### *$TiO_2$ or $TiO_2$ NPs*

#### *Mice*

#### *Mohamed (2015)*

*Test material*: $TiO_2$ NPs, rutile/anatase (77/22%), 47 nm (TEM).

*Scoring for nanoscale considerations (dispersion and/or confirmation of internal exposure), assigned according* to Appendix E, was 2.

*Internal exposure*: quantitative measurement in tissues, methodology with important flaws

Male Swiss Webster mice, aged 10–12 weeks, were housed in plastic cages under standard lighting conditions (12-h day/night cycle) with free access to water and food. Animals were randomly divided into four groups: the negative control group (treated with distilled water); and three experimental groups (15 mice per group) administered by gavage with $TiO_2$ NPs suspensions at the doses of 5, 50 or 500 mg/kg bw per day for 5 days. Animals from each group were sacrificed at 24 h, 7 days and 14 days after the last treatment. According to the authors, routine histopathology showed dose-dependent effects in the stomach. At 5 mg/kg, submucosal oedema was noted after 24 h that developed into ulcerations and mucosal necrosis after one and two weeks, respectively. After exposure to 50 mg/kg bw per day, submucosal vasculitis, massively degenerated glands and both mucosal and submucosal necrosis was evident after 24 h, 7 days and 14 days, respectively. Submucosal congested blood vessels, focal areas of leucocytic cell infiltrations and necrotic glands with mononuclear cell infiltrations (i.e. the highest grade of damage) were seen with $TiO_2$ NPs at 500 mg/kg bw per day at all time points.

The Panel noted that histopathological changes were not sufficient reported as no incidences and/or severity score were provided.

The Panel considered that these data suggest an inflammatory response in the stomach after short-term bolus exposure to $TiO_2$ NPs (47 nm).

#### *Li et al. (2019)*

*Three test materials*: (1) $TiO_2$ NPs, anatase, 25 nm; (2) $TiO_2$ NPs, anatase, 50 nm; (3) $TiO_2$ NPs, anatase, 80 nm. Purity not reported for none of them.

*Scoring for nanoscale considerations (dispersion and/or confirmation of internal exposure), assigned according* to Appendix E, was 2 ($TiO_2$ NPs 25 nm) and 2 (($TiO_2$ NPs 50–80 nm).

*Internal exposure*: quantitative analysis in tissues, methodology with important flaws.

Male C57BL/6 mice (8 weeks old) were randomly divided into four groups (n = 28): control, $TiO_2$ NPs (25 nm), $TiO_2$ NPs (50 nm) and $TiO_2$ NPs (80 nm). Mice were administered by gavage $TiO_2$ NPs suspended in PBS with a dose of 1 mg/kg bw per day for seven consecutive days. The control group was given an equal volume of PBS. Fresh faeces were collected 2 h after dosing every day, and the distal colonic contents from each mouse were collected 24 h after the last dosing, for subsequent analysis.

Following 3-day or 7-day $TiO_2$ NPs administration, 6 mice per group were randomly selected and sacrificed for analysis. Fresh brain, heart, spleen, liver, kidney, heart, serum and faeces were collected and the samples were weighed.

Mice were randomly selected and sacrificed for the macroscopic and histological examination of tissues. Pathological examinations of the colon were performed in a blinded manner.

The purpose of this study was to study the interaction between $TiO_2$ NPs of different dimensions, gut microbiota and intestinal barrier function.

Results indicate that short-term ingestion of $TiO_2$ NPs (25 nm) (1 mg/kg bw per day for 7 days) led to colonic epithelial injury, reduced expression levels of tight junction proteins and reduced thickness of the 'luminal mucus layer'. This was associated with altered gut microbiota composition, with reduction in number of Bifidobacterium compared with controls.

Regarding immunological organs, histological findings were not reported.

The Panel considered that this study provides no relevant information on immunological effects of $TiO_2$ NPs.

### *Rats*

#### *Hashem et al. (2020)*

*Test material*: $TiO_2$ (from Sigma, no information on constituent particle size distribution nor crystalline form).

*Scoring for nanoscale considerations (dispersion and/or confirmation of internal exposure), assigned according* to Appendix E, was 3.

*Internal exposure* not examined.

Adult male Wistar rats were housed in steel mesh cages at 21–24 °C, 50–60% relative humidity and a 12 -h light-dark cycle. The rats were weighed and randomly distributed into three groups, that were gavaged with 0.5% hydroxypropyl methylcellulose (HMC), or $TiO_2$ suspended in HMC at doses of 20 or 40 mg/kg bw per day for 90 consecutive days.

At the end of treatment, the body weight of low- and high-dose groups was 25% lower than that of the control group. Standard haematology using automated counting revealed a statistically significantly dose-dependent leucopenia and thrombocytopenia, as well as eosinophilia and neutrophilia. In the spleens that were evaluated histopathologically in a blinded fashion, statistically significantly dose-dependent alterations were observed that included lymphoid necrosis, white pulp expansion and increased numbers of macrophages. A marked increase in $CD4^+$ and $CD8^+$ immunolabelling was noted in the spleen. In addition, IgG and IgM measured by ELISA were statistically significantly elevated in $TiO_2$-treated rats. Phagocytic activity measured by a modified colorimetric nitro blue tetrazolium assay as well as lysozyme expression and nitric oxide levels measured by ELISA were significantly reduced following $TiO_2$ exposure. Lymphocyte proliferation in response to PHA, measured by a lymphocyte transformation assay and using MTT as a proxy for cell number was statistically significantly reduced.

The Panel concluded that these findings taken together indicate that $TiO_2$-induced haematological and immunological alterations after exposure for 90 days at all dose tested.

### *$TiO_2$ NPs < 30 nm*

### *Mice*

#### *Yu et al. (2016)*

*Test material*: $TiO_2$ NPs, anatase, 5–6 nm (further information on characterisation from Hu et al., 2011).

*Scoring for nanoscale considerations (dispersion and/or confirmation of internal exposure), assigned according* to Appendix E, was 2.

*Internal exposure* not examined.

CD-1 (ICR) female mice were housed in stainless steel cages in a ventilated animal room at 24 ± 2°C with a relative humidity of 60 ± 10% and a 12-h light/dark cycle and were randomly divided in 4 groups of 20 animals each. Control (treated with 0.5% w/v HPMC) and three experimental groups (2.5, 5 and 10 mg/kg bw $TiO_2$ NPs suspensions in HPMC) were administered daily by gavage for 90 days. After 90 days, the mice were weighed and then anaesthetised. After sacrifice, the hearts were excised, weighed and cryopreserved at –80°C.

Inflammatory lesions and tissue damage were seen histopathologically, and were more pronounced at the mid and high doses. It was not reported whether the histopathology was performed blinded, but the results were corroborated with objective measures. The expression of NF-κB, and of pro-inflammatory cytokines TNF-α, IL-1β, IL-6 and IFN-γ expression were increased in a dose-dependent fashion (statistically significant increase up to 1.8-fold compared with the control); expression of the NF-κB inhibitor I-κB was decreased in a dose-dependent fashion (statistically significant decrease up to 1.55-fold compared with the control), as evidenced by western blotting.

The Panel considered that these data indicate an effect of $TiO_2$ NPs (5–6 nm) exposure at all dose levels tested, as evidenced by histopathological lesions, corroborated by intermediate endpoints indicating disturbance of intracellular ion homeostasis that were adrenergic receptors in the heart. These lesions are accompanied by increases in the expression of intermediate inflammatory endpoints.

The Panel noted effects on inflammatory mediators with $TiO_2$ NPs (5–6 nm) at all doses tested and corroborated by histopathological lesions.

### *Li et al. (2018)*

*Two test materials*: 1) $TiO_2$ NPs, anatase, 20 nm (in water, DLS); 2) $TiO_2$ NPs, rutile, edged with corners morphology (SEM), 15 nm (in water, DLS).

*Scoring for nanoscale considerations (dispersion and/or confirmation of internal exposure), assigned according* to Appendix E, was 4.

*Internal exposure*: quantitative measurement in tissues, methodology with important flaws.

Male C57BL/6 mice, 8 weeks old, were kept in a humidity-controlled room on a 12-h/12-h light–dark cycle with food and water available ad libitum. The animals were randomly divided into 3 groups (n = 10 mice per group). $TiO_2$ NPs (either rutile or anatase) suspended in distilled water were administered by gavage at a dose of 100 mg/kg bw per day for 28 days, whereas the control group received an equal volume of distilled water. There were no effects on body weight. Whereas particles were observed in the spleen, examination of H&E-stained samples of the spleen revealed no histopathological changes. No histopathological changes were seen recorded in other tissues examined (the lung, jejunum, kidney, liver or brain). In colon, the increased length of villi was increased and irregularly arranged epithelial cells were reported irregularly arranged after exposure to $TiO_2$ NPs.

Phylogenetic analysis was performed by PCR of faecal DNA, extracted using the FastDNA® Spin Kit for Stool (MP Biomedicals, Santa Ana, USA) and amplified by barcoded composition primers flanking the V4/V5 regions of the 16S rRNA gene. Rutile NPs had a more pronounced influence on the gut microbiota than anatase NPs. The most influenced phylum was Proteobacteria, which was significantly increased by rutile NPs but not by anatase NPs. At the genus level, Rhodococcus was enriched by rutile NPs, Prevotella was significantly decreased by both the $TiO_2$ NPs.

The Panel considered that these data support an effect of $TiO_2$ NPs on the microbiota, but as no immunological parameters other than the histopathology of the spleen were included in this study, any consequence(s) associated with these changes in terms of inflammation and the immune system remain uncertain.

### *Zhang et al. (2020)*

*Test material*: $TiO_2$ NPs, 21 nm (TEM), crystalline form and purity unknown.

*Scoring for nanoscale considerations (dispersion and/or confirmation of internal exposure), assigned according* to Appendix E, was 4.

*Internal exposure* not examined.

C57BL/6J mice, aged 7 weeks, were maintained in the controlled conditions with temperature 23 ± 1°C and humidity 55 ± 10% on a 12-h light/dark cycle, with free access to a standard rodent diet and tap water. Thirty mice were randomly distributed over a vehicle group. Mice were treated with either vehicle or a suspension of $TiO_2$ NPs at 150 mg/kg bw per day by gavage for 30 days. Microbiota were evaluated by 16S ribosomal RNA (rRNA) gene sequencing in the faecal samples. Total genomic DNA was extracted from the faecal samples and the bacterial 16S rRNA were amplified by PCR. Subsequently PCR products were quantified using the QuantiFluor™-ST Blue Fluorescence Quantification. The results show that oral exposure to $TiO_2$ NPs resulted in significantly changed richness and composition of the gut microbiota. No changes in parameters indicating inflammation (IL-6 and IL-1β) in either intestines or brain were observed.

The Panel considered that exposure to $TiO_2$ NPs (21 nm) leads to changes in the microbiota composition, but the study does not indicate a local or systemic inflammatory action.

### *Rats*

### *Chen et al. (2015a)*

*Test material*: $TiO_2$ NPs, anatase 24 nm (TEM).

*Scoring for nanoscale considerations (dispersion and/or confirmation of internal exposure), assigned according* to Appendix E, was 2.

*Internal exposure* not examined.

Four-week-old healthy Sprague–Dawley rats were randomised into experimental and control groups, with 5 male and 5 female rats in each group. Suspensions in ultrapure water of $TiO_2$ NPs (0, 2, 10 and

50 mg/kg bw per day), glucose (1.8 g/kg bw per day) and $TiO_2$ NPs (0, 2, 10 and 50 mg/mg bw per day) + glucose (1.8 g/kg bw per day) were gavaged daily for 30 or 90 consecutive days.

Regarding the effects on spleen and white blood cells in animals exposed to $TiO_2$ NPs alone, no significant histopathological changes were observed in the spleen in all groups. On the contrary, increases in white blood cells parameters (white blood cell counts and granulocytes) were observed in female rats after exposure to $TiO_2$ NPs 50 mg/kg bw per day for 90 days and among male rats exposed to $TiO_2$ NPs 50 mg/kg bw per day for 30 (white blood cells counts, lymphocytes, monocytes absolute numbers and in the percentage of lymphocytes and granulocytes) and 90 days (percentage of monocytes); and a decrease in the while blood cells at 90 days in rats exposed to 10 mg/kg bw per day. The Panel considered that the increase in leucocytes may suggest an inflammatory response induced by $TiO_2$ NPs (24 nm) at the highest dose tested (50 mg/kg bw per day).

***Chen et al. (2019)***

*Test material:* $TiO_2$ NPs, anatase, 29 nm (SEM).

*Scoring for nanoscale considerations (dispersion and/or confirmation of internal exposure), assigned according to Appendix* E, was 2.

*Internal exposure* not examined.

Three-week-old male Sprague–Dawley rats, 6 per group, were administered via gavage with $TiO_2$ NPs at doses of 0, 2, 10, 50 mg/kg bw per day for 30 days. Morphology of colon was evaluated by routine histopathology and TEM. Serum levels of IL-6 and TNF-α were measured, as well as oxidative stress markers in colon tissue homogenates.

Histopathologically, reduced numbers of goblet cells were found as a result of exposure, as well as inflammatory infiltration, while in serum increased IL-6 expression was observed.

***Grissa et al. (2020)***

*Test material*: $TiO_2$ NPs, anatase, 5–12 nm (TEM, XRD).

*Scoring for nanoscale considerations (dispersion and/or confirmation of internal exposure), assigned according* to Appendix E, was 2.

*Internal exposure*: quantitative in tissues; methodology with important flaws.

Male Wistar rats were administered by gavage (aqueous suspension; volume of 10 mL/kg bw) with $TiO_2$ NPs (five times/week; 8 weeks) after being randomised over 4 groups (n = 8/group): 0 (control), 50, 100 and 200 mg $TiO_2$ NPs/kg bw per day. One day after the last $TiO_2$ NPs treatment, rats were euthanised and fresh brain from each rat was excised and weighed. A part of the frontal lobe of the left cerebral hemispheres was homogenised for biochemical assays, and the other part of the frontal lobe was used for the quantification of Ti. TNF-α and nitric oxide levels were measured using commercially available kits.

A statistically significant dose-related increase in the level of NO in 100 and 200 mg/kg bw per day $TiO_2$ NPs groups was observed together with a statistically significant increase in brain TNF-α in 200 mg/kg bw per day $TiO_2$ NPs group. The increase was dose-related for both parameters.

The Panel noted changes for the above-mentioned inflammatory markers at doses starting from 100 mg $TiO_2$ NPs (5–12 nm)/kg bw per day.

# Appendix I – Analysis of Ti-concentration in blood and urine in the EOGRT study

### *Sample collection*

Urine in F2 pups was collected via sterile bladder punctuation, whereas the urine in F0 and cohort 1A and 1B animals were collected by using metabolic cages. The applicant assumes that the Ti-levels in urine that were collected via metabolic cages was the result of contamination of urine from faeces in the metabolic cages.

### *Analysis Ti-concentration in blood and urine*

Blood and urine were microwave digested with sulfuric acid, followed by analysis with ICP-MS/MS. A comparison between analysis by ICP-MS/MS and ICP-OES was performed and it was concluded the use of $H_2SO_4$ in sample preparation did not result in interferences in the ICP-MSMS analysis. Background concentrations in blood and urine were determined and used to correct the Ti-concentrations. The instrumental LOD varied between 0.001 and 0.014 μg/L for blood, and between 0.002 and 0.026 μg/L for urine. The instrumental LOQ varied between 0.004 and 0.043 μg/L for blood, and between 0.006 and 0.077 μg/L for urine. Method LODs and LOQs were estimated to be substantially greater.

### *Assessment of background concentration in blood and urine*

The background concentrations in rat blood method blanks varied between 0.008 and 0.046 μg/L, and between 0.010 and 0.036 μg/g for urine. As uncorrected median Ti concentrations in blood for exposed animals ranged between 0.01 and 0.542 μg/g, and between 0.034 and 1.017 μg/g for urine, the background consisted of a considerable fraction of the Ti determined in many blood and urine samples. Together with the considerable variability in background concentrations and the magnitude of the estimated method LODs and LOQs, this suggests there is some uncertainty in the Ti-concentrations of blood and urine, especially in the lower values.

### *Kinetic profile*

For cohort F2, the Ti-concentration blood (n = 10/group) was increased in dose group 300 mg/kg bw/d and further increased in dose group 1,000 mg/kg bw/d. The Ti-concentration in blood for dose group 1,000 mg/kg bw/d in cohort F0 seems to be slightly increased, whereas cohorts 1A and 1B did not show a clear increase in Ti blood levels. The Panel noted that there are a few rats that showed an increased blood concentration that are considered outliers.

Urine (n = 10/group) Ti-levels generally increase in the exposed groups as compared to the control group. The variation between animals is large. For cohort F2, urine concentrations are low and there is not a clear increase with dose.

Increased Ti-levels in blood indicate that some Ti from the diet must have been systemically available (Figure I.1). Also Ti in urine suggests that Ti from the diet is absorbed, but for the cohorts F0, 1A and 1B, contamination of urine via faeces caused by the sampling of the excreta could have contributed to the Ti concentration in urine.

An increase in Ti concentrations in blood in the F2 cohort (dose group 300 and 1,000 mg/kg bw per day) at post-natal day 4–7 has been noted. The Panel considered that the pups may have been exposed to $TiO_2$ via inhalation of dust from powdered feed containing E171. However, the Panel concluded that the pups were primarily exposed to E171 in utero (i.e. via the placenta) and that pup Ti blood levels were able to accumulate to detectable levels soon after birth due to limited foetal excretion. Accordingly, although the blood concentrations of Ti in F1 females were not measurable, the Panel concluded that E171 was systemically available.

Small particles generally do not remain long in the blood stream (Landsiedel et al., 2012; ISO/TR, 2019). They are taken up by tissues rich in macrophages, such as liver and spleen. The blood kinetics therefore have limited value for assessing the toxicokinetic behaviour. Yet, the presence of Ti in blood suggests that some of the $TiO_2$ particles in E 171 were absorbed from the GI tract.

It should be noted that cohort F2 showed the highest blood concentration and the lowest urine concentration (Figure I.2). The low urine concentration as compared to the other cohorts may be explained by the manner of collecting urine, i.e. bladder punctuation for cohort F2 vs. metabolic cages for the other cohorts. In the latter case, contamination from faeces may have occurred.

The Panel considered that the blood concentrations in the F2 pups on PND 4–7 at concentrations clearly above the LOQ indicate intrauterine exposure and hence internal exposure of the F1 mothers. The percentage of the systemically available fraction is not known and the variability of the concentration data is high: Nevertheless, the F2 pup blood concentrations showed a dose-dependent increase, suggesting that the systemically available fraction in the F1 mothers also increased and that absorption from their GIT did not decrease with increasing dose of E 171 (i.e. any agglomeration of E 171 with increasing dose did not impact on absorption).

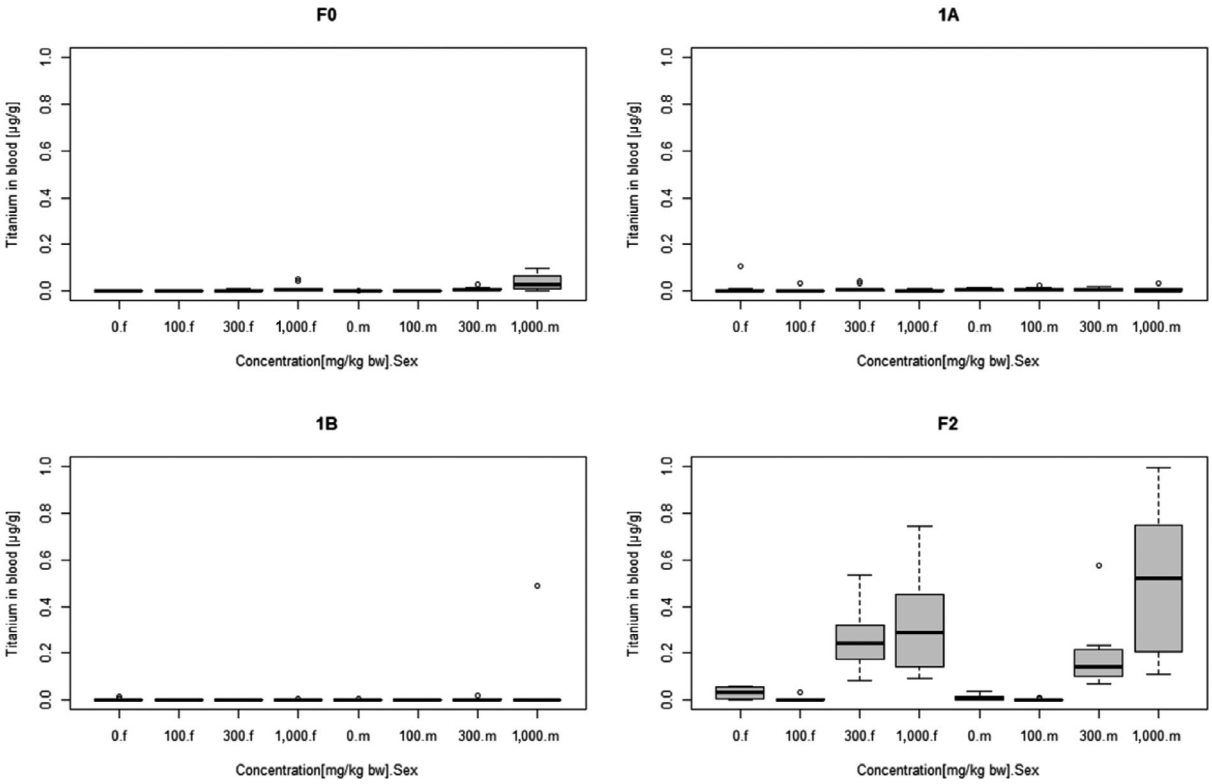


**Figure I.1:** Boxplots comparing Ti levels in blood (µg/g blood) for treatment concentration and sex combinations for each generation and cohort (Documentation provided to EFSA No 18) (for further explanation see text above)

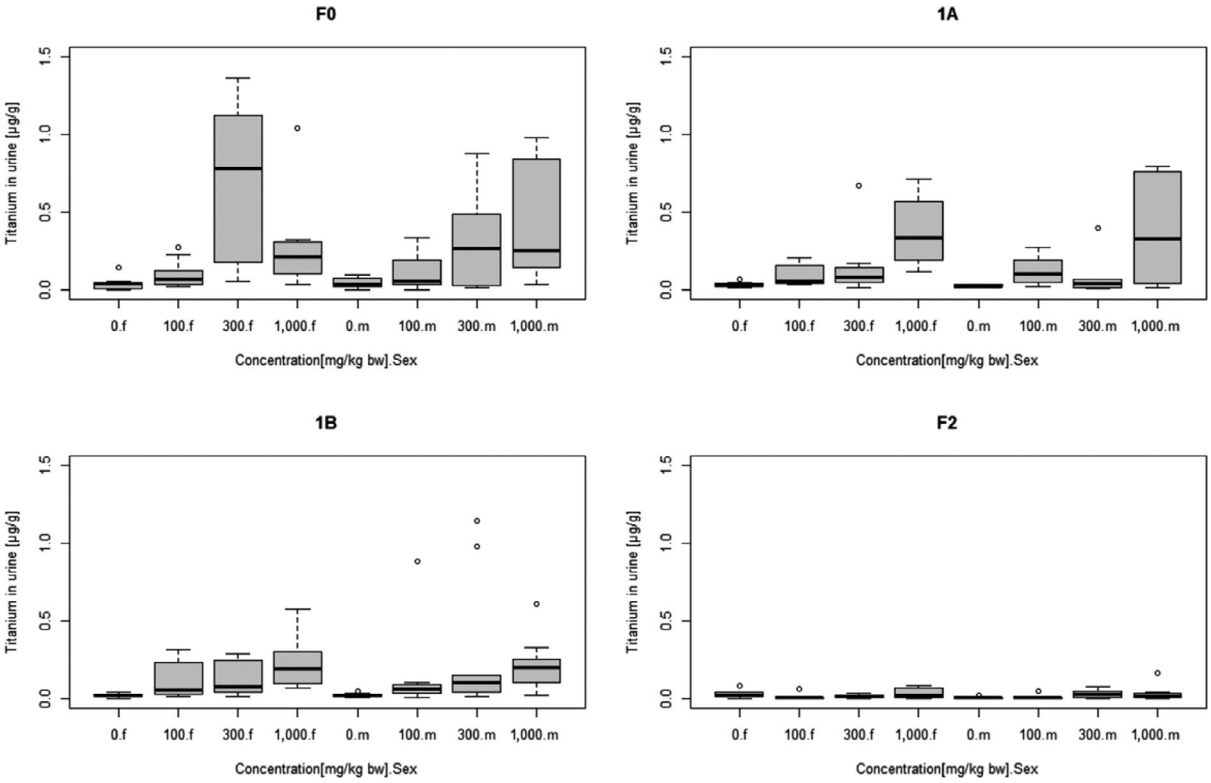


**Figure I.2:** Boxplots comparing Ti levels in urine (µg/g blood) for treatment concentration and sex combinations for each generation and cohort (Documentation provided to EFSA No 18)

## Appendix J – New *in vitro* genotoxicity studies

## Appendix K – New *in vivo* genotoxicity studies

## Appendix L – *In vitro* genotoxicity studies considered in the re-evaluation of E 171 (EFSA ANS Panel, 2016)

## Appendix M – *In vivo* genotoxicity studies considered in the re-evaluation of E 171 (EFSA ANS Panel, 2016)

## Appendix N – *In vitro* genotoxicity studies from OECD dossier (OECD, 2016)

## Appendix O – *In vivo* genotoxicity studies from OECD dossier (OECD, 2016)

## Appendix P – Genotoxicity studies submitted by Interested Business Operators

## Appendix Q – Concentration levels of E 171 used in the exposure assessment scenarios (mg/kg or mL/kg as appropriate)

## Appendix R – Number and percentage of food products labelled with E 171 out of the total number of food products present in the Mintel GNPD per food subcategory between 2016 and 2021

## Appendix S – Summary of total estimated exposure of E 171 from its use as a food additive for the maximum level exposure scenario and the refined exposure assessment scenarios per population group and survey: mean and 95th percentile (mg/kg bw per day)

## Appendix T – Summary of total estimated exposure of E 171 from its use as a food additive for the food supplements consumers only exposure assessment scenarios per population group and survey: mean and 95th percentile (mg/kg bw per day)

## Appendix U – Main food categories contributing to exposure to E 171 using the maximum level exposure assessment scenario and the refined exposure assessment scenarios (> 5% to the total mean exposure)

## Appendix V – Summary of total estimated exposure of E 171 from its use as a food additive considering reported use levels and analytical data (mg/kg bw per day)

Appendix J–V can be found in the online version of this output (in the 'Supporting information' section): https://efsa.onlinelibrary.wiley.com/doi/10.2903/j.efsa.2021.6585

# Appendix W – Reported data on the analysis of pristine E 171 (Verleysen et al., 2020, 2021)

Number- and mass-based percentages of $TiO_2$ particles smaller than threshold x are reported in Tables W.1 and W.2

**Table W.1:** Number percentage of particles

| x (nm) | Number % of particles with minimum Feret diameter smaller than threshold x | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Sample 1 | Sample 2 | Sample 3 | Sample 4 | Sample 5 | Sample 6 | E 171-A[(a)] | E 171-B[(a)] | E 171-C[(a)] | E 171-D[(a)] | E 171-E[(a)] | E 171-F[(a)] |
| 10 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 20 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 30 | 1 | 0 | 0 | 1 | 1 | 1 | 0 | 0 | 0 | 0 | 0 | 0 |
| 40 | 6 | 1 | 2 | 2 | 8 | 3 | 1 | 2 | 1 | 1 | 2 | 0 |
| 50 | 13 | 4 | 5 | 5 | 16 | 7 | 3 | 6 | 3 | 1 | 6 | 2 |
| 60 | 22 | 9 | 12 | 13 | 25 | 15 | 6 | 15 | 9 | 2 | 14 | 3 |
| 70 | 36 | 21 | 24 | 25 | 37 | 29 | 11 | 29 | 19 | 4 | 24 | 7 |
| 80 | 50 | 35 | 39 | 38 | 51 | 44 | 20 | 44 | 31 | 8 | 40 | 9 |
| 90 | 63 | 50 | 54 | 52 | 63 | 58 | 30 | 58 | 44 | 13 | 54 | 14 |
| **100** | **74** | **64** | **67** | **65** | **73** | **71** | **40** | **70** | **56** | **18** | **65** | **20** |
| 110 | 81 | 75 | 77 | 74 | 80 | 79 | 50 | 79 | 67 | 24 | 75 | 28 |
| 120 | 87 | 83 | 85 | 82 | 86 | 85 | 60 | 85 | 75 | 31 | 83 | 35 |
| 130 | 92 | 89 | 90 | 89 | 91 | 90 | 68 | 90 | 82 | 38 | 88 | 43 |
| 140 | 94 | 92 | 94 | 93 | 94 | 94 | 77 | 93 | 88 | 44 | 91 | 51 |
| 150 | 97 | 95 | 96 | 96 | 96 | 96 | 83 | 95 | 92 | 50 | 95 | 59 |
| 160 | 98 | 97 | 98 | 97 | 98 | 97 | 88 | 97 | 95 | 57 | 97 | 67 |
| 170 | 98 | 98 | 99 | 99 | 98 | 98 | 91 | 98 | 97 | 65 | 98 | 76 |
| 180 | 99 | 99 | 99 | 99 | 99 | 99 | 93 | 99 | 98 | 72 | 99 | 81 |
| 190 | 99 | 100 | 100 | 100 | 99 | 99 | 96 | 99 | 99 | 76 | 99 | 87 |
| 200 | 100 | 100 | 100 | 100 | 100 | 100 | 97 | 100 | 99 | 83 | 100 | 91 |
| 250 | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 95 | 100 | 99 |
| 300 | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 98 | 100 | 100 |
| 400 | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 100 |
| 500 | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 100 |

(a): Sample claimed to be the same of the E 171 for which data were evaluated in EFSA FAF Panel (2019).

**Table W.2:** Mass percentage of particles

| x (nm) | Mass % of particles with minimum Feret diameter smaller than threshold x | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Sample 1 | Sample 2 | Sample 3 | Sample 4 | Sample 5 | Sample 6 | E 171-A[(a)] | E 171-B[(a)] | E 171-C[(a)] | E 171-D[(a)] | E 171-E[(a)] | E 171-F[(a)] |
| 10 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 20 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 30 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 40 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 50 | 1 | 0 | 0 | 0 | 1 | 1 | 0 | 1 | 0 | 0 | 0 | 0 |
| 60 | 3 | 1 | 2 | 2 | 3 | 2 | 0 | 2 | 1 | 0 | 2 | 0 |
| 70 | 7 | 4 | 5 | 5 | 7 | 6 | 1 | 6 | 3 | 0 | 4 | 0 |
| 80 | 14 | 9 | 11 | 10 | 13 | 12 | 3 | 12 | 7 | 0 | 10 | 1 |
| 90 | 23 | 18 | 20 | 18 | 22 | 21 | 6 | 20 | 13 | 1 | 18 | 2 |
| **100** | **33** | **29** | **32** | **29** | **31** | **32** | **10** | **30** | **20** | **2** | **27** | **3** |
| 110 | 44 | 41 | 43 | 39 | 41 | 43 | 15 | 41 | 30 | 3 | 38 | 6 |
| 120 | 54 | 52 | 56 | 50 | 52 | 52 | 23 | 50 | 39 | 5 | 49 | 9 |
| 130 | 64 | 63 | 66 | 64 | 61 | 62 | 30 | 59 | 49 | 8 | 58 | 13 |
| 140 | 71 | 70 | 75 | 72 | 70 | 73 | 40 | 68 | 59 | 11 | 66 | 19 |
| 150 | 80 | 79 | 81 | 82 | 78 | 78 | 49 | 73 | 69 | 15 | 75 | 27 |
| 160 | 84 | 84 | 88 | 87 | 84 | 84 | 58 | 80 | 77 | 20 | 82 | 35 |
| 170 | 88 | 90 | 92 | 91 | 88 | 88 | 65 | 85 | 84 | 27 | 86 | 47 |
| 180 | 92 | 93 | 95 | 95 | 92 | 92 | 70 | 90 | 89 | 34 | 90 | 55 |
| 190 | 94 | 96 | 96 | 96 | 95 | 95 | 77 | 94 | 92 | 40 | 93 | 65 |
| 200 | 95 | 98 | 99 | 98 | 96 | 97 | 82 | 96 | 95 | 49 | 96 | 74 |
| 250 | 100 | 99 | 100 | 100 | 100 | 100 | 95 | 100 | 100 | 75 | 99 | 96 |
| 300 | 100 | 100 | 100 | 100 | 100 | 100 | 98 | 100 | 100 | 88 | 100 | 100 |
| 400 | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 99 | 100 | 100 |
| 500 | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 100 |

(a): Sample claimed to be the same of the E 171 for which data were evaluated in EFSA FAF Panel (2019).