

# Center for Hormonforstyrrende Stoffer

Litteraturgennemgang for perioden 15. maj 2014 – 1. september 2014

## **Indhold**

Humane studier ved Afd. for Vækst og Reproduktion, Rigshospitalet.....	2
Udvalgte publikationer .....	3
Bruttoliste .....	7
<i>In vitro</i> studier ved DTU Fødevareinstituttet .....	33
Udvalgte publikationer .....	34
Bruttolisten .....	35
In Vivo studier ved DTU - FOOD .....	43
Udvalgte publikationer .....	44
Bruttolisten in vivo .....	45
Wildlife studier ved Biologisk Institut, Syddansk Universitet (SDU).....	55
Udvalgte artikler .....	56
Bruttoliste .....	59

## **Humane studier ved Afd. for Vækst og Reproduktion, Rigshospitalet**

Søgning er udført på PubMed og dækker perioden 15. maj 2014 – 1. september 2014

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 fremkom ved de øvrige søgningsord.

De udvalgte artikler spredte sig over kemikaliegrupperne phenoler, phthalater, bromerede flammehæmmere og PFASer. En enkelt artikel, der ikke undersøger hormonforstyrrende stoffer, men udelukkende ser på sammenhæng mellem anogenital distance og henholdsvis kryptorkisme og hypospadi er også medtaget, grundet dens relevans for begrebet testikulært dysgenese syndrom.

God læselyst!

## Udvalgte publikationer

Braun JM, Lanphear BP, Calafat AM, Deria S, Khoury J, Howe CJ, Venners SA.

**Early-Life Bisphenol A Exposure and Child Body Mass Index: A Prospective Cohort Study.**

Environ Health Perspect. 2014 Jul 29. [Epub ahead of print]

**BACKGROUND:** Early life bisphenol A (BPA) exposure may increase childhood obesity risk, but few prospective epidemiological studies have investigated this relationship. **OBJECTIVE:** To determine if early life BPA exposure was associated with increased body mass index (BMI) at 2-5 years of age in 297 mother-child pairs from Cincinnati, OH (HOME Study). **METHODS:** Urinary BPA concentrations were measured in samples collected from pregnant women during the 2nd and 3rd trimesters and their children at 1 and 2 years of age. BMI z-scores were calculated from weight/height measures conducted annually from 2-5 years of age. We used linear mixed models to estimate BMI differences or trajectories with increasing creatinine-normalized BPA concentrations. **RESULTS:** After confounder adjustment, each 10-fold increase in prenatal ( $\beta = -0.1$ , 95% confidence limit [CL]: -0.5, 0.3) or early childhood ( $\beta = -0.2$ , CL: -0.6, 0.1) BPA concentrations was associated with a modest and non-significant reduction in child BMI. These inverse associations were suggestively stronger in girls compared to boys (prenatal effect measure modification [EMM] p-value = 0.30, early childhood EMM p-value = 0.05), but sex-specific associations were imprecise. Children in the highest early childhood BPA tercile had lower BMI at 2 years (difference = -0.3; CL: -0.6, 0) and larger increases in their BMI slope from 2-5 years (BMI increase per year = 0.12; CL: 0.07, 0.18) than children in the lowest tercile (BMI increase per year = 0.07; CL: 0.01, 0.13). All associations were attenuated without creatinine-normalization. **CONCLUSIONS:** Prenatal and early childhood BPA exposures were not associated with increased BMI at 2-5 years, but higher early childhood BPA exposures were associated with accelerated growth during this period.

Philippat C, Botton J, Calafat AM, Ye X, Charles MA, Slama R; EDEN Study Group

**Prenatal exposure to phenols and growth in boys**

Epidemiology. 2014 Sep;25(5):625-35. doi: 10.1097/EDE.0000000000000132

**BACKGROUND:** Phenols interact with nuclear receptors implicated in growth and adipogenesis regulation. Only a few studies have explored their effects on growth in humans. **OBJECTIVES:** We studied the associations of maternal exposure to phenols during pregnancy with prenatal and postnatal growth of male newborns. **METHODS:** Within a cohort of women recruited during pregnancy, we selected 520 mother-son pairs and quantified 9 phenols in spot urine samples collected during pregnancy. We used ultrasonography during pregnancy, together with birth measurements, to assess fetal growth. We modeled individual postnatal growth trajectories from repeated measures of weight and height in the first 3 years of life. **RESULTS:** Triclosan concentration was negatively associated with growth parameters measured at the third ultrasound examination but not earlier in pregnancy. At birth, this phenol tended to be negatively associated with head circumference (-1.2 mm for an interquartile range [IQR] increase in ln-transformed triclosan concentration [95% confidence interval = -2.6 to 0.3]) but not with weight or height. Parabens were positively associated with weight at birth. This positive association remained for 3 years for methylparaben ( $\beta = 193$  g [-4 to 389]) for an IQR increase in ln-transformed concentrations. **CONCLUSION:** We relied on only 1 spot urine sample to assess exposure; because of the high variability in phenol urinary concentrations reported during pregnancy, using only 1 sample may result in exposure misclassification, in particular for bisphenol A. Our study suggested associations between prenatal exposure to parabens and triclosan and prenatal or early postnatal growth.

*Ferguson KK, Peterson KE, Lee JM, Mercado-García A, Blank-Goldenberg C, Téllez-Rojo MM, Meeker JD. Prenatal and peripubertal phthalates and bisphenol A in relation to sex hormones and puberty in boys. Reprod Toxicol. 2014 Aug;47:70-6. doi: 10.1016/j.reprotox.2014.06.002. Epub 2014 Jun 16.*

Phthalates and BPA are known endocrine disruptors and exposure in pregnant mothers and children is ubiquitous. We explored the relationship of prenatal and childhood exposures with pubertal onset and sex hormones in boys (ages 8-14). Phthalate metabolites and BPA were measured in maternal 3rd trimester or childhood urine. Sex hormones DHEAS, estradiol, inhibin B, SHBG, and total testosterone were measured in serum. Adrenarche and puberty were assessed by pediatrician. Prenatal exposure to some phthalates was associated with decreased DHEAS and inhibin B levels, and with increased SHBG. Prenatal exposure to most phthalates and BPA was associated with greatly reduced odds of adrenarche (odds ratios [OR]=0.12-0.65) and slightly reduced odds of puberty (OR=0.50-0.98). Childhood exposure was not associated with adrenarche or puberty, but some phthalates and BPA were associated with increased SHBG levels and decreased total and free testosterone levels.

*Meeker JD, Ferguson KK*

**Urinary Phthalate Metabolites Are Associated With Decreased Serum Testosterone in Men, Women, and Children From NHANES 2011-2012**

*J Clin Endocrinol Metab. 2014 Aug 14:jc20142555. [Epub ahead of print]*

**Context:** There is evidence of declining trends in T levels among men in recent decades, as well as trends in related conditions at multiple life stages and in both sexes. There is also animal and limited human evidence that exposure to phthalates, chemicals found in plastics and personal care products, is associated with reduced androgen levels and associated disorders. **Objective:** To explore relationships between urinary concentrations of 13 phthalate metabolites and serum total T levels among men, women, and children when adjusting for important confounders and stratifying by sex and age (6-12, 12-20, 20-40, 40-60, and 60-80 y). **Design:** A cross-sectional study. **Setting:** US National Health and Nutrition Examination Survey, 2011-2012. **Patients or Other Participants:** US general population. **Interventions:** None **Main Outcome Measures:** Serum total T measured by isotope dilution-liquid chromatography-tandem mass spectrometry. **Results:** Multiple phthalates were associated with significantly reduced T in both sexes and in differing age groups. In females, the strongest and most consistent inverse relationships were found among women ages 40-60 years. In boys 6-12 years old, an interquartile range increase in metabolites of di-2-ethylhexyl phthalate was associated with a 29% (95% confidence interval, 6, 47) reduction in T. In adult men, the only significant or suggestive inverse associations between phthalates (metabolites of di-2-ethylhexyl phthalate and dibutyl phthalate) and T were observed among men ages 40-60 years. **Conclusions:** Because T plays an important role in all life stages for both sexes, future efforts should focus on better defining these relationships and their broader impacts.

*Frederiksen H, Kuiru-Hänninen T, Main KM, Dunkel L, Sankilampi U.*

**A Longitudinal Study of Urinary Phthalate Excretion in 58 Full-Term and 67 Preterm Infants from Birth through 14 Months.**

*Environ Health Perspect. 2014 Sep;122(9):998-1005. doi: 10.1289/ehp.1307569. Epub 2014 May 29.*

**BACKGROUND:** Some phthalates have shown antiandrogenic effects in rat offspring. Premature infants may be exposed to high amounts of specific phthalates during hospitalization, and thus are potentially at risk.

**OBJECTIVE:** We evaluated longitudinal phthalate exposure and metabolism in full-term (FT) and preterm (PT) infants. **METHODS:** Fifty-eight FT and 67 PT (gestational age, 24.7-36.6 weeks) infants were recruited at

birth and followed until 14 months (nine times). Urinary concentrations of metabolites of diethyl phthalate (DEP), dibutyl phthalate isomers (DiBP and DnBP), butylbenzyl phthalate (BBzP), di(2-ethylhexyl) phthalate (DEHP), and diisobutyl phthalate (DiNP) were measured in 894 samples. Daily intake and a hazard index for antiandrogenic effects were estimated, and excretion patterns of DEHP and DiNP metabolites were analyzed. **RESULTS:** Metabolites of BBzP, DiNP, and DEHP were 5-50 times higher at day 7 (D7) and month 1 (M1) in PT than in FT infants. Thereafter, metabolite concentrations were similar between the two groups. The estimated hazard index for combined DiBP, DnBP, BBzP, and DEHP exposures 7 days after birth exceeded the antiandrogenic threshold in > 80% of PT and > 30% of FT infants, and after M2, in 30% of all infants. The excretion pattern of DEHP and DiNP metabolites changed with age. **CONCLUSION:** Most PT infants and approximately one-third of healthy FT newborns were exposed to phthalates during early life at a potentially harmful level according to the European Food Safety Authority's recommended limits of daily exposure. Changes in the relative proportions of secondary phthalate metabolites over time were consistent with maturation of infant metabolic pathways during the first year of life. Further research is needed on the health effects of phthalate exposures and the influence of changes in metabolic capacity in neonates and infants.

*Chen A, Yolton K, Rauch SA, Webster GM, Hornung R, Sjödin A, Dietrich KN, Lanphear BP.*  
**Prenatal Polybrominated Diphenyl Ether Exposures and Neurodevelopment in U.S. Children through 5 Years of Age: The HOME Study.**  
Environ Health Perspect. 2014 Aug;122(8):856-62. doi: 10.1289/ehp.1307562. Epub 2014 May 21.

**BACKGROUND:** Polybrominated diphenyl ethers (PBDEs) are persistent chemicals that have been widely used as flame retardants in furniture, carpet padding, car seats, and other consumer products during the past three decades. **OBJECTIVE:** We examined whether in utero exposure to PBDEs is associated with child cognitive function and behavior in a U.S. study sample. **METHODS:** In a prospective birth cohort, we measured maternal serum concentrations of BDE-47 and other PBDE congeners in 309 women at 16 weeks of gestation during 2003-2006 and followed their children in Cincinnati, Ohio. We measured cognitive and motor abilities using the Bayley Scales of Infant Development-II at ages 1, 2, and 3 years; intelligence using the Wechsler Preschool and Primary Scale of Intelligence-III at age 5 years; and children's behaviors using the Behavioral Assessment System for Children-2 annually at ages 2-5 years. We used linear mixed models or generalized estimating equations with adjustment for potential confounders to estimate associations between these outcomes and log<sub>10</sub>-transformed PBDE concentrations. **RESULTS:** The geometric mean of BDE-47 in maternal serum (20.1 ng/g lipid) was comparable with U.S. adult national reference values. Prenatal BDE-47 was not significantly associated with Bayley Mental or Psychomotor Development Indices at 1-3 years, but a 10-fold increase in prenatal BDE-47 was associated with a 4.5-point decrease (95% CI: -8.8, -0.1) in Full-Scale IQ and a 3.3-point increase (95% CI: 0.3, 6.3) in the hyperactivity score at age 5 years. **CONCLUSIONS:** Prenatal exposure to PBDEs was associated with lower IQ and higher hyperactivity scores in children.

*Scheringer M, Trier X, Cousins IT, de Voogt P, Fletcher T, Wang Z, Webster TF*  
**Helsingør Statement on poly- and perfluorinated alkyl substances (PFASs)**  
Chemosphere. 2014 Nov;114:337-9. doi: 10.1016/j.chemosphere.2014.05.044. Epub 2014 Jun 14.

In this discussion paper, the transition from long-chain poly- and perfluorinated alkyl substances (PFASs) to fluorinated alternatives is addressed. Long-chain PFASs include perfluoroalkyl carboxylic acids (PFCAs) with 7 or more perfluorinated carbons, perfluoroalkyl sulfonic acids (PFSAs) with 6 or more perfluorinated carbons, and their precursors. Because long-chain PFASs have been found to be persistent, bioaccumulative and toxic, they are being replaced by a wide range of fluorinated alternatives. We summarize key concerns

about the potential impacts of fluorinated alternatives on human health and the environment in order to provide concise information for different stakeholders and the public. These concerns include, amongst others, the likelihood of fluorinated alternatives or their transformation products becoming ubiquitously present in the global environment; the need for more information on uses, properties and effects of fluorinated alternatives; the formation of persistent terminal transformation products including PFCAs and PFSAs; increasing environmental and human exposure and potential of adverse effects as a consequence of the high ultimate persistence and increasing usage of fluorinated alternatives; the high societal costs that would be caused if the uses, environmental fate, and adverse effects of fluorinated alternatives had to be investigated by publicly funded research; and the lack of consideration of non-persistent alternatives to long-chain PFASs.

*Thankamony A, Lek N, Carroll D, Williams M, Dunger DB, Acerini CL, Ong KK, Hughes IA*

**Anogenital distance and penile length in infants with hypospadias or cryptorchidism: comparison with normative data**

*Environ Health Perspect. 2014 Feb;122(2):207-11. doi: 10.1289/ehp.1307178*

**BACKGROUND:** Anogenital distance (AGD) in animals is a sensitive biomarker of fetal endocrine disruption and the associated testicular dysgenesis syndrome (TDS). However, AGD in human infants with cryptorchidism and hypospadias, which are potential manifestations of TDS during childhood, is not clearly described. **OBJECTIVE:** Our aim was to compare AGD in boys with cryptorchidism or hypospadias against normative data. **METHODS:** Boys with isolated cryptorchidism ( $n = 71$ , age  $13.4 \pm 5.8$  months) or hypospadias ( $n = 81$ , age  $11.4 \pm 6.2$  months) were recruited from a tertiary center for measurement of AGD and penile length; they were compared with 487 healthy full-term boys from a birth cohort by deriving age-specific standard deviation scores (SDS). **RESULTS:** Boys with cryptorchidism were older ( $p = 0.048$ ) compared with boys with hypospadias. Boys with hypospadias had shorter mean AGD and penile length SDS than healthy boys (both  $p < 0.0001$ ). Mean AGD and penile length SDS values in boys with cryptorchidism were longer than mean values in boys with hypospadias (both  $p < 0.01$ ) and shorter than mean values in healthy boys (both  $p < 0.0001$ ). Mean penile length SDS decreased as the severity of hypospadias increased ( $ptrend = 0.078$ ). **CONCLUSIONS:** In the study population, AGD and penile length were reduced in boys with hypospadias or cryptorchidism relative to normative data derived from a longitudinal birth cohort. The findings support the use of AGD as a quantitative biomarker to examine the prenatal effects of exposure to endocrine disruptors on the development of the male reproductive tract.

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### **Tributyltin / Triclosan / Triclocarban**

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11: Pollock T, Tang B, deCatanzaro D. Triclosan exacerbates the presence of 14C-bisphenol A in tissues of female and male mice. *Toxicol Appl Pharmacol*. 2014 Jul 15;278(2):116-23. doi: 10.1016/j.taap.2014.04.017. Epub 2014 Apr 29. PubMed PMID: 24784443.

12: Lee HR, Hwang KA, Nam KH, Kim HC, Choi KC. Progression of breast cancer cells was enhanced by endocrine-disrupting chemicals, triclosan and octylphenol, via an estrogen receptor-dependent signaling pathway in cellular and mouse xenograft models. *Chem Res Toxicol*. 2014 May 19;27(5):834-42. doi: 10.1021/tx5000156. Epub 2014 Apr 8. PubMed PMID: 24684733.

13: Engel LS, Buckley JP, Yang G, Liao LM, Satagopan J, Calafat AM, Matthews CE, Cai Q, Ji BT, Cai H, Engel SM, Wolff MS, Rothman N, Zheng W, Xiang YB, Shu XO, Gao YT, Chow WH. Predictors and variability of repeat measurements of urinary phenols and parabens in a cohort of shanghai women and men. *Environ Health Perspect*. 2014 Jul;122(7):733-40. doi: 10.1289/ehp.1306830. Epub 2014 Mar 20. PubMed PMID: 24659570; PubMed Central PMCID: PMC4080538.

14: Liao C, Kannan K. A survey of alkylphenols, bisphenols, and triclosan in personal care products from China and the United States. *Arch Environ Contam Toxicol*. 2014 Jul;67(1):50-9. doi: 10.1007/s00244-014-0016-8. Epub 2014 Mar 18.

PubMed PMID: 24639116.

15: Kastbjerg VG, Hein-Kristensen L, Gram L. Triclosan-Induced Aminoglycoside-Tolerant *Listeria monocytogenes* Isolates Can Appear as Small-Colony Variants. *Antimicrob Agents Chemother*. 2014 Jun;58(6):3124-32. doi: 10.1128/AAC.02266-13. Epub 2014 Mar 17. PubMed PMID: 24637686; PubMed Central PMCID: PMC4068453.

16: Gorrochategui E, Casas J, Pérez-Albaladejo E, Jáuregui O, Porte C, Lacorte S. Characterization of complex lipid mixtures in contaminant exposed JEG-3 cells using liquid chromatography and high-resolution mass spectrometry. *Environ Sci Pollut Res Int*. 2014 Jun 28. [Epub ahead of print] PubMed PMID: 24969426.

17: Rantakokko P, Main KM, Wohlfart-Veje C, Kiviranta H, Airaksinen R, Vartiainen T, Skakkebæk NE, Toppari J, Virtanen HE. Association of placenta organotin concentrations with growth and ponderal index in 110 newborn boys from Finland during the first 18 months of life: a cohort study. *Environ Health*. 2014 Jun 5;13(1):45. doi: 10.1186/1476-069X-13-45. PubMed PMID: 24899383; PubMed Central PMCID: PMC4061538.

18: Pereira-Fernandes A, Vanparys C, Vergauwen L, Knapen D, Jorens PG, Blust R. Toxicogenomics in the 3T3-L1 Cell Line, a New Approach for Screening of Obesogenic Compounds. *Toxicol Sci*. 2014 Aug 1;140(2):352-63. doi: 10.1093/toxsci/kfu092. Epub 2014 May 20. PubMed PMID: 24848799.

19: Belcher SM, Cookman CJ, Patisaul HB, Stapleton HM. In vitro assessment of human nuclear hormone receptor activity and cytotoxicity of the flame retardant mixture FM 550 and its triarylphosphate and brominated components. *Toxicol Lett*. 2014 Jul 15;228(2):93-102. doi: 10.1016/j.toxlet.2014.04.017. Epub 2014 Apr 28. PubMed PMID: 24786373.

20: Celada LJ, Whalen MM. Effects of butyltins on mitogen-activated-protein kinase kinase kinase and Ras activity in human natural killer cells. *J Appl Toxicol*. 2014 Sep;34(9):1002-11. doi: 10.1002/jat.2921. Epub 2013 Sep 5. PubMed PMID: 24038145; PubMed Central PMCID: PMC3868639.

### **Endocrine disruptors**

No publications that are not already included in the above list

## **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\*”.

Publiseret i perioden 2014/04/30 to 2014/12/30 present (Maj 2014 og fremefter)

Efter at have fjernet genganger fra forrige litteraturopdateringslister gav litteratursøgningen, med de angivne søgekriterier, tilsammen en liste med i alt 79 artikler (Bruttolisten). Artiklerne er blevet fordelt i 5 grupper: ”Perflourinated and Polyflourinated compounds”, ”Plastic derivatives (BPA, Phthalates and others)”, ”Pesticides/Fungicides/Insecticides/Biocides”, ”Various EDCs, Mixtures and Other endpoints” og ”Various Nano-materials/compounds”.

## 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 et *in vitro* studie, hvor man har undersøgt bisphenol A (BPA) og BPA analogen bisphenol S (BPS), for at sammenligne deres effekt på endpoints relateret til fedme og udvikling af fedtlever. Den anden artikel handler om de to fluorerede stoffer PFOS og PFOA og deres effekt på interleukin 2 (IL-2) produktionen i humane T-cell i *vitro*.

### Is bisphenol S a safe substitute for bisphenol A in terms of metabolic function? An *in vitro* study.

Héliès-Toussaint C, Peyre L, Costanzo C, Chagnon MC, Rahmani R.

As bisphenol A (BPA) has been shown to induce adverse effects on human health, especially through the activation of endocrine pathways, it is about to be withdrawn from the European market and replaced by analogues such as bisphenol S (BPS). However, toxicological data on BPS is scarce, and so it is necessary to evaluate the possible effects of this compound on human health. We compared the effect of BPA and BPS on obesity and hepatic steatosis processes using low doses in the same range as those found in the environment. Two *in vitro* models were used, the adipose cell line 3T3-L1 and HepG2 cells, representative of hepatic functions. We analyzed different parameters such as lipid and glucose uptakes, lipolysis, leptin production and the modulation of genes involved in lipid metabolism and energy balance. BPA and BPS induced an increase in the lipid content in the 3T3-L1 cell line and more moderately in the hepatic cells. We also observed a decrease in lipolysis after bisphenol treatment of adipocytes, but only BPS was involved in the increase in glucose uptake and leptin production. These latter effects could be linked to the modulation of SREBP-1c, PPAR $\gamma$ , aP2 and ERR $\alpha$  and  $\gamma$  genes after exposure to BPA, whereas BPS seems to target the PGC1 $\alpha$  and the ERR $\gamma$  genes. The findings suggest that both BPA and BPS could be involved in obesity and steatosis processes, but through two different metabolic pathways.

### In vitro evaluation of the effects of perfluorooctanesulfonic acid (PFOS) and perfluorooctanoic acid (PFOA) on IL-2 production in human T-cells.

Midgett K, Peden-Adams MM, Gilkeson GS, Kamen DL.

Perfluorinated compounds, such as perfluorooctane sulfonate (PFOS) and perfluorooctanoic acid (PFOA), have been shown to alter various immune functions suggesting they are immunotoxic. This study assessed the effects of PFOS and PFOA on interleukin (IL)-2 production in the human Jurkat T-cell line and PFOS in healthy human primary T cells. Jurkat cells were stimulated with phytohemagglutinin (PHA)/phorbol myristate acetate (PMA), anti CD-3/anti CD-28, or anti CD-3, and dosed with 0, 0.05, 0.1, 0.5, 1, 5, 10, 50, 75, or 100  $\mu\text{g ml}^{-1}$  PFOS or 0, 0.005, 0.01, 0.05, 0.1, 0.5, 1, 5, or 10  $\mu\text{g ml}^{-1}$  PFOA. Jurkat cells stimulated with PHA/PMA or anti CD-3 exhibited decreased IL-2 production beginning at 50  $\mu\text{g PFOS ml}^{-1}$  and 5  $\mu\text{g PFOS ml}^{-1}$  respectively, but stimulation with anti-CD3/anti-CD28 resulted in no changes compared with the control. Addition of the PPAR-alpha antagonist GW6471 to PFOS-dosed cells stimulated with PHA/PMA resulted in decreases in IL-2 production starting at 50  $\mu\text{g PFOS ml}^{-1}$ , which suggests PFOS affected T-cell IL-2 production via PPAR-alpha-independent mechanisms. Exposure to PFOA, PFOA + GW6471, or PFOS + PFOA in Jurkat cells resulted in no significant differences in IL-2 production. *In vitro* dosing studies using healthy primary human CD4+ T cells were consistent with the Jurkat results. These data demonstrated that PFOA did not impact IL-2 production, but PFOS suppressed IL-2 production in both a human cell line and human primary cells at dose levels within the high end of the human exposure range. A decrease in IL-2 production is characteristic of autoimmune diseases such as systemic lupus erythematosus and should be further investigated.

## Bruttolisten

### Perflourinated and Polyflourinated compounds

1.

[Perfluoroalkyl acid contamination of follicular fluid and its consequence for \*\*in vitro\*\* oocyte developmental competence.](#)

Petro EM, D'Hollander W, Covaci A, Bervoets L, Fransen E, De Neubourg D, De Pauw I, Leroy JL, Jorssen EP, Bols PE.

Sci Total Environ. 2014 Aug 1;496C:282-288. doi: 10.1016/j.scitotenv.2014.07.028. [Epub ahead of print]

2.

[Proteomic analysis of mouse testis reveals perfluoroctanoic acid-induced reproductive dysfunction via direct disturbance of testicular steroidogenic machinery.](#)

Zhang H, Lu Y, Luo B, Yan S, Guo X, Dai J.

J Proteome Res. 2014 Jul 3;13(7):3370-85. doi: 10.1021/pr500228d. Epub 2014 Jun 25.

3.

[8-Hydroxy-2'-deoxyguanosine as a biomarker of oxidative DNA damage induced by perfluorinated compounds in TK6 cells.](#)

Yahia D, Haruka I, Kagashi Y, Tsuda S.

Environ Toxicol. 2014 Aug 12. doi: 10.1002/tox.22034. [Epub ahead of print]

PMID: 25113910

4.

[Complement activation is involved in the hepatic injury caused by high-dose exposure of mice to perfluoroctanoic acid.](#)

Botelho SC, Saghafian M, Pavlova S, Hassan M, DePierre JW, Abedi-Valugerdi M.

Chemosphere. 2014 Aug 6. pii: S0045-6535(14)00855-8. doi: 10.1016/j.chemosphere.2014.06.093. [Epub ahead of print]

5.

[Cellular target recognition of perfluoroalkyl acids: In vitro evaluation of inhibitory effects on lysine decarboxylase.](#)

Wang S, Lv Q, Yang Y, Guo LH, Wan B, Zhao L.

Sci Total Environ. 2014 Aug 2;496C:381-388. doi: 10.1016/j.scitotenv.2014.07.034. [Epub ahead of print]

6.

[Activation of sterol regulatory element-binding proteins in mice exposed to perfluoroctanoic acid for 28 days.](#)

Yan S, Wang J, Dai J.

Arch Toxicol. 2014 Aug 6. [Epub ahead of print]

7.

[In vitro evaluation of the effects of perfluorooctanesulfonic acid \(PFOS\) and perfluoroctanoic acid \(PFOA\) on IL-2 production in human T-cells.](#)

Midgett K, Peden-Adams MM, Gilkeson GS, Kamen DL.

J Appl Toxicol. 2014 Jul 23. doi: 10.1002/jat.3037. [Epub ahead of print]

8.

[Comparative hepatic \*\*in vitro\*\* depletion and metabolite formation of major perfluorooctane sulfonate precursors in arctic polar bear, beluga whale, and ringed seal.](#)

Letcher RJ, Chu S, McKinney MA, Tomy GT, Sonne C, Dietz R.  
Chemosphere. 2014 Oct;112:225-31. doi: 10.1016/j.chemosphere.2014.04.022. Epub 2014 May 14.

9. [\*\*6:2 Fluorotelomer iodide \*in vitro\* metabolism by rat liver microsomes: Comparison with \[1,2-\(14\)C\] 6:2 fluorotelomer alcohol.\*\*](#)

Ruan T, Sulecki LM, Wolstenholme BW, Jiang G, Wang N, Buck RC.  
Chemosphere. 2014 Oct;112:34-41. doi: 10.1016/j.chemosphere.2014.02.068. Epub 2014 Apr 21.

10. [\*\*High-intensity focused ultrasound sonothrombolysis: the use of perfluorocarbon droplets to achieve clot lysis at reduced acoustic power.\*\*](#)

Pajek D, Burgess A, Huang Y, Hynynen K.  
Ultrasound Med Biol. 2014 Sep;40(9):2151-61. doi: 10.1016/j.ultrasmedbio.2014.03.026. Epub 2014 Jul 9.

11. [\*\*Structure-dependent binding and activation of perfluorinated compounds on human peroxisome proliferator-activated receptor  \$\gamma\$\*\*](#)

Zhang L, Ren XM, Wan B, Guo LH.  
Toxicol Appl Pharmacol. 2014 Jul 3. pii: S0041-008X(14)00249-X. doi: 10.1016/j.taap.2014.06.020. [Epub ahead of print]

12. [\*\*Structure-activity relations in binding of perfluoroalkyl compounds to human thyroid hormone T3 receptor.\*\*](#)

Ren XM, Zhang YF, Guo LH, Qin ZF, Lv QY, Zhang LY.  
Arch Toxicol. 2014 May 13. [Epub ahead of print]

13. [\*\*In vitro metabolic formation of perfluoroalkyl sulfonamides from copolymer surfactants of pre- and post-2002 scotchgard fabric protector products.\*\*](#)

Chu S, Letcher RJ.  
Environ Sci Technol. 2014 Jun 3;48(11):6184-91. doi: 10.1021/es500169x. Epub 2014 May 13.

### **Plastic derivatives" (BPA, Phthalates and others)**

1. [\*\*Is bisphenol S a safe substitute for bisphenol A in terms of metabolic function? An \*in vitro\* study.\*\*](#)

Héliès-Toussaint C, Peyre L, Costanzo C, Chagnon MC, Rahmani R.  
Toxicol Appl Pharmacol. 2014 Aug 8. pii: S0041-008X(14)00291-9. doi: 10.1016/j.taap.2014.07.025. [Epub ahead of print]

2. [\*\*Histone Methyltransferase EZH2 Is Transcriptionally Induced by Estradiol as Well as Estrogenic Endocrine Disruptors Bisphenol-A and Diethylstilbestrol.\*\*](#)

Bhan A, Hussain I, Ansari KI, Bobzean SA, Perrotti LI, Mandal SS.  
J Mol Biol. 2014 Aug 1. pii: S0022-2836(14)00373-8. doi: 10.1016/j.jmb.2014.07.025. [Epub ahead of print]

3. [\*\*Identification of mechanisms of action of bisphenol A-induced human preadipocyte differentiation by transcriptional profiling.\*\*](#)

Boucher JG, Husain M, Rowan-Carroll A, Williams A, Yauk CL, Atlas E.  
Obesity (Silver Spring). 2014 Jul 22. doi: 10.1002/oby.20848. [Epub ahead of print]

4. [\*\*Estrogen and bisphenol A affect male rat enamel formation and promote ameloblast proliferation.\*\*](#)

Jedeon K, Loiodice S, Marciano C, Vinel A, Canivenc Lavier MC, Berdal A, Babajko S.  
Endocrinology. 2014 Jul 8:en20132161. [Epub ahead of print]

5. [\*\*Non-monotonic dose responses in studies of endocrine disrupting chemicals: bisphenol a as a case study.\*\*](#)

Vandenberg LN.

Dose Response. 2013 Oct 7;12(2):259-76. doi: 10.2203/dose-response.13-020. Vandenberg. eCollection 2014 May.

6. [The Adverse Cardiac Effects of Di\(2-ethylhexyl\)phthalate and Bisphenol A.](#)

Posnack NG.

Cardiovasc Toxicol. 2014 May 9. [Epub ahead of print]

7. [Phthalate exposure and childhood obesity.](#)

Kim SH, Park MJ.

Ann Pediatr Endocrinol Metab. 2014 Jun;19(2):69-75. doi: 10.6065/apem.2014.19.2.69. Epub 2014 Jun 30. Review.

8. [Endocrine disruptors and human reproductive failure: the in vitro effect of phthalates on human luteal cells.](#)

Romani F, Tropea A, Scarinci E, Federico A, Dello Russo C, Lisi L, Catino S, Lanzone A, Apa R.

Fertil Steril. 2014 Jul 10. pii: S0015-0282(14)00504-4. doi: 10.1016/j.fertnstert.2014.05.041. [Epub ahead of print]

9. [Phthalates efficiently bind to human peroxisome proliferator activated receptor and retinoid X receptor  \$\alpha\$ ,  \$\beta\$ ,  \$\gamma\$  subtypes: an in silico approach.](#)

Sarath Josh MK, Pradeep S, Vijayalekshmi Amma KS, Balachandran S, Abdul Jaleel UC, Doble M, Spener F, Benjamin S.

J Appl Toxicol. 2014 Jul;34(7):754-65. doi: 10.1002/jat.2902. Epub 2013 Jul 11.

10. [Benzyl butyl phthalate induces migration, invasion, and angiogenesis of Huh7 hepatocellular carcinoma cells through nongenomic AhR/G-protein signaling.](#)

Tsai CF, Hsieh TH, Lee JN, Hsu CY, Wang YC, Lai FJ, Kuo KK, Wu HL, Tsai EM, Kuo PL.

BMC Cancer. 2014 Aug 1;14:556. doi: 10.1186/1471-2407-14-556.

11. [Primary neuronal-astrocytic co-culture platform for neurotoxicity assessment of di-\(2-ethylhexyl\) phthalate.](#)

Wu Y, Li K, Zuo H, Yuan Y, Sun Y, Yang X.

J Environ Sci (China). 2014 May;26(5):1145-53. doi: 10.1016/S1001-0742(13)60504-5.

12. [Effect of sunlight exposure on the release of intentionally and/or non-intentionally added substances from polyethylene terephthalate \(PET\) bottles into water: chemical analysis and in vitro toxicity.](#)

Bach C, Dauchy X, Severin I, Munoz JF, Etienne S, Chagnon MC.

Food Chem. 2014 Nov 1;162:63-71. doi: 10.1016/j.foodchem.2014.04.020. Epub 2014 Apr 13.

## Pesticides/Fungicides/Insecticides/Biocides

1. [Disruption of thyroid hormone functions by low dose exposure of tributyltin: An in vitro and in vivo approach.](#)

Sharan S, Nikhil K, Roy P.

Gen Comp Endocrinol. 2014 Aug 4. pii: S0016-6480(14)00300-1. doi: 10.1016/j.ygcen.2014.07.027. [Epub ahead of print]

2. [Tributyltin distribution and producing androgenic activity in water, sediment, and fish muscle.](#)

Shue MF, Chen TC, Bellotindos LM, Lu MC.

J Environ Sci Health B. 2014 Jun 3;49(6):432-8. doi: 10.1080/03601234.2014.894780.

3. [Disruption of the hormonal network and the enantioselectivity of bifenthrin in trophoblast: maternal-fetal health risk of chiral pesticides.](#)

Zhao M, Zhang Y, Zhuang S, Zhang Q, Lu C, Liu W.  
Environ Sci Technol. 2014 Jul 15;48(14):8109-16. doi: 10.1021/es501903b. Epub 2014 Jul 2.

4. [Maternal cypermethrin exposure during the perinatal period impairs testicular development in C57BL male offspring.](#)

Huang C, Li X.  
PLoS One. 2014 May 8;9(5):e96781. doi: 10.1371/journal.pone.0096781. eCollection 2014.

5. [Evaluation of potential endocrine activity of 2,4-dichlorophenoxyacetic acid using \*in vitro\* assays.](#)

Coady KK, Kan HL, Schisler MR, Gollapudi BB, Neal B, Williams A, LeBaron MJ.  
Toxicol In Vitro. 2014 Aug;28(5):1018-25. doi: 10.1016/j.tiv.2014.04.006. Epub 2014 May 6.

### Various EDCs, Mixtures and Other endpoints

1. [The role of P450 metabolism in the estrogenic activity of bifenthrin in fish.](#)

DeGroot BC, Brander SM.  
Aquat Toxicol. 2014 Jul 18;156C:17-20. doi: 10.1016/j.aquatox.2014.07.007. [Epub ahead of print]

2. [Endocrine disrupting potential of fipronil and its metabolite in reporter gene assays.](#)

Lu M, Du J, Zhou P, Chen H, Lu C, Zhang Q.  
Chemosphere. 2014 Aug 8;120C:246-251. doi: 10.1016/j.chemosphere.2014.07.015. [Epub ahead of print]

3. [Bioavailability and fate of sediment-associated trenbolone and estradiol in aquatic systems.](#)

Sangster JL, Zhang Y, Hernandez R, Garcia YA, Sivils JC, Cox MB, Snow DD, Kolok AS, Bartelt-Hunt SL.  
Sci Total Environ. 2014 Aug 7;496C:576-584. doi: 10.1016/j.scitotenv.2014.07.040. [Epub ahead of print]

4. [Selectivity of natural, synthetic and environmental estrogens for zebrafish estrogen receptors.](#)

Pinto C, Grimaldi M, Boudahtouf A, Pakdel F, Brion F, Aït-Aïssa S, Cavallès V, Bourguet W, Gustafsson JA, Bondesson M, Balaguer P.  
Toxicol Appl Pharmacol. 2014 Aug 7. pii: S0041-008X(14)00280-4. doi: 10.1016/j.taap.2014.07.020. [Epub ahead of print]

5. [Effect of estrogenic binary mixtures in the yeast estrogen screen \(YES\).](#)

Ramirez T, Buechse A, Dammann M, Melching-Kollmuß S, Woitkowiak C, van Ravenzwaay B.  
Regul Toxicol Pharmacol. 2014 Jul 11;70(1):286-296. doi: 10.1016/j.yrtph.2014.07.006. [Epub ahead of print]

6. [Effects of the mycotoxin patulin at the level of nuclear receptor transcriptional activity and steroidogenesis \*in vitro\*.](#)

Frizzell C, Elliott CT, Connolly L.  
Toxicol Lett. 2014 Sep 2;229(2):366-73. doi: 10.1016/j.toxlet.2014.06.847. Epub 2014 Jul 1.

7. [An assessment of endocrine activity in Australian rivers using chemical and \*in vitro\* analyses.](#)

Scott PD, Bartkow M, Blockwell SJ, Coleman HM, Khan SJ, Lim R, McDonald JA, Nice H, Nugegoda D, Pettigrove V, Tremblay LA, Warne MS, Leusch FD.  
Environ Sci Pollut Res Int. 2014 Jul 2. [Epub ahead of print]

8. [Assessment of multiple hormonal activities in wastewater at different stages of treatment.](#)

Bain PA, Williams M, Kumar A.  
Environ Toxicol Chem. 2014 Jun 30. doi: 10.1002/etc.2676. [Epub ahead of print]

9. [Analytical methodology for the profiling and characterization of androgen receptor active compounds in human placenta.](#)

- Indiveri P, Horwood J, Abdul-Sada A, Arreola JP, Olea N, Hill EM. Reprod Toxicol. 2014 Aug;47:102-10. doi: 10.1016/j.reprotox.2014.06.004. Epub 2014 Jun 24.
10. [Predictive endocrine testing in the 21st century using \*in vitro\* assays of estrogen receptor signaling responses.](#)  
Rotroff DM, Martin MT, Dix DJ, Filer DL, Houck KA, Knudsen TB, Sipes NS, Reif DM, Xia M, Huang R, Judson RS.  
Environ Sci Technol. 2014 Aug 5;48(15):8706-16. doi: 10.1021/es502676e. Epub 2014 Jul 10.
11. [Reprint of "Current perspectives on the androgen 5 alpha-dihydrotestosterone \(DHT\) and 5 alpha-reductases in teleost fishes and amphibians"](#)  
Martyniuk CJ, Bissegger S, Langlois VS.  
Gen Comp Endocrinol. 2014 Jun 19. pii: S0016-6480(14)00244-5. doi: 10.1016/j.ygcen.2014.06.011. [Epub ahead of print]
12. [Coactivator recruitment of AhR/ARNT1.](#)  
Endler A, Chen L, Shibasaki F.  
Int J Mol Sci. 2014 Jun 19;15(6):11100-10. doi: 10.3390/ijms150611100.
13. [Occurrences, toxicities, and ecological risks of benzophenone-3, a common component of organic sunscreen products: a mini-review.](#)  
Kim S, Choi K.  
Environ Int. 2014 Sep;70:143-57. doi: 10.1016/j.envint.2014.05.015. Epub 2014 Jun 14. Review.
14. [Effect-directed analysis for estrogenic compounds in a fluvial sediment sample using transgenic cyp19a1b-GFP zebrafish embryos.](#)  
Fetter E, Krauss M, Brion F, Kah O, Scholz S, Brack W.  
Aquat Toxicol. 2014 Sep;154:221-9. doi: 10.1016/j.aquatox.2014.05.016. Epub 2014 May 19.
15. [Evaluation of BDE-47 hydroxylation metabolic pathways based on a strong electron-withdrawing pentafluorobenzoyl derivatization gas chromatography/electron capture negative ionization quadrupole mass spectrometry.](#)  
Zhai C, Peng S, Yang L, Wang Q.  
Environ Sci Technol. 2014 Jul 15;48(14):8117-26. doi: 10.1021/es405446y. Epub 2014 Jun 20.
16. [Are \*in vitro\* methods for the detection of endocrine potentials in the aquatic environment predictive for \*in vivo\* effects? Outcomes of the Projects SchussenAktiv and SchussenAktivplus in the Lake Constance Area, Germany.](#)  
Henneberg A, Bender K, Blaha L, Giebner S, Kuch B, Köhler HR, Maier D, Oehlmann J, Richter D, Scheurer M, Schulte-Oehlmann U, Sieratowicz A, Ziebart S, Triebskorn R.  
PLoS One. 2014 Jun 5;9(6):e98307. doi: 10.1371/journal.pone.0098307. eCollection 2014.
17. [Critical analysis of endocrine disruptive activity of triclosan and its relevance to human exposure through the use of personal care products.](#)  
Witorsch RJ.  
Crit Rev Toxicol. 2014 Jul;44(6):535-55. doi: 10.3109/10408444.2014.910754. Epub 2014 Jun 4.
18. [Estrogenic activity of constituents of underarm deodorants determined by E-Screen assay.](#)  
Lange C, Kuch B, Metzger JW.  
Chemosphere. 2014 Aug;108:101-6. doi: 10.1016/j.chemosphere.2014.02.082. Epub 2014 Apr 12.
19. [Integrated assessment of runoff from livestock farming operations: Analytical chemistry, \*in vitro\* bioassays, and \*in vivo\* fish exposures.](#)

- Cavallin JE, Durhan EJ, Evans N, Jensen KM, Kahl MD, Kolpin DW, Kolodziej EP, Foreman WT, LaLone CA, Makynen EA, Seidl SM, Thomas LM, Villeneuve DL, Weberg MA, Wilson VS, Ankley GT. Environ Toxicol Chem. 2014 Aug;33(8):1849-57. doi: 10.1002/etc.2627. Epub 2014 Jun 27.
20. [Hypothesis-driven weight of evidence analysis to determine potential endocrine activity of MTBE](#).  
de Peyster A, Mihaich E.  
Regul Toxicol Pharmacol. 2014 Aug;69(3):348-70. doi: 10.1016/j.yrtph.2014.04.017. Epub 2014 May 6.
21. [Early Life Triclocarban Exposure During Lactation Affects Neonate Rat Survival](#).  
Kennedy RC, Menn FM, Healy L, Fecteau KA, Hu P, Bae J, Gee NA, Lasley BL, Zhao L, Chen J.  
Reprod Sci. 2014 May 6. [Epub ahead of print]
22. [Co-occurrence of estrogenic and antiestrogenic activities in wastewater: quantitative evaluation of balance by \*in vitro\* ER \$\alpha\$  reporter gene assay and chemical analysis](#).  
Ihara M, Ihara MO, Kumar V, Narumiya M, Hanamoto S, Nakada N, Yamashita N, Miyagawa S, Iguchi T, Tanaka H.  
Environ Sci Technol. 2014 Jun 3;48(11):6366-73. doi: 10.1021/es5014938. Epub 2014 May 13.
23. [Assessment of the sensitivity of three North American fish species to disruptors of steroidogenesis using \*in vitro\* tissue explants](#).  
Beitel SC, Doering JA, Patterson SE, Hecker M.  
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## In Vivo studier ved DTU - FOOD

### **Søgning er udført på PubMed og dækker perioden maj – sept 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 ((perfluor\* OR polyfluor\*)AND in vivo).

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 90 artikler (Bruttolisten)

Disse er efter Miljøstyrelsens ønske blevet fordelt i grupper efter stofnavne: "Parabens, "Plastic derivatives" (BPA, Phthalates and others), "Pesticides/fungicides" og " Various EDCs, Mixtures and Other endpoints".

## **Udvalgte publikationer**

To artikler er blevet udvalgt til nærmere beskrivelse (abstrakt og konklusion). Disse 2 artikler er valgt fordi vi mener de bidrager til ny viden om OECD test guideline studier (screeningsstudier) (Beekhuijzen et al. 2014). Den anden artikel omhandler et studie fra Michael Skinners gruppe om transgenerationelle effekter hos tredje generation efter vinclozolin eksponering og stress (Gillette et al 2014).

Ud fra bruttolisten (se længere nede i dokumentet) er udvalgt følgende 2 artikler til engelsk abstrakt.

### The underestimated value of OECD 421 and 422 repro screening studies: Putting it in the right perspective.

Beekhuijzen M, de Raaf MA, Zmarowski A, van Otterdijk F, Peter B, Emmen H.  
Reprod Toxicol. 2014 Sep;48:81-7. doi: 10.1016/j.reprotox.2014.04.003. Epub 2014 Apr 21.

#### **Abstract**

To assess the efficacy of reproduction/developmental screening studies (OECD 421 and 422), a retrospective evaluation of 134 studies was performed. The major findings were: (1) for up to half of the studies with developmental and reproductive toxicity, these effects would have been missed in other types of studies, which underscores that reproduction/developmental screening studies should not be waived by default based on negative 28-day and/or prenatal developmental data, (2) the required number of animals as stated in the guidelines, is appropriate for detecting developmental and reproductive toxicity, and (3) adding measurements like anogenital distance, internal sex determination and nipple retention, plus extending the postnatal period would add predictive value. Overall, the current reproduction/developmental screening studies are effective in providing unique data, especially considering the limited number of animals used. Some simple additions would enrich its value in risk assessment even further.

### Sexually dimorphic effects of ancestral exposure to vinclozolin on stress reactivity in rats.

Gillette R, Miller-Crews I, Nilsson EE, Skinner MK, Gore AC, Crews D.  
Endocrinology. 2014 Jul 22:en20141253. [Epub ahead of print] **Valgt**

#### **Abstract**

How an individual responds to the environment depends upon both personal life history as well as inherited genetic and epigenetic factors from ancestors. Using a ‘two-hit, 3 generations apart’ model, we tested how F3 descendants of rats given in utero exposures to the environmental endocrine-disrupting chemical (EDC) vinclozolin reacted to stress during adolescence in their own lives, focusing on sexually dimorphic phenotypic outcomes. In adulthood, male and female F3 vinclozolin- or vehicle-lineage rats, stressed or non-stressed, were behaviorally characterized on a battery of tests, then euthanized. Serum was used for hormone assays, and brains for qPCR and transcriptome analyses. Results showed that the effects of ancestral exposure to vinclozolin converged with stress experienced during adolescence in a sexually dimorphic manner. Debilitating effects were seen at all levels of the phenotype, including physiology, behavior, brain metabolism, gene expression, and genome-wide transcriptome modifications in specific brain nuclei. Additionally, females were significantly more vulnerable than males to transgenerational effects of vinclozolin on anxiety but not sociality tests. This fundamental transformation occurs in a manner neither predicted by the ancestral exposure or the proximate effects of stress during adolescence, an interaction we refer to as synchronicity.

## **Bruttolisten in vivo**

### **Plastic derivatives (BPA, Phthalates and others)**

#### **BPA**

##### **1. Sex differences in the adult HPA axis and affective behaviors are altered by perinatal exposure to a low dose of bisphenol A.**

Chen F, Zhou L, Bai Y, Zhou R, Chen L.

Brain Res. 2014 Jul 7;1571:12-24. doi: 10.1016/j.brainres.2014.05.010. Epub 2014 May 21.

##### **2. Investigation of the effects of subchronic low dose oral exposure to bisphenol A (BPA) and ethinyl estradiol (EE) on estrogen receptor expression in the juvenile and adult female rat hypothalamus.**

Rebuli ME, Cao J, Sluzas E, Delclos KB, Camacho L, Lewis SM, Vanlandingham MM, Patisaul HB. Toxicol Sci. 2014 Jul;140(1):190-203. doi: 10.1093/toxsci/kfu074. Epub 2014 Apr 20.

##### **3. Programming of metabolic effects in C57BL/6JxFVB mice by exposure to bisphenol A during gestation and lactation.**

van Esterik JC, Dollé ME, Lamoree MH, van Leeuwen SP, Hamers T, Legler J, van der Ven LT. Toxicology. 2014 Jul 3;321:40-52. doi: 10.1016/j.tox.2014.04.001. Epub 2014 Apr 13.

##### **4. Concentrations of phthalates and bisphenol A in Norwegian foods and beverages and estimated dietary exposure in adults.**

Sakhi AK, Lillegaard IT, Voorspoels S, Carlsen MH, Løken EB, Brantsæter AL, Haugen M, Meltzer HM, Thomsen C.

Environ Int. 2014 Aug 27;73C:259-269. doi: 10.1016/j.envint.2014.08.005. [Epub ahead of print]

##### **5. Models of science-policy interaction: exploring approaches to Bisphenol A management in the EU.**

Udovyk O.

Sci Total Environ. 2014 Jul 1;485-486:23-30. doi: 10.1016/j.scitotenv.2014.03.046. Epub 2014 Apr 1.

##### **6. Bisphenol A, obesity, and type 2 diabetes mellitus: genuine concern or unnecessary preoccupation?**

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Transl Res. 2014 Jul;164(1):13-21. doi: 10.1016/j.trsl.2014.03.003. Epub 2014 Mar 13. Review.

##### **7. Bisphenol-A and diethylstilbestrol exposure induces the expression of breast cancer associated long noncoding RNA HOTAIR in vitro and in vivo.**

Bhan A, Hussain I, Ansari KI, Bobzean SA, Perrotti LI, Mandal SS.

J Steroid Biochem Mol Biol. 2014 May;141:160-70. doi: 10.1016/j.jsbmb.2014.02.002. Epub 2014 Feb 14.

##### **8. Effects of bisphenol A and 4-nonylphenol on cellular responses through the different induction of LPA receptors in liver epithelial WB-F344 cells.**

Dong Y, Araki M, Hirane M, Tanabe E, Fukushima N, Tsujiuchi T.

J Recept Signal Transduct Res. 2014 Jun;34(3):201-4. doi: 10.3109/10799893.2013.876040. Epub 2014 Jan 24.

9. [Adverse effects of long-term exposure to bisphenol A during adulthood leading to hyperglycemia and hypercholesterolemia in mice.](#)

Marmugi A, Lasserre F, Beuzelin D, Ducheix S, Huc L, Polizzi A, Chetivaux M, Pineau T, Martin P, Guillou H, Mselli-Lakhal L.

Toxicology. 2014 Aug 25. pii: S0300-483X(14)00165-6. doi: 10.1016/j.tox.2014.08.006. [Epub ahead of print]

10. [Bisphenol A, oocyte maturation, implantation, and IVF outcome: review of animal and human data.](#)

Machtinger R, Orvieto R.

Reprod Biomed Online. 2014 Jul 10. pii: S1472-6483(14)00362-9. doi: 10.1016/j.rbmo.2014.06.013. [Epub ahead of print] Review.

11. [Perinatal exposure to bisphenol A exacerbates nonalcoholic steatohepatitis-like phenotype in male rat offspring fed on a high-fat diet.](#)

Wei J, Sun X, Chen Y, Li Y, Song L, Zhou Z, Xu B, Lin Y, Xu S.

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13. [Food intolerance at adulthood after perinatal exposure to the endocrine disruptor bisphenol A.](#)

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Endocrinology. 2014 Sep;155(9):3365-75. doi: 10.1210/en.2013-2161. Epub 2014 Jul 8.

15. [Sex-specific effects of long-term exposure to bisphenol-A on anxiety- and depression-like behaviors in adult mice.](#)

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18. [Mitochondrial dysfunction in early life resulted from perinatal bisphenol A exposure contributes to hepatic steatosis in rat offspring.](#)

Jiang Y, Xia W, Zhu Y, Li X, Wang D, Liu J, Chang H, Li G, Xu B, Chen X, Li Y, Xu S.

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## Phthalates and others

1. "Everybody's plastic": So what?

Erren TC, Meyer-Rochow VB, Groß JV.

Reprod Toxicol. 2014 Aug 9. pii: S0890-6238(14)00200-7. doi: 10.1016/j.reprotox.2014.07.079. [Epub ahead of print] No abstract available.

**2. In utero and peripubertal exposure to phthalates and BPA in relation to female sexual maturation.**  
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**7. Disruption of Rat Testis Development Following Combined In Utero Exposure to the Phytoestrogen Genistein and Anti-Androgenic Plasticizer Di-(2-Ethylhexyl) Phthalate.**  
Jones S, Boisvert A, Duong TB, Francois S, Thrane P, Culty M.  
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**8. Assessment of estrogenic potential of diethyl phthalate in female reproductive system involving both genomic and non-genomic actions.**  
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**9. In utero exposure to the endocrine disruptor di-(2-ethylhexyl) phthalate induces long-term changes in gene expression in the adult male adrenal gland.**  
Martinez-Arguelles DB, Campioli E, Lienhart C, Fan J, Culty M, Zirkin BR, Papadopoulos V.  
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### **Perflourinated and Polyflourinated compounds**

**1. Proteomic analysis of mouse testis reveals perfluoroctanoic acid-induced reproductive dysfunction via direct disturbance of testicular steroidogenic machinery.**  
Zhang H, Lu Y, Luo B, Yan S, Guo X, Dai J.  
J Proteome Res. 2014 Jul 3;13(7):3370-85. doi: 10.1021/pr500228d. Epub 2014 Jun 25.

## **Pesticides/Fungicides/Insecticides**

1. Maternal cypermethrin exposure during the perinatal period impairs testicular development in C57BL male offspring.

Huang C, Li X.

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2. The ameliorative effect of propolis against methoxychlor induced ovarian toxicity in rat.

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Exp Toxicol Pathol. 2014 Jul 14. pii: S0940-2993(14)00088-8. doi: 10.1016/j.etp.2014.06.003. [Epub ahead of print]

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4. Perinatal exposure to methoxychlor enhances adult cognitive responses and hippocampal neurogenesis in mice.

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

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Birth Defects Res B Dev Reprod Toxicol. 2014 Aug;101(4):308-24. doi: 10.1002/bdrb.21114. Epub 2014 Jul 30.

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## Wildlife studier ved Biologisk Institut, Syddansk Universitet (SDU)

Søgningen er udført på Web of Knowledge (all databases) og dækker perioden 28/4 – 15/8 2014.

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 fire artikler til medtagelse af abstract og yderligere kommentarer. Artikel 3 og 4 omhandler begge anvendelsen af ALP som biomarkør for hormonforstyrrende stoffer i muslinger og kommenteres samlet.

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:** Are In Vitro Methods for the Detection of Endocrine Potentials in the Aquatic Environment Predictive for In Vivo Effects? Outcomes of the Projects SchussenAktiv and SchussenAktiv plus in the Lake Constance Area, Germany. Henneberg, A.; Bender, K.; Blaha, L.; Giebner, S.; Kuch, B.; Koehler, H. R.; Maier, D.; Oehlmann, J.; Richter, D.; Scheurer, M.; Schulte-Oehlmann, U.; Sieratowicz, A.; Ziebart, S.; and Triebskorn, R. 2014. Plos One 9.

Abstract: Many studies about endocrine pollution in the aquatic environment reveal changes in the reproduction system of biota. We analysed endocrine activities in two rivers in Southern Germany using three approaches: (1) chemical analyses, (2) **in vitro** bioassays, and (3) **in vivo** investigations in fish and snails. Chemical analyses were based on gas chromatography coupled with mass spectrometry. For **in vitro** analyses of endocrine potentials in water, sediment, and waste water samples, we used the E-screen assay (human breast cancer cells MCF-7) and reporter gene assays (human cell line HeLa-9903 and MDA-kb2). In addition, we performed reproduction tests with the freshwater mudsnail **Potamopyrgus antipodarum** to analyse water and sediment samples. We exposed juvenile brown trout (**Salmo trutta f. fario**) to water downstream of a wastewater outfall (Schussen River) or to water from a reference site (Argen River) to investigate the vitellogenin production. Furthermore, two feral fish species, chub (**Leuciscus cephalus**) and spirlin (**Alburnoides bipunctatus**), were caught in both rivers to determine their gonadal maturity and the gonadosomatic index. Chemical analyses provided only little information about endocrine active substances, whereas the **in vitro** assays revealed endocrine potentials in most of the samples. In addition to endocrine potentials, we also observed toxic potentials (E-screen/reproduction test) in waste water samples, which could interfere with and camouflage endocrine effects. The results of our **in vivo** tests were mostly in line with the results of the **in vitro** assays and revealed a consistent reproduction-disrupting (reproduction tests) and an occasional endocrine action (vitellogenin levels) in both investigated rivers, with more pronounced effects for the Schussen river (e.g. a lower gonadosomatic index). We were able to show that biological **in vitro** assays for endocrine potentials in natural stream water reasonably reflect reproduction and endocrine disruption observed in snails and field-exposed fish, respectively.

**Artikel 2:** Population relevance of toxicant mediated changes in sex ratio in fish: An assessment using an individual-based zebrafish (*Danio rerio*) model. Hazlerigg, C. R.; Tyler, C. R.; Lorenzen, K.; Wheeler, J. R.; and Thorbek, P. 2014. Ecological Modelling 280, 76-88.

Abstract: Ecological risk assessments (ERAs) of toxicants are predominantly based on data from laboratory tests on individuals. However, the protection goal is generally at the population level. Ecological modelling has the potential to link individual-level effects to population-level outcomes. Here we developed an individual-based zebrafish population model to study the possible population-level relevance of toxicant-mediated changes in sex ratio. The model was structured with sub-models based on empirical data (e.g. growth, reproduction, mortality) derived from a combination of our own laboratory and field experiments, the literature and theoretical concepts. The outputs of the default model were validated against size distributions for wild populations of zebrafish sampled in Bangladesh. Sensitivity analysis showed that population abundance was most sensitive to changes in density-dependent survival and the availability of refugia for juveniles. The model was then used to determine the population-level relevance of changes in sex ratio caused by

an androgenic (dihydrotestosterone) and oestrogenic (4-tert-octylphenol) substance. Both were investigated under acute (10 day) and chronic (1 year) exposure regimes. Acute exposures to the test chemicals had little effect on population-level endpoints at any of the concentrations tested. Chronic exposures decreased population abundance at higher concentrations for both chemicals and most strongly with DHT. However, these concentrations were far in excess of environmentally realistic levels. Our study demonstrated that ecological models can be applied to link laboratory derived ecotoxicity data at the individual level to impacts at the population level and in our study we found different modes of action and potencies caused different levels of population perturbation. Ecological models can therefore help in assessing the ecological relevance of different organism-level effects of toxicants aiding future environmental protection strategies.

**Artikel 3:** Evaluation of yolk protein levels as estrogenic biomarker I bivalves; comparison of the alkali-labile phosphate method (ALP) and a species-specific immunoassay (ELISA). Morthorst, J.; Holbech, H.; Jeppesen, M.; Kinnberg, K. L.; Pedersen, K. L. and Bjerregaard, P. 2014. Comparative Biochemistry and Physiology C - Toxicology & Pharmacology 166, 88-95.

Abstract: Altered concentration of the vertebrate yolk protein precursor vitellogenin is a recognized biomarker for endocrine disruption in fish, and within recent years yolk protein alteration has also been associated with endocrine disruption in bivalves. Species-specific, direct and sensitive methods for quantification of vitellogenin in fish have been available for years whereas bivalve yolk protein levels have been estimated indirectly by alkali-labile phosphate (ALP) liberated from high molecular weight proteins because the sequence and biochemical structure of most bivalve yolk proteins are unknown. By applying a species-specific enzyme-linked immunosorbent assay (ELISA) for accurate determination of yolk protein level the impact of 17 $\beta$ -estradiol (57, 164 and 512 ng/L) on the freshwater bivalve *Unio tumidus* was investigated and compared with ALP estimations. Seven weeks of exposure during the pre-spawning and spawning period had no consistent effect on yolk protein concentration in hemolymph, and ALP levels in hemolymph also remained unchanged in both males and females. Further, basal male and female ALP levels were indistinguishable whereas the ELISA demonstrated that yolk protein levels of females exceeded male levels at the time of sampling, although male basal levels were high compared to fish. Altogether it is shown that individual ALP levels do not reflect yolk protein levels and hence hemolymph ALP levels cannot serve as biomarker for estrogenic exposure during the pre-spawning and spawning period in *U. tumidus*. The necessity of sensitive and validated biomarkers for reliable interpretation of data and the utility of ALP and yolk protein levels as biomarkers in bivalves are discussed.

**Artikel 4:** Biochemical and proteomic characterization of haemolymph serum reveals the origin of the alkali-labile phosphate (ALP) in mussel (*Mytilus galloprovincialis*). Oliveri, C.; Peric, L.; Sforzini, S.; Banni, M.; Viarengo, A.; Cavaletto, M. and Marsano, F. 2014. Comparative Biochemistry and Physiology D 11, 29-36.

Abstract: Mollusc haemolymph proteins are known to play several important physiological roles in the immune system, heavy metal transport and the tissue distribution of lipophilic compounds. In this study, we analysed acetone-extracted proteins from mussel haemolymph by one- and two-dimensional gel electrophoresis. The proteins were identified by comparing mass spectrometry data with the invertebrate EST database, allowing us to establish the mussel haemolymph serum proteome. Extrapallial protein (EP) precursor represents the most abundant serum protein;

astacin and CuZn superoxide dismutase were also detected. Slight contamination from muscle proteins, due to the sampling method, was also found. No differences were observed in the profiles obtained for male and female serum proteins. One aspect of interest was the previously reported finding that alkali-labile phosphate (ALP) from haemolymph serum may be representative of vitellogenin (vtg)-like protein content in the circulatory fluid of molluscs. In our analysis of mussel haemolymph serum, vitellogenin-like proteins were never found. To confirm these data, a typical methyl-tert-butyl-ether (MTBE) extraction, which is specific for vtg-like proteins, was performed, and the results of the electrophoretic analyses were compared with those obtained by acetic precipitation. The results showed that the electrophoretic profiles are similar and that vtg-like proteins cannot be identified. Moreover, the main phosphoprotein present in female and male extracts is EP protein precursor. In addition, agarose gel electrophoresis demonstrates that high-molecular-weight forms of vtg-like proteins are not detectable.

## **Bruttoliste**

### **Alkylphenoler**

Acute 4-nonylphenol toxicity changes the genomic expression profile of marine medaka fish, *Oryzias javanicus*.

Won, H.; Woo, S.; and Yum, S. 2014. Molecular & Cellular Toxicology 10, 181-195.

### **Bisphenol A**

Bisphenol A affects larval growth and advances the onset of metamorphosis in *Drosophila melanogaster*.

Weiner, A.; Ramirez, A.; Zintel, T.; Rose, R.; Wolff, E.; Parker, A.; Bennett, K.; Johndreau, K.; Rachfalski, C.; Zhou, J.; and Smith, S. 2014. Ecotoxicology and Environmental Safety 101, 7-13.

### **Phthalater**

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Phthalate ester leachates in aquatic mesocosms: Implications for ecotoxicity studies of endocrine disrupting compounds.

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### **UV-filtre**

The UV filter benzophenone 3 (BP-3) activates hormonal genes mimicking the action of ecdysone and alters embryo development in the insect *Chironomus riparius* (Diptera).

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### **Flammehæmmere**

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### **Pesticider**

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Evaluation of Organochlorine Pesticides and Sex Steroids in Lower Niagara River Lake Sturgeon. Jacobs, G. R.; Gundersen, D. T.; Webb, M. A.; Gorsky, D.; Kohl, K.; and Lockwood, K. 2014. Journal of Fish and Wildlife Management 5, 109-117.

Effects of Atrazine on Endocrinology and Physiology in Juvenile Barramundi, *Lates Calcarifer* (Bloch).

Kroon, F. J.; Hook, S. E.; Jones, D.; Metcalfe, S.; and Osborn, H. L. 2014. Environmental Toxicology and Chemistry 33, 1607-1614.

Two-dimensional proteomic analysis of gonads of air-breathing catfish, *Clarias batrachus* after the exposure of endosulfan and malathion.

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Lopez-Doval, J. C.; Poquet, M.; and Munoz, I. 2014. Limnetica 33, 205-215.

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Ibrahim, A. T. and Harabawy, A. S. 2014. Ecotoxicology and Environmental Safety 106, 33-39.

Agrichemicals chronically inhibit the cortisol response to stress in fish.

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