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Conference Highlights 2007/2008 from The ASCO Genitourinary Cancers Symposium, the Prostate Cancer Foundation Scientific Retreat, and the Upcoming American Urological Association Meeting

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Edited from PCRI ***Insights*** **May, 2008 v 11.2**

"The effectiveness of Abiraterone, a new and more powerful oral agent for men with hormone-resistant prostate cancer (link hormone refractory PC), has been established in several clinical trials. At the GCS, Abiraterone was evaluated (Abstract #3) in a pilot trial of 29 hormone resistant men previously treated with Taxotere. The median starting PSA was 115. Twelve men (41%) had a greater than 50% decline in PSA. Measurement of circulating tumor cells (CTC) was performed, and the number of cancer cells circulating in the blood decreased substantially in the men who responded. **Dr. Johann De Bono** from the Royal Marsden presented the results of another trial of Abiraterone in a group of men with an average PSA of 75 at the PCFSR. The PSA response rate was 61%, and one-fourth of the men had a greater than a 90% decline in PSA. Interestingly they also reported that five of the 16 patients who initially responded to Abiraterone and who later developed progression of disease, subsequently achieved another remission if a potent form of cortisol called Dexamethasone was added to the Abiraterone.

A phase III trial of Abiraterone is being initiated in the United States and Canada. Approximately 70 sites around the United States and Canada are conducting the trial including Prostate Oncology Specialists (where I work). The eligibility criteria for men who want to participate in the trial are (1) hormone resistance, (2) a PSA greater than five, and (3) previous exposure to Taxotere therapy. **This trial is designed to obtain FDA approval for Abiraterone.**

The ability of Abiraterone to induce responses in Taxotere and castration-resistant men **confirms recent reports that what has traditionally been called hormone resistance is really nothing of the kind.** In other words, ***research is showing that prostate cancer cells, even the "hormone resistant" ones, still require testosterone to grow.*** Hormone-resistant cancer cells use two mechanisms to proliferate in the face of low blood testosterone levels. First, by synthesizing an over abundance of testosterone receptors (GCS Abstract #18), they can magnify the androgen effect of the tiny amount of residual testosterone present after castration (with Lupron or Zoladex). Or second, they obtain the necessary testosterone by manufacturing their own testosterone inside the cancer cell. **The reason Abiraterone works in a hormone-resistant cell is because,** unlike Lupron or Casodex, **Abiraterone blocks testosterone synthesis inside the cancer cells."**