Eieren en chronische ziekten

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1 Introduction

This background document belongs to the advisory report *Dutch dietary guidelines: dietary protein sources and dietary patterns 2025 (DDG2025).*[1] It describes the methodology for the search, selection and evaluation of the literature regarding the effects and associations of egg consumption with health outcomes. It also describes the scientific evidence on this topic and the conclusions that have been drawn by the council's committee on Nutrition. These conclusions form the basis for the derivation of the 2025 dietary advices on eggs.

The committee on Nutrition has set up a working group to prepare this background document. The committee takes final responsibility for the content of the background document. A list of the committee members can be found in the advisory report.[1]
A list of the working group members can be found in Annex A of this background document.

1.1 Definition of eggs

Chicken egg consumption is the focus of this document. The committee used a weight of 50 grams as a reference for one medium egg.[2] Eggs belong to protein-providing staple foods, but are also a well-known source of dietary cholesterol (approximately 200 milligrams of cholesterol per egg).[3]

1.2 Intake of eggs in the Netherlands

According to the most recent Dutch Food Consumption Survey (DNFCS; 2019-2021),[4,5] Dutch adults consume eggs on 2 days per week on average. On such consumption days, the median consumption is 50 g (P5-P95: 9-135 g). The mean daily consumption of eggs is 18 g/d. This concerns eggs eaten as such or used in recipes; eggs incorporated into products such as cakes, pastries, pastas, sauces or deserts, were not surveyed. Women eat almost as many eggs as men. Egg intake increased compared to the DNFCS 2012-2016.[6]

2 Methodology

Below, the methodology used for the evaluation of the evidence is presented. In addition, a description is given of the selection of literature on the topic of eggs in relation to health outcomes.

2.1 Health outcomes

The committee focused on the health outcomes described below.

Chronic diseases:

Similar to the approach used for the Dutch dietary guidelines 2015 (DDG2015),[7] the committee focused on the most common chronic diseases (that are potentially nutrition related) in the Netherlands.[8,9] These include: coronary heart disease (CHD), stroke, heart failure, type 2 diabetes, cancer subtypes (colorectal cancer and breast cancer), obesity (in some evaluations combined with overweight), depression, chronic obstructive pulmonary disease (COPD) and dementia. Furthermore, evidence for total cardiovascular disease (CVD) and total cancer was included, but only in case the committee expected (based on the quick scans, described below) that such evidence would provide additional insights (on top of the evidence focused on the afore and below listed outcomes) that could be of added value for drawing up or changing a dietary guideline. The committee preferred to use evidence for CVD subtypes and cancer subtypes because these may have different aetiologic pathways related to different dietary exposures. Total CVD and total cancer comprise different subtypes and therefore provide less specific insights in the aetiology underlying the relationships between dietary factors and chronic diseases.

The committee focused on studies that addressed primary prevention of chronic diseases. This means that the relationship between a dietary factor and development of a chronic disease was investigated in people who did not have the disease at the start of the study. Moreover, in general, the committee preferred to base its evaluation on studies that addressed non-fatal events or the combined outcome of fatal and non-fatal events (thus: not solely focussed on fatal events) because the availability and quality of care may play a larger role in the outcome of fatal, compared to non-fatal events. Exceptions to this were made in case there were aetiological reasons to specifically address fatal events (e.g. for fish and fatal cardiovascular outcomes).

Causal risk factors and other short-term outcomes:
These include the following: blood pressure (systolic and diastolic), low-density lipoprotein (LDL) cholesterol, body weight, glycated haemoglobin (HbA1c) and estimated glomerular filtration rate (eGFR).

Causal risk factors are thought to capture the causal pathway that leads to the disease outcome and can be seen as replacement endpoints for the disease of interest.[10] Because of this, the committee considers that an effect on a causal risk factor will also lead to an effect on chronic disease risk. An example is LDL cholesterol as causal risk factor for CHD and stroke. The advantage of using causal risk factors (and other short-term outcomes) in experimental studies is that they usually need considerably fewer participants and shorter study durations (usually several weeks to months) than studies investigating morbidity or mortality outcomes. For instance, dietary effects on LDL cholesterol can be identified in just a few weeks, compared to several years for CHD. Similar to the approach used in 2015, blood pressure, LDL cholesterol and body weight were selected as causal risk factors. As explained in the DDG2015 background document on methodology,[7] these factors have been shown to have a causal relationship with at least one of the following chronic diseases: CHD, stroke, heart failure and type 2 diabetes. More recent evidence confirms the causality, presented below.

Blood pressure: A meta-analysis of 48 randomised controlled trials (RCTs) confirmed that multiple blood pressure lowering treatments, including more versus less intensive treatments, reduce the risk of cardiovascular events, including CHD and stroke. This was found in people with and without CVD.[11] Moreover, Mendelian randomisation studies support that blood pressure is causally associated with the risk of CHD and stroke.[12,13]

LDL cholesterol: Recent reports confirmed that numerous and different types of studies, including prospective cohort studies, RCTs and Mendelian randomisation studies, have convincingly shown that higher LDL cholesterol causes cardiovascular events, such as CHD.[14] The evidence from RCTs is based on various interventions performed among both primary and secondary prevention trials: A meta-analysis of 49 RCTs showed that the use of statin and various non-statin therapies, including diet, that reduce LDL cholesterol, reduce the risk of cardiovascular events.[15] In line with the committee, the European Food Safety Authority (EFSA) also acknowledges that reducing blood LDL cholesterol (by dietary modification and drugs) would generally reduce the risk of development of CHD.[16]

Body weight: Recent reports confirmed the importance of weight loss via various interventions for the prevention of type 2 diabetes, based on RCTs.[17,18] Moreover, the causal role of both general and central adiposity, indicated by body mass index and waist-to-hip ratio respectively, in the development of type 2 diabetes and other chronic diseases has been confirmed in Mendelian randomisation studies.[19] Besides body weight, the committee also searched for literature with waist circumference as outcome, because this may also reflect the causal pathway from adiposity to type 2 diabetes. However, very few relevant studies with this endpoint were found, and that evidence did not contribute to the guidelines.

HbA1c and eGFR: These factors were not yet included in 2015, and were added for the 2025 advisory report because these are important diagnostic indicators of chronic diseases (elevated HbA1c for type 2 diabetes and long-term decline in eGFR for chronic kidney disease).[20-22] However, in practice, insufficient research focussed on these factors was found to include in the committee's evaluations.

Other outcomes:
These include the following: all-cause mortality, quality of life, perceived health and fertility.
The committee additionally searched for evidence focused on quality of life, perceived health and fertility, which could be considered as supportive evidence for the guidelines. However, in practice, insufficient evidence on these outcomes, which fulfilled the inclusion criteria of the committee, was found.
Evidence focused on the all-cause mortality outcome was additionally evaluated, but only in case the committee expected (based on the quick scans, described below) that such evidence would provide additional insights that could be of added value for drawing up or changing a dietary guideline. The reason for this is similar to that described above, for the total CVD and total cancer outcomes.

2.2 Type of studies

The committee evaluated the state of science for each dietary factor – health outcome combination, based on meta-analyses (MAs) and pooled analyses. In MAs, reported results of multiple individual studies are combined and analysed. In pooled analyses, individual participant data of multiple studies are combined and analysed.
The committee selected MAs and pooled analyses of the following type of studies:

- Randomised controlled trials (RCTs) into effects of dietary factors on the incidence of morbidity/mortality due to a disease;
- RCTs into effects of dietary factors on causal risk factors;

- Prospective cohort studies into associations of dietary factors with morbidity or mortality due to disease.

Substitution analyses
In some of the background documents, substitution analyses were also included as a part of the evaluation. In RCTs, substitution was based on comparisons of the effects of consumption of two diets, each with another dietary protein source (keeping the rest of the diet as similar as possible). In observational cohort studies, substitution was hypothetically investigated by statistical modelling based on (baseline) dietary intake data. Because substitution did not actually take place in observational cohort studies, the committee interpreted the evidence as associations for dietary protein source 1 versus dietary protein source 2 with disease outcomes.

Mendelian randomisation studies
The committee additionally considered evidence from Mendelian randomisation studies, to serve as potential supportive evidence for certain guidelines. In Mendelian randomisation studies, genetic variants are used to investigate causal associations between dietary intake and risk of chronic diseases.[23] Only for the topic of dairy products, relevant Mendelian randomisation studies were found, as further explained in the background document on dairy products and chronic diseases.[24]

2.3 Quick scans

For efficiency reasons, the committee decided to make use of recent reports of other organisations where possible. To this end, quick scans were performed at the start of the advisory trajectory (2023). These quick scans were aimed at summarising the state of science in an efficient way and to identify aspects on which the dietary guidelines may need to be updated.

To identify differences and similarities compared to the DDG2015, the guidelines for each specific dietary factor (e.g. fruits and vegetables, fats and oils or dairy products) were compared to the recent advices on these dietary factors of the *Nordic Nutrition Recommendations 2023* (NNR2023),[25] and the *Dietary Guidelines for Americans 2020-2025* (DGA2020).[26] Moreover, the conclusions and findings regarding the relationships of dietary factors with health outcomes reported in the DDG2015 were compared to conclusions and findings reported in the background documents of the NNR2023 and of the DGA2020. In addition, and based on the above referred documents, the committee identified topics for which there may now be sufficient evidence to draw conclusions on, where this was not yet possible in 2015.

Based on the quick scans, the committee decided to focus its evaluation of eggs in particular on the following:

- The committee noted that there is now substantially more research from cohort studies that addressed the associations between egg consumption and risks of chronic diseases. Therefore, the committee aimed to evaluate whether it would be possible to derive a (quantitative) dietary guideline for eggs based on the recent state of science.
- In addition, the committee noted that for the following health outcomes, there is new and relevant literature that may be leading or supportive for the dietary guideline on eggs: CHD, stroke, heart failure, type 2 diabetes, LDL cholesterol and blood pressure. Therefore, the committee focused its evaluation of eggs on these health outcomes.

2.4 Consulted literature

For the evaluation of egg consumption and its effects or associations with the selected health outcomes, the committee used the following sources of literature:

- The committee used MAs addressed in the NNR2023 advisory report,[25] and in the NNR2023 background documents on eggs,[27] that include studies published until 13 September 2021 (PubMed) and 29 October 2021 (Web of Science).
- The committee additionally used pooled analyses of cohorts that contributed to the European Prospective Investigation into Cancer and nutrition (EPIC). EPIC publications were searched by the committee via a systematic literature search (search last updated on 8 July 2024). The committee used such pooled analyses in case these were not included in the above referred MAs, which was not the case for studies on egg consumption and health outcomes.
- For a selection of dietary factors, the committee additionally performed systematic literature searches in PubMed and Scopus to search for more recent literature (than NNR). This was only done in case the committee expected such a search would yield additional information that could potentially impact the conclusions drawn by the committee. This was applicable to the following situations:
  - For dietary factors on which, after publication of the recent reports (referred to above), there are scientific advances that could impact the dietary guidelines.
  - For dietary factors on which there is relatively much scientific discussion and/or no consensus on the health effects/associations in the field.

  For the topic of egg consumption, the committee decided that a search for more recent literature was not required.

From all MAs and pooled analyses retrieved via aforementioned sources, the most recent and complete MAs and pooled analyses that fit within the inclusion criteria of the

committee (i.e. based on selected study designs and health outcomes) and which together gave the most comprehensive overview of the studies available on each dietary factor – health outcome combination were selected for the committee's evaluation.

2.5 Evaluation of evidence

For each dietary factor - health outcome combination, an evaluation of the state of science was performed, further explained below.

2.5.1 Drawing conclusions based on a decision tree

The findings from prospective cohort studies and RCTs were separately evaluated by the committee. Per dietary factor – health outcome combination, a table summarising the main characteristics and results of the major MA(s) and/or pooled analyse(s) that contributed to the evaluation was presented. Below the summary table, the committee gave the conclusion, for which there are six fixed options:

- There is an association/effect with a strong level of evidence;
- There is an association/effect with a limited level of evidence;
- An association/effect is unlikely;
- The evidence is inconclusive;
- The evidence is contradictory;
- There is too little research.

The conclusions on (the certainty of) the evidence regarding the effect or association between egg consumption and health outcomes were based on the number of studies, number of participants and number of cases that contributed to the evaluation.
Also, the committee took the quality of the studies, in particular the risk of bias, and the heterogeneity in findings between studies, into account. The committee used the decision tree (presented in Annex B) as a guidance tool to support consistency in drawing conclusions. This decision tree was also applied by previous committees of the Health Council,[28-30] and includes elements that are also present in other evaluation methods for determining the quality and certainty of scientific evidence, such as GRADE (for example: heterogeneity and risk of bias).[31] The formulation of conclusions was different for RCTs than for cohort studies: RCTs allowed statements about effects (causality) to be made, whereas cohort studies only allowed statements about associations to be made.
The conclusion was followed by a text in which the conclusion was explained and in which the committee presented the publications assessed in connection with the conclusion. This text was accompanied by a table that showed relevant details related to the included studies.

2.5.2 Extensiveness of evaluation

Per dietary factor – health outcome combination, the committee decided on the level of detail of its evaluation. Extensive evaluations were performed for dietary factor – health outcome combinations of which conclusions could potentially be leading for drawing up a new guideline or changing an existing guideline. Remaining dietary factor – health outcome combinations, of which conclusions unlikely changed compared to 2015 or could only be supportive to a guideline, were evaluated less extensively. The differences between conclusions that are leading and those that are supportive for drawing up a guideline are described in Chapter 2 of the advisory report.[1]
In extensive evaluations, the committee drew formal conclusions based on the decision tree, and presented the evaluation according to the format described in Section 2.5.1. In less extensive evaluations, the committee did not draw such formal conclusions. Instead, the state of science was briefly (with less details) summarised in text with an accompanying table. Such a less extensive evaluation was in particular done to check whether potential effects or associations with remaining subtypes of exposures and remaining health outcomes were indeed in a supportive direction, or at least not in an opposite direction as compared to the conclusions on the exposure subtypes and outcomes that were extensively evaluated.

The committee performed, in principle, only (extensive and less extensive) evaluations of dietary factor – health outcome relationships when at least 5 individual studies of a specific study type were included in the selected MAs and/or pooled analyses.
This was done because at least 5 studies are needed to qualify the evidence as strong, and because only conclusions with a strong level of evidence are considered for drawing up the dietary guideline (see Chapter 2 of the advisory report[1]). The committee could decide to make exceptions in this approach, based on expert judgement of the evidential value, public interest and extent of scientific discussion.

For the topic of egg consumption, the committee noted, based on the quick scans and selected recent literature, that the evidence could potentially be (new and) leading for drawing up an egg guideline for the following topics, which were therefore extensively evaluated:

- Based on prospective cohort studies:
  - Consumption of eggs and the risk of CHD;
  - Consumption of eggs and the risk of stroke;
  - Consumption of eggs and the risk of type 2 diabetes.
- Based on RCTs:
  - Consumption of eggs and effects on LDL-cholesterol;
  - Consumption of eggs and effects on blood pressure.

Remaining topics were less extensively evaluated.

2.6 Formulating recommendations on eggs

Effects and associations of eggs with chronic disease risk are described in Chapter 3 of this background document. The committee used the totality of conclusions with strong evidence described in this document, together with conclusions based on substitution of dietary protein sources,[32] environmental and chemical food safety aspects,[33,34] for formulating the recommendations on eggs. The (methodology used for the) translation of the totality of evidence into dietary guidelines and recommendations is described in the advisory report.[1]

3 Evaluation of the evidence

This chapter describes the evaluation of the scientific evidence on the associations or effects of consumption of eggs with risks of chronic diseases or causal risk factors. Evaluations are grouped according to health outcome.

3.1 Coronary heart disease

The DDG2015 concluded that an association between egg consumption and risk of CHD is unlikely.[2] This was based on 2 MAs of 5 to 9 prospective cohort studies. There are now substantially more prospective cohort studies available on this topic. Therefore, the committee performed an extensive evaluation.

Summary of evidence for the association between egg consumption and risk of coronary heart disease

Aspect	Explanation
Available studies	One meta-analysis of 28 cohorts[35]
Heterogeneity	Moderate[35]
Strength of the association	• RR (95%CI) for highest versus lowest category of egg consumption: 0.97 (0.91, 1.04);[35] • RR (95%CI) per 1 egg per day increase: 0.96 (0.91, 1.03).[35]
Consumption level examined	• Highest – lowest category of egg consumption: >2 eggs to >7 eggs/week – no egg;[35] • Interquartile ranges (P25 to P75) in g/d: ♂ 7 to 27; ♀ 7 to 24.[36]
Study population	Europe, United States of America, Asia[35]

Conclusion: The evidence for the association between egg consumption and risk of coronary heart disease is inconclusive.
Evidence level: not applicable

Explanation

The committee used the MA of Drouin – Chartier et al.[35] for its evaluation of the association between egg consumption and incidence of (both fatal and non-fatal) CHD. The findings of this MA were based on 17 reports, that together included 28 prospective cohort studies, of which 14 were European (including the publication of Key et al.,[36] a pooled analysis of 9 cohorts that contributed to EPIC, counted as 9 cohorts by the committee). The follow-up time of the included studies varied from 9 to 32 years.

The definitions of eggs consumption in the included studies was not reported by Drouin – Chartier et al. Categories of egg consumption that were compared in the cohorts that contributed to this MA varied, for instance: <1 egg/month vs. ≥1 egg/d (a relatively large difference in exposure), and <1 egg/month vs. ≥2 eggs/week (a relatively small difference in exposure). In the pooled analysis of EPIC studies, the median (P25, P75) consumption level of eggs at baseline was 16 (7, 27) g/d in men and 15 (7,24) g/d in women.

The consumption of eggs was not statistically significantly associated with risk of CHD, both in dose-response and highest versus lowest category of intake analyses (Table 1). There was moderate heterogeneity between studies, which was not explained by the authors. No subgroup analyses were performed and no forest plots were presented. Drouin-Chartier et al. also addressed analyses into the health outcome CVD (with rather comparable results in the main analyses as for the outcome CHD), and these analyses suggested that geographical location was a source of heterogeneity, with lower risks of CVD for high versus low egg consumption in Asian populations, but not in populations from Europe and the United States of America (USA). Possibly, this also applies to the outcome of CHD, because CHD can be expected to make up u large part of total CVD.
Moreover, the pooled analysis of EPIC studies, that made a relatively large contribution to the MA of Drouin – Chartier et al. (including 409885 participants and 7198 cases), found suggestions for a reduced risk of CHD with relatively higher egg consumption, although not always statistically significant. RRs (95%CI) were 0.93 (0.86, 1.01) for high versus low egg consumption, and 0.93 (0.88, 0.99) per +20 g/d of egg consumption. The dose-response association attenuated to non-significant after exclusion of the first four years of follow-up. Subgroup analyses suggested differences in associations between men and women and between geographical regions, with statistically significant reduced CHD risks in men and Northern European populations, and no associations in women and central and Southern European populations.

In total, 21 out of 28 cohorts included in the MA of Drouin-Chartier et al. adjusted the associations for lipid profile or dyslipidaemia and/or use of lipid-lowering drugs, likely including or reflecting elevated LDL cholesterol, which is an established CHD risk factor that may well be on the causal pathway form egg consumption to CHD. Such adjustment may therefore lead to overadjustment and attenuation of associations with CHD. No subgroup analyses for studies with and without these adjustments were performed by Drouin-Chartier et al. In the pooled analysis of EPIC studies, the association was adjusted for hyperlipidemia at baseline. In subgroup analyses in people without chronic diseases (thus excluding people with hyperlipidemia), the association was not different compared to the overall association.

With respect to confounder adjustment, the committee also noted that 6 out of the 17 reports adjusted the associations for consumption of red and/or processed meat consumption. No subgroup analyses for studies with and without such adjustments were performed by Drouin-Chartier et al.

Drouin-Chartier et al. judged the risk of bias using the Newcastle-Ottawa scale. There were no major concerns with respect to risk of bias. For the health outcome CVD, the results from studies at low risk of bias were not substantially different from the main result. Possibly, this also applies to the studies on CHD (no such analyses were reported by the authors for the outcome of CHD), because the studies contributing to the MA of CHD and those contributing to the MA of CVD partly overlapped.
It was not reported whether there was evidence for publication bias with respect to the association of egg consumption and CHD risk. For the outcome of total CVD, there was no evidence for publication bias.
Drouin-Chartier et al. reported that the Nurses' Health Study (NHS) I, II and Health Professionals Follow-Up Study (HPFS), that contributed to the MA, were funded by non-commercial sources. For the other studies that contributed to the MA, funding sources were not reported. Moreover, some of the MA-authors reported relationships with industries of dairy and nuts, but not related to eggs.

Based on the above, and taking into account the decision tree, the committee concluded that the evidence for egg consumption and its association with CHD risk is inconclusive because there were both suggestions of neutral and protective associations reported, and findings were not always robust in subgroup analyses. Possibly, the associations were affected by differences in geographical regions, which may be due to differences in preparation methods of the eggs and/or residual confounding by foods that are consumed together with eggs.

Table 1 Characteristics and results of meta-analyses of prospective cohort studies on the association between egg consumption and risk of coronary heart disease

Author, year, *study design*	Egg consumption level examined	Type of analysis	N PCS	N participants; N cases	Strength of the association: RR (95% CI)	I^2	Study population (n PCS)	Risk of bias (n reports)
Drouin-Chartier 2020, *MA*	Highest intake category: >2 to >7 eggs/wk. Lowest intake category: no egg.	high vs. low	28 (22 estimates; 17 reports)	1421241; 59777	0.97 (0.91, 1.04)	42%	Europe (14), USA (8), Asia (6)	Low (7), Moderate (10)
Drouin-Chartier 2020, *MA*	Highest intake category: >2 to >7 eggs/wk. Lowest intake category: no egg.	Per +1 egg/d	NR (21 estimates)	1411261; 59713	0.96 (0.91, 1.03)	38%	NR	NR

Abbreviations: CI: confidence interval; MA: meta-analysis; NR: not reported; PCS: prospective cohort studies; RR: relative risk; USA: United States of America; vs: versus; wk: week.

3.2 LDL cholesterol

The DDG2015-committee concluded that there was strong evidence that the intake of cholesterol from eggs per 100 mg per day (half an egg per day) increases LDL cholesterol with 0.050 mmol/L.[2] This was based on a MA of 17 RCTs, that addressed the effects of cholesterol from eggs, and 3 individual RCTs. More recent evidence supports this conclusion, as described below. Since the committee aimed to evaluate the effect of eggs (the whole food product), and the recent MAs specifically reported on the effects for eggs, the committee decided to change the wording of the conclusion into the effect of one egg (containing approximately 200 mg dietary cholesterol) per day, as follows: There is strong evidence that consumption of eggs increases LDL cholesterol with approximately 0.10 mmol/L per one egg per day.

Two recent MAs, of Li et al.[10] and Khalighi Sikaroudi et al.,[37,38] support the 2015-conclusion that egg consumption increases LDL cholesterol (Table 2). Based on the DDG2015-conclusion, it can be concluded that 1 egg/d (that contains approximately 200 mg dietary cholesterol) increases LDL cholesterol on average with 0.10 mmol/L and that 2 eggs per day increase LDL cholesterol on average with 0.20 mmol/L. This is roughly in line with the findings of Li et al., showing that consumption of 1 to 4 eggs/d (median approximately 2 eggs/d) increases LDL cholesterol with 0.21 mmol/L, compared to various controls. Additionally, Khalighi Sikaroudi et al. showed an increase of 0.20 mmol/L, but did not clearly specify the range of egg intake in the intervention groups. Moreover, this MA showed substantial heterogeneity in size, but not in direction, of the effects, possibly due to differences in the range of egg intake

and different comparisons across studies. Heterogeneity was partly explained by subgroup analyses, showing that the effect of egg consumption on LDL cholesterol was larger in studies with durations of less than 12 weeks, studies with cross-over designs, studies in which intervention groups consumed more than one egg per day and studies that had a large contrast in egg consumption compared to the control groups.

Table 2 Characteristics and results of meta-analyses of randomised controlled trials on the effect of egg consumption compared to various controls on LDL cholesterol

Author, year	Intervention; comparison	N RCTs; N participants	Strength of the effect: MD (95% CI), mmol/L	I^2
Li, 2020	i: 1 to 4 eggs/d; c: No or less egg consumption, egg substitute, regular diet.	13; NR	0.21 (0.12, 0.31)	18%
Khalighi Sikaroudi, 2020	i: 1 egg/d (n=8) or >1 egg/d (n=44); c: No egg, less egg, egg substitute (without cholesterol), specific diet similar in macronutrient composition or other foods.	52; NR	0.19 (0.15, 0.23)[a]	77%

Abbreviations: c: comparison; CI: confidence interval; MD: mean difference; I: intervention; NR: not reported; mmol/L: millimol per liter; RCT, randomised controlled trial.

[a] Compared to the overall estimate, the estimate in healthy populations (n=40 RCTs) was rather similar, with MD 0.20 (95%CI: 0.15, 0.25) and I^2 79%.

3.3 Stroke

The DDG2015 concluded that an association between egg consumption and risk of stroke is unlikely.[2] This was based on 2 MAs, including 5 to 8 prospective cohort studies. There are now substantially more prospective cohort studies available on this topic. Based on that, the committee performed an extensive evaluation.

Summary of evidence for the association between egg consumption and risk of stroke

Aspect	Explanation
Available studies	One meta-analysis of 32 cohorts[39]
Heterogeneity	Yes, partly explained[39]
Strength of the association	• RR (95%CI) for highest versus lowest category of egg consumption: 0.92 (0.84, 1.01);[39] • Dose-response analysis: evidence for non-linearity, p-value 0.02.[39]
Consumption level examined	• Highest – lowest category of egg consumption: ≥2 eggs per day – no egg;[35,39] • Interquartile ranges (P25 to P75) in g/d: ♂ 7 to 26; ♀ 17 to 22.[40]
Study population	Europe, United States of America, Asia[39]

Conclusion: The evidence for the associations between egg consumption up to 300 grams per week and stroke risk is inconclusive.
Evidence level: not applicable
Additional remark: For egg consumption levels above 300 grams per week, there is too little research.

Explanation
The committee used the MA of Tang et al.[39] for its evaluation, including 32 prospective cohort studies (16 reports). Of these studies, 13 were European (including the publication of Tong et al.,[40] a pooled analysis of 9 cohorts that contributed to EPIC, counted as 9 cohorts by the committee). Studies into fatal and/or non-fatal stroke were included. The follow-up time of the included studies varied from 9 to 32 years.

In total, 9 out of 16 reports that contributed to this MA reported the definition of eggs. In these reports, egg intake was defined as whole egg intake or eggs consumed as components of recipes, including fried, boiled, poached, deviled eggs and egg salad. Two of the reports additionally noted that baked dishes (such as custards and puddings) were not included. No data regarding the effect of cooking method (e.g. boiled, fried, or other methods) was reported. The largest difference examined between the highest versus lowest category of egg intake level of the included studies was 0 egg/d versus ≥2 eggs/d and the smallest difference was <1 egg/month versus ≥2 eggs/week.

In the MA of Tang et al., the highest versus lowest category of egg intake analysis did show a tendency towards a reduced risk of total stroke, but not statistically significant, and with substantial heterogeneity between studies (Table 3). The forest plot showed that the associations were in particular in neutral and protective directions, with a few studies tending towards harmful associations (but not statistically significant).
In subgroup analyses, there were differences in associations between geographical locations, with a reduced stroke risk in Asian studies, but not in studies from the USA or Europe. The potential non-linearity of the association (described below) could also have contributed to the heterogeneity.
The dose-response analysis demonstrated a nearly J-shaped association between egg consumption and risk of stroke. A decreased risk was observed for the intake of one to four eggs weekly (0-200 g/week), and an increased risk for the intake of more than six eggs weekly (>300 g/week), although the latter was not statistically significant.
The increased risk became statistically significant at intakes of 10 eggs (>500 g/week) weekly. However, the committee noted that there were relatively few observations, and only from small studies, for intakes higher than 300 g/week, and only three observations for intakes higher than 500 g/week.

Regarding subtypes of stroke, Tang et al. showed that there was a tendency towards a reduced risk of ischemic stroke with high versus low egg consumption, but this was not statistically significant. For hemorrhagic stroke, no association was found for high versus low egg consumption, but with substantial heterogeneity between studies that was not further explained.
The committee noted that the vast majority of studies included in the MA of Tang et al. adjusted the associations for systolic blood pressure or hypertension at baseline. Theoretically, blood pressure might be on the causal pathway form egg consumption to stroke. Therefore, the committee cannot entirely exclude the possibility that the associations reported in the MA may be, to some extent, underestimated.

Tang et al. judged the risk of bias using the Newcastle-Ottawa scale. Based on that, 12 out of 16 reports were judged to be of high quality (low risk of bias), and the remaining to be of moderate quality. The associations were not different between subgroups based on low and moderate risk of bias.
No evidence for publication bias was observed by Tang et al.
The MA-work was funded by non-commercial sources, and the MA-authors reported no conflicts of interest. No information was given on the funding sources of the individual studies that were included in the MA.

Based on the above and taking into account the decision tree, the committee concluded that the evidence for the association between egg consumption and stroke risk is inconclusive up to the consumption of 300 g/week because there were both suggestions for neutral and for protective associations. Moreover, findings were inconsistent in subgroups by geographical location. The committee judged that there is too little research to form a conclusion for higher egg intakes because there were relatively few observations from only small-scale studies with intakes of >300 g/week.

Table 3 Characteristics and results of meta-analyses of prospective cohort studies on the association between egg consumption and risk of stroke

Author, year, *study design*	Egg consumption level examined	Type of analysis	N PCS	N participants; N cases	Strength of the association: RR (95% CI)	I^2	Study population (n PCS)	Risk of bias (n reports)
Tang, 2020, *MA*	Highest intake category: ≥2 eggs/d Lowest intake category: no egg	High vs. low	32 (16 reports)[a]	1387653; 58451[a]	Total stroke: 0.92 (0.84, 1.01); Total stroke, in Europe: 1.02 (0.91, 1.16); Total stroke, in USA: 0.95 (0.77,1.16); Total stroke, in Asia: 0.83 (0.73, 0.94); Ischemic stroke: 0.94 (0.88, 1.00); Hemorrhagic stroke: 0.96 (0.74, 1.26)	64% 24% 51% 61% 15% 75%	Europe (13), USA (14), Asia (4), Middle east (1)[a]	Low (12), Moderate (4)[a]
Tang, 2020, *MA*	0 to 800 g/wk	Dose-response analysis for egg intake in g/wk	26 (15 reports)	1364129; 57149	Total stroke: Evidence for a non-linear dose-response curve (p-value 0.02)	65%	Europe (13), USA (8), Asia (4), Middle east (1)	Low (11), Moderate (4)

Abbreviations: CI: confidence interval; MA: meta-analysis; NR: not reported; PCS: prospective cohort studies; RR: relative risk; USA: United States of America; vs: versus; wk: week.

[a] Applies to the analysis of total stroke. The analyses in subgroups by continent and subtype of stroke include fewer studies, participants and cases.

3.4 Blood pressure

In the DDG2015, no evaluation on the effects of egg consumption on blood pressure could be performed.[2] There is now sufficient evidence available on egg consumption and its effects on blood pressure to draw conclusions on this topic. Therefore, the committee performed an extensive evaluation.

Summary of evidence for the effects of egg consumption compared to various controls on blood pressure

Aspect	Explanation
Available studies	One meta-analysis of 15 randomised controlled trials[41]
Heterogeneity	• Systolic blood pressure: No;[41] • Diastolic blood pressure: Moderate.[41]
Strength of the effect	Mean differences (95%CI) for intervention versus control group: • Systolic blood pressure: 0.046 (-0.792, 0.884) mmHg;[41] • Diastolic blood pressure: -0.603 (-1.521, 0.315) mmHg.[41]
Consumption level examined	One egg to three eggs per day[41]
Study population	United States of America and other non-European countries[41]

Conclusion 1: An effect of consumption of 1 to 3 eggs per day for 3 to 12 weeks on systolic blood pressure is unlikely.
Evidence level: Strong

Conclusion 2: The evidence for the effect of egg consumption on diastolic blood pressure is inconclusive
Evidence level: not applicable

Explanation
The committee used the MA of Kolahdouz-Mohammadi et al.[41] for its evaluation of the effects of egg consumption on systolic and diastolic blood pressure. The MA included 15 RCTs. Intervention durations varied between RCTs from 3 to 12 weeks.
The committee noted that the RCTs included in the MA originated from the USA (n=10) or other non-European countries (n=5). In the USA, preparation methods and use of eggs might be different compared to some European countries, such as the Netherlands.

The interventions used in the RCTs were based on the intake of whole eggs and ranged from 1 to 3 eggs/d. Comparison foods varied between RCTs. For instance, some RCTs used no or less egg consumption as comparison, and others used eggs substitutes or oatmeal.

Kolahdouz-Mohammadi et al. found no effects of egg consumption on systolic and diastolic blood pressure (Table 4). There was no heterogeneity between studies for systolic blood pressure. There was moderate heterogeneity between studies for diastolic blood pressure, with most of the RCTs reporting a neutral effect and a few reporting reducing effects. The committee found no plausible explanation for the heterogeneity. In subgroup analyses, the effects were not substantially different in subgroups based on study design, sex, population health status, obesity and hypertension status, diet adherence, study duration, and amount of egg consumption, although the number of RCTs in some of the subgroups was small.

Kolahdouz-Mohammadi et al. used the Cochrane Collaboration's tool to judge the risk of bias. One RCT was judged as good quality (low risk of bias), 7 as fair quality and 7 as poor quality. For a large proportion of RCTs, there was a lack of blinding of participants, personnel and/or outcome assessors or of allocation concealment. However, the committee judged it unlikely that this impacted the MA conclusions because a lack of blinding would rather have led to an overestimation of the effects, whereas no effects were found in this MA. In addition, studies of poor quality lacked

random sequence generation or had incomplete outcome data. No subgroup analyses were performed for low and fair quality studies. However, the committee considered it likely that this did not noticeably impact the MA conclusions, because there was no or only moderate heterogeneity between the studies in the MAs.
There was no evidence for publication bias reported by Kolahdouz-Mohammadi et al. The MA-authors reported no conflicts of interest. No information on funding sources of the MA-work and individual studies that contributed to the MA was provided.

Based on the above and taking into account the decision tree, the committee concluded that an effect of egg consumption on systolic blood pressure is unlikely. The effect estimation observed in the MA was close to zero and the confidence interval was narrow and evenly distributed around zero. Furthermore, there was no substantial heterogeneity, there were sufficient RCTs and participants included in the MA for drawing a conclusion with strong evidence, and there were no other major considerations that might impact the certainty of the evidence.
The committee concluded that the evidence for diastolic blood pressure is inconclusive. Although the effect estimate was close to 0, the confidence interval was relatively broad and skewed to the left, which precludes the conclusion that the effect is classified as unlikely.

Table 4 Characteristics and results of meta-analyses of randomised controlled trials on the effects of egg consumption compared to various controls on blood pressure

Author, year, *study design*	Intervention (n); comparison (n)	N RCTs; N participants	Strength of the effect: MD (95%CI), mmHg	I^2	Study population (n)	Risk of bias (n)
Kolahdouz-Mohammadi, 2020, *MA*	i: 1 to 3 eggs/d (15); c: Less or no egg consumption (5), egg substitute incl. egg-beaters (4), oatmeal (3) or other foods (3)	15; 748	SBP, overall: 0.046 (-0.792, 0.884); SBP, in healthy populations: 0.167 (-0.881, 1.215); DBP, overall: -0.603 (-1.521, 0.315); DBP in healthy populations: -1.166 (-2.626, 0.295).	0% 38%	USA (10), South America (2), Australia (1), Asia (1)[a]	Study quality: High (1), Fair (7), Poor (7)

Abbreviations: c: comparison; CI: confidence interval; DBP: diastolic blood pressure; MD: mean difference; i: intervention; incl.: including; mmHg: millimeters of mercury; RCT: randomised controlled trial, SBP: systolic blood pressure; USA: United States of America.
[a] In terms of health status, the following populations were included: healthy population (n=8), people with type 2 diabetes (n=4), people with metabolic syndrome (n=1), people with hyperlipidemia (n=1), people with coronary artery disease (n=1).

3.5 Heart failure

The DDG2015 concluded there was limited evidence that consumption of eggs is associated with a higher risk of heart failure in prospective cohort studies from Northern America.[2] This was based on 2 cohort studies from the USA. There are now a few more cohort studies available on this topic, summarised in the MA of Bechthold et al.[42] (Table 5). Bechthold et al. also reported the increased risk of heart failure observed in the 2 cohort studies from the USA. In addition, 2 cohort studies from Europe were included in the MA of Bechthold et al. One of these European studies also found increased risks of heart failure with higher egg consumption, although this was only statistically significant in the high versus low analysis and not in the dose-response analysis. The other European cohort study found no associations, with rather broad confidence intervals. Since there were only 4 cohort studies included in the MA, and the higher risk of heart failure was in particular apparent in the 2 cohort studies from the USA, and not consistent in the 2 cohort studies from Europe, the evidence seems inconclusive.

Table 5 Characteristics and results of meta-analyses of prospective cohort studies on the association between egg consumption and risk of heart failure

Author, year	N PCS; N cases	Type of analysis	Strength of the association: RR (95% CI)	I^2
Bechthold, 2019	4; 5059	Highest versus lowest egg intake category	1.25 (1.12, 1.39)	0%
Bechthold, 2019	4; 5059	Per +50 g/d of eggs	1.16 (1.03, 1.31)	55%

Abbreviations: CI: confidence interval; PCS: prospective cohort studies; RR: relative risk.

3.6 Type 2 diabetes

The DDG2015 concluded that there is strong evidence that consumption of at least 1 egg/d compared to less than 1 egg/month is associated with an approximately 40% higher risk of type 2 diabetes.[2] This was based on a MA of 4 cohort studies from the USA, and one individual cohort study from Europe. There are now substantially more cohort studies available on this topic, also including more data from Europe. Therefore, the committee performed an extensive evaluation on the association between egg consumption and risk of type 2 diabetes.

Summary of evidence for the association between egg consumption and risk of type 2 diabetes

Aspect	Explanation
Available studies	One meta-analysis of 19 prospective cohort studies[43]
Heterogeneity	Yes, in magnitude for studies from USA and Asia, and in direction for studies from Europe[43]
Strength of the association	RR (95%CI) per +1 egg/d:[43] • Overall: 1.07 (0.99, 1.15); • In studies from Europe: 0.99 (0.85, 1.15); • In studies from USA: 1.18 (1.10, 1.27); • In studies from Asia: 0.82 (0.62, 1.09).
Consumption level examined	Highest – lowest category of egg consumption: ≥7 eggs per week – no egg[43]
Study population	Europe, United States of America, Asia[43]

Conclusion: The evidence for the association between egg consumption and risk of type 2 diabetes is contradictory.
Evidence level: not applicable

Explanation:
The committee used the MA of Drouin – Chartier et al.[43] for the evaluation of the association between egg consumption and risk of type 2 diabetes. This MA included 16 reports that together included 19 prospective cohort studies. Of these, 8 cohort studies were European. The reported durations of follow-up in the included studies varied from 5 to 32 years.

The definitions of eggs consumption in the included studies was not reported by Drouin – Chartier et al. Categories of egg consumption that were compared in the cohorts that contributed to the MA varied, for instance: 0 egg/week vs. ≥7 eggs/week (a relatively large difference in exposure) and <1 egg/week vs. ≥4 eggs/week (a relatively small difference in exposure).

The MA of Drouin – Chartier et al. observed no associations between egg consumption and risk of type 2 diabetes in both the dose-response analysis and in the highest versus lowest category of intake analysis (Table 6). However, there was substantial heterogeneity between studies. The associations differed by geographical location, with a harmful association observed in studies from the USA, but not in studies from Europe and Asia. Heterogeneity remained present within the subgroups of geographical location. Visual inspection of the forest plot showed that the heterogeneity was mainly in magnitude of the associations in studies from the USA (RRs above 1) and from Asia (RRs below 1). For European studies, heterogeneity was also present in the directions of the associations, with both suggestions for beneficial and harmful associations.

A possible explanation for the heterogeneity in findings between countries and continents might be related to egg consumption habits. In the USA, egg consumption may be reflective of adherence to a Western dietary pattern because eggs are often consumed with red or processed meat, refined grains, and sugary beverages.
In Europe, this varies between different countries and eating patterns (for instance, between Mediterranean and Northern European eating patterns). Differences in egg consumption levels are less likely an explanation for the heterogeneity because, overall, low amounts of egg intake were observed in the included prospective cohort studies (most individuals consumed 1 to <5 eggs/week).

The committee noted that 15 out of 16 reports adjusted the associations for BMI. BMI could be on the causal pathway between egg consumption and occurrence of type 2 diabetes. Therefore, the associations reported in the MA may be, to some extent, underestimated. This was also suggested by the individual prospective cohort study by Ericson et al.,[44] that contributed to this MA. Ericson et al. showed that high versus low egg consumption was associated with 27% higher risk of type 2 diabetes. Additional adjustment for BMI attenuated this risk estimate to 14%.

Drouin – Chartier et al. judged the risk of bias using the Newcastle Ottawa scale.
In total, 10 out of the 16 reports were judged to have a low risk of bias, and the remaining were judged to have moderate or high risk of bias. The associations were not substantially different between subgroups based on the risk of bias.
Drouin – Chartier et al. reported significant funnel plot asymmetry (significant Egger's test), indicating that unfavorable risk estimates with high standard errors were perhaps not reported. This might be the result of publication bias, but might also be due to heterogeneity (e.g. between larger and smaller studies).
The MA-work was funded by non-commercial sources. Two of the MA-authors reported relationships with dairy or nut industries, but not with egg companies. Funding sources of the individual studies that contributed to the MA were not reported by the MA-authors.

Based on the above and taking into account the decision tree, the committee concluded that results from prospective cohort studies on the association between egg consumption and risk of type 2 diabetes are contradictory. This holds for the overall association between egg consumption and type 2 diabetes risk, but also within European cohorts, reporting suggestions for neutral, harmful as well as beneficial associations. This heterogeneity is possibly due to differences in background diet and egg consumption habits between countries.

Table 6 Characteristics and results of meta-analyses of prospective cohort studies on the association between egg consumption and risk of type 2 diabetes

Author, year, *study design*	Egg consumption level examined	Type of analysis	N PCS	N participants; N cases	Strength of the association: RR (95% CI)	I^2	Study population (n PCS)	Risk of bias (n reports)
Drouin - Chartier, 2020, *MA*	Highest intake category: ≥7/wk Lowest intake category: no egg	High vs. low	19 (16 reports; 22 estimates)[a]	589559; 41248[a]	Overall: 1.08 (0.98, 1.19); In studies from Europe: 0.98 (0.84, 1.13); In studies from USA: 1.26 (1.11, 1.42); In studies from Asia: 0.87 (0.69, 1.10)	61% 59% 47% 38%	Europe (8), USA (9), Asia (2)[a]	Low (10), Moderate (5), High (1)[a]
Drouin – Chartier, 2020, *MA*	Highest intake category: ≥7/wk Lowest intake category: no egg	Per +1 egg/d	19 (16 reports, 22 estimates)	589559; 41248[a]	Overall: 1.07 (0.99, 1.15); In studies from Europe: 0.99 (0.85, 1.15); In studies from USA: 1.18 (1.10, 1.27); In studies from Asia: 0.82 (0.62, 1.09)	70% 74% 51% 59%	Europe (8), USA (9), Asia (2)[a]	Low (10), Moderate (5), High (1)[a]

Abbreviations: RR: relative risk; CI: confidence interval; PCS: prospective cohort studies; USA: United States of America, vs: versus, wk: week.

[a] Applies to the overall analysis. The number of participants and cases by continent are as follows: Europe: 179714 and 10698, respectively; USA: 339377 and 28528, respectively; Asia: 70468 and 2022, respectively.

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Annexes

A Working group

The following working group prepared this background document:

Working group on nuts and eggs and chronic diseases

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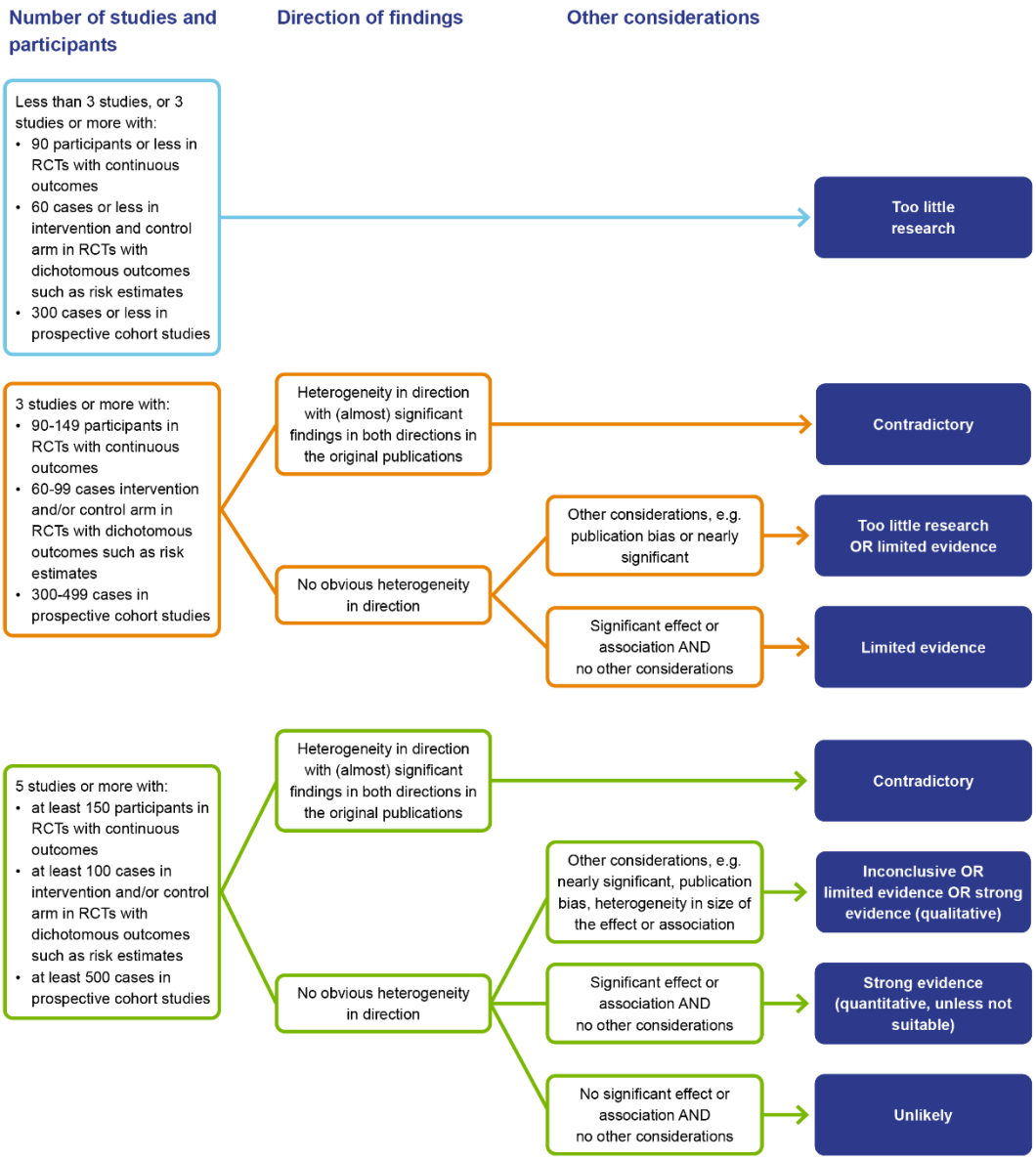
B Decision tree

The decision tree below was used as a guidance tool by the committee on Nutrition for drawing conclusions based on scientific evidence from meta-analyses and pooled analyses of randomised controlled trails or prospective cohort studies.



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