



ERYTHROSINE

Explanation

Erythrosine (FD&C Red No. 3) is widely used as a coloring agent for foods, beverages, pharmaceutical preparations, and cosmetics. It has been evaluated for acceptable daily intake by the Joint FAO/WHO Expert Committee on Food Additives (see Annex 1, Ref. Nos. 8, 19 & 35) in 1964, 1969 and 1974. At the 18th Meeting (1974) of the Committee, an ADI of 0 - 2.5 mg/kg body weight was allocated. Toxicological monographs, were published in 1970 and 1975 (Annex 1, Ref. 20 and 36).

Since the previous evaluation, additional data have become available and are summarized and discussed in the following monograph. The previously published monographs have been expanded and are reproduced in their entirety below.

BIOLOGICAL DATA

BIOCHEMICAL ASPECTS

The metabolic behaviour and excretory pattern for erythrosine have been studied in adult rats. The colour was given by stomach tube in log-spaced doses from 0.5-500 mg per kg body weight. In five days the recovery in the excreta was 102%. After an intravenous application of 3 mg per kg body weight the urine and bile for the initial two to four hours was collected, an average of 55% (50.4 - 58.0%) of the administered quantity was found in bile. In the urine, the recovery was 1.3% (0.3 - 1.8%). No glucuronic acid conjugation was found. The colour was found to be largely excreted in the faeces by rats (55 -72%) and despite the presence of two groups capable of undergoing conjugations, no colour could be identified in the urine. A small amount of the colour (0.4 - 1.7%) was excreted in the bile (Daniel, 1962).

Consideration has been given to the possibility that iodide may be liberated from erythrosine and may disturb thyroid function. In the rat, erythrosine is metabolically stable and 100% of the amount is ingested and excreted with its iodine content intact after administration of 500 mg/kg (Webb et al., 1962). Protein-bound and total blood iodine levels were elevated in rats given erythrosine by stomach tube twice weekly in a chronic study (Bowie et al., 1966). However, the elevated PBIs (protein-bound iodine) were due to interference by erythrosine in PBI determinations rather than thyroid

dysfunction in rats and gerbils (Anonymous, 1969).

In man, oral administration of 16 mg of erythrosine daily for 10 days resulted in an increase of protein-bound iodine in the serum from 6-11 µg/100 ml after 15 to 20 days, followed by a sharp decline in iodine levels in the next 10 days with gradual return to the initial

value in three months (Anderson et al., 1964). Erythrosine could be an adventitious source of iodide (Vought et al., 1972). No biologically significant increases in plasma inorganic iodine or in urinary iodine excretion were found in six patients (ages 25-68 years, sex not reported) after oral exposure to 1.9 µmol (1,680 µg)/day of erythrosine for ten days. In other assays of thyroid function, thyroid radioiodine uptake, levels of thyroxine and protein bound iodine (PBI) in plasma remained unchanged (Berstein et al., 1975).

Large doses of erythrosine labelled with I₁₃₁ given orally to rats inhibited uptake of ¹³¹I by the thyroid of treated animals. Daily doses over 1 mg are necessary for this effect (Marignan et al., 1965).

When cherries coloured with erythrosine are stored in plain cans, fluorescein is readily formed by interaction of the tin-iron couple present. This does not occur in lacquered cans. The production of fluorescein (with 4 I atoms) from erythrosine occurs in presence of metallic iron and/or tin and free organic acid (result of electrochemical reduction in the can) (Dickinson & Raven, 1962).

It was found that this colour in a concentration of 200-400 mg/l, inhibited the action of pepsin but had no effect on lipase activity (Diemair & Hausser, 1951).

It was also found that this colour had in vivo as well as in vitro haemolytic effect. In the in vivo studies the mouse was used (Waliszewski, 1952).

Erythrosine was administered to rats in doses of 5, 10, 15 and 50 g per rat weighing 200-250 g twice weekly for six months. Haemoglobin was reduced at three months as was the red cell count. The cholesterol levels of males were depressed. Excretion of the dye was mainly in the faeces and predominantly unchanged (Bowie et al., 1966).

TOXICOLOGICAL STUDIES

Special studies on carcinogenicity

(see under long-term studies)

Special studies on mutagenicity

This colour was tested for mutagenic activity and showed a very slight but statistically significant mutagenic effect on Escherichia

coli in concentrations of 0.5 g/100 ml. It was found that the xanthene molecule itself was the causative factor and that the substituent groups only modify the effect (Lück et al., 1963; Lück & Rickeryl, 1960).

The lack of mutagenic activity of erythrosine for Salmonella typhimurium strains (TA 1535, TA 100, TA 1538, TA 98 and TA 1537) was observed when tested in the Ames test at concentrations ranging

from 1 to 10,000 µg/plate with or without metabolic activation system (Auletta et al., 1977; Bonin & Baker, 1980; Brown et al., 1978). Erythrosine was inactive in the host-mediated rec - Assay (Kada et al., 1972), in DNA-repair, fluctuation and treat-and-plate assays (Haveland-Smith et al., 1981) and did not induce rat embryo calls transformation in vitro (Price et al., 1978).

Special studies on reproduction

Rat

Four groups of Charles River CD rats (23-25 males and females/group) received erythrosine in the diet at dose levels of 0, 0.25, 1.0 or 4.0% for 3 consecutive generations. The F₀ parental rats received their respective diets for 69 days prior to mating. The study showed that during the gestation period slight to moderate reduction in mean material body weight gain was noted in females of all generations at the 1.0% and 4.0% dose levels. Slight to moderate reductions in mean pup body weight was recorded at the 4.0% level on lactation days 0, 4, 14 and 21 in all generation. These reductions were statistically significant only on lactation day 21. These were no consistent compound related effects on the reproductive performance of males and females and pups survival at any dose level in any generation (Albridge et al., 1981).

Groups of 18-22 pairs (males and females, weighing 200-220 g) of adult Sprague-Dawley rats were fed diets containing erythrosine at levels of 0, 0.25, 0.5 or 1.0% for 2 weeks before mating and during mating period. The diets were continued for the females throughout gestation and lactation and were provided continuously to their offspring until they reached 90-100 days. Positive control group did not receive erythrosine in the diet but offspring were injected daily with 50 mg/kg of hydroxyurea on post-natal days 2-10 of life. Two years later, a second experiment, a replication of the first one with the same dose groups and number of animals per group was performed. In both experiments, parental animals were evaluated for weight and food consumption and females for reproductive success. The offspring were assessed for behaviour toxicity plus weight, food consumption, physical development, and brain weight. Erythrosine produced no reductions in paternal or offspring weight or food consumption. Erythrosine significantly increased preweaning offspring mortality at

the 1.0 and 0.5% dose levels in the first experiment, but not in the second. Mean litter size was not adversely affected by erythrosine in both experiments. Behaviourally, erythrosine produced no dose-dependent effects that replicated across the two experiments. It was concluded that no evidence was obtained that erythrosine via dietary exposure at levels as high as 1.0% is psychotoxic to developing rats (Vorhees et al., 1983).

Acute toxicity

Animal	Route	LD ₅₀ mg/kg bw	Reference
Mouse	Oral	6800	Butterworth et al., 1976a
	i.p.	360	Butterworth et al., 1976a
	i.v.	370	Waliszewski, 1952, DFG, 1957
Rat	i.p.	300	Emerson & Anderson, 1934
		350	Butterworth et al., 1976a
	Oral	1895	Lu & Lavallee, 1964
		7100	Butterworth et al., 1976a
Rabbit	i.v.	1840	Hansen et al., 1973a
		200	Emerson & Anderson, 1934
Gerbil	Oral	1930	Anonymous, 1969

A group of five young rats was given subcutaneous injections twice daily for three days. The rats were killed on the fourth day. The colour was administered in aqueous solution at a level of 250 mg/kg body weight each injection. No oestrogenic activity (normal uterine weight) was detected (Graham & Allmark, 1959).

In experiments with guinea-pigs, it was found that this colour had no sensitization activity (Bär & Griepentrog, 1960).

Short-term studies

Rat

In a 90-day study on five groups of 15 male and 15 female rats, erythrosine was given in the diets at 0.25%, 0.5%, 1% and 2%. No adverse effects were noted as regards body weight, food intake, haematology, blood and urine analyses which were related to administration of test substance. Organ weights were normal except that absolute and relative caecal enlargement was seen at all levels tested. It was dose-related but histology was normal. Absolute and

relative thyroid weight was increased at the 2% level. Histopathology showed no abnormalities except pigment deposition in renal tubules in females only at the 2% level but in males at all levels again in a dose-related manner. The pigment was identified as protein-bound erythrosine. In addition, total PBI was raised at all levels in a dose-related manner, protein-bound erythrosine in serum behaved similarly and non-protein bound iodine also increased with dose levels. Thyroxine iodine however remained unchanged and I^{131} uptake was reduced (Hansen et al., 1973b).

Five groups of Carworth Farm E strain SPF rats (15 males and 15 females/group) received 0, 0.25, 0.5, 1.0 or 2.0% erythrosine in the diet for 90 days. There were no effects attributable to treatment on the rate of body weight gain, food intake, results of hematological examination, serum analyses or renal function tests. Thyroid weight relative to body weight was slightly increased in rats receiving 1.0% and 2.0% erythrosine. Thyroid activity was not impaired by any dietary level of erythrosine. This was indicated by the normal histopathology of the organ, the lack of effect on serum thyroxine levels, and the normal rates of oxygen consumption in the treated animals (Butterworth et al., 1976a).

Groups of Sprague-Dawley female rats (12-20 animals per group) were exposed to erythrosine in the diet at dose levels of 0 or 2.0% for either 6 or 12 months. During the last 12 weeks of the experimental period, a slight decrease of body weight gain was observed in rats exposed for 12 months. Other parameters such as food consumption, hematology, clinical chemistry, urinalysis and organ weights were comparable among treated and control rats in both the six and twelve month groups. Sporadic pathological changes were observed in treated and control rats (Sekigawa et al., 1978).

Gerbil

Three groups of gerbils (15 males and 15 females/group) received erythrosine in the diet at dose levels of 200, 750 or 900 mg/kg for 19 months (those animals in the 900 mg dosage group received 1200 mg/kg for the first 3 months). The control group consisted of 30 males and 30 females. Body weight decreases were seen in male gerbils at all feeding levels. However, this weight loss was observed in females only at the 900 mg/kg level. Elevated PBIs, due to interference by erythrosine with PBI determination, were seen. No other haematological differences were seen. No adverse gross pathology was noted. Histopathology was not performed (Anonymous, 1969).

Dog

Two-year feeding studies were conducted with groups of three male and three female beagles at levels of 0, 0.5, 1.0 and 2.0% in the diet. All dogs survived the study. No gross or microscopic pathology

related to colour administration was seen (Hansen et al., 1973b).

Pig

Four groups of the Large White strain pigs (3 males and 3 females/group) weighing approximately 20 kg) were fed erythrosine in their diet at dose levels of 0, 167, 500 or 1500 mg/kg/day for 14 weeks. The treated pigs exhibited decreased levels of serum thyroxine when compared with controls. There were dose-related increases in the serum levels of protein-bound iodine, iodine not bound to protein and

protein-bound erythrosine in animals of all treated groups. A dose related increase in thyroid weight was noted, although the differences were statistically significant only in female pigs at the higher dose levels (500 and 1500 mg/kg/day) when compared with the controls. None of the treated pigs revealed pathological changes of the thyroid (Butterworth et al., 1976b).

Long-term studies

Mouse

A total of 122 male and female mice produced by mixed breeding from five different strains were given a diet containing 1 mg per animal per day of the colour. Mice at the age of 50-100 days were used. A number of the animals were sacrificed after an observation period of 500 days and the remaining mice after 700 days. A total of 168 mice was used as the control group. Positive control groups which were given 0-aminoazotoluene and dimethylaminoazobenzene were also included. In these groups the formation of liver tumours was noted after approximately 200 days. The incidence of tumours in mice receiving the colour was not significantly greater than in the controls (Waterman & Lignac, 1958).

Chronic feeding studies were conducted with mice. Seventy mice were fed at 1 and 2%. Because of the small number of animals surviving the experiment and the small number of tumours found, no effect of tumour formation could be attributed to the colour (Anonymous, 1969).

Five groups of Charles River CD-1 mice (60 males and 60 females/group) were exposed to erythrosine in the diet at dose levels of 0 (two control groups were used), 0.3, 1.0 or 3.0% for 24 months. (Average consumption of erythrosine, for males - 0, 424, 1474 or 4759 mg/kg/day and for females - 0, 507, 1834 or 5779 mg/kg/day). With the exception of significant decreased body weights (throughout the entire study) of males and females at the 3.0% dose level, other investigated parameters (mortality, food intake, hematology, gross and histopathology) were not adversely affected by erythrosine treatment at any dose level (Richter et al., 1981).

Two groups of 7-week old ICR mice, weighing 27-38 g (50 males and

50 females/group) were fed a diet containing erythrosine at dose levels of 1.25% or 2.5% for 18 months. All animals of experimental groups were fed the basic diet free of erythrosine for the additional 6 months then sacrificed and autopsied. The control group consisted of 45 males and 45 females. Treated mice received erythrosine in a cube diet for the first 20 weeks, and thereafter, the erythrosine was mixed with the basic powder diet. The mortality was greater among animals exposed to erythrosine than among the controls (approximately 61% animals died in the 2.5% group, 59% in the 1.25% group and 36% in the control group). Body weight gains were not adversely affected by

erythrosine ingestion. Animals of all experimental groups exhibited high incidence of lymphocytic leukemia and sporadic cases of pulmonary adenomas were also observed. The frequency of both lesions was in the range spontaneously occurring in this strain of mice. The results indicated that erythrosine was not carcinogenic to ICR mice under the experimental conditions utilized (Yoshii & Isaka, 1984).

Rat

Groups of 24 weanling rats, evenly divided by sex, were fed this colour at 0, 0.5, 1.0, 2.0 and 5.0% for two years. Slight growth depression was observed in the animals at the 5% level, and those above 0.5% had distended caeca but microscopically the distended caeca showed normal histology. The statistical evaluation of the rat study revealed no significant changes in organ weights at the highest level. There was some diarrhoea at the 5% level. There was no difference in survival (Anonymous, 1969).

The colour was fed at a level of 4% of the diet to five male and five female rats for periods up to 18 months. Gross staining was observed in the glandular stomach and small intestine and granular deposits in the stomach, small intestine and colon. Hepatic cirrhosis was noted in one out of four rats living up to 12 months. Fifty control animals observed for 20 months or more failed to develop tumours, or hepatic cirrhosis (Willheim & Ivy, 1953).

Groups of 12 male and 12 female weanling Osborne-Mendel rats were fed 0, 0.5, 1.0, 2.0 and 5.0% erythrosine in their diet for two years. Growth depression was observed in rats given 5%. The relative spleen weight was depressed in all male test groups and in females at the 5% level. Slight caecal enlargement was noted at 1% and increased with dose but the histology of the enlarged caeca was normal. No other gross or histopathological findings related to colour administration were noted (Hansen et al., 1973b).

Groups of 25 male and 25 female 100-day-old rats and a group of 50 male and 50 female controls were fed 0, 0.5, 1.0, 2.0 and 4.0% erythrosine in their diet for 86 weeks. Other groups of 25 male and 25 female rats aged 100 days were intubated twice a week for 85 weeks with erythrosine at 0, 100, 235, 750 and 1500 mg/kg body weight. After

this treatment animals were kept on normal diets for two years. Body weight decreases were seen at 2 and 4%. Elevated PBI, due to interference by erythrosine with PBI determination rather than thyroid dysfunction, were seen. Thyroxine-iodine levels were not affected. There were no other haematological differences and no anaemia was seen. No adverse gross pathology was noted; histopathology had not shown any colour related abnormalities (Hansen et al., 1973b).

Groups of 70 males and 70 females Charles River CD weanling rats were fed erythrosine in the diet at levels of 0.1, 0.5 or 1.0% for 30 months after in utero exposure. Two concurrent control groups (70 animals/sex/group) received no colour in the diet. The average consumption of erythrosine was, for males - 0, 49, 251 or 507 mg/kg/day and for females 0, 61, 307 or 642 mg/kg/day. There were no consistent significant compound related effects during the in utero phase. In the main study, there were no consistent significant compound-related effects on the following; physical observation, behaviour, mortality, food consumption, hematology, clinical chemistry, urinalysis and ophthalmological findings. Mean body weights of control and treated rats did not differ significantly during the exposure period. The gross pathological changes noted could not be attributed to treatment with erythrosine. The incidence of non neoplastic lesions was comparable between treated and control groups. There was a statistically significant increase in the incidence of benign thyroid tumours (follicular adenoma): 6/68 in the 1.0% female test group vs. 0/140 in the control group. The incidence of malignant tumors in rats of treated groups was comparable with that of the controls (Brewer et al., 1981).

Two groups of Charles River CD weanling rats (70 males and 70 females/group) were given erythrosine in the diet at dose levels of 0 or 4.0% for a period of approximately 29 months after in utero exposure. The average consumption of the erythrosine was: male 0 or 2465 mg/kg/day, female 0 or 3029 mg/kg/day. There were no consistent significant compound-related effects on the following; physical observations, behaviour, mortality, food consumption, hematology, clinical chemistry, urinalysis and ophthalmological findings. Mean body weights of treated rats (both sexes) were slightly lower throughout the study than the control rats. These differences were statistically significant except weeks 3-5 and 122 (male) and weeks 0-4, 6 and 114 (female). The mean absolute and relative thyroid weights for treated males (4.0%) were more than doubled when compared with the controls. The histopathological examination revealed that the incidence of thyroid hyperplasia (follicular and C-cell) was significantly increased in treated males. There was a statistically significant increase in the incidence of follicular adenoma of the thyroid in treated male rats (16/68 in treated group vs. 0/69 in the control group) when compared with the controls. The incidence of malignant tumors, including thyroid C-cell and follicular carcinoma, was comparable among treated and control rats (Brewer et al., 1982).

Groups of 6-week old pathogen-free Fischer (F344) rats (50 males and 50 females) were fed diets containing erythrosine at levels of 1.25 or 2.5% for 18 months. The control group consisted of 30 males and 30 females and received a diet free of erythrosine. For the first 20 weeks of treatment, erythrosine was given in pelleted diet and for the remaining treatment period in powder diets. Rats exposed to erythrosine were sacrificed at 18 months and the control rats at 24

months after the start of the study. No parameters other than histopathology were reported. Histopathology revealed sporadic cases of spontaneous neoplasms (tumors of genital system, endocrine system, hematopoietic system and digestive system) but their frequencies were similar among animals of erythrosine treated groups and comparable to the controls. No pathological changes were observed in the thyroid glands (Fukunishi et al., 1984).

A study was undertaken to investigate whether the thyroid tumors found after chronic feeding of erythrosine to male rats at a dose level of 4.0% in the diet resulted from excess iodine (either as a contaminant of the colour or as iodine metabolized from the colour) or from another non-iodine-related property of the erythrosine. The study was composed of six dose groups each containing 70 (35 males and 35 females) Charles River CD rats.

Group 1 - received unadulterated diet.

Group 2 - received 80 µg of NaI (sodium iodide)/g of diet.

Group 3 - received purified erythrosine at 4.0% level in the diet.

Group 4 - received purified erythrosine at 4.0% level in the diet plus 80 µg of NaI/g of diet.

Group 5 - received purified erythrosine at 4.0% level in the diet plus 160 µg of NaI/g of diet.

Group 6 - received commercial erythrosine at 4.0% level in the diet.

Exposure continued for 27 weeks. The study demonstrated that feeding of commercial erythrosine at a level of 4% in the diet produces an endocrine state of hyperthyroidism. Thyroid stimulating hormone (TSH) and thyroxine (T₄) levels were elevated and tri-iodothyronine (T₃) concentrations were depressed. Changes in clinical chemistry parameters, body weight, and food consumption were also indicative of hyperthyroidism. Additional purification of the commercial preparation of erythrosine to remove free iodide did not modify the responses described. These responses were not found after feeding a diet spiked with NaI only (80 µg/g of diet). This study demonstrated that thyroid changes observed in this and former studies are associated with increased TSH concentration. However, the results

of this study do not indicate the mechanism for these effects of erythrosine (Couch et al., 1983).

Twenty rats were subject to weekly subcutaneous injections of 1 ml of a 5% aqueous solution for 596 days (85 weeks). The total quantity of colour administered was 2.65 g/animal. Seven rats survived 300 days or more. No tumours were observed (Umeda, 1956).

Eighteen rats were injected subcutaneously with aqueous solutions of erythrosine at 12 mg/animal once per week for two years. No tumours either at the injection sites or in other parts of the body were observed (Hansen et al., 1973b).

Gerbil

Three groups (15-16 animals/sex/group) of Mongolian gerbils, approximately 6 months old, were fed diets containing erythrosine at levels of 1.0, 2.0 or 4.0% for 105 weeks. Control groups (31 animals of each sex) were fed diets free of erythrosine. Animals of all treated groups exhibited a statistically significant dose-related decrease in body weight gain when compared with the controls. In general, there were slight, and in some isolated cases, significant depressions of hematocrit and hemoglobin values, and leucocyte and reticulo-cyte counts in animals of treated groups. The relative weights of heart, liver and spleen were significantly decreased in animals of both sexes at the two high dose levels (2.0 and 4.0%). Dose-related changes such as enlargement of follicles and, in some cases, focal hyperplasia were observed in the thyroid of treated animals. Histopathology did not reveal any treatment related effects (Collins & Long, 1976).

Groups of 20-24 males and 20-24 females Mongolian gerbils approximately 6 months old received erythrosine (dissolved in water) by stomach intubation at dose levels of 200, 750 or 900 mg/kg twice weekly for 97 weeks. A control group (32 animals/sex) was intubated with distilled water only. The dosages were administered in a volume of 10 ml/kg body weight. No treatment-related adverse effects were observed for investigated parameters such as clinical toxicity, mortality, body weight gain, hematology, organ weights, gross and histopathology (Collins & Long, 1976).

OBSERVATIONS IN MAN

Five human volunteers (four males and one female, ages 21-35 years) received erythrosine in a diet at dose levels of 5, 10 or 25 mg/day in weekly increments for a period of 3 weeks. The study demonstrated slowly and slightly increasing levels of total serum iodine and protein bound iodine (PBI) associated with the weekly increasing erythrosine doses. In the other tests for serum T₄, T₃, TSH, erythrosine concentration, urinary iodine and erythrosine

excretion, and T₃ - resin uptake remained unchanged throughout the 3 weeks. Increases in serum PBI and total serum iodine during exposure period indicates that a portion of the iodine ingested as erythrosine appears to be absorbed from the gastrointestinal tract. No changes in concentration of TSH, T₄ and T₃ in serum indicate that both the thyroid function and thyroregulatory mechanisms were unaffected by the ingestion of erythrosine during a three week period at a dose as high as 25 mg/day (Ingbar et al., 1983).

Comments

The Committee considered information obtained since 1974 which included: measurements of thyroid function in human subjects ingesting erythrosine; data on mutagenicity; data on reproduction and behavioural toxicity; the results of long-term feeding studies in mice and rats; and the results of 90-day and 6-month studies in rats, in which effects on the thyroid function were demonstrated. In the latter studies it was shown that the effects on the thyroid function were not due to sodium iodide, which is normally present in the commercial product. The results of tests for mutagenicity were negative. The Committee considered that the development of thyroid tumours in the long-term studies on rats might be mediated by a hormone effect, although the mechanism for this was not demonstrated. One way of determining the no-effect level would have been by assessing the extent of diffuse hyperplasia in the thyroid glands of erythrosine-treated rats, as this was likely to have accompanied the observed increase in thyroid weight and would indicate an effect on thyroid function. However, the data for this purpose were not available to the Committee. Because insufficient data were available to determine a no-effect level, the existing ADI was reduced to 0-1.25 mg/kg of body weight and made temporary.

EVALUATION

Level causing no toxicological effect

Rat: 0.5% (=5000 ppm) in the diet equivalent to 250 mg/kg body weight.

Estimate of temporary acceptable daily intake for man

0-1.25 mg/kg body weight.

FURTHER WORK OR INFORMATION

Required by 1986

1. The histopathology (including the assessment of diffuse hyperplasia) of all thyroid glands from the recent long-term studies in rats.

2. The mechanism of the effects of erythrosine on the thyroid gland, in terms of the biochemical and histopathological parameters; and the existence of a threshold level of these effects and their reversibility.

Desirable

Information on the pharmacokinetics of erythrosine and its effect on the thyroid functions of human subjects.

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See Also:

[Toxicological Abbreviations](#)

[Erythrosine \(FAO Nutrition Meetings Report Series 46a\)](#)

[Erythrosine \(WHO Food Additives Series 6\)](#)

[Erythrosine \(WHO Food Additives Series 21\)](#)

[Erythrosine \(WHO Food Additives Series 24\)](#)

[Erythrosine \(WHO Food Additives Series 28\)](#)

[Erythrosine \(WHO Food Additives Series 44\)](#)

[ERYTHROSINE \(JECFA Evaluation\)](#)