

EVALUATION OF CERTAIN FOOD ADDITIVES AND CONTAMINANTS

Seventy-fourth report of the
Joint FAO/WHO Expert Committee on
Food Additives



Food and Agriculture
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United Nations



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3. Specific food additives

The Committee evaluated four food additives for the first time and re-evaluated a number of others. Information on the safety evaluations and on specifications is summarized in Annex 2. Details on further toxicological studies and other information required for certain substances are given in Annex 3.

3.1 Safety evaluations

3.1.1 *Aluminium-containing food additives*

Explanation

Aluminium can occur in food as a result of its natural occurrence in the environment, contamination from various sources, leaching from food contact materials and the use of aluminium-containing food additives.

Various aluminium compounds were evaluated by the Committee at its thirteenth, twenty-first, twenty-sixth, twenty-ninth, thirtieth, thirty-third and sixty-seventh meetings (Annex 1, references 20, 44, 59, 70, 73, 83 and 184). At its thirteenth meeting, the Committee established an acceptable daily intake (ADI) “not specified” for sodium aluminosilicate and aluminium calcium silicate (Annex 1, reference 20). At its twenty-sixth meeting, the Committee established a temporary ADI of 0–0.6 mg/kg body weight (bw) for sodium aluminium phosphate (Annex 1, reference 59). At its thirtieth meeting, the Committee noted concerns about a lack of precise information on the aluminium content of the diet and a need for additional safety data. The Committee extended the temporary ADI of 0–0.6 mg/kg bw expressed as aluminium to all aluminium salts added to food and recommended that aluminium in all its forms should be reviewed at a future meeting (Annex 1, reference 73).

The Committee evaluated aluminium as a contaminant at its thirty-third meeting, placing emphasis on estimates of consumer exposure, absorption and distribution of dietary aluminium and possible neurotoxicity, particularly the relationship between exposure to aluminium and Alzheimer disease. The Committee established a provisional tolerable weekly intake (PTWI) of 0–7.0 mg/kg bw for aluminium, and a consolidated monograph was produced

(Annex 1, reference 84). The Committee concluded that there was no need to set a separate ADI for the food additives sodium aluminium phosphate basic or sodium aluminium phosphate acidic, because the PTWI included aluminium exposure arising from food additive uses.

At its sixty-seventh meeting, the Committee re-evaluated aluminium used in food additives and from other sources and concluded that aluminium compounds have the potential to affect the reproductive system and developing nervous system at doses lower than those used in establishing the previous PTWI (Annex 1, reference 186). The Committee noted that the lowest lowest-observed-effect levels (LOELs) for aluminium in a range of different dietary studies in mice, rats and dogs were in the region of 50–75 mg/kg bw per day. The Committee selected the lower end of this range of LOELs (50 mg/kg bw per day) and established a PTWI of 1 mg/kg bw by applying an uncertainty factor of 100 to allow for interspecies and intraspecies differences and an additional uncertainty factor of 3 for deficiencies in the database, notably the absence of no-observed-effect levels (NOELs) in the majority of the studies evaluated and the absence of long-term studies on the relevant toxicological end-points. The PTWI applied to all aluminium compounds in food, including food additives. The previously established ADIs and PTWI for aluminium compounds were withdrawn. The Committee noted that the PTWI was likely to be exceeded to a large extent by some population groups, particularly children, who regularly consume foods that include aluminium-containing food additives. The Committee also noted that dietary exposure to aluminium is expected to be very high for infants fed on soya-based formula. The Committee noted a need for:

- further data on the bioavailability of different aluminium-containing food additives;
- an appropriate study of developmental toxicity and a multigeneration study incorporating neurobehavioural end-points using relevant aluminium compounds;
- studies to identify the forms of aluminium present in soya-based formula and their bioavailability.

Aluminium-containing food additives were re-evaluated by the Committee at its present meeting, as requested by CCFA. The Committee was asked to consider all data necessary for safety evaluation (bioavailability, developmental toxicity and multigeneration reproductive toxicity) and data on actual use levels in food. In addition, the Committee was asked to consider all data necessary for the assessment of safety, dietary exposure and specifications for aluminium lactate and potassium aluminium silicate, which had not been evaluated previously by the Committee for use as food additives. Potassium

aluminium silicate is mined from natural sources and then further purified for use as a carrier substrate for potassium aluminium silicate-based pearlescent pigments. Potassium aluminium silicate-based pearlescent pigments are produced by reaction of potassium aluminium silicate with soluble salts of titanium and/or iron followed by calcination at high temperatures. The pigments can be produced with a variety of different pearlescent colour effects depending upon particle size and the combination of titanium dioxide and/or iron oxide deposited on the potassium aluminium silicate.

The Committee received submissions from a number of sponsors, including unpublished studies of bioavailability and toxicity and a review of the scientific literature. Additional information was identified from the scientific literature. No information was received on the forms of aluminium present in soya-based infant formula.

Toxicological data

As recommended by the Committee at its sixty-seventh meeting, new studies had been conducted on the bioavailability of aluminium compounds. The new data indicated that absorption of aluminium following the ingestion of various aluminium compounds by rats is generally in the region of 0.01–0.3% and support the assumption that the more water-soluble aluminium compounds are generally more bioavailable. As a result of limitations in the sensitivity of the analytical methods, inter-animal variation and methodological differences between studies, including the administered doses, it is not possible to draw firm conclusions on quantitative differences in absorption between different compounds. There are indications that there are sex differences in absorption in rats and that the proportion of the dose absorbed is lower following repeated administration than following single administration. The reported absorptions of the food additives for which data were available (sodium aluminium phosphate acidic, sodium aluminium phosphate basic, sodium aluminosilicate, aluminium sulfate, FD&C aluminium lake, aluminium metal, aluminium ammonium sulfate) are within the overall range of 0.01–0.3% in rats. A possible exception relates to potassium aluminium silicate-based pearlescent pigments. These products are marketed in particulate form. The solubility of the particulates is very low, and therefore it is likely that the bioavailability is lower than for other aluminium-containing food additives. However, direct data to support a conclusion that aluminium is appreciably less available from these pigments than from other aluminium compounds were not available.

In studies reviewed previously by the Committee, absorption of aluminium in human volunteers was within the same range as that in rats, with some indication of increased absorption in the elderly. The absorption can be modified

by substances in foods that bind to the aluminium ion, such as citrate, which increases absorption, and phosphate, which forms an insoluble aluminium salt, thereby decreasing absorption. The newly available data indicate that absorption in humans is likely to vary widely, but did not support an estimation of bioavailability.

New studies in rats have confirmed that absorbed aluminium is able to cross the placental barrier into the fetus and then into the fetal brain and that it is also transferred to the offspring via lactation. The new studies have also confirmed that administration of a number of aluminium salts to rats can result in increased concentrations of aluminium in bone, kidney and spinal cord. About 90% of Al^{3+} in plasma is bound to transferrin, and about 10% to citrate. Cellular uptake is thought to occur from the aluminium bound to transferrin by transferrin receptor-mediated endocytosis.

No new data on excretion were identified. Studies reviewed previously by the Committee have shown that urine is the primary route of excretion of absorbed aluminium in experimental animals and in humans. Initial half-lives of 2–5 hours have been reported in rats, mice, rabbits and dogs after intravenous administration and less than 1 day in humans after intravenous administration. In different studies and species, multiple half-lives have been reported, arising from slower rates of elimination from different tissues.

Based on the available data relating to the absorption, distribution and elimination of aluminium from a variety of different aluminium compounds, the Committee concluded that there was no basis for deriving a chemical-specific adjustment factor for either interspecies or intraspecies differences in toxicokinetics.

As recommended by the Committee at its sixty-seventh meeting, new multigeneration reproductive and developmental toxicity studies incorporating neurobehavioural end-points had been conducted.

The multigeneration reproductive studies conducted with aluminium sulfate and aluminium ammonium sulfate administered to rats in the drinking-water did not provide evidence of reproductive toxicity. The major developmental effects observed in both studies were delayed maturation of the female offspring, decreased body weight gain and changes in some organ weights. These effects are likely to have been related to the reported decrease in maternal fluid and feed consumption. Thus, it is not possible to attribute the findings to a direct effect of the aluminium. No effects on motor activity or learning ability were observed in these studies.

The available developmental toxicity studies include two published studies involving dosing of aluminium chloride by oral gavage to pregnant rats. These studies provided evidence of fetotoxicity, but it was unclear if the findings

were secondary to maternal toxicity. There were no effects on pregnancy outcome in a developmental study of aluminium chloride basic.

Cognitive deficits were observed in a number of new studies of neurotoxicity and neurobehavioural end-points. Most of these studies have limitations for use in risk assessment, such as administration of only one high dose level, failure to consider aluminium content in the diet, lack of assessment of other forms of toxicity and assessment of only a limited number of outcomes. The lowest aluminium dose linked with cognitive effects was 0.5 mg/kg bw per day administered to rats as aluminium chloride in the drinking-water, which was reported to be associated with impaired memory in old rats. In this study, the rats were given a restricted amount of feed twice weekly in order to reduce the rats' weight to approximately 85% of the free-feeding weight and hence prolong their lifespan. Typically, they ate the feed in the first 2–3 days and had a day or more with no feed. Whereas impaired cognitive function in old age is a potentially relevant observation, the impact of the restricted feeding regimen used in this study is unknown, and impaired cognitive function has been observed in other studies only at much higher levels of exposure, albeit in younger animals. The Committee therefore concluded that the results of this study require independent verification and were not suitable for use in the risk assessment.

In a developmental and chronic neurotoxicity study of aluminium citrate administered to rats in drinking-water, the major treatment-related effects were renal damage (hydronephrosis, urethral dilatation, obstruction and/or presence of calculi) and reduced grip strength, but not cognitive impairment, in the pups. Renal damage was not observed in a control group of rats given sodium citrate at the molar equivalent of the high-dose aluminium citrate, demonstrating that the effect was not due to the citrate ion. Dosing with both aluminium citrate and sodium citrate resulted in a significant increase in fluid consumption compared with control animals. The no-observed-adverse-effect level (NOAEL) and lowest-observed-adverse-effect level (LOAEL) for these effects were at target aluminium doses of 30 and 100 mg/kg bw per day. However, because the aluminium citrate was administered in the drinking-water, the actual dose was influenced by the water consumption, which varied in the different stages of the study. Mean doses at the NOAEL were 10–14% below target during gestation, up to 50% above target during lactation, up to about 30% above target in the weaned pups for the first few weeks, but then 15–45% of target for the remainder of the study. At the LOAEL, the mean dosage level was approximately at target during gestation, up to 90% above target during lactation and the first few weeks post-weaning, and then 25–50% of target for the remainder of the study. Hence, if the effects in the pups were mediated in utero, the NOAEL is slightly overestimated; conversely, however, if the effects were mediated during lactation

or the first few weeks after weaning, the NOAEL is underestimated. As the effect on grip strength was more pronounced in younger animals, exposure in utero and/or during lactation is likely to be more important than exposure during the later stages, when exposure was decreased due to decreased fluid consumption. The Committee concluded that, taking into account the greater bioavailability of aluminium from aluminium citrate than from other aluminium compounds, it was appropriate to assume that the NOAEL was 30 mg/kg bw per day. In view of the uncertainty regarding the doses at different times of this study as a result of changes in water consumption, the Committee decided not to model the dose–response data.

The Committee received a submission specifically on potassium aluminium silicate–based pearlescent pigments. No effects were observed in subchronic or chronic toxicity studies at doses of the test material up to 2500 mg/kg bw per day, equivalent to 360 mg/kg bw per day as aluminium, but no studies were available regarding reproductive or neurobehavioural effects.

Most epidemiological studies reviewed addressed the potential neurotoxicity of aluminium in drinking-water or antacids, by means of different designs: experimental, prospective cohort or case–control studies or ecological studies. The results of these studies were controversial; some of the drinking-water studies showed an association of aluminium with dementia or Alzheimer disease, whereas others reported an absence of neuropsychological effects measured in several ways. None of these studies took into account the ingestion of aluminium in food. The coincidental observation of neuropathological features of Alzheimer disease and aluminium in brain reported in some cases does not demonstrate a causal role of aluminium in Alzheimer disease. Occupational exposure to aluminium does not seem to have an impact on cognitive performance, motor performance or adverse reproductive outcomes in exposed workers. Although recent studies do not definitively rule out a positive association between aluminium in drinking-water and Alzheimer disease, the information available remains inconsistent and does not support a causal association. Neonates who were exposed to aluminium from solutions for parenteral nutrition had reduced lumbar spine and hip bone mass in adolescence. However, in elderly people, the aluminium content in bones was not associated with increased risk of hip fractures. There was no information from the epidemiological literature about the potential effects of oral exposure to aluminium in food. Given these limitations, no pivotal epidemiological studies are available for risk assessment.

Assessment of dietary exposure

Owing to their multiple functions, aluminium-containing food additives are permitted for use in a large variety of foods. At its present meeting, the

Committee was asked to evaluate the safety of potassium aluminium silicate-based pearlescent pigments based on the recommendation of the Forty-second Session of CCFA (6). This aluminium-containing food additive has not previously been evaluated by the Committee.

Potassium aluminium silicate (mica) is used as a carrier substrate for titanium dioxide and/or iron oxide. Potassium aluminium silicate is not intended to be placed on the market as such, but only when coated with the food colours titanium dioxide and/or iron oxide. In the European Union (EU), E555 potassium aluminium silicate is approved as a carrier for E171 titanium dioxide and E172 iron oxides and hydroxides (maximum 90% potassium aluminium silicate relative to the pigment) (7). In the United States of America (USA), pearlescent pigments consisting of potassium aluminium silicate coated with titanium dioxide are approved for use as a colour additive at levels up to 1.25% in cereals, confections and frostings, gelatine desserts, hard and soft candies (including lozenges), nutritional supplement tablets and capsules, and chewing gum (8). Potassium aluminium silicate-based pearlescent pigments are proposed to be used in confectionery, chewing gums and beverages at usage levels ranging from a minimum of 0.02% up to a maximum of 1.25%.

The Committee noted that no actual usage data were submitted for aluminium ammonium sulfate (INS 523), sodium aluminium phosphate basic (541(ii)), aluminium silicate (INS 559), aluminium powder or aluminium potassium sulfate (INS 522). Currently used aluminium-containing food additives are aluminium sulfate (INS 520), sodium aluminosilicate (INS 554), sodium aluminium phosphate acidic (INS 541(i)) and aluminium lakes of food colour.

At the sixty-seventh meeting, the Committee considered only consumer exposure to aluminium in the diet; occupational exposure and other routes or commodities were not considered. Dietary sources of exposure include natural dietary sources, drinking-water, migration from food contact materials and food additives. The potential range of exposure to aluminium from dietary sources reviewed at the sixty-seventh meeting by the Committee was 14–280 mg/week (Table 1).

For the evaluation of potassium aluminium silicate-based pearlescent pigments as a new food additive, the Committee evaluated an anticipated dietary exposure assessment based on food consumption data from the EU and the USA with the maximum proposed levels of use of potassium aluminium silicate-based pearlescent pigments. The Committee concluded that anticipated dietary exposure in the general population from the use of this food colour at the maximum proposed use levels (0.5% in beverages and 1.25% by weight in solid food) would range from 10 mg/kg bw per day at the mean to 323

Table 1

Estimated ranges of mean exposure of the adult population to aluminium from different dietary sources

Country/region	Estimated exposure from food additives used in cereals and cereal-based products (mg/person per week)	Estimated exposure from overall diet including natural sources, water consumption, food contact materials and food additives (mg/person per week)
WHO ^a	—	14–280
WHO ^b	2–124	11–136
Australia	4	17
Brazil	40–70	—
China	4–124	23–136
China, Hong Kong Special Administrative Region	30	36
Europe (EFSA)	2–46	11–91
Japan	—	84
USA	24–30	60

EFSA, European Food Safety Authority

^a Estimated ranges from the sixty-seventh meeting of the Committee (Annex 1, reference 184).

^b Estimated ranges from the data reviewed at this meeting.

mg/kg bw per day for consumers with a high consumption of non-alcoholic beverages. When converted to an aluminium basis, this corresponds to an aluminium exposure from potassium aluminium silicate-based pearlescent pigments of 1.8 mg/kg bw per day up to 58 mg/kg bw per day.

The Committee recognizes that its estimates are conservative, as it is assumed that all processed foods and beverages contain the colour added at the maximum proposed use levels. The Committee noted that non-alcoholic flavoured drinks are the major contributor in these estimates, accounting for 20–70% of overall dietary exposure.

For other aluminium-containing food additives under re-evaluation, a tentative estimate of dietary exposure from food additive sources has been made, taking into account previous assessments and other publications or submissions reviewed by the Committee at the current meeting. The Committee noted, from the report of its sixty-seventh meeting and from a European Food Safety Authority (EFSA) scientific opinion, that the range of estimates was mainly based on dietary exposure calculated with the total diet study method, which takes into account water consumption. It is known from the literature that the main sources of migration of aluminium into food are from the use of cookware or aluminium utensils. It is also known that the design of total

diet studies generally tries to control any bias of additional contamination that may result from the use of containers, cookware or utensils containing aluminium during the preparation and storage of food as consumed.

The Committee noted that estimates of the contribution to overall mean dietary exposure from all sources (including natural sources, water consumption, food contact materials and food additives) were in the range of 10–140 mg/week in adult populations (0.2–2.3 mg/kg bw per week as aluminium, assuming a body weight of 60 kg; Table 1). Major contributors to these estimates were cereals and cereal-based food products, with a proportion of 20–90%, depending on the country, equivalent to a dietary exposure of approximately 2–120 mg/week (0.03–2 mg/kg bw per week as aluminium, assuming a body weight of 60 kg).

This assessment is consistent with previous evaluations made by the Committee in which cereal products were considered as potentially high contributors to dietary aluminium exposure. The Committee also noted from its review that high levels of the actual uses of aluminium-containing food additives were reported for cereals and cereal-based products, in particular for sodium aluminosilicate (INS 554) and sodium aluminium phosphate acidic (INS 541(i)). Based on this, the Committee concluded that aluminium from the consumption of cereals and cereal-based products could reasonably be assumed to be mainly from food additive sources.

The Committee noted that the estimated dietary exposures related to average adult populations and that high dietary exposures (e.g. 90th or 95th percentile) are generally assumed to be 2 times higher than the reported average. It also noted that children generally have higher food intake than adults when expressed on a body weight basis and therefore represent the highest potential exposure to aluminium per kilogram of body weight.

Evaluation

The new data submitted to the Committee and available in the published literature addressed some of the research needs identified previously, including studies of bioavailability and reproductive, developmental and neurobehavioural effects.

The absorption of aluminium compounds is generally in the region of 0.01–0.3%. Soluble aluminium compounds appear to be more bioavailable, but it is not possible to draw conclusions on quantitative differences in the overall toxicokinetics of different aluminium-containing food additives or between experimental animals and humans.

The recent evidence did not show effects of aluminium on reproductive outcomes. The new studies support previous observations of neurodevelop-

mental effects in experimental animals, but there continues to be a lack of consistency regarding the reported effects, and there are some limitations to all of the studies. Most of the studies involved administration of aluminium compounds in drinking-water, rather than in the diet.

At its current meeting, the Committee noted that the new data did not substantially change the LOAEL range of 50–75 mg/kg bw per day, but one of the studies also provided a NOAEL of 30 mg/kg bw per day. This NOAEL was identified from a study in which aluminium citrate was administered in drinking-water. Aluminium citrate is more soluble than many other aluminium compounds and is likely to be more bioavailable from drinking-water than from food. The Committee concluded that the NOAEL of 30 mg/kg bw per day was an appropriate basis for establishing a PTWI for aluminium compounds. Because long-term studies on the relevant toxicological endpoints had become available since the sixty-seventh meeting, there was no longer a requirement for an additional uncertainty factor for deficiencies in the database. The Committee therefore established a PTWI of 2 mg/kg bw from the NOAEL of 30 mg/kg bw per day by applying an uncertainty factor of 100 for interspecies and intraspecies differences. The previous PTWI of 1 mg/kg bw was withdrawn.

The data submitted on aluminium lactate and potassium aluminium silicate-based pearlescent pigments were insufficient to demonstrate that these food additives differ from other forms of aluminium in their bioavailability or toxicity. The PTWI applies to all aluminium compounds in food, including food additives. The Committee emphasized that whereas substances that have long half-lives and accumulate in the body are not generally considered suitable for use as food additives, consumption of aluminium-containing food additives would not be a health concern, provided that total dietary exposure to aluminium is below the PTWI.

The Committee concluded that, for adults, the estimates of mean dietary exposure to aluminium-containing food additives from consumption of cereals and cereal-based products are up to the PTWI of 2 mg/kg bw. Estimates of dietary exposure of children to aluminium-containing food additives, including high-level dietary exposure, can exceed the PTWI by up to 2-fold.

For potassium aluminium silicate-based pearlescent pigments at the maximum proposed use levels and using conservative estimates, the Committee noted that anticipated dietary exposure at the highest range of estimates is 200 times higher than the PTWI of 2 mg/kg bw.

Therefore, the Committee recommended that provisions for food additives containing aluminium included in the GSFA should be compatible with the

revised PTWI for aluminium compounds of 2 mg/kg bw as aluminium from all sources.

There is a need for convincing data to demonstrate that aluminium is not bioavailable from potassium aluminium silicate-based pearlescent pigments.

No data were available to identify the forms of aluminium present in soya-based formula and their bioavailability. Such studies were requested at the sixty-seventh meeting and are still required.

An addendum to the toxicological monograph was prepared.

The Committee received no data on the use of aluminium lactate as a food additive or on the manufacture, assay, impurities or use levels in food. The Committee decided that it was not appropriate to develop specifications for aluminium lactate.

The Committee prepared new tentative specifications for pearlescent pigments containing potassium aluminium silicate at the request of CCFA at its Forty-second Session (6). Limited data were received on potassium aluminium silicate itself as well as for the three general types of pearlescent pigments made using potassium aluminium silicate with titanium dioxide, iron oxide or both titanium dioxide and iron oxide. Based on the data received, the Committee decided to prepare specifications for potassium aluminium silicate itself, as well as a combined specification for the three general types of potassium aluminium silicate-based pearlescent pigments manufactured using potassium aluminium silicate combined with titanium dioxide, iron oxide or both titanium dioxide and iron oxide.

In the case of potassium aluminium silicate, information is required on preparation and purification methods, particle size distribution, methods of identification for silicate and aluminium, data on the levels of the inorganic impurities, the suitability of an inductively coupled plasma atomic emission spectroscopy (ICP-AES) method for the determination of inorganic impurities, and the suitability of a proposed method based on alkali fusion followed by ICP-AES for the assay for potassium aluminium silicate based on the determination of aluminium.

In the case of potassium aluminium silicate-based pearlescent pigments, information is required on their manufacture, stability in food, particle size distribution, pH range, methods for the identification of iron, titanium and aluminium, data on the levels of the inorganic impurities, the suitability of an ICP-AES method for the determination of inorganic impurities, a filtration method appropriate for the small particle sizes associated with the pigments, and the suitability of a proposed method based on alkali fusion followed by ICP-AES for the assay for titanium, iron and aluminium.

New specifications for potassium aluminium silicate and potassium aluminium silicate-based pearlescent pigments were prepared and made tentative. The requested information should be made available by the end of 2012.

3.1.2 *Benzoe Tonkinensis*

Explanation

Benzoe Tonkinensis was placed on the agenda of the current meeting at the request of CCFA during its Forty-second Session (6). The Committee has not previously evaluated *Benzoe Tonkinensis*.

Benzoe Tonkinensis is a balsamic resin from the *Styrax tonkinensis* (Pierre) Craib ex Hartwich tree, which belongs to the Styracaceae family. It is variously referred to as Siam benzoin gum, Siam benzoin and benzoin Laos or in a generic way as “benzoin gum”.

Two varieties of benzoin gums occur: *Benzoe Tonkinensis* and *Benzoe Sumatranus*. These two resins differ in their botanical source, geographical origin and chemical composition. The term “benzoin gum” can include resins from one or the other of the two sources or their mixtures.

The Committee previously considered benzoin gum at its twenty-first and fifty-fifth meetings (Annex 1, references 44 and 149) but did not evaluate it owing to the lack of analytical and toxicological data. At its twenty-first meeting, the Committee prepared a tentative specification covering the two forms of benzoin gum. However, no ADI was established, and no monograph was prepared. At its fifty-fifth meeting, the Committee withdrew the tentative specification for benzoin gum, as the relevant information was not provided.

Benzoe Tonkinensis is intended to be used as a flavouring agent in foods and beverages.

Chemical and technical considerations

Benzoe Tonkinensis has an opaque appearance and consists of grainy, ovoid, flattened almond-like splits, sometimes agglomerated by a brown-red transparent resin. The product has a strong vanilla flavour and is insoluble in water and soluble in ethanol.

The resin is composed mainly of coniferyl benzoate (15–60%) and benzoic acid (15–45%), with lesser amounts of vanillin (<5%), benzyl benzoate (<2%), 2-hydroxy-1-phenylethanone and 1-(4-hydroxy-3-methoxyphenyl)-2-propanone.

The Committee noted that there are large variations between samples of *Benzoe Tonkinensis* in the amounts of the four main components (coniferyl