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IN THE COURT OF ARBITRATION FOR SPORT

IN THE MATTER OF FLOYD LANDIS,

CAS 2007/A/1394

FLOYD LANDIS V. UNITED STATES ANTI-DOPING AGENCY

DECLARATION OF JOHN AMORY, M.D.

I, Dr. John Amory, declare and state as follows:

1. I am over the age of 18 and have personal knowledge of the following facts and, if called as a witness, could and would competently testify to them.

2. I am 40 years old, and currently reside in Seattle, Washington. My office address is:

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1959 NE Pacific Street
Seattle, WA 98195
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I. QUALIFICATIONS

PROFESSIONAL POSITIONS

3. I am a physician and a scientist, practicing in the field of andrology, which is the study of the male reproductive system. Since 1997, I have been employed on the faculty at the University of Washington medical school located in Seattle, Washington. I began as an assistant professor, and currently hold an academic appointment as associate professor of medicine at that institution.

4. In addition to my academic appointment at the University of Washington, I served as director of the preoperative clinic at the Veterans Affairs Center in Seattle, a position I held from 1997-2001. I am also a faculty mentor at the University of Washington's Men's Reproductive Health Research (MRHR) Career Development Training Center. This Center is one of two selected and funded by the National Institutes of Health beginning in 2006. The Center has as its long-term goal the recruitment and career development of urologists, endocrinologists, and physicians who have demonstrated research potential and are committed to a career focusing on male reproductive health.

5. I have an active clinical practice, in which I treat men with testosterone deficiencies – a condition known as hypogonadism – men who need testosterone replacement therapy. Since 2001, I have been both a hospital inpatient physician and an outpatient physician, at the University of Washington. I am board certified in Internal Medicine.

6. I am also active as a research scientist, and am currently working on developing new formulations of testosterone, and new methods of administering testosterone to men who need it. My research activities also focus on development of male contraceptives using testosterone. For example, I am actively involved in developing a clinically available oral formulation of testosterone, which our research team hopes to have ready for use in two to three years.

7. My curriculum vitae, attached as Exhibit 1 to this Declaration, contains a complete list of the Professional Positions that I have held over the course of my career.

EDUCATION

8. I completed my undergraduate degree in biology at Harvard University. I attended medical school at the University of California in San Francisco, and was awarded an M.D. from that institution. I completed my residency at UCSF as well.

9. In addition to my undergraduate and medical degrees, I hold a master's degree in public health, with an emphasis in biostatistics and epidemiology, a degree I earned at the University of Washington.

HONORS AND AWARDS

10. I am a recent recipient of the Young Andrologist Award, given by the American Society for Andrology. This award is given to researchers under the age of 45.

11. I also received the Endocrine Society International Award for Excellence in Published Clinical Research in 2005 and was the Helen and Phillip Fialkow Scholar in the Department of Medicine at the University of Washington in 2004.

MEMBERSHIPS

12. I have been a member of the American Society of Andrology for ten years. I am also a member of the Endocrine Society of the American College of Endocrinology.

PUBLICATIONS

13. My curriculum vitae, which is attached as Exhibit 1 to this Declaration, contains a current list of my publications.

14. In addition to authoring my own presentations and studies, I also peer-review articles submitted for publication in a number of scholarly journals, including, among others, the Journal of Clinical Endocrinology and Metabolism, and the Journal of Andrology.

CONSULTING AND RELATED ACTIVITIES

15. For the past three years, I have served as a member of USADA's anti-doping review board. I was recently reappointed to this board; my current term expires in January, 2009. The anti-doping review board is a body of independent experts, educated in laboratory science, clinical science, and/or law. This board looks at evidence generated in support of a doping case and decides whether or not there is sufficient evidence to justify progressing to a full hearing. I have served on an anti-doping review board in 8-10 individual cases over the past three years, two or three of these cases involved the use of the testosterone to epitestosterone ratio and Carbon Isotope Ratio ("CIR") tests.

II. OPINIONS

INTRODUCTION

16. I became interested in this case because of my research interest in testosterone and its effects, and because the test results generated by the LNDD after Appellant's Stage 17 sample did not correspond with what I would have expected to see in a testosterone user. Nor did it make sense to me that an athlete in an endurance sport would have chosen testosterone to boost performance, given the absence of evidence supporting a link between testosterone use and either endurance or an accelerated recovery time. My interest in the case became heightened after reviewing the documents; not only did the lab results continue to puzzle me, but it was apparent to me that there had been many troubling irregularities in the handling of Appellant's sample. Like Dr. Gary Wadler, a member of the World Anti-Doping Agency, I believed that Appellant's test results just did not add up.¹

17. In preparation for my testimony in this case, I was provided with the discovery documents produced in this case, including the laboratory documentation package prepared by the LNDD. I have also reviewed the briefs and the transcript of the hearing before the AAA Panel of arbitrators.

18. I made the decision to testify in this case on a pro bono basis. Prior to my testimony before the AAA Panel, I had probably spent 40-50 hours of time reviewing documents in this case and formulating my opinions. Since testifying, I have spent many additional hours preparing and refining my opinions, and in reviewing additional documents, including the

¹ "Doping expert thinks Landis result 'doesn't add up,'" (July 26, 2006), *at* <http://sports.espn.go.com/oly/cycling/news/story?id=2532029>, attached as Exhibit 2.

transcript and the Appellee's Brief. I am not charging for my time in this case and I believe that to uphold an anti-doping sanction on the evidence in this case is morally and ethically wrong.

There is no evidence that testosterone augments endurance

19. Testosterone is the most important male sexual hormone, and is the hormone responsible for transforming a preadolescent boy into a man. Testosterone works on almost every tissue in the body, from the head to the toe. It increases red cell mass, causing men to have higher hematocrit levels than women. Over time, it dramatically increases muscle mass and decreases fat mass. It has a marked effect on sexual function, mood, energy and hair growth. Secretion of testosterone by the testes assists in the sperm maturation process, directly impacting male fertility.

20. While testosterone is produced naturally by the body, some men benefit from administration of exogenous testosterone because they do not produce sufficient amounts. Some boys, for example, do not produce enough testosterone to get them through puberty. Some men are born with a condition called Klinefelter's syndrome, characterized by a failure to produce testosterone. Some men lose their testicles, due either to accident or cancer. All of these men will require testosterone replacement therapy in order to maintain healthy muscle mass and bone mineral density, not to mention normal sexual function.

21. Testosterone can be administered in a number of ways. The traditional method was to use an intramuscular injection to deliver a dose of testosterone enanthate. However, the shot is quite painful because the testosterone is suspended in an oil base, requiring a large-bore needle. About 15 years ago, patches became available, but they had several side effects, including irritating skin reactions. More recently, we have begun to use testosterone gels; a patient puts gel in his hand then rubs it over his back and chest on a daily basis.

22. When exogenous testosterone is administered to a man with normal levels of testosterone, however, that man's natural or endogenous testosterone production will significantly decrease. This is, in fact, the theory behind the use of testosterone as a male contraceptive. Administration of exogenous testosterone will suppress secretion of luteinizing hormone and follicle-stimulating hormone from the pituitary gland. As the levels of these hormones fall in response to the administration of exogenous testosterone, the amount of testosterone secreted by the testis also decreases, depriving developing sperm of the signals needed to reach full maturity. The fewer sperm that mature, the fewer there are produced and the less fertile the man becomes. Decreases in luteinizing hormone production can be seen in less than one month.

23. One reason that the accusation against Appellant did not make sense to me is that there is no convincing evidence—that is, evidence published in a peer-reviewed journal—demonstrating that testosterone use boosts endurance. While it *is* effective in increasing strength and power in a manner that would be useful to a sprinter or weightlifter, there is no evidence that it helps with endurance sports like cycling. Indeed, the benefit received by an endurance athlete engaging in “microdosing,” which means applying small doses to testosterone to avoid doping detection, is even more remote. USADA witness, Joe Papp, testified about the “benefits” of microdosing before the Panel, but that testimony must be viewed as entirely anecdotal, as the medical literature provides no support for the notion that testosterone at *large* doses provides endurance benefits, let alone “micro” doses.

24. It is well-accepted now that testosterone produces strength benefits in men already producing normal amounts of testosterone (“eugonadal” men). The best study of this was authored by Dr. Shalendar Bhasin and published in 1996 in the *New England Journal of*

Medicine. This study clearly established a link between testosterone and strength.² The *New England Journal of Medicine* has the most rigorous peer-review process of any medical journal in the world, and Dr. Bhasin's study is widely regarded as the most well-designed and authoritative study on this subject.

25. Peer-review is an important feature in the development of scientific knowledge. In this system, researchers submit research papers to journals, which then send the article to independent reviewers—usually between three and six of them—who are not only anonymous, but experts in the relevant field of inquiry. These scientists review the methodology used by the researcher submitting the paper, as well as the conclusions reached. The goal is two-fold. First, the reviewers will be looking to see whether the methodology selected was rigorous, whether it was an appropriate tool to use to answer the question posed, and whether it was correctly implemented in the particular study. They will also be looking to see if the conclusions reached were properly supported by the study. Due to the extensive review by these anonymous reviewers, studies are almost never accepted upon initial submission. It is typical for the reviewers to be harsh, and to request that additional data be collected or additional experiments be conducted before the paper is accepted for publication. This process helps to insure that only well-presented data from properly conducted studies is published, and that only data that meets the highest standards of clarity and methodology makes it into the published literature. Once a paper is published in the literature, it is often accepted as true and authoritative, though it is important to remember that peer-review continues after publication as well. That is, once

² S. Bhasin et al., *The Effects of Supraphysiologic Doses of Testosterone on Muscle Size and Strength in Normal Men*, *New Eng. J. Med.* 335(1):1- 7 (July 4, 1996), Exhibit GDC 272-278. The study was supported by a grant from the U.S. National Institutes of Health.

published, all scientists are provided an opportunity to review the published study in the journal, and to comment upon its merits. Both the pre- and post-publication phases of peer-review are important quality control mechanisms in the development of scientific knowledge.

26. To return to Dr. Bhasin's study, his study subjects were 40 normal (eugonadal), healthy men with normal testosterone levels,³ who were randomly assigned to either a 10-week course of 600 mg of testosterone enanthate injected weekly, or a placebo. The testosterone dose administered was about six times higher than the dose that would be given as part of testosterone replacement therapy. Dr. Bhasin then further randomized the study populations into exercise and no-exercise groups. So both the testosterone and placebo groups had an exercise and a no-exercise subgroup. Those exercising did so three times per week. At the beginning of the study period, a baseline was established for each man by determining fat-free mass by underwater weighing, determining muscle size by magnetic resonance imaging (MRI), and determining muscle strength by assessing the man's ability to perform bench press and squatting exercises. Diet and exercise levels were controlled to eliminate the potential for these variables to confound the result, a significant improvement over prior studies.

27. Dr. Bhasin concluded that high (superphysiologic) doses of testosterone in normal healthy men who did not exercise increased both muscle size and strength. Even greater effects in muscle size and strength were seen among the subjects that both took testosterone and exercised. In these subjects, fat-free muscle mass increased, muscle size increased, as did muscle strength. Dr. Bhasin was thus able to conclude that there are synergistic effects between testosterone administration and strength—the two work together to increase muscle size and

³ Of the 43 men that commenced the treatment phase of the study, three dropped out or were eliminated for illicit drug use.

strength. Prior to Dr. Bhasin's study, there was no consensus in the literature about whether testosterone actually produced strength benefits, but Dr. Bhasin's study conclusively established that it did, given sufficient dose and time.⁴

28. Finally, Dr. Bhasin concluded that there were *no* observed changes in mood or behavior among the subjects taking testosterone. This conclusion is consistent with one reached by Dr. O'Connor and his team and published in the journal *Physiology and Behavior* in 2002.⁵ In this study, Dr. O'Connor administered either 200 mg of testosterone enanthate or a placebo to eugonadal study subjects on a weekly basis for eight weeks.⁶ Both the subjects and their partners were asked to answer a number of questionnaires designed to measure aggression and assertiveness. Healthy men experienced neither increases in aggression nor other mood changes, while those men that started out testosterone deficient actually reported improvements, becoming less hostile.

29. In a later study, Dr. Bhasin concluded that testosterone's effects are dose-related, and that there was a threshold below which effects will not occur, though that threshold dose has not yet been determined.⁷ In 2001, Dr. Bhasin studied 61 normal men who were randomized to

⁴ See also Ferrando et al., *Testosterone administration to older men improves muscle function: molecular and physiological mechanisms*, Am. J. Physiol. Endocrin. Metab. 282: E601-07 (2002).

⁵ D. O'Connor et al., *Exogenous Testosterone, aggression, and mood in eugonadal and hypogonadal men*, Phys. & Behav. 75(2002); 557-66, Exhibit GDC 633-642.

⁶ Men with testosterone deficiencies were also studied in Dr. O'Connor's research.

⁷ S. Bhasin et al., *Testosterone dose-response relationships in healthy young men*, Am. J. Physiol. Endocrinol. Metab. 281:E1172-1181 (2001), Exhibit GDC 279; *see also* Hartgens F, Kuipers H., *Effects of androgenic-anabolic steroids in athletes*, Sports Medicine, 34(8): 513-

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one of five different groups. Each group received a monthly injection to suppress their natural testosterone production, and then received weekly injections of varying doses of exogenous testosterone [25, 50, 125, 300, 600 mg testosterone enanthate] over 20 weeks. He again measured for changes in muscle size, muscle strength and fat-free mass. Dr. Bhasin's results indicated that changes in fat-free mass, muscle strength and size were dose-dependent, and that the dose-response curve is linear.

30. Based on these and other studies, we can now confidently say that testosterone administration does, in fact, increase strength and muscle size and mass. The same *cannot* be said, however, for testosterone's impact on endurance or recovery. In contrast to testosterone's impact on strength, the medical literature does *not* support a link between testosterone administration and either endurance or recovery.

31. For example, in a study conducted by Dr. Baume and others (including Dr. Martial Saugy, Director of Switzerland's WADA-accredited lab), no endurance benefits were seen after study subjects were given testosterone for one month, nor was there an increase in serum biomarkers that are thought to indicate recovery activity.⁸ Dr. Baume's study was conducted in order to test the accuracy of anecdotal accounts given by anonymous athletes, who

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554, at 519 (2004) [hereinafter, "Hartgens & Kuipers"] (testosterone's effects are dose-related); Friedl, K., *Effect of Anabolic Steroid Use on Body Composition and Physical Performance*, in Yesalis C., et al., *Anabolic Steroids in Sport and Exercise* (2000) at 145 (noting that failure to detect weight gain in studies examining testosterone effects is evidence demonstrating that there is a threshold below which testosterone will not cause biological effects in normal men, and suggesting that this threshold lies at the "replacement" level for normal men).

⁸ N. Baume et al., *Effect of multiple oral doses of androgenic anabolic steroids on endurance performance and serum indices of physical stress in healthy male subjects*, Eur. J. Appl. Physiol. (2006) 98; 329-40, Exhibit GDC 621-632.

believed that taking testosterone improved either endurance or recovery. Dr. Baume studied the endurance effects—if any—of testosterone and nandrolone administration on healthy men who were active in sports. In his study, 25 subjects were randomized into three groups, one of which was given a placebo, one of which was given testosterone undecanoate, and one of which was given 19-norandrostenedione (nandrolone). The doses were administered 12 times in a one-month study period. The study subjects' ability to run on a treadmill was assessed before and after the study period. During the study period, the subjects were placed on an endurance training regimen. Finally, Baume and his team evaluated serum biomarkers believed to be indicative of recovery. No differences were detected between the three study populations with respect to their endurance during the running test, or in their recovery biomarkers. Dr. Baume's study fails to support the conclusion that administration of exogenous testosterone provides an endurance boost or a recovery boost.

32. Although Dr. Don Catlin, former director of UCLA's WADA-accredited lab testified before the AAA Panel that the effects of testosterone on recovery were "well known,"⁹ his entire opinion consisted of this sole statement. In contrast to Dr. Bhasin and Dr. Baume, Dr. Catlin has published nothing about this particular question, and in fact cited to no studies published by others to support his opinion. Dr. Bhasin's conclusions are based upon his own work, work subjected to rigorous peer-review and public comment.

33. Similarly, I cannot credit the anecdotal testimony provided by cyclist Joe Papp, another witness providing testimony at USADA's request in the May 2007 arbitration hearing. Mr. Papp admitted to having taken many kinds of prohibited substances, including EPO, human

⁹ See Tr. of Proceeding at 1199.

growth hormone, thyroid medicine, amphetamines, human growth hormone and AndroGel, which contains testosterone.¹⁰ Mr. Papp testified that he believed that the testosterone in the AndroGel gave him a recovery benefit. There are several problems with this sort of testimony. It is, of course, the anecdotal account of one person giving a subjective account of his own performance. This type of anecdotal accounts are usually discounted by scientists.¹¹ The problem is compounded by the fact that Mr. Papp was not taking testosterone alone, but a number of different prohibited substances. It would be difficult to tease apart the effects of any one drug in a subject taking many drugs, even in a scientifically designed and controlled study. An individual is simply in no position to do this in a scientifically credible fashion. The AAA Panel was right to disregard Mr. Papp's testimony.¹²

34. I understand that USADA has indicated that it will call cyclist Patrick Sinkewitz to provide testimony in the appeal of Appellant's case. It has been publicly reported that Mr. Sinkewitz tested positive for testosterone in June, 2007 after applying a 25 mg packet of testosterone gel to his arm the night before a doping control test that revealed a testosterone: epitestosterone ratio of 24:1. Mr. Sinkewitz later stated that he had used EPO in the past as well.¹³ If Mr. Sinkewitz offers the same type of testimony that Mr. Papp offered—that

¹⁰ Transcript, 1018-9, 1026-1030.

¹¹ For the most part, scientists are compelled to rely upon individual self-reports for information about mood.

¹² AAA Award, page 79, ¶299.

¹³ "Saturday's EuroFile: Sinkewitz speaks; Cipo' won't Rock," *Agence France Presse*, November 3, 2007, <http://www.velonews.com/article/13612>, attached here as Exhibit 3; "Sinkewitz ne demande pas de contre-expertise," Publié le 31 juillet B 11:37, <http://tour-de->

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testosterone aided his recovery—I would have the same reservations about accepting his testimony as “science” that I had about accepting Mr. Papp’s testimony. I would also note that both Mr. Sinkewitz and Mr. Papp admitted to having used EPO, which, in contrast to testosterone, *does* provide great benefits to endurance athletes, effects that have been demonstrated convincingly in a number of studies.

35. It should be noted that both Dr. Bhasin and I were interviewed by the *New York Times* about testosterone effects after Appellant’s test results were publicized in 2006.¹⁴ Dr. Bhasin generally discussed researchers’ attempts to identify whether testosterone produced endurance benefits by, among other things, administering testosterone to study subjects and monitoring their ability to perform endurance exercises like running on a treadmill. Such studies have detected no significant differences in the endurance abilities of those taking testosterone and those taking placebo, causing Dr. Bhasin to remark that “no one has been able to show clearly that testosterone increases endurance.” Nor do the studies show that the hormone increases aggression or provides the sort of euphoric benefit that might allow an athlete to train harder. Dr. Bhasin himself looked for such effects in his 1996 study and found none. That testosterone causes such effects is “folklore.”

36. If testosterone cannot improve endurance or recovery at supraphysiologic doses, it cannot do so at “microdoses.” I do not know whether, or to what extent, athletes engage in this activity but as I have stated, the peer-reviewed medical literature does not provide support for the

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france.france2.fr/le-tour-une.php?id_article=773 (Mr. Sinkewitz’s “A” Sample T/E ratio was 24:1), attached as Exhibit 5; *L’Equipe*, “Cyclisme - Les regrets de Sinkewitz,” (July 31, 2007), http://www.lequipe.fr/Cyclisme/20070731_093445Dev.html, attached as Exhibit 6.

¹⁴ G. Kolata, Cycling: Some Researchers Question the Tests for Testosterone, New York Times, July 28, 2006, USADA Exhibit 122.

theory that testosterone augments endurance, even at doses that would easily be detected with standard tests like the T/E ratio test. That being the case, there is no reason to suspect that microdoses of testosterone would provide endurance benefits that larger doses do not.

37. WADA member, Dr. Gary Wadler, has publicly commented on the state of the scientific literature, and its support for the notion that testosterone increases strength after weeks of administration, but not endurance:

“Steroids can increase strength and improve recovery time and prevent the breakdown of muscle, maybe make him more assertive and aggressive. All of those could have some positive attribute. But most steroids are given in cycles [6-12 weeks] and in context of working out in a gym with weights. It makes no sense to me why an athlete would take testosterone the day of a race when it doesn't work that way. It doesn't make sense in terms of the pharmacology of the drug, and it really doesn't have the attributes that would be attractive to a cyclist -- particularly one running the risk of violating anti-doping regulations.”¹⁵

38. Dr. Wadler went on to note that he thought that something was “missing” from the facts known about Appellant’s case, particularly since the risks of taking the drug when Appellant was sure to be tested so significantly outweighed the “benefits” which are nonexistent for one-time use:

“Everybody knew the spotlight was on cycling. For eight years, the world has been watching cycling particularly closely. It would be the ultimate form of denial, or the ultimate sense of invincibility, to think you're going to evade that. And when the pharmacology of the

¹⁵ “WADA panel member Wadler Q & A,” July 27, 2006, emphasis added. See <http://sports.espn.go.com/oly/tdf2006/news/story?id=2531677>, Exhibit 4 to this declaration.

drug doesn't really, in my judgment, seem like a drug of particular note to a cyclist, it doesn't really compute.”¹⁶

39. I agree with Dr. Wadler's conclusion. Appellant's results do *not* “compute.”¹⁷ Because LNDD's results are implausible both because there would have been no reasoned justification for a cyclist to take testosterone, but also because the results generated do not reveal a testosterone metabolite profile consistent with known science.

The results of LNDD's analysis of Appellant's Stage 17 samples do not correspond with known science

If Appellant had taken testosterone on five days during the Tour de France, his T/E ratio should have reflected that use, but it did not

40. The LNDD reported an Adverse Analytical Finding (AAF) only as to the sample collected from Appellant on July 20, after he won Stage 17. It was on that date that Appellant's T/E ratio exceeded the positivity threshold of 4:1, a finding that LNDD “confirmed” with the results of CIR tests revealing an abnormal value for the testosterone metabolite, 5-alpha.¹⁸

The results of Appellant's seven other Tour de France samplings were subsequently analyzed with the GC/C/IRMS instrument; though these samples did not have a positive T/E test, USADA now claims that these results support the AAF because abnormal 5-alpha diol values

¹⁶ Exhibit 3, “WADA panel member Wadler Q & A,” (July 27, 2006), emphasis added. See <http://sports.espn.go.com/oly/tdf2006/news/story?id=2531677>.

¹⁷ *Id.*; Exhibit 2, “Doping expert thinks Landis result ‘doesn’t add up,’ (July 26, 2006), <http://sports.espn.go.com/oly/cycling/news/story?id=2532029>.

¹⁸ The value for Andro 11-keto could not be considered positive once the .8 measurement uncertainty was taken into account.

were reported on four other dates during the tour; including the final parade stage.¹⁹ Two scenarios present themselves.

41. I am aware of no published, peer-reviewed, studies in which the researchers looked at the impact on the T/E ratio of a single use of testosterone gel. However, I note that USADA has indicated it intends to call cyclist Patrick Sinkewitz to testify in Appellant's appeal. Mr. Sinkewitz has publicly stated that he used a single 25 mg gel package of Testogel the night before a doping control test, but his T/E ratio was measured at 24:1 the next day according to published accounts.²⁰ If Mr. Sinkewitz's experience is indicative of either "microdosing" or the relative detectability of small doses of testosterone, we can conclude from his experience that one 25 mg gel package certainly wasn't a dose small enough to escape detection.

42. The impact of chronic and intermittent testosterone use on the T/E ratio is discussed in USADA's own Exhibit 34, a March 2007 progress report by USADA witness Dr. Schanzer entitled, "The misuse of testosterone gel." This is a progress report from what appears to be an ongoing WADA research project being conducted at the Institute of Biochemistry at the German Sport University in Cologne. This progress report details the

¹⁹ See GDC 1363; USADA Exhibit 30. An abnormal value for 5-beta diol was reported for one of these dates, July 13, 2006.

²⁰ "Sinkewitz ne demande pas de contre-expertise," Publié le 31 juillet B 11:37, http://tour-de-france.france2.fr/le-tour-une.php?id_article=773 (Mr. Sinkewitz's "A" Sample T/E ratio was 24:1), attached as Exhibit 5; *L'Equipe*, "Cyclisme - Les regrets de Sinkewitz," (July 31, 2007), http://www.lequipe.fr/Cyclisme/20070731_093445Dev.html, attached as Exhibit 6.

findings to date of a study looking at the chronic administration of testosterone, and the intermittent administration of testosterone gel.²¹

43. In this progress report, Dr. Schanzer describes the results to date of a study he is conducting on normal healthy men in order to identify the best diagnostic parameter to use to detect doping with testosterone gel. Eighteen study subjects, who were also participants in a study on the endocrine effects of testosterone gel, were placed into two groups, one of which was given daily transdermal applications of 2 x 5 g Testogel corresponding to a 100 mg dose. This was administered to them for six weeks. The second group received intermittent doses of Testogel—one week daily administration of 2 x 5 g Testogel, and one week without Testogel—for a total study period of six weeks. Urine and blood samples were collected before, during, and after the study period; all urine samples were analyzed by GC/MS, while the samples of two of the subjects were also analyzed using the CIR test.

44. It is probably worth noting that from a methodological standpoint, Dr. Schanzer's study is not described by him as either a randomized or a blinded study. It is also worth noting that as to the CIR test for *testosterone* and certain of its metabolites, Dr. Schanzer notes that "new purification methods have been developed," new methods that were used in place of the Cologne lab's standard operating procedures. It is not clear to me what those methods are, or

²¹ While this study was presented at a scientific conference, it is not at all clear what pre-publication peer review took place. Since it is a progress report in what appears to be a WADA-funded project, it seems reasonable to conclude that the authors were required to submit this progress report as a condition of continued funding, irrespective of any peer-review. And, of course, since the paper was dated March 2007 and the hearing at which it was relied upon by USADA took place only two months later, there would not have been an opportunity for much post-publication comment on the study by other researchers.

how they could have impacted the analysis or the results.²² But if new and improved lab purification methods have been developed, it gives rise to questions about the need for such change, and the need to implement analogous changes in all other WADA labs.

45. The administration of the gel consistently increased the T/E ratio amongst Schanzer's study subjects, though not all values were above the 4:1 threshold positivity cutoff because some of the subjects were low-mode (normal T/E ratio is low, even as low as 0.1). The low-mode individuals experienced less change in their T/E ratios than did high-mode individuals.²³ But among those study subjects who used gel during the entire period, increases were seen in the T/E ratio when compared to the individual's normal reference range, even when the ratios did not actually exceed 4:1. When the T/E ratio exceeded 4:1, it did so consistently, which would be expected given the standardization of dose and the fact that a person's "mode" does not tend to fluctuate over time.²⁴ As figure 9 indicates, the administration of testosterone gel changed the values of testosterone and epitestosterone (and other steroids), increasing testosterone modestly, while suppressing epitestosterone to a much greater degree. So this Cologne study confirms that when chronic administration of testosterone gel increases the T/E, it does so consistently.

²² The paper indicates that the method is presented at Annex 1, but this annex is comprised entirely of box plots of steroid profile parameters and scatter diagrams.

²³ At the AAA hearing, Dr. Catlin testified that there are in fact up to 22 genetic types of persons, all responding somewhat differently to steroids. Tr. of Proceedings at 1255.

²⁴ See, e.g., Aguilera et al., "Performance Characteristics of a Carbon Isotope Ratio Method for Detecting Doping with Testosterone Based on Urine Diols: Controls and Athletes with Elevated Testosterone/Epitestosterone Ratios," USADA Exhibit 40, USADA 1225, USADA 1232.

46. At the AAA hearing, I compared the results in Dr. Schanzer's study with the results gathered by LNDD from the urine samples collected from Appellant during the 2006 Tour de France, results presented in a table found at Exhibit GDC 1363. The Panel, however, concluded that the LNDD's T/E ratio results could not be relied upon to support the adverse analytical finding because LNDD did not both acquire and analyze three diagnostic ions. Even if we assume that LNDD's results were accurate and reliable,²⁵ one must conclude that Appellant's sample results were not consistent with the use of testosterone gel on all dates. The only date on which his T/E ratio exceeded the 4:1 level was July 20; as GDC 1363 indicates, the "B" sample T/E ratio was reported to be 11:1 on that day. On this date, an abnormal value was reported as to only one metabolite, 5-alpha, though it is well-established that 5-alpha and 5-beta ought to change in tandem. That they did not is a troubling anomaly. Further, Appellant's results show an elevated T/E ratio on only one day, but abnormal 5-alpha values on five days, accompanied on only one of these days by an abnormal 5-beta diol value. If Appellant, indeed, used testosterone on all dates for which an abnormal CIR test 5-alpha value was reported, one would have expected that the T/E ratio would have been higher on those dates as well (July 13, 18, 22, 23), not just on July 20. Yet they were not. None of these results is consistent with the profile of study subjects consistently administered testosterone gel in Dr. Schanzer's study. These study results do not support the conclusion that Appellant used testosterone on the five days for which an abnormal 5-alpha diol value was reported. They are less consistent with oral testosterone use or testosterone injections, because in addition to causing a spike in T/E ratios after administration

²⁵ The AAA Panel concluded that those T/E results could *not* be relied upon because LNDD failed to both acquire and analyze three diagnostic ions when conducting the GC/MS analysis.

in high mode individuals like Appellant, the testosterone persists, gradually dissipating over time. This effect is not confirmed by these results.

47. Dr. Catlin's testimony before the AAA Panel is consistent with my conclusion. Dr. Catlin testified about Exhibit 30, Appellant's longitudinal steroid profile, a document which includes the T/E ratios depicted on GDC 1363, but also includes T/E ratios generated from tests dating back to 2002. When presented with Exhibit 30, Dr. Catlin stated that the profile was very "ordinary up until the box," and was not consistent with the type of suppression he had seen upon administration of designer drugs, with the caveat that there are individual variations in response.²⁶

48. What must also be noted is that Appellant's absolute testosterone concentrations, as noted on Exhibit GDC 1363 (and on Exhibit 30) are in the low part of the normal range, which is between 40 and 160-200. The same can be said of the epitestosterone numbers. That these figures are low is significant for two reasons. First, if Appellant had been *taking* testosterone, one would have expected to see high testosterone values. As a corollary to this observation, the low values in the epitestosterone figures undermine any suggestion that Appellant had used epitestosterone cream to mask his testosterone use. If he had been taking both testosterone and epitestosterone cream to mask the testosterone use by maintaining a stable T/E ratio, *both* concentrations would have been *high*, though the resulting ratio could well have been normal. So, for example, on July 13, when Appellant had an abnormal finding for both 5-alpha and 5-beta, the total testosterone value reported was extremely low for both testosterone and epitestosterone. An epitestosterone value of 7.6 would simply not support the conclusion that

²⁶ Tr. of Proceeding at 1254-56. The "box" appears on exhibit 30 around the data for July 20, the results of Appellant's Stage 17 sample.

Appellant had used an epitestosterone cream to mask testosterone doping. The fact that Appellant's concentrations of both testosterone and epitestosterone were at the very lowest end of the normal range excludes such masking as a realistic possibility.

49. Dr. Schanzer's paper is consistent with the "case report," if we can call it that, of Patrick Sinkewitz. Just as Mr. Sinkewitz's reported one-time use of testosterone gel less than 24 hours before a doping test resulted in a T/E ratio of 24:1, so Dr. Schanzer's results demonstrated that even intermittent use of testosterone gel increased the T/E ratio in a marked and stable manner. Even as to those study subjects whose T/E ratio did not exceed 4:1, the gel increased the T/E ratio over the individual's normal reference range.

50. The LNDD T/E ratio results, when combined with the CIR test results, simply do not support the contention that Appellant took testosterone over a several-day period during the Tour de France, as is alleged. If testosterone administration caused an excessive T/E ratio on one day, one would have expected to see an excess on all five of the days for which an abnormal 5-alpha value was reported. A person's T/E ratio does not simply change as a result of T/E administration on one day, but not on the next. We do not see cases in the published literature in which a person takes testosterone over a period of days, and on some of those days, the person exhibits both a high T/E ratio and abnormal metabolite values, but on subsequent days, exhibits a normal T/E ratio and abnormal metabolite values. Low-mode individuals might have abnormal metabolite values with a normal T/E ratio, but this is a pattern that would be consistent, not intermittent. And of course, Appellant is not a low-mode individual, he is a high-mode individual. So the administration of testosterone should have caused his T/E ratio to react in a consistent fashion, and it did not.

Appellant's CIR test results do not support the Adverse Analytical Finding

51. As part of my work as an independent reviewer on the USADA anti-doping review board, I have developed a familiarity with the CIR test, which has been used as a confirmatory test for the T/E ratio test. The CIR test is intended to detect testosterone of exogenous origin. The test identifies exogenous testosterone on the basis of the ratio between ^{12}C and ^{13}C found in the sample's testosterone metabolites and an the ratio found in an endogenous reference compound.

52. The metabolism of testosterone is extraordinarily complicated. First, testosterone serves as a pro hormone for formation of dihydrotestosterone and estradiol for men. From that point, testosterone is metabolized in a variety of ways, and is chemically converted to certain compounds.

53. Testosterone is not only converted into various metabolites, but it impacts hormones in the body. For example, when exogenous testosterone is administered, imbalances can be expected in the body's testosterone/epitestosterone ratio, but effects on other hormone levels occur as well. The main hormonal effect seen when exogenous testosterone is administered is the suppression of the body's luteinizing hormone and follicle-stimulating hormone, which are secreted by the pituitary gland. When testosterone is taken over weeks and months, the serum levels of luteinizing hormone and follicle-stimulating hormone will fall. These effects of testosterone administration are depicted graphically at Exhibit GDC 620. This chart summarizes the effects on luteinizing hormone levels observed when 100 mg testosterone enanthate was administered to seven men with normal luteinizing hormone levels over a twenty-eight day period. As the chart reveals, some depression in the luteinizing hormone levels occurs almost immediately, and will fall to subnormal levels within 28 days.

54. In the 1996 Bhasin study discussed earlier, Dr. Bhasin measured luteinizing and follicle-stimulating hormone levels twice during the control period of his study, and eight times during the 10-week treatment period. While baseline levels of luteinizing and follicle-stimulating hormone were similar amongst all study subjects, those taking testosterone experienced significant decreases in those hormones over the course of the study period.²⁷

55. The ratio of testosterone to luteinizing hormone is thought to be a more sensitive indicator of testosterone use than the T/E ratio.²⁸ That ratio has been shown to be sensitive enough to detect testosterone use for a longer period after the last use.

56. In addition to depressing luteinizing hormones, testosterone is metabolized into four important metabolites: androsterone, etiocholanolone, 5-alpha diol and 5-beta diol. Evaluating changes in these metabolites is another way to supplement or confirm the results of a T/E ratio or a T/LH ratio. The GC/C/IRMS instrument evaluates the testosterone metabolites by evaluating the ¹³C to ¹²C ratio of these metabolites.

57. The ratio of ¹³C to ¹²C ratio can be measured by GC/C/IRMS instrument, and compared against an international standard. Differences between a measured compound and the standard are reported as a delta value. Synthetic testosterone has a lower delta value than endogenous testosterone because it is made primarily of plant materials with a different ¹³C to ¹²C ratio than would be found in a typical person's diet. The CIR analysis measures the delta values of these testosterone metabolites in a urine sample and these values can be compared to

²⁷ Exhibit GDC 275, *NEJM* at 4

²⁸ P. Perry et al., *Detection of anabolic steroid administration: ratio of urinary testosterone to epitestosterone vs the ratio of urinary testosterone to luteinizing hormone*, Clin. Chem. 43(5); 731-5 (1997), Exhibit GDC 652-56.

the values measured for an endogenous reference compound known to be unaffected by the administration of exogenous testosterone. The difference between the delta value of the target testosterone metabolite and the delta value of the endogenous reference compound is known as the delta/delta value. Because the use of synthetic testosterone should result in a delta value more negative than it would ordinarily be in the absence of testosterone use, one will see a corresponding negative change in the delta/delta values of these metabolites. The variation should tightly correspond, particularly the 5-alpha and 5-beta values; the difference seen in the delta/delta values for these testosterone metabolites will increase rapidly and in tandem with one another after the administration of testosterone. This is well-supported in the peer-reviewed scientific literature. The CIR method, while useful for detecting exogenous testosterone, is known to be “inherently difficult” to perform correctly, requiring “scrupulous attention to detail” and “strict adherence” to quality control.²⁹

58. The fact that testosterone metabolites—particularly 5-alpha and 5-beta — tend to change in a parallel fashion upon testosterone administration is well illustrated in a 1997 paper authored by USADA witness, Dr. Cedric Shackleton, published in the journal, *Steroids*.³⁰ This is presented by USADA as Exhibit 40, beginning at USADA 1241. Drs. Catlin and Aguilera, both USADA witnesses, also cite this paper in their own study, “Performance Characteristics of a Carbon Isotope Ratio Method for Detecting Doping with Testosterone Based on Urine Diols:

²⁹ R. Aguilera et al., *Performance Characteristics of a Carbon Isotope Ratio Method for Detecting Doping with Testosterone Based on Urine Diols: Controls and Athletes with Elevated Testosterone/Epitestosterone Ratios*, USADA Exhibit 40, beginning at USADA 1225, at USADA 1226, 1231.

³⁰ C. Shackleton et al., *Confirming testosterone administration by isotope ratio mass spectrometric analysis or urinary androstane diols*, *Steroids*, 62:379-387 (1997).

Controls and Athletes with Elevated Testosterone/Epitestosterone Ratios,” USADA Exhibit 40, beginning at USADA 1225.

59. In Dr. Shackleton’s study, the study subjects were given a single intramuscular injection of testosterone enanthate at a dose of 250 mg. Figure 4 appears on page 383 of the article (USADA 1245), and illustrates the changes one would expect to see with exogenous testosterone administration. The top axis is time measured in days, starting at 0 and continuing through day 14, while the y-axis is the delta ^{13}C value. The figure depicts the changes in the metabolites of testosterone as compared to the endogenous reference compound (pregnanediol) over time. So if we look, for example, at Subject 3, in the top right panel of figure 4, we can see that prior to testosterone administration, the difference between the delta value for pregnanediol (the line with open circles) and the delta value for 5-alpha and the 5-beta is almost two. Once testosterone is administered, however, there is a dramatic and parallel decrease in the delta value of both the 5-alpha and the 5-beta, resulting in a delta delta of approximately four as compared to the pregnanediol. The 5-alpha and the 5-beta values stay down, beginning to rise together as the exogenous testosterone attenuates. We know that after a single injection, testosterone will persist in the system for about 14 days, and that is consistent with what we see in this figure. The 5-alpha and the 5-beta drop together, and then they begin to rise together as the 14th day approaches. The difference between the 5-alpha and the 5-beta, however, appears to be one or less at the greatest point of divergence between the two. This effect is seen in the other panels of figure 4 as well (though only 5-beta is presented in Figure 4A).

60. So this Figure 4 nicely illustrates how testosterone metabolites behave in normal men after administration of testosterone: 5-alpha and the 5-beta drop acutely after injection, they stay down while the testosterone is in residence and, as the testosterone wears off, they rise in

tandem back to a baseline delta-delta of between 1 and 2. Others have published data similar to Dr. Shackleton's and generated similar findings.

61. We can also see this effect in the paper authored by Dr. Schanzer and discussed earlier. Recall that Dr. Schanzer conducted CIR analysis on the urine samples of two of his subjects, who had been given testosterone in gel form, not orally or by injection. Dr. Schanzer presents his CIR results in Figures 18-21 of that paper. There are differences to note, most notably that the endogenous reference compound used by Dr. Schanzer is not the same that LNDD used (pregnanediol). And of course, Dr. Schanzer's sample size was small; he conducted CIR analysis on the samples of only two of his subjects.

62. In Figure 18, Dr. Schanzer presents both the T/E ratios and the CIR results of the urine analysis conducted on samples submitted by subject P10 before, during and after gel administration. All six of the samples collected during gel administration had T/E ratios above 4, and when testosterone was analyzed as the target compound, all six of these samples were found to be positive, with a delta-delta difference greater than 3. The T/E ratio is actually positive at all times when the testosterone gel is being administered. So this reveals a good correlation between the T/E ratio and the CIR results. Figures 20 and 21, the CIR test results for subject P9 (intermittent use) also show that values of the metabolites behave as you would expect, though it must be noted that as Figures 20 and 21 indicate, they are not as sensitive as one might hope, given that some of the CIR values are not in the positive range when testosterone is being administered. Though the test is not perfect, at least the metabolites are behaving in a manner that is physiologically plausible, with the peaks moving together in a parallel direction. Though there is some difference in the 5-alpha and 5-beta, the samples are still behaving in the manner that you would expect, based on the published literature.

63. While one might expect some difference in the behavior of the testosterone metabolites based upon mode of administration, the Dr. Schanzer and Dr. Shackleton papers do not really support this hypothesis because both confirm that the T/E ratio and the delta values of the metabolites will all be affected by the testosterone administration, and will cause analogous and consistent changes in the direction and timing of delta value changes amongst the target metabolites. While one mode of administration might take effect more quickly than another, or wash out more quickly than another, when the changes do occur, they should occur in a consistent pattern. While Dr. Schanzer did hypothesize that the 5-alpha reductase in the skin was responsible for an unexpectedly large difference between the 5-alpha and 5-beta delta values with respect to study subject P9, this hypothesis was simply incorrect.³¹ I have treated hundreds of men with hypogonadism by prescribing this gel to them, and studies have been done in thousands of men. We simply do not see the effect Dr. Schanzer hypothesizes, that being a disproportionate increase in the dihydrotestosterone (DHT) over testosterone due to 5-alpha reductase in skin, resulting in a discrepancy between 5-alpha and 5-beta. The only time this effect was seen was with the very first testosterone patch that came out, which was applied to the scrotum. In that case, there was a high elevation of DHT, but when the gel is applied to the rest of the torso, the effect is not seen. There is an abundance of data on this point. This is the sort of conclusion that would have been identified by post-publication peer review.

64. To my knowledge, the peer-reviewed scientific literature does not contain a report in which the ¹³C to ¹²C ratio of only one of the 5-alpha or 5-beta differs significantly from the other after testosterone administration, nor does it contain a physiologic explanation for such an

³¹ See Exhibit 34, Schanzer report at page 13

effect. One would expect a difference between the 5-alpha and the 5-beta delta-delta values of no more than one, two at the most. That parallel movement is not, however, what we see when we look at Appellant's sample results.

65. Now, if we return to Appellant's CIR results, presented on the GDC 1363 exhibit discussed earlier, we can see results that are *not* consistent with the published literature with respect to the behavior of the diols. In clear contrast to what the published literature would support, on July 20, there is a very large difference in the value for the two diols (5-alpha and 5-beta), a difference of almost four, which is much greater than any difference we saw in the Dr. Shackleton's paper, or that I've seen in similar studies. We would expect those values to move in tandem, and parallel each other closely, but they do not.

66. The same anomaly between the 5-alpha and 5-beta is seen on July 22nd and 23rd, but these figures are even more suggestive that something has gone wrong in the analysis itself. Because on these dates, the T/E ratio is clearly well within the normal range, as are all of the other metabolites. It is only on July 20th that you see a correspondence between a positive T/E ratio and positive CIR, revealing very poor correlation between the tests at this lab. While the July 22-23 values again present a very large difference between the 5-alpha and 5-beta diols (3 to 3.5), a difference that you wouldn't expect to see based on the literature, one must note that it is *only* the 5-alpha that falls outside the normal range on these two dates. *All* other values, including the T/E ratio, were well within normal ranges. This type of profile is not anything we've seen in studies of high-mode men who have been administered exogenous testosterone. There is just not a physiological process that would yield such an abnormal for 5-alpha while all other values are normal, particularly days after the 5-alpha abnormality was accompanied by a high T/E ratio.

67. So what is being suggested by USADA is that Appellant not only took testosterone before the July 20th test, but also before the July 22nd and 23rd tests, with that conclusion being based solely on the abnormal 5-alpha-pdiol delta-delta value seen on July 22-23. This is completely implausible. If the administration of testosterone before the July 20 test resulted in an 11:1 T/E ratio--which is what Exhibit GDC 1363 shows--then you would *not* expect administration of testosterone before the July 22nd test to yield a T/E ratio of 2.5, nor would you expect to see a T/E ratio on July 23 of 1. While it is certainly true that individual T/E ratios may vary naturally, with some individuals being "high mode" and some "low mode," we do not see variations like this in the same individual, particularly the same individual when tested in three of four consecutive days. July 23--which one must remember was the *parade stage day*, in which the Tour leader is apparently never seriously challenged--presents particularly jarring results. The T/E ratio of 1 is a textbook normal ratio, but we see a large discrepancy (about 3.5) between 5-alpha and 5-beta. There is just no physiological explanation for this.

68. The T/E ratios reported for Appellant are those of a high-mode individual. If such an individual takes testosterone, his T/E ratio will always go into the abnormal range, and that is not what is depicted here for July 22 and particularly with July 23. Further, when such an individual takes testosterone, you would expect the high T/E ratio to be confirmed by increases in both 5-alpha and 5-beta on each day. This is not what we see in Appellant's results. Instead, there are abnormal values for 5-alpha alone on four of the five days that USADA claims that Appellant used testosterone, and on only one day is that abnormal reading confirmatory of a high T/E ratio. On only one day (and a different day, at that) is the abnormal 5-alpha value tracked by an abnormal 5-beta value. And of course, on this day (July 13), the T/E ratio was well within the

normal range. These results are simply inconsistent with one another, and inconsistent with what we know about the metabolism of testosterone based on the published literature.

69. As an aside, Appellant's T/E ratios, as depicted in Exhibit GDC 1363 and Exhibit 30, are those of a high-mode individual, in contrast to a study subject referred to as S1 in another paper by Baume and his colleagues: "Use of isotopic ratio mass spectrometry to detect doping with oral testosterone undecanoate: Inter-individual variability of $^{13}\text{C}/^{12}\text{C}$ ratio."³²

70. USADA's counsel, Mr. Young, questioned me about this individual at the hearing before the AAA Panel. As I indicated at that time, S1's T/E ratio did not change significantly with testosterone administration, though his testosterone metabolites, **androsterone and etiocholanolone** do behave precisely as expected—they both drop together and precipitously after the administration of testosterone, and then rise back to baseline in tandem.³³ So while his T/E ratio never became elevated, his testosterone metabolites *did* behave as one would have expected them to after testosterone administration. These sorts of results would be consistent with the profile of a low-mode individual, which S1 clearly was, having a baseline T/E ratio of 0.1, not 1.0 or higher. The results gathered from S1's urine would be particularly consistent in persons having a metabolism that rapidly converts testosterone. This was, in fact, the conclusion drawn by Baume and his team.³⁴ But the important thing is that S1's results were internally consistent; while his T/E ratio never rose significantly, his metabolites did. This stands in contrast to Appellant's results, which show a positive T/E ratio on July 20 accompanied by an abnormal

³² See USADA Exhibit 43 beginning at USADA 804.

³³ See *id.* at 807 (article page 367).

³⁴ See *id.* at 806 (article page 366).

diol value, but very *normal* T/E ratios on other days, still accompanied by the abnormal diol value. These results, unlike S1's, are internally *inconsistent*, and do not present a pattern that might allow one to conclude that Appellant's was either a low-mode individual like S1 (he was not)³⁵ or a person that metabolized testosterone quickly.

71. Nor are the results generated for Appellant's Stage 17 sample like those depicted for subject S2, shown in Figure 1 at USADA 807/Exhibit 43. Unlike Appellant's result, the results for subject S2 do exactly what you would expect: there is an elevated T/E ratio that goes up at the same time that the delta-deltas increase for androsterone and etiocholanolone. The metabolites all travel in tandem and parallel each nicely. The same can be said for subject S4-S7. These are all high-mode individuals like Appellant, but you see not only a spike in the T/E ratio after the testosterone administration, but you see androsterone and etiocholanolone moving in tandem with one another. This is exactly what we *don't* see in Appellant's results.

72. The results for subject S3 also differ markedly from Appellant's because none of S3's values are even abnormal. So you don't have a situation with one abnormal value, with all the remaining values reported as normal, which is what we see in Appellant's case.

73. The LNDD's CIR test results are also anomalous with respect to the results for the andro minus 11-keto value and the etio minus 11-keto values. One would also expect, based on the peer-reviewed literature, to see parallel changes in andro minus 11-keto value and the etio minus 11-keto values. That is certainly not the case with Appellant's July 20 results, as presented at Exhibit GDC 1363, which also adds to the implausibility of these results.

³⁵ Exhibit 30 and GDC 1363 reveal that Appellant's baseline T/E ratio is 1 or higher, confirming that he is a high-mode individual, unlike S1, whose T/E ratio was in the 0.1 range.

74. Nor can Appellant's results be explained by concluding that he must have taken oral testosterone, a synthetic steroid product that has a very short half-life in the body. That would in no way explain why some values were abnormal (5-alpha), while the T/E ratio and/or 5-beta diol were completely normal, which is what we see in Appellant's case. The implausibility of this hypothesis is heightened when we consider that there would be no benefit from taking testosterone that would wash out so quickly. If a person takes a single dose of 80-160 mg of testosterone undecanoate, it might wash out quickly, allowing the athlete to avoid detection, but it would also have no effect.

75. The LNDD results should not have been used to confirm that doping occurred because the results generated are implausible when compared to what we know about the metabolic pathways of testosterone, based on the published literature. In only two of the eight samples do we see good correlation between the 5-alpha and 5-beta (July 13 and July 14; on July 14, both values correlated well, but were negative), with an additional sample showing a high but plausible correlation between the two (July 11). There is only one day on which a positive 5-alpha occurs with a positive 5-beta (July 13), and no days on which a positive 5-alpha occurs with both a positive T/E ratio and a positive 5-beta.³⁶ In fact, given the AAA Panel's conclusion that the T/E ratio should not be relied upon at all because of LNDD's failure to analyze three diagnostic ions, the 5-alpha metabolite is the *only* positive result on *any* day other than July 13. This profile is not consistent with what we know about the behavior of metabolites in general,

³⁶ Only the T/E ratio and the 5-alpha are positive on July 20 once the .8 measurement of uncertainty for CIR is taken into consideration. Of course, the AAA Panel concluded that the T/E ratios could not be relied upon, so that 5-alpha metabolite no longer "confirms" any of the other values. Similarly, 5-beta is not positive on July 18 once the .8 measurement uncertainty for CIR is taken into consideration.

and it is not internally consistent because 5-alpha is repeatedly abnormal on days when all other values are normal.

The LNDD's chain of custody was compromised.

76. As a member of the USADA independent review board, I have participated in the review of 8-10 anti-doping cases. In these cases, we discuss the chain of custody in our conferences; establishing that a rigorous chain of custody was maintained is always an important part of the review.

77. When I received the lab documentation package generated by LNDD after analyzing Appellant's Stage 17 sample, I reviewed that package for purposes of satisfying myself that a proper chain of custody had been documented. As I have publicly stated—and testified about—a proper chain of custody is of *paramount* importance.

78. My belief that a proper chain of custody is of paramount concern is shared by WADA lab directors Don Catlin and Manfred Donike, who state that “scrupulous attention to chain of custody is required.”³⁷

79. There were errors in LNDD's chain of custody, including glaring errors with respect to misnumbering of the sample.³⁸ In my own experience as a doctor—both a researcher and a clinician—if you send a sample to the lab with one number and you receive lab results back that bear another number, you throw those lab results away because you just cannot be certain that those results belong to that particular patient. The ramifications could be immense, so you absolutely must retest the sample if a lab result sheet comes back bearing the wrong number. If

³⁷ “*Testing Urine for Drugs*,” International Federation of Clinical Chemistry, (1992), J. Automatic Chem., 14(3): 85-92 at 85 (1992), Exhibit GDC0219-232.

³⁸ See, e.g., USADA 008, 009, 024, 229, 288.

you are making treatment decisions based upon lab results, you must be confident that those treatment decisions are based upon data actually generated from your patient's own samples. Lives and health are at stake.

80. In this case, of course, LNDD was not making treatment decisions. Nevertheless, the ramifications of its conduct are immense for Appellant—to his reputation and his ability to make a living. Sample result records with obvious errors like incorrect sample numbers should have been retested, with the records corrected, and there is no evidence that this occurred.

81. I have reviewed the Declaration of Dr. Bruce Goldberger and concur with his testimony on the deficiencies in LNDD's chain of custody documentation. These deficiencies seriously undermine the reliability of the test results because there are no assurances that the integrity of the sample was not compromised.

CONCLUSION

82. It is my opinion that Appellant's CIR results are evidence that an unsupportable Adverse Analytical Finding was reported by the LNDD. The results are inconsistent with any physiologic process that has been reported in the scientific literature. As testified at length above, the T/E ratio and CIR data presented (see Ex. GDC 1363) are simply inconsistent with what we know about the results generated when exogenous testosterone is administered. Far from proving that Appellant took testosterone, these anomalous values actually support the conclusion that there was an error in the analysis, an error that caused implausible CIR results. The lack of correlation between the T/E ratio and CIR values, as well as the poor correlation between 5-alpha and 5-beta are, in my opinion, proof that the Adverse Analytical Finding is unreliable.

COMMENTS ON AAA PANEL DECISION

The Panel failed to consider the scientific implausibility of Appellant's results as evidence that mistakes had caused an unreliable AAF

83. The Panel repeatedly states that Appellant's CIR results contain evidence of the presence of exogenous testosterone.³⁹ The bulk of my testimony expresses an opinion to the contrary. Because the metabolic pattern presented by LNDD's results does not correspond to a pattern caused by any known metabolic process, caused by any known metabolic process, and are inconsistent with a large amount of published, peer-reviewed, scientific literature in the area, one must conclude that they are inaccurate. Far from supporting the conclusion that testosterone had been taken, those CIR results convince me that there was a problem with the analysis that lead to the report of implausible results.

84. As a corollary to this opinion, it is my opinion that one questionable set of data cannot "confirm" another. For example, on page 73, ¶268 and page 74, ¶270, the Panel indicated that it was confident that a technical breach of the chain of custody could not have caused the AAF because the results of the A sample were "confirmed" by the B sample. It is my opinion that this statement is insupportable. Appellant's B sample results revealed the same anomaly that the A sample results did—the 5-alpha and the 5-beta did not vary in tandem with one another. One questionable set of data cannot confirm another.

COMMENTS ON USADA'S APPELLATE BRIEF

85. I have reviewed the brief that USADA submitted to this Panel in response to Appellant's appeal. As a general proposition, of course, I disagree with the brief insofar as it

³⁹ See, e.g., Panel Decision at 43.

attempts to defend the lab results generated by LNDD, which appear to me to be scientifically indefensible, for the reasons outlined in detail above. In addition, I have a few specific responses to particular statements made in that brief.

LNDD's CIR results do not establish that Appellant took exogenous testosterone

86. As one would expect, USADA repeatedly asserts that the CIR result confirms that Appellant took exogenous testosterone.⁴⁰ The bulk of my opinion concerning the CIR results generated by LNDD makes the point that these results, far from confirming a positive T/E ratio--are scientifically unreliable because those results simply do not correspond in any way to what we know about testosterone metabolism. First, you do not see individuals whose 5-alpha and 5-beta fail to move in tandem, which is what you see in Appellant's CIR results, including those generated after Stage 17. In normal circumstances, the difference between the 5-alpha and the 5-beta delta-delta values would not exceed one, and only in rare cases would it rise as high as two. Yet, as the figures at page 27 and 28 of USADA's brief reveal,⁴¹ the difference between the 5-alpha diol and 5-beta diol values on the A sample was 3.99, while the difference on the B sample was 3.74. This is a variance that is unsupported in the literature, and not caused by any known physiologic process. Worse, you simply do not see accounts in the peer-reviewed literature documenting cases in which a subject takes testosterone on Day 1 [e.g., July 20], and then submits a urine sample generating an abnormal T/E ratio and an abnormal value for 5-alpha diol alone (with a difference of almost 4 between the 5-alpha diol and the 5-beta diol), and then see

⁴⁰ See, e.g., USADA Brief at 24, 27, 87.

⁴¹ It does appear that USADA concedes that only the 5-alpha values lie in the abnormal range once the measurement uncertainty of .8 is accounted for, a position the agency appeared to contest before the AAA Panel.

the same individual take testosterone on Days 3 and 4 [July 22 and 23], evidencing a completely *normal* T/E ratio and an abnormal value for 5-alpha diol, again with the difference between 5-alpha and 5-beta ranging in excess of 3. Nor does the literature support the contention that a one-time administration of testosterone before the Day 1 test would persist in this preferential manner, favoring the 5-alpha diol so heavily. So I cannot agree that his CIR test results establish that he took exogenous testosterone. Instead, those results suggest to me that the LNDD failed to pay the “scrupulous attention to detail” and “strict adherence” to quality control that Dr. Aguilera asserted was crucial to proper use of the CIR technique, resulting in the generation of unreliable results.⁴²

LNDD’s results *are* inconsistent with known science.

87. Beginning at page 80 of its brief, USADA directly challenges the testimony I offered before the Panel. USADA’s argument obfuscates several important scientific points by painting with too broad a brush, and with too little supporting authority.

88. For example, USADA implies that because individual metabolism varies, because different modes of administration affect metabolism, because the dosage and timing of testosterone influence results, then generalizations cannot be made, based on the scientific literature, about the way testosterone is metabolized in normal, healthy men. This is incorrect.

89. It is true, for example, that some individuals metabolize testosterone at different rates, while some produce less testosterone in relation to epitestosterone. This fact does *not* mean, however, that when testosterone is converted into its metabolites, it does so randomly or

⁴² R. Aguilera et al., “Performance Characteristics of a Carbon Isotope Ratio Method for Detecting Doping with Testosterone Based on Urine Diols: Controls and Athletes with Elevated Testosterone/Epitestosterone Ratios,” USADA Exhibit 40, beginning at USADA 1225, at USADA 1226, 1231.

unpredictably, or that an individual's metabolism of testosterone will change dramatically from day to day. The scientific literature confirms that the metabolites, particularly 5-alpha diol and 5-beta diol will vary together, in close parallelism. While the *values* of the change might vary between the high and low mode individual, the patterns do not. Nor is it plausible to suggest that the same individual might metabolize testosterone in a widely varying fashion over the course of a week's time, which appears to be what USADA suggests, based on the results presented in GDC 1363. These points are neatly demonstrated by the Baume study discussed above, where subject S1—clearly a low-mode individual—had a low T/E ratio, even after testosterone gel administration, but had high values of urinary testosterone metabolites, which moved as we would expect them to and in a consistent fashion, in tandem and at the same direction at approximately the same time.⁴³

90. USADA makes the same mistake with respect to mode of administration. While mode of administration impacts timing, it does not significantly impact the patterns we see with respect to the changes in delta delta values of the various metabolites. As discussed above, one might have *expected* to see variation depending upon whether the testosterone was injected, taken orally, or administered as a gel, but we do not. The scientific literature simply does not support the implication that the conversion of testosterone into its metabolites varies markedly by mode of administration. USADA cites a paper by Dr. Schanzer, who has drawn a conclusion about the influence of 5-alpha reductase involvement in the metabolism of testosterone administered in gel form that is completely inconsistent with the peer-reviewed literature (Dr. Schanzer's study is *not* peer-reviewed) and with the clinical experience.

⁴³ See Baume, Exhibit 34, at USADA 806-7.

91. USADA states on page 81 that the “studies which use CIR to look at the metabolism of exogenous testosterone support the conclusions that there are significant differences in the innate metabolic preferences between individuals favoring either the 5-alpha or 5-beta pathways.”⁴⁴ USADA then cites five studies measuring 5-alpha and 5-beta, which it argues “were discussed” at the hearing before the Panel. While they may have been *discussed*, it cannot be said that the studies cited all provide support for the proposition that 5-alpha and 5-beta vary in non-parallel fashion.

92. For example, the Shackelton study does not support USADA’s position, but instead clearly supports the contention that one would expect to see 5-alpha and 5-beta move in close parallelism. I testified extensively about this paper in the hearing below, and I have discussed this paper at length above, paying particular attention to Figure 1, at USADA 1245 (Ex. 40), which shows that the 5-alpha and 5-beta do move in tandem in the manner described, a pattern we do *not* see repeated in Appellant’s results.

93. USADA itself concedes that the results of the 1999 Aguilera study cited overwhelmingly support the notion that 5-alpha and 5-beta will move in tandem. On page 83 of its brief, USADA concedes that in nine of the 10 subjects studied in the 1999 paper, 5-alpha and 5-beta metabolised in a “relatively similar” manner.

94. The Schanzer “study,” has been extensively discussed above. It was not a peer-reviewed study, but only a progress report analyzing CIR tests from only two individuals, hardly sufficient to challenge the scientific consensus on this point. It also contains the incorrect hypothesis about the involvement of 5-alpha reductase in metabolism of testosterone

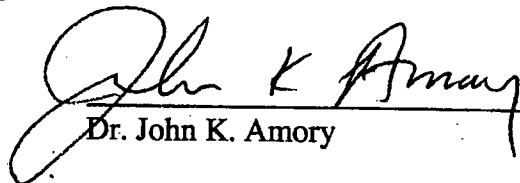
⁴⁴ USADA Response Brief at 81.

administered by gel, a conclusion supported by neither clinical results nor published literature, something that surely would have been pointed out had the study been submitted for publication in a peer-reviewed journal.

95. Notably absent from the list of studies cited by USADA was the 2006 study by Baume, which USADA presented at the hearing as Exhibit 43, and about which I testified extensively at the arbitration hearing. That study very clearly demonstrates at Figure 1 that even where you have clear differences in the modes among study subjects (compare, e.g., Subjects S1 and S3 with the other subjects), the testosterone metabolites **androsterone and etiocholanolone** move in parallel to one another.

96. In short, LNDD's CIR test results convince me that the data are unreliable because they do not correspond to what is known about the physiologic metabolism of testosterone. They are internally inconsistent, documenting a metabolic process that varies markedly from day to day, and they are inconsistent with the literature. So I cannot agree with USADA's conclusion that these CIR test results confirm either the flawed T/E ratio test or the general contention that Appellant took synthetic testosterone.

I declare under penalty of perjury under the laws of the United States of America that the foregoing is true and correct. This declaration was executed on March 7, 2008, in Seattle Washington.


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PERSONAL INFORMATION

Born: April 19, 1967, Vancouver, British Columbia, Canada (US citizen)

Married: October 7, 1995 to Josephine Harris Amory MD (Obstetrics)

Children: William Glendinning Amory, born July 5th, 2002

Thomas Gerard Amory born Feb 1st, 2005

EDUCATION

2004-2006 MPH, University of Washington, Seattle, WA

1989-1994 MD with Thesis, University of California, San Francisco, CA

1985-1989 BA Biology-*Magna cum Laude*, Harvard University, Cambridge, MA

POSTGRADUATE TRAINING

1994-1997 Resident in Internal Medicine, University of California, San Francisco, CA

1992-1993 Research Fellow, National Institutes of Health, Bethesda, MD

FACULTY POSITIONS

2005- Associate Professor of Medicine, University of Washington, Seattle, WA

2001-2005 Assistant Professor of Medicine, University of Washington, Seattle, WA

1997-2001 Acting Instructor of Medicine, University of Washington, Seattle, WA

HOSPITAL POSITIONS

2001- Attending Physician, University of Washington, Seattle, WA

1997-2001 Staff Physician and Director Pre-op clinic, VA-Puget Sound, Seattle, WA

HONORS & AWARDS

2007	Young Andrologist Award, American Society of Andrology
2006	Named one of "Seattle's Best Doctors" by <i>Seattle Magazine</i> (Internal Med)
2005	Endocrine Society International Award for Excellence in Published Clinical Research
2004	Helen and Phillip Fialkow Scholar, Department of Medicine, University of Washington
2003	Paul Beeson Award for excellence in Housestaff Instruction, Department of Medicine, University of Washington
2003	AOA honorary inductee, University of Washington Medical School graduating class of 2003
2003	Fellowship, American College of Physicians
1997	Weingarten Memorial Award for outstanding third-year medicine resident, University of California, San Francisco
1996	Martin Memorial Award for outstanding second-year medicine resident, University of California, San Francisco
1992	Dean's Prize for Medical Student Research, UCSF
1990	Dr. June Colburn Research Fellow, UCSF
1987	John Harvard Scholar
1985	National Merit Scholar

CERTIFICATIONS & LICENSURE

1997-present	American Board of Internal Medicine, Board Certified in Internal Medicine (renewed 2007-2017)
1997-present	Washington Medical License
1996-present	DEA License

PROFESSIONAL ORGANIZATIONS

1997-present	American College of Physicians
1998-present	The Andrology Society
2003-present	The Endocrine Society
2003-present	Association of Reproductive Health Professionals (Planned Parenthood)

TEACHING RESPONSIBILITIES**YEARLY:**

1. Four weeks ward attending in general inpatient medicine. Responsible for teaching rounds and overseeing patient management.
2. Multiple lectures to residents on general medicine topics including thyroid disease, nutrition, contraception, reproductive disorders, medication side effects and peripheral vascular disease.
3. Human Biology 565 (2nd year medical student course on human reproduction). Co-course director (with Robert Steiner, PhD). 5 hours of course lectures yearly: "The

Adult Male," "Male Infertility," "Male Contraception," "Population Dynamics,"
 "Androgens in the Aging Male."

4. Human Biology 665 (3rd year medical student medical clerkship lecture series). 4 hours of course lectures yearly (Acute Renal Failure)
5. Human Biology 544 (2nd year medical student course on endocrinology). Small group leader "Hypothyroidism" "Adrenal Insufficiency"

EDITORIAL RESPONSIBILITIES

2008-present Editorial board for *Journal of Andrology*,

At-large reviewer for *Journal of Clinical Endocrinology and Metabolism*, *Endocrinology*, *Human Reproduction*, *Asian Journal of Andrology* and many others

SPECIAL NATIONAL RESPONSIBILITIES

2004-2007 United States Anti-Doping Agency, anti-doping review board
 2005-present Reviewer, Faculty of 1000, Reproductive Physiology
 2008-present Therapeutic Use Exemption Advisory Committee, US Professional Golf Association (PGA) Tour
 2008-present Scientific and Medical Advisory Board, Agency for Cycling Ethics

UNIVERSITY SERVICE

2004-present Human Subjects (IRB) Committee A-member
 2004-2007 Medical School Admissions Committee
 2003-present Residency Selection Committee
 2002-2004 Faculty Senate-member
 2001-present UW Hospital Nutrition Committee (Chair)

CURRENT RESEARCH FUNDING

1. NIH/NICHD
 1K23HD045386, 9/04-9/09
PI: John K. Amory Total costs: \$629,668
 Oral Androgen Administration and Function in Man
2. NIH/NICHD
 U54 HD012629-27
 Center P.I: Robert Braun
 Project V PI: William J. Bremner 4/06-3/11
John K. Amory, Co-investigator Total costs: \$10.6 m
 Project V: Gonadotropin Regulation Project V costs: 1.5m
 Steroidogenesis, spermatogenesis and gene
 Expression in man
3. NIH/NICHD
 5 U54 HD 042454

Center PI: William J. Bremner
 Project I PI: **John K. Amory**
 Oral testosterone for male hormonal contraception

3/07-2/12
 Total costs: 13.5m
 Project I costs: 2.2m

4. NIH/NICHD
 5 K12 HD-053984
 PI: William J. Bremner
 Research Mentor/Program Director: **John K. Amory**
 Men's Reproductive Health Research
 at the University of Washington/
 K12 Training program
 9/06-9/11
 Total costs: 2.2 m
5. GlaxoSmithKline
 TDC106220
 PI: **John K. Amory**
 Oral nanomilled testosterone in hypogonadal men
 Dates: 1/07-1/08
 Total costs: \$310,821
6. Merrion Pharmaceuticals
 PI: **John K. Amory**
 Oral administration of the GnRH antagonist acyline
 In normal men
 Dates: 3/07-3/08
 Total costs: \$134,000

COMPLETED RESEARCH FUNDING

1. American Professors of Infection Control.
 P.I. **John K. Amory**
 Computer-based prevention of urinary tract infection
 1/00-1/01
 Total costs: \$8,000
2. University of Washington, Center for Research in
 Reproduction and Contraception
 PI: **John K. Amory**
 Contraceptive Pilot Grant: Glycosphingolipid Inhibition
 and Spermatogenesis in Men
 5/04-5/06
 Total costs: \$68,211
5. Schering Pharmaceuticals
 PI: **John K. Amory**
 Glycosphingolipid Inhibition and Spermatogenesis in Men
 3/05-8/06
 Total costs: \$150,000
4. GlaxoSmithKline
 PI: **John K. Amory**
 Oral Androgens in Man
 12/04-12/06
 Total costs: \$169,000
5. NIH/NICHD
 Contraceptive Clinical Trials Network
 PI: William J. Bremner
John K. Amory, Co-investigator
 Contract #: HHSN27520040337

- | | |
|--|--|
| <p>Safety and Gonadotropin Suppressive Activity of
Nestorone Gel and Testosterone Gel in Men</p> | <p>4/05-4/07
Total costs: \$225,866</p> |
| <p>6. University of Washington, Center for Research in
Reproduction and Contraception
PI: John K. Amory
Contraceptive pilot grant: Gonadotropin suppression
with oral testosterone enanthate and finasteride
in man</p> | |
| | <p>6/06-5/07
Total costs: \$35,000</p> |
| <p>7. NIH/NICHD
U54 HD42454
Center PI: William J. Bremner
Project I PI: William J. Bremner
John K. Amory, Co-investigator
Project I: Hormonal Contraception
In the Male</p> | |
| | <p>9/02-9/07

Total costs: \$9.5 m
Project I costs: 1.5m</p> |

PUBLICATIONS

PEER-REVIEWED ARTICLES

1. **Amory JK**, Chou T, Redberg R, Blake L, Vartanian R. Diagnosis of a Primary Cardiac Leiomyosarcoma by Endomyocardial Biopsy. *Cardiovascular Pathology* 1996, Vol 5(2): 113-117.
2. Eisner M, **Amory JK**, Mullaney B, Tierney L Jr., Browner WS. Necrotizing lymphadenitis associated with systemic lupus erythematosus. *Seminars in Arthritis and Rheumatism* 1996, 26(1): 477-482.
3. **Amory JK**, Matsumoto AM. The therapeutic potential of testosterone patches. *Expert Opinion in Investigational Drugs* 1998 7(12): 1977-1985.
4. **Amory JK**, Bremner WJ. The Use of Testosterone as a Male Contraceptive. *Balliere's Clinics in Endocrinology* 1998 12(3): 471-483.
5. **Amory JK**, Bremner WJ. Newer agents for hormonal contraception in the male. *Trends in Endocrinology and Metabolism* 2000, 11(2): 61-66.
6. **Amory JK**, Anawalt BD, Paulsen CA, Bremner WJ. Klinefelter syndrome: a brief review with a biography of Dr. Klinefelter. *Lancet* 2000, 356:333-335.
7. Kahn JG, Becker BJ, MacIssac L, **Amory JK**, Neuhaus J, Olkin I, Creinin M. The efficacy of medical abortion: A meta-analysis. *Contraception* 2000, 61:29-40.
8. Saint S, Wiese J, **Amory JK**, Bernstein ML, Patel UD, Zemencuk JK, Bernstein SJ, Lipsky BA, Hofer TP. Are physicians aware of which of their patients have an indwelling urinary catheter? *American Journal of Medicine* 2000, 109:476-480.
9. **Amory JK**, Bremner WJ. Endocrine regulation of testicular function in men. *Molecular and Cellular Endocrinology* 2001, 182:175-179.

10. Goldstein AS, **Amory JK**, Martin SM, Vernon C, Matsumoto AM, Yager P. Testosterone delivery using glutamide based complex high axial ratio microstructures. *Bioorganic and Medicinal Chemistry* 2001, 9:2819-2825.
11. **Amory JK**, Anawalt BD, Bremner WJ, Matsumoto AM. Daily testosterone and gonadotropin levels are similar in azoospermic and nonazoospermic normal men administered weekly testosterone: implications for male contraceptive development. *Journal of Andrology* 2001, 22:1053-1060.
12. Anawalt BD, **Amory JK**. Male hormonal contraceptives. *Expert Opinion in Pharmacotherapeutics* 2001, 2:1389-1398.
13. Anawalt BD, **Amory JK**. Male contraception. *Annals of Medicine* 2001; 587-595.
14. **Amory JK**, Anawalt BD, Blaskovich PD, Gilchrist J, Nuwayser ES, Matsumoto AM. Testosterone release from a subcutaneous, biodegradable microcapsule formulation (Viatrel) in hypogonadal men. *Journal of Andrology* 2002, 23:84-91.
15. Amory DW, Grigore A, **Amory JK**, Gerhardt MA, White WD, Smith PK, Schwinn DA, Reves JG, Newman MF. Neuroprotection is associated with beta-adrenergic receptor antagonists during cardiac surgery: Evidence from 2,575 patients. *Journal of Cardiothoracic and Vascular Anesthesia* 2002, 16:270-277.
16. Cherrier MM, Anawalt BD, Herbst KL, **Amory JK**, Craft S, Matsumoto AM, Bremner WJ. Cognitive effects of short-term manipulation of serum sex steroids in healthy young men. *Journal of Clinical Endocrinology and Metabolism* 2002, 87:3090-3096.
17. Herbst KL, Anawalt BD, **Amory JK**, Bremner WJ. Acyline: the first study in humans of a potent, new gonadotropin-releasing hormone antagonist. *Journal of Clinical Endocrinology and Metabolism* 2002, 87:3215-3220.
18. **Amory JK**, Chansky HA, Chansky KL, Camuso M, Hoey C, Anawalt BD, Matsumoto AM, Bremner WJ. Preoperative supraphysiological testosterone in older men undergoing knee replacement surgery. *Journal of American Geriatrics Society* 2002, 50:1698-1701.
19. Herbst KL, Anawalt BD, **Amory JK**, Matsumoto AM, Bremner WJ. The male contraceptive regimen of testosterone and levonorgestrel significantly increases lean mass in healthy young men in 4 weeks but attenuates a decrease in fat mass induced by testosterone alone. *Journal of Clinical Endocrinology and Metabolism* 2003, 88:1167-1173.
20. **Amory JK**. Male Contraception. *A.R.T. and Science* 2003; 2(4):8-11.
21. Cornia P, **Amory JK**, Fraser S, Saint S, Lipsky B. Computer-based order entry decreases duration of indwelling urinary catheterization in hospitalized patients. *American Journal of Medicine* 2003, 114:404-407.
22. **Amory JK**, Bremner WJ. Regulation of testicular function in experimental male contraceptive development. *Journal of Steroid Biochemistry and Molecular Biology* 2003, 86:357-361.
23. Herbst KL, **Amory JK**, Brunzell JD, Chansky HA, Bremner WJ. Testosterone administration to men increases hepatic lipase activity and decreases HDL and LDL size in 3 weeks. *American Journal of Physiology: Endocrinology and Metabolism* 2003, 284:E1112-E1118.

24. **Amory JK**, Scriba GKE, Amory DW, Bremner WJ. Oral testosterone-triglyceride conjugate in rabbits: Single-dose pharmacokinetics and comparison with oral testosterone undecanoate. *Journal of Andrology* 2003; 24:716-720.
25. **Amory JK**. George Washington's infertility: why the father of our country was never a father. *Fertility and Sterility* 2004; 81:495-499.
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41. **Amory JK.** Male Hormonal Contraception: Current status and future prospects *Treatments in Endocrinology* 2005; 4(6):333-41.
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43. ***Amory JK, Page ST, Bremner WJ.** Oral testosterone in oil: Pharmacokinetic effects of 5 α reduction with finasteride or dutasteride and food intake in men. *Journal of Andrology* 2006; 27:72-78.
44. **Amory JK, Page ST, Bremner WJ.** Recent progress in male hormonal contraception. *Nature Clinical Practice: Endocrinology and Metabolism* 2006;2:32-41.
45. Page ST, Plymate SR, Bremner WJ, Hess DL, Matsumoto AM, Lin DW, **Amory JK**, Nelson PS, Wu JD. Effects of medical castration and testosterone replacement on CD4+CD25+ regulatory T cells, CD8+ T-cell IFN γ expression and NK cells: Evidence for a physiological role for testosterone and/or its metabolites in cellular immune function *American Journal of Physiology: Endocrine and Metabolism* 2006 290(5):E856-63
46. **Amory JK, Bremner WJ.** Male hormonal contraception: the future of male contraception? *Medicine* 2006 34:1 25-26.
47. **Amory JK, Rosen H, Sukit C, Wallace F, Saint S.** A Jaundiced Eye: Clinical Problem Solving. *New England Journal of Medicine* 2006 324; 14:62-66.
48. **Amory JK.** Male hormonal contraceptives. *Minerva Ginecol.* 2006 58:215-26.
49. Page ST, Lin DW, Mostaghel EA, Hess DL, True LD, **Amory JK**, Nelson PS, Matsumoto AM, Bremner WJ. Persistent intraprostatic androgen concentrations

- after medical castration in healthy men. *Journal of Clinical Endocrinology and Metabolism* 2006 91:3850-3856.
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 51. Kalus R, Shojania KG, **Amory JK**, Saint S. Lost in Transcription: Clinical Problem Solving. *New England Journal of Medicine* 2006 355:1487-1491.
 52. Arias E, **Amory JK**. Testosterone and the Male Heart: Friend or Foe? *Renal and Urology News* 2006 1(4):1-8.
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 54. **Amory JK**, Muller CH, Page ST, Pagel ER, Bhandari A, Leifke E, Subramanyam B, Bone W, Radlmaier A, Bremner WJ. Miglustat has no apparent effect on spermatogenesis in normal men. *Human Reproduction* 2007 22:702-707.
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 56. ***Amory JK**, Wang C, Swerdloff R, Anawalt BD, Matsumoto AM, Bremner WJ, Walker SE, Haberer LJ, Clark RV. The Effect of 5 α -Reductase Inhibition with Dutasteride and Finasteride on Semen Parameters and Serum Hormones in Healthy Men. *Journal of Clinical Endocrinology and Metabolism* 2007; 92:1659-1665.
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- suppression induced by male hormonal contraceptive treatment *Journal of Andrology* 2007 28 (5): 734-41.
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65. **Amory JK**, Kalhorn T, Page ST. Pharmacokinetics and pharmacodynamics of oral testosterone enanthate in oil plus dutasteride for four weeks in normal men: Implications for male hormonal contraception. *Journal of Andrology* Epub 11-07
66. **Amory JK**. Progress and prospects in male hormonal contraception. *Current Opinion in Endocrinology and Diabetes* (in press).
67. **Amory JK**, Anawalt BD, Matsumoto AM, Page ST, Bremner WJ, Wang C, Swerdloff RS, Clark RV. The effect of 5 α -reductase inhibition with dutasteride and finasteride on bone mineral density, serum lipoproteins, hemoglobin, PSA and sexual function in healthy young men. *Journal of Urology* (in press).
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69. **Amory JK**. "Common problems of the elbow." In: Frances C, Bent S and Saint S Eds. *Saint-Francis Guide to Outpatient Medicine*. Lippincott Williams and Wilkins, Philadelphia 1999:365-368.
70. **Amory JK**. "An approach to injection and aspiration of joints." In: Frances C, Bent S and Saint S Eds. *Saint-Francis Guide to Outpatient Medicine*. Lippincott Williams and Wilkins, Philadelphia, 1999:335-339.
71. **Amory JK**, Bremner WJ. "Male contraception" In *Principles and Practice of Endocrinology and Metabolism* (3rd Ed.) Becker KL (Ed.) Lippincott Williams and Wilkins, Philadelphia 2001: 1220-1224.
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81. **Amory JK**. "Klinefelter Syndrome" in *The Encyclopedia of Human Development*. Salkind, Neil J. (Ed.). Sage Publications, Thousand Oaks, CA, 2005.
82. **Amory JK**. "Adult diabetic ketoacidosis" in *Manual of Evidence-Based Admitting Orders and Therapeutics* (5th Ed.). Eds. Eric B. Larson and Karen McDonough, Elsevier, London, 2006: 43-47.
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OTHER PUBLICATIONS

84. **Amory JK**: Preparation, titer, affinity and use of immunoglobulin isolated from eggs of immunized hens. (Undergraduate Thesis) Harvard College, 1989.
85. **Amory JK**: The role of Pr60^{gag} in the immunopathogenesis of Murine AIDS. (MD Thesis) University of California, San Francisco, 1994.
86. **Amory JK**. Cancer therapy and sperm banking (letter to the editor). *New England Journal of Medicine*, 2004 351:510.
87. **Amory JK**, Amory DW. Oral erythromycin and the risk of sudden death (letter to the editor). *New England Journal of Medicine* 2005 352:301-304.
88. **Amory JK**. The effect of 5 α -reductase inhibition on spermatogenesis in normal men. (MPH Thesis), University of Washington, Seattle, 2006.
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CURRICULAR PUBLICATIONS

1. **Amory JK.** Human Biology 565 syllabus: "The Adult Male," "Male Infertility," "Male Contraception," "Population Dynamics," "Androgens in the Aging Male." (yearly)
2. Bussell S, DeHoog S, Billingsly S, **Amory JK.** "Hospital Nutrition for 3rd year medical students." <http://courses.washington.edu/med665/nutrition/>

PATENTS

1. "Oral Androgen Therapy Using Modulators of Testosterone Bioavailability" US Patent # 10,990,118, authors: John K. Amory and William J. Bremner (Holder University of Washington)-issued 11/21/06.

MANUSCRIPTS SUBMITTED

1. Matthiesson KL, Meachem SJ, **Amory JK**, Robertson DM, Bremner WJ, McLachlan RI. Relationship of serum INSL3 to germ cell and Leydig cell parameters in normal men receiving male hormonal contraceptive treatment. (Submitted to *Journal of Clinical Endocrinology and Metabolism*).
2. Kalus A, Fredericks LP, Presland R, Livingston B, **Amory JK**, Sonesson A, Back O, Dale B. Human-B-defensin 1 polymorphism is associated with allergic sensitization in atopic dermatitis. (Submitted to *American J Dermatology*)
3. Roth M, **Amory JK**, Page ST. Male infertility in the setting of morbid obesity. (Submitted to *Nature Clinical Practice: Endocrinology and Metabolism*)
4. Page ST, **Amory JK**, Matsumoto AM. Effect of testosterone supplementation on function mobility. (Submitted to *Nature Clinical Practice: Endocrinology and Metabolism*)
5. Cherrier M, **Amory JK**, Shen D. Cognitive effects of opiates in older adults (manuscript in preparation)

ABSTRACTS

1. **Amory J**, Kliks S, Levy J: Effect of Anti-HIV Monoclonal Antibodies on HIV-1 Replication *in vitro*. Clinical Research, 1991 39:58A.
2. **Amory J**, Martin N, Levy J, and Wara D: The Large Molecular Weight Glycoprotein MG1, A Component of Human Saliva Inhibits HIV-1 Infectivity. Clinical Research, 1992 40:51A.
3. Anawalt BD, **Amory JK**, Herbst KL, Matsumoto AM, Bremner WJ. Testosterone administration to normal men decreases truncal and total body fat. Clinical Research, 1999
4. **Amory JK**, Chanksy H, Chansky K, Hoey C, Anawalt BD, Matsumoto AM and Bremner WJ. The effects of supraphysiologic testosterone on functional outcomes after joint replacement surgery in elderly men. ENDO 99, OR 9-3 (oral presentation).
5. **Amory JK**, Anawalt BD, Matsumoto AM. Safety and pharmacokinetics of testosterone release from an injected microcapsule in hypogonadal men. American Society of Andrology, 2000.

6. **Amory JK**, Bremner WJ, Herbst KL, Matsumoto AM, Anawalt BD. Testosterone rapidly increases vertebral bone mineral density in eugonadal men. ENDO 2000, Abstract #852276.
7. Herbst KL, Anawalt BD, **Amory JK**, Matsumoto AM, Bremner WJ. Exogenous testosterone administration to normal men changes body composition from fat to lean without changing body weight. ENDO 2000, Abstract #2354.
8. Dick SE, Anawalt BD, **Amory JK**, Herbst KL, Bremner WJ, Matsumoto AM, Lower dose levonorgestrel plus testosterone enanthate effectively suppresses spermatogenesis with little or no weight gain. ENDO 2000, abstract #2353.
9. Herbst KL, Deeb SS, Bremner WJ, **Amory JK**. Testosterone increases hepatic lipase and decreases HDL-C in three weeks in elderly men. *W Soc Clin Investigation* 2002.
10. Anawalt BD, **Amory JK**, Wang C, Swerdloff RS, Dobs AS, Meikle AW, Elbers JMH, Houwing NS. A pharmacokinetic study of oral testosterone undecanoate. *Andrology Society* 2002.
11. Herbst KL, Anawalt BD, Coviello A, **Amory JK**, Bremner WJ. Acyline: a New potent, long-acting Gonadotropin-releasing hormone (GnRH) antagonist safely and significantly suppresses testosterone for greater than two weeks after a single dose. Endocrine Society 2002, Abstract # P2-654.
12. Herbst KL, **Amory JK**, Chansky HA, Bremner WJ. Supraphysiological testosterone administration rapidly increases hepatic lipase activity (HLA) and decreases HDL(2) and HDL (3) and LDL size in older men. Endocrine Society 2002, Abstract #P2-653.
13. Coviello A, **Amory JK**, Anawalt BD, Matsumoto AM, Bremner WJ. Gonadotropins are higher in men who fail to suppress to azoospermia compared to those who do not. Endocrine Society 2002, Abstract #
14. Voie A, Amory D, **Amory JK**, Moehring M, Spencer M. Ultrasound and tPA enhanced thrombolysis: influence of ultrasound frequency and pulse length. European Society of Neurosonology and Cerebral Hemodynamics, 2002, P157
15. Coviello AD, Herbst KL, **Amory J**, Anawalt B, Jarow JP, Brown T, Wright B, Bremner W, Matsumoto AM. Intratesticular testosterone is 40 fold higher than serum testosterone in normal men but is successfully suppressed by testosterone enanthate and levonorgestrel. *W Soc Clin Investigation*, 2003
16. Herbst KL, Coviello AD, **Amory JK**, Anawalt BD, Murdoch SJ, Brunzell JD, Bremner WJ. Acyline-induced hypogonadism rapidly increases high-density lipoprotein cholesterol, low-density lipoprotein buoyancy and insulin levels. *W Soc Clin Investigation* Abstract # 2003
17. **Amory JK**, Watts NB, Easley KA, Sutton PR, Anawalt BD, Matsumoto AM, Bremner WJ, Tenover JL. Testosterone plus finasteride for 3 years increases bone mineral density without increasing prostate volume in older men with low serum T. *American Society for Andrology*, 2003, Abstract #10 (Platform presentation).
18. Coviello KL, Herbst KL, **Amory JK**, Anawalt BD, Yan X, Brown T, Wright B, Bremner WJ, Matsumoto AM, Jarow JP. Exogenous testosterone plus levonorgestrel profoundly suppresses intratesticular testosterone and androgenic bioactivity. *American Society for Andrology*, 2003, Abstract #96.

19. Page ST, Herbst KL, **Amory JK**, Coviello AD, Anawalt BD, Matsumoto AM, Bremner WJ Short term effects of androgen manipulation on adiponectin in normal men *Journal of Investigative Medicine*. 52 (1): S97-S98 JAN 2004
20. Coviello AD, Herbst KL, **Amory JK**, Anawalt BD, Sutton PL, Wright WW, Brown T, Yan X, Bremner WJ, Matsumoto AM, Zirkin BR, Jarow JP Low dose human chorionic gonadotropin maintains intratesticular testosterone in normal men following gonadotropin suppression. *Journal of Investigative Medicine*. 52 (1): S99-S99 JAN 2004
21. Coviello AD, Herbst KL, **Amory JK**, Anawalt BD, Jarow JP, Brown T, Wright W, Bremner WJ, Matsumoto AM Intratesticular testosterone concentrations comparable to serum levels are not sufficient to maintain normal sperm production in men. *Journal of Investigative Medicine* 51: 365 Suppl. 1 FEB 2003
22. Herbst KL, Coviello AD, **Amory JK**, Anawalt BD, Murdoch SJ, Brunzell JD, Bremner WJ. Acyline-induced hypogonadism rapidly increases HDL cholesterol, LDL buoyancy and insulin levels. *Journal of Investigative Medicine* 51: 410 Suppl. 1 FEB 2003
23. Page ST, **Amory JK**, Bowman ED, Anawalt BD, Matsumoto AM, Bremner WJ, Tenover JL. Exogenous testosterone or testosterone with finasteride improves physical performance and increases lean body mass in older men with low serum testosterone. International conference on endocrinology, 9/04.
24. **Amory JK**, Page S, Bremner WJ. Absorption of oral testosterone in oil is augmented by 5a reductase inhibition in man. *American Society of Andrology* 4/05 (oral presentation).
25. Page ST, Lin D, Nelson P, **Amory JK**, Matsumoto AM, Bremner WJ. The effect of medical castration on hormones PSA and prostate size in normal middle-aged men. *Endocrine Society* 2005.
26. Page ST, **Amory JK**, Anawalt BD, Matsumoto AM, Brockenbrough AT, Irwig MS, Bremner WJ. Is there a role for GnRH antagonists in male hormonal contraception? *Journal of Investigative Medicine* 54:S95.
27. **Amory JK**, Muller C, Page ST, Pagel E, Bhandari A, Leifke E, Bone W, Radlmiel A, Bremner WJ. The effect of miglustat on spermatogenesis in normal men: A pilot study. ENDO 88th Annual meeting,
28. Page ST, Bremner WJ, Clark RB, Bush MA, Carifcofe R, Smith PM, Amory JK. Oral nanomilled testosterone (T) plus dutasteride normalizes serum T in medically castrated men: A pharmacokinetic study. ENDO 88th Annual meeting.
29. **Amory JK**, Page ST, Anawalt BD, Coviello AD, Matsumoto AM, Bremner WJ. Elevated serum INSL3 is associated with failure to completely suppress spermatogenesis in men receiving male hormonal contraception. ASA meeting 4/22/07
30. **Amory JK**, Coviello AD, Page ST, Anawalt BD, Bremner WJ. Serum 17-hydroxyprogesterone strongly correlates with intratesticular testosterone in gonadotropin-suppressed normal men receiving various dosages of human chorionic gonadotropin ENDO 89th annual meeting

Mentoring

1. Kati Matthiesson MD, 2003-2004, Effect of acyline (GnRH antagonist) on spermatogenesis, gene expression and tissue hormone levels, Monash University (PhD) candidate
2. Lindsay Bunk, MPH, 2004-2005, Acceptability of testosterone gel for male hormonal contraception, Public Health.
3. Women's Reproduction Health Research (K-12) Mentor 2005-current
4. Stephanie Page, MD, PhD. Oral Nanomilled testosterone in men and intratesticular testosterone in men undergoing male hormonal contraception (UW)

Selected Presentations:

1. Amory JK. Medical Abortion. Medicine Service Conference, UCSF, 5/96
2. Amory JK. The development of the oral contraceptive for women and prospects for a pill for men. Medicine Service Conference, UCSF, 5/97
3. Amory JK. Pre-operative Evaluation. Internal Medicine Ambulatory care conference. University of Washington. 9/4/97.
4. Amory JK. Pre-operative cardiac evaluation. Chief of Medicine Rounds, VA-Puget Sound, University of Washington, 2/20/98
5. Amory JK. Pre-operative evaluation of abnormal coagulation tests. Chief of Medicine Rounds, VA Puget Sound, University of Washington, 4/28/98
6. Amory JK. Pre-operative Evaluation. General Medicine Conference, University of Washington, 9/98.
7. Amory JK. Update on hormonal male contraceptives. Reproductive Endocrinology seminar, University of Washington, Department of OB/GYN-10/23/98
8. Amory JK. An analysis of the HERS studies. Women's Health Rounds 10/19/98
9. Amory JK. Unintentional Weight Loss. Chief of Medicine Rounds, VA-Puget Sound Health Care System, University of Washington, 9/8/98
10. Amory JK. Management of patients with angina in need of non-cardiac surgery. Chief of Medicine Rounds. VA-Puget Sound Health Care System, University of Washington. 1/12/99.
11. Amory JK. Hematuria. Chief of Medicine Rounds. VA-Puget Sound Health Care System, University of Washington. 9/99
12. Amory JK. Pre-operative assessment of the geriatric patient. Department of Geriatrics grand rounds. Harborview Medical Center and University of Washington. 1/00
13. Amory JK. Pre-operative medical assessment. Noon conference. Harborview Medical Center, 8/14/00
14. Amory JK. Oral Contraceptives. Primary Care Conference, UWMC 9/17/00.
15. Amory JK. Hepatopulmonary Syndrome, UWMC, 9/13/01
16. Amory JK. Pre-operative evaluation. ACP Annual Meeting, Seattle, WA, 10/20/01
17. Amory JK. Male Contraception. Urology Grand Rounds 2/14/02
18. Amory JK. Newer contraceptives for women. Primary Care Conference 3/7/02

19. Amory JK and Hirsh I. Outpatient Management of Diabetes. Chairman's Rounds 3/20/02
20. Amory JK. Male Contraception and Infertility. Ob/Gyn Grand Rounds 5/1/02
21. Amory JK. Clostridium Difficile Colitis. Chairman's Rounds 6/11/02
22. Amory JK. Update in Contraception. Primary Care Conference 8/29/02
23. Amory JK. Peripheral Vascular Disease. Resident's Teaching Conference 9/19/02
24. Amory JK. Thyroid Function Tests: A Practicum. Primary Care Conference 9/19/02
25. Amory JK. TPMT deficiency and pancytopenia. Chairman's Rounds 10/29/02
26. *Amory JK. George Washington's Infertility: Why the father of our country was never a father. **Medicine Grand Rounds** 10/31/02
27. Amory JK. Klinefelter' Syndrome. Pediatric Endocrine Rounds. Children's Hospital, Seattle, 11/21/02.
28. Amory JK. University of Washington CPC: 23 yo male with diarrhea and Orthostasis (Autoimmune Polyglandular Syndrome). 11/20/02
29. Amory JK. Harborview Medicine CPC: 36 yo male with itching and lymphadenopathy (sarcoidosis). 11/27/02.
30. Amory JK. George Washington's Infertility. King County Medical Society. 3/12/03.
31. Amory JK. Vitamin Deficiencies in Outpatient Medicine, Chairman's Rounds, 7/17/03
32. Amory JK. Female reproductive disorders. VA endocrine conference 8/23/03
33. Amory JK. Thyroid function tests: A practicum. UW resident conference 8/27/03
34. Amory JK. Male Contraception: 2003. Association of Reproductive Professionals National Conference, La Jolla, CA (Plenary session) 9/10/03
35. Amory JK. Peripheral Vascular Disease. Resident Teaching Conference-Providence Hospital. 9/22/03 and University of Washington 10/13/03
36. Amory JK. Androgens in the Aging Male. Geriatrics Grand Rounds, Harborview Hospital, 11/7/03
37. Amory JK. Medical Jeopardy, UW resident conference, 1/9/04.
38. Amory JK. Female Reproductive Disorders. UW Primary Care Conference 4/15/04
39. Amory JK, Linden H. Breast cancer: screening, diagnosis and treatment. UW Chairman's Rounds 6/15/04.
40. Amory JK. Thyroid Function Tests, UW resident conference, 7/7/04
41. Amory JK. Vascular Disease, UW Primary Care Conference, 8/26/04
42. Amory JK. Male Contraception: Update 2004, Seattle Gynecological Society Fall Assembly, 9/10/04
43. Amory JK. "Looking up the Kilt" Adventures in Andrology Research" Helen and Phillip Fialkow Award Presentation. **Medicine Grand Rounds**, 10/28/04.
44. Amory JK. Male contraception and infertility. MEDEX grand rounds 1/3/05
45. Amory JK. Female gonadal disorders. Med 556 "Endocrinology" 1/5/05

46. Amory JK. Vitamin Deficiencies. Resident Lunch Conference, 3/2/05
47. Amory JK. Male and Female gonadal disorders. Resident teaching conf, 3/3/05
48. Amory JK. Update in Andrology. Current Concepts in Drug Therapy 3/24/05
49. Amory JK. Absorption of oral testosterone in oil is augmented by 5alpha reductase inhibition in man. American Society of Andrology Annual Meeting, Platform Presentation 4/3/05
50. Amory JK. New Developments in Male Contraception. Symposium speaker (S-39C). Endocrine Society Annual Meeting 6/3/05.
51. Amory JK. Hot Flashes in an Elderly Male. Chairman's Rounds. UW Medical Center 7/19/05.
52. Amory JK. Thyroid Disease. ACP review course 7/21/05. Seattle, WA
53. Amory JK. Peripheral Vascular Disease. Resident's noon conference, 9/1/05, Seattle, WA
54. Amory JK. Serious Adverse Medication Events. Resident's noon conference, 9/15/05, Seattle, WA
55. Amory JK. A pill for him: progress towards oral androgen therapy and oral hormonal contraceptives for men. "New Frontiers in Clinical Research: From Diagnosis to Therapy, 1st annual General Clinical Research Center Symposium, Seattle, WA 9/23/05
56. Amory JK. Postpartum Endocrinology. Endocrine Days, Semiahoo, WA 9/24/05
57. Amory JK. Thyrotoxicosis Factitia. Chairman's Rounds, UW Medical Center. 10/4/05
58. Amory JK. Update on GnRH antagonists for male contraception. 9th Summit Meeting on Hormonal Male Contraception, Geneva, Switzerland 9/11/05
59. Amory JK. Male infertility and contraception. Endocrine Teaching Conference, Harborview Medical Center, 11/22/05
60. Amory JK. Male infertility. ENDO days 1/29/06
61. Amory JK. Male infertility and contraception. Obstetrics and Gynecology Grand Rounds, University of Washington. 3/29/06
62. Amory JK. Serious adverse drug effects. UW Capstone course 5/17/06
63. Amory JK. Male Contraception. "Meet the Professor" ENDO 88th Annual meeting, Boston, MA, June 24th, 2006
64. Amory JK. Serious Back Pain. Chairman's Rounds, UWMC 7/11/06
65. Amory JK. Thyroid Disease. ACP review, Bellevue, WA 7/27/06
66. Amory JK. Peripheral vascular disease. Noon conference. Providence 8/2/06
67. Amory JK. Thyroid function tests. Noon Conference Providence 9/6/06
68. Amory JK. Peripheral vascular disease. Primary care conference 9/7/06, UWMC
69. Amory JK. Serious adverse drug effects. Noon conference. UWMC 2/14/07
70. Amory JK. Male reproductive disorders. UWMC 3/1/07,

71. Amory JK. Update in Men's Health. Current Concepts in Drug Therapy, UW (CME course) 5/17/07
72. Amory JK. Markers for responders and non-responders, e.g. INSL3. 11th Summit Meeting on Male Contraception, 9/26/07
73. Amory JK. Oral testosterone enanthate plus dutasteride: implication for male contraceptive development. The Future of Male Contraception Meeting, 9/28/07
74. Amory JK. Update in Men's Health. ACP-Washington Chapter Scientific meeting 11/3/07
75. Amory JK. Acyline pharmacokinetics and Pharmacodynamic in normal young men. Bioqual Inc. Rockville, MD 12/4/07
76. Amory JK. Peripheral vascular disease. Primary care conference 12/20/07, UWMC
77. Amory JK. Androgens in Woman? Women's Healthcare Update, University of Washington. 3/6/08