Center for Hormonforstyrrende Stoffer

Litteraturgennemgang for perioden 1. april – 30. juni 2012

Indhold

Humane studier ved Afd. for Vækst og Reproduktion, Rigshospitalet

Søgning er udført på PubMed og dækker perioden 1. april – 30. juni 2012

Følgende søgeprofil er benyttet:

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

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

Som det fremgår af bruttolisten for humane studier, er der ganske mange hits i denne omgang. Derfor vil der også være relevante artikler, der ikke er blevet kommenteret. De udvalgte artikler omhandler BPA, PFOA, phthalater, parabener og paracetamol. God læselyst.

Udvalgte publikationer

Occup Environ Med. 2012 May 31. [Epub ahead of print]
Perfluorooctanoic acid exposure is associated with elevated homocysteine and hypertension in US adults
Min JY, Lee KJ, Park JB, Min KB.
Institute of Health and Environment, Seoul National University, Seoul, Republic of Korea.

Objective: To investigate the association between serum perfluorooctanoic acid (PFOA) concentration and cardiovascular disease, as measured by homocysteine level and blood pressure in a representative sample of US adults.
Methods: A cross-sectional study of 2934 adults (≥20 years) who participated in the 2003-2004 and 2005-2006 National Health and Nutrition Examination Survey and had detectable levels of PFOA in their serum. The health effects analysed as potentially associated with PFOA exposure included homocysteine level and blood pressure.
Results: The geometric mean value (95% CI) of the study participants' serum PFOA concentration was 4.00 μg/l (95% CI 3.86 to 4.13). The homocysteine and systolic blood pressure were shown to increase significantly with an increase in the log-transformed serum PFOA concentration, after adjusting for potential confounding variables. Adjusted ORs comparing participants at the 80th versus the 20th percentiles were 2.62 for hypertension (95% CI 2.09 to 3.14), and a positive association was also evident in models based on quartiles or based on restricted cubic splines.
Conclusion: These findings suggest that background exposure to PFOA may continue a risk factor for the development of cardiovascular diseases.

ASN Neuro. 2012 May 30;4(4).
Di-(2-ethylhexyl) phthalate and autism spectrum disorders
Testa C, Nuti F, Hayek J, De Felice C, Chelli M, Rovero P, Latini G, Papini AM
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ASDs (autism spectrum disorders) are a complex group of neurodevelopment disorders, still poorly understood, steadily rising in frequency and treatment refractory. Extensive research has been so far unable to explain the aetiology of this condition, whereas a growing body of evidence suggests the involvement of environmental factors. Phthalates, given their extensive use and their persistence, are ubiquitous environmental contaminants. They are EDs (endocrine disruptors) suspected to interfere with neurodevelopment. Therefore they represent interesting candidate risk factors for ASD pathogenesis. The aim of this study was to evaluate the levels of the primary and secondary metabolites of DEHP [di-(2-ethylhexyl) phthalate] in children with ASD. A total of 48 children with ASD (male: 36, female: 12; mean age: 11±5 years) and age- and sex-comparable 45 HCs (healthy controls; male: 25, female: 20; mean age: 12±5 years) were enrolled. A diagnostic methodology, based on the determination of urinary concentrations of DEHP metabolites by HPLC-ESI-MS (HPLC electrospray ionization MS), was applied to urine spot samples. MEHP [mono-(2-ethylhexenyl) 1,2-benzenedicarboxylate], 6-OH-MEHP [mono-(2-ethyl-6-hydroxyhexyl) 1,2-benzenedicarboxylate], 5-OH-MEHP [mono-(2-ethyl-5-hydroxyhexyl)1,2-benzenedicarboxylate] and 5-oxo-MEHP [mono-(2-ethyl-5-oxohexyl)1,2-benzenedicarboxylate] were measured and compared with unequivocally characterized, pure synthetic compounds (>98%) taken as standard. In ASD patients, significant increase in 5-OH-MEHP (52.1%, median 0.18) and 5-oxo-MEHP (46.0%, median 0.096) urinary concentrations were detected, with a significant positive correlation between 5-OH-MEHP and 5-oxo-MEHP (r_s=0.668, P<0.0001). The fully oxidized form 5-oxo-MEHP showed 91.1% specificity in identifying patients with ASDs. Our findings demonstrate for the first time an association between phthalates exposure and ASDs, thus suggesting a previously unrecognized role for these ubiquitous environmental contaminants in the pathogenesis of autism.

Environ Health Perspect. 2012 Apr 27. [Epub ahead of print]
Prenatal Bisphenol A Exposure and Child Behavior in an Inner City Cohort
Perera F, Vishnevetsky J, Herbstman JB, Calafat AM, Xiong W, Rauh V, Wang S
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Background: Experimental laboratory evidence suggests that bisphenol A (BPA), an endocrine disruptor, is a neurodevelopmental toxicant. However, there have been limited and inconclusive results with respect to sex-specific BPA effects on child behavior. Objectives: We examined the association between prenatal BPA exposure and child behavior, adjusting for postnatal BPA exposure, and hypothesizing sex-specific effects.
Methods: We followed African-American and
Dominican women and their children from pregnancy to age 5 years, collecting spot urine samples from the mothers during pregnancy (34 weeks on average) and from children between 3-4 years to estimate BPA exposure. We assessed child behavior between 3-5 years using the Child Behavior Checklist (CBCL) and used generalized linear models to test the association between BPA exposure and child behavior, adjusting for potential confounders.
Results: The analysis was conducted on 198 children (87 boys and 111 girls). Among boys, high prenatal BPA exposure highest quartile versus the lowest three quartiles) was associated with significantly higher CBCL scores (more problems) on Emotionally Reactive (1.62 times greater, 95% CI 1.13-2.32) and Aggressive Behavior syndromes (1.29 times greater, 95% CI 1.09, 1.53). Among girls, higher exposure was associated with lower scores on all syndromes, reaching statistical significance for Anxious/Depressed (0.75 times as high, 95% CI 0.57, 0.99) and Aggressive Behavior (0.82 times as high, 95% CI 0.70, 0.97).
Conclusion: These results suggest that prenatal exposure to BPA may affect child behavior, and differently among boys and girls.

Environ Int. 2012 Aug;43:21-8. Epub 2012 Mar 30.
4-Nonylphenol and bisphenol A in Swedish food and exposure in Swedish nursing women
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4-Nonylphenol (NP) and bisphenol A (BPA) are phenolic substances used in high volumes by the industry. Studies on cells and in experimental animals have shown that both these compounds can be classified as estrogenic hormone disrupters. Information about the exposure of humans to NP and BPA is still scarce, especially regarding levels in human blood. The first aim of this study was to investigate possible sources of NP and BPA exposure from food, by analyzing the levels of NP and BPA from a Swedish food market basket, based on the Swedish per capita food consumption. A second aim was to investigate blood serum levels of NP and BPA, as well as NP-ethoxylates, among young women in Sweden (n=100). Moreover, associations between food consumption and blood NP and BPA levels were studied. In food, NP was to some extent found at levels above limit of quantification (LOQ 20 ng/g fresh weight) in fruits, cereal products, vegetables, and potatoes. BPA levels above LOQ (2 ng/g fresh weight) were found in fish, meats, potatoes, and dairy products. The estimated mean intakes per capita were (medium bound) 27 μg NP/day and 3.9 μg BPA/day, showing that food is a source of BPA and NP in the general Swedish population. In blood serum, free NP above limit of detection (LOD 0.5 ng/g) was detected in 46% of the study participants while detectable levels of total NP (LOD 0.8 ng/g) were observed in 43%. The corresponding percentages for BPA were 25% and 22%, respectively. The results indicate that there is a continuous source of exposure to NP and BPA that is high enough for free NP and BPA to be detected in some consumers. Among the participants with quantifiable levels of free and total NP (n=38), 85% (median, range: 38-112%) of the NP was present as free NP. For BPA 76% (49-109%) was detected as free BPA (n=15). All women had levels of ethoxylates of NP below LOD

(0.1-0.7 ng/g). A significantly higher total consumption of fruits and vegetables was reported in questionnaires by participants with NP levels at or above LOD than among women with levels below LOD. This result is supporting the market basket results of relatively high NP levels in these types of food.

Hum Reprod. 2012 Apr;27(4):1191-201. Epub 2012 Feb 2.
Intrauterine exposure to mild analgesics during pregnancy and the occurrence of cryptorchidism and hypospadia in the offspring: the Generation R Study
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BACKGROUND: Recently, over-the-counter mild analgesic use during pregnancy has been suggested to influence the risk of reproductive disorders in the offspring. We examined the influence of maternal exposure to mild analgesics during pregnancy on the occurrence of cryptorchidism and hypospadia in their offspring.
METHODS: Associations between maternal exposure to mild analgesics during pregnancy and cryptorchidism or hypospadia in the offspring were studied in 3184 women participating in a large population-based prospective birth cohort study from early pregnancy onwards in the Netherlands (2002-2006), the Generation R Study. Cryptorchidism and hypospadia were identified during routine screening assessments performed in child health care centres by trained physicians. The use of mild analgesics was assessed in three prenatal questionnaires in pregnancy, resulting in four periods of use, namely, periconception period, first 14 weeks of gestation, 14-22 weeks of gestation and 20-32 weeks of gestation. Logistic regression analyses were used to study the associations between maternal exposure to mild analgesics and cryptorchidism and hypospadia.
RESULTS: The cumulative prevalence over 30 months of follow up was 2.1% for cryptorchidism and 0.7% for hypospadia. Use of mild analgesics in the second period of pregnancy (14-22 weeks) increased the risk of congenital cryptorchidism [adjusted odds ratio (OR) 2.12; 95% confidence interval (CI) 1.17-3.83], primarily due to the use of acetaminophen (paracetamol) (adjusted OR 1.89; 95% CI 1.01-3.51). Among mothers of cryptorchid sons, 33.8% reported (23 of 68) the use of mild analgesics during pregnancy, compared with 31.8% (7 of 22) of mothers with a boy with hypospadia and 29.9% (926 of 3094) of mothers with healthy boys.
CONCLUSIONS: Our results suggest that intrauterine exposure to mild analgesics, primarily paracetamol, during the period in pregnancy when male sexual differentiation takes place, increases the risk of cryptorchidism.

J Allergy Clin Immunol. 2012 Jun 14. [Epub ahead of print]
Urinary levels of triclosan and parabens are associated with aeroallergen and food sensitization
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BACKGROUND: Endocrine-disrupting compounds (EDCs) have immune-modulating effects. We were interested in determining their association with allergic sensitization.
OBJECTIVE: We sought to determine the association between EDCs and allergic sensitization and whether this relationship depends on the antimicrobial properties of the EDCs, sex, or both.
METHODS: Data were obtained from the 2005-2006 National Health and Nutrition Examination Survey in which urinary bisphenol A; triclosan; benzophenone-3; propyl, methyl, butyl, and ethyl parabens; and specific IgE levels were available for 860 children. Aeroallergen and food sensitizations were defined as having at least 1 positive (≥0.35 kU/L) specific IgE level to an

aeroallergen or a food. Logistic regression was used to determine the association of EDCs and sensitization. Analyses were adjusted for urinary creatinine level, age, sex, ethnicity, and poverty index ratio.
RESULTS: The odds of aeroallergen sensitization significantly increased with the level of the antimicrobial EDCs triclosan and propyl and butyl parabens (P ≤ .04). The odds of food sensitization significantly increased with the level of urinary triclosan among male subjects (odds ratio for third vs first tertiles, 3.9; P = .02 for trend). There was a significant interaction between sex and triclosan level, with male subjects being more likely to be food sensitized with exposure (P = .03). Similar associations were not identified for the nonantimicrobial EDCs bisphenol A and benzophenone-3 (P > .2).
CONCLUSIONS: As a group, EDCs are not associated with allergen sensitization. However, levels of the antimicrobial EDCs triclosan and parabens were significantly associated with allergic sensitization. The potential role of antimicrobial EDCs in allergic disease warrants further study because they are commonly used in Western society.

Bruttoliste

Bisphenol A

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5: Azzouz A, Ballesteros E. Gas chromatography-mass spectrometry determination of pharmacologically active substances in urine and blood samples by use of a continuous solid-phase extraction system and microwave-assisted derivatization. J Chromatogr B Analyt Technol Biomed Life Sci. 2012 Apr 1;891-892:12-9.

6: Gonzalo-Lumbreras R, Sanz-Landaluze J, Guinea J, Cámara C. Miniaturized extraction methods of triclosan from aqueous and fish roe samples. Bioconcentration studies in zebrafish larvae (Danio rerio). Anal Bioanal Chem. 2012 May;403(4):927-37.

7: Geens T, Neels H, Covaci A. Distribution of bisphenol-A, triclosan and n-nonylphenol in human adipose tissue, liver and brain. Chemosphere. 2012 May;87(7):796-802.

Flame retardants

1: Chen D, Letcher RJ, Burgess NM, Champoux L, Elliott JE, Hebert CE, Martin P, Wayland M, Chip Weseloh DV, Wilson L. Flame retardants in eggs of four gull species (Laridae) from breeding sites spanning Atlantic to Pacific Canada. Environ Pollut. 2012 Sep;168:1-9.

2: Roberts SC, Macaulay LJ, Stapleton HM. In Vitro Metabolism of the Brominated Flame Retardants 2-Ethylhexyl-2,3,4,5-Tetrabromobenzoate (TBB) and Bis(2-ethylhexyl) 2,3,4,5-Tetrabromophthalate (TBPH) in Human and Rat Tissues. Chem Res Toxicol. 2012 May 23. [Epub ahead of print]

3: van der Veen I, de Boer J. Phosphorus flame retardants: Properties, production, environmental occurrence, toxicity and analysis. Chemosphere. 2012 Aug;88(10):1119-53.

4: Eng ML, Elliott JE, MacDougall-Shackleton SA, Letcher RJ, Williams TD. Early exposure to 2,2',4,4',5-pentabromodiphenyl ether (BDE-99) affects mating behavior of zebra finches. Toxicol Sci. 2012 May;127(1):269-76.

5: Norrgran J, Jones B, Lindquist NG, Bergman A. Decabromobiphenyl, polybrominated diphenyl ethers, and brominated phenolic compounds in serum of cats diagnosed with the endocrine disease feline hyperthyroidism. Arch Environ Contam Toxicol. 2012 Jul;63(1):161-8.

In vitro studier ved DTU-FOOD

Søgt i Pubmed med følgende kriterier:

" Endocrine disrupt* AND in vitro*AND expose*" og "Paraben* AND in vitro*"

Publiceret fra i perioden 2012-03-01-2012-06-31 (Marts - Juni 2012)

Efter at have fjernet gengangere, fra forrige litteraturopdateringslister, gav litteratursøgningen, med de angivne søgekriterier, tilsammen en liste med i alt 21 artikler herudover fandtes 3 yderligere artikler:

Udvalgte publikationer

The estrogenic potential of salicylate esters and their possible risks in foods and cosmetics.
Zhang Z, Jia C, Hu Y, Sun L, Jiao J, Zhao L, Zhu D, Li J, Tian Y, Bai H, Li R, Hu J.
Abstract:

Salicylate esters (SEs), a class of chemicals extensively used as flavor and fragrance additives in foods, beverages and a wide variety of consumer products, are suspected to have estrogenic activity based on chemical analysis of in silica molecular docking. We evaluated the estrogenic potentials of phenyl salicylate (PhS), benzyl salicylate (BzS), phenethyl salicylate (PES), ethyl salicylate (ES) and methyl salicylate (MS) using an in vitro human estrogen receptor α (hERα)-coactivator recruiting assay and in vivo immature rodent uterotrophic bioassays. We found that PhS, BzS and PES showed obvious in vitro hERα agonistic activities; BzS in particular exhibited a higher estrogenic activity compared to bisphenol A (BPA). The uterine weights were significantly increased in mice treated with 11.1, 33.3, 100 and 300 mg/kg/day BzS and 33.3mg/kg/day PES and rats treated with 3.7, 11.1, 33.3 and 100mg/kg/day BzS for 3 days ($P<0.05$). Finally, we transformed the daily intakes and the dermal exposures of SEs in the real world into estradiol equivalent concentrations (EEQs). We found that the EEQ of BzS daily intake in consumers in the U.S. and the EEQs of dermal BzS and PES exposure among high-volume users worldwide were higher than the maximum secure daily estradiol intake recommended by the U.S. Food and Drug Administration (FDA). In particular, the EEQ for dermal BzS exposure was up to 162 ng EEQ/kg, which is 3.3 times higher than the maximal acceptable daily E(2) intake recommended by the Joint FAO/WHO Expert Committee on Food Additives (JECFA).

BLTK1 Murine Leydig Cells: A Novel Steroidogenic Model for Evaluating the Effects of Reproductive and Developmental Toxicants.
Forgacs AL, Ding Q, Jaremba RG, Huhtaniemi IT, Rahman NA, Zacharewski TR.

Abstract:
Leydig cells are the primary site of androgen biosynthesis in males. Several environmental toxicants target steroidogenesis resulting in both developmental and reproductive effects including testicular dysgenesis syndrome. The aim of this study was to evaluate the effect of several structurally diverse endocrine disrupting compounds (EDCs) on steroidogenesis in a novel BLTK1 murine Leydig cell model. We demonstrate that BLTK1 cells possess a fully functional steroidogenic pathway that produces low basal levels of testosterone (T) and express all the necessary steroidogenic enzymes including Star, Cyp11a1, Cyp17a1, Hsd3b1, Hsd17b3, and Srd5a1. Recombinant human chorionic gonadotropin (rhCG) and forskolin (FSK) elicited concentration- and time-dependent induction of 3',5'-cyclic adenosine monophosphate, progesterone (P), and T, as well as the differential expression of Star, Hsd3b6, Hsd17b3, and Srd5a1 messenger RNA levels. The evaluation of several structurally diverse male reproductive toxicants including 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD), atrazine, prochloraz, triclosan, monoethylhexyl phthalate

(MEHP), glyphosate, and RDX in BLTK1 cells suggests different modes of action perturb steroidogenesis. For example, prochloraz and triclosan antifungals reduced rhCG induction of T, consistent with published in vivo data but did not alter basal T levels. In contrast, atrazine and MEHP elicited modest induction of basal T but antagonized rhCG-mediated induction of T levels, whereas TCDD, glyphosate, and RDX had no effect on basal or rhCG induction of T in BLTK1 cells. These results suggest that BLTK1 cells maintain rhCG-inducible steroidogenesis and are a viable in vitro Leydig cell model to evaluate the effects of EDCs on steroidogenesis. This model can also be used to elucidate the different mechanisms underlying toxicant-mediated disruption of steroidogenesis.

Differential effects of environmental chemicals and food contaminants on adipogenesis, biomarker release and PPARγ activation.
Taxvig C, Dreisig K, Boberg J, Nellemann C, Schelde AB, Pedersen D, Boergesen M, Mandrup S, Vinggaard AM.
Abstract:
Eleven environmental relevant chemicals were investigated for their ability to affect adipogenesis *in vitro*, biomarker release from adipocytes and PPARα and γ activation. We found that butylparaben stimulated adipogenesis in 3T3-L1 adipocytes and increased release of leptin, adiponectin and resistin from the cells. Butylparaben activated PPARγ as well, which may be a mediator of the adipogenic effect. Polychlorinated biphenyl (PCB)153 also stimulate adipogenesis and biomarker release, but did not affect PPARs. The data indicates that PPARγ activating chemicals often stimulate adipocyte differentiation although PPARγ activation is neither a requirement nor a guarantee for stimulation. Four out of the eleven chemicals (bisphenol A, mono-ethylhexyl phthalate, butylparaben, PCB 153) caused increased adipogenesis. The release of adipocyte-secreted hormones was sometimes but not always correlated with the effect on adipocyte differentiation. Eight chemicals were able to cause increased leptin release. These findings strengthen the hypothesis that chemicals can interfere with pathways related to obesity development.

Bruttoliste

1. ICCVAM recommends **in vitro** test method for **endocrine-disruptors**.

[No authors listed]
Altern Lab Anim. 2012 Mar;40(1):11. No abstract available.

2. Paracetamol (acetaminophen), aspirin (acetylsalicylic acid) and indomethacin are anti-androgenic in the rat foetal testis.

Kristensen DM, Lesné L, Le Fol V, Desdoits-Lethimonier C, Dejucq-Rainsford N, Leffers H, Jégou B.
Int J Androl. 2012 Jun;35(3):377-84. doi: 10.1111/j.1365-2605.2012.01282.x.

3. Screening Estrogenic Activities of Chemicals or Mixtures In Vivo Using Transgenic (cyp19a1b-GFP) Zebrafish Embryos.

Brion F, Le Page Y, Piccini B, Cardoso O, Tong SK, Chung BC, Kah O.
PLoS One. 2012;7(5):e36069. Epub 2012 May 7.

4. Expression and DNA methylation changes in human breast epithelial cells after bisphenol A exposure.

Fernandez SV, Huang Y, Snider KE, Zhou Y, Pogash TJ, Russo J.
Int J Oncol. 2012 Jul;41(1):369-77. doi: 10.3892/ijo.2012.1444. Epub 2012 Apr 20.

5. Changes in concentrations of hydrophilic organic contaminants and of **endocrine-disrupting** potential downstream of small communities located adjacent to headwaters.

Jarosova B, Blaha L, Vrana B, Randak T, Grabic R, Giesy JP, Hilscherova K.
Environ Int. 2012 Sep;45:22-31. Epub 2012 May 8.

6. Serum and follicular fluid concentrations of polybrominated diphenyl ethers and **in-vitro** fertilization outcome.

Johnson PI, Altshul L, Cramer DW, Missmer SA, Hauser R, Meeker JD.
Environ Int. 2012 Sep;45:9-14. Epub 2012 May 7.

7. A testing strategy for the identification of mammalian, systemic **endocrine disruptors** with particular focus on steroids.

Kolle SN, Ramirez T, Kamp HG, Buesen R, Flick B, Strauss V, van Ravenzwaay B.
Regul Toxicol Pharmacol. 2012 Jul;63(2):259-78. Epub 2012 Apr 24.

8. Induced growth of BG-1 ovarian cancer cells by 17β-estradiol or various **endocrine disrupting** chemicals was reversed by resveratrol via downregulation of cell cycle progression.

Kang NH, Hwang KA, Kim TH, Hyun SH, Jeung EB, Choi KC.
Mol Med Report. 2012 Jul;6(1):151-6. doi: 10.3892/mmr.2012.887. Epub 2012 Apr 23.

9. Human endometrial cell coculture reduces the **endocrine disruptor** toxicity on mouse embryo development.

Song HY, Lee MS, Lee YS, Lee HH.
J Occup Med Toxicol. 2012 Apr 30;7(1):7. [Epub ahead of print]

10. Long-term effects of a binary mixture of perfluorooctane sulfonate (PFOS) and bisphenol A (BPA) in zebrafish (Danio rerio).

Keiter S, Baumann L, Färber H, Holbech H, Skutlarek D, Engwall M, Braunbeck T.
Aquat Toxicol. 2012 Aug 15;118-119:116-29. Epub 2012 Apr 11.

11. Quantification of steroids and **endocrine disrupting** chemicals in rat ovaries by LC-MS/MS for reproductive toxicology assessment.

Quignot N, Tournier M, Pouech C, Cren-Olivé C, Barouki R, Lemazurier E.
Anal Bioanal Chem. 2012 Jun;403(6):1629-40. Epub 2012 Apr 22.

12. Bisphenol AF may cause testosterone reduction by directly affecting testis function in adult male rats.

Feng Y, Yin J, Jiao Z, Shi J, Li M, Shao B.
Toxicol Lett. 2012 Jun 1;211(2):201-9. Epub 2012 Apr 6.

13. GPR30, the non-classical membrane G protein related estrogen receptor, is overexpressed in human seminoma and promotes seminoma cell proliferation.

Chevalier N, Vega A, Bouskine A, Siddeek B, Michiels JF, Chevallier D, Fénichel P.
PLoS One. 2012;7(4):e34672. Epub 2012 Apr 4.

14. Differential Estrogenic Actions of **Endocrine-Disrupting** Chemicals Bisphenol A, Bisphenol AF and Zearalenone through Estrogen Receptor α and β **in Vitro**.

Li Y, Burns KA, Arao Y, Luh CJ, Korach KS.
Environ Health Perspect. 2012 Apr 11. [Epub ahead of print]

15. Evaluation of the Daphnia magna reproduction test for detecting **endocrine disruptors**.

Dang Z, Cheng Y, Chen HM, Cui Y, Yin HH, Traas T, Montforts M, Vermeire T.
Chemosphere. 2012 Jul;88(4):514-23. Epub 2012 Apr 1.

16. BLTK1 Murine Leydig Cells: A Novel Steroidogenic Model for Evaluating the Effects of Reproductive and Developmental Toxicants.

Forgacs AL, Ding Q, Jaremba RG, Huhtaniemi IT, Rahman NA, Zacharewski TR.
Toxicol Sci. 2012 Jun;127(2):391-402. Epub 2012 Mar 29.

17. Exposure of alveolar macrophages to polybrominated diphenyl ethers suppresses the release of pro-inflammatory products **in vitro**.

Hennigar SR, Myers JL, Tagliaferro AR.
Exp Biol Med (Maywood). 2012 Apr 1;237(4):429-34. Epub 2012 Mar 27.

18. The estrogenic potential of salicylate esters and their possible risks in foods and cosmetics.

Zhang Z, Jia C, Hu Y, Sun L, Jiao J, Zhao L, Zhu D, Li J, Tian Y, Bai H, Li R, Hu J.
Toxicol Lett. 2012 Mar 7;209(2):146-53. Epub 2011 Dec 16.

19. Endocrine-active chemicals in mammary cancer causation and prevention.

Jenkins S, Betancourt AM, Wang J, Lamartiniere CA.
J Steroid Biochem Mol Biol. 2012 Apr;129(3-5):191-200. Epub 2011 Jun 23. Review.

20. The influence of volatile solvents on transport across model membranes and human skin.

Oliveira G, Hadgraft J, Lane ME.
Int J Pharm. 2012 May 24. [Epub ahead of print]

21. Effect of Direction (Epidermis-To-Dermis and Dermis-To-Epidermis) on the Permeation of Several Chemical Compounds through Full-Thickness Skin and Stripped Skin.

Oshizaka T, Todo H, Sugibayashi K.
Pharm Res. 2012 May 24. [Epub ahead of print]

Herudover er der yderligere 3 artikel, som ikke blev fanget af de valgte søgekriterier:

Differential effects of environmental chemicals and food contaminants on adipogenesis, biomarker release and PPARγ activation.
Taxvig C, Dreisig K, Boberg J, Nellemann C, Schelde AB, Pedersen D, Boergesen M, Mandrup S, Vinggaard AM.
Mol Cell Endocrinol. 2012 Apr 14. [Epub ahead of print]

QSAR model for human pregnane X receptor (PXR) binding: Screening of environmental chemicals and correlations with genotoxicity, endocrine disruption and teratogenicity.
Dybdahl M, Nikolov NG, Wedebye EB, Jónsdóttir SO, Niemelä JR.
Toxicol Appl Pharmacol. 2012 May 22. [Epub ahead of print]
PMID: 22627063

QSAR Model for Androgen Receptor Antagonism - Data from CHO Cell Reporter Gene Assays

Gunde Egeskov Jensen, Nikolai Georgiev Nikolov, Karin Dreisig, Anne Marie Vinggaard and Jay Russel Niemelä

J Steroids Horm Sci 2012, S:2http://dx.doi.org/10.4172/2157-7536.S2-006

In Vivo studier ved DTU - FOOD

Søgning er udført på PubMed og dækker perioden 1/4 2012 – 20/6 2012

(april - juni 2012)

Følgende søgeprofil er benyttet: "(endocrine disrupt*) AND (utero*) AND (rat OR mice OR mammal*)" samt "(endocrine disrupt*) AND (rat OR mice OR mammal*)". Efter at have fjernet gengangere fra dem vi havde med på den forrige litteraturopdateringsliste, gav litteratursøgningen tilsammen en liste med i alt 31 (+ yderligere 3) artikler (Bruttolisten):

Udvalgte publikationer:

Adverse effects on sexual development in rat offspring after low dose exposure to a mixture of endocrine disrupting pesticides.
Hass U, Boberg J, Christiansen S, Jacobsen PR, Vinggaard AM, Taxvig C, Poulsen ME, Herrmann SS, Jensen BH, Petersen A, Clemmensen LH, Axelstad M.

The present study investigated whether a mixture of low doses of five environmentally relevant endocrine disrupting pesticides, epoxiconazole, mancozeb, prochloraz, tebuconazole and procymidone, would cause adverse developmental toxicity effects in rats. In rat dams, a significant increase in gestation length was seen, while in male offspring increased nipple retention and increased incidence and severity of genital malformations were observed. Severe mixture effects on gestation length, nipple retention and genital malformations were seen at dose levels where the individual pesticides caused no or smaller effects when given alone. Generally, the mixture effect predictions based on dose-additivity were in good agreement with the observed effects. The results indicate that there is a need for modification of risk assessment procedures for pesticides, in order to take account of the mixture effects and cumulative intake, because of the potentially serious impact of mixed exposure on development and reproduction in humans.

Reprod Toxicol. 2012 Jun 4. [Epub ahead of print]
Persistent developmental toxicity in rat offspring after low dose exposure to a mixture of endocrine disrupting pesticides.
Jacobsen PR, Axelstad M, Boberg J, Isling LK, Christiansen S, Mandrup KR, Berthelsen LO, Vinggaard AM, Hass U.

There is growing concern of permanent damage to the endocrine and nervous systems after developmental exposure to endocrine disrupting chemicals. In this study the permanent reproductive and neurobehavioral effects of combined exposure to five endocrine disrupting pesticides, epoxiconazole, mancozeb, prochloraz, tebuconazole and procymidone, were examined. Pregnant and lactating rat dams were dosed with a mixture of the five pesticides at three different doses, or with the individual pesticides at one of two doses. Adverse effects were observed in young

and adult male offspring from the group exposed to the highest dose of the mixture. These included reduced prostate and epididymis weights, increased testes weights, altered prostate histopathology, increased density of mammary glands, reduced sperm counts, and decreased spatial learning. As no significant effects were seen following single compound exposure at the doses included in the highest mixture dose, these results indicate cumulative adverse effects of the pesticide mixture.

Gestational Exposure to Bisphenol A Produces Transgenerational Changes in Behaviors and Gene Expression.
Wolstenholme JT, Edwards M, Shetty SR, Gatewood JD, Taylor JA, Rissman EF, Connelly JJ.
Endocrinology. 2012 Jun 15. [Epub ahead of print]

Bisphenol A (BPA) is a plasticizer and an endocrine-disrupting chemical. It is present in a variety of products used daily including food containers, paper, and dental sealants and is now widely detected in human urine and blood. Exposure to BPA during development may affect brain organization and behavior, perhaps as a consequence of its actions as a steroid hormone agonist/antagonist and/or an epigenetic modifier. Here we show that BPA produces transgenerational alterations in genes and behavior. Female mice received phytoestrogen-free chow with or without BPA before mating and throughout gestation. Plasma levels of BPA in supplemented dams were in a range similar to those measured in humans. Juveniles in the first generation exposed to BPA in utero displayed fewer social interactions as compared with control mice, whereas in later generations (F(2) and F(4)), the effect of BPA was to increase these social interactions. Brains from embryos (embryonic d 18.5) exposed to BPA had lower gene transcript levels for several estrogen receptors, oxytocin, and vasopressin as compared with controls; decreased vasopressin mRNA persisted into the F(4) generation, at which time oxytocin was also reduced but only in males. Thus, exposure to a low dose of BPA, only during gestation, has immediate and long-lasting, transgenerational effects on mRNA in brain and social behaviors. Heritable effects of an endocrine-disrupting chemical have implications for complex neurological diseases and highlight the importance of considering gene

Perinatal ethinyl oestradiol alters mammary gland development in male and female Wistar rats.
Mandrup KR, Hass U, Christiansen S, Boberg J.
Int J Androl. 2012 Jun;35(3):385-96. doi: 10.1111/j.1365-2605.2012.01258.x. Epub 2012 Mar 19.

Increased attention is being paid to human mammary gland development because of concerns for environmental influences on puberty onset and breast cancer development. Studies in rodents have showed a variety of changes in the mammary glands after perinatal exposure to endocrine disrupting chemicals, indicating progressed development of mammary glands when exposed to oestrogens early in life. However, laboratories use different parameters to evaluate the development of mammary glands, making studies difficult to compare. Moreover, studies of whole mounts in Wistar rats are lacking. In the present study, Wistar rats were exposed to 0, 5, 15 or 50 μg/kg of ethinyl oestradiol per day during gestation and lactation. A wide range of morphological parameters were evaluated in whole mounts of

mammary glands from male and female offspring PD21-22. This study showed that in both male and female pre-pubertal Wistar rats, mammary gland development was accelerated after perinatal oestrogen exposure with increase in size, density and number of terminal end buds (TEBs). In female rats, the most sensitive parameters were the distance to the fifth gland, the relative growth towards the lymph node and the overall density. The sensitive endpoints in male rats were TEB numbers, both in the whole gland and in the zone C, the overall- and the highest density. The overall density was sensitive in both male and female rats and was considered a good representative of both branching and budding of the gland. The number of TEBs in zone C was representative of the number of TEBs in the whole gland. Further studies in older Wistar rats and with weak oestrogenic compounds could be performed to validate mammary gland examination as an endpoint in reproductive toxicity studies and to examine how early life environmental exposures may alter mammary gland development, disrupt lactation and alter susceptibility to breast cancer.

A critique of the European Commission Document, "State of the Art Assessment of Endocrine Disrupters".
Rhomberg LR, Goodman JE, Foster WG, Borgert CJ, Van Der Kraak G.
Crit Rev Toxicol. 2012 Jul;42(6):465-73. Epub 2012 May 26.
(Kun abstract)

Abstract
In this commentary, we critique a recently finalized document titled "State of the Art Assessment of Endocrine Disrupters" (SOA Assessment). The SOA Assessment was commissioned by the European Union Directorate-General for the Environment to provide a basis for developing scientific criteria for identifying endocrine disruptors and reviewing and possibly revising the European Community Strategy on Endocrine Disrupters. In our view, the SOA Assessment takes an anecdotal approach rather than attempting a comprehensive assessment of the state of the art or synthesis of current knowledge. To do the latter, the document would have had to (i) distinguish between apparent associations of outcomes with exposure and the inference of an endocrine-disruption (ED) basis for those outcomes; (ii) constitute a complete and unbiased survey of new literature since 2002 (when the WHO/IPCS document, "Global Assessment of the State-of-the-Science of Endocrine Disruptors" was published); (iii) consider strengths and weaknesses and issues in interpretation of the cited literature; (iv) follow a weight-of-evidence methodology to evaluate evidence of ED; (v) document the evidence for its conclusions or the reasoning behind them; and (vi) present the evidence for or reasoning behind why conclusions that differ from those drawn in the 2002 WHO/IPCS document need to be changed. In its present form, the SOA Assessment fails to provide a balanced and critical assessment or synthesis of literature relevant to ED. We urge further evidence-based evaluations to develop the needed scientific basis to support future policy decisions.

Bruttoliste

1. The **endocrine disruptor** Diethylstilbestrol induces adipocyte differentiation and promotes obesity in **mice**.
Hao CJ, Cheng XJ, Xia HF, Ma X.
Toxicol Appl Pharmacol. 2012 Jun 15. [Epub ahead of print]

2. Chemoprotective effects of kolaviron on ethylene glycol monoethyl ether-induced pituitary-thyroid axis toxicity in male rats.
Adedara IA, Farombi EO.
Andrologia. 2012 Jun 19. doi: 10.1111/j.1439-0272.2012.01321.x. [Epub ahead of print]

3. Gestational Exposure to Bisphenol A Produces Transgenerational Changes in Behaviors and Gene Expression.
Wolstenholme JT, Edwards M, Shetty SR, Gatewood JD, Taylor JA, Rissman EF, Connelly JJ.
Endocrinology. 2012 Jun 15. [Epub ahead of print]

4. Thyroid hormone receptors: The challenge of elucidating isotype-specific functions and cell-specific response.
Flamant F, Gauthier K.
Biochim Biophys Acta. 2012 Jun 12. [Epub ahead of print]

5. Of **mice** and men (and rats): phthalate-induced fetal testis **endocrine disruption** is species-dependent.
Johnson K, Heger N, Boekelheide K.
Toxicol Sci. 2012 Jun 14. [Epub ahead of print]

6. Effects of commercial formulations of deltamethrin and/or thiacloprid on thyroid hormone levels in **rat** serum.
Sekeroglu V, Sekeroglu ZA, Demirhan ES.
Toxicol Ind Health. 2012 Jun 7. [Epub ahead of print]

7. Persistent developmental toxicity in **rat** offspring after low dose exposure to a mixture of **endocrine disrupting** pesticides.
Jacobsen PR, Axelstad M, Boberg J, Isling LK, Christiansen S, Mandrup KR, Berthelsen LO, Vinggaard AM, Hass U.
Reprod Toxicol. 2012 Jun 4. [Epub ahead of print]

8. Phthalate Exposure Changes the Metabolic Profile of Cardiac Muscle Cells.
Posnack NG, Swift LM, Kay MW, Lee NH, Sarvazyan N.
Environ Health Perspect. 2012 Jun 6. [Epub ahead of print]

9. Adverse effects on sexual development in **rat** offspring after low dose exposure to a mixture of **endocrine disrupting** pesticides.
Hass U, Boberg J, Christiansen S, Jacobsen PR, Vinggaard AM, Taxvig C, Poulsen ME, Herrmann SS, Jensen BH, Petersen A, Clemmensen LH, Axelstad M.
Reprod Toxicol. 2012 May 29. [Epub ahead of print]

10. Paracetamol (acetaminophen), aspirin (acetylsalicylic acid) and indomethacin are anti-androgenic in the **rat** foetal testis.
Kristensen DM, Lesné L, Le Fol V, Desdoits-Lethimonier C, Dejucq-Rainsford N, Leffers H, Jégou B.
Int J Androl. 2012 Jun;35(3):377-84. doi: 10.1111/j.1365-2605.2012.01282.x.

11. A testing strategy for the identification of **mammalian**, systemic **endocrine disruptors** with particular focus on steroids.
Kolle SN, Ramirez T, Kamp HG, Buesen R, Flick B, Strauss V, van Ravenzwaay B.
Regul Toxicol Pharmacol. 2012 Jul;63(2):259-78. Epub 2012 Apr 24.

12. Acetaldehyde at a Low Concentration Synergistically Exacerbates Allergic Airway Inflammation as an **Endocrine-Disrupting** Chemical and as a Volatile Organic Compound.
Kawano T, Matsuse H, Fukahori S, Tsuchida T, Nishino T, Fukushima C, Kohno S.
Respiration. 2012 Apr 25. [Epub ahead of print]

13. Quantification of steroids and **endocrine disrupting** chemicals in **rat** ovaries by LC-MS/MS for reproductive toxicology assessment.
Quignot N, Tournier M, Pouech C, Cren-Olivé C, Barouki R, Lemazurier E.
Anal Bioanal Chem. 2012 Jun;403(6):1629-40. Epub 2012 Apr 22.

14. Position paper: The membrane estrogen receptor GPER - Clues and questions.
Barton M.
Steroids. 2012 Apr 10. [Epub ahead of print]

15. Accelerated Mammary Tumor Onset in a HER2/Neu Mouse Model Exposed to DDT Metabolites Locally Delivered to the Mammary Gland.
Johnson NA, Ho A, Cline JM, Hughes CL, Foster WG, Davis VL.
Environ Health Perspect. 2012 Apr 18. [Epub ahead of print]

16. Human Fetal Testis Xenografts Are Resistant To Phthalate-Induced **Endocrine Disruption**.
Heger NE, Hall SJ, Sandrof MA, McDonnell EV, Hensley JB, McDowell EN, Martin KA, Gaido KW, Johnson KJ, Boekelheide K.
Environ Health Perspect. 2012 Apr 17. [Epub ahead of print]

17. The metabonomics of combined dietary exposure to phthalates and polychlorinated biphenyls in **mice**.
Zhang J, Yan L, Tian M, Huang Q, Peng S, Dong S, Shen H.
J Pharm Biomed Anal. 2012 Jul;66:287-97. Epub 2012 Apr 3.

18. Coexpression of nuclear receptors and histone methylation modifying genes in the testis: implications for **endocrine disruptor** modes of action.
Anderson AM, Carter KW, Anderson D, Wise MJ.
PLoS One. 2012;7(4):e34158. Epub 2012 Apr 4.

19. High dose bisphenol A impairs hippocampal neurogenesis in female **mice** across generations.
Jang YJ, Park HR, Kim TH, Yang WJ, Lee JJ, Choi SY, Oh SB, Lee E, Park JH, Kim HP, Kim HS, Lee J.
Toxicology. 2012 Jun 14;296(1-3):73-82. Epub 2012 Apr 3.

20. Identification of iopanoic acid as substrate of type 1 deiodinase by a novel nonradioactive iodide-release assay.
Renko K, Hoefig CS, Hiller F, Schomburg L, Köhrle J.
Endocrinology. 2012 May;153(5):2506-13. Epub 2012 Mar 20.

21. Strain specific induction of pyometra and differences in immune responsiveness in **mice** exposed to 17α-ethinyl estradiol or the **endocrine disrupting** chemical bisphenol A.
Kendziorski JA, Kendig EL, Gear RB, Belcher SM.
Reprod Toxicol. 2012 Aug;34(1):22-30. Epub 2012 Mar 10.

22. Maternal bisphenol A oral dosing relates to the acceleration of neurogenesis in the developing neocortex of mouse fetuses.
Komada M, Asai Y, Morii M, Matsuki M, Sato M, Nagao T.
Toxicology. 2012 May 16;295(1-3):31-8. Epub 2012 Mar 7.

23. The novel **endocrine disruptor** tolylfluanid impairs insulin signaling in primary rodent and human adipocytes through a reduction in insulin receptor substrate-1 levels.
Sargis RM, Neel BA, Brock CO, Lin Y, Hickey AT, Carlton DA, Brady MJ.
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Herudover er der yderligere 3 artikler, som ikke blev fanget af de valgte søgekriterier:

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(udvalgt)

Perinatal exposure to xenoestrogens impairs mammary gland differentiation and modifies milk composition in Wistar rats.

Kass L, Altamirano GA, Bosquiazzo VL, Luque EH, Muñoz-de-Toro M.

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A critique of the European Commission Document, "State of the Art Assessment of **Endocrine** Disrupters".
Rhomberg LR, Goodman JE, Foster WG, Borgert CJ, Van Der Kraak G.
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Wildlife studier ved Biologisk Institut, Syddansk Universitet (SDU)

Søgningen er udført på Web of Science og dækker perioden 27/3 2012 - 26/6 2012.

Søgeprofilen kombinerer:	Endocrine disrupt*	og	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

Marie,B., Huet,H., Marie,A., Djediat,C., Puiseux-Dao,S., Catherine,A., Trinchet,I., and Edery,M., 2012. Effects of a toxic cyanobacterial bloom (Planktothrix agardhii) on fish: Insights from histopathological and quantitative proteomic assessments following the oral exposure of medaka fish (Oryzias latipes). Aquatic Toxicology 114, 39-48.

Abstract: Cyanobacterial toxic blooms often occur in freshwater lakes and constitute a potential health risk to human populations, as well as to fish and other aquatic organisms. Microcystin-LR (the cyanotoxin most commonly detected in the freshwater environment) is a potent hepatotoxin, deregulating the kinase pathway by inhibiting phosphatases 1 and 2A. Although toxicological effects have been clearly linked to the in vitro exposure of fish to purified microcystins, cyanotoxins are produced by the cyanobacteria together with numerous other potentially toxic molecules, and their overall and specific implications for the health of fish have still not been clearly established and remain puzzlingly difficult to assess.
The medaka fish (Oryzias latipes) was chosen as an in vitro model for studying the effects of a cyanobacterial bloom on liver protein contents using a gel free quantitative approach, iTRAQ in addition to pathology examinations on histological preparations. Fish were gavaged with 5 mu L cyanobacterial extracts (Planktothrix agardhii) from a natural bloom (La Grande Paroisse, France) containing 2.5 mu g equiv. MC-LR. 2 h after exposure, the fish were sacrificed and livers were collected for analysis. Histological observations indicate that hepatocytes present glycogen storage loss, and cellular damages, together with immunological localization of MCs. Using a proteomic approach, 304 proteins were identified in the fish

livers, 147 of them with a high degree of identification confidence. Fifteen of these proteins were statistically significantly different from those of controls (gavaged with water only). Overall, these protein regulation discrepancies clearly indicate that oxidative stress and lipid regulation had occurred in the livers of the exposed medaka fish. In contrast to previous pure microcystin-LR gavage experiments, marked induction of vitellogenin 1 protein was observed for the first time with a cyanobacterial extract. This finding was confirmed by ELISA quantification of vitellogenin liver content, suggesting that the Planktothrix bloom extract had induced the occurrence of an endocrine-disrupting effect.

Vosges,M., Kah,O., Hinfray,N., Chadili,E., Le Page,Y., Combarnous,Y., Porcher,J.M., and Brion,F., 2012. 17 alpha-Ethinylestradiol and nonylphenol affect the development of forebrain GnRH neurons through an estrogen receptors-dependent pathway. Reproductive Toxicology 33, 198-204.

Abstract: There is growing evidence that neuroendocrine circuits controlling development and reproduction are targeted by EDCs. We have previously demonstrated that low concentrations of 17alpha-ethinylestradiol (EE2) disrupt the development of forebrain GnRH neurons during zebrafish development. The objectives of the present study were to determine whether the weak estrogenic compound, nonylphenol (NP), could elicit similar effects to EE2 and to what extent the estrogen receptors are involved in mediating these effects. Using immunohistochemistry, we confirmed that EE2 exposure induces an increase in the number of GnRH-ir neurons and we demonstrated that NP is able to produce similar effects in a concentration-dependent manner. The effects of both NP and EE2 were shown to be blocked by the estrogen receptors (ERs) antagonist ICI 182-780, demonstrating the involvement of functional ERs in mediating their effects. Altogether, these results highlight the need to consider neuroendocrine networks as critical endpoints in the field of endocrine disruption.

DeQuattro,Z.A., Peissig,E.J., Antkiewicz,D.S., Lundgren,E.J., Hedman,C.J., Hemming,J.D., and Barry,T.P., 2012. Effects of progesterone on reproduction and embryonic development in the fathead minnow (Pimephales promelas). Environmental Toxicology and Chemistry 31, 851-856.

Abstract: High concentrations (375 ng/L) of the steroid hormone progesterone (P4) were measured in snowmelt runoff associated with large livestock-feeding operations in Wisconsin. To gain insight into the potential endocrine-disrupting effects of P4 in fish, experiments were conducted to evaluate the effects of short-term exposure to environmentally relevant concentrations of P4 on reproduction and embryonic development in the fathead minnow (Pimephales promelas). For the reproduction assay, groups of reproductively mature fish were exposed for 21 d to nominal concentrations of 0, 10, 100, and 1,000 ng/L P4 in a flow-through system, and various key reproductive endpoints (e.g., egg number, fertilization success) were quantified throughout the exposure period. The embryonic development assay consisted of incubating fathead minnow eggs in static culture to quantify the effects of P4 on early development and hatching success. Progesterone caused dose-dependent decreases in fecundity and fertility and significantly reduced gonadosomatic index and vitellogenin gene expression in females. There were no effects of P4 on early embryonic development or hatching success. Progesterone may be a significant endocrine-disrupting chemical in fish.

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