



The prevalence of Infectious agents in oesophageal adenocarcinoma: a systematic review

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A. Kunzmann, S. Graham, C. McShane, J. Doyle, M. Tommasino, J. Jamison, J. James, B. Johnston, D. McManus, T. Gheit & L. Anderson
¹Centre for Public Health, School of Medicine, Dentistry and Biomedical Science. ²Belfast Health and Social Care Trust, Royal Hospitals, Belfast.

Background

Globally infectious agents are known to cause approximately 20% of cancers.

Human papillomavirus (HPV), which may reach the oesophagus via oro-genital transmission, has previously been postulated to be associated with oesophageal adenocarcinoma development.

Other infectious agents have also been postulated to be involved in the development of oesophageal adenocarcinoma including *Helicobacter pylori* infection and John Cunningham virus.

A systematic review of the literature investigating the prevalence of infectious agents in oesophageal adenocarcinoma is currently being undertaken by researchers at Queen's University Belfast.

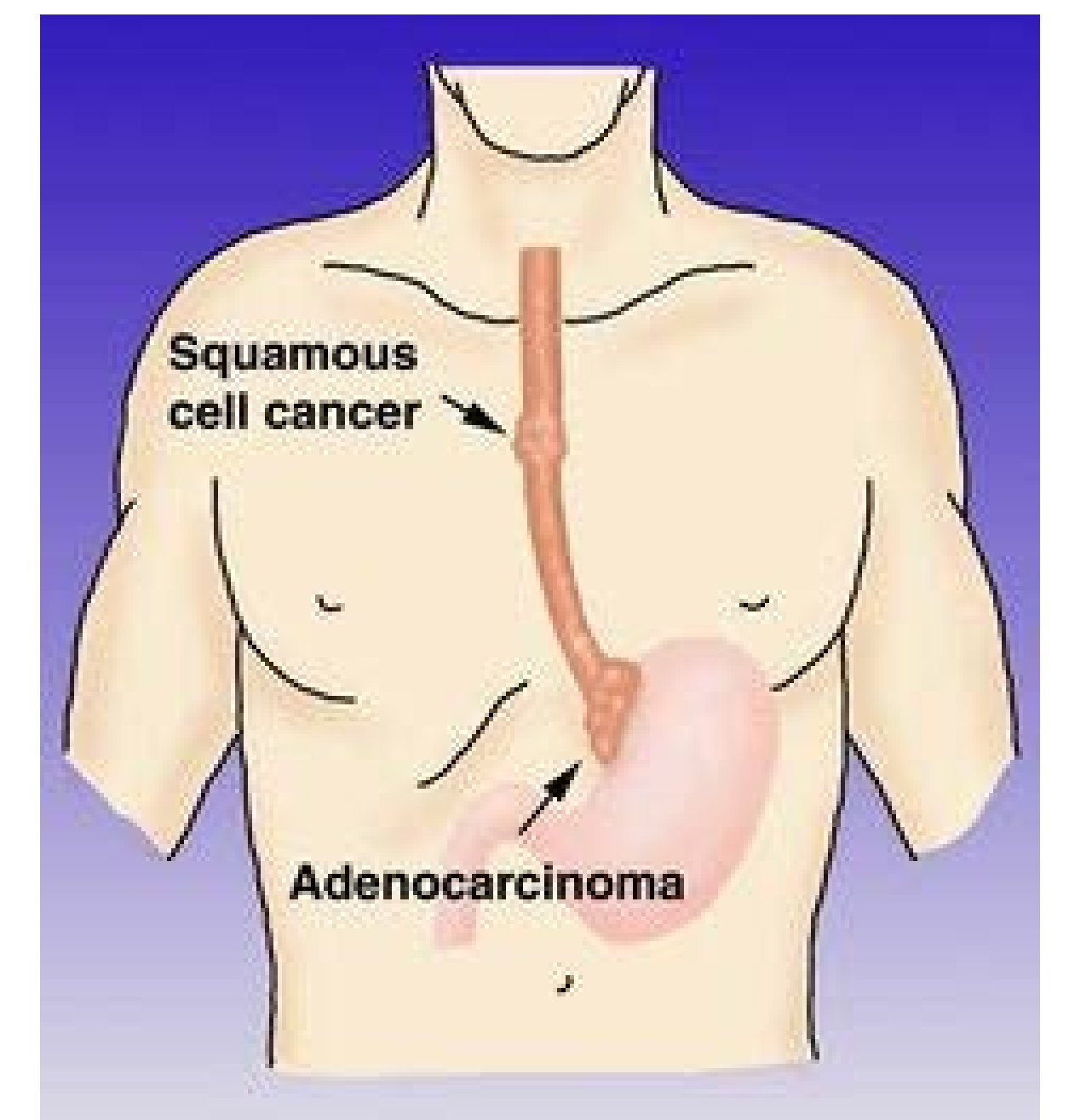


Figure 1: Location of oesophageal adenocarcinomas

Methods

To identify studies that assessed the prevalence of infectious agents in oesophageal adenocarcinoma and Barrett's oesophagus, three electronic databases, namely Medline, Embase and Pubmed were systematically searched from inception through to July 2015 using terms for infectious agents, oesophageal adenocarcinoma and Barrett's oesophagus.

No language restriction was applied.

Titles and abstracts followed by full texts of those selected were screened independently in duplicate.

Random effects meta-analysis of proportions were used to examine the association between each infectious agent and BO and OAC, separately, when 4, or more, studies were eligible.

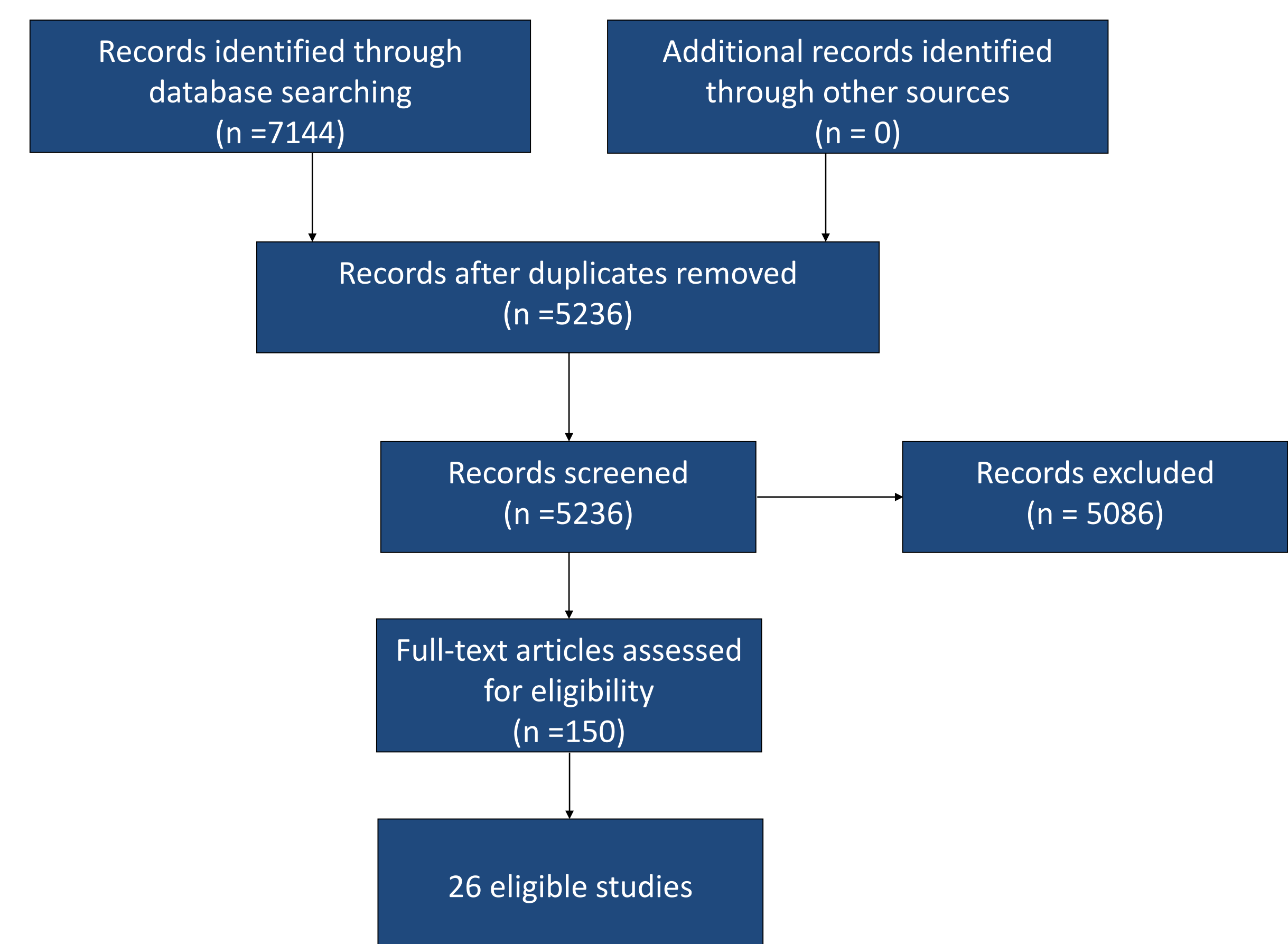


Figure 2: Flow diagram of study selection

Results

The pooled prevalence of HPV in oesophageal adenocarcinoma tumour samples was 17% (n=16 studies, 95% CI: 5-33%) (see figure 3)

- HPV prevalence was higher in oesophageal adenocarcinoma tissue than in oesophageal tissue from healthy controls (n=5 studies, pooled OR=3.83, 95% CI: 1.26-11.63).

The prevalence of HPV in Barrett's oesophagus samples/serum was 27% (n=5, 95% CI: 3-61%).

The prevalence of Epstein-Barr virus in oesophageal adenocarcinoma was 6% (n=5, 95% CI: 0-27%).

Too few studies have assessed other infectious agents.

There was considerable between study variation for each of the analyses (I²=88-97%).

- Sensitivity analyses by study size, study quality, type of tissue and geography did not reveal the source of heterogeneity.

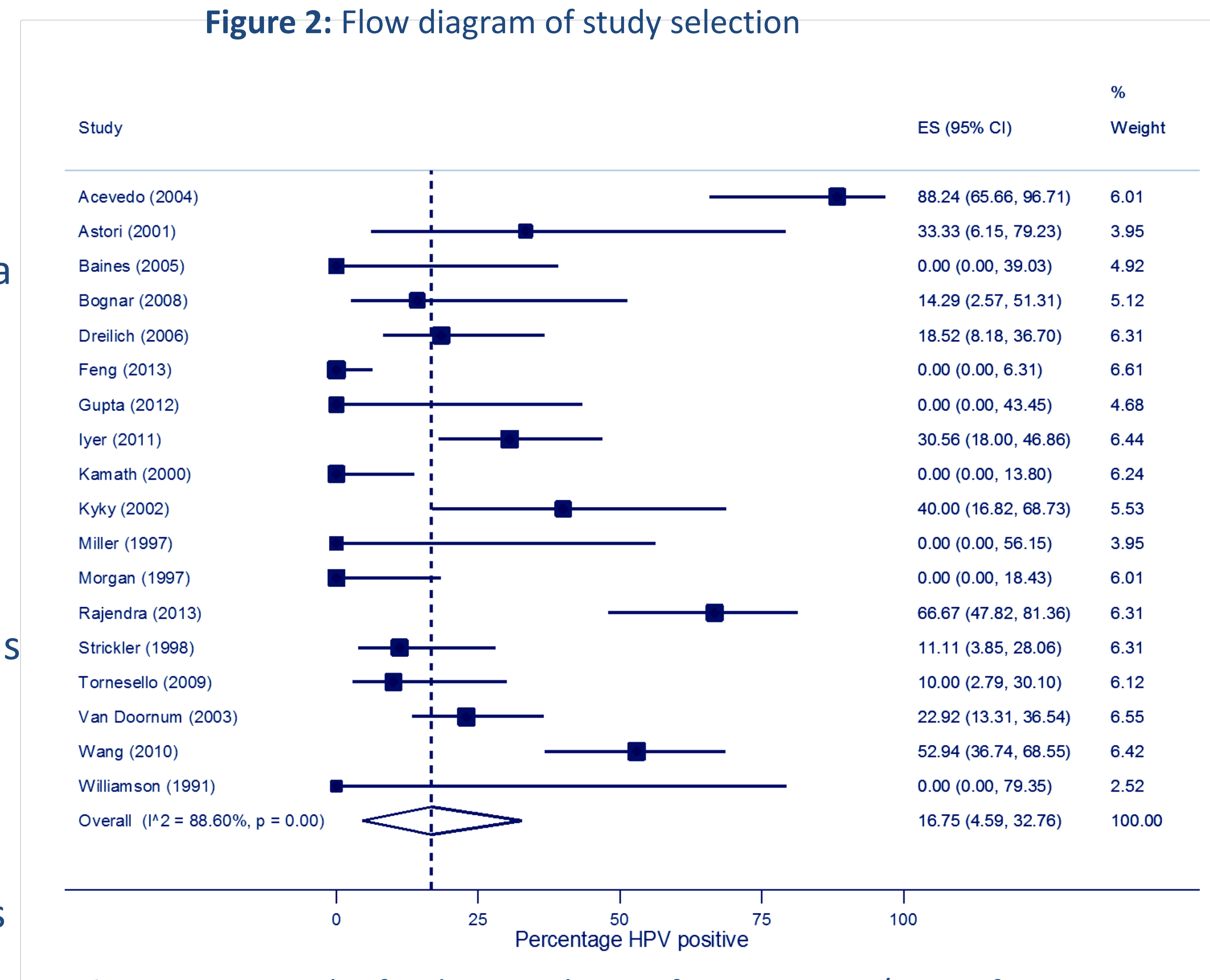


Figure 3: Forest plot for the prevalence of HPV in tissue/serum from oesophageal adenocarcinoma patients

Discussion

The prevalence of HPV and EBV in oesophageal adenocarcinoma is low compared to other viral associated cancers but may have been hampered by small sample sizes and detection methods susceptible to fixation processes.

Additional research with adequate sample size and high quality detection methods is required.

Following completion of this review, a nested case-control study using high quality infectious agent detection methods is planned within a population based sample from Northern Ireland to assess the role of infectious agents in progression from Barrett's Oesophagus to oesophageal adenocarcinoma.