

# Mary J. Shomon

## Patient Advocate, Author

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November 25, 2005

Bill Law Jr., MD  
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American Association of Clinical Endocrinologists  
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Dear Dr. Law,

I appreciate your acknowledging my contact with you and AACE by responding in your letter of November 23, 2005. Thank you for writing.

As a patient advocate, whose objective is clear communications with the thyroid patient community, I must point out, however, that you neglected to answer any of the questions in my letter. By failing to address any of the legitimate questions, your assertions still lack specific citations or evidence-based supporting information.

For the sake of the credibility of AACE and your public positions, I had hoped that you were willing and able to stand behind your statements about hypothyroidism and its treatment by providing the "solid scientific evidence" to support them. I remain hopeful.

I will willingly publish your letter of response as soon as I receive it. So, again, to recap the questions detailed in my last letter.

\* \* \*

FIRST, you have said that while "such symptoms are also very common in the general population, most of who *[sic]* do NOT have hypothyroidism and will NOT experience any sustained improvement in their symptoms with thyroid hormone therapy."

By AACE's own recommendation, as many as 1 in 5 people in the U.S. are hypothyroid -- and likely suffering from its symptoms -- but most are not yet diagnosed. AACE has strongly recommended that the "normal" range of the Thyroid Stimulating Hormone (TSH) test be narrowed to 0.3 to 3.0, from the current range of approximately 0.5 to 5.0. A study reported on in the *Journal of the American Medical Association* found that using a TSH upper normal range of 5.0, approximately 5% of the population is hypothyroid.

However, if the upper portion of the normal range was lowered to 3.0, approximately 20% of the population -- as many as 59 million people -- would be hypothyroid. (1)

**QUESTION: We don't know how many members of the public suffer from symptoms such as fatigue, depression, weight gain and are NOT hypothyroid, but the current standards -- vis a vis AACE's recommended standards -- exclude 15% of the people who are hypothyroid. If by AACE's own standards, as many as 20% of the population are hypothyroid, how can you say that most of the people in the general population who have hypothyroid-like symptoms will not improve with thyroid hormone treatment?**

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SECOND, you also claim that "Inappropriate thyroid hormone treatment with ANY preparation can lead to thin bones, known as osteoporosis..."

This claim is most definitely *not* backed up by a large body of solid scientific evidence. Certainly, there are some studies that show a concern regarding bone density. But there are valid, reputable studies that fail to support this position as well. The issue is an ongoing, undecided and quite controversial topic. To suggest otherwise is misleading, and not acknowledging the valid positions of researchers.

For example, a study released in June, 2000, at the World Congress on Osteoporosis in Chicago, Illinois, found that taking thyroxine does not increase the risk of osteoporosis. The research, presented by Dr. Martin Stenstrom of the University of Gothenburg in Sweden, studied more than 750 women who were taking prescribed thyroid medication for thyroid disease. Over an 18 month period, bone mineral density was measured, and compared to a control group who were not taking thyroid hormone. No differences were noted in bone mineral density between those taking the thyroid hormone, and the control group.

An October, 1998 study reported on in the *Journal of Gynecological Endocrinology* found that levothyroxine suppressive therapy, if carefully carried out and monitored, has no significant effect on bone mass. (2)

The highly regarded *Journal of Clinical Endocrinology and Metabolism* found that even "suppressive," levothyroxine therapy -- prescribing medicine that lowers TSH levels to hyperthyroid levels below normal range -- if carefully carried out and monitored, has no significant effect on bone metabolism or bone mass. (3)

A major thyroid-related journal, *Thyroid*, found that long-term levothyroxine therapy using suppressive doses has no significant adverse effects on bone. (4)

In 1998, the *Journal of Hormonal and Metabolic Research* found that there was no difference in bone mineral density between thyroid patients and controls, and that the main factor in bone density and bone turnover is menopausal status. The researchers found that slightly suppressive levothyroxine doses constitute neither an actual risk factor for bone loss nor, consequently, for osteoporotic fractures. (5)

These are only a selection of the peer-reviewed research articles published in major medical journals that dispute your statement.

**QUESTION: How can AACE claim that "Inappropriate thyroid hormone treatment with ANY preparation can lead to thin bones, known as osteoporosis..." without mentioning that this position is quite controversial, that there is legitimate and reputable research that disputes this position, and that the position is certainly *not* definitive or scientifically conclusive?**

\* \* \*

THIRD, you claim that "Animal-derived desiccated thyroid...is not a natural form of thyroid replacement for humans at all."

This argument is fundamentally misleading. Since Armour is derived from the thyroid glands of pigs, it is true that it is not "natural" to humans. But the favored drug of the AACE, levothyroxine, is synthetically manufactured. Synthroid and levothyroxine, therefore, are not a natural form of thyroid replacement for humans either.

**QUESTION: Are you suggesting that levothyroxine is somehow natural to humans, or "more natural" than desiccated thyroid?**

FOURTH, you also refer to desiccated thyroid as "an obsolete product."

I am not aware of any double-blind, peer-reviewed, journal published research demonstrating solid scientific evidence that establishes that today's prescription desiccated thyroid drugs are obsolete, or anything less than equally effective as synthetic thyroid drugs.

**QUESTION: Can you please point patients and practitioners to some specific research that scientifically supports the opinion that prescription desiccated thyroid drugs are obsolete?**

\* \* \*

FIFTH, you claim that that desiccated thyroid is "obtained from ground-up cattle and pig thyroid glands." It's true that some of the non-prescription glandular supplements are made of various thyroid gland ular material from various animals. But physicians and practitioners who *prescribe* natural thyroid are working with prescription desiccated thyroid drugs that are regulated by the Food and Drug Administration. These drugs are all made of the thyroid glands of pigs. Cow thyroid has not been used for years.

**QUESTION: Can you please explain why you make the claim that prescription desiccated thyroid is made from cattle, when that is not true of the currently prescribed prescription drugs, nor has it been true for years?**

\* \* \*

SIXTH, you claim that it's "extremely difficult for even a trained specialist to properly adjust the dose to fit each patient's needs."

In 2004, more than 2 million prescriptions were written for Armour Thyroid alone. There are clearly many doctors who are able to properly adjust the dose to fit their patients' needs, and who do not find it extremely difficult.

Also, a substantial percentage of patients taking the AACE preferred form of thyroid hormone replacement -- levothyroxine -- are not being properly adjusted by their physicians. (For comparison purposes, in 2000, approximately 40 million prescriptions were written for Synthroid brand levothyroxine, vs. less than 2 million for Armour Thyroid, the leading brand of desiccated thyroid.) In 2000, the *Archives of Internal Medicine* published the results of the Colorado Thyroid Prevalence Study. (6) That study found that a full 40% of patients taking thyroid medications had abnormal TSH levels. The fact that *40% of thyroid patients -- millions of Americans -- are taking thyroid hormone (most often levothyroxine) but still not in TSH range*, is evidence that proper adjustment of levothyroxine is a huge challenge for practitioners, and should be a major focus of AACE's educational efforts.

**Note:** that study was based on the older .5 to 5.0 TSH guidelines, and according to the newer .3 to 3.0 range recommended by AACE, the number of treated patients who fall outside the range is almost certainly much higher, perhaps even more than half.

**QUESTION: I am not aware of any scientific evidence to show that patients being treated with Armour or desiccated thyroid are not receiving proper doses to maintain euthyroidism or resolve symptoms. Can you share any citations or evidence that supports this? Additionally, how does AACE explain the difficulty in achieving euthyroidism among patients on levothyroxine, as evidenced by the Colorado Thyroid Prevalence Study? Does AACE have any plans to help educate endocrinologists and other physicians on proper dosage adjustment of levothyroxine, to help reduce the 40% rate of treat patients who fall outside the euthyroid TSH range?**

SEVENTH, you encouraged Michael Bass of CBS to "view AACE's guidelines on thyroid disorders at [www.aace.com](http://www.aace.com)." These guidelines for hypothyroidism and hyperthyroidism were last updated in December 2002, three years ago. In your recent letter to me, you mentioned that "all of our medical guidelines for clinical practice are evidence-based." Yet, as you know, numerous important research findings and advancements in the treatment of these conditions have been made. Specifically, recent findings on hypothyroidism's impact on fertility and pregnancy, substantially depart from these guidelines. Physicians following these guidelines will compromise a woman's ability to get pregnant, and should she become pregnant, she is at increased risk of miscarriage if these guidelines are followed, rather than newer recommendations for early testing, dosage increases, and frequent monitoring of more than just TSH levels in pregnant patients.

Just a few of the post-2002 hypothyroidism research findings that provide new evidence on fertility, pregnancy and infant diagnosis and treatment practice include:

- Alexander, Erik K. M.D., et. al. Timing and Magnitude of Increases in Levothyroxine Requirements during Pregnancy in Women with Hypothyroidism, *New England Journal of Medicine*, Volume 351:241-249 July 15, 2004 Number 3
- Casey. B. "Maternal Hypothyroidism: Maternal Fetal Outcomes." Endocrine Society Annual Meeting, May 2005. [S7-2]
- Wolfberg A, et. al. "Obstetric and neonatal outcomes associated with maternal hypothyroid disease." *J Matern Fetal Neonatal Med.* 2005 Jan;17(1):35-9
- Spencer, Carole. "Thyroid Function Tests & Pregnancy: What's Normal?[S7-1]" *Endo 2005 Abstracts*
- Forrest, Douglas, "News & Views: The Developing Brain and Maternal Thyroid Hormone: Finding the Links," *Endocrinology*, 145 (9):4034-4036, 2004.
- Fisher, Delbert, "Editorial: Next Generation Newborn Screening for Congenital Hypothyroidism?" *The Journal of Clinical Endocrinology & Metabolism*,90 (6):3797-3799, 2005.
- Maniatis, Aristides et. al. "Congenital Hypothyroidism and the 2nd Newborn Metabolic Screen in Colorado [OR10-1]." *Endo 2005 Abstracts*
- Kempers, M.J.E., et.al., "Disturbance of the Fetal Thyroid Hormone State Has Long-Term Consequences for Treatment of Thyroidal and Central Congenital Hypothyroidism," *The Journal of Clinical Endocrinology & Metabolism*, 90 (7):4094-4100, 2005.
- Spencer, et. al. "Thyroid Reference Ranges in Pregnancy: Studies on an Iodine Sufficient Cohort" Abstract, 13th International Thyroid Congress, Argentina, Fall 2005

These are just a few of the many studies that affect many aspects of hypothyroidism diagnosis and treatment practice.

**QUESTION: Given the number of new advances in thyroid treatment -- changes that can have an impact on the evidence-based approaches to successful treatment of thyroid disease in pregnancy, and may help prevent disabilities in newborns -- why has it been three years since the AACE's hypothyroidism and hyperthyroidism practice guidelines have been updated to reflect the new evidence?**

\* \* \*

While it's not a medical question or issue per se, I do believe it's important to ask why, when communicating publicly regarding your positions in support of levothyroxine, and critical of desiccated thyroid, AACE does not publicly disclose its close financial relationship with Abbott Labs, the manufacturer of Synthroid?

Naturally, the failure to openly disclose this relationship raises ethical questions regarding AACE's ability to provide unbiased recommendations to physicians and patients.

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**QUESTION: Will AACE be publicly disclosing its financial relationships with Abbott and other drug companies when promoting drugs from whom it receives financial support, or attacking the competitors of its financial supporters? What percentage of AACE's overall operating budget and/or outside support does Abbott Labs' support constitute?**

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My objective is to provide honest, unbiased information to my readers in the thyroid community. Since the use of desiccated thyroid, the risk of osteoporosis, obsolescence of drugs, the use of potentially dangerous ingredients (such as cattle) , and successful pregnancy with thyroid disease are all very important issues for patients with hypothyroidism, I know that AACE will stand behind its positions, and clarify for patients by providing the "solid scientific evidence" to support your positions.

Thank you again, and I look forward to your response and clarification of these issues of importance to the thyroid community.

Sincerely,

Mary J. Shomon  
Patient Advocate, Author

#### **Citations**

- 1 Fatourehchi V, Klee GG, Grebe SK, et al. Effects of reducing the upper limit of normal TSH values. *Journal of the American Medical Association*. 2003;290:3195-3196.
- 2 "Bone mineral density in premenopausal women receiving levothyroxine suppressive therapy."). *Gynecol Endocrinol* 1998 Oct;12(5):333-7,
- 3 "Carefully monitored levothyroxine suppressive therapy is not associated with bone loss in premenopausal women." *J Clin Endocrinol Metab* 1994 Apr;78(4):818-23,
- 4 "Suppressive doses of thyroxine do not accelerate age-related bone loss in late postmenopausal women.") *Thyroid*, 1995 Feb;5(1):13-7,
- 5 "A slightly suppressive dose of L-thyroxine does not affect bone turnover and bone mineral density in pre- and postmenopausal women with nontoxic goitre.") *Horm Metab Res* 1995 Nov;27(11):503-7,
- 6 Canaris, Gay J. MD, MSPH; et. al. "The Colorado Thyroid Disease Prevalence Study," *Arch Intern Med*. 2000;160:526-534.