

Article

Chronic Dietary Exposure to Pesticide Residues from Apples, Oranges, and Bananas in the HELLANS 2024–2025 Adult Cohort: A Deterministic and Probabilistic Screening Assessment

Konstantinos B. Simoglou ^{1,2,*} , Zisis Vryzas ³, Eleftherios Alissandrakis ²  and Emmanouil Roditakis ^{2,4,*} 

¹ Department of Quality, Phytosanitary Inspections & Food Safety, Rural Economy Directorate, Regional Unit of Drama, 66133 Drama, Greece

² Department of Agriculture, School of Agricultural Sciences, Hellenic Mediterranean University, Estavromenos, 71004 Heraklion, Greece; ealiss@hmu.gr

³ Pesticide Science Laboratory, School of Agriculture, Faculty of Agriculture, Forestry and Natural Environment, Aristotle University of Thessaloniki, 54124 Thessaloniki, Greece; zvryzas@agro.auth.gr

⁴ Institute of Agri-Food and Life Sciences, Hellenic Mediterranean University Research Centre, 71410 Heraklion, Greece

* Correspondence: simoglouk@pamth.gov.gr (K.B.S.); eroditakis@hmu.gr (E.R.)

Abstract

Chronic dietary exposure to pesticide residues links environmental contamination and food safety. This study assessed substance-specific and cumulative exposure from apples, oranges, and bananas among 1318 adults participating in the HELLANS 2024–2025 cohort. Consumption was estimated from a 24-h dietary recall and a food-frequency questionnaire (FFQ), while residue data derived from the Greek national monitoring for 2022–2024. Deterministic chronic exposure was calculated using EFSA PRIMo revision 3.1. Endpoint-specific total margins of exposure (MOETs) were estimated for chronic brain and/or erythrocyte acetylcholinesterase inhibition and hypothyroidism under lower-bound, middle-bound, and upper-bound scenarios. Participant-level cumulative exposure was calculated directly for all 1318 participant records, while uncertainty in monitoring-derived mean concentrations was quantified using 5000 stratified outer-bootstrap replicates. All calculable deterministic estimates were below 100% of the ADI; the highest was phosmet (2.34% ADI). Under the UB scenario, P95 and P99 MOETs were 1324 and 1070 for acetylcholinesterase inhibition and 3944 and 3194 for hypothyroidism; the corresponding P99.9 screening-tail estimates were 928 and 2854. The 95% monitoring-sample uncertainty intervals for the UB P99.9 estimates were 897–954 and 2740–2957, respectively. All point estimates and lower 95% monitoring-sample uncertainty limits exceeded the screening value of 100. Sensitivity analyses using midpoint FFQ frequencies and hypothetical reductions in orange and banana residues did not alter the risk interpretation. Overall, the results did not indicate a chronic screening-level concern from the three assessed fruits within the study cohort, a volunteer sample with high educational attainment, but should not be extrapolated to total dietary exposure or the Greek population.



Academic Editor: Paolo Pastorino

Received: 10 August 2026

Revised: 15 October 2026

Accepted: 16 October 2026

Published: 20 October 2026

Copyright: © 2026 by the authors.

Licensee MDPI, Basel, Switzerland.

This article is an open access article distributed under the terms and conditions of the [Creative Commons Attribution \(CC BY\)](https://creativecommons.org/licenses/by/4.0/) license.

Keywords: outer bootstrap; EFSA PRIMo; total margin of exposure; cumulative risk assessment; chronic dietary exposure; pesticide monitoring; left-censored data

1. Introduction

Pesticides contribute substantially to crop protection, agricultural productivity, and food security [1]. However, their use may result in residues in food commodities, creating

a pathway between environmental contamination and human exposure [2]. For the general population, diet—particularly the consumption of fruits, vegetables, and other plant-derived foods—is a major route of exposure to pesticide residues [3]. Nevertheless, the detection of a residue does not necessarily imply a consumer health risk, which depends on the magnitude of exposure relative to an appropriate toxicological reference value.

Dietary risk assessments have traditionally evaluated active substances individually by comparing estimated exposure with health-based guidance values such as the acceptable daily intake (ADI). Consumers may, however, be exposed simultaneously to multiple residues through different foods. Substance-by-substance assessments may therefore provide an incomplete characterization of combined exposure when several substances affect the same target organ or biological system [4–7].

Deterministic and probabilistic methods provide complementary information. Deterministic assessments use fixed exposure inputs and are suitable for initial screening, whereas probabilistic assessments characterize distributions of modelled exposure and allow participant variability and sampling uncertainty to be examined separately [3,8,9]. EFSA coordinates pesticide-residue monitoring throughout the European Union, and recent results indicate that most food samples comply with established maximum residue limits and that estimated dietary exposure generally does not raise a concern for consumer health [10]. Dietary exposure assessments can also support the prioritization of active substances and food commodities for monitoring and risk-management actions [11].

To support cumulative pesticide assessment, EFSA has established cumulative assessment groups (CAGs), in which active substances are grouped according to common toxicological effects. EFSA has applied this framework to effects on the nervous system and thyroid gland [12,13] and has continued to prioritize substances and target-organ systems for further evaluation [14,15]. For cumulative risk characterisation, substance-specific exposures can be normalised by dividing each exposure estimate by the endpoint-specific no-observed-adverse-effect level (NOAEL) assigned within the relevant cumulative assessment group. The resulting normalised exposures are summed across substances affecting the same endpoint, and the reciprocal of the cumulative normalised exposure is expressed as the total margin of exposure (MOET) [5–7]. European studies have generally reported low chronic dietary exposure relative to health-based guidance values [3,8,9,16]. Greek investigations have similarly documented pesticide residues in commonly consumed foods while generally reporting low estimated dietary risk under the assumptions applied [17–21]. Nevertheless, public concern regarding pesticide residues in Greece emphasizes the need for transparent, context-specific exposure estimates and effective risk communication [22].

Evidence integrating individual-level consumption data, national residue-monitoring results, and both deterministic and probabilistic exposure assessment remains limited in Greece. The present study therefore aimed to characterize chronic dietary exposure to pesticide residues from apples, oranges, and bananas among adults participating in the HELLANS 2024–2025 cohort. Cohort-specific consumption and body-weight information was combined with Greek residue-monitoring data from 2022–2024. Substance-specific exposure was assessed using EFSA PRIMo revision 3.1, while cumulative risk characterisation focused on the established EFSA chronic endpoints of brain and/or erythrocyte acetylcholinesterase inhibition and hypothyroidism. The study also examined the influence of left-censored monitoring results and uncertainty in monitoring-derived mean concentrations.

2. Materials and Methods

2.1. Study Design and Consumption Assessment

Data were collected through an online questionnaire administered via the European Commission's EUSurvey platform between May 2024 and April 2025 as part of the HEL-LANS 2024–2025 study. Adult volunteers were recruited by convenience sampling through email invitations, social media, websites, and online news platforms, yielding 1318 completed responses. Participation was voluntary and anonymous, and informed consent was obtained from all participants. The study protocol was approved by the Research Ethics and Deontology Committee of the Hellenic Mediterranean University (approval No. 12656, 16 May 2024) and complied with the Declaration of Helsinki and the General Data Protection Regulation.

The questionnaire was developed based on previous studies [23,24] and comprised sections on sociodemographic characteristics, attitudes towards pesticide residues and food safety, and food consumption. The variables used in the present exposure assessment were participant body weight and the consumption of apples, oranges, and bananas. These fruits were selected because they were among the most frequently consumed fruits in the cohort, were broadly available throughout the year, and had sufficient residue-monitoring data.

Food consumption was assessed using a single 24 h dietary recall (24HR) and a short food-frequency questionnaire (FFQ). The 24HR recorded the foods and corresponding amounts consumed during the preceding day. It was completed online without interviewer assistance, and NHANES 2019–March 2020/FNDDS data were used to select reference portion weights [25]. The research team prepared, weighed, and photographed the corresponding fruit portions, and participants selected the image closest to the amount consumed. The image library was standardized by direct weighing but was not formally validated.

The FFQ recorded consumption frequency during the preceding year as never, less than one day per month, 1–3 days per month, one day per week, 2–3 days per week, 4–5 days per week, or almost daily/daily. The upper bound of each interval was used as a conservative estimate, corresponding to 0, 12, 36, 52, 156, 260, and 365 days/year, respectively. For participant (j) and fruit (f), the corresponding probability of consumption was calculated as follows:

$$P_{jf} = \frac{D_{jf}}{365}$$

where D_{jf} is the assigned annual number of consumption days.

Because the FFQ did not collect portion sizes, expected long-term daily intake was estimated as follows:

$$IR_{jf} = P_{jf} \times \overline{A_f}$$

where P_{jf} is the consumption probability for participant (j) and fruit (f), and $\overline{A_f}$ is the mean amount consumed among 24HR consumers of that fruit. The complete participant row—body weight and expected intakes of all three fruits—was retained as a joint observation in all participant-level analyses. These values represent modelled expected daily intake rather than individual usual-intake distributions because only one 24HR was available and between-person variation in portion size was not fully represented [26–28].

Data collection covered a complete annual cycle. However, because the volunteer sample was not nationally representative and population weights were not applied, all consumption and exposure distributions were interpreted as specific to the HEL-LANS cohort.

2.2. Pesticide Residue Data and Treatment of Left-Censored Results

Pesticide residue data for apples, oranges, and bananas were obtained from the Greek national monitoring program for 2022–2024 [29–31]. Sampling strategy was identified from the EFSA Standard Sample Description metadata. Following EFSA cumulative-risk

assessment practice [5], only samples coded as objective (ST10A) or selective (ST20A) sampling and belonging to eligible official programme types were retained. Suspect samples (ST30A) and samples from EU increased-control or national third-country import-control programmes were excluded. Of 1072 primary samples, 997 were retained (539 apple, 329 orange, and 129 banana samples); the 75 excluded samples and the monitoring-data audit are documented in Supplementary Data S1. The annual datasets were harmonised with respect to commodity, active-substance identity, result value, quantification status, and the reported limit of quantification (LOQ). Substance names and official residue-definition codes referring to the same active substance were consolidated before calculating occurrence and concentration statistics. Annual datasets were pooled separately by commodity; thus, each year contributed in proportion to its available number of samples. Substance–commodity combinations are reported in Table A2 only when at least one result was quantified.

For the deterministic PRIMo assessment, results below the LOQ were assigned LOQ/2. For the cumulative MOET assessment, three separate scenarios were evaluated:

- Lower bound (LB): results below the LOQ were assigned zero;
- Middle bound (MB): results below the LOQ were assigned LOQ/2;
- Upper bound (UB): results below the LOQ were assigned the LOQ.

Arithmetic mean concentrations were calculated separately for every substance–commodity–scenario combination.

No peeling or processing factors were applied because reliable, directly matching factors were not consistently available for all substance–commodity combinations. The assessment was therefore conducted as a whole-commodity screening analysis. Comparable analyses have noted that the application of suitable processing factors would generally be expected to reduce estimated exposure [32].

2.3. Deterministic Exposure Assessment

Chronic deterministic exposure was assessed using the official EFSA Pesticide Residue Intake Model (PRIMo), revision 3.1 [33]. The unrounded deterministic calculation audit is provided in Supplementary Data S1, and a completed three-fruit PRIMo worked example is provided in Supplementary File S1. Study-specific arithmetic mean consumption and body-weight values and arithmetic mean residue concentrations from the pooled 2022–2024 monitoring data were entered into the relevant input cells. Exposure was aggregated across apples, oranges, and bananas for each active substance and expressed as a percentage of the substance-specific ADI. Current EU ADIs were used when available [34]. When no current ADI could be identified, the occurrence result was retained, as shown in Table A2, but the deterministic %ADI is reported as “not available” and was not calculated.

2.4. Endpoint-Specific Chronic Cumulative Exposure and Risk Characterisation

Cumulative risk characterisation was based on established EFSA cumulative assessment groups (CAGs) and endpoint-specific no-observed-adverse-effect levels (NOAELs). Two chronic endpoints were evaluated: (i) brain and/or erythrocyte acetylcholinesterase inhibition and (ii) hypothyroidism. Active-substance assignments and endpoint-specific reference points were obtained from the corresponding EFSA nervous-system and thyroid CAG reports [12,13] and are documented in Table A4. Assignments were non-exclusive, and only substances quantified in at least one assessed commodity and having both an eligible CAG assignment and an endpoint-specific NOAEL were included.

The primary estimand was the MOET corresponding to specified percentiles of the empirical participant-level cumulative normalised-exposure distribution. Within each censoring scenario, commodity–substance arithmetic mean concentrations were fixed, and

exposure was calculated for all 1318 participant records, thereby preserving the observed joint distribution of body weight and consumption of the three fruits. Sex was not used for stratification, and sex-specific exposure distributions were not estimated.

Cumulative normalised exposure (NE) was calculated as follows:

$$NE = \Sigma[(\bar{C} \times I/1000)/(BW \times NOAEL)],$$

where $\bar{C}$ is the scenario-specific arithmetic mean concentration (mg/kg), I is the participant's expected daily intake of a fruit (g/day), BW is body weight (kg), and $NOAEL$ is the endpoint-specific reference point (mg/kg bw/day). For each endpoint and scenario, the P50, P95, P99, and P99.9 of the participant-level normalised-exposure distribution were calculated from all 1318 values. The total margin of exposure (MOET) at exposure percentile P was then calculated as follows: $MOET = 1/Q_p(NE)$, where Q_p is the corresponding percentile of cumulative normalised exposure across the 1318 participant records.

Thus, the reported MOET at a given exposure percentile is not the same percentile of the participant-specific MOET distribution [35]. EFSA cumulative risk assessments report MOETs at defined exposure percentiles and use a value of 100 at P99.9 as the threshold for regulatory consideration [5,35]. LB, MB, and UB were analysed separately to assess sensitivity to the treatment of left-censored monitoring results [5,35,36].

2.5. Outer-Bootstrap Analysis of Monitoring-Sample Uncertainty

Sampling uncertainty in the commodity–substance arithmetic mean concentrations was quantified using a 5000-replicate outer non-parametric bootstrap implemented in Microsoft Excel 365 with XLRisk version 1.0 [37]. Separate formula-based workbooks were used for the LB, MB, and UB scenarios (Supplementary File S1), with a fixed random seed of 20260826. XLRisk was configured for ordinary Monte Carlo sampling with Latin hypercube sampling disabled. Within each active substance $\times$ commodity $\times$ year stratum, analysed monitoring observations were resampled with replacement while preserving the original stratum sample size; the resulting resamples were pooled across years, thereby retaining the original sample-size weighting of the 2022–2024 monitoring data.

In each replicate, scenario-specific commodity–substance arithmetic mean concentrations were recalculated and propagated through the cumulative exposure model using all 1318 participant records. Participant inputs, endpoint assignments, and endpoint-specific NOAELs were held fixed. The P50, P95, P99, and P99.9 of the participant-level normalised-exposure distribution and the corresponding MOETs were recalculated for each replicate.

Iteration-level MOET outputs were summarised in JASP version 0.98.1 [38], with quantile type 7 selected for percentile estimation. For each endpoint, scenario, and exposure percentile, the 95% monitoring-sample uncertainty interval (UI) was defined as the 2.5th–97.5th percentile interval of the bootstrap MOET distribution [7,35]. These intervals quantify sampling uncertainty in the monitoring-derived mean concentrations, conditional on the observed participant-level body weight and consumption inputs and the specified toxicological model. They do not incorporate uncertainty in the participant sample, consumption model, residue definitions, CAG membership, endpoint selection, or toxicological reference points and should not be interpreted as intervals for the overall uncertainty of the exposure assessment.

Bootstrap convergence was assessed by comparing the 2.5th and 97.5th percentile estimates after 500, 1000, 2500, and 5000 replicates (Supplementary Data S2).

Sparse monitoring strata are documented in Supplementary Data S1. Iteration-level MOET outputs, JASP percentile summaries, and convergence diagnostics are provided in Supplementary Data S2.

2.6. Sensitivity Analyses

Two sensitivity analyses were performed to examine model assumptions. First, the FFQ frequency categories were recoded using category midpoints rather than the upper-bound values used in the primary analysis. The sensitivity coding was 0, 6, 24, 52, 130, 234, and 338 days/year for never, <1 day/month, 1–3 days/month, 1 day/week, 2–3 days/week, 4–5 days/week, and almost daily/daily, respectively. Fruit-specific mean 24HR portion amounts and individual body weights were held fixed. Participant expected daily intakes were recalculated and propagated through the deterministic %ADI and endpoint-specific MOET calculations, while residue concentrations, endpoint assignments, and NOAELs remained unchanged.

Second, to quantify the potential influence of using whole-commodity residue concentrations rather than edible-portion concentrations for oranges and bananas, the UB cumulative assessment was repeated after applying hypothetical 25%, 50%, and 75% reductions to the orange and banana mean residue concentrations, with apple concentrations unchanged. These scenarios were intended solely as an illustrative quantitative sensitivity analysis of possible edible-portion effects and should not be interpreted as validated commodity- or substance-specific processing factors. The sensitivity results were compared with the primary point estimates; the monitoring-sample outer bootstrap was not repeated because the objective was to examine the effect of fixed model-input assumptions. Complete results are provided in Supplementary Data S2.

3. Results

3.1. Study Sample and Participant Characteristics

The cohort comprised 1318 adults, including 763 females (57.9%) and 555 males (42.1%). The largest age groups were 45–54 years (40.9%) and 55–64 years (25.6%), while 84.4% of participants had completed tertiary education. Participants were distributed across Northern (35.8%), Central (31.9%), and Southern Greece (32.2%); complete sociodemographic characteristics are presented in Table A1. Mean body weight was 78.8 kg (95% bootstrap CI: 77.9–79.7 kg). Participant body weight and the modelled expected intakes of all three fruits were retained jointly in the endpoint-specific cumulative calculations and were held fixed during the monitoring-data bootstrap.

3.2. Selection and Consumption of the Assessed Fruits

Apples, oranges, and bananas were selected because of their relatively high consumption frequencies, broad year-round availability, and sufficient residue-monitoring samples. Modelled expected daily consumption relative to body weight was highest for oranges, followed by apples and bananas (Table 1). These values represent FFQ-frequency-weighted expected daily intakes rather than empirical distributions of individual usual intake. Summary statistics for body weight and the fruit-consumption inputs are provided in Table S2 within Supplementary Data S1.

Table 1. Modelled expected daily consumption of the three assessed fruits in the study cohort (N = 1318).

Fruit	Mean, g/kg bw/day (95% Bootstrap CI)	P97.5, g/kg bw/day
Apples	1.119 (1.063–1.178)	3.614
Oranges	1.437 (1.369–1.506)	4.411
Bananas	1.027 (0.979–1.077)	3.165

Note: Consumption estimates combine participant-specific FFQ probabilities with the mean amount reported by consumers in the 24 h recall. Accordingly, P97.5 refers to the distribution of modelled expected daily intake within the study cohort. CI, 95% bootstrap confidence interval based on 10,000 resamples (JASP version 0.98.1).

3.3. Occurrence of Pesticide Residues

Analytical coverage varied by substance and commodity: analyte-specific denominators ranged from 2 to 539 samples for apples, 15 to 329 for oranges, and 17 to 129 for bananas. Consequently, quantification percentages were interpreted together with their corresponding analysed-sample counts. Sparse analytical coverage was observed for propamocarb, for which only two samples were analysed and both were quantified, and for fosetyl-Al and dithianon, for which 12 samples each were analysed and 5 and 4 results, respectively, were quantified. Among substances with broad apple coverage, acetamiprid was quantified in 133/539 samples (24.7%), boscalid in 106/474 (22.4%), etofenprox in 97/539 (18.0%), and tebuconazole in 96/539 (17.8%). The highest apple concentrations were 4.915 mg/kg for imazalil and 4.390 mg/kg for pyrimethanil.

In oranges, pyriproxyfen and imazalil were the most frequently quantified substances with broad analytical coverage, occurring in 51/329 (15.5%) and 46/329 (14.0%) samples, respectively. Fosetyl-Al was quantified in 2/15 samples (13.3%), whereas phosmet was quantified in 19/326 (5.8%). The highest orange concentrations were 2.700 mg/kg for thiabendazole and 1.880 mg/kg for imazalil.

In bananas, copper was quantified in 16/17 analysed samples (94.1%), although its analytical coverage was limited. Azoxystrobin, thiabendazole, and pyriproxyfen were quantified in 94/129 (72.9%), 80/129 (62.0%), and 35/129 (27.1%) samples, respectively. Maximum concentrations were 2.300 mg/kg for copper, 1.174 mg/kg for thiabendazole, and 0.790 mg/kg for azoxystrobin. Complete analyte-specific occurrence data and LB, MB, and UB arithmetic mean concentrations are presented in Table A2.

3.4. Substance-Specific Deterministic Chronic Exposure Assessment

All substance-specific deterministic estimates were below 100% of the corresponding ADI (Table A3). Phosmet had the highest aggregate exposure across the three fruits at 2.343% ADI, followed by lambda-cyhalothrin (0.800%), acetamiprid (0.464%), imazalil (0.363%), tau-fluvalinate (0.332%), pyrimethanil (0.254%), and fenpropimorph (0.210%). The highest fruit-specific values were 1.024% ADI for phosmet from apples, 1.319% ADI for phosmet from oranges, and 0.210% ADI for fenpropimorph from bananas.

The %ADI values were interpreted separately by active substance and were not summed across toxicologically unrelated substances. Chlorpyrifos, piperonyl butoxide, dithiocarbamates measured as CS₂, and total measured copper were retained in the occurrence assessment but were not expressed as %ADI: suitable current ADIs were unavailable for chlorpyrifos and piperonyl butoxide, the non-specific CS₂ result could not be assigned to an individual dithiocarbamate active substance, and pesticide-source attribution was not possible for total copper. However, chlorpyrifos was retained in the AChE MOET analysis because an endpoint-specific NOAEL was available for this effect. Complete fruit-specific and three-fruit PRIMo estimates are presented in Table A3.

3.5. Endpoint-Specific Chronic Cumulative Exposure and Monitoring-Sample Uncertainty

Endpoint-specific MOETs decreased from the LB to the MB and UB scenarios, consistent with increasing substituted concentrations for left-censored results (Table 2). Greater interpretive emphasis was placed on P95 and P99 because these percentiles are less dependent on the most extreme participant-level exposure observations than P99.9. Under the UB scenario, the AChE MOETs were 1324 at P95 and 1070 at P99, while the corresponding hypothyroidism MOETs were 3944 and 3194. The UB P99.9 screening-tail MOETs were 928 for AChE inhibition and 2854 for hypothyroidism. Complete percentile-specific results for all scenarios are provided in Tables 2 and A5. Among the 67 active substances/residue definitions quantified in at least one assessed commodity, 22 met the criteria for inclu-

sion in at least one of the two endpoint-specific cumulative assessments; the remaining 45 lacked an eligible EFSA CAG assignment and/or endpoint-specific reference point for these endpoints.

Table 2. Endpoint-specific chronic cumulative total margins of exposure (MOETs) under the lower-bound, middle-bound, and upper-bound scenarios, with monitoring-sample outer-bootstrap uncertainty at the P99.9 exposure level.

Endpoint-Specific Cumulative Assessment Group	Scenario	MOET at P50 Exposure	MOET at P95 Exposure	MOET at P99 Exposure	MOET at P99.9 Exposure	95% Monitoring-Sample UI for MOET at P99.9
Chronic brain and/or erythrocyte acetylcholinesterase inhibition	LB	55,270	18,121	15,021	13,023	8317–23,276
Chronic brain and/or erythrocyte acetylcholinesterase inhibition	MB	7395	2482	1995	1732	1617–1832
Chronic brain and/or erythrocyte acetylcholinesterase inhibition	UB	3964	1324	1070	928	897–954
Hypothyroidism	LB	48,656	17,434	13,818	12,065	10,216–14,244
Hypothyroidism	MB	19,649	6482	5200	4666	4354–4971
Hypothyroidism	UB	12,251	3944	3194	2854	2740–2957

Notes: MOET, total margin of exposure; UI, uncertainty interval. LB, MB, and UB assign results below the LOQ to zero, LOQ/2, and the LOQ, respectively. MOETs are the reciprocals of the corresponding percentiles of cumulative normalised exposure. The 95% UIs were derived from 5000 stratified outer-bootstrap replicates of the monitoring observations and are conditional on the observed participant-level consumption and body-weight inputs and the specified toxicological model; they do not represent overall uncertainty in the exposure assessment. Complete percentile-specific UIs are provided in Table A5. A MOET of 100 at P99.9 was used as the screening reference value.

For the UB scenario, the 95% monitoring-sample UIs were 1286–1369 and 1031–1101 for the AChE P95 and P99 MOETs, respectively, and 3797–4092 and 3061–3317 for the corresponding hypothyroidism estimates. The P99.9 UIs were 897–954 for AChE inhibition and 2740–2957 for hypothyroidism. All point estimates and lower monitoring-sample UI limits remained above 100. All 5000 bootstrap replicates yielded valid MOET estimates for every reported combination, and the maximum relative change in either UI bound between 2500 and 5000 replicates was <1% across all censoring scenarios (Supplementary Data S2).

3.6. Sensitivity Analyses Results

Recoding the FFQ categories to midpoint frequencies reduced the mean modelled daily intake of apples, oranges, and bananas by 10.9–11.8% relative to the primary upper-bound frequency coding. The highest deterministic estimate, phosmet, decreased from 2.343% ADI to 2.069% ADI. Endpoint-specific MOETs increased under the midpoint coding. In the UB scenario, the AChE MOETs at P95, P99, and P99.9 increased from 1324, 1070, and 928 to 1467, 1155, and 1002, respectively; the corresponding hypothyroidism MOETs increased from 3944, 3194, and 2854 to 4312, 3451, and 3088. The interpretation therefore remained unchanged (Supplementary Data S2).

The hypothetical edible-portion residue-reduction analysis also moved the MOETs away from the screening value of 100. Under UB, a 50% reduction in orange and banana mean residue concentrations increased the AChE MOETs at P95, P99, and P99.9 to 1848, 1481, and 1284, respectively, and the corresponding hypothyroidism MOETs to 4614, 3789, and 3322. The 25% and 75% reduction scenarios showed the same direction of change. These scenarios are illustrative and should not be interpreted as validated processing-factor corrections (Supplementary Data S2).

4. Discussion

This study combined recent Greek residue-monitoring data with cohort-specific consumption and body-weight information to characterize chronic dietary exposure to pesticide residues from apples, oranges, and bananas. The assessment produced two complementary findings. First, all substance-specific deterministic exposures estimated with EFSA PRIMo revision 3.1 were well below 100% of the corresponding ADI. Second, the endpoint-specific cumulative MOET estimates at P95 and P99, as well as the P99.9 screening-tail estimates and their lower 95% monitoring-sample uncertainty limits, remained above the screening reference value of 100 under the LB, MB, and UB scenarios. These findings do not indicate a chronic screening-level concern from the three assessed fruits within this cohort under the assumptions applied, but they should not be interpreted as evidence of zero risk or as an assessment of total dietary exposure.

The substance-specific deterministic PRIMo assessment and the endpoint-specific cumulative assessment served different purposes. EFSA PRIMo revision 3.1 estimated substance-specific chronic exposure relative to each substance's ADI [33], whereas the cumulative analysis normalised each substance–commodity exposure by the endpoint-specific NOAEL assigned within an established EFSA chronic cumulative assessment group [12,13]. Direct numerical agreement between %ADI and MOET was therefore neither expected nor required. All calculable deterministic estimates remained well below 100% ADI. Chlorpyrifos and piperonyl butoxide were not expressed as %ADI because suitable current ADIs were unavailable, while dithiocarbamates measured as CS₂ could not be attributed to an individual active substance, and total measured copper could not be attributed specifically to pesticide use. These distinctions illustrate why regulatory status, residue occurrence, residue definition, source attribution, and toxicological eligibility must be documented separately.

The cumulative results were sensitive to the treatment of left-censored measurements, as expected. MOETs decreased progressively from LB to MB and UB because substituting higher values for results below the LOQ increased normalised exposure. The reduction was particularly marked for the AChE endpoint, showing that substituted values materially influenced the estimated distribution even though the risk interpretation did not change. Reporting all three scenarios is therefore preferable to relying on LOQ/2 alone because it makes the influence of left-censoring assumptions explicit.

The outer bootstrap addressed sampling uncertainty in the monitoring means, which is distinct from participant-level exposure variability. In each of 5000 replicates, monitoring observations were resampled within active substance $\times$ commodity $\times$ year strata, scenario-specific arithmetic mean concentrations were recalculated, and the endpoint-specific exposure model was rerun while participant records, CAG assignments, and NOAELs were held fixed. The resulting intervals are therefore conditional on the observed participant-level body weight and consumption inputs and the specified toxicological model. They do not quantify uncertainty in the participant sample, consumption model, residue definitions, end-point assignments, NOAELs, or other structural assumptions and should not be interpreted as overall uncertainty intervals for the exposure assessment.

Commodity-specific patterns were also evident in the deterministic assessment. Phosmet provided the highest fruit-specific %ADI estimate for apples (1.024%) and oranges (1.319%), whereas fenpropimorph provided the highest estimate for bananas (0.210%). These rankings cannot be inferred from quantification frequency alone because exposure also depends on the mean concentration, fruit consumption, body weight, and toxicological reference value. Detection or quantification frequency should therefore not be interpreted as a direct measure of health risk.

No peeling or processing factors were applied in the primary analysis; residue concentrations therefore referred to the monitored commodities rather than exclusively to their edible portions. This assumption is particularly relevant to oranges and bananas and may overestimate edible-portion exposure when residues are concentrated in the peel. Published processing studies indicate that washing, peeling, and juice production can substantially reduce pesticide residues in citrus, although the magnitude varies among active substances and processing conditions [32,39]. Because directly applicable sub-stance-commodity-specific factors were unavailable for all relevant combinations, generic processing corrections were not introduced into the primary model. Instead, the sensitivity analysis applied hypothetical 25%, 50%, and 75% reductions to orange and banana residues under UB. All three scenarios increased the endpoint-specific MOETs; for example, a 50% reduction increased the AChE P95/P99/P99.9 MOETs from 1324/1070/928 to 1848/1481/1284 and the corresponding hypothyroidism MOETs from 3944/3194/2854 to 4614/3789/3322. These results illustrate the likely direction and magnitude of edible-portion effects but are not validated processing-factor estimates.

Previous European studies using Belgian, Danish, Hungarian, and Greek data generally reported low chronic dietary exposure under their respective study conditions [3,7–10,16–21]. These findings provide qualitative context only. Direct numerical comparisons are not appropriate because the studies differed in their populations, foods covered, monitoring periods, processing assumptions, treatment of left-censored results, consumption inputs, model structures, toxicological groupings, and risk metrics. In particular, earlier HI estimates based on sums of ADI-normalised HQs are not directly comparable with the endpoint-specific MOETs reported here. Although probabilistic methods have also been used in previous cumulative assessments [5–9,16], differences in model design preclude direct numerical comparison. The present findings should therefore be interpreted within the HELLANS cohort and the defined three-fruit exposure scenario and not as confirmation of or a benchmark against previous estimates.

The joint empirical participant records preserved the observed relationship between body weight and consumption of the three fruits. Cross-commodity dependence in residue concentrations could not be evaluated because monitoring samples were not linked across fruits by consumer, purchase occasion, origin, or batch. Accordingly, the reported uncertainty intervals quantify monitoring-sample uncertainty conditional on the observed participant data and specified exposure model rather than uncertainty from a full two-dimensional probabilistic analysis.

Interpretation of occurrence data also depends on analyte-specific analytical coverage. The pooled monitoring files did not contain an identical number of valid results for every substance, commodity, and year; consequently, occurrence percentages and mean concentrations were calculated only among samples in which the substance was analysed. Analytical coverage varied markedly among substance-commodity combinations, and quantification percentages should therefore be interpreted together with the corresponding numbers analysed. For example, propamocarb was quantified in both two apple samples in which it was analysed, whereas cyantraniliprole was quantified only once despite much broader analytical coverage. These contrasting cases illustrate that quantification frequency alone does not provide a reliable indication of residue prevalence or exposure relevance.

Several limitations should also be considered. The HELLANS cohort was a volunteer sample without population weighting with high educational attainment, which may have systematically influenced fruit-consumption patterns and, consequently, the estimated exposure distribution; the estimated intake and MOET distributions should not be generalized to all Greek adults. Probabilities derived from FFQ were combined with the mean amount consumed from a single 24HR, producing modelled expected long-term intake

rather than complete individual usual-intake distributions [26–28]. The self-administered 24HR used a study-specific library of directly weighed fruit portions but did not implement the complete NHANES Automated Multiple-Pass Method, interviewer assistance, or formal independent validation of the image library [25]. Using the upper bound of each FFQ frequency interval may overestimate modelled long-term intake relative to midpoint coding; however, the midpoint sensitivity analysis did not alter the overall risk interpretation. Caution is warranted when interpreting P99.9. Given the cohort size ($n = 1318$), this extreme percentile is strongly influenced by a small number of high-exposure participant records. P99.9 was retained because of its relevance to the EFSA cumulative-risk screening framework, but greater interpretive emphasis was placed on P95 and P99 because they are less sensitive to the most extreme observations in the empirical exposure distribution.

The assessment was restricted to three fruits and therefore does not characterize total dietary exposure; exclusion of other foods may underestimate overall exposure despite conservative assumptions for the included commodities. Monitoring data from 2022–2024 were pooled in proportion to the available sample numbers [29–31], preserving the empirical dataset but not modelling temporal trends or uncertainty in annual sampling intensity. Substances not quantified in any assessed commodity and substances without an eligible endpoint assignment or reference point could not contribute to the corresponding MOET. Accordingly, the UB scenario is an upper-bound substitution for left-censored observations within the included endpoint model, not an upper bound for every pesticide that may occur in the total diet.

Despite these limitations, the assessment integrated national monitoring data with participant-level dietary and body-weight information, distinguished substance-specific deterministic exposure from endpoint-specific cumulative risk characterization, and explicitly addressed uncertainty and key modelling assumptions through outer-bootstrap and sensitivity analyses. Inclusion decisions were based on established EFSA endpoint assignments and reference points rather than contribution thresholds. Overall, these features provide a transparent screening characterization of chronic cumulative exposure from apples, oranges, and bananas in the HELLANS cohort while clearly defining the boundaries of inference and the remaining sources of uncertainty.

5. Conclusions

Pesticide residues were quantified in apples, oranges, and bananas, but their presence alone does not imply a consumer health risk. All substance-specific deterministic chronic exposure estimates calculated with EFSA PRIMo revision 3.1 were substantially below 100% of the corresponding ADI. In the endpoint-specific cumulative assessment, the P95 and P99 MOET estimates, as well as the P99.9 screening-tail estimates and their lower 95% monitoring-sample uncertainty limits, remained above the screening reference value of 100 under the LB, MB, and UB scenarios. The most conservative primary result was the UB P99.9 MOET for acetylcholinesterase inhibition, at 928 (95% monitoring-sample UI: 897–954).

Overall, the findings did not indicate a chronic screening-level dietary concern from pesticide residues in the three assessed fruits within the HELLANS cohort, under the assumptions applied. The interpretation was unchanged in the FFQ midpoint and hypothetical orange/banana residue-reduction sensitivity analyses. Because the cohort was a non-representative volunteer sample with high educational attainment, the findings should not be extrapolated to the broader Greek adult population or to total dietary exposure. Future assessments should extend food coverage, nationally representative consumption data, more uniform analytical coverage, and more fully characterize uncertainty in participant-derived inputs and toxicological assumptions.

Supplementary Materials: The following supporting information can be downloaded at <https://www.mdpi.com/article/10.3390/pollutants6040059/s1>: Supplementary Data S1: Analytical inputs and audit package, comprising the monitoring-data audit and LOQ inputs, Table S1. harmonised sample-level monitoring records; Table S2. summary distributions of participant-derived body-weight and fruit-consumption inputs; Table S3. unrounded deterministic PRIMo calculation outputs; Supplementary File S1: Computational-workbook package, comprising the completed EFSA PRIMo revision 3.1 three-fruit worked example and the formula-based XLRisk outer-bootstrap workbooks for the LB, MB, and UB scenarios, with individual-level HELLANS participant data removed; Supplementary Data S2: Exported 5000-replicate MOET outputs, JASP percentile summaries, and bootstrap convergence diagnostics, FFQ midpoint sensitivity analysis, hypothetical orange/banana residue-reduction sensitivity analysis, and empirical percentile-rank context.

Author Contributions: Conceptualization, K.B.S. and E.R.; methodology, K.B.S.; software, K.B.S.; validation, K.B.S.; formal analysis, K.B.S.; investigation, K.B.S.; resources, K.B.S.; data curation, K.B.S.; writing—original draft preparation, K.B.S.; writing—review and editing, Z.V., E.A. and E.R.; visualization, K.B.S.; supervision, E.R. All authors have read and agreed to the published version of the manuscript.

Funding: This research received no external funding.

Data Availability Statement: Publicly available pesticide-monitoring datasets were analyzed in this study and are available from the repositories cited in References [29–31]. The author-generated data supporting the reported results are included in the article and Supplementary Materials. Individual-level HELLANS participant data are not publicly available due to ethical and data-protection restrictions imposed by the Research Ethics and Deontology Committee of the Hellenic Mediterranean University.

Acknowledgments: The authors gratefully acknowledge Emmanouil Karazafeiris for his invaluable advice and technical support, as well as the three anonymous reviewers for their insightful comments and suggestions which greatly improved the quality of this manuscript.

Conflicts of Interest: The authors declare no conflicts of interest.

Abbreviations

The following abbreviations are used in this manuscript:

24HR	24 h dietary recall
AChE	Acetylcholinesterase
ADI	Acceptable daily intake
bw	Body weight
CAG	Cumulative assessment group
CI	Confidence interval
EFSA	European Food Safety Authority
FFQ	Food frequency questionnaire
LB	Lower-bound scenario
LOD	Limit of detection
LOQ	Limit of quantification
MB	Middle-bound scenario
MOET	Total margin of exposure
MRL	Maximum residue limit
NE	Cumulative normalised exposure
NHANES	National Health and Nutrition Examination Survey
NOAEL	No-observed-adverse-effect level
P50	50th percentile
P95	95th percentile
P97.5	97.5th percentile

P99	99th percentile
P99.9	99.9th percentile
PRIMo	Pesticide Residue Intake Model
UB	Upper-bound scenario
UI	Uncertainty interval

Appendix A

Table A1. Sociodemographic characteristics of the respondents (N = 1318).

Demographic Variables		Frequency	Percentage
Sex	Female	763	57.9%
	Male	555	42.1%
Age	18–24	96	7.3%
	25–34	89	6.8%
	35–44	194	14.7%
	45–54	539	40.9%
	55–64	338	25.6%
	≥65	62	4.7%
Educational background	Less than high school	3	0.2%
	High school—Technical education	203	15.4%
	Bachelor’s degree	478	36.3%
	Master’s degree	519	39.4%
	Doctoral degree	115	8.7%
Residential geographical area	Northern Greece	472	35.8%
	Central Greece	421	31.9%
	Southern Greece	425	32.2%
Occupation	Civil servants	729	55.3%
	Private employees	227	17.2%
	Self-employed	142	10.8%
	Farmers	25	1.9%
	Unemployed	44	3.3%
	University students	89	6.8%
	Retired	62	4.7%

Table A2. Occurrence and residue concentrations of pesticide active substances quantified in apple, orange and banana samples.

(a) Apples							
Active Substance/Residue Definition	Analysed Samples, n	Quantified Samples, n (%)	Arithmetic Mean in All Analysed Samples (mg/kg)			Mean in Quantified Samples (mg/kg)	Maximum Quantified (mg/kg)
			LB	MB	UB		
2-Phenylphenol	539	4 (0.7)	0.001130	0.006093	0.011056	0.152250	0.440000
Acetamiprid	539	133 (24.7)	0.009059	0.012825	0.016591	0.036711	0.220000
Boscalid	474	106 (22.4)	0.017250	0.021132	0.025014	0.077137	0.320000
Captan	263	2 (0.8)	0.000764	0.005726	0.010688	0.100500	0.110000
Carbendazim and benomyl (expressed as carbendazim)	539	6 (1.1)	0.000469	0.005414	0.010358	0.042167	0.120000
Chlorantraniliprole	539	84 (15.6)	0.004272	0.008493	0.012714	0.027414	0.220000
Chlorpyrifos	539	1 (0.2)	0.000111	0.005102	0.010093	0.060000	0.060000
Cyantraniliprole	383	1 (0.3)	0.000444	0.005431	0.010418	0.170000	0.170000
Cyflumetofen	82	2 (2.4)	0.000927	0.005805	0.010683	0.038000	0.046000
Cyprodinil	539	29 (5.4)	0.002590	0.007321	0.012052	0.048141	0.270000
Deltamethrin	539	2 (0.4)	0.000087	0.005069	0.010050	0.023500	0.034000
Difenoconazole	539	40 (7.4)	0.001602	0.006231	0.010860	0.021590	0.074000
Diphenylamine	461	1 (0.2)	0.000024	0.005013	0.010002	0.011000	0.011000
Dithianon	12	4 (33.3)	0.007333	0.010667	0.014000	0.022000	0.041000
Dithiocarbamates (expressed as CS ₂)	147	1 (0.7)	0.000116	0.005082	0.010048	0.017000	0.017000
Dodine	400	53 (13.2)	0.006023	0.010361	0.014698	0.045458	0.199000
Etofenprox	539	97 (18.0)	0.009312	0.013412	0.017512	0.051744	0.200000
Fenbuconazole	539	2 (0.4)	0.000054	0.005035	0.010017	0.014500	0.016000
Fenoxycarb	539	4 (0.7)	0.000150	0.005113	0.010076	0.020250	0.050000
Fenpyroximate	539	2 (0.4)	0.000058	0.005039	0.010020	0.015500	0.019000
Flonicamid	406	19 (4.7)	0.002562	0.007328	0.012094	0.054737	0.250000
Fluazinam	135	2 (1.5)	0.001048	0.005974	0.010900	0.070750	0.122600
Fludioxonil	383	48 (12.5)	0.009212	0.013586	0.017959	0.073506	0.699300
Fluopyram	539	77 (14.3)	0.005661	0.009947	0.014233	0.039629	0.340000
Flupyradifurone	383	12 (3.1)	0.001046	0.005890	0.010733	0.033400	0.050000
Tau-Fluvalinate	539	25 (4.6)	0.001793	0.006561	0.011329	0.038652	0.160000
Fluxapyroxad	461	45 (9.8)	0.002579	0.007091	0.011603	0.026418	0.140000
Fosetyl-Al	12	5 (41.7)	0.343917	0.346833	0.349750	0.825400	1.742000

Table A2. Cont.

(a) Apples							
Active Substance/Residue Definition	Analysed Samples, n	Quantified Samples, n (%)	Arithmetic Mean in All Analysed Samples (mg/kg)			Mean in Quantified Samples (mg/kg)	Maximum Quantified (mg/kg)
			LB	MB	UB		
Hexythiazox	539	1 (0.2)	0.000083	0.005074	0.010064	0.044700	0.044700
Imazalil	539	7 (1.3)	0.012675	0.017610	0.022545	0.975971	4.915000
Imidacloprid	539	1 (0.2)	0.000019	0.005009	0.010000	0.010000	0.010000
Indoxacarb	539	12 (2.2)	0.000514	0.005403	0.010291	0.023083	0.046000
Isopyrazam	248	1 (0.4)	0.000044	0.005024	0.010004	0.011000	0.011000
Lambda-cyhalothrin	539	10 (1.9)	0.000458	0.005365	0.010273	0.024690	0.110000
Mefentrifluconazole	383	26 (6.8)	0.001870	0.006531	0.011191	0.027550	0.190000
Methoxyfenozide	539	2 (0.4)	0.000041	0.005022	0.010004	0.011000	0.012000
Myclobutanil	539	1 (0.2)	0.000035	0.005025	0.010016	0.018700	0.018700
Phosmet	539	37 (6.9)	0.004490	0.009147	0.013803	0.065405	0.420000
Piperonyl butoxide	262	14 (5.3)	0.006966	0.011698	0.016431	0.130357	0.510000
Pirimicarb	539	65 (12.1)	0.006241	0.010638	0.015035	0.051754	0.300000
Propamocarb	2	2 (100.0)	0.033500	0.033500	0.033500	0.033500	0.035000
Propargite	539	2 (0.4)	0.000382	0.005364	0.010345	0.103000	0.170000
Pyraclostrobin	539	66 (12.2)	0.004628	0.009016	0.013404	0.037797	0.120000
Pyridaben	539	1 (0.2)	0.000020	0.005011	0.010002	0.011000	0.011000
Pyrimethanil	539	26 (4.8)	0.012858	0.017617	0.022376	0.266562	4.389800
Pyriproxyfen	539	13 (2.4)	0.000766	0.005645	0.010524	0.031746	0.100000
Spinosad	314	1 (0.3)	0.000143	0.005127	0.010111	0.045000	0.045000
Spirodiclofen	539	1 (0.2)	0.000076	0.005067	0.010058	0.041000	0.041000
Spirotetramat	523	7 (1.3)	0.000266	0.005199	0.010132	0.019857	0.041000
Sulfoxaflor	383	16 (4.2)	0.000945	0.005736	0.010527	0.022619	0.051000
Tebuconazole	539	96 (17.8)	0.008297	0.012406	0.016516	0.046583	0.510000
Tebufoenozide	539	36 (6.7)	0.003581	0.008247	0.012913	0.053608	0.240000
Tebufoenpyrad	539	7 (1.3)	0.000566	0.005501	0.010436	0.043571	0.130000
Tetraconazole	196	1 (0.5)	0.000148	0.005122	0.010097	0.029000	0.029000
Thiabendazole	539	4 (0.7)	0.007523	0.012486	0.017449	1.013725	1.459100
Thiacloprid	539	3 (0.6)	0.000095	0.005067	0.010039	0.017000	0.025000
Thiophanate-methyl	461	2 (0.4)	0.000330	0.005308	0.010286	0.076000	0.120000
Trifloxystrobin	539	13 (2.4)	0.000432	0.005312	0.010191	0.017923	0.036000

Table A2. Cont.

(b) Oranges							
Active Substance/Residue Definition	Analysed Samples, n	Quantified Samples, n (%)	Arithmetic Mean in All Analysed Samples (mg/kg)			Mean in Quantified Samples (mg/kg)	Maximum Quantified (mg/kg)
			LB	MB	UB		
2-Phenylphenol	329	6 (1.8)	0.001793	0.006702	0.011611	0.098333	0.300000
Acetamiprid	329	11 (3.3)	0.001330	0.006163	0.010996	0.039791	0.110000
Chlorpyrifos	329	4 (1.2)	0.000626	0.005565	0.010505	0.051500	0.080000
Clofentezine	329	3 (0.9)	0.000213	0.005167	0.010122	0.023333	0.037600
Cypermethrin	260	1 (0.4)	0.000304	0.005285	0.010265	0.079000	0.079000
Deltamethrin	329	5 (1.5)	0.000447	0.005371	0.010295	0.029400	0.076000
Fenpyroximate	329	4 (1.2)	0.000204	0.005143	0.010082	0.016750	0.022000
Fludioxonil	195	3 (1.5)	0.002047	0.006970	0.011893	0.133067	0.282200
Fluopyram	329	1 (0.3)	0.000030	0.005015	0.010000	0.010000	0.010000
Flupyradifurone	195	2 (1.0)	0.000251	0.005200	0.010149	0.024500	0.038000
Tau-Fluvalinate	329	6 (1.8)	0.001538	0.006447	0.011356	0.084350	0.220000
Fosetyl-Al	15	2 (13.3)	0.021600	0.086600	0.151600	0.162000	0.230000
Hexythiazox	329	7 (2.1)	0.000497	0.005390	0.010284	0.023343	0.045000
Imazalil	329	46 (14.0)	0.045159	0.049460	0.053761	0.322985	1.880000
Lambda-cyhalothrin	329	15 (4.6)	0.001349	0.006121	0.010893	0.029587	0.070000
Metalaxyl and metalaxyl-M (sum of isomers, expressed as metalaxyl)	329	2 (0.6)	0.000122	0.005091	0.010061	0.020000	0.027000
Phosmet	326	19 (5.8)	0.004469	0.009178	0.013887	0.076684	0.290000
Piperonyl butoxide	179	4 (2.2)	0.000793	0.005682	0.010570	0.035500	0.062000
Pyraclostrobin	329	14 (4.3)	0.001704	0.006491	0.011278	0.040036	0.141400
Pyrimethanil	329	8 (2.4)	0.004443	0.009322	0.014200	0.182725	0.640000
Pyriproxyfen	329	51 (15.5)	0.005105	0.009330	0.013555	0.032935	0.230000
Tebuconazole	329	1 (0.3)	0.000246	0.005231	0.010216	0.081000	0.081000
Tebuconazole	329	7 (2.1)	0.000921	0.005815	0.010708	0.043286	0.120000
Thiabendazole	329	1 (0.3)	0.008207	0.013191	0.018176	2.700000	2.700000

Table A2. Cont.

(c) Bananas							
Active Substance/Residue Definition	Analysed Samples, n	Quantified Samples, n (%)	Arithmetic Mean in All Analysed Samples (mg/kg)			Mean in Quantified Samples (mg/kg)	Maximum Quantified (mg/kg)
			LB	MB	UB		
Atrazine	129	1 (0.8)	0.000705	0.005667	0.010628	0.091000	0.091000
Azoxystrobin	129	94 (72.9)	0.111173	0.112529	0.113886	0.152567	0.790000
Bifenthrin	106	4 (3.8)	0.000500	0.005311	0.010123	0.013250	0.018000
Total measured copper	17	16 (94.1)	0.698118	0.698412	0.698706	0.741750	2.300000
Dithiocarbamates (expressed as CS ₂)	26	1 (3.8)	0.000654	0.005462	0.010269	0.017000	0.017000
Fenpropidin	129	1 (0.8)	0.000155	0.005116	0.010078	0.020000	0.020000
Fenpropimorph	101	7 (6.9)	0.001495	0.006149	0.010802	0.021571	0.035000
Fludioxonil	73	2 (2.7)	0.003630	0.008493	0.013356	0.132500	0.190000
Lambda-cyhalothrin	129	1 (0.8)	0.000093	0.005054	0.010016	0.012000	0.012000
Myclobutanil	129	7 (5.4)	0.004578	0.009306	0.014035	0.084357	0.190000
Piperonyl butoxide	72	1 (1.4)	0.000153	0.005083	0.010014	0.011000	0.011000
Pyriproxyfen	129	35 (27.1)	0.010360	0.014003	0.017647	0.038183	0.170000
Thiabendazole	129	80 (62.0)	0.096109	0.098008	0.099907	0.154975	1.174000

Note: All active substances quantified in at least one sample are presented. Quantified samples were defined as results at or above the corresponding limit of quantification (LOQ), and percentages were calculated using the analyte-specific denominator. For analysed results below the LOQ, arithmetic mean concentrations in all samples were calculated separately under the three censoring scenarios: lower bound (LB) = 0, middle bound (MB) = LOQ/2, and upper bound (UB) = LOQ. Mean concentrations in quantified samples and maximum concentrations were calculated using only results at or above the LOQ. Substances that were never quantified are not presented.

Table A3. Substance-specific deterministic chronic dietary exposure to pesticide residues from apples, oranges, and bananas under the middle-bound scenario, expressed as a percentage of the acceptable daily intake (%ADI) and calculated using EFSA PRIMo revision 3.1.

Active Substance/Residue Definition	ADI (mg/kg bw/day)	%ADI–Apples	%ADI–Oranges	%ADI–Bananas	Aggregate %ADI–Three Fruits
2-Phenylphenol	0.4	0.001705	0.002408	—	0.004113
Acetamiprid	0.005	0.287092	0.177169	—	0.464261
Atrazine	0.02	—	—	0.029087	0.029087
Azoxystrobin	0.2	—	—	0.057761	0.057761
Bifenthrin	0.015	—	—	0.036350	0.036350
Boscalid	0.04	0.059131	—	—	0.059131

Table A3. Cont.

Active Substance/Residue Definition	ADI (mg/kg bw/day)	%ADI–Apples	%ADI–Oranges	%ADI–Bananas	Aggregate %ADI–Three Fruits
Captan	0.25	0.002564	—	—	0.002564
Carbendazim and benomyl (expressed as carbendazim)	0.02	0.030297	—	—	0.030297
Chlorantraniliprole	1.56	0.000609	—	—	0.000609
Chlorpyrifos	NA	—	—	—	—
Clofentezine	0.017	—	0.043687	—	0.043687
Total measured copper	NA	—	—	—	—
Cyantraniliprole	0.01	0.060786	—	—	0.060786
Cyflumetofen	0.17	0.003822	—	—	0.003822
Cypermethrin	0.005	—	0.151913	—	0.151913
Cyprodinil	0.03	0.027315	—	—	0.027315
Deltamethrin	0.01	0.056732	0.077195	—	0.133928
Difenoconazole	0.01	0.069744	—	—	0.069744
Diphenylamine	0.075	0.007481	—	—	0.007481
Dithianon	0.01	0.119390	—	—	0.119390
Dithiocarbamates (expressed as CS ₂)	NA	—	—	—	—
Dodine	0.1	0.011597	—	—	0.011597
Etofenprox	0.03	0.050040	—	—	0.050040
Fenbuconazole	0.006	0.093931	—	—	0.093931
Fenoxycarb	0.053	0.010798	—	—	0.010798
Fenpropidin	0.02	—	—	0.026262	0.026262
Fenpropimorph	0.003	—	—	0.210401	0.210401
Fenpyroximate	0.01	0.056400	0.073919	—	0.130319
Flonicamid	0.025	0.032806	—	—	0.032806
Fluazinam	0.01	0.066867	—	—	0.066867
Fludioxonil	0.37	0.004110	0.002708	0.002356	0.009174
Fluopyram	0.012	0.092778	0.060070	—	0.152848
Flupyradifurone	0.064	0.010301	0.011678	—	0.021979
Fluxapyroxad	0.02	0.039682	—	—	0.039682
Fosetyl-Al	1	0.038820	0.012447	—	0.051267
Hexythiazox	0.03	0.018929	0.025825	—	0.044754
Imazalil	0.025	0.078842	0.284357	—	0.363199

Table A3. Cont.

Active Substance/Residue Definition	ADI (mg/kg bw/day)	%ADI–Apples	%ADI–Oranges	%ADI–Bananas	Aggregate %ADI–Three Fruits
Imidacloprid	0.06	0.009345	—	—	0.009345
Indoxacarb	0.005	0.120940	—	—	0.120940
Isopyrazam	0.03	0.018745	—	—	0.018745
Lambda-cyhalothrin	0.0025	0.240211	0.351909	0.207547	0.799667
Mefentrifluconazole	0.035	0.020885	—	—	0.020885
Metalaxyl and metalaxyl-M (sum of isomers, expressed as metalaxyl)	0.08	—	0.009147	—	0.009147
Methoxyfenozide	0.1	0.005621	—	—	0.005621
Myclobutanil	0.025	0.022499	—	0.038215	0.060714
Phosmet	0.001	1.023755	1.319150	—	2.342905
Piperonyl butoxide	NA	—	—	—	—
Pirimicarb	0.035	0.034020	—	—	0.034020
Propamocarb	0.29	0.012930	—	—	0.012930
Propargite	0.03	0.020011	—	—	0.020011
Pyraclostrobin	0.03	0.033638	0.031098	—	0.064736
Pyridaben	0.01	0.056089	—	—	0.056089
Pyrimethanil	0.013	0.151680	0.103061	—	0.254741
Pyriproxyfen	0.05	0.012637	0.026821	0.028751	0.068209
Spinosad	0.024	0.023912	—	—	0.023912
Spirodiclofen	0.015	0.037808	—	—	0.037808
Spirotetramat	0.05	0.011638	—	—	0.011638
Sulfoxaflor	0.04	0.016051	—	—	0.016051
Tau-fluvalinate	0.005	0.146868	0.185330	—	0.332198
Tebuconazole	0.03	0.046287	0.025062	—	0.071349
Tebufenozide	0.02	0.046151	—	—	0.046151
Tebufenpyrad	0.01	0.061571	0.083574	—	0.145144
Tetraconazole	0.004	0.143336	—	—	0.143336
Thiabendazole	0.1	0.013975	0.018960	0.100614	0.133550
Thiacloprid	0.01	0.056711	—	—	0.056711
Thiophanate-methyl	0.02	0.029706	—	—	0.029706
Trifloxystrobin	0.1	0.005945	—	—	0.005945

Note: %ADI was calculated as $100 \times$ the estimated chronic daily intake divided by the corresponding acceptable daily intake (ADI). The middle-bound (MB) scenario was applied, with results below the limit of quantification (LOQ) assigned LOQ/2. NA indicates that no suitable current ADI was identified and %ADI was therefore not calculated. A dash (—) indicates that no commodity-specific %ADI was calculated because the active substance was not quantified in the corresponding commodity.

Table A4. Active substances and endpoint-specific reference points used in the EFSA CAG-based MOET assessment.

Established Chronic CAG Endpoint	Active Substance	Endpoint-Specific NOAEL (mg/kg bw/day)	Toxicological Indicator	Commodity with Quantified Residue	Use in MOET Model	Source
Chronic brain and/or erythrocyte acetylcholinesterase inhibition	Chlorpyrifos	0.1	AChE inhibition (erythrocytes)	Apple, Orange	Included	[12]
Chronic brain and/or erythrocyte acetylcholinesterase inhibition	Dimethoate	0.1	AChE inhibition (brain, erythrocytes)	None	Excluded—no quantified residue	[12]
Chronic brain and/or erythrocyte acetylcholinesterase inhibition	Malathion	17	AChE inhibition (erythrocytes)	None	Excluded—no quantified residue	[12]
Chronic brain and/or erythrocyte acetylcholinesterase inhibition	Omethoate	0.027	AChE inhibition (erythrocytes)	None	Excluded—no quantified residue	[12]
Chronic brain and/or erythrocyte acetylcholinesterase inhibition	Phosmet	1.1	AChE inhibition (brain)	Apple, Orange	Included	[12]
Chronic brain and/or erythrocyte acetylcholinesterase inhibition	Pirimicarb	10	AChE inhibition (brain, erythrocytes)	Apple	Included	[12]
Hypothyroidism	2,4-D	5	Increased relative thyroid weight	None	Excluded—no quantified residue	[13]
Hypothyroidism	Boscalid	22	Increased relative thyroid weight	Apple	Included	[13]
Hypothyroidism	Clofentezine	1.72	Follicular cell adenoma	Orange	Included	[13]
Hypothyroidism	Cyantraniliprole	7	Follicular cell hypertrophy	Apple	Included	[13]
Hypothyroidism	Cyflufenamid	20	Increased relative thyroid weight; follicular cell hypertrophy, adenoma and carcinoma	None	Excluded—no quantified residue	[13]
Hypothyroidism	Cyprodinil	3.14	Follicular cell hypertrophy	Apple	Included	[13]

Table A4. Cont.

Established Chronic CAG Endpoint	Active Substance	Endpoint-Specific NOAEL (mg/kg bw/day)	Toxicological Indicator	Commodity with Quantified Residue	Use in MOET Model	Source
Hypothyroidism	Dithianon	7.9	Increased relative thyroid weight	Apple	Included	[13]
Hypothyroidism	Etofenprox	25.5	Increased relative thyroid weight	Apple	Included	[13]
Hypothyroidism	Fenbuconazole	3	Increased relative thyroid weight; follicular cell hyperplasia and adenoma/carcinoma	Apple	Included	[13]
Hypothyroidism	Fenoxycarb	10.1	Follicular cell hypertrophy	Apple	Included	[13]
Hypothyroidism	Fluopicolide	32	Follicular cell hypertrophy/hyperplasia	None	Excluded—no quantified residue	[13]
Hypothyroidism	Fluopyram	1.2	Follicular cell hypertrophy	Apple, Orange	Included	[13]
Hypothyroidism	Fluxapyroxad	2.7	Increased relative thyroid weight	Apple	Included	[13]
Hypothyroidism	Myclobutanil	15	Increased relative thyroid weight	Apple, Banana	Included	[13]
Hypothyroidism	Penthiopyrad	60	Follicular cell hypertrophy	None	Excluded—no quantified residue	[13]
Hypothyroidism	Pyrimethanil	17	Follicular cell hypertrophy/hyperplasia	Apple, Orange	Included	[13]
Hypothyroidism	Spinosad	2.7	Increased relative thyroid weight	Apple	Included	[13]
Hypothyroidism	Spirodiclofen	19.88	Follicular cell hyperplasia	Apple	Included	[13]

Table A4. Cont.

Established Chronic CAG Endpoint	Active Substance	Endpoint-Specific NOAEL (mg/kg bw/day)	Toxicological Indicator	Commodity with Quantified Residue	Use in MOET Model	Source
Hypothyroidism	Sulfoxaflor	79.4	Increased relative thyroid weight	Apple	Included	[13]
Hypothyroidism	Tetraconazole	1	Follicular cell hypertrophy	Apple	Included	[13]
Hypothyroidism	Thiabendazole	10	Follicular cell hypertrophy/hyperplasia; follicular cell adenoma	Apple, Banana, Orange	Included	[13]
Hypothyroidism	Thiacloprid	1.2	Follicular cell hypertrophy	Apple	Included	[13]
Hypothyroidism	Thiophanate-methyl	8	Increased relative thyroid weight; follicular cell hypertrophy/hyperplasia	Apple	Included	[13]

Notes: AChE, acetylcholinesterase; CAG, cumulative assessment group; MOET, total margin of exposure; NOAEL, no-observed-adverse-effect level. Endpoint assignments and reference points were taken from [12], Table B.8, and [13], Table B.1. Assignments were non-exclusive. Only substances quantified at least once in an assessed commodity contributed to cumulative normalised exposure and MOET. Quantified substances not listed in Table A4 lacked an eligible EFSA CAG assignment and/or endpoint-specific NOAEL for either of these two endpoints and were therefore not included in the MOET calculation. Chlorpyrifos was retained in the AChE MOET model using its endpoint-specific NOAEL despite the absence of a current ADI; this affected only the deterministic %ADI assessment. Numerical MOET estimates and uncertainty intervals are reported in Table A5.

Table A5. Endpoint-specific chronic cumulative total margins of exposure (MOETs) under the lower-bound, middle-bound, and upper-bound scenarios, with complete 95% monitoring-sample outer-bootstrap uncertainty intervals.

Endpoint-Specific Cumulative Assessment Group	Scenario	MOET at P50 Exposure (95% UI)	MOET at P95 Exposure (95% UI)	MOET at P99 Exposure (95% UI)	MOET at P99.9 Exposure (95% UI)
Chronic brain and/or erythrocyte acetylcholinesterase inhibition	LB	55,270 (35,697–100,050)	18,121 (11,435–33,309)	15,021 (9627–26,848)	13,023 (8317–23,276)
Chronic brain and/or erythrocyte acetylcholinesterase inhibition	MB	7395 (6925–7841)	2482 (2309–2628)	1995 (1857–2113)	1732 (1617–1832)

Table A5. Cont.

Endpoint-Specific Cumulative Assessment Group	Scenario	MOET at P50 Exposure (95% UI)	MOET at P95 Exposure (95% UI)	MOET at P99 Exposure (95% UI)	MOET at P99.9 Exposure (95% UI)
Chronic brain and/or erythrocyte acetylcholinesterase inhibition	UB	3964 (3833–4087)	1324 (1286–1369)	1070 (1031–1101)	928 (897–954)
Hypothyroidism	LB	48,656 (40,504–59,197)	17,434 (14,649–20,675)	13,818 (11,644–16,370)	12,065 (10,216–14,244)
Hypothyroidism	MB	19,649 (18,027–21,095)	6482 (6077–6901)	5200 (4883–5550)	4666 (4354–4971)
Hypothyroidism	UB	12,251 (11,659–12,851)	3944 (3797–4092)	3194 (3061–3317)	2854 (2740–2957)

Notes: Values are exact point estimates followed by 95% monitoring-sample outer-bootstrap uncertainty intervals (UIs) in parentheses. MOET, total margin of exposure; LB, lower-bound scenario, in which results below the limit of quantification (LOQ) were assigned zero; MB, middle-bound scenario, in which results below the LOQ were assigned LOQ/2; UB, upper-bound scenario, in which results below the LOQ were assigned the LOQ. Point estimates were calculated using the commodity–substance arithmetic mean concentrations and the joint empirical distribution of body weight and corrected fruit consumption among all 1318 participants. MOET at each exposure percentile was calculated as the reciprocal of the corresponding percentile of cumulative normalised exposure and therefore does not represent the same percentile of the participant-specific MOET distribution. For each censoring scenario, 95% UIs were obtained as the 2.5th and 97.5th percentiles of 5000 outer non-parametric bootstrap replicates of the analysed monitoring observations, stratified by active substance, commodity, and year, with the original stratum sample sizes preserved. In each bootstrap replicate, the resulting commodity–substance arithmetic mean concentrations were propagated through the cumulative exposure model using all 1318 original participant records. Participant records, endpoint assignments, and endpoint-specific NOAELs were held fixed; consequently, the intervals quantify sampling uncertainty in the monitoring means but not uncertainty in the participant sample, common 24HR-based portion estimates, toxicological reference points, or other structural assumptions and should not be interpreted as overall uncertainty intervals. A MOET of 100 was used as the screening reference value. A MOET of 100 was used as the screening reference value. All point estimates and all lower bounds of the 95% UIs exceeded 100.

References

1. Popp, J.; Pető, K.; Nagy, J. Pesticide Productivity and Food Security. A Review. *Agron. Sustain. Dev.* **2013**, *33*, 243–255. [\[CrossRef\]](#)
2. Lebelo, K.; Malebo, N.; Mochane, M.J.; Masinde, M. Chemical Contamination Pathways and the Food Safety Implications along the Various Stages of Food Production: A Review. *Int. J. Environ. Res. Public Health* **2021**, *18*, 5795. [\[CrossRef\]](#) [\[PubMed\]](#)
3. Claeys, W.L.; Schmit, J.-F.; Bragard, C.; Maghuin-Rogister, G.; Pussemier, L.; Schiffers, B. Exposure of Several Belgian Consumer Groups to Pesticide Residues through Fresh Fruit and Vegetable Consumption. *Food Control* **2011**, *22*, 508–516. [\[CrossRef\]](#)
4. Crépet, A.; Vanacker, M.; Sprong, C.; De Boer, W.; Blaznik, U.; Kennedy, M.; Anagnostopoulos, C.; Christodoulou, D.L.; Ruprich, J.; Rehurkova, I.; et al. Selecting Mixtures on the Basis of Dietary Exposure and Hazard Data: Application to Pesticide Exposure in the European Population in Relation to Steatosis. *Int. J. Hyg. Environ. Health* **2019**, *222*, 291–306. [\[CrossRef\]](#) [\[PubMed\]](#)
5. European Food Safety Authority (EFSA); Craig, P.S.; Dujardin, B.; Hart, A.; Hernandez-Jerez, A.F.; Hougaard Bennekou, S.; Kneuer, C.; Ossendorp, B.; Pedersen, R.; Wolterink, G.; et al. Cumulative Dietary Risk Characterisation of Pesticides That Have Chronic Effects on the Thyroid. *EFSA J.* **2020**, *18*, e06088. [\[CrossRef\]](#) [\[PubMed\]](#)
6. European Food Safety Authority (EFSA); Craig, P.S.; Dujardin, B.; Hart, A.; Hernández-Jerez, A.F.; Hougaard Bennekou, S.; Kneuer, C.; Ossendorp, B.; Pedersen, R.; Wolterink, G.; et al. Cumulative Dietary Risk Characterisation of Pesticides That Have Acute Effects on the Nervous System. *EFSA J.* **2020**, *18*, e06087. [\[CrossRef\]](#) [\[PubMed\]](#)
7. European Food Safety Authority (EFSA); Dujardin, B. Comparison of Cumulative Dietary Exposure to Pesticide Residues for the Reference Periods 2014–2016 and 2016–2018. *EFSA J.* **2021**, *19*, e06394. [\[CrossRef\]](#) [\[PubMed\]](#)
8. Jensen, B.H.; Petersen, A.; Nielsen, E.; Christensen, T.; Poulsen, M.E.; Andersen, J.H. Cumulative Dietary Exposure of the Population of Denmark to Pesticides. *Food Chem. Toxicol.* **2015**, *83*, 300–307. [\[CrossRef\]](#) [\[PubMed\]](#)
9. Ambrus, Á.; Szenczi-Cseh, J.; Bíró, L.; Vászárhelyi, A.; Dobrik, H.S. Assessment of Hungarian Consumers' Exposure to Pesticide Residues Based on the Results of Pesticide Residue Monitoring between 2017 and 2021. *Agrochemicals* **2023**, *2*, 458–483. [\[CrossRef\]](#)
10. European Food Safety Authority (EFSA); Medina Pastor, P.; Carrasco Cabrera, L.; Di Piazza, G.; González Ciria, C. The 2024 European Union Report on Pesticide Residues in Food. *EFSA J.* **2026**, *24*, e10054. [\[CrossRef\]](#) [\[PubMed\]](#)
11. Nougadère, A.; Merlo, M.; Héraud, F.; Réty, J.; Truchot, E.; Vial, G.; Cravedi, J.-P.; Leblanc, J.-C. How Dietary Risk Assessment Can Guide Risk Management and Food Monitoring Programmes: The Approach and Results of the French Observatory on Pesticide Residues (ANSES/ORP). *Food Control* **2014**, *41*, 32–48. [\[CrossRef\]](#)
12. European Food Safety Authority (EFSA); Crivellente, F.; Hart, A.; Hernández-Jerez, A.F.; Hougaard Bennekou, S.; Pedersen, R.; Terron, A.; Wolterink, G.; Mohimont, L. Establishment of Cumulative Assessment Groups of Pesticides for Their Effects on the Nervous System. *EFSA J.* **2019**, *17*, e05800. [\[CrossRef\]](#) [\[PubMed\]](#)
13. European Food Safety Authority (EFSA); Crivellente, F.; Hart, A.; Hernández-Jerez, A.F.; Hougaard Bennekou, S.; Pedersen, R.; Terron, A.; Wolterink, G.; Mohimont, L. Establishment of Cumulative Assessment Groups of Pesticides for Their Effects on the Thyroid. *EFSA J.* **2019**, *17*, e05801. [\[CrossRef\]](#) [\[PubMed\]](#)
14. Bredsdorff, L.; Nørby, K.; Petersen, A.B.; Beltoft, V.; Boberg, J. Update of a Database of Toxicological Effects of Pesticides in View of Performing Cumulative Risk Assessments. Part 1: Catalogue of Toxicity Endpoints and Data Model for the Collection of Toxicity Endpoints. *EFSA Support. Publ.* **2024**, *21*, 9137E. [\[CrossRef\]](#)
15. European Food Safety Authority (EFSA); Di Piazza, G.; Dujardin, B.; Levorato, S.; Medina, P.; Mohimont, L.; Solazzo, E.; Costanzo, V. Prioritisation of Pesticides and Target Organ Systems for Dietary Cumulative Risk Assessment Based on the 2019–2021 Monitoring Cycle. *EFSA J.* **2024**, *22*, e8554. [\[CrossRef\]](#) [\[PubMed\]](#)
16. Larsson, M.O.; Sloth Nielsen, V.; Bjerre, N.; Laporte, F.; Cedergreen, N. Refined Assessment and Perspectives on the Cumulative Risk Resulting from the Dietary Exposure to Pesticide Residues in the Danish Population. *Food Chem. Toxicol.* **2018**, *111*, 207–267. [\[CrossRef\]](#) [\[PubMed\]](#)
17. Gkountouras, D.; Boti, V.; Albanis, T. High Resolution Mass Spectrometry Targeted Analysis and Suspect Screening of Pesticide Residues in Fruit Samples and Assessment of Dietary Exposure. *Environ. Pollut.* **2024**, *352*, 124143. [\[CrossRef\]](#) [\[PubMed\]](#)
18. Gkountouras, D.; Boti, V.; Albanis, T. Pesticides and Transformation Products Footprint in Greek Market Basket Vegetables: Comprehensive Screening by HRMS and Health Risk Assessment. *Sci. Total Environ.* **2024**, *953*, 176085. [\[CrossRef\]](#) [\[PubMed\]](#)
19. Tsoutsis, C.S.; Konstantinou, I.K.; Hela, D.G. Organophosphorus Pesticide Residues in Greek Virgin Olive Oil: Levels, Dietary Intake and Risk Assessment. *Food Addit. Contam. Part A* **2008**, *25*, 1225–1236. [\[CrossRef\]](#) [\[PubMed\]](#)
20. Tzatzarakis, M.; Kokkinakis, M.; Renieri, E.; Goumenou, M.; Kavvalakis, M.; Vakonaki, E.; Chatzinikolaou, A.; Stivaktakis, P.; Tsakiris, I.; Rizos, A.; et al. Multiresidue Analysis of Insecticides and Fungicides in Apples from the Greek Market. Applying an Alternative Approach for Risk Assessment. *Food Chem. Toxicol.* **2020**, *140*, 111262. [\[CrossRef\]](#) [\[PubMed\]](#)
21. Vlassi, E.; Bempelou, E.; Liapis, K.; Arapis, G. Consumer Safety Evaluation after Monitoring of Endocrine Disruptor Pesticide Residues: A Case Study of Thessaly, Central Greece. *Toxicol. Environ. Chem.* **2020**, *102*, 105–123. [\[CrossRef\]](#)
22. Simoglou, K.B.; Skarpa, P.E.; Roditakis, E. Pesticides and Eroding Food Citizenship: Understanding Individuals' Perspectives on the Greek Food System. *Agrochemicals* **2025**, *4*, 3. [\[CrossRef\]](#)

23. Simoglou, K.B.; Roditakis, E. Consumers' Benefit—Risk Perception on Pesticides and Food Safety—A Survey in Greece. *Agriculture* **2022**, *12*, 192. [CrossRef]
24. Trichopoulou, A.; Orfanos, P.; Valanou, E.; Martimianaki, G.; Peppas, E.; Androulidaki, A.; Anifantis, E.; Katsoulis, M.; Kritikou, M.; Pantzarlis, M.; et al. The EFSA-funded Collection of Dietary and Related Data in the General Population Aged 10–74 Years in Greece. *EFSA Support. Publ.* **2018**, *15*, 1499E. [CrossRef]
25. USDA. Food and Nutrient Database for Dietary Studies Documentation and Databases. Available online: <https://www.ars.usda.gov/northeast-area/beltsville-md-bhnrc/beltsville-human-nutrition-research-center/food-surveys-research-group/docs/fndds-download-databases/> (accessed on 15 April 2024).
26. Willett, W. *Nutritional Epidemiology*, 3rd ed.; Oxford University Press: Oxford, UK, 2012; ISBN 978-0-19-975403-8.
27. Kirkpatrick, S.I.; Baranowski, T.; Subar, A.F.; Toozé, J.A.; Frongillo, E.A. Best Practices for Conducting and Interpreting Studies to Validate Self-Report Dietary Assessment Methods. *J. Acad. Nutr. Diet.* **2019**, *119*, 1801–1816. [CrossRef] [PubMed]
28. Kirkpatrick, S.I.; Guenther, P.M.; Subar, A.F.; Krebs-Smith, S.M.; Herrick, K.A.; Freedman, L.S.; Dodd, K.W. Using Short-Term Dietary Intake Data to Address Research Questions Related to Usual Dietary Intake among Populations and Subpopulations: Assumptions, Statistical Techniques, and Considerations. *J. Acad. Nutr. Diet.* **2022**, *122*, 1246–1262. [CrossRef] [PubMed]
29. Ministry of Rural Development and Food. *Greek Results from the Monitoring of Pesticide Residues in Food (2022)*; Ministry of Rural Development and Food: Athens, Greece, 2024. [CrossRef]
30. Ministry of Rural Development and Food. *Greek Results from the Monitoring of Pesticide Residues in Food (2023)*; Ministry of Rural Development and Food: Athens, Greece, 2025. [CrossRef]
31. Ministry of Rural Development and Food. *Greek Results from the Monitoring of Pesticide Residues in Food (2024)*; Ministry of Rural Development and Food: Athens, Greece, 2026. [CrossRef]
32. Li, Z.; Zhang, Y.; Zhao, Q.; Cui, Y.; He, Y.; Li, J.; Yang, Q.; Lin, Z.; Wang, C.; Liang, G.; et al. Determination, Distribution and Potential Health Risk Assessment of Insecticides and Acaricides in Citrus Fruits of China. *J. Food Compos. Anal.* **2022**, *111*, 104645. [CrossRef]
33. European Food Safety Authority (EFSA); Anastassiadou, M.; Brancato, A.; Carrasco Cabrera, L.; Ferreira, L.; Greco, L.; Jarrah, S.; Kazocina, A.; Leuschner, R.; Magrans, J.O.; et al. Pesticide Residue Intake Model-EFSA PRIMo Revision 3.1. *EFSA Support. Publ.* **2019**, *16*, 1605E. [CrossRef]
34. European Commission. EU Pesticides Database (v3.4.). Available online: https://food.ec.europa.eu/plants/pesticides/eu-pesticides-database_en (accessed on 22 July 2026).
35. European Food Safety Authority (EFSA); Anastassiadou, M.; Choi, J.; Coja, T.; Dujardin, B.; Hart, A.; Hernandez-Jerrez, A.F.; Jarrah, S.; Lostia, A.; Machera, K.; et al. Cumulative Dietary Risk Assessment of Chronic Acetylcholinesterase Inhibition by Residues of Pesticides. *EFSA J.* **2021**, *19*, e06392. [CrossRef] [PubMed]
36. EFSA Panel on Plant Protection Products and Their Residues (PPR). Guidance on the Use of Probabilistic Methodology for Modelling Dietary Exposure to Pesticide Residues. *EFSA J.* **2012**, *10*, 2839. [CrossRef]
37. Vlahos, K. XLRISK Excel Add-In for Performing Monte Carlo Simulation. 2019. Available online: <https://github.com/pyscripter/XLRisk> (accessed on 14 June 2026).
38. JASP Team. *JASP*, Version 0.98.12026. Available online: <https://jasp-stats.org/> (accessed on 7 July 2026).
39. Liang, Y.; Liu, Y.; Ding, Y.; Liu, X.J. Meta-Analysis of Food Processing on Pesticide Residues in Fruits. *Food Addit. Contam. Part A* **2014**, *31*, 1568–1573. [CrossRef] [PubMed]

Disclaimer/Publisher's Note: The statements, opinions and data contained in all publications are solely those of the individual author(s) and contributor(s) and not of MDPI and/or the editor(s). MDPI and/or the editor(s) disclaim responsibility for any injury to people or property resulting from any ideas, methods, instructions or products referred to in the content.