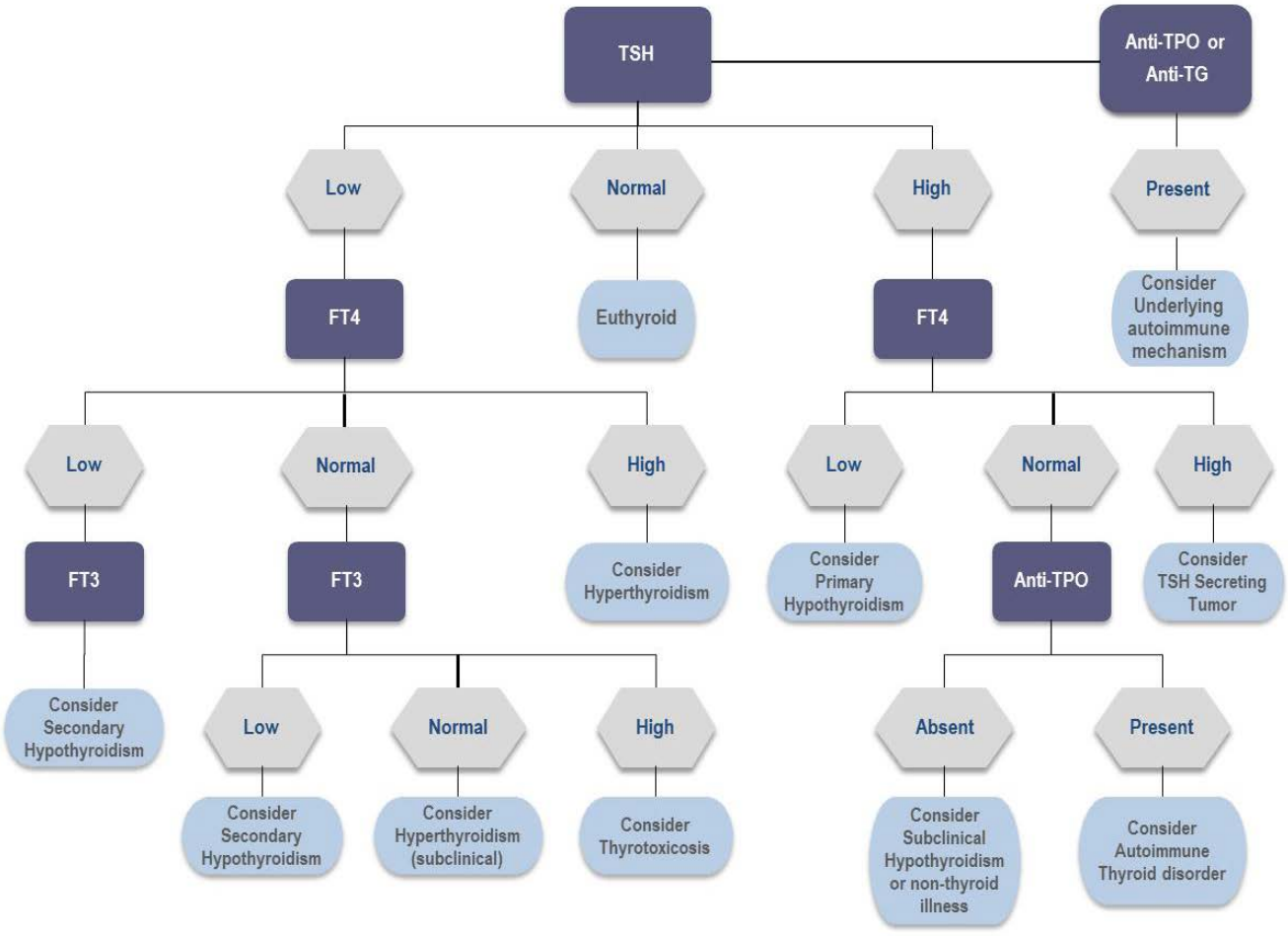
LAB #: Sample Report
PATIENT: Sample Patinet
ID:
SEX: Female
DOB: 01/01/1951 AGE: 67

CLIENT #: 12345
DOCTOR: Sample Doctor
Doctor's Data, Inc.
3755 Illinois Ave.
St. Charles, IL 60174 U.S.A.

Thyroid Profile; serum

	RESULT / UNIT	REFERENCE INTERVAL	PERCENTILE (2.5th 16th 50th 84th 97.5th; 95th 99th)
Free T3	**3.9 pg/mL**	2.2- 4.0	
Free T4	**0.8 ng/dL**	0.6- 1.3	
Thyroid Stimulating Hormone (TSH)	**0.01 µIU/mL**	0.30- 4.5	
Thyroglobulin Antibody (Anti-TG)	**1.1 IU/mL**	< 4.0	
Thyroid Peroxidase Antibody (Anti-TPO)	**250 IU/mL**	< 9.0	



This diagnostic algorithm is intended for baseline assessments only and may not be accurate for patients on thyroid medications.

SPECIMEN DATA

Comments:

Date Collected: **02/18/2019** Time Collected:
Date Received: **02/20/2019** Fasting:
Date Completed: **02/21/2019**
Methodology: **Chemiluminesescent Immunoassay**

Intro

The analysis of thyroid hormones and antibodies together may improve the accuracy of diagnosis and clinical success. The American Thyroid Association estimates that approximately 20 million Americans have thyroid disease, and approximately 60% of those with thyroid disease are unaware of their condition. The analysis of thyroid stimulating hormone, free thyroid hormones and thyroid antibodies may best distinguish thyrotoxicosis from hypothyroidism and the euthyroid state. Less than one percent of thyroid hormone is free unbound hormone; this one percent is the biologically active fraction. Total T4 and T3 values cannot be reliably used to diagnose conditions due to inherited and acquired variations in the concentration of thyroid hormone binding proteins. The recognition of auto-immunity as a leading cause of thyroid dysfunction has led to the evaluation of auto-antibodies in thyroid testing. Thyroid antibody tests are used to distinguish autoimmune thyroid disorders from other thyroid dysfunction.

The synthesis and metabolism of thyroid hormone requires the precursor amino acid tyrosine, iodine, selenium and vitamin A. Abnormal results on the Thyroid Profile may prompt additional testing to evaluate nutrient status. Commentaries are pesented in this report when abnormal results have been detected.

References:
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Laboratory Evaluation of Thyroid Function.
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Weetman, Anthony P (2005) Non-thyroid autoantibodies in autoimmune thyroid disease.
Best practice & research. Clinical endocrinology & metabolism vol. 19 (1) p. 17-32

Thyroid Stimulating Hormone (TSH) Low

The level of thyroid stimulating hormone (TSH) is lower than expected in this sample. Serum TSH is considered the single best test of thyroid function, and with thyroid hormone measurements, provides the most comprehensive assessment of thyroid function. TSH is secreted from the pituitary gland when the pituitary is stimulated by thyrotropin-releasing hormone (TRH), which is released from the hypothalamus. The release of thyroxine (T4) and triiodothyronine (T3) from the thyroid gland into circulation is controlled by the secretion of TSH. T3 and T4 control the synthesis and secretion of TSH and thyrotropin releasing hormone (TRH) at the level of pituitary and hypothalamus, through a negative feedback loop. Normally, if T3 and T4 increase, TSH decreases. Conversely, if T3 and T4 decrease, TSH increases.

Low TSH with high or normal T4 and T3 indicates hyperthyroidism or thyroiditis; this may also occur with thyroid hormone replacement therapy.

Low TSH with low T4 and T3 indicates secondary hypothyroidism. The hypothyroid condition is secondary to a disorder in the pituitary gland or the hypothalamus. This pattern may also occur if a non-thyroid illness is present.

Low TSH may occur during the first trimester of pregnancy. Lower TSH levels may occur in geriatric patients, after hyperthyroid episodes, or in chronic diseases ("sick" euthyroid). Medications such as glucocorticoids, dobutamine and dopamine may decrease TSH levels. Metformin has been shown to decrease TSH levels in type II diabetics with hypothyroidism.

TSH levels may also decrease if thyroid hormone replacement is excessive, if gastrointestinal absorption is altered, or if T4 clearance is altered. The liver is the primary site of T4 and T3 degradation, via iodothyronine deiodinase enzymes. Thyroxine may also be metabolized in the kidney. Thyroid hormones may also be metabolized through conjugation with glucuronides and sulfates. They are excreted directly into the bile, which is released into the gastrointestinal tract, where the thyroid hormones may undergo enterohepatic recirculation.

References:
Fournier, Jean-Pascal; Yin, Hui; Yu, Oriana Hoi Yun; Azoulay, Laurent (2014)
Metformin and low levels of thyroid-stimulating hormone in patients with type 2 diabetes mellitus
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http://www.clevelandclinicmeded.com/medicalpubs/diseasemanagement/endocrinology/hypothyroidism-and-hyperthyroidism/#cesec6
Accessed 09 January 2015

Thyroid peroxidase Antibodies (Anti-TPO) High

The level of thyroid peroxidase (TPO) is higher than expected in this patient sample. TPO antibody levels are considered the most sensitive measure for autoimmune thyroid disease, including Hashimoto's disease, idiopathic myxedema, and Grave's disease. TPO may also be elevated if other autoimmune diseases are present. Good association between elevated anti-TPO and the confirmation of thyroiditis by histology. Up to 90% of Hashimoto's patients and 60-80% of Grave's disease patients will have elevated anti-TPO antibodies. Elevated anti-TPO levels increase the risk of hypothyroidism in sub-clinical hypothyroid patients, and increases the risk that other autoimmune diseases may develop.

TPO is an enzyme on the thyroid hormone synthesis pathway; it is normally found only in the follicular cells of the thyroid gland. Congenital hypothyroidism occurs when mutations in the DNA coding for TPO cause defective enzyme function. Thyroid follicles may be destroyed by inflammation, goiter, or rarely, cancer. The release of TPO into the blood causes an antigenic response, and anti-TPO is formed. Anti-TPO enzymes activate complement, and may contribute to thyroid dysfunction and the development

of hypothyroidism.

References:

1. Feldt-Rasmussen U: Analytical and clinical performance goals for testing autoantibodies to thyroperoxidase, thyroglobulin, and thyrotropin receptor. Clin Chem 1996;42:160-163

2. Genetics Home Reference TPO http://ghr.nlm.nih.gov/gene/TPO Accessed 13 January 2015

3. Gharib H, Tuttle RM, Baskin HJ, et al: Consensus Statement #1, Subclinical thyroid dysfunction: a joint statement on management from the American Association of Clinical Endocrinologists, The American Thyroid Association, and The Endocrine Society. Thyroid 2005;15:24-28 National Academy of Clinical Biochemistry: Laboratory Medicine Practice Guidelines.

4. Edited by LM Demers, CA Spencer. Laboratory support for the diagnosis and monitoring of thyroid disease, Section D. Thyroid antibodies (TPOAb, TgAb, TRAb), pp 43-54, and Section E. Thyroglobulin (Tg), pp 55-65 - reprinted in unchanged form in Thyroid 2003;13(1):45-56, 57-67

Thyroid Antibodies

Thyroid antibody tests are used to distinguish autoimmune thyroid disorders from other thyroid dysfunction. Thyroid antibody tests are most important in patients with other, pre-existing autoimmune conditions such as systemic lupus erythematosus, rheumatoid arthritis, Celiac disease, etc. Autoimmune thyroid antibodies may result in high or low thyroid hormone levels.

Thyroxine (T4) is converted to triidothyronine (T3) in the peripheral tissues. Most T4 is converted to T3 by selenium-dependent iodothyronine deiodinase enzymes. Iodine is essential for the synthesis of thyroid hormones. The thyroid gland expresses several selenoproteins as protection against the hydrogen peroxide and oxidative stress generated during thyroid hormone synthesis. Individuals with mutations in the DNA that codes for selenoprotein synthesis factor have an increased risk of thyroid function defects. Iodine deficiency may be exacerbated by selenium, iron or Vitamin A. Low selenium levels have been associated with goiter and thyroid nodules in European women. Several randomized controlled trials have found that selenium supplementation decreases thyroid-disease-specific antibody levels, and may improve time to remission. The effects of selenium supplementation appear to be dependent on the patient's initial selenium status; those with the lowest selenium levels demonstrated the greatest responses to selenium supplementation. If thyroid antibodies are elevated, consider:

-Iodine status and presence of goitrogenic halides (Urine Iodine or Urine Halides)
-Mineral status (RBC Elements)
-Digestion and absorption of nutrient minerals (Comprehensive Stool Analysis with Parasitology x 3)

References:

1. American Association for Clinical Chemistry (2014) Thyroid Antibodies www.labtestsonline.org Accessed 08 January 2014

2. Ch'ng, Chin Lye; Jones, M Keston; Kingham, Jeremy G C (2007) Celiac disease and autoimmune thyroid disease. Clinical medicine & research vol. 5 (3) p. 184-92

3. Weetman, Anthony P (2005) Non-thyroid autoantibodies in autoimmune thyroid disease. Best practice & research. Clinical endocrinology & metabolism vol. 19 (1) p. 17-32

4. Schomburg, Lutz (2012) Selenium, selenoproteins and the thyroid gland: interactions in health and disease. Nature reviews. Endocrinology vol. 8 (3) p. 160-71