

Table 21

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Table 21. Developmental toxicity studies for PFCAs - PFOA

*Derived by contractor; ** calculated according to EFSA. (2012); NR – not reported; NA – not applicable.

Substance & / CAS no. / purity / reference	Strain & species / sex / no. of animals	Dose (mg/kg bw/day) / vehicle / route of admin / duration / Guideline (GL) study) / Good Laboratory Practice (GLP) status	PFAS concentration (µg/mL / µg/g)	Observed effects at LOAEL (Published NOAEL / LOAEL (mg/kg bw/day)	Study aut comment
				controls vs treated groups). Recovery (
				controls vs treated groups).		

					Females (mean ± SD):	
					↑ absolute and relative liver weight: data only provided in figures.	Histopatho changes, characteriz enlargeme disarrange of hepatoc cytoplasm nuclear migration, acidophilb inflammato cell infiltra and reduct glycogen storage, w observed i maternal n the PFOA exposed g Serum ALT AST were a significan increased.
					↑ AST and ALT: data only provided in figures.	
		0 or 1, Milli Q water,			Hepatocyte hypertrophy, disarrangement, cytoplasmic loss, nuclear migration, acidophil bodies and inflammatory cell infiltration.	
PFOA (ammonium salt)	Balb/c mice.	Gavage,				
CAS No. 3825-26-1	Pregnant females.	GD0 to parturition,	NR			Females: NA / 1*
98.4%.	8/dose.	Non-GL study,				
Xu et al. (2022)		GLP not stated.			↑ mRNA levels of genes related to inflammation: Tlr4, Myd88, Traf6, Rela, IL1b and Tnf.	Gestationa exposure t PFOA induc maternal h alterations through th liver axis.
					↑ apoptosis in liver: protein expression of PARP-1, cleaved caspase-3 and Bax.	
					Recovery not assessed.	

PFOA	CAS No. not given	Kunming mice. Pregnant females, 10/dose.	Distilled water, Gavage, GD1-7, Non-GL study, GLP not stated.	NR	Males (mean ± SD):	
					0, 1, 5, 10, 20 or 40	↑ liver index: data only provided in figures.
	99.2%.				↓ SOD and GSH-Px in liver: data only provided in figures	Females: NA / 1*
Zhang et al. (2021)					↑ MDA in liver: data only provided in figures.	
					Recovery not assessed.	

The present study results suggest that PFOA is hepatotoxic in a dose-dependent manner, and short-term exposure can cause swelling of the liver cells, which explains the increase in liver index.

The higher PFOA levels administered led to the lower levels of SOD and GSH-Px, and the greater the accumulation of MDA. The results suggested that oxidative damage is a potential mechanism of PFOA hepatotoxicity.