SUPREME COURT OF THE STATE OF NEW YORK
COUNTY OF NEW YORK

NEW YORK UNIVERSITY,	:	Index No. _____
Plaintiff,	:	
v.	:	**COMPLAINT**
PFIZER, INC.,	:	
Defendant.	:	

Plaintiff New York University ("NYU"), by and through its undersigned counsel, as its complaint against Defendant Pfizer, Inc. ("Pfizer"), alleges as follows:

NATURE OF THE ACTION

1. This is an action brought by NYU against Pfizer for damages resulting from Pfizer's breach of an agreement. To provide guidance with respect to the following detailed description, simply stated, this action is for breach of contract based on Pfizer's failure to comply with the terms of the agreement to pay royalties to NYU based on sales of its drug XALKORI, which is used to treat non-small cell lung cancer harboring an ALK mutation. Under an agreement between NYU and Sugen, Inc. ("Sugen"), which was acquired by Pfizer, NYU is due royalties for any drug product derived from or based on "NYU Know-How," if that drug product was developed based on a receptor target which was not adopted into its drug discovery prior to the acquisition of Sugen. The active ingredient in XALKORI is a small molecule inhibitor named crizotinib, which was based on or derived from NYU Know-How and research. Further, XALKORI is a drug that was developed based on a receptor target which was not adopted into drug discovery at the time of the acquisition of Sugen. Thus, royalties are due NYU under the agreement.

2. The contract Pfizer is accused of breaching was originally executed by NYU and Sugen and is entitled "Second Amended Restated NYU/Sugen Research and License Agreement" ("The Agreement"), that was effective as of September 1, 1991. Pfizer has specifically breached Section 9 of The Agreement, because Pfizer, as the successor in interest of Sugen, has failed to pay NYU the royalty fees required in that Section based on Pfizer's sales of its XALKORI drug product.

3. The Agreement at issue provides, *inter alia,* for:

A. Sugen to make payments to NYU to fund ten years of NYU's investigations and research in a very specific field of receptors, e.g. receptor tyrosine kinases (the "NYU Research Project'), and where the information or materials discovered, developed or acquired by NYU in the NYU Research Project is designated as the "Research Technology" (The Agreement Sections 1(i),(k),(m),(o), 3, 4, Appendix II);

B. NYU to grant to Sugen "the exclusive worldwide license to practice the [NYU's] Research Technology for the research, development, manufacture . . . of SUGEN Products" which include "any product for the diagnosis, treatment or prevention of human disease which contains . . . any substance which activates or prevents activation of a Receptor," *e.g.*, receptor tyrosine kinases (The Agreement Sections 7, 1(e),(m),(q)); and

C. in the event that Sugen is acquired or merged with another company, the acquiring company agrees to pay NYU certain royalties based on sales of certain SUGEN Products, e.g. where the utility of the SUGEN Product "is derived from or based on the Research Technology," and where the SUGEN Product "is developed based on Receptor targets which were not adopted into drug discovery at the time of the. . . acquisition, merger. . . " (The Agreement Section 9).

4. NYU's Research Technology included determination of a three dimensional crystal structure of the tyrosine kinase domain of the FGFR1. The foregoing NYU information enabled Sugen and later Pfizer to systematically design and efficiently test candidate small molecule compounds as inhibitors of additional receptor tyrosine kinases. One such

investigation for an inhibitor of the Met receptor resulted in the design of a small molecule inhibitor named crizotinib (PF-02341066).

5. Subsequently, after the acquisitions of Sugen by Pharmacia and later by Pfizer, and based upon the inhibitor effect of crizotinib on MET, crizotinib was further screened for activity against other receptor tyrosine kinases ("RTK") and found to have inhibitory effect on at least thirteen other human kinases, including ALK, another member of the RTK family. In 2007, Japanese scientists at Jichi Medical University published a scientific paper implicating EML4-ALK, a mutated version of the RTK ALK, in a population of non-small cell lung cancers. When Pfizer became aware of this paper, Pfizer adopted EML4-ALK as a target for drug discovery for the treatment of non-small cell lung cancer, leading to the development of XALKORI with its active ingredient crizotinib.

PARTIES AND VENUE

6. Plaintiff NYU is a corporation organized and existing under the laws of the State of New York. NYU operates an internationally prominent research university including its medical center, in the City and County of New York with its principal office at 70 Washington Square South, New York, New York 10012.

7. Defendant Pfizer is an internationally prominent pharmaceutical corporation organized and existing under the laws of the State of Delaware having its principal place of business in the City and County of New York at 234 East 42nd Street, New York, New York 10017.

8. Venue is proper in this Court under CPLR 503 because NYU and Pfizer reside in New York County.

9. Pursuant to Section 21(d) of The Agreement, Pfizer has consented to the jurisdiction and venue of this Court.

FACTUAL BACKGROUND

A. Pertinent Corporate Entities And Individuals

10. NYU is a prestigious internationally recognized academic institution. In addition to the quality education it provides to its undergraduate and graduate students, NYU is recognized as a premier research institution, renowned for generating new ideas, insights, and knowledge. The goals of its basic research activities are to promote and nurture scientific progress. This commitment to research excellence is a key factor in NYU's ability to attract and secure grant money and financial gifts necessary, not only to support the University's existing and future educational initiatives and academic research programs, but also to attract the highest quality and expert faculty, and to enhance NYU's physical facilities.

11. In 1990, NYU Medical Center hired Dr. Joseph Schlessinger to serve as Professor and Chairman of the Department of Pharmacology. Before joining NYU, Dr. Schlessinger had established himself as a pioneer and expert in elucidating the mechanism of action of RTKs and other tyrosine kinase as well as the role played by these enzymes and their signaling pathways in cancer and other diseases. The general objective of his work was to investigate the role of RTKs and their signaling pathways as a means to inhibit or control the growth of cells, such as cancer cells.

12. NYU understood the significance of Dr. Schlessinger's accomplishments, expertise, and field of his research, and recognized the potential of his work, which included the discovery and development of medicines for treating cancers and other diseases. In particular, NYU appreciated Dr. Schlessinger's unique insight in this regard, including his belief that novel anticancer agents could be designed by conducting research in the structure and function of the tyrosine kinase receptor family, including research focusing on elucidating the structural interactions between these receptor proteins in complex with small molecule inhibitors.

13. In 1991, while an NYU employee, Dr. Schlessinger co-founded Sugen, a drug discovery company located in Redwood City, California to help carry out Dr. Schlessinger's visions, specifically, with respect to the design of drug compounds. This would complement NYU's research on the mechanisms underlying the activity of receptor tyrosine kinase function as a foundation for a drug discovery platform.

14. On August 16, 1991, NYU and Sugen entered into an agreement under which Sugen agreed to sponsor and pay NYU annual fees to conduct an "NYU Research Project" in the field of "Receptors," which included receptor tyrosine kinases.

15. The NYU Research Project as defined in 1991 was to be led by NYU scientist Dr. Joseph Schlessinger. Dr. Schlessinger actually held two leadership roles in the NYU/Sugen research. In addition to his leadership role at NYU, he was on the Scientific Advisory Board at Sugen.

16. On information and belief, Sugen was acquired by Pharmacia and Upjohn in or about 1999, which company was later renamed Pharmacia. On information and belief, after being acquired, Sugen remained a wholly owned subsidiary and continued to function in its research as a separate entity within Pharmacia.

17. In 2001, after Sugen's acquisition by Pharmacia, Dr. Schlessinger left NYU to take a position as Professor and Chairman of the Department of Pharmacology at Yale University School of Medicine. This was about the same time that the ten-year NYU Research Project funded by Sugen came to an end.

18. In about 2003, Defendant Pfizer acquired Pharmacia, which included Sugen. On information and belief, after the acquisition, Sugen did not retain a separate corporate identity from Pfizer.

19. Pfizer has been recognized as one of the world's largest research-based pharmaceutical companies. Pfizer has received numerous awards and commendations for its innovative prescription medicines to fight cancer and other diseases, and to help people live longer, healthier lives.

B. The Agreement At Issue

20. In about 1996, NYU and Sugen entered into The Agreement, to be effective as of September 1, 1991. In the "Recitals" of The Agreement, the Parties expressed the desire that The Agreement "amend" and "restate" two prior agreements between NYU and Sugen: (i) the "NYU/SUGEN Research and License Agreement," of September 1, 1991, and (ii) the "Amended and Restated NYU/SUGEN Research And License Agreement" of November 1993.

21. The Agreement defines the "Research Period" to mean the "ten year period" commencing on September 1, 1991, during which Sugen agrees to fund a research program or project at NYU, the subject and goals of the NYU Research Project being described in Appendix II of the three NYU/Sugen Agreements. The annual dollar amounts for Sugen's funding to NYU appears in Section 4(a) in each of the three agreements. The Agreement states the amounts Sugen is to pay NYU during the seventh through the tenth years.

22. Pfizer has breached The Agreement by failing to pay NYU the royalty payments due and owing based on at least Pfizer's sales of its XALKORI drug product, pursuant to Section 9 of The Agreement.

23. The Agreement was and is supported by good and valuable consideration.

24. NYU has fully and adequately performed its obligations under The Agreement.

25. The obligations of Sugen and its successors to pay royalties to NYU under The Agreement have not expired, have not been terminated, and remain in effect.

26. Pfizer has admitted that The Agreement applies to Pfizer as a result of its acquisition of Sugen as part of the acquisition of Pharmacia, and that Pfizer is obligated to pay royalties to NYU based on certain products that are marketed by Pfizer. In a letter from Pfizer's Vice-President Patricia S. Andrews to NYU dated February 7, 2006, Pfizer acknowledged its obligation under The Agreement to pay NYU license royalty fees specified in The Agreement based on Pfizer's sales of its drug product SUTENT, which was first sold on or about February 6, 2006. Subsequently, Pfizer has provided NYU with its sales data for the SUTENT drug product and has paid NYU royalties in accordance with The Agreement.

C. The NYU Research Project Under The Agreement, Conducted Under the Supervision Of Dr. Schlessinger

27. The first "WHEREAS" clause of The Agreement refers to Dr. Joseph Schlessinger of NYU as "the NYU Scientist," and in Section 3(a) he is designated to lead the NYU Research Project during the Research Period.

28. Section 1(k) of The Agreement defines "NYU Research Project" to mean "the investigations during the Research Period into the field of the Receptors under the direction of the NYU Scientist [Dr. Schlessinger] which are funded by SUGEN and include the research programs described in Appendix II hereto which forms an integral part hereof." The research program described in Appendix II reflects the pioneering approach to anticancer drug discovery envisioned by NYU and Sugen under Dr. Schlessinger's leadership, and is clearly described as follows:

> The proposed research is an investigation of the mechanisms underlying the action of receptors which control the level of cellular phosphotyrosine by regulating the activity of either protein tyrosine kinases or protein tyrosine phosphatases.

Among the specific goals listed in Appendix II for NYU in this joint research program was:

> To determine the three dimensional structure of the protein tyrosine kinase domain in complex with inhibitors using x-ray crystallography.

In Section 1(m) of The Agreement, "Receptor" is defined as follows:

> receptor tryrosine kinases, intracellular tyrosine kinases, or receptors that directly or indirectly activate non-receptor tyrosine kinases; and/or
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> receptor serine/threonine kinases, intracellular serine/threonine kinases, or receptors that directly or indirectly activate serine/threonine kinases; and/or
>
> receptor tyrosine phosphatases, intracellular tyrosine phosphatases, or receptors that directly or indirectly activate tyrosine phosphatases; and/or
>
> molecules that regulate the signaling of the above receptors.

29. Dr. Schlessinger was convinced that small molecule inhibitors of tyrosine kinases could be successfully designed which selectively interfered with the binding of these receptors with adenosine triphosphate ("ATP"). By envisioning and pursuing a drug development strategy based on designing small molecule inhibitors of the ATP binding site of the tyrosine kinase receptor, Dr. Schlessinger led a pioneering effort in the field of anticancer drug development. Indeed, conventional scientific wisdom suggested that an ATP antagonist would produce serious adverse effects *in vivo* due to cellular toxicity, and thus such inhibitors would prove unacceptable as a pharmaceutical drug product. *See* J. Schlessinger, *SU11248: Genesis of a New Cancer Drug*, The Scientist Magazine (Apr. 11, 2005). Notwithstanding the general skepticism exhibited by the pharmaceutical industry, NYU joined Dr. Schlessinger in the research for small molecule inhibitors of the ATP binding site of receptor tyrosine kinases. They have proven to be right in that the use of small molecule inhibitors of the ATP binding site has led to targeted therapy, which has become a major focus of oncology research.

30. Under Dr. Schlessinger's leadership, several scientists were included in NYU's research team, and NYU soon began publishing its findings and substantial accomplishments. One of the leading NYU scientists conducting work in this project was Dr. Moosa Mohammadi, who focused on characterizing the structure and function of fibroblast growth factor receptor 1 ("FGFR1"), and specifically the understanding of the structure of the tyrosine kinase domain of

this receptor. Another important NYU scientist working with Dr. Schlessinger on this project was Dr. Stevan Hubbard, whose research also focused on receptor tyrosine kinases. Dr. Hubbard's work under the NYU Research Project included the use of X-ray crystallography to elucidate the molecular mechanisms associated with ligand binding and associated structural changes in the FGFR1 tyrosine kinase domain upon ligand binding.

31. NYU's research efforts soon resulted in the elucidation of the X-ray crystallographic structures of FGFR1 kinase, both alone and in complex with an ATP analog. This information was the result of research performed solely by NYU's scientists, and reflect the scientific expertise and know-how of Drs. Mohammadi, Schlessinger, and Hubbard, including know-how and expertise which permitted not only the successful expression and purification of the FGFR1 kinase, but also the crystallization of the FGFR1 kinase. Moosa Mohammadi *et al.*, *Structure of the FGF Receptor Tyrosine Kinase Domain Reveals a Novel Autoinhibitory Mechanism*, 86 Cell, 577-87 (Aug. 23, 1996).

32. Further, under the NYU Research Project, NYU appreciated that the discovery of the crystals and atomic structure coordinates of FGFR1 kinase provided a unique and powerful means and technique to screen and identify compounds that inhibit receptor and nonreceptor tyrosine kinases, and thus could be used to facilitate the design and development of new therapeutic agents. In addition, NYU recognized that the discovery of the crystals and atomic structure coordinates of FGFR1 kinase provided a key tool and important know-how for creating homology models of other tyrosine kinases based on this information, as well as for facilitating the elucidation of crystal structures of other tyrosine kinase domains, including the kinase domain of the MET receptor, and the structures of co-crystals of such domains with ligands such as inhibitors, agonists, and antagonists.

33. With the crystal structure of the FGFR1 kinase now in hand, NYU and Sugen used this unique research tool and X-ray crystallography to determine the three-dimensional structure of the FGFR1 tyrosine kinase domain in complex with other compounds to screen for potential tyrosine kinase inhibitors. NYU disclosed its information, discoveries, and know-how to Sugen, which was facilitated by Dr. Schlessinger's roles at both NYU and Sugen. Also, Sugen candidate compounds were brought to NYU for screening by NYU scientists. Thus, the crystal structures of the FGFR1 kinase became an important tool used by NYU under the NYU Research Project in the quest to identify tyrosine kinase inhibitors with potential therapeutic value.

34. During the NYU Research Project, NYU and Sugen worked to identify inhibitors of several different tyrosine kinase receptors. For example, Sugen prepared compounds SU4984 and SU5402, two oxindole-based compounds, and Sugen initially tested them using biochemical and cell culture assays as possible inhibitors of Vascular Endothelial Growth Factor receptors. After Sugen's initial tests, Sugen then asked NYU to test the oxindole/indolinone compounds SU4984 and SU5402. Accordingly, NYU made crystals of FGFR1 kinase according to the methodology described in NYU's 1996 *Cell* publication, separately soaked these crystals with SU4984 and SU5402, and determined the crystal structures of FGFR1 kinase in complex with these two compounds. NYU's X-ray crystallographic data with SU4984 and SU5402 revealed that compounds with the oxindole/indolinone chemical structure had promise as potential protein tyrosine kinase inhibitors. Moosa Mohammadi *et al.*, *Structures of the Tyrosine Kinase Domain of Fibroblast Growth Factor Receptor in Complex with Inhibitors*, 276(5314) Science 955-60 (May 9, 1997).

35. As shown in the *Science* article, NYU's FGFR1 kinase crystals could be used to rationally design modified inhibitors based upon an oxindole core which could possess enhanced binding affinity and improved selectivity. In this article, NYU and Sugen not only reported the identification of a new class of protein tyrosine kinase inhibitors based on an oxindole core (indolinones), but also concluded that the structural information from these studies could be used to "facilitate the design of new inhibitors for use in the treatment of cancer and other diseases in which cell signaling by tyrosine kinases plays a crucial role in disease pathogenesis." *Id.* Abstract. NYU's FGFR1 kinase crystals were also used to validate the inhibitory potential of the oxindole compound SU6668, and the FGFR1/SU6668 co-crystal was also used to create homology models of SU6668 with other receptor tyrosine kinases. A. Douglas Laird *et al.*, *SU6668 Is a Potent Antiangiogenic and Antitumor Agent That Induces Regression of Established Tumors*, 60 Cancer Research 4152-60 (Aug. 1, 2000).

36. Research performed at NYU based on the use of the FGFR1 kinase crystal resulted in the discovery of compound SU11248 (sunitinib). As reported in a publication by Dr. Schlessinger in 2005: "availability of the crystal structure of SU5402 and SU6668 in complex with the PTK domain of FGFR1, combined with modeling and iterative analyses . . . facilitated the discovery of . . . , SU11248," which is the active ingredient in the blockbuster drug product SUTENT. J. Schlessinger, *SU11248: Genesis of a New Cancer Drug*, The Scientist Magazine (Apr. 11, 2005). This illustrates how Sugen, and thereafter Pfizer, utilized and relied upon NYU's "Research Technology" to streamline and accelerate their drug discovery processes.

37. During the NYU Research Project, Sugen's drug discovery team also decided to pursue small-molecule inhibitors of the MET receptor. *See* Xueyan Wang *et al., Development of*

the First Generation c-Met Kinase Inhibitors: Beginning of a Path to a New Treatment for Cancer, 10(11) Mol. Cancer Ther. 2022-23 (Nov. 2011). These efforts to characterize the chemical structure of an inhibitory molecule of MET also relied on and were derived from NYU's pioneering studies characterizing the X-ray crystal structure of the kinase domain of FGFR1. Information gained from these efforts facilitated later studies with homology models and X-ray crystal structures of the MET receptor kinase domain, which led to the small-molecule inhibitor crizotinib. The specific research leading to crizotinib is discussed below in the context of the development of the drug XALKORI.

D. <u>The Information And Research Leading To XALKORI</u>

38. XALKORI is a prescription drug sold by Pfizer to treat patients having Non-Small Cell Lung Cancer ("NSCLC") harboring ALK mutations that is advanced or has spread to other parts of the patient's body. Crizotinib is the active ingredient in XALKORI.

39. The crizotinib compound was first identified as an inhibitor of the target MET receptor. The efforts leading to the chemical structure of crizotinib as an inhibitory molecule of MET relied on NYU's pioneering studies as illustrated above.

40. While crizotinib can inhibit the MET receptor, MET is not the receptor target of Pfizer's FDA-approved drug XALKORI. The target receptor for XALKORI is EML4-ALK, a mutated form of the ALK receptor. According to the FDA's Accelerated Approval letter to Pfizer for XALKORI, dated August 26, 2011, XALKORI was approved for the treatment of patients with locally advanced or metastatic NSCLC that is anaplastic lymphoma kinase (ALK)-positive as detected by an FDA–approved test. Therefore, the target receptor for XALKORI is ALK, and more particularly, EML4-ALK, a mutated form of the ALK receptor, which must be found in a patient in accordance with said Accelerated Approval letter of August 26, 2011, before administering or prescribing XALKORI to a patient. In addition, the

FDA-approved label for XALKORI confirms that administration of XALKORI to a patient requires the presence of the mutated ALK receptor in the patient.

41. The ALK target receptors (including mutated forms such as EML4-ALK) are members of the protein tyrosine kinase family of receptors. Pfizer's earlier studies involving the ALK target receptors focused on the mutated NPM-ALK receptor and the possible treatment of Anaplastic Large Cell Lymphoma (ALCL). Pfizer modified its drug development strategy in 2007, after learning from an article from a Japanese university published in *Nature* that there was a link between the EML4-ALK mutant receptor and NSCLC. Manabu Soda *et al.*, *Identification of the transforming EML4-ALK fusion gene in non-small-cell lung cancer*, 448(7153) Nature 561-66 (Aug. 2, 2007 (e-published July 11, 2007)). Therefore, in 2007, Pfizer began to investigate the specific targeting of the EML4-ALK receptor to treat NSCLC.

42. The target receptor *EML4-ALK* was only first adopted into drug discovery by Pfizer in about 2007, after the foregoing Japanese publication. This is confirmed by Pfizer's own publications describing the development of XALKORI. Eunice L. Kwak *et al*., *Anaplastic Lymphoma Kinase Inhibition in Non–Small-Cell Lung Cancer,* 363(18) New England J. Med. 1693-1703 (Oct. 28, 2010).

43. On information and belief, prior to the Japanese publication, Pfizer had pending before the FDA an Investigational New Drug application (IND), which identified crizotinib and listed MET among the target receptors for this compound. On information and belief, after the Japanese publication, Pfizer notified the FDA of its desire to change the target receptor for development of the drug from MET to ALK. This is documented, for example, in the FDA's Drug Approval Package for XALKORI. The Drug Approval Package indicates that Pfizer switched the focus of clinical development of crizotinib as a drug from targeting the MET

receptor, to targeting the ALK receptor. To this end, Pfizer amended its IND to include studies of ALK-positive lung cancer patients, and met several times with the FDA in this regard. Pfizer also obtained and submitted specific data under its filed NDA in order to obtain accelerated approval of crizotinib in XALKORI as a drug for the treatment of ALK-positive (which includes EML-ALK) advanced NSCLC in patients. Further details in this regard are provided below.

44. A passage in the section from the FDA Drug Approval Package for XALKORI entitled, "Clinical Pharmacology and Biopharmaceutics Review(s)" states: "Although initially developed against c-MET, Crizotinib showed pre-clinical and clinical activity against ALK. Due to ALK emergence as a potentially relevant oncogenic target at the time, Crizotinib development was shifted to target ALK+ NSCLC." It is thus clear that in 2007 the newly recognized *EML4-ALK* receptor became a new target Receptor and crizotinib became the subject of a new drug discovery program targeting the *EML4-ALK* receptor for treating NSCLC.

E. Relationship Between XALKORI And NYU's Know-How Developed During The NYU Research Project

45. XALKORI is a SUGEN Product according to The Agreement that was developed based on the Receptor target *EML4-ALK* for treatment of NSCLC. *EML4-ALK* is a Receptor target which was not adopted into drug discovery by Sugen prior to 1999, when Sugen was acquired by Pharmacia, and was not adopted into drug discovery prior to Pfizer's acquisition of Pharmacia in 2003.

46. NYU's "Research Technology" under The Agreement is illustrated above, in paragraphs 27-37.

47. Crizotinib and its development was "derived from or based on" NYU's "Research Technology" under The Agreement as is well documented and illustrated below:

- In 2003, an article by scientists at Sugen reported the identification of oxindole inhibitors of c-MET and later the identification of an oxindole/indolinone lead compound SU11274, by using a MET kinase homology model developed from NYU's FGFR1 kinase, and a high-throughput screening assay. Xueyan Wang *et al.*, *Potent and selective inhibitors of the Met [hepatocyte growth factor/scatter factor (HGF/SF) receptor] tyrosine kinase block HGF/SF-induced tumor cell growth and invasion*, 2 Mol. Cancer Ther. 1085-92 (2003) ("Sugen/Wang article").

- The Sugen/Wang article cites to the NYU work reported in the 1997 Mohammadi *et al. Science* paper, and to NYU's data for the structure of FGFR1 kinase with ATP analog bound, as reported in NYU's 1996 *Cell* paper. Sugen would later characterize SU11274 as a "breakthrough" in the rational targeting of the MET receptor in cancer therapy, reporting that SU11274 was "the first small molecule that was designed to specifically inactivate the MET kinase function." S. Berthou *et al.*, *The Met kinase inhibitor SU11274 exhibits a selective inhibition pattern toward different receptor mutated variants*, 23 Oncogene 5387-93, 5391 (July 8, 2004).

- The chemical structure of SU11274 was later optimized by Sugen to generate PHA-665752, another oxindole/indolinone compound. James G. Christensen *et al.*, *A Selective Small Molecule Inhibitor of c-Met Kinase Inhibits c-Met-Dependent Phenotypes in Vitro and Exhibits Cytoreductive Antitumor Activity in Vivo*, 63 Cancer Research 7345-55 (Nov. 1, 2003).

- PHA-665752 was further optimized to generate crizotinib (PF-02341066). It was developed from studying and comparing the binding of Sugen's various candidate compounds in complex with the FGFR1 kinase crystals, with MET homology models

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derived from NYU's FGFR1 kinase crystals, and later in complex with a Met kinase crystal. *See, e.g.*, J. Jean Cui *et al*., *Structure Based Drug Design of Crizotinib (PF-02341066), a Potent and Selective Dual Inhibitor of Mesenchymal—Epithelial Transition Factor (c-MET) Kinase and Anaplastic Lymphoma Kinase (ALK)*, 54 J Med Chem. 6342-63 (2011), 54 J Med Chem. 6342-63 (2011); Xueyan Wang *et al.*, *Development of the First Generation c-Met Kinase Inhibitors: Beginning of a Path to a New Treatment for Cancer*, 10(11) Mol. Cancer Ther. 2022-23 (Nov. 2011). Therefore, information and direction from NYU's drug discovery process led to the rational deconstruction of the indolinone drug scaffold (oxindole core) of Sugen's earlier lead compounds, and the design of the benzyloxypyridine compound, crizotinib.

48. NYU's pioneering work with FGFR1 kinase crystals provided a key research tool which provided Sugen with a vastly accelerated drug development pathway that led to XALKORI. Thus, as contemplated under The Agreement, NYU should be compensated accordingly. *See, e.g.*, J. Schlessinger, The Scientist Magazine (Apr. 11, 2005).

F. <u>The Effect Of Pfizer's Activities On NYU</u>

49. While NYU is recognized as a prestigious educational and research institution, it nevertheless depends on accurate and continued acknowledgments of its ongoing scientific contributions and successes to maintain its reputation and to compete with other institutions for grants and funding by third parties.

50. NYU is a party to many contracts with third parties seeking NYU's assistance and collaboration for research, development and investigations in many technical fields, including those of drug discovery, drug research and development, and the sciences relating thereto. NYU considers its reputation to be essential to the success of the University and to be of great pecuniary value.

51. In the association and relationship of a third party with NYU, NYU provides to such third party, a meaningful value, credibility and prestige, which can have pecuniary advantage where such third party seeks its own funding and support, or where results or conclusions reached based on the association with NYU are judged or assessed for reliability, credibility, correctness or significance.

52. To demonstrate its successful reputation to the public, NYU publicly displays and discloses its accomplishments and successes based on its endeavors, programs and projects, including those that proceed in collaboration with or association with third parties. Illustrative of NYU's promotion of its research accomplishments is NYU's webpage entitled "Success Stories." http://www.nyu.edu/about/university-initiatives/entrepreneurship-at-nyu/inspire/success-stories.html. On this website, NYU provides examples of scientific achievements made by NYU's scientists which have resulted in significant successes. NYU's successful research collaboration with Sugen/Pfizer is specifically mentioned in the disclosure of Pfizer's FDA-approved drug product SUTENT (which contains the chemical substance referred to as SU11248, or sunitinib), which is described therein as being a first-in-class cancer drug which resulted from collaboration with Sugen. With respect to SUTENT, Pfizer's acknowledgment of NYU's role in developing the substance which is the active ingredient in this "SUGEN Product" by payment of royalties to NYU under The Agreement permitted NYU to include SUTENT in its webpage of "success stories."

53. Such "success stories," statements of praise, and acknowledgments of NYU's contributions by its research partners are considered to be important and valuable assets of NYU and important bases for attracting other research partners as well as third parties willing to fund and support future endeavors, programs and projects involving NYU's resources, including its

faculty, students, and personnel in the future. Such "success stories" add to NYU's reputation and provide a basis for attracting new and high quality faculty and students to NYU, which is vital to maintain the reputation and prestige of the University.

Pfizer's Breach Of The Agreement

54. NYU repeats and realleges the preceding allegations as though fully set forth herein.

55. In a letter from Pfizer's Vice-President Patricia S. Andrews to NYU dated February 7, 2006, Pfizer acknowledged that The Agreement was in force, that Pfizer was subject to its terms, and that Pfizer was assuming the obligations of Sugen under The Agreement for paying royalties. In that letter, Pfizer agreed to pay royalties to NYU based on Pfizer's new drug product SUTENT. Pfizer has continued to make royalty payments to NYU based on sales of SUTENT.

56. NYU is entitled to a payment of at least 2.5% royalties for sales of XALKORI under Section 9 of The Agreement, entitled, "SUGEN Ownership Change." In relevant part, Section 9 reads:

> SUGEN Products that are developed based on Receptor targets which were not adopted into drug discovery at the time of the effective date of such acquisition, merger, or joint venture shall be subject to a). a royalty of 2.5% on Net Sales of SUGEN, and/or Corporation Entity, which may be offset by 50% of the royalties paid by SUGEN to third parties (other than MPG), provided that the royalties due to NYU shall not be less than 1.5% of Net Sales of SUGEN and/or Corporation Entity and b). 10% of License Revenues with respect to any SUGEN Product, provided that with respect to such SUGEN Product there exists a Patentable Invention with respect to such target and/or its utility which is derived from or based on the Research Technology, and provided further that such SUGEN Product shall include a product irrespective of whether an IND application is filed with respect thereto within 4 years from the end of the Research Period, or not.

57. XALKORI is a SUGEN Product as defined in The Agreement, that was developed based on the Receptor target ALK, more particularly, EML4-ALK. ALK, which

includes EML4-ALK, are Receptor targets which were not adopted into drug discovery by Sugen at the time of the effective date that Sugen was acquired by Pharmacia in 1999, or at the time Pfizer acquired Pharmacia in 2003. Therefore, under The Agreement, XALKORI is a "SUGEN Product" that was developed to target the receptor *EML4-ALK*, which was not a receptor target that had been adopted into drug discovery as of the date that Sugen was acquired by Pharmacia and by Pfizer.

58. Patentable Inventions with respect to ALK and EML4-ALK as a Receptor target and/or its utility exist and include at least the patents listed by Pfizer in the Orange Book for XALKORI. Illustrative of the intentions of both NYU and Sugen with regard to the breadth of the scope of the Agreement, is the definition of "Patentable Invention" referred to in Section 9, which is not restricted to only those patents claiming inventions made by employees or students of NYU. Rather, "Patentable Invention" is defined broadly in Section 1(l) as "a claim in an issued, unexpired patent that has not been held invalid by any final decision of a court in the relevant country. It also includes claims in a pending application that has priority from a specification filed less than seven years previous."

59. The patents listed in the Orange Book for XALKORI include information derived from or based on NYU's "Research Technology." Under Section 1(o) of The Agreement, "Research Technology" includes all "NYU Patents and NYU Know-How." "NYU Know-How" is broadly defined in Section 1(i) of The Agreement as any information and materials discovered, developed, or acquired by or on behalf of NYU during the term and in the course of the "NYU Research Project", defined in Section 1(k) of the Agreement.

60. XALKORI is a "SUGEN Product" within the meaning of Section 9 of The Agreement, because "SUGEN Product" is defined in Section 1(q) to mean, *inter alia*,

> any product for the diagnosis, treatment or prevention of human disease which contains or comprises:
>
> i) any Receptor (as hereinafter defined); and/or
>
> ii) any substance which activates or prevents activation or otherwise modulates activation of a Receptor; and/or
>
> iii) any substance which induces, prevents or otherwise modulates intracellular activity of either the activated or resting Receptor; and/or
>
> iv) any substance which otherwise physically interacts with a Receptor . . .

XALKORI is an FDA-approved drug product for the treatment of NSCLC in patients expressing a mutated ALK receptor. ALK receptors are tyrosine kinase receptors, which are included in the definition of "Receptors" under Section 1(m) of The Agreement.

61. As illustrated above, the scientific literature authored by Sugen and Pfizer scientists establish that XALKORI was derived from or based on NYU Patents or NYU Know-How. For example, XALKORI (containing the active ingredient known as crizotinib, PF-02341066) is a benzyloxypyridine that was derived from PHA-66572, an oxindole inhibitor of the MET receptor, which was identified at Sugen. J. Jean Cui *et al*., *Structure Based Drug Design of Crizotinib (PF-02341066), a Potent and Selective Dual Inhibitor of Mesenchymal—Epithelial Transition Factor (c-MET) Kinase and Anaplastic Lymphoma Kinase (ALK)* 54 J Med Chem. 6342-63 (2011), 54 J Med Chem. 6342-63 (2011); 10(11) Xueyan Wang *et al.*, *Development of the First Generation c-Met Kinase Inhibitors: Beginning of a Path to a New Treatment for Cancer*, Mol. Cancer Ther. 2022-23 (Nov. 2011). PHA-66572 was derived from SU11274, Xueyan Wang *et al*., 2 Mol Cancer Ther. 1085-92 (2003), whose development relied on a homology model of MET kinase based on NYU's pioneering crystallographic studies of the tyrosine kinase domain of FGFR 1, Mohammadi *et al.*, *Structure*

of the FGF Receptor Tyrosine Kinase Domain Reveals a Novel Autoinhibitory Mechanism, 86 Cell 577-87 (1996); Mohammadi *et al.*, *Structures of the Tyrosine Kinase Domain of Fibroblast Growth Factor Receptor in Complex with Inhibitors*, 276(5314) Science 955-60 (1997). (*See also* Mohammadi *et al.*, U.S. Patent No. 5,942,428, entitled "Crystals of the tyrosine kinase domain of non-insulin receptor tyrosine kinases.") Thus, in accordance with The Agreement, XALKORI is a "SUGEN Product" which is "derived from or based on the Research Technology" which includes at least "NYU Know-How."

62. Under Section 9 of The Agreement, royalty payments have been due to NYU based on 2.5% of Pfizer's sales of XALKORI, but Pfizer has failed to pay to NYU any royalty payments for Pfizer's sales of XALKORI.

63. By letter dated February 28, 2013, NYU advised Pfizer that it was required to make payments of 2.5% royalty to NYU for sales of XALKORI under Section 9 of The Agreement. Notwithstanding Pfizer's receipt of the foregoing letter, including the basis for the contractual requirement by Pfizer to pay royalties based on its sales or License Revenues with respect to XALKORI, Pfizer has still failed to comply with its royalty obligations.

64. Pfizer's failure to comply with its royalty obligations under The Agreement constitutes a material breach of the Agreement.

65. NYU has been damaged by Pfizer's breach of The Agreement in failing to pay NYU the royalties due and owing under Section 9 of The Agreement based on Pfizer sales of the XALKORI drug product.

WHEREFORE, Plaintiff NYU requests that the Court enter judgment against Defendant Pfizer and in favor of NYU in an amount to be determined at trial, together with interest and costs, and for such other and further relief as the Court deems just and proper.

Respectfully submitted,

Date: October 16, 2013
New York, New York

By: /s/ Michael S. Oberman

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