CHAPTER 1

INTRODUCTION

Chlamydia trachomatis is considered to be the most commonly diagnosed treatable bacteria causing sexually-transmitted diseases (WHO, 2012). *C. trachomatis* infection, however, can remain asymptomatic and without evidences of complication at time of diagnosis (Geisler, 2010). The tendency of chlamydial infection to become asymptomatic may lead to the lack of awareness on chlamydial screening and eventually lead to severe complications. Although the disease can affect both men and women, however women are more susceptible to the adverse effects of prolonged chlamydial infection. Like most of STIs, chlamydial infection is studied more thoroughly investigated in women, as the consequences and manifestations are generally more damaging to women's reproductive system compared to men's (Paavonen & Eggert-Kruse, 1999). Specifically, prolonged and untreated chlamydial infection in women can result in pelvic inflammatory disease (PID), spontaneous abortion, premature birth, ectopic pregnancy, and neonatal infections (Chan et al., 2016; Mårdh, 2002).

C. trachomatis is known to infect endometrial epithelial cells at their mucosal surfaces (Rasmussen et al., 1997; Rödel et al., 2012). Therefore, endometrial epithelial cell lines have been utilised for studying *C. trachomatis in vitro*, due the obligate intracellular nature of the bacteria. As a site of chlamydial infection, endometrial epithelial cells are constantly exposed to hormones, which have direct and indirect effects on endometrial epithelial cells (Kintner, Schoborg, Wyrick, & Hall, 2015). Progesterone, on the other hand, is a steroid hormone particularly important for developing and maintaining the regulation of hormone-responsive tissues, including the reproductive tract (Diep, Daniel, Mauro, Knutson, & Lange, 2015). Furthermore progesterone is suspected to inhibit the accumulation of *C. trachomatis* in endometrial cells (Kintner et al., 2015).

In that sense, considering the significant influence of progesterone on endometrial epithelial cells, further investigation of progesterone's effect upon the infectivity of chlamydia may provide more insight on mechanism underlying chlamydial infection. Furthermore, the study may also provide a new insight on methods of interventions, possibly by incorporating treatment with progesterone.

This study was conducted with the aim of identifying the effects of *C. trachomatis* infection on the expression of progesterone-sensitive genes in endometrial epithelial cells, as well as investigating the effects of progesterone exposure upon the expression of the particular genes during chlamydial infection. We therefore hypothesised that *C. trachomatis* infection and progesterone exposure can affect the expression of progesterone-sensitive genes in endometrial epithelial cells.