These informational questions are designed to assist you in your decision of whether or not to inject lupron into your body. These questions should be posed to the physician who is prescribing lupron to you, and they can also serve to inform future physicians and lawyers. All emphasis has been added fyi, except where noted.

1. Why can’t anyone receive substantive answers to the following questions?

2. What is causing so many lupron victims to be so sick?

3. How come the first Food and Drug Administration (FDA) approval of lupron (New Drug Application [NDA] 19-010 in 1985) was approved out of the “Office of Biologics Research and Review” and not out of the “Office of Drugs”?

4. Why does the FDA’s Summary Basis of Approval (SBA) for the first approved indication of lupron (obtained under the Freedom of Information Act), contain redacted (whited out, censored) words, sentences, paragraphs (i.e. “Toxicology”), and is missing page after page after page (i.e., 19 consecutive pages in one instance), along with blank pages that contain the words “INFORMATION DELETED FROM ORIGINAL REVIEWS [emphasis original]”?

5. Why can’t I read the censored words, sentences, paragraphs, and pages in lupron’s SBAs’, and what do the censored words, sentences, paragraphs, and pages say?

6. Why does the National Institute’s of Health (NIH) Clinical Center Nursing Department, in cooperation with the Occupational Safety and Health Administration (OSHA), recommend the use of two pairs of chemotherapy gloves (among other protective gear) when healthcare workers handle lupron (leuprolide)(www.cc.nih.gov/nursing/shhdpro.html)?

7. How come lupron is classified as “a hazardous drug” according to the NIH and OSHA?

8. Why does TAP, in its lupron brochures, state “None of the components is hazardous. No special handling or disposal procedures are needed.”?

10. What is the “OTHER” in lupron?

11. Why is lupron not classified within the category "10:00.10 - Antineoplastic/HORMONE [emphasis original]”?

12. Why did an FDA Medical Officer involved with lupron’s initial approval for prostate cancer (1985) state in a 1984 book: “the long-term safety of LHRH [GnRH] analogues [such as lupron] have not yet been fully investigated, especially when we are dealing with structures drifting farther and farther from the original model” [Gueriguian, JL; Schaffenburg, CA; Chiu, Y; Berliner, V. Trends in Drug Development with Special Reference to the Testing of LHRH [GnRH] Analogues. In LHRH and its Analogues, Elsevier Science Publishers B.V. 1984, p. 507]?


14. Why is it that lupron is listed as one of the drugs that has been “implicated in a syndrome that resembles systemic lupus erythematus” [Lupus (1994);3:455]?

15. Is lupron “metabolized by enzymes in hypothalamus and anterior pituitary” with a “half-life of 3 hours” as stated by published literature [Appleton & Lange’s 1999 Drug Guide, p.788], or is lupron metabolized by being “destroyed within the GI track” with a half-life up to “4.25 hours” as stated by published literature [Anesthesiology & Critical Care Drug Handbook, 1998-1999, p.491], or does lupron have a half-life of up to “6.8 hours” as stated by published literature [Pharmaceutical Research (1992); 9(2):247]?

16. If lupron is reported to “be out of your system before an embryo will implant”, why is it “[o]ne could reasonably conclude that suppression of endogenous gonadotropin release is continued for another 12 days or so after cessation of LA [leuprolide acetate/lupron] [Fertility and Sterility,57(3):601].”?

17. If lupron is reported to “be out of your system before an embryo will implant”, why did (former) TAP Medical Director James D. Miller state “subclinical but detectable levels of lupron have been found in 1/3 of subjects 11 weeks after [last] injection” [Current Concepts in Endometriosis(1990):337]?

18. Since lupron (leuprolide) is listed as “a hazardous drug” by the NIH, and is listed as a Pregnancy category X drug (meaning fetal risk outweighs benefit and should not be taken by any woman who is or who may become pregnant), and lupron is listed as a “reproductive toxicant” and a “developmental toxicant” (www.scorecard.org) … why is lupron/leuprolide given to women attempting to conceive a child (or to women serving as a surrogate or egg donor)?

19. What caused the death of 1 of 25 hens given lupron in a 1994 study of chickens, and why
had all the chicken egg shells become “thinned” at the end of the 30 day experiment [‘Molting single comb white leghorns with the use of the Lupron depot formulation of leuprolide acetate.’ Poultry Science(1994);73:1226]?

20. What would explain the reason why there were no controlled, double-blind, placebo safety studies undertaken or required in order to approve lupron for the indication of endometriosis?

21. What would explain the reason why there were no formal dosing studies undertaken or required in order to approve lupron for the indication of endometriosis?

22. What is the significance of the following statement?: “The severity of the lesions were greater in testes of rats sacrificed 7 days after cessation of [lupron] indicating that the effects CONTINUED AFTER drug withdrawal” [‘Review and Evaluation of Pharmacology and Toxicology Data’, NDA 19-010, 3/1/84]

23. What explains the fact that the lupron “flare may be observed as late as six months after initiation of leuprolide [lupron] therapy” in men with prostate cancer, and that this flare “must not be confused with progression of metastases” [Clinical Nuclear Medicine (July 1990):485]?

24. In a 1986 forum sponsored by TAP, the question was asked whether this flare effect might be “hazardous” to the prostate cancer patient – and the reply was “While we are not certain what to make of flare … one has to look at hundreds of patients before determining what this blip on the screen really means.” [Urology Supplement (January 1986);27(1):3,20] – what has been determined in the interim regarding the lupron flare?

25. In the 1991 study of lupron’s metabolites whereby “the urinary M-I-like immunoreactivity levels” of Lupron Depot 3.75 were as follows: 1 day after lupron (“4.12 ng/ml”), 5 days after lupron (“0.93 ng/ml”), 7 days after lupron (“1.60 ng/ml”), 15 days after lupron (“0.85 ng/ml”), 25 days after lupron (“1.79 ng/ml”), 29 days after lupron (“1.74 ng/ml”) … what does it mean when metabolites increase over time instead of decrease? [Journal of Chromatography, 566 (1991):57-66]

26. Why is it that in the initial data for lupron’s first approval reviewed by FDA, there were identified only isolated cases of impotence during the prostate cancer clinical trials (NDA 19-010) – yet following approval of lupron, there became ample published literature documenting near universal impotence amongst subjects [do PubMed search of CAS # 53714-56-0 and impotence]?

27. Why would the manufacturer of lupron (TAP) advise in its lupron depot brochures (i.e. 4/94, 2/95) that “[a] nonhormonal or barrier method of contraception should be continued until 2 months after therapy. Pregnancy should not be attempted before this time” --- yet, in fact, women are being given lupron depot (long-acting) in In Vitro Fertilization (IVF) cycles [i.e., Journal of Assisted Reproduction and Genetics (1995),12 (1):15-19; Ibid (1995);12(3):Supplement, Abstract PP-133:156S; Fertility and Sterility (1993), Program
28. Why does the pharmaceutical manufacturer of lupron state in its lupron depot brochures that “GnRH ... acts on the pituitary to stimulate two other hormones, LH and FSH. ... When LUPRON DEPOT is administered monthly, production of these hormones is reduced to the very low levels found after menopause” – especially since any medical textbook describes menopause as FSH and LH being “produced thereafter in large and continuous quantities”?

29. What were the results of the clinical trials described as being “underway to determine the effectiveness of Lupron in infertility / in vitro fertilization (IVF) programs [1988 - 1992: Abbott ‘Annual Report’]”?

30. WHY were the clinical trials for lupron’s use in infertility and in vitro fertilization “discontinued” [Abbott, personal communication, 1995]?

31. WHY has lupron never been approved by the FDA for the indication of in/fertility treatment, or in vitro fertilization, or for egg donors or surrogates?

32. WHY has lupron/leuprolide been commonly used in in/fertility treatment despite never having gained FDA approval?

33. Were the clinical trials that were conducted for lupron’s use in infertility / IVF programs discontinued for “Efficacy reasons”? “Safety reasons”? “Both reasons”? “OTHER reasons”?

34. What is the significance (given the widespread promotion of lupron’s use in infertility/IVF) of the Public Citizen’s Health Research Group’s Comments on “Dissemination of Information on Unapproved/New Uses for Marketed Drugs, Biologics, and Devices [Docket No. 98N-0222, July 23, 1998], whereby it is stated: “Unquestionably, a patient prescribed a drug for a use that has not been shown to be safe and effective is receiving that drug for an experimental purpose” [Section 3: Full Public Access to Safety and Effectiveness Information]?


37. How come “The most frequently observed malformation in rabbits [given lupron] were vertebral anomalies and hydrocephalus” [J.D. Miller, personal communication, TAP, 1992;
Drugs in Pregnancy and Lactation, 4th Ed. 1994; p.481/1


39. Since the first published long-term study of babies born after accidental exposure to GnRHa’s revealed that 4 out of 6 babies have severe neurodevelopmental abnormalities, and the conclusion of this study was that “[t]his observation … justifies the need for long-term follow-up of more children previously exposed to GnRHa” [Human Reproduction (October 1999);14(10):2656. See also Human Reproduction, 15(6):1412] – has there been any further long-term follow-up of children exposed to GnRHa?


41. What is the significance of the following statement?: “Inclusion of patients with a poor response to GnRH-a therapy has not always occurred in outcome analysis in the published literature.” [Fertility and Sterility (1994);61(2):405]

42. Why is it that human “oocytes matured in vitro by GnRH-a [lupron, buserelin] are not necessarily cytoplasmically mature” [Fertility and Sterility (1992);57(5):1091]?

43. Given that lupron is widely used in infertility treatment, why did the United States Pharmacopeia Convention state regarding leuprolide acetate/daily lupron: “your doctor should be told if you intend to have children. Leuprolide causes sterility which may be permanent. Be sure that you have discussed this with your doctor before receiving this medicine.” [USP DI (1988); 8th Ed., Vol III: ‘Advice for the Patient’;663]?

44. What is the mechanism of action of lupron whereby it caused human uterus and fallopian tube “strips [to] show[] tetanic contractions that lasted between 2 and 40 (+) minutes.” ['Leuprolide acetate stimulates smooth muscle of the human reproductive tract’. Fertility and Sterility (1991);56(5):993]?

45. If literature indicates “the possible role of endogenous opioids in the [GnRH agonist analog] peptide effects” [Physiology & Behavior (1992);51(3):601], and “opiate mediated processes are involved in the initiation and control over seizures to the brain” [Neuropeptides (1990);17:81], and “opioid peptides caused seizures that are brief, have a short latency … and are not associated with behavioral convulsions” ['Epilepsy and Sudden Death’ (Lathers CM, Schraeder PS). Marcel Dekker; New York, p.437] … what causes the twitches and spasms on and following lupron?

46. What causes the “vaguely described visual and auditory abnormalities and seizures reported with another GnRHa (histerelin) ['Neurotoxic Side Effects of Prescription Drugs’, J.C.M. Brust, 1996, p.194], and what causes the hallucinations and convulsions and grand mal convulsions
47. Since it is known that GnRH’a’s (i.e. lupron/leuprolide) cause a “hypophysectomy” [Birth(1988):15(3):134], which is by definition the “destruction or removal of the pituitary”, and it is known that “sustained treatment with GnRH agonists [i.e., lupron/leuprolide] most likely abolishes pituitary function” [Gynecologic Obstetric Investigation(1988);25:130] – what long-term effects will result from lupron destroying/abolishing my pituitary function; and why do you want to prescribe lupron to me?

48. Are you one of the umpteen doctors TAP has given money to? Or alternatively: Have you ever received any monies from TAP in the form of speakers fees, or monies from TAP for grants, or for clinical trials, or for honoraria, or consultant fees, or monies or gifts (i.e. free lupron samples) from TAP? [for further information, google “TAP” and “bribes”; and/or see the U.S. government’s lawsuit against TAP for bribing doctors to prescribe lupron, in which TAP (labeled as a “criminal enterprise”) paid the highest fine ever recorded at that time in history - $875 million … United States of America, ex rel. Joseph Gerstein and Tufts Associated Health Maintenance Organization, Inc., Plaintiffs v. TAP Holdings, Inc. and TAP Pharmaceuticals, Inc., Defendants. Civil Action No. 98 CV10547GAO. U.S. District Court for the Eastern District of MA. March 26, 1998; i.e. www.usdoj.gov/opa/pr/2001/October/513.civ.htm]

49. In a 1993 study, ‘Adverse effects of leuprolide acetate depot treatment’, it is stated “The cause of some adverse effects reported has no clear endocrine mechanism. … The mechanism of joint pain remains unclear and warrants further investigation.” [Fertility and Sterility,59(2):448] – what investigation has taken place, and what are the mechanism(s) of action?

50. What is the significance of the statement “whether leuprolide alters gastrointestinal motility as part of its action is unknown.”[American Journal of Physiology (1992);262(1):G185]?

51. What is responsible for the following adverse reactions reported to the FDA (Spontaneous Reporting System) for women receiving lupron: “gastrointestinal disorder”, “esophagitis (inflammation of the esophagus/throat)”, “ulcerative colitis”, “acute abdominal syndrome”, “abdominal enlargement”, “anorexia”, “dyspepsia”, “diarrhea”, “bloody diarrhea”, “dysphagia (difficulty swallowing)”, “abdominal pain”, “nausea”, and “vomiting”?

52. What does the following statement mean?: “Whatever risks [lupron] may pose to women who take it, the drug has a noticeable effect on the embryos the women conceive, according to clinical embryologists. The Lupron embryos grow faster, develop more rapidly. They are more fragile when frozen and less likely to survive thawing. Nobody knows why or what it means for the long-term health of the woman or any resulting child.” [Holtz R. Atlanta Journal and Constitution, 10/27/91: ‘A Risky Fertility Revolution: Drugs may bear long-term danger for moms, babies’; p.1]

53. If lupron is classified as “chemotherapy” and an “antineoplastic”, and women who’ve taken lupron have suffered hair loss (alopecia) like women undergoing chemotherapy – however, they fail to grow their hair back unlike the women undergoing chemotherapy who do grow their hair
What would cause permanent alopecia?

Since healthcare workers (according to NIH/OSHA guidelines) handle hazardous drugs with protective gear (and leuprolide is on the “Hazardous Drug” list), why are they advised to “avoid contact” with leuprolide for “a specific time period (e.g., 3 months)” if the worker is “trying to conceive or father a child” [AHFS (American Hospital Formulary Service Drug Information 1999: American Society of Hospital Pharmacists (ASHP) Technical Assistance Bulletin on Handling Cytotoxic and Hazardous Drugs;1029-1038)]?

If health care workers, whose ‘only’ exposure to lupron would be through the recommended protective gear (two pairs of chemotherapy gloves, mask/respirator, gown, etc.), are advised to “avoid contact” for 3 months prior to conception attempts – how come women are told to inject lupron directly into their body on a daily basis in an attempt to conceive?

Why have “these agents” (GnRH agonists; i.e. leuprolide) been used as a “probe” (a device used to get information) [e.g. American Journal of Obstetric Gynecology (1990);163:1114]?

What is the significance of the following statement?: “The long-term disruption of the pituitary-ovarian relationship which followed a single injection of LHRH-AS [GnRH antiserum] illustrates the fact that interruption of the pituitary stimulation and of the hypothalamic triggering mechanism for a relatively short period of time can induce what may be irreversible effects” [Endocrinology (1976);98:1539]


When the FDA found that TAP-funded lupron brochures “focus upon administration of Lupron for these unapproved uses to an excessive degree” and “actively promote [lupron] administration in a range of indications for which [lupron] is not approved and apparently has not been adequately demonstrated to be safe and effective” [FDC Reports, 4/2/90, p.11] … what effect might this “excessive degree” of promotion for unapproved uses have had upon the prescribing practices of the medical community?

Why did the manufacturer of lupron (TAP) donate $5000 to RESOLVE (an alleged “grassroots” organization for the infertile) in 1989 [RESOLVE Annual Report (Form PC), 1989], when in 1989 lupron only had FDA approval for prostate cancer and did not have any female indication approved at all?

Why did TAP have a booth at the 1989 American Fertility Society [now American Society of Reproductive Medicine] meeting, as acknowledged by TAP in a May 4, 1990 response to the FDA’s Notice of Adverse Findings?

This is a long one - so bear with me: Since LH and FSH responses to GnRH “were studied in a group of female subjects from the 1st to 8th decades” in 1985 [Journal of Steroidal
Biochemistry (1985); 23(5B):823], along with “a decade of investigation [of] GnRH and its agonistic and antagonistic analogs ... [in which] “intense investigation in all species, including man” took place [Fertility and Sterility (1983); 39(3):257] -- then why is it stated in 1988 that “there have been no studies regarding the effect of leuprolide in humans on the health of the oocyte” and “final evidence of the absence of a toxic effect on human embryo development awaits the analysis of a large number of pregnancies and ovulation induction cycles” [Fertility and Sterility (1988); 50(4):612] --- YET by 1990 alone, 97% of reported IVF stimulation cycles were using GnRH-a’s [1990 MRI/SART IVF-ET Registry Results. Fertility and Sterility (1992); 57(1):15], of which lupron is the most widely prescribed; so, therefore, ... why is it stated in 1993 that “there is a paucity of published data regarding [GnRH-a] outcomes in human gestation” and “[c]learly, much larger numbers of GnRH-a exposed pregnancies are required before reliable conclusions regarding the drug’s effects on pregnancy can be made” [Fertility and Sterility (1993); 60(6):980]? 

63. Why was it that, in 1993, “[d]espite the popularity of using GnRHa in ovulation induction, there [was] only one indirect report on the meiotic maturity of oocytes retrieved from women using this medication ... this is the first report of asynchrony between cumulus-coronal morphology and nuclear maturity at follicular aspiration in GnRH-a and norethindrone-programmed stimulations” [Fertility and Sterility (1993); 59(2):375]? 

64. Since it was stated in the 1998 ‘Current Protocols in Pharmacology’ [vol. 1, p.10.3.22] that “GnRH analogs had originally been developed for the treatment of prostate cancer, and were accordingly subjected to a less rigorous toxicology program than the standard. The current focus with these agents on less serious conditions such as fertility disorders, and the modifications in the structure of the native compound have made it necessary to examine them in a more traditional way [acute toxicity, subchronic and chronic toxicology, genetic toxicology, reproductive toxicology, carcinogenicity, special studies]” --- therefore, what ‘traditional’ examination(s) of GnRH analogs is/are underway? 


67. What is the risk, in terms of effect(s) on my bones, of the following information from a
study [Bone (1995); 16(2):261-7] which examined the effects of GnRH agonists on the bones of women with endometriosis?: “results suggest that bone loss induced by GnRH analogs may be associated with adverse effects on cancellous [trabecular] microstructure which are unlikely to be reversed following cessation of therapy.”

68. How can the short and long term safety of my bones be determined if the mechanism of joint pain from lupron “remains unclear”?

69. What is the significance of the 4/13/90 FDA Medical Officer’s statement (in ‘Review of Revised Labeling’ for the endo NDA, 4/13/90): “The most common adverse event was hot flashes, the majority of which occurred within 3 months after stop of study”?

70. If GnRH analogs/lupron are supposed to “stimulate” your system during the first few weeks before it then “suppresses” - why do women “escape” this suppression months into treatment, and why did a 1987 study (with GnRH analogs buserelin and nafarelin) of over 200 women in the U.S. and abroad show that “over 100 women showed that signs of endometrial overstimulation occurred [] during the first few months” [Lancet, (May 23, 1987):1179]?

71. If hot flashes are not related to release of LH since women receiving GnRH analogs experience hot flashes (hot flushes, vasodilation, flush) in the absence of LH [Journal of Clinical Endocrinology and Metabolism (1983);56(3):445] - how do you explain the hot flashes in women on lupron who have “escaped” lupron [above-menopausal levels of estrogen], and how do you explain the hot flashes in women on lupron and ‘add back’ estrogen, and how do you explain the hot flashes in women who have stopped lupron?

72. What does the following mean regarding the “flush” seen after GnRH agonist hypophysectomy: “dysregulation of the GnRH releasing clock center in the nucleus arcuatus in the mediobasal hypothalamus is associated with altered central alpha-receptor activity which results in lowering of the set point of the central thermostat and the circulatory changes.” [European Journal of Obstetrical Gynecology and Reproductive Biology (1994);57(3):137]?

73. If lesions or stimulation of the preoptic anterior hypothalamus (which connects to and exerts control over the pituitary) causes vasodilation of all skin vessels of the body and causes sweating [”Textbook of Medical Physiology’, Guyton (1981):353/703], and if lesions of the preoptic region produce insomnia [Neurobiology, 2nd Ed (1988), Shepherd:525]”, and if stimulation in the amygdala (which has “abundant direct connection with the hypothalamus”) causes ovulation and uterine activity [”Textbook of Medical Physiology’, Guyton (1981):705] ... what causes lesions?

74. What significance does the following FDA Medical Officer’s statement have?: “It is difficult to understand why symptoms seem to decrease within two - four weeks of starting [lupron], prior to even well-established hypogonadism and amenorrhea.” [endo NDA 20011]

75. Does the following statement answer the latter question?: A GnRHa was “studied in several pharmacological tests in mice, and the peptide [GnRHa] was found to exert sedative-anxiolytic,
neuroleptic or dopamine antagonist and analgesic activities” and “the central inhibitory actions of [the analog] were demonstrated to be partially reversible by the opiate antagonist naloxone, indicating the possible role of endogenous opioids in the peptide effects.” [Physiology and Behavior(1992); 51(3):601]

76. How come “GnRH analogs may exert direct effects upon the Autonomic Nervous System” [Fertility and Sterility(1991);56(2):357.]?

77. Do you find the following statement alarming?: “GnRH analogs are not like any other medication currently available for treatment of disease. As we continue to learn more about these analogs’ mechanisms of action, it is increasingly apparent that they do not just affect the gonadal [sex] hormones, but are powerful modulators of autonomic neural function.” [June 1995. ‘Placebo Controlled Study Randomizing Leuprolide Acetate’. Digestive Diseases and Sciences, 40(6):1405].

78. What does the following statement mean?: During IVF using GnRHa’s, various neurological symptoms (severe headaches/migraines, numbness, tingling, paresthesias, weakness, ataxia) were observed in the young female patients, and it was reported that “the mechanisms of action responsible for this complication is not clear. … [however] … a direct effect of potent GnRH-analog on the central nervous system resulting in neurological effects independent of the hypothalamus-pituitary-gonadal axis is possible.” ['Adverse neurological symptoms after GnRHa therapy for IVF cycles’. Fertility and Sterility(1990);53(4):738]

79. What caused “a significant proportion of women, more than half in some cases, [to] show[] significantly worse performance on one or more memory tests while on leuprolide acetate [lupron]. When memory tests performances were combined, a substantial majority of the patients [72%] showed difficulty with memory while on leuprolide acetate” [Journal of Assisted Reproduction and Genetics (1993), 10(1):53-7]?

80. Since it is known that lupron “shuts down blood flow to the frontal lobes of the brain” [Gannett News Service, 11/17/94, ‘Research Links Sex Hormones to Human Brain and Learning’], what will happen to the frontal lobe of my brain during and after lupron?

81. Why is lupron “effective in decreasing blood flow to the uterus” [American Journal of Obstetric Gynecology(1994);170(6):1623]?

82. What do you think of the following information?: “Transient cerebral ischemia (TCI) is one possibility that may explain the symptoms of numbness, headache, paresthesia and paresis [during GnRHa use in IVF]. … This could explain the various neurological symptoms occurring by means of vasospasm of intracerebral blood vessels. Furthermore, a direct effect of potent GnRH-analog on the central nervous system resulting in neurological effects independent of the hypothalamic-pituitary-gonadal axis is possible … it is quite possible that mild cases have escaped notice; thus, the occurrence of this type of complication may be far more common than we realize.” ['Adverse neurological symptoms after gonadotropin-releasing hormone analog therapy for in vitro fertilization cycles’. Fertility and Sterility(1990);53(4):738]
83. Why is Clonidine used to treat the hot flashes from lupron?

84. What risk does the following statement (from FDA’s “Evaluation” in the 1985 SBA for lupron’s original NDA for palliative treatment of prostate cancer) have for my pituitary gland?: in lab rats receiving lupron “[t]here was approximately a two-fold increase in pituitary adenomas in both males and females at the low dose with no no-effect dose demonstrated. ... the possibility exists that Leuprolide [lupron] at the same time may be acting as a stimulator of other cell functions which could result in pituitary adenomas. There is no obvious reason to suggest that the same process could not occur in humans.”?

85. Why is it recommended that patients undergoing anesthesia who are on lupron have “a peripheral neurologic examination and evaluation of paresthesias [before] regional anesthetic techniques” [Manuel of Drug Interactions for Anesthesiology, 2nd Ed. (Mueller RA; Lundberg DBA), 1992]?

86. Why is it that, regarding the possibility of GnRHa’s causing human pituitary adenomas, “We [FDA] cannot exclude that [GnRHa] may cause not only adenomas in rat pituitary glands as reported previously, but also a (nodular) hyperplasia of the pituitary gland in man” [Neuropathology and Applied Neuropathology (1991); 17:75-81]?

87. How come, if lupron is supposed to “suppress” the pituitary, all of the rats treated at 0.6 - 4.0 mg/kg/day had “drug-related adenomatous, hypertrophic [morbid enlargement] and hyperplastic [abnormal multiplication or increase in the number of normal cells] changes with cellular atypia [condition of being irregular or not conformed to type] seen in the anterior lobe of the pituitary gland of [all] males and females” [Study No. TA78-537, Reference #14, NDA 19-010]?

88. Since the vast majority of pituitary adenomas (which occur in 10-20% of the population) are microscopic (and may not be evident radiographically), and “typically remain clinically silent for years (except for subtle signs, i.e., headache, diminished libido, impotence, neurological symptoms, visual field defects, endocrine hypo/hyperfunction) and “unless the signs of altered pituitary function are grossly apparent” – the diagnosis of pituitary disease can be overlooked ['‘Medicine’, 2nd Ed.; Fishman et al (1985);198] … then how is it known whether some lupron patients or (like the rats in TA78-537, Reference #14, NDA 19-010) all lupron patients have pituitary adenomas?

89. What is the explanation for the statement in Dr. Varney’s study of “neuropsychological dysfunction associated with [use of lupron in IVF]” where it was concluded that “memory and coordination [ ] were commonly observed patient complaints” and that “There was no correlation between estradiol levels and test results on any test [‘Neuropsychologic dysfunction in women following leuprolide acetate induction of hypoestrogenism’. Journal of Assisted Reproduction and Genetics(1993); 10(1)53-57]?
90. If memory loss following lupron administration was a “commonly observed patient complaints” in 1993, how is it that in 1995 the FDA ranked “amnesia” as only the 23rd most common adverse reaction to lupron [FDA Spontaneous Reporting System/Division of Epidemiology and Surveillance]?

91. Why did “a substantial majority of the patients show [] difficulty with memory while on leuprolide acetate [lupron]”, and why were problems with “memory and coordination … commonly observed patient complaints” [Journal of Assisted Reproduction and Genetics(1993); 10(1):53-57]?

92. What would explain the discrepancy between Dr. Andrew Friedman’s fibroid study using lupron in which only 6% of women experienced memory loss [‘Adverse effects of leuprolide acetate depot treatment’. Fertility and Sterility(1993);59(2):448.], while another study by Dr. Varney et al. using lupron in IVF found 72% of women reporting memory decline [‘Neuropsychologic Dysfunction in Women Following Leuprolide Acetate Induction of Hypoestrogenism’. Journal of Assisted Reproduction and Genetics(1993);10(1):53]?

93. Why did Dr. Andrew Friedman (who was a consultant for TAP and had received numerous TAP grants to study lupron) “falsify[] and fabricat[e] approximately 80% of data” in 4 lupron studies [Federal Register. May 1, 1996. Findings of Scientific Misconduct. Vol 61(85):19295-6]?

94. Given that Dr. Andrew Friedman fabricated data in 4 lupron studies, and given that Dr. Friedman was an opinion leader in the use of lupron with hundreds of publications (studies, articles, chapters, and books), and given that Dr. Friedman submitted data to the FDA for lupron’s endometriosis approval – have any other of the hundreds of publications of Dr. Friedman been reviewed for falsified and fabricated data?

95. Why did the use of lupron in infertility treatment go from “only used in certain diagnoses” (in 1990) to “widely prescribed” (in 1991)? [Brigham & Women IVF Clinic Brochures, 1990, 1991. Dr. Andrew Friedman, Director]

96. What is the mechanism(s) whereby GnRHa’s result in “DNA damage” [American Journal of Obstetrics and Gynecology(August 1997);177(2):417]?

97. If “the shrinkage in fibroid and uterine volumes that is associated with chronic administration of a GnRH-a [lupron] could be the result of cell death” [1990. Obstetrics and Gynecology,76(3): Part One, p.388] – what would cause the cell death?

98. If a study (supported by TAP) stated that lupron “is a very effective treatment for carefully selected patients with severe, perimenstrual migraine headaches [Fertility and Sterility (1997);67(2):390]” – why do women taking, and after taking, lupron experience “migraine-like” headaches [‘Neurotoxic Side Effects of Prescription Drugs’, J.C.M. Brust, 1996, p.193] that require treatment/medication?
99. What is the mechanism(s) of action responsible for the common, intense, headaches reported following lupron exposure?

100. What is the mechanism(s) of action responsible for memory loss following lupron exposure?

101. What is the mechanism(s) of action responsible for bone loss following lupron exposure?

102. Should I be concerned about the following information?: In the bioavailability study submitted to the FDA (and deemed by the FDA as “unacceptable”), it was acknowledged that “the great change” in one parameter “was probably a result of the drug kinetics being more complex than assumed, although no explanation is readily apparent” [NDA 19-010; ‘Single-Dose Pharmacokinetics of Leuprolide in Humans Following Intravenous and Subcutaneous Administration’. Journal of Pharmaceutical Sciences (Feb 1986);75(2):158]. And in a study co-authored by Abbott employees (who hold patents involving lupron), the solution behavior of lupron was analyzed (by a machine funded in part by Abbott) wherein it was determined that “[t]hese experiments suggest that nascent secondary structure exists in leuprolide acetate” [1994. ‘Solution behavior of leuprolide acetate, an LHRH agonist, as determined by circular dichronism spectroscopy’. International Journal of Pharmaceutics, 108:49-55].

103. When it is stated in the FDA’s SBA Medical Officer’s Review of Revised Labeling Submission for the endometriosis approval (5/29/90) “Since the upper limits of normal range for cholesterol and LDL appear to be extremely high in the labs used by the sponsor, we suggest you leave out all upper normal ranges. This information would only tend to confuse the practitioner, rather than offer any worthwhile information. We would still like to know why and how many labs had high upper limits. However, this need not delay the approvability.” - why was the approvability not delayed?

104. Since significant numbers of lupron victims report significantly and sustained elevated lipid levels (i.e., hypercholesteremia and hyperlipemia are reported adverse events to lupron), yet there has been a lack of establishment of causal relationship to lupron – what is being done or has been done to clear up this “confusion”?

105. Since “deferral of the bioavailability requirements [was] recommended under CFR 320.22(5) (e) because leuprolide [was] an important [older male] oncologic drug” [according to the FDA’s 12/11/84 ‘Deferral of in vivo Bioavailability/Bioequivalence Requirements” for NDA 19-010] - why were no formal dosing studies done prior to lupron’s approval for an indication involving young, healthy women with endometriosis?

106. Why were no formal dosing studies conducted before the FDA approved lupron for the treatment of endometriosis in human females, yet when lupron was administered to baboons “[t]he doses used were derived from preliminary human data [Fertility and Sterility (1993), 59(5):1124-8]”?

107. How come “significant effects on both primary [thymus and bone marrow] and secondary
[Peripheral blood, spleen, lymph nodes] lymphoid tissues ... unrelated to plasma estradiol levels” were observed in mice following lupon injection [Journal of Reproductive Immunology (1993);25:167], and how come in women with endometriosis it was found that “[lupon] alters circulating lymphocyte and granulocyte subpopulations” [Fertility and Sterility Supplement (1994);S148]?

109. If, in the report of results from a study of lupon for the unapproved indication of functional bowel disorder, the results were reported as positive, yet “there were no significant differences at the end of treatment between active drug [lupon] and placebo” and “[another] concern is that it appears that the baseline results, particularly for the original placebo control, are somewhat different from that reported in the placebo-controlled study” [Digestive Diseases and Sciences (1995); 40(6):1405] ... should I question other published “positive” results from lupon studies?

110. If the original patent describing lupon (Patent #3,914,412 - Oct. 25, 1975) states “the new peptide induces ovulation in warm-blooded animals ... produc[ing] almost certain ovulation ... beneficially employed in animal husbandry” -- why is lupon not approved by the FDA for “ovulation induction”?

111. Since leuprolide acetate (the daily lupon) injected by consumers contains the diluent “benzyl alcohol”, but the required animal toxicology studies for lupon’s initial FDA approval were done not by dissolving leuprolide acetate/lupon in benzyl alcohol, but in normal saline only ... so how could these toxicology studies not contain the exact injection (lupon [leuprolide acetate] plus benzyl alcohol) that humans get?

112. What significance would the following findings have on a human (testicle, ovary, whole body, or embryo)? In animal toxicology studies done for the initial FDA approval of lupon, rats received two (2) days of 1 mg/kg/day of lupon - and autopsy showed “various degrees of testicular degeneration” [Study No. 76-129, Reference #8, NDA 19-010]?

113. What is responsible for the results of an in vitro fertilization (IVF) embryo cryopreservation study which showed “a rise in the percentage of embryos with <50% intact blastomeres when using the leuprolide acetate [lupon] - hMG protocol” [Fertility and Sterility (1993);59(5):1065]?

114. If “[f]urther large scale studies seem indicated to further investigate the risks from inadvertent GnRH-a expo exposure in early pregnancy” because 43.5% of women in one study experienced adverse pregnancy outcome after lupon exposure in early pregnancy [Fertility and Sterility Program Supplement, Nov. 1996, Abstract P-034, p.A27] -- what studies are being conducted, and how many women/babies are involved?

116. How come in a study of women with fibroids using lupron, their LH and FSH levels revealed that “[n]either of these gonadotrophins showed any significant changes after 4 weeks of therapy” [Fertility and Sterility (1987);48(4):560-4]?

117. Given that the FDA stated that the clinical studies undertaken for lupron’s approval for the indication of endometriosis “had a great number of dropouts” and “the lack of adequate blinding may cause bias” -- what impact would “a great number of dropouts” and “lack of adequate blinding” and possible “bias” have upon the validity of the conclusions of the ‘study’?

118. Since the evaluation of the risk/benefit ratio for lupron’s use in palliative treatment of prostate cancer was classified as less stringent, what does the following FDA statement regarding its evaluation of lupron for prostate cancer have for (young and otherwise healthy) women with endometriosis, infertility, fibroids, PMS, etc.: “There are other [word or words censured] inconsistent effects of Leuprolide [lupron] in the various toxicology studies but potentially the most serious effect of Leuprolide, in my view, is its effect on spinal column bone marrow. This increased fat deposition and subsequent hypocellularity was explained as a physiological response to the drug.” [NDA 19-010]

119. If “bone marrow hypocellularity resulting from a pharmacological increase in fat content was observed in all [rats treated with lupron for 2 years (Study No. TA78-537, Reference #17, NDA 19-010)] and no “meaningful changes” were seen in the rats hematological [blood] findings --- how come in the FDA’s “twelve-month interim report” of Study TA78-537 (2/27/84) it is stated that “bone marrow hypocellularity was present in [all] treated animals” and “mean hematocrit, hemoglobin and RBC [red blood count] were higher in treated females than in controls”?

120. Are the findings of many decades ago relevant [in Clinical Hematology (1947); Wintrobe, 2nd Ed. p.39], whereby it was reported that there was “a tendency to high erythrocyte [red blood cell] counts observed in cases of pituitary basophilism” [a syndrome due to overgrowth of the basophil cells of the anterior lobe of the pituitary, which causes among others, amenorrhea (loss of period) in females]?

121. Would bone marrow (which produces blood cells) that has “hypocellularity” (a state of abnormal decrease in the number of cells present) cause an increase in the red blood cell count?


123. Have you read “GnRH Agonist Induces Suppression of Lymphocyte Subpopulations in Secondary Lymphoid tissues of Prepubertal Female Mice [American Journal of Reproductive Immunology (1993);30:15]?
124. In “GnRH Agonist Induces Suppression of Lymphocyte Subpopulations in Secondary Lymphoid tissues of Prepubertal Female Mice [American Journal of Reproductive Immunology (1993);30:15], it is stated: “Analysis of light-scattering characteristics of cells from both control (lypholized microcapsules alone) and Lupron (lypholized microcapsules with Leuprolide Acetate) treated animals revealed 3 readily identifiable populations. **The precise identity of these cells has not been established.**” Why wasn’t the precise identify of these cells established?

125. Why did a “single i.m. injection of agonist [lupron] significantly decrease[] both absolute and relative thymic weights and absolute thymocyte counts.” [‘Alterations in thymic and bone marrow lymphocyte subpopulations in GnRHa agonist treated prepubertal female mice’. American Journal of Reproductive Immunology (1993);25:167]?

126. [adrenal weights]

127. What causes the “aplastic anemia” reported to the FDA as adverse events following administration of lupron?


129. What causes the development of osteoporosis (severe bone loss) in lupron treated patients?

130. Since osteoporosis is historically known as the “silent disease” (that is, it is asymptomatic - without symptoms), what causes the bone pain in women who’ve taken lupron and developed osteoporosis?

131. If the mechanism of bone pain on lupron “remains unclear”, how can the short and long term safety of my bones be determined?

132. Why do GnRHa’s cause “severe disruption of the cancellous microstructure” of the bone … suggest[ing] that bone loss induced by GnRH analogs may be associated with adverse effects on cancellous microstructure which are unlikely to be reversed following cessation of therapy.”? [‘The Effects of GnRHa on Iliac Crest Cancellous Bone Structure in Women with Endometriosis. Bone(1995);16(2):261]

133. Why were the initial prostate clinical trials **limited** to “Stage D2” prostate cancer patients[NDA 19-010]? (“Stage D2” was designated as cancer that had metastasized [spread] to the bone)

134. How could the effects of lupron on bone be evaluated if these lupron-treated subjects had cancer in their bones?

135. If “QCT [quantitative computerized tomography/bone scan] always shows significant
**trabecular** bone loss of the vertebrae and hip with GnRH agonists” and “DPA [bone scans] may not detect significant early changes and an apparent lack of change may be observed” [1994. Acta Obstetricia et Gynecologica Scandinavica;73:159.]” – why did so many early lupron studies use DPA/DEXA scans when assessing bone loss?

136. From 1991 through 1995, lupron’s package insert for endometriosis states that after 6 months, the vertebral **trabecular** bone density as measured by QCT was decreased by an average of 13.5% -- but in 1996 the package insert was changed to read the vertebral bone density (but not trabecular) as measured by DEXA was decreased by an average of 3.9% (1991 – 1996 Physicians Desk Reference. Lupron: endometriosis). ... Why do you think this change was made?

137. Do you recommend your patients undergo a QCT bone scan before, during, and after lupron?

138. How come if the follow-up studies of bone loss in the endometriosis clinical trials (M86-031 and M86-039) showed that “3 [of 12 {25%}] patients continued to have significant loss of bone at the follow-up period [-13.4%; -10.8%; and -9.6%]”, the FDA could conclude on 2/15/90 that “**Lupron causes some irreversible decrease in bone mass**, but during one 6 month treatment period, this finding should not be significant”?

139. Why would the FDA conclude that -9.6%, -10.8%, and -13.4% bone loss in 25% of these follow-up endo patients “should not be significant”, if the FDA states in TAP submission 19-943 (for the indication of lupron for fibroids [submitted prior to the endo NDA]), the bone loss “by CT scan [...] was -8.7% … The **variability** between all these studies are troublesome.”?

140. What is the “unpublished data” mentioned in Journal of American Obstetric Gynecology [(1993);168(2):674], in which it is mentioned that lupron depot caused a “15.3%” bone loss?

141. If the osteoporosis that women experience following lupron administration is allegedly due to the “menopausal side effects” – why do women using lupron lose far more bone density than women from natural menopause?

142. Why doesn’t the hormonal profile of a menopausal woman (increased FSH/LH, decreased estrogen – **hypergonadotrophic hypogonadism**) match the hormonal profile of a woman on lupron (decreased FSH/LH, decreased {sometimes} estrogen – **hypogonadotropic hypogonadism**)?

143. How come in the lupron endometriosis clinical trials 63% of women had “instances of spotting [] on at least one occasion after suppression”, and 68% of women “noted irregular bleeding” [NDA 20-011]

144. What is meant in the (PDR-like) “MIMS Annual Phillippines” [1997, p.625-6] by the following statement concerning ‘Luprolex’ (leuprerelin acetate/lupron)?: “**Unexpected metrorrhagia** ("irregular bleeding") during treatment is abnormal; it needs [sic] to verify plasma estrogen level and, if lower than 50 pg/mL, to search the eventual organic associated lesions”.
145. Why do people who are both on lupron and those who have stopped lupron complain of “joint problems, joint pain, arthralgias, bone pain, bone loss, bone disorder, bone fracture, osteopenia, osteoporosis”?

146. Why does there not appear to be any values listed for the LH and FSH levels of the lupron endometriosis clinical study subjects within the FDA’s NDA?

147. What causes women to continue to experience hot flashes long after lupron is stopped?

148. Why were estradiol levels conducted monthly in the placebo-controlled study for lupron for endometriosis (M86-031), but in the study comparing lupron with danazol (M86-039) estradiol levels were only conducted on months 3 and 6, thereby “providing incomplete information” [NDA 20-011]?

149. Why were estradiol levels higher at months 2 and 4 during the M86-031 lupron for endometriosis clinical study?

150. Since estradiol levels were not checked on month 1, or month 3, or month 5, of the 6 month endometriosis clinical trial -- how is it known that these levels were not strikingly inconsistent and abnormal?

151. Why was it “most striking [that] throughout [the study of lupron in monkey endometriosis in 1983] there was [] instability of serum estradiol levels” and “it is apparent that the ovarian cycles of some [monkeys] were not fully suppressed in the first month [and] ... in the third month of treatment ... the hypothalamic-pituitary ovarian axis gradually was coming under the inhibitory influence of [lupron]”?

152. If the above monkey study evidenced “gradual” suppression from lupron - how does one explain the manufacturer’s toxicity data (NDA 19-010) in monkeys receiving lupron for 3 months where “atrophic changes in the uterus and vagina and cessation of follicular development in the ovaries was seen in all treated groups”?

153. How common is it in the drug approval process that, as in the case of rats treated with lupron, the “maximum-tolerated dosage [was] not established because pituitary hyperplasia” occurred “at all doses” [NDA 19-010]?

154. When it is stated that “when the analog [lupron] is discontinued, gonadotropin and sex steroid levels usually return to normal gradually, but occasionally the levels remain low [Drug Evaluations, Annual 1995:2189] --- can you qualify “usually” and “occasionally” relative to my risk of failure to return to pre-lupron hormonal levels?

155. While data from studies of lupron’s anti-tumor effect are “inconsistent”, what is meant by the determination in the following ovarian cancer cell study?: “the high viability of cells exposed to Lupron suggest that the drug was interfering with a cellular regulatory mechanism
rather than acting as a toxin … between day 14 and 21 the experimental cells divided faster than the control cells” [Journal of Clinical Endocrinology and Metabolism (1991);72(5):1036]

156. In the 1984 FDA documents detailing the toxicological review of these studies, it is written “Other tumors which were significantly increased by [rat] Lupron treatment included pancreatic islet-cell adenoma and testicular interstitial-cell adenoma. [end of discussion censured] [NDA 19-010]” – why was the end of the discussion censured, whited-out, redacted?

157. If lupron depot contains “injectable microspheres composes of lactic acid-glycolic acid copolymer and leuprolide acetate”, and polymers such as “lactic acid-glycolic acid enable release of molecules for over 100 days … [w]hen encapsulated proteins remain in the body for a long time, they may denature or aggregate as a result of exposure to moisture at 37 degrees Celsius, resulting in a loss of biological activity and possible changes in immunogenicity” [Patent #5,216,126. June 1, 1993] – what is known about lupron’s effect on the immune system?

158. What caused the following results, and what do these results mean relative to my immune system?: “Decreased bone marrow B-cells and thymocyte counts were observed in the primary lymphoid tissues of prepubertal female mice following GnRH agonist [lupron] treatment … Previous studies of secondary lymphoid tissues in these same mice also suggested decreased levels of lymphocytes and neutrophils in peripheral blood and reduced splenic and blood B-cells in lupron-treated mice. These results indicate a general suppression of lymphocyte maturation and suggests a potential effect of GnRH agonists [lupron] at an early stem cell stage of leukocyte [white blood cell] development. … Thus, GnRH agonist [lupron] appears to have immunomodulatory effects … which results in suppressed immune responses.” [Journal of Reproductive Immunology (1993);25:167].

159. And what does this mean relative to my immune system?: In women with endometriosis and fibroids, “GnRH agonist [lupron] alters circulating lymphocyte and granulocyte subpopulations” [Fertility and Sterility (1994);S148].

160. Is it significant that within the lupron fibroid NDA it is stated that “decreases in total WBC [white blood count] and neutrophils were observed.” [NDA 19-943]; and also that within the lupron endometriosis NDA it is stated “white blood cell count fell with Lupron. Slight leukopenia has been noted with other analogues.” [NDA 20-011]?

161. Regarding the statement in an article discussing lupron’s use in palliative treatment of prostate cancer [Urology, Supplement (1986);27(1):9-13] that “Although clinical trials investigating LH-RH analogs in the treatment of metastatic prostatic cancer only recently have been completed, the safety and efficacy of these drugs are already established” … yet the very same journal contains another statement in a panel discussion on GnRH analogs: “It is important to remember that all of these newer compounds have not yet undergone the test of time.” Is there a discrepancy between these two statements?

162. Since “[a] physician could get a $103 return from prescribing a 7.5 milligram Lupron dosage because Medicare paid $515, which was 25 percent higher than TAP’s list price of $412
… [called] more bluntly, ‘profit’” ['Congress probes Medicare price ploy: Discounts on drugs enriching doctors. Chicago Tribune. October 15, 2000], as well as TAP giving doctors free samples for which the total cost of lupron ($515) was pocketed [***********google “TAP”, “fraud”, “free samples”] … what caused lupron to become dominant in the field of medicine?

163. In “Species Differences in the Sensitivity to GnRH Analogs” [Journal of Steroidal Biochemistry (1985);23(5B):811-7] it is stated that “the choice of species used for experiments is critical for the extrapolation of the results obtained” … so, what species, and what progression of order) have been used in the “experiments” with lupron? And, what is “shuttling”?

164. What prospective, epidemiologically sound clinical trials are open to all women undergoing assisted reproductive technologies (ART) using lupron?


166. What retrospective studies are underway to assess the long-term health of lupron recipients?

167. Since lupron has NOT received approval by the FDA for the indication of fertility (or IVF or egg donation or surrogacy, etc.) – and therefore its use in IVF is thus characterized as “off-lable” – what significance does the following statement have?: “New Pressure on ‘Off-Label’ Animal Drugs: [Veterinary] Researchers beware: If you administer drugs in animal studies that aren’t specifically approved for the species you’re studying, you could soon wind up in trouble with the FDA, which has recently come under pressure to enforce a federal law that prohibits ‘off-label’ use of veterinary drugs … unlike doctors, who can legally prescribe drugs [to humans] for use other than those for which they were originally approved.” [Science. August 21, 1992. 257:1031]

168. Why was it that animal researchers need “beware” when prescribing an off-label use of a drug for an animal – yet doctors were and are prescribing lupron for hundreds of off-label uses, ranging from Alzheimers to ZIFT? [for a fairly comprehensive (yet incomplete) list of A – Z off-label uses of lupron, see ‘The Check is in the Fe/male’,
http://docs@commerce.senate.gov/hearings/testimony.cfm?id=685&wit_id=1802.]

169. What is the Doctrine of Informed Consent?

170. Will I receive written informed consent regarding lupron, and if so, what will this written informed consent state?

171. The National Lupron Victims Network had compiled over ten thousand questionnaires from victims, and had a presence on the internet (www.lupronvictims.com) – but then they disappeared into thin air. Where did they - and their information - go?
172. If lupron victims report that their physicians deny any correlation between the use of lupron and the development of subsequent health problems, would these same physicians be reporting adverse events” following lupron to the FDA’s Spontaneous Reporting System? ... And if not, are the numbers of adverse events following the use of lupron that have been reported to the FDA’s Spontaneous Reporting System accurately reflective of the number of adverse events?

173. If I develop any adverse events during or following lupron, will you report these adverse events to the FDA’s Spontaneous Reporting System?

174. As of March 2007, according to the FDA’s Spontaneous Reporting System, there were 15,571 total adverse reactions to lupron, with 296 deaths … how many total adverse reactions and deaths from lupron are there today?

175. In a discussion of lupron’s use and its side effects, a physician stated that “societal litigiousness may mandate discussion of even remote possibilities to avoid risk of suit” [American Journal of Obstetric Gynecology (1995);172(4)(1):1323] – why then aren’t published risks of lupron discussed, where are the mandated discussions of these “remote possibilities”, and why have lupron victims encountered a lack of medicolegal advocacy wherever they get shuffled?

176. What is responsible for all the lupron victims - and why are so many so sick?

These ‘TOP144 lupron SUQS’ are a work in progress. There are many more questions than just 144 -- ‘TOP144’ was chosen merely as a pun on words, because Takeda/TAP’s early designation of lupron was “TAP144”.

Today there are 176 ‘TOP144 lupron SUQS’ … tomorrow there may be ‘ABOUT 43818’! (Abbott’s early designation of lupron was “ABBOTT 43818”).