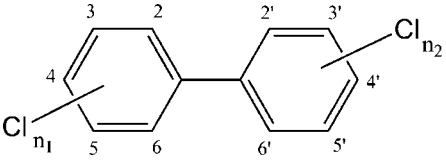
Chlorinated biphenyls

BAR (2011)*	**PCB 28 0.02 µg/l plasma** **PCB 52 < 0.01 µg/l plasma** **PCB 101 < 0.01 µg/l plasma** Sampling time: not fixed
Chemical description	Chlorinated biphenyls (mono- to decachlorobiphenyl)
Synonyms	CB Chlorobiphenyls Chlorodiphenyls PCB Polychlorobiphenyl Polychlorodiphenyl Chlorinated biphenyls
Molecular formula	$C_{12}H_{(10-n)}Cl_n$
Structural formula	
MAK values (2012)*	**Mono-, di-, trichlorinated biphenyls:** **–** **Higher chlorinated (≥ 4 Cl) biphenyls:** **0.003 mg/m³ I****
Peak limitation (2012)	Mono-, di-, trichlorinated biphenyls: –* Higher chlorinated (≥ 4 Cl) biphenyls: Category II, excursion factor 8
Absorption through the skin (1978)	H

The MAK Collection for Occupational Health and Safety

DOI: 10.1002/3527600418.bb133636e1918

Sensitization	–
Carcinogenicity (2012)	Mono-, di-, trichlorinated biphenyls:
	Carcinogen Category 3 B*
	Higher chlorinated (≥ 4 Cl) biphenyls:
	Carcinogen Category 4
Prenatal toxicity (1989)	Mono-, di-, trichlorinated biphenyls:
	–*
	Higher chlorinated (≥ 4 Cl) biphenyls:
	Pregnancy Risk Group B
Germ cell mutagenicity (2012)	Mono-, di-, trichlorinated biphenyls:
	Germ cell mutagen Category 3A*
	Higher chlorinated (≥ 4 Cl) biphenyls:
	Germ cell mutagen Category 5

*for updates please see http://onlinelibrary.wiley.com/book/10.1002/9783527805983
**(PCB 28 + PCB 52 + PCB 101 + PCB 138 + PCB 153 + PCB 180) · 5

Substance	Chemical name	Formula	CAS No.	Molecular weight [g/mol]
PCB	Polychlorinated biphenyls	$C_{12}H_{(10-n)}Cl_{(n)}$	1336-36-3	
PCB 28	2,4,4′-Trichlorobiphenyl	$C_{12}H_7Cl_3$	7012-37-5	257.5
PCB 52	2,2′,5,5′-tetrachlorobiphenyl	$C_{12}H_6Cl_4$	35693-99-3	292.0
PCB 101	2,2′,4,5,5′-pentachlorobiphenyl	$C_{12}H_5Cl_5$	37680-73-2	326.4
PCB 138	2,2′,3,4,4′,5′-hexachlorobiphenyl	$C_{12}H_4Cl_6$	35065-28-2	360.9
PCB 153	2,2′,4,4′,5,5′-hexachlorobiphenyl	$C_{12}H_4Cl_6$	35065-27-1	360.9
PCB 180	2,2′,3,4,4′,5,5′-heptachlorobiphenyl	$C_{12}H_3Cl_7$	35065-29-3	395.3

Among other purposes, chlorinated biphenyls (PCB) are used in the electrical industry as cooling and insulating fluids in transformers and condensers, as heat exchangers and hydraulic liquids, as plasticizers in plastics, paints and resins, as cutting oils and lubricants, as flame retardants in the electrical industry, and as permanent elastic sealing materials. Since 1989, it is not allowed to produce or sell PCB and PCB-containing products and equipment in Germany. Since July 2000, the use of equipment (transformers) using fluids containing more than one litre having a PCB content of over 50 mg/kg has been prohibited, and PCB and PCB-containing equipment was to be disposed of by the end of 2010 as determined by the Chemicals Prohibition Ordinance (Chemikalien-Verbotsverordnung 2010) passed in the same year.

As a result of the PCB prohibition in Germany it can no longer be assumed that workplace exposure from direct handling of PCB is still relevant. Over recent years, therefore, environmental medical aspects have been in the foreground, for example the results of mother-child studies (Wilhelm et al. 2008 a, b; Wittsiepe et al. 2008).

Nevertheless, the exposure of workers in waste disposal and decontamination do show that occupational exposure to PCBs must still be considered. In such cases, the determination of low chlorinated PCBs in the blood of the workers involved is of particular importance, as they are preferentially absorbed by inhalation unlike higher chlorinated PCBs.

1 Metabolism and Toxicokinetics

1.1 Absorption, distribution and excretion

Highly chlorinated PCBs are mainly absorbed via animal-derived foods. The daily absorption by adults in the late 1980s in Germany was estimated to be about 0.05 µg PCB/kg body weight. Due to a decreasing PCB content in foods a daily amount of about 0.001–0.02 µg PCB/kg body weight is absorbed at present (UBA 1999; WHO 2003). In the total exposure of humans, the inhalation of low chlorinated PCBs, especially from contaminated indoor air, plays a rather subordinate role (UBA 2003 a). However, low chlorinated PCB congeners can be metabolized more easily. In the case of this route of exposure, therefore, an increased formation of metabolites having a toxicity still not known up to now can be expected (UBA 1999). The various PCB congeners can also be absorbed through the skin to different extents (Henschler 1978). The initial elimination half-lives of PCB congeners are given as up to five years. It is assumed, however, that individual congeners can have a half-life of a few days (Bühler et al. 1988; Ryan et al. 1993). Excretion of the PCB mainly takes place with the faeces (Henschler 1978). On account of the extremely long half-life of high chlorinated PCBs, the internal PCB exposure in elderly humans is higher than in younger persons (see Section 6 for more).

1.2 Metabolism

Low chlorinated PCBs can partly be metabolized via epoxides. Thereof, soluble metabolites such as hydroxy- or methylsulfone-PCB are formed. Little is known about the toxicity of the intermediates formed (UBA 1999).

2 Critical Toxicity

From animal studies, evidence for the carcinogenic, immuno-, neuro- and reproductive toxic effects of PCB are available (WHO 2003). As critical endpoints, immunotoxicity and neurotoxicity occurred in Rhesus monkeys, the most sensitive animal species for PCB effects, and were observed already at PCB doses of 5 µg/kg body weight and day and 7.5 µg/kg body weight and day (Aroclor 1254), respective-

ly (Rice 1997; Rice and Hayward 1997; Tryphonas et al. 1989, 1991). Especially high chlorinated PCBs produce liver tumours in rodents (WHO 2003). In the few animal studies on toxicity after inhalation exposure, effects in the liver, kidneys and nerves as well as changes in the thyroid hormones were observed (Casey et al. 1999; Treon et al. 1956).

Specific PCBs (4 non-ortho as well as 8 mono-ortho PCBs according to WHO) have effects similar to those of dioxin and are therefore designated as "dioxin-like PCBs". Among the 20 different coplanar PCB congeners, the highest toxicity is attributed to the non-ortho-substituted congeners PCB 77, PCB 126 and PCB 169 (Safe 1994).

Dioxin-like non-ortho PCBs, such as PCB 77, PCB 81, PCB 126 and PCB 169 can bind to the aryl hydrocarbon receptor (Ah receptor) and induce, for example, CYP 1A1 and CYP 1A2 in a way similar to that of TCDD. Dioxin-like mono-ortho PCBs such as PCB 105, PCB 118, PCB 167 are so-called mixed type inducers, i.e. they induce both CYP 1A1 as well as CYP 2B1. Adverse effects of PCB in humans after exposure to PCB at high levels were observed, such as those that affected the inhabitants of Japan and Taiwan who were exposed to PCBs between 1968 and 1979 by eating contaminated rice oil (Yusho and Yu-Cheng disease). In adults, somatic effects and skin changes (chloracne, hyperkeratosis, hyperpigmentation) followed by hematological changes, disturbances of the lung function and an increased number of still born were found. Children whose mothers had consumed the PCB-contaminated rice oil during pregnancy were particularly affected. As, in both cases, the rice oil had also been considerably contaminated with polychlorinated dibenzofuranes and the PCB blood levels could only be roughly estimated at that time, this means that the role of PCBs involved in the health effects observed remain unclear (Henschler 1978; Ross 2004).

In addition, workers handling transformers and condensers containing PCBs were exposed to higher levels. The evaluation of nearly 40 studies on occupational PCB exposure has, however, only shown chloracne to be a convincingly confirmed toxic effect. In a number of other cases, the occurrence of toxic effects was considered possible or suspected, but altogether rather questionable (Henschler 1978). In children, immunological effects and disturbances in psychomotor and mental development are already produced by PCB exposure via mother's milk and food consumption (Walkowiak et al. 2001; Weisglas-Kuperus et al. 2000).

The carcinogenicity of PCBs in humans is at present under discussion (IARC 2011).

3 Exposure and Effects

Due to the varying composition of PCB mixtures, it is questionable whether selected PCBs as biomarkers correlate closely enough with the effects (biological effects) that derivation of health-based threshold limit values is possible. As no plasma concentration-effect relationships can be derived so far, only biological reference values are established at present.

4 Selection of Indicators

It is usual to estimate internal exposure by analyzing three indicator congeners for low chlorinated PCBs (28, 52, 101) and three indicator congeners for high chlorinated PCBs (138, 153, 180) in plasma samples (Beck and Mathar 1985); in selecting these, toxicological aspects were not in the foreground. An investigation of all PCBs is not feasible (UBA 1999).

In addition, experience also exists as regards a valid determination of other congeners (Humphrey et al. 2000; Schettgen et al. 2008). However, the limited database does not allow the derivation of a BAR ("Biologischer Arbeitsstoff-Referenzwert") for these congeners at present.

5 Methods

Established methods for quantitative analysis in blood do exist for 6 so-called indicator congeners and 12 coplanar PCBs. As based on a procedure tested and approved by the Commission (Schulte et al. 1991), a specific and sensitive method has been developed with which 12 further dioxin-like congeners can be determined in the plasma in addition to the 6 indicator congeners (Schettgen et al. 2008). Here, formic acid is added to 2 ml plasma, the PCB extracted with iso-octane, purified in a silicagel column, concentrated to the desired level and finally detected specifically using GC/MS. The detection limit for the individual congeners is 0.01 µg/l plasma. As internal standard for dioxin-like PCBs, ^{13}C-labeled analogues to the analytes are used to ensure accuracy of the measurement.

6 Background Exposure

In 1999, reference values for the high chlorinated PCBs 138, 153 and 180 as well as their sum in whole blood and plasma for different age groups were published by the Human Biomonitoring Commission at the Umweltbundesamt (Federal Environment Agency) (UBA 1999). The blood samples for measurement were taken in 1994 and 1995. The PCB reference values in plasma increased with age and their sums were 3.2 µg/l in the 18–25 year age group and 12.2 µg/l in the 56–65 year age group (see Table 1). The whole blood concentrations were lower by approximately one half in the higher age groups, though no such relationship could be discovered in the low age groups. No reference values were given for the low chlorinated and coplanar PCBs.

In 2003, an updating of the reference values was published by the the Human Biomonitoring Commission at the Umweltbundesamt (Federal Environment Agency) (UBA 2003 b). The blood samples on which this updating is based were taken between 1997 and 1999. Again a clear age dependence is found in these updated values. Although the blood samples were taken only a few years later, a slight decrease in exposure can already be found in the reference values for the younger age groups up to 49 years (whole blood concentrations for the sum of the PCBs from 1.1 µg/l (18–19 years) up to 7.8 µg/l (60–69 years)) (see Table 2). When interpreting these

Table 1 Reference values for PCBs 138, 153, 180 and their sum in human blood (Σ of PCBs = PCB 138 + PCB 153 + PCB 180) (UBA 1999)

Age [years]	PCB 138 [µg/l]		PCB 153 [µg/l]		PCB 180 [µg/l]		Σ of the PCBs [µg/l]	
	Whole blood	Plasma	Whole blood	Plasma	Whole blood	Plasma	Whole blood	Plasma
7–10	0.5	–	0.5	–	0.3	–	1.3	–
18–25	0.8	0.8	1.0	1.0	0.7	0.8	2.5	3.2
26–35	1.0	1.5	1.5	1.9	1.0	1.5	3.5	5.6
36–45	1.3	2.2	2.0	2.8	1.4	2.2	4.6	7.6
46–55	1.6	3.0	2.5	3.7	1.9	2.9	5.7	10.0
56–65	1.8	3.7	3.0	4.6	2.2	3.5	6.8	12.2

Table 2 Reference values for PCBs in whole blood (UBA 2003 b)

Age [years]	PCB 138 [µg/l]	PCB 153 [µg/l]	PCB 180 [µg/l]	Σ of the PCBs [µg/l]
18–19	0.4	0.6	0.3	1.1
20–29	0.6	0.9	0.6	2.0
30–39	0.9	1.6	1.0	3.2
40–49	1.4	2.2	1.6	5.1
50–59	1.7	2.8	2.1	6.4
60–69	2.2	3.3	2.4	7.8

data, the fact must be taken into consideration that no measurements in plasma were carried out in the environmental survey of 1998, from which these samples were obtained.

The PCB concentrations in the plasma of the general population as based on the six indicator PCBs as well as the 12 coplanar PCBs have been published (Schettgen 2011 b; Schettgen et al. 2008, 2009).

After a blood sampling in 2003, the concentrations of low chlorinated, high chlorinated and coplanar PCBs in the plasma of 105 not occupationally exposed persons aged 5 to 84 years were investigated (Schettgen et al. 2008). The data necessary to derive a BAR for the adult general population was calculated from the original data (n = 67, age 18–65 years) (Schettgen 2011 a). For the adult general population, the 95th percentiles for PCBs 28, 52 and 101 were at 0.03 µg/l, below the analytical detection limit of 0.01 µg/l and at 0.02 µg/l, respectively (see Table 3). In the same study, 95th percentiles of 1.3, 1.7 and 1.4 µg/l for PCB 138, 153 and 180 were found for the three high chlorinated indicator PCBs. Compared with the reference values

published by the Umweltbundesamt (Federal Environment Agency), a lower exposure than in the 1990s was therefore already found at this time (see Table 3).

In this study (Schettgen et al. 2008), volume-based data for the coplanar PCBs were also presented for the first time. The presence of PCBs 77, 81, 114, 123, 126 and 169 could not be detected in the samples. For the 6 coplanar PCBs which were demonstrated, the 95th percentiles are given in Table 3. In the same way as with the high chlorinated indicator PCBs, an age-dependency could also be found for the coplanar PCBs.

On the occasion of an indoor exposure to PCBs within a public building, a control group without occupational exposure was analyzed in a further study (Schettgen et al. 2009). Sampling was carried out in 2007 and 2008. The results for low chlorinated, high chlorinated and coplanar PCBs are given in Table 4.

The 95th percentiles for the low chlorinated PCBs were in the range of the analytical detection limit of the method. Compared with the values from 2003, a further decrease in exposure of the general population is once more found for the high chlorinated PCBs.

Table 3 Concentrations of low chlorinated, high chlorinated and coplanar PCBs in the plasma of non-exposed groups of persons aged 18–65 years (n = 67), blood sampling in 2003 (Schettgen 2011 a; Schettgen et al. 2008)

	PCB	Median [µg/l]	95th percentile [µg/l]	Maximum [µg/l]
Low chlorinated PCBs	PCB 28	0.01	0.03	0.05
	PCB 52	< 0.01	< 0.01	0.02
	PCB 101	< 0.01	0.02	0.03
High chlorinated PCBs	PCB 138	0.36	1.3	1.8
	PCB 153	0.5	1.7	2.5
	PCB 180	0.5	1.4	2.6
Coplanar PCBs	PCB 77	< 0.01	< 0.01	< 0.01
	PCB 81	< 0.01	< 0.01	< 0.01
	PCB 105	< 0.01	0.02	0.07
	PCB 114	< 0.01	< 0.01	0.03
	PCB 118	0.05	0.14	0.30
	PCB 123	< 0.01	< 0.01	< 0.01
	PCB 126	< 0.01	< 0.01	0.06
	PCB 156	0.06	0.16	0.28
	PCB 157	< 0.01	0.03	0.04
	PCB 167	0.02	0.06	0.09
	PCB 169	< 0.01	0.02	0.04
	PCB 189	0.01	0.03	0.09

Table 4 Concentrations of low chlorinated, high chlorinated and coplanar PCBs in the plasma of non-exposed groups of persons aged of 18–65 years (n = 97), blood sampling in 2007/2008 (Schettgen 2011 a; Schettgen et al. 2009)

	PCB	Median [µg/l]	95th percentile [µg/l]	Maximum [µg/l]
Low chlorinated PCBs	PCB 28	< 0.01	0.02	0.06
	PCB 52	< 0.01	< 0.01	0.03
	PCB 101	< 0.01	< 0.01	0.02
High chlorinated PCBs	PCB 138	0.26	0.92	2.44
	PCB 153	0.39	1.49	3.52
	PCB 180	0.30	1.15	3.19
Coplanar PCBs	PCB 77	< 0.01	< 0.01	< 0.01
	PCB 81	< 0.01	< 0.01	< 0.01
	PCB 105	< 0.01	0.02	0.14
	PCB 114	< 0.01	0.01	0.04
	PCB 118	0.04	0.13	0.73
	PCB 123	< 0.01	< 0.01	0.01
	PCB 126	< 0.01	0.02	0.06
	PCB 156	0.05	0.18	0.59
	PCB 157	< 0.01	0.03	0.10
	PCB 167	0.02	0.05	0.20
	PCB 169	< 0.01	< 0.01	0.01
	PCB 189	< 0.01	0.03	0.08

7 Evaluation of the BAR

The BAR for low chlorinated PCB congeners in plasma are based on the 95th percentile of the results in the general population, without referring to health effects. The fact that, among other factors, the reference value for background exposure can be influenced by age, sex, social status, living area and lifestyle factors must be taken into consideration. In more recent studies, the analytical detection limit for the PCBs is given as 0.01 µg/l blood (Schettgen et al. 2008, 2009).

As regards the low chlorinated PCBs, detectable PCB concentrations indicate an additional exposure. This can result, for example, from contaminated indoor air or from occupational handling of PCBs. In the two studies by Schettgen et al., the 95th percentile for PCB 28 was 0.02 and 0.03 µg/l, and the 95th percentile for PCB 52 was in both studies below the analytical detection limit of 0.01 µg/l. For PCB 101, the 95th percentile was 0.02 µg/l in one study and below the analytical detection limit of 0.01 µg/l in the second study (Schettgen et al. 2008, 2009).

For low chlorinated PCBs, BAR of

0.02 µg PCB 28 /l plasma

< 0.01 µg PCB 52 /l plasma

< 0.01 µg PCB 101/l plasma

are established.

There is no fixed sampling time.

For high chlorinated PCBs, only the older reference values in the plasma (1999) or whole blood (2003) from the Umweltbundesamt (Federal Environment Agency) exist (UBA 1999, 2003 b). Recent studies show that the present exposure profile of the general population for high chlorinated PCBs is clearly below the published reference values of the Federal Environment Agency (Schettgen et al. 2008, 2009). For the coplanar and high chlorinated PCBs, however, no sufficient data for derivation of a BAR are available at present.

Occupational exposure to high and low chlorinated PCBs becomes recognizable in a sensitive way when the BAR for low chlorinated PCBs is exceeded.

8 Interpretation of Data

Low chlorinated PCBs

Exposure to low chlorinated PCBs can principally be caused by contaminated indoor air both in an occupational environment as well as (more rarely) in a private context. An interpretation of the results is thus basically only possible in association with a qualified occupational medical and environmental medical anamnesis. Also, direct occupational handling can result in an additional exposure, for example in the disposal of aggregates containing PCBs.

High chlorinated and coplanar PCBs

An additional occupational exposure, resulting in an increase in high chlorinated PCBs, becomes demonstrable when the BAR for low chlorinated PCBs is exceeded. High chlorinated and coplanar PCBs are mainly taken up via food.

In women, with increasing age and after breast feeding, exposure may be lower than in men subjected to the same exposure.

9 References

Beck H, Mathar W (1985) Analysenverfahren zur Bestimmung von ausgewählten PCB-Einzelkomponenten in Lebensmitteln. Bundesgesundheitsblatt Gesundheitsforschung Gesundheitsschutz 28: 1–12

Bühler F, Schmid P, Schlatter C (1988) Kinetics of PCB elimination in man. Chemosphere 17: 1 717–1 726

Casey AC, Berger DF, Lombardo JP, Hunt A, Quimby F (1999) Aroclor 1242 inhalation and ingestion by Sprague-Dawley rats. J Toxicol Environ Health A 56: 311–342

Chemikalien-Verbotsverordnung (2010) Verordnung über Verbote und Beschränkungen des Inverkehrbringens gefährlicher Stoffe, Zubereitungen und Erzeugnisse nach dem Chemikaliengesetz in der Fassung der Bekanntmachung vom 13. Juni 2003 (BGBl. I S. 867), zuletzt geändert durch Artikel 5 Absatz 10 der Verordnung vom 26. November 2010 (BGBl. I S. 1 643)

Henschler D (Ed.) (1978) Chlorierte Biphenyle. Toxikologisch-arbeitsmedizinische Begründungen von MAK-Werten, 6. Lieferung, VCH, Weinheim

Humphrey H, Gardiner J, Pandya J, Sweeney A, Gasior D, McCaffrey R, Schantz S (2000) PCB congerer profile in the serum of human consuming Great Lakes fish. Environ Health Persp 108: 167–172

IARC (International Agency for Research on Cancer) (2011) Chemical agents and related occupations. IARC-Monographs on the Evaluation of Carcinogenic Risks of Chemicals to Humans, Vol. 100 F, Lyon

Rice DC (1997) Effect of postnatal exposure to a PCB mixture in monkeys on multiple fixed interval-fixed ratio performance. Neurotoxicol Teratol 19: 429–434

Rice DC, Hayward S (1997) Effects of postnatal exposure to a PCB mixture in monkeys on nonspatial discrimination reversal and delayed alternation performance. Neurotoxicol 18: 479–494

Ross G (2004) The public health implications of polychlorinated biphenyls (PCBs) in the environment. Ecotoxicol Environ Saf 59: 275–291

Ryan JJ, Levesque D, Panopio LG, Sun WF, Masuda Y, Kuroki H (1993) Elimination of polychlorinated dibenzofurans (PCDFs) and polychlorinated biphenyls (PCBs) from human blood in the Yusho and Yu-Cheng rice oil poisonings. Arch Environ Contam Toxicol 24: 504–512

Safe SH (1994) Polychlorinated biphenyls (PCBs): environmental impact, biochemical and toxic responses, and implications for risk assessment. Crit Rev Toxicol 24: 87–149

Schettgen T, Alt A, Angerer J, Drexler H, Kraus T (2008) Pilotstudie zur Hintergrundbelastung der deutschen Allgemeinbevölkerung gegenüber koplanaren polychlorierten Biphenylen. In: Baur X, Glensk E (Eds.) Dokumentation über die 48. Jahrestagung der Deutschen Gesellschaft für Arbeitsmedizin und Umweltmedizin e. V., Gentner Verlag, Stuttgart, 151–155 (CD)

Schettgen T, Alt A, Keller D, Preim D, Kraus T (2009) Innere Belastung von Beschäftigten eines PCB-kontaminierten Gebäudes mit dioxin-ähnlichen und nicht-dioxin-ähnlichen PCB Kongeneren. In: Kraus T (Ed.) Dokumentation über die 49. Jahrestagung der Deutschen Gesellschaft für Arbeitsmedizin und Umweltmedizin e. V., Gentner Verlag, Stuttgart, 325–329 (CD)

Schettgen T (2011 a) Mitteilung vom 27.04.2011 an die Kommission. Originaldaten der Studien von Schettgen et al. 2008 und Schettgen et al. 2009 für die erwachsene Allgemeinbevölkerung

Schettgen T, Gube M, Alt A, Fromme H, Kraus T (2011 b) Pilot study on the exposure of the German general population to dioxin-like and non-dioxin-like PCBs. Int J Hyg Environ Health 214: 319–325

Schulte E, Lewalter J, Ellrich D, Barchet R, Eisenmann R, Arbeitsgruppe „Analysen in biologischem Material" (1991) Polychlorierte Biphenyle. In: Angerer J, Schaller KH, Henschler D (Eds.) Analytische Methoden zur Prüfung gesundheitsschädlicher Arbeitsstoffe, Band 2: Analysen in biologischem Material, 10. Lieferung, VCH, Weinheim

Treon JF, Cleveland FP, Cappel JW, Atchley RW (1956) The toxicity of the vapors of aroclor 1242 and aroclor 1254. Am Ind Hyg Assoc 17: 204–213

Tryphonas H, Hayward S, O'Grady L, Loo JCK, Arnold DL, Bryce F, Zawidzka ZZ (1989) Immunotoxicity studies of PCB (Aroclor 1254) in the adult rhesus (Macaca Mulatta) monkey – preliminary report. Int J Immunopharmacol 11: 199–206

Tryphonas H, Luster MI, White KL Jr, Naylor PH, Erdos MR, Burleson GR, Germolec D, Hodgen M, Hayward S, Arnold DL (1991) Effects of PCB (Aroclor 1254) on non-specific immune parameters in rhesus (Macaca mulatta) monkeys. Int J Immunopharmacol 13: 639–648

UBA (Umweltbundesamt) (1999) Stoffmonographie PCB – Referenzwerte für Blut. Stellungnahme der Kommission „Human-Biomonitoring“ des Umweltbundesamtes. Bundesgesundheitsblatt Gesundheitsforschung Gesundheitsschutz 42: 511–521

UBA (Umweltbundesamt) (2003 a) Abschätzung der zusätzlichen Aufnahme von PCB in Innenräumen durch die Bestimmung der PCB-Konzentration in Plasma bzw. Vollblut. Stellungnahme der Kommission „Human-Biomonitoring“ des Umweltbundesamtes. Bundesgesundheitsblatt Gesundheitsforschung Gesundheitsschutz 10: 923–927

UBA (Umweltbundesamt) (2003 b) Aktualisierung der Referenzwerte für PCB-138, -153, -180 im Vollblut sowie Referenzwerte für HCB, β-HCH und DDE im Vollblut. Stellungnahme der Kommission „Human-Biomonitoring“ des Umweltbundesamtes. Bundesgesundheitsblatt Gesundheitsforschung Gesundheitsschutz 46: 161–168

Walkowiak J, Wiener JA, Fastabend A, Heinzow B, Krämer U, Schmidt E, Steingrüber H, Wundram S, Winneke G (2001) Environmental exposure to polychlorinated biphenyls and quality of the home Environment: effects on psychodevelopment in early childhood. The Lancet 358 (9293): 1 602–1 607

Weisglas-Kuperus N, Patandin S, Berbers GA, Sas TC, Mulder PG, Sauer PJ, Hooijkaas H (2000) Immunologic effects of background exposure to polychlorinated biphenyls and dioxins in Dutch preschool children. Environ Health Perspect 108: 1203–1207

WHO (World Health Organization) (2003) Polychlorinated biphenyls: human health aspects. Concise International Chemical Assessment Document 55, WHO, Genf

Wilhelm M, Ranft U, Krämer U, Wittsiepe J, Lemm F, Fürst P, Eberwein G, Winneke G (2008 a) Lack of neurodevelopmental adversity by prenatal exposure of infants to current lowered PCB levels: comparison of two German birth cohort studies. J Toxicol Environ Health A71: 700–702

Wilhelm M, Wittsiepe J, Lemm F, Ranft U, Krämer U, Fürst P, Röseler SC, Greshake M, Imöhl M, Eberwein G, Rauchfuss K, Kraft M, Winneke G (2008 b) The Duisburg birth cohort study: influence of the prenatal exposure to PCDD/Fs and dioxin-like PCBs on thyroid hormone status in newborns and neurodevelopment of infants until the age of 24 months. Mutat Res 659: 83–92

Wittsiepe J, Schrey P, Lemm F, Eberwein G, Wilhelm M (2008) Polychlorinated dibenzo-p-dioxins/polychlorinated dibenzofurans (PCDD/Fs), polychlorinated biphenyls (PCBs), and organochlorine pesticides in human blood of pregnant women from Germany. J Toxicol Environ Health A 71: 703–709

Authors: Th. Kraus, A.W. Rettenmeier
Approved by the Working Group: 25 January 2011