Human Receptor Activation By Aroclor 1260, A Polychlorinated Biphenyl Mixture

Supplementary Table 1

Selected PCB congeners present in Aroclor 1260 (≥1% of total composition). Table is adapted from www.atsdr.cdc.gov/toxprofiles/tp17-c4.pdf.

PCB congener	No. of chlorine	Ortho substitution	%
180	7	(Non-coplanar, di- <i>ortho</i>)	11.38
153	6	(Non-coplanar, di- <i>ortho</i>)	9.39
149	6	(Non-coplanar, tri- <i>ortho</i>)	8.75
138	6	(Non-coplanar, di- <i>ortho</i>)	6.54
187	7	(Non-coplanar, tri- <i>ortho</i>)	5.40
174	7	(Non-coplanar, tri- <i>ortho</i>)	4.96
170	7	(Non-coplanar, di- <i>ortho</i>)	4.11
101	5	(Non-coplanar, di- <i>ortho</i>)	3.13
151	6	(Non-coplanar, tri- <i>ortho</i>)	3.04
132	6	(Non-coplanar, tri- <i>ortho</i>)	2.90
141	6	(Non-coplanar, di- <i>ortho</i>)	2.62
177	7	(Non-coplanar, tri- <i>ortho</i>)	2.57
95	5	(Non-coplanar, tri- <i>ortho</i>)	2.45
163	6	(Non-coplanar, di- <i>ortho</i>)	2.42
183	7	(Non-coplanar, tri- <i>ortho</i>)	2.41
194	8	(Non-coplanar, di- <i>ortho</i>)	2.07
179	7	(Non-coplanar, tetra-ortho)	2.03
136	6	(Non-coplanar, tetra-ortho)	1.46
203	8	(Non-coplanar, tri- <i>ortho</i>)	1.40
110	5	(Non-coplanar, di- <i>ortho</i>)	1.33
146	6	(Non-coplanar, di- <i>ortho</i>)	1.15
171	7	(Non-coplanar, tri- <i>ortho</i>)	1.11
196	8	(Non-coplanar, tri- <i>ortho</i>)	1.09
135	6	(Non-coplanar, tri- <i>ortho</i>)	1.08



Aroclor 1260 activation of the human LXRα. HepG2 cells were transiently transfected with the expression plasmid pSG5-hLXRα and reporter plasmid pTK-LXRE-Luc. T0901317 (100nM, T) was used as a positive control. (A) Cells were exposed to Aroclor 1260 at 0, 5, 10, 15 and 20 µg/mL and luciferase induction was normalized and compared to DMSO-exposed cells (0 µg/mL Aroclor 1260). (B) Cells were exposed to 100 nM T or T plus Aroclor 1260 at 0, 5, 10, 15 and 20 µg/mL. The luciferase induction was normalized to that of cells exposed only to DMSO solvent carrier (as in A, not shown). Luciferase activity in cells exposed to T and Aroclor 1260 was compared to that of T-exposed cells. Data were normalized to luciferase activity in cells exposed only to DMSO and are expressed as mean ± SEM, n=4, * *p* <0.05.



Aroclor 1260 activation of the human FXR. HepG2 cells were transiently transfected with the expression plasmid pSG5-hFXR and reporter plasmid pGL3-IR1-Luc. GW4064 (0.5 μ M, GW) was used as a positive control. (A) Cells were exposed to Aroclor 1260 at 0, 5, 10, 15 and 20 μ g/mL and luciferase induction was normalized and compared to DMSO-exposed cells (0 μ g/mL Aroclor 1260). (B) Cells were exposed to 0.5 μ M GW or GW plus Aroclor 1260 at 0, 5, 10, 15 and 20 μ g/mL. The luciferase induction was normalized to that of cells exposed only to DMSO solvent carrier (as in A, not shown). Luciferase activity in cells exposed to GW and Aroclor 1260 was compared to that of GW-exposed cells. Data were normalized to luciferase activity in cells exposed only to DMSO and are expressed as mean ± SEM, n=4, * *p* <0.05.



Aroclor 1260 activation of the human PPARy. HepG2 cells were transiently transfected with the expression plasmid pCMV6-h PPARy and reporter plasmid pGL3-DR1-Luc. Pioglitazone (10 μ M, P) was used as a positive control. (A) Cells were exposed to Aroclor 1260 at 0, 5, 10, 15 and 20 μ g/mL and luciferase induction was normalized and compared to DMSO-exposed cells (0 μ g/mL Aroclor 1260). (B) Cells were exposed to 10 μ M P or P plus Aroclor 1260 at 0, 5, 10, 15 and 20 μ g/mL. The luciferase induction was normalized to that of cells exposed only to DMSO solvent carrier (as in A, not shown). Luciferase activity in cells exposed to P and Aroclor 1260 was compared to that of P-exposed cells. Data were normalized to luciferase activity in cells exposed only to DMSO and are expressed as mean ± SEM, n=4, * p <0.05.



Effects of Aroclor 1260 on target genes. Primary human hepatocytes were exposed to different concentrations of Aroclor 1260. After a 24 hour incubation, RNA was isolated and RT-PCR was performed. Relative mRNA levels of target genes were measured in Aroclor 1260-exposed cells namely (A) FAS, (B) SHP, and (C) CPT1A.