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8 UNITED STATES DISTRICT COURT  
9  
10 NORTHERN DISTRICT OF CALIFORNIA

11 In re RIGEL PHARMACEUTICALS, INC. )  
SECURITIES LITIGATION )

No. 3:09-cv-00546-JSW

) CLASS ACTION

12 \_\_\_\_\_ )  
13 This Document Relates To: )

14 ALL ACTIONS. )

) CONSOLIDATED AMENDED  
) COMPLAINT FOR VIOLATIONS OF THE  
) FEDERAL SECURITIES LAWS

) DEMAND FOR JURY TRIAL

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1 **I. INTRODUCTION**

2 1. This is a securities class action on behalf of all persons who acquired the securities of  
3 Rigel Pharmaceuticals, Inc. (“Rigel” or the “Company”) between December 13, 2007 and February  
4 3, 2009 (the “Class Period”), including all persons who acquired the common stock of Rigel  
5 pursuant and/or traceable to a false and misleading registration statement and prospectus  
6 (collectively, the “Registration Statement”) issued in connection with the Company’s February 2008  
7 offering (the “Offering”). This action asserts strict liability claims under the Securities Act of 1933  
8 (“1933 Act”) and fraud claims under the Securities Exchange Act of 1934 (“1934 Act”) against  
9 Rigel, its senior insiders and the investment banks which underwrote the Offering (collectively,  
10 “defendants”).

11 2. Rigel is a clinical-stage drug development company that discovers and develops  
12 novel, small-molecule drugs for the treatment of inflammatory/autoimmune diseases and cancer, as  
13 well as viral and metabolic diseases. The Company was founded in 1996 and is based in South San  
14 Francisco, California.

15 3. Before the Class Period, Rigel was developing a new drug, R788, for the treatment of  
16 rheumatoid arthritis (“RA”), an autoimmune disease characterized by chronic inflammation that  
17 affects multiple tissues but typically produces its most pronounced symptoms in the joints. R788  
18 was the Company’s lead product candidate, and Rigel designed, paid for and conducted a Phase IIa  
19 clinical trial to evaluate the safety and preliminary clinical efficacy of R788 in patients with active  
20 RA despite methotrexate therapy (the “Phase IIa RA clinical trial”). Rigel described the Phase IIa  
21 RA clinical trial as a multi-center, randomized, double-blind, placebo-controlled, ascending-dose  
22 study involving 189 patients in the U.S. and Mexico. The patients were divided into 3  
23 approximately equal-size cohorts receiving 50, 100 or 150mg of R788 twice a day. The Phase IIa  
24 RA clinical trial was conducted over a 12-week treatment period in patients who had RA for at least  
25 12 months.

26 4. On December 13, 2007, Rigel issued a press release and held a conference call  
27 (attended by defendants James M. Gower (“Gower”), Elliott B. Grossbard (“Grossbard”), Donald G.  
28 Payan (“Payan”), Raul R. Rodriguez (“Rodriguez”), and Ryan D. Maynard (“Maynard”)) touting the

1 purportedly positive summary results of the recently completed Phase IIa RA clinical trial. The  
2 press release was an exhibit to a Form 8-K filed with the United States Securities and Exchange  
3 Commission (“SEC”) the same day. In the press release, titled “Rigel’s R788 Demonstrates  
4 Significant Improvement in Rheumatoid Arthritis in Phase 2 Clinical Study,” the Company  
5 presented a single set of data for all patients which indicated an ascending dose response (*i.e.*, the  
6 higher the dose a patient received, the greater the improvement in their symptoms) and efficacy  
7 results purportedly demonstrating statistically significant improvement over placebo. Defendants  
8 also purportedly reported safety data regarding R788’s effect on blood pressure, liver enzymes,  
9 neutropenia, diarrhea and gastrointestinal side effects.

10 5. The purportedly positive results of the Phase IIa RA clinical trial reported in the  
11 December 13, 2007 press release and conference call were repeated to the market in reports issued  
12 by analysts following the Company. The analysts highlighted the better-than-expected efficacy  
13 results, the ascending dose response and the lack of dose-dependent hypertension. The analysts  
14 increased their price target for the Company’s stock and reported that they expected the price of the  
15 Company’s stock to increase due to the positive reported results. They were right. In response to  
16 the announcement of the summary results of the Phase IIa RA clinical trial, Rigel’s common stock  
17 price more than tripled in one day, from \$8 per share to \$25.95.

18 6. The results of the Phase IIa RA clinical trial, however, were neither “statistically  
19 significant,” “impressive,” a “major milestone” nor did they demonstrate that R788 was “highly  
20 effective” as defendants falsely led investors to believe. Properly applying basic principals of  
21 statistics, the results of Rigel’s Phase IIa RA clinical trial were not statistically significant and  
22 demonstrated that the drug was not as effective as defendants claimed it to be.

23 7. Plaintiffs have retained the services of Stanford University Professor (Emeritus) of  
24 Biostatistics, Dr. Daniel A. Bloch, who has published 196 original articles in peer-reviewed journals,  
25 including co-authoring over 30 articles which appeared in *Arthritis and Rheumatism*. Dr. Bloch  
26 reviewed defendants’ December 13, 2007 press release, the per-country data disclosed on October  
27  
28

1 27, 2008 and the subsequent publication of the Phase IIa RA clinical trial in the November 2008  
2 issue of *Arthritis and Rheumatism*.<sup>1</sup> He summarized his findings as follows:

3 ***When the data is properly analyzed, all of the nominal p-values are higher***  
4 ***[i.e., show less statistical significance] than those reported with the December 13,***  
5 ***2007 press release . . . . The comparisons of both 100mg and 150mg groups to***  
***Placebo for the primary outcome variable, the ACR20, with or without accounting***  
***for multiple comparisons, are not statistically significant.***<sup>2</sup>

6 Bloch Decl. at 12-13.

7 8. In addition to defendants' claims that R788 exhibited statistically significant results  
8 (when it did not), defendants presented only the combined data for U.S. and Mexico patients in  
9 Rigel's December 13, 2007 press release. The presentation of the combined data concealed from  
10 investors contained material information including: (i) the existence of a country interaction so  
11 substantial that the placebo in Mexico was more effective than any dose in the U.S. at ACR20;<sup>3</sup> (ii)  
12 that the distribution by dose in the Phase IIa RA clinical trial was not balanced among the U.S. and  
13 Mexico patients *i.e.*, the low dose was tested in the U.S. and high dose was tested in Mexico; and  
14 (iii) there was no ascending dose response.

15 \_\_\_\_\_  
16 <sup>1</sup> The Declaration of Dr. Daniel A. Bloch ("Bloch Decl."), dated January 23, 2010, including  
17 his curriculum vitae, is attached hereto as Exhibit A. Plaintiffs have incorporated Dr. Bloch's  
18 analysis and conclusions that defendants' statements regarding the statistical significance and  
efficacy of R788 violated basic statistical principals and were thus false and misleading herein.

19 <sup>2</sup> A "p-value" is a statistical measure of the probability that a difference between groups in a  
20 clinical trial happened by chance. In this case, the lower the p-value, the more likely it is that the  
21 difference between the dosed and placebo groups was caused by R788, rather than by chance alone.  
22 Statistical significance consisting of a p-value of 0.05 or below has traditionally been considered  
23 convincing evidence in clinical trials and by the U.S. Food and Drug Administration. A p-value of  
24 0.05 means that there is a 5% likelihood that the observed result occurred by chance alone, and  
25 conversely a 95% likelihood that the improvement was attributable to the drug. Or in other words, if  
the drug performed no better than placebo, one would expect to see such results in only 5 out of 100  
trials. For p-values less than 0.05, the conclusion is that the result is "statistically significant" and  
thus the patients' improvement is attributed to the function of R788. A p-value of 0.06 (*i.e.*, 6%), is  
considered too large to claim statistically significant improvement in the context of clinical trials; it  
is "statistically insignificant." Very large p-values, for example, p=.20, are statistically insignificant  
and indicate that the observed result would occur as a function of chance alone 20% of the time.

26 <sup>3</sup> ACR20, ACR50 and ACR70 refer to measures of improvement under the American College  
27 of Rheumatology ("ACR") criteria. To meet ACR criteria, a patient must show either 20, 50 or 70%  
28 improvement in tender or swollen joint counts and at least the same level of improvement in 3 of 5  
other measures of symptoms. *See* ¶52.

1           9.       Defendants’ statements about the safety results of the Phase IIa RA clinical trial were  
2 also materially false and misleading. Defendants knew, but failed to disclose, that (i) there was a  
3 dose-dependent increase in average systolic blood pressure of 3-5mm Hg in 100mg patients, and 8-  
4 9mm Hg in the 150mg patients; (ii) 5 patients (not 2, as reported on December 13, 2007)  
5 experienced hypertension, with blood pressure increases as high as 20-30mm Hg; (iii) hypertension  
6 was one of the two most common clinically meaningful drug related adverse events; (iv) 9 patients  
7 (not 3, as reported on December 13, 2007) experienced increased liver enzymes compared to patients  
8 taking the placebo; (v) 20 patients (not 15, as reported on December 13, 2007) experienced  
9 neutropenia; (vi) 34 patients (not 15, as reported on December 13, 2007) experienced diarrhea; and  
10 (vii) 35 patients (not 15, as reported on December 13, 2007) experienced upper gastrointestinal side  
11 effects. Rigel, Gower, Grossbard, Payan, Rodriguez and Maynard knew that these undisclosed  
12 toxicities could undermine the Company’s investment thesis, *i.e.*, partnership, and present hurdles to  
13 regulatory approval.

14           10.       Rigel, Gower, Grossbard, Payan, Rodriguez and Maynard also knew it was important  
15 to report the positive results for the R788 Phase IIa RA clinical trial for several reasons: R788 was  
16 the Company’s lead product candidate; the worldwide market for RA drugs exceeded \$13 billion in  
17 2007; and Rigel was racing to develop the first oral pill for RA before Pfizer Inc. (“Pfizer”).

18           11.       Rigel, Gower, Grossbard, Payan, Rodriguez and Maynard also knew that the  
19 Company needed to raise additional funds and that the false and misleading statements about the  
20 Phase IIa RA clinical trial – and the inflated stock price those false and misleading statements caused  
21 – would allow Rigel to raise substantially greater funds than if the adverse information were  
22 disclosed.

23           12.       Moreover, Rigel, Gower, Grossbard, Payan, Rodriguez and Maynard knew that the  
24 Company would become insolvent if it did not raise additional capital. The Company reported net  
25 losses every year since its December 2000 initial public offering (“IPO”). After reporting a net loss  
26 of \$74.3 million in 2007, Rigel reported just \$44.5 million of cash and \$82.2 million of capital at  
27 December 31, 2007. Additional losses were projected for 2008 and Rigel reported a \$132.3 million  
28

1 net loss in 2008 which would have rendered the Company insolvent if it did not raise additional  
2 capital in February 2008.

3 13. Rigel, Gower, Grossbard, Payan, Rodriguez and Maynard further knew that the  
4 Company's stock price would likely decline if they disclosed the adverse information about the  
5 Phase IIa RA clinical trial because the Company's stock price declined 40% after it reported the  
6 results of Phase II clinical trial of R788 in patients with immune thrombocytopenic purpura ("ITP")  
7 (the "Phase II ITP clinical trial") on November 9, 2007. Analysts following the Company reported  
8 the price decline was due to concerns about R788's adverse event profile, including, *inter alia*, blood  
9 pressure and gastrointestinal side effects.

10 14. Gower, Grossbard, Payan, Rodriguez and Maynard also knew that they would receive  
11 higher salaries, bonuses and stock option awards, and that the value of their existing stock options  
12 would increase substantially, if Rigel reported positive results from the Phase IIa RA clinical trial.  
13 As detailed below, the compensation of Gower, Grossbard, Payan, Rodriguez and Maynard was  
14 directly dependent on the results of the Phase IIa RA clinical trial, and they each received salary  
15 increases, bonuses and stock awards at the end of 2007 due to the reported positive results of the  
16 Phase IIa RA clinical trial and the inflated price of the Company's stock caused by their false and  
17 misleading statements about the Phase IIa RA clinical trial.

18 15. Defendants took advantage of the inflated stock price caused by their false and  
19 misleading statements and omissions on December 13, 2007, by causing Rigel to issue 5 million  
20 shares of Rigel stock at \$27 per share in an offering that raised \$135 million. On January 24, 2008,  
21 Rigel filed with the SEC an S-3ASR Registration Statement and Form 424B3 Preliminary  
22 Prospectus for the Offering, which incorporated by reference the materially false and misleading  
23 December 13, 2007 Form 8-K. On February 1, 2008, Rigel filed with the SEC a Form 424B5  
24 Prospectus that also incorporated by reference the December 13, 2007 press release and repeated the  
25 false and misleading summary results of the Phase IIa RA clinical trial. Absent the false and  
26 misleading statements about the results of the Phase IIa RA clinical trial, Rigel's stock price would  
27 not have increased and the Company would not have been able to complete the Offering at \$27 per  
28 share.

1           16.     The four Underwriter Defendants (as defined *infra* ¶¶43-47) were paid more than \$7  
2 million to underwrite the Offering and failed to require disclosure of the adverse information about  
3 the Phase IIa RA clinical trial. Public investors relied on the Underwriter Defendants to conduct a  
4 reasonable investigation and to obtain and verify the information contained in the Registration  
5 Statement and to make sure essential facts about the Company were disclosed. Indeed, the  
6 Underwriter Defendants had access to the adverse information at a critical time in Rigel’s corporate  
7 life – when it was seeking to raise capital. The Underwriter Defendants either knew about the  
8 adverse information and failed to require its disclosure or did not know by failing to conduct a  
9 reasonable investigation and independently verifying the representations in the Registration  
10 Statement. Either way, the Underwriter Defendants failed to meet their “gatekeeper” function of  
11 protecting investors.

12           17.     After the Offering, Rigel, Gower and Rodriguez continued to tout the purportedly  
13 positive results of the Phase IIa RA clinical trial but concealed the adverse information related to the  
14 trial. On February 11, 2008, Gower touted the results of the Phase IIa RA clinical trial during the  
15 BIO CEO & Investor Conference. On July 8, 2008, Rodriguez touted the results of the Phase IIa RA  
16 clinical trial of R788 during the Collins Stewart 4th Annual Growth Conference. As a result, the  
17 Company’s stock price continued to trade at artificially inflated prices.

18           18.     On October 27, 2008, Rigel presented additional results of the Phase IIa RA clinical  
19 trial at a meeting of the ACR and on an investor conference call. Those results included the results  
20 on a per-country basis and the adverse safety information omitted from the Company’s December  
21 13, 2007 press release, as well as from the Registration Statement and the presentations on February  
22 11, 2008 and July 8, 2008. When this adverse information was finally disclosed, Rigel’s stock price  
23 plunged 38% in a single day, from \$14.41 to \$8.84.

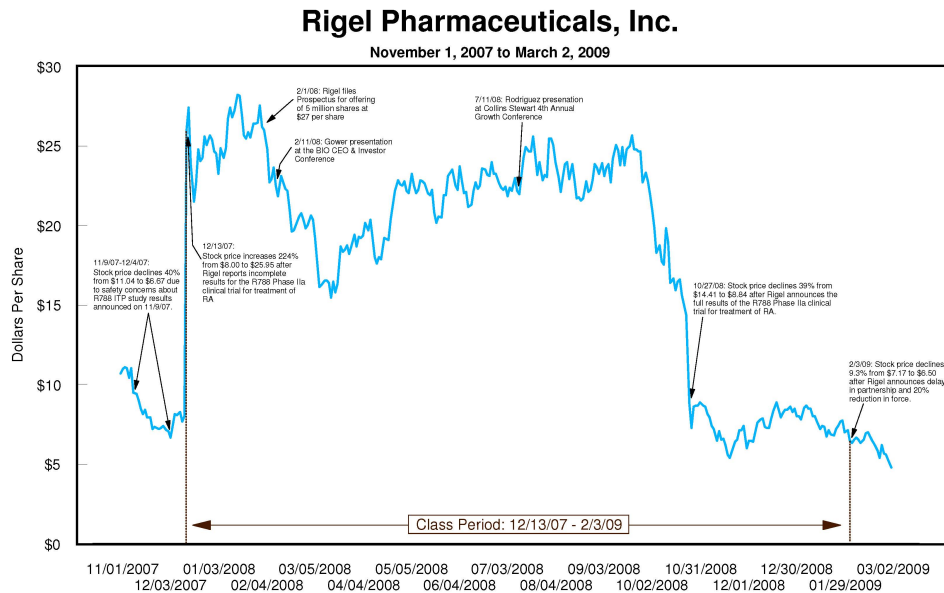
24           19.     Analysts following the Company issued reports in which they downgraded the  
25 Company’s stock and wrote that the adverse information about the Phase IIa RA clinical trial of  
26 R788 raised significant concerns about the efficacy and safety of the drug, whether Rigel would be  
27 able to close a partnership deal and the ultimate commercialization of R788. Gower stated that Rigel  
28

1 remained in detailed licensing discussions with several potential partners and expected a lucrative  
2 partnership in early 2009.

3 20. On February 3, 2009, Rigel announced that it would delay partnership discussions  
4 regarding R788 until after results from its Phase IIb clinical studies were available and that the  
5 Company laid off 36 employees, or 20% of its workforce. The price of the Company's stock  
6 declined 9.3%, from \$7.17 on February 2, 2009 to \$6.50 on February 3, 2009.

7 21. On July 23, 2009, Rigel announced that one of the Phase IIb clinical studies failed to  
8 meet efficacy endpoints because patients did not report statistically significant higher ACR response  
9 rates than the placebo group. The Company also stated that hypertension and diarrhea were the most  
10 common clinically meaningful drug related adverse events. Rigel's stock price dropped from a high  
11 of \$14.60 on July 22, 2009 to a low of \$9.31 on July 24, 2009, a drop of 36.2%.

12 22. The following chart (which is also attached hereto) illustrates how defendants' false  
13 and misleading statements and omissions about the Phase IIa RA clinical trial caused the price of  
14 Rigel's stock to be artificially inflated and how class members were damaged when the results of the  
15 trial – and its impact on Rigel's current and future business prospects – were revealed to the market:



1 **II. JURISDICTION AND VENUE**

2 23. The claims alleged herein arise under §§10(b) and 20(a) of the 1934 Act (15 U.S.C.  
3 §§78j(b) and 78t(a)) and Rule 10b-5 promulgated thereunder by the SEC (17 C.F.R. §240.10b-5),  
4 and §§11, 12(a)(2) and 15 of the 1933 Act (15 U.S.C. §§77k, 77l(a)(2) and 77o).

5 24. This Court has jurisdiction over the subject matter of this action pursuant to 28 U.S.C.  
6 §§1331, 22 of the 1933 Act and §27 of the 1934 Act.

7 25. Venue is proper pursuant to §22 of the 1933 Act and §27 of the 1934 Act. The  
8 Company is located in this District, and the false and misleading statements were made in this  
9 District.

10 **III. PARTIES**

11 26. Lead plaintiff Inter-Local Pension Fund GCC/IBT acquired the common stock of  
12 Rigel pursuant or traceable to the Offering as described in the attached certification and was  
13 damaged thereby.

14 27. Defendant Rigel is headquartered in South San Francisco, California. The Company  
15 was incorporated in Delaware in 1996 and completed its IPO in December 2000, issuing 5 million  
16 shares at \$7 per share. In 2Q07, Rigel completed a public offering of 5.75 million shares of common  
17 stock at \$9.75 per share that raised net proceeds of \$52.3 million. The Company completed another  
18 public Offering in February 2008, issuing 5 million shares at \$27 per share.

19 28. Its stock trades in an efficient market on the NASDAQ under the symbol "RIGL."

20 29. Defendant Gower was, at all relevant times, Chairman of the Board and Chief  
21 Executive Officer ("CEO") of the Company. Gower made false and misleading statements about the  
22 results of the Phase IIa RA clinical trial in the December 13, 2007 press release, during the  
23 Company's December 13, 2007 conference call and during the February 11, 2008 conference call.  
24 He also signed or authorized the signing of the false and misleading Registration Statement for the  
25 Company's February 2008 Offering.

26 30. Defendant Maynard was, at all relevant times, Chief Financial Officer ("CFO") of the  
27 Company. Maynard attended the December 13, 2007 conference call, during which defendants  
28 Gower and Grossbard made false and misleading statements about the results of the Phase IIa RA

1 clinical trial, and signed or authorized the signing of the false and misleading Registration Statement  
2 for the Company's February 2008 Offering.

3 31. Defendant Payan was, at all relevant times, Executive Vice President of Discovery  
4 and Research of the Company. He is a medical doctor and was a co-founder of the Company. Payan  
5 attended the December 13, 2007 conference call, during which defendants Gower and Grossbard  
6 made false and misleading statements about the results of the Phase IIa RA clinical trial, and signed  
7 or authorized the signing of the false and misleading Registration Statement for the Company's  
8 February 2008 Offering.

9 32. Defendant Rodriguez was, at all relevant times, Executive Vice President and Chief  
10 Operating Officer ("COO") of the Company. Rodriguez attended the December 13, 2007 conference  
11 call, during which defendants Gower and Grossbard made false and misleading statements about the  
12 results of the Phase IIa RA clinical trial, and made false and misleading statements about the results  
13 of the Phase IIa RA clinical trial during the July 8, 2008 Collins Stewart 4th Annual Growth  
14 Conference.

15 33. Defendant Grossbard was, at all relevant times, Executive Vice President and Chief  
16 Medical Officer of the Company. Grossbard made false and misleading statements about the results  
17 of the Phase IIa RA clinical trial in the December 13, 2007 press release and during the Company's  
18 December 13, 2007 conference call. He was a contributing author for an article on the Phase IIa RA  
19 clinical trial that was published in *Arthritis & Rheumatism*, Vol. 58, No. 11 (Nov. 2008). According  
20 to the article, Grossbard participated in the design of the Phase IIa RA clinical trial, had full access  
21 to the data from the trial, analyzed and interpreted the data from the study, was a member of the  
22 publication committee, prepared the manuscript and vouched for the completeness of the data and  
23 analysis.

24 34. The defendants referenced above in ¶¶29-33 are herein referred to as the "Officer  
25 Defendants."

26 35. Defendant Jean Deleage ("Deleage") was, at all relevant times, a director of the  
27 Company. Deleage signed or authorized the signing of the false and misleading Registration  
28 Statement for the Company's February 2008 Offering.

1           36. Defendant Bradford S. Goodwin (“Goodwin”) was, at all relevant times, a director of  
2 the Company. Goodwin signed or authorized the signing of the false and misleading Registration  
3 Statement for the Company’s February 2008 Offering.

4           37. Defendant Gary A. Lyons (“Lyons”) was, at all relevant times, a director of the  
5 Company. Lyons signed or authorized the signing of the false and misleading Registration  
6 Statement for the Company’s February 2008 Offering.

7           38. Defendant Walter H. Moos (“Moos”) was, at all relevant times, a director of the  
8 Company. Moos signed or authorized the signing of the false and misleading Registration Statement  
9 for the Company’s February 2008 Offering.

10           39. Defendant Hollings C. Renton (“Renton”) was, at all relevant times, a director of the  
11 Company. Renton signed or authorized the signing of the false and misleading Registration  
12 Statement for the Company’s February 2008 Offering.

13           40. Defendant Peter S. Ringrose (“Ringrose”) was, at all relevant times, a director of the  
14 Company. Ringrose signed or authorized the signing of the false and misleading Registration  
15 Statement for the Company’s February 2008 Offering.

16           41. Defendant Stephen A. Sherwin (“Sherwin”) was, at all relevant times, a director of  
17 the Company. Sherwin signed or authorized the signing of the false and misleading Registration  
18 Statement for the Company’s February 2008 Offering.

19           42. The defendants referenced above in ¶¶29-33, 35-41 are referred to herein as the  
20 “Individual Defendants.”

21           43. Defendant Credit Suisse Securities (USA) LLC (“Credit Suisse”) operates as an  
22 investment bank in the U.S. Its businesses include securities underwriting, sales and trading,  
23 investment banking, private equity, alternative assets, financial advisory services, investment  
24 research, and asset management. Credit Suisse acted as an underwriter in connection with the  
25 Offering.

26           44. Defendant Oppenheimer & Co. Inc. (“Oppenheimer”) is an investment bank and full-  
27 service investment firm. Oppenheimer acted as an underwriter in connection with the Offering.

1           45. Defendant Thomas Weisel Partners LLC (“Thomas Weisel”) is an investment bank  
2 founded in 1998 focused primarily on the growth sectors of the economy. Thomas Weisel acted as  
3 an underwriter in connection with the Offering.

4           46. Defendant Jefferies & Company, Inc. (“Jefferies”) is a full-service global investment  
5 bank and institutional securities firm focused on growing and middle-market companies and their  
6 investors. Jefferies provides clients with capital markets and financial advisory services,  
7 institutional brokerage, securities research and asset management. Jefferies acted as an underwriter  
8 in connection with the Offering.

9           47. Pursuant to the 1933 Act, the defendants referenced in ¶¶43-46 above are referred to  
10 herein as the “Underwriter Defendants.”

11           48. The Underwriter Defendants are liable for the false and misleading statements in the  
12 Registration Statement. In connection with the Offering, the Underwriter Defendants drafted and  
13 disseminated the Registration Statement and were paid over *\$7 million* in gross fees in connection  
14 therewith. The Underwriter Defendants’ failure to conduct an adequate due diligence investigation  
15 was a substantial factor leading to the harm complained of herein.

16 **IV. DEFENDANTS MAKE MATERIALLY FALSE AND MISLEADING**  
17 **STATEMENTS AND OMISSIONS ABOUT THE PHASE IIa RA**  
18 **CLINICAL TRIAL**

19 **A. Prior to the Class Period, Rigel Was Conducting a Phase IIa RA**  
20 **Clinical Trial to Evaluate the Efficacy and Safety of R788, the**  
21 **Company’s Lead Product Candidate and a Potential Treatment for**  
22 **Rheumatoid Arthritis**

23           49. In the Company’s 2007 Form 10-K, Rigel describes itself as a clinical-stage drug  
24 development company that discovers and develops novel, small molecule drugs for treatment of  
25 inflammatory/autoimmune diseases, cancer and viral diseases. In 2007, the Company was  
26 developing multiple small molecule drug candidates whose specialized mechanisms of action were  
27 intended to provide therapeutic benefits for a range of inflammatory/autoimmune diseases, as well as  
28 cancers.

          50. According to the Company’s 2007 Form 10-K, R788 was the Company’s lead  
product candidate and was being developed to treat RA, an autoimmune disease characterized by

1 chronic inflammation that affects multiple tissues but typically produces its most pronounced  
2 symptoms in the joints. The Company reported on its website that RA was a progressive disease  
3 often leading to chronic pain and severe, incapacitating disabilities by causing the body's immune  
4 system to become inflamed, which in turn destroys soft tissue and erodes bone and cartilage. Rigel  
5 also reported that RA is often progressive and debilitating and affects nearly 2.1 million people in  
6 the U.S.

7 51. Rigel reported in its 2007 Form 10-K that treatments other than R788 had significant  
8 potential side effects and other shortfalls, including gastrointestinal complications and kidney  
9 damage. Most RA patients receive multiple drugs depending on the extent and aggressiveness of the  
10 disease, and most patients also eventually require some form of disease modifying anti-rheumatic  
11 drug ("DMARD"), including methotrexate.

12 52. Rigel focused its RA program on the development of a safe oral DMARD. Prior to  
13 the Class Period, Rigel was conducting a Phase IIa RA clinical trial to evaluate the safety and  
14 preliminary clinical efficacy of R788 in patients with active RA despite methotrexate therapy. The  
15 Company described the design of the clinical study in its December 13, 2007 press release as  
16 follows:

17 The clinical trial was a multi-center, randomized, double blind, placebo  
18 controlled, ascending dose study involving 189 patients in three approximately equal  
19 size cohorts receiving 50, 100, or 150 mg po bid. Within each cohort, patients were  
20 assigned on a 3:1 basis to R788 or placebo. The clinical trial was conducted over a  
21 12-week treatment period in patients who had RA for at least 12 months. These  
22 patients had active disease despite receiving adequate stable doses of methotrexate  
23 over the preceding 6 months. All of the patients continued to receive their same  
24 stable dose of methotrexate throughout the clinical trial period and extension.  
25 Efficacy assessments for each participant were based on the American College of  
26 Rheumatology criteria, which denote at least a 20% (ACR 20) improvement, at least  
27 a 50% (ACR 50) improvement, or at least 70% (ACR 70) improvement, from the  
28 baseline assessment at the end of the 12-week treatment period. The ACR  
measurement factors include, reported physician and patient global assessment of  
disease activity, patient reported pain score, and any change in C-reactive protein  
(CRP) in the patients' blood. ***The primary efficacy endpoint for the study was the  
percent of patients who were ACR 20 responders at the end of week 12.*** Secondary  
efficacy endpoints were ACR 50 and ACR 70 scores as well as Disease Activity  
Score (DAS) at the end of week 12.

53. In the Company's September 6, 2006 press release, Rigel reported that it had initiated  
enrollment and dosing for the Phase IIa RA clinical trial. In the Company's April 11, 2007 press

1 release, Rigel reported that it had completed the 50mg dose group in the Phase IIa RA clinical trial,  
2 was enrolling patients in the 100mg dose group and expected to receive results from the completed  
3 clinical trial in the second half of 2007. In the Company’s November 6, 2007 press release, Rigel  
4 announced that it would report the results of the Phase IIa RA clinical trial by the end of the year.

5 **B. Just Prior to Class Period, Rigel’s Stock Falls When Defendants**  
6 **Reveal Blood Pressure Concerns and Detailed Safety Data in the**  
7 **Phase II Clinical Trial of R788 for Idiopathic Thrombocytopenic**  
8 **Purpura**

9 54. Just weeks before defendants announced the Phase IIa RA clinical trial results, Rigel  
10 presented results from its Phase II ITP clinical trial of R788.<sup>4</sup> On November 9, 2007, defendants  
11 published a press release which included the following:

12 Nine of the first 14 patients (64%) studied responded favorably to R788  
13 treatment. . . . The *primary side effects* were GI-related symptoms. *R788 elevated*  
14 *blood pressure in some patients* but appeared not to have significant effect on  
15 neutrophil counts.

16 55. On November 9, 2007, Adam Walsh of Jefferies described the efficacy results as  
17 “positive” and stated “There is no question that R788 has impressive efficacy for treatment of ITP, in  
18 our view.” He also noted that “key safety data” included that “R788 elevated blood pressure in 14%  
19 (2/14) of patients between 5-10mm Hg.”

20 56. After release of the Phase II ITP clinical trial results, Rigel’s stock price declined  
21 40%.

22 57. On December 4, 2007, Rigel held a conference call to discuss the results, including  
23 the side effects which were present in the Phase II ITP clinical trial. Defendants published slides,  
24 including one titled “R788 Phase 2 Trial in ITP – Safety” which provided the following information:  
25

---

26  
27 <sup>4</sup> Defendants were testing, or sought to test, R788 as a treatment for a variety of indications  
28 including RA, ITP (a bleeding disorder) lymphoma, multiple sclerosis and lupus.

Adverse Reaction	N=16
Nausea	4
Diarrhea	4
<b><i>SBP [Systolic Blood Pressure] increased by &gt; 10</i></b>	4
Drowsiness	3
Weight gain >5 kg	3
Constipation	2
<b><i>ALT &gt;2x ULN</i></b>	2
Vomiting	1
Abdominal pain	1
Headache	1

58. As the chart in ¶57 depicts, defendants' disclosures in connection with the Phase II ITP clinical trial of R788 set expectations by including the disclosure of increased blood pressure (not just hypertension) and elevations in liver enzymes ("ALT") which were twice the upper limit of normal ("2x ULN") (as opposed to ALT >3x ULN reported on December 13, 2007).

59. On a conference call the same day, Grossbard indicated that the Company would know the mean change in blood pressure in the Phase IIa RA clinical trial "within a week or two" and flagged that a mean increase in systolic blood pressure of 5mm Hg might present a problem if observed:

Finally, blood pressure. We are going to get a good fix on the general effect on blood pressure in the rheumatoid arthritis trial, because we have three doses, a placebo group, ***and a we will see what the mean change in blood pressure is, and we should know that within a week or two***, there are occasional patients in the whom an investigator reports, that the patient has had an increase in blood pressure on the drug, in the lymphoma study, and this now kind of a rumor or anecdote, it seems to occur in a few patients that pre-existing hypertension.

In the rheumatoid arthritis study, while I don't know we are still blinded, the overall incidence of that is quite low, so I don't think it's a very, very prevalent problem, ***although I can't say that at a higher dose, the mean population increase in systolic pressure won't be 5 millimeters or what, I just don't know that yet.***

**C. December 13, 2007: Defendants Present Misleading Phase IIa RA Clinical Trial Results, Falsely Claim that R788 Demonstrated Statistically Significant Efficacy Results and Failed to Disclose Material Adverse Information Concerning the Safety of R788**

60. **False Statement:** On December 13, 2007, the Company issued a press release entitled "Rigel's R788 Demonstrates Significant Improvement in Rheumatoid Arthritis in Phase 2 Clinical Study; Achieves Statistically Significant ACR20, ACR50 & ACR70 Results." The press

1 release was also an exhibit to a Form 8-K that Rigel filed with the SEC on December 13, 2007. The  
 2 release stated in part (false and misleading statements are in bold and italics):

3 Rigel Pharmaceuticals, Inc. . . . today announced that its oral syk kinase inhibitor,  
 4 ***R788 (tamatnib fosdium), has demonstrated statistically significant results in***  
 5 ***treating Rheumatoid Arthritis (RA) patients in a recently completed Phase 2***  
 6 ***clinical trial. Groups treated with R788 at 100mg and 150mg po bid (orally, twice***  
 7 ***daily), showed higher ACR20, ACR50, ACR70 and DAS28 response rates than the***  
 8 ***placebo group. The efficacy results for the 100mg and the 150mg dose groups***  
 9 ***were fairly comparable.*** Dramatically, the onset of the effect in these dose groups  
 10 occurred as early as one week after initiation of therapy. We believe that ***the***  
 11 ***significant ACR scores and good tolerability observed in this clinical trial,*** and the  
 12 further benefit of oral delivery may make R788 a favorable alternative to the  
 13 currently marketed biological agents.

14 \* \* \*

15 ***“This clinical study has shown that R788 treatment can achieve impressive***  
 16 ***ACR response rates,”*** said Elliott Grossbard, M.D., senior vice president of medical  
 17 development at Rigel. ***“In this clinical trial both the 100mg and 150mg doses***  
 18 ***improved arthritis symptoms and did so quickly. We plan to initiate the next clinical***  
 19 ***trial with R788 in RA in 2008,”*** he added.

20 ***Efficacy Results***

<b><i>Treatment Assigned</i></b>	<b><i>Number (N)</i></b>	<b><i>ACR 20 % (N)</i></b>	<b><i>ACR 50 % (N)</i></b>	<b><i>ACR 70 % (N)</i></b>	<b><i>DAS28-CRP 2.6, % (N)</i></b>
<b><i>po bid</i></b>	<b><i>(N)</i></b>	<b><i>% (N)</i></b>	<b><i>% (N)</i></b>	<b><i>% (N)</i></b>	<b><i>% (N)</i></b>
<b><i>Placebo</i></b>	<b><i>47</i></b>	<b><i>38% (18)</i></b>	<b><i>19% (9)</i></b>	<b><i>4% (2)</i></b>	<b><i>17% (8)</i></b>
<b><i>50 mg</i></b>	<b><i>46</i></b>	<b><i>32% (15)</i></b>	<b><i>17% (8)</i></b>	<b><i>2% (1)</i></b>	<b><i>20% (9)</i></b>
<b><i>100 mg</i></b>	<b><i>49</i></b>	<b><i>65% (32)</i></b> <b><i>(p=.008)</i></b>	<b><i>49% (24)</i></b> <b><i>(p=.002)</i></b>	<b><i>33% (16)</i></b> <b><i>(p&lt;.001)</i></b>	<b><i>35% (17)</i></b> <b><i>(p=.005)</i></b>
<b><i>150 mg</i></b>	<b><i>47</i></b>	<b><i>72% (34)</i></b> <b><i>(p&lt;.001)</i></b>	<b><i>57% (27)</i></b> <b><i>(p&lt;.001)</i></b>	<b><i>40% (19)</i></b> <b><i>(p&lt;.001)</i></b>	<b><i>47% (22)</i></b> <b><i>(p&lt;.001)</i></b>

21 \* \* \*

22 James M. Gower, chairman and chief executive officer of Rigel said, ***“These***  
 23 ***very important clinical trial results are a major milestone for Rigel as we establish***  
 24 ***the potential of R788 in RA and its value as an alternative to current therapies.*** In  
 25 addition, ***given these results*** and the recent results in ITP, ***we believe that R788 may***  
 26 ***be a useful drug in the treatment of autoimmune diseases.”***

27 **Safety Results**

28 ***The most common clinically meaningful adverse events noted in the clinical***  
 29 ***trial were dose-related neutropenia, mild elevations of liver function tests, and***  
 30 ***gastrointestinal (GI) side effects.*** Dose reduction (to one half the assigned dose, by  
 31 taking the drug once per day) was pre-specified in the protocol, contingent on  
 32 neutrophil counts and/or liver function tests. Notably, a vast majority of the patients

(19 out of 21) who had their dose reduced, successfully completed the clinical trial with minimal safety issues.

*The key safety results are shown in the table below:*

	<i>Placebo po BID N=47</i>	<i>50mg po BID N=46</i>	<i>100mg po BID N=49</i>	<i>150mg po BID N=47</i>
Completed Study at Reduced Dose (N)	1	0	5	13
Dropouts (N):	11	6	6	8
Withdrawn Consent	6	3	2	1
Adverse Event	2	1	3	6
Other	3	2	1	1
<i>Neutropenia (N) Requiring dose reduction</i>	<i>0</i>	<i>0</i>	<i>5</i>	<i>10</i>
<i>ALT &gt; 3XULN (N)</i>	<i>2</i>	<i>0</i>	<i>0</i>	<i>3</i>
<i>Diarrhea (N) (severity moderate or greater)</i>	<i>0</i>	<i>3</i>	<i>2</i>	<i>10</i>
<i>Upper GI side effects (N) (gastritis, nausea, dyspepsia) (severity moderate or greater)</i>	<i>2</i>	<i>1</i>	<i>2</i>	<i>12</i>
<i>Hypertension (N) (severity moderate or greater)</i>	<i>0</i>	<i>0</i>	<i>2</i>	<i>0</i>

61. On December 13, 2007, the Company also held a conference call attended by defendants Gower, Grossbard, Payan, Maynard, and Rodriguez. During the call, defendants Gower and Grossbard repeated the positive results of the Phase IIa RA clinical trial (false and misleading statements are in bold and italics):

[Gower:] We were very pleased to be able to announce ***highly statistically significant results of a Phase 2 trial of 788 in patients with rheumatoid arthritis.*** And I would like to introduce Dr. Elliot Grossbard to take us through the study results. Elliot?

\* \* \*

[Grossbard:] The efficacy results are shown in the graph on the handout that many of you may have downloaded. ***As you can see, the highly significant effect for both the ACR 20, 50, 70 and DAS28 score. The p values are uniformly less than .008, usually less than .001.*** Of note, although not included in this graph, is that the onset of the effect was within one week, and you could see significant differences between the patients at one week after the initiation of treatment.



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[Weinblatt:] *All I can tell you is that this study showed that there is a clinical effect which is significant at all major ACR response rates.*

63. The positive results of the Phase IIa RA clinical trial reported in the December 13, 2007 press release and conference call were repeated to the market in reports issued by analysts following the Company, including reports issued on December 13, 2007 by CIBC World Markets (“CIBC”) analyst Brian Abrahams, Jefferies analyst Adam A. Walsh and Credit Suisse analyst Michael Aberman, M.D.

64. Credit Suisse analyst Aberman increased the price target of Rigel stock from \$12 to \$25 and highlighted the efficacy results, including the ascending dose response, and the absence of dose dependent hypertension:

It is hard to imagine better results than Rigel achieved with R788 in RA and we think this compound has a good chance of becoming a blockbuster for autoimmune diseases. Key efficacy highlights include 1) a no effect dose, 2) a dose response between all doses, and 3) arguably the best efficacy seen in a PII RA trial . . . . Of particular note . . . [there was] *no evidence of dose dependent hypertension.*

65. Jefferies analyst Walsh increased the price target for Rigel stock from \$16 per share to \$19 per share and highlighted the statistically significant efficacy results:

***R788 Shows Home Run Efficacy and Good Tolerability in RA***

\* \* \*

Efficacy exceeded best-case expectations, with *highly statistically significant results* on all primary and secondary outcome measures at the likely go forward dose (100mg).

\* \* \*

The 100mg dose group hit the primary endpoint of ACR20 improvement at 12 weeks vs placebo as well as all secondary measures including ACR50, ACR70 and DAS28-CRP. . . . We view these efficacy results as significantly better than our and Street expectations.

66. Abrahams reported that CIBC expected upside in Rigel’s stock price because the results of the Phase IIa RA clinical trial provided “strong proof-of-concept for systemic Syk kinase inhibition in rheumatoid arthritis, and unlocks the potential for the agent to be used in other chronic autoimmune conditions as well.” Abrahams, highlighted the “dose-dependent activity” and stated that

1 On the primary endpoint, the ACR20 rate in the 100mg 2x/day group was 65%  
 2 (p=0.008), and the ACR20 rate in the highest-dose 150mg 2x/day group was 72%  
 3 (p<0.001). For the 100mg and 150mg doses, ACR50, ACR70, and DAS28-CRP  
 4 *response rates were statistically significantly higher than placebo*. The 50mg dose  
 did not appear to be efficacious. Importantly, there were clear dose-dependent  
 effects on all efficacy measures of the three R788 doses, which should facilitate  
 determination of an optimal therapeutic window.

5 67. Rigel's stock price more than tripled, from \$8 per share on December 12, 2007 to  
 6 \$25.95 on December 13, 2007, the day defendants announced the results of the Phase IIa RA clinical  
 7 trial. By comparison, the peer group declined 0.9% and the NASDAQ declined 0.1%.<sup>5</sup>

8 **D. Reasons Why Defendants Knew the Statements Made on December**  
 9 **13, 2007 Were Materially False and Misleading**

10 **1. R788 Was Neither as Effective as Reported and Did Not**  
 11 **Achieve Statistically Significant Results at the Primary**  
 12 **Efficacy Endpoint**

13 68. The Officer Defendants and Rigel knew the statements made about the efficacy of  
 14 R788 made on December 13, 2007 were materially false and misleading as follows:

15 69. **Defendants failed to report ACR response data by country.** As set forth in ¶60,  
 16 on December 13, 2007, Rigel reported the following results for the Phase IIa RA clinical trial:

	<b>Placebo</b>	<b>50MG</b>	<b>100MG</b>	<b>150MG</b>
<b># of U.S. &amp; Mexico Patients</b>	<b>47</b>	<b>46</b>	<b>49</b>	<b>47</b>
ACR20	18 (38%)	15 (33%)	32 (65%) (p=.008)	34 (72%) (p<.001)
ACR50	9 (19%)	8 (17%)	24 (49%) (p=.002)	27 (57%) (p<.001)
ACR70	2 (4%)	1 (2%)	16 (33%) (p<.001)	19 (40%) (p<.001)

21 70. On October 27, 2008, Rigel revealed for the first time the study results on a per-  
 22 country basis for patients in the U.S. and Mexico as follows:  
 23  
 24  
 25

26  
 27 <sup>5</sup> The peer group is the NASDAQ Biotechnology Index. In the Company's 2007 Form 10-K,  
 28 Rigel compared its stock price to the NASDAQ and the NASDAQ Biotechnology Index.

	Placebo	50MG	100MG	150MG
# of U.S. Patients	25	46	21	5
ACR20	6 (24%)	15 (33%)	11 (52%)	2 (40%)
ACR50	1 (4%)	8 (17%)	6 (29%)	2 (40%)
ACR70	0 (0%)	1 (2%)	3 (14%)	2 (40%)

	Placebo	50MG	100MG	150MG
# of Mexico Patients	22	0	28	42
ACR20	12 (55%)	0 (0%)	21 (75%)	32 (76%)
ACR50	8 (36%)	0 (0%)	18 (64%)	25 (60%)
ACR70	2 (9%)	0 (0%)	13 (46%)	17 (40%)

71. On October 27, 2008, Grossbard also acknowledged he knew about the differing response rates on December 13, 2007:

The issue of Mexico/US interaction before the study – I think we actually mentioned this at our original discussion on the Web after the study was over. I was concerned that there might be such an interaction.

And so, I requested before the study was unblinded that we do a country interaction and it turned out there was one. And the issue of the interaction was that *the placebo rate was much higher in Mexico than in the US. And the response rate was much higher in Mexico than in the US.*

72. **Analysts recognized that the per-country data disclosed on October 27, 2008 undermined the efficacy results announced on December 13, 2007.** The differing response rates and unbalanced dose distribution revealed on October 27, 2008 was important information because it indicated that the higher response rates by Mexican patients overstated the dose response. In fact, numerous analysts reported this was important information when the Company disclosed it for the first time on October 27, 2008. In an October 28, 2008 report, RBC Capital Markets (“RBC”) analyst Jason Kantor wrote that the impact of the Mexican data may have overstated the dose response:

*The formal presentation of the R788 Phase IIa data and subsequent investor event at ACR were confounded by two disclosures: 1) a dose-dependent increase in mean systolic blood pressure of 3-5 mmHg at 100mg; and 2) higher placebo and on-treatment response rates among patients treated in Mexico vs the US.*

\* \* \*

**Response rates differ by geography.** The drug clearly worked in both the US and Mexico, and the benefit over placebo was similar in both countries. However,

1 patients in Mexico had higher response rates in both the placebo treated arms. *The*  
2 *higher response rates at the Mexican sites may have contributed disproportionately*  
3 *to the benefit observed at the higher doses*, as nearly all patients in the 150mg cohort  
4 and no patients in the 50mg cohort were from Mexico.

5 \* \* \*

6 In the Phase IIa dose-ranging trial, 189 moderate-to-severe RA patients were  
7 dosed twice daily (BID) for 3 months at three doses vs. placebo. The trial was  
8 conducted in the US and Mexico with approximately 50% in each countr[y]. *The*  
9 *company provided the ACR response data by country for the first time. Patients in*  
10 *Mexico had a higher response rate in both placebo and treatment arms than the*  
11 *US patients*. The placebo group and the 100mg group were relatively evenly split  
12 between US and Mexican patients. However, *there were no Mexican patients*  
13 *treated at the 50mg dose and approximately 70% of the 150 mg dose was enrolled*  
14 *in Mexico*.

15 We do not view the efficacy differences as being a significant clinical or  
16 regulatory risk. The drug clearly worked in both the US and Mexico, and the benefit  
17 over placebo was similar in both countries. Moreover, this phenomenon has been  
18 reported in other clinical trials for RA drugs.

19 *The main concern is that the impact of the Mexican data may have been to*  
20 *overstate the dose response*. Patients in Mexico had higher response rates in general,  
21 and they represented a larger portion of the population at increasing doses. *The*  
22 *investment thesis for R788 in RA is based largely on the very robust efficacy results*  
23 *and strong dose response*.

24 73. Credit Suisse analyst Aberman also wrote in a report issued on October 27, 2008 that  
25 Rigel had presented the differences in efficacy in Mexico versus the U.S. for the first time and that it  
26 was a particular concern because the ratio of Mexico patients to U.S. patients was higher in the  
27 higher-dosing groups, which could skew the data in favor of R788:

28 One of the first areas of concerns that was raised from today’s data presentation was  
the fact that patients enrolled in Mexico had a higher placebo response rate than in  
the US. A high placebo response could also lead to higher treatment response. *This*  
*is a particular concern since the ratio of Mexican patients to US patients was*  
*higher in the higher dosing groups, which could skew the data in favor of R788.*

The analysts were correct, the distribution of doses – with 50mg tested only in the U.S. and 150mg  
tested almost only in Mexico – and the very high placebo response rate in Mexico – 55% – did skew  
the data in favor of R788, as described further in ¶¶74-90.

74. **The Phase IIa RA clinical trial results were not statistically significant as**  
**reported by defendants on December 13, 2007.** Employing “a basic set of analyses applying  
standard statistical methodologies,” R788 failed to show statistically significant improvement over  
placebo for 100mg or 150mg at ACR20, the primary efficacy endpoint. Bloch Decl. at 6.

1 When the data is properly analyzed, all of the nominal p-values are higher than those  
2 reported with the December 13, 2007 press release, even without accounting for  
3 multiple comparisons. ***The comparisons of both 100mg and 150mg groups to  
Placebo for the primary outcome variable, the ACR20, with or without accounting  
for multiple comparisons, are not statistically significant.***

4 *Id.* at 12. Because the drug failed to achieve statistically significant results at any of the stated  
5 efficacy endpoints, the statements at ¶¶60-62 touting the purported efficacy of R788 were false and  
6 misleading. As explained below, the improper pooling of country data, the use of an inappropriate  
7 statistical test and the failure to correct for multiple pair-wise comparisons rendered the statistical  
8 results and efficacy claims touted on December 13, 2007 false.

9 75. **Defendants improperly pooled the data inflating the efficacy results of R788.** It  
10 is axiomatic that a statistical analysis of data collected in a multi-site clinical study (such as the  
11 Phase IIa RA clinical trial) is analyzed separately and the results are combined. As explained in  
12 *Pitfalls of Multisite Randomized Clinical Trials of Efficacy and Effectiveness*:

13 ***The simple answer, of course, is that site effects and site x treatment  
14 interactions should always be considered in the analysis of the results of a multisite  
RCT [randomized clinical trial].*** Ignoring site differences can produce a statistical  
15 artifact called Simpson's Paradox (Blyth 1972; Bickel and Hammel 1975; Hand  
16 1979; Wagner 1982); which may result in a conclusion that the treatment is effective  
when in fact it is not effective at any site, or in a conclusion that the treatment is  
ineffective when in fact it is effective at every site.

17 Kraemer, H.C., *Schizophrenia Bulletin*, Vol. 26, No. 3, 538-39 (2000).

18 76. Indeed, basic statistical principals requires that the data for the U.S. and Mexico be  
19 analyzed separately and that defendants calculate two p-values. Bloch Decl. at 5. These p-values  
20 are then combined using Fisher's method to produce an overall p-value for the U.S. and Mexico  
21 data.<sup>6</sup> *Id.* at 5-9. Analysis of the pooled data from the U.S. and Mexico, as defendants did, "resulted  
22 in misleading success rates and false p-values":

23 Inspection of the ACR rates by Country ***easily reveals*** that the analysis of the  
24 combined data from the U.S. and Mexico reported with the December 13, 2007 press

25 <sup>6</sup> Fisher's method, also known as Fisher's combined probability test, is a technique for data  
26 fusion or "meta-analysis" (analysis of analyses). In its basic form, it is used to combine the results  
27 from several independent tests bearing upon the same overall hypothesis. Dr. Bloch used this  
28 method to "combine p-values from 2 independent studies, in order to obtain an accurate p-value for  
the combined results from the U.S. and Mexico." Bloch Decl. at 6.

1 release is *not appropriate and resulted in misleading success rates and false p-*  
2 *values*. Specifically, Defendant failed to account for the fact that patients in Mexico  
3 had higher success rates in all treatment groups than the U.S[.] patient groups.  
4 *Because the Defendant did not report an analysis which adjusts for these large*  
5 *differences in success rates between countries, the results presented in Rigel’s*  
6 *December 13, 2007 Press Release bias results in favor of R788.*

7 *Id.* at 5.

8 77. Correcting for this manipulation, the p-values for the 100mg and 150mg doses at  
9 ACR20 and ACR50 are all higher (*i.e.*, indicating less statistical significance) than those falsely  
10 reported on December 13, 2007, and R788 did not achieve statistically significant results at the  
11 primary efficacy endpoint, ACR20, for the 150mg dose. *Id.* at 7-9 (the p-values are “much bigger”  
12 at ACR20, the primary efficacy endpoint). The following chart depicts the correction of defendants’  
13 improper pooling of the data (but does not account for the improper use of the chi-squared test or the  
14 failure to account for the multiple comparisons problem discussed in ¶¶78-81, below, both of which  
15 further increase the p-values):

	<b>December 13, 2007 Reported P-Values</b>	<b>Calculated by Country with Chi- Squared Method</b>	<b>Not Statistically Significant</b>
ACR20 100mg	0.008	0.037	
ACR20 150mg	<0.001	0.152	x
ACR50 100mg	0.002	0.003	
ACR50 150mg	<0.001	0.026	

16 78. **Defendants further inflated the efficacy of R788 by using an improper statistical**  
17 **analysis.** In addition to inappropriately pooling the per-country data prior to analyzing it, defendants  
18 used the chi-squared analysis to calculate p-values. *Id.* at 6. Because the sample sizes are small in  
19 the Phase IIa RA clinical trial, application of the chi-squared test is inappropriate and yielded p-  
20 values that were indicative of greater statistical significance than the data supported. *Id.* at 6-7.  
21 Because of the small sample sizes at issue, “Fisher Exact test rather than a chi-square test is the  
22 appropriate statistical procedure.” *Id.* at 9.

23 79. Using Fisher’s Exact test to calculate p-values for the U.S. and Mexico, (and then  
24 properly combining them, *see* footnote 6), the p-values were higher and demonstrated less statistical  
25 significance than defendants reported on December 13, 2007. Bloch Decl. at 9-11. Indeed, when the  
26  
27  
28

1 improper use of the chi-square analysis, and the improper pooling of data, is corrected for, the p-  
 2 values for the 100mg and 150mg doses at ACR20, ACR50 and ACR70 are all higher (*i.e.*, indicating  
 3 less significance) than those falsely reported on December 13, 2007 and ***at the primary efficacy***  
 4 ***endpoint, ACR20 for the 100mg or 150mg doses R788 did not achieve statistically significant***  
 5 ***results.*** *Id.* at 9-11. The corrected results accounting for the improper pooling of the data and the  
 6 use of the chi-squared analysis (but not accounting for the multiple comparisons problem) are  
 7 summarized in the following table:

	<b>December 13, 2007 Reported P-Values</b>	<b>P-Values Calculated by Country with Fisher's Exact Test</b>	<b>Not Statistically Significant</b>
ACR20 100mg	0.008	0.056	x
ACR20 150mg	<0.001	0.216	x
ACR50 100mg	0.002	0.022	
ACR50 150mg	<0.001	0.043	
ACR70 100mg	<0.001	0.004	
ACR70 150mg	<0.001	0.002	

13  
 14 **80. Defendants failed to correct the multiple comparisons problem which further**  
 15 **inflated the efficacy results of R788.**<sup>7</sup> Defendants made multiple pair-wise comparisons between  
 16 groups – *i.e.*, 100mg versus placebo at ACR20, 100mg versus placebo at ACR50, 100mg versus  
 17 placebo at ACR70 – which increased the likelihood that one comparison would be statistically  
 18 significant by chance alone. Bloch Decl. at 3-4, 6, 11-12. When a set of statistical inferences is  
 19 considered simultaneously, a proper statistical analysis should account for the multiple comparisons  
 20 being made. *Id.* Defendants failed to do so, which resulted in p-values which were lower (*i.e.*, more  
 21 statistically significant) than that data supported and which overstated the efficacy of R788.

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 23 <sup>7</sup> “Multiple comparisons” arise when a statistical analysis encompasses a number of formal  
 24 comparisons, with the presumption that attention will focus on the strongest differences among all  
 25 comparisons that are made. For example, in considering the efficacy of a drug in terms of the  
 26 reduction of any one of a number of disease symptoms. As more symptoms are considered, it  
 27 becomes more likely that the drug will appear to be an improvement over existing drugs in terms of  
 28 at least one symptom. However a difference between the groups is only meaningful if it generalizes  
 to an independent sample of data (*e.g.*, to an independent set of people treated with the same drug).  
 The confidence that a result will generalize to independent data should generally be weaker if it is  
 observed as part of an analysis that involves multiple comparisons, rather than an analysis that  
 involves only a single comparison.

1 Applying Tukey’s Studentized Range test, “control[s] for differences between the U.S[.] and Mexico  
2 and for multiple pair-wise comparisons that were made between groups.” *Id.* at 12. Application of  
3 Tukey’s Studentized Range test to the R788 data on a per-country basis – *i.e.*, the proper statistical  
4 analysis for the data obtained in the Phase IIa RA clinical trial – ***the p-values at the primary efficacy***  
5 ***endpoint, ACR20 for 100mg and 150mg were very large and not statistically significant.*** *Id.* at 11-  
6 12.

7 81. The p-values obtained by an appropriate statistical analysis of 100mg and 150mg at  
8 the primary efficacy endpoint, ACR20, were  $p=0.198$  and  $p=0.444$ , respectively. *Id.* at 12. These  
9 values were more than 20 times larger for 100mg dose, and 400 times larger for 150mg dose, than  
10 reported by defendants on December 13, 2007, and demonstrate the falsity of defendants’ claims of  
11 efficacy, as explained by Dr. Bloch:

12 With this analysis I have controlled for differences between U.S[.] and Mexico and  
13 for multiple pair-wise comparisons that were made between groups. ***The overall p-***  
14 ***values comparing both 100mg and 150mg groups to Placebo for the primary***  
15 ***outcome variable, the ACR20, are large and the correct conclusion is that success***  
16 ***rates between either of the high dose groups and Placebo are not statistically***  
17 ***significantly different. The nominal p-values reported with the December 13, 2007***  
18 ***press release are false and misleading.***

16 *Id.*

17 82. **Presentation of the combined data on December 13, 2007 was additionally**  
18 **misleading because it skewed the efficacy results of R788.** Not providing investors with data on a  
19 per-country basis concealed (i) the existence of a country interaction so substantial that the placebo  
20 in Mexico was more effective than any dose in the U.S. at ACR20 as set forth in ¶70; (ii) that the  
21 distribution by dose was not balanced across the U.S. and Mexico (*Id.*); and (iii) that there was no  
22 ascending dose response.

23 83. Indeed, defendant Grossbard falsely stated on December 13, 2007, that “overall there  
24 was a good dose response,” when in fact there was not. *See* ¶60. By combining the data defendants  
25 mislead investors into believing there was a strong ascending dose response.

26 84. The appearance of an ascending dose response and the apparent lack of response at  
27 50mg, were highlighted by analysts as positive indicators of efficacy. For instance, Credit Suisse  
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1 analyst Aberman stated “Key efficacy highlights include 1) a no effect dose, 2) a dose response  
2 between all doses, and 3) arguably the best efficacy seen in a PII RA trial.”

3 85. As reflected in the chart below the combined data misleadingly portrayed an  
4 ascending dose-response. For example, at ACR20, when defendants reported the pooled results on  
5 December 13, 2007, it appeared that patients were 5% worse than placebo at 50mg, 27% better than  
6 placebo at 100mg and 34% better than placebo at 150mg. Failing to provide a per-country  
7 breakdown disguised that 100mg was only 19% better than placebo in the U.S., not 32% better, as it  
8 appeared in the pooled data reported on December 13, 2007 and that **150mg in the U.S. was worse**  
9 **than 100mg, by 12%, not 7% better as it was presented in the pooled data.** In Mexico, where only  
10 100mg and 150mg were tested, 150mg was only 1% better than 100mg, not 7% as it appeared in the  
11 pooled data.

<b>Improvement Over Placebo at ACR20</b>	<b>50mg</b>	<b>100mg</b>	<b>150mg</b>
U.S. Data	9%	28% ( $\Delta$ 19)	16% ( $\Delta$ -12)
Mexico Data	NA	20% (NA)	21% ( $\Delta$ 1)
Combined Data	-5%	27% ( $\Delta$ 32)	34% ( $\Delta$ 7)

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14  
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16 86. By not presenting the per-country data (*i.e.*, U.S. and Mexico) on December 13, 2007,  
17 R788 appeared more effective at the primary efficacy endpoint, ACR20, for the 150mg dose, than it  
18 actually was in either the U.S. or Mexico. As the chart above reflects, the combined results show an  
19 improvement of 34% over placebo when the sites individually achieved improvement of only 16%  
20 and 21%, respectively. “[T]he difference reported with the December 13, 2007 press release is 34%,  
21 a success rate over twice that achieved in the U.S[.] and 13% higher than that achieved in Mexico.”  
22 *Id.* at 5.

23 87. At 50mg, a dose which analysts characterized as the “no effect dose,” combining the  
24 data had the opposite effect, making R788 appear less effective than placebo. This contributed to the  
25 false impression that there was a strong escalating dose response at the 100mg and 150mg, *i.e.*, the  
26 larger dose of R788 a patient received, the greater the improvement in their symptoms, as the  
27 following chart depicts:  
28

	Placebo	50MG	Improvement Over Placebo
<b># of U.S. &amp; Mexico Patients</b>	47	46	
ACR20	18 (38%)	15 (33%)	-5%
ACR50	9 (19%)	8 (17%)	-2%
ACR70	2 (4%)	1 (2%)	-2%

88. However, when compared to the U.S. placebo group the 50mg dose performed better than placebo, although at a level that was not statistically significant, as the following chart depicts:

	Placebo	50MG	Improvement Over Placebo
<b># of U.S. Patients</b>	25	46	
ACR20	6 (24%)	15 (33%)	9%
ACR50	1 (4%)	8 (17%)	13%
ACR70	0 (0%)	1 (2%)	1%

89. Dr. Bloch noted this distortion, which made the 50mg dose appear 15% less effective than it was, and the distortions in ¶¶82-88 above, as evidence of why combining the data was misleading:

Referencing the patient populations who received the 50mg dose or Placebo and were assessed for response with the ACR20, the primary outcome variable, the differences in success rates of the 50mg group versus the Placebo group was 9% in the U.S. No such comparison can be made for patients treated in Mexico since no patients received the 50mg dose in Mexico. However, the difference reported with the December 13, 2007 press release is -5%, a difference in success rates which is 14% lower than that achieved in the only country that gave patients the 50mg dose, the U.S. These examples illustrate that ***the data from the U.S. and Mexico should not [be] pooled, and that a proper, overall, analysis must combine the results of pair-wise comparisons obtained from each country.***

Bloch Decl. at 5-6.

90. On October 27, 2008, Grossbard attempted to downplay the significance of the differences in response rate per-country by claiming that the only “important factor is the difference between the active and control group.” However, both the total response rate and the difference between active and placebo groups is essential to accurately assessing the study. The total response rate was material to investors because the investment thesis in R788 required it to be highly effective as Rigel was in a competition with other pharmaceutical companies, including Pfizer (See ¶¶122-25),

1 and higher ACR response rates would indicate that R788 could beat out possible competing drugs  
2 and warranted a lucrative partnership deal. The combined data concealed the difference between  
3 the relevant active and control groups – *i.e.*, the U.S. active group versus the U.S. control group,  
4 making the drug appear to have an ascending dose-response (which was not actually present) and  
5 more effective than it was.

6 **2. R788 Exhibited Greater Toxicity than Defendants Led**  
7 **Investors to Believe**

8 91. Defendants’ statements about the safety results of the Phase IIa RA clinical trial were  
9 also materially false and misleading because defendants knew but failed to disclose that (i) there was  
10 a dose-dependent increase in average systolic blood pressure of 3-5mm Hg in 100mg patients and 8-  
11 9mm Hg in the 150mg patients which signaled the potential for increased cardiovascular risk,  
12 presented additional hurdles to regulatory approval and commercial partnership, particularly because  
13 the mechanism that caused the increase was not well understood; (ii) 5 patients (not 2, as reported on  
14 December 13, 2007) experienced hypertension, with blood pressure increases as high as 20-30mm  
15 Hg; (iii) hypertension was one of the two most common clinically meaningful drug related adverse  
16 events (iv) 9 patients (not 3, as reported on December 13, 2007) experienced increased liver enzymes  
17 compared to patients taking the placebo; (v) 20 patients (not 15, as reported on December 13, 2007)  
18 experienced neutropenia; (vi) 34 patients (not 15, as reported on December 13, 2007) experienced  
19 diarrhea; and (vii) 35 patients (not 15, as reported on December 13, 2007) experienced upper  
20 gastrointestinal side effects. The undisclosed adverse events were material as they signaled the  
21 strength of the side effects, presented complications for regulatory approval, impacted the need for  
22 and design of future studies and jeopardized Rigel’s ability to obtain a partnership for the further  
23 development of R788 – *i.e.*, its investment thesis.

24 92. **R788 caused a dose-dependent increase in mean blood pressure.** On December  
25 13, 2007, Rigel reported that 2 patients in the 100mg cohort experienced moderate hypertension<sup>8</sup> and

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26  
27 <sup>8</sup> Hypertension is a clinical term for people who have systolic or diastolic blood pressure  
28 equal to or above a certain level, measured in mmHg (“millimeters of mercury”). Typically,

1 Grossbard stated that “[t]he incidence of reported moderate hypertension was quite low.”  
2 Defendants statements were false and misleading because they failed to disclose (i) additional  
3 incidences of hypertension and that some of the increases were an alarming 20-30mm Hg; (ii) that  
4 there was a clinically significant and dose dependent increase in average blood pressure in all  
5 patients; and (iii) that hypertension and increased blood pressure were dose dependent side effects.

6 93. Rigel would later concede that hypertension was one of the “most common clinically  
7 meaningful” side effects of R788 in the Phase IIa RA clinical trial. On July 23, 2009, Rigel  
8 announced that one of the Phase IIb clinical studies failed to meet efficacy endpoints because  
9 patients did not report statistically significant higher ACR response rates than the placebo group.  
10 Despite failing to note the significance of hypertension in the Phase IIa RA clinical trial on  
11 December 13, 2007, defendants admitted to its significance in announcing the Phase IIb results:

12 *Similar to [the Phase IIa study,] the most common clinically meaningful drug*  
13 *related adverse events* noted in [the Phase IIb study] were diarrhea and  
*hypertension.*

14 94. On the December 13, 2007 conference call, Grossbard explained that elevated liver  
15 enzymes and neutropenia were dose-related adverse events that were ameliorated by reduced  
16 dosages of R788. See ¶61. However, defendants disclosed only two incidences of moderate  
17 hypertension and described the incidence of moderate hypertension as “quite low.” This gave  
18 investors the misleading impression that blood pressure effects were not dose-dependent (when they  
19 were), would not pose a clinical or regulatory risk (which they did), and would not be a barrier for  
20 potential partners (which it was).

21 95. The misleading nature of defendants’ statements is echoed in analyst reports. Indeed,  
22 in his December 13, 2007 report, entitled “RA Phase II Data Look Great – Increase PT,” Credit  
23 Suisse analyst Aberman highlighted the absence of dose dependent hypertension: “*Of particular*  
24 *note, . . . no evidence of dose dependent hypertension.*”

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27 blood pressure of 140-159/90-99mm Hg is considered “Stage 1” hypertension and blood pressure  
28 of 160+/100+mm Hg is considered “Stage 2” hypertension.

1           96.     On October 27, 2008, defendants disclosed that in fact 5 patients experienced  
2 hypertension and the reported increase in blood pressure were as high as 20-30mm Hg. On the same  
3 day, defendants also disclosed that there was a dose-dependent increase in average blood pressure  
4 across all patients, *i.e.*, the more R788 a patient received the higher their blood-pressure. The mean  
5 systolic blood pressure increase was 3-4mm Hg in 50mg dose, 3-5mm Hg in the 100mg dose and 8-  
6 9mm Hg in the 150mg dose in all patients, regardless of whether or not their blood pressure reached  
7 the threshold for hypertension.

8           97.     Defendants were aware that the effect of R788 on blood pressure was of key  
9 importance to investors even prior to the Class Period. On November 9, 2007, Rigel released a press  
10 release announcing the results of ITP study and stating “**R788 elevated blood pressure in some**  
11 **patients**” which also coincided with a 40% drop in Rigel’s share price. On December 4, 2007,  
12 defendants admitted that they were looking to the results of the Phase IIa RA clinical trial to “get a  
13 good fix on the general effect on blood pressure.” Defendants even stated “***we will see what the***  
14 ***mean change in the blood pressure is, and we should know that within a week or two.***”

15           98.     Despite indicating that they would know of the “mean change” in blood pressure,  
16 defendants concealed this very information from investors when they presented the Phase IIa RA  
17 clinical trial data nine days later. On December 13, 2007, Defendants deliberately announced only  
18 incidences of moderate to severe hypertension – and downplayed their significance to make the drug  
19 appear safer than it was. Defendants provided no definition of what constituted “moderate” or  
20 “severe” blood pressure increases and the subsequently reported results on October 27, 2008 belie  
21 their prior representations, as reflected in the market’s reaction.

22           99.     During the October 27, 2008 ACR investor update, Grossbard acknowledged that five  
23 patients (not two, as reported on December 13, 2007) experienced hypertension, that there was a  
24 dose-dependent increase in blood pressure, that the increase was “of crushing importance to  
25 everybody” at the ACR conference, that additional studies would be necessary to get more precise  
26 estimates and that “the final word on blood pressure’s pretty far down the road”:

27           [T]here were a total of ***five patients*** in the two high dose groups that the investigators  
28 wrote hypertension.

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Then there's a question of blood pressure. And we have noted, and it is in the paper coming out in the next two weeks, that ***our drug at doses of 100mg twice a day, for example, over 12 weeks, has an average increase in blood pressure of about 4mm systolic relative to their baseline.***

\* \* \*

***I believe it's real and I believe there's an effect on blood pressure.*** The magnitude of the effect that we measured in this study in the 100 mg twice a day dose was about 4 mm [for the patients in the 100mg cohort].

The studies we're doing now are much larger. We'll get another estimate from those studies. And it's possible over time we would end up doing an ambulatory blood pressure study to get an even more precise estimate of the blood pressure effect. So that's where we stand on blood pressure.

\* \* \*

It's every person compared to his baseline that detects it. And so doing that, we had seven different changes from the baseline, one for each visit, and then we averaged them to get this number that we've given to you. The diastolic number is a little smaller than 4 mm. It was 2 mm or 3 mm. Systolic number was 4 mm to 5 mm.

\* \* \*

The systolic was about 8 mm and the diastolic was about 4 mm [for the patients in the 100 mg cohort]. That's my recollection.

\* \* \*

***And when the drug was withdrawn at the end of the study, at the end of the 12-week period, it came pretty much down to baseline. So I believe it's a real effect.*** I just can't be precise about the magnitude of the effect, whether it's going to turn out to be three or five or seven. That's just harder to know because we at it that hard as part of this study.

We're paying more attention to it now in a Phase 2b study and I – my guess is it'll probably be a little better as we get large numbers and we've now alerted investigators to the fact that blood pressure can be a side effect and that they should treat it when it occurs and so on and so forth. But that's a prediction, it's not a fact.

\* \* \*

It depends on the dose. The dose of 100 mg twice a day, it was four, and the dose 150 twice a day, it was seven to eight. And it would depend if it was sitting, if it was standing. But qualitatively, those are the way the numbers go. ***It's dose dependent. There's no question about it.***

\* \* \*

***In the course of the study, five patients either had antihypersensitive treatment initiated, or had their blood pressure medicine changed as a result of the blood pressure that was observed during the study,*** which coincided usually with the five

1 patients – I don't want to say it was person to person, but it coincided with the *five*  
2 *patients who were identified as having the adverse event of hypertension.*

3 \* \* \*

4 When we do the next study we're going to have 400 or 500 patients for fairly  
5 long periods of time. So *I know it's of crushing importance to everybody here*  
6 *whether its three or five, but that's not going to be the final word on blood pressure.*  
7 *The final word on blood pressure's pretty far down the road.*

8 100. Gower also acknowledged that the blood pressure increases presented a regulatory  
9 hurdle: "The regulatory risks are more tangible . . . in that you never know, especially in this climate,  
10 what you are going to run into that will cause concern on the part of the FDA or an advisory panel."

11 101. The dose-dependent increase in blood pressure was important because (i) it might  
12 signal an increase in cardiovascular risk; (ii) the mechanism by which R788 increased blood pressure  
13 was not well understood; and (iii) it could be a stumbling block for some pharmaceutical companies  
14 in considering licensing the drug. Defendants would later admit that hypertension was one of two  
15 most "common clinically meaningful drug related adverse events" in the Phase IIa RA clinical trial.

16 102. In fact, when this information was first disclosed on October 27, 2008, several  
17 analysts noted their concern. In his October 28, 2008 report, RBC analyst Jason Kantor downgraded  
18 the stock due to "heightened safety concerns for R788" and noted that: (i) the previously undisclosed  
19 increase in blood pressure was viewed as a "potentially significant concern" to independent  
20 physicians attending the October 27, 2008 ACR conference; and (ii) the new negative information  
21 caused one pharmaceutical company to walk away from a potential partnership with Rigel. Kantor  
22 also reported that the defendants stated during the webcast that the blood pressure data was available  
23 to potential partners throughout the process:

24 The formal presentation of the R788 Phase IIa data and subsequent investor  
25 event at ACR were confounded by two disclosures: 1) *a dose-dependent increase in*  
26 *mean systolic blood pressure of 3-5 mmHg at 100mg*; and 2) higher placebo and on-  
27 treatment response rates among patients treated in Mexico vs the US. Based on our  
28 conversations with the company, one principle investigator, and other physicians, we  
believe *the perceived risk/benefit ratio has worsened slightly, and in the absence of*  
*new data, concerns are likely to persist.* We still anticipate a partnership in early  
2009, but no longer expect a deal sooner in 2008. We are downgrading to Sector  
Perform, Speculative Risk, and would not be aggressively buying on the current  
weakness.

- *Increase in blood pressure. Rigel previously disclosed a modest increase in hypertensive adverse events with R788, and now disclosed a dose-*

1 *dependent increase in average blood pressure. The blood pressure increase*  
2 *is somewhat concerning in that 1) it may signal an increase in*  
3 *cardiovascular risk; 2) the mechanism is not well understood; and 3) it*  
4 *could be a stumbling block for some pharma companies in considering*  
5 *licensing the drug. In speaking to some independent physicians at the*  
6 *conference, the blood pressure issue was viewed as a potentially significant*  
7 *concern.*

8 \* \* \*

- 9 • **R788 partnership in early 2009.** According to management, Rigel remains  
10 in detailed licensing discussions with several potential partners, and  
11 confirmed that *the blood pressure data has been available to potential*  
12 *partners throughout the process.* Management continues to expect a  
13 lucrative partnership in early 2009, pending successful completion of the  
14 thorough QT study. The company emphatically stated that no QT  
15 prolongation signal has been observed so far. A partnership would be a  
16 catalyst for outperformance.

17 103. Kantor also wrote that the dose-dependent increase in mean systolic and diastolic  
18 blood pressure was surprising and raised several concerns:

19 As reported in prior top-line results, there were dose-dependent increases in  
20 several important adverse events, specifically neutropenia, elevated liver enzymes,  
21 GI side effects, and hypertension. However, *what surprised us was that there was a*  
22 *dose-dependent increase in mean systolic and diastolic blood pressure.* The clinical  
23 significance of the increased blood pressure is unclear, but we have several concerns:

24 1. *An increase in blood pressure raises the potential concern of increased*  
25 *cardiovascular events in larger trials or future commercial experience. This*  
26 *increases both clinical and regulatory risk.*

27 2. The mechanism of the blood pressure increase is unknown, which could raise  
28 additional regulatory concerns. One of the principle investigators suggested that the  
effect could be due to cross reactivity with the VEGF receptor. If there is a  
physiologically measurable impact on VEGF, it could raise further safety concerns.

3. The blood pressure increase appears to be inseparable from efficacy because  
a slight increase is also observed at the low, non-efficacious dose.

4. *There is a steep dose-related increase in blood pressure.*

5. The increase in blood pressure may necessitate a larger and longer Phase III  
program, and may limit ultimate commercial uptake of the drug.

6. Potential cardiovascular risk could limit partnering opportunity. *Rigel*  
*management informed us that potential partners have had access to this data for*  
*some time, and in fact at least one potential partner was discouraged enough to*  
*walk away.*



1 dependent increase in ALT – 2 patients in the 50mg cohort, 3 patients in the 100mg cohort and 4  
2 patients in the 150mg cohort.

3 107. Analysts noted the difference in defendants’ disclosures. On October 28, 2008,  
4 Oppenheimer analyst Abrahams issued a report in which he wrote the following:

5 ACR data highlighted neutropenia and LFT [liver function test] elevations as  
6 potential risks worth watching. *Neutropenia was slightly higher than previously*  
7 *reported and not completely confined to the first several weeks, and LFT elevations*  
8 *were associated with R788. While R788’s side effect profile is not benign, as*  
9 *suggested by prior data*, we believe it is acceptable thus far.

10 108. While Abrahams noted that the December 2007 data “suggested a possible  
11 association with LFT elevations,” he wrote that the data presented on October 27, 2008 “showed  
12 mild dose-dependent ALT elevations greater than 1.2x ULN [upper limit of normal] at all doses,  
13 *which confirms R788’s association with LFT increases*, in our opinion.” He also wrote that “there  
14 are clearly some additive liver abnormalities as evidenced by the increase in patients who had LFT  
15 levels of more than 1.2X ULN.”

16 109. On December 4, 2007, in connection with the Phase II ITP clinical trial, defendants  
17 released all incidences of elevated liver enzymes (ALT) which were twice the upper limit of normal.  
18 *See ¶57.* However, on December 13, 2007, in connection with Phase IIa RA clinical trial,  
19 defendants released only liver enzyme elevations that were three times the upper limit of normal.  
20 Having reported two times elevations only nine days earlier, defendants set expectation for the safety  
21 results that would be reported by the Company and the failure to disclose the extent of elevated liver  
22 enzymes on December 13, 2007 was part of a deliberate effort to conceal the frequency of drug  
23 related adverse events.

24 110. **Neutropenia:** On December 13, 2007, Rigel reported that 15 patients experienced a  
25 dose-dependent increase in neutropenia – 5 patients in the 100mg cohort and 10 patients in the  
26 150mg cohort. However, during the October 27, 2008 ACR presentation and in the November 2008  
27 article, it was reported that 20 patients experienced neutropenia – 1 in the 50mg cohort, 5 in the  
28 100mg cohort and 14 in the 150mg cohort.

111. Like the other adverse safety data, analysts noted the difference. For example,  
Oppenheimer analyst Abrahams wrote the following in his October 28, 2008 report: “**Full phase IIa**

1 *data showed an increase in neutropenia from previously reported top-line data. . . . There were*  
2 *five additional cases of neutropenia in the full phase IIa data.”*

3 112. **Gastrointestinal side effects:** On December 13, 2007, Rigel reported that 15 patients  
4 experienced upper gastrointestinal side effects – 1 in the 50mg cohort, 2 in the 100mg cohort and 12  
5 in the 150mg cohort. However, during the October 27, 2008 ACR presentation and in the November  
6 2008 article, it was reported that 35 patients experienced upper gastrointestinal side effects – 4 in the  
7 50mg cohort, 14 in the 100mg cohort and 17 in the 150mg cohort.

8 113. **Diarrhea:** On December 13, 2007, Rigel reported that 15 patients experienced  
9 diarrhea – 3 in the 50mg cohort, 2 in the 100mg cohort and 10 in the 150mg cohort. However,  
10 during the October 27, 2008 ACR presentation and in the November 2008 article, it was reported  
11 that 34 patients experienced diarrhea – 5 in the 50mg cohort, 8 in the 100mg cohort and 21 in the  
12 150mg cohort.

13 114. **Rigel, Gower, Grossbard, Payan, Rodriguez, and Maynard knew about the**  
14 **adverse safety data on December 13, 2007, because the Phase IIa RA clinical trial was**  
15 **completed.** All of the above adverse information was known by the defendants on December 13,  
16 2007, because the study was complete and the data was in. In fact, during the December 13, 2007  
17 conference call, Grossbard acknowledged that the December 13, 2007 press release was an abstract  
18 of the November 2008 article in *Arthritis & Rheumatism* that references (i) the differing response  
19 rates between patients in Mexico and the U.S.; and (ii) the significantly worse adverse safety  
20 information. Further, defendants told investors that they would know the blood pressure data in  
21 December 2007 (*see* ¶59) and defendants admitted to analysts that the blood pressure data had been  
22 available throughout the process (*see* ¶102).

23 115. Further, Rigel had 159 employees as of December 31, 2007, according to its Form  
24 10-K. According to the Company’s April 8, 2008 Proxy Statement, Gower, Grossbard, Payan,  
25 Rodriguez and Maynard were its most senior executives. They were the highest paid officers in the  
26 Company, and each attended the December 13, 2007 conference call. Given the importance of the  
27 Phase IIa RA clinical trial to the Company’s success and the senior positions of Gower, Grossbard,  
28

1 Payan, Rodriguez and Maynard, as well as their admissions, it would be absurd to suggest they did  
2 not know about the adverse information that was concealed from investors until October 27, 2008.

3 116. Yet, the per-county data and statistically insignificant efficacy results as well as  
4 substantially worse safety information were not disclosed on December 13, 2007.

5 117. **Rigel, Gower, Grossbard, Payan, Rodriguez, and Maynard knew the undisclosed**  
6 **adverse safety data was important.** Rigel, Gower, Grossbard, Payan, Rodriguez, and Maynard  
7 knew the undisclosed safety data was important to the Company's financial results. Rigel reported  
8 in its 2007 Form 10-K that it must demonstrate the safety of any drug before it could be approved for  
9 commercial sale:

10 Our ongoing development activities are and will continue to be subject to  
11 extensive regulation by numerous governmental authorities in the United States and  
12 other countries, including the Food and Drug Administration, or FDA, under the  
13 Federal Food, Drug and Cosmetic Act.

14 \* \* \*

15 Our failure, or the failure of our strategic partners, to adequately demonstrate the  
16 safety and efficacy of our products under development will prevent receipt of FDA  
17 and similar foreign regulatory approval and, ultimately, commercialization of our  
18 products.

19 118. Following the Company's December 13, 2007 press release and conference call,  
20 analysts that followed Rigel also reported that a key risk to the Company's stock price was adverse  
21 safety data. For example, CIBC analyst Abrahams reported on December 13, 2007 that "key risks"  
22 to the Company's 12- to 18-month stock price target included the "emergence of an unacceptable  
23 safety signal with R788."

24 119. In the November 2008 article on the Phase IIa RA clinical trial published in *Arthritis*  
25 *& Rheumatism* (of which Grossbard was a contributing author), it was written that the Phase IIa RA  
26 clinical trial was designed to evaluate the safety and preliminary clinical efficacy of R788 in patients  
27 with RA despite methotrexate therapy and that all patients were monitored for adverse events and  
28 serious adverse events. Moreover, it was stated in the article that "whether the adverse events seen  
in this study, including gastrointestinal intolerance, neutropenia, effects on blood pressure, and liver  
enzyme elevations, will be a major issue in longer studies of RA patients will also need to be  
determined." Yet, none of this information was disclosed on December 13, 2007.





1 in the expected partnership for R788 until after the Phase IIb data in Q3:09; 2) a dwindling cash  
2 balance; and 3) limited visibility regarding safety signals in the upcoming TASKi2 and TASKi3  
3 trials, whose results are expected in July and August, respectively.” He also noted the filing of the  
4 first complaint in this lawsuit and wrote the following:

5           We typically do not put much emphasis on these types of lawsuits because  
6 they are often frivolous. However, the concerns raised in the suit are similar to those  
7 we raised at the time the data was presented (please refer to our note titles “Safety  
Concerns Heightened Following Phase IIa Presentation to ACR” published on  
October 28, 2008 for more details).

8           **127. Rigel, Gower, Grossbard, Payan, Rodriguez and Maynard knew the Company**  
9 **needed to raise funds to avoid insolvency.** In addition to knowing about the undisclosed adverse  
10 safety information, the statistically insignificant results at the primary efficacy endpoint and the  
11 differing response rates by patients in Mexico and the U.S., the defendants knew the Company  
12 needed to raise funds given the Company’s deteriorating financial condition. Indeed, in every Form  
13 10-Q filed by Rigel in 2007, the first risk factor reported was that Rigel needed additional capital in  
14 the future to sufficiently fund its operations and research. For example, Rigel reported the following  
15 in its 3Q07 Form 10-Q filed with the SEC on November 6, 2007:

16           *We will need additional capital in the future to sufficiently fund our operations*  
17 *and research.*

18           We have consumed substantial amounts of capital to date, and operating  
19 expenditures are expected to increase over the next several years as we expand our  
20 research and development activities. We believe that our existing capital resources  
21 and anticipated proceeds from current collaborations will be sufficient to support our  
22 current operating plan through at least the next 12 months. ***In the foreseeable***  
23 ***future, our operations will require significant additional funding in large part due***  
24 ***to our research and development expenses, future preclinical and clinical-testing***  
***costs, and the absence of any meaningful revenues.*** The amount of future funds  
needed will depend largely on the timing and structure of potential future  
collaborations. ***We do not know whether additional financing will be available***  
***when needed, or that, if available, we will obtain financing on terms favorable to***  
***our stockholders or us.*** As of September 30, 2007 and December 31, 2006, our  
cash, cash equivalents and available-for-sale securities were \$112.5 million and  
\$104.5 million, respectively.

25           To the extent we raise additional capital by issuing equity securities, our  
26 stockholders could at that time experience substantial dilution. To the extent that we  
27 raise additional funds through collaboration and licensing arrangements, we may be  
28 required to relinquish some rights to our technologies or product candidates, or grant  
licenses on terms that are not favorable to us.

1           128. In fact, Rigel, Gower, Grossbard, Payan, Rodriguez and Maynard knew the Company  
2 would become insolvent if it did not raise funds. After reporting a net loss of \$74.3 million in 2007,  
3 Rigel reported just \$44.5 million of cash and \$82.2 million of capital at December 31, 2007. Rigel's  
4 February 2008 Offering raised \$127.5 million for the Company, but Rigel reported a \$132.3 million  
5 net loss in 2008. Thus, absent the \$127.5 million raised in the February 2008 Offering, Rigel would  
6 have become insolvent before the end of 3Q08. Rigel reported a \$99 million net loss for the nine  
7 months ending September 30, 2008, which would have more than depleted the \$82.2 million of  
8 capital the Company reported as of December 31, 2007.

9           129. Rigel, Gower, Grossbard, Payan, Rodriguez and Maynard also knew disclosure of the  
10 adverse safety information and ineffective results would make raising funds more difficult, if not  
11 impossible. Indeed, the stock price declined more than 38% following the disclosure of the adverse  
12 safety information and the data on a per-country basis reflecting the differing response rates and  
13 skewed dosing on October 27, 2008. Instead of disclosing the full results of the Phase IIa RA  
14 clinical trial, Rigel, Gower, Grossbard, Payan, Rodriguez and Maynard concealed the adverse  
15 information and accurate efficacy results concerning R788, and the Company's stock price increased  
16 from \$8 per share on December 12, 2007 to \$25.95 on December 13, 2007. Then, in February 2008,  
17 Rigel sold 5 million shares for \$27 per share for net proceeds of \$127.5 million. If Rigel, Gower,  
18 Grossbard, Payan, Rodriguez and Maynard disclosed the adverse safety information and accurate  
19 efficacy results, the Company's stock price would not have increased, and Rigel could not have sold  
20 the 5 million shares for \$27 per share and may not have been able to raise any funds.

21           130. Rigel, Gower, Grossbard, Payan, Rodriguez and Maynard also knew that the  
22 Company's stock price would decline if they reported the adverse safety information about the Phase  
23 IIa RA clinical trial because that is precisely what occurred on November 9, 2007 when Rigel  
24 announced the results of its Phase II ITP clinical trial of R788. The Company reported that a  
25 majority of the patients involved in the clinical study responded favorably to R788 and that the  
26 clinical study showed that R788 could improve platelet counts in ITP but also revealed safety  
27 concerns, including increases in blood pressure. Following the release of this information, the  
28

1 Company's stock price declined 14.1% from \$11.04 on November 8, 2007 to \$9.48 on November 9,  
2 2007. By December 4, 2007, the Company's stock price had declined to \$6.67.

3 131. Analysts attributed the decline to concerns about adverse safety data. For example,  
4 after the Company's December 4, 2007 Phase II ITP conference call, Jefferies analyst Walsh wrote  
5 in a December 5, 2007 report that Rigel shares had plummeted 40% since the Phase II ITP results  
6 were announced on November 9, 2007 due to concerns about "key safety data" including gastro  
7 intestinal side effects and elevated blood pressure.

8 132. **Need for partnership to raise funds.** Rigel, Gower, Grossbard, Payan, Rodriguez  
9 and Maynard also knew that it was important for investors to believe that the Company would be  
10 able to establish a partnership with a pharmaceutical or biotechnology company because such a  
11 partnership would provide the funds necessary to continue the development of R788 for RA. Rigel  
12 reported in its 2007 Form 10-K that one of its strategies was to "establish strategic collaborations  
13 with pharmaceutical and biotechnology companies to develop and market our product candidates."  
14 In addition, the Company reported that its future funding requirements depended on many uncertain  
15 factors, including the "ability to establish new collaborations and to maintain . . . existing  
16 collaboration partnerships" and that most of its expected future revenues were contingent upon  
17 collaborative and license agreements.

18 133. Wall Street analysts following the Company also reported that a partnership was  
19 important. For example, in his December 13, 2007 report, Credit Suisse analyst Aberman wrote that  
20 "a commercial partnership in Europe and/or Asia should also be a corporate goal *for 2008* on the  
21 strength of [the Phase IIa RA clinical trial] data."

22 134. During the October 27, 2008 ACR investor update, Gower falsely stated that Rigel  
23 was still on track for putting partnership in place in the early part of 2009 and that the Company  
24 needed to get the partnership in place before the second half of 2009.

25 *[S]till on track for what we've been saying all along, which is putting the*  
26 *partnership in place as early as the early part of next year.* I doubt it will be this  
27 *year. It's certainly not in our control that it would be. But we need to get the*  
*partnership put in place, ideally a few months before we go to the end-of-Phase 2b*  
*meeting and start the Phase 3s, which starts in the second half of next year.*



1 138. RBC analyst Kantor issued a report on November 3, 2008, in which he wrote the  
2 following:

3 With recent safety concerns arising from ACR, a lucrative partnership would  
4 alleviate many investor concerns. Rigel forecasts completing a partnership in early  
2009, likely after completing the formal Qt study.

5 139. Oppenheimer analyst Abrahams issued a report on November 3, 2008, in which he  
6 wrote the following:

7 Importantly, mgmt reiterated that partnership discussions remain on track.

8 \* \* \*

9 Mgmt said R788's safety profile has had no impact on partnership  
10 discussions, timing, or deal structure. Mgmt noted term sheets are actively being  
11 exchanged, and have been since ACR. Importantly, while BP effects have been a  
point of discussion with partners, they have not been singled out as a particularly  
concerning side effect.

12 140. Similar reports were issued on November 4, 2008 by Jefferies analyst Walsh and SIG  
13 analyst Jellinek.

14 141. On February 3, 2009, however, Rigel reported that it would *delay* partnership  
15 discussions regarding R788 until after results from its Phase IIb clinical studies were available and  
16 that the Company laid off 36 employees, or 20% of its workforce. The price of the Company's stock  
17 declined 9.3% from \$7.17 on February 2, 2009 to \$6.50 on February 3, 2009, compared to a 1.8%  
18 *increase* in the peer group and a 1.5% *increase* in the NASDAQ. On July 9, 2009, Rigel reported  
19 that it would not conduct corporate partnership discussions until 2010.

20 142. **Defendants' compensation:** Gower, Grossbard, Payan, Rodriguez and Maynard also  
21 knew that they would receive higher salaries, bonuses and stock option awards, and that the value of  
22 their existing stock options would increase substantially, if Rigel reported positive results from the  
23 Phase IIa RA clinical trial. According to the Company's Proxy Statement filed with the SEC on  
24 April 8, 2008, Rigel's "performance driven compensation program" consisted of three components:  
25 base salary, short-term cash incentive compensation (*i.e.*, bonuses) and long-term equity incentive  
26 compensation. For 2007, the amount of each component was based upon the achievement of  
27 Company goals and objectives related to (i) the clinical development of Rigel's new product  
28

1 candidates; (ii) expansion of the Company's pipeline; and (iii) Rigel's cash position at the end of  
2 2007.

3 143. The April 8, 2008 Proxy Statement confirms that defendants Gower, Grossbard,  
4 Payan, Rodriguez and Maynard each received salary increases, bonuses and stock awards at the end  
5 of 2007 because Rigel achieved significant milestones with regard to clinical development, and the  
6 first significant milestone listed was the reported results of the Phase IIa RA clinical trial. As shown  
7 in the following chart, Gower, Grossbard, Payan, Rodriguez and Maynard each received substantial  
8 salary increases:

<b>Defendant</b>	<b>2007 Salary</b>	<b>2008 Salary</b>	<b>% Increase</b>
Gower	\$500,000	\$600,000	20%
Payan	\$420,000	\$483,000	15%
Grossbard	\$390,000	\$450,200	15.4%
Rodriguez	\$380,000	\$430,000	13.2%
Maynard	\$260,000	\$300,000	15.4%

9  
10  
11  
12  
13 144. In addition, it was reported in the April 8, 2008 Proxy Statement that individuals were  
14 eligible to receive a bonus of 0% to 60% of their 2007 base salary but that Gower, Grossbard, Payan,  
15 Rodriguez and Maynard each received bonuses that were substantially greater than 60% of their  
16 2007 base salaries due to the Company's exceptional success in 2007, including the reported results  
17 of the Phase IIa RA clinical trial. Further, it was reported in the April 8, 2008 Proxy Statement that  
18 while the stock price was not expressly stated as a goal under the 2007 non-equity incentive plan in  
19 determining bonuses, the \$25.39 closing price of Rigel's stock on December 31, 2007 was  
20 considered. As alleged above, the Company's stock price more than tripled from \$8 per share on  
21 December 12, 2007 to \$25.95 on December 13, 2007 as a result of defendants' false and misleading  
22 statements about the Phase IIa RA clinical trial. The following chart shows the bonuses received by  
23 Gower, Grossbard, Payan, Rodriguez and Maynard:

<b>Defendants</b>	<b>Bonus</b>	<b>% of 2007 Salary</b>
Gower	\$600,000	120%
Payan	\$420,000	100%
Grossbard	\$351,000	90%
Rodriguez	\$380,000	100%
Maynard	\$208,000	80%

1 145. Gower, Grossbard, Payan, Rodriguez and Maynard also received additional stock  
 2 awards on January 31, 2008. Further, it was reported in the April 8, 2008 Proxy Statement that  
 3 options generally vested over a four-year period from the date of the grant but that the options  
 4 granted in January 2008 vested monthly over a one-year period:

Defendants	Options Awarded in 1/07	Options Awarded in 1/08	% Increase
Gower	100,000	165,000	65%
Payan	80,000	140,000	75%
Grossbard	80,000	130,000	62.5%
Rodriguez	80,000	125,000	56.25%
Maynard	50,000 <sup>9</sup>	75,000	50%

9 146. The increase in the Company's stock price caused by the false and misleading  
 10 statements about the Phase IIa RA clinical trial also increased the value of the stock options owned  
 11 by Gower, Grossbard, Payan, Rodriguez and Maynard. As of December 31, 2007, Gower, Payan,  
 12 Grossbard, Rodriguez and Maynard owned options to purchase millions of the Company's shares.  
 13 Many of the options were "out-of-the-money" until the false and misleading statements about the  
 14 Phase IIa RA clinical trial caused the price of Rigel's stock to increase from \$8 per share on  
 15 December 12, 2007 to \$25.95 on December 13, 2007:

Defendant	Options	Exercise Price
<b>Gower</b>	50,000	\$1.80
	270,000	\$8.25
	80,000	\$17.66
	15,000	\$22.17
	200,000	\$24.56
	60,000	\$7.40

26 <sup>9</sup> Does not include the supplemental promotion option grant of 56,911 shares awarded to  
 27 Maynard upon his promotion from Vice President and Acting CFO to Vice President and CFO in  
 28 January 2007.

<b>Defendant</b>	<b>Options</b>	<b>Exercise Price</b>
	100,000	\$11.73
<b>Payan</b>	3,334	\$1.80
	250,000	\$8.25
	40,000	\$17.66
	11,250	\$22.17
	93,000	\$24.56
	55,000	\$7.40
	80,000	\$11.73

<b>Grossbard</b>	150,000	\$8.25
	35,000	\$17.66
	32,222	\$22.17
	65,000	\$24.56
	60,000	\$7.40
	27,778	\$9.56
	80,000	\$11.73

<b>Rodriguez</b>	150,000	\$8.25
	75,000	\$17.66
	12,500	\$22.17
	55,000	\$24.56
	65,000	\$7.40
	43,889	\$9.56
	80,000	\$11.73

<b>Maynard</b>	7,504	\$8.15
	11,895	\$23.00
	60,000	\$23.32
	4,600	\$7.88
	90,000	\$10.20
	1,000	\$9.56
	106,911	\$11.73

147. The exercise price of the options received on January 31, 2008 was \$26.45. The value of those options declined when the Company's stock price fell after the full results of the R788 Phase IIa RA clinical trial were disclosed on October 27, 2008. But Gower, Grossbard, Payan, Rodriguez and Maynard received additional options in 2009 with a substantially lower exercise price to replace the "out-of-the-money" options granted on January 31, 2008:

<b>Defendant</b>	<b>Options Awarded on 3/30/09</b>	<b>Exercise Price</b>
Gower	190,000	\$6.49
Payan	145,000	\$6.49
Grossbard	115,000	\$6.49
Rodriguez	115,000	\$6.49
Maynard	150,000	\$6.49

1           **E.      February 2008: Rigel’s Registration Statement Contains False and**  
2           **Misleading Statements About the Safety and Efficacy Results of the**  
3           **Phase IIa RA Clinical Trial**

4           148.    On or about January 24, 2008, Rigel filed with the SEC a Form S-3ASR Registration  
5           Statement and Form 424B3 Prospectus with the SEC for the Offering.    The Form S-3ASR  
6           Registration Statement was signed by Gower, Maynard, Payan, DeLeage, Goodwin, Lyons, Moos,  
7           Renton, Ringrose and Sherwin.

8           149.    On February 1, 2008, Rigel filed with the SEC a Form 424B5 Prospectus for the  
9           Offering.

10          150.    The Registration Statement contained untrue statements of material fact or omitted to  
11          state other facts necessary to make the statements made therein not misleading and was not prepared  
12          in accordance with applicable SEC rules and regulations.    Specifically, the Registration Statement  
13          provided “the following documents filed with the SEC are incorporated by reference . . . .    Our  
14          current report on Form 8-K, filed with the SEC on December 13, 2007.”    The Form 8-K Rigel filed  
15          with the SEC on December 13, 2007 included the December 13, 2007 press release quoted above at  
16          ¶60.

17          151.    In addition, the Form 424B5 Prospectus contained the following statements (false and  
18          misleading statements in bold and italics):

19                    We recently completed a Phase 2, multicenter, ascending dose, randomized,  
20                    double-blind, placebo-controlled, dose-ranging study evaluating three doses of R788  
21                    over a 12-week period in RA patients.    All of these patients continued to receive their  
22                    same previously scheduled dose of methotrexate.    ***In this clinical trial, R788***  
23                    ***demonstrated statistically significant efficacy results in treating RA patients at two***  
24                    ***dose levels.***    Efficacy assessments for each participant were based on the American  
25                    College of Rheumatology criteria which denote a 20% (ACR 20) improvement, at  
26                    least a 50% (ACR 50) improvement, or at least a 70% (ACR 70) improvement from  
27                    the baseline assessment at the end of the 12-week treatment period.    ***Groups treated***  
28                    ***with R788 at 100mg and 150mg po bid (orally, twice daily) showed higher ACR20,***  
                    ***ACR50, ACR70 and DAS28 response rates than the placebo group.***    ***The most***  
                    ***common clinically meaningful adverse events noted in the clinical trial were dose-***  
                    ***related neutropenia, mild elevations of liver function tests and gastrointestinal side***  
                    ***effects.***    Dose reduction (to one-half the assigned dose by taking the drug once per  
                    day) was pre-specified in the protocol and contingent on neutrophil counts and/or  
                    liver function tests.    Notably, a vast majority of the patients who had their dose  
                    reduced successfully completed the clinical trial with minimal safety issues.    We  
                    expect to initiate a Phase 2b clinical trial evaluating dosing and x-rays of bones over  
                    a 24-week period.    We also expect to initiate a second Phase 2b clinical trial treating  
                    a sub-population of RA patients with R788 by the end of the first half of 2008.

1           152. On February 6, 2008, at least 5 million shares of Rigel stock were sold to the public at  
2 \$27.00 per share, raising \$135 million.

3           153. **Reasons why the Registration Statement contained untrue statements of**  
4 **material fact or omissions:** As described in ¶¶68-90, Rigel, Gower, Grossbard, Payan, Rodriguez  
5 and Maynard knew that their presentation of, and statements about, the efficacy results of the Phase  
6 IIA RA clinical trial were materially false and misleading and that R788 had not “demonstrated  
7 statistically significant improvement efficacy results in treating RA patients at two dose levels  
8 [100mg and 150mg]” in the Phase IIA RA clinical trial. The Registration Statement was false  
9 because the defendants manipulated the statistical analysis of the Phase IIA RA clinical trial data by  
10 (i) pooling the data from the U.S. and Mexico prior to statistical analysis in contravention of basic  
11 statistical principals; (ii) improperly calculating p-values using the chi-squared analysis, as opposed  
12 to Fisher’s Exact test; and (iii) failing to account for the multiple comparisons problem. *See* ¶¶68-  
13 90; *see also*, Bloch Decl. at 5-12. Each of these manipulations caused the p-values reported to  
14 appear lower (*i.e.*, more significant) than was supported by the data. Properly analyzed, none of the  
15 efficacy results at the primary efficacy endpoint, ACR20, were statistically significant. *Id.* at 12-13.  
16 The p-values at ACR20 were “much bigger” than reported.

17           154. Not disclosing the data on a per-country basis in the Registration Statement,  
18 defendants also concealed (i) the existence of a country interaction so substantial that the placebo in  
19 Mexico was more effective than any dose in the U.S. at ACR20; (ii) that the distribution of doses  
20 was not balanced across the U.S. and Mexico, and (iii) that the appearance of an ascending dose  
21 response was attributable to the uneven does distribution and the country interaction. *See* ¶¶82-90.

22           155. Defendants’ statements in the Registration Statement about the safety results of the  
23 Phase IIA RA clinical trial in the offering document were also materially false and misleading  
24 because defendants knew but failed to disclose that (i) there was a dose-dependent increase in  
25 average systolic blood pressure of 3-5mm Hg in 100mg patients and 8-9mm Hg in the 150mg  
26 patients which increased both the clinical and regulatory risk; (ii) 5 patients (not 2, as reported on  
27 December 13, 2007) experienced hypertension, with blood pressure increases as high as 20-30mm  
28 Hg; (iii) hypertension, which was omitted from defendants list of common clinically meaningful

1 adverse events in the prospectus, was specifically admitted later as was one of the two most common  
2 clinically meaningful drug related adverse events; (iv) 9 patients (not 3, as reported on December 13,  
3 2007) experienced increased liver enzymes compared to patients taking the placebo; (v) 20 patients  
4 (not 15, as reported on December 13, 2007) experienced neutropenia; (vi) 34 patients (not 15, as  
5 reported on December 13, 2007) experienced diarrhea; and (vii) 35 patients (not 15, as reported on  
6 December 13, 2007) experienced upper gastrointestinal side effects. See ¶¶91-119. The undisclosed  
7 adverse events were material as they signaled the strength of the side effects, presented  
8 complications for regulatory approval, impacted the need for and design of future studies and  
9 jeopardized Rigel’s ability to obtain a partnership for the further development of R788 – *i.e.*, its  
10 investment thesis.

11           156. Defendants’ omission of the dose-dependent increase in mean blood pressure was  
12 particularly egregious in light of the fact that defendants specifically told the market in the February  
13 1, 2008 prospectus that: “*The most common clinically meaningful adverse events noted in the*  
14 *clinical trial were dose-related neutropenia, mild elevations of liver function tests and*  
15 *gastrointestinal side effects.*” A statement made less than two months after defendant Grossbard  
16 acknowledged concerns regarding blood pressure increases and at a time when defendants had the  
17 data regarding the clinically significant dose dependent increase in average blood pressure. ¶¶59,  
18 102, 114.

19           157. As reported in the Form 8-K filed with the SEC on February 14, 2008, the Rigel  
20 board of directors approved the bonuses awarded to Gower, Grossbard, Payan, Rodriguez and  
21 Maynard on February 11, 2008, just days after the Company raised \$127.5 million that it needed to  
22 prevent Rigel from becoming insolvent in 2008. Rigel and the Officer Defendants knew at the time  
23 of the Offering that the safety and efficacy of R788 was critical to Rigel’s success as well as their  
24 own compensation. ¶¶120-47.

25           158. Rigel paid the four Underwriter Defendants more than \$7 million to underwrite the  
26 Offering which failed to require disclosure of the adverse information about the Phase IIa RA  
27 clinical trial. According to Rigel’s January 31, 2008 press release, Credit Suisse was the sole book-  
28 runner for the Offering with Thomas Weisel, Jefferies and Oppenheimer, acting as co-managers.

1 According to the January 31, 2008 Underwriting Agreement between Rigel and the Underwriter  
2 Defendants, Rigel provided the Underwriter Defendants with a “General Disclosure Package” that  
3 included, among other things, a listing of the Company’s clinical trials and “descriptions of the  
4 results of the studies, tests and trials.”

5 **F. February 11, 2008: Gower Makes Materially False and Misleading**  
6 **Statements about the Phase IIa RA Clinical Trial During the BIO**  
7 **CEO & Investor Conference**

8 159. On February 11, 2008, at the BIO CEO & Investor Conference, defendant Gower  
9 made the following statements (false and misleading statements in bold and italics):

10 The Phase II study that we announced in December was a study on 190  
11 patients, double-blind, placebo-controlled in 30 centers in the US and Mexico. *We*  
12 *saw rather unprecedented numbers in terms of the ACR scoring. As you can see*  
13 *on the chart, significantly different as is noted by the stars in both the 100*  
14 *milligram orally BID dose and 150 milligram orally BID dose across the board and*  
15 *all of ACR20, ACR50, ACR70 and DAS scoring. Rather spectacular numbers for*  
16 *the higher two dose groups specifically in the ACR50’s and ‘70s where we got*  
17 *between 50 and 60% ACR50 response and over one-third ACR70’s at 90 days*  
18 *which is relatively unprecedented in these kind of studies if you want to look at*  
19 *previous studies done in these same populations with the same protocol.*

20 This was a very strict intense treat protocol. And done using the same  
21 protocols that have been used for pretty much everything from Enbrel on forward,  
22 certainly the same protocols and the same, some of the same groups used in the  
23 studies done in the last few years with Rituxan and Orencia for approvals IL-6 and  
24 the JAK3’s in terms of study. So you can never compare studies directly one-to-one  
25 that aren’t done in exactly the same time but these are using the same protocols and  
26 the same approach so they should be roughly comparable.

27 *The safety results were also good. We did have two dose dependent*  
28 *toxicities that were noted. One was neutropenia, which we’ve known from the*  
29 *animal studies on forward that we carry a certain amount of neutropenia along with*  
30 *the mechanism of this growth comes most likely from its ability to regulate adhesion*  
31 *molecules and the monocytes. And there you are seeing a dose dependent matter that*  
32 *increased from about slightly under 10% to just under 20% of between the higher*  
33 *two dose groups.*

34 We had prespecified a protocol based dose reduction, which cut the dose in  
35 half for any patients that got a grade 2 neutropenia. This is a neutrophil count of  
36 1500. We didn’t see any grade 3 or grade 4 neutropenias in the study, and as many  
37 of you know those are the ones that are associated with infections. But because this  
38 was an early study we wanted to be extra cautious and we cut the dose in half. But  
39 when those patients hit a neutrophil count of 1500, all of those patients however did  
40 fine on the reduced dose. Actually we got, if you look at those as a group although  
41 we didn’t – this is not prespecified as a statistical endpoint, their ACR20 at 90 days  
42 was 82% and those that continued on the study with the dose reductions. So they did  
43 quite well and maintained the efficacy and the neutropenia has not recurred nor has  
44 anyone dropped off the study because of neutropenia. But it is something which is  
45 not uncommon for this patient population. As many of you know, RA patients are

1 predisposed to neutropenia. Methotrexate adds to it. Wheat appears added to that.  
2 That is something the rheumatologists have to watch but doesn't seem at this point to  
be something that is not manageable.

3 *The other thing that we saw that seems dose-related was lower GI*  
4 *disturbance*, also something fairly common in this disease. Methotrexate alone as  
5 you would notice in the placebo group, those were all methotrexate plus a dummy  
6 788, has a number of patients that have lower GI symptoms. We had a modest  
7 number in the intermediate dose group, slightly higher number in the upper dose  
8 group. As with the neutropenia no patients found this uncomfortable enough to want  
9 to drop off the study. None were hospitalized. None had to be rehydrated. But  
10 certainly it is a tolerance issue. *Everything else that showed up is no different*  
11 *between the placebo group and the control group on the safety elements of the*  
12 *study. So, so far, so good.*

13 160. **Reasons why Gower knew his statements at the BIO CEO & Investor**  
14 **Conference were materially false and misleading:** As described in ¶¶68-90, Gower, knew that his  
15 presentation of, and statements about, the efficacy results of the Phase IIa RA clinical trial were  
16 materially false and misleading and that R788 had not demonstrated “unprecedented” or  
17 “significant[]” results or “Rather spectacular numbers” in the Phase IIa RA clinical trial. In fact,  
18 R788 showed statistically insignificant improvement over placebo in the Phase IIa RA clinical trial.  
19 Bloch Decl. at 5-12.

20 161. As discussed in ¶¶68-90, defendants manipulated the statistical analysis of the Phase  
21 IIa RA clinical trial data including by:

22 (a) **Improperly pooling the data from the U.S. and Mexico:** Basic statistical  
23 principals require (i) that the data for the U.S. and Mexico be analyzed separately, with the  
24 calculation of two p-values; and (ii) then combining the two p-values using Fisher's method to  
25 produce an overall p-value for the combined U.S. and Mexico data. Bloch Decl. at 5-9. Correcting  
26 solely for the improperly pooled data, the p-values for the 100mg and 150mg doses at ACR20 and  
27 ACR50 are all higher (*i.e.*, indicating less significance) than those falsely reported on December 13,  
28 2007 and February 11, 2008, and R788 did not achieve statistically significant results at the primary  
efficacy endpoint, ACR20, for the 150mg dose. *Id.* at 9; *see* ¶¶75-77.

(b) **Improperly applying the chi-squared analysis:** In addition to pooling the  
data prior to analyzing it, defendants used the chi-squared analysis to calculate p-values. Because of  
the small sample size, the proper analysis required using Fisher's Exact test to obtain p-values,

1 which are all larger (*i.e.*, indicating less significance) than the p-values obtained by the chi-squared  
2 analysis. Bloch Decl. at 9. Correcting for the improper pooling of the data and applying Fisher’s  
3 Exact test, the p-values for the 100mg and 150mg doses at ACR20, ACR50 and ACR70 are all  
4 substantially higher (*i.e.*, indicating less significance) than those falsely reported on December 13,  
5 2007 and February 11, 2008; further R788 did not achieve statistically significant results at ACR20  
6 for the 100mg or 150mg doses as defendants had claimed. *See* ¶¶78-79.

7           (c)     **Failing to account for the multiple comparisons problem:** Defendants  
8 made multiple pair-wise comparisons between groups – *i.e.*, 100mg versus placebo at ACR20,  
9 ACR50, ACR70 and DAS28 – which increased the likelihood that one comparison would be  
10 statistically significant by chance alone. Bloch Decl. at 11-12. Analyzed appropriately with Tukey’s  
11 Studentized Range test on a per-country basis – the p-values at the primary efficacy endpoint,  
12 ACR20 for 100mg and 150mg were very large and not statistically significant. *See* ¶¶80-81.

13           162.    Thus, contrary to Gower’s claims that R788 had demonstrated “unprecedented” or  
14 “significant[.]” results or “Rather spectacular numbers” in the Phase IIa RA clinical trial, R788 had in  
15 fact failed to show statistically significant improvement over placebo.

16           163.    Moreover, by failing to disclose the data on a per-country basis on December 13,  
17 2007 or in subsequent statements, defendants also concealed (i) the existence of a country interaction  
18 so substantial that the placebo in Mexico was more effective than any dose in the U.S. at ACR20  
19 (¶¶69-70); (ii) that the dose distribution was not balanced across the U.S. and Mexico (¶¶69-70); and  
20 (iii) that the appearance of an ascending dose response was attributable to the uneven does  
21 distribution and the country interaction (¶¶69-70; 82-90).

22           164.    Further, Gower’s statements about the safety results of the Phase IIa RA clinical  
23 study were materially false and his affirmative statement misleading, in particular his omission of  
24 hypertension as a dose-dependent side effect and his affirmative statement that safety results  
25 appeared “good.” Gower knew but failed to disclose that (i) there was a dose-dependent increase in  
26 average systolic blood pressure of 3-5mm Hg in 100mg patients and 8-9mm Hg in the 150mg  
27 patients which increased both the clinical and regulatory risk; (ii) 5 patients (not 2, as reported on  
28 December 13, 2007) experienced hypertension, with blood pressure increases as high as 20-30mm

1 Hg; (iii) hypertension was one of the two most common clinically meaningful drug related adverse  
2 events; (iv) 9 patients (not 3, as reported on December 13, 2007) experienced increased liver  
3 enzymes compared to patients taking the placebo; (v) 20 patients (not 15, as reported on December  
4 13, 2007) experienced neutropenia; (vi) 34 patients (not 15, as reported on December 13, 2007)  
5 experienced diarrhea; and (vii) 35 patients (not 15, as reported on December 13, 2007) experienced  
6 upper gastrointestinal side effects. The undisclosed adverse events were material as they signaled  
7 the strength of the side effects, presented complications for regulatory approval, impacted the need  
8 for and design of future studies and jeopardized Rigel's ability to obtain a partnership for the further  
9 development of R788 – *i.e.*, its investment thesis. Gower knew that R788 was critical to Rigel's  
10 success and his own compensation. ¶¶120-47.

11 **G. July 8, 2008: Rodriguez Makes Materially False and Misleading**  
12 **Statements About the Phase IIa RA Clinical Trial During the Collins**  
13 **Stewart 4th Annual Growth Conference**

14 165. On July 8, 2008, at the Collins Stewart 4th Annual Growth Conference, defendant  
15 Rodriguez made the following statements (false statements in bold and italic):

16 Speaking of that, we last year started – reported a Phase II RA clinical trial.  
17 This is the data we reported in December of last year. This is a three-month study  
18 looking at R788 in patients with active RA all on a methotrexate background. It's a  
19 three-month study looking at those signs and symptoms.

20 ***What we saw, and you see in this graph, is that we had some dramatic***  
21 ***improvement in the signs and symptoms looking at ACR20, ACR50, and ACR70 at***  
22 ***the 100 milligram and the 150 milligram dose groups.*** This is all b.i.d. The 50  
23 looked pretty much like placebo. ***The others looked quite dramatic[]***.

24 In fact compared to other TNF agents or other products that are in the market  
25 now or in development now, this is in the higher range of those efficacy measures.  
26 So very dramatic improvement. We also saw a couple of things that we saw the  
27 benefit occur within the first two weeks of therapy. That is, even within the first  
28 week, we are able to see a dramatic improvement in signs and symptoms into the  
trial. That was sustained throughout the three months of the trial. So very nice  
results. Per the protocol, if we ran into any trouble with say neutropenia or elevated  
liver enzymes, the protocol required us to cut the dose in half. That is what occurred  
in a few cases.

29 ***You see some of the safety background on these various doses in this chart.***  
30 ***We had some cases of neutropenia, five in the 100 milligram and 10 in the 150***  
31 ***milligram dose groups that required the dose to be reduced.*** A few liver enzymes  
32 elevated in 150 milligram. I should note that all the patients that had their dosage  
33 reduced, about 18 of them, completed the trial and their ACR20 scores, 82% of them  
34 met their ACR20 scores. So they had a very nice benefit even though their dose was  
reduced.

1 So effectively, if you had a benefit it occurred early in the trial and then if  
2 you needed your dose reduced it didn't seem to undermine the benefit that you did  
3 receive. So we were very satisfied with this. *We had some GI side effects and they  
4 were somewhat random and transient, more in the 150 than the 100. A bit of  
5 hypertension here and there, but, basically, a fairly good safety profile.*

6 *The 100 milligram dose group had a very nice and profound efficacy result  
7 and a pretty good safety profile.* So that is going to be the lead dose that we go  
8 forward. However, the drug does have a very good PKA; we have about a 17-hour  
9 half-life. So we are going to try to push that a little bit and see if once a day works.

10 166. **Reasons why Rodriguez knew his statements at the Collins Stewart 4th Annual  
11 Growth Conference were materially false and misleading:** As described in ¶¶68-90, Rodriguez,  
12 knew that his presentation of, and statements about, the efficacy results of the Phase IIa RA clinical  
13 trial were materially false and misleading and that patients taking R788 had not shown “dramatic  
14 improvement” or that there was “profound efficacy” results in the Phase IIa RA clinical trial. In fact,  
15 R788 showed statistically insignificant improvement over placebo in the Phase IIa RA clinical trial.  
16 Bloch Decl. at 12-13.

17 167. As discussed in ¶¶68-90, defendants manipulated the statistical analysis of the Phase  
18 IIa RA clinical trial data by:

19 (a) **Improperly pooling the data from the U.S. and Mexico:** Basic statistical  
20 principals require that (i) the data for the U.S. and Mexico be analyzed separately, with the  
21 calculation of two p-values; and (iii) then in combining the two p-values using Fisher's method to  
22 produce an overall p-value for the combined U.S. and Mexico data. *Id.* at 5-9. Correcting solely for  
23 the improperly pooled data, the p-values for the 100mg and 150mg doses at ACR20 and ACR50 are  
24 all higher (*i.e.*, indicating less significance) than those falsely reported on December 13, 2007 and  
25 July 8, 2008 and R788 did not achieve statistically significant results at the primary efficacy  
26 endpoint, ACR20, for the 150mg dose. *See* ¶¶75-77.

27 (b) **Improperly applying the chi-squared analysis:** In addition to pooling the  
28 data prior to analyzing it, defendants used the chi-squared analysis to calculate p-values. Because of  
the small sample size, the proper analysis required using Fisher's Exact test to obtain p-values,  
which are all larger (*i.e.*, indicating less significance) than the p-values obtained by the chi-squared  
analysis. Bloch Decl. at 9-11. Correcting for the improper pooling of the data and applying Fisher's

1 Exact test, the p-values for the 100mg and 150mg doses at ACR20, ACR50 and ACR70 are all  
2 substantially higher (*i.e.*, indicating less significance) than those falsely reported on December 13,  
3 2007 and July 8, 2008, and R788 did not achieve statistically significant results at ACR20 for the  
4 100mg or 150mg doses as defendants had claimed. *See* ¶¶78-79.

5 (c) **Failing to account for the multiple comparisons problem:** Defendants  
6 made multiple pair-wise comparisons between groups – *i.e.*, 100mg versus placebo at ACR20,  
7 ACR50, ACR70 and DAS28 – which increased the likelihood that one comparison would be  
8 statistically significant by chance alone. Bloch Decl. at 11-12. Analyzed appropriately with Tukey’s  
9 Studentized Range test on a per-country basis – the p-values at the primary efficacy endpoint,  
10 ACR20 for 100mg and 150mg were very large and not statistically significant. *Id.* at 11-13. *See*  
11 ¶¶80-81. Thus, the 100 mg dose group did not have a “very nice and profound efficacy result” or  
12 demonstrate “dramatic improvement.”

13 168. Further, by failing to disclose the data on a per-country basis on December 13, 2007,  
14 or in subsequent statements, defendants also concealed (i) the existence of a country interaction so  
15 substantial that the placebo in Mexico was more effective than any dose in the U.S. at ACR20 (¶¶69-  
16 70); (ii) that the dose distribution was not balanced across the U.S. and Mexico (¶¶69-70); and (iii)  
17 that the appearance of an ascending dose response was attributable to the uneven does distribution  
18 and the country interaction (¶¶69-70; 82-90).

19 169. Rodriguez’s July 8, 2008 statements about the safety results of the Phase IIa RA  
20 clinical trial were also materially false and misleading. In particular he deliberately down played the  
21 adverse hypertension results: “A of bit hypertension here and there, but, basically a fairly good  
22 safety profile.” Rodriguez knew but failed to disclose that (i) there was a dose-dependent increase in  
23 average systolic blood pressure of 3-5mm Hg in 100mg patients and 8-9mm Hg in the 150mg  
24 patients which increased both the clinical and regulatory risk; (ii) 5 patients (not 2, as reported on  
25 December 13, 2007) experienced hypertension, with blood pressure increases as high as 20-30mm  
26 Hg; (iii) hypertension was one of the two most common clinically meaningful drug related adverse  
27 events; (iv) 9 patients (not 3, as reported on December 13, 2007) experienced increased liver  
28 enzymes compared to patients taking the placebo; (v) 20 patients (not 15, as reported on December

1 13, 2007) experienced neutropenia; (vi) 34 patients (not 15, as reported on December 13, 2007)  
2 experienced diarrhea; and (vii) 35 patients (not 15, as reported on December 13, 2007) experienced  
3 upper gastrointestinal side effects. Thus, R788 did not have a “pretty good safety profile” as  
4 Rodriguez touted to investors. The undisclosed adverse events were material as they signaled the  
5 strength of the side effects, presented complications for regulatory approval, impacted the need for  
6 and design of future studies and jeopardized Rigel’s ability to obtain a partnership for the further  
7 development of R788 – *i.e.*, its investment thesis. Rodriguez knew that R788 was critical to Rigel’s  
8 success and his own compensation. ¶¶120-47.

9 **H. October 27, 2008: Rigel’s Stock Price Declines Substantially After It**  
10 **Begins to Reveal Previously Concealed Adverse Information About**  
11 **the Phase IIa RA Clinical Trial and Its Impact on the Company’s**  
12 **Condition; Defendants Continue to Mislead Investors About the**  
13 **Prospectus for a Partner**

14 170. On October 27, 2008, the Company presented additional results of the Phase IIa RA  
15 clinical trial at the ACR meeting and during a conference call that was attended by Gower,  
16 Grossbard, Payan, Rodriguez and Maynard. The Company disclosed the differing response rates  
17 between patients in the U.S. and Mexico and additional adverse safety data regarding R788’s effect  
18 on blood pressure, liver enzymes, neutropenia, diarrhea and gastrointestinal side effects.

19 171. In response to this previously undisclosed negative information, the price of the  
20 Company’s stock declined 38% from \$14.41 on October 24, 2008 to \$8.84 on October 27, 2008.  
21 Analysts following the Company issued reports in which they wrote that the previously undisclosed  
22 negative information raised questions about the efficacy and safety of the drug and caused the stock  
23 price to plummet.

24 172. In an October 28, 2008 report, RBC analyst Jason Kantor downgraded the stock due  
25 to “heightened safety concerns for R788,” and noted that (i) the impact of the Mexican data may  
26 have overstated the dose response; (ii) the previously undisclosed increase in blood pressure was  
27 viewed as a “potentially significant concern” to independent physicians attending the October 27,  
28 2008 ACR conference; and (iii) the new negative information caused one pharmaceutical company  
to walk away from a potential partnership with Rigel. ¶¶72, 102.

1           173. Similar reports were issued by SIG analyst Jellinek, Oppenheimer analyst Abrahams,  
2 Jefferies analyst Walsh, Merrill Lynch analyst Andrew Berens and Credit Suisse analyst Aberman.  
3 Credit Suisse analyst Aberman reported that Rigel had presented the differences in efficacy in  
4 Mexico versus the U.S. for the first time and that it was a particular concern because the ratio of  
5 Mexican patients to U.S. patients was higher in the higher dosing groups which could skew the data  
6 in favor of R788 (which in fact it did). ¶73. He also reported that the magnitude of the increase in  
7 blood pressure was disclosed for the first time and that there was no question the increase in blood  
8 pressure was one of the risks of the program. Aberman wrote that it was an issue because of the  
9 FDA's increased scrutiny over cardiac toxicity and the well known association of elevated blood  
10 pressure with cardiac events. He also wrote that one investigator suggested that the elevated blood  
11 pressure would be a show stopper clinically.

12           174. Merrill Lynch analyst Andrew Berens reported that the detailed presentation revealed  
13 dose-related blood pressure increase with R788, an imbalance in response rates noted at the Mexico  
14 trial sites, and more granularity on elevated liver enzymes noted with R788, which were likely to  
15 increase regulatory risk for the drug and which could delay a partnership with a large  
16 pharmaceutical/biotech company.

17           175. On November 3, 2008, Rigel reported its financial results for the quarter ending  
18 September 30, 2008. The Company also held its first ever earnings conference call but the focus of  
19 the call was the toxicity concerns with R788 following the ACR presentation. Analysts following  
20 the Company asked numerous questions about the increase in blood pressure and then issued reports.  
21 Credit Suisse analyst Aberman issued a report on November 3, 2008 in which he wrote that “[b]ased  
22 on the questions on the call, investors clearly remain wary over the toxicity profile of R788 and we  
23 think this may not wane until (1) a commercial partnership is signed in 1H09, and/or (2) Phase IIb  
24 data are released in 3Q09.” He also wrote that “There is no question that the elevated blood pressure  
25 seen in the Phase IIa is a risk for the long term prospects of R788.”

26           176. While defendants disclosed the per-country data and additional adverse safety results  
27 on October 27, 2008, defendants continued to mislead investors by falsely assuring them that Rigel  
28

1 was “still on track” for putting a partnership in place in the early part of 2009 (false and misleading  
2 statements are in bold and italic):

3 [Gower:] ***[S]till on track for what we’ve been saying all along, which is putting the***  
4 ***partnership in place as early as the early part of next year.*** I doubt it will be this  
5 year. It’s certainly not in our control that it would be. But we need to get the  
6 partnership put in place, ideally a few months before we go to the end-of-Phase 2b  
7 meeting and start the Phase 3s, which starts in the second half of next year.

8 177. On November 3, 2008, defendants also falsely stated:

9 [Gower:] We remain committed to doing everything possible to develop and  
10 commercialize R788 in RA. ***We expect to establish a collaboration partnership to***  
11 ***further these ends, and that in fact is going quite well.***

12 178. In fact, Rigel and the Officer Defendants knew that the Phase IIa RA clinical trial  
13 results had derailed the Company’s partnership prospects. Defendants admitted on the October 27,  
14 2008 conference call that Rigel’s potential partners had the underlying data related to the trial.  
15 Defendants knew that the data would inform potential partners that R788, based on the Phase IIa RA  
16 clinical trial, did not demonstrate statistically significant results at the primary efficacy endpoint.  
17 ¶¶68-90. Any potential partner of Rigel’s, before making a substantial financial commitment (*i.e.*, in  
18 excess of \$100 million), would want demonstrated efficacy and would perform its own due diligence  
19 with respect to the data. Thus, Rigel was not on track for partnership because it had demonstrated  
20 statistically significant results at the primary efficacy endpoint to its potential partners.

21 179. The R788 per-country and additional safety data was made available to the investors  
22 on October 27, 2008, alerting the investment community to efficacy concerns and increased toxicity  
23 results. Subsequently an *Arthritis and Rheumatism* article entitled “Treatment of Rheumatoid  
24 Arthritis With a Syk Kinase Inhibitor: A Twelve-Week, Randomized, Placebo-Controlled Trial,”  
25 Vol. 58, No. 11, was published in November 2008. The article contained the same improper  
26 conclusions that R788 was effective made by defendants throughout the Class Period, as well as  
27 inaccurately claiming that R788 showed statistically significant changes in certain biomarkers, IL-6  
28 and MMP-3, which are thought to be associated with joint damage progression. Bloch Decl. at 13-  
18. The Phase IIa RA clinical trial was less robust than the article claimed, certain secondary  
efficacy endpoints (including DAS28 response rates and the IL-6 and MMP-3 biomarkers) were  
skewed in R788’s favor and did not exhibit statistically significant results, as claimed. *Id.* All

1 conclusions that Rigel's potential partners likely had already drawn from the data in the course of  
2 their due diligence.

3 **I. February 3, 2009: Rigel Stock Price Declines Significantly After It**  
4 **Reveals a Delay in Partnership Discussions Regarding R788**

5 180. On February 3, 2009, Rigel finally announced, what defendants already knew  
6 internally, that it would delay partnership discussions regarding R788 until after results from its  
7 Phase IIb clinical studies were available and that the Company laid off 36 employees, or 20% of its  
8 workforce. The price of the Company's stock declined 9.3%, from \$7.17 on February 2, 2009 to  
9 \$6.50 on February 3, 2009, compared to a 1.8% *increase* in the peer group and a 1.5% *increase* in  
10 the NASDAQ.

11 **V. LOSS CAUSATION**

12 181. The false and misleading statements and omissions caused and maintained the  
13 artificial inflation in Rigel's stock price throughout the Class Period. During the Class Period,  
14 defendants made materially false and misleading statements about the Phase IIa RA clinical trial,  
15 failed to disclose adverse data related to the clinical trial and the effect of the clinical trial on its  
16 prospect for a partner.

17 182. The false and misleading statements and omissions caused Rigel's stock to trade at  
18 artificially inflated prices. After the Company reported the false and misleading results of the Phase  
19 IIa RA clinical trial on December 13, 2007, Rigel stock price more than tripled, from \$8 per share on  
20 December 12, 2007 to \$25.95 per share on December 13, 2007, compared to a 0.9% *decline* in the  
21 peer group and a 0.1% *decline* in the NASDAQ. The stock continued to trade at artificially inflated  
22 prices, which allowed the Company to issue 5 million shares for \$27 per share in February 2008.

23 183. Rigel made two partial disclosures on October 27, 2008 and February 3, 2009 that  
24 disclosed some of the previously concealed problems and some of the impact those problems were  
25 having on Rigel's financial condition and would have on the Company's future results. The partial  
26 disclosures caused Rigel's stock price to decline significantly more than the changes in the peer  
27 group and the NASDAQ. As a result, the price declines following the partial disclosures provide a  
28 measurement of class members' economic losses. Indeed, investors were alerted to the fact that the

1 defendants may have previously overstated the efficacy results in Phase IIa RA clinical trial and that  
2 there were additional toxicity results, including a dose dependent increase in blood pressure.

3 184. On October 27, 2008, Rigel disclosed additional results of the Phase IIa RA clinical  
4 trial, including the data on a per-country basis, which revealed that the prior representations of the  
5 safety and efficacy results of the Phase IIa RA clinical trial were materially false and misleading  
6 including (i) the existence of a country interaction so substantial that the placebo was more effective  
7 than any dose in the U.S. at ACR20; (ii) that the distribution of doses was not balanced across the  
8 U.S. and Mexico; and (iii) that the efficacy results may have been overstated based on the country  
9 interaction. Defendants also revealed additional safety results of the Phase IIa RA clinical trial  
10 including that (i) there was a dose-dependent increase in average systolic blood pressure of 3-5mm  
11 Hg in 100mg patients and 8-9mm Hg in the 150mg patients which increased both the clinical and  
12 regulatory risk; (ii) 5 patients (not 2, as reported on December 13, 2007) experienced hypertension,  
13 with blood pressure increases as high as 20-30mm Hg; (iii) 9 patients (not 3, as reported on  
14 December 13, 2007) experienced increased liver enzymes compared to patients taking the placebo;  
15 (iv) 20 patients (not 15, as reported on December 13, 2007) experienced neutropenia; (v) 34 patients  
16 (not 15, as reported on December 13, 2007) experienced diarrhea; and (vi) 35 patients (not 15, as  
17 reported on December 13, 2007) experienced upper gastrointestinal side effects.

18 185. On this news, Rigel's stock dropped 38.7% from the previous day's closing price of  
19 \$14.41, to \$8.84. By comparison, the peer group declined 5.3% and the NASDAQ declined 3%.  
20 Thus, some of the inflation of Rigel's stock price was removed upon the negative disclosures,  
21 causing economic loss (damages) to investors.

22 186. However, the Company's stock price remained artificially inflated because Rigel did  
23 not reveal the full extent of the Phase IIa RA clinical trial results on the Company's ability to partner  
24 with a pharmaceutical company for the continued development of R788. In fact, during the October  
25 27, 2008 and November 3, 2008 conference calls, Gower assured investors that Rigel was still on  
26 track for putting a partnership in place in the early part of 2009 and the establishment of a  
27 partnership was "going quite well."

28

1           187. On February 3, 2009, however, Rigel disclosed that it would delay partnership  
2 discussions regarding R788 until after results from its Phase IIb clinical studies were available and  
3 that the Company laid off 36 employees, or 20% of its workforce. The price of the Company's stock  
4 declined 9.3%, from \$7.17 on February 2, 2009 to \$6.50 on February 3, 2009, compared to a 1.8%  
5 increase in the peer group and a 1.5% increase in the NASDAQ.

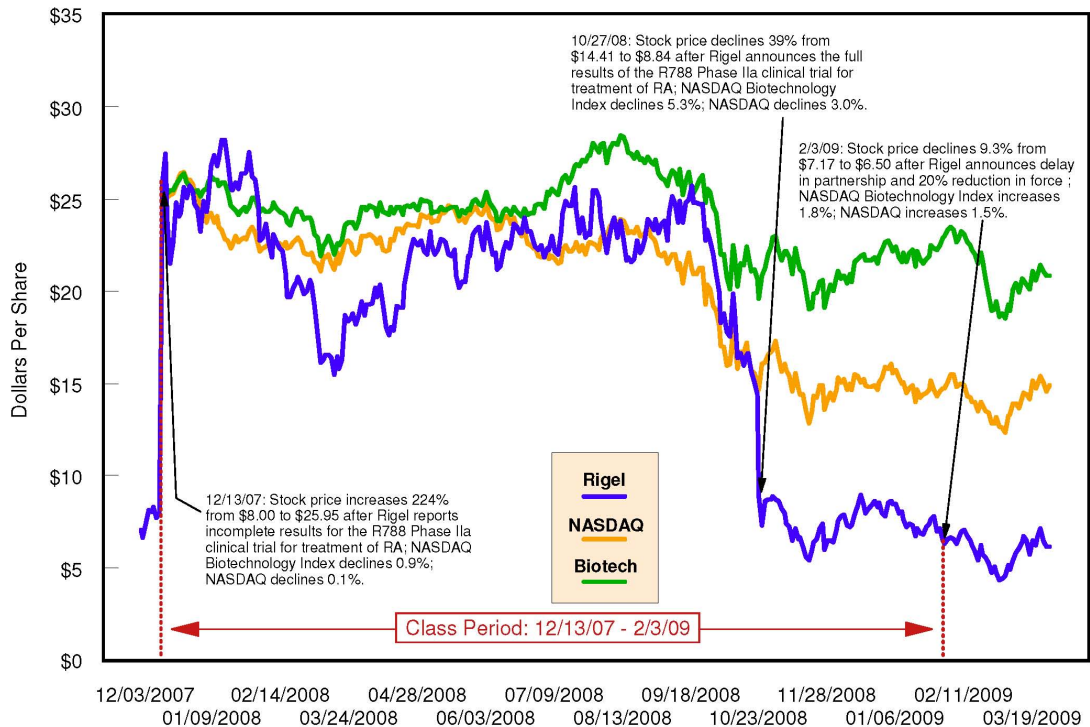
6           188. The declines in Rigel's stock price as the negative news was incrementally disclosed,  
7 revealing that the Company's condition was not as previously reported, were directly related to  
8 defendants' prior misrepresentations and omissions. Each partial disclosure of adverse facts that  
9 removed inflation from Rigel's stock price (thereby causing damages) was directly related to the  
10 false statements and omissions about the Phase IIa RA clinical trial and their impact on the  
11 Company's financial condition and future prospects.

12           189. The declines in Rigel's stock price following the partial disclosures compared to the  
13 changes in the peer group and NASDAQ negate any inference that the losses suffered by class  
14 members were caused by changed market or industry conditions or Company-specific facts unrelated  
15 to the fraudulent conduct. The following chart (which is also attached hereto) illustrates the changes  
16 in Rigel's stock price during the Class Period compared to the peer group and the NASDAQ:

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# Rigel Pharmaceuticals, Inc.

Indexed vs NASDAQ Composite, NASDAQ Biotechnology Indices  
October 1, 2007 to October 31, 2007



## VI. FEBRUARY 2008 OFFERING: VIOLATIONS OF THE 1933 ACT

190. On or about January 24, 2008, Rigel filed with the SEC a Form S-3ASR Registration Statement and Form 424B3 Prospectus with the SEC for the Offering. The Form S-3ASR Registration Statement was signed by Gower, Maynard, Payan, DeLeage, Goodwin, Lyons, Moos, Renton, Ringrose and Sherwin. On February 1, 2008, Rigel filed with the SEC a Form 424B5 Prospectus for the Offering. In connection with the Offering the Underwriter Defendants drafted and disseminated the Registration Statement.

191. The Registration Statement contained untrue statements of material fact or omitted to state other facts necessary to make the statements made therein not misleading and was not prepared in accordance with applicable SEC rules and regulations. Specifically, the Registration Statement provided “the following documents filed with the SEC are incorporated by reference . . . . Our current report on Form 8-K, filed with the SEC on December 13, 2007.” The Form 8-K Rigel filed

1 with the SEC on December 13, 2007 included false statements in the December 13, 2007 press  
2 release quoted above at ¶60.

3 192. For purposes of the 1933 Act claims, plaintiff alleges that defendants made false and  
4 misleading representations in the December 13, 2007 press release, part of the Registration  
5 Statement. The false statements in the December 13, 2007 press release are quoted in ¶60 and are  
6 summarized as follows:

- 7 • The p-values provided in the December 13, 2007 press release – i.e. “(p=.008),”  
8 “(p=.002)” and “(p<.001)” [as depicted in the table “Efficacy Results.”]
- 9 • “Rigel’s R788 Demonstrates Significant Improvement in Rheumatoid Arthritis in  
10 Phase IIa Clinical Study; Achieves Statistically Significant ACR20, ACR50 &  
11 ACR70 Results.”
- 12 • “Rigel Pharmaceuticals, Inc. . . . today announced that its oral syk kinase inhibitor,  
13 R788 (*tamatinib fosdium*), has demonstrated statistically significant results in treating  
14 Rheumatoid Arthritis (RA) patients in a recently completed Phase 2 clinical trial.”
- 15 • “We believe that the significant ACR scores and good tolerability observed in this  
16 clinical trial, and the further benefit of oral delivery may make R788 a favorable  
17 alternative to the currently marketed biological agents.”
- 18 • “This clinical study has shown that R788 treatment can achieve impressive ACR  
19 response rates, . . . In this clinical trial both the 100mg and 150mg doses improved  
20 arthritis symptoms and did so quickly.”
- 21 • “These very important clinical trial results are a major milestone for Rigel as we  
22 establish the potential of R788 in RA and its value as an alternative to current  
23 therapies.” “The most common clinically meaningful adverse events noted in the  
24 clinical trial were dose-related neutropenia, mild elevations of liver function tests,  
25 and gastrointestinal (GI) side effects.”
- 26 • The number of patients who experienced hypertension, increased liver enzymes,  
27 neutropenia, diarrhea, and upper gastrointestinal side effects [as depicted in the table  
28 of “Safety Results.”]

193. The Form 424B5 Prospectus contained the following additional false and misleading  
statements:

We recently completed a Phase 2, multicenter, ascending dose, randomized,  
double-blind, placebo-controlled, dose-ranging study evaluating three doses of R788  
over a 12-week period in RA patients. All of these patients continued to receive their  
same previously scheduled dose of methotrexate. ***In this clinical trial, R788  
demonstrated statistically significant efficacy results in treating RA patients at two  
dose levels.*** Efficacy assessments for each participant were based on the American  
College of Rheumatology criteria which denote a 20% (ACR 20) improvement, at  
least a 50% (ACR 50) improvement, or at least a 70% (ACR 70) improvement from  
the baseline assessment at the end of the 12-week treatment period. ***Groups treated  
with R788 at 100mg and 150mg po bid (orally, twice daily) showed higher ACR20,***

1 ***ACR50, ACR70 and DAS28 response rates than the placebo group. The most***  
2 ***common clinically meaningful adverse events noted in the clinical trial were dose-***  
3 ***related neutropenia, mild elevations of liver function tests and gastrointestinal side***  
4 ***effects.*** Dose reduction (to one-half the assigned dose by taking the drug once per  
5 day) was pre-specified in the protocol and contingent on neutrophil counts and/or  
6 liver function tests. Notably, a vast majority of the patients who had their dose  
7 reduced successfully completed the clinical trial with minimal safety issues. We  
8 expect to initiate a Phase 2b clinical trial evaluating dosing and x-rays of bones over  
9 a 24-week period. We also expect to initiate a second Phase 2b clinical trial treating  
10 a sub-population of RA patients with R788 by the end of the first half of 2008.

11 194. On February 6, 2008, at least 5 million shares of Rigel stock were sold to the public at  
12 \$27.00 per share, raising \$135 million.

13 195. Inter-Local Pension Fund GCC/IBT acquired the common stock of Rigel pursuant or  
14 traceable to the Offering.

15 196. **Reasons why the Registration Statement contained untrue statements of**  
16 **material fact or omissions:** The presentation of, and the statements about, the efficacy results of the  
17 Phase IIa RA clinical trial in the Registration Statement were materially false and misleading.  
18 Contrary to the representations in the Registration Statement, R788 did not achieve statistically  
19 significant efficacy results at the primary efficacy endpoint, ACR20, for any dose. Bloch Decl. 12-  
20 13. Further, the p-values given in the Registration Statement were false (*i.e.*, show greater statistical  
21 significance than the results support) because they were not computed using basic principals of  
22 statistics. *Id.* at 5-13.

23 197. The Phase IIa RA clinical trial data presented in the Registration Statement was also  
24 false for the following reasons:

25 (a) **The statements of efficacy in the Registration Statement were based upon**  
26 **improperly pooled data.** Basic statistical principals requires that the data for the U.S. and Mexico  
27 be analyzed separately and that two p-values be calculated. These p-values are to then be combined  
28 using Fisher's method to produce an overall p-value for the pooled U.S. and Mexico data. *Id.* at 5-6.  
Application of this basic statistical principal (but not correcting for the improper chi-squared test or  
multiple comparison problem, described below) renders the p-values for the 100mg and 150mg  
doses at ACR20 and ACR50 all higher (*i.e.*, indicating less significance) than those falsely reported

1 in the Registration Statement and further establishes that *R788 did not achieve statistically*  
2 *significant results at the primary efficacy endpoint, ACR20, for the 150mg dose. Id.* at 7-9.

3 (b) **Application of the chi-squared analysis produced false results.** In addition  
4 to pooling the data prior to analyzing it, defendants used the chi-squared method to obtain p-values  
5 reported in the Registration Statement. Because of the small sample size, the proper analysis  
6 required using Fisher's Exact test, which when applied, yielded p-values that were all larger (*i.e.*,  
7 indicating less significance) than the p-values obtained by the chi-squared analysis. Bloch Decl. at  
8 9-11. Application of Fisher's Exact test on a per-country basis (but not correcting for the multiple  
9 comparisons problem), yields p-values for the 100mg and 150mg doses at ACR20, ACR50 and  
10 ACR70 that are all substantially higher (*i.e.*, indicating less significance) than those falsely reported  
11 in the Registration Statement and further establishes that *R788 did not achieve statistically*  
12 *significant results at ACR20 for the 100mg or 150mg doses. Id.*

13 (c) **The efficacy results in the Registration Statement did not account for the**  
14 **multiple comparisons problem rendering the results false and misleading.** The Phase IIa RA  
15 clinical trial made multiple pair-wise comparisons between groups – *i.e.*, 100mg versus placebo at  
16 ACR20, ACR50, ACR70 and DAS28 – which increased the likelihood that one comparison would  
17 be statistically significant by chance alone. Under this situation, multiple pair-wise comparisons  
18 must be accounted for (and were not in the Registration Statement). Bloch Decl. at 11-12. Tukey's  
19 Studentized Range test properly accounts for the multiple pair-wise comparisons performed in the  
20 Phase IIa RA clinical trial. Applying Tukey's Studentized Range test on a per-country basis results  
21 in *p-values at the primary efficacy endpoint, ACR20, for 100mg and 150mg that are very large*  
22 *and not statistically significant as falsely reported in the Registration Statement. Id.* at 11-12.

23 198. Thus, the efficacy results were falsely reported by defendants in the Registration  
24 Statement. Further, the Registration Statement omitted the per-country data (*i.e.*, Mexico and the  
25 U.S.) which concealed the following adverse material information (i) the existence of a country  
26 interaction so substantial that the placebo in Mexico was more effective than any dose in the U.S. at  
27 ACR20 (¶70); (ii) that the dose distribution was not balanced across the U.S. and Mexico (*Id.*); and  
28

1 (iii) that the appearance of an ascending dose response was attributable to the uneven does  
2 distribution and the country interaction (¶¶85-89).

3 199. The statements about the safety results of the Phase IIa RA clinical study in the  
4 Registration Statement were also materially false and misleading because defendants failed to  
5 disclose that (i) there was a dose-dependent increase in average systolic blood pressure of 3-5mm Hg  
6 in 100mg patients and 8-9mm Hg in the 150mg patients which increased both the clinical and  
7 regulatory risk; (ii) 5 patients (not 2, as reported on December 13, 2007) experienced hypertension,  
8 with blood pressure increases as high as 20-30mm Hg; (iii) hypertension was one of the two most  
9 common clinically meaningful drug related adverse events; (iv) 9 patients (not 3, as reported on  
10 December 13, 2007) experienced increased liver enzymes compared to patients taking the placebo;  
11 (v) 20 patients (not 15, as reported on December 13, 2007) experienced neutropenia; (vi) 34 patients  
12 (not 15, as reported on December 13, 2007) experienced diarrhea; and (vii) 35 patients (not 15, as  
13 reported on December 13, 2007) experienced upper gastrointestinal side effects. The undisclosed  
14 adverse events were material as they signaled the strength of the side effects, presented  
15 complications for regulatory approval, impacted the need for and design of future studies and  
16 jeopardized Rigel's ability to obtain a partnership for the further development of R788 – *i.e.*, its  
17 investment thesis.

18 200. Rigel and the Individual Defendants were responsible for the contents and  
19 dissemination of the Registration Statement. Rigel, Gower, Maynard, Payan, Deleage, Lyons, Moos,  
20 Renton, Ringrose and Sherwin signed or authorized the signing of the Registration Statement. None  
21 of these defendants made a reasonable investigation or possessed reasonable grounds for the belief  
22 that the statements contained in the Registration Statement were true and without omissions of any  
23 material facts and were not misleading.

24 201. The four Underwriter Defendants that were paid more than \$7 million to underwrite  
25 the Offering failed to require disclosure of the adverse information about the Phase IIa RA clinical  
26 trial. According to Rigel's January 31, 2008 press release, Credit Suisse was the sole book-runner  
27 for the Offering with Thomas Weisel, Jefferies and Oppenheimer, acting as co-managers. According  
28 to the January 31, 2008 Underwriting Agreement between Rigel and the Underwriter Defendants,

1 Rigel provided the Underwriter Defendants with a “General Disclosure Package” that included,  
2 among other things, a listing of the Company’s clinical trials and “descriptions of the results of the  
3 studies, tests and trials.”

4 202. Public investors relied on the Underwriter Defendants to conduct a reasonable  
5 investigation and to obtain and verify the information contained in the Registration Statement and to  
6 make sure essential facts about the Company were disclosed. Indeed, the Underwriter Defendants  
7 had access to the adverse information at a critical time in Rigel’s corporate life – when it was  
8 seeking to raise capital. The Underwriter Defendants failed to conduct a reasonable investigation  
9 and independently verify the representations in the Registration Statement. The Underwriter  
10 Defendants failed to meet their “gatekeeper” function of protecting investors.

11 203. The Underwriter Defendants purchased the 5 million shares for \$25.5825 and sold the  
12 shares to the public for \$27 per share. As a result, the Underwriter Defendants received \$7,087,500  
13 from the Offering.

14 **VII. CLASS ACTION ALLEGATIONS AND FRAUD-ON-THE-MARKET**  
15 **PRESUMPTION OF RELIANCE**

16 204. Plaintiff brings this action as a class action pursuant to Rule 23 of the Federal Rules  
17 of Civil Procedure on behalf of all persons who purchased Rigel securities during the Class Period,  
18 including persons who acquired the common stock of Rigel pursuant and/or traceable to the false and  
19 misleading Registration Statement, and were damaged thereby (the “Class”). Excluded from the  
20 Class are defendants, directors and officers of Rigel and their families and affiliates.

21 205. The members of the Class are so numerous that joinder of all members is  
22 impracticable. During the Class Period, there were approximately 31 million to 36.7 million  
23 outstanding shares owned by hundreds, if not thousands, of persons. Thus, the disposition of their  
24 claims in a class action will provide substantial benefits to the parties and the Court.

25 206. There is a well-defined community of interest in the questions of law and fact  
26 involved in this case. Questions of law and fact common to the members of the Class that  
27 predominate over questions which may affect individual Class members include:

- 28 (a) Whether the 1933 and 1934 Acts were violated by defendants;

1 (b) Whether defendants engaged in a fraudulent scheme and omitted and/or  
2 misrepresented material facts;

3 (c) Whether defendants' statements omitted material facts necessary to make the  
4 statements made, in light of the circumstances under which they were made, not misleading;

5 (d) Whether defendants knew or recklessly disregarded that their statements were  
6 materially false and misleading;

7 (e) Whether the prices of Rigel securities were artificially inflated;

8 (f) Whether defendants' fraudulent scheme, misrepresentations and omissions  
9 caused Class members to suffer economic losses, *i.e.*, damages; and

10 (g) The extent of damage sustained by Class members and the appropriate  
11 measure of damages.

12 207. Plaintiff's claims are typical of those of the Class because plaintiff and the Class  
13 purchased Rigel common stock during the Class Period and sustained damages from defendants'  
14 wrongful conduct. Plaintiff will adequately protect the interests of the Class and has retained  
15 counsel who are experienced in class action securities litigation. Plaintiff has no interests that  
16 conflict with those of the Class.

17 208. A class action is superior to other available methods for the fair and efficient  
18 adjudication of this controversy. A class action will achieve economies of time, effort and expense  
19 and provide uniformity of decision to the similarly situated members of the Class without sacrificing  
20 procedural fairness or bringing about other undesirable results. Class members have not indicated an  
21 interest in prosecuting separate actions as none have been filed. The number of Class members and  
22 the relatively small amounts at stake for individual Class members make separate suits  
23 impracticable. No difficulties are likely to be encountered in the management of this action as a  
24 class action.

25 209. In addition, a class action is superior to other methods of fairly and efficiently  
26 adjudicating this controversy because the questions of law and fact common to the Class  
27 predominate over any questions affecting only individual Class members. Although individual Class  
28 members have suffered disparate damages, the fraudulent scheme and the misrepresentations and

1 omissions causing damages are common to all Class members. Further, there are no individual  
2 issues of reliance that could make this action unsuited for treatment as a class action because all  
3 Class members relied on the integrity of the market and are entitled to the fraud-on-the-market  
4 presumption of reliance.

5 210. The market for Rigel's common stock was open, well developed and efficient at all  
6 relevant times. Rigel's stock met the requirements for listing, and was listed and actively traded, on  
7 the NASDAQ, a highly efficient and automated market. As a regulated issuer, Rigel filed periodic  
8 public reports with the SEC. Rigel regularly communicated with public investors via established  
9 market communication mechanisms, including through regular disseminations of press releases on  
10 the national circuits of major newswire services and through other wide-ranging public disclosures,  
11 such as communications with the financial press and other similar reporting services.

12 211. As alleged herein, the change in the price of Rigel's stock – compared to the changes  
13 in the peer group and NASDAQ – in response to the release of unexpected material positive and  
14 negative information about the Company shows there was a cause-and-effect relationship between  
15 the public release of the unexpected information about Rigel and the price movement in the  
16 Company's stock. The average weekly trading volume of Rigel's stock during the Class Period was  
17 approximately 4.25 million shares, or 11.6% of total outstanding shares. Numerous analysts  
18 followed Rigel, attended the Company's conference calls and issued reports throughout the Class  
19 Period. The Company was eligible to register and did register securities on Form S-3 during the  
20 Class Period.

21 212. As a result of the foregoing, the market for Rigel common stock promptly digested  
22 current information regarding Rigel from all publicly available sources and reflected such  
23 information in the Company's stock price. Under these circumstances, all purchasers of Rigel  
24 common stock during the Class Period suffered similar injury through their purchases of Rigel  
25 common stock at artificially inflated prices and the subsequent revelations concerning declines in  
26 price, and a presumption of reliance applies.

1 **VIII. NO SAFE HARBOR**

2 213. Rigel’s verbal “Safe Harbor” warnings accompanying its oral forward-looking  
3 statements issued during the Class Period were ineffective to shield those statements from liability.

4 214. Defendants are also liable for any false or misleading forward-looking statements  
5 pled because, at the time each forward-looking statements was made, the speaker knew the forward-  
6 looking statements was false or misleading and the forward-looking statements was authorized  
7 and/or approved by an executive officer of Rigel who knew that the forward-looking statements was  
8 false. None of the historic or present tense statements made by defendants was an assumption  
9 underlying or relating to any plan, projection or statement of future economic performance, as they  
10 were not stated to be such assumptions underlying or relating to any projection or statement of future  
11 economic performance when made, nor were any of the projections or forecasts made by defendants  
12 expressly related to or stated to be dependent on those historic or present tense statements when  
13 made.

14 **COUNT I**

15 **For Violations of Section 10(b) of the 1934 Act and Rule 10b-5**  
16 **Against Defendants Rigel, Gower, Maynard, Payan, Grossbard and Rodriguez**

17 215. Plaintiff incorporates ¶¶1-189; 204-14 by reference.

18 216. During the Class Period, defendants named in this Count disseminated or approved  
19 the false statements specified above, which they knew or recklessly disregarded were misleading in  
20 that they contained misrepresentations and failed to disclose material facts necessary in order to  
21 make the statements made, in light of the circumstances under which they were made, not  
22 misleading.

23 217. These defendants violated §10(b) of the 1934 Act and Rule 10b-5 in that they:

24 (a) Employed devices, schemes, and artifices to defraud;

25 (b) Made untrue statements of material facts or omitted to state material facts

26 necessary in order to make the statements made, in light of the circumstances under which they were  
27 made, not misleading; or

1 (c) Engaged in acts, practices, and a course of business that operated as a fraud or  
2 deceit upon plaintiff and others similarly situated in connection with their purchases of Rigel  
3 securities during the Class Period.

4 218. Plaintiff and the Class have suffered damages in that, in reliance on the integrity of  
5 the market, they paid artificially inflated prices for Rigel securities. Plaintiff and the Class would  
6 not have purchased Rigel securities at the prices they paid, or at all, if they had been aware that the  
7 market prices had been artificially and falsely inflated by defendants' misleading statements.

8 219. As a direct and proximate result of these defendants' wrongful conduct, plaintiff and  
9 the other members of the Class suffered damages in connection with their purchases of Rigel  
10 securities during the Class Period.

## 11 **COUNT II**

### 12 **For Violations of Section 20(a) of the 1934 Act** 13 **Against Defendants Rigel, Gower, Maynard,** 14 **Payan, Grossbard and Rodriguez**

15 220. Plaintiff incorporates ¶¶1-189; 204-19 by reference.

16 221. Gower, Maynard, Payan, Grossbard and Rodriguez acted as controlling persons of  
17 Rigel within the meaning of §20 of the 1934 Act. By virtue of their positions and their power to  
18 control public statements about Rigel, Gower, Maynard, Payan, Grossbard and Rodriguez had the  
19 power and ability to control the actions of Rigel and its employees. Rigel controlled Gower,  
20 Maynard, Payan, Grossbard and Rodriguez and its other officers and employees. By reason of such  
21 conduct, defendants are liable pursuant to §20(a) of the 1934 Act.

## 22 **COUNT III**

### 23 **For Violations of Section 11 of the 1933 Act** 24 **Against All Defendants, Except Grossbard and Rodriguez**

25 222. Plaintiff incorporates ¶¶1-4; 7-8; 15; 23-48, 190-203 by reference.

26 223. This Count is brought pursuant to §11 of the 1933 Act, 15 U.S.C. §77k, on behalf of  
27 the Class, against all defendants except Grossbard and Rodriguez. For purposes of this Count,  
28 plaintiff expressly excludes and disclaims any allegation that could be construed as alleging fraud or  
intentional or reckless misconduct, as this Court is based solely on claims of strict liability and/or

1 negligence under the 1933 Act. Plaintiff does not repeat or reallege any paragraph that alleges  
2 defendants' misconduct was done intentionally, knowingly or with a reckless disregard or otherwise  
3 sounds in fraud.

4           224. The Registration Statement was false and misleading, contained untrue statements of  
5 material facts, omitted to state other facts necessary to make the statements made not misleading,  
6 and omitted to state material facts required to be stated therein.

7           225. Rigel is the registrant for the Offering. As issuer of the shares, Rigel is strictly liable  
8 to plaintiff and the Class for the misstatements and omissions.

9           226. The Individual Defendants named herein were responsible for the contents and  
10 dissemination of the Registration Statement. Each of the Individual Defendants named in this Count  
11 signed or authorized the signing of the Registration Statement. None of the defendants named herein  
12 made a reasonable investigation or possessed reasonable grounds for the belief that the statements  
13 contained in the Registration Statement were true and without omissions of any material facts and  
14 were not misleading.

15           227. Defendants issued and disseminated, caused to be issued and disseminated, and  
16 participated in the issuance and dissemination of materially false and misleading written statements  
17 to the investing public that were contained in the February 2008 Offering, which misrepresented or  
18 failed to disclose the facts set forth above. By reason of the conduct alleged herein, each of the  
19 defendants violated §11 of the 1933 Act, employed a person who violated §11 of the 1933 Act  
20 and/or controlled a person who violated §11 of the 1933 Act.

21           228. Plaintiff acquired Rigel shares pursuant and/or traceable to the Registration Statement  
22 for the Offering.

23           229. Plaintiff and the Class have sustained damages. As a direct and proximate result of  
24 the acts and omissions of the defendants, in violation of the 1933 Act, the market price of Rigel  
25 common stock was artificially inflated, and plaintiffs and the Class suffered substantial damages in  
26 connection with their purchases of Rigel common stock pursuant to the February 2008 Offering.

27           230. At the times plaintiff purchased or otherwise acquired Rigel common stock, they and  
28 the other members of the class were without knowledge of the facts concerning the wrongful conduct

1 alleged herein and could not have reasonably discovered those facts prior to October 27, 2008. Less  
2 than one year has elapsed from the time when plaintiff discovered or reasonably could have  
3 discovered the facts upon which this action is based to the time that they filed this action. Less than  
4 three years have elapsed from the time that the securities upon which this claim for relief is brought  
5 were bona fide offered to the public and this action was filed.

#### 6 **COUNT IV**

##### 7 **For Violations of Section 12(a)(2) of the 1933 Act** 8 **Against All Defendants, Except Grossbard and Rodriguez**

9 231. Plaintiff repeats and realleges the allegations set forth above in Count III, only ¶¶1-4;  
10 7-8; 15; 23-48; 190-203; 222-30. For purposes of this Count, plaintiff expressly excludes and  
11 disclaims any allegation that could be construed as alleging fraud or intentional or reckless  
12 misconduct, as this Count is based solely on claims of strict liability and/or negligence under the  
13 1933 Act.

14 232. By means of the defective Prospectus, defendants named in this Count assisted in the  
15 sale of shares of the Company's securities to plaintiff and other members of the Class.

16 233. The Prospectus contained untrue statements of material fact, and concealed and failed  
17 to disclose material facts, as detailed above. Defendants owed plaintiff and the other members of the  
18 Class who purchased Rigel securities pursuant to the Prospectus the duty to make a reasonable and  
19 diligent investigation of the statements contained in the Prospectus to ensure that such statements  
20 were true and that there was no omission to state a material fact required to be stated in order to  
21 make the statements contained therein not misleading. These defendants, in the exercise of  
22 reasonable care, should have known of the misstatements and omissions contained in the Prospectus  
23 as set forth above.

24 234. Plaintiff did not know, nor in the exercise of reasonable diligence could have known,  
25 of the untruths and omissions contained in the Prospectus at the time it acquired the Company's  
26 securities.

27 235. By reason of the conduct alleged herein, defendants violated §12(a)(2) of the 1933  
28 Act. As a direct and proximate result of such violations, plaintiff and the other members of the Class

1 who purchased Rigel common stock pursuant to the Prospectus sustained substantial damages in  
2 connection with their purchases of Rigel stock. Accordingly, plaintiff and the other members of the  
3 Class who hold such stock have the right to rescind and recover the consideration paid for their  
4 shares, and hereby tender their shares to the defendants sued herein. Class members who have sold  
5 their shares seek damages to the extent permitted by law.

6 **COUNT V**

7 **For Violations of Section 15 of the 1933 Act**  
8 **Against the Individual Defendants, Except Grossbard and Rodriguez**

9 236. Plaintiff repeats and realleges each and every allegation contained in Count III and IV  
10 only ¶¶1-4; 7-8; 15; 23-48; 190-203; 222-35.

11 237. This Count is brought pursuant to §15 of the 1933 Act against the Individual  
12 Defendants, except Grossbard and Rodriguez.

13 238. Each of the Individual Defendants named in this Count was a control person of Rigel  
14 by virtue of his position as a director and/or senior officer of Rigel which allowed each of these  
15 defendants to exercise control over Rigel, its employees and its operations.

16 239. Each of the Individual Defendants was a participant in the violations of §11 of the  
17 1933 Act alleged in the Count above, based on their having signed or authorized the signing of the  
18 Registration Statement and having otherwise participated in the process which allowed the Offering  
19 to be successfully completed.

20 **IX. PRAYER FOR RELIEF**

21 WHEREFORE, plaintiff prays for relief and judgment, as follows:

- 22 A. Declaring this action to be a proper class action pursuant to Fed. R. Civ. P. 23;  
23 B. Awarding plaintiff and the members of the Class damages and interest;  
24 C. With respect to Count IV, ordering rescission or rescissory damages for purchasers of  
25 Rigel common stock in the Offering;  
26 D. Awarding plaintiff's reasonable costs, including attorneys' fees; and  
27 E. Awarding such equitable and/or injunctive or other relief as the Court may deem just  
28 and proper.



1 CERTIFICATE OF SERVICE

2 I hereby certify that on January 27, 2010, I electronically filed the foregoing with the Clerk  
3 of the Court using the CM/ECF system which will send notification of such filing to the e-mail  
4 addresses denoted on the attached Electronic Mail Notice List, and I hereby certify that I have  
5 mailed the foregoing document or paper via the United States Postal Service to the non-CM/ECF  
6 participants indicated on the attached Manual Notice List.

7 I further certify that I caused this document to be forwarded to the following Designated  
8 Internet Site at: <http://securities.stanford.edu>.

9 I certify under penalty of perjury under the laws of the United States of America that the  
10 foregoing is true and correct. Executed on January 27, 2010.

11  
12 /s/  
13 WILLOW E. RADCLIFFE  
14 COUGHLIN STOIA GELLER  
15 RUDMAN & ROBBINS LLP  
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17 San Francisco, CA 94111  
18 Telephone: 415/288-4545  
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