SUPPLEMENTAL MATERIAL

Table 1: List of Abbreviations

Abbreviation	Full form
BBP	butyl benzyl phthalate
BEHP	bis (2-ethylhexyl) phthalate
BHC	benzene hexachloride
BPA	bisphenol A
CALUX	chemical activated luciferase gene expression
Cd	Cadmium
DDT	dichlorodiphenyltrichloroethane
DEHP	di (2-ethyl hexyl) phthalate
DEN	deep endometriotic nodules
DEP	diethyl phthalate
DES	Diethylstilbestrol
DL	dioxin like
DLC	dioxin like compounds
DMP	dimethyl phthalate
DnBP	di-n-butyl phthalate
DnOP	di-n-octyl phthalate
EDC	endocrine disrupting chemical
ETAAS	electrothermal atomic absorption spectrometry
GC	Gas chromatography
HCB	hexachlorobenzene
Hg	Mercury
HPLC	high performance liquid chromatography
HRMS	high resolution mass spectometry
ICP-MS	inductively coupled plasma mass spectrometry
MS	mass spectometry
mBP / MBP	mono-n-butyl phthalate
mBzP / MBzP	mono-benzyl phthalate
mCHP /MCHP	monocyclohexyl phthalate
mCMHP / MCMHP	mono-[(2-carboxymethyl) hexyl] phthalate
mCPP / MCPP	mono (3-carboxypropyl) phthalate

mECPP / MECPP	mono (2-ethyl-5-carboxyphentyl) phthalate
mEHHP / MEHHP	mono (2-ethyl-5- hydroxyhexyl) phthalate
mEHP / MEHP	mono (2-ethylhexyl) phthalate
mEOHP / MEOHP	mono (2-ethyl-5-oxohexyl) phthalate
mEP / MEP	monoethyl phthalate
miBP	mono (2-isobutyl) phthalate
mNP	monoisonoyl phthalate
mOP /MOP	monooctyl phthalate
MRI	magnetic resonance imaging
MT	metallothioneins
Ni	Nickel
OCEP	organochlorinated environmental pollutant
OCP	organochlorinated pesticides
OHAT	Office of Health Assessment and Translation
OR	Odds ratio
Pb	Lead
PBB	Polybrominated-biphenyl
PBDE	Polybrominated-diphenyl-ether
PCB	polychlorinated biphenyl
PCDD	polychlorinated dibenzo dioxins
PCDF	polychlorinated dibenzo furans
PE	phthalate ester
POP	Persistent organochlorine pollutants
p-p'-DDE	1-1-dichloro-2,2-bis 4-chlorophenyl ethene
t-nonachlor	Trans-nonachlor
TCDD	2,3,7,8 tetrachloro dibenzo-p-dioxin
TEF	toxic equivalent factor
TEQ	toxic equivalent quotient
TMS	tandem mass spectometry
WHO	World Health Organization
UNEP	United Nations Environmental Program

USG	ultrasonography
95% CI	95% confidence interval

Table 2: PRISMA-P (Preferred Reporting Items for Systematic review and Meta-Analysis Protocols) 2015 checklist: recommended items to address in a systematic review protocol			
Section and topic	**Item No**	**Checklist item**	**Comments**
ADMINISTRATIVE INFORMATION			
Title:			
Identification	1a	Identify the report as a protocol of a systematic review	The PRISMA guidelines were used to conduct the systematic review.
Update	1b	If the protocol is for an update of a previous systematic review, identify as such	Not applicable
Registration	2	If registered, provide the name of the registry (such as PROSPERO) and registration number	Not applicable
Authors:			
Contact	3a	Provide name, institutional affiliation, e-mail address of all protocol authors; provide physical mailing address of corresponding author	Dr. Diksha Sirohi, School of Public Health, Faculty of Medicine, The University of Queensland (d.sirohi@uqconnect.edu.au); Dr. Ruqaiya AL Ramadhani, School of Public Health, Faculty of Medicine, The University of Queensland (r.alramadhani@uq.net.au); Dr. Luke D. Knibbs, School of Public Health, Faculty of Medicine, The University of Queensland, (l.knibbs@uq.edu.au) Physical mailing address of corresponding author: Dr. Diksha Sirohi, School of Public Health, Faculty of Medicine, The University of Queensland, Herston QLD - 4006
Contributions	3b	Describe contributions of protocol authors and identify the guarantor of the review	Not applicable

Amendments	4	If the protocol represents an amendment of a previously completed or published protocol, identify as such and list changes; otherwise, state plan for documenting important protocol amendments	Not applicable
Support:			
Sources	5a	Indicate sources of financial or other support for the review	Not applicable
Sponsor	5b	Provide name for the review funder and/or sponsor	Not applicable
Role of sponsor or funder	5c	Describe roles of funder(s), sponsor(s), and/or institution(s), if any, in developing the protocol	Not applicable
INTRODUCTION			
Rationale	6	Describe the rationale for the review in the context of what is already known	Rationale for the review is addressed in the main manuscript on pages 5 and 6 in the Introduction section.
Objectives	7	Provide an explicit statement of the question(s) the review will address with reference to participants, interventions, comparators, and outcomes (PICO)	The PICO statement is explicitly described in the main manuscript on pages 6 and 7 under Search Strategy and Data Sources in the Materials and Methods section.
METHODS			
Eligibility criteria	8	Specify the study characteristics (such as PICO, study design, setting, time frame) and report characteristics (such as years considered, language, publication status) to be used as criteria for eligibility for the review	The PICO statement is described in the main manuscript on pages 6 and 7 under Search Strategy and Data Sources in the Material and Methods section. The inclusion criteria are described on pages 7 and 8 under the Material and Methods sections.
Information sources	9	Describe all intended information sources (such as electronic databases, contact with study authors, trial registers or other grey literature sources) with planned dates of coverage	Three electronic databases (PubMed, EMBASE and Scopus) were searched up to 2 July, 2018.
Search strategy	10	Present draft of search strategy to be used for at least one electronic database, including planned limits, such that it could be repeated	Details of the search strategy are described in Table-3 of the Supplemental Materials.

Study records:			
Data management	11a	Describe the mechanism(s) that will be used to manage records and data throughout the review	The flow-chart showing the selection of studies is mapped out on page 10 of the main manuscript. The details on data management is described on page 11 of the main manuscript under the Result and Discussion section.
Selection process	11b	State the process that will be used for selecting studies (such as two independent reviewers) through each phase of the review (that is, screening, eligibility and inclusion in meta-analysis)	Two reviewers will conduct the search of the databases independently and will apply the eligibility criteria independently to arrive at the final number of studies to be included in the review. Findings will be discussed and discrepancies will be resolved through mutual consensus.
Data collection process	11c	Describe planned method of extracting data from reports (such as piloting forms, done independently, in duplicate), any processes for obtaining and confirming data from investigators	The data extraction process is described on page 11 in the main manuscript under the Results and Discussion section. Table-7 in the Supplemental Materials provides the summary of studies included in the review.
Data items	12	List and define all variables for which data will be sought (such as PICO items, funding sources), any pre-planned data assumptions and simplifications	Data is sought for population (cases, operative or population cohort), exposure to different types of EDCs (BPA, PE, metals and trace elements, dioxins and DLC, organochlorinated environmental pollutants, PCB), controls (controls) and outcome (endometriosis). This systematic review did not receive any funding.
Outcomes and prioritization	13	List and define all outcomes for which data will be sought, including prioritization of main and additional outcomes, with rationale	Outcome is the diagnosis of endometriosis. Studies in which endometriosis was diagnosed surgically and/or by histopathology or by the means of ultrasonography or magnetic resonance imaging were included in this review. Surgical visualization and histopathology are the gold-standard for diagnosis, while

			ultrasonography and magnetic resonance imaging have decent sensitivity in diagnosing endometriosis.
Risk of bias in individual studies	14	Describe anticipated methods for assessing risk of bias of individual studies, including whether this will be done at the outcome or study level, or both; state how this information will be used in data synthesis	The Risk of Bias Assessment is described on page 9 in the main manuscript. Table-6 of the Supplemental Materials gives the risk of bias assessment ratings.
Data synthesis	15a	Describe criteria under which study data will be quantitatively synthesised	Not applicable
	15b	If data are appropriate for quantitative synthesis, describe planned summary measures, methods of handling data and methods of combining data from studies, including any planned exploration of consistency (such as I^2, Kendall's τ)	Not applicable
	15c	Describe any proposed additional analyses (such as sensitivity or subgroup analyses, meta-regression)	Not applicable
	15d	If quantitative synthesis is not appropriate, describe the type of summary planned	Effect estimates, 95% confidence interval, and an assessment of confounding variables are used to review the studies.
Meta-bias(es)	16	Specify any planned assessment of meta-bias(es) (such as publication bias across studies, selective reporting within studies)	Not applicable
Confidence in cumulative evidence	17	Describe how the strength of the body of evidence will be assessed (such as GRADE)	The quality assessment of individual studies was carried out using the New Castle Ottawa Checklist for case-control and cohort studies. This is described on page 8 of the main manuscript and in Table-4 and Table-5 of the Supplemental Materials. The NTP / OHAT guidelines for Human and Animal Studies was used to evaluate the body of evidence. This is described on page 9 of the main manuscript under Risk of Bias Assessment and in Table-6 of Supplemental Materials.

Table 3 - Details of search terms

Sr. No	Category	Search Terms
1	Clinical condition	Endometriosis OR Adenomyosis OR Endometrio* OR Chocolate cyst
2	EDC types	Endocrine disrupting chemical OR endocrine disruptors OR trace element OR metal OR organochlorine, OR polychlorinated biphenyl OR polybrominated diphenyl ether OR dioxin OR phthalate OR bisphenol A OR OCP OR PCB OR PBB OR EDC OR BPA
3	Study Design	cohort OR follow up OR longitudinal OR prospective OR case-control OR study OR studies OR stud*

Table 4: QUALITY ASSESSMENT FOR CASE-CONTROL STUDIES

Case- Control Study	selection				comparability		exposure			overall
	Case definition	Case representativeness	Control selection	Control definition	Main confounder	Other factors	Ascertainment of exposure	Same method for case and control	Non-response rate	
1. Upson et al, **A population-based case–control study of urinary bisphenol A concentrations and risk of endometriosis**	*	*	*	*	*	*	*	*	*	9
2. Huang et al, **Association between phthalate exposure and glutathione S-transferase M1 polymorphism in adenomyosis, leiomyoma and endometriosis**	*	-	-	*	*	*	*	*	*	7
3. BS Reddy, **Association of phthalate esters with endometriosis in Indian women**	*	-	-	*	*	_	*	*	_	5

4. Heilier et al, **Assessment of cadmium impregnation in women suffering from endometriosis: a preliminary study**	*	*	-	*	*	*	*	*	-	7
5. Hiroaki Itoh, **a case-control study of the association between urinary cadmium concentration and endometriosis in infertile Japanese women**	*	-	-	-	*	*	*	*	*	6
6. Elena De Felip , **Dioxin-like compounds and endometriosis: a study on Italian and Belgian women of reproductive age**	*	-	-	*	*	-	*	*	*	5
7. Cai LY et al, **Dioxins in ascites and serum of women with endometriosis: a pilot study**	*	-	-	*	*	*	*	*	*	7
8. Porpora et al, **Endometriosis and OCEP: A case control study on Italian women of reproductive age**	*	-	-	*	*	*	*	*	*	7
9. Silva et al**, Elevated levels of whole blood nickel in a group of Sri Lankan women**	*	- hospital	- hospital	*	* age matched	-	*	*	*	6

with endometriosis: a case control study										
10. JF Heilier et al, **Environmental and host-associated risk factors in endometriosis and deep endometriotic nodules: A matched case–control study**	*	-	-	-	-	-	*	*	_	3
11. Heilier et al, **Cadmium, Lead and Endometriosis**	*	-	-	*	-	-	*	*	*	5
12. Roya Rozati et al, **Evaluation of the Phthalate Esters in South Indian Women with Endometriosis**	*	-	-	*	*	-	*	*	-	5
13. Trabert et al , **Non–Dioxin-Like Polychlorinated Biphenyls and Risk of Endometriosis**	*	*	*	*	*	*	*	*	*	9
14. Upson et al , **Phthalates and risk of endometriosis**	*	*	*	*	*	*	*	*	*	9
15. Germaine Lebel et al, **Organochlorine exposure and risk of endometriosis**	*	-	-	*	*	*	*	*	*	7
16. Niskar AS, **Serum Dioxin, Polychlorinated Biphenyls and Endometriosis: A case**	*	-	-	*	*	*	*	*	*	7

control study in Atlanta										
17. A.Pauwels **,The risk of endometriosis and exposure to dioxins and PCB: a case control study of infertile women**	*	-	-	*	*	*	*	*	*	7
18. Reddy et al, **High plasma concentrations of polychlorinated biphenyls and phthalate esters in women with endometriosis: a prospective case control study**	*	-	-	*	-	-	*	*	-	4
19. Porpora et al, **Increased levels of polychlorobiphenyls in Italian women with endometriosis**	*	-	-	*	*	*	*	*	*	7
20. Heilier JF et al, **Increased dioxin-like compounds in the serum of women with peritoneal endometriosis and deep endometriotic (adenomyotic) nodules**	*	-	-	-	*	*	*	*	*	6

21. MA Martinez et al, **Increased levels of dioxin-like substances in adipose tissue in patients with deep infiltrating endometriosis**	*	-	-	*	*	*	*	*	*	7
22. Simsa et al, **Increased exposure to dioxin-like compounds is associated with endometriosis in a case–control study in women**	*	-	-	*	*	-	*	*	*	6
23. Kim SH et al, **Increased plasma levels of phthalate ester in women with advanced-stage endometriosis: a prospective case-control study**	*	-	-	*	*	*	*	*	*	7
24. Schiattarella A et al, **Plasma and urinary levels of lead and cadmium in patients with endometriosis**	*	-	-	*	-	-	*	*	-	4
25. Rashidi B.H e al, **A case–control study of bisphenol A and endometrioma among subgroup of Iranian women**	*	-	-	-	*	*	*	*	*	6

26. Porpora M. G et al, **Endometriosis and Organochlorinated Environmental Pollutants: A case-control Study on Italian Womenof Reproductive Age**	*	-	-	*	*	*	*	*	*	7
27. Ploteau S et al, **Associations between internal exposure levels of persistent organic pollutants in adipose tissue and deep infiltrating endometriosis with or without concurrent ovarian endometrioma**	*	-	-	*	*	*	*	*	-	6
28. Simonelli A et al, **Environmental and occupational exposure to bisphenol A and endometriosis: urinary and peritoneal fluid concentration levels**	*	-	-	*	-	-	*	*	*	5

Table 5: QUALITY ASSESSMENT FOR COHORT STUDIES										
Cohort Study	Selection				comparability		outcome			overall
	Exposed cohort representativeness	non exposed cohort selection	Ascertainment of exposure	outcome of interest was not present at the start of study	Main confounder	Other factors	Assessment of outcome	Was follow-up long enough for outcomes to occur	Adequacy of follow up of cohorts	
1. Louis GM Buck et al, **Environmental PCB exposure and risk of endometriosis**	*	*	*	*	*	*	*	-	*	8
2. Louis GM Buck et al, **Bisphenol A and phthalates and endometriosis: the Endometriosis: Natural History, Diagnosis and Outcomes Study**	*	*	*	*	*	*	*	-	*	8
3. Louis GM Buck et al, **Persistent Lipophilic Environmental Chemicals and Endometriosis: The ENDO Study**	*	*	*	*	*	*	-	-	*	7
4. Pollack AZ et al, **Trace elements and endometriosis: The ENDO Study**	*	*	*	*	*	*	-	-	*	7

5. Cooney MA et al, **Organochlorine pesticides and endometriosis**	*	*	*	*	*	*	*	-	-	7

TABLE 6: RISK OF BIAS ASSESSMENT RATINGS

Bias Domains and Questions / Name of the study	**Upson et al, 2014**	**Simonelli et al 2016**	**Rashidi et al 2017**	**Buck Louis et al 2013**	**Upson et al 2013**	**Rozati et al 2008**	**Reddy at el 2006**	**Kim et al 2011**	**Huang et al 2010**	**Pollack et al 2013**	**Silva et al 2013**	**Itoh et al 2008**	**Heilier et al 2004**	**Heilier et al 2006**	**Pauwels et al 2001**
Selection Bias															
Did selection of study participants result in appropriate comparison groups?	(++)	(+)	(+)	(++)	(++)	(++)	(++)	(++)	(++)	(++)	(++)	(-)	(-)	NR	(-)
Confounding Bias															
Did the study design or analysis account for important confounding and modifying variables? (Key Domain)	(+)	NR	(+)	(+)	(+)	(+)	(+)	(+)	(+)	(+)	(-)	(+)	(+)	(-)	(+)
Attrition / Exclusion Bias															
Were outcome data complete without attrition or exclusion from analysis?	(+)	(-)	(-)	(+)	(+)	(-)	(-)	(+)	(+)	(++)	(+)	(+)	(++)	NR	(+)
Detection Bias															
Can we be confident in the exposure characterization? (Key Domain)	(++)	(++)	(++)	(++)	(++)	(++)	(++)	(++)	(++)	(++)	(++)	(++)	(++)	(++)	(++)
Can we be confident in the outcome assessment? (Key Domain)	(++)	(++)	(+)	(++)	(++)	(++)	(++)	(++)	(++)	(+)	(++)	(++)	(--)	(+)	(+)
Selective Reporting Bias															
Were all measured outcomes reported?	(++)	(++)	(++)	(++)	(++)	(++)	(++)	(++)	(++)	(++)	(++)	(++)	(++)	(++)	(++)

Other Sources of Bias															
Were there no other potential threats to internal validity (e.g., statistical methods were appropriate and researchers adhered to the study protocol)?	(+)	(+)	(+)	(+)	(+)	(+)	(+)	(+)	(+)	(++)	(+)	(+)	(+)	(+)	(+)
Conflict of Interest	(+)	(+)	(+)	(+)	(+)	(+)	(+)	(+)	(+)	(+)	(+)	(+)	(+)	(+)	(+)
Summary of Tiered Classification	Tier 1	Tier 2	Tier 2	Tier 1	Tier 1	Tier 2	Tier 2	Tier 1	Tier 1	Tier 1	Tier 2	Tier 2	Tier 2	Tier 2	Tier 2

[Key: (++) Definitely low risk of bias, (+) Probably low risk of bias, (-) Probably high risk of bias, (--) Definitely high risk of bias, NR Reasons not recorded]

TABLE 6 CONTINUED: RISK OF BIAS ASSESSMENT RATINGS

Bias Domains and Questions / Name of the study	**Niskar et al 2009**	**Martinez et al 2015**	**Simsa et al 2010**	**Heilier et al 2005**	**Cai et al 2011**	**De Felip et al 2004**	**Ploteau et al 2017**	**Lebel et al 1998**	**Porpora et al 2009**	**Buck Louis et al 2012**	**Cooney et al 2010**	**Louis et al 2005**	**Trabert et al 2010**	**Porpora et al 2006**
Selection Bias														
Did selection of study participants result in appropriate comparison groups?	NR	(++)	NR	NR	NR	(--)	(+)	(+)	(+)	(++)	NR	(+)	(++)	(NR)
Confounding Bias														
Did the study design or analysis account for important confounding and modifying variables? (Key Domain)	(+)	(+)	(+)	(+)	(+)	(-)	(+)	(+)	(+)	(+)	(-)	(+)	(+)	(+)
Attrition / Exclusion Bias														
Were outcome data complete without attrition or exclusion from analysis?	(+)	(+)	(+)	(+)	(+)	(+)	(+)	(+)	(+)	(+)	(+)	(+)	(+)	(+)
Detection Bias														
Can we be confident in the exposure characterization? (Key Domain)	(++)	(++)	(++)	(++)	(++)	(++)	(++)	(++)	(++)	(++)	(++)	(++)	(++)	(++)
Can we be confident in the outcome assessment? (Key Domain)	NR	(+)	(++)	(-)	(+)	(++)	(+)	(+)	(+)	(+)	(+)	(+)	(+)	(++)
Selective Reporting Bias														

Were all measured outcomes reported?	(++)	(++)	(++)	(++)	(++)	(++)	(++)	(++)	(++)	(++)	(++)	(++)	(++)	(++)
Other Sources of Bias														
Were there no other potential threats to internal validity (e.g., statistical methods were appropriate and researchers adhered to the study protocol)?	(+)	(+)	(+)	(+)	(+)	(-)	(+)	(+)	(+)	(+)	(+)	(+)	(+)	(+)
Conflict of Interest	(+)	(++)	(+)	(+)	(+)	(+)	(+)	(+)	(+)	(+)	(+)	(+)	(+)	(+)
Summary of Tiered Classification	**Tier 2**	Tier 1	Tier 1	**Tier 2**	**Tier 2**	**Tier 2**	Tier 1	Tier 1	Tier 1	Tier 1	**Tier 2**	Tier 1	Tier 1	**Tier 2**

[Key: (++) Definitely low risk of bias, (+) Probably low risk of bias, (-) Probably high risk of bias, (--) Definitely high risk of bias, NR Reasons not recorded]

Table 7: Summary of studies included

Study / Reference no.	Location	Study design	N	Age Mean (+ SD) Or Range	Outcome Assessment	Confounders	Exposure Assessment	
			Ca / Co	Ca / Co	Diagnosis		Chemical	Specimen
Upson et al, 2014 / [13]	USA	Population based case control	144 / 291	17–49 years	Ca: Sur Co: MRI	Age, reference year, natural logarithm transformed urinary creatinine, education, alcohol consumption, smoking status and race	BPA	Urine
Simonelli et al 2016 / [20]	Italy	Case control	60 / 68	<30 - ≥40 years	Ca: Sur Co: Unclear	Not explicitly stated	BPA	Urine and peritoneal fluid
Rashidi et al 2017 / [19]	Iran	Case control	50 / 50	Ca: 32.22 +5.34 Co: 33.20 +5.46	Ca: Sur Co: USG	Age, BMI, parity and education	BPA	Urine
Buck Louis et al 2013 / [21]	USA	Matched cohort	Operative Cohort 190 / 283 Population Cohort 14 / 127	Operative Cohort Ca: 32.0 +6.8 Co: 33.6 +7.1 Population Cohort Ca: 33.1 +8.3 Co: 32.1 +7.8	Operative Cohort: Sur Population Cohort: MRI	Age, BMI, urinary creatinine	BPA and PE	Urine
Upson et al 2013 / [31]	USA	Population based case control	92 / 195	18–49 years	Ca: Sur Co: Unclear	Age, reference year, natural logarithm transformed urinary creatinine, education, alcohol consumption and smoking status	PE	Urine
Rozati et al 2008 / [30]	India	Case control	99 / 135	Ca: 25.6 +4.2 Co: 26.4 +4.7	Ca; Sur Co: Sur	BMI, age at menarche, duration of infertility and clinical symptoms	PE	Blood
Reddy at el 2006 / [29]	India	Case control	49 / 59	Ca: 26.2 +4.2 Co I: 27.4 +4.7 Co II: 27.1 +3.4	Ca: Sur Co: Sur	Not explicitly stated	PE	Blood

Kim et al 2011 / [32]	Korea	Prospective case control	97 / 169	Ca: 34.8 +0.7 Co: 34.9 +0.5	Ca: Sur Co: Sur	Number of deliveries, BMI and plasma MEPH levels and DEHP levels	PE	Blood
Huang et al 2016 / [28]	Taiwan	Case control	80 / 29	Ca: EN: 34.3 +7.5 LEI: 41.1 +6.8 AD: 43.2 +6.5 Co: 36.2 +9.0	Ca: Sur Co: Sur	GSTM1 polymorphism and BMI for EN GSTM1 genotype and age for AD and LEI. Adjusted for creatinine	PE	Urine and serum (peripheral lymphocytes)
Pollack et al 2013 / [38]	USA	Matched cohort	Operative Cohort 190 / 283 Population Cohort 14 / 113	Operative Cohort Ca: 32.0 +6.8 Co: 33.6 +7.1 Population Cohort Ca: 33.1 +8.3 Co: 32.1 +7.8	Operative Cohort: Sur Population Cohort: MRI	Age, BMI, smoking site, race, vitamin use and creatinine	Cd, Pb, Hg, and trace elements	Urine and blood
Silva et al 2013 / [40]	Sri-Lanka	Case control	50 / 50	Ca: 33.0 +5.4 Co: 32.7 +5.4	Ca: Sur Co: Sur	Age	Cd, Ni, Pb	Blood
Itoh et al 2008 / [47]	Japan	Case control	54 / 74	20- 45 years	Ca: Sur Co: Sur	Menstrual regularity, BMI, age at menarche, alcohol consumption and smoking status	Cd	Urine
Heilier et al 2004 / [46]	Belgium	Prospective case control	59 / 21	Ca: EN: 30.7 +6.9 DEN: 29.7 +6.3 Co: 31.4 +5.5	Ca: MRI / Rectal USG / CA 125 Co: Asymptomatic	Age, smoking	Cd	Urine and blood
Heilier et al 2006 / [45]	Belgium	Case control	81 / 25	Ca: ENDO: 31.5 +6.5 DEN: 29.3 +6.8 BD: 29.4 +4.8 Co: 30.5 +5.6	Ca: Sur Co: Vaginal echography and CA 125	Smoking	Cd, Pb	Urine and blood
Pauwels et al 2001 / [71]	Belgium	Case control	42 / 27	Ca: 25-42 years Co: 24-41 years	Ca: Sur Co: Sur	Age, BMI, smoking status, alcohol consumption, caffeine intake and ovulatory dysfunction	Dioxins, DL Co-planar PCB, Non-Co-planar PCB	Blood
Niskar et al 2009 / [70]	USA	Case control	60 / 64	20-45 years	Ca: Sur Co: 48% by Sur	Lipid	PCDD, PCDF, PCB, p,p'-DDE	Blood
Martinez et al 2015 / [66]	Spain	Case control	30 / 30	Ca: 32.5 +3.8 Co: 31.1 +4.9	Ca: Sur Co: Sur	Age, smoking and BMI.	PCDD, PCDF, PCB	Adipose tissue
Simsa et al 2010 / [68]	Belgium	Case Control	96 / 105	Ca: 24 - 42 years Co: 20 - 46 years	Ca: Sur Co: Sur	Age	DLC	Blood

[N: number of participants, Ca: cases, Co: controls, EN: endometriosis, LEI: leiomyoma, AD: adenomyosis, DIE: deep infiltrating endometriosis, DEN: deep endometriotic nodules, ENDO: peritoneal endometriosis, OvE: ovarian endometrioma, BD: Both deep endometriotic nodules (DEN) and peritoneal endometriosis (ENDO), Sur: surgery, MRI: magnetic resonance imaging, USG: ultrasonography, HP: histopathology]

Heilier et al 2005 / [65]	Belgium	Case control	50 / 21	Ca: ENDO: 31.8 +6.5 DEN: 30.8 +7.0 Co: 31.1 +6.0	Ca: Sur, HP, MRI Co: Vaginal echography and CA 125	Age, BMI	PCDD, PCDF, DL-PCB	Blood
Cai et al 2011 / [64]	Japan	Pilot case control	10 / 7	Ca: 33.5 +3.6 Co: 36.4 +5.9	Ca: Sur Co: Sur	Age, parity, smoking and BMI	PCDD, PCDF, PCB	Serum and peritoneal fluid
De Felip et al 2004 / [69]	Belgium and Italy	Pilot case control	23 / 17	18- 40 years	Ca: Sur Co: Sur	Parity and diet	PCDD, PCDF, PCB	Blood
Ploteau et al 2017 / [67]	France	Case control	55 / 44	Ca: DIE only: 32.7 +6.4 DIE+OvE: 36.7 +4.7 Co: 32.7 +6.4	Ca: Sur Co: Asymptomatic	Age and BMI	PBDE, PBB, PCDD, DL-PCB, NDL-PCB, OCP	Serum and adipose tissue
Lebel et al 1998 / [82]	Canada	Case control	86 / 70	Ca: 32.2 +5.9 Co: 33.2 +7.0	Ca: Sur Co: Sur	Age, BMI, parity and indication for laparoscopy	PCB, OCP	Blood
Porpora et al 2009 / [72]	Italy	Case control	80 / 78	Ca: 31.6 +6.0 Co: 29.5 +6.1	Ca: Sur Co: Sur	Age, BMI, smoking habits and weight loss	HCB, p.p'-DDE, PCDD, PCDF, DL-PCB, NDL-PCB	Blood
Buck Louis et al 2012 / [59]	USA	Matched cohort	Operative Cohort 190 / 283 Population Cohort 14 / 113	Operative Cohort Ca: 32.0 +6.8 Co: 33.6 +7.1 Population Cohort Ca: 33.1 +8.3 Co: 32.1 +7.8	Operative Cohort: Sur Population Cohort: MRI	Age, BMI, breastfeeding, serum cotinine, lipid	PCB, PBDE, HCB, HCH, beta-HCH, gamma-HCH	Blood, urine and omental fat
Cooney et al 2010 / [88]	USA	Cohort	29 / 51	Ca: 32 +4 Co: 31 +5	Ca: Sur Co: Not explicitly stated	Lipid and smoking	OCP	Blood
Louis et al 2005 / [79]	USA	Cohort	32 / 54	Ca: 32.7 +4.44 Co: 31.6 +4.96	Ca: Sur Co: Sur	Gravidity, parity, current smoking status and serum lipids	PCB	Blood
Trabert et al 2010 / [81]	USA	Case control	251 / 538	18-49 years	Ca: Sur Co: Asymptomatic	Alcohol intake, income, age, lipid and quartile of serum p-p'-DDE	PCB	Blood
Porpora et al 2006 / [80]	Italy	Case control	40 / 40	22 – 40 years	Ca: Sur Co: Sur	Age and smoking.	PCB	Blood

[N: number of participants, Ca: cases, Co: controls, EN: endometriosis, LEI: leiomyoma, AD: adenomyosis, DIE: deep infiltrating endometriosis, DEN: deep endometriotic nodules, ENDO: peritoneal endometriosis, OvE: ovarian endometrioma, BD: Both deep endometriotic nodules (DEN) and peritoneal endometriosis (ENDO), Sur: surgery, MRI: magnetic resonance imaging, USG: ultrasonography, HP: histopathology]

Table 8: Results showing the effect estimates for urinary bisphenol-A (BPA) and endometriosis

Author and Reference	Effect Estimate	
Upson et al, 2014 / [13]	Total BPA in microgram / L	OR[a] for NPE[b] / 95% CI[c]
	</= 0.364	1.0
	>0.364 – 0.863	3.3 (1.3, 8.3)
	>0.863 – 2.01	2.9 (1.1, 7.6)
	>2.01	1.7 (0.6, 1.1)
Buck Louis et al, 2013 / [21]	OR for operative cohort 0.96 95% CI (0.79, 1.19) OR for population cohort 1.68 95% CI (0.96, 2.92)	
Rashidi et al, 2017 / [19]	OR = 1.74 (95% CI 1.40 -2.16) P <0.001	
Simonelli et, al, 2016 / [20]	The relationship between endometriosis and the expected occupational exposure shows statistically significant dependence ($\chi 2$ = 6.830, p = 0.01) and an OR of 0.38 (95 % CI, 95 % CI 0.18–0.79), with a higher percentage of patients in the unexpected exposed workers.	

Key: a = odds ratio, b = Non-ovarian pelvic endometriosis, c = 95% confidence interval

Table 9: Results showing the effect estimates for phthalate esters (PEs) and endometriosis

Author and Reference	Effect Estimate
Buck Louis et al, 2013 / [21]	Limit of quantitation measured in ng/mL
	mBP (Operative cohort)- OR[a] = 1.11 95% CI[b] (0.86, 1.43) (Population cohort)- OR = 2.62 95% CI (1.14, 6.05)
	mCMHP (Operative cohort)- OR = 0.98 95% CI (0.77, 1.26) (Population cohort)- OR = 2.65 95% CI (1.33, 5.31)
	mOP (Operative cohort)- OR = 1.06 95% CI (0.87, 1.29). Sensitivity analysis OR = 1.38 95% CI (1.10, 1.72) (Population cohort)- OR = 0.84 95% CI (0.40, 1.78)
	mEHP (Operative cohort)- OR = 1.20 95% CI (0.97, 1.49). Sensitivity analysis OR = 1.35 95% CI (1.03, 1.78) (Population cohort)- OR = 2.59 95% CI (1.17, 5.75)
	Sum of DEHP metabolites OR = 2.81 95% CI (1.42,5.56)
Upson K et al, 2013 / [31]	Phthalate ester quartiles in ng/mL
	Sum of DEHP P = 0.053
	</= 0.06 OR = 1.0
	>0.06-0.18 OR = 1.1 95% CI (0.5, 2.5)
	>0.18-0.50 OR= 0.7 95% CI (0.3, 1.8)
	>0.50 OR = 0.4 95% CI (0.1, 1.13)
	Sum of BzBP P = 0.559
	</= 0.03 OR = 1.0
	>0.03 – 0.07 OR = 1.7 95% CI (0.7, 4.2)
	>0.07- 0.16 OR = 0.9 95% CI (0.3, 2.8)
	>0.16 OR = 1.2 95%CI (0.3, 4.2)
	Sum of DBP P = 0.986:
	</= 0.03 OR = 1.0
	>0.03 – 0.06 OR = 1.3 95% CI (0.5, 3.1)
	>0.06 – 0.12 OR = 1.3 95% CI (0.5, 3.5)
	>0.12 OR = 1.3 95% CI (0.4, 4.3)
	MEHP OR = 0.3 95% CI (0.1, 0.7) P=0.012

Rosati et al, 2008 / [30]	Mean in micrograms/ml (SD), Control vs cases t-value; p-value:
	DMP: Cases: 0.03 (0.72), Controls: 0.04 (0.10), t: 5.13; p <0.0001
	DEP: Cases: 0.89 (0.84), Controls: 0.9 (0.18), t: 5.13; p <0.0001
	DnBP: Cases: 0.98 (0.96), Controls: 0.55 (0.89), t: -9.52; p <0.0001
	BBP: Cases: 3.32 (2.17), Controls: 0.15 (0.21), t: 5.22; p <0.0001
	DEHP: Cases: 2.15 (1.99), Controls: 0.11 (0.22), t: 0.33; p=0.0014
	The correlation between PEs concentration (control group) and endometriosis different severity was strong and significant: DMP: r=+0.03, p<0.0001; DnBP r=+0.39, p<0.0001; BBP: r=+0.89, p<0.0001; DnOP: r=+0.66, p<0.0001 and BEHP: r=+0.33, p<0.0014)
Reddy et al, 2006 / [29]	Mean in micrograms/ml (SD), Control vs cases t-value; p-value:
	DnBP: Cases: 0.44 (0.41), Controls I: 0.08 (0.14), Control II: 0.15 (0.21), $t_{(Control\ I\ vs\ cases)}$: 5.13; p <0.0001, $t_{(Control\ II\ vs\ cases)}$: 3.01, p=0.004
	BBP: Cases: 0.66 (0.61), Controls I: 0.12 (0.20), Control II: 0.11 (0.22), $t_{(Control\ I\ vs\ cases)}$: 5.13; p <0.0001, $t_{(Control\ II\ vs\ cases)}$: 3.94, p=0.0002
	DnOP: Cases: 3.32 (2.17), Controls I: 0, Control II: 0,
	DEHP: Cases: 2.44 (2.17), Controls I: 0.50 (0.80), Control II: 0.45 (0.68), $t_{(Control\ I\ vs\ cases)}$: 5.22, p<0.0001, $t_{(Control\ II\ vs\ cases)}$: 4.10, p=0.0001
	The correlation between PEs concentration (control group) and endometriosis different severity was strong and significant: DnBP = r = +0.73; P <0.0001, BBP = r = +0.78; P <0.0001, DnOP = r =+0.73; P <0.000, DEHP = r = +0.44 P <0.0014
Kim et al, 2011 / [32]	Mean concentrations in plasma in ng / mL 1. DEHP: Cases: 179.7 Controls: 92.5 OR = 1.001 95% CI (1.000, 1.002) P= 0.161
	2.MEPH: Cases: 17.4 Controls: 12.4 OR =1.020 95% CI (1.003, 1.038) P=0.020
Huang et al, 2010 / [28]	Patients with GSTM1 null genotype increased risk for AD[c] (OR ¼ 10.4;95% CI, 1.26–85.0) and LEI[d] (OR ¼ 5.93; 95% CI, 1.10–31.9) after adjustment for age, compared with those with GSTM1[e] wild-typeand low urinary level of SMEHP.

Key: a = odds ratio, b= 95% CI = 95% confidence interval, c = Adenomyosis, d = Leiomyoma, e = Glutathione S-transferase M1

Table 10: Results showing the effect estimates for metals and endometriosis

Author and Reference	Effect Estimate
Pollack et al, 2013 / [38]	Blood Cd: (Operative cohort)- $OR^{a}_{(3rd\ vs\ 1st\ tertile)}$ = 0.55 95% CI[b] (0.31, 0.98) and $OR_{(2nd\ vs\ 1st\ tertile)}$ = 0.74 95% CI (0.46, 1.21) (Population cohort)- $OR_{(3rd\ vs\ 1st\ tertile)}$ = 0.14 95% CI (0.01, 1.58) and $OR_{(2nd\ vs\ 1st\ tertile)}$ = 1.73 95% CI (0.45, 6.67) Urinary Cd[c]: (Operative cohort)- $OR_{(3rd\ vs\ 1st\ tertile)}$ = 0.74 95% CI (0.38, 1.43) and $OR_{(2nd\ vs\ 1st\ tertile)}$ = 1.19 95% CI (0.70, 2.02) (Population cohort)- $OR_{(3rd\ vs\ 1st\ tertile)}$ = 0.22 95% CI (0.01, 4.13) and $OR_{(2nd\ vs\ 1st\ tertile)}$ = 1.18 95% CI (0.20, 7.17)
	Blood Cr: NA[d]

	Urinary Cr[e]: (Operative cohort)- $OR_{(3rd\ vs\ 1st\ tertile)}$ = 0.91 95% CI (0.55, 1.51) and $OR_{(2nd\ vs\ 1st\ tertile)}$ = 1.97 95% CI (1.21, 3.19) (Population cohort)- $OR_{(3rd\ vs\ 1st\ tertile)}$ = 1.29 95% CI (0.27, 6.23) and $OR_{(2nd\ vs\ 1st\ tertile)}$ = 0.94 95% CI (0.20, 4.44)
	Blood Cu: NA Urinary Cu[f]: (Operative cohort)- $OR_{(3rd\ vs\ 1st\ tertile)}$ = 2.66 95% CI (1.26, 5.64) and $OR_{(2nd\ vs\ 1st\ tertile)}$ = 1.30 95% CI (0.76, 2.25) (Population cohort)- $OR_{(3rd\ vs\ 1st\ tertile)}$ = 1.59 95% CI (0.13, 19.1) and $OR_{(2nd\ vs\ 1st\ tertile)}$ = 1.59 95% CI (0.31, 8.14)
	Blood Pb[g]: (Operative cohort)- $OR_{(3rd\ vs\ 1st\ tertile)}$ = 0.84 95% CI (0.50, 1.41) and $OR_{(2nd\ vs\ 1st\ tertile)}$ = 0.73 95% CI (0.45, 1.17) (Population cohort)- $OR_{(3rd\ vs\ 1st\ tertile)}$ = 1.70 95% CI (0.39, 7.38) and $OR_{(2nd\ vs\ 1st\ tertile)}$ = 0.92 95% CI (0.20, 4.16) Urinary Pb: (Operative cohort)- $OR_{(3rd\ vs\ 1st\ tertile)}$ = 1.36 95% CI (0.80, 2.31) and $OR_{(2nd\ vs\ 1st\ tertile)}$ = 1.33 95% CI (0.80, 2.21) (Population cohort)- $OR_{(3rd\ vs\ 1st\ tertile)}$ = 1.94 95% CI (0.35, 10.7) and $OR_{(2nd\ vs\ 1st\ tertile)}$ = 1.32 95% CI (0.29, 6.09)
	Blood Hg[h]: (Operative cohort)- $OR_{(3rd\ vs\ 1st\ tertile)}$ = 1.00 95% CI (0.60, 1.67) and $OR_{(2nd\ vs\ 1st\ tertile)}$ = 1.08 95% CI (0.67, 1.74) (Population cohort)- $OR_{(3rd\ vs\ 1st\ tertile)}$ = 0.81 95% CI (0.19, 3.39) and $OR_{(2nd\ vs\ 1st\ tertile)}$ = 0.69 95% CI (0.16, 2.90) Urinary Hg: (Operative cohort)- $OR_{(3rd\ vs\ 1st\ tertile)}$ = 0.78 95% CI (0.45, 1.36) and $OR_{(2nd\ vs\ 1st\ tertile)}$ = 1.00 95% CI (0.60, 1.65) (Population cohort)- $OR_{(3rd\ vs\ 1st\ tertile)}$ = 0.14 95% CI (0.01, 1.52) and $OR_{(2nd\ vs\ 1st\ tertile)}$ = 0.19 95% CI (0.02, 1.87)
Silva et al, 2013 / [40]	Whole blood concentrations in Geometric Means and 95% CI; unit of measurement: micrograms/L (Cases)
	Ni[i]: 26 95% CI (1.9, 3.3) P = 0.016
	Pb: 11.0 95% CI (8.6, 13.1) P = 0.389
	Cd: 0.7 95% CI (0.7, 0.9) P = 0.423
	Whole blood concentrations in Geometric Means and 95% CI; unit of measurement: micrograms/L (Controls):
	Ni: 0.8 95% CI (0.7, 0.9) P = 0.016
	Pb: 6.9 95% CI (5.7, 8.0) P = 0.389
	Cd: 0.8 95% CI(0.6, 1.0) P = 0.423
Itoh et al, 2008 / [47]	Urinary Cd concentration tertile cut-off points: P for trend – 0.79
	0.184 – 0.389 OR = 1.00 (Referent)
	0.393 – 0.699 OR = 1.69 95% CI (0.64, 4.44)
	0.707 – 7.92 OR = 0.86 95% CI (0.30, 2.49)
Heilier et al, 2004 / [46]	Geometric Means of Cd concentration in urine in micrograms / gram of creatinine (geometric SD), p-value:
	PE[j]: 0.25 (1.50), DEN[k]: 0.29 (1.76), Controls: 0.26 (1.46), p-value not given, however the author stated that no significant difference in means
	Geometric Means of Cd concentration in blood in micrograms / litre of blood (geometric SD):
	PE: 0.1 (1.5), DEN: 0.1 (1.4), Controls 0.1 (1.6), P =0.80
Heilier et al, 2006 / [45]	Blood concentrations in micrograms / litre, and urine concentration in micrograms / gram creatinine are presented below as geometric means (geometric SD):
	Blood Pb: controls= 22 (1.5); cases= 17 (1.7)
	Blood Cd: controls= 0.4 (2.1); cases= 0.4 (1.9);

	Urinary Cd: controls= 0.3 (1.9); cases= 0.3 (2.3);
	Analysis of variance: Pb (Blood) – 0.10; Cd (Blood)-0.51; Cd (Urine) – 0.78

Key: a = odds ratio, b = 95% confidence interval, c = Cadmium, d = Not available, e = Chromium, f = Copper, g = Lead, h = Mercury, i = Nickel, j = peritoneal endometriosis k = deep endometriotic nodule

Table 11: Results showing the effect estimates dioxins, dioxin-like compounds (DLCs), non-dioxin like polychlorinated biphenyls (NDL-PCBs), polychlorinated biphenyls (PCBs), other persistent organochlorine pollutants (POPs) and endometriosis

Author and Reference	Effect Estimate
Pauwels et al, 2001 / [71]	CALUX based TEQ[a] values for dioxins and dioxin co-planar PCBs (median values)
	Cases: 29 pg TEQ / g lipid; Controls: 27 pg TEQ / g lipid. OR[b] = 4.5 95% CI[c] (0.48, 43.62)
Niskar et al, 2009 / [70]	Lipid adjusted PCB OR = 1.04 95%CI (0.44, 2.44)
	Non-Lipid adjusted PCB OR = 1.02 95%CI (0.43, 2.41)
	Unadjusted TEQ OR = 1.02 95%CI (0.95, 1.08)
Martinez et al, 2015 [66]	Median concentration in adipose tissue in pg / gram lipid:
	Dioxins + Furans: Cases: 6.90 Controls: 6.10 OR = 1.72 95%CI 1.16-3.15 P<0.05
	PCBs: Cases: 4.64 Controls: 6.10 OR = 1.97 95%CI 1.36 - 2.77 P =0.01
	TEQ values of 2,3,7,8-TCDD and 1,2,3,7,8-PeCDD and 2,3,4,7,8-PeCDF were also statistically significantly higher in cases than in controls (P<0.001 for each)
Simsa et al, 2010 / [68]	Endometriosis in women with higher exposure (>75 centile - > 25 pg CALUX TEQ/ g / lipd) versus women with lower exposure (<25 pg CALUX TEQ / g/ lipid): OR = 2.44 (95%CI 1.05-5.70 P=0.04) in women with higher compared with lower DLC versus controls.
	OR = 3.01 (95%CI 1.06 -9.04 P=0.03) in women with moderate to severe endometriosis with higher concentration of DLC as compared to women with lower concentration of DLC.
Heilier et al, 2005 / [65]	Log Regression was significant for DEN[d] – OR = 3.3 95% CI (1.4, 7.6) for an increment of 10 pg in total TEQ levels / g lipid.
	An increased risk was also found for PE[e] – OR = 1.9 95% CI (0.9, 3.8) for total TEQ levels and also for dioxins alone at OR 3.2 95% CI (1.0, 9.9)
Cai et al, 2011 / [64]	OR =2.5 95%CI (1.17, 5.34)
De Felip et al, 2004 / [69]	Analyte concentrations in pgTE/g lb (picogram Toxic Equivalent/gram of lipid base, based on WHO-Toxic Equivalent) in
	Italian Cases:
	PCDD+PCDF: 10 - 11
	Non-ortho PCB: 3.9 - 4.0
	Mono-ortho PCB: 3.8 - 4.3
	Belgian Cases:
	PCDD+PCDF: 18- 27
	Non-ortho PCB: 8.6 - 11
	Mono-ortho PCB: 7.7 – 11
	Analyte concentrations in pgTE/g lb (picogram Toxic Equivalent/gram of lipid base, based on WHO-Toxic Equivalent) in
	Italian Controls:
	PCDD+PCDF: 8.9 +/- 1.3
	Non-ortho PCB: 3.92 +/- 0.58
	Mono-ortho PCB: 4.83 +/- 0.71

	Belgian Controls:
	PCDD+PCDF: 24.7 +/- 3.7
	Non-ortho PCB: 9.4 +/- 1.4
	Mono-ortho PCB: 10.4 +/- 1.5
Ploteau et al, 2017 / [67]	1.Association between the cases with DIE[f]+ OvE[g] for PBB 153 showed aOR[h] (95%CI) of 8.26 (2.27–44.41)1-SD increase 2. OCDF = adjusted ORs (95% CI) of 5.42 (2.73–12.85) per 1-SD increase
Louis et al, 2005 / [79]	Risk for anti-estrogenic PCB congeners and risk of endometriosis OR = 3.30 95%CI (0.87, 12. 46)
Trabert et al, 2010 / [81]	Sum of all PCB congeners: OR = 1.3 95%CI (0.8, 2.2)
	Sum of Estrogenic congeners: OR = 1.1 95%CI (0.8, 1.4)
Porpora et al, 2006 / [80]	Median concentration in ng / g lipid
	All PCBs:
	Cases: 410 Controls: 250 P = 0.0003
	Categories: 250-360 OR = 6.5 95%CI (1.5, 28)
	Categories: > 360 OR = 5.3 95% CI (1.3, 23)
	PCB-153
	Cases: 150 Controls: 95 P = 0.0004
	Categories: 93-130 OR = 10.0 95%CI (2.1, 48)
	Categories: > 130 OR = 9.1 95% CI (1.9, 43)
	PCB-180
	Cases: 65 Controls: 45 P = 0.0002
	Categories: 37-64 OR = 5.8 95%CI (1.4, 24)
	Categories: > 64 OR = 4.0 95% CI (1.0, 16)
Lebel et al, 1998 / [82]	Geometric means in micrograms / kg lipid:
	Sum of all PCB congeners:
	Cases: 123.5 95%CI (113.3, 134.7) Controls: 119.3 95%CI (108.9, 130.5)
Porpora et al, 2009 / [72]	**Total PCBs:**
	209-305: Cases: 41.25; Controls: 25.68 OR = 4.64 95%CI (1.93, 11.16)
	>/=306: Cases: 42.50; Controls: 22.90 OR = 5.63 95% CI(2.25, 14.70)
Buck Louis et al, 2012 / [59]	PCB-74 in fat (Operative cohort) – OR = 0.72 95%CI (0.55, 0.93) (Population cohort)- NA as no fat available
	PCB 156 in fat (Operative cohort) – OR = 0.74 95%CI (0.57, 0.96) (Population cohort)- NA as no fat available
	PBDE - 47 in fat (Operative cohort) – OR = 0.70 95%CI (0.55, 0.90) (Population cohort)- NA as no fat available

Key: a = Toxic equivalency factor, b = odds ratio, c = 95% confidence interval, d = Deep endometriotic nodules, e = Peritoneal endometriosis, f = Deep Infiltrating Endometriosis, g = Ovarian Endometriosis, h = adjusted odds ratio

Table 12: Results showing the effect estimates of organochlorinated pesticides (OCPs) and endometriosis

Author and Reference	Effect Estimate
Lebel et al, 1998 / [82]	**Sum of all Chlordanes (pesticides) in Geometric Means**
	Cases: 22.4 95%CI[a] (20.9, 23.9) Controls: 22.3 95%CI (20.7, 24.1)
	Sum of DDT in Geometric Means
	Cases: 238.2 95%CI (209.8, 270.6) Controls: 229.0 95%CI (195.6, 268.1)
Porpora et al, 2009 / [72]	**HCB:**
	32-54 (concentration in ng/g fat): Cases: 35.0 Controls: 30.77; OR[b] = 0.91 95%CI (0.40, 2.08)

	32-54 (concentration in ng/g fat): Cases: 35.0 Controls: 30.77; OR[b] = 0.91 95%CI (0.40, 2.08)
	p-p'DDE:
	32-54 (concentration in ng/g fat): Cases: 35.0 Controls: 30.77; OR[b] = 0.91 95%CI (0.40, 2.08)
	32-54 (concentration in ng/g fat): Cases: 35.0 Controls: 30.77; OR[b] = 0.91 95%CI (0.40, 2.08)
	pgC-TEQs/g fat:
	32-54 (concentration in ng/g fat): Cases: 35.0 Controls: 30.77; OR[b] = 0.91 95%CI (0.40, 2.08)
	32-54 (concentration in ng/g fat): Cases: 35.0 Controls: 30.77; OR[b] = 0.91 95%CI (0.40, 2.08)
Niskar et al, 2009 / [70]	P,P'-DDE Median concentrations (pgg-1), p-value for median test: cases=18900, controls=14600, p=0.15
Buck Louis et al, 2012 / [59]	Gamma HCH in fat (Operative cohort) - OR = 1.27 95%CI (1.01, 1.59) (Population cohort)- NA as no fat available
	Beta HCH in serum (Operative cohort) - OR = 0.77 95%CI (0.54, 1.14) (Population cohort) - OR = 1.72 95%CI (1.09, 2.72)
Cooney et al, 2010 / [88]	HCB OR = 6.4 95%CI (1.0, 42.8)
	t-nonachlor OR = 4.6 95%CI (0.5 , 41.6)
	Aldrin OR = 1.2 95%CI (0.2, 8.1)
	Beta-BHC OR = 2.0 95%CI (0.3, 15.8)
	After grouping for structure – aromatic fungicides OR= 5.3 95%CI (1.2, 23.6) for highest tertile
	After grouping for structure – cyclodiene insecticide OR = 2.7 95% CI (0.8, 9.5)
	After grouping for structure –chlorinated insecticide OR = 1.6 95% CI (0.5, 5.3) for mid-range tertile
Ploteau et al, 2017 /[67]	1. Association between the cases with DIE[c]+ OvE[d] for oxychlordane showed aOR (95%CI) of 5.82(1.84–27.69) for 1-SD increase 2. cis-heptachlor epoxide 5.36 (2.44–14.84) per 1-SD increase

Key: a = 95% confidence interval, b = odds ratio, c = Deep Infiltrating Endometriosis, d = Ovarian Endometrioma