

Toxicological Summary**PFOS****Dietary Acute Northern Bobwhite Study**

Test Substance: Perfluorooctanesulfonate (PFOS)

Structure: 1-Octanesulfonic acid, 1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptafluoro-, potassium salt, CAS# 2795-39-3

Remarks: The test substance is a white powder (3M lot #217). The sample was stored under ambient conditions and purity was originally determined to be 90.49% by LC/MS, ¹H-HMR, ¹⁹F-NMR and elemental analyses techniques. In a subsequent analysis of the test substance, purity was determined to be 86.9%. The nominal and measured dietary PFOS concentrations in this summary are based on the 86.9% purity value.

The in-life phase report was originally completed in 2000, and reported values based on the 90.40% purity estimation. The report was revised in 2004 to reflect the subsequent revision in sample purity. This Toxicological Summary reflects these changes as well as including results from all of the associated reports of analyses of tissues collected during the in-life phase.

METHODS

Method: OPPTS 850.2200 (Draft), FIFRA Subdivision E. Section 71-2, OECD 205, and ASTM Standard E857-87 (1997)

Type: Dietary Acute

Year: 1999 in life test, 2000 for report, 2004 for amended report

Species: Northern Bobwhite (*Colinus virginianus*)

Test Phases: Acclimation – 10 days
Exposure period – 5 days
Post-exposure recovery period – 3 or 17 days (with the study ending on either Day 8 or Day 22, respectively)

Test quail Age: 10 days old at test initiation

Number of Replicates: There were six replicates in the control treatment, and two replicates in each test substance treatment. There were five quail per replicate (pen) for a total of 30 quail in the control treatment and 10 quail in each test substance treatment (which included eight dose groups). The animals were all exposed for five days, and then held for a recovery period. At Day 8, half the replicates from each treatment group (3 of

6 replicates from the control and 1 from each dose level) were sacrificed while the other half were continued in the study until Day 22.

Feed and Water: Food and water were provided *ad libitum* during acclimation and testing phases of the study. The quail did not receive any form of antibiotic treatment during either the acclimation or test phase of the study.

Analytical Monitoring: Test substance concentrations in feed and quail liver and serum were determined by reverse-phase HPLC and mass spectrometry.

Statistical Methods: LC50 (Lethal concentration that results in 50% mortality of a population for a given exposure time) and LT50 (Lethal time that results in 50% mortality of a population for a given dose) values were calculated by probit analysis using the Statistical Analysis System (SAS/STAT: PROC PROBIT). LD50 (Lethal dose, as measured by average daily intake, that results in 50% mortality of a population for a given exposure time) values were calculated by probit analysis (SAS/STAT:PROC PROBIT). Body weight data were compared by Dunnett's test using TOXSTAT software (Dunnett, 1955; Gulley, 1990). All other statistical evaluations were conducted with Excel worksheets including regressions with concentrations of PFOS in tissues.

Average Daily Intake (ADI) of PFOS for each treatment group was estimated on a pen basis and did not take into account potential differences between male and females. Food consumption and adult quail body weight data were averaged over the duration of the exposure period and the ADI was calculated as follows (Equation 1)

$$\text{ADI (mg/kg/day)} = \frac{\text{Average Feed Consumption (g/bird/day)}}{\text{Average Body weight (g/bird)}} \times \text{Feed Conc. (ppm PFOS)} \quad (\text{Equation 1})$$

Test Diet Preparation: The test substance was mixed directly into the feed ration by means of a Hobart mixer. No carrier was used. Nominal dietary concentrations of PFOS used in this study were 0 (control), 17.6, 35.1, 70.3, 141, 281, 562, and 1125 parts per million (ppm).

RESULTS

Measured Diet Concentrations: Wildlife International Ltd. determined concentrations of PFOS in the test diet. The mean percent recovery of the matrix-spiked feed was 94.7%, while the test feed samples had measured concentrations of PFOS that ranged from 92 to 119% of the nominal value. Measured mean concentrations of PFOS were: <LOQ, 18.7, 38.6, 71.5, 167, 279, 516, and 1148 ppm.

Mortalities and Clinical Observations: A summary of the percent mortality for each treatment group is given (Table 1).

Table 1. Cumulative treatment-related percent mortality of juvenile bobwhite quail exposed to PFOS

Nominal PFOS Conc. (ppm)	Measured PFOS Conc. (ppm)	Average Daily Intake ^A (mg/kg-day)	Days									
			Exposure Period					Recovery Period				22 ^B
			1	2	3	4	5	6	7	8		
Control	<LOQ ^C	<LOQ	0	0	0	0	0	0 ^D	0	0	0	
17.6	18.7	4.83	0	0	0	0	0	0	0	0	0	
35.1	38.6	8.52	0	0	0	0	0	0	0	0	0	
70.3	71.5	23.8	0	0	0	0	0	0	0	0	0	
141	167	44.7	0	0	0 ^E	0	0	0	11	11	11	
281	279	76.4	0	0	0	0	20	40	80	80	80	
562	516	193	0	0	10	20	50	80	100	100	100	
1125	1148	225	0	0	30	100	100	100	100	100	100	

^A Average daily intake (ADI; mg PFOS/kg body weight per day) based on Day 0-5 body weight and feed consumption data.

^B No mortalities occurred in any treatment levels in the extended recovery period from Day 8 to Day 22.

^C Limit of quantitation (LOQ) for PFOS in the diet was 1.10 ppm

^D One quail was euthanized on Day 6 after sustaining a broken leg on Day 5.

^E One quail was euthanized on Day 3 after sustaining a broken leg.

One quail in the control group of 30 was found with a broken leg on Day 5, and was subsequently euthanized on Day 6. Two other quail in the control group had foot lesions that were associated with cage mate aggression. All other quail in the control treatment were normal in appearance and behavior throughout the test. There were neither treatment-related mortalities nor overt signs of toxicity at PFOS concentrations $\leq$ 70.3 ppm in the diet.

In the 141 ppm treatment group, one bird was euthanized on Day 3 after sustaining a broken leg and there was one treatment-related mortality (11%) on Day 7. No other mortality was noted for this treatment. Two additional quail displayed clinical signs of toxicity (wing droop). However, by Day 9, the two quail had recovered and their appearance and behavior returned to normal. All other quail in the 141 ppm treatment had a normal appearance and behaved normally for the duration of the study.

In the 281 ppm treatment, 80% mortality (8/10 quail) was observed by Day 7 with no additional mortalities being observed for the duration of the test. Quail mortality occurred on Days 5, 6 and 7. Signs of toxicity were observed prior to death and included ruffled appearance, reduced reaction to stimuli (sound and motion), lethargy, wing droop, loss of coordination, lower limb weakness and convulsions. After Day 9, surviving quail had normal appearance and behavior for the duration of the study.

In the 562 ppm treatment, mortality was first observed on Day 3 and reached 100% by Day 7. Signs of toxicity observed prior to death included a ruffled appearance, reduced reaction to stimuli, lethargy, depression, wing droop, loss of coordination, lower limb weakness, lower limb rigidity, prostate posture and convulsions.

In the 1125 ppm treatment, mortality was first observed on Day 3 and reached 100% by Day 4. Signs of toxicity observed prior to death included a ruffled appearance, reduced reaction to stimuli, lethargy, depression, wing droop, loss of coordination, lower limb weakness, and lower limb rigidity.

Both LC50s and LT50s for PFOS were determined by probit analysis (Table 2). The dietary 8-day LC50 was 212 ppm PFOS with a 95% confidence interval of 158 to 278 ppm. The slope of the concentration-response curve was 7.04. Body weight and food consumption data collected during the 5-day exposure period were used to estimate an average daily intake of PFOS and an additional probit analyses was conducted. The LD50 based on Average Daily Intake (ADI) of PFOS was 61 mg PFOS/kg body weight/day with a 95% confidence interval of 48 to 77 mg PFOS/kg body weight/day. The slope of the dose-response curve was 8.88. The LT50, estimated with the 1125 ppm PFOS treatment data was 3.06 days. This result agreed with cumulative mortality data where 30% mortality was observed at day 3 and 100% mortality was observed at day 4 (Table 1).

Table 2. Estimates of the lethal concentration of PFOS (LC₅₀) and the lethal time (LT₅₀) of a population of juvenile northern bobwhite quails. ^A

Effect metric	Time	LC50 (ppm)	95% CI	Slope
LC50	Day 3	1,593	-	3.174
	Day 5	482	356-672	4.722
	Day 6	319	228-448	3.923
	Day 8	212	158-278	7.036
	Day 22	212	158-278	7.036
	Dose	LT50 (days)	95% CI	Slope
LT50	281 ppm	6.41	5.69-7.61	9.54
	562 ppm	4.78	4.17-5.48	8.29
	1125 ppm	3.06	-	60.5

^A All statistical measures estimated by Probit analysis (SAS/STAT: PROC PROBIT)

Body Weight: In comparison to controls, there were no apparent PFOS related effects on body weight for quails in the 17.6, 35.1 or 70.3 ppm treatments. However, quail in the 141, 281 and 562 ppm treatments had body weights that were significantly reduced in comparison to controls ($p < 0.01$) for Days 0-5. While there was a statistically significant reduction in body weight at PFOS concentrations > 70.3 ppm, this body weight reduction was no longer apparent by Day 15 (10 days post exposure). As was observed for body weight, significant treatment-related effects on body weight gain from Day 0 to 5 were observed at concentrations > 70.3 ppm PFOS. However, after Day 8, no treatment-related effects on body weight gain were noted at any concentration through study termination. Effects on body weight or body weight gain could not be determined for 281, 562 and 1125 ppm treatment groups after Day 8 due to most of the quail dying in these groups.

Table 3. Average body weight (g) of juvenile northern bobwhites exposed to various dietary concentrations of PFOS.

Treatment (ppm)	Body Weight Day 0 ^A	Body Weight Day 5	Body Weight Day 8	Body Weight Day 15	Body Weight Day 22
Control	20 ± 1	30 ± 4	38 ± 5	59 ± 10	82 ± 13
17.6	21 ± 1	31 ± 4	40 ± 5	68 ± 8	87 ± 7
35.1	20 ± 1	31 ± 3	39 ± 3	65 ± 5	89 ± 7
70.3	20 ± 1	30 ± 2	37 ± 3	60 ± 4	79 ± 4
141	20 ± 1	27* ± 3	33* ± 3	58 ± 3	79 ± 2
281	20 ± 1	18* ± 2	18* ± 4	35	55
562	20 ± 1	16* ± 2	na	na	na
1125	20 ± 1	na	na	na	na

^A Body weight is reported as means and standard deviations on a wet weight basis.

na = not applicable due to 100% mortality in treatment group.

* Statistically different from control group at p < 0.05 (Dunnett's t-test).

In a second set of analyses, the growth rates for each treatment group were calculated for various spans of time during the study, including 0-8 days, 8-22 days, and 0-22 days. Growth rate (1/day) was estimated with a general growth model (Equation 2) that was linearized and then fitted to the body weight data collected during the study.

$$W_A = W_0 \exp^{gt} \quad (\text{Equation 2})$$

Where: W_A is the body weight (g) of a mallard at time (t) of the study
 W_0 is the body weight (g) of a mallard at the onset of the study (t=0)
g is the overall growth rate (1/day)
t is time (days)

Data from the 562 and 1125 ppm PFOS treatments were not included, since all quail had died by Day 8. For Day 0-8, the growth rates of the quail in treatment groups of less than 70.3 ppm were not statistically different from the control growth rate (Table 4). In agreement with the body weight data, quail from the 141 and 281 ppm treatments had Day 0-8 growth rates that were reduced from that observed in the controls. However, after Day 8 (Day 3 of the recovery period) the quail in the 141 and 281 ppm treatments grew at a rate that was equivalent to that observed in quail from treatment groups less than or equal to 70.3 ppm PFOS. The overall growth rates for quail in the 17.6, 35.1, 70.3, 141, and 281 ppm treatments did not differ from the observed growth rates in the control group.

Table 4. Growth rates (1/day) for juvenile northern bobwhite quails exposed to PFOS. ^A

Days	Treatment group (ppm)					
	0	17.6	35.1	70.3	141	281
0 to 8	0.0803	0.0803	0.0839	0.0773	0.0623	-0.0140
8 to 22	0.0549	0.0555	0.0589	0.0542	0.0624	0.0798
0 to 22	0.0639	0.0681	0.0681	0.0628	0.0648	0.0526

^A Growth rates estimated by linear regression: (Ln Body weight (g)= growth rate (time) + y-intercept).

Feed Consumption: There were no PFOS-related effects on feed consumption during the exposure (Days 0-5) or recovery period (Days 6-22) of the study. A reduction in feed consumption was noted at the 281, 562 and 1125 ppm treatment groups during the exposure period (Days 0-5) but these reductions were not statistically significant. Overall, the juvenile quail averaged a consumption rate of approximately 0.298 g feed/g body weight/day for the study. While there was a slight decrease in the feed consumption rate during the recovery period, this result was observed in all treatments groups, including the control.

Table 5. Average feed consumption (FC) of juvenile northern bobwhite quail exposed to dietary concentrations of PFOS

Treatment (ppm)	FC	FC	FC
	Days 0-8 ^A	Days 8-22	Days 0-22
0 (Control)	0.325	0.182	0.218
17.6	0.311	0.173	0.211
35.1	0.317	0.227	0.263
70.3	0.401	0.241	0.280
141	0.347	0.219	0.227
281	0.376	0.230	0.242
562	0.681	NA ^B	NA
1125	0.200	NA	NA

^A Average feed consumption given as g/g body weight/day on a pen basis.

^B NA =Not applicable due to 100% mortality.

Gross Necropsy: Half of the surviving quail were subjected to necropsy on Day 8 and the remaining birds were necropsied on Day 22 following test termination. On Day 8, one bird in the 70.3 ppm treatment was noted with a slightly pale liver but this was not considered treatment-related. A single bird euthanized from the 281 ppm treatment was observed to have a lack of muscle mass and general thinness. Since these finding correlated with treatment-related effects on body weight, this necropsy finding was considered treatment related. The remaining quail that survived to Day 8 and 22 had necropsy findings that were unremarkable.

All quail that died during the study were subjected to a gross necropsy. For quail found dead during the study, the necropsy results included thin condition, loss of muscle mass, altered spleen color, autolysis of tissues and pale organs (Table 6). These findings were considered to be treatment related.

Table 6. Gross pathological observations from quail that died during the dietary acute study with PFOS.

Finding	Treatment Group (ppm)				
	Control (N=1)	141 (N=2)	281 (N=8)	562 (N=10)	1125 (N=10)
Abdominal cavity, some autolysis	0	0	2	2	4
Abdominal cavity, autolysis throughout	0	0	0	1	1
Crop, empty	0	0	2	5	2
Emaciated	0	0	2	5	8
Fractured leg	1	1	0	0	0
G.I. tract empty	0	0	1	1	0
Gizzard contents bile stained	0	0	2	5	1
Heart, anterior portion mottled white color	0	0	1	0	0
Heart pale	0	0	0	2	1
Intestinal contents tar-like	0	0	0	2	0
Keel, prominent	0	0	1	3	10
Kidneys, pale	0	0	0	2	0
Liver, pale and mottled	0	1	0	0	0
Loss of muscle mass	0	0	4	7	9
Muscular-skeletal, pale	0	1	0	0	0
Small in stature	0	0	3	0	0
Spleen, black	0	0	0	1	0
Spleen, small and pale or both	0	0	1	3	2
Spleen, dark, grey or grey-brown	0	0	0	1	2
Thin	0	0	0	4	2
Not Remarkable	0	0	1	0	0

Concentrations of PFOS in Tissues: Liver concentration data are presented separately for quail that died during the study and for those sacrificed at study termination. Serum data were also collected from quail at study termination. Liver samples collected from quail that died during Days 0 to 7 of the study were analyzed for PFOS content, and the data are presented in Table 7. During this period, mortality in quail was associated with liver concentrations that were greater than 110 µg/g PFOS. In the 1125 ppm PFOS treatment, this concentration was achieved by Day 3, while for quail in the 281 and 562 ppm treatments, this level was achieved by Day 5. Since birds were not sampled in a systematic manner, a pharmacokinetic analysis of the data could not be conducted to evaluate the uptake and disposition kinetics of PFOS in these quail.

Table 7. Measured concentrations of PFOS ($\mu\text{g/g}$) in the liver of select quail with treatment-related mortality during the acute dietary study. ^A

Day	Nominal PFOS Concentrations (ppm)			
	141	281	562	1125
3	No mortality	No mortality	Not analyzed	134 (N=1)
4	No mortality	No mortality	Not analyzed	126 (N=3)
5	No mortality	249 (N=1)	126 (N=1)	-
6	No mortality	202 (N=1)	235 (N=1)	-
7	111 (N=1)	139 (N=2)	111 (N=1)	-

^A All PFOS concentrations reported in units of $\mu\text{g/g}$ wet weight. The data represent an average for all quail that died in a treatment group on a specified day. The number in parenthesis is the number of samples analyzed and not the number of birds that were subjected to analysis. Some samples were composites.

Results from liver and serum analyses of samples collected from surviving quail on Day 8 and Day 22 show that PFOS remained present after the exposure period (Table 8). No mortality was associated with liver concentrations = $45.0 \mu\text{g/g}$ PFOS or serum concentrations = $41.2 \mu\text{g/ml}$ PFOS.

Table 8. Measured concentrations of PFOS in the liver and serum of bobwhite quail collected at Day 8 and Day 22 of the study. ^A

Dose Group (ppm)	Day 8		Day 22	
	Liver ($\mu\text{g/g}$)	Serum ($\mu\text{g/ml}$)	Liver ($\mu\text{g/g}$)	Serum ($\mu\text{g/ml}$)
Control	<LOQ ^B	<LOQ	0.21 ± 0.09	0.12 ± 0.25
17.6	18.5 ± 3.76	<LOQ	3.25 ± 0.81	5.89 ± 1.28
35.1	25.8 ± 4.65	3.04 ± 3.92	2.92 ± 1.56	7.27 ± 4.17
70.3	44.0 ± 4.46	41.2 ± 31.1	10.0 ± 3.29	26.2 ± 24.5
141	70.3	41.5 ± 49.1	25.8 ± 6.84	40.5 ± 14
281	NA	43.6	39.3	112

^A All concentrations reported as means and standard deviations on a wet weight basis.

^B Limit of quantitation (LOQ). LOQ= $0.005 \mu\text{g/ml}$ (serum), LOQ= $0.03 \mu\text{g/g}$ (liver)

NA= not applicable; samples were not collected

The average ratio of concentrations of PFOS in blood serum to that in liver (S:L) for quail was 0.515 on Day 8 and 2.42 on Day 22. The relatively great difference between the Day 8 and Day 22 S:L ratio was due in part to the low concentrations of PFOS in serum that was observed across all treatment groups. In particular, the serum PFOS concentrations in the 17.6 and 35.1 ppm treatment groups were less than the limit of quantitation thus reducing the S:L ratio. Finally, there were no significant differences in the S:L ratio between male and female mallards. The lack of significance was due in part to the small sample size and relatively great variability that was observed in both serum and liver PFOS concentrations for all treatments. Therefore these ratios were not reported separately.

The relationships between PFOS treatment dose and liver and serum PFOS concentrations measured on Day 8 are given:

$$\text{Liver PFOS } (\mu\text{g/g}) = 0.5087 * (\text{Dose, ppm}) - 3.0744 \quad R^2 = 0.9585$$

$$\text{Serum PFOS } (\mu\text{g/ml}) = 0.3399 * (\text{Dose, ppm}) - 0.6851 \quad R^2 = 0.3985$$

As with the S:L ratios, there were no sex specific differences in serum and liver concentrations on Day 8. This was due to the small sample size and relatively great variability observed in the serum and liver PFOS concentrations. For serum, the small sample size was due in part to the fact that serum concentrations were not measured in birds that died prior to Day 8. The variability in the data is also reflected in the relatively small coefficient of determination (R^2) for the serum PFOS concentrations to treatment concentration. The relationship between PFOS dose and tissue concentrations on Day 22 were not analyzed since growth and depuration may have significantly altered the relationships, and the information gained would not be applicable to environmental situations.

Post exposure, the concentration of PFOS in the liver decreased throughout the recovery period (Days 8-22). This decrease was most likely due to two factors, growth dilution and excretion. In addition, it was assumed that the biological mechanism for elimination is the same at lower body burden levels. To estimate the loss of PFOS from liver due to depuration from quail, a simple one-compartment model was employed (Equation 3). The following model evaluates the rate of loss of PFOS from liver:

$$C_A = C_0 * \exp^{-k't} \quad (\text{Equation 3})$$

Where: C_A is the concentration at time (t) during the elimination phase ($\mu\text{g/g}$)
 C_0 is the concentration at the onset of elimination ($\mu\text{g/g}$)
 k' is the overall elimination rate constant (day^{-1})
 t is time (days)

The overall elimination rate constant (k') was estimated by determining the slope of the concentration in liver plotted as a function of time during the elimination phase of the study (Days 8-22). It is important to remember that the estimated elimination rate constant takes into account losses from two concurrent processes, growth dilution and excretion. These two processes may differ in importance based on age, sex, and season. For this analysis, it was assumed that both processes acted in an independent but additive manner such that the over all elimination or loss could be represented as follows:

$$k' = k_2 + g$$

In this equation, g is the growth rate that was estimated from body weight measurements recorded during the study while k' was estimated as the overall rate of decrease in measured liver PFOS concentrations. Once k' and g were calculated, the elimination rate was estimated (k_2). Based on the measured liver PFOS concentrations and growth rates from Days 8 through 22 of study period, elimination rate constants were estimated for the 17.6, 35.1, 70.3, 141 and 281 ppm PFOS treatments (Table 9). The average elimination

rate of PFOS (based on liver concentrations) was 0.0542 day^{-1} with the half-life in liver estimated to be approximately 12.8 days.

Table 9. Growth rates, liver PFOS elimination constants and excretion rate constants for PFOS in bobwhite quail. ^A

Treatment (ppm)	Growth Rate (day^{-1})	k' (day^{-1})	k_2 (day^{-1})
0 (Control)	0.0549	--	--
17.6	0.0555	0.1253	0.0698
35.1	0.0589	0.1652	0.1063
70.3	0.0542	0.1103	0.0561
141	0.0624	0.0831	0.0207
281	0.0798	0.0980	0.0182

^A All rate constants were based on data derived from measurement made during the recovery period (Days 8 to 22).

Serum concentrations of PFOS did not behave in a manner that was consistent with that observed for liver PFOS concentrations. During the recovery period, mean concentrations of PFOS in the serum increased from <LOQ to $5.89 \mu\text{g/ml}$ and from 3.04 to $7.27 \mu\text{g/ml}$ in the 17.6 and 35.1 ppm treatments, respectively. In contrast, PFOS concentrations in serum of quail from the 70.3 ppm treatment decreased from 41.2 to 26.2 mg/L over the same time period. In addition to these inconsistencies, the variability in measured serum concentrations on Day 8 was relatively great ($\text{CV} > 100\%$) for all treatment groups, making it difficult to evaluate treatment related differences during the recovery period. As a result, kinetic analyses to estimate clearance times were not conducted with the serum data.

CONCLUSIONS:

The dietary 8-day LC50 was 212 ppm (CI= 158-278 ppm) while the 8-day LD50 based on ADI was 61 mg PFOS/kg body weight/day (CI= 48-77 mg PFOS/kg body weight/day). The no mortality concentration was 70.3 ppm (or 23.8 mg PFOS/kg body weight/day). Based on the reduction of body weight at Day 8, the No Observable Adverse Effect Concentration (NOAEC) was determined to be 70.3 ppm (or 23.8 mg PFOS/kg body weight/day). The Lowest Observable Adverse Effect Concentration (LOAEC) based on body weight at Day 8 was 141 ppm (equivalent to $44.7 \text{ mg PFOS/kg body weight/day}$). The 8-day No Observable Adverse Effect Level (NOAEL) and Lowest Observable Adverse Effect Level (LOAEL) values based on liver concentrations were 44.0 and $70.3 \mu\text{g/g}$, respectively. The 8-day NOAEL and LOAEL values based on serum concentrations were 41.2 and $41.5 \mu\text{g/ml}$, respectively. However, some caution is needed when interpreting serum PFOS values relative to conducting a risk assessment with monitoring data. The source of this caution is due to the fact that serum PFOS concentrations did not follow the trend observed with liver concentrations, wherein PFOS levels decreased during the recovery phase. It is assumed that the biological mechanism for elimination is the same at lower body burden levels for both liver and serum. However, the data indicate that this assumption may not be true and that the mechanisms

of elimination from these two tissues may not be similar. The NOAEL, LOAEL, and threshold values (geometric mean of NOAEL and LOAEL) are summarized (Table 10).

Table 10. NOAEC, LOAEC, and threshold doses (TD) of PFOS derived from a northern bobwhite quail acute feeding study ^A

Endpoint	NOAEL ^B	LOAEL	TD
Dietary (ppm)	70.3	141	103
Average Daily Intake (mg/kg/day)	23.8	44.7	32.6
Serum (µg/ml)	41.2	41.5	41.4
Liver (µg/g)	44.0	70.3	55.6

^A All values were based on the reduction of body weight in treated quail. Liver and serum PFOS concentrations are based on Day 8 data.

^B NOAEL and LOAEL values for diet and the ADI are reported as dietary concentrations. Serum and liver effect values are measured tissue values.

DATA QUALITY:

Reliability: Klimish ranking = 1

REFERENCES:

Dunnett, C.W. 1955. A multiple comparison's procedure for comparing several treatments with a control. J. Amer. Statis. Assoc. 50:1096-1121.

Gallagher, S.P., Casey, C.S., Beavers, J.B., and Van Hoven, R.L. 2004. PFOS: A dietary LC50 study with the Northern Bobwhite. Amended report, Wildlife International Ltd., Project NO.: 454-103.

Gulley, D.D. 1990. TOXSTAT Release 3.2. The University of Wyoming.

Hansen, K. 2001. Analytical Report of Data for PFOS Dietary LC50 Study in Northern Bobwhites (Sera and Liver). Revision 1. 3M Environmental Laboratory. Report No.: FACT-TOX-129.

SAS Institute. 1999. SAS/STAT User's Guide, Release 8.02 Edition, SAS Institute, Cary NC. USA.

Wagner, J.G. 1979. Fundamentals of Clinical Pharmacokinetics. Drug Intelligence Publications Inc., Hamilton, Ill. 461 p.

OTHER:

Last changed: 5/05/04