruptions.
Rosenfeld CS, Trainor BC.
Curr Environ Health Rep. 2014 Dec;1(4):287-301.

4. **Bisphenol A** exposure induces metabolic disorders and enhances atherosclerosis in hyperlipidemic rabbits.
Fang C, Ning B, Waqar AB, Niimi M, Li S, Satoh K, Shiomi M, Ye T, Dong S, Fan J.
J Appl Toxicol. 2015 Jan 23. doi: 10.1002/jat.3103. [Epub ahead of print]

5. Identification of secretaglobin Scgb2a1 as a target for developmental reprogramming by **BPA** in the rat prostate.
Wong RL, Wang Q, Treviño LS, Bosland MC, Chen J, Medvedovic M, Prins GS, Kannan K, Ho SM, Walker CL.
Epigenetics. 2015 Jan 22:0. [Epub ahead of print]

6. Impact of gestational **bisphenol a** on oxidative stress and free Fatty acids: human association and interspecies animal testing studies.
Veiga-Lopez A, Pennathur S, Kannan K, Patisaul HB, Dolinoy DC, Zeng L, Padmanabhan V.
Endocrinology. 2015 Mar;156(3):911-22. doi: 10.1210/en.2014-1863. Epub 2015 Jan 20.

7. Sustained reprogramming of the estrogen response after chronic exposure to **endocrine disruptors**.
Patterson AR, Mo X, Shapiro A, Wernke KE, Archer TK, Burd CJ.
Mol Endocrinol. 2015 Mar;29(3):384-95. doi: 10.1210/me.2014-1237. Epub 2015 Jan 16.

8. Alterations of neurotransmitter norepinephrine and gamma-aminobutyric acid correlate with murine **behavioral** perturbations related to **bisphenol A** exposure.
Ogi H, Itoh K, Ikegaya H, Fushiki S.
Brain Dev. 2015 Jan 7. pii: S0387-7604(14)00293-9. doi: 10.1016/j.braindev.2014.12.008. [Epub ahead of print]

9. Structural bisphenol analogues differentially target steroidogenesis in murine MA-10 Leydig cells as well as the glucocorticoid receptor.
Roelofs MJ, Berg Mv, Bovee TF, Piersma AH, Duursen MB.
Toxicology. 2015 Mar 2;329:10-20. doi: 10.1016/j.tox.2015.01.003. Epub 2015 Jan 8.

10. 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, Leranth C.
J Comp Neurol. 2014 Dec 30. doi: 10.1002/cne.23735. [Epub ahead of print]

11. **Bisphenol-A** exposure during adolescence leads to enduring alterations in cognition and dendritic spine density in adult male and female rats.
Bowman RE, Luine V, Diaz Weinstein S, Khandaker H, DeWolf S, Frankfurt M.
Horm Behav. 2014 Dec 30;69C:89-97. doi: 10.1016/j.yhbeh.2014.12.007. [Epub ahead of print]

12. **Bisphenol A** as epigenetic modulator: setting the stage for carcinogenesis?
Ferreira LL, Couto R, Oliveira PJ.
Eur J Clin Invest. 2015 Jan;45 Suppl 1:32-6. doi: 10.1111/eci.12362.

13. A new chapter in the **bisphenol A** story: bisphenol S and bisphenol F are not safe alternatives to this compound.
Eladak S, Grisin T, Moison D, Guerquin MJ, N'Tumba-Byn T, Pozzi-Gaudin S, Benachi A, Livera G, Rouiller-Fabre V, Habert R.
Fertil Steril. 2015 Jan;103(1):11-21. doi: 10.1016/j.fertnstert.2014.11.005. Epub 2014 Dec 2.

14. **BPA**, an energy balance disruptor.
Le Corre L, Besnard P, Chagnon MC.
Crit Rev Food Sci Nutr. 2015;55(6):769-77. doi: 10.1080/10408398.2012.678421.

15. **Bisphenol a** and risk management ethics.
Resnik DB, Elliott KC.
Bioethics. 2015 Mar;29(3):182-9. doi: 10.1111/bioe.12079. Epub 2014 Jan 29.

16. The effect of **bisphenol A** on some oxidative stress parameters and acetylcholinesterase activity in the heart of male albino rats.
Aboul Ezz HS, Khadrawy YA, Mourad IM.
Cytotechnology. 2015 Jan;67(1):145-55. doi: 10.1007/s10616-013-9672-1. Epub 2013 Dec 12.

17. Bisphenol A regulates the estrogen receptor alpha signaling in developing hippocampus of male **rats** through estrogen receptor.
Xu XB, He Y, Song C, Ke X, Fan SJ, Peng WJ, Tan R, Kawata M, Matsuda K, Pan BX, Kato N.
Hippocampus. 2014 Dec;24(12):1570-80. doi: 10.1002/hipo.22336. Epub 2014 Aug 13.

18. Alterations of neurotransmitter norepinephrine and gamma-aminobutyric acid correlate with murine behavioral perturbations related to bisphenol A exposure.
Ogi H, Itoh K, Ikegaya H, Fushiki S.
Brain Dev. 2015 Jan 7. pii: S0387-7604(14)00293-9. doi: 10.1016/j.braindev.2014.12.008. [Epub ahead of print]

Phthalates and others
1. **Comparative Effects** of **Di(n-Butyl) Phthalate** Exposure on Fetal Germ Cell Development in the Rat and in Human Fetal Testis Xenografts.

van den Driesche S, McKinnell C, Calarrão A, Kennedy L, Hutchison GR, Hrabalkova L, Jobling MS, Macpherson S, Anderson RA, Sharpe RM, Mitchell RT.
Environ Health Perspect. 2015 Mar;123(3):223-30. doi: 10.1289/ehp.1408248. Epub 2014 Dec 16. (**valgt**)

2. **Phthalate esters affect maturation** and **function** of primate testis tissue ectopically grafted in mice.
Rodriguez-Sosa JR, Bondareva A, Tang L, Avelar GF, Coyle KM, Modelski M, Alpaugh W, Conley A, Wynne-Edwards K, França LR, Meyers S, Dobrinski I.
Mol Cell Endocrinol. 2014 Dec;398(1-2):89-100. doi: 10.1016/j.mce.2014.10.004. Epub 2014 Oct 27(**valgt**)

3. The effects of di(2-ethylhexyl) phthalate and/or selenium on trace element levels in different organs of **rats**.
Erkekoglu P, Arnaud J, Rachidi W, Kocer-Gumusel B, Favier A, Hincal F.
J Trace Elem Med Biol. 2015 Jan;29:296-302. doi: 10.1016/j.jtemb.2014.08.002. Epub 2014 Aug 19.

4. Influence of in utero di-n-hexyl phthalate and dicyclohexyl phthalate on fetal testicular development in **rats**.
Aydoğan Ahbab M, Barlas N.
Toxicol Lett. 2015 Mar 4;233(2):125-37. doi: 10.1016/j.toxlet.2015.01.015. Epub 2015 Jan 28.

5. Mixture effects of nonylphenol and di-n-butyl phthalate (monobutyl phthalate) on the tight junctions between Sertoli cells in male **rats** in vitro and **in vivo**.
Hu Y, Wang R, Xiang Z, Qian W, Han X, Li D.
Exp Toxicol Pathol. 2014 Dec;66(9-10):445-54. doi: 10.1016/j.etp.2014.07.003. Epub 2014 Sep 5.

6. In utero exposure to the **endocrine disruptor** di(2-ethylhexyl) phthalate targets ovarian theca cells and steroidogenesis in the adult female **rat**.
Meltzer D, Martinez-Arguelles DB, Campioli E, Lee S, Papadopoulos V.
Reprod Toxicol. 2014 Dec 18;51C:47-56. doi: 10.1016/j.reprotox.2014.12.005. [Epub ahead of print]

Perflourinated and Polyflourinated compounds

1. Possible role of serotonin and neuropeptide Y on the disruption of the reproductive axis activity by perfluorooctane sulfonate.
López-Doval S, Salgado R, Fernández-Pérez B, Lafuente A.
Toxicol Lett. 2015 Mar 4;233(2):138-47. doi: 10.1016/j.toxlet.2015.01.012. Epub 2015 Jan 23.

2. Developmental **toxicity** of **perfluorononanoic** acid in mice.
Das KP, Grey BE, Rosen MB, Wood CR, Tatum-Gibbs KR, Zehr RD, Strynar MJ, Lindstrom AB, Lau C.
Reprod Toxicol. 2014 Dec 25;51C:133-144. doi: 10.1016/j.reprotox.2014.12.012. [Epub ahead of print]

3. Effects of **perfluorooctane** sulfuric acid on placental PRL-family hormone production and fetal growth retardation in mice.
Lee CK, Kang SG, Lee JT, Lee SW, Kim JH, Kim DH, Son BC, Kim KH, Suh CH, Kim SY, Park YB.
Mol Cell Endocrinol. 2015 Feb 5;401:165-72. doi: 10.1016/j.mce.2014.10.026. Epub 2014 Nov 6.

4. Evaluation of the Chronic **Toxicity** and Carcinogenicity of **Perfluorohexanoic** Acid (PFHxA) in Sprague-Dawley Rats.
Klaunig JE, Shinohara M, Iwai H, Chengelis CP, Kirkpatrick JB, Wang Z, Bruner RH.
Toxicol Pathol. 2015 Feb;43(2):209-20. doi: 10.1177/0192623314530532. Epub 2014 May 28.

5. Analysis of apoptosis induced by **perfluorooctane** sulfonates (PFOS) in mouse Leydig cells in vitro.
Zhang DY, Xu XL, Shen XY, Ruan Q, Hu WL.
Toxicol Mech Methods. 2015 Jan;25(1):21-5. doi: 10.3109/15376516.2014.971140. Epub 2014 Oct 13.

Pesticides/Fungicides/Insecticides

1. The ameliorative effect of propolis against methoxychlor induced ovarian toxicity in **rat**.
El-Sharkawy EE, Kames AO, Sayed SM, Nisr NA, Wahba NM, Elsherif WM, Nafady AM, Abdel-Hafeez MM, Aamer AA.
Exp Toxicol Pathol. 2014 Dec;66(9-10):415-21. doi: 10.1016/j.etp.2014.06.003. Epub 2014 Jul 14.

2. Fetal and neonatal exposure to the **endocrine disruptor**, methoxychlor, reduces lean body mass and bone mineral density and increases cortical porosity.
Fagnant HS, Uzumcu M, Buckendahl P, Dunn MG, Shupper P, Shapses SA.
Calcif Tissue Int. 2014 Dec;95(6):521-9. doi: 10.1007/s00223-014-9916-x. Epub 2014 Oct 18.

3. Dietary exposure to the **endocrine disruptor** tolylfluanid promotes global metabolic dysfunction in male **mice**.
Regnier SM, Kirkley AG, Ye H, El-Hashani E, Zhang X, Neel BA, Kamau W, Thomas CC, Williams AK, Hayes ET, Massad NL, Johnson DN, Huang L, Zhang C, Sargis RM.
Endocrinology. 2015 Mar;156(3):896-910. doi: 10.1210/en.2014-1668. Epub 2014 Dec 23.

Various EDCs, Mixtures and Other endpoints

1. Differential Gene Expression Patterns in Developing Sexually Dimorphic Rat Brain Regions Exposed to Anti-androgenic, Estrogenic, or Complex **Endocrine Disruptor** Mixtures: Glutamatergic Synapses as Target.
Lichtensteiger W, Bassetti-Gaille C, Faass O, **Axelstad M, Boberg J, Christiansen S**, Rehrauer H, Georgijevic JK, **Hass U**, Kortenkamp A, Schlumpf M. Endocrinology. 2015 Jan 21:en20141504. [Epub ahead of print]

2. Metabolism of UV-filter benzophenone-3 by **rat** and human liver microsomes and its effect on **endocrine-disrupting** activity.
Watanabe Y, Kojima H, Takeuchi S, Uramaru N, Sanoh S, Sugihara K, Kitamura S, Ohta S.
Toxicol Appl Pharmacol. 2015 Jan 15;282(2):119-28. doi: 10.1016/j.taap.2014.12.002. Epub 2014 Dec 17

3. Estrogenic exposure alters the spermatogonial stem cells in the developing testis, permanently reducing crossover levels in the adult.
Vrooman LA, Oatley JM, Griswold JE, Hassold TJ, Hunt PA.
PLoS Genet. 2015 Jan 23;11(1):e1004949. doi: 10.1371/journal.pgen.1004949. eCollection 2015 Jan.

4. Epigenetic effects of low perinatal doses of flame retardant BDE-47 on mitochondrial and nuclear genes in **rat** offspring.
Byun HM, Benachour N, Zalko D, Frisardi MC, Colicino E, Takser L, Baccarelli AA.
Toxicology. 2015 Feb 3;328:152-9. doi: 10.1016/j.tox.2014.12.019. Epub 2014 Dec 19.

5. Development of Phenotypic and Transcriptional Biomarkers to Evaluate Relative Activity of Potentially Estrogenic Chemicals in Ovariectomized **Mice**.

Hewitt SC, Winuthayanon W, Pockette B, Kerns RT, Foley JF, Flagler N, Ney E, Suksamrarn A, Piyachaturawat P, Bushel PR, Korach KS.
Environ Health Perspect. 2015 Jan 9. [Epub ahead of print]

6. Oral exposure to low-dose of nonylphenol impairs memory performance in Sprague-Dawley **rats**.
Kawaguchi S, Kuwahara R, Kohara Y, Uchida Y, Oku Y, Yamashita K.
J Toxicol Sci. 2015;40(1):43-53.

7. Effect of maternal exposure to **endocrine disrupting** chemicals on reproduction and mammary gland development in female Sprague-Dawley **rats**.
Manservisi F, Gopalakrishnan K, Tibaldi E, Hysi A, Iezzi M, Lambertini L, Teitelbaum S, Chen J, Belpoggi F.
Reprod Toxicol. 2014 Dec 29. pii: S0890-6238(14)00324-4. doi: 10.1016/j.reprotox.2014.12.013. [Epub ahead of print]

8. Persistent organic pollutants and obesity: are they potential mechanisms for breast cancer promotion?
Reaves DK, Ginsburg E, Bang JJ, Fleming JM.
Endocr Relat Cancer. 2015 Apr;22(2):R69-R86. Epub 2015 Jan 26. Review.

9. Alterations in gene expression during sexual differentiation in **androgen** receptor knockout mice induced by environmental **endocrine disruptors**.
Liu D, Shen L, Tao Y, Kuang Y, Cai L, Wang D, He M, Tong X, Zhou S, Sun J, Shi C, Wang C, Wu Y.
Int J Mol Med. 2015 Feb;35(2):399-404. doi: 10.3892/ijmm.2014.2015. Epub 2014 Nov 28.

10. Polychlorinated Biphenyls (PCBs) Inhibit Hepcidin Expression through an Estrogen-Like Effect Associated with Disordered Systemic Iron Homeostasis.
Qian Y, Zhang S, Guo W, Ma J, Chen Y, Wang L, Zhao M, Liu S.
Chem Res Toxicol. 2015 Feb 24. [Epub ahead of print]

11. [Corrigendum] Alterations in gene expression during sexual differentiation in androgen receptor knockout **mice** induced by environmental **endocrine disruptors**.
Liu D, Shen L, Tao Y, Kuang Y, Cai L, Wang D, He M, Tong X, Zhou S, Sun J, Shi C, Wang C, Wu Y.
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van der Burg B, Wedebye EB, Dietrich DR, Jaworska J, Mangelsdorf I, Paune E, Schwarz M, Piersma AH, Kroese ED.
Reprod Toxicol. 2015 Feb 2. pii: S0890-6238(15)00009-X. doi: 10.1016/j.reprotox.2015.01.008. [Epub ahead of print]

13. **Endocrine Disruption** of Cadmium in **Rats** Using the OECD Enhanced TG 407 Test System.
Wang HJ, Liu ZP, Jia XD, Chen H, Tan YJ.
Biomed Environ Sci. 2014 Dec;27(12):950-9. doi: 10.3967/bes2014.135.

14. Dietary cholesterol affects expression of prostatic acid phosphatase in reproductive organs of male **rats**.
Lim W, Bae H, Sohn JY, Jeong W, Kim SH, Song G.
Biochem Biophys Res Commun. 2015 Jan 2;456(1):421-7. doi: 10.1016/j.bbrc.2014.11.100. Epub 2014 Dec 4.

15. Dietary early-life exposure to contaminated eels does not impair spatial cognitive performances in adult offspring **mice** as assessed in the Y-maze and the Morris water maze.
Dridi I, Leroy D, Guignard C, Scholl G, Bohn T, Landoulsi A, Thomé JP, Eppe G, Soulimani R, Bouayed J.
Nutr Res. 2014 Dec;34(12):1075-84. doi: 10.1016/j.nutres.2014.06.011. Epub 2014 Jun 26.

16. Dietary phytoestrogens present in soy dramatically increase cardiotoxicity in male **mice** receiving a chemotherapeutic tyrosine kinase inhibitor.
Harvey PA, Leinwand LA.
Mol Cell Endocrinol. 2015 Jan 5;399:330-5. doi: 10.1016/j.mce.2014.10.011. Epub 2014 Oct 30.

17. Exposures, mechanisms, and impacts of endocrine-active flame retardants.
Dishaw LV, J Macaulay L, Roberts SC, Stapleton HM.
Curr Opin Pharmacol. 2014 Dec;19:125-33. doi: 10.1016/j.coph.2014.09.018. Epub 2014 Oct 10

18. **Estrogenic exposure alters** the spermatogonial stem cells in the developing testis, permanently reducing crossover levels in the adult.
Vrooman LA, Oatley JM, Griswold JE, Hassold TJ, Hunt PA.
PLoS Genet. 2015 Jan 23;11(1):e1004949. doi: 10.1371/journal.pgen.1004949. eCollection 2015 Jan.

Burden & Cost of exposure (med links til artiklerne)

1. Neurobehavioral Deficits, Diseases and Associated Costs of Exposure to **Endocrine Disrupting** Chemicals in the European Union.
Bellanger M, Demeneix B, Grandjean P, Zoeller RT, Trasande L.
J Clin Endocrinol Metab. 2015 Mar 5:jc20144323. [Epub ahead of print]
http://press.endocrine.org/doi/abs/10.1210/jc.2014-4323

2. Estimating Burden and Disease Costs of Exposure to **Endocrine-Disrupting** Chemicals in the European Union.
Trasande L, Zoeller RT, Hass U, Kortenkamp A, Grandjean P, Myers JP, DiGangi J, Bellanger M, Hauser R, Legler J, Skakkebaek NE, Heindel JJ.
J Clin Endocrinol Metab. 2015 Mar 5:jc20144324. [Epub ahead of print]
http://press.endocrine.org/doi/abs/10.1210/jc.2014-4324

3. Male Reproductive Disorders, Diseases, and Costs of Exposure to **Endocrine-Disrupting**Chemicals in the European Union.
Hauser R, Skakkebaek NE, Hass U, Toppari J, Juul A, Andersson AM, Kortenkamp A, Heindel JJ, Trasande L.
J Clin Endocrinol Metab. 2015 Mar 5:jc20144325. [Epub ahead of print]
http://press.endocrine.org/doi/abs/10.1210/jc.2014-4325

4. Obesity, Diabetes, and Associated Costs of Exposure to **Endocrine-Disrupting** Chemicals in the European Union.
Legler J, Fletcher T, Govarts E, Porta M, Blumberg B, Heindel JJ, Trasande L.
J Clin Endocrinol Metab. 2015 Mar 5:jc20144326. [Epub ahead of print]
http://press.endocrine.org/doi/abs/10.1210/jc.2014-4326

Wildlife studier ved Biologisk Institut, Syddansk Universitet

Søgningen er udført på Web of Knowledge (all databases) og dækker perioden 1/12 2014 - 20/3 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 publikationer

Artikel 1: Climate change and pollution speed declines in zebrafish populations.
Brown, A. R.; Owen, S. F.; Peters, J.; Zhang, Y.; Soffker, M.; Paull, G. C.; Hosken, D. J.; Wahab, M. A. and Tyler, C. R. Proceedings of the National Academy of Sciences 112, 1237-1246.

Abstract: Endocrine disrupting chemicals (EDCs) are potent environmental contaminants, and their effects on wildlife populations could be exacerbated by climate change, especially in species with environmental sex determination. Endangered species may be particularly at risk because inbreeding depression and stochastic fluctuations in male and female numbers are often observed in the small populations that typify these taxa. Here, we assessed the interactive effects of water temperature and EDC exposure on sexual development and population viability of inbred and outbred zebrafish (Danio rerio). Water temperatures adopted were 28 °C (current ambient mean spawning temperature) and 33 °C (projected for the year 2100). The EDC selected was clotrimazole (at 2 μg/L and 10 μg/L), a widely used antifungal chemical that inhibits a key steroidogenic enzyme [cytochrome P450(CYP19) aromatase] required for estrogen synthesis in vertebrates. Elevated water temperature and clotrimazole exposure independently induced maleskewed sex ratios, and the effects of clotrimazole were greater at the higher temperature. Male sex ratio skews also occurred for the lower clotrimazole exposure concentration at the higher water temperature in inbred fish but not in outbred fish. Population viability analysis showed that population growth rates declined sharply in response to male skews and declines for inbred populations occurred at lower male skews than for outbred populations. These results indicate that elevated temperature associated with climate change can amplify the effects of EDCs and these effects are likely to be most acute in small, inbred populations exhibiting environmental sex determination and/or differentiation.

Artikel 2: Combined effects of silver nanoparticles and 17 alpha-ethinylestradiol on the freshwater mudsnail Potamopyrgus antipodarum.
Voelker, C.; Graef, T.; Schneider, I.; Oetken, M.; and Oehlmann, J. 2014. Environmental Science and Pollution Research 21, 10661-10670.

Abstract: Ecotoxicological studies have shown that nanosilver is among the most toxic nanomaterials to aquatic organisms. However, research has so far focused on the determination of acute effects. Combined effects of nanosilver with other substances have not yet been studied in aquatic organisms. The present study aimed to investigate the chronic toxicity of nanosilver as well as the potential of nanosilver to influence the effects of co-occurring substances on the freshwatermudsnail Potamopyrgus antipodarum. In 28-day chronic toxicity experiments, the effects of nanosilver on the reproduction of P. antipodarum were assessed. In order to evaluate the influence of nanosilver on other substances, 17α-ethinylestradiol (EE2) was chosen as model compound due to the well-characterized effects on P. antipodarum. In addition to effects on reproduction, exposure to nanosilver and EE2 was monitored by determining the expression of estrogen-responsive transcripts (estrogen receptor and vitellogenin encoding genes). Exposure to nanosilver decreased the reproduction of P. antipodarum (EC10: 5.57 μg l−1; EC50: 15.0 μg l−1). Exposure to EE2 significantly stimulated the embryo production at 25 ng l−1. The presence of nanosilver led to increased EE2 effects at EE2 concentrations that had no influence on reproduction when applied in absence of nanosilver. In contrast, combined exposure to nanosilver decreased

EE2 effects at concentrations that stimulated reproduction and the expression of estrogen responsive genes when applied in the absence of nanosilver. This is the first study demonstrating an influence of nanosilver on the effects of cocontaminants on aquatic organisms. The study further highlights the need for chronic experiments to properly assess environmental risks of nanosilver and their effects on co-occurring contaminants.

Artikel 3: Effects of the brominated flame retardant TBCO on fecundity and profiles of transcripts of the HPGL-axis in Japanese medaka.
Saunders, D. M. V.; Podaima, M.; Wiseman, S.; and Giesy, J. P. 2015. Aquatic toxicology (Amsterdam, Netherlands) 160, 180-187.

Abstract: The novel brominated flame retardant, 1,2,5,6-tetrabromocyclooctane (TBCO) is an additive flame retardant which is marketed under the trade name Saytex BCL-48. TBCO has recently been investigated as a potential alternative to the major use brominated flame retardant, hexabromocyclododecane (HBCD), which could have major implications for significant increases in amounts of TBCO used. Yet there is a lack of information regarding potential toxicities of TBCO. Recently, results of in vitro experiments have demonstrated the potential of TBCO to modulate endocrine function through interaction with estrogen and androgen receptors and via alterations to the synthesis of 17-β-estradiol and testosterone. Further research is required to determine potential endocrine disrupting effects of TBCOin vivo. In this experiment a 21-day fecundity assay with Japanese medaka (Oryzias latipes) was conducted to examine endocrine disrupting effects of TBCO in vivo. Medaka were fed a diet containing either 607 or 58μg TBCO/g food, wet mass (wm). Fecundity, measured as cumulative deposition of eggs and fertilization of eggs, as well as abundances of transcripts of 34 genes along the hypothalamus–pituitary–gonadal–liver (HPGL) axis were measured as indicators of holistic endocrine disruption and to determine mechanisms of effects, respectively. Cumulative fecundity was 18% lesser by medaka exposed to 58μg TBCO/g, wm food. However, fecundity of medaka exposed to 607μg TBCO/g,wm food was not significantly different from that of controls. Organ-specific and dose-dependent alterations to abundances of transcripts were observed in male and female medaka. A pattern of down-regulation of expression of genes involved in steroidogenesis, metabolism of cholesterol, and regulatory feedback mechanisms was observed in gonads from male and female medaka which had been exposed to the greater concentration of TBCO. However, these effects on expression of genes were not manifested in effects on fertilization of eggs or fecundity. In livers from male and female medaka exposed to the lesser concentration of TBCO greater expression of genes that respond to exposure to estrogens, including vitellogenin II, choriogenin H, and ERα, were observed. The results reported here confirm the endocrine disrupting potential of TBCO and elucidate potential mechanisms of effects which include specific patterns of alterations to abundances of transcripts of genes in the gonad and liver of medaka.

Bruttoliste

Alkylphenoler og Bisphenol A

Liver histology and ultrastructure of the Italian newt (Lissotriton italicus): Normal structure and modifications after acute exposure to nonylphenol ethoxylates.
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Park, C. J. and Gye, M. C. 2014. Aquatic Toxicology 156, 191-200.

Induction of vitellogenin synthesis in immature male yellowfin seabream (Acanthopagrus latus) exposed to 4-nonylphenol and 17beta-estradiol.
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Lee, C. C.; Jiang, L. Y.; Kuo, Y. L.; Chen, C. Y.; Hsieh, C. Y.; Hung, C. F.; and Tien, C. J. 2015. Science of the Total Environment 502, 417-425.

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Flammehæmmere

Effects of the brominated flame retardant TBCO on fecundity and profiles of transcripts of the HPGL-axis in Japanese medaka.
Saunders, D. M. V.; Podaima, M.; Wiseman, S.; and Giesy, J. P. 2015. Aquatic toxicology (Amsterdam, Netherlands) 160, 180-187.

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Climate change and pollution speed declines in zebrafish populations.
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