**IN THE UNITED STATES DISTRICT COURT**

**FOR THE DISTRICT OF SOUTH CAROLINA**

**CHARLESTON DIVISION**

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| PALMETTO PHARMACEUTICALS LLC,<br>Plaintiff,<br>v.<br>ASTRAZENECA PHARMACEUTICALS LP,<br>Defendant. | C/A No. 2:11-CV-00807-MBS<br><br>PLAINTIFF'S FIRST AMENDED COMPLAINT AND DEMAND FOR JURY TRIAL |

**FIRST AMENDED COMPLAINT FOR PATENT INFRINGEMENT**

Plaintiff Palmetto Pharmaceuticals LLC ("Palmetto") for its first amended complaint against defendant AstraZeneca Pharmaceuticals LP ("AstraZeneca" or "Defendant") hereby alleges as follows:

**Parties**

1. Palmetto is a small, privately funded pharmaceutical research company headquartered in Charleston, South Carolina, and is engaged in research in the southeastern United States. Palmetto is a limited liability corporation organized under the laws of Delaware, having a principal place of business at 211 King Street, Charleston, South Carolina 29401.

2. AstraZeneca is a limited partnership organized under the laws of Delaware, which does business throughout the United States. AstraZeneca has a principal place of business at 1800 Concord Pike, Wilmington, Delaware 19850. AstraZeneca maintains a registered agent in South Carolina, being CT Corporation System, 2 Office Park Court, Suite 103, Columbia, South Carolina 29223.

## Jurisdiction and Venue

3. This Court has jurisdiction over the subject matter of this action pursuant to 28 U.S.C. § 1338 because this action arises under the patent laws of the United States.

4. AstraZeneca does extensive business in South Carolina and has committed acts of infringement within the state, as herein alleged. As noted, AstraZeneca also maintains a registered agent in the state. By virtue of these contacts, this Court has personal jurisdiction over AstraZeneca.

5. Venue in this district is proper under 28 U.S.C. §§ 1391(c) and 1400 because Defendant has committed acts of infringement in this district and is subject to personal jurisdiction in the state.

## The Patent-in-Suit

6. Palmetto is the assignee and lawful owner of United States Patent No. 6,465,516 B1, entitled "Method of Stimulating Nitric Oxide Synthase," which issued on October 15, 2002, as amended by Reexamination Certificate No. 6,465,516 C1, which issued on issued on April 5, 2011 (collectively the "Palmetto '516 patent"). A copy of United States Patent No. 6,465,516 B1 is attached as Exhibit A. A copy of Reexamination Certificate No. 6,465,516 C1 is attached as Exhibit B.

7. The Palmetto '516 patent claims methods of treating "nonhyperlipidemic" subjects (i.e., people who do not have "hyperlipidemia") who would benefit from increased Nitric Oxide (or "NO") production. These claims, thus, claim a method of use.

8. Hyperlipidemia is a medical condition that usually includes having a high cholesterol level. Persons who are nonhyperlipidemic do not have high cholesterol levels. High cholesterol levels are considered to be abnormal.

9. Method of use claims are patentable in the United States. "Whoever invents or discovers any new and useful process . . . may obtain a patent therefor . . . ." 35 U.S.C. § 101. "The term 'process' . . . includes a new use of a known . . . manufacture, composition of matter, or material." 35 U.S.C. § 100(b).

10. The patented methods of the Palmetto '516 patent involve, *inter alia*, administering an "Hmg-CoA reductase inhibitor" in an amount effective to increase the Nitric Oxide production in a nonhyperlipidemic subject.

11. The claims of the Palmetto '516 patent are directed to a new and useful process by claiming a new use of Hmg-CoA reductase inhibitors such as statins.

**Background Information**

12. The allegations of paragraphs 1-11 are realleged and incorporated as fully alleged herein.

13. In 2003, AstraZeneca began marketing a statin, rosuvastatin calcium, under the trademark "CRESTOR®." CRESTOR has become a widely prescribed statin.

14. In 2003, the United States Food & Drug Administration ("FDA") approved CRESTOR for three uses or "indications," which included treatment of people with hyperlipidemia including elevated cholesterol levels.

**A. The JUPITER Trial and the JUPITER Indications**

15. On information and belief, in 2003, the same year that AstraZeneca first obtained FDA approval to administer CRESTOR to people with hyperlipidemia, AstraZeneca also began enrolling patients in a new clinical trial known as "JUPITER."

16. On information and belief, the purpose of the JUPITER Trial was to evaluate whether people who did not have hyperlipidemia, but who did have other cardiovascular risk factors, could benefit from taking CRESTOR.

17. On information and belief, in approximately 2008, following completion of the JUPITER Trial, AstraZeneca requested FDA approval to market CRESTOR for treating people who do *not* have hyperlipidemia, but who do have other cardiovascular risk factors (the "JUPITER Indications").

18. On December 15, 2009, an Advisory Committee of the FDA convened a meeting to consider AstraZeneca's request to market CRESTOR for the JUPITER Indications (the "12/15/09 FDA Advisory Committee Meeting"). At that meeting, the Advisory Committee received presentations and public comments relating to the JUPITER Indications.

19. Dr. Paul Ridker, a researcher at the Brigham and Women's Hospital in Boston, Massachusetts, served as the Principal Investigator of the JUPITER Trial, and Chaired the Independent Steering Committee of the JUPITER Trial. At the 12/15/09 FDA Advisory Committee Meeting, Dr. Ridker, together with senior scientists from AstraZeneca, made presentations in support of approving marketing CRESTOR for the JUPITER Indications.

20. On information and belief, Dr. Ridker also served as a consultant to AstraZeneca and received research and other support from AstraZeneca.

21. In addition to serving as AstraZeneca's Principal Investigator, Dr. Ridker also is an inventor of U.S. Patent No. 7,030,152 ("the Ridker '152 patent") that is licensed to AstraZeneca. The Ridker '152 patent claims, *inter alia*, a method for treating a nonhyper-cholesterolemic human who has a "high sensitivity C-reactive protein" (also called "hsCRP" or simply "CRP") level above 2.0 by administering a statin.

22. On information and belief, also presenting at the meeting on behalf of AstraZeneca were senior AstraZeneca scientists (i) Dr. Jonathan Fox, Vice President of Clinical Development for Cardiovascular and Gastrointestinal Diseases at AstraZeneca, and (ii) Dr. Michael Cressman, Medical Science Director for the rosuvastatin calcium (CRESTOR) clinical development program at AstraZeneca. On information and belief, Dr. Antonio Gotto, who served on the steering committee of the JUPITER Trial and as a consultant to AstraZeneca, also presented to the Committee.

23. On information and belief, at the 12/15/09 Advisory Committee Meeting, Dr. Ridker explained that the purpose of the JUPITER Trial was to determine if there is a benefit to administering statins to individuals who were nonhyperlipidemic, but who nevertheless had risk factors for cardiovascular disease. Specifically, on information and belief, Dr. Ridker told the 12/15/09 Advisory Committee Meeting that the JUPITER Trial was "conducted among individuals who would not qualify for the drugs because they lack hyperlipidemia," but who did "have evidence of systemic inflammation," i.e., elevated hsCRP. Dr. Ridker has stated as much. Paul M. Ridker et al., *Rosuvastatin to Prevent Vascular Events in Men and Women with Elevated C-Reactive Protein*, 359 N. Eng. J. Med. 2195-2207, 2195 (2008) ("In this trial of apparently healthy persons without hyperlipidemia but with elevated high-sensitivity C-reactive protein levels, rosuvastatin significantly reduced the incidence of major cardiovascular events.").

24. One risk factor for including a nonhyperlipidemic person in the JUPITER Trial was whether he/she had an elevated level of hsCRP or CRP. Specifically, a minimum hsCRP value of 2.0 was required for participation in the JUPITER Trial.

**B. FDA Approval of JUPITER Indications**

25. The FDA approved the use of CRESTOR for the JUPITER Indications on February 8, 2010. Following the approval, the FDA announced on its website: "This is the first time CRESTOR has been approved for use in the prevention of heart disease in individuals with 'normal' low-density lipoprotein (LDL) cholesterol levels and no clinically evident heart disease."

26. On information and belief, AstraZeneca took steps to list the Ridker '152 patent in the FDA's Publication of Approved Drug Products with Therapeutic Equivalence Evaluations (the "Orange Book"). The listing indicates that the Ridker '152 patent covers the use of CRESTOR for the primary prevention of cardiovascular disease.

27. In March 2010, the FDA published the Ridker '152 patent in the Orange Book, and in April 2010, AstraZeneca began suing a number of generic drug manufacturers on the Ridker '152 patent.

28. On information and belief, although none of the generic drug manufacturers had yet included the JUPITER Indications in their proposed labels for generic CRESTOR, AstraZeneca nevertheless sued them on the basis that they eventually would have to include the JUPITER Indications in their labels, and that merely selling generic CRESTOR with a label that included the JUPITER Indications in the label would constitute infringement of the Ridker '152 patent.

**C. Administering a Statin to a Person with Elevated hsCRP Is Administering a Statin to a Person in Need of Increased Nitric Oxide**

29. Scientific publications in the field of cardiovascular disease, including multiple publications by Dr. Ridker, establish that administering a statin to a person with elevated hsCRP is administering a statin to a person in need of increased Nitric Oxide production.

30. Peer-reviewed scientific literature in the field of cardiovascular science, including publications by Dr. Ridker himself, have established that (i) decreased Nitric Oxide production is implicated in all known cardiovascular disease, (ii) elevated hsCRP is associated with reduced production of Nitric Oxide, and (iii) statins can increase the production of Nitric Oxide in people with elevated hsCRP. *See* James T.Willerson & Paul M. Ridker, *Inflammation as a Cardiovascular Risk Factor*, 109Circulation II-2–II-10, supp. II at II-3 (2004) ("Decreased NO production is implicated in the clinical course of all known CVD. NO inhibits platelet adherence and aggregation, suppresses vasoconstriction, reduces the adherence of leukocytes to the endothelium, and suppresses the proliferation of vascular SMC. Therefore, a reduction in NO activity contributes to a proinflammatory and prothrombotic milieu. CRP may itself play a role in repressing the production of NO and diminishing NO bioavailability."); Subodh Verma, et al., *A Self-Fulfilling Prophecy: C-Reactive Protein Attenuates Nitric Oxide Production and Inhibits Angiogenesis*, 106 Circulation 913-19, 913 (2002) ("Verma et al. 2002") ("CRP, at concentrations known to predict adverse vascular events, directly quenches the production of the NO. . . . Through decreasing NO synthesis, CRP may facilitate the development of diverse cardiovascular diseases . . . ."); Senthil Kumar Venugopal et al., *Demonstration That C-Reactive Protein Decreases eNOS Expression and Bioactivity in Human Aortic Endothelial Cells***,** 106 Circulation 439-1441, 1441 (2002) ("[u]sing a variety of techniques, we convincingly show that CRP causes a decrease in eNOS expression and bioactivity, especially at the higher

concentration. . . . ."); Uma Singh et al., *C-Reactive Protein Decreases Endothelial Nitric Oxide Synthase Activity via Uncoupling*, 43 J. Molecular & Cellular Cardiology 780–91, 787 (2007) ("CRP levels are correlated with increased risk for CVD and with endothelial dysfunction related to reduced NO bioavailability.").

31. Dr. Ridker likewise recognized that elevated hsCRP is associated with decreased Nitric Oxide production. In fact, in his 2003 publication explaining the rationale for the JUPITER Trial (the "2003 JUPITER Rationale"), Dr. Ridker cited Verma et al. 2002, among others, as support for administering CRESTOR to nonhyperlipodemic patients with elevated hsCRP. Paul M. Ridker, *Rosuvastatin in the Primary Prevention of Cardiovascular Disease Among Patients With Low Levels of Low-Density Lipoprotein Cholesterol and Elevated High-Sensitivity C-Reactive Protein: Rationale and Design of the JUPITER Trial*, 108 Circulation 2292-97, 2292 (2003) ("evidence has recently accumulated that shows CRP to be a direct participant in the atherothrombotic process capable of augmenting the innate inflammatory response, triggering expression of adhesion molecules and monocyte chemoattractant protein-1, attenuating expression of endothelial NO synthase . . ."") (citing Verma et al. 2002 and other publications).

32. Dr. Ridker illustrated the relationship between increased hsCRP and decreased NO production in Figure 2, noting that "CRP attenuates NO production, decreases eNOS expression."[1]

[1] Endothelial Nitric Oxide synthase or "eNOS" is an enzyme type that is responsible for the production of Nitric Oxide in humans. The Nitric Oxide synthases exist in a variety of forms, including a constitutive form (cNOS) and an inducible form (iNOS).

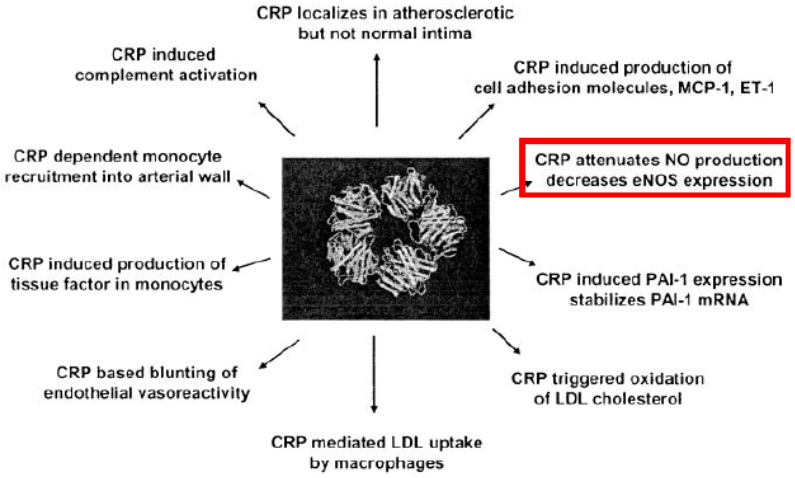


**Figure 2.** Mechanisms relating C-reactive protein (CRP) to development and progression of the atherothrombotic process. eNOS indicates endothelial NO synthase; ET-1, endothelin-1; MCP-1, monocyte chemoattractant protein-1; and PAI-1, plasminogen activator inhibitor-1.

*Id*. at 2293 (Figure 2) (rectangle added).

33. Dr. Ridker also had earlier credited Verma et al. for their observation that hsCRP is associated with decreased Nitric Oxide production. Gavin J. Blake & Paul M. Ridker, *C-Reactive Protein, Subclinical Atherosclerosis, and Risk of Cardiovascular Events*, 22 Arterioscler. Thromb. Vasc. Biol. 1512-13, 1513 (2002) ("Very recently, Verma and colleagues have reported that CRP, at concentrations known to predict future cardiovascular events, directly quenches the production of NO . . . These data suggest that by suppressing NO synthesis, CRP plays a direct role in the pro-atherogenic process.").

34. Dr. Ridker has written on the involvement of cytokines with inflammation, Nitric Oxide and C-reactive protein. *See* Peter Libby & Paul M. Ridker, *Novel Inflammatory Markers of Coronary Risk: Theory Versus Practice*, 100 Circulation 1148-50, 1148-49 (1999), and Peter Libby et al., *Inflammation in Atherosclerosis: From Pathophysiology to Practice*, 54 J. Am. Coll. Cardiol. 2129-38, 2130 (2009).

35. Dr. Donald Hunninghake of AstraZeneca's U.S. Medical Affairs has written:

> Statin Therapy
>
> The proposed mechanisms underlying the clinical benefits of therapy with statin drugs, which inhibit 3-hydroxy-3-methylglutaryl coenzyme-A (HMG-CoA)

> reductase, include depletion of plaque lipid, decreased inflammation, and increased nitric oxide (NO) production. NO has antithrombotic and vasodilator effects and produces many effects similar to other antioxidants. A thicker fibrous cap is formed and there is overall plaque stabilization. The proinflammatory cytokines also stimulate the liver with increased release of C-reactive protein (CRP) and serum amyloid A into the systemic circulation (Figure 2). Statin therapy is associated with reductions in CRP levels.

Donald B. Hunninghake, *Cardiovascular Disease in Chronic Obstructive Pulmonary Disease*, 2 Proc. Am. Thorac. Soc. 44-49, 44-45 (2005).

**D. The Reexamination**

36. On February 12, 2010, Palmetto filed a request in the United States Patent & Trademark Office (USPTO) for reexamination of claim 1 of its Palmetto '516 patent. On March 26, 2010, Palmetto filed a replacement request in the USPTO providing additional information concerning Palmetto's request for reexamination.

37. Both requests identified the Ridker '152 patent as a patent that had not been considered during the original prosecution of the Palmetto '516 patent. (The Ridker '152 patent did not issue as a patent until April 2006, i.e., more than three years after the Palmetto '156 patent issued.) Ultimately, the Ridker '152 patent was not found to be prior art against the Palmetto '516 patent because, *inter alia*, the USPTO determined that the earliest applications leading to the Ridker '152 patent did not disclose the invention of the Palmetto '516 patent.

38. Palmetto also identified Dr. Ridker's 2003 JUPITER Rationale (discussed *supra*), and included the same Figure 2 (discussed *above*) in laying out the basis for its reexamination request.

39. The USPTO agreed with Palmetto that the Ridker's 2003 JUPITER Rationale makes clear that "a subject with an elevated CRP level" is a subject recited in claim 1 of the Palmetto '516 patent, stating in a May 19, 2010 Order Granting Request for Ex Parte

Reexamination that "[a] publication by Dr. Ridker in 2003 (Circulation: 108: 2292 - 2292) which is not prior art, reported that increased CRP levels correlate to an attenuation in nitric oxide production, thereby identifying a subject with an elevated CRP level as 'a subject who would benefit from increased nitric oxide production in a tissue' as recited in claim 1 of the Kaesemeyer Palmetto '516 patent."

**E. Palmetto's Offer to License the Palmetto '516 Patent: Notice to AstraZeneca**

40. Following publication of the JUPITER Trial results, Palmetto offered AstraZeneca the opportunity to license or acquire the Palmetto '516 patent via emails on November 8, 2008, and November 13, 2008. Dr. Jamieson of Palmetto also provided AstraZeneca with an article disclosing that elevated hsCRP is associated with decreased Nitric Oxide production and that administering statins overcomes the decreased production.

41. On December 9, 2008, AstraZeneca declined Palmetto's offer.

**Infringing Acts**

42. The allegations of paragraphs 1-41 are realleged and incorporated as fully alleged herein.

43. AstraZeneca makes, offers to sell, and sells an Hmg-CoA reductase inhibitor called CRESTOR, also known as rosuvastatin calcium.

44. In the package insert accompanying CRESTOR and elsewhere, AstraZeneca indicates and instructs the use of CRESTOR

> In individuals without clinically evident coronary heart disease but with an increased risk of cardiovascular disease based on age ≥ 50 years old in men and ≥ 60 years old in women, hsCRP ≥ 2mg/L, and the presence of at least one additional cardiovascular disease risk factor such as hypertension, low HDL-C, smoking, or a family history of premature coronary heart disease, CRESTOR is indicated to:

- reduce the risk of stroke
- reduce the risk of myocardial infarction
- reduce the risk of arterial revascularization procedures

45. The CRESTOR package insert more fully instructs doctors, other medical professionals, users, and potential users of CRESTOR that using CRESTOR for the "Primary Prevention of Cardiovascular Disease" benefits individuals who qualify as nonhyperlipidemic:

> **14.8 Primary Prevention of Cardiovascular Disease**
>
> In the **J**ustification for the **U**se of Statins in **P**rimary Prevention: An **I**ntervention **T**rial **E**valuating **R**osuvastatin (JUPITER) study, the effect of CRESTOR (rosuvastatin calcium) on the occurrence of major cardiovascular (CV) disease events was assessed in 17,802 men (≤ 50 years) and women (≤ 60 years) who had no clinically evident cardiovascular disease, LDL-C levels <130 mg/dL (3.3 mmol/l) and hs-CRP levels ≥2 mg/L. The study population had an estimated baseline coronary heart disease risk of 11.6% over 10 years based on the Framingham risk criteria and included a high percentage of patients with additional risk factors such as hypertension (58%), low HDL-C levels (23%), cigarette smoking (16%), or a family history of premature CHD (12%). Study participants had a median baseline LDL-C of 108 mg/dL and hsCRP of 4.3 mg/L. Study participants were randomly assigned to placebo (n=8901) or rosuvastatin 20 mg once daily (n=8901) and were followed for a mean duration of 2 years. The JUPITER study was stopped early by the Data Safety Monitoring Board due to meeting predefined stopping rules for efficacy in rosuvastatin-treated subjects.

46. As noted above, treating a nonhyperlipidemic individual having an elevated hsCRP (e.g., above 2.0 as set forth on the label) by administering CRESTOR is treating a subject who would benefit from increased Nitric Oxide production by administering an Hmg-CoA reductase inhibitor in an amount effective to increase Nitric Oxide production. Nonhyperlipidemic individuals include individuals having an LDL-C (low density lipoprotein cholesterol) of less than 130 mg/DL or less than 108 mg/dL, as set forth in 14.8 of the CRESTOR packaging insert, as shown Paragraph 45, *supra*.

47. Since at least November 2008, AstraZeneca has actual knowledge of the Palmetto '516 patent, and is aware that the use of CRESTOR as described in Paragraphs 43-46 infringes at least claims 1, 4, 7 and 15-20 of the Palmetto '516 patent. The use of CRESTOR as described in Paragraph 43-46 also infringes reexamined claims 15-20 of the Palmetto '516 patent.

48. As set forth in the CRESTOR package insert, the administration of CRESTOR is beneficial because it reduces the risks of events such as stroke, angina, and arterial revascularization procedures. *See* Figures 2 and 3 of Section 14.8. The risk posed by any attendant reperfusion injury is thereby reduced.

49. The CRESTOR package insert identifies hypertension as a risk factor that indicates a need for the administration of CRESTOR. *See* Section 1.6.

**Count One: Direct Infringement of the Palmetto '516 Patent**

50. The allegations of paragraphs 1-49 are realleged and incorporated as fully alleged herein.

51. AstraZeneca makes, offers to sell, and sells CRESTOR throughout the United States.

52. AstraZeneca directly infringes the claims of the Palmetto '516 patent by providing CRESTOR to nonhyperlipidemic individuals who would benefit from increased Nitric Oxide production.

53. AstraZeneca's acts constitute infringement of the Palmetto '516 patent under 35 U.S.C. § 271(a).

**Count Two: Induced Infringement of the Palmetto '516 Patent**

54. The allegations of paragraphs 1-53 are realleged and incorporated as fully alleged herein.

55. The Palmetto '516 patent is directly infringed by the provision of CRESTOR by, *inter alia*, doctors, other medical professionals, and/or patients to nonhyperlipidemic subjects who would benefit from increased Nitric Oxide production.

56. Through its label/package insert, website, and promotional materials, AstraZeneca instructs the public, including nonhyperlipidemic individuals who would benefit from increased Nitric Oxide production, to use CRESTOR in an infringing manner, and to contact their physicians or other medical professionals to seek CRESTOR for use in an infringing manner.

57. Through its label/package insert, website, and funded publications, AstraZeneca instructs and encourages doctors and/or other medical professionals to prescribe or/or provide CRESTOR in a way that when so used infringes the claims of the Palmetto '516 patent.

58. On information and belief, AstraZeneca employs pharmaceutical sales specialists in South Carolina who encourage, *inter alia*, doctors and/or other medical professionals to prescribe and/or provide CRESTOR in a way that when so used infringes the claims of the Palmetto '516 patent.

59. AstraZeneca has knowledge of the Palmetto '516 patent and that the use indicated and promoted on its label infringes the claims of the Palmetto '516 patent. AstraZeneca intentionally encourages this infringing use.

60. AstraZeneca's acts constitute active inducement of infringement of the Palmetto '516 patent, and it is liable as an infringer under 35 U.S.C. § 271(b).

**Count Three: Contributory Infringement of the Palmetto '516 Patent**

61. The allegations of paragraphs 1-60 are realleged and incorporated as fully alleged herein.

62. AstraZeneca sells and offers to sell in the United States a material— CRESTOR —with a package insert containing instructions indicating and promoting use of CRESTOR in manner that infringes claims of the Palmetto '516 patent.

63. CRESTOR and its package insert are especially made for, and intended for, a use that infringes the claims of the Palmetto '516 patent and are not a staple article or commodity of commerce suitable for noninfringing use.

64. Astra Zeneca's acts make it liable as a contributing infringer under 35 U.S.C. § 271(c).

**Count Four: Willful Infringement of the Palmetto '516 Patent**

65. The allegations of paragraphs 1-64 are realleged and incorporated as fully alleged herein.

66. AstraZeneca's infringement of the Palmetto '516 patent was intentional and willful.

**Prayer for Relief**

WHEREFORE, Palmetto seeks judgment against AstraZeneca as follows:

1. For monetary damages sufficient to compensate Palmetto for AstraZeneca's infringement of the Palmetto '516 patent;
2. For enhanced damages due to the willful nature of AstraZeneca's conduct;
3. For a permanent injunction prohibiting AstraZeneca from infringing the Palmetto '516 patent in the future;
4. For its attorneys fees and costs; and
5. For such further relief as to which Palmetto may be entitled.

**Jury Demand**

Palmetto hereby demands a trial by jury.

Dated: May 16, 2011

By: S/ A. Camden Lewis

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