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Farming, Reported Pesticide Use, and Prostate Cancer

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Abstract

Prostate cancer is the leading cancer type diagnosed in American men and is the second leading cancer diagnosed in men worldwide. Although studies have been conducted to investigate the association between prostate cancer and exposure to pesticides and/or farming, the results have been inconsistent. We performed a meta-analysis to summarize the association of farming and prostate cancer. The PubMed database was searched to identify all published case–control studies that evaluated farming as an occupational exposure by questionnaire or interview and prostate cancer. Ten published and two unpublished studies were included in this analysis, yielding 3,978 cases and 7,393 controls. Prostate cancer cases were almost four times more likely to be farmers compared with controls with benign prostate hyperplasia (BPH; meta odds ratio [OR], crude = 3.83, 95% confidence interval [CI] = 1.96–7.48, *Q*-test *p* value = .352; two studies); similar results were obtained when non-BPH controls were considered, but with moderate heterogeneity between studies (meta OR crude = 1.38, 95% CI = 1.16–1.64, *Q*-test *p* value = .216, I^2 = 31% [95% CI = 0–73]; five studies). Reported pesticide exposure was inversely associated with prostate cancer (meta OR crude = 0.68, 95% CI = 0.49–0.96, *Q*-test *p* value = .331; four studies), whereas no association with exposure to fertilizers was observed. Our findings confirm that farming is a risk factor for prostate cancer, but this increased risk may not be due to exposure to pesticides.

Keywords

hormone disruptors; case–control; Caribbean; meta-analysis

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Declaration of Conflicting Interests
The authors declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Introduction

Prostate cancer is the leading cancer type diagnosed and the second leading cause of cancer-related death in American men (Siegel, Naishadham, & Jemal, 2012). An estimated 241,740 new prostate cancer cases and 28,170 deaths are expected in 2012 in the United States (Siegel et al., 2012). Although family history, age, and Black race are known risk factors, pesticide exposure is also thought to contribute to prostate cancer development. Pesticides are substances or mixtures of substances that are used for preventing, destroying, repelling, or mitigating any pest. Though often misunderstood to refer only to insecticides, the term *pesticide* also applies to herbicides, fungicides, and various other substances used to control pests, insects, mice and other animals, unwanted plants (weeds), fungi, and microorganisms such as bacteria and viruses. Exposure to pesticides may occur in several ways: during preparation of mixtures of pesticides for use, such as by mixing a concentrate with water or loading the pesticide into application equipment; by applying pesticides, such as in an agricultural or commercial setting; or by entering an area where pesticides have been applied to perform allowed tasks, such as picking crops. Defining types of pesticides involved and modalities of individual exposure is a complex task for epidemiologists.

Androgens, particularly testosterone and dihydrotestosterone, are essential to the normal growth and functioning of the prostate, but are also known carcinogens; variations in these hormone levels might be related to prostate cancer development (Sawada et al., 2010). Exposure to pesticides such as methyl bromide and organochlorines are thought to increase the risk of prostate cancer through their biologic effects on hormone levels and function (Soto et al., 1995; van der Gulden, Kolk, & Verbeek, 1995). Organochlorines such as dichlorodiphenyltrichloroethane and polychlorinated biphenyls are believed to disrupt normal endocrine activity by acting as estrogen-like compounds and by increasing the levels of endogenous estrogens (Safe, 2000; Sawada et al., 2010) and act as tumor promoters (Hansen & Matsumura, 2001; Xu et al., 2010). A meta-analysis on 18 cohort studies showed an increased risk of developing or dying of prostate cancer (Maele-Fabry, Libotte, Willems, & Lison, 2006) with pesticide exposure in pesticide manufacturing workers.

Studies suggest that farming is one of the most common occupational risk factors for prostate cancer because of the use of pesticides (Alavanja et al., 2003). Although multiple studies on this topic have been conducted, the findings have been inconsistent. More than a decade has passed since published findings from a review (Blair, Malker, Cantor, Burmeister, & Wiklund, 1985) and two meta-analyses (Acquavella et al., 1998; Keller-Byrne, Khuder, & Schaub, 1997) of farming and prostate cancer have been reported. These publications summarized reported results from studies on prostate cancer mortality as well as case–control studies. There were very few studies at that time that involved noncancer controls, and there was a paucity of studies that involved non-White populations and specifically Caribbean populations. Therefore, we have reviewed and performed an updated meta-analysis of the current literature to summarize the association between farming and prostate cancer, and for the first time included unpublished data on non-White populations from the Caribbean. A summary of pesticide use among these farmers also has been reported.

Method

Literature Search and Selection Criteria

A specialized search using the database PubMed on May 1, 2012, was performed to identify studies that evaluated farming exposure and prostate cancer. The search was broadened to include occupational exposure risks. The search terms used were: (prostate) AND (cancer OR neoplasms OR tumor OR carcinoma OR carcinogenesis) AND (pesticide OR

organochlorine OR Organophosphate OR Carbamate OR Pyrethroid OR agriculture OR farm OR farmer OR farming OR occupational exposure), which yielded the query in PubMed reported in Table 1. In addition, all the MeSH terms attached to each reference have been further searched. The use of wild cards such as farm* substantially increased the number of articles retrieved but did not improve the quality of the search.

The search yielded 684 studies that were evaluated based on the following inclusion criteria: (a) studies written in English, (b) studies that include data pertinent to calculate odds ratios (ORs) for prostate cancer with farming, and (c) studies that include incident prostate cancer cases. Studies that included other cancer types as their control groups (Brownson, Reif, Chang, & Davis, 1989; Fincham, Hanson, & Berkel, 1992; Keller & Howe, 1994) and those for which both the cases and controls were selected from a cohort of pesticide applicators or farmers (Gunnarsdottir & Rafnsson, 1991; Rafnsson & Gunnarsdottir, 1989; Stark, Chang, Fitzgerald, Riccardi, & Stone, 1987; Wigle et al., 1990) were excluded from this analysis because the study design was such that the whole population was exposed to pesticides and/or was composed of farmers; thus, no unexposed, control group was available. Twenty-two articles were excluded because they were written in languages other than English; however, the English abstracts were checked to make sure that no relevant study was excluded because of a language barrier.

Twelve articles fit the inclusion criteria. Four publications were identified with overlapping patient populations (Franceschi et al., 1993; Talamini, Franceschi, La Vecchia, Guarneri, & Negri, 1993; van der Gulden, Kolk, & Verbeek, 1992; van der Gulden et al., 1995) for which only the more recent publication with the larger study population was included (Franceschi et al., 1993; van der Gulden et al., 1995). Overall, 10 published articles were included in this meta-analysis, one of which reported on both incident and prevalent cases (Le Marchand, Kolonel, & Yoshizawa, 1991). In addition, data sets from two unpublished prostate cancer studies (Bunker et al. and Jackson et al.) were included. Overall, this analysis includes 12 studies consisting of 3,988 cases and 7,531 controls. From each study, the following data were extracted and tabulated: country in which the study was conducted, race/ethnicity, recruitment period, number of cases and controls, source of the control population, type of controls (whether diagnosed with benign prostatic hyperplasia [BPH] or not), type of exposure (farming or any pesticide), and method of exposure assessment (self-administered or interviewer-administered questionnaires). Incident cases were those subjects newly diagnosed with prostate cancer and histologically confirmed.

Unpublished Studies—Additional unpublished data sets were obtained through a partnership of the Genetic Susceptibility to Environmental Carcinogens study (GSEC; www.gsec.net) and the African-Caribbean Cancer Consortium (AC3; www.ac-ca-consortium.org), which constituted the GSEC-AC3 consortia. A request for data was sent to all the research groups partnering with the GSEC-AC3 consortia.

Jamaica study—Two hundred and forty-three men aged 40 to 80 years with incident prostate cancer histologically confirmed were recruited from urology clinics at the two main tertiary hospitals and from private practitioners in the Kingston Metropolitan area in Jamaica between March 2005 and July 2007. Controls were subjects attending the same urology outpatient clinics, with a negative digital rectal examination and total prostate-specific antigen (PSA) <4.0 μg/L and free: total PSA ≤ 0.24. All the subjects answered an interviewer-administered questionnaire primarily aimed at investigating diet and other lifestyle factors. Medical records were reviewed to ensure that there was no previous history of prostate surgery or hormonal/finasteride treatment.

The study was approved by the ethics committee of the University of the West Indies, and subjects gave written informed consent prior to participation in the study.

Tobago study—This is a population-based longitudinal study of prostate cancer screening, as well as risk, in ≥40-year-old men living in Tobago. Between September 1997 and September 2007, the Tobago Prostate Study used public service announcements, flyers, local health care workers, and word of mouth to solicit 40- to 79-year-old men in Tobago for participation in a study of periodic prostate cancer screening; 3,264 men accepted to enter the study, signed written informed consent, answered a risk factor questionnaire, underwent blood collections and prostate cancer screening examinations, with PSA serum concentrations ≥4 ng/mL or abnormal digital rectal examinations prompting referral for ultrasound-guided transrectal prostate biopsy. A simple age-matched random sample of the at-risk men screened for prostate cancer represented the control group. The institutional review boards of the University of Pittsburgh and Tobago Division of Health and Social Services approved the research protocol.

Statistical Analysis

The association between farming, pesticide, herbicide, and/or fertilizer and prostate cancer was investigated by calculating the crude OR with 95% confidence intervals (CI) for each study. The combined OR (meta ORs) were calculated when data were provided by two or more studies. To minimize heterogeneity between studies, stratified analyses were performed based on the type of control (BPH, non-BPH, or Mixed). Mixed controls referred to participants for whom it was not specified whether a BPH diagnosis was present. When the stratified analysis did not resolve the heterogeneity between studies, a sensitivity analysis was performed to identify individual studies that might have determined heterogeneity. When a study was identified, the analysis was repeated after excluding the study to confirm that there was no longer heterogeneity between the remaining studies.

The Q statistics were used to test for heterogeneity between the studies included in the meta-analyses. I^2 was used as a confirmatory estimate of the degree of heterogeneity among studies (Higgins, Thompson, Deeks, & Altman, 2003; Ioannidis, Patsopoulos, & Evangelou, 2007), with I^2 < 25%, 25% to 50%, and >50% representing low, moderate, and high degree of heterogeneity, respectively. When significant heterogeneity was observed (Q-test p value < .05), the meta OR was not reported in the results. A fixed-effects model was used to calculate the meta ORs when there was no heterogeneity observed between the studies. “Small study effects” were assessed by performing the Egger’s test. All statistical analyses were performed using STATA SE, Version 10 (StataCorp LP, College Station, TX).

Results

Farming and Prostate Cancer

Twelve studies reported data on the association of farming and prostate cancer (3,978 cases and 7,393 controls); there was large heterogeneity between the studies (Q-test p value = .002, I^2 = 63% [95% CI = 32–80]) and no statistical evidence of publication bias (Eggers-test p value = .0751). To explore the observed between-study heterogeneity, the studies were stratified based on the type of controls included in the study (BPH vs. non-BPH; Table 2). The meta OR for studies involving BPH controls suggested that cases were almost fourfold more likely to have been farmers compared with BPH controls (meta OR = 3.83, 95% CI = 1.96–7.48, Q-test p value = .352). Given that there are only two studies in this group, the I^2 statistic and publication bias for this stratum could not be performed.

There was also an increased risk of prostate cancer among farmers compared with non-BPH controls, and there was moderate heterogeneity between studies (meta OR = 1.38, 95% CI = 1.16–1.64, Q-test p value = .216, I^2 = 31% [95% CI = 0–73]). No evidence of publication bias was observed for these studies (Eggers-test p value = .494). As expected, for study designs that included mixed controls (both BPH and non-BPH), large heterogeneity was observed (Q-test p value = .024, I^2 = 64% [95% CI = 7–86]), and no statistical evidence of publication bias was observed (Eggers-test p value = .888). Sensitivity analysis did not identify a single study that appeared to influence the meta OR.

Caribbean studies—A subgroup analysis of studies including Caribbean populations (n = 3) yielded a meta OR = 1.23 (95% CI = 1.0–1.51), with no heterogeneity among studies.

Pesticide Exposure

Information on pesticide exposure was reported for 5 of the 12 studies (Table 3). Four studies reported data on pesticide exposure among farmers only, and the fifth publication presented the risk of prostate cancer in relationship to combinations of leisure and occupational exposure to pesticides or garden sprays, but a separation among the two activities was not possible (Sharpe, Siemiatycki, & Parent, 2001). Pesticide exposure among farmers (269 cases and 535 controls) was inversely associated with prostate cancer (meta OR = 0.68, 95% CI = 0.49–0.96, Q-test p value = .331, I^2 = 12% [95% CI = 0–87]; Table 3); no association between pesticide exposure and prostate cancer was reported by Sharpe et al. (2001; crude OR = 1.2, 95% CI = 0.5–2.5; adjusted OR = 1.2, 95% CI = 0.6–2.8). In this single study, when the analysis was restricted to people exposed to pesticides or garden sprays during leisure, the OR for prostate cancer was 2.3, 95% CI = 1.3–4.1 (adjusted OR = 2.5, 95% CI = 1.4–4.6; Sharpe et al., 2001). Among the farmers included in these studies, no association between prostate cancer and exposure to fertilizers was observed (meta OR = 0.74, 95% CI = 0.40–1.34). However, there was high heterogeneity among studies (Q-test p value = .04, I^2 = 68.9%).

Discussion

This meta-analysis suggests that farming is associated with an increased risk of prostate cancer, consistent with what was reported by previously published meta-analyses (Acquavella et al., 1998; Blair et al., 1985), which showed a small but significant increase in prostate cancer risk when mortality and case–control studies were analyzed. The current analysis includes additional studies that were not available at the time the other meta-analyses were published and includes three studies conducted in the Caribbean populations. These studies show that the association between prostate cancer and farming is present in that geographic area as well.

An interesting observation deriving from this analysis is the heterogeneity observed between the studies; the stratification according to type of controls was able to resolve this heterogeneity, suggesting that there are distinct differences between BPH and non-BPH controls that could contribute to inconsistencies in the associations between studies.

One concern when conducting meta-analyses of published data is the possibility of publication bias deriving from studies that are not published in the current literature because of null results or small sample size. Although the issue of nonpublished results is difficult to address, the present analysis did not seem to be hampered by publication bias, according to the statistical analysis conducted to detect such bias; in addition, two unpublished studies were included in an attempt to be as inclusive as possible. However, we cannot exclude the presence of unpublished studies on this topic, especially in geographic area such as the Caribbean or Africa. Another limitation is that some of the studies (5 of 12) did not match

on age; thus, it is possible that prostate cancer cases, being older, are also more likely to have been farmers. However, the studies that did not match included cases and controls of similar mean ages, and the study-specific estimates do not indicate that a significant association between prostate cancer and farming is restricted to the studies that did not match on age. Therefore, this limitation, although methodologically important, does not seem to affect the reported estimates.

We have also evaluated the specific contribution of pesticide exposure among studies on farming and prostate cancer. Reported pesticide exposure among farmers does not appear to significantly contribute to prostate cancer risk, and if any, the studies show an inverse association between pesticide exposure during farming and prostate cancer. This may suggest that controls are actually exposed to pesticides under different conditions, such as leisure time, but they do not report it. One main limitation of the present analysis is that pesticides exposure was self-reported. It is likely that reporting bias may have played a role, by introducing a random misclassification of exposure that could have biased the results toward the null. The study by Sharpe et al. (2001) suggests that pesticide exposure might be more relevant among people exposed during leisure rather than when farming. Some studies indicate an increased risk of prostate cancer with occupational pesticide exposure (e.g., Strom, Yamamura, Flores-Sandoval, Pettaway, & Lopez, 2008), whereas others show no association (Cockburn et al., 2011; Fritschi, Glass, Tabrizi, Leavy, & Ambrosini, 2007; Kumar et al., 2010), and similarly in these studies specific occupational exposure was not defined. It is possible that subjects exposed to pesticides during leisure take fewer precautions than subjects who use pesticides in the workplace.

In addition to pesticides exposure, farmers may experience exposures to a large variety of other factors. Exposure to chemicals such as fertilizers, to sun, and to organic dust from harvesting could be present as well and could vary on an individual basis. The only other chemicals we were able to test in the present analysis, fertilizers, was significantly associated with prostate cancer; however, the analysis suffered due to small sample size and high heterogeneity among studies. Exposures to pesticides and other chemicals in farmers may vary by type of farm operation, environmental temperature, and other climate factors (Blair & Freeman, 2009).

Unfortunately, no details on duration and quality of pesticides exposure are reported by the studies included in this analysis. Nevertheless, Multigner et al. (2010) reported a significantly higher prostate cancer risk among cases in the highest quartile of cumulative exposure (OR = 1.73, 95% CI = 1.04–2.88) compared with cases in the lowest quartile of cumulative exposure (OR = 1.06, 95% CI = 0.62–1.82). This suggests that duration of pesticide exposure plays an important role in prostate cancer development. Considering the biological effects of pesticides as endocrine disruptors, it is conceivable that chronic exposure for years may be relevant to prostate cancer progression.

Prostate cancer is a multifactorial disease, where age, race, family history, hormonal levels, and smoking play a significant role; such cofactors should be always analyzed in relation to pesticide exposure. However, the present analysis relies on previously published data and as such is not suitable for conducting subgroup analyses or adjustment for confounders.

Future studies should target the full range of exposures in farmers, individual differences in quality and quantity of exposure to pesticides, and biomarkers of individual pesticide exposure; detailed information on personal behavioral risk factors should also be collected and analyzed in relation to pesticide exposure.

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Table 1

Medical Subject Headings (MeSH) Terms

(“prostate”[MeSH Terms] OR “prostate”[All Fields]) AND
((“neoplasms”[MeSH Terms] OR “neoplasms”[All Fields] OR “cancer”[All Fields]) OR (“neoplasms”[MeSH Terms] OR “neoplasms”[All Fields]) OR (“tumour”[All Fields] OR “neoplasms”[MeSH Terms] OR “neoplasms”[All Fields] OR “tumor”[All Fields]) OR (“carcinoma”[MeSH Terms] OR “carcinoma”[All Fields]) OR (“Carcinogenesis”[Journal] OR “carcinogenesis”[All Fields])) AND
((“pesticides”[MeSH Terms] OR “pesticides”[All Fields] OR “pesticide”[All Fields] OR “pesticides”[Pharmacological Action]) OR organochlorine[All Fields] OR (“phosphoric acid esters”[MeSH Terms] OR (“phosphoric”[All Fields] AND “acid”[All Fields] AND “esters”[All Fields]) OR “phosphoric acid esters”[All Fields] OR “organophosphate”[All Fields]) OR (“carbamates”[MeSH Terms] OR “carbamates”[All Fields] OR “carbamate”[All Fields]) OR (“pyrethrins”[MeSH Terms] OR “pyrethrins”[All Fields] OR “pyrethroid”[All Fields]) OR (“agriculture”[MeSH Terms] OR “agriculture”[All Fields]) OR farming[All Fields] OR (“occupational exposure”[MeSH Terms] OR (“occupational”[All Fields] AND “exposure”[All Fields]) OR “occupational exposure”[All Fields])

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Table 2

Summary Estimates of the Association Between Farming and Prostate Cancer, Stratified by Type of Controls

Study	Country	Ethnic groups (*n*)	Year of recruitment	Cases	Controls	Control source	Age range/average (Years)	Matching factors	OR (95% CI) farmer vs. nonfarmer	Definition of farming/ pesticide use
Cases vs. BPH controls										
Checkoway et al. (1987)	United States	White (69), Black (35)	1984–1985	40	64	Hospital	≥50 (cases: 68.8; controls: 67.3)	None	5.00 (2.08–12.01)	Ever employed as farmer/ever exposed to pesticides-herbicides
van der Gulden et al. (1995)	Netherlands	White (1,691)	1988–1990	345	1346	Hospital	45–91 (cases: 72; controls: 69)	None	2.63 (0.93–7.44)	Farmers working in mixed farming (i.e., cattle/stock); mean number of days per year during which pesticide/ fertilizer was applied, 1960–1970
Meta OR									3.83 (1.96–7.48)	
Q-test *p* value									.352	
Cases vs. non-BPH controls										
Franceschi et al. (1993)	Italy	White (1,669)	1985–1991	161	1508	Hospital	16–79	None	2.06 (1.41–3.00) (age, smoking, drinking adjusted: 1.9; 06–1.4)	Ever occupation as farmer
Ilic et al. (1996)	Serbia	White (303)	1990–1994	101	202	Hospital	Cases: 70.5; controls: 71.5	Age, hospital, admittance, residence	1.16 (0.71–1.91)	Ever occupation as farmer
Bunker (unpublished)	Tobago	Afro-Caribbean (574)	1997–2003	263	311	Healthy	40–79	Age	1.21 (0.86–1.71)	Ever worked in a farm/ plantation ever exposed to pesticides
Jackson (unpublished)	Jamaica	Afro-Caribbean (518)	2005–2007	243	275	Healthy	40–80 (cases: 61.8; controls: 67.8)	None	1.39 (0.89–2.17)	Current occupation as farmer

Study	Country	Ethnic groups (*n*)	Year of recruitment	Cases	Controls	Control source	Age range/average (Years)	Matching factors	OR (95% CI) farmer vs. nonfarmer	Definition of farming/ pesticide use
Multigner et al. (2010)	Guadalupe	Afro-Caribbean (1,432)	2004–2007	709	723	Healthy	54–72 (cases: 60.6; controls: 66.2)	None	1.22 (0.86–1.73)	Ever banana farming/ever exposed to chlordecone (plasma concentration)
Meta OR									1.38 (1.16–1.64)	
Q-test *p* value									.216	
I^2,95% CI									31% (0–73)	
Eggers-test *p* value									.494	
Cases vs. mixed controls[a]										
Le Marchand et al. (l99l)[b]	United States	Asian (545),White (178), Hawaiian (48)	1977–1983	263	508	Healthy	N/A(data stratified <70, ≥70)	Frequency matched 5-year age groups	0.98(0.60–1.60)	Farmer as the longest job held by men 70+ years; Ever exposure to pesticides from any occupation
Ewings and Bowie (1996)	England	White (484)	1989–1991	159	325	Hospital	N/A	Age	0.74 (0.48–1.15)	Ever farming (includes horticultural workers, groundsmen, etc.); ever used pesticides-fertilizers
Krstev et al. (1998)	United States	White (1,223), Black (1,073)	1986–1989	981	1,315	Healthy	40–79	Age frequency matched	2.40 (1.32–4.38)	Ever employed as farmer; ever pesticide exposure
Sharpe et al. (2001)	Canada	French Canada (565), other (311)	1979–1985	400	476	Healthy	47–70	Frequency matched—age and residence	1.11 (0.83–1.49)	Ever gardening and or farming; weekly exposure to pesticides for ≥6 months
Meyer et al. (2007)	United States	White (464), Black (333)	1999–2001	405	392	Hospital[c]	65–79	Frequency matched—age	1.36 (1.03–1.79)	Ever worked on farms since age 14; ever mixed/applied pesticides

Study	Country	Ethnic groups (*n*)	Year of recruitment	Cases	Controls	Control source	Age range/average (Years)	Matching factors	OR (95% CI) farmer vs. nonfarmer	Definition of farming/ pesticide use
Q-test *p* value									.024	
I^2,95% CI									64% (7–86)	
Eggers-test *p* value									.888	

Note. OR = odds ratio; CI = confidence interval; N/A = not available; BPH = benign prostate hyperplasia.

[a]Included both BPH and non-BPH controls or did not confirm the nature of the controls.

[b]Includes incident and prevalent cases.

[c]Medicare beneficiaries.

Table 3

Reported Pesticide, Fertilizers Exposure, and Prostate Cancer Risk Among Farmers

Study	Cases (*n*)	Controls (*n*)	Cases exposed (*n*)	Controls exposed (*n*)	OR (95% CI), Pesticide exposed vs. nonexposed	Cases exposed (*n*)	Controls exposed (*n*)	OR (95% CI)
Checkoway et al. (1987)	30	24	4	4	0.77 (0.17–3.46)			
van der Gulden et al. (1995)	64	259	19	73	1.08 (0.59–1.96)	27	102	1.12 (0.64–1.96)
Ewings et al. (1996)	40	106	15	54	0.58 (0.27–1.22)	23	65	0.85 (0.41–1.78)
Bunker, (unpublished)	135	146	34	57	0.52 (0.30–0.90)	35	93	0.45 (0.28–0.71)
Total	269	535	72	188		85	260	
Meta OR					0.68 (0.49–0.96)			0.74 (0.40–1.34)
Q-test *p* value					.331			.04
I^2, 95% CI					12% (0–87)			68.9% (0–88.8)
Eggers-test *p* value					.808			—

Note. OR = odds ratio; CI = confidence interval.