Toxicology Reports 10 (2023) 367–375

Contents lists available at ScienceDirect

Toxicology Reports

journal homepage: www.elsevier.com/locate/toxrep

Meta-analysis of the effects of smoking and smoking cessation on triglyceride levels

Angela van der Plas *, Meagan Antunes, Sandrine Pouly, Guillaume de La Bourdonnaye, Matthew Hankins, Annie Heremans

PMI R&D, Philip Morris Products S.A., Quai Jeanrenaud 5, CH-2000 Neuchâtel, Switzerland

ARTICLE INFO

Handling Editor: Prof. L.H. Lash

Keywords:
Cigarette smoking
Smoking cessation
Triglycerides
Tobacco
Cardiovascular disease

ABSTRACT

Smoking increases lipid levels, including triglycerides, leading to increased cardiovascular disease risk. We performed a meta-analysis to quantify the effects of smoking and smoking cessation on triglyceride levels. The PubMed and Scopus databases were searched to identify studies reporting either triglyceride levels in smokers and non-smokers or the effects of smoking cessation on triglyceride levels. Fixed- and random-effects models were used to perform the analyses when three or more studies/comparisons were available. We identified 169 and 21 studies evaluating the effects of smoking and smoking cessation, respectively, on triglyceride levels. Triglyceride levels were 0.50 mmol/L (95% confidence interval: 0.49–0.50 mmol/L) higher in smokers than non-smokers, but the effect differed widely across studies. No statistically significant effect was observed on triglyceride levels between baseline and 6 weeks (mean difference [MD] = 0.02 [−0.09, 0.12] mmol/L), 2 months (MD = 0.03 [−0.21, 0.27] mmol/L), 3 months (MD = 0.08 [−0.03, 0.21] mmol/L), or 1 year (MD = 0.04 [−0.06, 0.14] mmol/L) after quitting. However, a slightly significant decrease in triglyceride levels was observed at 1 month after cessation (MD = −0.15 [−0.15, −0.01] mmol/L). The results of this meta-analysis provide a basis for understanding the effects of smoking and smoking cessation on triglyceride levels, which could have important implications for public health.

1. Introduction

Cigarette smoke contains large amounts of free radicals and pro-oxidants [1], and cigarette smoking is a well-known risk factor for cardiovascular disease (CVD) such as coronary artery disease [2], atherosclerosis, and heart failure [3]. One mechanism through which smoking increases the risk of CVD includes the alteration of lipid levels [3]. Cigarette smoking is associated with increased levels of triglycerides (TGs) and lower levels of high-density lipoprotein cholesterol (HDL-C). However, no significant effects of cigarette smoking on low-density lipoprotein-cholesterol (LDL-C) levels have been observed [4,5]. Whether the nutritional behavior of smokers also influences their cholesterol levels remains unclear [4]. On average, current smokers have 3% higher total cholesterol levels, approximately 10–15% higher TG levels, and approximately 6.5% lower HDL-C levels than non-smokers [4].

Most international guidelines recommend lowering LDL-C levels to lower the risk of developing CVD, but other lipid molecules have also been associated with CVD [6]. For instance, HDL-C plasma levels were shown to be inversely related to CVD risk [7], including coronary heart disease [6], and this association persists even at very low levels of LDL-C. Additionally, a 1996 meta-analysis performed showed that elevated TG levels were associated with a 14% increased risk of CVD in males and 37% increased risk in females per 1 mmol/L increase in TG levels, after adjustment for HDL-C levels [8].

There is abundant evidence supporting a link between smoking and smoking cessation with HDL-C levels. Two meta-analyses found increased HDL-C levels following smoking cessation [9,10]; however, no meta-analyses have examined the effects of smoking and smoking cessation on TG levels. Therefore, the aim of the present study was to summarize the available literature through a meta-analysis of the effects of smoking on TG levels.

Abbreviations: BMI, body mass index; CVD, cardiovascular disease; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; TG, triglycerides.

* Corresponding author.
E-mail address: Angela.vanderplas@pmi.com (A. van der Plas).

https://doi.org/10.1016/j.toxrep.2023.03.001
Received 25 January 2023; Received in revised form 21 February 2023; Accepted 3 March 2023
Available online 4 March 2023
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2. Methods

2.1. Study selection

MEDLINE searches were performed by querying the PubMed and Scopus databases for studies that evaluated the relationship between smoking or smoking cessation and TG levels. The PubMed search was performed on July 9, 2021, using the following query: ("triglycerides"[MeSH Terms] OR "triglycerides"[All Fields]) AND (("smoking"[MeSH Terms] OR "smoking"[All Fields]) OR ("tobacco"[MeSH Terms] OR "tobacco"[All Fields] OR "tobacco products"[MeSH Terms] OR ("tobacco"[All Fields] AND "products"[All Fields]) OR "tobacco products"[All Fields]) OR quitting[All Fields] OR cessation[All Fields]). The search using the Scopus database was performed on the same date, using the following query: "Triglycerides AND (smoking OR tobacco OR cessation OR quitting)."

The abstracts obtained through these searches were screened to confirm whether they contained the relevant information regarding smoking status and TG levels. To verify that all available studies were retrieved, the reference lists of the publications obtained through the original search were checked for any additional articles. Finally, all retrieved articles were assessed according to the following inclusion and exclusion criteria.

The inclusion criteria were as follows: (1) case control, cohorts, or interventional studies such as randomized controlled trials, which included healthy adult subjects; (2) studies reporting measurements of TG levels by smoking exposure (smokers vs. non-smokers and baseline vs. a defined time after smoking cessation) with the following measures available: mean values by group, standard deviation (SD) or standard error (SE), sample size per group, or enough information to allow for the calculation of mean and SD; and (3) studies that were published in English, Spanish, Italian, Portuguese, or German.

The exclusion criteria were as follows: (1) review articles, case reports, or editorials; or (2) studies reporting insufficient data (such as missing values for triglyceride levels by exposure group, SD or SE and sample size; or enough information to calculate means and SD) or those reporting data used in a more recent, included study.

2.2. Data extraction

Two investigators (M.A. and A.vdP.) independently identified relevant articles, extracted the data (mean values by group, SD or SE, sample size per group, or other information to sufficiently allow for the calculation of mean and SD) and discussed disagreements to reach a consensus. The study-specific information that was extracted included the name of the first author, the year of publication, country, study design and population characteristics, number of participants per group, and effect adjustment variables (e.g., age and sex). Estimate-specific information for each exposure group (smokers, non-smokers) and time point (including time after cessation) consisted of TG levels (including mean as well as SD or SE) and group size.

Values reported in mg/dL were converted into mmol/L using the multiplier 0.01129 [11]. Median and range values were converted to mean ± SD using the formulas reported by Hozo et al. [12].

2.3. Statistical analysis

All analyses were performed using the 'meta' [13] and 'dmetar' [14] packages in R 4.0.5 [15].

To quantify the effects of smoking on TG levels, pooled mean differences (Δ) between smokers and non-smokers (when assessing effects of smoking on TGs) or differences between baseline and follow-up measures after smoking cessation (when assessing the effects of quitting smoking on TG levels) and 95% confidence intervals (CIs) were calculated using the fixed-effects model in the 'metamean' function [13]. The 'metamean' function uses the inverse variance method for pooling, giving studies with small variance relatively higher weight and studies with larger variance relatively smaller weights [13].

Adjusted estimates were preferred to unadjusted ones. When estimates were provided by strata (e.g., sex or age groups), they were included separately. For the smoking cessation data, estimates were grouped by the duration of quitting and compared using subgroup analyses.

The degree of heterogeneity across estimates was assessed by the I^2 value, which describes the percentage of variation across studies that is not caused by sampling error. In general, when interpreting the I^2 value, a value less than 25% represents low heterogeneity, a value between 25% and 75% represents moderate heterogeneity, and a value greater than 75% represents substantial heterogeneity [16]. Funnel plot symmetry and Egger's regression test were used to evaluate publication bias [17]. Statistical significance was assessed at $\alpha = 0.05$.

To explore possible sources of heterogeneity, the meta-analysis was performed using the random-effects model, and sensitivity analyses were conducted using the 'InfluenceAnalysis' function to eliminate studies that contributed the most to heterogeneity. The Baujat plot and forest plot sorted by I^2 produced by the 'InfluenceAnalysis' function were used to identify the studies with high heterogeneity contribution and low influence on the overall results. These studies represent outliers and were removed to reduce the amount of between-study heterogeneity. Additionally, subgroup analyses were performed based on the definition of smoking, geographical region, study design, period of publication, and sex.

3. Results

A flow diagram depicting the retrieval process of articles used in the analysis is presented in Fig. 1. We identified 300 publications that presented the effect estimates of smoking status and its association with TG levels. A total of 169 publications reporting 239 effect estimates were included in the analyses, and their characteristics are listed in Supplementary Table 1. The reasons for exclusion were as follows: 1 study had been retracted [18], 1 was published in Japanese [19], 2 compared ever-smokers to non-smokers [20,21], 2 were duplicates of included studies [22,23], 2 were conducted on mice [24,25], 3 lacked non-smoker data [26–28], 5 used post-prandial data [29–33], 5 examined effects of other tobacco products (i.e., not cigarettes) [34–38], 6 presented data regarding the acute effects of smoking on TG levels [39–44], 8 provided geometric means or log-transformed data [45–52], 8 had underage populations [53–60], 16 included diseased populations [61–76], and 70 publications presented incomplete data [77–147]. We identified 21 studies that included data on the effects of smoking cessation and TG levels; their characteristics are listed in Supplementary Table 2. Ten publications were excluded for the following reasons: 1 study compared reducers (those who reduced the amount of cigarettes they smoked) and non-quitters [148], 2 compared immediate changes after smoking [43,149], 9 did not specify the cessation period [91,150–157], 1 was conducted on a diseased population [67], and 3 had incomplete data [158–160].

3.1. Effects of smoking status on TG levels

An overview of the 169 included studies can be found in Supplementary Table 1, along with the effect estimates retrieved from these studies. The meta-analysis revealed that the mean TG level of smokers (1.6181 [1.6158, 1.6203] mmol/L) was approximately 38% higher than that of non-smokers (1.1734 [1.1726, 1.1741] mmol/L). This value is higher than previously reported TG levels; a 2010 review stated they were approximately 10% higher in smokers than in non-smokers [4]. The overall meta-analysis (Table 1) revealed a statistically significant increase in mean TG levels in smokers compared to non-smokers, using both fixed-effects (Δ = 0.41 [0.41, 0.42] mmol/L) and random-effects models (Δ = 0.27 [0.23, 0.32] mmol/L). Visual inspection of the

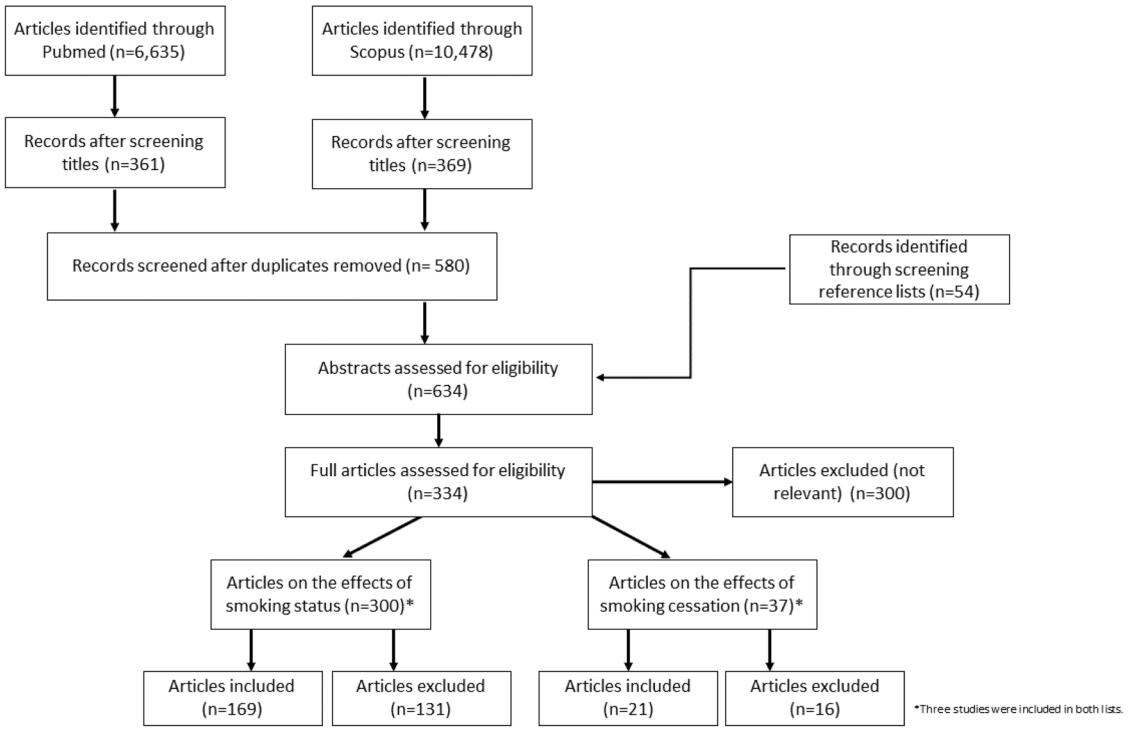

Fig. 1. Flow diagram for the article retrieval process.

funnel plot (Fig. 2) revealed evidence of asymmetry, with several large studies showing significant positive effects. Funnel plot asymmetry was confirmed using Egger's regression test (intercept = −3.43; 95% CI: −5.20, −1.66, t = −3.08 p = 0.0002), and it may be an indication of publication bias. The heterogeneity of the individual effect estimates was also very high (I^2 = 99%).

Due to the high heterogeneity, we conducted sensitivity analyses, which identified four studies (Park et al., 2021 [161], Chimura et al. [162], Cuschieri et al. [163], and Pasupathi et al. [164]) that had large impacts on between-study heterogeneity. Exclusion of these studies, however, did not substantially reduce the I^2 value (I^2 = 96%). Thus, we performed subgroup analyses by geographic region, study design, publication period, smoking definition, and sex to further explore possible sources contributing to the high heterogeneity (Table 1). Studies conducted in Australia and Latin America had the lowest heterogeneity (I^2 = 11% and I^2 = 36%, respectively). Stratification by study design did not substantially decrease heterogeneity (I^2 = 89–99%). Studies conducted in the 1980 s had lower heterogeneity (I^2 = 53%) than studies published in other periods (I^2 = 82–99%). Estimates obtained for females had lower heterogeneity (I^2 = 78%) than those for males (98%) or those where both sexes were combined (99%).

We identified 64 comparisons from 48 publications available on individuals smoking more than 15 cigarettes per day (Table 2). The results showed higher mean TG levels in smokers than in non-smokers, and while heterogeneity was slightly lower, it was still considerable (Δ = 0.60 [0.60, 0.61] mmol/L; I^2 = 95%). The random-effects model of this meta-analysis yielded similar results for the overall group (Δ = 0.32 [0.25, 0.40] mmol/L). Subgroup analyses of these estimates revealed that USA- and Canada-based studies had lower heterogeneity (I^2 = 42%) than studies conducted in other geographic regions (I^2 = 86–99%). Significant heterogeneity was also found in studies conducted in the 1970 s (I^2 = 92%), 2000 s (I^2 = 84%), and 2010 s (I^2 = 96%), but not in those conducted in the 1980 s (I^2 = 35%) or 1990 s (I^2 = 72). While heterogeneity was high in both sex-specific analyses (I^2 = 88–93%), it was slightly lower when estimates that combined sexes were included (I^2 = 68%).

3.2. *Effect of smoking cessation on TG levels*

We used 21 studies with 31 estimates to evaluate the effect of smoking cessation on TG levels. An overview of the studies is provided in Supplementary Table 2. The overall meta-analysis showed no statistically significant difference in TG levels from baseline to the end of the observation period after smoking cessation, using either a fixed-effects model (Δ = 0.00 [−0.05, 0.05] mmol/L) or a random-effects model (Δ = −0.01 [−0.06, 0.05] mmol/L) (Table 3). No substantial heterogeneity was identified (I^2 = 12%), and visual inspection of the funnel plot (Fig. 3) revealed no evidence of publication bias, which was confirmed using Egger's regression test (intercept = 0.28, 95% CI: −0.49, 1.05, t = 0.71, p = 0.49).

We also conducted subgroup analyses to determine if there were different effects depending on the follow-up duration (Table 3). Three studies provided four comparisons on TG level changes after 6 weeks of cessation [165–167]. However, pooled analysis revealed no significant difference between TG levels after 6 weeks of smoking cessation and those at baseline (Δ = 0.02 [−0.09, 0.12] mmol/L, I^2 = 70%). Heterogeneity was mainly driven by the study by Allen et al. [165], which was the only one to report increased TG levels 6 weeks after smoking cessation. Four studies had a follow-up duration of 1 month after smoking cessation, which revealed a slightly significant decrease in TG levels (Δ = −0.15 [−0.29, –0.01] mmol/L, I^2 = 0%). For the 2-month cessation comparison, a non-significant increase in TG levels was found based on four studies [168–171] (Δ = 0.03 [−0.21, 0.27] mmol/L, I^2 = 0%). A total of 7 studies reported 11 comparisons of TG levels after 3 months of cessation [172–178]. The pooled analysis showed a non-significant increase after this cessation period (Δ = 0.08 [−0.04, 0.19] mmol/L, I^2 = 0%). Three studies assessed mean TG levels 1 year after cessation [5,167,177], and a non-significant increase in TG levels was found (Δ = 0.04 [−0.06, 0.14] mmol/L, I^2 = 0%).

4. Discussion

The aim of this analyses was to quantify the effects of smoking and smoking cessation on serum triglycerides levels. The meta-analysis

Table 1
Results of the meta-analysis conducted on smoking status and triglyceride (TG) level (mmol/L) data from previous studies. All estimates.

Region	Studies	Effect Estimates	Mean Differences in TG levels (mmol/L) (smokers – non-smokers)		
			Fixed Effects [95% CI]	I^2 (%)	Random Effects [95% CI]
US & Canada	23	34	0.08 [0.07, 0.10]	82	0.18 [0.12, 0.23]
Europe	51	73	0.24 [0.24, 0.25]	96	0.21 [0.17, 0.26]
Asia	43	72	0.46 [0.46, 0.47]	100	0.29 [0.20, 0.38]
Latin America	5	5	0.15 [0.00, 0.30]	36	0.17 [− 0.03, 0.36]
Middle East	18	21	0.11 [0.09, 0.13]	97	0.35 [0.23, 0.46]
Indian Subcontinent	23	26	0.76 [0.74, 0.79]	98	0.49 [0.30, 0.68]
Australia	2	4	0.16 [0.03, 0.30]	11	0.18 [0.02, 0.34]
Africa	4	4	0.13 [0.08, 0.17]	87	0.13 [0.00, 0.26]
Study Design					
Cross Sectional	143	202	0.13 [0.12, 0.13]	98	0.26 [0.23, 0.30]
Cohort	19	29	0.58 [0.58, 0.58]	99	0.27 [0.19, 0.35]
Case Control	5	6	0.29 [0.25, 0.32]	97	0.40 [0.17, 0.64]
RCT	2	2	NA	NA	NA
Period of Publication					
1970 s	4	5	0.43 [0.38, 0.47]	95	0.30 [0.03, 0.56]
1980 s	24	40	0.25 [0.22, 0.28]	53	0.24 [0.19, 0.30]
1990 s	37	54	0.17 [0.16, 0.18]	82	0.22 [0.18, 0.26]
2000 s	51	72	0.25 [0.24, 0.26]	99	0.23 [0.15, 0.32]
2010 s	43	58	0.08 [0.08, 0.09]	98	0.33 [0.28, 0.38]
2020 s	10	10	0.59 [0.58, 0.59]	100	0.35 [0.24, 0.47]
Smoking Definition[a]					
None/Other	128	163	0.16 [0.16, 0.17]	98	0.25 [0.21, 0.28]
> 10 CPD	11	12	0.37 [0.31, 0.43]	65	0.34 [0.22, 0.46]
> 15 CPD	9	11	0.41 [0.35, 0.48]	97	0.24 [− 0.19, 0.66]
> 20 CPD	40	53	0.60 [0.60, 0.61]	93	0.33 [0.26, 0.40]
Sex[a]					
Males	82	105	0.30 [0.29, 0.30]	98	0.31 [0.24, 0.37]
Females	35	48	0.05 [0.05, 0.06]	86	0.16 [0.12, 0.19]
Combined	70	86	0.52 [0.52, 0.52]	99	0.29 [0.22, 0.35]
Total	**169**	**239**	**0.41 [0.41, 0.42]**	**99**	**0.27 [0.23, 0.32]**

[a] The sum of studies does not add to 169, as some studies provided multiple stratum-specific estimates.

revealed higher TG levels in smokers than in non-smokers (1.6181 [1.6158, 1.6203] mmol/L vs. 1.1734 [1.1726, 1.1741] mmol/L, respectively). We found that most of the heterogeneity came from European and Asian studies (I^2 = 96–100%), studies performed in the 1970 s (I^2 = 95) or 2000 s or later (I^2 = 99–100%), and studies with male subjects (I^2 = 98%) or both sexes combined (I^2 = 99). Studies

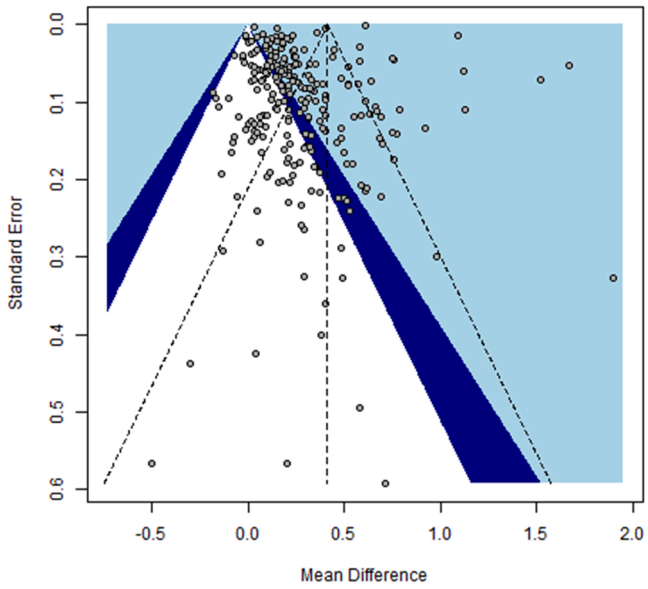

Fig. 2. Funnel plot for studies reporting triglyceride levels in smokers and non-smokers.

assessing smokers who consumed 15 or more cigarettes per day exhibited lower heterogeneity (I^2 = 95%), which was non-negligible. Our analysis of the effect of smoking cessation on TG levels revealed a slightly significant decrease at 1 month after smoking cessation. However, no significant difference in TG levels was observed at 6 weeks, 2 months, 3 months, or 1 year after smoking cessation.

The high heterogeneity in the meta-analysis of smoking status and TG levels can partially be explained by differences in smoking intensity, study design, geographical region, and sex. The notorious intra-individual variability in serum TG levels [179], even after standardizing sampling procedures and laboratory techniques, may also contribute to the heterogeneity seen across studies [180–182]. TG levels can also vary by up to 25% in healthy fasted subjects when measured 2.5 months apart, and hyper-triglyceridemic individuals can exhibit even greater fluctuations [183]. Besides smoking status, many factors can influence TG levels, including age, body mass index (BMI), oral contraceptive use, stress, alcohol use, and lack of physical exercise [184, 185].

A possible reason for no observable effect after smoking cessation for most follow-up durations might be that weight gain often occurs after quitting smoking [186,187]. The association between weight gain and TG levels has long been established [188]. Albrink et al. [188] reported results from a prospective cohort of 215 males, where they evaluated the effect of weight gain on various cardiovascular risk parameters. The authors found that in males who gained 4.5 kg or more, TG levels were higher than in those who gained fewer than 4.5 kg during the follow-up period (7.1 mmol/L vs. 4.6 mmol/L). A meta-analysis of the impact of weight loss on lipid parameters [189], which included 64 studies, revealed that for each 1 kg of weight loss, TG levels decreased by 1.93% or 0.017 mmol/L.

Botella-Carretero et al. analyzed the association between smoking cessation and weight gain [190] and reported increases in weight, BMI, waist–hip ratio, and diastolic blood pressure after quitting smoking, which were independent of obesity at baseline or the use of nicotine patches or bupropion. Moreover, a systematic review and meta-analysis designed to quantify weight gain after smoking cessation analyzed data from 63,403 quitters and 388,432 continuing smokers and found that the mean weight gain was 4.10 kg [2.69–5.51] among quitters [191]. The authors also reported that, compared to continuing smoking, cessation was associated with absolute weight gain (Δ = 2.61 kg [95% CI: 1.61–3.60]).

Table 2
Results from the meta-analysis of smoking and triglyceride (TG) level (mmol/L) data from previous studies. Estimates for smokers smoking ≥ 15 cigarettes per day.

	Studies	Effect Estimates	Mean Differences in TG levels (mmol/L) (smokers – non-smokers)		
Region			Fixed Effects [95% CI]	I^2 (%)	Random Effects [95% CI]
USA & Canada	11	14	0.16 [0.09, 0.22]	42	0.17 [0.07, 0.27]
Europe	14	21	0.33 [0.31, 0.36]	89	0.22 [0.11, 0.33]
Asia	16	21	0.61 [0.61, 0.61]	86	0.41 [0.30, 0.51]
Middle East	2	3	0.82 [0.73, 0.91]	99	0.64 [− 0.28, 1.56]
Indian Subcontinent	4	4	0.68 [0.56, 0.79]	95	0.66 [0.16, 1.16]
Africa	1	1	NA	NA	NA
Study Design					
Cross Sectional	42	55	0.38 [0.35, 0.40]	92	0.31 [0.21, 0.40]
Cohort	6	9	0.61 [0.61, 0.61]	95	0.42 [0.26, 0.58]
Period of Publication					
1970 s	3	3	0.53 [0.48, 0.58]	92	0.35 [0.05, 0.64]
1980 s	15	23	0.19 [0.13, 0.25]	35	0.19 [0.10, 0.27]
1990 s	14	19	0.28 [0.24, 0.31]	72	0.28 [0.21, 0.36]
2000 s	9	11	0.19 [0.13, 0.25]	84	0.20 [0.04, 0.36]
2010 s	6	7	0.82 [0.76, 0.88]	96	0.85 [0.52, 118]
2020 s	1	1	NA	NA	NA
Sex[a]					
Males	27	32	0.38 [0.34, 0.41]	93	0.34 [0.19, 0.48]
Females	10	12	0.18 [0.14, 0.23]	88	0.15 [0.06, 0.24]
Combined	17	20	0.61 [0.61, 0.61]	68	0.43 [0.34, 0.51]
Total	**48**	**64**	0.60 [0.60, 0.61]	95	0.32 [0.25, 0.40]

[a] The sum of studies does not add to 48, as some studies provided multiple stratum-specific estimates.

Another feasible explanation for the lack of a decrease in TG levels after smoking cessation could be the relatively short follow-up period of only up to 1 year. A recent study by Noh et al. [192] showed that TG levels decreased 3 years after smoking cessation (1.64 ± 0.83 mmol/L vs. 1.47 ± 0.82 mmol/L). A limitation of our baseline vs. time after smoking cessation meta-analysis is that the numbers of evaluated participants in studies typically decrease with longer follow-up duration. Indeed, when examining the sample sizes of the studies included in this meta-analysis, the studies using a follow-up time of 6 weeks after cessation included 467 quitters, while the analyses after 3 months included 446 quitters, and the analyses that were conducted after 1 year included only 356 participants. This was also seen in the study by Noh et al. [192], where only 49 out of the 779 participants quit and were successfully followed for 3 years. To address this issue, larger and longer follow-up studies after smoking cessation are needed to evaluate the impact of quitting on TG levels.

Our analysis has some limitations, for instance it is difficult to adjust for confounding variables that could affect triglyceride levels. While we tried to use adjusted values as much as possible, these were not always available. Another potential limitation is that we only used Pubmed and Scopus as our sources for literature identification, we did also check the reference lists of all identified publications to make sure we had all literature available possible.

Based on the findings of the present meta-analysis, we conclude that smoking is clearly associated with higher TG levels in current smokers than in non-smokers. These data need to be interpreted with caution due to the high heterogeneity encountered in the analyses. A longer follow-up of larger groups of quitters is needed for more comprehensive assessment of the possible effects of smoking cessation on TG levels. Furthermore, the impact of lifestyle and physical changes (e.g., weight gain) on TG levels following smoking cessation should also be investigated.

This study represents the first meta-analysis published on the effects

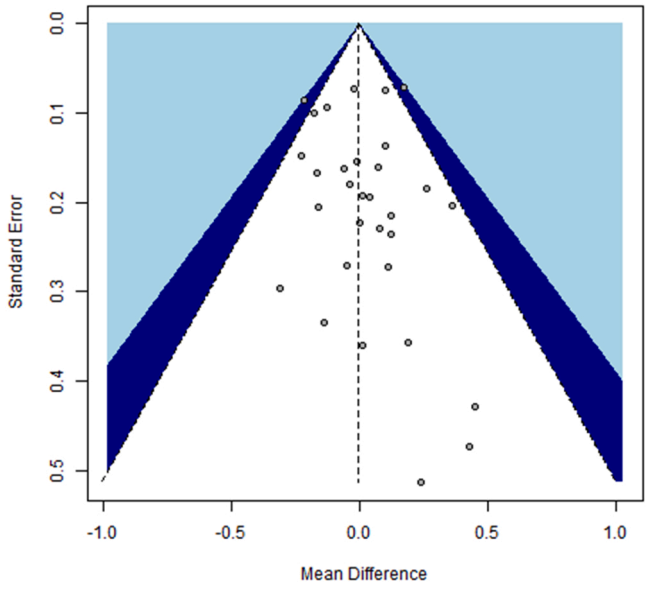

Fig. 3. Funnel plot for studies reporting triglyceride levels before and after smoking cessation.

Table 3
Smoking cessation and triglyceride (TG) levels (mmol/L).

	Studies	Effect Estimates	Mean Difference (after quitting – baseline)		
Follow-up[a]			Fixed Effects [95% CI]	I^2 (%)	Random Effects [95% CI]
2 weeks	1	1	NA	NA	NA
3 weeks	1	1	NA	NA	NA
6 weeks	3	4	0.02 [− 0.09, 0.12]	70	-0.05 [− 0.27, 0.17]
1 month	4	4	-0.15 [− 0.29, − 0.01]	0	-0.15 [− 0.29, − 0.01]
2 months	4	4	0.03 [− 0.21, 0.27]	0	0.03 [− 0.21, 0.27]
3 months	7	11	0.08 [− 0.04, 0.19]	0	0.08 [− 0.04, 0.19]
1 year	3	3	0.04 [− 0.06, 0.14]	0	0.04 [− 0.06, 0.14]
3 years	1	1	NA	NA	NA
Total	**21**	**31**	**0.00 [− 0.05, 0.05]**	**12**	**-0.01 [− 0.06, 0.05]**

[a] The sum of studies does not add to 21, as some studies provided estimates for multiple follow-up times.

of smoking and smoking cessation on TG levels. The results could be relevant to public health policy and clinicians hoping to improve outcomes for patients after smoking cessation.

Funding

Philip Morris International is the sole source of funding and sponsor of this research. All authors are employees of Philip Morris International, thus, the funder was involved in study design, the collection, analysis and interpretation of data, the writing of the report, and in the decision to submit the article for publication.

CRediT authorship contribution statement

Meagan Antunes: Data curation, Formal analysis, Validation, Writing – original draft, Writing – review & editing. **Angela van der Plas:** Conceptualization, Data curation, Writing – original draft, Writing – review & editing. **Guillaume de La Bourdonnaye:** Validation, Writing – review & editing. **Sandrine Pouly:** Conceptualization, Validation, Writing – review & editing. **Matthew Hankins:** Conceptualization, Writing – review & editing, Supervision. **Annie Heremans:** Resources, Writing – review & editing, Supervision.

Author contributions

Meagan Antunes: was responsible for the data curation; statistical analysis and review and editing of the original draft, Guillaume de La Bourdonnaye was responsible for the data validation and review of the original draft, Sandrine Pouly was responsible for the conceptualization and data validation, Matthew Hankins was responsible for resources and review of the original draft, Annie Heremans was responsible for resources and review of the original draft, Angela van der Plas for the conceptualization, and writing of the original draft.

Declaration of Competing Interest

The authors declare the following financial interests/personal relationships which may be considered as potential competing interests: All authors are employed by Philip Morris International.

Data Availability

Data can be found in cited publications and is summarized in supplementary tables.

Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.toxrep.2023.03.001.

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