Adverse outcomes of (sub) clinical thyroid disorders on fertility and subsequent pregnancy may critically determine the necessity to screen and treat women of infertile couples. Thyroid hormones can interfere with normal ovulatory cycle. TSH, its receptor, and thyroid hormone receptors (TR-a1 and TR-b1) have been located in ovarian surface epithelium and in oocytes of primordial, primary, and secondary follicles, thus participating in the regulation of ovarian function. In animal models thyroid hormones synergize with FSH to exert direct stimulatory effects on granulosa cell functions. Thyroid antibodies have been associated with varying causes of infertility, including PCOD, endometriosis and premature ovarian failure. The reason underlying these associations remain largely speculative. Age of patients, organ and non-organ specific autoimmunity, and finally the development of (sub) clinical hypothyroidism have been incriminated. Moreover, the presence of thyroid antibodies when compared with the absence of thyroid antibodies was associated with an increased risk of miscarriage in cohort studies, and in case-control studies. Recurrent miscarriage and preterm birth were more frequently reported. Subclinical hypothyroidism in early pregnancy, compared with normal thyroid function, was associated with an increased risk of pre-eclampsia and perinatal mortality. Although subclinical hypothyroidism in pregnancy might influence neuropsychological development of the offspring, the only controlled intervention study with L Thyroxine during pregnancy was unable to show benefit on this outcome parameter (CAT study). Our own meta-analysis of intervention trials in women with thyroid autoimmunity and/or subclinical hypothyroidism with L Thyroxine in a setting of assisted reproduction technologies, showed that LT4 treatment resulted in a significantly higher delivery rate, with a pooled relative risk (RR) of 2.76 (95% confidence limits 1.20-6.44), a pooled absolute risk difference (ARD) of 36.3% (3.5-69.0%) and a summary number needed to treat (NNT) of 3 (1-28) in favor of LT4 supplementation. LT4 treatment significantly lowered miscarriage rate with a pooled RR of 0.45 (0.24-0.82), a pooled ARD of 231.3% (248.2 to 214.5%) and a summary NNT of 3 (2-7) in favor of LT4 supplementation. LT4 treatment had no effect on clinical pregnancy (RR 1.75; 0.90-3.38). These data indicate that systematic screening of women, consulting for infertility and eligible to undergo ART might be cost saving.