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Letter to the Editor OMICS International

Infertility and Thyroid Autoimmunity

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The birth rate in Europe has reached the very low rate of 1.58 children per woman [1]; in Italy, the rate is even lower, with just 1.35 children per couple recorded in 2015 [2]. In the industrial city of Turin, the rate is less than one child per couple, whilst a rate of two children per woman is considered the standard replacement rate for a population, resulting in relative stability in terms of total numbers. Rates above two children indicate growing populations with a declining median age. Conversely, there is a significant migration rate from foreign countries, mostly from Africa and the Middle East, which causes social unrest. A contributing factor to this low birth rate is infertility, for which cures are thus required. Thyroid autoimmunity can cause low fertility [3], which can be treated with immunosuppressive drugs, even in the absence of functional thyroid damage. Anti-thyroid autoantibodies are often present in women of child-bearing age [4], and up to 51.4% of women with thyroid disorders have circulating anti-thyroid autoantibodies [5].

The age for having the first and only child for working couples in Northern Italy is often ≥ 40 years old, when there is a higher prevalence of thyroid problems. Deroux et al. suggested the use of therapeutic immunomodulators on a case-by-case basis to favor pregnancy in infertile couples with thyroid autoimmunity, even in the case of euthyroid patients, who still carry anti-thyroperoxidase or anti-phospholipid (APL) systemic antibodies [6]. Curing thyroid autoimmunity with immunosuppressive drugs is not without risks for both the woman and the fetus. Hence, we believe it could be worthwhile to assess the presence of causes that have been reported to generate anti-thyroid autoantibodies, such as infection by pathogenic strains of *Helicobacter pylori*, i.e. those expressing the CagA protein; these strains generate an autoantibody response via molecular mimicry [7].

We therefore suggest a less aggressive strategy than immunosuppression, namely to firstly cure the *H. pylori* infection in those infertile couples found to be positive for the bacterium. *H. pylori* infection has been shown in several instances to elicit the abovementioned immune response, specifically anti-peroxidase antibodies, which, despite being specific for the bacterium, could cross react against the human thyrocytes [7,8]. *H. pylori* infection has, in fact, been found to be associated with hypothyroidism in several countries [7,8]. *H. pylori* possess the unique ability to disguise itself as human; it can therefore generate autoimmunity against cells in the stomach, the thyroid, and the trophoblast via molecular mimicry. This may also be the case of APL antibodies that also disappear after eradication of the *H. pylori* infection [9,10].

Regardless of the mechanisms, *H. pylori* infection has been suggested to be an important cause of infertility [11,12]. We therefore propose to include the test for pathogenic strains of *H. pylori* when treating cases of infertility.

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