

# My Personal "trial" of Cyclic Testosterone Therapy



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I previously posted about my proposed personal experimental "trial" of high dose cyclic testosterone therapy for my severe hypogonadism (testosterone deficiency syndrome from ADT). Now I have completed both parts of the initial cycle. Note that this is NOT a formal clinical trial. It is a personal experiment of an unproven therapy in my context of non-metastatic hormone-sensitive advanced PC.

My PSA over the past 16 months (following PLN RT and 6 months ADT) has been low but not undetectable. My prostate and pelvic lymph nodes (including 2 PSMA positive nodes) have been previously treated. So I presumably have micro-metastatic: cancer somewhere at sites unknown, similar to BCR. I am going to have a repeat PSMA PET scan done next month.

My experiment with cyclic testosterone is being done with the knowledge and cooperation of my MO, but only after much lobbying, and finally support from another clinician, Michael Schweitzer in Seattle who is an active researcher in the BAT trials. And furthermore, giving written informed consent of my acceptance of the risks, known and unknown.

Yesterday I had an additional video consultation with Dr. Tomaz Beer at OHSU, who also gave a vote of support for my use of cyclic testosterone at this time.

My hypogonadism was not just unpleasant symptoms such as hot flushes, etc. But I had lost much muscle strength and lean body mass to where I had a spinal disc collapse resulting in nerve root compression requiring 2 surgeries over the past 2 months. And my body had not responded to high-level resistance training. My weight and strength just kept dropping (called sarcopenia).

Previously it has been noted on this site that, in the BAT (bipolar androgen therapy) clinical trials for advanced prostate cancer, that it was not possible to predict which patients would respond favorably to BAT and who would respond adversely with rapidly and persistent rise of their PSA. However, it was possible identify who was responding favorably and who was not. Essentially all PCa patients have some rise of PSA when starting testosterone. This is a permissive effect from overcoming the suppression of castrate T levels. But for favorable responders, this was a moderate rise that did not continue. Their clinical course on BAT was benign and in many cases beneficial clinically. I won't review the BAT trials results again here as this has been done previously. Unfavorable responders to BAT had persistent and progressive rises of PSA. In the trials those patients stopped it and were removed from further participation in BAT. The trend was that their PSA, back on ADT, dropped back down and there were generally seen to not have progression clinically on scans.

My experiment was to do one test cycle of one month of high dose testosterone. Then I would do one month off testosterone and monitor my PSA. This should indicate my personal response to testosterone therapy and indicate whether I am a "favorable" responder at this time.

My starting PSA, most recent before testosterone, was 0.081, very low but not undetectable. I then did 200 mg of intramuscular testosterone cypionate weekly for 4 weeks. After that my PSA went up to 0.180. This I took to be an acceptably moderate rise. Then in part 2, after 5 weeks off testosterone (and no added ADT), my PSA came back down to 0.123.

To me and my physicians, both my MO and Dr. Beer, this suggested that I may reasonably do more cycles of T-cypionate, perhaps one month "on" and 2-3 months off (to be determined) without undue risk and enjoy great benefit to my well being and hypogonadism. I am re-gaining muscle and strength after my surgeries and feel excellent.

Dr. Beer said yesterday that my current and proposed use of cyclic supra-physiologic testosterone: "It is unlikely to be dangerous (for me) to try intermittent

exposure to testosterone in the near term." He also added that I should not add ADT during the off cycles initially, but only if indicated by PSA rises. And then he would consider relugolix (oral ADT) for the fast-on and fast-off properties.

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