The Impact of Thyroid Dysfunction on Lactation
Lisa Marasco, MA, IBCLC

Thyroid hormones play an integral role in mammary function. During lactation, they aid in the regulation of both prolactin and oxytocin. Nevertheless, studies on the impact of thyroid dysfunction on human lactation have been scant. It is generally understood that hypothyroidism can interfere with milk production, but the potential impact of hyperthyroidism or postpartum thyroiditis has remained obscure. Recent studies may provide some new insights. A decade ago, Joshi et al. (1993) noted that lactation failure preceded the clinical evidence of thyroid problems in a number of their study subjects. It was their conclusion that such history should be considered when assessing for potential thyroid dysfunction, implying that lactation failure due to thyroid dysfunction could occur without clear laboratory evidence.

Thyroid disorders can be primary or secondary, clinical or subclinical. Many thyroid problems are the result of autoimmune problems. Thyroid disorders are more common in women and may begin before or during pregnancy, postdelivery, or later in life. They can also occur concomitantly with other medical conditions; for instance, women with polycystic ovary syndrome (PCOS) have an increased incidence of autoimmune thyroiditis.

Hypothyroidism. The most common form of hypothyroidism is Hashimoto’s disease. It is usually diagnosed based on high levels of thyroid stimulating hormone (TSH) and low levels of triiodothyronine (T3)/tetra-iodothyronine (thyroxine or T4). Standard laboratory ranges for these hormones are derived from nonpregnant populations, though some experts suggest that the TSH upper limit for early pregnancy be lowered to 2.0-2.5 uIU/L. This remains controversial. Tighter standards might also be applied to pre-conception and later pregnancy, and if extended through lactation might allow some struggling breastfeeding mothers to obtain help sooner. When hypothyroidism is diagnosed, thyroid hormone replacement is the first-line treatment. Untreated, hypothyroidism during pregnancy can cause a number of problems, including pregnancy-induced hypertension and low birth weight. These in themselves are risk factors for delayed lactation or insufficient milk production.

Buckshee et al. found a lactation deficiency rate of 19.2% in their study mothers with hypothyroidism despite hormone replacement treatment during pregnancy. In a case with a positive outcome, a 30-year-old mother of three children presented with her four-month-old infant who was beginning to fall off his weight curve. The mother complained of fatigue and depression, and the pediatrician noted a lowered feeding frequency and
attributed baby’s weight-gain problem to mother’s stress and exhaustion. When the mother’s sister, a medical student, commented on the mother’s puffy face and suggested the possibility of a hormone imbalance, lab tests led to a diagnosis of Hashimoto’s thyroiditis (hypothyroid phase). Thyroid replacement was begun, the mother’s energy returned quickly, her milk production increased, and formula supplementation was no longer necessary.

Despite a general awareness that hypothyroidism can interfere with successful lactation, there has been little research into the mechanisms of that interference until recently. In 2003, Hapon et al. induced hypothyroidism in rats before mating and compared these rats to controls during lactation. They discovered no differences in suckling-induced prolactin release but did find a reduction in circulating oxytocin after suckling. The treated mothers released less milk than did control mothers, resulting in poor milk transfer and poor litter growth. 11

Hyperthyroidism. Hyperthyroidism is evidenced by a reduction in TSH and an increase in T3/T4. The most common form is Grave’s disease. The rate of hyperthyroidism during pregnancy is 2:1000. 12 Pregnancy tends to induce a mild degree of hypothyroidism in most women due to increased rates of plasma clearance of T3 and T4. 11 Previously hyperthyroid women may experience an attenuation of their symptoms, especially during the second and third trimesters, but symptoms may rebound shortly after delivery. 13,14 Hyperthyroidism in pregnancy can result in obstetric complications such as preterm delivery, pre-eclampsia, fetal growth restriction, and increased maternal and fetal mortality.

Recent animal studies have shed some light on the impact of hyperthyroidism on lactation. Excessive thyroid hormone seems to accelerate mammary growth. While hypothyroid rats have smaller litters and longer gestations, hyperthyroid rats have larger litters, prolonged labors, and earlier onset of labor and lactogenesis. When severe hyperthyroidism was induced before mating and maintained through pregnancy, Rosato et al. 15 noted good lobulo-alveolar growth and evidence of lactogenesis II, yet complete lactation failure occurred. This suggests a problem with oxytocin release and milk ejection. The authors also observed defects in maternal behavior among many of the mother rats which were likely related to the oxytocin deficit.

A study by Varas et al. 16 examined the effects of induced moderate hyperthyroidism in rats. The rats were able to lactate, but impairment of milk ejection led to apoptosis, involution, and litter death over time. Histological studies again showed functional mammary tissue with “distended alveoli” but “almost total absence of adipose tissue.” In an acute suckling test at 21 days postpartum, significantly smaller increases in serum prolactin and oxytocin concentration were found in treated rats compared to controls after 30 minutes of suckling.

Two recent human cases have involved multigravid women who delivered prematurely secondary to poorly controlled hyperthyroidism. In each case, lactation was severely suppressed with neither woman able to express colostrum. Standard lactation management strategies were tried without success. 17 When lactogenesis stage II seems to have occurred, yet the milk “just isn’t coming out,” the possibility of hyperthyroidism should be
considered, in the absence of other explanations. If diagnosed, the first line of treatment is to lower the level of thyroid hormones. Lao recommends propylthiouracil (PTU) as the drug of choice for breastfeeding mothers because it is excreted in insignificant amounts in breast milk and does not depress neonatal thyroid function. Methimazole is suggested as an acceptable option if the infant can be monitored frequently. 14

Postpartum thyroid dysfunction. There are four possible types of postpartum thyroid dysfunction. Postpartum thyroiditis (PPT) is the most common, while postpartum Graves’ disease comprises 15% of cases. Postpartum pituitary infarction (Sheehan’s Syndrome) and lymphocytic hypophysitis are secondary forms of postpartum thyroid dysfunction that have a negative impact on lactation. They are more rare and affect thyroid hormones through pituitary dysfunction. 18 Postpartum thyroiditis (PPT) occurs after approximately 5-7% of all pregnancies. 20 Women with diabetes mellitus type 1 have 3 times the risk of developing PPT. 21 The risk is also 3 times as great among smokers who smoke more than 20 cigarettes daily. 22 The classic form of PPT starts with a transient hyperthyroidism that lasts for a few to several weeks, then transitions to a hypothyroid state, which may last a few to several months. 19 The hypothyroid state is usually more obvious clinically, leading to diagnosis. In some cases onset may be days after delivery, accompanied not only by signs of hyperthyroidism but also by severe hypertension. 20 Historically, when detected early, physicians have often elected not to treat until the hypothyroid stage, unless symptoms are severe. (For symptomatic thyrotoxicosis, Lao recommends a short course of beta-blockers such as propanolol or atenolol, but discourages antithyroid drugs. 14) Non-classic forms of PPT may start with hypothyroidism and move to hyperthyroidism, or they may simply be one or the other exclusively. In a review of 13 prevalence studies, Stagnaro-Green found the most common presentation of PPT was hypothyroidism without preceding hyperthyroidism (43%). Hyperthyroidism presenting alone represented 32% of cases, while the classic presentation of hyperthyroidism followed by hypothyroidism occurred in only 25% of cases. Regardless of the form, 80% of mothers with PPT return to normal thyroid function by the end of 12 months, though the risk of permanent thyroid disease with postpartum hypothyroidism may be as great as 50%. There is also a high recurrence risk with future pregnancies. 19

Improving lactation outcomes. Impaired release of milk, rather than impairment of milk synthesis, appears to be the greater obstacle with both hyper- and hypothyroidism. Without adequate milk removal, increased concentrations of feedback inhibitor of lactation protein in residual milk trigger downward regulation of milk synthesis, resulting in suppressed milk production and eventual involution of the gland. Thus, improving milk release may improve lactation when thyroid hormones are dysfunctional. Exogenous pitocin (i.e., pitocin nasal spray) might provide the oxytocin necessary to eject milk. Massaging the breast from the chest toward the nipple prior to feeding may make more milk available to baby. Breast compression, which mechanically increases internal pressure, may also help to propel milk from the breast during the feed. 23 Galactogogues would be effective only in the presence of a functioning milk ejection reflex and
will work best when thyroid hormones are in balance. They should be considered supportive, not first-line therapy, but may be useful adjunct therapy when milk production has suffered.

Greater recognition of the impact of thyroid dysfunction on lactation and timely, accurate diagnosis with appropriate treatment of affected mothers will enable mothers to provide sufficient milk to their babies. In time, a greater understanding will also lead to more effective treatments and improved outcomes.

References:
15. Rosato, R., M. Gimenez, and G. Jahn. Effects of chronic thyroid hormone administration on pregnancy, lactogenesis, and lactation in the
Last edited October 22, 2006 by chj.
Copyright © 2006 by La Leche League International and those posting information. All Rights Reserved.
This site is for your information only. For medical advice consult a health professional. For more information, please see our Terms of Use.
Contact Us | Terms of Use | Privacy Policy