

The Honorable Ricardo S. Martinez, Chief District Judge

UNITED STATES DISTRICT COURT
FOR THE WESTERN DISTRICT OF WASHINGTON
AT SEATTLE

SAMIT PATEL, individually and on
behalf of all others similarly situated,

Plaintiff,

v.

SEATTLE GENETICS, INC.,
CLAY B. SIEGALL,
TODD E. SIMPSON, and
JONATHAN DRACHMAN,

Defendants.

No. 2:17-cv-00041-RSM

**CONSOLIDATED AMENDED
COMPLAINT — CLASS ACTION —
FOR VIOLATION OF FEDERAL
SECURITIES LAWS**

Jury Trial Demanded

Lead Plaintiff Carl Johnson (“Lead Plaintiff”), individually and on behalf of all other persons similarly situated, by his undersigned attorneys, for his Consolidated Amended Complaint against Seattle Genetics, Inc. (“Seattle Genetics” or the “Company”), Clay B. Siegall (“Siegall”), Todd E. Simpson (“Simpson”), and Jonathan Drachman (“Drachman”) (Siegall, Simpson, and Drachman are referred to as the “Individual Defendants”) (Seattle Genetics and the Individual Defendants collectively are referred to as the “Defendants”), alleges the following based upon personal knowledge as to Lead Plaintiff and his own acts, and upon information and belief as to all other matters, based upon, *inter alia*, the independent investigation conducted by and through

1 his attorneys, which included, among other things, a review of the Defendants’ public documents,
2 United States Securities and Exchange Commission (“SEC”) filings, wire and press releases
3 published by, and regarding, Seattle Genetics, conference calls and announcements made by
4 Defendants, economic analysis of Seattle Genetics’ stock price movement and pricing volume
5 data, analysts’ reports and advisories about the Company, private investigation, and information
6 readily obtainable on the internet. Lead Plaintiff believes that substantial evidentiary support will
7 exist for the allegations set forth herein after a reasonable opportunity for discovery.

8 **NATURE OF THE ACTION**

9 1. This is a class action on behalf of persons or entities other than Defendants, who
10 purchased or otherwise acquired Seattle Genetics’ common stock between October 27, 2016 and
11 December 27, 2016, both dates inclusive (the “Class Period”), seeking to recover damages caused
12 by Defendants’ violations of the federal securities laws, and to pursue remedies under §§10(b) and
13 20(a) of the Securities Exchange Act of 1934 (“Exchange Act”).

14 2. Seattle Genetics is a development stage biopharmaceutical company traded on the
15 NASDAQ exchange under the symbol “SGEN.” This case arises out of misrepresentations and
16 omissions that Defendants made during the class period related to known liver toxicity of one of
17 Seattle Genetics’ most important drug candidates, SGN-CD33A, also known as Vadastuximab
18 Talirine. SGN-CD33A is a type of cancer treatment known as an antibody-drug conjugate
19 (“ADC”). ADCs are a drug technology that uses antibodies to target specific antigens on the
20 surface of cancerous cells, and deliver locally strong anticancer agents that would be too toxic to
21 administer otherwise. Seattle Genetics’ trials of SGN-CD33A focused on developing the drug to
22 treat a type of blood cancer called Acute Myeloid Leukemia (“AML”).

23 3. SGN-CD33A is the successor to an earlier ADC developed by Pfizer known as
24 Mylotarg (Gemtuzumab ozogamicin). Mylotarg was manufactured and marketed by Pfizer from
25 2000 to 2010 as a treatment for AML. In June 2010, Pfizer withdrew Mylotarg from the market at
26 the request of the FDA because an advanced stage clinical trial demonstrated that the fatal rate of
27

1 treatment-related toxicity was significantly higher than standard chemotherapy with no
2 corresponding benefit to cancer patients.¹

3 4. Seattle Genetics repeatedly claimed that SGN-CD33A had a superior design and
4 more advanced ADC technology than Mylotarg, allowing it to kill cancerous cells effectively
5 without the toxicity that doomed the earlier drug. Specifically, throughout the Class Period,
6 Defendants claimed SGN-CD33A did not share the toxic side effects of Mylotarg, and
7 misleadingly touted the absence of liver disease in clinical trials, while omitting that internal
8 information disseminated to Defendants and others within Seattle Genetics unquestionably
9 demonstrated that SGN-CD33A was hepatotoxic.

10 5. Each of the Defendants was well aware throughout the Class Period that SGN-
11 CD33A posed a high risk of liver toxicity (hepatotoxicity). Specifically:

- 12 a. Data from prior studies of a similar drug confirmed hepatotoxicity. Between 2011
13 and 2012, Seattle Genetics collaborated with Spirogen Ltd. (“Spirogen”) to develop
14 a drug that was very close to SGN-CD33A. Animal studies for this drug showed a
15 high risk of toxicity, and Seattle Genetics ultimately abandoned a clinical trial
16 comprised of 8 to 12 patients because it posed an unreasonable risk of death.
- 17 b. Internal documents confirmed hepatotoxicity. Seattle Genetics maintained Safety
18 Data Sheets for each of the drugs it developed, and the components used in its labs.
19 For SGN-CD33A, the Safety Data Sheets confirmed high levels of hepatotoxicity.
- 20 c. A third-party risk assessment confirmed toxicity. According to a confidential
21 witness who had direct access to the study, in the middle of 2016, Seattle Genetics
22 procured a third party risk assessment, which concluded that the levels of toxicity
23 associated with the drug were unacceptably high.

24
25 ¹ Besides the now-withdrawn approval for Mylotarg, the FDA has only approved two other
26 ADCs: Adcetris (Brentuximab vedotin), developed by Seattle Genetics to treat relapsed or
27 refractory Hodgkin lymphoma and systemic anaplastic large cell lymphoma, and Kadcyła
(Trastuzumab emtansine), developed by Genentech to treat metastatic breast cancer.

- 1 d. Contract manufacturer raised concerns about toxicity. Seattle Genetics relied on a
2 contract manufacturing organization (“CMO”) to help produce the payload and
3 linker components of the ADC technology utilized in SGN-CD33A. After seeing
4 the third-party risk assessment discussed above, this CMO suspended production
5 of the components that it helped produce for SGN-CD33A.
- 6 e. Company reaction to CMO manufacturing suspension indicates its awareness of
7 toxicity concerns. The Company was aware of both the CMO’s suspension and the
8 concerns. In response, the Company directed its health & safety engineer to attempt
9 to deflect those concerns.
- 10 f. In-house toxicologist raised concerns. According to a confidential witness, Seattle
11 Genetics’ in-house toxicologist expressed concerns about the level of toxicity in
12 SGN-CD33A, before being pressured not to do so.
- 13 g. Regulations required awareness. Seattle Genetics was required to make itself aware
14 of, and report to the United States Food and Drug Administration (“FDA”), deaths
15 and other serious adverse events in clinical trials. Under FDA regulations, sponsors
16 conducting clinical trials are required to “promptly review all information relevant
17 to the safety of the drug” and to “notify FDA of any unexpected fatal or life-
18 threatening suspected adverse reaction as soon as possible but in no case later than
19 7 calendar days after the sponsor's initial receipt of the information.” 21 U.S.C.
20 §312.32. Accordingly, to fulfill regulatory reporting duties, Defendants had to have
21 known and informed the FDA of the four deaths due to hepatotoxicity in SGN-
22 CD33A clinical trials, and the six cases of observed liver toxicity.

23 6. On December 27, 2016, the FDA placed a full clinical hold on the Company’s
24 Phase I/II trial of SGN-CD33A administered to stem cell transplant patients (“Stem Cell Phase
25 I/II”).

26 7. The FDA also placed partial clinical holds on two other Phase I trials of SGN-
27 CD33A administered in combination with chemotherapy regimens in AML patients. The

1 Company told investors that the trials subject to partial clinical holds would not enroll new patients,
2 and that existing patients could continue to participate if they signed a revised consent form.

3 8. On this news, Seattle Genetics' stock price declined by \$9.50 per share, or by over
4 15%, to close at \$52.36 on December 27, 2016.

5 9. Like investors, analysts were stunned by Defendants' revelation. That same day,
6 for example, Credit Suisse analyst Kennen McKay lowered the Company's price target by \$10,
7 and remarked that the announcement was surprising given that Defendants had created the false
8 impression that SGN-CD33A had unique technology to "avoid the [toxicity] pitfalls" of Mylotarg.

9 10. On March 6, 2017, the Company announced that it would abandon entirely the Stem
10 Cell Phase I/II trial and would adopt substantial risk mitigation measures to address hepatotoxicity
11 in all other trials of SGN-CD33A. With these hepatotoxicity risk mitigation measures in place, the
12 FDA lifted the partial clinical holds it had placed on two other Phase I trials.

13 JURISDICTION AND VENUE

14 11. The claims asserted herein arise under Sections 10(b) and 20(a) of the Exchange
15 Act (15 U.S.C. §§ 78j(b) and 78t(a)), and SEC Rule 10b-5 promulgated thereunder (17 C.F.R. §
16 240.10b-5).

17 12. This Court has jurisdiction over the subject matter of this action pursuant to 28
18 U.S.C. §§ 1331 and 1337 and Section 27 of the Exchange Act (15 U.S.C. § 78aa).

19 13. Venue is proper in this District pursuant to Section 27 of the Exchange Act (15
20 U.S.C. § 78aa) and 28 U.S.C. § 1391(b) given that a significant portion of the Defendants' actions,
21 and the subsequent damages, took place within this District. Seattle Genetics is a corporation
22 incorporated in Delaware with its principal place of business in Bothell, Washington, within this
23 District, and the Defendants Siegall, Simpson and Drachman reside in or around Bothell,
24 Washington.

25 14. In connection with the acts, conduct and other wrongs alleged in this Complaint,
26 Defendants, directly or indirectly, used the means and instrumentalities of interstate commerce,
27

1 including but not limited to, the United States mail, interstate telephone communications and the
2 facilities of a national securities exchange.

3 **PARTIES**

4 15. Lead Plaintiff purchased Seattle Genetics' securities at artificially inflated prices
5 during the Class Period as set forth in his previously-filed Certification (Dkt. No. 7-2), and suffered
6 damages as a result of the disclosure of federal securities laws violations alleged herein.

7 16. Defendant Seattle Genetics is a Delaware corporation with its executive offices
8 located at 21823 30th Drive, Suite 300 SE, Bothell, WA 98021. Seattle Genetics' shares trade on
9 the NASDAQ national market system under the ticker symbol "SGEN."

10 17. Defendant Siegall is Seattle Genetics' co-founder, and at all relevant times, has
11 been Seattle Genetics' President, Chief Executive Officer and Chairman of the Company's Board
12 of Directors.

13 18. Defendant Simpson became Seattle Genetics' Chief Financial Officer in October
14 2005, and held that position at all times relevant hereto.

15 19. Defendant Drachman was appointed as Seattle Genetics' Chief Medical Officer and
16 Executive Vice President, Research and Development in October 2013, and held that position at
17 all times relevant hereto.

18 20. Defendants Siegall, Simpson, and Drachman are sometimes referred to herein as
19 the "Individual Defendants."

20 **BACKGROUND AND PRE-CLASS PERIOD EVENTS**

21 **Clinical Trials and Clinical Holds**

22 21. A biopharmaceutical company generally conducts clinical trials in three phases.
23 These phases are codified in FDA regulations.

24 22. Phase I studies "are designed to determine the metabolism and pharmacologic
25 actions of the drug in humans, the side effects associated with increasing doses, and, if possible,
26 to gain early evidence on effectiveness." 21 C.F.R. § 312.21. They are typically open label, which
27 means that trial data is provided to, not blinded from, the sponsor.

1 23. Phase II studies are “typically well controlled” studies “conducted to evaluate the
2 effectiveness of the drug for a particular indication or indications in patients with the disease or
3 condition under study and to determine the common short-term side effects and risks associated
4 with the drug.” *Id.*

5 24. Phase III studies are expanded studies “performed after preliminary evidence
6 suggesting effectiveness of the drug has been obtained, and are intended to gather the additional
7 information about effectiveness and safety that is needed to evaluate the overall benefit-risk
8 relationship of the drug and to provide an adequate basis for physician labeling. Phase III studies
9 usually include from several hundred to several thousand subjects.” *Id.*

10 25. Occasionally, a sponsor will designate a single clinical trial as spanning two
11 different phases of study (*e.g.*, a Phase I/II trial or a Phase II/III trial).

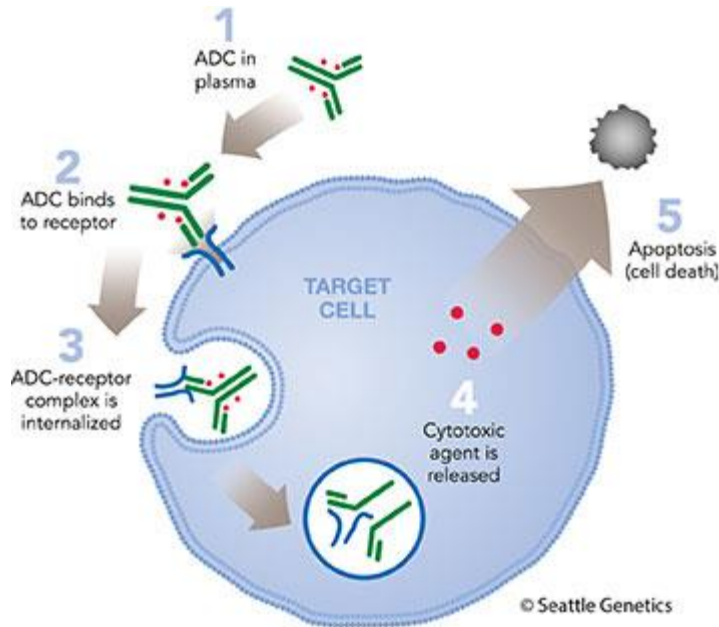
12 26. During clinical trials, sponsors are required to “promptly review all information
13 relevant to the safety of the drug” and to “notify FDA of any unexpected fatal or life-threatening
14 suspected adverse reaction as soon as possible but in no case later than 7 calendar days after the
15 sponsor's initial receipt of the information.” 21 U.S.C. §312.32.

16 27. If the FDA determines that a trial protocol exposes subjects to unreasonable and
17 significant risk, the FDA can halt or limit the trial by placing it on a total or partial “clinical hold.”
18 21 C.F.R. § 312.42(b). FDA Regulations require the agency to inform the sponsor of a deficiency
19 and attempt to resolve the matter *before* issuing a clinical hold, unless patients are exposed to a
20 serious and immediate risk. 21 C.F.R. §312.42(c).

21 **ADCs and SGN-CD33A**

22 28. Defendant Seattle Genetics is a clinical-stage biotechnology company that focuses
23 on the use of ADCs to treat cancer. ADCs aim to harness antibodies to target delivery of toxic
24 payloads to cancer cells while simultaneously seek to spare healthy cells susceptible to damage in
25 non-targeted treatments like chemotherapy and radiation. Because ADC’s are targeted and, aim to
26 deliver the payload only where needed, they are considered to be a means of delivering more potent
27 payloads to cancerous regions than could be delivered systemically.

29. The following illustrations show the elements of an ADC and the targeted delivery of a toxic payload:



30. Specifically, SGN-CD33A uses the following components:
- Cytotoxic agent (payload): SGN-CD33A uses a highly toxic payloads known as pyrrolobenzodiazepine (“PBD”) dimers. PBD dimers bind to the DNA of tumor cells to block cell division, the process by which cancer spreads.
 - Linker: Seattle Genetics claims that SGN-CD33A “employs a novel linker system and proprietary, site-specific conjugation technology (EC-mAb) that allows uniform drug-loading of the cell-killing PBD agent to the anti-CD33 antibody.”²

² See <http://www.seattlegenetics.com/pipeline/vadastuximab-talirine>.

1 c. Antibody: SGN-CD33A antibodies target a receptor called CD33, which is
2 expressed on AML cancer cells.

3 31. According to Seattle Genetics, this design allows SGN-CD33A “to be stable in the
4 bloodstream and to release its potent DNA binding agent upon internalization into CD-33
5 expressing cells.”³

6 32. Seattle Genetics has worked on PBD dimer payloads since 2008 under an exclusive
7 licensing arrangement with Spirogen. Spirogen is an ADC developer based in the United Kingdom,
8 which was acquired by the AstraZeneca Group in 2013.

9 33. SGN-CD33A is a successor to Mylotarg. Mylotarg was an ADC developed by
10 Pfizer. It was approved in 2000 with a “black box” warning label that stated, in relevant part,
11 “Hepatotoxicity, including severe hepatic veno-occlusive disease (VOD), has been reported in
12 association with the use of Mylotarg as a single agent....” After post-marketing surveillance
13 demonstrated that the rate of VOD was even higher than expected, and a post-approval clinical
14 trial showed no clinical benefit, Pfizer voluntarily withdrew Mylotarg from the market in June
15 2010.

16 34. Beginning in July 2013, Seattle Genetics began clinical trials of SGN-CD33A. Its
17 initial clinical trial was a Phase I open-label study of SGN-CD33A in combination with
18 hypomethylating agents (“HMA Phase I”). The full name of the HMA Phase I study is: “A Phase
19 1 trial of SGN-CD33A in Patients With CD33-positive Acute Myeloid Leukemia.” HMAs,
20 including decitabine or azacitidine, are considered standard treatment for older patients with AML.
21 The trial also evaluates anti-leukemic activity, pharmacokinetics and overall survival in patients
22 with AML. On the basis of interim data from the HMA Phase I, in May 2016, Seattle Genetics
23 initiated a Phase III, randomized, double-blind, placebo-controlled clinical trial called CASCADE.
24 The CASCADE trial is designed to determine if SGN-CD33A in combination with the HMAs can
25 extend overall survival in older patients with AML compared to patients treated with HMAs alone.
26

27 ³ See <http://www.seattlegenetics.com/SGN-CD33A>.

1 In December 2016, the FDA placed the HMA Phase I trial of SGN-CD33A on a partial clinical
2 hold.

3 35. In December 2014, Seattle Genetics initiated a Phase I study to evaluate SGN-
4 CD33A administered in combination with a chemotherapy regimen known as 7+3 for younger
5 patients with newly diagnosed AML (“7+3 Phase I”). The 7+3 Phase I study is entitled: “A Phase
6 1b Dose Escalation Study of SGN-CD33A in Combination With Standard-of-care for Patients with
7 Newly Diagnosed Acute Myeloid Leukemia.” This open-label clinical trial seeks to determine the
8 maximum tolerated dose and safety profile of SGN-CD33A. It also seeks to evaluate anti-leukemic
9 activity, pharmacokinetics and overall survival. In December 2016, the FDA placed the 7+3 Phase
10 I trial on a partial clinical hold.

11 36. In November 2015, Seattle Genetics initiated the Stem Cell Phase I/II trial, an open-
12 label clinical study of SGN-CD33A in patients with relapsed or refractory AML. This study was
13 entitled: “A Phase 1/2 Study of Vadastuximab Talirine Administered in Sequence With Allogeneic
14 Hematopoietic Stem Cell Transplant in Patients With Relapsed or Refractory Acute Myeloid
15 Leukemia (AML).” This trial sought to evaluate SGN-CD33A as a pre-conditioning regimen
16 before the administration of an allogeneic stem cell transplant and as a maintenance therapy after a
17 stem cell transplant. On December 27, 2016, Seattle Genetics announced that the FDA placed the
18 Stem Cell Phase I/II trial on full clinical hold. On March 6, 2017, Seattle Genetics announced that
19 it would abandon this trial, citing challenges associated with developing an effective treatment in
20 this setting.

21 37. Even before the Class Period, Defendants claimed that SGN-CD33A’s superior
22 linker and cytotoxic payload differentiated SGN-CD33A from Mylotarg’s known deficiencies. For
23 example, on January 11, 2016, Defendant Siegall attended the J. P. Morgan Health Care
24 Conference and made the following claims regarding the difference between SGN-CD33A and
25 Mylotarg:

1 <Q>: [Question Inaudible] (09:17-09:30)

2 <A - Clay B. Siegall>: Right. The question from [ph] Richard (09:32) was about
3 this new ADC drug conjugate, 33A, why are we more excited than Mylotarg, which
4 was a 33 drug conjugate of the past. Mylotarg had a lot of issues and problems, but
5 it was a phenomenal pioneering molecule.

6 ****

7 And when you think about Mylotarg, the biggest toxicity that with a problem, and
8 it was written about a lot was veno-occlusive disease. And we've treated couple of
9 hundred patients that with 33A without VOD. And so, we –you just don't see the
10 same toxicity that was probably because the drug was falling off very readily, a
11 drug being calicheamicin. And so, I think we have used all our engineering skill to
12 make what I think is one of the best ADC technologies there is. And we put it into
13 this – using this target 33, which we like a lot. And despite all of the problems that
14 Mylotarg had, it still had some really nice subjective responses. It still had some
15 survival data. Not huge, but had some despite its problems in toxicities. And so, I
16 just think that we used the right target with all the new technologies. Is there
17 anything else to add to that? Okay. Yeah.

18 **Known Hepatotoxicity Associated with SGN-CD33A**

19 38. Prior to the start of the Class Period, Seattle Genetics and the Individual Defendants
20 were well aware that SGN-CD33A posed a high risk of hepatotoxicity. According to Confidential
21 Witness 1 (“CW 1”), hepatotoxicity was known, *inter alia*, from prior experience with a
22 predecessor drug using similar components, from internal Safety Data Sheets made available to
23 Seattle Genetics employees, and from a third-party risk assessment. As discussed below, the
24 particular hepatotoxic adverse events experienced in the open label Phase I trials were also made
25 known to Seattle Genetics as they occurred.

26 39. CW 1 has seventeen years of experience in the biotechnology industry, and served
27 as the Senior Environmental Health and Safety Engineer at Seattle Genetics from March 2015 to
February 2017. As Senior Environmental Health and Safety Engineer at Seattle Genetics, CW1
was responsible for providing information to Seattle Genetics employees about the risks of the
environment in which they worked, including the risks of exposure to toxic drug compounds in
Seattle Genetics labs, and the stringent handling requirements for those drugs. CW1 primarily dealt

1 with the division at Seattle Genetics that was responsible for synthesizing drugs, including SGN-
2 CD33A. CW1 reported to David Moore (“Moore”), the Associate Director of Facilities, and Tina
3 Bailey, the Senior Manager of Facilities, at Seattle Genetics. Moore reported to Mike Mabrito, the
4 Director of Facilities at Seattle Genetics, and both Moore and Mabrito directly reported to
5 Defendant Simpson.

6 40. According to CW1, in 2011 or 2012, Seattle Genetics collaborated with Spirogen
7 to initiate a clinical trial that enrolled eight to twelve patients on a standalone therapy similar to
8 SGN-CD33A. Early data from animal studies for this predecessor drug indicated high risk of
9 treatment-related toxicity, and Seattle Genetics ultimately terminated the clinical trial because of
10 hepatotoxicity. CW1 explained that the development of SGN-CD33A was based on a similar
11 molecule to the one utilized in the earlier trial, and “almost all of the safety correlations [with
12 SGN-CD33A] were based off that Spirogen compound.” Because SGN-CD33A and the
13 predecessor that the Company developed in collaboration with Spirogen were similar, the data
14 from the predecessor was used to develop the safety protocols that CW1 was responsible for
15 communicating to Seattle Genetics’ various divisions.

16 41. CW1 coordinated with the Company’s in-house toxicologist to prepare Safety Data
17 Sheets that listed specific levels of toxicity associated with each organ in the human body. These
18 reports were available to the Company’s officers, including Defendants, and any employees
19 authorized to access the reports. For SGN-CD33A, the Safety Data Sheets indicated a risk of
20 hepatotoxicity.

21 42. According to CW1, in the middle of 2016, Seattle Genetics procured a third party
22 risk assessment of the toxicity associated with SGN-CD33A, and that assessment concluded that
23 the risks were high. CW1 explained that the contract manufacturer that Seattle Genetics used to
24 manufacture the cytotoxic payload and linker components of SGN-CD33A learned of the third-
25 party risk assessment, and suspended manufacturing these components as a result.

1 43. CW1 was asked to collaborate with the in-house toxicologist to respond to the third
2 party's risk assessment in an effort to convince the contract manufacturer to continue production
3 of SGN-CD33A's components.

4 44. After CW1 raised concerns about the risks of exposure to SGN-CD33A, CW1 was
5 instructed by Mabrito not to discuss the issue with the in-house toxicologist, and the Company
6 threatened to fire CW1 if he violated this directive.

7 45. CW1 stated Seattle Genetics' in-house toxicologist initially also expressed
8 concerns about SGN-CD33A's level of toxicity (consistent with the findings of the third-party
9 assessment), but the in-house toxicologist was coerced to moderate his views by Moore and
10 Mabrito. CW1 understood this coercion originated from Defendant Simpson.

11 46. CW1 approached several senior-level administrative divisions within the Company
12 about his concerns with no success. CW1 attempted to directly reach Defendant Simpson to discuss
13 his concerns, but Defendant Simpson rebuffed CW1. CW1 also emailed Defendant Siegall's
14 executive assistant to seek a meeting, and copied the executive assistant on various emails with
15 Human Resources. However, Defendant Siegall did not respond to CW1.

16 47. In addition to knowing the undisclosed *risks* of hepatotoxicity, Defendants also
17 learned of the six specific hepatotoxic events at issue here, including four deaths, as they occurred
18 in the open-label Phase I trials of SGN-CD33A. Seattle Genetics, as the sponsor of these trials,
19 was required to report each of those events to the FDA within seven (7) days. While the exact
20 timing of each hepatotoxic event is not known, Plaintiffs are informed and believe that they
21 occurred at least several weeks, if not months, prior to the end of the Class Period. Plaintiffs base
22 this information and belief on the following facts: (a) it is highly improbable that six separate
23 hepatotoxic events in trials that had been ongoing since 2013-2015 suddenly occurred at a single
24 moment in time in late 2016. An even distribution of those events during the time of the clinical
25 trials would suggest that multiple adverse hepatotoxic events had already occurred prior to the
26 Class Period; (b) Seattle Genetics was permitted a week after each event before they were required
27 to report the event to the FDA; (c) the FDA would require time to process and review the adverse

1 event reports; and (d) once the FDA decided to consider initiating clinical holds, as it ultimately
2 did at the end of the Class Period, regulations specified that it should generally attempt to resolve
3 the issue with the sponsor before initiating the hold, all of which suggests that the adverse events
4 here were part of a chain of events that began long before December 27, 2016.

5 **DEFENDANTS' MATERIALLY FALSE AND MISLEADING**
6 **STATEMENTS AND OMISSIONS DURING THE CLASS PERIOD**

7 48. The Class Period begins on October 27, 2016. On a conference call that day held in
8 connection with the Company's earnings report for the third quarter of 2016, Defendant Drachman
9 made the following misrepresentations in response to an analyst question about the results of the
10 7+3 Phase I study:

11 <Q - Cory W. Kasimov>: Hey, good afternoon, guys. Thanks for taking the
12 questions. I guess, first, to follow up on the SGN-CD33A program. With the data
13 that we're going to see at ASH in combination with 7+3, are you able to say roughly
14 how many patients or how much follow-up we might see in San Diego?

15 <A - Clay B. Siegall>: I'll turn that over to Jonathan and see what Jonathan is
16 willing to tell you.

17 <A - Jonathan Drachman>: All right. So, Cory, thanks for the question. We can't
18 disclose exactly the details of the number of patients or the follow up. The study
19 has been going on for a while, not a huge amount of time. And just as a reminder
20 to people about the frontline younger patients and what the goal is. *This is really*
21 *an area where more than half the patients are treated aggressively with 7+3 or a*
22 *similar regimen with an intent to try to cure those patients. There're pretty high*
23 *CR [complete remission] rates, and there is a substantial number of patients who*
24 *are cured with 7+3 consolidation with or without allogeneic transplant in*
25 *remission.* So, it is a complicated space. It's really like trying to redefine frontline
26 therapy with curative intent. *So it's something that we're looking at really closely.*
27 *We're excited about our interim data.* And when we present it, I think it'll be a
good time to evaluate whether we feel like we're making a difference at that time
and then what our next steps will be.

1 49. The statements identified in paragraph 48 were materially false and misleading
2 when made because they omitted the following information necessary to make the statements not
3 misleading under the circumstances under which they were made: (a) that SGN-CD33A had high,
4 known risks of liver toxicity; and (b) as a result, patients exposed to SGN-CD33A in clinical trials
5 were experiencing serious adverse hepatotoxic events.

6 50. On the October 27, 2016 conference call, Defendant Siegall made the following
7 materially false and misleading statements in response to a question from an analyst concerning
8 the difference between SGN-CD33A and AML therapies under development by Seattle Genetics'
9 competitors:

10 <Q - **Tazeen Ahmad**>: Hi. Good afternoon. Thanks for taking my question. One
11 on SGN-CD33A, if I might, just to follow up on some of the conversations we've
12 been having so far on the call. Obviously, this is going to be something that we're
13 all going to be looking for at the ASH meeting, but in terms of how you're thinking
14 of its profile versus other drugs that are in development, there are few companies
15 that are trying to go for similar populations in AML, whether it be AbbVie, Otsuka,
16 Boehringer or a number of other companies. What should we really be looking for?
17 Should we be looking for differences in efficacy or safety at this early stage?

18 <A - **Clay B. Siegall**>: So, there are few [competing drug candidates], but as
19 far as a drug targeted with an antibody and delivering cytotoxic like our antibody-
20 drug conjugate, *we're very happy with our positioning in this field and think that
21 this could make a big difference for patients. And because SGN-CD33A is
22 expressed on all these tumors that with AML, basically all of them could be very
23 user-friendly from a combination standpoint, much like ADCETRIS is user-
24 friendly from a combination standpoint in Hodgkin lymphoma.* Because CD30 is
25 expressed on all Hodgkin lymphoma, basically, and we could use it in combination
26 with other types of therapies, whether they be old-school therapies, like cytotoxics
27 or newer therapies like some of the checkpoint inhibitors. So, we think that ADCs
make extraordinary regimen partners, if you will, with other drugs. And we're going
to continue doing that.

51. The statements identified in paragraph 50 were materially false and misleading
when made because they omitted the following information necessary to make the statements not

1 misleading under the circumstances under which they were made: (a) that SGN-CD33A had high,
2 known risks of liver toxicity; and (b) as a result, patients exposed to SGN-CD33A in clinical trials
3 were experiencing serious adverse hepatotoxic events.

4 52. On November 8, 2016, Defendant Siegall attended the Credit Suisse Health Care
5 Conference and made the following materially false and misleading statements:

6 We're taking this very active single agent drug that we presented more than a
7 year ago and combining it with hypomethylating agents, which are used to treat
8 older AML patients. And what you could see on this slide is a 71% CR/CRi rate,
9 which is way higher than you would see with HMAs alone.

10 And we're excited with the data. The median OS is interim and is moving around,
11 but what's really important is that we have a low 30 and 60-day mortality rate,
12 which we see in these patients and the data that we have presented really support
13 going forward into our phase 3 CASCADE trial.

14

15 *And so we know that there's a good safety profile there and we'll be presenting*
16 *data at the ASH conference to show the detailed safety and efficacy of what we've*
17 *come up with so far.*

18 53. The statements identified in paragraph 52 were materially false and misleading
19 when made because there was not a “good safety profile” for SGN-CD33A, and because the
20 statements omitted the following information necessary to make the statements not misleading
21 under the circumstances under which they were made: (a) that SGN-CD33A had high, known risks
22 of liver toxicity; and (b) as a result, patients exposed to SGN-CD33A in clinical trials were
23 experiencing serious adverse hepatotoxic events.

24 54. On December 3, 2016, Seattle Genetics issued a press release announcing partial
25 results from the 7+3 Phase I study, which included the following statements:

26 **Seattle Genetics Presents Phase 1b Data from Vadastuximab Talirine**
27 **(SGNCD33A; 33A) in Combination with Standard of Care in Frontline Acute**
Myeloid Leukemia at ASH Annual Meeting

1 *-Clinical Data Featured in Press Program and Oral Presentation Indicate 33A is*
2 *Well Tolerated in Combination with 7+3 Induction Therapy in Younger Newly*
3 *Diagnosed AML Patients*

4 *-Antileukemic Activity Data Show Remission Rate of 76 Percent, with 78 Percent*
5 *of Those Remissions Negative for Minimal Residual Disease-*

6

7 “Our clinical trial data reported at ASH demonstrate that adding vadastuximab
8 talirine, also known as 33A, to standard of care treatment results in a rapid, high
9 rate of remissions in frontline, younger AML patients with poor prognosis. Notably,
10 seventy-eight percent of patients who achieved remissions in this trial tested
11 negative for minimal residual disease, which means no cancer could be detected
12 with a sensitive test,” said Jonathan Drachman, M.D., Chief Medical Officer and
13 Executive Vice President, Research and Development at Seattle Genetics. ***“In this***
14 ***trial, 33A in combination with 7+3 was well-tolerated, with a low early mortality***
rate. Based on these promising, early data, we plan to initiate a randomized phase
2 clinical trial in 2017 in younger newly diagnosed AML patients to further evaluate
the potential benefit of adding 33A to standard of care.”

15 “People with acute myeloid leukemia die of infections or bleeding within weeks or
16 a few months of diagnosis without effective, aggressive chemotherapy. Even with
17 current treatment regimens, fewer than 50% of younger adults are successfully
18 treated. ***The phase 1 results of 33A in combination with standard of care show a***
high rate of remissions in younger newly diagnosed AML patients without
significantly adding to the toxicity of the treatment.

19

20 Data were reported from 42 newly diagnosed AML patients with a median age of
21 46 years and intermediate or adverse cytogenetic risk of 50 percent and 36 percent,
22 respectively. Seventeen percent of patients had secondary AML. Key findings
include:

23

- 24
- 25 • ***The most common Grade 3 or 4 treatment-emergent adverse events occurring***
26 ***in 20 percent or more of patients were febrile neutropenia, thrombocytopenia,***
27 ***anemia and neutropenia. No non-hematologic treatment-emergent adverse***
events of Grade 3 or higher were reported in 15 percent or more of patients.

No veno-occlusive disease/sinusoidal obstruction syndrome or significant hepatotoxicity was observed on treatment.

• *The most common Grade 1 and 2 treatment-emergent adverse events occurring in 20 percent or more of patients were nausea, diarrhea, constipation, hypokalemia and decreased appetite. No infusion-related reactions occurred.*

•

55. The statements identified in paragraph 54 were materially false and misleading when made because they omitted the following information necessary to make the statements not misleading under the circumstances under which they were made: (a) that SGN-CD33A had high, known risks of liver toxicity; and (b) as a result, patients exposed to SGN-CD33A in clinical trials were experiencing serious adverse hepatotoxic events, including veno-occlusive disease, even if the observations did not occur in this particular trial.

56. On December 5, 2016, Seattle Genetics issued a press release announcing partial results from the HMA Phase I study, which included the following statements:

– Both Combination and Monotherapy Data Show 33A is Well-Tolerated with Rapid, High Remission Rates for AML Patients in Multiple Phase 1 Trials; Data Highlighted in Three Oral Presentations –

.

.

“We are pleased with the growing body of data demonstrating that vadastuximab talirine, also known as 33A, has a promising overall tolerability and activity profile in clinical trials for patients with AML,” said Jonathan Drachman, M.D., Chief Medical Officer and Executive Vice President, Research and Development at Seattle Genetics.

.

1 Data were reported from 53 frontline AML patients with a median age of 75 years
2 and predominantly intermediate or adverse cytogenetic risk who had declined
3 intensive therapy. Regarding additional poor-prognosis indicators, 42 percent of
4 patients had evidence of underlying myelodysplasia, 11 percent had FLT3-mutated
5 disease and 43 percent had secondary AML, which is AML that arises from prior
6 chemotherapy, a pre-existing MDS or myeloproliferative disease. Key findings
7 include:

8

- 9 • With a median follow-up of 14.7 months, median overall survival for all patients
10 was 11.3 months and 28 percent of patients remained alive and on study as of last
11 follow-up. The 30- and 60-day mortality rates were two and eight percent, with no
12 treatment-related deaths occurring during that time.

13

- 14 • *The most common Grade 3 or 4 treatment-emergent adverse events occurring in
15 20 percent or more of patients were thrombocytopenia, febrile neutropenia,
16 anemia and neutropenia.*
- 17 • *The most common Grade 1 and 2 treatment-emergent adverse events occurring
18 in 20 percent or more of patients were fatigue, nausea, constipation, peripheral
19 edema and decreased appetite.*

20 **Vadastuximab Talirine Monotherapy in Older Patients with Treatment Naive
21 CD33-Positive Acute Myeloid Leukemia (Abstract #590, oral presentation
22 on Monday, December 5, 2016 at 7:15 a.m. PT)**

23 Interim results from 93 patients in the ongoing phase 1 study evaluating 33A
24 monotherapy in AML patients were previously presented at the 2015 ASH Annual
25 Meeting. New results describing the safety and activity of the recommended 33A
26 monotherapy dose of 40 micrograms per kilogram (mcg/kg) in an expansion cohort
27 of treatment-naïve older AML patients were presented by Dr. Anjali
Advani, Cleveland Clinic.

Data were reported from 27 treatment-naïve older AML patients with a median age
of 74 years and intermediate or adverse cytogenetic risk of 70 percent and 26
percent, respectively. Regarding additional poor-prognosis indicators, 48 percent

1 of patients had evidence of underlying myelodysplasia and 22 percent had FLT3
2 mutated disease. Key findings include:

3

- 4 • *The most common Grade 3 or higher treatment-emergent adverse events*
5 *occurring in 20 percent or more of patients were thrombocytopenia, febrile*
6 *neutropenia and anemia.*
- 7 • *The most common Grade 1 and 2 treatment-emergent adverse events occurring*
8 *in 20 percent or more of patients were peripheral edema, decreased appetite,*
9 *fatigue, diarrhea and dizziness.*

10 57. The statements identified in paragraph 56 were materially false and misleading
11 when made because they omitted the following information necessary to make the statements not
12 misleading under the circumstances under which they were made: (a) that SGN-CD33A had high,
13 known risks of liver toxicity; and (b) as a result, patients exposed to SGN-CD33A in clinical trials
14 were experiencing serious adverse hepatotoxic events.

15 **The Truth Emerges**

16 58. On December 27, 2016, Seattle Genetics issued a press release, which was also
17 filed with the SEC as an attachment to a Current Report on Form 8-K, disclosing that six patients
18 in SGN-CD33A trials experienced hepatotoxic events, including “several cases of veno-occlusive
19 disease” and four deaths, and that as a result “several Phase I trials” were placed on clinical hold
20 by the FDA:

21 **Seattle Genetics Announces Clinical Hold on Several Phase 1 Trials of**
22 **Vadastuximab Talirine (SGN-CD33A)**

23 -Enrollment Continues on Phase 3 CASCADE Trial in Acute Myeloid Leukemia
24 and Phase 1/2 Trial in Myelodysplastic Syndrome-

25 BOTHELL, Wash.--(BUSINESS WIRE)--Dec. 27, 2016-- Seattle Genetics,
26 Inc. (Nasdaq:SGEN), a global biotechnology company, today announced that *it has*
27 *received notice from the U.S. Food and Drug Administration (FDA) that a*

1 *clinical hold or partial clinical hold has been placed on several early stage trials*
2 *of vadastuximab talirine (SGN-CD33A) in acute myeloid leukemia (AML). The*
3 *clinical holds were initiated to evaluate the potential risk of hepatotoxicity in*
4 *patients who were treated with SGN-CD33A and received allogeneic stem cell*
5 *transplant either before or after treatment. Six patients have been identified with*
6 *hepatotoxicity, including several cases of veno-occlusive disease, with four fatal*
7 *events.* Overall, more than 300 patients have been treated with SGN-CD33A in
8 clinical trials across multiple treatment settings. Seattle Genetics is working
9 diligently with the FDA to determine whether there is any association between
10 hepatotoxicity and treatment with SGN-CD33A, to promptly identify appropriate
11 protocol amendments for patient safety and to enable continuation of these trials.

12 The phase 1/2 trial of SGN-CD33A monotherapy in pre- and post-allogeneic
13 transplant AML patients has been placed on full clinical hold. Two phase 1 trials
14 have been placed on partial clinical hold (no new enrollment, existing patients may
15 continue treatment with re-consent). These studies are SGN-CD33A monotherapy,
16 including a subset of older AML patients in combination with hypomethylating
17 agents, and SGN-CD33A combination treatment with 7+3 chemotherapy in newly
18 diagnosed younger AML patients. No new studies will be initiated until the clinical
19 holds are lifted.

20 Seattle Genetics' other ongoing trials of SGN-CD33A, including the phase 3
21 CASCADE trial in older AML patients and phase 1/2 trial in myelodysplastic
22 syndrome, are proceeding with enrollment.

23 59. On this news, Seattle Genetics' stock price declined by \$9.50 per share, or by over
24 15%, to close at \$52.36 on December 27, 2016.

25 60. Analysts expressed surprise. In particular, Kennen MacKay, a research analyst at
26 Credit Suisse wrote that:

27 This morning, SGEN announced several clinical holds imposed by the FDA upon
SGN-CD33A ph1 trials driven by cases of [HVOD]. This comes as a surprise to us
given: 1) SGN-CD33A was designed to address the [ADC] linker technology
pitfalls of its predecessor Mylotarg, thought to be the cause of Mylotarg-associated
HVOD, and 2) prior to this report no HVOD concerns had been observed in early
stage SGN-CD33A testing. Recall that it was a greater number of HVOD-related
deaths which negatively skewed Mylotarg's risk/reward profile and drove Pfizer to

1 voluntarily withdraw the product from the market after having received accelerated
2 approval. Furthermore, while the incidence of Mylotarg's HVOD has been
3 commonly attributed to premature cleavage of the cytotoxic payload, concern
4 continues to exist surrounding potential CD33 target-mediated hepatotoxicity.
5 Given these dynamics, we lower our PoS for SGN-CD33A in frontline older/unfit
6 AML to 30% (from 70% previously) and to 15% in frontline younger/fit AML
7 (from 30% previously), resulting in our (\$10) reduction to \$60 (from \$70
8 previously), and remain Neutral-rated.

9 McKay also noted that Defendant Siegall, in a private conversation, declined to specify in which
10 of the Phase I trials the deaths occurred.

11 **Post-Class Period Events**

12 61. On March 6, 2017, Seattle Genetics issued a press release and filed the same with
13 the SEC as an attachment to a Current Report on Form 8-K, announcing that the Company had
14 decided to abandon the Stem Cell Phase I/II trial of SGN-CD33A.

15 62. The Company also announced that it had implemented a series of risk mitigation
16 measures with respect to hepatotoxicity for all other trials of SGN-CD33A, including modifying
17 eligibility standards to exclude patients with liver cirrhosis, and constituting an adjudication
18 committee to verify incidences of HVOD and potentially terminating the treatment if future
19 incidences of HVOD are found. With these measures, the Company indicated that the FDA had
20 agreed to allow it to resume enrollment in the HMA Phase I and 7+3 Phase I trials.

21 **CLASS-ACTION ALLEGATIONS**

22 63. Lead Plaintiff brings this action as a class action pursuant to Federal Rule of Civil
23 Procedure 23(a) and (b)(3) on behalf of all persons or entities that purchased or otherwise acquired
24 Seattle Genetics' common stock between October 27, 2016 and December 27, 2016, both dates
25 inclusive, seeking to pursue remedies under §§10(b) and 20(a) of the Exchange Act. Excluded are
26 Defendants herein, the officers and directors of the Company, at all relevant times, members of
27 their immediate families and their legal representatives, heirs, successors or assigns and any entity
in which Defendants have or had a controlling interest.

1 64. Class members are so numerous that joinder of all members is impracticable.
2 Throughout the Class Period, Seattle Genetics' common stock was actively traded on the
3 NASDAQ Global Select Market. Because the overwhelming majority of owners hold shares in
4 street name, Lead Plaintiff believes that there are hundreds or thousands of members in the
5 proposed Class. Potential Class members may be identified from records maintained by Seattle
6 Genetics, its transfer agents, and brokers and banks that hold shares beneficially for investors in
7 street name, and may be notified of the pendency of this action by mail, using the form of notice
8 similar to that customarily used in securities class actions.

9 65. Lead Plaintiff's claims are typical of the claims of those of the Class, as all Class
10 members were similarly affected by Defendants' wrongful conduct in violation of federal law
11 complained of herein.

12 66. Lead Plaintiff will fairly and adequately protect the interests of the members of the
13 Class and has retained counsel competent and experienced in class action and securities litigation.

14 67. Common questions of law and fact exist as to all Class members and predominate
15 over any questions solely affecting individual Class members. Among the questions of law and
16 fact common to the Class are:

- 17 a. whether the Defendants Siegall, Simpson and Drachman are control persons of
18 Seattle Genetics for purposes of the Exchange Act;
- 19 b. whether Seattle Genetics and the Individual Defendants failed to disclose material
20 information regarding SGN-CD33A therapy and its known risk of hepatotoxicity;
- 21 c. whether Seattle Genetics and the Individual Defendants made misrepresentations
22 or omissions with scienter;
- 23 d. whether the federal securities laws were violated by Defendants' acts as alleged
24 herein;
- 25 e. whether the prices of Seattle Genetics' securities during the Class Period were
26 artificially inflated because of the Defendants' conduct complained of herein; and
- 27 f. whether the Class has sustained damages as a result of the disclosures alleged herein

1 with respect to their Exchange Act claims and, if so, what is the proper measure of
2 damages.

3 68. A class action is superior to all other available methods for the fair and efficient
4 adjudication of this controversy since joinder of all members is impracticable. Furthermore, as the
5 damages suffered by individual Class members may be relatively small, the expense and burden
6 of individual litigation make it impossible for Class members to individually redress the wrongs
7 done to them. There will be no difficulty in the management of this action as a class action.

8 69. With respect to the Exchange Act claims, Lead Plaintiff will rely, in part, upon the
9 presumption of reliance established by the fraud-on-the-market doctrine in that:

- 10 a. Defendants made public misrepresentations or failed to disclose material facts
11 during the Class Period;
- 12 b. the omissions and misrepresentations were material;
- 13 c. Seattle Genetics' securities are traded in efficient markets;
- 14 d. the Company's shares were liquid and traded with moderate to heavy volume
15 during the Class Period;
- 16 e. the Company traded on the NASDAQ, and was covered by multiple analysts;
- 17 f. the misrepresentations and omissions alleged would tend to induce a reasonable
18 investor to misjudge the value of the Company's securities; and
- 19 g. Lead Plaintiff and the Class members purchased and/or otherwise acquired Seattle
20 Genetics' common stock between the time the Defendants failed to disclose or
21 misrepresented material facts and the time the true facts were disclosed, without
22 knowledge of the omitted or misrepresented facts.

23 70. Based upon the foregoing, Lead Plaintiff and other Class members are entitled to a
24 presumption of reliance upon the integrity of the market.

25 71. Alternatively, Lead Plaintiff and the Class members are entitled to the presumption
26 of reliance established by the Supreme Court in *Affiliated Ute Citizens of the State of Utah v.*
27

1 *United States*, 406 U.S. 128, 92 S. Ct. 2430 (1972), as Defendants omitted material information in
2 violation of a duty to disclose such information, as detailed above.

3 **COUNT I**

4 **Violation of § 10(b) of the Exchange Act and Rule 10b-5**

5 **(against all Defendants)**

6 72. Plaintiffs repeat and reallege the allegations contained in Paragraphs 1 to 71 above
7 as if fully set forth herein.

8 73. This Count is asserted against Seattle Genetics and each of the Individual
9 Defendants for violations of Section 10(b) of the Exchange Act, 15 U.S.C. § 78j(b), and Rule 10b-
10 5 promulgated thereunder by the SEC.

11 74. During the Class Period, Defendants engaged in a plan, scheme, conspiracy and
12 course of conduct, pursuant to which they knowingly or recklessly engaged in acts, transactions,
13 practices and courses of business which operated as a fraud and deceit upon the Lead Plaintiff and
14 the other members of the Class; made various untrue statements of material facts and omitted to
15 state material facts necessary in order to make the statements made, in light of the circumstances
16 under which they were made, not misleading; and employed devices, schemes and artifices to
17 defraud in connection with the purchase and sale of securities. Such scheme was intended to, and,
18 throughout the Class Period, did: (i) deceive the investing public, including Lead Plaintiff and
19 other Class members, as alleged herein; (ii) artificially inflate and maintain the market price of
20 Seattle Genetics' securities; and (iii) cause Lead Plaintiff and other members of the Class to
21 purchase or otherwise acquire Seattle Genetics' securities and options at artificially inflated prices.

22 75. Specifically, Seattle Genetics and Defendants Siegall, Simpson and Drachman
23 made material misrepresentations and omissions as particularized in Paragraphs ___ to ___.

24 76. By virtue of their positions at Seattle Genetics, Defendants Siegall, Simpson and
25 Drachman had actual knowledge of the materially false and misleading statements and material
26 omissions alleged herein and intended thereby to deceive Lead Plaintiff and the other members of
27 the Class, or, in the alternative, acted with reckless disregard for the truth in that they failed or

1 refused to ascertain and disclose such facts as would reveal the materially false and misleading
2 nature of the statements made, although such facts were readily available to Seattle Genetics and
3 Defendants Siegall, Simpson and Drachman. In addition to the facts alleged herein demonstrating
4 a strong inference of scienter, certain information showing that Defendants Siegall, Simpson and
5 Drachman acted knowingly or with reckless disregard for the truth is peculiarly within these
6 Individual Defendants' knowledge and control. As the senior managers of Seattle Genetics, these
7 Individual Defendants had knowledge of the details of Seattle Genetics' internal affairs, SGN-
8 CD33A and its known risk of hepatotoxicity.

9 77. As officers and/or directors of a publicly-held company, Defendants Siegall,
10 Simpson and Drachman had a duty to disseminate timely, accurate, full and truthful information
11 regarding Seattle Genetics' business, operations, and financial controls. As a result of the
12 dissemination of the aforementioned false and misleading reports and filings, the market price of
13 Seattle Genetics' securities was artificially inflated throughout the Class Period.

14 78. In ignorance of the adverse facts concerning Seattle Genetics' operations which
15 were concealed by the misrepresentations and omissions alleged herein, Lead Plaintiff and the
16 other members of the Class purchased or otherwise acquired Seattle Genetics securities at
17 artificially inflated prices and relied upon the price of the securities, the integrity of the market for
18 the securities and/or upon statements disseminated by Defendants, and were damaged upon the
19 disclosure of Defendants' wrongdoing described herein.

20 79. During the Class Period, Seattle Genetics' securities were traded on an active and
21 efficient market. Lead Plaintiff and the other members of the Class, directly relying on the
22 materially false and misleading statements described herein, and/or relying upon the integrity of
23 the market, purchased or otherwise acquired shares of Seattle Genetics securities at prices
24 artificially inflated by Defendants' wrongful conduct. Had Lead Plaintiff and the other members
25 of the Class known the truth, they would not have purchased or otherwise acquired said securities,
26 or would not have purchased or otherwise acquired them at the inflated prices that were paid. At
27 the time of the purchases and/or acquisitions by Lead Plaintiff and the Class, the true value of

1 Seattle Genetics' securities was substantially lower than the prices paid by Lead Plaintiff and the
2 other members of the Class. The market price of Seattle Genetics' securities declined sharply upon
3 public disclosure of the facts alleged herein to the injury of Lead Plaintiff and Class members.

4 80. By reason of the conduct alleged herein, Seattle Genetics and the Individual
5 Defendants knowingly or recklessly violated Section 10(b) of the Exchange Act and Rule 10b-5
6 promulgated thereunder.

7 81. As a direct and proximate result of these Defendants' wrongful conduct, Lead
8 Plaintiff and the other Class members suffered damages in connection with their respective
9 purchases, acquisitions and sales of the Company's securities during the Class Period upon the
10 disclosures alleged herein. Seattle Genetics and Individual Defendants are liable for damages in
11 connection with these losses under Section 10(b) of the Exchange Act and Rule 10b-5 promulgated
12 thereunder.

13 **COUNT II**

14 **Violation of § 20(a) of the Exchange Act**

15 **(against Defendants Siegall, Simpson, and Drachman)**

16 82. Lead Plaintiff repeats and realleges allegations contained in Paragraphs 1 to 81
17 above, as if fully set forth herein.

18 83. During the Class Period, Defendants Siegall, Simpson, and Drachman participated
19 in the operation and management of Seattle Genetics, and conducted and participated, directly and
20 indirectly, in the conduct of Seattle Genetics' business affairs. Because of their senior positions,
21 they knew the adverse non-public information about Seattle Genetics' operations, SGN-CD33A
22 and its known hepatotoxicity.

23 84. As officers of a publicly owned company, these Defendants had a duty to
24 disseminate accurate and truthful information with respect to Seattle Genetics' reports and filings
25 and to correct promptly any public statements issued by Seattle Genetics, which had become
26 materially false or misleading.

1 85. Because of their positions of control and authority as senior officers of the
2 Company, Defendants Siegall, Simpson, and Drachman were able to, and did, control the contents
3 of the various reports, press releases, investor conferences and public filings which Seattle
4 Genetics disseminated in the marketplace during the Class Period. Throughout, the Class Period,
5 Defendants Siegall, Simpson, and Drachman exercised power and authority to cause Seattle
6 Genetics to engage in the wrongful conduct complained of herein.

7 86. As control persons, Defendants Siegall, Simpson, and Drachman are liable pursuant
8 to Section 20(a) of the Exchange Act for the primary violations of the Exchange Act committed
9 by Seattle Genetics as set forth in Count I.

10 **REQUEST FOR RELIEF**

11 Lead Plaintiff requests judgment against Defendants as follows:

12 A. Determining that this action may be maintained as a class action under Rule 23 of
13 the Federal Rules of Civil Procedure, and certifying Lead Plaintiff as the Class Representative;

14 B. Requiring Defendants to pay damages sustained by Lead Plaintiff and the Class
15 by reason of the acts and transactions alleged herein;

16 C. Awarding Lead Plaintiff and the other members of the Class prejudgment and post-
17 judgment interest, as well as their reasonable attorneys' fees, expert fees, and other costs to the
18 extent allowable; and
19

20 D. Awarding such other and further relief as this Court may deem just and proper.

21 **DEMAND FOR JURY TRIAL**

22 Lead Plaintiff hereby demands a trial by jury of all issues so triable.

23 Dated: June 6, 2017

24 Respectfully submitted,

25 *s/ Cliff Cantor*

26 By: Cliff Cantor, WSBA # 17893

27 LAW OFFICES OF CLIFFORD A. CANTOR, P.C.

627 208th Ave. SE
Sammamish, WA 98074
Tel: (425) 868-7813
Fax: (425) 732-3752
email: cliff.cantor@outlook.com

Plaintiffs' Liaison Counsel

POMERANTZ LLP
Patrick V. Dahlstrom
Joshua B. Silverman
Omar Jafri
Ten South La Salle Street, Suite 3505
Chicago, Illinois 60603
Tel: (312) 377-1181
Fax: (312) 377-1184
email: pdahlstrom@pomlaw.com
jbsilverman@pomlaw.com
ojafri@pomlaw.com

POMERANTZ LLP
Jeremy A. Lieberman
J. Alexander Hood II
600 Third Avenue, 20th Floor
New York, New York 10016
Tel: (212) 661-1100
Fax: (212) 661-8665
email: jalieberman@pomlaw.com
ahood@pomlaw.com

Plaintiffs' Lead Counsel

Certificate of Service

I certify that, on the date stamped above, I caused this document to be filed with the Clerk of the Court using the CM/ECF system, which will send notification of filing by email to counsel of record for all parties.

s/ Cliff Cantor, WSBA #17893