

## Maternal Hypothyroidism and Rheumatoid Arthritis

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The amount of thyroid hormones (THs) is important for the progress of the development (El-bakry et al., 2010; Ahmed, 2011, 2012a,b, 2013, 2014, 2015a-c, 2016a-d, 2017a-p; Ahmed et al., 2008, 2010, 2012, 2013a,b, 2014; 2015a,b; Ahmed and Ahmed, 2012; Ahmed and Incerpi, 2013; Van Herck et al., 2013; Ahmed and El-Gareib, 2014; Incerpi et al., 2014; Candelotti et al., 2015; De Vito et al., 2015; El-Ghareeb et al., 2016; Ahmed and El-Gareib, 2017; Endendijk et al., 2017; Gigena et al., 2017), particularly the energy expenditure, the differentiation of bone and cartilage and the developing joints and skeletal muscles (contractile function and regeneration) (McLean and Podell, 1995; Croteau et al., 1996; Yu et al., 2000; Peeters et al., 2003; Mebis et al., 2007; Simonides and van Hardeveld, 2008; Grozovsky et al., 2009; Heemstra et al., 2009; Dentice et al., 2010; Marsili et al., 2010 & 2011; Novak and Soukup, 2011; Brent, 2012; Salvatore et al., 2014).

On the other hand, the deviation in the levels of THs can cause muscle pain/weakness or joint disorders (soreness and swelling), myalgias, arthralgias, myopathies, neuropathic and arthritis (McLean and Podell, 1995; Raterman et al., 2008; Garber et al., 2012; Suresh and Wimalaratna, 2013; Bengtsson et al., 2014; Elattar et al., 2014; Villar et al., 2015). In addition, thyroid disorders can cause rheumatoid arthritis (RA; chronic autoimmune systemic inflammatory multisystem disease) (Chan et al., 2001; El-Sherief et al., 2004; Raterman et al., 2008; Mousa et al., 2012; Raterman and Nurmohamed, 2012; Kerola et al., 2014) by increasing the levels of thyroid autoantibody titers (Ilias et al., 1999; Staykova, 2007). More importantly, hypothyroidism can cause aseptic necrosis, epiphyseal dysgenesis, and crystal-

induced arthritis in the knees, wrists, and hands (McLean and Podell, 1995; Elattar et al., 2014). Moreover, hypothyroidism might exacerbate or precipitate the musculoskeletal disease including the malaise, fatigue and dyslipidemia (original RA symptoms) (Arnaout et al., 1994; Surks and Sievert, 1995; Tunbridge and Vanderpump, 2000; Porkodi et al., 2004; Elattar et al., 2014). Several authors reported that the association between the thyroid diseases and RA might increase the risk of cardiovascular disease (Biondi and Klein, 2004; Dessein et al., 2007; Dhawan and Quyyumi, 2008; Raterman et al., 2008, 2010 & 2012; Schott et al., 2009). Overall, the communication between all these disorders can cause symmetric polyarthritis, bone destruction, extra-articular manifestations (EAMs), disability, comorbidities, and premature mortality (Cadena et al., 2003; Anaya, 2006; Carlé et al., 2006; Rojas-Villarraga et al., 2009).

On the basis of these data, it can be depicted that the normal functions of the maternal THs may be important for the development of muscle and joint. In addition, any disturbance in the levels of maternal THs (hypothyroidism) may perturb the burn energy, the amount of fluid builds in joints and their metabolites causing swelling and pain. These disorders may increase the risk of developing rheumatoid arthritis. Though, the mechanism of this interruption remains indefinite. I advise to measure the levels of THs [thyroxine (T4), 3,5,3'-triiodothyronine (T3)], thyroid-stimulating hormone (TSH) and thyroid autoantibodies in all cases of RA. Obviously, investigation in this issue is still in its infancy.

### CONFLICT OF INTEREST

The author declares that no competing financial interests exist.

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