

Current Evidence on Thiamin (Vitamin B1) Status in Healthy European Adults: A Systematic Review of Biomarkers and Deficiency Prevalence

Vid VIČIČ

`vid.vicic@zf.uni-lj.si`

University of Ljubljana, Faculty of Health Sciences

Sergej PIRKMAJER

University of Ljubljana, Faculty of Medicine

Andreja KUKEC

University of Ljubljana, Faculty of Medicine

Systematic Review

Keywords: Thiamin, Micronutrient deficiency, Wernicke encephalopathy, beriberi, Europe, Vitamin B1, ThDP, ETKAC

Posted Date: April 13th, 2026

DOI: <https://doi.org/10.21203/rs.3.rs-8997297/v1>

License:   This work is licensed under a Creative Commons Attribution 4.0 International License. [Read Full License](#)

Additional Declarations: No competing interests reported.

Version of Record: A version of this preprint was published at Journal of Health, Population and Nutrition on September 29th, 2026. See the published version at <https://doi.org/10.1186/s41043-026-01469-6>.

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: A total of 19 cross-sectional studies from 11 countries, published between 1976 and 2024, met the inclusion criteria, providing data on 1687 individuals. 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 %. When pregnant and post-partum women were excluded, the prevalence of thiamin deficiency ranged from 0 % to 10 %.

Discussion: The prevalence of thiamin deficiency in Europe has not been reliably established owing to the lack of representative, population-based studies. Representative studies using harmonized thiamin biomarkers are urgently needed to establish the prevalence and public health significance of thiamin deficiency in Europe. Particular attention should be given to prevention of thiamin deficiency in high-risk groups, such as pregnant and lactating women, individuals following low-carbohydrate high-fat (LCHF) diets, and people with alcohol use disorder.

Conclusions: It is commonly assumed that thiamin deficiency is not a problem in Europe, but the main finding of this systematic review is that there is virtually no representative data to support this assumption.

1. BACKGROUND

Thiamin (vitamin B1) is a water-soluble vitamin essential for carbohydrate and amino acid metabolism, energy production and neuronal function [1]. Thiamin is ingested mainly in phosphorylated forms and free thiamin is produced by intestinal phosphatases [2, 3]. Once dephosphorylated, free thiamin is absorbed in the small intestine primarily via thiamin transporters THTR1 (SLC19A2) and THTR2 (SLC19A3) [4, 5]. THTR1 and THTR2 also mediate thiamin transport in the kidney and other tissues [6, 7]. In addition to these classical thiamin transporters, several other carriers are likely involved in thiamin handling [8]. For instance, reduced folate carrier 1 (RFC1 aka SLC19A1) may contribute to thiamin absorption by transporting thiamin monophosphate [9].

Inside the cell, thiamin is phosphorylated to thiamin diphosphate (ThDP) (older name: thiamin pyrophosphate -TPP), its major physiological form, by thiamin pyrophosphokinase [10–12]. In the cytoplasm, ThDP participates in the pentose phosphate pathway as a cofactor of transketolase (Fig. 1), which catalyses the conversion of pentose sugars to intermediates of glycolysis, thus enabling production of NADPH even when ribose-5-phosphate is not needed for nucleotide synthesis [2, 13–15]. Mitochondrial carrier SLC25A19

transports ThDP to the mitochondrial matrix [16], where it serves as a cofactor in oxidative decarboxylation of α -keto acids, including pyruvate [16, 17], α -ketoglutarate [16, 18], and α -keto acids of branched-chain amino acids [19, 20] (Fig. 1). Besides the role of ThDP as a cofactor, thiamin and its phosphorylated derivatives affect membrane function and seem to play a role in regulation of neuronal excitability [21, 22].

Thiamin deficiency is still widespread in low- and middle-income countries that rely heavily on processed staple foods, such as polished rice. Although thiamin is naturally present in a wide range of foods, its content is significantly diminished by bran removal (milling, polishing), refining, processing, and cooking [23]. The best sources of thiamin are pork, wholegrain cereals and legumes [24].

Although 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 or people with alcohol use disorder (AUD) [25], this assumption is hardly supported by evidence. There are few general population-based studies [26, 27] that have investigated thiamin status in European countries, highlighting a critical gap in public health research.

While some micronutrients like vitamin D have been extensively studied in high income populations, thiamin status remains under-investigated in epidemiological research [28].

Thiamin must be supplied regularly, as body stores are minimal. Diet almost devoid of thiamin produces first sign of deficiency in just 10 days [29]. Clinical thiamin deficiency may present with a spectrum of neurological, psychiatric, and cardiovascular signs and symptoms [30]. Subclinical deficiencies can also impair cognitive and physical functions [23]. Early signs of thiamin deficiency are often unrecognized [31], none of the signs and symptoms are pathognomonic, and can be mistaken for psychosomatic disorders [32].

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 [33], while wet beriberi presents with increased cardiac output, cardiomegaly, heart failure, and peripheral oedema [34, 35]. Cardiac dysfunction, which can be severe, is a major feature of infantile beriberi which appears in infants during first months of life [36], while the nervous system dysfunction may predominate in older children [37]. Thiamin deficiency, especially in alcohol use disorder, may manifest also as Wernicke's encephalopathy and Korsakoff syndrome [25]. Wernicke's encephalopathy is characterized by mental changes, abnormal eye movements, and/or ataxia [38–40]. If untreated, Wernicke's encephalopathy may progress to Korsakoff's syndrome, an irreversible condition which manifests with amnesia and confabulation [40]. In one study the researchers examined material from 6,964 autopsies. 52 cases of Wernicke's encephalopathy were found, of which 12 (23%) were found in people without alcohol dependence. Only 4 cases (7.7%) were diagnosed based on clinical symptoms in living patients. None of the people without alcohol dependence were given thiamin therapy [41].

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 [42]. Thiamin deficiency also occurs in malnourished patients, patients after bariatric surgery [30], people living with obesity [43], patients on total parenteral nutrition [30, 44] and patients with diabetes [45]. 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) [46]. Lastly, ultra-processed foods, snacks and soft drinks contribute significantly to energy intake while being low in thiamin [47]. The population reference intake for thiamin is the same for all population groups: 0.1 mg/MJ (0.4 mg/1000 kcal) as it is dependent on energy intake [48]. The ultra-processed food share in energy intake in Europe ranges from 14–44%, the highest being in United Kingdom [49]. All risk factors contributing to thiamin deficiency are presented in Fig. 2.

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

2. 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].

2.1 STUDY POPULATION, EXPOSURE AND OUTCOME

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

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].

There is also a possibility of measurement of excreted thiamin in 24h urine, which has good correlation with thiamin intake [48, 60], but is not a biomarker of thiamin status, especially when thiamin intake is low [61].

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.

2.2 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 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.

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 [30].	Deficiency < 70 nmol/L [62].
ThDP - thiamin diphosphate in erythrocytes (older name: thiamin pyrophosphate -TPP)	Washed erythrocytes		Deficiency < 280 ng/g Hb RBC ThDP [63].
Total thiamin in whole blood (free thiamin plus its esters)	Whole blood	Includes inactive thiamin forms. Less reliable biomarker of thiamin status than ThDP - thiamin diphosphate in whole blood or erythrocytes [23].	< 75nmol/L [62].
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)[61].	Deficiency: ETKAC > 1.25 (α > 25%) Insufficiency: ETKAC 1.15–1.25 (α 15–25%). Adequate (sufficiency): ETKAC < 1.15 (α < 15%) [23, 48, 61].

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	24h urine	1 mg of thiamin intake predicts 268.2 µg of thiamin excreted in urine [60]	Biomarker of thiamin intake, not status. Not reliable when thiamin intakes are low. Requires at least 24h urine collection [48, 60, 61].
Basal ETK - Basal erythrocyte transketolase activity	Washed erythrocytes	Complementary biomarker [64].	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 [64]. This allows differentiation between whether the low activity is due to thiamin deficiency or reduced synthesis of the enzyme itself.

2.3 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 excel spreadsheet.
- The data was then rechecked by second researcher.

Study risk of bias assessment

Newcastle-Ottawa Scale adapted for cross-sectional studies was used [65]. Grading is available in supplemental data.

DATA SYNTHESIS AND ANALYSIS

Only one study included at least a regionally representative population sample [27] and many countries were not represented. The meta-analysis was not feasible.

3. RESULTS

A total of 19 cross-sectional studies from 11 countries published between 1976 and 2024 met the inclusion criteria and provided data on the thiamin status of 1687 healthy European adults. The results are stratified by the biomarkers erythrocyte transketolase activity coefficient (ETKAC) in Table 3, whole blood thiamin diphosphate (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 was a single (Spanish) study with a representative sample of the healthy adult population [27].

3.1 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%.

Table 3
Studies reporting erythrocyte transketolase activity coefficients (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
Ortega et al. 2004 [66]	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 [67]	Healthy post-partum women	Coast of Catalonia province, Spain	131 (#131)	26 $\pm$ 5	1.25 $\pm$ 0.17	41.2%	62.6%
Malara et al. 2013 [68]	Healthy volunteers with low or high physical activity	Warsaw, Poland	80 (#40)	19.7 $\pm$ 0.71	1.053 $\pm$ 0.054	0%	
Mataix et al. 2003 [27]	Representative sample of healthy population	Andalusia, Spain	372 (#191)	25–60	1.01 $\pm$ 0.17	6.4%	13.6% (ETKAC > 1.16)
Hötzel and Bitsch 1976 [69]	Healthy students	Bonn, West Germany	130 (#84)	adult	1.126 $\pm$ 0.121	10.00%	20%
Jones et al. 2023 [64]	Selected samples from nationally representative study NDNS RP	United Kingdom	134 (#134)	44.0 $\pm$ 11.8	1.09 $\pm$ 0.05	0%	13%
Fabian et al. 2008 [70]	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	
Studies using healthy volunteers for controls or reference values establishment are marked with (#) and presented in grey.							
Herve et al. 1995 [71]	Healthy volunteers (#)	Clichy, France	52 (#28)	21–65	1.12 $\pm$ 0.1	N/A	N/A
Elmadfa et al. 2001 [53]	Healthy volunteers (#)	Vienna, Austria	12 (#6)	25–30	1.12 $\pm$ 0.1	N/A	

Author, year	Population	Location	No. of subjects	Age (mean ± SD or range yrs)	ETKAC (mean ± SD)	Thiamin deficiency (%) ETKAC > 1.25	Thiamin insufficiency (%) ETKAC > 1.15
Jermendy 2006 [72]	Healthy control (#)	Budapest, Hungary.	60 (28)	54.1 ± 2.5	1.08 ± 0.02	N/A	
Boni et al. 1980 [73]	Healthy medical and laboratory personnel (#)	Leuven, Belgium	12 (NR)	adult	1.135 (range 1.08– 1.185)	0%	N/A
SD, standard deviation; ETKAC, erythrocyte transketolase activity coefficients.							

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.						
Mancinelli et al. 2003 [74]	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
Verstraete and Stove 2021 [75]	Healthy volunteers (#)	Ghent, Belgium	50 (NR)	adult	145 $\pm$ 22.1	0%
Körner et al. 2009 [76]	Healthy volunteers (#)	North Rhine-Westphalia, Germany	30 (16)	40.5 (18–65)	194.7 $\pm$ 27.5	0%
Herve et al. 1995 [71]	Healthy volunteers (#)	Clichy, France	52 (28)	21–65	176 $\pm$ 28	N/A
Lynch et al. 1997 [77]	Healthy laboratory staff (#)	Belfast, United Kingdom	45 (16)	38 (20–60) median (range)	174.2 $\pm$ 34.05	N/A
Tallaksen et al. 1991 [78]	Healthy volunteers (#)	Oslo, Norway	30 (15)	43 (32–54) median (range)	143 $\pm$ 35	N/A
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.						
Schrijver et al. 1982 [79]	Healthy volunteers (#)	Utrecht, Netherlands	98 (#nd)	adult	115 (range 70–185)	1–2%
Herve et al. 1995 [71]	Healthy volunteers (#)	Clichy, France	52 (#28)	21–65	186 $\pm$ 30	N/A
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
Churuangsuk et al. 2024 [26]	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 %
Studies using healthy volunteers for controls or reference values establishment are marked with (#) and presented in grey.						
Talwar et al. 2000 [63]	Laboratory staff and people attending a cardiovascular risk clinic (#)	Glasgow, United Kingdom	147 (♀68)	54 $\pm$ 8.2	433 $\pm$ 76.5	2.5 %
Tallaksen et al. 1991 [78]	Healthy volunteers (#)	Oslo, Norway	30 (♀15)	43 (32-54) median (range)	325 $\pm$ 76.95	N/A
SD, standard deviation; RBC ThDP, erythrocyte thiamin diphosphate; IQR, interquartile range						

4. DISCUSSION

To the best of our knowledge, and according to the available literature, this systematic review represents the first comprehensive summary of thiamin status in healthy European adults. On a global level the prevalence of thiamin deficiency was reviewed by Johnson et al. [80] and Whitfield et al. [23] and systematic review of thiamin deficiency cases in paediatric population in high-income countries was done by Rakotoambinina et al. [81].

The prevalence of thiamin deficiency and insufficiency in Europe remains largely unknown. The available data, limited to 19 studies from only eleven countries, highlights the need for additional research. One study using a representative sample from the Andalusian region in Spain reported a deficiency prevalence of 6.4% (ETKAC > 1.25) and insufficiency 13.6% (ETKAC > 1.16 (authors used slightly different cutoff)) [27]. Another study done in the United Kingdom used selected samples from nationally representative study found prevalence of thiamin insufficiency (ETKAC > 1.15) in 13%. This suggests that the extent of the issue may be significantly higher than previously assumed [64].

Inadequate thiamin intake was recently estimated in a modelling analysis, which compared age and sex specific nutrient requirements with available dietary intake data. Based on these estimates 29.7% of the global population (2.2 billion people) have inadequate thiamin intake. For European countries the prevalence of estimated inadequate intake ranged from 8.4% (Belgium) to 56.2% (Italy). The model did not account for intake from food fortification or food supplements [82]. As dietary intake data are typically self-reported, they are subject to bias and must be interpreted with caution [83]. Additionally methodological differences and errors in data collection can produce significant errors. For example, in Cambodia, a country with one of the highest prevalences of thiamin deficiency [80] their estimated prevalence of inadequate thiamin intake was only 24% [82].

4.1 SPECIAL SUBPOPULATIONS

The risk of thiamin deficiency is higher in populations with increased relative thiamin requirements, such as pregnant and post-partum women (European Food Safety Authority, 2016; Oudman et al., 2019). A Spanish study in women in the third trimester of pregnancy reported thiamin deficiency in 13.7% of participants (ETKAC > 1.25) (Ortega et al., 2004), while another Spanish study in post-partum women 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) [67]. Moreover, breast milk of thiamin-deficient mothers contains reduced thiamin levels, leading to deficiency in the offspring [66, 84]. Conditions such as hyperemesis gravidarum reduce dietary intake. Additionally i/v glucose administration during labor increases thiamin needs [54]. Cases of Wernicke encephalopathy with long-term consequences following hyperemesis gravidarum have also been reported [52]. Thiamin deficiency has been documented in families exhibiting a high incidence of Sudden Infant Death Syndrome (SIDS) [25, 85].

A systematic review by Rakotoambinina et al. [81] summarized pediatric thiamin deficiency in high-income countries and identified predisposing factors including genetic causes, diabetes, obesity, excessive consumption of sugar-sweetened beverages, eating disorders, gastrointestinal disorders/surgeries, critical illness, cancer, and artificial nutrition. In the European union, this risk is mitigated by mandatory fortification of processed cereal-based baby foods with thiamin at 25 µg/100 kJ (100 µg/100 kcal) [86] and the composition of infant formulas in EU, including thiamin content is tightly regulated [87].

General fortification of cereals in most European countries is not mandatory [23]. This raises concerns regarding population-level adequacy, particularly given the high proportion of thiamin-devoid ultra-processed foods in the diet. Across Europe, ultra-processed foods account for 14–44% of total energy intake, with the highest share reported in the United Kingdom [49]. Despite this high prevalence of thiamin deficiency

(ETKAC > 1.25) was found in Norwich (UK) adolescents. 13.5% had thiamin deficiency and 26.8% had thiamin insufficiency (ETKAC > 1.15) [88]. Korean study found association of higher ultra-processed food intake with lower thiamin intake [47]. However, a Brazilian study has shown that the consumption of ultra-processed foods is associated with a lower risk of inadequate thiamin intake due to fortification of ultra processed food [89].

Alcohol dependence significantly contributes to thiamin deficiency; 30–80% alcohol dependent individuals are thiamin deficient [42]. 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%) [90].

Restrictive diets also increase risk for deficiency. Thiamin deficiency (α -ETK > 1.25) was found in 8% of volunteers following unsupplemented LCHF (Low-carbohydrate high-fat) and in 2% of healthy unsupplemented Glasgow (UK) adults following general diet and [26].

High prevalence of thiamin deficiency was reported in people living with obesity; in those preparing for bariatric surgery the pooled prevalence was 7% (95% CI: 4–12%). Many studies included patients already on weight loss diet [51]. Two studies not included in the systematic review were conducted in the northern regions of the Russian Federation, where thiamin deficiency (ETKAC > 1.25) was highly prevalent [91, 92]. Reported prevalence of deficiency for Cargopol, Arkangelsk province was 57.7% in males and 68.8% in females (ETKAC > 1.25) [92].

4.2 LIMITATIONS AND STRENGTHS

The main limitation of this review is inclusion of studies that were not using representative samples of population. Study populations included in this review are very heterogeneous and include pregnant women [66], post-partum women [67], convenience samples comprising of staff, that are usually healthier better nourished, and socioeconomically advantaged [63, 73, 77], university students [69], and employees undergoing routine medical examinations [74]. The remaining studies utilized cohorts of healthy volunteers serving as control groups or for reference value establishment [53, 70–72, 68, 79, 75, 76, 78]. However, the main strength of this review is the strict definition of appropriate thiamin status biomarkers. Many studies continue to be published, cited, and included in systematic reviews using inappropriate biomarkers, such as plasma thiamin or plasma thiamin monophosphate (ThMP). Studies that did not specify or cite the method used were excluded from this review. The cutoffs for thiamin deficiency, insufficiency, and adequacy are not universally standardized. Johnson et al. [80] 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.

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 [64, 80, 93].

4.3 PRACTICAL CONSIDERATIONS

In contrast to vitamin D, for which adequate intake from unfortified foods is practically unachievable and population sufficiency can only be ensured through fortification [94], sufficient thiamin intake in high-income

adult populations can easily be achieved through a varied diet. Thiamin deficiency could be considered a sign of alcohol abuse [42], restrictive diets [26, 95] or generally very poor diet. In thiamin deficient populations, deficiency of other micronutrients should be expected [67, 95]. Therefore public health interventions should promote balanced diet that includes cereals, especially fortified breakfast cereals, seeds, legumes and meat, particularly lean pork [48]. Considering the increased needs during pregnancy and lactation, severe consequences of thiamin deficiency [25, 85] and safety of thiamin, routine supplementation by prenatal micronutrient food supplements containing thiamin is reasonable. Even at dosages of several hundred milligrams, there are no reports of adverse effects of oral thiamin supplementation [96]. This also ensures sufficient thiamin content in breast milk [66, 84]. Infants and children should be provided with varied diet, and fortified cereals should be used. Thiamin supplementation in clinical settings is covered in ESPEN micronutrient guidelines [30] and thiamin use in persons with alcohol use disorder (AUD) was described by Shakory [97].

4.3 FUTURE RESEARCH

Representative, population-based studies using harmonized biomarkers are urgently needed to establish the prevalence and risk factors for thiamin deficiency in 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.

5. CONCLUSIONS

Until general population-level data become available, it is scientifically unjustified to conclude that thiamin deficiency is not a public health concern in Europe.

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

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

Declarations

Author Contributions:

Conceptualisation: V.V.; writing: V.V., S.P.; data synthesis: V.V., A.K., supervision: A.K. All authors have read and agreed to the published version of the manuscript.

Funding: This work was supported by the Slovenian Research and Innovation Agency (ARIS), projects: 1. MULTIPART (J7-60123); 2. SLORapro (P3-0429); 3. Molekularni mehanizmi razvoja in delovanja skeletne mišice (P3-0043) and 4. NeuroMyo (J7-60125).

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

Conflict of interest: There are no conflicts of interest to declare.

Ethics approval: Not applicable.

References

1. Manzetti S, Zhang J, van der Spoel D (2014) Thiamin function, metabolism, uptake, and transport. *Biochemistry* 53:821–835. <https://doi.org/10.1021/bi401618y>
2. Brown G (2014) Defects of thiamine transport and metabolism. *J Inherit Metab Dis* 37:577–585. <https://doi.org/10.1007/s10545-014-9712-9>
3. Said HM, Nexø E (2018) Gastrointestinal Handling of Water-Soluble Vitamins. *Compr Physiol* 8:1291–1311. <https://doi.org/10.1002/cphy.c170054>

4. Rajgopal A, Edmondson A, Goldman ID, Zhao R (2001) SLC19A3 encodes a second thiamine transporter ThTr2. *Biochim Biophys Acta* 1537:175–178. [https://doi.org/10.1016/s0925-4439\(01\)00073-4](https://doi.org/10.1016/s0925-4439(01)00073-4)
5. Reidling JC, Lambrecht N, Kassir M, Said HM (2010) Impaired intestinal vitamin B1 (thiamin) uptake in thiamin transporter-2-deficient mice. *Gastroenterology* 138:1802–1809. <https://doi.org/10.1053/j.gastro.2009.10.042>
6. Boulware MJ, Subramanian VS, Said HM, Marchant JS (2003) Polarized expression of members of the solute carrier SLC19A gene family of water-soluble multivitamin transporters: implications for physiological function. *Biochem J* 376:43–48. <https://doi.org/10.1042/BJ20031220>
7. Kono S, Miyajima H, Yoshida K, et al (2009) Mutations in a thiamine-transporter gene and Wernicke's-like encephalopathy. *N Engl J Med* 360:1792–1794. <https://doi.org/10.1056/NEJMc0809100>
8. Overton E, Emelyanova A, Bunik VI (2025) Thiamine, gastrointestinal beriberi and acetylcholine signaling. *Front Nutr* 12:1541054. <https://doi.org/10.3389/fnut.2025.1541054>
9. Zhao R, Gao F, Goldman ID (2002) Reduced folate carrier transports thiamine monophosphate: an alternative route for thiamine delivery into mammalian cells. *Am J Physiol Cell Physiol* 282:C1512-1517. <https://doi.org/10.1152/ajpcell.00547.2001>
10. Zhao R, Gao F, Goldman ID (2001) Molecular cloning of human thiamin pyrophosphokinase. *Biochim Biophys Acta* 1517:320–322. [https://doi.org/10.1016/s0167-4781\(00\)00264-5](https://doi.org/10.1016/s0167-4781(00)00264-5)
11. Mayr JA, Freisinger P, Schlachter K, et al (2011) Thiamine pyrophosphokinase deficiency in encephalopathic children with defects in the pyruvate oxidation pathway. *Am J Hum Genet* 89:806–812. <https://doi.org/10.1016/j.ajhg.2011.11.007>
12. Nosaka K, Onozuka M, Nishino H, et al (1999) Molecular cloning and expression of a mouse thiamin pyrophosphokinase cDNA. *J Biol Chem* 274:34129–34133. <https://doi.org/10.1074/jbc.274.48.34129>
13. Boyle L, Wamelink MMC, Salomons GS, et al (2016) Mutations in TKT Are the Cause of a Syndrome Including Short Stature, Developmental Delay, and Congenital Heart Defects. *Am J Hum Genet* 98:1235–1242. <https://doi.org/10.1016/j.ajhg.2016.03.030>
14. Kochetov GA, Solovjeva ON (2014) Structure and functioning mechanism of transketolase. *Biochim Biophys Acta* 1844:1608–1618. <https://doi.org/10.1016/j.bbapap.2014.06.003>
15. McCool BA, Plonk SG, Martin PR, Singleton CK (1993) 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* 268:1397–1404
16. Lindhurst MJ, Fiermonte G, Song S, et al (2006) Knockout of Slc25a19 causes mitochondrial thiamine pyrophosphate depletion, embryonic lethality, CNS malformations, and anemia. *Proc Natl Acad Sci U S A* 103:15927–15932. <https://doi.org/10.1073/pnas.0607661103>
17. Naito E, Ito M, Takeda E, et al (1994) Molecular analysis of abnormal pyruvate dehydrogenase in a patient with thiamine-responsive congenital lactic acidemia. *Pediatr Res* 36:340–346. <https://doi.org/10.1203/00006450-199409000-00013>
18. Yap ZY, Strucinska K, Matsuzaki S, et al (2021) A biallelic pathogenic variant in the OGDH gene results in a neurological disorder with features of a mitochondrial disease. *J Inherit Metab Dis* 44:388–400.

<https://doi.org/10.1002/jimd.12248>

19. Yang N, Han L, Gu X, et al (2012) Analysis of gene mutations in Chinese patients with maple syrup urine disease. *Mol Genet Metab* 106:412–418. <https://doi.org/10.1016/j.ymgme.2012.05.023>
20. Chuang JL, Wynn RM, Moss CC, et al (2004) 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* 279:17792–17800. <https://doi.org/10.1074/jbc.M313879200>
21. Nghiê m HO, Bettendorff L, Changeux JP (2000) Specific phosphorylation of Torpedo 43K rapsyn by endogenous kinase(s) with thiamine triphosphate as the phosphate donor. *FASEB J Off Publ Fed Am Soc Exp Biol* 14:543–554. <https://doi.org/10.1096/fasebj.14.3.543>
22. Sasa M, Takemoto I, Nishino K, Itokawa Y (1976) The role of thiamine on excitable membrane of crayfish giant axon. *J Nutr Sci Vitaminol (Tokyo)* 22 SUPPL:21–24. https://doi.org/10.3177/jnsv.22.supplement_21
23. Whitfield KC, Bourassa MW, Adamolekun B, et al (2018) Thiamine deficiency disorders: diagnosis, prevalence, and a roadmap for global control programs. *Ann N Y Acad Sci* 1430:3–43. <https://doi.org/10.1111/nyas.13919>
24. European Food Safety Authority (2025) Food composition data. <https://www.efsa.europa.eu/en/data-report/food-composition-data>. Accessed 15 Sept 2025
25. 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>
26. Churuangsuk C, Catchpole A, Talwar D, et al (2024) Low thiamine status in adults following low-carbohydrate / ketogenic diets: a cross-sectional comparative study of micronutrient intake and status. *Eur J Nutr* 63:2667–2679. <https://doi.org/10.1007/s00394-024-03459-y>
27. Mataix J, Aranda P, Sánchez C, et al (2003) Assessment of thiamin (vitamin B1) and riboflavin (vitamin B2) status in an adult Mediterranean population. *Br J Nutr* 90:661–666. <https://doi.org/10.1079/BJN2003926>
28. Brown KH, Hess SY, Moore SE, et al (2025) Neglected micronutrients—considering a broader set of vitamins and minerals in public health nutrition programs worldwide: a narrative review. *Am J Clin Nutr* 122:680–694. <https://doi.org/10.1016/j.ajcnut.2025.06.030>
29. Anderson M, Mickelsen O, Keys A (1946) Diets and food items for the experimental production of thiamin deficiency in man. *J Am Diet Assoc* 22:1–7
30. Berger MM, Shenkin A, Schweinlin A, et al (2022) ESPEN micronutrient guideline. *Clin Nutr Edinb Scotl* 41:1357–1424. <https://doi.org/10.1016/j.clnu.2022.02.015>
31. Karakonstantis S, Galani D, Korela D, et al (2020) Missing the early signs of thiamine deficiency. A case associated with a liquid-only diet. *Nutr Neurosci* 23:384–386. <https://doi.org/10.1080/1028415X.2018.1507964>
32. Lonsdale D (2018) Thiamin. *Adv Food Nutr Res* 83:1–56. <https://doi.org/10.1016/bs.afnr.2017.11.001>
33. Mauermann ML, Staff NP (2026) Peripheral Neuropathy: A Review. *JAMA* 335:255–266. <https://doi.org/10.1001/jama.2025.19400>

34. Pereira AG, Cunha LN de P, Paiva SAR, et al (2025) An Overview of Beriberi. *Med Princ Pract Int J Kuwait Univ Health Sci Cent* 1–7. <https://doi.org/10.1159/000547719>
35. Inoue A, Itabashi K, Iwai T, et al (2021) Imaging findings of vitamin deficiencies: are they forgotten diseases? *BJR Open* 3:20210011. <https://doi.org/10.1259/bjro.20210011>
36. Coats D, Shelton-Dodge K, Ou K, et al (2012) Thiamine Deficiency in Cambodian Infants with and without Beriberi. *J Pediatr* 161:843–847. <https://doi.org/10.1016/j.jpeds.2012.05.006>
37. Smith TJ, Johnson CR, Koshy R, et al (2021) Thiamine deficiency disorders: a clinical perspective. *Ann N Y Acad Sci* 1498:9–28. <https://doi.org/10.1111/nyas.14536>
38. Li S, Xing C (2025) Wernicke encephalopathy: a mini review of the clinical spectrum, atypical manifestations, and diagnostic challenges. *Front Neurol* 16:1566366. <https://doi.org/10.3389/fneur.2025.1566366>
39. Brust JCM (2008) A 74-year-old man with memory loss and neuropathy who enjoys alcoholic beverages. *JAMA* 299:1046–1054. <https://doi.org/10.1001/jama.299.5.jrr80000>
40. Harper CG, Giles M, Finlay-Jones R (1986) Clinical signs in the Wernicke-Korsakoff complex: a retrospective analysis of 131 cases diagnosed at necropsy. *J Neurol Neurosurg Psychiatry* 49:341–345. <https://doi.org/10.1136/jnnp.49.4.341>
41. Lindboe CF, Løberg EM (1989) Wernicke's encephalopathy in non-alcoholics. An autopsy study. *J Neurol Sci* 90:125–129. [https://doi.org/10.1016/0022-510x\(89\)90095-6](https://doi.org/10.1016/0022-510x(89)90095-6)
42. Mancinelli R, Ceccanti M (2009) Biomarkers in alcohol misuse: their role in the prevention and detection of thiamine deficiency. *Alcohol Alcohol Oxf Oxf* 44:177–182. <https://doi.org/10.1093/alcalc/agn117>
43. Kerns JC, Arundel C, Chawla LS (2015) Thiamin Deficiency in People with Obesity. *Adv Nutr* 6:147–153. <https://doi.org/10.3945/an.114.007526>
44. Salvatori G, Mondì V, Piersigilli F, et al (2016) Thiamine Deficiency in a Developed Country: Acute Lactic Acidosis in Two Neonates Due to Unsupplemented Parenteral Nutrition. *JPEN J Parenter Enteral Nutr* 40:886–889. <https://doi.org/10.1177/0148607114568316>
45. Ziegler D, Reiners K, Strom A, Obeid R (2023) Association between diabetes and thiamine status - A systematic review and meta-analysis. *Metabolism* 144:155565. <https://doi.org/10.1016/j.metabol.2023.155565>
46. da Silva JSV, Seres DS, Sabino K, et al (2020) ASPEN Consensus Recommendations for Refeeding Syndrome. *Nutr Clin Pract* 35:178–195. <https://doi.org/10.1002/ncp.10474>
47. Shim J-S, Shim SY, Cha H-J, et al (2022) Association between Ultra-processed Food Consumption and Dietary Intake and Diet Quality in Korean Adults. *J Acad Nutr Diet* 122:583–594. <https://doi.org/10.1016/j.jand.2021.07.012>
48. European Food Safety Authority (2016) Dietary reference values for thiamin. *EFSA J* 14:4653. <https://doi.org/10.2903/j.efsa.2016.4653>
49. Mertens E, Colizzi C, Peñalvo JL (2022) Ultra-processed food consumption in adults across Europe. *Eur J Nutr* 61:1521–1539. <https://doi.org/10.1007/s00394-021-02733-7>
50. O'Brien M, Quirke R, Gowan R, McNicholas F (2025) Prevalence of thiamine deficiency in anorexia nervosa: a systematic review and narrative synthesis. *Eur Child Adolesc Psychiatry* 34:1281–1293.

<https://doi.org/10.1007/s00787-024-02576-7>

51. Karimi Behnagh A, Eghbali M, Abdolmaleki F, et al (2024) Pre- and Post-surgical Prevalence of Thiamine Deficiency in Patients Undergoing Bariatric Surgery: a Systematic Review and Meta-analysis. *Obes Surg* 34:653–665. <https://doi.org/10.1007/s11695-023-06896-6>
52. Oudman E, Wijnia JW, Oey M, et al (2019) Wernicke's encephalopathy in hyperemesis gravidarum: A systematic review. *Eur J Obstet Gynecol Reprod Biol* 236:84–93. <https://doi.org/10.1016/j.ejogrb.2019.03.006>
53. Elmadfa I, Majchrzak D, Rust P, Genser D (2001) The thiamine status of adult humans depends on carbohydrate intake. *Int J Vitam Nutr Res Int Z Vitam- Ernährungsforschung J Int Vitaminol Nutr* 71:217–221. <https://doi.org/10.1024/0300-9831.71.4.217>
54. Clark AF (2024) Use of Thiamine Supplementation in Pregnant Women Diagnosed With Hyperemesis Gravidarum and Wernicke Encephalopathy. *Nurs Womens Health* 28:222–226. <https://doi.org/10.1016/j.nwh.2024.02.003>
55. Saka Y, Naruse T, Kato A, et al (2018) Thiamine status in end-stage chronic kidney disease patients: a single-center study. *Int Urol Nephrol* 50:1913–1918. <https://doi.org/10.1007/s11255-018-1974-y>
56. Lautenschläger T, Neinhuis C, Monizi M, et al (2017) Edible insects of Northern Angola. *Afr Invertebr* 58:55–82. <https://doi.org/10.3897/afrinvertebr.58.21083>
57. Nishimune T, Watanabe Y, Okazaki H, Akai H (2000) Thiamin is decomposed due to Anaphe spp. entomophagy in seasonal ataxia patients in Nigeria. *J Nutr* 130:1625–1628. <https://doi.org/10.1093/jn/130.6.1625>
58. Vid Vičič, Andreja 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 (2021) The PRISMA 2020 statement: an updated guideline for reporting systematic reviews. *Syst Rev* 10:89. <https://doi.org/10.1186/s13643-021-01626-4>
60. Tasevska N, Runswick SA, McTaggart A, Bingham SA (2008) Twenty-four-hour urinary thiamine as a biomarker for the assessment of thiamine intake. *Eur J Clin Nutr* 62:1139–1147. <https://doi.org/10.1038/sj.ejcn.1602829>
61. Jones KS, Parkington DA, Cox LJ, Koulman A (2021) Erythrocyte transketolase activity coefficient (ETKAC) assay protocol for the assessment of thiamine status. *Ann N Y Acad Sci* 1498:77–84. <https://doi.org/10.1111/nyas.14547>
62. Lu J, Frank EL (2008) Rapid HPLC Measurement of Thiamine and Its Phosphate Esters in Whole Blood. *Clin Chem* 54:901–906. <https://doi.org/10.1373/clinchem.2007.099077>
63. Talwar D, Davidson H, Cooney J, St JO'Reilly D (2000) 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* 46:704–710
64. Jones KS, Parkington DA, Bourassa MW, et al (2023) Protocol and application of basal erythrocyte transketolase activity to improve assessment of thiamine status. *Ann N Y Acad Sci* 1521:104–111. <https://doi.org/10.1111/nyas.14962>

65. Herzog R, Álvarez-Pasquin MJ, Díaz C, et al (2013) Are healthcare workers' intentions to vaccinate related to their knowledge, beliefs and attitudes? a systematic review. *BMC Public Health* 13:154. <https://doi.org/10.1186/1471-2458-13-154>
66. Ortega RM, Martínez RM, Andrés P, et al (2004) Thiamin status during the third trimester of pregnancy and its influence on thiamin concentrations in transition and mature breast milk. *Br J Nutr* 92:129–135. <https://doi.org/10.1079/BJN20041153>
67. Sánchez DJ, Murphy MM, Bosch-Sabater J, Fernández-Ballart J (1999) 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* 53:27–38. <https://doi.org/10.1038/sj.ejcn.1600674>
68. Malara M, Hübner-Wozniak E, Lewandowska I (2013) Assessment of intake and nutritional status of vitamin B1, B2, and B6 in men and women with different physical activity levels. *Biol Sport* 30:117–123. <https://doi.org/10.5604/20831862.1044430>
69. Hötzel D, Bitsch R (1976) Thiamine status of human subjects, estimated by biochemical methods. *J Nutr Sci Vitaminol (Tokyo)* 22:41–45. https://doi.org/10.3177/jnsv.22.Supplement_41
70. Fabian E, Majchrzak D, Dieminger B, et al (2008) Influence of probiotic and conventional yoghurt on the status of vitamins B1, B2 and B6 in young healthy women. *Ann Nutr Metab* 52:29–36. <https://doi.org/10.1159/000114408>
71. Herve C, Beyne P, Lettéron P, Delacoux E (1995) Comparison of erythrocyte transketolase activity with thiamine and thiamine phosphate ester levels in chronic alcoholic patients. *Clin Chim Acta Int J Clin Chem* 234:91–100. [https://doi.org/10.1016/0009-8981\(94\)05980-7](https://doi.org/10.1016/0009-8981(94)05980-7)
72. Jermendy G (2006) Evaluating thiamine deficiency in patients with diabetes. *Diab Vasc Dis Res* 3:120–121. <https://doi.org/10.3132/dvdr.2006.014>
73. Boni L, Kieckens L, Hendriks A (1980) An Evaluation of a Modified Erythrocyte Transketolase Assay for Assessing Thiamine Nutritional Adequacy. *J Nutr Sci Vitaminol (Tokyo)* 26:507–514. <https://doi.org/10.3177/jnsv.26.507>
74. Mancinelli R, Ceccanti M, Guiducci MS, et al (2003) Simultaneous liquid chromatographic assessment of thiamine, thiamine monophosphate and thiamine diphosphate in human erythrocytes: a study on alcoholics. *J Chromatogr B* 789:355–363. [https://doi.org/10.1016/S1570-0232\(03\)00139-9](https://doi.org/10.1016/S1570-0232(03)00139-9)
75. Verstraete J, Stove C (2021) Volumetric absorptive microsampling (VAMS) as a reliable tool to assess thiamine status in dried blood microsamples: a comparative study. *Am J Clin Nutr* 114:1200–1207. <https://doi.org/10.1093/ajcn/nqab146>
76. Körner RW, Vierzig A, Roth B, Müller C (2009) 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* 877:1882–1886. <https://doi.org/10.1016/j.jchromb.2009.05.013>
77. Lynch PLM, Trimble ER, Young IS (1997) High-performance liquid chromatographic determination of thiamine diphosphate in erythrocytes using internal standard methodology. *J Chromatogr B Biomed Sci App* 701:120–123. [https://doi.org/10.1016/S0378-4347\(97\)00353-8](https://doi.org/10.1016/S0378-4347(97)00353-8)

78. Tallaksen CME, Bøhmer T, Bell H, Karlsen J (1991) Concomitant determination of thiamin and its phosphate esters in human blood and serum by high-performance liquid chromatography. *J Chromatogr B Biomed Sci App* 564:127–136. [https://doi.org/10.1016/0378-4347\(91\)80075-N](https://doi.org/10.1016/0378-4347(91)80075-N)
79. Schrijver J, Speek AJ, Klosse JA, et al (1982) 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* 19:52–56. <https://doi.org/10.1177/000456328201900111>
80. Johnson CR, Fischer PR, Thacher TD, et al (2019) Thiamin deficiency in low- and middle-income countries: Disorders, prevalences, previous interventions and current recommendations. *Nutr Health* 25:127–151. <https://doi.org/10.1177/0260106019830847>
81. Rakotoambinina B, Hiffler L, Gomes F (2021) Pediatric thiamine deficiency disorders in high-income countries between 2000 and 2020: a clinical reappraisal. *Ann N Y Acad Sci* 1498:57–76. <https://doi.org/10.1111/nyas.14669>
82. Passarelli S, Free CM, Shepon A, et al (2024) Global estimation of dietary micronutrient inadequacies: a modelling analysis. *Lancet Glob Health* 12:e1590–e1599. [https://doi.org/10.1016/S2214-109X\(24\)00276-6](https://doi.org/10.1016/S2214-109X(24)00276-6)
83. Hebert JR, Clemow L, Pbert L, et al (1995) Social desirability bias in dietary self-report may compromise the validity of dietary intake measures. *Int J Epidemiol* 24:389–398. <https://doi.org/10.1093/ije/24.2.389>
84. Gallant J, Chan K, Green TJ, et al (2021) Low-dose thiamine supplementation of lactating Cambodian mothers improves human milk thiamine concentrations: a randomized controlled trial. *Am J Clin Nutr* 114:90–100. <https://doi.org/10.1093/ajcn/nqab052>
85. Lonsdale D (2015) Sudden infant death syndrome and abnormal metabolism of thiamin. *Med Hypotheses* 85:922–926. <https://doi.org/10.1016/j.mehy.2015.09.009>
86. European Commission. Commission Directive 2006/125/EC of 5 December 2006 on processed cereal-based foods and baby foods for infants and young children (Text with EEA relevance). *OJ L* 339, 6.12.2006, pp. 16–35. Brussels: European Union.
87. European Commission. Commission Delegated Regulation (EU) 2016/127 of 25 September 2015 supplementing Regulation (EU) No 609/2013 of the European Parliament and of the Council as regards the specific compositional and information requirements for infant formula and follow-on formula and as regards requirements on information relating to infant and young child feeding (Text with EEA relevance). *OJ L* 25, 2.2.2016, pp. 1–29. Brussels: European Union.
88. Bailey AL, Finglas PM, Wright AJA, Southon S (1994) Thiamin intake, erythrocyte transketolase (EC 2.2.1.1) activity and total erythrocyte thiamin in adolescents. *Br J Nutr* 72:111–125. <https://doi.org/10.1079/BJN19940014>
89. Falcão RCTM de A, Lyra C de O, Morais CMM de, et al (2019) Processed and ultra-processed foods are associated with high prevalence of inadequate selenium intake and low prevalence of vitamin B1 and zinc inadequacy in adolescents from public schools in an urban area of northeastern Brazil. *PloS One* 14:e0224984. <https://doi.org/10.1371/journal.pone.0224984>
90. Rehm J, Anderson P, Barry J, et al (2015) Prevalence of and potential influencing factors for alcohol dependence in Europe. *Eur Addict Res* 21:6–18. <https://doi.org/10.1159/000365284>

91. Bichkaeva FA, Tkachev AV, Boiko ER, et al (2003) Vitamin B1 Levels in Residents of the European North. *Hum Physiol* 29:119–121. <https://doi.org/10.1023/A:1022073123466>
92. Bojko ER (2001) Thiamine Status in Inhabitants of the Russian European North. *Int J Circumpolar Health* 60:466–468. <https://doi.org/10.1080/25761900.2022.12220617>
93. Whitfield KC, Smith TJ, Rohner F, et al (2021) Thiamine fortification strategies in low- and middle-income settings: a review. *Ann N Y Acad Sci* 1498:29–45. <https://doi.org/10.1111/nyas.14565>
94. Vičič V, Mikuš RP, Kugler S, et al (2023) Vitamin D fortification of eggs alone and in combination with milk in women aged 44-65 years: Fortification model and economic evaluation. *Zdr Varst* 62:30–38. <https://doi.org/10.2478/sjph-2023-0005>
95. Churuangsuk C, Griffiths D, Lean MEJ, Combet E (2019) Impacts of carbohydrate-restricted diets on micronutrient intakes and status: A systematic review. *Obes Rev Off J Int Assoc Study Obes* 20:1132–1147. <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
97. Shakory S (2020) Thiamine in the management of alcohol use disorders. *Can Fam Physician* 66:165–166

Figures

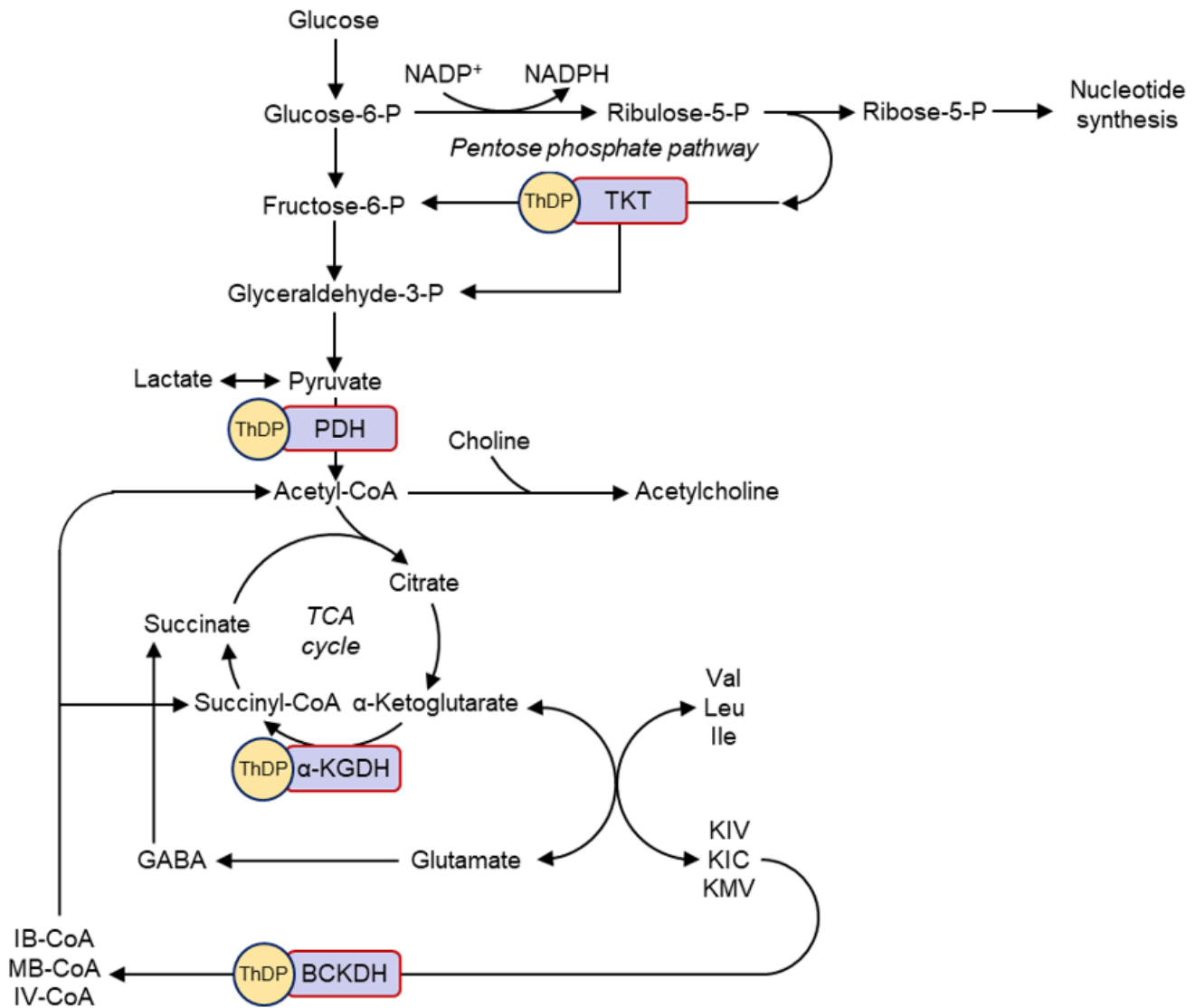


Figure 1

Role of ThDP in energy metabolism. Abbreviations: α-KGDH: α-ketoglutarate dehydrogenase complex, BCKDH: branched-chain α-keto acid dehydrogenase complex, KIC: α-ketoisocaproate, KIV: α-ketoisovalerate, KMV: α-keto β-methylvalerate, IB-CoA: isobutyryl-CoA, Ile: isoleucine; Leu: Leucine, MB-CoA: methylbutyryl-CoA, IV-CoA: isovaleryl-CoA, PDH: pyruvate dehydrogenase complex, TCA: tricarboxylic acid, TKT: transketolase, Val: Valine.

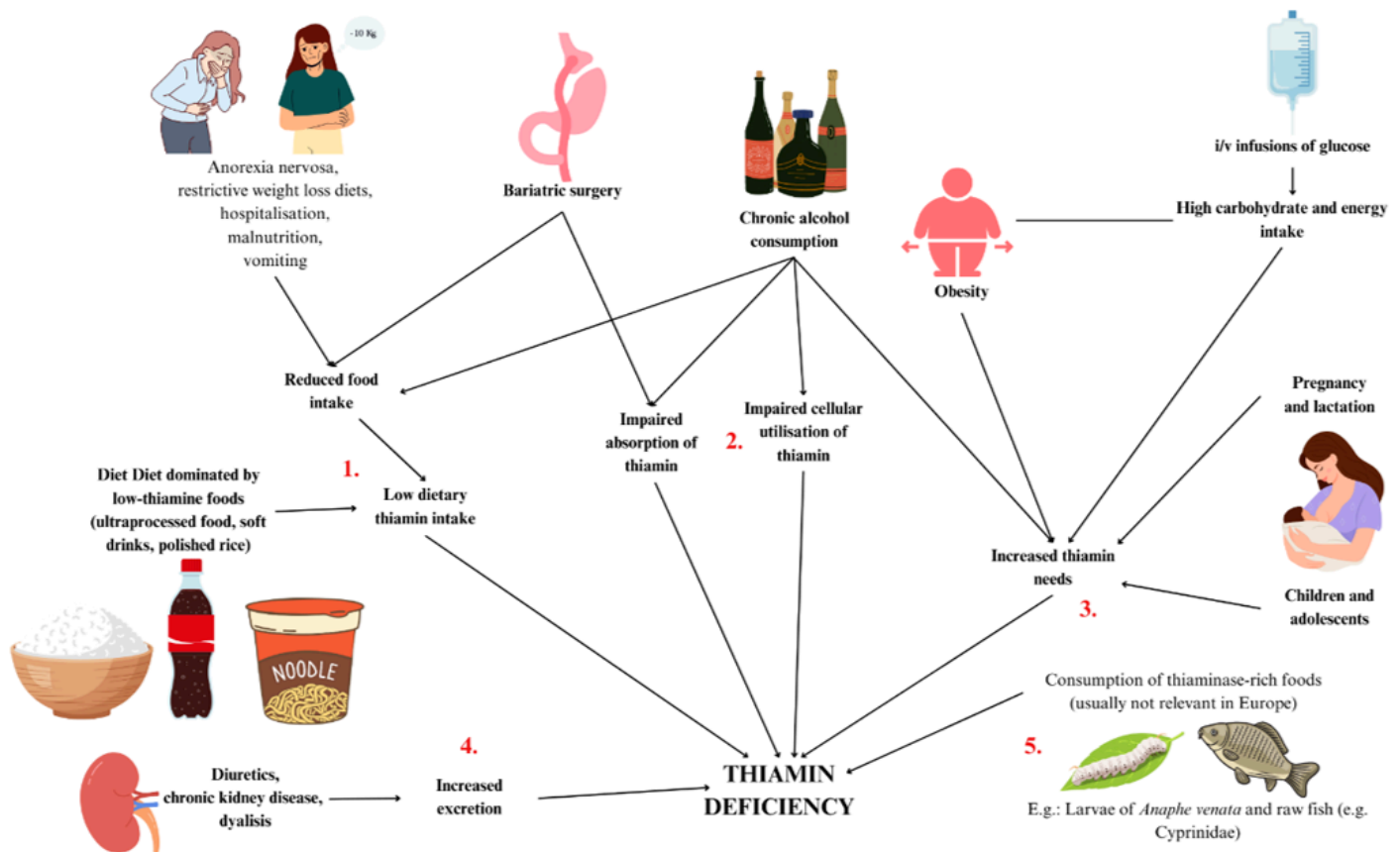


Figure 2

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. [42]. 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.

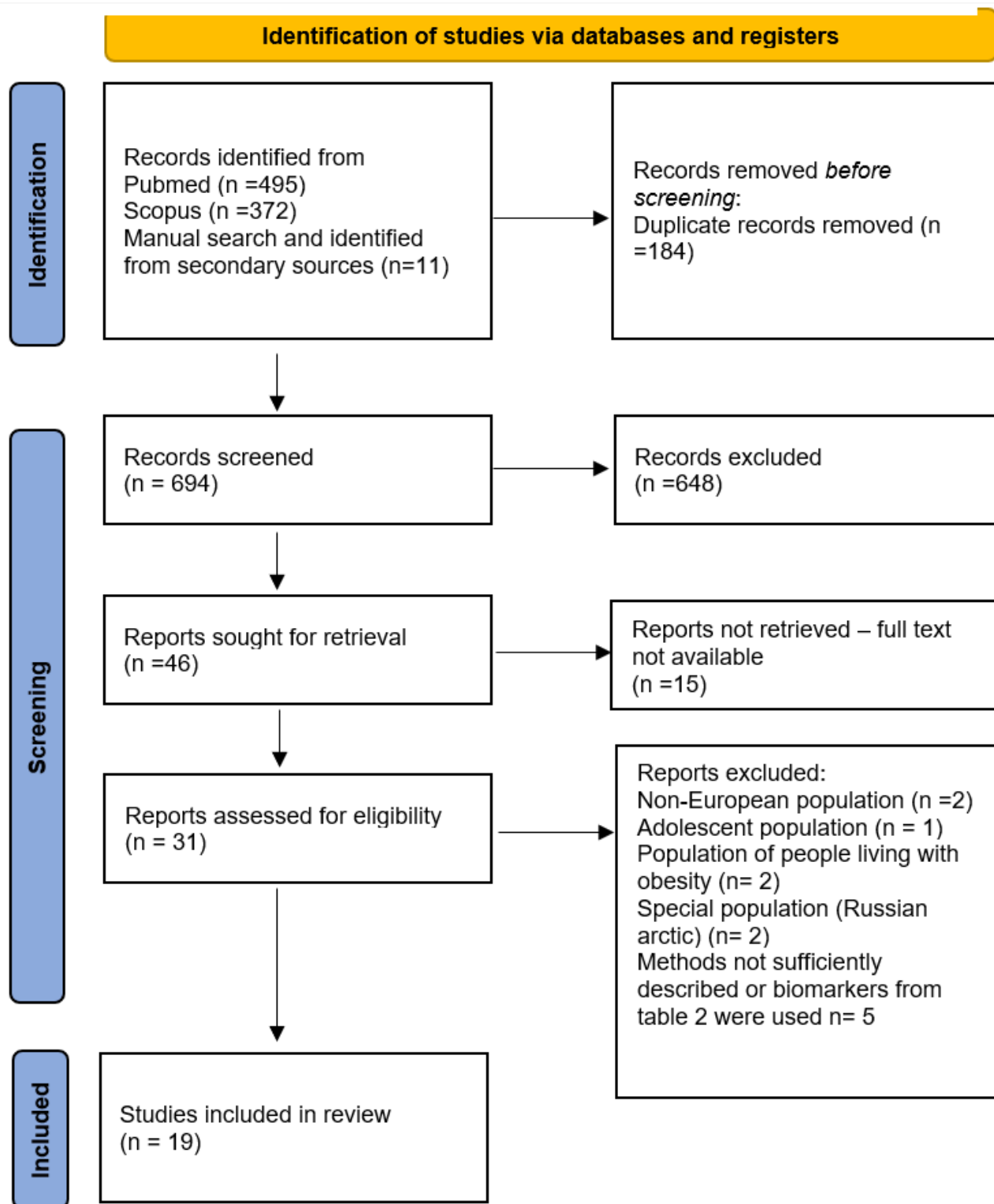


Figure 3

Study flow diagram. Made according to PRISMA guidelines [59].

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- [24.2.2026SUPP.docx](#)
- [PRISMA2020checklist.docx](#)