

Current evidence on thiamin (vitamin B1) status in healthy European adults: a systematic review of deficiency prevalence

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Abstract

Objective This systematic review evaluates the thiamin status of the European adult population, identifying critical gaps and priorities for future research.

Methods The review was conducted according to a pre-registered protocol (PROSPERO: CRD420251101630) and PRISMA guidelines. A systematic search of PubMed and Scopus was carried out to identify observational studies reporting relevant thiamin status biomarkers (erythrocyte transketolase activity coefficient (ETKAC), whole blood thiamin diphosphate (WB ThDP), total thiamin in whole blood, or erythrocyte thiamin diphosphate - RBC ThDP) in apparently healthy, community-dwelling adults (≥ 18 years) from European countries.

Results and discussion A total of 21 cross-sectional studies from 11 countries, published between 1976 and 2026, met the inclusion criteria, providing data on 7,344 individuals. The prevalence of thiamin deficiency (ETKAC > 1.25 , WB ThDP < 70 nmol/L, total thiamin in whole blood < 75 nmol/L, RBC ThDP < 280 ng/g Hb) ranged from 0% to 41.2%. When pregnant and post-partum women were excluded, the prevalence of thiamin deficiency ranged from 0% to 10%. The prevalence of thiamin deficiency in Europe remains poorly established because nationally representative studies in the general population are scarce. The largest and most recent data come from the United Kingdom, where wheat flour is fortified with thiamin.

Conclusions Further population-based studies using harmonised biomarkers are required before reliable conclusions can be drawn regarding the prevalence and public health significance of thiamin deficiency in Europe.

Keywords Thiamin · Micronutrient deficiency · Wernicke encephalopathy · Beriberi · Europe · Vitamin B1 · ThDP · ETKAC

Abbreviations

α -KGDH	α -ketoglutarate dehydrogenase complex
AUD	Alcohol use disorder
BCKDH	Branched-chain α -keto acid dehydrogenase complex
BMI	Body mass index
EFSA	European Food Safety Authority
ESPEN	European Society for Clinical Nutrition and Metabolism

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ETK	Erythrocyte transketolase
ETKAC	Erythrocyte transketolase activity coefficient
EU	European Union
Hb	Hemoglobin
HPLC	High-performance liquid chromatography
IB-CoA	Isobutyryl-CoA
Ile	Isoleucine
IQR	Interquartile range
i/v	Intravenous
IV-CoA	Isovaleryl-CoA
KIC	α -ketoisocaproate
KIV	α -ketoisovalerate
KMV	α -keto β -methylvalerate
LCHF	Low-carbohydrate high-fat
Leu	Leucine
MB-CoA	Methylbutyryl-CoA
NADPH	Nicotinamide adenine dinucleotide phosphate
PDH	Pyruvate dehydrogenase complex
PRISMA	Preferred Reporting Items for Systematic Reviews and Meta-Analyses
PROSPERO	International Prospective Register of Systematic Reviews
RBC	Erythrocyte (Red blood cell)
RFC1	Reduced folate carrier 1
SD	Standard deviation
SIDS	Sudden infant death syndrome
SLC19A1	Solute carrier family 19 member 1
SLC19A2	Solute carrier family 19 member 2
SLC19A3	Solute carrier family 19 member 3
SLC25A19	Solute carrier family 25 member 19
TCA	Tricarboxylic acid cycle
ThDP	Thiamin diphosphate
ThMP	Thiamin monophosphate
THTR1	Thiamin transporter 1
THTR2	Thiamin transporter 2
TKT	Transketolase
TPP	Thiamin pyrophosphate
Val	Valine
WB	Whole blood
WHO	World Health Organization

Background

Thiamin (vitamin B1), a water-soluble vitamin essential for carbohydrate and amino acid metabolism, energy production and neuronal function [1], is ingested mainly in phosphorylated forms. Once dephosphorylated by intestinal phosphatases [2, 3], free thiamin is absorbed in the small intestine primarily via the thiamin transporters THTR1 (SLC19A2) and THTR2 (SLC19A3) [4, 5], which also mediate thiamin transport in the kidney and other tissues [6, 7]. In addition to these classical thiamin transporters, several other carriers, such as reduced folate

Thiamin deficiency

Glucose

Glucose-6-P

Fructose-6-P

Glyceraldehyde-3-P

Pyruvate

Acetyl-CoA

Citrate

Succinyl-CoA

α-Ketoglutarate

Succinate

Glutamate

GABA

IB-CoA

MB-CoA

IV-CoA

Lactate

Acetylcholine

Nucleotide synthesis

Ribulose-5-P

Ribose-5-P

Choline

Val

Leu

Ile

KIV

KIC

KMV

ThDP

TKT

PDH

α-KGDH

BCKDH

NADP⁺

NADPH

Pentose phosphate pathway

TCA cycle

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and legumes [25]. It is often assumed that thiamin deficiency is rare in the general population in Europe and is limited to high-risk groups such as malnourished individuals or people with alcohol use disorder (AUD) [26].

Thiamin must be supplied regularly, as body stores are minimal. A diet almost devoid of thiamin produces first sign of deficiency in just 10 days [27]. Clinical thiamin deficiency may present with a spectrum of neurological, psychiatric, and cardiovascular signs and symptoms [28]. Subclinical deficiencies can also impair cognitive and physical functions [23]. Early signs of thiamin deficiency are often unrecognized [29], none of the signs and symptoms are pathognomonic, and they can be mistaken for psychosomatic disorders [30].

Dietary thiamin deficiency may ultimately lead to beriberi (dry, wet, or infantile) and Wernicke-Korsakoff syndrome. In adults, dry beriberi presents with peripheral sensory and motor neuropathy [31], while wet beriberi presents with increased cardiac output, cardiomegaly, heart failure, and peripheral oedema [32, 33]. Cardiac dysfunction, which can be severe, is a major feature of infantile beriberi which appears in infants during the first months of life [34], while the nervous system dysfunction may predominate in older children [35]. Thiamin deficiency, especially in alcohol use disorder, may also manifest as Wernicke's encephalopathy and Korsakoff syndrome [36]. Wernicke's encephalopathy is characterized by mental changes, abnormal eye movements, and/or ataxia [37–39]. If untreated, Wernicke's encephalopathy may progress to Korsakoff's syndrome, an irreversible condition which manifests with amnesia and confabulation [39]. Clinical symptoms are not reliable for detecting Wernicke's encephalopathy. In one autopsy study of 6,964 cases, 52 cases of Wernicke's encephalopathy were identified, of which 12 (23%) occurred in people without alcohol dependence. Only four cases (7.7%) had been diagnosed clinically during life, and none of the people without alcohol dependence had received thiamin therapy [40].

Long-term alcohol consumption can lead to thiamin deficiency by reducing dietary intake of thiamin, inhibiting absorption in the gastrointestinal tract and disrupting its cellular utilization [41]. Thiamin deficiency also occurs in malnourished patients, patients after bariatric surgery [28], people living with obesity [42], patients on total parenteral nutrition [28, 43] and patients with diabetes [44]. Thiamin deficiency may also occur in the context of refeeding syndrome, since the transition from starvation to feeding greatly increases the demand for thiamin, which serves as a cofactor for glucose-dependent metabolic pathways (Fig. 1) [45]. Lastly, ultra-processed foods, snacks and soft drinks contribute significantly to energy intake while being low in thiamin [46]. The ultra-processed food share in energy intake in Europe ranges from 14 to 44%, the highest being in United Kingdom [47]. All risk factors contributing to thiamin deficiency are presented in Fig. 2.

Thiamin status is usually assessed through two approaches:

1. Direct measurement of thiamin or its phosphorylated metabolites (thiamin diphosphate (ThDP) in whole blood (WB) or erythrocytes (RBC) or total thiamin in whole blood (WB)). Thiamin deficiency is generally associated with low total thiamin or ThDP concentrations in whole blood and erythrocytes [48].
2. Functional assessment via the erythrocyte transketolase activity coefficient (ETKAC), an indirect marker of thiamin-dependent enzymatic saturation ($\text{ETKAC} = \text{ETK activity with added ThDP (U/g Hb)} / \text{baseline ETK activity (U/g Hb)}$). ETKAC is a sensitive marker for thiamin function and status [48].

This systematic review evaluates thiamin status of the European adult population, identifying critical gaps and priorities for future research. On a global level the prevalence of thiamin deficiency was reviewed by Johnson et al. [24] and Whitfield et al. [23]. Rakotoambinina et al. [49] reviewed published reports of paediatric thiamin deficiency in high-income countries.

Materials and methods

The study was conducted according to a previously registered protocol in the international prospective register of systematic reviews PROSPERO ID - CRD420251101630 [58]. The systematic review was conducted according to PRISMA guidelines [59].

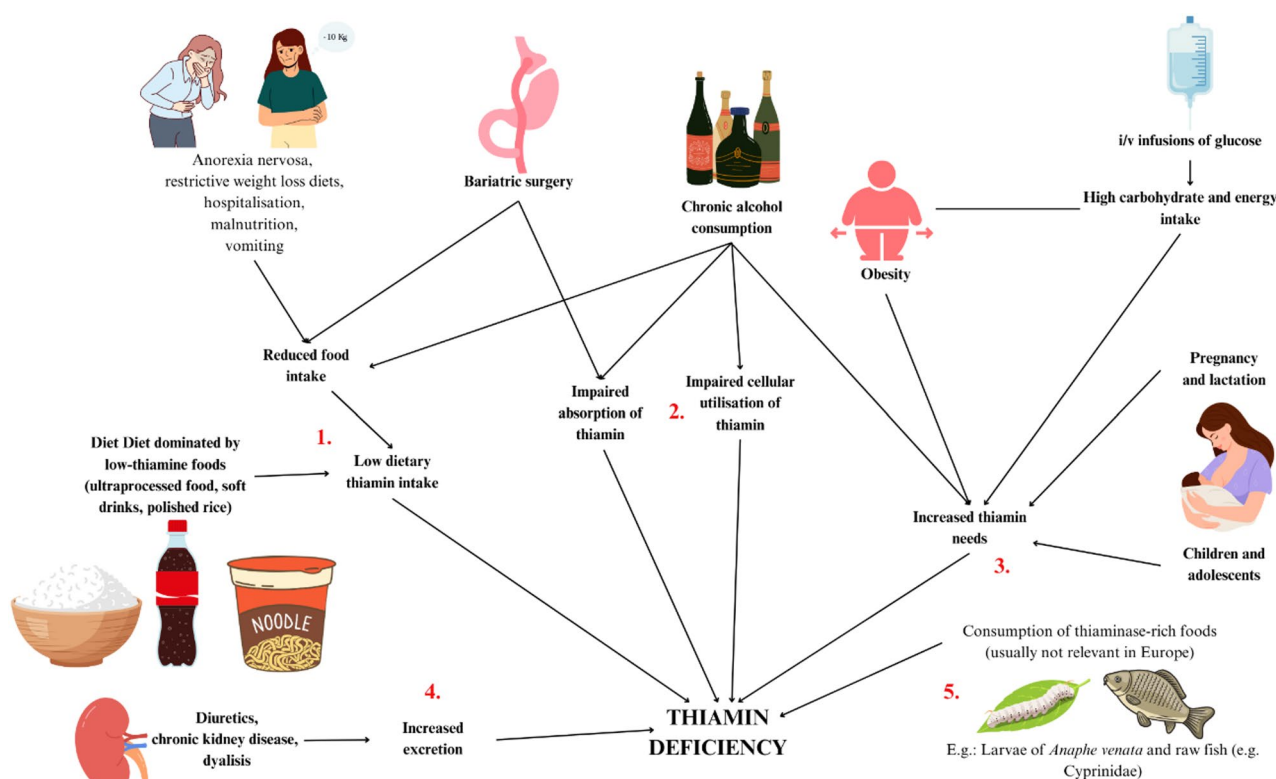


Fig. 2 Risk factors contributing to thiamin deficiency: (1) Low thiamin intake. The reason for low thiamin intake may be generally low food intake for example in people with anorexia nervosa [50], bariatric surgery [51] or vomiting [52]. More often the cause is diet dominated by foods low in thiamin (polished rice, soft drinks, ultra-processed foods) [23]. (2) Impaired absorption and cellular utilization of thiamin. Chronic alcohol consumption affects food intake, thiamin absorption, cellular utilization and increases thiamin needs. Additionally, alcoholic beverages provide substantial energy while contributing negligible amounts of thiamin [41]. Bariatric surgery decreases thiamin intake and absorption [51]. (3) High carbohydrate and energy intake. Thiamin requirements depend on carbohydrate and energy intake [48, 53]. Children, adolescents, pregnant women, lactating women, and people living with obesity have increased thiamin needs. Intravenous infusions of glucose given to thiamin deficient patients increase thiamin requirements and exacerbate deficiency [54]. (4) Increased thiamin excretion. Use of diuretics, chronic kidney disease and dialysis can increase thiamin excretion [55]. (5) High intake of thiaminase from insufficiently cooked larvae of African silkworm *Anaphe venata* causes seasonal thiamin deficiency in Nigeria [56, 57]. In Europe, thiaminase contribution to thiamin deficiency is negligible. Source of images: Canva.

Study population, exposure and outcome

The study population included apparently healthy, community-dwelling adults aged ≥ 18 years from European countries meeting the inclusion criteria.

The primary exposure was thiamin status, assessed using the biomarkers defined in Table 1. The outcomes included: (1) absolute measurements of biomarkers and (2) the prevalence of thiamin deficiency defined by the cut-off values listed in Table 1.

The inclusion and exclusion criteria

The inclusion criteria were (1) apparently healthy, community-dwelling adults aged ≥ 18 years, including pregnant and lactating women, (2) observational studies (cohort, case-control, or cross-sectional) and baseline data from interventional studies collected prior to any intervention, (3) studies reporting selected thiamin biomarkers

Table 1 Thiamin biomarkers included in the systematic review

Biomarker	Biol. sample	Use	Cutoff for deficiency
ThDP - thiamin diphosphate in whole blood (older name: thiamin pyrophosphate -TPP)	Whole blood	Biologically active vitamer and indicator of thiamin status [28]	Deficiency < 70 nmol/L [76]
ThDP - thiamin diphosphate in erythrocytes (older name: thiamin pyrophosphate -TPP)	Washed erythrocytes		Deficiency < 280 ng/g Hb RBC ThDP [64]
Total thiamin in whole blood (free thiamin plus its esters)	Whole blood	Includes active and inactive thiamin forms. Less reliable biomarker of thiamin status than ThDP - thiamin diphosphate in whole blood or erythrocytes [23]	< 75 nmol/L [76]
ETKAC - Erythrocyte transketolase activity coefficient	Washed erythrocytes	Functional test of biological activity. Sensitive marker of thiamin function and status [48] ETKAC = ETK activity with added ThDP (U/g Hb)/baseline ETK activity (U/g Hb) [77]	Deficiency: ETKAC > 1.25 (α > 25%) Insufficiency: ETKAC 1.15–1.25 (α 15–25%). Adequate (sufficiency): ETKAC < 1.15 (α < 15%) [23, 48, 77]

Table 2 Thiamin biomarkers not included in the systematic review

Biomarker	Biol. sample	Use	Reason for exclusion
Plasma thiamin	Plasma	Reflects recent intake	Not an indicator of thiamin status [23, 48]
ThMP- thiamin monophosphate, older: TMP	Plasma	Reflects recent intake	
24-h urinary thiamin	24 h urine	Reflects recent intake [78]	Biomarker of thiamin intake, not status. Not reliable when thiamin intakes are low. Requires at least 24 h urine collection [48, 77, 78]
Basal ETK - Basal erythrocyte transketolase activity	Washed erythrocytes	Complementary biomarker [79]	Useful in specific populations. Aids in interpretation of thiamin status in context of variable apoenzyme concentrations. Cutoff for deficiency is not clearly defined; < 0.50 U/gHb or < 0.59 U/gHb are used as cutoffs for deficiency [79]. This allows differentiation between whether the low activity is due to thiamin deficiency or reduced synthesis of the enzyme itself

listed in Table 1, (4) studies including healthy European adults, (5) no restrictions on language, (6) no restrictions on publication date, and (7) a minimum sample size of 10 participants.

The exclusion criteria were (1) case reports, narrative reviews, and conference abstracts, (2) studies reporting biomarkers that do not reflect thiamin status, as specified in Table 2, (3) individuals taking thiamin food supplements, (4) participants with alcohol use disorder (AUD), obesity (BMI > 30 kg/m²), type 1 or type 2 diabetes mellitus, chronic kidney disease, liver dysfunction, malabsorption syndromes (e.g., celiac disease, Crohn's disease), or other conditions known to affect thiamin status, (5) studies involving institutionalized populations or specific clinical subgroups such as individuals post-bariatric surgery or with Wernicke–Korsakoff syndrome, and (6) studies focusing on special populations such as seamen, or aboriginal groups.

Due to the limited number of published studies, studies based on non-representative population samples were also included; however, these were clearly marked in the result section. Study populations included in this review are heterogeneous and include pregnant women [60], post-partum women [61], convenience samples comprising of staff, that are usually healthier, better nourished, and socioeconomically advantaged [62–64], university students [65], and employees undergoing routine medical examinations [66]. The remaining studies utilized cohorts of healthy volunteers serving as control groups or for reference value establishment [53, 67–75].

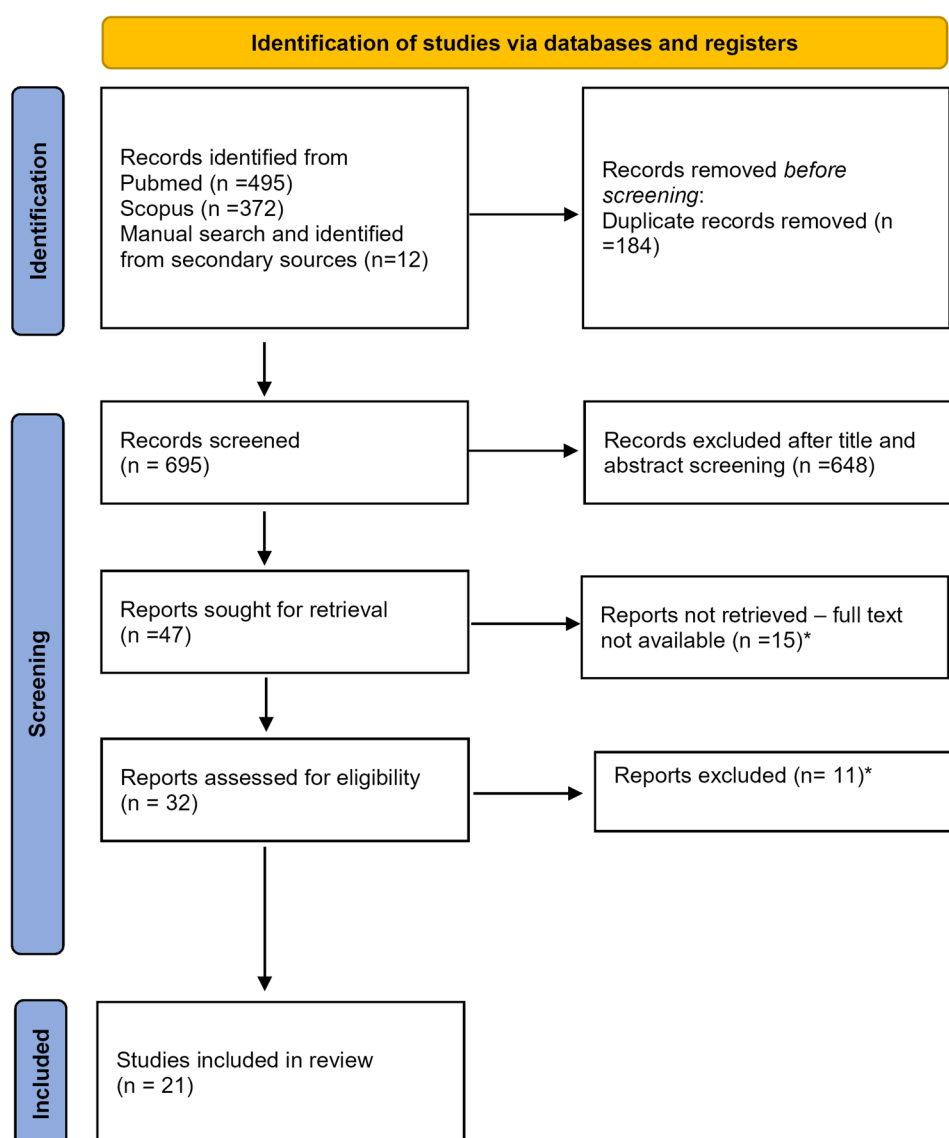
Search and screening strategy

The review team comprises two experts in public health and nutrition. The literature search was conducted in PubMed and Scopus and supplemented by snowballing (screening reference lists and citations of included studies).

Search process:

- We defined the search criteria. The PubMed and SCOPUS search combined terms for European populations with thiamin-related keywords (e.g., thiamin, vitamin B1, ThDP, ETK, ETKAC, ThDP etc.). The search was limited to human studies. Full search syntax is available in supplemental data.
- The titles and abstracts were searched manually and selected as described in Study Flow Diagram (Fig. 3).
- The data from selected studies was manually extracted into an MS Excel spreadsheet.
- The data was then rechecked by a second researcher.

Fig. 3 Study flow diagram. Made according to PRISMA guidelines [59]. * Complete list is available in supplemental data



Study risk of bias assessment Newcastle-Ottawa Scale adapted for cross-sectional studies was used [80]. Grading is available in supplemental data.

Data synthesis and analysis

Only one study used a nationally representative sample from the United Kingdom [81], and one Spanish study used a regionally representative sample [82] and many countries were not represented. The meta-analysis was not feasible.

Results

A total of 21 cross-sectional studies from 11 countries published between 1976 and 2026 met the inclusion criteria and provided data on the thiamin status of 7,344 healthy European adults. The results are stratified by the biomarkers erythrocyte transketolase activity coefficient (ETKAC) in Table 3, whole blood thiamin diphosphate

Table 3 Studies reporting erythrocyte transketolase activity coefficient (ETKAC)

Author, year	Population	Location	No. of subjects	Age (mean $\pm$ SD or range yrs)	ETKAC (mean $\pm$ SD)	Thiamin deficiency (%) ETKAC > 1.25	Thiamin insufficiency (%) ETKAC > 1.15
Fabian et al. 2008 [67]	Healthy, with no fermented food consumption in last 7 days	Vienna, Austria	33 (♀33)	24.4 $\pm$ 2.8	1.085 $\pm$ 0.024	N/A	N/A
Hötzel and Bitsch 1976 [65]	Healthy students	Bonn, West Germany	130 (♀84)	adult	1.126 $\pm$ 0.121	10.00%	20%
Jones et al. 2023 [79]	Selected samples from nationally representative study NDNS RP	United Kingdom	134 (♀134)	44.0 $\pm$ 11.8	1.09 $\pm$ 0.05	0%	13%
Koulman et al. 2026 [81]	Nationally representative study NDNS RP	United Kingdom	5657 (♀3096)	39 (16–57) median (IQR)	1.11 (1.07–1.14) median (IQR)	0.7%	18%*
Malara et al. 2013 [70]	Healthy volunteers with low or high physical activity	Warsaw, Poland	80 (♀40)	19.7 $\pm$ 0.71	1.053 $\pm$ 0.054	0%	N/A
Mataix et al. 2003 [82]	Representative sample of healthy population	Andalusia, Spain	372 (♀191)	25–60	1.01 $\pm$ 0.17	6.4%	13.6% (ETKAC > 1.16)
Ortega et al. 2004 [60]	Healthy women in third trimester pregnancy	Cuenca, Spain	51 (♀51)	29.9 $\pm$ 3.8	1.06 $\pm$ 0.22	13.7%	31.3%
Sánchez et al. 1999 [61]	Healthy women sampled at delivery	Coast of Catalonia province, Spain	131 (♀131)	26 $\pm$ 5	1.25 $\pm$ 0.17	41.2%	62.6%
Studies using healthy volunteers for controls or reference values establishment are marked with (#) and presented in grey.							
Boni et al. 1980 [63]	Healthy medical and laboratory personnel (#)	Leuven, Belgium	12 (♀NR)	adult	1.135 (range 1.08–1.185)	0%	N/A
Elmadfa et al. 2001 [53]	Healthy volunteers (#)	Vienna, Austria	12 (♀6)	25–30	1.12 $\pm$ 0.1	N/A	N/A
Herve et al. 1995 [68]	Healthy volunteers (#)	Clichy, France	52 (♀28)	21–65	1.12 $\pm$ 0.1	N/A	N/A
Jermendy 2006 [69]	Healthy control (#)	Budapest, Hungary.	60 (♀28)	54.1 $\pm$ 2.5	1.08 $\pm$ 0.02	N/A	N/A
Michalak et al. 2013 [75]	Healthy control (#)	Poznań, Poland	20 (♀16)	49.1 (23–70)	1.0 (range 0.0–0.0)	0%	0%

SD, standard deviation; ETKAC, erythrocyte transketolase activity coefficient, * calculated by the authors from original data [83]

Table 4 Studies reporting whole blood thiamin diphosphate - WB ThDP (nmol/L)

Author, year	Population	Location	No. of subjects	Age (mean $\pm$ SD or range yrs)	WB ThDP (nmol/L) (mean $\pm$ SD)	Thiamin deficiency (%), WB ThDP < 70 nmol/L
Studies using healthy volunteers for controls or reference values establishment are marked with (#) and presented in grey.						
Herve et al. 1995 [68]	Healthy volunteers (#)	Clichy, France	52 (♀28)	21–65	176 $\pm$ 28	N/A
Körner et al. 2009 [73]	Healthy volunteers (#)	North Rhine-Westphalia, Germany	30 (♀16)	40.5 (18–65)	194.7 $\pm$ 27.5	0%
Lynch et al. 1997 [62]	Healthy laboratory staff (#)	Belfast, United Kingdom	45 (♀16)	38 (20–60) median (range)	174.2 $\pm$ 34.05	N/A
Mancinelli et al. 2003 [66]	Random selection of healthy employees undergoing routine medical examinations (#)	Rome, Italy	103 (♀58)	42.9 $\pm$ 10.2	222.2 $\pm$ 56.3	N/A
Tallaksen et al. 1991 [74]	Healthy volunteers (#)	Oslo, Norway	30 (♀15)	43 (32–54) median (range)	143 $\pm$ 35	N/A
Verstraete and Stove 2021 [72]	Healthy volunteers (#)	Ghent, Belgium	50 (♀NR)	adult	145 $\pm$ 22.1	0%

SD, standard deviation; WB ThDP, whole blood thiamin diphosphate

Table 5 Studies reporting total whole blood thiamin (nmol/L)

Author, year	Population	Location	No. of subjects	Age (mean $\pm$ SD or range yrs)	Total WB thiamin (nmol/L) (mean $\pm$ SD)	Thiamin deficiency (%), Total WB thiamin < 75 nmol/L
Studies using healthy volunteers for controls or reference values establishment are marked with (#) and presented in grey.						
Herve et al. 1995 [68]	Healthy volunteers (#)	Clichy, France	52 (♀28)	21–65	186 $\pm$ 30	N/A
Schrijver et al. 1982 [71]	Healthy volunteers (#)	Utrecht, Netherlands	98 (♀NR)	adult	115 (range 70–185)	1–2%

SD, standard deviation; WB, whole blood

Table 6 Studies reporting erythrocyte thiamin diphosphate - RBC ThDP (ng/g Hb)

Author, year	Population	Location	No. of subjects	Age (mean $\pm$ SD or range yrs)	RBC ThDP (ng/g Hb) (mean $\pm$ SD)	Thiamin deficiency (%), RBC ThDP < 280 ng/g Hb
Studies using healthy volunteers for controls or reference values establishment are marked with (#) and presented in grey.						
Churuangsuk et al. 2024 [84]	Healthy volunteers, following general diet, without thiamin food supplements	Glasgow, United Kingdom	42 (♀nd)	27 (24–34) median (IQR)	621 $\pm$ 200	2%
	Healthy volunteers, following LCHF (low carbohydrate high fat diet), without thiamin food supplements		36 (♀nd)	36 (25–49) median (IQR)	378 $\pm$ 68	8%
Tallaksen et al. 1991 [74]	Healthy volunteers (#)	Oslo, Norway	30 (♀15)	43 (32–54) median (range)	325 $\pm$ 76.95	N/A
Talwar et al. 2000 [64]	Laboratory staff and people attending a cardiovascular risk clinic (#)	Glasgow, United Kingdom	147 (♀68)	54 $\pm$ 8.2	433 $\pm$ 76.5	2.5%

SD, standard deviation; RBC ThDP, erythrocyte thiamin diphosphate; IQR, interquartile range

(WB ThDP; nmol/L) in Table 4, total thiamin in whole blood (nmol/L) in Table 5 and erythrocyte thiamin diphosphate (RBC ThDP; ng/g Hb) in Table 6.

While most studies included community-dwelling adults, several investigated specific subgroups, including pregnant and postpartum women, university students, individuals following LCHF diet (low carbohydrate high

fat diet), and convenience samples of healthcare workers. Study populations were recruited from 11 countries: Spain, United Kingdom, Austria, Poland, Germany, Hungary, France, Belgium, Italy, Norway and the Netherlands. There were two studies with a representative sample of the healthy adult population [81, 82].

Summary of biomarker findings

The prevalence of thiamin deficiency (ETKAC > 1.25, WB ThDP < 70 nmol/L, total thiamin < 75 nmol/L, RBC ThDP < 280 ng/g Hb), ranged from 0% to 41.2%. If pregnant and postpartum women were excluded, the prevalence of thiamin deficiency ranged from 0% to 10%.

Discussion

This systematic review identified limited and heterogeneous evidence on thiamin status in apparently healthy European adults. Across 21 cross-sectional studies from 11 countries, reported deficiency prevalence widely from 0% to 41.2%, and from 0% to 10% after excluding pregnant and post-partum women. Current evidence is insufficient to rule out thiamin deficiency as a public health concern across Europe.

Of the 7,344 included participants, 6,061 (83%) were from five studies conducted in the United Kingdom. As the United Kingdom has mandated thiamin fortification of non-wholemeal wheat flour since 1956, extrapolation of these findings to other European countries, which do not require general cereal fortification [23], is limited [23, 85]. Therefore, the overall prevalence estimates presented in this review are likely to underestimate the prevalence of thiamin deficiency in Europe. In a recent nationally representative study from the United Kingdom, the prevalence of thiamin deficiency was affecting 0.7% of participants overall (ETKAC > 1.25), while thiamin insufficiency affected 18.0% (ETKAC > 1.15). Deficiency prevalence differed by ethnicity, with 0.5% in White, 1.3% in Asian, and 5.4% in Black participants. Thiamin intake was significantly lower in Black than in White participants, but differences in thiamin status persisted after adjustment for thiamin intake [81]. Another study from the United Kingdom showed that restrictive diets, such as low-carbohydrate high-fat (LCHF) diets, may also increase the risk of thiamin deficiency because they restrict foods naturally rich in thiamin, particularly whole grains, and reduce intake of thiamin-fortified wheat flour. Thiamin deficiency (α -ETK > 1.25) was found in 8% of volunteers following un-supplemented LCHF and in 2% of healthy un-supplemented Glasgow (UK) adults following general diet [84].

Significantly higher prevalence of thiamin deficiency and insufficiency was reported by a Spanish study using a representative sample from the Andalusian region of Spain (Table 3) [82]. These findings suggest that the prevalence of thiamin deficiency may vary significantly across Europe and may be influenced by national food fortification policies.

Relationship between biomarkers and dietary thiamin intake

Dietary thiamin intake is important for interpreting thiamin status biomarker findings. In its assessment, EFSA included intake data from 13 dietary surveys in nine European Union countries. Mean thiamin intake in adults (≥ 18 years) varied across countries from 0.88 to 1.99 mg/day, corresponding to 0.11–0.24 mg/MJ (0.46–1.0 mg/1000 kcal). Relative to the requirement of 0.10 mg/MJ, this corresponds to 110–240% of the requirement [48]. Higher thiamin intake has been associated with a lower risk of biochemical thiamin deficiency, defined by ETKAC [81] or RBC ThDP concentrations [84]. This relationship depends strongly on the dietary assessment method. In a study of older Chinese adults, thiamin intake was not associated with RBC ThDP concentration. The authors attributed this discrepancy partly to measurement error in dietary assessment, as the Chinese food composition database was largely based on raw food weight and did not adequately account for the substantial loss of water-soluble vitamins during food preparation [86]. Low dietary thiamin intake may indicate an increased

risk of inadequate thiamin status, but it cannot replace biomarker-based assessment. EFSA panel also noted that ETKAC, erythrocyte ThDP and whole-blood ThDP are not valid markers of habitual thiamin intake within the usual intake range [48]. Urinary thiamin reflects recent intake and may be useful for validating dietary intake estimates as 1 mg of thiamin intake predicts 268.2 µg of thiamin excreted in urine [78]. Collectively, these findings indicate that dietary intake data and biochemical biomarkers provide complementary, rather than interchangeable, information on thiamin status.

Special subpopulations

The risk of thiamin deficiency is higher in populations with increased relative thiamin requirements, such as pregnant and post-partum women [48, 52]. A Spanish study in women in the third trimester of pregnancy reported thiamin deficiency in 13.7% of participants (ETKAC > 1.25) [60], while another Spanish study in women at delivery found a prevalence of 41.2% (ETKAC > 1.25). Only 14.8% of infants born to thiamin deficient mothers had adequate thiamin status (ETKAC < 1.15) [61]. Moreover, breast milk of thiamin-deficient mothers contains reduced thiamin levels, leading to deficiency in the offspring [60, 87]. Thiamin deficiency has been documented in families exhibiting a high incidence of Sudden Infant Death Syndrome (SIDS) [36, 88].

When interpreting findings from population-based studies, alcohol dependence should be considered as a potential contributor to thiamin deficiency as 30–80% alcohol dependent individuals are thiamin deficient [41]. The prevalence of alcohol dependence among adults aged 18–64 years in the EU was estimated at 3.4%, with significant variation between sexes and countries. The lowest prevalence was observed in Spain (0.7%) and the highest prevalence was observed in Latvian men (21.2%) [89].

Two studies conducted in northern regions of the Russian Federation were not eligible for inclusion in the present systematic review. They report high prevalence of thiamin deficiency [90, 91]. Bojko [90] reported thiamin deficiency (ETKAC > 1.25) prevalences of 57.7% in males and 68.8% in females in Kargopol (Arkhangelsk Region).

Limitations and strengths

However, the main strength of this review is the strict definition of appropriate thiamin status biomarkers (Table 1). Studies using biomarkers (Table 2), such as plasma thiamin [92, 93], thiamin monophosphate (ThMP) and plasma thiamin diphosphate (ThDP) [93] were excluded, but are presented in supplemental material. The cut-off values for thiamin deficiency, insufficiency, and adequacy are not universally standardized, partly because methods for assessing thiamin status are also not standardized. Johnson et al. [24] reviewed proposed cutoffs. For this review, we applied the cutoffs defined in Table 1, which are also reported in brackets whenever deficiency or insufficiency is reported. Although cut-off values varied between studies, studies were not excluded solely on this basis. Instead, differences in cut-off values were reported and considered when interpreting the results. The synthesis of results was further complicated by the use of different biomarkers and a lack of standardized diagnostic cut-offs, which impedes direct comparability across studies [24, 79, 94]. A further limitation is that most available evidence comes from a single country (the United Kingdom), which limits the geographical representativeness of the evidence base.

Practical considerations

Sufficient thiamin intake in high-income adult populations can be achieved through a varied diet that includes cereals, especially fortified breakfast cereals, seeds, legumes and meat, particularly lean pork [48]. Thiamin deficiency could be considered a sign of alcohol abuse [41], restrictive diets [84, 95] or generally very poor diet. In thiamin deficient populations, deficiency of other micronutrients should be expected [61, 95]. Considering the increased needs during pregnancy and lactation, severe consequences of thiamin deficiency [36, 88] and the

safety of thiamin, supplementation with prenatal micronutrient food supplements containing thiamin may be reasonable. Routine thiamin supplementation is currently not recommended in most European countries. WHO recommends thiamin supplementation as part of multiple micronutrient supplementation during emergencies [36]. Even at doses of several hundred milligrams, there are no reports of adverse effects of oral thiamin supplementation [96]. Sufficient intake also ensures sufficient thiamin content in breast milk [60, 87].

Future research

Representative, population-based studies using harmonized biomarkers are urgently needed to establish the prevalence and risk factors for thiamin deficiency in continental Europe. Further targeted investigations are warranted among subpopulations with potentially increased requirements or reduced intake, including pregnant and lactating women and other groups with increased physiological requirements, individuals adhering to restrictive or low-carbohydrate diets, people living with obesity, persons with alcohol use disorder (AUD) and persons of lower socioeconomic status. Harmonisation of laboratory methods and diagnostic cut-offs should be prioritised to improve comparability between studies.

Conclusions

Available evidence does not support the conclusion that thiamin deficiency has been eliminated as a public health concern across Europe. The current evidence base is limited by the predominance of data from the United Kingdom and the scarcity of representative studies from continental Europe. Further population-based studies using harmonised biomarkers are required before reliable conclusions regarding the prevalence and public health significance of thiamin deficiency in Europe can be drawn.

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Data availability Search strategy is available in supplemental data and on PROSPERO: [<https://www.crd.york.ac.uk/PROSPEROFILES/dd0680a43ba5a2be5fad83d708c13cd8.pdf>].

Declarations

Ethics approval and consent to participate Not applicable.

Competing interests The authors declare no competing interests.

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References

- Manzetti S, Zhang J, van der Spoel D. Thiamin function, metabolism, uptake, and transport. *Biochemistry*. 2014;53:821–35. <https://doi.org/10.1021/bi401618y>.
- Brown G. Defects of thiamine transport and metabolism. *J Inher Metab Dis*. 2014;37:577–85. <https://doi.org/10.1007/s10545-014-9712-9>.
- Said HM, Nexø E. Gastrointestinal Handling of Water-Soluble Vitamins. *Compr Physiol*. 2018;8:1291–311. <https://doi.org/10.1002/cphy.c170054>.
- Rajgopal A, Edmondson A, Goldman ID, Zhao R. SLC19A3 encodes a second thiamine transporter ThTr2. *Biochim Biophys Acta*. 2001;1537:175–8. [https://doi.org/10.1016/s0925-4439\(01\)00073-4](https://doi.org/10.1016/s0925-4439(01)00073-4).
- Reidling JC, Lambrecht N, Kassir M, Said HM. Impaired intestinal vitamin B1 (thiamin) uptake in thiamin transporter-2-deficient mice. *Gastroenterology*. 2010;138:1802–9. <https://doi.org/10.1053/j.gastro.2009.10.042>.
- Boulware MJ, Subramanian VS, Said HM, Marchant JS. Polarized expression of members of the solute carrier SLC19A gene family of water-soluble multivitamin transporters: implications for physiological function. *Biochem J*. 2003;376:43–8. <https://doi.org/10.1042/BJ20031220>.
- Kono S, Miyajima H, Yoshida K, et al. Mutations in a thiamine-transporter gene and Wernicke's-like encephalopathy. *N Engl J Med*. 2009;360:1792–4. <https://doi.org/10.1056/NEJMc0809100>.
- Zhao R, Gao F, Goldman ID. Reduced folate carrier transports thiamine monophosphate: an alternative route for thiamine delivery into mammalian cells. *Am J Physiol Cell Physiol*. 2002;282:C1512–1517. <https://doi.org/10.1152/ajpcell.00547.2001>.
- Overton E, Emelyanova A, Bunik VI. Thiamine, gastrointestinal beriberi and acetylcholine signaling. *Front Nutr*. 2025;12:1541054. <https://doi.org/10.3389/fnut.2025.1541054>.
- Zhao R, Gao F, Goldman ID. Molecular cloning of human thiamin pyrophosphokinase. *Biochim Biophys Acta*. 2001;1517:320–2. [https://doi.org/10.1016/s0167-4781\(00\)00264-5](https://doi.org/10.1016/s0167-4781(00)00264-5).
- Mayr JA, Freisinger P, Schlachter K, et al. Thiamine pyrophosphokinase deficiency in encephalopathic children with defects in the pyruvate oxidation pathway. *Am J Hum Genet*. 2011;89:806–12. <https://doi.org/10.1016/j.ajhg.2011.11.007>.
- Nosaka K, Onozuka M, Nishino H, et al. Molecular cloning and expression of a mouse thiamin pyrophosphokinase cDNA. *J Biol Chem*. 1999;274:34129–33. <https://doi.org/10.1074/jbc.274.48.34129>.
- Nghiêm HO, Bettendorff L, Changeux JP. Specific phosphorylation of Torpedo 43K rapsyn by endogenous kinase(s) with thiamine triphosphate as the phosphate donor. *FASEB J*. 2000;14:543–54. <https://doi.org/10.1096/fasebj.14.3.543>.
- Sasa M, Takemoto I, Nishino K, Itokawa Y. The role of thiamine on excitable membrane of crayfish giant axon. *J Nutr Sci Vitaminol (Tokyo)*. 1976;22(SUPPL):21–4. https://doi.org/10.3177/jnsv.22.supplement_21.
- Boyle L, Wamelink MMC, Salomons GS, et al. Mutations in TKT are the cause of a syndrome including short stature, developmental delay, and congenital heart defects. *Am J Hum Genet*. 2016;98:1235–42. <https://doi.org/10.1016/j.ajhg.2016.03.030>.
- Kochetov GA, Solovjeva ON. Structure and functioning mechanism of transketolase. *Biochim Biophys Acta*. 2014;1844:1608–18. <https://doi.org/10.1016/j.bbapap.2014.06.003>.
- McCool BA, Plonk SG, Martin PR, Singleton CK. Cloning of human transketolase cDNAs and comparison of the nucleotide sequence of the coding region in Wernicke-Korsakoff and non-Wernicke-Korsakoff individuals. *J Biol Chem*. 1993;268:1397–404.
- Lindhurst MJ, Fiermonte G, Song S, et al. Knockout of Slc25a19 causes mitochondrial thiamine pyrophosphate depletion, embryonic lethality, CNS malformations, and anemia. *Proc Natl Acad Sci U S A*. 2006;103:15927–32. <https://doi.org/10.1073/pnas.0607661103>.
- Naito E, Ito M, Takeda E, et al. Molecular analysis of abnormal pyruvate dehydrogenase in a patient with thiamine-responsive congenital lactic acidemia. *Pediatr Res*. 1994;36:340–6. <https://doi.org/10.1203/00006450-199409000-00013>.
- Yap ZY, Strucinska K, Matsuzaki S, et al. A biallelic pathogenic variant in the OGDH gene results in a neurological disorder with features of a mitochondrial disease. *J Inher Metab Dis*. 2021;44:388–400. <https://doi.org/10.1002/jimd.12248>.
- Yang N, Han L, Gu X, et al. Analysis of gene mutations in Chinese patients with maple syrup urine disease. *Mol Genet Metab*. 2012;106:412–8. <https://doi.org/10.1016/j.ymgme.2012.05.023>.
- Chuang JL, Wynn RM, Moss CC, et al. Structural and biochemical basis for novel mutations in homozygous Israeli maple syrup urine disease patients: a proposed mechanism for the thiamin-responsive phenotype. *J Biol Chem*. 2004;279:17792–800. <https://doi.org/10.1074/jbc.M313879200>.
- Whitfield KC, Bourassa MW, Adamolekun B, et al. Thiamine deficiency disorders: diagnosis, prevalence, and a roadmap for global control programs. *Ann N Y Acad Sci*. 2018;1430:3–43. <https://doi.org/10.1111/nyas.13919>.
- Johnson CR, Fischer PR, Thacher TD, et al. Thiamin deficiency in low- and middle-income countries: Disorders, prevalences, previous interventions and current recommendations. *Nutr Health*. 2019;25:127–51. <https://doi.org/10.1177/0260106019830847>.

25. European Food Safety Authority. (2025) Food composition data. <https://www.efsa.europa.eu/en/data-report/food-composition-data>. Accessed 15 Sept 2025.
26. Bourassa MW, Bergeron G, Brown KH. A fresh look at thiamine deficiency—new analyses by the global thiamine alliance. *Ann N Y Acad Sci.* 2021;1498:5–8. <https://doi.org/10.1111/nyas.14594>.
27. Anderson M, Mickelsen O, Keys A. Diets and food items for the experimental production of thiamin deficiency in man. *J Am Diet Assoc.* 1946;22:1–7.
28. Berger MM, Shenkin A, Schweinlin A, et al. ESPEN micronutrient guideline. *Clin Nutr.* 2022;41:1357–424. <https://doi.org/10.1016/j.clnu.2022.02.015>.
29. Karakonstantis S, Galani D, Korela D, et al. Missing the early signs of thiamine deficiency. A case associated with a liquid-only diet. *Nutr Neurosci.* 2020;23:384–6. <https://doi.org/10.1080/1028415X.2018.1507964>.
30. Lonsdale D. Thiamin. *Adv Food Nutr Res.* 2018;83:1–56. <https://doi.org/10.1016/bs.afnr.2017.11.001>.
31. Mauermann ML, Staff NP. Peripheral neuropathy: a review. *JAMA.* 2026;335:255–66. <https://doi.org/10.1001/jama.2025.19400>.
32. Pereira AG, Cunha LN, de Paiva P SAR, et al. An Overview of Beriberi. *Med Princ Pract Int J Kuwait Univ Health Sci Cent.* 2025;1–7. <https://doi.org/10.1159/000547719>.
33. Inoue A, Itabashi K, Iwai T, et al. Imaging findings of vitamin deficiencies: are they forgotten diseases? *BJR|Open.* 2021;3:20210011. <https://doi.org/10.1259/bjro.20210011>.
34. Coats D, Shelton-Dodge K, Ou K, et al. Thiamine deficiency in Cambodian infants with and without beriberi. *J Pediatr.* 2012;161:843–7. <https://doi.org/10.1016/j.jpeds.2012.05.006>.
35. Smith TJ, Johnson CR, Koshy R, et al. Thiamine deficiency disorders: a clinical perspective. *Ann N Y Acad Sci.* 2021;1498:9–28. <https://doi.org/10.1111/nyas.14536>.
36. World Health Organization. Thiamine deficiency and its prevention and control in major emergencies. WHO Reference Number: WHO/NHD/99.13. Geneva: World Health Organization. 1999. Available from: <https://www.who.int/publications/i/item/WHO-NHD-99.13>
37. Li S, Xing C. Wernicke encephalopathy: a mini review of the clinical spectrum, atypical manifestations, and diagnostic challenges. *Front Neurol.* 2025;16:1566366. <https://doi.org/10.3389/fneur.2025.1566366>.
38. Brust JCM. A 74-year-old man with memory loss and neuropathy who enjoys alcoholic beverages. *JAMA.* 2008;299:1046–54. <https://doi.org/10.1001/jama.299.5.jrr80000>.
39. Harper CG, Giles M, Finlay-Jones R. Clinical signs in the Wernicke-Korsakoff complex: a retrospective analysis of 131 cases diagnosed at necropsy. *J Neurol Neurosurg Psychiatry.* 1986;49:341–5. <https://doi.org/10.1136/jnnp.49.4.341>.
40. Lindboe CF, Løberg EM. Wernicke's encephalopathy in non-alcoholics. An autopsy study. *J Neurol Sci.* 1989;90:125–9. [https://doi.org/10.1016/0022-510x\(89\)90095-6](https://doi.org/10.1016/0022-510x(89)90095-6).
41. Mancinelli R, Ceccanti M. Biomarkers in alcohol misuse: their role in the prevention and detection of thiamine deficiency. *Alcohol and alcoholism (Oxford, Oxfordshire).* 2009;44:177–82. <https://doi.org/10.1093/alcalc/agn117>.
42. Kerns JC, Arundel C, Chawla LS. Thiamin Deficiency in People with Obesity12. *Adv Nutr.* 2015;6:147–53. <https://doi.org/10.3945/an.114.007526>.
43. Salvatori G, Mondì V, Piersigilli F, et al. Thiamine Deficiency in a Developed Country: Acute Lactic Acidosis in Two Neonates Due to Unsupplemented Parenteral Nutrition. *JPEN J Parenter Enter Nutr.* 2016;40:886–9. <https://doi.org/10.1177/0148607114568316>.
44. Ziegler D, Reiners K, Strom A, Obeid R. Association between diabetes and thiamine status - a systematic review and meta-analysis. *Metabolism.* 2023;144:155565. <https://doi.org/10.1016/j.metabol.2023.155565>.
45. da Silva JSV, Seres DS, Sabino K, et al. ASPEN consensus recommendations for refeeding syndrome. *Nutr Clin Pract.* 2020;35:178–95. <https://doi.org/10.1002/ncp.10474>.
46. Shim J-S, Shim SY, Cha H-J, et al. Association between ultra-processed food consumption and dietary intake and diet quality in Korean adults. *J Acad Nutr Diet.* 2022;122:583–94. <https://doi.org/10.1016/j.jand.2021.07.012>.
47. Mertens E, Colizzi C, Peñalvo JL. Ultra-processed food consumption in adults across Europe. *Eur J Nutr.* 2022;61:1521–39. <https://doi.org/10.1007/s00394-021-02733-7>.
48. European Food Safety Authority. Dietary reference values for thiamin. *EFSA J.* 2016. <https://doi.org/10.2903/j.efsa.2016.4653>.
49. Rakotoambinina B, Hiffler L, Gomes F. Pediatric thiamine deficiency disorders in high-income countries between 2000 and 2020: a clinical reappraisal. *Ann N Y Acad Sci.* 2021;1498:57–76. <https://doi.org/10.1111/nyas.14669>.
50. O'Brien M, Quirke R, Gowan R, McNicholas F. Prevalence of thiamine deficiency in anorexia nervosa: a systematic review and narrative synthesis. *Eur Child Adolesc Psychiatry.* 2025;34:1281–93. <https://doi.org/10.1007/s00787-024-02576-7>.
51. Karimi Behnagh A, Eghbali M, Abdolmaleki F, et al. Pre- and post-surgical prevalence of thiamine deficiency in patients undergoing bariatric surgery: a systematic review and meta-analysis. *Obes Surg.* 2024;34:653–65. <https://doi.org/10.1007/s11695-023-06896-6>.
52. Oudman E, Wijnia JW, Oey M, et al. Wernicke's encephalopathy in hyperemesis gravidarum: a systematic review. *Eur J Obstet Gynecol Reprod Biol.* 2019;236:84–93. <https://doi.org/10.1016/j.ejogrb.2019.03.006>.

53. Elmadfa I, Majchrzak D, Rust P, Genser D. The thiamine status of adult humans depends on carbohydrate intake. *Int J Vitam Nutr Res*. 2001;71:217–21. <https://doi.org/10.1024/0300-9831.71.4.217>.
54. Clark AF. Use of thiamine supplementation in pregnant women diagnosed with hyperemesis gravidarum and Wernicke encephalopathy. *Nurs Womens Health*. 2024;28:222–6. <https://doi.org/10.1016/j.nwh.2024.02.003>.
55. Saka Y, Naruse T, Kato A, et al. Thiamine status in end-stage chronic kidney disease patients: a single-center study. *Int Urol Nephrol*. 2018;50:1913–8. <https://doi.org/10.1007/s11255-018-1974-y>.
56. Lautenschläger T, Neinhuis C, Monizi M, et al. Edible insects of northern Angola. *Afr Invertebr*. 2017;58:55–82. <https://doi.org/10.3897/afrinvertebr.58.21083>.
57. Nishimune T, Watanabe Y, Okazaki H, Akai H. Thiamin is decomposed due to *Anaphe* spp. entomophagy in seasonal ataxia patients in Nigeria. *J Nutr*. 2000;130:1625–8. <https://doi.org/10.1093/jn/130.6.1625>.
58. Vid Vičič A. Kukec (2025) Systematic review of thiamine (vitamin B1) status in healthy European adults ID - CRD420251101630 ER -. In: <https://www.crd.york.ac.uk/PROSPERO/view/CRD420251101630>
59. Page MJ, McKenzie JE, Bossuyt PM, et al. The PRISMA 2020 statement: an updated guideline for reporting systematic reviews. *Syst Rev*. 2021;10:89. <https://doi.org/10.1186/s13643-021-01626-4>.
60. Ortega RM, Martínez RM, Andrés P, et al. Thiamin status during the third trimester of pregnancy and its influence on thiamin concentrations in transition and mature breast milk. *Br J Nutr*. 2004;92:129–35. <https://doi.org/10.1079/BJN20041153>.
61. Sánchez DJ, Murphy MM, Bosch-Sabater J, Fernández-Ballart J. Enzymic evaluation of thiamin, riboflavin and pyridoxine status of parturient mothers and their newborn infants in a Mediterranean area of Spain. *Eur J Clin Nutr*. 1999;53:27–38. <https://doi.org/10.1038/sj.ejcn.1600674>.
62. Lynch PLM, Trimble ER, Young IS. High-performance liquid chromatographic determination of thiamine diphosphate in erythrocytes using internal standard methodology. *J Chromatogr B Biomed Sci Appl*. 1997;701:120–3. [https://doi.org/10.1016/S0378-4347\(97\)00353-8](https://doi.org/10.1016/S0378-4347(97)00353-8).
63. Boni L, Kieckens L, Hendriks A. An evaluation of a modified erythrocyte transketolase assay for assessing thiamine nutritional adequacy. *J Nutr Sci Vitaminol (Tokyo)*. 1980;26:507–14. <https://doi.org/10.3177/jnsv.26.507>.
64. Talwar D, Davidson H, Cooney J, St JO'Reilly D. Vitamin B(1) status assessed by direct measurement of thiamin pyrophosphate in erythrocytes or whole blood by HPLC: comparison with erythrocyte transketolase activation assay. *Clin Chem*. 2000;46:704–10.
65. Hötzel D, Bitsch R. Thiamine status of human subjects, estimated by biochemical methods. *J Nutr Sci Vitaminol (Tokyo)*. 1976;22:41–5. https://doi.org/10.3177/jnsv.22.Supplement_41.
66. Mancinelli R, Ceccanti M, Guiducci MS, et al. Simultaneous liquid chromatographic assessment of thiamine, thiamine monophosphate and thiamine diphosphate in human erythrocytes: a study on alcoholics. *J Chromatogr B*. 2003;789:355–63. [https://doi.org/10.1016/S1570-0232\(03\)00139-9](https://doi.org/10.1016/S1570-0232(03)00139-9).
67. Fabian E, Majchrzak D, Dieminger B, et al. Influence of probiotic and conventional yoghurt on the status of vitamins B1, B2 and B6 in young healthy women. *Ann Nutr Metab*. 2008;52:29–36. <https://doi.org/10.1159/000114408>.
68. Herve C, Beyne P, Lettéron P, Delacoux E. Comparison of erythrocyte transketolase activity with thiamine and thiamine phosphate ester levels in chronic alcoholic patients. *Clin Chim Acta Int J Clin Chem*. 1995;234:91–100. [https://doi.org/10.1016/0009-8981\(94\)05980-7](https://doi.org/10.1016/0009-8981(94)05980-7).
69. Jermendy G. Evaluating thiamine deficiency in patients with diabetes. *Diab Vasc Dis Res*. 2006;3:120–1. <https://doi.org/10.3132/dvdr.2006.014>.
70. Malara M, Hübner-Wozniak E, Lewandowska I. Assessment of intake and nutritional status of vitamin B1, B2, and B6 in men and women with different physical activity levels. *Biol Sport*. 2013;30:117–23. <https://doi.org/10.5604/20831862.1044430>.
71. Schrijver J, Speek AJ, Klosse JA, et al. A reliable semiautomated method for the determination of total thiamine in whole blood by the thiochrome method with high-performance liquid chromatography. *Ann Clin Biochem*. 1982;19:52–6. <https://doi.org/10.1177/000456328201900111>.
72. Verstraete J, Stove C. Volumetric absorptive microsampling (VAMS) as a reliable tool to assess thiamine status in dried blood microsamples: a comparative study. *Am J Clin Nutr*. 2021;114:1200–7. <https://doi.org/10.1093/ajcn/nqab146>.
73. Körner RW, Vierzig A, Roth B, Müller C. Determination of thiamin diphosphate in whole blood samples by high-performance liquid chromatography—a method suitable for pediatric diagnostics. *J Chromatogr B Analyt Technol Biomed Life Sci*. 2009;877:1882–6. <https://doi.org/10.1016/j.jchromb.2009.05.013>.
74. Tallaksen CME, Böhmer T, Bell H, Karlsen J. Concomitant determination of thiamin and its phosphate esters in human blood and serum by high-performance liquid chromatography. *J Chromatogr B Biomed Sci Appl*. 1991;564:127–36. [https://doi.org/10.1016/0378-4347\(91\)80075-N](https://doi.org/10.1016/0378-4347(91)80075-N).
75. Michalak S, Michałowska-Wender G, Adamcewicz G, Wender MB. Erythrocyte transketolase activity in patients with diabetic and alcoholic neuropathies. *Folia Neuropathol*. 2013;51:222–6. <https://doi.org/10.5114/fn.2013.37706>.
76. Lu J, Frank EL. Rapid HPLC Measurement of Thiamine and Its Phosphate Esters in Whole Blood. *Clin Chem*. 2008;54:901–6. <https://doi.org/10.1373/clinchem.2007.099077>.

77. Jones KS, Parkington DA, Cox LJ, Koulman A. Erythrocyte transketolase activity coefficient (ETKAC) assay protocol for the assessment of thiamine status. *Ann N Y Acad Sci.* 2021;1498:77–84. <https://doi.org/10.1111/nyas.14547>.
78. Tasevska N, Runswick SA, McTaggart A, Bingham SA. Twenty-four-hour urinary thiamine as a biomarker for the assessment of thiamine intake. *Eur J Clin Nutr.* 2008;62:1139–47. <https://doi.org/10.1038/sj.ejcn.1602829>.
79. Jones KS, Parkington DA, Bourassa MW, et al. Protocol and application of basal erythrocyte transketolase activity to improve assessment of thiamine status. *Ann N Y Acad Sci.* 2023;1521:104–11. <https://doi.org/10.1111/nyas.14962>.
80. Herzog R, Álvarez-Pasquin MJ, Díaz C, et al. Are healthcare workers' intentions to vaccinate related to their knowledge, beliefs and attitudes? A systematic review. *BMC Public Health.* 2013;13:154. <https://doi.org/10.1186/1471-2458-13-154>.
81. Koulman A, Jones KS, Collins D, et al. Ethnic differences in thiamine status persist after adjusting for diet; findings from the UK National Diet and Nutrition Survey. *Clin Nutr ESPEN.* 2026;72:102893. <https://doi.org/10.1016/j.clnesp.2025.102893>.
82. Mataix J, Aranda P, Sánchez C, et al. Assessment of thiamin (vitamin B1) and riboflavin (vitamin B2) status in an adult Mediterranean population. *Br J Nutr.* 2003;90:661–6. <https://doi.org/10.1079/BJN2003926>.
83. UK Data Service. <https://datacatalogue.ukdataservice.ac.uk/studies/study/6533#doi>. Accessed 22 May 2026.
84. Churuangsk C, Catchpole A, Talwar D, et al. Low thiamine status in adults following low-carbohydrate / ketogenic diets: a cross-sectional comparative study of micronutrient intake and status. *Eur J Nutr.* 2024;63:2667–79. <https://doi.org/10.1007/s00394-024-03459-y>.
85. Bread. and flour: labelling and composition. In: GOV.UK. <https://www.gov.uk/guidance/bread-and-flour-labelling-and-composition>. Accessed 17 July 2025.
86. Zhang G, Ding H, Chen H, et al. Thiamine nutritional status and depressive symptoms are inversely associated among older Chinese adults. *J Nutr.* 2013;143:53–8. <https://doi.org/10.3945/jn.112.167007>.
87. Gallant J, Chan K, Green TJ, et al. Low-dose thiamine supplementation of lactating Cambodian mothers improves human milk thiamine concentrations: a randomized controlled trial. *Am J Clin Nutr.* 2021;114:90–100. <https://doi.org/10.1093/ajcn/nqab052>.
88. Lonsdale D. Sudden infant death syndrome and abnormal metabolism of thiamin. *Med Hypotheses.* 2015;85:922–6. <https://doi.org/10.1016/j.mehy.2015.09.009>.
89. Rehm J, Anderson P, Barry J, et al. Prevalence of and potential influencing factors for alcohol dependence in Europe. *Eur Addict Res.* 2015;21:6–18. <https://doi.org/10.1159/000365284>.
90. Bojko ER. Thiamine status in inhabitants of the Russian European North. *Int J Circumpolar Health.* 2001;60:466–8. <https://doi.org/10.1080/25761900.2022.12220617>.
91. Bichkaeva FA, Tkachev AV, Boiko ER, et al. Vitamin B1 levels in residents of the European North. *Hum Physiol.* 2003;29:119–21. <https://doi.org/10.1023/A:1022073123466>.
92. Thornalley PJ, Babaei-Jadidi R, Al Ali H, et al. High prevalence of low plasma thiamine concentration in diabetes linked to a marker of vascular disease. *Diabetologia.* 2007;50:2164–70. <https://doi.org/10.1007/s00125-007-0771-4>.
93. Nix WA, Zirwes R, Bangert V, et al. Vitamin B status in patients with type 2 diabetes mellitus with and without incipient nephropathy. *Diabetes Res Clin Pract.* 2015;107:157–65. <https://doi.org/10.1016/j.diabres.2014.09.058>.
94. Whitfield KC, Smith TJ, Rohner F, et al. Thiamine fortification strategies in low- and middle-income settings: a review. *Ann N Y Acad Sci.* 2021;1498:29–45. <https://doi.org/10.1111/nyas.14565>.
95. Churuangsk C, Griffiths D, Lean MEJ, Combet E. Impacts of carbohydrate-restricted diets on micronutrient intakes and status: a systematic review. *Obes Rev.* 2019;20:1132–47. <https://doi.org/10.1111/obr.12857>.
96. European Commission, Scientific Committee on Food. Opinion of the Scientific Committee on Food on the Tolerable Upper Intake Level of Vitamin B1 (expressed on 11 July 2001). SCF/CS/NUT/UPPLEV/46 Final, 16 July 2001. Brussels: European Commission. Available at: https://food.ec.europa.eu/system/files/2020-12/sci-com_scf_out93_en.pdf

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