



Data Article

Dataset of testicular germ cell tumors (TGCT) risk associated with serum polychlorinated biphenyl (PCB) by age at diagnosis and histologic types



Zhiyuan Cheng^a, Xichi Zhang^b, Bryan Bassig^c, Russ Hauser^d, Theodore R. Holford^e, Elizabeth Zheng^f, Dian Shi^{a,g}, Yong Zhu^e, Stephen Marc Schwartz^h, Chu Chen^h, Kunchong Shi^a, Bo Yang^a, Zhengmin Qianⁱ, Peter Boyle^j, Tongzhang Zheng^{a,*}

^aSchool of Public Health, Brown University, Providence, RI 02903, USA

^bDepartment of Biostatistics and Bioinformatics, Rollins School of Public Health, Emory University, 1518 Clifton Rd, NE, Atlanta, GA 30322, USA

^cDivision of Cancer Epidemiology and Genetics, National Cancer Institute, NIH, DHHS, Bethesda, MD 20892, USA

^dDepartment of Environmental Health, T.H. Chan School of Public Health, Harvard University, MA 02115, USA

^eDepartment of Biostatistics, Yale School of Public Health, Yale University, CT 06510, USA

^fUniversity of Vermont, Burlington, VT 05405, USA

^gSchool of Basic Medicine, Lanzhou University, Lanzhou, Gansu 730000, China

^hEpidemiology Program, Fred Hutchinson Cancer Research Center, Seattle, WA 98109, USA

ⁱInstitute for Global Health & Wellbeing College for Public Health & Social Justice, Saint Louis University, St. Louis, MO 63103, USA

^jInternational Prevention and Research Institute, International Agency for Research on Cancer (IARC), Lyon 69006, France

ARTICLE INFO

Article history:

Received 12 January 2021

Revised 19 February 2021

Accepted 23 March 2021

Available online 27 March 2021

ABSTRACT

In a population-based case control study of testicular germ cell tumors (TGCT), we reported a strong positive association between serum levels of Wolff's Group 1 (potentially estrogenic) polychlorinated biphenyl (PCBs) and risk of TGCT, and the observed associations were similar for both seminoma and non-seminoma. While the observed specific associations between TGCT and Wolff's Group 1 PCBs cannot

DOI of original article: [10.1016/j.envpol.2021.116458](https://doi.org/10.1016/j.envpol.2021.116458)

* Corresponding author.

E-mail address: Tongzhang_zheng@Brown.edu (T. Zheng).

<https://doi.org/10.1016/j.dib.2021.107014>

2352-3409/© 2021 The Authors. Published by Elsevier Inc. This is an open access article under the CC BY-NC-ND license (<http://creativecommons.org/licenses/by-nc-nd/4.0/>)

Keywords:

Case-control study
 Endocrine disruptors
 Persistent organic pollutants
 Polychlorinated biphenyl
 Testicular germ cell tumor

be easily explained by bias or confounding, a question can still be asked, that is, could the relationship between PCBs and TGCT differ by age at diagnosis? PCBs tend to bioaccumulate, with more heavily chlorinated PCB congeners tending to have longer half-lives. Half-lives of PCB congeners were reported ranging from 4.6 years for PCB-28 to 41.0 years for PCB-156. The half-life for the heavy PCB congeners (17.8 years) was found to be approximately twice that for the light PCBs (9.6 years) in early studies. Therefore, the same PCB concentration measured in a 20-year-old vs. a 55-year-old is unlikely to represent the same lifetime PCB exposure or type of PCB exposure. In this analysis, we stratified the data by median age of diagnosis of TGCT and further stratified by histologic type of TGCT (seminoma vs non-seminoma) to explore if the risk of TGCT associated with PCB exposures differs by age.

© 2021 The Authors. Published by Elsevier Inc.

This is an open access article under the CC BY-NC-ND license (<http://creativecommons.org/licenses/by-nc-nd/4.0/>)

Specifications Table

Subject	Epidemiology
Specific subject area	Cancer Epidemiology, Environmental Exposures
Type of data	Tables
How data were acquired	Serum levels of PCBs were analyzed by chemical laboratory at Harvard University and confounding factors were self-reported
Data format	Raw
Parameters for data collection	Men diagnosed with testicular germ cell tumors took part in this research project after the disease was diagnosed and health controls were selected by random digit dialing. All the participants signed the Informed Consent Form.
Description of data collection	The trained interviewers conducted in-person interviews with a standardized and structured questionnaire for demographic and lifestyle factors, past medical history, et al., and following the completion off the in-person interview, at least 5cc venous blood was collected and stored at -84°C until laboratory analyses.
Data source location	Data were collected either at the participants' homes or at locations convenient for the participants in Massachusetts and Connecticut, USA.
Data accessibility	Raw data are present in .CSV available with the article [1].
Related research article	Zhiyuan Cheng, Xichi Zhang, Bryan Bassig, Russ Hauser, Theodore R. Holford, Elizabeth Zheng, and et al. Serum polychlorinated biphenyl (PCB) levels and risk of testicular germ cell tumors: a population-based case-control study in Connecticut and Massachusetts. Environ. Pollut. 2021, 273: 116,458 https://doi.org/10.1016/j.envpol.2021.116458 [2]

Value of the Data

- The incidence rate of testicular germ cell tumors (TGCT) has continuously increased in Western countries over the last several decades [3-9]. Little is known about the polychlorinated biphenyl (PCBs) exposures that could explain the observed long-term increasing trend in the U.S. This dataset provides the results of serum level of 56 congeners of PCBs, the histological types of TGCT, as well as the epidemiology survey dataset including the demographic data and majority of the confounding factors of TGCT for further adjustment [1].
- This dataset is unique and utility to researchers who were interesting in exploring the TGCT risks associated with the exposure level of single PCBs, functional PCBs groups. Particularly under the inconsistent association between PCBs exposure and TGCT risk. [10-14]

- The dataset is also valuable to test the risk of TGCT associated with PCB exposures by age at diagnosis in differ. Since age at diagnosis could be a potential impact on the observed association since PCBs tend to bioaccumulate, individual PCB congeners have wide variation in half-lives [15], and thus cases diagnosed at different ages may have different PCB exposure profiles.
- This data presented here describe the association of PCBs and TGCT risk by age of diagnosis of TGCT and by histologic type of TGCT (seminoma vs non-seminoma). The results presented here offer a deep investigation of the relationship between PCB exposures and the risk of TGCT.
- The data are of considerable importance in cancer prevention and control area, especially considering the fact that testicular cancer has been increasing during the past decades in the US and several European populations.

1. Data Description

Table 1 present the composed variables and descriptions of .CSV file which contains raw data related to this article. A total of 356 histologically confirmed incident TGCT patients and 323 population-based controls were included in this database. All of the 56 PCBs congeners were presented as total lipid (total cholesterol and triglycerides) adjusted residue values.

Table 2 presents the risk of TGCT associated with functional categories of PCBs congeners among those under age 36 years old (median age of the study participants). A significant association was observed between total serum level of Group 1 (potential estrogenic) PCBs and risk of TGCT. Multi-covariate adjusted OR of 2.27 (95%CI:1.02–5.04) was observed when the fourth quartile was compared with the lowest quartile group. Further stratification of the Group 1 PCBs into Group 1A (estrogenic, weak phenobarbital inducers, not persistent) and Group 1B (weak phenobarbital inducers, persistent) PCBs showed that the Group 1B PCB congeners were significantly associated with the increased risk of TGCT. An OR of 2.48 (95%CI: 1.14–5.84) was observed for the Group 1B when the fourth quartile group was compared with the lowest. Group 2 (potentially antiestrogenic) and the sub-groups of Group 2 PCB congeners, and Group 3 (phenobarbital, CYP1A, and CYP2B inducers) PCBs showed no increased risk of TGCT.

Table 3 shows the results linking serum levels of PCBs stratified by the Wolff's classification to seminoma risk for those under 36 years old. The results for seminoma were similar to that of the total TGCT as presented in **Table 2** for those under age 36. A multi-covariate adjusted OR of 3.15 (95%CI: 1.37–7.22) was observed for Group 1 PCBs, and 3.63 (95%CI: 1.44–9.18) for the PCB 1B group when the fourth quartile of serums levels of PCBs was compared with the lowest

Table 1
Contents of the dataset.

Variables	Description
TGCT_Subtype	1=Seminoma 2=Non-seminoma
Case_cntrl	1=TGCT cases 2=Matched controls
Age	Age at diagnose
High_inch	High at diagnose
Weight_pound	Weight at diagnose (pound)
Undescended_testis	1=Undescended testis history 2=Non history of undescended testis
Education	1=High school or less, 2=College, 3=Postgraduate, 4=Master and above
BMI	Body Mass index
Study_site	1= Connecticut, 2=Massachusetts
Race	1=White, 2=Others
Testicle_injury	1=Have testicle injury history, 2=No history of testicle injury
birthweight_Kg	Birth weight (Kilogram)
Pcbxxx_fa	Lipo-adjusted concentration of 56 PCBs congeners

Table 2

Risk of TGCT associated with specific groups of PCBs for those with age < 36 (median age), Connecticut and Massachusetts, 2006–2010.

PCBs concentration (ng/g) ^a	Cases	Controls	OR (95% CI) ^b	ρ for trend ^c	ρ for homogeneity ^d
Group 1: Estrogenic PCBs (Congeners 25, 28, 31, 44, 49, 52, 70, 101, 174, 177, 187, 201)					
0.8–2.6	44	55	1.00		
2.7–5.4	27	28	1.13 (0.46–2.76)		
5.5–11.1	22	24	0.87 (0.35–2.18)		
11.2–88.2	66	26	2.27 (1.02–5.04)	<0.05	0.1
Group 1A (Congeners 25, 28, 31, 44, 49, 52, 70)					
0.1–0.8	34	33	1.00		
0.9–1.7	17	23	0.65 (0.23–1.89)		
1.8–3.3	21	35	0.72 (0.26–1.95)		
3.4–24.5	87	42	1.71 (0.78–3.79)	<0.05	0.2
Group 1B (Congeners 101, 174, 177, 187, 201)					
0.1–0.7	39	59	1.00		
0.8–3.3	40	30	1.86 (0.80–4.32)		
3.4–7.8	34	23	1.74 (0.71–4.26)		
7.9–75.1	46	21	2.48 (1.14–5.84)	<0.05	0.1
Group 2: Antiestrogenic PCBs (Congeners 66, 74, 95, 105, 110, 118, 128, 138, 156, 167, 170, 171)					
1.3–18.5	70	60	1.00		
18.6–29.8	52	40	1.02 (0.49–2.12)		
29.9–46.0	27	20	1.13 (0.49–2.61)		
46.1–193.4	9	7	1.55 (0.30–8.16)	0.8	0.2
Group 2A (Congeners 66, 74, 95, 105, 110, 171, 118, 156, 167)					
0.3–9.9	67	58	1.00		
10.0–14.6	41	40	0.86 (0.40–1.83)		
14.7–23.8	41	25	1.01 (0.44–2.31)		
23.9–114.1	10	10	1.93 (0.53–6.98)	0.9	0.4
Group 2B (Congeners 128, 138, 170)					
0.2–7.8	76	66	1.00		
7.9–13.8	44	41	0.67 (0.32–1.40)		
13.9–22.2	31	18	1.23 (0.53–2.86)		
22.3–170.6	8	8	0.68 (0.13–3.66)	0.5	0.7
Group 3: Enzyme induction PCBs (Congeners 99, 153, 180, 183, 196, 203)					
0.3–19.8	74	73	1.00		
19.9–37.2	58	41	1.09 (0.54–2.18)		
37.3–66.8	21	12	1.75 (0.62–5.00)		
66.9–339.8	6	7	0.63 (0.12–3.27)	0.4	0.4

^a Quartiles of PCBs groups were categorized by the quartile distribution of serum levels of PCBs among the controls.

^b Adjusted for race (white/other), BMI (17.7–24.8, 24.9–29.9, 30.0–50.1), undescended testis (yes/no), history of injuries to testis or groin (yes/no), family history of cancer (yes/no/unknown), birth weight (0.7–2.4, 2.5–3.9, 4.0–5.4), education (high school or less, college, postgraduate, Master and above), and study site (CT or MA).

^c ρ represents the Cochran–Armitage test for trend.

^d ρ represents the homogeneity test for the log-transformed PCBs concentration.

quartile group. No significantly increased or decreased risk of seminoma was observed for the Group 2 or subgroups of Group 2 PCB congeners and Group 3 PCBs.

Tables 4 presents the association of PCB congeners with non-seminoma risk for those under 36 years old. A multi-covariate adjusted OR of 2.73 (95%CI:1.30–5.73) was observed for the Group 1, and 3.39 (95%CI: 1.50–7.67) for the Group 1B PCBs congeners when the fourth quartile was compared with the lowest quartile group. Again, no significant increased or decreased risk of non-seminoma was observed for the population under age 36 years old.

Table 5 presented the TGCT risk associated with functional PCBs stratified by the Wolff's classification for those aged 36 and over. A significant association was observed between serum levels of Group 1 PCBs and the risk of TGCT. A multi-covariate adjusted OR of 1.91 (95%CI:1.04–3.69) was observed for Group 1 PCBs when the fourth quartile was compared with the lowest. A significantly higher OR of 6.26 (95%CI: 2.21–10.74) was observed for Group 1B PCBs when the fourth quartile was compared with the lowest quartile group. Group 2 (potentially

Table 3

Risk of seminoma associated with specific groups of PCBs for those with age <36 (median age), Connecticut and Massachusetts, 2006–2010.

PCBs concentration (ng/g) ^a	Cases	Controls	OR (95% CI) ^b	ρ for trend ^c	ρ for homogeneity ^d
Group 1: Estrogenic PCBs (Congeners 25, 28, 31, 44, 49, 52, 70, 101, 174, 177, 187, 201)					
0.8–2.6	17	55	1.00		
2.7–5.4	11	28	1.12 (0.43–2.95)		
5.5–11.1	10	24	1.21 (0.44–3.30)		
11.2–88.2	29	26	3.15 (1.37–7.22)	<0.05	0.2
Group 1A (Congeners 25, 28, 31, 44, 49, 52, 70)					
0.1–0.8	12	33	1.00		
0.9–1.7	5	23	0.45 (0.13–1.56)		
1.8–3.3	9	35	0.59 (0.20–1.80)		
3.4–24.5	41	42	1.99 (0.83–4.76)	<0.05	0.1
Group 1B (Congeners 101, 174, 177, 187, 201)					
0.1–0.7	13	59	1.00		
0.8–3.3	21	30	2.99 (1.21–7.42)		
3.4–7.8	14	23	2.24 (0.81–6.19)		
7.9–75.1	19	21	3.63 (1.44–9.18)	0.1	0.1
Group 2: Antiestrogenic PCBs (Congeners 66, 74, 95, 105, 110, 118, 128, 138, 156, 167, 170, 171)					
1.3–18.5	28	64	1.00		
18.6–29.8	27	40	1.40 (0.67–2.92)		
29.9–46.0	9	20	1.07 (0.40–2.93)		
46.1–193.4	3	7	0.90 (0.19–4.20)	0.6	0.4
Group 2A (Congeners 66, 74, 95, 105, 110, 171, 118, 156, 167)					
0.3–9.9	30	58	1.00		
10.0–14.6	18	40	0.73 (0.33–1.63)		
14.7–23.8	15	25	0.92 (0.38–2.20)		
23.9–114.1	4	10	0.90 (0.24–3.46)	0.8	0.8
Group 2B (Congeners 128, 138, 170)					
0.2–7.8	27	66	1.00		
7.9–13.8	24	41	1.24 (0.58–2.65)		
13.9–22.2	13	18	1.88 (0.75–4.73)		
22.3–170.6	3	8	0.81 (0.18–3.68)	0.6	0.7
Group 3: Enzyme induction PCBs (Congeners 99, 153, 180, 183, 196, 203)					
0.3–19.8	28	73	1.00		
19.9–37.2	29	41	1.62 (0.78–3.36)		
37.3–66.8	7	12	1.55 (0.51–4.69)		
66.9–339.8	3	7	1.01 (0.22–4.58)	0.5	0.5

- ^a Quartiles of PCBs groups were categorized by the quartile distribution of serum levels of PCBs among the controls.
^b Adjusted for race (white/other), BMI (17.7–24.8, 24.9–29.9, 30.0–50.1), undescended testis (yes/no), history of injuries to testis or groin (yes/no), family history of cancer (yes/no/unknown), birth weight (0.7–2.4, 2.5–3.9, 4.0–5.4), education (high school or less, college, postgraduate, Master and above), and study site (CT or MA).
^c ρ represents the Cochran–Armitage test for trend.
^d ρ represents the homogeneity test for the log-transformed PCBs concentration.

antiestrogenic) or subgroups of Group 2 (Group 1A and Group 1B) and Group 3 (phenobarbital, CYP1A, and CYP2B inducers) PCBs showed no increased or decreased risk of TGCT.

Table 6 presents the results for seminoma only for those aged 36 and over. Group 1A showed a significantly increased risk of non-seminoma with a multi-covariate adjusted OR of 7.46 (95%CI: 2.27–12.53). Group 2 and Group 3, however, showed no significantly increased or decreased risk of seminoma.

Table 7 shows the results for non-seminoma only for those aged 36 and over. Group 1A PCBs showed a significantly increased risk of non-seminoma with an OR of 3.11 (95%CI: 1.08–8.97). On the other hand, a significantly decreased risk of non-seminoma with an OR of 0.16 (95%CI: 0.04–0.68) and 0.12 (95%CI: 0.03–0.51) were observed for Group 2A PCBs (congeners 66, 74, 95, 105, 110, 171, 118, 156, 167) when the fourth and third quartile groups compared with lowest reference quartile. Group 2 and Group 3 PCB congeners showed no significant association with non-seminoma risk.

Table 4

Risk of non-seminoma associated with specific groups of PCBs for those with age < 36 (median age), Connecticut and Massachusetts, 2006–2010.

PCBs concentration (ng/g) ^a	Cases	Controls	OR (95% CI) ^b	ρ for trend ^c	ρ for homogeneity ^d
Group 1: Estrogenic PCBs (Congeners 25, 28, 31, 44, 49, 52, 70, 101, 174, 177, 187, 201)					
0.8–2.6	25	55	1.00		
2.7–5.4	15	28	1.26 (0.55–2.87)		
5.5–11.1	12	24	1.21 (0.49–2.97)		
11.2–88.2	35	26	2.73 (1.30–5.73)	0.1	0.2
Group 1A (Congeners 25, 28, 31, 44, 49, 52, 70)					
0.1–0.8	20	33	1.00		
0.9–1.7	11	23	0.71 (0.27–1.86)		
1.8–3.3	12	35	0.56 (0.23–1.37)		
3.4–24.5	44	42	1.51 (0.70–3.25)	0.1	0.1
Group 1B (Congeners 101, 174, 177, 187, 201)					
0.1–0.7	24	59	1.00		
0.8–3.3	18	30	2.03 (0.88–4.71)		
3.4–7.8	19	23	1.94 (0.83–4.52)		
7.9–75.1	26	21	3.39 (1.50–7.67)	0.3	0.2
Group 2: Antiestrogenic PCBs (Congeners 66, 74, 95, 105, 110, 118, 128, 138, 156, 167, 170, 171)					
1.3–18.5	39	64	1.00		
18.6–29.8	24	40	0.87 (0.43–1.77)		
29.9–46.0	17	20	1.84 (0.80–4.22)		
46.1–193.4	6	7	1.53 (0.40–5.90)	0.4	0.3
Group 2A (Congeners 66, 74, 95, 105, 110, 171, 118, 156, 167)					
0.3–9.9	34	58	1.00		
10.0–14.6	22	40	0.89 (0.44–1.83)		
14.7–23.8	25	25	1.86 (0.86–4.02)		
23.9–114.1	6	10	1.23 (0.36–4.25)	0.6	0.4
Group 2B (Congeners 128, 138, 170)					
0.2–7.8	46	66	1.00		
7.9–13.8	19	41	0.58 (0.29–1.19)		
13.9–22.2	17	18	1.64 (0.70–3.85)		
22.3–170.6	5	8	1.31 (0.36–4.82)	0.3	0.2
Group 3: Enzyme induction PCBs (Congeners 99, 153, 180, 183, 196, 203)					
0.3–19.8	43	73	1.00		
19.9–37.2	27	41	1.24 (0.64–2.43)		
37.3–66.8	14	12	2.51 (0.97–6.49)		
66.9–339.8	3	7	1.00 (0.22–4.54)	0.1	0.2

^a Quartiles of PCBs groups were categorized by the quartile distribution of serum levels of PCBs among the controls.

^b Adjusted for race (white/other), BMI (17.7–24.8, 24.9–29.9, 30.0–50.1), undescended testis (yes/no), history of injuries to testis or groin (yes/no), family history of cancer (yes/no/unknown), birth weight (0.7–2.4, 2.5–3.9, 4.0–5.4), education (high school or less, college, postgraduate, Master and above), and study site (CT or MA).

^c ρ represents the Cochran–Armitage test for trend.

^d ρ represents the homogeneity test for the log-transformed PCBs concentration.

Table 8 shows the Pearson correlation coefficients among the individual PCB congeners studied in this population. As shown in Table 7, the less chlorinated PCBs (mainly referred to non-ortho or one ortho PCBs congeners) generally have weaker correlations, while more chlorinated PCB congeners (mainly referred to those PCBs congeners with total of four or more chlorine substituents or with more than two of the meta positions chlorinated) generally have stronger correlations with other PCB congeners studied in this population. More specifically, Group 1 PCBs congeners showed non-significant correlation with Group 2 or Group 3 PCBs congeners, while strong correlation was observed between Group 2 and Group 3 PCBs congeners. These correlations strengthen our observed associations mainly among less chlorinated PCBs and TGCT risk in this study.

Table 5

Risk of total TGCT associated with specific groups of PCBs for those with age ≥ 36 (median age), Connecticut and Massachusetts, 2006–2010.

PCBs concentration (ng/g) ^a	Cases	Controls	OR (95% CI) ^b	ρ for trend ^c	ρ for homogeneity ^d
Group 1: Estrogenic PCBs (Congeners 25, 28, 31, 44, 49, 52, 70, 101, 174, 177, 187, 201)					
0.8–2.6	19	24	1.00		
2.7–5.4	19	53	0.57 (0.16–2.01)		
5.5–11.1	44	57	0.78 (0.25–2.41)		
11.2–88.2	71	56	1.91 (1.04–3.69)	<0.05	0.2
Group 1A (Congeners 25, 28, 31, 44, 49, 52, 70)					
0.1–0.8	28	47	1.00		
0.9–1.7	27	58	1.84 (0.64–5.33)		
1.8–3.3	26	46	1.11 (0.38–3.27)		
3.4–24.5	68	39	6.26 (2.21–10.74)	<0.05	0.1
Group 1B (Congeners 101, 174, 177, 187, 201)					
0.1–0.7	10	21	1.00		
0.8–3.3	32	51	1.10 (0.28–4.28)		
3.4–7.8	37	57	0.99 (0.26–3.76)		
7.9–75.1	70	61	2.65 (0.72–9.72)	<0.05	0.1
Group 2: Antiestrogenic PCBs (Congeners 66, 74, 95, 105, 110, 118, 128, 138, 156, 167, 170, 171)					
1.3–18.5	19	15	1.00		
18.6–29.8	34	42	0.70 (0.21–2.41)		
29.9–46.0	47	56	0.53 (0.16–1.77)		
46.1–193.4	48	75	0.50 (0.15–1.70)	0.9	0.4
Group 2A (Congeners 66, 74, 95, 105, 110, 171, 118, 156, 167)					
0.3–9.9	27	23	1.00		
10.0–14.6	38	40	0.82 (0.28–2.41)		
14.7–23.8	41	57	0.36 (0.13–1.02)		
23.9–114.1	43	70	0.45 (0.16–1.33)	0.9	0.4
Group 2B (Congeners 128, 138, 170)					
0.2–7.8	14	14	1.00		
7.9–13.8	31	40	0.61 (0.16–2.31)		
13.9–22.2	55	64	0.88 (0.27–2.89)		
22.3–170.6	49	72	0.64 (0.19–2.19)	0.8	0.5
Group 3: Enzyme induction PCBs (Congeners 99, 153, 180, 183, 196, 203)					
0.3–19.8	12	8	1.00		
19.9–37.2	35	40	0.82 (0.22–3.09)		
37.3–66.8	58	69	0.73 (0.21–2.62)		
66.9–339.8	44	73	0.32 (0.08–1.22)	0.7	0.3

^a Quartiles of PCBs groups were categorized by the quartile distribution of serum levels of PCBs among the controls.

^b Adjusted for race (white/other), BMI (17.7–24.8, 24.9–29.9, 30.0–50.1), undescended testis (yes/no), history of injuries to testis or groin (yes/no), family history of cancer (yes/no/unknown), birth weight (0.7–2.4, 2.5–3.9, 4.0–5.4), education (high school or less, college, postgraduate, Master and above), and study site (CT or MA).

^c ρ represents the Cochran–Armitage test for trend.

^d ρ represents the homogeneity test for the log-transformed PCBs concentration.

2. Experimental Design, Materials and Methods

Participants. The study population has been described previously [16]. The subjects were recruited between 2006 and 2010 among male residents of CT and MA. The case group includes 356 histologically confirmed incident TGCT patients aged from 15 to 55. 323 population-based controls were selected by random digit dialing frequency-matched to the cases on the basis of age (± 5), sex and state.

Experimental Design. The trained interviewers conducted in-person interviews with a standardized and structured questionnaire for demographic and lifestyle factors, past medical history, etc. and following the completion off the in-person interview, at least 5cc venous blood was collected and stored at -84 °C until laboratory analyses.

The serum samples collected in CT and MA were transferred to the study laboratory at Harvard University where lipid level (total cholesterol, triglyceride) and 56 PCB congeners were

Table 6Risk of seminoma associated with specific groups of PCBs for those with age ≥ 36 (median age), Connecticut and Massachusetts, 2006–2010.

PCBs concentration (ng/g) ^a	Cases	Controls	OR (95% CI) ^b	ρ for trend ^c	ρ for homogeneity ^d
Group 1: Estrogenic PCBs (Congeners 25, 28, 31, 44, 49, 52, 70, 101, 174, 177, 187, 201)					
0.8–2.6	14	24	1.00		
2.7–5.4	7	53	0.34 (0.07–1.54)		
5.5–11.1	28	57	0.67 (0.19–2.36)		
11.2–88.2	52	56	1.59 (0.59–5.18)	<0.05	0.1
Group 1A (Congeners 25, 28, 31, 44, 49, 52, 70)					
0.1–0.8	20	47	1.00		
0.9–1.7	15	58	1.13 (0.31–4.13)		
1.8–3.3	17	46	1.10 (0.32–3.79)		
3.4–24.5	49	39	7.46 (2.27–12.53)	<0.05	0.1
Group 1B (Congeners 101, 174, 177, 187, 201)					
0.1–0.7	5	21	1.00		
0.8–3.3	20	51	0.94 (0.19–4.62)		
3.4–7.8	24	57	1.06 (0.23–4.96)		
7.9–75.1	52	61	2.35 (0.54–10.27)	0.1	0.1
Group 2: Antiestrogenic PCBs (Congeners 66, 74, 95, 105, 110, 118, 128, 138, 156, 167, 170, 171)					
1.3–18.5	13	15	1.00		
18.6–29.8	19	42	0.47 (0.12–1.88)		
29.9–46.0	33	56	0.54 (0.15–1.92)		
46.1–193.4	35	75	0.42 (0.12–1.50)	0.8	0.6
Group 2A (Congeners 66, 74, 95, 105, 110, 171, 118, 156, 167)					
0.3–9.9	13	23	1.00		
10.0–14.6	27	40	1.71 (0.48–6.11)		
14.7–23.8	29	57	0.68 (0.20–2.39)		
23.9–114.1	32	70	0.74 (0.21–2.57)	0.1	0.3
Group 2B (Congeners 128, 138, 170)					
0.2–7.8	9	14	1.00		
7.9–13.8	19	40	0.44 (0.09–2.02)		
13.9–22.2	39	64	0.81 (0.23–2.91)		
22.3–170.6	34	72	0.52 (0.14–1.90)	0.8	0.5
Group 3: Enzyme induction PCBs (Congeners 99, 153, 180, 183, 196, 203)					
0.3–19.8	9	8	1.00		
19.9–37.2	21	40	0.75 (0.18–3.17)		
37.3–66.8	41	69	0.75 (0.20–2.80)		
66.9–339.8	30	73	0.27 (0.07–1.07)	0.1	0.2

^a Quartiles of PCBs groups were categorized by the quartile distribution of serum levels of PCBs among the controls.^b Adjusted for race (white/other), BMI (17.7–24.8, 24.9–29.9, 30.0–50.1), undescended testis (yes/no), history of injuries to testis or groin (yes/no), family history of cancer (yes/no/unknown), birth weight (0.7–2.4, 2.5–3.9, 4.0–5.4), education (high school or less, college, postgraduate, Master and above), and study site (CT or MA).^c ρ represents the Cochran–Armitage test for trend.^d ρ represents the homogeneity test for the log-transformed PCBs concentration.

analyzed (PCBs 6, 8, 16, 18, 25, 26, 28, 31, 33, 37, 41, 44, 47, 49, 52, 60, 66, 70, 74, 84, 87, 95, 97, 99, 101, 105, 110, 118, 128, 135, 136, 138, 141, 146, 149, 151, 153, 156, 157, 167, 170, 171, 174, 180, 183, 187, 189, 194, 195, 196, 199, 201/177, 203, 206 and 209). In brief, serum extraction is based on analytical procedures developed by the U.S. Centers for Disease Control and Prevention (CDC), with modifications to conform to ultra-trace levels analysis. The PCBs concentration in serum extracts were analyzed by gas chromatography with electron capture detection using Hewlett Packard 6980 GC with dual injection, dual capillary columns and dual Micro-ECDs (GC/_LECD). Confirmatory analyses were done on a capillary column of different polarity and similar instrumental conditions. Total lipid concentration was calculated for each subject using measurements of total cholesterol and triglycerides. Method detection limits (MDLs) were determined as three times the standard deviation obtained from the analysis of eight aliquots of bovine serum fortified with target analytes as recommended in U.S. EPA method (EPA 1984), MDL values for most of the congeners below 0.01 ng/g with all PCB

Table 7Risk of non-seminoma associated with specific groups of PCBs for those with age \geq 36 (median age), Connecticut and Massachusetts, 2006–2010.

PCBs concentration (ng/g) ^a	Cases	Controls	OR (95% CI) ^b	ρ for trend ^c	ρ for homogeneity ^d
Group 1: Estrogenic PCBs (Congeners 25, 28, 31, 44, 49, 52, 70, 101, 174, 177, 187, 201)					
0.8–2.6	4	24	1.00		
2.7–5.4	12	53	1.70 (0.44–6.66)		
5.5–11.1	11	57	1.33 (0.33–5.38)		
11.2–88.2	17	56	2.22 (0.57–8.59)	0.6	0.4
Group 1A (Congeners 25, 28, 31, 44, 49, 52, 70)					
0.1–0.8	7	47	1.00		
0.9–1.7	11	58	1.63 (0.54–4.97)		
1.8–3.3	9	46	1.18 (0.36–3.84)		
3.4–24.5	17	39	3.11 (1.08–8.97)	0.2	0.1
Group 1B (Congeners 101, 174, 177, 187, 201)					
0.1–0.7	5	21	1.00		
0.8–3.3	10	51	0.81 (0.22–2.96)		
3.4–7.8	11	57	0.81 (0.23–2.87)		
7.9–75.1	18	61	1.28 (0.38–4.34)	0.3	0.2
Group 2: Antiestrogenic PCBs (Congeners 66, 74, 95, 105, 110, 118, 128, 138, 156, 167, 170, 171)					
1.3–18.5	6	15	1.00		
18.6–29.8	14	42	1.45 (0.41–5.16)		
29.9–46.0	12	56	0.72 (0.20–2.58)		
46.1–193.4	12	75	0.67 (0.18–2.46)	0.1	0.2
Group 2A (Congeners 66, 74, 95, 105, 110, 171, 118, 156, 167)					
0.3–9.9	13	23	1.00		
10.0–14.6	10	40	0.29 (0.07–1.27)		
14.7–23.8	11	57	0.12 (0.03–0.51)		
23.9–114.1	10	70	0.16 (0.04–0.68)	0.4	0.2
Group 2B (Congeners 128, 138, 170)					
0.2–7.8	5	14	1.00		
7.9–13.8	11	40	1.32 (0.20–8.59)		
13.9–22.2	13	64	0.91 (0.16–5.24)		
22.3–170.6	15	72	0.70 (0.12–3.87)	0.4	0.3
Group 3: Enzyme induction PCBs (Congeners 99, 153, 180, 183, 196, 203)					
0.3–19.8	3	8	1.00		
19.9–37.2	13	40	1.23 (0.18–8.57)		
37.3–66.8	14	69	0.67 (0.10–4.62)		
66.9–339.8	14	73	0.34 (0.05–2.44)	0.1	0.1

^a Quartiles of PCBs groups were categorized by the quartile distribution of serum levels of PCBs among the controls.^b Adjusted for race (white/other), BMI (17.7–24.8, 24.9–29.9, 30.0–50.1), undescended testis (yes/no), history of injuries to testis or groin (yes/no), family history of cancer (yes/no/unknown), birth weight (0.7–2.4, 2.5–3.9, 4.0–5.4), education (high school or less, college, postgraduate, Master and above), and study site (CT or MA).^c ρ represents the Cochran–Armitage test for trend.^d ρ represents the homogeneity test for the log-transformed PCBs concentration.

congeners below 0.05 ng/g. The analytical methods and QA/QC procedures have been described elsewhere [2].

Statistic methods. Unconditional logistic regression models were used to assess the association between serum PCB levels and TGCT risk and to adjust for potential confounders. The total serum levels of PCBs, groups of PCBs and individual PCB congeners were compared between the controls and all the TGCT cases or two broad histologic groups of TGCT (seminoma and non-seminoma). Total PCBs were calculated by summing the concentrations of all 56 measured PCBs analytes. We grouped PCB congeners into 3 PCB subgroups as proposed by Wolff et al. [17], based on PCB structure and biological-activity.

Table 8
Continued

PCBs groups	PCB 6	PCB 8	PCB 16	PCB 18	PCB 26	PCB 28	PCB 33	PCB 37	PCB 41	PCB 47	PCB 60	PCB 70	PCB 78	PCB 84	PCB 87	PCB 95	PCB 97	PCB 101	PCB 105	PCB 135	PCB 136	PCB 141	PCB 146	PCB 149	PCB 151	PCB 157	PCB 171	PCB 189	PCB 194	PCB 195	PCB 199	PCB 206	PCB 209			
Not among Wolff's classification	PCB 6	1.00																																		
	PCB 8	0.52	1.00																																	
	PCB 16	0.30	0.48	1.00																																
	PCB 18	0.34	0.57	0.45	1.00																															
	PCB 26	0.25	0.42	0.32	0.40	1.00																														
	PCB 28	-0.03	0.06	0.09	0.12	0.01	1.00																													
	PCB 33	0.23	0.22	0.33	0.26	0.35	0.05	1.00																												
	PCB 37	0.15	0.33	0.36	0.41	0.47	0.15	0.31	1.00																											
	PCB 41	0.08	0.09	0.08	-0.03	0.07	0.16	0.11	0.21	1.00																										
	PCB 47	0.15	0.13	0.04	-0.13	0.08	0.08	0.18	0.13	0.17	1.00																									
	PCB 60	0.03	0.00	-0.01	0.01	0.03	0.46	-0.01	0.07	0.19	0.05	1.00																								
	PCB 70	0.13	0.31	0.22	0.46	0.40	0.17	0.20	0.39	0.09	0.04	0.06	1.00																							
	PCB 84	0.20	0.03	-0.05	-0.03	-0.05	-0.05	0.07	-0.07	0.06	0.17	0.05	-0.03	1.00																						
	PCB 87	0.14	0.15	0.09	0.22	0.20	0.13	0.06	0.24	0.05	-0.01	0.17	0.42	0.05	1.00																					
	PCB 95	0.14	0.22	0.20	0.43	0.16	0.09	0.21	0.33	-0.04	0.01	-0.02	0.45	0.03	0.49	1.00																				
	PCB 97	0.10	0.24	0.18	0.33	0.42	0.16	0.26	0.32	0.10	0.08	0.07	0.58	-0.12	0.47	0.32	1.00																			
	PCB 101	0.12	0.18	0.21	0.46	0.24	0.19	0.18	0.33	0.05	0.08	0.11	0.59	0.01	0.70	0.70	0.54	1.00																		
	PCB 135	0.16	0.16	0.08	0.27	0.27	0.15	0.19	0.25	0.18	0.13	0.16	0.32	0.11	0.59	0.37	0.47	0.62	1.00																	
	PCB 136	0.16	0.15	0.09	0.30	0.26	0.15	0.10	0.25	0.13	0.06	0.09	0.42	-0.05	0.57	0.37	0.49	0.60	0.63	1.00																
	PCB 141	0.12	0.14	0.13	0.35	0.22	0.14	0.11	0.22	0.07	0.06	0.08	0.42	-0.05	0.57	0.37	0.49	0.60	0.63	0.78	1.00															
	PCB 146	0.01	0.06	-0.03	0.05	0.04	0.26	-0.03	0.14	0.28	0.06	0.35	0.03	0.02	0.17	0.01	0.11	0.09	0.05	0.04	1.00															
	PCB 149	0.21	0.25	0.19	0.48	0.22	0.12	0.17	0.23	0.04	0.06	0.35	0.00	0.37	0.54	0.42	0.58	0.66	0.60	0.73	-0.01	1.00														
	PCB 151	0.16	0.24	0.15	0.36	0.20	0.21	0.08	0.21	0.08	0.04	0.08	0.57	-0.03	0.57	0.49	0.54	0.67	0.67	0.77	0.81	0.14	0.78	1.00												
	PCB 157	0.03	0.12	0.04	0.11	0.10	0.18	0.02	0.18	0.22	0.06	0.27	0.08	-0.03	0.28	0.07	0.13	0.12	0.11	0.11	0.77	0.01	1.00													
	PCB 171	-0.02	0.05	-0.10	-0.09	-0.05	0.19	-0.08	0.10	0.25	0.08	0.28	-0.06	-0.03	-0.21	-0.16	0.05	0.01	0.00	-0.05	-0.06	0.68	-0.13	-0.03	0.58	0.57	1.00									
	PCB 189	-0.04	-0.03	-0.07	-0.11	-0.04	0.18	-0.07	-0.01	0.24	0.08	0.24	-0.17	-0.02	-0.31	-0.21	0.00	-0.09	-0.06	-0.03	-0.08	0.65	-0.18	-0.08	0.54	0.56	0.73	1.00								
	PCB 194	0.00	0.07	0.05	0.19	0.07	0.29	0.02	0.13	0.16	0.03	0.23	0.20	-0.09	0.13	0.11	0.21	0.21	0.21	0.22	0.26	0.65	0.29	0.34	0.49	0.67	0.56	0.65	1.00							
	PCB 195	-0.06	-0.04	-0.06	-0.02	-0.06	0.22	-0.09	0.04	0.19	0.03	0.27	0.00	-0.01	0.01	-0.07	0.07	0.01	0.06	0.05	0.04	0.71	0.02	0.11	0.54	0.59	0.64	0.86	0.77	1.00						
	PCB 206	-0.05	-0.08	-0.11	-0.16	-0.11	0.17	-0.11	-0.06	0.21	0.02	0.25	-0.19	0.03	-0.06	-0.21	-0.05	-0.11	-0.08	-0.08	-0.13	0.65	-0.19	-0.10	0.47	0.49	0.61	0.89	0.57	0.87	1.00					
	PCB 209	-0.04	-0.05	-0.06	0.01	-0.04	0.25	-0.06	0.02	0.22	0.04	0.25	0.01	0.00	0.05	-0.08	0.10	0.09	0.11	0.08	0.02	0.65	-0.02	0.09	0.40	0.49	0.60	0.71	0.55	0.71	0.76	1.00				

Ethics Statement

The study (HIC number: 0,602,001,111) was approved by the Institutional Review Boards (IRBs) at both Yale and Harvard Universities, and by the Human Investigation Committees (HIC) at the Department of Public Health in CT and MA, at the Dana Farber Cancer Institute, and the 28 participating hospitals in Connecticut. All study participants provided informed consents.

CRediT Author Statement

Zhiyuan Cheng: Design, Software, Formal analysis, Writing - Original Draft, Writing - Review & Editing; **Xichi Zhang:** Design, Software, Investigation; **Bryan Bassig:** Software, Validation, Writing - Review & Editing; **Russ Hauser:** Supervision, Project administration, Funding acquisition; **Theodore R. Holford:** Conceptualization, Methodology, Data collection; **Elizabeth Zheng:** Investigation, Software; **Dian Shi:** Software, Validation. **Yong Zhu:** Methodology, Resources; **Stephen Marc Schwartz:** Conceptualization, Design, Methodology; **Chu Chen:** Conceptualization, Design; **Kunchong Shi:** Resources, Methodology; **Bo Yang:** Software; **Zhengmin Qian:** Conceptualization, Design; **Peter Boyle:** Methodology; **Tongzhang Zheng:** Conceptualization, Methodology, Writing - Review & Editing, Resources, Supervision, Project administration, Funding acquisition.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships which have, or could be perceived to have, influenced the work reported in this article.

Acknowledgements

This work was partly supported by grants from the National Institutes of Health (US): [R01CA104786](#); [R01ES029082](#); [D43TW 008323](#); [D43TW 007864-01](#); [R01ES029082](#); The National Natural Science Fund from the National Natural Science Foundation of China (grant [81172757](#)).

The cooperation of 28 Connecticut hospitals, including Charlotte Hungerford Hospital, Bridgeport Hospital, Danbury Hospital, Hartford Hospital, Middlesex Hospital, New Britain General Hospital, Bradley Memorial Hospital, Yale/New Haven Hospital, St. Francis Hospital and Medical Center, St. Mary's Hospital, Hospital of St. Raphael, St. Vincent's Medical Center, Stamford Hospital, William W. Backus Hospital, Windham Hospital, Eastern Connecticut Health Network, Griffin Hospital, Bristol Hospital, Johnson Memorial Hospital, Day Kimball Hospital, Greenwich Hospital, Lawrence and Memorial Hospital, Milford Hospital, New Milford Hospital, Norwalk Hospital, MidState Medical Center, John Dempsey Hospital and Waterbury Hospital, in allowing patient access, is gratefully acknowledged. Rajni Mehta from the Yale Comprehensive Cancer Center's RCA provided great assistance in both IRB approvals and field implementation of the study. Certain data used in this study were obtained from the Tumor Registry located in the Connecticut Department of Public Health, and Massachusetts Department of Public Health. PCBs quantitative determination and total lipids were tested by Study laboratory at Harvard School of Public Health.

Supplementary Materials

Supplementary material associated with this article can be found in the online version at doi:[10.1016/j.dib.2021.107014](https://doi.org/10.1016/j.dib.2021.107014).

References

- [1] C. Zhiyuan, Z. Xichi, B. Bryan, H. Russ, R.H. Theodore, Z. Elizabeth, et al. Dataset of Testicular Germ Cell Tumors (TGCT) Risk Associated with Serum Polychlorinated Biphenyl (PCB) by Age at Diagnosis and Histologic Type. In., V1 edn: Harvard Dataverse; 2021. doi:10.7910/DVN/XSO8FK.
- [2] Z. Cheng, X. Zhang, B. Bassig, R. Hauser, T.R. Holford, E. Zheng, et al., Serum polychlorinated biphenyl (PCB) levels and risk of testicular germ cell tumors: a population-based case-control study in Connecticut and Massachusetts, *Environ. Pollut.* 273 (2021) 116458.
- [3] F. Bray, L. Richiardi, A. Ekblom, E. Pukkala, M. Cuninkova, H. Moller, Trends in testicular cancer incidence and mortality in 22 European countries: continuing increases in incidence and declines in mortality, *Int. J. Cancer* 118 (12) (2006) 3099–3111.
- [4] V.M. Chia, S.M. Quraishi, S.S. Devesa, M.P. Purdue, M.B. Cook, K.A. McGlynn, International trends in the incidence of testicular cancer, 1973–2002, *Cancer Epidemiol. Biomarkers. Prev* 19 (5) (2010) 1151–1159.
- [5] P. Filippou, J.E. Ferguson 3rd, M.E. Nielsen, Epidemiology of prostate and testicular cancer, *Semin. Intervent. Radiol* 33 (3) (2016) 182–185.
- [6] A.A. Ghazarian, B. Trabert, S.S. Devesa, K.A. McGlynn, Recent trends in the incidence of testicular germ cell tumors in the United States, *Andrology* 3 (1) (2015) 13–18.
- [7] J.K. Gurney, A.A. Florio, A. Znaor, J. Ferlay, M. Laversanne, D. Sarfati, et al., International trends in the incidence of testicular cancer: lessons from 35 years and 41 countries, *Eur. Urol* 76 (5) (2019) 615–623.
- [8] Z.L. Smith, R.P. Werntz, S.E. Eggener, Testicular cancer: epidemiology, diagnosis, and management, *Med. Clin. North Am* 102 (2) (2018) 251–264.
- [9] A. Znaor, J. Lortet-Tieulent, M. Laversanne, A. Jemal, F. Bray, International testicular cancer incidence trends: generational transitions in 38 countries 1900–1990, *Cancer Causes Control* 26 (1) (2015) 151–158.
- [10] J.S. Piltoft, S.B. Larsen, S.O. Dalton, C. Johansen, J.L. Baker, L. Cederkvist, et al., Early life risk factors for testicular cancer: a case-cohort study based on the Copenhagen School Health Records Register, *Acta Oncol* 56 (2) (2017) 220–224.
- [11] T. Fukawa, H.O. Kanayama, Current knowledge of risk factors for testicular germ cell tumors, *Int J Urol* 25 (4) (2018) 337–344.
- [12] M.H. Greene, C.P. Kratz, P.L. Mai, C. Mueller, J.A. Peters, G. Bratslavsky, et al., Familial testicular germ cell tumors in adults: 2010 summary of genetic risk factors and clinical phenotype, *Endocr Relat Cancer* 17 (2) (2010) R109–R121.
- [13] H.W. Herr, Prognostic risk factors in low stage testicular germ cell tumors: unanswered questions regarding clinically useful prognosticators for extratesticular disease, *Cancer* 82 (1) (1998) 230–232.
- [14] K.A. McGlynn, Environmental and host factors in testicular germ cell tumors, *Cancer Invest* 19 (8) (2001) 842–853.
- [15] R.F. Seegal, E.F. Fitzgerald, E.A. Hills, M.S. Wolff, R.F. Haase, A.C. Todd, et al., Estimating the half-lives of PCB congeners in former capacitor workers measured over a 28-year interval, *J. Expo. Sci. Environ. Epidemiol* 21 (3) (2011) 234–246.
- [16] N. Li, R. Hauser, T. Holford, Y. Zhu, Y. Zhang, B.A. Bassig, et al., Muscle-building supplement use and increased risk of testicular germ cell cancer in men from Connecticut and Massachusetts, *Br. J. Cancer* 112 (7) (2015) 1247–1250.
- [17] M.S. Wolff, D. Camann, M. Gammon, S.D. Stellman, Proposed PCB congener groupings for epidemiological studies, *Environ. Health Perspect* 105 (1) (1997) 13–14.