UNITED STATES DISTRICT COURT
SOUTHERN DISTRICT OF NEW YORK

JENNIFER TUNG, Individually and on Behalf of All Others Similarly Situated, Plaintiff, v. BRISTOL-MYERS SQUIBB COMPANY, MICHAEL GIORDANO, FOUAD NAMOUNI, FRANCIS M. CUSS, GIOVANNI CAFORIO, LAMBERTO ANDREOTTI, and CHARLES A. BANCROFT, Defendants.	Civil Action No. 1:18-cv-01611-JPO CLASS ACTION **JURY TRIAL DEMANDED**

CONSOLIDATED SECOND AMENDED CLASS ACTION COMPLAINT FOR VIOLATIONS OF THE FEDERAL SECURITIES LAWS

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Lead Plaintiffs Arkansas Public Employees Retirement System ("APERS") and the Louisiana Sheriffs' Pension & Relief Fund ("Louisiana Sheriffs"; with APERS, "Lead Plaintiffs"), together with named plaintiff Erste Asset Management GmbH ("Erste AM"; with Lead Plaintiffs, "Plaintiffs"), by and through their counsel, bring this class action under Sections 10(b), 20(a), and 20A of the Securities Exchange Act of 1934 (the "Exchange Act") and Rule 10b-5 promulgated thereunder on behalf of themselves and all persons or entities who purchased or otherwise acquired the securities of Defendant Bristol-Myers Squibb Company ("Bristol-Myers" or the "Company") during the period from January 27, 2015 through October 9, 2016 (the "Class Period"), and were damaged thereby (the "Class"). Plaintiffs allege the following upon information and belief, except as to those allegations concerning Plaintiffs, which Plaintiffs allege upon personal knowledge. Plaintiffs' information and belief are based upon Lead Counsel's investigation, which included review and analysis of, *inter alia*: (i) regulatory filings made by Bristol-Myers with the United States Securities and Exchange Commission ("SEC"); (ii) press releases and public statements by the Company; (iii) analyst reports concerning Bristol-Myers; (iv) interviews with former Bristol-Myers employees; (v) consultation with experts, including Ronald H. Blum M.D., an expert in the field of medical oncology with more than forty years' experience as a clinician, researcher, clinical trials expert, academic, and teacher; and (vi) other public information relevant to this action. Lead Counsel's investigation into the factual allegations contained herein is continuing, and many of the relevant facts are known only by Defendants or are exclusively within their custody or control. Plaintiffs believe that substantial additional evidentiary support will exist for the allegations set forth herein after a reasonable opportunity for further investigation or discovery.

I. INTRODUCTION

1. Throughout the Class Period, Bristol-Myers and the Company's top executives made materially false and misleading statements and omissions concerning the design, primary

outcome, and inherent risk of a closely-watched clinical study—Checkmate-026. Checkmate-026 sought new label approval by the United States Food and Drug Administration ("FDA") for the use of the Company's heralded new immuno-oncology drug, Opdivo, as a first line treatment for non-small cell lung cancer ("NSCLC"). Lung cancer impacts hundreds of thousands of new patients every year, and Opdivo and its potential use as a first-line treatment for NSCLC raised the hopes of patients desperate to prolong their lives. This action arises from materially false, incomplete, and misleading statements and omissions made or caused to be made by Defendants during the Class Period in violation of Sections 10(b), 20(a), and 20A of the Exchange Act.

2. By way of background, Checkmate-026 was at the center of Bristol-Myers' strategy to focus on the emerging field of immuno-oncology that involves cancer therapies that look to use the body's own immune system in place of, or in combination with, cancer treatments such as chemotherapy. The *Wall Street Journal* called immuno-oncology "***the next big hope for the drug sector***" in search of "blockbuster sales." Defendants had largely pinned their hopes on Bristol-Myers' flagship immuno-oncology drug, Opdivo. Opdivo works by blocking the interaction between two proteins: "programmed death receptor 1" ("PD-1"), a protein on the surface of immune system cells; and PD-L1, a ligand that binds with PD-1 and causes the immune system to stop attacking. After the discovery of PD-L1 on the surface of cancerous cells, Opdivo was developed to switch back on the body's natural defenses by preventing the two proteins from binding and resulting in the selective killing of cancer cells.

3. Only a certain subset of cancer patients express PD-L1, including (according to Defendants) up to an estimated 70% of NSCLC patients. The strength of a patient's expression of PD-L1 is represented as a percentage, with the higher the percentage referred to as a higher strength of expression. By the start of the Class Period, well-publicized immuno-oncology research and

studies determined that 25% of cancer patients had cancerous cells that exhibited a level of 50% PD-L1 expression. A 50% level was widely defined to be "strong" and increased the chance that the cancer patient would benefit from PD-1 inhibitors, such as Opdivo. Conversely, research suggested that mere PD-L1 positivity alone—which Bristol-Myers and others in the field had, since the earliest studies, most commonly defined as 5% PD-L1 expression—was insufficiently predictive of therapeutic benefit from PD-1 inhibitors.

4. Defendants hailed Opdivo as one of the Company's most "important asset[s] in [its] immuno-oncology portfolio." With Checkmate-026, Defendants sought to achieve something remarkable: the replacement of chemotherapy with Opdivo as a first-line monotherapy for patients diagnosed with NSCLC and exhibiting "strong" or "high positive" PD-L1 expression. The success of Checkmate-026 could not only give Opdivo access to what was widely considered the most lucrative market in cancer treatment, but also secure a superior label for Opdivo and establish it as the premiere immuno-oncology therapy—and Bristol-Myers as the premiere immuno-oncology developer.

5. At the start of the Class Period, numerous competitors raced to catch up with Opdivo's early successes, but two companies dominated the PD-1 inhibitor field—Bristol-Myers and Merck & Co. ("Merck")—with Merck's parallel treatment, Keytruda, posing the biggest threat to Opdivo's dominance. Experts predicted that immuno-therapy treatment would soon be a $40 billion industry, and that the treatment of lung cancer was seen as its "most lucrative use." Checkmate-026 promised to open a breakthrough market—the replacement of chemotherapy with first-line treatment of NSCLC, the most common form of lung cancer that impacts hundreds of thousands of people each year. Thus, investors closely watched Checkmate-026 and awaited its heavily anticipated conclusion.

6. This is not a case about a clinical trial that failed. This is a case about Defendants' misleading statements and omissions concerning the design and inherent risk posed by the unusually aggressive and secret design of Checkmate-026. Defendants' misrepresentations made it impossible for investors to accurately assess, and in fact actively misled investors about, the undisclosed risk that the study's focus on a patient pool with a minimal 5% PD-L1 expression level—"one of the most important parameters in patient selection," according to a former Bristol-Myers employee involved in the Checkmate-026 study—increased exponentially the risk that Checkmate-026 would fail to achieve its secretly expansive primary outcome. This risk was directly linked to Defendants' make-or-break strategy to control what the *Wall Street Journal* called "***the next big hope for the drug sector***"—and for the hundreds of thousands suffering from lung cancer and their families.

7. Defendants designed Checkmate-026 to target patients with just a minimal and relatively weak 5% expression of PD-L1. This was an aggressive gamble that, if successful, could have resulted in a superior label for Opdivo over its competitors but, if unsuccessful, could deprive lung cancer patients from a much-anticipated treatment option and cost Bristol-Myers its status as the frontrunner in immuno-oncology. In reality—and undisclosed to investors—Defendants' gamble added considerable risks to Checkmate-026 and threatened Bristol-Myers' ability to achieve any FDA approval at all for the use of Opdivo to treat NSCLC as a first-line alternative to chemotherapy. The 5% PD-L1 expression focus also threatened the ability of the clinical trial to provide meaningful data about the performance of Opdivo at stronger expression levels, like those used by Merck. Yet, throughout the Class Period, Defendants actively misled investors about Checkmate-026's use of a mere 5% expression level. While Defendants declined to enumerate the exact expression level used, even in the face of direct questioning, they publicly maintained that

Checkmate-026 focused on only those with cancer cells exhibiting a "strong" or "high positive" expression of PD-L1, the same terminology Bristol-Myers' primary competitor used to define 50% expression. Defendants' representations reasonably and predictably led the market to believe that patients with a far higher level of PD-L1 expression (and thus with a far greater chance of successful treatment) were the focus of the study.

8. Throughout the Class Period, Defendants responded to the market's questions about the clinical trial by stating in publicly-available reports and in other statements to investors that Checkmate-026's primary objective was to demonstrate that Opdivo was superior to chemotherapy in "***strongly*** PD-L1+ tumor expressing patients." By repeatedly stating that the clinical trial targeted "strongly" or "high positive" PD-L1 expressing patients, Defendants falsely indicated that they had designed Checkmate-026 conservatively and maximized its chances for success. Further, in response to the market's continued interest in Checkmate-026 throughout the Class Period—including in particular the clinical study's focus on "strong" expressors—Defendants repeatedly emphasized the clinical trial's superior design, Bristol-Myers' consideration of the role of PD-L1 expression in the study's design, and their confidence in a positive outcome, even just days before announcing its disastrous results. The market understood that the use of the words "strong" or "high" commonly referred to 50% or greater expression of PD-L1 in cancerous cells.

9. Defendants' mischaracterization of Checkmate-026's primary focus on a "strong" or "high" PD-L1 expression patient pool (when it was secretly a 5% PD-L1 expression cutoff) was directly contrary to Bristol-Myers' own consistent and public definition of 5% PD-L1 expression as a marker of mere or minimal positive expression. Defendants' mischaracterization was also in clear conflict with the immuno-oncology field's use of those terms—based in large part on

Bristol-Myers' own published Checkmate studies—a fact confirmed not only by various contemporaneous articles and research papers in prominent medical publications, but by a top clinician and academic in the immuno-oncology field. In addition to their review of, and citation to, numerous contemporaneous clinical trials and papers, Lead Counsel consulted with Ronald H. Blum, M.D., a Medical Oncologist with more than forty years' experience as a clinician, researcher, clinical trials expert, academic, and researcher, with a focus on the study and treatment of lung cancer. As discussed further herein, Dr. Blum has directly participated in the independent oversight of late phase clinical trials, including clinical trials examining PD-1 checkpoint inhibitors such as Checkmate-026, and has led international clinical trials focused on lung cancer sponsored by the National Cancer Institute and the National Institute of Health. Dr. Blum states that, by no later than the start of the Class Period, there was an industrywide consensus among all major participants in the immuno-oncology industry (which includes Bristol-Myers and the named Individual Defendant executives) that (a) a 5% PD-L1 expression level denoted a low or minimal expression; (b) a 50% PD-L1 expression level was commonly referred to as "strong" or "high"; and (c) Defendants' persistent characterization of Checkmate-026's 5% expression as "strong" or "high" until August 5, 2016 was inconsistent with the industry's contemporaneous use of "strong" PD-L1 expression.

10. If successful, Defendants' secret gamble would have resulted in a superior label for Opdivo over Merck's Keytruda, which was undergoing a contemporaneous trial—Keynote-024—aimed at "strong" PD-L1 expressors, meaning at least at the 50% level. In the new and rapidly evolving industry of PD-1 checkpoint inhibitors, the explicit definition of "strong" PD-L1 expression by one of the industry's dominant players settled the industry consensus that "strong" referred to 50% or greater PD-L1 expression—a consensus that, for nearly the entire Class Period,

Defendants expressly refused to contradict. Defendants' secret strategy backfired, however, because Merck's Keynote-024 was an absolute success, and bestowed Merck with the first—and, as of this filing, still only—label to market a PD-1 checkpoint inhibitor as a first-line treatment for NSCLC.

11. Meanwhile, Defendants' hidden gamble to misrepresent Checkmate-026's primary focus in order to capture an outsized portion of the market share for Opdivo completely failed. On August 5, 2016, Bristol-Myers stunned investors by revealing that the purportedly "strong" PD-L1 expression cutoff of Checkmate-026 ***had been just 5%***, a level that investors knew had most commonly indicated only the ***bare minimum expression of PD-L1 positivity*** since the very first published studies in the industry for PD-1 checkpoint inhibitors. Bristol-Myers relatedly reported that, in contrast to Merck's Keynote-024 study (which expressly targeted 50% or greater PD-L1 expressors), Checkmate-026 failed to show that Opdivo was more effective than chemotherapy.

12. Medical professionals and investors alike reacted with uncharacteristic disbelief in August 2016. One seasoned industry observer called the failure a "MAJOR SURPRISE – ***possibly the biggest clinical surprise of my career***." BMO Capital Markets stated in its analyst report issued on August 5, 2016, that it was "***completely puzzled*** by Bristol's decision to evaluate . . . at a threshold this low [*i.e.* 5%], particularly given that the trial description indicated patients would be strongly expressing PD-L1." Another analyst noted its belief that Defendants had misled the market all along, stating "Based on the available data, ***we had confidence in the CheckMate-026 trial; however, the expression level cut-off for the primary analysis of 5% had not been previously disclosed***."

13. After disclosing Checkmate-026's initial failure on August 5, 2016, Defendants raised investors' expectations that the Company could still extract data for Opdivo's use in treating

NSCLC in the 50% PD-L1 expressors. By this time, however, Defendants knew or should have known that no meaningful extraction of data for 50% expressors was possible because, as a consequence of Defendants' study design, the study did not have a meaningful number of Opdivo-receiving patients with a 50% or greater expression of PD-L1. Indeed, just two months later, on October 9, 2016, Defendants disclosed that Checkmate-26 failed at all levels, with the limited subgroup data it collected at the 50% level insufficient to show any benefit as compared to chemotherapy. With the study yielding no useful information, even at the 50% expression level, analysts commented that Bristol-Myers had experienced its "worst-case scenario."

14. Defendants soon admitted that "the failure of CM-026 was due to [Bristol-Myers'] choosing the wrong end point" of 5% PD-L1 expression—the same end point that, during the Class Period, Defendants had deceptively described as "strong," knowingly using the identical language employed by Merck in describing its 50% cutoff in a competing parallel study. Indeed, Defendant Fouad Namouni, who was responsible for Bristol-Myers' development of Opdivo, ***admitted*** that Defendants' repeated representations that Checkmate-026 focused on "strong" PD-L1 expressors was false, stating weeks after the end of the Class Period, in November 2016, that "[Checkmate-026] was ***not designed to look at the smaller subgroup of . . . high expression of PD-L1***" and "that's the reason [the] study . . . [did] not meet its primary endpoint." Likewise, Bristol-Myers' new research and development chief, who had served on Bristol-Myers' Board of Directors during the Class Period, stated to *Forbes* on June 2, 2017 that "the most likely" cause of the study's failure was the "lower cutoff," the very metric that Defendants doggedly concealed from investors until August 5, 2016.

15. The Individual Defendants (defined below) all knew from the start of the Class Period that Checkmate-026's primary outcome focused on those with 5% expression levels. As

discussed herein, multiple former senior-level Bristol-Myers employees tie the Individual Defendants directly to Bristol-Myers' decision to focus Checkmate-026 on a weak 5% focus, and to publicly describe that cutoff as "strong" and "high." Yet, despite having been repeatedly questioned by analysts about the Checkmate-026 patient pool, the Individual Defendants concealed and actively misled investors about that 5% threshold and, as a result, the true known risk inherent to the clinical trial's design. Moreover, Defendants, who directed the design of Checkmate-026 and were fully aware of its undisclosed aggressive and risky design, sold over ***$75 million*** of Bristol-Myers stock during the Class Period, reaping in profits of nearly ***$55 million***, while purchasing no shares on the open market.

16. Defendants' knowing misrepresentations are evidenced by their own historical, contemporaneous, and post-Class Period statements concerning the minimal indication of expression shown by 5% expression, the industry's acceptance of Bristol-Myers' historical usage of 5% expression to denote mere positivity as well as Merck's and other competitors' usage of 50% expression to denote "strong" expression, and Dr. Blum's confirmation of industry understanding of PD-L1 expression levels, which should be imputed to Defendants who were directly and actively involved in the design and communication of Checkmate-026.

17. Defendants misled investors as to the design and riskiness of Checkmate-026 throughout the Class Period. As a result, investors were caught off guard by Defendants' failure, and the price of Bristol-Myers' stock fell a total of $25.51 per share, from $75.32 at the close of August 4, 2015, to just $49.81 at the close of October 10, 2016, ***a decline of nearly 34%***, causing massive losses to the Class.

II. JURISDICTION AND VENUE

18. The claims asserted herein arise under and pursuant to Sections 10(b), 20(a), and 20A of the Exchange Act (15 U.S.C. §§ 78j(b), 78t(a), and 78t-1) and SEC Rule 10b-5 promulgated thereunder (17 C.F.R. § 240.10b-5).

19. This Court has jurisdiction over the claims asserted in this Complaint pursuant to Section 27 of the Exchange Act, 15 U.S.C. § 78aa, and 28 U.S.C. § 1331.

20. Venue is proper in this judicial district pursuant to Section 27 of the Exchange Act, 15 U.S.C. § 78aa, and 28 U.S.C. § 1391(b). The Company maintains its principal executive offices in this District and did so at all relevant times, and many of the acts and conduct that constitute the violation of law complained of herein, including dissemination to the public of materially false and misleading information, occurred in and/or were issued from this District.

21. In connection with the wrongful acts and conduct alleged in this Complaint, Defendants, directly or indirectly, used the means an instrumentalities of interstate commerce including, but not limited to, the mails, interstate and international telephone communications and the facilities of the New York Stock Exchange ("NYSE"), a national securities exchange located in this District.

III. PARTIES

A. Plaintiffs

22. Co-Lead Plaintiff APERS, is a public pension fund that provides retirement benefits for qualified public employees of the State of Arkansas. APERS was established in 1957 and, as of June 2017, managed assets totaling $8 billion for approximately 92,000 members. As set forth in APERS' previously-filed Certification (ECF No. 26-3), APERS purchased a significant amount of Bristol-Myers common stock during the Class Period and suffered substantial losses as a result of the violations of the federal securities laws as alleged in this action.

23. Co-Lead Plaintiff Louisiana Sheriffs is a multi-employer, defined benefit, governmental retirement plan providing retirement, disability and death benefits to approximately 20,000 active and retired employees of the sheriff's offices in all 64 Louisiana parishes. Louisiana Sheriffs was created by an act of the Louisiana legislature in 1946 and manages more than $3 billion in assets. As set forth in Louisiana Sheriffs' previously-filed Certification (ECF No. 26-3), Louisiana Sheriffs purchased a significant amount of Bristol-Myers common stock during the Class Period and suffered substantial losses as a result of the violations of the federal securities laws as alleged in this action.

24. Additional Named Plaintiff Erste AM is an investment company based in Vienna, Austria. As part of Erste AM's asset management services, it is responsible for managing mutual funds, private funds, and institutional funds. As set forth in Erste AM's previously-filed Certification (ECF No. 18-1), Erste AM purchased a significant amount of Bristol-Myers common stock during the Class Period and suffered substantial losses as a result of the violations of the federal securities laws as alleged in this action.

B. Defendants

25. Defendant Bristol-Myers is a corporation organized under the laws of Delaware with its principal executive offices at 345 Park Avenue, New York, New York. Bristol-Myers is one of the world's largest pharmaceutical companies, and engages in the discovery, development, licensing, manufacturing, marketing, distribution, and sale of pharmaceuticals and related health care products worldwide in several therapeutic areas, including cancer, HIV/AIDS, cardiovascular disease, diabetes, hepatitis, rheumatoid arthritis and psychiatric disorders. As of September 30, 2016, the Company had 1,671,229,946 shares of Bristol-Myers common stock outstanding. During the Class Period, Bristol-Myers common stock was actively and efficiently traded on the NYSE under the ticker symbol "BMY."

26. Defendant Michael Giordano ("Giordano") served as the Company's Senior Vice President and Head of Development for Oncology and Immuno-oncology at all relevant times until July 25, 2016, when he abruptly retired from the Company. In that role, Giordano was responsible for the development strategy of the Company in the areas of oncology and immuno-oncology, providing direction for teams working on Opdivo and other medications. Thus, Giordano was intimately involved in and fully conversant with the development, research, and testing of Opdivo, and was one of Bristol-Myers' chief spokespersons in connection with information provided to the public about Opdivo and Bristol-Myers' strategy for Opdivo, including with respect to clinical testing. As detailed further herein, Giordano directly participated in the study design of Checkmate-026, including specifically the level of PD-L1 expression chosen as its primary focus.

27. Defendant Fouad Namouni ("Namouni") has served as the Company's Head of Oncology Development since July 25, 2016, and prior to that had served as development lead for Opdivo and Yervoy, another of the Company's immuno-oncology drugs, during the relevant time period. In those roles, Namouni was responsible for leading Bristol-Myers' execution of its comprehensive development strategy for the Company's oncology pipeline (including immuno-oncology). Thus, Namouni was intimately involved in and fully conversant with the development, research, and testing of Opdivo, and was also one of Bristol-Myers' chief spokespersons in connection with information provided to the public about Opdivo and Bristol-Myers' strategy for Opdivo, including with respect to clinical testing. As detailed further herein, Namouni directly participated in the study design of Checkmate-026 including, specifically, the level of PD-L1 expression chosen as its primary focus.

28. Defendant Francis M. Cuss ("Cuss") served at all relevant times as the Company's Chief Scientific Officer ("CSO") and Executive Vice President. Prior to becoming CSO, Cuss had

been a key member of Bristol-Myers' research and development team, leading teams in the development of several blockbuster medicines and making significant contributions in the advancement of Bristol-Myers' immuno-oncology portfolios, particularly PD-1 inhibitors such as Opdivo. Since 2010, Cuss had been a part of the Company's Senior Management Team, where he worked closely with Defendant Andreotti. Thus, Cuss was intimately involved in and fully conversant with the development, research, and testing of Opdivo, and was one of Bristol-Myers' chief spokespersons in connection with information provided to the public about Opdivo and Bristol-Myers' strategy for Opdivo, including with respect to clinical testing. In March 2017, as a consequence of the events described herein, Cuss resigned from his role at Bristol-Myers. As described further herein, Cuss specifically reviewed and approved the Company's strategy in Checkmate-026 as part of his role in multiple different committees, including the Brand Development Operating Committee.

29. Defendant Giovanni Caforio ("Caforio") served as the Company's Chief Operating Officer ("COO") from June 5, 2014 to May 5, 2015, and served as its CEO from May 5, 2015 through the end of the Class Period. Prior to becoming COO, Caforio had been at Bristol-Myers for more than a decade, during which time he was heavily involved in the Company's oncology efforts (including immuno-oncology), including serving previously as President of U.S. Operations and as Senior Vice President of Oncology. Thus, Caforio was intimately involved in and fully conversant with the development, research, and testing of Opdivo, and spoke frequently to the public about the strategic importance of immuno-oncology to Bristol-Myers' strategy, and specifically concerning the Company's Opdivo clinical trials. As described further herein, Caforio not only approved the 5% PD-L1 expression cutoff used in Checkmate-026, but Caforio personally

pushed for the Company to pursue "the high case"—meaning the maximum forecasted patient pool with just 5% PD-L1 expression.

30. Defendant Lamberto Andreotti ("Andreotti") began working at Bristol-Myers in 1998, served as the Company's CEO from May 2010 to May 5, 2015, and then as Executive Chairman for the Company's Board of Directors until May 2, 2017. As CEO, Andreotti continued Bristol-Myers' "string of pearls" strategy, which entailed the pursuit of acquisitions of, and partnerships with, pharmaceutical companies whose drugs held promise for replenishing Bristol-Myers' pipeline as it lost patent protection for its older treatments. Relevant to this litigation, that strategy included Bristol-Myers' acquisition of nivolumab (the technical name for the drug Bristol-Myers later marketed as Opdivo) through its purchase of Medarex, Inc. ("Medarex"), and later the Company's collaboration agreement to develop and market nivolumab with Ono Pharmaceutical Co., Ltd. ("Ono").

31. Andreotti's leadership and control of Bristol-Myers continued after he stepped down as CEO in May 2015. As described in the January 20, 2015 press release announcing his planned resignation as CEO, investors were assured that Andreotti would continue to stay actively involved after becoming Executive Chairman. Quoting the Board's then-chairman, the press release stated that Andreotti's "appointment as chairman . . . ensure[s] the continuation of both the leadership and strategy that have made BMS successful." Andreotti's involvement was further detailed in the Company's December 21, 2016 press release, which quoted the Company's lead independent director stating that, as CEO and chairman, Andreotti had "played a critical role in the company's transformation to a BioPharma leader and in shaping the strategy that continues to guide the company today."

32. Accordingly, Andreotti was intimately involved in and fully conversant with the development, research, and testing of Opdivo, and had ultimate discretion over the Company's decision to begin the clinical trial at issue, Checkmate-026. For example, as CEO, Andreotti signed the Company's 2014 and 2015 Forms 10-K (along with Bancroft and (for 2014 only) Caforio), which acknowledged that Defendants were "focusing our efforts and resources in certain disease areas . . . . In particular, Opdivo[.]" Andreotti also served as one of Bristol-Myers' chief spokespersons in connection with information provided to the public about Opdivo and Bristol-Myers' strategy for Opdivo, including with respect to clinical testing.

33. Defendant Charles A. Bancroft ("Bancroft") served at all relevant times as the Company's Chief Financial Officer ("CFO"). In that role, Bancroft was intimately involved in and fully conversant with the development, research, and testing of Opdivo, and was one of Bristol-Myers' chief spokespersons in connection with information provided to the public about Opdivo and Bristol-Myers' strategy for Opdivo, including with respect to clinical testing.

34. The Defendants referenced in ¶¶25-33 above are referred to herein as "Defendants," with Defendants Giordano, Namouni, Cuss, Caforio, Andreotti, and Bancroft referred to herein as the "Individual Defendants."

35. During the Class Period, the Individual Defendants regularly spoke in public, at investors conferences, on earnings calls, and to the media about the Company's immuno-oncology strategy, Opdivo, Checkmate-026, and other relevant subjects as discussed herein.

IV. SUBSTANTIVE ALLEGATIONS

A. Bristol-Myers And Its Competitors Race To Develop PD-1 Checkpoint Inhibitors And Control The Developing Field Of Immuno-Oncology.

36. Prior to and throughout the Class Period, Bristol-Myers focused considerable research and development efforts on the rapidly-evolving field of cancer research and treatment

called immuno-oncology. In contrast to traditional cancer treatments—chemotherapy, radiation therapy, and surgery, all of which focus on attacking the intrinsic properties of the cancerous tumor—immuno-oncology-based cancer treatment attempts to harness the human body's own immune system to fight cancer by targeting pathways that cancer cells use to evade recognition and destruction.

37. Cancer is among the leading causes of death worldwide. In 2012, there were 14.1 million new cases and 8.2 million cancer-related deaths worldwide. The number of new cancer cases per year is expected to rise to 23.6 million by 2030. In the United States alone, approximately 1.7 million new cancer cases are expected to be diagnosed and over 600,000 Americans are expected to die of the disease in 2018. The development of immuno-oncology-based cancer treatments presented an enormous breakthrough in the treatment of cancer and, consequentially, an unprecedented market opportunity for Bristol-Myers and other pharmaceutical companies. On January 1, 2015, *The Guardian* discussed Bristol-Myers' leading role in the development of immuno-oncology treatments, writing that "[i]mmunotherapy is the most exciting development in cancer treatment in years, beginning to take off at a time when much cancer drug research seems to be hitting a brick wall." In 2016, the *Wall Street Journal* called immuno-oncology "***the next big hope for the drug sector***" in search of "blockbuster sales."

38. One of the most promising immuno-oncology treatments concerns so-called PD-1 "checkpoint inhibitors." The body's immune system has a braking mechanism, referred to as a "checkpoint," to prevent immune responses to normal cells. Without this checkpoint, autoimmune disease could flourish. One immune system checkpoint involves the interaction between two proteins. PD-1 is a protein on the surface of immune system attack cells known as "T-cells." PD-L1 is a protein that binds with PD-1 and prevents the T-cell from attacking. While PD-L1 is ordinarily

present on healthy cells, researchers discovered the existence of PD-L1 on the surface of cancerous cells that effectively turns off the immune system T-cells and allows the cancer to grow unchecked.

39. In response to this discovery, Bristol-Myers raced against its competitors to develop a drug to harness the potential of PD-1 in treating cancer by blocking these immune checkpoint proteins, disabling the brake that prevents the immune system from attacking cancer cells. In 2009, Bristol-Myers acquired Medarex for $2.4 billion, largely on the strength of Medarex's checkpoint inhibitor program. In particular, Medarex had developed in the early 2000s a PD-1 checkpoint inhibitor called "nivolumab" (later marketed as Opdivo), and Bristol-Myers' acquisition of the drug through the deal positioned Bristol-Myers as an early leader in immuno-oncology. In 2011, Bristol-Myers secured its rights to develop and commercialize nivolumab by entering into a collaboration agreement with Ono, with whom Medarex had previously licensed certain rights concerning nivolumab. By mid-2012, promising results in one of Bristol-Myers' early nivolumab clinical trials caused considerable excitement, with the *New York Times* writing on June 1, 2012:

> One of the great frustrations for researchers in the war on cancer is that the body's own defense system does not do a better job fighting the disease. Tumors, it turns out, have a molecular shield that repels attacks from the immune system.
>
> Now, a new study says, an experimental drug is showing promise in disabling that shield, unleashing the immune system and causing shrinkage of some lung, skin and kidney cancers that had defied treatment with existing drugs.

The *New York Times* article also quoted an analyst at Leerink Partners ("Leerink") as stating that PD-1 inhibitors "could be the most exciting clinical and commercial opportunity in oncology."

40. Within a year, Bristol-Myers disclosed in SEC filings that it owned a patent covering nivolumab, and that nivolumab had been granted a "Fast Track" designation by the FDA for the treatment of three tumor types: NSCLC, renal cell carcinoma, and advanced melanoma. Investors seized on the fact that Bristol-Myers held an early advantage in the immuno-oncology field, as analysts noted that the Company's stock price was driven upwards in 2013 due to investor

"recognition about the disruptive potential of immunotherapy." Indeed, the market news website *www.streetwisereports.com* noted on July 8, 2013 that shares of Bristol-Myers increased nearly 20% following the days surrounding the May 2013 American Society of Clinical Oncology's (ASCO) 2013 Annual Meeting, at which the Company discussed its various immuno-oncology studies and plans. Moreover, a May 22, 2013 research report titled "Immunotherapy—The Beginning of the End for Cancer," issued by analysts at Citigroup, predicted that the annual market for immunotherapies—defined as including checkpoint agents, vaccines and cell therapy—will exceed $35 billion and become the backbone of treatment in up to 60% of cancers over the next decade.

41. Accordingly, nivolumab quickly became one of the most promising—and most watched—drugs in Bristol-Myers' portfolio, and Defendants recognized the importance of nivolumab to Bristol-Myers investors. For example, on January 24, 2014, during the Company's fourth quarter 2013 earnings conference call, Defendant Andreotti emphasized that "PD-1 is a ***cornerstone for immunotherapy and we are very committed to it***." Defendant Cuss "fully agree[d] with Andreotti's characterization of the promise of PD1." Three weeks later, Defendants spoke specifically about the market's focus on nivolumab, stating in the Company's February 14, 2014 Form 10-K for fiscal year 2013 (signed by Defendants Andreotti and Bancroft):

> As we are evolving to a specialty care biopharmaceutical company, we are focusing more of our efforts and resources in certain disease areas such as oncology, virology, immunology, and specialty cardiovascular disease. With our more focused portfolio, investors are placing heightened scrutiny on some of our late-stage compounds. ***In particular, nivolumab is an important asset in our immuno-oncology portfolio***. . . . The announcement of data from our clinical studies or news of any developments related to our late-stage immuno-oncology compounds, such as nivolumab, is likely to cause significant volatility in our stock price.

Defendants Bristol-Myers, Andreotti, Bancroft, and Caforio similarly stated in Bristol-Myers' February 13, 2015 Form 10-K for fiscal year 2014 that they were "focusing our efforts and resources in certain disease areas . . . . In particular, Opdivo[.]"

42. Investors focused particularly on the potential for Bristol-Myers to secure the first-mover advantage in PD-1 checkpoint inhibitors. The "first-mover advantage" or "first-to-market advantage" refers to the well-documented concept that being first to bring a product to a particular market provides distinct advantages by displacing competitors and capturing incremental market share. For example, a study published by McKinsey & Company in September 2014 reported that the market share of first entrants was an average of six percentage points higher ten years after the drug's launch. McKinsey & Company concluded that "the value of being first should not be overestimated, particularly when the lead time is short (less than two years) and a market is expected to be crowded with more than two players," as it was expected that the immunotherapy market would be. *See* Myoung Cha and Flora Yu, "Pharma's first-to-market advantage," McKinsey & Company (September 2014).

43. Bristol-Myers took an early lead in the race to bring PD-1 checkpoint inhibitors to market when, on July 4, 2014, Japanese regulatory authorities approved nivolumab for the treatment of certain patients with a skin cancer known as melanoma. Nivolumab became the first anti-PD-1 antibody approved anywhere in the world. Defendants immediately began marketing and manufacturing nivolumab under the name "Opdivo."

44. Just a week later, Bristol-Myers' immuno-oncology strategy appeared to accelerate. Defendants announced on July 10, 2014 that Bristol-Myers expected to complete in the third quarter of 2014 its application to the FDA for the approval of the use of nivolumab with certain melanoma patients.

45. However, Bristol-Myers' early lead was short-lived. In September 2014, the Company's primary competitors in the nascent industry for PD-1 checkpoint inhibitors—Merck—received an earlier-than-expected first FDA approval for a PD-1 checkpoint inhibitor, permitting the use of Merck's drug pembrolizumab in the treatment of certain patients with melanoma. Merck immediately began manufacturing and marketing pembrolizumab in the United States under the name "Keytruda." As *Forbes* noted on September 5, 2014, "The rapid [FDA] approval, which allowed Keytruda, also known as pembrolizumab, to leapfrog a similar drug from Bristol-Myers Squibb, is a triumph for research head Roger Perlmutter, who arrived at Merck 18 months ago and has significantly improved the company's record of getting drugs through the FDA."

46. Though Opdivo had received the first approval for a PD-1 checkpoint inhibitor in the world, that approval had been limited to Japan. Opdivo needed FDA approval to access the United States market, which was both far larger and also home to critical key opinion makers in the medical community, whose familiarity (or lack thereof) with Opdivo could make or break the sales potential of the medication. Accordingly, by the start of the Class Period, Defendants understood that Keytruda's FDA approval posed an enormous threat to Bristol-Myers' ability to secure control of the PD-1 checkpoint inhibitor market, both in the United States and globally.

47. Defendants immediately took steps to undercut Merck's advantage. On September 4, 2014—the very same day that Keytruda received its FDA approval for the treatment of melanoma—Bristol-Myers and Ono filed a patent infringement lawsuit against Merck in the District Court of the District of Delaware. The suit alleged that Keytruda infringed Bristol-Myers' Opdivo patent, and that Merck had developed Keytruda based on knowledge gained from Opdivo's family of patents. *See Bristol-Myers Squibb Co. et al. v. Merck & Co. Inc.*, No. 14-cv-1131 (D. Del.). The infringement suit makes clear that Bristol-Myers viewed itself as the true innovator,

alleging that "Merck started developing pembrolizumab ***after***" Bristol-Myers, and only with "knowledge of the family of the family of patents that includes the [Opdivo] patent for many years." The infringement suit concedes that Opdivo and Keytruda are functionally identical: both are "PD-1 antibodies [that] allow a patient's immune system to resume its ability to recognize, attack, and destroy cancer cells" by "binding to PD-1 and blocking the PD-1 checkpoint pathway." Indeed, by March 14, 2016, the *Wall Street Journal* would note that "doctors consider the drugs to be roughly comparable, and . . . showed similar efficacy and safety in similar patient groups from separate studies," and, in June 2017, even Bristol-Myers' own head of research and development would state to *Forbes* that "there probably are greater similarities between [Keytruda] and [Opdivo] than there are differences between these drugs[.]"

B. Investors Understood That The Efficacy Of PD-1 Checkpoint Inhibitors Is Related To The Level Of PD-L1 Expression In Cancer Cells.

48. Though PD-1 checkpoint inhibitors did not begin to receive regulatory approvals until 2014, research and development concerning those drugs—and the science underlying PD-1 checkpoint inhibitors generally—had been underway since the first discovery of PD-1 in 1992. By the start of the Class Period, research—funded primarily by a handful of companies, including most prominently Bristol-Myers—had established the industry understanding that certain factors played important roles in the development, testing, and presentation of PD-1 checkpoint inhibitors.

49. Chief among those factors was the level of PD-L1 present in a patient's cancer cells, referred to as "expression." As summarized in an article published in the November 2014 issue of *The American Journal of Hematology/Oncology*:

> ***[L]evels of PD-L1 expression have been shown to predict higher tumor grade and poor prognosis in multiple different malignancies***, likely because the PD-1/PD-L1 interaction suppresses [tumor fighting immune cells]. Indeed, some studies have suggested that PD-L1 expression by cancer cells provides them with an "immune shield" and protects them from effector T-cell cytotoxicity.

50. By the start of the Class Period, PD-L1 expression was understood to have a critical relationship with the efficacy of PD-1 checkpoint inhibitors such as Opdivo and Keytruda. As a result, the strength of the patient's PD-L1 expression often defined clinical trials concerning those drugs and ultimately could limit the number of patients who could benefit from those drugs in practice. As noted by the *New York Times* in a June 2012 profile of Bristol-Myers' early clinical results with Opdivo, "preliminary evidence [indicates] that PD-1 blockers will ***not work in people whose tumors do not make PD-L1***, as determined by studying a biopsy sample. That might allow the drug to be used only for patients most likely to benefit, researchers said."

51. This understanding continued throughout the Class Period. For example, in a June 2, 2015 article, *Reuters* referred to Bristol-Myers and Merck as the "[d]rugmakers . . . testing which patients will most benefit from new cancer treatments based on a protein found in their tumors." The article noted that Bristol-Myers' own data, published just days earlier, "showed that Opdivo was most helpful to lung cancer patients with ***the highest levels of PD-L1 in their tumors***."

52. Dr. Ronald Blum, whose expertise is discussed further herein at ¶¶116-120, states that, based on his own involvement in, and oversight of, clinical trials examining PD-1 checkpoint inhibitors—including Merck's clinical trials of Keytruda—by the start of the Class Period, it was widely understood by participants in the development of PD-1 inhibitors, that the strength of the PD-L1 expression was a critical variable in assessing the probability of successful response to PD-1 checkpoint inhibitors.

53. The Individual Defendants were well aware of the industry's understanding of the role of PD-L1 expression in the likely success of PD-1 checkpoint inhibitors. The Individual Defendants were the key executives at Bristol-Myers who, as discussed further herein, spoke regularly about the Company's immuno-oncology program—including specifically the Head of

Immuno-oncology (Defendant Giordano), the Head of Oncology Development and development lead for Opdivo (Defendant Namouni), the Chief Scientific Officer with direct experience in the Company's immuno-oncology portfolio (Defendant Cuss), and the former Chief Operating Officer and then Chief Executive Officer who was directly involved in the design of Checkmate-026 and other immuno-oncology studies (Defendant Caforio). Moreover, at all relevant times, as detailed herein, the Individual Defendants spoke regularly about Opdivo and the dozens of ongoing clinical trials involving Opdivo, at Bristol-Myers, and routinely represented themselves as experts in the field of PD-1 checkpoint inhibitors. Accordingly, the Individual Defendants knew (or were extremely reckless in not knowing) of the consensus that "strong" PD-L1 expression was inconsistent with a 5% PD-L1 expression level, for at least two reasons: (a) the most common marker for mere PD-L1 positivity was 5%, as established by Bristol-Myers itself; and (b) "strong" or "high" PD-L1 expression was commonly considered to be expressions greater than 50%, as established by published articles and clinical studies by the start of the Class Period.

1. The most common marker for mere PD-L1 positivity was 5%.

54. In assessing whether a patient expressed PD-L1 at all—referred to as "PD-L1 positivity"—many leading industry researchers reported that a level of 5% expression of PD-L1 in a patient's cancerous tumors was the bare minimum level of expression for a patient to be considered as PD-L1 "positive." For example, an article published on March 28, 2012 in the medical journal *Science Translation Medicine* cited studies dating back to 2006 and 2007 that reported that "***a 5% expression threshold***" for PD-L1 positivity was "in keeping with previous studies." Indeed, an article published on May 26, 2016 in the peer-reviewed industry journal *Drugs*, cited data going back as far as 2002 and concluded that, though "[t]he best cut-off percentage of scored cells to determine PD-L1 positivity . . . remains an unresolved question,"

"***[s]ince the first published data [in 2002], the threshold most often chosen is >5% expression***." Festino, L., Botti, G., Lorigan, P. et al., "Cancer Treatment with Anti-PD-1/PD-L1 Agents: Is PD-L1 Expression a Biomarker for Patient Selection?," *Drugs* (May 26, 2016).

55. The industry practice of using a 5% threshold as the bare minimum for PD-L1 positivity was specifically observed in studies of NSCLC—the cancer targeted in Checkmate-026—as shown by a chart presented in *Journal of Thoracic Oncology* in July 2015. This chart reveals that the majority of published findings concerning NSCLC and PD-L1 —five of which were studies conducted by Bristol-Myers of Opdivo (nivolumab)—consistently defined PD-L1 positivity as 5% or greater by the start of the Class Period:

TABLE 1. Summary of Published Findings for PD-L1 Immunohistochemistry in Therapeutic Trials

Drug	Biomarker Antibody	Rx Line	Definition of "Positive"[a] (%)	N Positive (%)	Positive Predictive Outcome	ORR % IHC pos. Cases	ORR % IHC neg. Cases	Ref.
Nivolumab	Dako 28-8	1st	≥5 in >100 cells	59	Yes	31[b]	10	7,8[f]
Nivolumab	Dako 28-8	≥2nd	≥5, ≥1	49, 56	No	15, 13	14, 17	9,10
Nivolumab + Ipilimumab	Dako 28-8	1st	≥5 in >100 cells	42	No	19	14	11
Nivolumab	Dako 28-8	≥2nd	≥5	33[c]	Yes	24	14	12[f]
Nivolumab	5H1[d]	≥2nd	≥5, also studied TIICs	67	Yes	No data for lung	No data for lung	13
Pembrolizumab	Dako 22C3	Any	"Strong" ≥50, "Weak" 1–49	25, 70	Yes, Yes	37, 17	9	14
Pembrolizumab	Dako 22C3	1st	≥50, ≥1	?	Yes	47, 26	?	15
MPDL3280A	Roche Ventana, SP142	≥2nd	≥10,[e] ≥5, ≥1 TIICs	13, 28, 56	Yes	83, 46, 31	18, 18, 20	16–18
MEDI-4736	Roche Ventana, SP263	≥2nd	Data not available	41	Yes	25	3	19,20

[a]Expression in tumor cells unless otherwise stated.
[b]The 31% figure is for all tumors. The ORR was 37% in nonsquamous tumors and 12% in squamous cases. In PDL-1 negative cases, ORR was 14% in nonsquamous tumors and 0% in squamous tumors.
[c]This study concerned squamous cell carcinomas only.
[d]These authors also used the anti-PD-1 monoclonal M3 in their immunohistochemical analysis.
[e]IHC score 3, ≥10% TIICs positive; IHC score 2–3, ≥5% TIICs positive; IHC score 1–2–3, ≥1% TIICs positive.
[f]ORR quoted are those actually presented, as opposed to those published in the abstract
IHC, immunohistochemistry; TIICs, tumor infiltrating immune cells; ORR, overall response rate (response evaluation criteria in solid tumors).

Keith M. Kerr et al., "Programmed Death-Ligand 1 Immunohistochemistry in Lung Cancer," *Journal of Thoracic Oncology*, 2015;10: 985-989, at 986 (July 2015).

56. As made apparent by the above *Journal* chart and the Company's own published study results, Bristol-Myers—one of two primary players at this time in the developing PD-1 checkpoint inhibitor industry—itself established the industry standard of 5% PD-L1 expression as the bare minimum for positivity.

57. For example, on July 11, 2013, before the start of the Class Period, the results of a Bristol-Myers'-funded study of Opdivo (nivolumab) that treated patients with the drug from December 2009 through February 2013, was published in the *New England Journal of Medicine*. This study confirmed that Bristol-Myers defined PD-L1 positivity as an expression level of 5% or greater: "A sample was defined as ***PD-L1–positive if at least 5%*** of the tumor cells exhibited membrane PD-L1 staining of any intensity in a section containing at least 100 cells that could be evaluated." *See* Jedd D. Wolchok, M.D., et al., "Nivolumab plus Ipilimumab in Advanced Melanoma," *N. Engl. J. Med.* 2013 (July 11, 2013). Bristol-Myers' use of 5% as the definition of mere positivity was repeated in Bristol-Myers' Checkmate-066 study, a clinical trial testing Opdivo (nivolumab) against melanoma in patients with no prior treatment. Checkmate-066 again defined PD-L1 positivity as a "tumor specimen ***with [at least] 5%"*** expression in the Company's September 20, 2012 Clinical Trial Protocol, and referred to any patient with less than 5% expression as "PD-L1 negative." Bristol-Myers' use of the 5% expression positivity marker was repeated to investors on November 16, 2014, when Bristol-Myers disclosed the results of Checkmate-066, stating that "***PD-L1 positivity was defined as at least 5% of tumor cells showing cell-surface PD-L1 staining***." The 5% expression positivity threshold was repeated in the study results in the *New England Journal of Medicine* published just five days before the start of the Class Period. *See* Caroline Robert, M.D., et al., "Nivolumab in Previously Untreated Melanoma without BRAF Mutation," N. Engl. J. Med. 2015 (Jan. 22, 2015) ("***PD-L1 positivity was defined as at least 5% of tumor cells*** showing cell-surface PD-L1 staining of any intensity in a section containing at least 100 tumor cells that could be evaluated.").

58. Given the fact that Bristol-Myers, itself, established the industry standard defining 5% PD-L1 expression as the marker for PD-L1 positivity, the Individual Defendants—directly

involved in the Company's trial design and promotion of Opdivo (discussed further herein, *e.g.* ¶¶86-90)—were well aware of industry consensus, and in fact frequently spoke of the standard 5% PD-L1 expression minimum. For example, on June 2, 2014, Defendants Giordano and Namouni presented the results of the clinical trials Checkmate-012 (studying nivolumab against NSCLC in patients with no history of chemotherapy, including as a monotherapy and in combination with other drugs) and Checkmate-003 (studying nivolumab as a monotherapy against NSCLC in patients with a history of chemotherapy). During this presentation, Namouni described the "durable" and "interesting" responses with specific reference to the 5% "cutoff that was used to [do] this analysis," specifically explaining that detecting "good expression" was critical because "PDL-1 is a very ubiquitous protein." Similarly, during the Bank of America Merrill Lynch Global Healthcare Conference on September 18, 2014, Bristol-Myers Senior Vice President John Elicker stated that Bristol-Myers was calibrating the "percent of patients with PDL1 expression in the lung cancer studies . . . we expect" "based on the data we have seen, generally speaking at a 5% cutoff." Likewise, on October 30, 2014, Defendants disclosed the results of Checkmate-063 (studying nivolumab as a monotherapy against NSCLC in patients who have progressed after at least two prior systemic treatments), which also used 5% expression of PD-L1 as the baseline threshold for PD-L1 positivity. In an article published in March 2015 in *Lancet Oncology*, the clinical investigators for Bristol-Myers's Checkmate-063 study specifically explained that they "***used a cutoff of 5% to define PD-L1 positivity***[] on the basis of preliminary findings" in Bristol-Myers's earlier studies. Similarly, on April 20, 2015, Defendants announced the results of Checkmate-069, which an article published shortly thereafter in the *New England Journal of Medicine* explained likewise "considered . . . PD-L1–positive ***if at least 5%*** of tumor cells showed cell-surface PD-L1 staining of any intensity in a section containing at least 100 tumor cells that could be evaluated."

59. Bristol-Myers' repeated and consistent use of 5% PD-L1 expression as the minimal level of positivity demonstrates clearly Defendants' understanding of (and participation) in the industry consensus that 5% PD-L1 expression indicated a minimally positive—or weak—indication of PD-L1 positivity.

60. The market was well aware that Bristol-Myers used a 5% expression as a baseline for PD-L1 positivity in the Company's own Opdivo studies. For example, in a January 23, 2014 analyst report titled "Bristol-Myers Squibb: Why Biomarkers Matter . . . & More Thoughts on BMY's IO Strategy," a Leerink analyst discussed the announcement of Checkmate-026, as well as a "review of biomarker data published to date." In this report, Leerink discussed in detail prior data from Bristol-Myers that clearly "defined" "PD-L1 expressing tumors" as those demonstrating just 5% expression. For example, Leerink published the following chart, demonstrating that Bristol-Myers, as well as Merck and competitor Roche, defined positive PD-L1 expression as equal to or greater than 5%:

Clinical Activity of PD1/PDL1 Agents in Lung Cancer by PDL1 Expression

Efficacy Comparison by PDL1 Status as of October 2013

	Anti-PD1				Anti-PDL1	
Lung	**Nivolumab**		**MK-3475**		**MPDL3280a**	
	n	*RR*	*n*	*RR*	*n*	*RR*
PDL1 (+)*	**31**	**16%**	**7**	**57%**	**13**	**46%**
PDL1 (-)*	32	13%	22	9%	40	15%
Unknown	66	20%				
All Patients	**129**	**17%**	**29**	**21%**	**53**	**23%**

** ≥ 5% cells PDL1 (+) - BMY looking at tumor cells (through retrospective analysis), Roche looking at TILs; PDL1 status for MK-3475 based on Youden Index-based cut point*

Notes: Roche PDL1 (-) population includes 7 patients with unknown PDL1 status

RRs reported in RECIST criteria; RRs include uPRs

Source: ESMO 2013, IASLC 2013, Leerink Partners

61. In May 2015, Bristol-Myers presented the results of additional Opdivo studies at the ASCO industry conference. As summarized by Leerink in a May 14, 2015 report on the Biopharma landscape, Bristol-Myers again defined mere PD-L1 positivity "as ***≥ 5% tumor membrane staining in ≥ 1 biopsy***."

2. The immuno-oncology field understood "strong" or "high" PD-L1 expression to be expressions greater than 50%.

62. With 5% expression the most common minimum to constitute PD-L1 expression at all, researchers and market observers typically reserved "strong" or "high" characterizations for PD-L1 expressions many times greater.

63. By the start of the Class Period, industry consensus for "strong" or "high" PD-L1 expression regularly referred to PD-L1 expression levels of at least 50%. Specifically, on April 6, 2014, Merck—one of Bristol-Myers' principal competitors in the nascent PD-1 checkpoint inhibitor industry—issued a press release related to pembrolizumab (soon to be marketed as Keytruda) and presented on "early findings from studies exploring the relationship between tumor PD-L1 expression and clinical outcomes following monotherapy treatment with [pembrolizumab], an investigational anti-PD-1 immunotherapy, in patients with advanced melanoma and advanced non-small cell lung cancer (NSCLC)." Merck concluded that "the preliminary analysis suggests that the optimal cut-point is ≥50 percent of tumor cells" and that "[w]hen using this [50%] measurement, approximately 25 percent of advanced NSCLC patients had tumors that ***strongly expressed PD-L1***."

64. Similarly, in the materials for Merck's June 2, 2014 presentation at annual industry convention ASCO 2014, Merck explicitly defined "strong" PD-L1 as ≥50%, and defined "weak" PD-L1 as 1-49%. During that presentation, Dr. Tanguy Seiwert, Associate Professor of Medicine

for the University of Chicago, described PD-L1 expressions greater than 50% as "strongly and diffusively positive."

65. The industry quickly took note of the definition for "strong" PD-L1 expression adopted by one of the key companies studying PD-1 checkpoint inhibitors, and in turn adopted this definition itself. For example, the European Society for Medical Oncology's 2014 Congress Meeting Report described Merck's findings as concerning "patients with strong PD-L1 expression (≥50% staining) [versus] in patients with weak/negative PD-L1 expression" and "patients with PD-L1 strong-positive vs. PD-L1 weak-positive/negative tumours."

66. The understanding that 5% PD-L1 expression was weak, or minimal positivity of PD-L1 expression, and that 50% PD-L1 expression was "strong" PD-L1 expression, continued during the Class Period. For example, an article published in the peer-reviewed scientific journal *PLOS One* on March 16, 2015 noted that, while studies had defined PD-L1 positivity as either 1% or 5%, "strong" expression was at least 50%, stating: "[i]n NSCLC immune and tumor cells . . . ***a strong expression (staining ≥50%)*** derived greater clinical benefit than patients with weak or negative score PD-L1 expression."

67. Likewise, in July 2015, the industry publication *Journal of Thoracic Oncology* published an article describing PD-L1 expressions of "1% ***or even 5%***" as "***very low staining thresholds***" that pose "a greater risk that scoring will be inconsistent and is more likely to reflect inaccurately the patient's tumor burden overall," Keith M. Kerr et al., "Programmed Death-Ligand 1 Immunohistochemistry in Lung Cancer," *Journal of Thoracic Oncology*, 2015;10: 985-989, at 986 (July 2015). The article also explicitly referred to PD-L1 expressions of 1-49% as "weak," and above 50% as "strong."

68. Similarly, a scholarly article published in *Translational Oncology* on March 3, 2016 titled "PD-L1 Expression and Survival Among Patients With Advanced Non-Small Cell Lung Cancer Treated With Chemotherapy" determined that:

> Using the clinical trial assay to identify levels of PD-L1 expression that maximally predict clinical response to pembrolizumab, ***PD-L1 weak positive*** is defined as membranous PD-L1 expression in 1% to 49% of tumor cells, and ***PD-L1 strong positive*** is defined as expression in ≥ 50% of tumor cells.

69. Other pharmaceutical companies in the industry also adopted Merck's definition of "strong" PD-L1 expression. For example, in a presentation on March 25, 2015, the Vice President of Pre-Clinical Research at Peregrine Pharmaceuticals gave a presentation entitled "Expansion and Activation of T-cells via the Targeting of the Immunosuppressive Ligand Phosphatidylserine: Combination Strategy with Other Checkpoint Inhibitors," which referred to both Opdivo and Keytruda and also incorporated Merck's conception of "High" and "Low" PD-L1 tumor expression in NSCLC. Likewise, in a presentation on October 8, 2015, competitor Nektar spoke about Keytruda's application for the "subset of ***high PD-L1 expression of more than 50%.***"

70. Given the widespread industry usage of "strong" to denote 50% PD-L1 expression, Bristol-Myers' use of that same descriptor was clearly—and reasonably—going to be understood in the same manner. The Individual Defendants were aware of the convention of characterizing 50% PD-L1 expression as "strong" or "high," and indeed had focused on the immuno-oncology industry's (and specifically Merck's) characterization of "strong" PD-L1 expression to be equal to 50% or greater expression.

71. For example, Bristol-Myers' Senior Vice President for Global Commercialization for Oncology until early 2014, when the Checkmate-026 trial was announced (FE-1[1]), discussed

[1] This complaint refers to former employees with the shorthand of "FE-." Former employees' roles are delineated when introduced and in Appendix A.

the levels of PD-L1 expression employed by Bristol-Myers and Merck with both Defendants Giordano and Namouni, who were each involved in the design on Checkmate-026. FE-1 worked at Bristol-Myers since 1989 and, in early 2014, FE-1 co-chaired Bristol-Myers' Disease Strategy Committee meetings with Giordano, the Head of Development for Oncology and Immuno-oncology. According to FE-1, during this time FE-1 participated in discussions with Giordano and Namouni (who was at the time head of the Opdivo program) about what level of PD-L1 expression was "good" commercially and about Merck's use of a 50% PD-L1 expression level.

72. In addition, FE-2, a former Associate Director of Global Oncology Forecasting & Strategic Planning, worked at Bristol-Myers from January 2010 through March 2013 and was directly involved in the commercial development of Checkmate-026 before the study was announced in January 2014 as the Company's lead global forecaster for Opdivo. FE-2 explained that Bristol-Myers maintained competitive intelligence teams to gather information on the trials performed by the Company's competitors, including Merck. FE-2 described how the Company would typically run 40-100 scenarios using that data to inform Bristol-Myers' executive team about what the competitors were doing, what Bristol-Myers was poised to do, and how those two situations would interact under various conditions. FE-2 specifically stated that Bristol-Myers would have immediately incorporated any information learned about the 50% "strong" PD-L1 threshold used by Merck into new forecasting to understand Merck's position in the market and to compare to Bristol-Myers's efforts in Checkmate-026, and that this crucial information and resulting forecast updates would have gone to Defendant Namouni.

C. Defendants Mislead Investors By Defining Checkmate-026's Primary Focus As Patients That "Strongly" Expressed PD-L1.

73. At the start of the Class Period, the market had great expectations for PD-1 checkpoint inhibitors. Just before the start of the Class Period, in a January 8, 2015 report, a

Leerink analyst estimated that immuno-oncology pharmaceuticals represented a $40 billion-plus market opportunity, noting that "the breadth, durability, and tolerability of PD1/PDL1 antibodies demonstrated over the last three years should establish this class as the backbone of a new pillar of cancer care, immuno-oncology." By the end of 2015, in a December 1, 2015 article titled "Bristol-Myers: Big Spending, Big Reward," the *Wall Street Journal* attributed "Bristol's lofty valuation" in large part to the "commercial potential" for Bristol-Myers' PD-1 checkpoint inhibitor, with analysts projecting $6.8 billion sales by 2019—"one-third of that year's projected total revenue."

74. However, having lost the first-mover advantage in the treatment of melanoma, and with its drug considered functionally equivalent to its closest competitor's, Bristol-Myers' ability to wrestle control of the PD-1 checkpoint inhibitor market relied on its approach in designing its clinical trials, and whether it could secure better—broader, quicker—approvals than the competition. Importantly, the most promising approval necessary to take control of the PD-1 checkpoint inhibitor market still remained up for grabs: the use of PD-1 checkpoint inhibitors as a monotherapy to treat NSCLC.

75. NSCLC is the most common type of lung cancer, accounting for 85% to 90% of all lung cancer diagnoses. The National Cancer Institute estimates 228,150 Americans will be diagnosed with lung cancer this year, and more than 80% will not survive, making it one of the most important commercial markets for immuno-oncology. A successful new treatment for this disease stood to generate billions of dollars worldwide for the market leader. Just two weeks before the start of the Class Period, *Reuters* wrote "[s]ome industry analysts expect . . . PD-1 or PD-L1 inhibitors, to generate more than $30 billion in worldwide annual sales by 2025, ***with lung cancer seen as the most lucrative use***."

76. Bristol-Myers and Merck engaged in numerous clinical studies of their respective PD-1 checkpoint inhibitors, eager to secure dominance in this sought-after field. By the start of the Class Period, Bristol-Myers had concluded at least one dozen (12) Opdivo "Checkmate" studies, and was actively engaged in an additional twenty-one (21) Opdivo "Checkmate" studies. Similarly, Merck reported at least twenty-two (22) Keytruda "Keynote" studies, both completed and ongoing, at the start of the Class Period. As industry website *FiercePharma* wrote on December 23, 2014, Opdivo and Keytruda were "among the hottest tickets in oncology these days" and Bristol-Myers was "arm[ed] for PD-1 battle" and "doing its best to keep Merck's competitor [Keytruda] in check."

77. In January 2014, Bristol-Myers announced a new clinical trial—Checkmate-026—that sought, in simple terms, to prove that Opdivo would perform better than chemotherapy as a first-line monotherapy (a treatment used on patients who had not previously received treatment and without combination with other drugs) in stopping the progression of NSCLC. Specifically, Defendant Cuss stated during the Company's January 24, 2014 earnings conference call that "as many of you have seen, we recently posted on *clin[ical]trials.gov* a first-line Phase III trial with nivolumab monotherapy in lung cancer that we will start soon." Not much else was said publicly about Checkmate-026 over the following year, as the Company was still recruiting candidates for the study.

78. Industry analysts and investors, as well as (as confirmed by Plaintiffs' expert Dr. Blum) clinicians and clinical investigators, all rely on *ClinicalTrials.gov* as an unparalleled means to analyze relevant, detailed information about a study's focus and goals. *ClinicalTrials.gov* is a registry and results data bank operated by the National Library of Medicine of the National Institute of Health, and is the primary tool used by pharmaceutical companies to communicate

material details concerning ongoing clinical trials to the market. Analysts and investors relied on this information because drug companies, and Bristol-Myers specifically, are statutorily required to post truthful and accurate information to *ClinicalTrials.gov*. (*See generally* 42 C.F.R. § 11 (among other things, requiring disclosure of "[d]escription of the metric used to characterize the specific primary outcome measure" that "shall not be false or misleading in any particular").) During the Class Period, Defendants explicitly acknowledged that they controlled the information disclosed on *Clinicaltrials.gov* concerning Checkmate-026. For example, on September 8, 2015, Defendant Giordano referred to information on *Clinicaltrials.gov* by stating that "Of course, we communicate with the FDA anything ***we might disclose in Clinicaltrials.gov***." As prompted by Defendants, in fact the market did rely on *ClinicalTrials.gov* with respect to Checkmate-026.

79. In its initial *ClinicalTrials.gov* submission on January 19, 2014, the Company's Study Description stated that "the purpose of this study is to show that Nivolumab will improve progression free survival ***in subjects with strongly Stage IV or Recurrent PD-L1+ non-small cell lung cancer*** when compared to chemotherapy." While a subject who merely expressed any PD-L1 positivity—which had historically been most commonly defined in numerous other studies as 5% expression—was eligible for inclusion, the Company stated that Checkmate-026's "Primary Outcome Measure'' (defined as the "most important" measure of success) was "***progression free survival (PFS) . . . in subjects with strongly Programmed death-ligand 1+ (PD-L1+) tumor expression***." Two of the four "Secondary Outcome Measures" for Checkmate-026 were also focused solely on those with strong PD-L1 expression: the "[o]bjective response rate (ORR) . . . in subjects with ***strongly PD-L1+ tumor expression***" and "[o]verall survival (OS) in subjects with ***strongly PD-L1+ tumor expression***." Only the last two (of four) Secondary Outcome Measures were directed at "subjects with any PD-L1+ tumor expression."

80. Based on Dr. Blum's own extensive experience, including his involvement in, and oversight of, clinical trials examining PD-1 checkpoint inhibitors (such as Merck's clinical trials of Keytruda) (¶¶116-120), Dr. Blum states that industry practice required Bristol-Myers to accurately disclose the PD-L1 expression cutoff targeted by Checkmate-026 because it was understood by this time that the strength of the PD-L1 expression was a critical variable in assessing the probability of successful response to PD-1 checkpoint inhibitors, and Bristol-Myers' failure to reveal its cutoff while repeatedly describing it as "strong" was highly unusual and misleading.

81. The market understood that Checkmate-026's focus on "strong" expressors reflected the consensus understanding that the level of PD-L1 expression was a critical variable in assessing the probability of Opdivo's efficacy, and thus that Checkmate-026's likelihood of success was related to the PD-L1 expression cutoff. Indeed, during a Bristol-Myers investor presentation on October 31, 2014, Defendants Giordano and Namouni directly explained that, in Checkmate-026, "we're not dismissing any scientific clinical evidence that helps us to get to patients quickly," and that while Defendants were looking to "patients across the continuum [of] PD-L1 expression . . . if speed can be achieved with a ***more select population***, we will certainly not hesitate to take that approach."

82. Defendants and analysts focused heavily on Checkmate-026 and its potential beginning in 2015, when the recruitment of patients for the study was nearly complete. On the first day of the Class Period, January 27, 2015, Bristol-Myers updated the Checkmate-026 data on *ClinicalTrials.gov*, but made no change the Checkmate-026's Primary Measure Outcome or Secondary Measure Outcomes. Defendants also discussed Checkmate-026 during the Company's fourth quarter 2014 earnings call on that same date. By this time, Bristol-Myers had secured its

first FDA approval for Opdivo in the treatment of melanoma, had initiated its patent infringement suit against Merck, and was racing to win the first-mover advantage in the treatment of NSCLC.

83. Moreover, as discussed above, by this time, numerous clinical trials and research papers had been published firmly setting the industry understanding for what was considered "strong" (or "high") PD-L1 expression positivity (*i.e.*, 50%) and what was considered minimal or weak PD-L1 expression positivity (1% - 5%). As described more fully above, this includes both the industrywide consensus that 5% PD-L1 expression constituted the bare minimum for positivity—which Defendants themselves helped define—as well as Merck's prominent adoption of the industry's description of "strong" PD-L1 expression as referring to 50% or greater expression. (¶¶54-72.)

84. Perhaps the single most important development between the initial announcement of Checkmate-026 in January 2014 and the start of the Class Period on January 27, 2015, however, was Merck's announcement of a parallel study vying for the approval of Keytruda as a first-line monotherapy alternative to chemotherapy for patients.

85. In May 2014, Merck published a study titled "Study of MK-3475 Compared to Platinum-Based Chemotherapies in Participants With Metastatic Non-Small Cell Lung Cancer (KEYNOTE-024)."[2] Keynote-024's Study Description paralleled that of Checkmate-026: "This is a study to assess the efficacy and safety of pembrolizumab (MK-3475 / SCH 900475) [Keytruda] compared to standard of care (SOC) platinum-based chemotherapies in the treatment of participants with previously untreated stage IV, ***programmed cell death ligand 1 (PD-L1) strong expressing Non-Small Cell Lung Cancer (NSCLC)***." Checkmate-026 made its study eligible to all patients with positive PD-L1 expression and focused its study on only those with "***strongly PD-***

[2] MK-3475 was Merck's earliest name for pembrolizumab/Keytruda.

L1+ tumor expression." Merck similarly limited Keynote-024's eligibility to patients with "PD-L1 strong expressing tumor"—but while Merck's "strong" matched industry expectations of 50% or greater, investors were unaware that Bristol-Meyers' "strong" misleadingly referred to PD-L1 expression of as little as 5%, or mere positive expression. As evidenced by the opinion of Dr. Blum and numerous industry sources discussed herein, Bristol-Myers' description of its 5% expression cutoff was inconsistent with "strong" PD-L1 expression.

86. Former employees confirm that the Individual Defendants were not only aware of the concealed 5% PD-L1 expression level used in Checkmate-026, but personally participated in and/or approved the decision to focus Checkmate-026 on patients expressing just 5% PD-L1 expression.

87. FE-2, discussed above, was a former Associate Director, Global Oncology Forecasting & Strategic Planning, who worked at Bristol-Myers from January 2010 through March 2013 and was directly involved in the development of Checkmate-026 as the Company's lead global forecaster for Opdivo. FE-2 attended several Checkmate-026 strategy meetings along with Defendants Caforio and Namouni, and stated that both Caforio and Namouni were required to approve the PD-L1 expression threshold used in Checkmate-026. FE-2 explained that, as Checkmate-026's protocol was refined, FE-2 spent a lot of time with Caforio discussing strategic decisions about the value of Opdivo and how to position it. FE-2 explained that there was uncertainty around the size of the patient pool and the level of PD-L1 expression that would respond meaningfully, and that the Company was focused on accelerating the process to avoid missing that segment of the market due to competition. FE-2 noted that Caforio would "keep pushing and pushing to drive the high case," meaning the maximum forecasted patient pool and lowest expression level.

88. FE-3—the Company's former Head of Product Portfolio and Access Strategy for Oncology when FE-3 left Bristol-Myers in October 2016, and Vice President, Head of Access Strategy for Immuno-oncology from 2013-2015—explained that due to Opdivo's importance to Bristol-Myers, senior management engaged in a constant dialogue with senior management, including those in the C-suite level, about any developments concerning Opdivo. Specifically, FE-3 (who worked at Bristol-Myers for 33 years) stated that it was internally known that Checkmate-026 was looking at 5% PD-L1 expression, with discussions about that threshold going "all the way up" to Defendants Cuss, Namouni, Giordano, and Caforio. FE-3 added that a development team consisting of representatives from Bristol-Myers' Regulatory, Clinical, and Commercial groups were in charge of the development of Checkmate-026, and reported to Defendants Cuss and Giordano, who would have reviewed and approved Checkmate-026's strategy. FE-3 also explained that Cuss and Giordano were also members of Bristol-Myers' Brand Development Operating Committee, which was responsible for endorsing the Company's approach to lung cancer, including high-level study design (i.e., why a particular strategy was chosen), prior to execution.

89. Bristol-Myers' former Vice President and Head, Clinical Pharmacology and Pharmacometrics, FE-4, who worked at the Company from October 2004 through June 2017, confirmed that Checkmate-026 was a "critical study" for Bristol-Myers, and learned from colleagues that Checkmate-026 had to be approved at the highest level of the Company.

90. FE-5, a former Group Medical Director, Immuno-oncology, worked at the Company from August 2012 through January 2016 and oversaw a group of physicians on project teams involved in establishing the safety measures for various clinical trials, including Checkmate-026. FE-5 stated that each of Bristol-Myers' senior executives would have known the cutoffs for

the Opdivo trials because PD-L1 expression is "one of the most important parameters in patient selection." Moreover, FE-5 confirmed that Defendant Giordano was directly involved in the decision to have a 5% PD-L1 expression cutoff in Checkmate-026, as he was in charge of Bristol-Myers' clinical strategy and ultimately gave approval for the protocol and design structure. FE-5 also stated that Defendant Cuss's approval of Checkmate-026's strategy was required.

91. Merck's entry into the race for approval of a first-line monotherapy immuno-oncology treatment for NSCLC heightened the scrutiny on Checkmate-026, and increased the pressure for both companies to win the first-mover advantage.

92. Defendants' January 27, 2015 reaffirmation on *ClinicalTrials.gov* that Checkmate-026 was focused on those subjects with "strong" PD-L1 expression communicated at least two material facts to investors: (a) the likely FDA approval to generate from the study would be confined to the roughly 25% of NSCLC patients with strong 50% PD-L1 expression, but because of this focus; and (b) the study had a strong chance of success. Moreover, the fact that Checkmate-026 could possibly have secondary outcomes that proved Opdivo's efficacy in treating even those with weaker PD-L1 positivity made the study potentially more valuable that Keynote-024.

93. Unlike Merck, Defendants steadfastly refused to reveal the exact percentage employed to determine subjects with "***strongly PD-L1+ tumor expression***." On January 24, 2014, an analyst asked whether Defendants could "tell us what percent of non-small cell patients are high expressors of PDL lung using your cutoff criteria? If I look at the Merck and Roche data, it suggests maybe it's 25% [of the patients]"—meaning, the expression cutoff was 50% as typically understood. In response, Defendant Cuss refused to reveal the percent of strong PD-L1 expressors that were used in the Primary Outcome Measure of the study, stating "***I'm not going to comment***

on our particular percentages." Cuss did, however, acknowledge and confirm the importance of the strong PD-L1 expression cutoff to the study. Specifically, Defendant Cuss noted that "***directional data [] shows that PD-L1 expression tends to be related to higher response rates***," and described Checkmate-026 as a "biomarker rich" study.

94. Defendants' refusal to be truthful and disclose Checkmate-026's weak 5% cutoff criteria, and reaffirmation of the purported "strength" of the cutoff, led analysts to understand, based on past studies and research, that the industry consensus was that "strong" expression of PD-L1 was likely close to, if not equal to, 50%, and by no means was as low as 5%. For example, on March 7, 2016, analysts from Cowen issued a report titled "Statistical Analysis of IO First Line Lung Studies" that focused heavily on the odds of success of Keynote-024 and Checkmate-026. Cowen "assum[ed] primary analysis on PD-L1 enriched population (expression >50%)."

95. The market's impression continued to be affirmed as Defendants regularly announced the results of Bristol-Myers' other studies. For example, on April 20, 2015, Defendants announced the results of Checkmate-069, which also used a 5% cutoff for mere positivity. Likewise, on May 31, 2015, Defendants announced the results of Checkmate-017, which considered "positivity . . . [a]cross prespecified expression levels (1%, 5%, and 10%)."

96. Developments related to Keynote-024 by mid-2015 further solidified the market's conclusion that 50% was the most likely "strong" expression cutoff employed by Bristol-Myers in Checkmate-026. Though the Keynote-024 study information posted by Merck on *ClinicalTrials.gov*, like the Checkmate-026 information, did not specify the percentage of PD-L1 expression Merck defined as "strong," since well before the Class Period Merck had expressly defined 50% or greater PD-L1 expression as "strong" expression. (¶63.) In any event, by June 1, 2015, Merck implicitly confirmed that Keynote-024 defined "strong" expression to include only

those subjects whose cancer cells expressed PD-L1 at least a 50% level. On that date, Merck provided an investor presentation in connection with ASCO 2015, in which one of the slides stated that Merck's "NSCLC File Accepted by FDA," noting ranges of ≥ 50%, 1-49%, and less than 1%. The market immediately understood that this range applied to Keynote-024. For example, in its report the next day analyzing the impact of developments at ASCO on Bristol-Myers, Barclays noted Keynote-024's expression cutoff was greater than 50%. Ultimately, by February 2016, Merck's President Roger Perlmutter openly acknowledged that—consistent with industry consensus that "strong" PD-L1 expression referred to 50% or greater—"[Keytruda] 024 . . . ***looks at those that are PD-L1 high . . . those with the proportion of scores above 50%***."

97. Given the overlap and parallels between Checkmate-026 and Keynote-024, and the fact that both studies described their focus in identical terms of subjects with "strongly" expressing tumors, the market reasonably understood that the two studies were effectively testing for similar results. As Dr. Blum explains, because it was understood by the start of the Class Period that the strength of the PD-L1 expression was a critical variable in assessing the probability of successful response to PD-1 checkpoint inhibitors, industry convention required Bristol-Myers to disclose the PD-L1 expression cutoff targeted by Checkmate-026. Given that Defendants did not specifically provide this critical information, Dr. Blum explained that it would have been understood that Defendants had nonetheless followed the industry convention through their use of the exact same terms (e.g. "strong" and "high") used by Merck, as had already been defined by Merck and adopted by the industry. In light of the industry's historical convention, Dr. Blum described it as disingenuous for Defendants to secretly use a 5% threshold, which was inconsistent with "strong" PD-L1 expression.

98. Defendants further reinforced the market's false understanding that Bristol-Myers and Merck had similar (if not identical) approaches to PD-L1 expression by directly telling investors that they were working to harmonize the approach to measuring PD-L1. Specifically, both Bristol-Myers and Merck collaborated with the same company, Dako, to develop the diagnostic assays that each company used to evaluate PD-L1 expression for their respective studies. At ASCO 2015 on June 1, 2015—incidentally, the same event and the same day that Merck implicitly confirmed that Keynote-24's "strong" expression referred to PD-L1 expression levels of 50% or greater, consistent with industry understanding—an analyst asked Defendant Giordano about the discussion around "trying to harmonize how [PD-L1] is done across companies" and "how you're thinking about that approach and ***working with the other sponsors of these PD-L1 assets [assays]*** [sic] to have something that's more useful for clinicians?" In response, Defendant Giordano stated that "there are ongoing approaches to harmonizing the assays, and ***Bristol-Myers Squibb, as a leader in the field and committed to the patient's benefit, will participate in the efforts to harmonize PD-L1 expression assays to enable helping the most patients***." In an article published the next day, June 2, 2015, *Reuters* wrote, "Drugmakers including Bristol-Myers Squibb Co and Merck & Co are testing which patients will most benefit from new cancer treatments based on a protein found in their tumors [PD-L1]. . . . Drug companies are working to refine and ***standardize their PD-L1 testing***."

1. For most of the Class Period, Defendants conceal that Checkmate-026 is focused on subjects with less than 50% PD-L1 expression.

99. Throughout the Class Period, in response to investors' repeated questions, Defendants misleadingly emphasized Defendants' consideration of—and confidence in—the role of PD-L1 expression in Checkmate-026. For example, Defendant Cuss, Bristol-Myers' Chief

Scientific Officer, made statements during a July 23, 2015 earnings call that described Checkmate-026 as part of Defendants' "very important" "first-line lung strategy" and providing "***the quickest way to bring Opdivo to first-line patients***." Months later, on September 8, 2015, Defendants Giordano and Namouni spoke at length about their "lead study," Checkmate-026, at the World Conference on Lung Cancer ("WCLC"), during which they explicitly referred the public back to information Defendants had disclosed on *Clinicaltrials.gov*:

> [Defendant Giordano]: Importantly, we have recapitulated the role of PD-L1 expression in non-small cell lung cancer and have identified that the magnitude of benefit in the PD-L1 expressors confirms our first-line monotherapy approach to study the PD-L1 expressors in study 026. ***[Checkmate-026 is] our lead study***, the first study that will read out as a Phase III in non-small cell lung cancer first-line setting. ***We have great confidence that approach will give us an early read of the role of PD-L1 expressing patients in the first-line setting***. . . .
>
> As a reminder, the 026 study design [—]***for which we had great confidence***[—]***the study has been designed appropriately*** to read out significantly in the next several months in which we're studying the PD-L1 positive population with end points of PFS. . . .
>
> [Checkmate] 026 is the first study to read out for patients in the first-line study – first-line setting. ***We've designed that study in order to get speed to market and be able to treat patients in the first-line setting***. Selection of PD-L1 positives turned out to be a very wise chose based upon the magnitude of benefit that we're expecting to see in that patient population in that study design. . . .
>
> [Defendant Namouni]: . . . [F]or the 026 timeline as a question, obviously this is a study that is ongoing. We completed the recruitment recently for -026. ***It is a PFS as main end point, progression free survival as main end point.*** We will follow the events. When the events will be ready, we will run the analysis. ***We will not have any additional information to share at this point on -026***. . . .
>
> [Defendant Giordano]: ***We've indicated in Clinicaltrials.gov, that the 026 study will*** mature ***– will have its end point in August.*** Of course, we communicate with the FDA anything ***we might disclose in Clinicaltrials.gov***.

100. The following day, Defendants Caforio, Bancroft, Giordano, and Namouni, as well as Bristol-Myers' Head of Research Carl DeCicco, met with analysts from Evercore ISI. In Evercore's September 10, 2015 report describing the meeting, its analyst wrote that Defendants

"believe[] the data for Opdivo in PDL1+ tumors is strong, ***has confidence in the design of -026*** and is ***more than optimistic about the results***."

101. On October 1, 2015, Defendant Giordano spoke at length about Checkmate-026 at the Leerink Partners Immuno-Oncology Roundtable. Giordano was asked specifically to discuss Checkmate-026 and, specifically, the Company's "conviction and confidence with regard to your first-line strategy with monotherapy?" Giordano responded that "***we are very confident that that study will be a positive study***; in particular the characterization of the survival. We have chosen a PFS endpoint because of potential cross-over, which increasingly will be a challenge in the future. ***We are tracking very carefully data on PFS and OS and how those track with regard to Opdivo on the longer term.*** Our team, I think, has a very good sense of how to model what we might expect to see in PFS and OS and use that on a regular basis to determine the timing of the analysis and any adjustments to the number of events we need to see."

102. In 2016, investors focused even more acutely on Checkmate-026. Throughout 2015, as Bristol-Myers secured approvals for use of Opdivo as a combination therapy or in other cancers such as melanoma, Opdivo became a major driver in Bristol-Myers' financial performance—for example, on October 27, 2015, industry observer *ThePharmaLetter* wrote that "Opdivo drives Bristol-Myers Squibb success." However, as *Reuters* wrote in a February 26, 2016 article, while Bristol-Myers had "pulled ahead in the race to dominate the hot new cancer immunotherapy market . . . much is still to play for in a market that has grabbed the attention of investors and which some analysts believe could eventually be worth more than $40 billion a year." *Reuters* noted that "[d]evelopments this year and next will be crucial in determining if Bristol's rivals can catch up"—namely in "first-line lung cancer."

103. In response to the heightened interest, Defendants continued to express confidence in Checkmate-026—including in their consideration of the role of PD-L1 expression in the clinical trial. For example, during Bristol-Myers' January 28, 2016 earnings call, Defendant Cuss explicitly stated the Company's focus on PD-L1 expression, which was based on "both published and our internal data" in taking "great care" in designing the study (rather than disclose their aggressive gamble underway):

> [A]s far as 026 is concerned, ***we have actually taken great care in the design of this study. We have paid real attention to the choice of end point***, the optimal timing of the analysis, the role of the non-proportional hazard ratio, and ***of course, the role of PD-L1 expression and the sample size.*** And we've used a wealth of data, both published and our internal data, on Opdivo to help guide us.

104. By early 2016, some analysts publicly emphasized and relied upon their reasonable assumption that Bristol-Myers was employing the same 50% cutoff for strong expressors in Checkmate-026 as Merck employed in Keynote-024.

105. On March 7, 2016, Cowen issued a report analyzing the potential success of several ongoing first-line lung cancer clinical trials, which focused heavily on Checkmate-026 and Keynote-024. While the analysts noted that Checkmate-026's "cut-off for strong positivity has not been disclosed," Cowen "***believe[d] a cut-off of ≥50% is likely***, given that ***a high threshold gives the study a higher chance of success***." In support of this conclusion, Cowen ***expressly stated*** that Bristol-Myers had classified "strongly PD-L1+ patients" as those expressing greater than 50% PD-L1 based on "Company data," including Defendants' Checkmate -012 clinical trial.

106. During Cowen's Health Care Conference later that same day, Defendant Namouni was specifically asked to correct any statements made by Cowen in its March 7, 2016 report—including, as just noted, Cowen's explicit statement that Bristol-Myers had classified "strong" PD-L1 expression as 50% or greater. Defendant Namouni did not correct any statements. Specifically, Cowen's analyst stated:

> ***CheckMate-026 is obviously on everyone's mind. Bristol seems very confident in the outcome.*** You always say no more about the trial than we do, but can you help us think about it? What are the key things we should be thinking about with -026 as we think about possible scenarios for the outcome? ***And feel free to correct anything in the report that was issued this morning, if anything, that needs to be corrected***.

In response, Namouni confirmed that Checkmate-026 "***was well-designed***" and "represents all the knowledge in terms of design that was really made at Bristol-Myers Squibb from our understanding, from a number of positive Phase 3s looking at different endpoints." Specifically, Namouni confirmed that Checkmate-026 looked at high—or strong—expressors, as well as low expressors: "***[T]he study has been well-designed and well-powered*** to detect the difference in all the positives but also ***in what we define [as] the high positives***." Despite being explicitly put on notice that the analyst believed the expression cutoff was 50%, Namouni ***never*** conveyed that the "high positives" were not 50% or anything close to 50%.

107. Further, Defendant Namouni's statement that Checkmate-026 was "well-powered" was also false. A clinical trial's "statistical power" is a technical term that indicates the probability that a clinical trial will have a statistically significant positive result, and that the samples studied in a clinical trial had been appropriately selected to prevent results unrelated to the treatment being tested. By describing Checkmate-026 as "***well-powered*** to detect the difference in all the positives but also ***in what we define [as] the high positives***," Namouni communicated that Checkmate-026 was designed to provide meaningful data concerning different levels of PD-L1 expression. In reality, Namouni and the other Defendants knew that Checkmate-026 was not "well-powered," and was not designed to provide any statistically sound data concerning "high positive" PD-L1 expression.

2. Defendants eventually concede that the Checkmate-026 cutoff for "strong" expression may be less than 50%, but still refuse to provide any details to investors.

108. By spring 2016, some analysts began to consider whether Checkmate-026's cutoff was something less than 50%, but still assumed that the Company's use of "strong" expression had to equate to a cutoff of 25%, at a minimum. For example, on March 7, 2016, Leerink issued an analyst report titled "The Battle for PD1 Monotherapy Supremacy in 1L NSCLC: BMY vs MRK." In this report, Leerink stated that while Bristol-Myers in Checkmate-026 was "evaluating the 70% of pts with PDL1 expression >1%," "BMY's primary analysis will first be in PDL1 'high' expressors, followed by an evaluation of the overall PDL1+ group – giving BMY two "bites at the apple" to succeed."

109. Leerink's report continued with an in-depth analysis that attempted to calculate the odds of Checkmate-026's success. Leerink concluded that "In the 20-35% of patients with high levels of PDL1 expression (***the primary analysis population for both [Bristol-Myers and Merck] studies***), we expect both KN-024 and CM-026 to succeed, leading to regulatory approvals of Keytruda & Opdivo in pts expressing high levels of PDL1." Leerink explained that while Merck's PD-L1 expression for Keynote-024 was 50%, representing approximately 20%-25% of first-line lung cancer patients, "BMY's PDL1 'high' likely represents 30-40% of 1L lung cancer patients (broader label)," a figure that translated to at least 25% PD-L1 expression.

110. Therefore, by March 2016, while Bristol-Myers and the Individual Defendants refused to explicitly state what cutoff the Company employed to define strong PD-L1 expression in Checkmate-026, the market reasonably understood that cutoff to be somewhere between 25% - 50% expression, representing 35% to 25% of the patient population, respectively.

111. Nonetheless, Defendants continued to mislead investors as to the PD-L1 strong expression cutoff and the risks to success for Checkmate-026 through August 5, 2016.

112. On April 28, 2016, Defendant Cuss for the first time acknowledged what the Leerink analysts had concluded on March 7, 2016 and acknowledged that their "actual level of what strongly expressing PD-1 is [is] lower than 50%." However, Cuss emphasized that "***we are very comfortable about the design of our trial. We are very comfortable about the depth of understanding that went into to the statistical plans***." The "statistical plan" that Cuss referred to was likely the statistical action plan for Checkmate-026, an element of the clinical trial design that required advance approval by the study's data safety monitoring committee. Similar to Defendant Namouni on March 7, 2016, Cuss's statements conveyed not just that Defendants were "very comfortable" that Checkmate-026 had been conservatively designed to succeed, but also that Checkmate-026's statistical action plan meant that the clinical trial would provide meaningful data on the performance of Opdivo.

113. On June 16, 2016, Merck announced that Keynote-024 had succeeded in its primary endpoint, establishing that Keytruda worked better than chemotherapy in lung cancer patients with "strong" PD-L1 expression levels greater than 50%. In a June 2016 report, a BMO analyst wrote that "Merck has established a high bar with its positive [Keynote-024] study," but noted that BMO nonetheless "expect[s] that CM-026 will hit its primary PFS endpoint in patients who are 'strongly' PD-L1+ (***we believe this means at least 10%***)."

114. In the face of Keytruda's new success and analysts' reasonable conclusions about the true cutoff employed by Bristol-Myers in Checkmate-026, Defendants continued to conceal their use of a weak PD-L1 expression of 5% for Checkmate-026's cutoff while expressing extreme confidence in the outcome of Checkmate-026. Indeed, on July 28, 2016, during Bristol-Myers' earnings call, Defendant Cuss refused to answer a pointed question about the Checkmate-026 cutoff, stating, "I will say again, we're not going to talk about the high [expressive] cutoff at this

point. But you, obviously, will have an opportunity to see that when we talk about the data. . . . [***A]s I have noted in the past and will note again today, I'm confident in the study design of 026***." Defendant Caforio echoed this sentiment, and emphasized that "we are very confident in . . . the design of the clinical trial."

115. Analysts accepted Defendants' representations of confidence, and reasonably understood them to indicate that Checkmate-026 had been conservatively designed. For example, on July 28, 2016, a Barclays analyst wrote that it "remain[ed] comfortable with the PFS primary endpoint among the both high and low PD-L1 expressers." Similarly, Leerink's analyst report that day that they "have high confidence" that Defendants' "critical" Checkmate-026 study would "succeed on PFS in both PDL-1 high and all PDL1+ [patients]."

3. A Prominent Expert in the Immuno-Oncology Field States That Defendants' Representations That 5% PD-L1 Expression Was "Strong" or "High" Contradicted Known Industry Convention When Made

116. Ronald H Blum, M.D., is a Medical Oncologist with more than forty years' experience as a clinician, academic and researcher. Dr. Blum's research interests are in the clinical trials of new agents and their integration into combined modality approaches to cancer treatment. As a clinical trialist, he has co-authored peer reviewed publications that have defined new practice standards for lung cancer, breast cancer, melanoma, sarcomas, prostate cancer, and supportive care. As an example, he led international clinical trials focused on lung cancer sponsored by National Cancer Institute, National Institute of Health.

117. For more than 20 years, Dr. Blum has been engaged in the independent oversight of late phase clinical trials, through chartered committees known variously as Data Safety Monitoring Boards (DSMB), Data Safety Monitoring Committees (DSMC), Data Monitoring Committees (DMC), or Independent Data Monitoring Committees (IDMC). These committees are

sponsored by academic institutions or pharmaceutical companies. Over the years, he has been the chair of more than 12 such committees, including the Harvard Cancer Center's DSMB and committees focused on lung cancer clinical trials.

118. Dr. Blum has been a consultant to the NCI and served on editorial boards for peer reviewed journals. The American Society of Clinical Oncology (ASCO) honored him as Fellow of ASCO, FASCO, for his contributions to the society having served as chair of multiple committees.

119. Until his retirement from his clinical and administrative duties in 2014, Dr. Blum was the Director of the Beth Israel Cancer Center and Director of Cancer Centers and Programs, Continuum Cancer Centers of New York, and a Medical Director, Continuum Hospice Care, New York, NY and Professor of Medicine, Albert Einstein College of Medicine. Prior to that Dr. Blum was at New York University Medical Center, a Professor of Medicine and the deputy director of the NCI designated Comprehensive Cancer Center. He is now an Adjunct Clinical Professor at the Icahn School of Medicine at Mount Sinai, New York, New York. Dr. Blum continues to teach and participate in interdisciplinary clinical activities and to serve as chair of data safety monitoring committees both for academic and industry sponsored clinical trials. Dr. Blum is a thesis honors graduate of the State University of New York at Buffalo and did postgraduate training at Johns Hopkins University, National Cancer Institute (NCI), and Harvard Medical School.

120. Dr. Blum reviewed Bristol-Myers' representations that Checkmate-026 focused on patients that exhibited a "strong" or "high positive" expression of the PD-L1 biomarker, and certain source documents referenced herein. Based on this review and Dr. Blum's substantial practical and academic experience, including his own involvement in, and oversight of, clinical trials examining PD-1 checkpoint inhibitors—including the Keynote-006 study, one of Merck's

clinical trials of Keytruda—Dr. Blum concluded that Defendants' representations of the strength and level of Checkmate-026's PDL-1 expression focus were contrary to the medical oncology industry's standard use of a 5% PD-L1 expression level to be a measure of low or minimal PD-L1 expression, and usage of "strong expression" to denote a level closer or equal to 50%. Dr. Blum opines that, at all relevant times, Defendants' persistent refusal to disclose their definition of "strong" PD-L1 expression was disingenuous because Defendants' focus on a 5% expression level was inconsistent with the industry's use of "strong" PD-L1 expression during the Class Period.

121. In making this conclusion, Dr. Blum states that Defendants' description of a 5% PD-L1 expression level as "strong" and/or "high" was inconsistent with the widely accepted definition of "strong" or "high" expression during the Class Period. By the start of the Class Period, the industry understood that the strength of PD-L1 expression was a critical variable in assessing the probability of successful response to PD-1 checkpoint inhibitors such as Opdivo and Keytruda. Thus, Dr. Blum explained, the strength of the PD-L1 expression cutoff used in a clinical trial such as Checkmate-026 was a critical study variable in the design, conduct, and interpretation of PD-L1 trials, which was required to be accurately disclosed. As a result, Dr. Blum concluded that Defendants' use of the terms "strong" and/or "high"—terms used in Merck's earlier studies (and specifically in Merck's parallel Keynote-024 study) and in other industry publications to denote 50% or, at the very least, levels above 25%—falsely suggested that Bristol-Myers and the Individual Defendants were using those terms consistent with historical and contemporaneous convention.

V. THE TRUTH IS REVEALED

A. Defendants Announce That Checkmate-026 Failed To Meet Its Primary Endpoint, And Reveal The Shocking Cause—That The Purportedly "Strong" PD-L1 Expression Level Cutoff Was Just 5%.

122. On August 5, 2016, Bristol-Myers shocked the market when the Company revealed for the first time that Checkmate-026 defined patients with "strong" PD-L1 expression as those with just 5% expression—the very minimal level of expression that the Company had historically employed to define the mere baseline of positivity. In a press release that day, Defendants stated that Checkmate-026 "did not meet its primary endpoint of progression-free survival in patients with previously untreated advanced non-small cell lung cancer (NSCLC) whose tumors expressed ***PD-L1 at ≥ 5%***." The press release also quoted Defendant Caforio as stating that Defendants were "disappointed Checkmate-026 did not meet its primary endpoint in this ***broad patient population***."

123. Analysts reacted with shock, anger, and disappointment at not only the news of Checkmate-026's failure, but at the fact that Defendants had concealed from the market that the cutoff for "strong" PD-L1 express was only 5%. For example, in its analyst report issued later that day, BMO Capital Markets wrote that it was

> ***completely puzzled*** by Bristol's decision to evaluate … at a threshold [*i.e.* 5%] this low, particularly given that the *trial description indicated patients would be strongly expressing PD-L1*. . . . ***[BMO] and probably most of the market, thought the threshold was at least 10%, and therefore expected that the trial had a reasonably high probability of success***. . . . [We] suspect that the reason the trial failed is that the PD-1 threshold of at least 5% was too low."

124. Likewise, Cowen wrote that day:

> We published on April 1st that, at a 5% PD-L1 expression cut-off, odds of CM-026 success were 0.5%. Had the expression cut off been 10%, odds of success would have been 67%. Had BMY used the expression cut-off adopted by MRK (50%), then ***CM-026 success would have been certain***.

125. Similarly, Evercore wrote that Checkmate-026 was

> [A] MAJOR SURPRISE – possibly the biggest clinical surprise of my career . . . ***our only lead is the much broader patient population in BMY's trial: their high-expresser cutoff was 5% PD-L1 expression, a much lower bar than MRK's 50%***.

126. William Blair wrote:

> Based on the available data, ***we had confidence in the CheckMate-026 trial; however, the expression level cut-off for the primary analysis of 5% had not been previously disclosed.*** . . . [I]t is apparent that Bristol-Myers designed the CheckMate-026 trial in hopes of addressing a broader patient population, a similar strategy to what was used in second-line NSCLC. ***The broader patient population in the CheckMate-026 trial*** combined with a primary endpoint of progression free survival, which has consistently not been as robust an endpoint with PD-1 inhibitors as overall survival, ***were likely the contributing factors to the failure of CheckMate-026***.

127. J.P. Morgan analysts concurred, writing that it was "disappointed and highly surprised by the outcome and s[aw] ***the failure as largely driven by the study's broad design***"—in other words, the unexpectedly low cutoff.

128. News media reacted similarly. In an article entitled "Bristol-Myers Squibb's Hubris Cost It $21 Billion," *The New York Times* wrote that the Company "has suffered a $21 billion ***self-inflicted wound***":

> The amount is the value that investors wiped off the pharmaceutical company on Friday morning after its trial to greatly broaden the use of one of its most promising cancer drugs failed. ***It was an unnecessarily risky move for Bristol***, whose immunotherapy has been outselling Merck's. The stumble will allow its more cautious rival to clean up.

Similarly, *Investors Business Daily* published an article entitled, "Bristol-Myers Plunges on Surprise Opdivo Failure in Lung Cancer," noting that it was Opdivo's "first conspicuous miss for the drug since it launched in late 2014. . . . ***Bristol-Myers went for a broader patient population, potentially winning a bigger market but increasing its risk of failure***."

129. *Forbes* published an interview with Defendants Caforio, Cuss, and Namouni on August 5, 2016, in the wake of the Company's disastrous announcement. In that article including the interview, entitled "Bristol-Myers Executives: We Took A Bold Risk With Failed Lung

Cancer," *Forbes* wrote: "This morning, Bristol-Myers Squibb shocked Wall Street and the world of cancer medicine with the news that its drug Opdivo had failed to show a benefit as a first choice of treatment in non-small cell lung cancer. Investor forecasts had shown Bristol getting billions of dollars in sales for this use, and shares in Bristol-Myers Squibb slumped as much as 23% in morning trading." *Forbes* noted that, in the wake of this news, "shares of rival Merck, which makes Keytruda, a similar drug, are up 11%."

130. The market reacted harshly to the disclosure of Checkmate-026's previously-concealed, low Primary Outcome endpoint and resulting failure, and Bristol-Myers stock price dropped from $75.32 on August 4, 2016, to $63.28 on August 5, 2016—a stunning one-day decline of ***nearly 16%***.

131. As the public continued to catch its breath from the shocking failure of Checkmate-026, additional consensus formed that Defendants' cutoff selection caused the failure. For example, on August 9, 2016, Cowen published details of a conference call it conducted with "physician experts," who were "not surprised CM-026 failed given the 5% PD-L1 threshold." The experts further stated that it was "unlikely that other factors played a role" in the failure—"***PD-L1 expression level was the key difference***. Opdivo and Keytruda are therapeutically equivalent."

132. Defendants' August 5, 2016 revelation that Checkmate-026 focused on patients with just a 5% PD-L1 expression was news to the market, but was well-known to and in fact devised by Defendants years earlier. While Defendants refused to provide the actual PD-L1 cutoff that they described on *ClinicalTrials.gov* and elsewhere throughout the Class Period as "strongly PD-L1+ tumor expression," the trial protocol for Checkmate-026, which was first issued on December 20, 2013—but only made public in heavily redacted form in June 2017[3]—explicitly

[3] Though the publicly available copy of the protocol indicates that it was revised several times

stated that "Strongly PD-L1+ status is defined as . . . ≥5% tumor cells in a minimum of 100 evaluable tumor cells." This measure was a level of expression so low that it was the most common level used to define the ***bare minimum of PD-L1 positivity at all*** (including in some of Defendants' own studies) (¶¶54-61), and in fact had been referred to as "weak" in industry commentary (¶¶64-68).

133. The Checkmate-026 trial protocol was kept from the public until June 2017, when the *New England Journal of Medicine* published a heavily-redacted version. However, Defendants had helped craft and were aware of and had access to the protocol since its creation. The protocol explicitly states that it is "the confidential and proprietary information of Bristol-Myers Squibb Company and its global affiliates," and in several instances describes the Company's heavy involvement in and oversight of the protocol, including that "[a]ll revisions to the protocol must be discussed with, and be prepared by, BMS"; that "[r]epresentatives of BMS must be allowed to visit all study site locations periodically to assess the data quality and study integrity"; and that "Bristol-Myers Squibb will provide investigational staff training prior to study initiation."

B. The Full Risk Concealed By Defendants' Fraud Materializes When Defendants Reveal Checkmate-026 Is A Total Failure.

134. Even though Checkmate-026 had failed to meet its primary objective, due to Defendants' secret, aggressive, and misleadingly described cutoff, Defendants continued to sow

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over the Class Period, there is no indication that the 5% PD-L1 expression threshold was revised at any point since the protocol's inception. First, as a material element of the clinical trial, federal regulation would have required this threshold to be fixed before beginning the trial. Second, none of the descriptions of the amendments indicate that the expression threshold was changed. Third, Defendant Cuss himself stated on April 28, 2016 that Defendants "***[had] not changed our analysis at all***. The PFS analysis will be on the hazard ratio of the PFS curves ***in the strongly expressing population*** . . ." Finally, even if the expression cutoff had been modified, the final revision was issued on May 31, 2016—months before the end of the Class Period, and even after which time Defendants Namouni, Caforio, and Cuss continued to make false and misleading statements concerning Checkmate-026.

hope that the full, still-nonpublic data from the clinical trial would provide useful information about Opdivo's efficacy through subgroup analyses focusing on patients in the trial who met a higher, 50% PD-L1 threshold. However, such post-hoc analyses of the greater than 50% expression pool lacked validity because it was subject to bias, as that threshold had not been pre-defined at the start of the study. Therefore, Defendants' statements continued to mislead investors as to the design of Checkmate-026.

135. Indeed, in the *Forbes* interview published on August 5, 2016, discussed above in ¶129, Defendants Cuss, Caforio, and Namouni tried to paint the study's failure in a somewhat positive light. Defendant Namouni painted the Company's undisclosed risks as simply an attempt to be "bold," stating "think ***we obviously took a risk to be more bold,*** to try to cover a broader population of patients, because our strategy was really to never leave patients behind." Defendant Caforio then described how Defendants believed that Checkmate-026 could still provide useful data with respect to "the 25% of patients that express PD-1 at the ***very high level***"—in other words, those with 50% PD-L1 expression. However, Caforio did not explain how the study would be able to still elicit this information or the risks involved—even though Defendants (and not investors) had access to the full data at this time.

136. Defendant Caforio expanded on this statement further on September 13, 2016, during the Morgan Stanley Global Healthcare Conference. Caforio stated that, while "[t]he results of Study 26 are clearly disappointing," once Defendants presented "the study in full" in the fall of 2016, "***we will be able to really show the performance of Opdivo in this patient population***. . . . ***We will also be able to communicate some of the exploratory analysis that are being conducted in the trial, including the efficacy of Opdivo in some of the subsets at higher levels of expression of PD-1.*** And obviously, as you know, we have not disclosed that data." Again, Caforio's reference

to "exploratory analysis" suggested the possibility of post-hoc analysis that had been pre-defined (as it would otherwise lack any validity as subject to bias), and therefore continued to mislead investors as to the design of Checkmate-026. Moreover, Caforio's statements clearly admitted that in fact Checkmate-026 had not previously focused on "higher levels of expression," in direct contrast to Defendants' prior characterization of the study's focus as "strong" and "high."

137. Relying on his extensive direct experience in clinical trials (including specifically trials evaluating PD-1 checkpoint inhibitors), Dr. Blum explained that Bristol-Myers and those in charge of the study should have known certain characteristics of the patient population, including specifically the stratification of the PD-L1 expression levels among patients, once the study was populated at the start of the Class Period. As a result, Dr. Blum believes, based on his experience overseeing similar clinical trials, that Defendants' August 5 and September 13, 2016 statements were disingenuous as Defendants did or should have known that Checkmate-026 was not sufficiently powered to stratify results at a 50% PD-L1 expression level. In addition, according to the publicly available version of the protocol only made available months later (*e.g.*, ¶¶132-33), the analyses of PD-L1 expression levels above 5% were not preplanned or specified, limiting the statistical validity of any findings.

138. Defendants' representation that the still-nonpublic results of Checkmate-026 could elicit any useful information was revealed to be without any basis on October 9, 2016, when Defendants for the first time presented the clinical trial's full data at the annual European Society for Medical Oncology ("ESMO"). This presentation fully disclosed the risks concealed by Defendants' secret gamble: not only had Checkmate-026 failed to meet its primary endpoint, but the clinical trial data also indicated that Defendants' concealed aggressive trial design had left

Checkmate-026 inadequate by being underpowered to explore the results on smaller subsets of patients in a range of PD-L1 expressions comparable to Merck's Keytruda-024 study.

139. In a report issued that day, an analyst with Sanford Bernstein characterized Defendants' presentation as a "'worst case scenario' for Opdivo"—

> While it was known that [Checkmate-026] failed, ***most investors at least expected to see a trend towards efficacy in patients with high levels of PDL1 expression*** (mirroring the population MRK studied). ***In fact, there was nothing.*** Now, Opdivo is left with a big question mark, with various theoretical possibilities to explain the findings, but nothing concrete can be proven.

140. The "big question mark" related directly to the same issue that Defendants had concealed from investors: their choice of expression cutoff. Specifically, by designing the clinical trial with a 5% expression cutoff, Defendants ensured only that the data was "powered" for statistical analysis for the entire study population at that cutoff. This design decision—which, as described above (¶¶86-90), Defendants were involved in—created a significant risk that the clinical trial would not be "powered" with an appropriate sample selection to provide meaningful analysis comparing those "high levels of PDL1 expression (mirroring the population MRK studied)" (i.e., greater than 50% PD-L1 expression).

141. The data presented by Defendants on October 9, 2016 revealed that this risk had in fact materialized: Defendants disclosed that they had no means under accepted statistical methodologies of finding a significant difference between the performance of Opdivo and chemotherapy. Moreover, this revelation called into serious question Defendants' earlier statements that the full data from Checkmate-026 could still provide useful information (¶¶135-136) because this data had been available to Defendants since at least August 5, 2016, when Defendants announced that Checkmate-026 had failed.

142. The misleading nature of Defendants' purported "strong" PD-L1 expression cutoff was further reinforced during Defendants' October 9, 2016 presentation at ESMO, when Bristol-

Myers Vice President Nick Botwood explained that Defendants had "stratified the Checkmate-026 study for histology and at the 5% level. ***We didn't stratify at high levels of PD-L1.***" In other words, even more bluntly than Defendant Caforio had weeks prior (¶136), Botwood admitted that Defendants did not consider 5% to be "high"—a clear disconnect from Defendants' earlier claims, such as explicitly made by Defendant Namouni on March 7, 2016, that Checkmate-026 had been "well-designed and well-powered to detect the difference in all the positives but also in ***what we define [as] the high positives***." (¶106.) Further, Botwood also conceded the possibility—that Defendants would later admit—that "had we done a trial specifically and prospectively randomized at 50% [PD-L1 expression cutoff], . . . it seems possible we may have seen a favorable outcome."

143. As a result of Defendants' revelation that the concealed focus on 5% PD-L1 expressors had left the Company unable even to have meaningful data at the 50% expressor level, and thus the clinical trial was a total failure, the price of Bristol-Myers' common stock dropped again, falling more than 10% on one trading day, falling from $55.43 at the close on October 7, 2016 (the nearest prior trading day) to $49.81 on October 10, 2016.

144. In total, the price of Bristol-Myers' stock fell $25.51 per share, from $75.32 at the close of August 4, 2015, to just $49.81 at the close of October 10, 2016, ***a decline of nearly 34%***, causing massive losses to the Class.

145. Market observers and journalists alike linked this decline to the disappointment from Defendants' surprising—and now total—failure in Checkmate-026. For example, on October 9, 2016, investor website *TheStreet.com* wrote that "Merck Solidifies Lung Cancer Immunotherapy Lead Over Bristol-Myers Squibb," stating that Bristol-Myers "leaves the same conference with a competing immunotherapy, Opdivo, that's a total failure in newly diagnosed

lung cancer. There was speculation Bristol might salvage something positive for Opdivo from the negative clinical trial first announced in August. That didn't happen, so now Bristol is in a bad place." Likewise, on October 10, 2016, Leerink wrote in its report, "Bottom Line: BMY's much anticipated Checkmate-026 data . . . . was disappointing."

146. In an article published the same day, *Reuters* wrote that the "lung-cancer setback puts rival Merck drug in the driver seat," with the "[n]ew data is likely ***to prompt doctors to abandon Bristol-Myers Squibb's immunotherapy Opdivo in favor of Merck & Co's rival Keytruda*** in a large segment of the lucrative lung cancer market." Similarly, on October 14, 2016, J.P. Morgan wrote that "[E]xpectations in BMY shares clearly having reset post the failure of -026 (shares are down ~33% since August 5) . . . . CM-026 was clearly disappointing[.]" A Sanford Bernstein analyst report on October 18, 2016, confirmed the hypothesis, noting that in "a survey of 130 US and EU oncologists," 69% "said their perception is moderately / significantly / substantially worse" after seeing the results of the failed Checkmate-026.

147. The market understood that these revelations directly related to the risk concealed by Defendants in failing to disclose the gamble they took in Checkmate-026's design. For example, in its October 10, 2016 article titled "Bristol's Failure of Leadership," *Bloomberg* wrote that Defendants' "aggressive trial designs are hobbling Opdivo, not helping"—

> ***Self-inflicted wounds hurt the most***.
>
> Bristol-Myers Squibb's immune-boosting cancer drug Opdivo had been dominating Merck's rival drug Keytruda because it is marketed to a broader patient population. Opdivo outsold Keytruda by more than $500 million in the second quarter.
>
> But Bristol's attempt to use the same broad-brush approach in treating newly diagnosed lung-cancer patients blew up in its face back in August. In a trial with those patients, Opdivo did not outperform chemotherapy, while a more-conservative trial by Merck succeeded. That result carved $30 billion from Bristol's market cap at the time.

> ***The news got worse this weekend***: Updated data from the same trial show Opdivo didn't even beat chemo among a more-promising patient group. Merck's drug, on the other hand, reduced death in those patients by 40 percent compared to chemo. Markets shaved another $9 billion in Bristol's value on Monday, as shares fell to a two-year low. . . .
>
> Bristol didn't limit trials to patients whose tumors have a very high level of a protein (PD-L1) thought to predict success for this drug class. . . .
>
> It seems unlikely Opdivo is a fundamentally worse drug than Keytruda, given its success over the past few years. . . .
>
> ***But in trying to differentiate Opdivo from its rivals, Bristol's approach has been too aggressively broad, raising the possibility of more design failures to come***.

VI. POST CLASS PERIOD EVENTS

148. The failure of Checkmate-026 crippled Bristol-Myers' lead in the immuno-oncology market. For example, on October 19, 2016, *FierceBiotech* reported:

> The National Comprehensive Cancer Network (NCCN) has weighed in on the checkpoint inhibitor race to first line treatment in non-small cell lung cancer. It gave Merck's Keytruda a thumb's up, but denied both Bristol-Myers Squibb's Opdivo.
>
> All three are FDA-approved to treat second line NSCLC. . . . The NCCN vote was unanimously in favor for treatment of first-line NSCLC with Keytruda based on its Keynote-024 data, observed Wall Street analyst Timothy Anderson at Sanford Bernstein in an Oct. 18 note

149. To date, Opdivo has not secured FDA approval for usage as a first-line monotherapy in treating NSCLC, meaning that Bristol-Myers has been limited to prescriptions of Opdivo in second-line treatments—limiting both its revenue and influence as against Merck, whose Keytruda can be prescribed for both first- and second-line monotherapy.

150. In the wake of their failed study, in addition to the September 13, 2016 and October 9, 2016 concessions described above (¶¶136, 138-142), Defendants made several admissions that Checkmate-026's fundamental flaw was the very thing they had concealed from investors during the Class Period: the aggressive, but undisclosed, endpoint PD-L1 expression cutoff selection.

Moreover, Defendants' admissions indicate that the 5% expression level was known to be neither strong nor high at the time of the study.

151. For example, on November 8, 2016, speaking at the Credit Suisse Health Care conference, Defendant Namouni ***admitted that Bristol-Myers did not focus on tumors exhibiting a strong, or "high" expression of PD-L1, a shocking acknowledgment that their multiple Class Period statements saying just that were entirely false***:

> Obviously, the study ***was not designed*** to look at the smaller subgroup of highly inflamed tumor or ***high expression of PD-L1***. Basically, ***that's the reason we are having a study that did not meet its primary endpoint.***

152. Later, Defendants Caforio, Bancroft, Namouni, and Gordon met with analysts from Leerink, during which they spoke about Checkmate-026. As summarized in Leerink's December 2, 2016 report, "***Overall, mgmt. believes the failure of CM-026 was due to it choosing the wrong endpoint***." However, as described above, the endpoint that was chosen—5%—had been concealed from investors until August 5, 2016.

153. On March 8, 2017, Bristol-Myers announced that Defendant Cuss, Bristol-Myers' Chief Scientific Officer and Head of Research and Development since July 2013, was leaving the Company. That Cuss departed so close on the heels of the Checkmate-026 debacle did not go unnoticed. On March 9, 2017, industry website *FiercePharma* published an article titled "Bristol-Myers' R&D revamp mirrors upheaval in Opdivo's lunch cancer fortunes," which attributed Cuss' departure to Checkmate-026:

> Bristol-Myers Squibb has a new R&D chief. ***That's not much of a surprise to those who've been following the company's fall from grace, thanks to its unexpected and brutal failure in a cancer immunotherapy trial last fall, or the precipitous decline in its share price since***.
>
> With billions cut from peak sales estimates for its PD-1 checkpoint inhibitor Opdivo—which until the trial fiasco was seen as the natural market leader—and shares down to $57.40 Wednesday from more than $72 before the data went public,

> Chief Scientific Officer Francis Cuss made for the exit Wednesday. Board member and cancer doctor Thomas Lynch stepped into his place.

154. Dr. Cuss's replacement, Dr. Thomas J. Lynch, is a highly-esteemed oncologist who has served as the Professor of Medical Oncology at Yale and Chief of Hematology/Oncology at Massachusetts General Hospital Cancer Center. Dr. Lynch had served as a director of Bristol-Myers since January 1, 2014, having been elected specifically because—in the words of Defendant Andreotti—he was "a world class oncologist" whose perspectives would be "invaluable" as Defendants "advance our work in immuno-oncology and build our leadership position in oncology."

155. On June 2, 2017, *Forbes* published a discussion of Checkmate-026's failure with Dr. Lynch, who—speaking with both an impressive scientific pedigree and from having been a director at Bristol-Myers throughout the Class Period and the Checkmate-026 study—also conceded that Defendants' aggressive and undisclosed endpoint selection had caused the failure:

> Last August, before Lynch took over, Bristol's cancer drug Opdivo failed to help patients with previously untreated lung cancer. Just months before, Keytruda, a rival drug from Merck, had succeeded in a similar study. The main question: ***why had Bristol failed and Merck succeeded?***
>
> Lots of reasons have been proposed: that there were differences in the way diagnostic tests measured levels of the programmed cell death receptor one, which predicts how well these drugs will work. That there were unplanned differences between the control group in the Bristol study and the main trial. Lynch, building on his decades as a lung cancer specialist at the Massachusetts General Hospital in Boston, doesn't buy any of those. He says ***the most likely difference is the obvious one: Bristol chose a lower cutoff*** of a diagnostic test that measures levels what's called the programmed death receptor ligand one, or PDL1. It predicts who will benefit from drugs like Keytruda and Opdivo, known as PD-1 inhibitors.
>
> 'I'm a lung cancer guy,' Lynch says. 'And I know that lung cancer is a very hard disease to treat. I also know that I believe that there probably are greater similarities between [Keytruda] and [Opdivo] than there are differences between these drugs. . . .
>
> ***Quite simply, Lynch says, it looks as if Merck's predictive cutoff was 'superior' to Bristol's. No excuses, no mysteries.***

156. While Lynch pointed out that Merck's expression cutoff was "superior," he did not address the fact that Defendants had consistently described that cutoff to investors ***identically*** to Merck, despite knowing otherwise. On June 26, 2017, nearly one year after the August 5, 2016 disclosure, Bristol-Myers tried to rewrite history, and altered their *ClinicalTrials.gov* description of Checkmate-026's "Primary Outcome." That important metric was transformed from "Progression-Free Survival (PFS) . . . in subjects with ***strongly Programmed death-ligand 1+ (PD-L1+) tumor expression***" to "Progression-Free Survival in Participants ***With PD-L1 Expression >= 5%***." In other words, Defendants finally came clean that 5% was not "strongly Programmed death-ligand 1+ (PD-L1+) tumor expression" nearly one year too late.

VII. DEFENDANTS' FALSE AND MISLEADING STATEMENTS AND OMISSIONS

157. Defendants made materially false and misleading statements and omissions during the Class Period concerning, among other things, the primary outcome, focus and design of the clinical trial known as Checkmate-026. Defendants made the following materially false and misleading statements and omissions during the Class Period with knowledge or reckless disregard for their falsity at the time they were made.

A. Defendants Claim That Checkmate-026 Is Focused On Patients With "Strong" PD-L1 Tumor Expression

158. On January 27, 2015, the first day of the Class Period, Bristol-Myers updated its Checkmate-026 study, the details of which were published and made publicly-available on the website *ClinicalTrials.gov*. As discussed above, FDA rules required Bristol-Myers to submit clinical trial information to the Director of the National Institutes of Health (NIH) for inclusion in the registry and results database established via *ClinicalTrials.gov*. (¶78; *see* 42 C.F.R. § 11.) As described by the University of California Office of Research, "[t]he purpose of *ClinicalTrials.gov* is to disclose to the public key information about clinical trials that are currently available or that

have been conducted." Federal law requires that clinical trial information submitted to *ClinicalTrials.gov* "shall not be false or misleading in any particular." (*See* 42 U.S.C. § 282 (j)(5).) Further, also as noted above, Defendants acknowledged during the Class Period that they had control of their *ClinicalTrials.gov* disclosures concerning Checkmate-026. For example, on September 8, 2015, Defendant Giordano referred to information on *ClinicalTrials.gov* by stating that "Of course, we communicate with the FDA anything ***we might disclose in Clinicaltrials.gov***."

159. The Checkmate-026 study was titled "An Open-Label, Randomized Phase 3 Trial of Nivolumab Versus Investigator's Choice Chemotherapy as First-Line Therapy for Stage IV or Recurrent PD-L1+ Non-Small Cell Lung Cancer (Checkmate 026)." In the information concerning Checkmate-026 published on *ClinicalTrials.gov*, Bristol-Myers provided a "Brief Summary" of the Study: "[t]he purpose of this study is to show that Nivolumab will improve progression free survival in subjects ***with strongly Stage IV or Recurrent PD-L1+ non-small cell lung cancer when compared to chemotherapy***." According to federal regulation, the Brief Summary disclosed on *ClinicalTrials.gov* must provide "a short description of the clinical trial, including a brief statement of the clinical trial's hypothesis, written in language intended for the lay public." (42 C.F.R. § 11.10.)

160. In its *ClinicalTrials.gov* disclosure, the Company also defined the "Primary Outcome Measure" of the study:

> Progression Free Survival (PFS) as assessed by independent radiology review committee (IRRC) ***in subjects with strongly Programmed death-ligand 1+ (PD-L1+) tumor expression***.
>
> PFS is defined as the time from randomization to the date of the first documented tumor progression as determined by the IRRC (per RECIST 1.1), or death due to any cause Progression Free Survival (PFS) as assessed by independent radiology review committee (IRRC) ***in subjects with strongly Programmed death-ligand 1+ (PD-L1+) tumor expression***.

According to federal regulation, the Primary Outcome Measure disclosed on *ClinicalTrials.gov* conveys to the public "the outcome measure(s) of ***greatest importance*** specified in the protocol," and "the planned outcome measure that is the ***most important*** for evaluation [of] the effect of an intervention/treatment." Therefore, Bristol-Myers represented that the Company's "most important" outcome from Checkmate-026 was to yield positive results concerning progression free survival for patients with "***strongly***" PD-L1+ tumor expression. Bristol-Myers also represented that the top two of its four "secondary outcome measures" were focused on the "objective response rate" and "overall survival" in patients "with ***strongly PD-L1+ expression***."

161. Defendant Bristol-Myers' statements in the Brief Summary, Primary Outcome Measure, and Secondary Outcome Measures of Checkmate-026, described above in ¶¶159-160, were materially false and misleading because Defendants knew or recklessly disregarded from the first day of the Class Period that Checkmate-026 was focused on patients with PD-L1 expression greater than or equal to just 5%—a weak level of expression that most commonly defined the bare minimum for PD-L1 positivity. (¶¶54-61, 120.) Defendants also knew or recklessly disregarded that the clinical trial was neither focused on nor designed to capture data concerning, those patients with "strong" PD-L1 expression—as frequently understood to mean PD-L1 expression levels greater than or equal to 50%. (¶¶62-72, 121.) The level of PD-L1 expression employed by Bristol-Myers in this prominent clinical study was highly material to investors, in part because data indicated that the level of expression was a critical variable in assessing Opdivo's efficacy and therefore the study's odds of success. Numerous prominent research publications, as well as prior clinical studies conducted both by Bristol-Myers and its competitors, indicated that Checkmate-026's focus on "strong" expressors of PD-L1 meant that the primary focus of the clinical trial was the efficacy of Opdivo as a first-line monotherapy on the 25% of the NSCLC patient population

that exhibited 50% or greater PD-L1 expression, and for which the drug was expected to be most successful. (¶¶54-72, 120-21.) Through their deception, Defendants misled investors as to the true risk inherent in Checkmate-026's primary focus—that the clinical trial targeted the majority (55%) of the NSCLC population with weak 5% levels of PD-L1 expression for whom the data indicated Opdivo was much less likely to be effective. Moreover, the low expression cutoff chosen also created an undisclosed risk that the study would not be powered to detect statistically meaningful results when stratified at higher levels of expression.

162. In addition, on the following dates in the Class Period, Defendants again exercised their control over their *ClinicalTrials.gov* disclosures concerning Checkmate-026 by further updating information concerning the clinical trial: February 16, 2015, March 5, 2015, March 23, 2015, April 13, 2015, April 20, 2015, May 6, 2015, May 20, 2015, June 3, 2015, June 22, 2015, July 3, 2015, July 17, 2015, August 4, 2015, August 18, 2015, September 3, 2015, September 16, 2015, October 5, 2015, October 15, 2015, November 4, 2015, November 18, 2015, December 8, 2015, December 21, 2015, January 6, 2016, January 21, 2016, January 26, 2016, February 10, 2016, February 25, 2016, March 11, 2016, March 28, 2016, April 12, 2016, April 21, 2016, April 25, 2016, May 10, 2016, May 25, 2016, June 9, 2016, June 24, 2016, July 11, 2016, and July 26, 2016.

163. In each instance, Defendants did not change the Brief Summary, Primary Outcome Measure, or Secondary Outcome Measures for the Checkmate-026 discussed above (¶¶159-60), thus repeating (and thereby making anew) each time Defendants' statements that "[t]he purpose of this study is to show that Nivolumab will improve progression free survival in subjects ***with strongly Stage IV or Recurrent PD-L1+ non-small cell lung cancer when compared to chemotherapy***," that the Primary Outcome Measure of Checkmate-026 was "Progression Free

Survival (PFS) as assessed by independent radiology review committee (IRRC) ***in subjects with strongly Programmed death-ligand 1+ (PD-L1+) tumor expression***," and that the top two of four Secondary Outcome Measures of Checkmate-026 focused on the "objective response rate" and "overall survival" in patients "with ***strongly PD-L1+ expression***." All of the updated versions of the Checkmate-026 Study were false and misleading for the reasons stated above in ¶161.

164. Moreover, Defendants' *ClinicalTrials.gov* statements and omissions—including those made again in connection with their May, June, and July 2016 updates—continued to be false and misleading following Defendant Cuss's acknowledgment during an April 28, 2016 conference call (discussed *supra* at ¶112 and *infra* at ¶¶193-97) that Bristol-Myers' definition of "strongly expressing PD-[L]1 [was] lower than 50%." While Defendant Cuss did acknowledge for the first time that 50% was not the PD-L1 expression cutoff in Checkmate-026, Cuss nonetheless: a) reaffirmed that Checkmate-026 was still "looking at [] strongly expressing patients;" b) contemporaneously refused to define the level of PD-L1 expression; and c) failed to inform investors that Checkmate-026 in fact was focused on those patients with a weak 5% expression. Moreover, Defendant Cuss emphasized in the April 28, 2016 call that the Company had not changed its efficacy endpoints and statistical analysis plan, noting that "***we have not changed our analysis at all***. The PFS analysis will be on the hazard ratio of the PFS curves ***in the strongly expressing population*** . . ." Cuss also made clear that Checkmate-026 would look at the weaker PD-L1 expressors only "[i]f the primary endpoint of PFS is strongly – is met in the strongly expressing patients, [then] we'll also look at all the randomized population, which includes all those that express at the greater than 1% level." Accordingly, Defendants' statements on *ClinicalTrials.gov* in May, June, and July 2016 continued to mislead investors as to the true risk inherent in Checkmate-026's primary focus, by falsely and misleadingly stating that the clinical

trial targeted "strong" PD-L1 expressors rather than weak, 5% PD-L1 expressors for whom the data indicated Opdivo was much less likely to be effective, as well as the additional risk that the study would not be powered to detect statistically meaningful results when stratified at higher levels of expression.

165. Bristol-Myers finally acknowledged the falsity of their statements concerning Checkmate-026 on *ClinicalTrials.gov* when, after the end of the Class Period, on June 26, 2017, Bristol-Myers updated its disclosures. On that date, the Company changed its *ClinicalTrials.gov* entry to correctly state the Primary Outcome Measure, revising that important metric from the original "Progression-Free Survival (PFS) . . . . in subjects with strongly Programmed death-ligand 1+ (PD-L1+) tumor expression" to "Progression-Free Survival in ***Participants With PD-L1 Expression >= 5%.***" Bristol-Myers likewise revised at that time the two of its four "secondary outcome measures" that examined patients "with ***strongly PD-L1+ expression***" to instead "***Participants With PD-L1 Expression >= 5%***."

B. Additional Statements Throughout The Class Period Concerning Checkmate-026's Design And Likelihood Of Success All Failed To Disclose That The Study Was Aggressively Designed To Focus On Weak PD-L1 Expressors And Had A Substantial Risk Of Failure

1. The July 23, 2015 Conference Call

166. On July 23, 2015, Defendants Cuss, Caforio, and Bancroft participated in Bristol-Myers' earnings call, during which Cuss stated that Checkmate-026 was one of the "registration potential studies" in Defendants' "very important" "first-line lung strategy." Defendant Cuss also stated that "in monotherapy, [Checkmate] -026 in PD-L1 expressing patients . . . provides ***the quickest way to bring Opdivo to first-line patients*** . . ."

167. Defendant Cuss's statement described above in ¶166 was false and misleading because, as Bristol-Myers' Chief Scientific Officer, Cuss knew from the first day of the Class

Period that Checkmate-026 focused on patients with PD-L1 expression greater than or equal to just 5%, and not the "strong" or "high positive" expression level communicated to investors. For example, former employees of Bristol-Myers have confirmed that Defendant Cuss was part of a committee that made the decision to invest in Checkmate-026, and through that position, was informed as to all material aspects of the study. (*E.g.*, ¶¶88, 90.) Defendant Cuss knew or was reckless in not knowing that Bristol-Myers in numerous prior and contemporaneous studies of Opdivo used the 5% expression of PD-L1 to indicate only mere positivity. Moreover, Defendant Cuss knew or was reckless in not knowing that the industry wrongly understood that Checkmate-026 focused on "strong" expressors of PD-L1 that carried relatively low risk and for which the drug was expected to be most successful. Moreover, the weak expression focus chosen also created an undisclosed risk that the study would not be powered to detect statistically meaningful results when stratified at higher levels of expression.

168. Accordingly, Defendant Cuss's statement that Checkmate-026 was designed to "provide[] ***the quickest way to bring Opdivo to first-line patients***" falsely communicated to investors that the study was designed conservatively to focus on "strong" or "high positive" expressors of PD-L1 that would be successful and quickly approved for market and made available to first-line patients who were most suitable for Opdivo monotherapy. This misleading statement created an affirmative duty to disclose that the study was not directed at PD-L1 positive patients that expressed "strong" or "high positive" levels of PD-L1 positivity, as represented, but was instead a much riskier study directed at PD-L1 positive patients that expressed only weak or low levels of PD-L1 positivity.

169. Further, Defendants Caforio and Bancroft also participated in the July 23, 2015 earnings call and, as Bristol-Myers' Chief Operating Officer and Chief Financial Officer

(respectively), also had knowledge concerning the truth about Checkmate-026. (*E.g.*, ¶¶53, 86-90.) Thus, Caforio and Bancroft understood that Cuss's statement described above in ¶166 was false and misleading for the reasons described above. Accordingly, Caforio and Bancroft had a duty to correct Cuss's statement by providing the full truth about the true risk inherent in Checkmate-026 to investors, but did not do so.

2. The September 8, 2015 WCLC 2015 Review And Conference Call

170. On September 8, 2015, Bristol-Myers published on its website the "WCLC 2015 Review"—a powerpoint presentation discussing the data presented by Bristol-Myers at the June 6-9, 2015 16th Annual World Conference on Lung Cancer. In this presentation, Bristol-Myers commented on the Company's "Front Line NSCLC Strategy" and stated that, based on its ongoing clinical trials, the ***"[m]agnitude of benefit seen in PD-L1 expressors confirms first line monotherapy strategy in PD-L1 expressing patients (-026)***."

171. This statement described above in ¶170 was false and misleading because the Company represented to investors the importance of the "***[m]agnitude of benefit seen in PD-L1 expressors***," but failed to disclose that the Company's published statements in *ClinicalsTrial.gov*, which had been updated by the Company as recently as September 3, 2015, concerning Checkmate-026's focus on "strong" PD-L1 expressors was untrue.

172. Defendants Giordano and Namouni discussed this presentation during a September 8, 2015 investor conference call. Defendant Giordano spoke directly about Checkmate-026, and informed investors that "***[Checkmate-026 is] our lead study***, the first study that will read out as a Phase III in non-small cell lung cancer first-line setting." Defendant Giordano also confirmed the importance of PD-L1 expression to the success of Checkmate-026, stating "we have recapitulated the role of PD-L1 expression in non-small cell lung cancer and have ***identified that the magnitude***

of benefit in the PD-L1 expressors confirms our first-line monotherapy approach to study the PD-L1 expressors in study 026."

173. Defendant Giordano also indicated to investors during the September 8, 2015 conference call that Defendants expected to extract useful interim data from Checkmate-026, stating: "***[w]e have great confidence that [first-line monotherapy] approach will give us an early read of the role of PD-L1 expressing patients in the first-line setting***." Giordano attributed the "early read" on Checkmate-026 to the study's superior design: "As a reminder, the 026 study design[,] ***for which we had great confidence***[,] ***the study has been designed appropriately*** to read out significantly in the next several months in which we're studying the PD-L1 positive population with end points of PFS." Defendant Giordano continued:

> [Checkmate] 026 is the first study to read out for patients in the first-line study – first-line setting. ***We've designed that study in order to get speed to market and be able to treat patients in the first-line setting***. Selection of PD-L1 positives turned out to be a very wise chose based upon the magnitude of benefit that we're expecting to see in that patient population in that study design. . . .

174. While Defendant Giordano noted that "approximately 70% of patients in [Checkmate-026] with regard to screening are PD-L1 expressors," he pointedly did not disclose what percentage of those patients exhibited the "strong" PD-L1 expression discussed in the official Checkmate-026 study published on *ClinicalTrials.gov*.

175. Defendant Giordano's statements described above in ¶¶172-74 were false and misleading because, as Bristol-Myers' head of immuno-oncology development who was directly involved in the design and oversight of Checkmate-026, Giordano knew from the first day of the Class Period that Checkmate-026 focused on patients with PD-L1 expression greater than or equal to just 5%, and not the "strong" expression level communicated to investors from the start of the Class Period. (*E.g.*, ¶¶53, 88, 90.) Defendant Giordano knew or was reckless in not knowing that numerous prominent research publications, as well as prior clinical studies conducted both by

Bristol-Myers and its competitors, indicated that Defendants' contemporaneous statements on *ClinicalTrials.gov*, which had been updated as recently as September 3, 2015, that Checkmate-026 focused its Primary Outcome and Secondary Outcomes on "strong" expressors of PD-L1 meant that the primary focus of the clinical trial was the efficacy of Opdivo as a first-line monotherapy on the 25% of the NSCLC patient population that exhibited 50% or greater PD-L1 expression, and for which the drug was expected to be most successful. Moreover, the low expression cutoff chosen also created an undisclosed risk that the study would not be powered to detect statistically meaningful results when stratified at higher levels of expression.

176. Accordingly, Defendant Giordano knew or was reckless in not knowing that the deceptive statements on *ClinicalTrials.gov* had misled investors as to the true risk inherent in Checkmate-026's primary focus—that the clinical trial targeted the majority (55%) of the NSCLC population with weak 5% levels of PD-L1 expression for whom the data indicated Opdivo was much less likely to be effective—and Giordano had a duty to correct those statements by providing the full, highly material truth that the study was primarily focused on patients with a weak 5% PD-L1 tumor expression, but did not do so.

177. Further, Giordano's statements that Defendants had "great confidence" in Checkmate-026, and that the clinical trial was "designed appropriately," were false and misleading because they misled investors about the true risk inherent in Checkmate-026's primary focus—that the clinical trial targeted the majority (55%) of the NSCLC population with weak 5% levels of PD-L1 expression for whom the data indicated Opdivo was much less likely to be effective—and the additional risk that the study would not be powered to detect statistically meaningful results when stratified at higher levels of expression.

178. Further, Defendant Namouni also participated in the September 8, 2015 conference call and, as Bristol-Myers' development lead for Opdivo who was also directly involved in the study's design and oversight (*e.g.*, ¶¶53, 58, 71-72, 87-88, 90), also had knowledge concerning the truth about Checkmate-026. Namouni thus understood that Giordano's statements described above in ¶¶172-74 were false and misleading for the reasons described above. Accordingly, Namouni had a duty to correct Giordano's statements by providing the full truth about the true risk inherent in Checkmate-026 to investors, but did not do so.

3. The January 28, 2016 Conference Call

179. On January 28, 2016, Defendants Cuss, Caforio, and Bancroft participated in Bristol-Myers' January 28, 2016 earnings call. During that call, Cuss once again misled investors about Checkmate-026, stating "[A]s far as 026 is concerned, ***we have actually taken great care in the design of this study. We have paid real attention to the choice of end point***, the optimal timing of the analysis, the role of the non-proportional hazard ratio, and ***of course, the role of PD-L1 expression and the sample size.***" Cuss continued, "we believe we really have got ***the ultimate balance of speed, and the design to deliver positive results*** in the widest population of first-line lung patients as we've done in other studies of Opdivo recently. . . ."

180. Defendant Cuss's statements described above in ¶179 were false and misleading because, as Bristol-Myers' Chief Scientific Officer and his role approving the design and cost of Checkmate-026, Cuss knew from the first day of the Class Period that Checkmate-026 focused on patients with PD-L1 expression greater than or equal to just 5%, and not the "strong" expression level communicated to investors from the start of the Class Period. (*E.g.*, ¶¶53, 88, 90.) Defendant Cuss knew or should have known that numerous prominent research publications, as well as prior clinical studies conducted both by Bristol-Myers and its competitors, indicated that Defendants'

contemporaneous statements on *ClinicalTrials.gov*, which had been updated as recently as January 26, 2016, that Checkmate-026 focused its Primary Outcome and Secondary Outcomes on "strong" expressors of PD-L1 meant that the primary focus of the clinical trial was the efficacy of Opdivo as a first-line monotherapy on the 25% of the NSCLC patient population that exhibited 50% or greater PD-L1 expression, and for which the drug was expected to be most successful. Moreover, the low expression cutoff chosen also created an undisclosed risk that the study would not be powered to detect statistically meaningful results when stratified at higher levels of expression.

181. Accordingly, Defendant Cuss's statements concerning the "***great care***" taken "***in the design of [Checkmate-026]***" and the "***real attention***" paid to "***the role of PD-L1 expression and the sample size***" were false and misleading and created an affirmative duty to disclose that the study was not directed at PD-L1 positive patients that expressed strong or high levels of PD-L1 positivity, as represented, but was instead a much riskier study directed at PD-L1 positive patients that expressed only weak or low levels of PD-L1 positivity. Moreover, Cuss's assertion that Checkmate-026 had "***the design to deliver positive results***" was also false and misleading because it misled investors about the true risk inherent in Checkmate-026's primary focus—that the clinical trial targeted the majority (55%) of the NSCLC population with weak 5% levels of PD-L1 expression for whom the data indicated Opdivo was much less likely to be effective—and the additional risk that the study would not be powered to detect statistically meaningful results when stratified at higher levels of expression.

182. Further, Defendants Bancroft and Caforio also participated in the January 28, 2016 conference call and, as Bristol-Myers' Chief Financial Officer and Chief Operating Officer (respectively), also had knowledge concerning the truth about Checkmate-026. (*E.g.*, ¶¶53, 87-90.) Indeed, FE-2 described how Caforio directly participated in the decision to focus on the maximum

forecasted patient pool and lowest expression level. (¶87.) Thus, Bancroft and Caforio understood that Cuss's statements described above in ¶179 were false and misleading for the reasons described above. Accordingly, Bancroft and Caforio had a duty to correct Cuss's statements by providing the full truth about the true risk inherent in Checkmate-026 to investors, but did not do so.

4. The February 10, 2016 Leerink Conference

183. On February 10, 2016, Defendant Bancroft participated in the Leerink Partners Global Healthcare Conference, stating:

> Well, lung is a big unmet medical need. It's a large number of patients and . . . . So that has been very, very important area for Bristol-Myers Squibb. . . .
>
> [O]ur objective is . . . to really raise the standard of care . . . [a]nd also ultimately replace chemotherapy altogether. . . .
>
> If you look at our 026 trial, which is first-line lung monotherapy with Opdivo, which we expect to read out later this year, we have a fairly broad patient population in that trial. ***So we have greater than 1%, and we also have high expressers and low expressers in that trial***.
>
> And ***we feel very good about how that trial was designed*** and the overall patient population that that covers. . . .

184. Defendant Bancroft's statements described above in ¶183 were false and misleading because, as Bristol-Myers' Chief Financial Officer, Bancroft knew from the first day of the Class Period that Checkmate-026 focused on patients with PD-L1 expression greater than or equal to just 5%, and not the "strong" expression level communicated to investors from the start of the Class Period. (*E.g.*, ¶¶53, 88-89.) Defendant Bancroft knew or should have known that numerous prominent research publications, as well as prior clinical studies conducted both by Bristol-Myers and its competitors, indicated that Defendants' contemporaneous statements on *ClinicalTrials.gov*, which had been updated as recently that day on February 10, 2016, that Checkmate-026 focused its Primary Outcome and Secondary Outcomes on "strong" expressors of PD-L1 meant that the primary focus of the clinical trial was the efficacy of Opdivo as a first-line

monotherapy on the 25% of the NSCLC patient population that exhibited 50% or greater PD-L1 expression, and for which the drug was expected to be most successful. Moreover, the low expression cutoff chosen also created an undisclosed risk that the study would not be powered to detect statistically meaningful results when stratified at higher levels of expression.

185. Accordingly, Defendant Bancroft's statement that "we feel very good about how that trial was designed," and his assertion that Checkmate-026 had "high expressors and low expressors," were misleading and created an affirmative duty to disclose that the study was not directed at PD-L1 positive patients that expressed strong or "high" levels of PD-L1 positivity, as represented, but was instead a much riskier study directed at PD-L1 positive patients that expressed only weak or low levels of PD-L1 positivity. Moreover, Bancroft's assertion of Defendants' confidence in the design of Checkmate-026 was false and misleading because it misled investors about the true risk inherent in Checkmate-026's primary focus—that the clinical trial targeted the majority (55%) of the NSCLC population with weak 5% levels of PD-L1 expression for whom the data indicated Opdivo was much less likely to be effective—and the additional risk that the study would not be powered to detect statistically meaningful results when stratified at higher levels of expression.

5. The February 25, 2016 Meeting With Analysts

186. On or around February 25, 2016, management from Defendant Bristol-Myers met with Credit Suisse. In its February 25, 2016 report discussing that meeting, Credit Suisse analysts noted that "the most significant catalysts for [Bristol-Myers and Merck] in 2016" were "the results from the pivotal studies for BMY's Opdivo (Checkmate 026) and MRK's Keytruda (KEYNOTE 024) in the first-line non-small cell lung cancer (NSCLC) setting." Credit Suisse wrote that at that

meeting, Bristol-Myers "management team" stated that, with respect to Checkmate-026, they had "***confidence that they are utilizing the correct study designs in the first-line setting***."

187. Defendant Bristol-Myers' statement described above in ¶186 was false and misleading because the Company and its management (including the Individual Defendants) knew from the first day of the Class Period that Checkmate-026 focused on patients with PD-L1 expression greater than or equal to just 5%, and not the "strong" expression level communicated to investors from the start of the Class Period. The Company and its management knew or should have known that numerous prominent research publications, as well as prior clinical studies conducted both by Bristol-Myers and its competitors, indicated that Defendants' contemporaneous statements on *ClinicalTrials.gov*, which had been updated that day, on February 25, 2016, that Checkmate-026 focused its Primary Outcome and Secondary Outcomes on "strong" expressors of PD-L1 meant that the primary focus of the clinical trial was the efficacy of Opdivo as a first-line monotherapy on the 25% of the NSCLC patient population that exhibited 50% or greater PD-L1 expression, and for which the drug was expected to be most successful. Moreover, the low expression cutoff chosen also created an undisclosed risk that the study would not be powered to detect statistically meaningful results when stratified at higher levels of expression.

188. Accordingly, the assertion by the Company and its management of their "confidence that they are utilizing the correct study designs" was false and misleading because it misled investors about the true risk inherent in Checkmate-026's primary focus—that the clinical trial targeted the majority (55%) of the NSCLC population with weak 5% levels of PD-L1 expression for whom the data indicated Opdivo was much less likely to be effective—and the additional risk that the study would not be powered to detect statistically meaningful results when stratified at higher levels of expression.

6. The March 7, 2016 Cowen Health Care Conference

189. On March 7, 2016, Cowen published an analyst report covering Checkmate-026 that reported that it "believe[d] a cut-off of ≥50% is likely" for Checkmate's primary patient endpoint. Later that day, Defendant Namouni participated in Cowen's Health Care Conference, during which the Cowen analyst invited Defendant Namouni to "correct anything in the report that was issued this morning, if anything, that needs to be corrected." Defendant Namouni responded, stating:

> ***This study was well-designed***, and this study represents all the knowledge in terms of design that was really made at Bristol-Myers Squibb from our understanding, from a number of positive Phase 3s looking at different endpoints, and this helps us to design these type of studies. In this study, we are looking at this population. ***We are looking at high expresser. We're looking at low expressers***. Our level of expression are different from other markers who is with other medicines. Just need to be careful there, but ***this study is more powered and well-designed to detect a PFS difference at the right time in this population of patients.***
>
> ***[T]he study has been well-designed and well-powered to detect the difference*** in all the positives but also ***in what we define [as] the high positives***. . . .

190. Defendant Namouni's statements described above in ¶189 were false and misleading because, as Bristol-Myers' development lead for Opdivo who directly participated in the design and oversight of Checkmate-026, Namouni knew from the first day of the Class Period that Checkmate-026 focused on patients with PD-L1 expression greater than or equal to just 5%, and not the "strong" or "high positive" expression level communicated to investors from the start of the Class Period. (*E.g.*, ¶¶53, 58, 71-72, 87-88.) Defendant Namouni knew or should have known that numerous prominent research publications, as well as prior clinical studies conducted both by Bristol-Myers and its competitors, indicated that Defendants' contemporaneous statements on *ClinicalTrials.gov*, which was updated as recently as February 25, 2016, that Checkmate-026 focused its Primary Outcome and Secondary Outcomes on "strong" expressors of PD-L1 meant that the primary focus of the clinical trial was the efficacy of Opdivo as a first-line monotherapy

on the 25% of the NSCLC patient population that exhibited 50% or greater PD-L1 expression, and for which the drug was expected to be most successful. Moreover, the low expression cutoff chosen also created an undisclosed risk that the study would not be powered to detect statistically meaningful results when stratified at higher levels of expression.

191. Accordingly, Defendant Namouni's statements concerning the design of Checkmate-026, and, specifically, the use of "high expresser[s]" and "high positives" in Checkmate-026, were false and misleading and created an affirmative duty to disclose that the study was not directed at PD-L1 positive patients that expressed strong or high levels of PD-L1 positivity, as represented, but was instead a much riskier study directed at PD-L1 positive patients that expressed only weak or low levels of PD-L1 positivity. Namouni's statements were further false and misleading in this respect because he was specifically invited to correct statements in a Cowen analyst report that explicitly predicted that Defendants' "strong" PD-L1 expression cutoff was 50%, and did not correct that report.

192. Moreover, Defendant Namouni's assertions that Checkmate-026 had "been well-designed" and was "well-powered to detect the difference in . . . what we define [as] the high positives" were also false and misleading because they were made despite knowing that the study was not powered to detect statistically meaningful results when stratified at higher levels of expression. In fact, Dr. Ronald Blum—relying on his extensive experience of involvement in, and oversight of, clinical trials (including specifically trials evaluating PD-1 checkpoint inhibitors)—states that Defendants would have known at all times the characteristics of the patient population, including the PD-L1 expression levels, which would have revealed all along that the study was not sufficiently powered to stratify at truly "strong" expression levels.

7. The April 28, 2016 Conference Call

193. On April 28, 2016, Defendants Caforio and Cuss participated in Bristol-Myers' earnings call, during which they made the following statements:

> [Analyst]: ***Obviously investors are very focused on the upcoming front-line long studies from both Merck and you guys.*** Can you compare and contrast the first – the two first line monotherapy trials of Opdivo versus Keytruda, since that's obviously what we're focused on? . . . .
>
> [T]here's also worry out there about PFS being a challenging endpoint, just given that chemotherapy works pretty well in front line lung. . . .
>
> [Defendant Caforio]: We have, I am convinced, the right approach of investigating monotherapy, both in a broader population of PD-1-positive patients and ***in a subset of patients that express PD-1 at a higher level***. . . .
>
> [Defendant Cuss]: [L]et me emphasize, ***we've really taken great care in the design of study 026 in its choice of its endpoint***, the optimal timing of the analysis, which I just mentioned, the role of non-proportional hazard ratio, ***the role of PDL-1 expression***, sample size. And we've used the results of many of our published and unpublished data to look at this and essentially ***we remain very confident***. We think we've got the optimum balance of speeds and design, and to deliver results not just in a narrow population but in the widest population of first line lung patients, as we've done with other studies of Opdivo recently.
>
> Just to be very specific, in the trial design we're looking at both ***strongly expressing patients*** and patients with any level of expression. We've not actually disclosed the actual level of what strongly expressing PD-1 is, but it's lower than 50%. It's not clear to us that you need to have very high levels of PDL-1 expression to benefit from treatment and if you recall, the OS curve from O57, the shapes of the Kaplan Meier curves were similar across the 1%, 5% and 10% cut-offs. . . .
>
> So let me say again, and I want to reiterate, we have not changed our analysis at all. The PFS analysis will be on the hazard ratio of the PFS curves in the strongly expressing population, as we've said before and of course this takes into account the totality of the Kaplan Meier curve. If the primary endpoint of PFS is strongly – ***is met in the strongly expressing patients***, we'll also look at all the randomized population, which includes all those that express at the greater than 1% level. That hasn't changed either. . . .
>
> Let me just reiterate, ***we are very comfortable about the design of our trial. We are very comfortable about the depth of understanding that went into to the statistical plans***. . . .

194. Defendants Caforio's and Cuss's statements described above in ¶193 were materially false and misleading because both Defendants knew or recklessly disregarded from the first day of the Class Period that Checkmate-026 focused on patients with PD-L1 expression greater than or equal to just 5%—a weak level of expression that most commonly defined the bare minimum for PD-L1 positive expression at all. (*E.g.*, ¶¶53, 87-90.) Moreover, the low expression cutoff chosen also created an undisclosed risk that the study would not be powered to detect statistically meaningful results when stratified at higher levels of expression.

195. Accordingly, Defendants Caforio and Cuss knew or was reckless in not knowing that the deceptive statements on *ClinicalTrials.gov*, which had been updated as recently as April 25, 2016, had misled investors as to the true risk inherent in Checkmate-026's—that the clinical trial targeted the majority (55%) of the NSCLC population with weak 5% levels of PD-L1 expression for whom the data indicated Opdivo was much less likely to be effective—and Defendants had a duty to correct those statements by providing the full, highly material truth that the study was primarily focused on patients with a weak 5% PD-L1 tumor expression, but did not do so.

196. While Defendant Cuss did acknowledge for the first time that 50% was not the PD-L1 expression cutoff in Checkmate-026, Cuss's statements were false and misleading because he nonetheless: a) reaffirmed that Checkmate-026 was still "looking at [] strongly expressing patients;" b) contemporaneously refused to define the level of PD-L1 expression; and c) failed to inform investors that Checkmate-026 in fact was focused on those patients with a weak 5% expression. Accordingly, Cuss continued to mislead investors as to the true risk inherent in Checkmate-026's primary focus, by falsely and misleadingly stating that the clinical trial targeted "strong" PD-L1 expressors rather than the weak, 5% PD-L1 expressors for whom the data

indicated Opdivo was much less likely to be effective, as well as the additional risk that the study would not be powered to detect statistically meaningful results when stratified at higher levels of expression.

197. Moreover, Defendant Cuss's assertion of "confiden[ce]" in Checkmate-026 was further false and misleading because it misled investors about the true risk inherent in Checkmate-026's primary focus—that the clinical trial targeted the majority (55%) of the NSCLC population with weak 5% levels of PD-L1 expression for whom the data indicated Opdivo was much less likely to be effective—and the additional risk that the study would not be powered to detect statistically meaningful results when stratified at higher levels of expression.

8. The June 4, 2016 2016 ASCO Conference

198. On June 4, 2016, Defendants Namouni and Caforio participated in the 2016 ASCO Conference, during which Defendant Namouni responded to an analyst question by confirming that Defendants had "not disclosed what the [Checkmate-026] cutoff is, but we have disclosed that it's lower than 50%."

199. Defendant Namouni's statement described above in ¶198 was materially false and misleading because Namouni knew or recklessly disregarded from the first day of the Class Period that Checkmate-026 focused on patients with PD-L1 expression greater than or equal to just 5%—a weak level of expression that most commonly defined the bare minimum for PD-L1 positive expression at all. (*E.g.*, ¶¶53, 58, 87-88.) Moreover, the low expression cutoff chosen also created an undisclosed risk that the study would not be powered to detect statistically meaningful results when stratified at higher levels of expression.

200. Accordingly, Defendant Namouni knew or recklessly disregarded that the deceptive statements on *ClinicalTrials.gov*, which had been updated as recently as May 25, 2016,

had misled investors as to the true risk inherent in Checkmate-026's—that the clinical trial targeted the majority (55%) of the NSCLC population with weak 5% levels of PD-L1 expression for whom the data indicated Opdivo was much less likely to be effective—and Defendants had a duty to correct those statements by providing the full, highly material truth that the study was primarily focused on patients with a weak 5% PD-L1 tumor expression, but did not do so.

201. Further, Defendant Caforio participated in the June 4, 2016 conference call and, as Bristol-Myers' Chief Operating Officer, also had knowledge concerning the truth about Checkmate-026. (*E.g.*, ¶¶87-88.) Thus, Caforio understood that Namouni's statement described in ¶198 was false and misleading for the reasons described above. Accordingly, Caforio had a duty to correct Namouni's statements by providing the full truth about the true risk inherent in Checkmate-026 to investors, but did not do so.

9. The July 28, 2016 Conference Call

202. On July 28, 2016, Defendant Cuss and Caforio participated in Bristol-Myers' earnings call, during which they made the following statements:

> [Defendant Cuss]: ***[A]s I have noted in the past and will note again today, I'm confident in the study design of 026. . . .*** I will say again, we're not going to talk about the ***high*** [expressive] cutoff at this point. But you, obviously, will have an opportunity to see that when we talk about the data. . . .
>
> [Defendant Caforio]: ***[W]e are very confident in . . . the design of the clinical trial***.

203. Defendant Cuss's and Caforio's statements described above in ¶202 were materially false and misleading because both knew or recklessly disregarded from the first day of the Class Period that Checkmate-026 focused on patients with PD-L1 expression greater than or equal to just 5%—a weak level of expression that most commonly defined the bare minimum for PD-L1 positive expression at all. (*E.g.*, ¶¶53, 58, 87-88.) Moreover, the low expression cutoff

chosen also created an undisclosed risk that the study would not be powered to detect statistically meaningful results when stratified at higher levels of expression.

204. Accordingly, Defendants Cuss and Caforio knew or should have known that the deceptive statements on *ClinicalTrials.gov*, which had been updated as recently as July 26, 2016, had misled investors as to the true risk inherent in Checkmate-026—that the clinical trial targeted the majority (55%) of the NSCLC population with weak 5% levels of PD-L1 expression for whom the data indicated Opdivo was much less likely to be effective—and Defendants had a duty to correct those statements by providing the full, highly material truth that the study was primarily focused on patients with a weak 5% PD-L1 tumor expression, but did not do so.

205. Moreover, Defendant Cuss's statement regarding the "high" expressor cutoff and Defendant Cuss's and Caforio's assertions of Defendants' "confidence" in Checkmate-026 were false and misleading because they misled investors about the true risk inherent in Checkmate-026's primary focus—that the clinical trial targeted the majority (55%) of the NSCLC population with weak 5% levels of PD-L1 expression for whom the data indicated Opdivo was much less likely to be effective, and the additional risk that the study would not be powered to detect statistically meaningful results when stratified at higher levels of expression.

VIII. LOSS CAUSATION

206. Defendants' wrongful conduct, as alleged herein, directly and proximately caused Plaintiffs and the Class to suffer substantial losses.

207. During the Class Period, Plaintiffs and the Class purchased or otherwise acquired Bristol-Myers securities at artificially inflated prices, and were damaged thereby when the truth was revealed and when the risks Defendants concealed with their false and misleading statements materialized. The price of Bristol-Myers securities declined significantly (causing investors to suffer losses) when the Defendants' misrepresentations, and/or the information alleged herein to

have been concealed from the market, and/or the effects thereof, were revealed, and/or the foreseeable risks that had been fraudulently concealed by the Defendants materialized.

208. Defendants' materially false and misleading statements and omissions misrepresented, among other things described above, that Bristol-Myers' closely-watched first-line monotherapy clinical trial of its lead immuno-oncology drug Opdivo had as its focus in both Primary Outcome and Secondary Outcomes the efficacy of Opdivo in treating those NSCLC patients who exhibited "strong" or "high positive" expression of PD-L1 in their cancerous cells. These statements represented to investors that Checkmate-026 was a conservative, well-designed study with a high likelihood of success, which stood to generate tens of billions of dollars in additional new revenue for Bristol-Myers. When these statements were corrected and the risks concealed by them materialized, investors suffered losses as the price of Bristol-Myers securities declined. As a result of the disclosure of the truth of Defendants' fraud, Bristol-Myers' common stock price suffered statistically significant declines, and ultimately fell approximately 34%, from a closing price of $75.32 at the close of August 4, 2015, to just $49.81 at the close of October 10, 2016.

209. Specifically, a disclosure on August 5, 2016 partially revealed that the Defendants' materially false and misleading statements misrepresented, among other things, the risk in their design of Checkmate-026. Those statements began to be corrected, and the risks concealed by them began to materialize, on August 5, 2016, when Defendants revealed that Checkmate-026's purportedly "strong" PD-L1 expression cutoff was only 5%, and that this nominal cutoff caused Checkmate-026 to fail to meet its primary endpoint. This failure caused investors to suffer losses as the price of Bristol-Myers securities declined $12.04 per share, from $75.32 at the close of

August 4, 2016 to $63.28 at the close of August 5, 2016—a statistically significant one-day decline of 16%. For purposes of comparison, the S&P 500 Index increased by 1% at that time.

210. On October 9, 2016, the truth was fully revealed, and the concealed risks fully materialized, when Defendants disclosed that the full data for Checkmate-026 showed no discernible benefit for Opdivo monotherapy on any level, including for those exhibiting strong 50% PD-L1 expression, causing investors to suffer losses as the price of Bristol-Myers securities declined $5.62 per share, from $55.43 at the close on October 7, 2016 to $49.81 (the prior trading day) at the close on October 10, 2016—a statistically significant one-day decline of 10%. For purposes of comparison, the S&P 500 Index increased a marginal amount (0.5%) during that time.

211. Accordingly, as a result of their purchases or acquisitions of Bristol-Myers' publicly traded common stock during the Class Period, Plaintiffs and other members of the Class suffered economic loss and damages.

IX. PRESUMPTION OF RELIANCE

212. At all relevant times, the market for Bristol-Myers common stock was an efficient market for the following reasons, among others:

a) Bristol-Myers common stock met the requirements for listing, and was listed and actively traded on NYSE, a highly efficient and automated market;

b) Bristol-Myers securities were actively traded throughout the Class Period, with substantial trading volume, average weekly turnover, and institutional investor population. The average weekly trading volume for Bristol-Myers common stock during the Class Period was over 35 million shares, and the average weekly turnover was 2.15%;

c) As a regulated issuer, Bristol-Myers filed periodic public reports with the SEC;

d) Bristol-Myers was eligible to file registration statements with the SEC on Form S-3;

e) Defendants regularly communicated with public investors by means of established market communication mechanisms, including through regular dissemination of press releases on the major news wire services and through other wide-ranging public disclosures, such as communications with the financial press, securities analysts, and other similar reporting services;

f) The market reacted promptly to public information disseminated by Bristol-Myers; and

g) Bristol-Myers common stock was covered by numerous securities analysts employed by major brokerage firms, including Sanford C. Bernstein; Cowen & Company; BMO Capital Markets; J.P. Morgan; Guggenheim; Evercore ISI Research; Leerink Partners; SunTrust Robinson Humphrey; Credit Suisse; Barclays; Societe Generale; Morgan Stanley; William Blair; Hilliard Lyons; and UBS. Each of these reports was publicly available and entered the public marketplace.

213. Accordingly, the market for Bristol-Myers securities promptly digested current information with respect to Bristol-Myers from all publicly-available sources and reflected such information in the prices of those securities. Under these circumstances, all purchasers of the Company's publicly traded common stock during the Class Period suffered similar injury through their purchases at artificially inflated prices, and a presumption of reliance applies.

214. Plaintiffs are also entitled to a presumption of reliance under *Affiliated Ute Citizens of Utah v. United States*, 406 U.S. 128 (1972), because the claims asserted herein against Bristol-Myers are predicated upon omissions of material fact that there was a duty to disclose. Specifically, particularly given the enormous importance of Checkmate-026, reasonable investors would have considered—and did consider—important in making investment decisions the fact that the purportedly "strong" or "high positive" expression level cutoff for the clinical trial endpoint was actually just 5%, a fact that had a material impact on the risks that the clinical trial would fail and/or provide meaningful data.

X. INAPPLICABILITY OF STATUTORY SAFE HARBOR

215. The statutory safe harbor applicable to forward-looking statements under certain circumstances does not apply to any of the false or misleading statements pleaded in this Complaint. The statements complained of herein were historical statements or statements of current facts and conditions at the time the statements were made. Further, to the extent that any of the false or misleading statements alleged herein can be construed as forward-looking, the statements were not accompanied by any meaningful cautionary language identifying important facts that could cause actual results to differ materially from those in the statements.

216. Alternatively, to the extent the statutory safe harbor otherwise would apply to any statements pleaded herein, Defendants are liable for those false and misleading forward-looking statements because at the time each of those statements was made, the speakers knew the statement was false or misleading, or the statement was authorized or approved by an executive officer of Bristol-Myers who knew that the statement was materially false or misleading when made. Such a statement cannot be protected under the PSLRA safe harbor.

XI. SUMMARY OF SCIENTER ALLEGATIONS

217. As alleged herein, numerous facts give rise to the strong inference that, throughout the Class Period, Defendants knew or recklessly disregarded that their statements and omissions, as set forth in Section VII, were materially false and misleading when made. The information in this section is a summary of certain of the allegations detailing Defendants' scienter that are set forth more fully above. All allegations must be considered holistically in evaluating Defendants' scienter. The cumulative knowledge of all members of the Company's senior management team, including the Individual Defendants, regarding the matters addressed herein is properly imputed to Bristol-Myers.

A. Defendants Have Admitted That Checkmate-026 Was Not Designed To Target "High" PD-L1 Expressors, Contrary To Prior Representations.

218. Defendants' scienter is supported by Defendant Namouni's remarkable admission at the Credit Suisse Health Care Conference on November 8, 2016, when he stated:

> I think the major driver [in Checkmate-026's failure] is really . . . it was focused on a larger population of biomarker. Obviously, ***the study was not designed to look at the smaller subgroup of highly inflamed tumor or high expression of PD-L1.*** Basically, that's the reason we are having a study that did not meet its primary endpoint.

In other words, Defendant Namouni *admitted* that Bristol-Myers knew that it did not design Checkmate-026 to focus on tumors exhibiting a strong, or "high" expression of PD-L1, despite Defendants claiming otherwise throughout the Class Period.

219. Defendants made similar admissions in the wake of the August 5, 2016 and October 9, 2016 disclosures (¶¶135-36, 142), and new Chief Scientific Officer Lynch, who was a member of the Bristol-Myers Board of Directors during the Class Period, also admitted that the Company's choice of a "lower cutoff" was the cause of Checkmate-026's failure (¶155).

B. Bristol-Myers And The Individual Defendants Repeatedly Defined 5% Expression As A Marker Of Minimal PD-L1 Positivity In Checkmate Studies Prior To And During The Class Period.

220. The Individual Defendants' scienter is further supported by the fact that their public statements were directly contradicted by the Company's own use of 5% expression to denote weak or minimal PD-L1 expression.

221. The allegations made herein clearly establish that (a) prior to and during the Class Period, Bristol-Myers repeatedly and publicly defined 5% PD-L1 expression as the minimal indication of positivity (¶¶54-61, 120); (b) Bristol-Myers and the Individual Defendants made an intentional decision to change the Company's historical conventions and define 5% PD-L1 expression as "strong" expression in their public statements concerning Checkmate-026 (¶¶71-72,

87-90); and (c) Bristol-Myers and the Individual Defendants actively and repeatedly concealed from the market the actual expression used in Checkmate-026 until the August 5, 2016 disclosure (¶¶99-115).

222. Defendants' own, repeated usage of 5% expression as the lowest level of mere PD-L1 positivity rendered their secret characterization of 5% expression as "strong" or "high" with respect to the primary focus of Checkmate-026 knowingly false and/or misleading, especially when Bristol-Myers' Checkmate-026 study was in a direct race with Merck's Keynote-024 study, where "strong" expression denoted a 50% expression threshold. Defendants misled the market by secretly changing, in Checkmate-026, the definition of 5% PD-L1 expression from weak or minimal to "strong" (*e.g.*, ¶¶62-72, 121) without any disclosure or acknowledgment of this significant change to, as FE-5 stated, "one of the most important parameters in patient selection" in clinical studies of PD-1 checkpoint inhibitors (¶90).

C. Individual Defendants Giordano, Namouni, Cuss, And Caforio Were All Involved In The Design And Approval Of Checkmate-026's Focus On 5% PD-L1 Expression And Its Characterization as "Strong".

223. The Individual Defendants' scienter is further supported by the fact that, as senior executives of the Company, the Individual Defendants had direct involvement in the design and oversight of the Company's clinical trials. Indeed, certain Individual Defendants explicitly described their involvement in Checkmate-026 in particular:

Defendant Giordano, Head of Bristol-Myers' Oncology Department:

> "***We've designed*** that study [Checkmate-026] in order to get speed to market and be able to treat patients in the first-line setting." (September 8, 2015.)
>
> "[W]e are very confident that that study [Checkmate-026] will be a positive study . . . ***We have chosen a PFS endpoint*** [for the clinical trial] because of potential cross-over. . . ." (October 1, 2015.)

Defendant Cuss, Bristol-Myers' Chief Scientific Officer:

> "[A]s far as 026 is concerned, ***we have actually taken great care in the design of this study. We have paid real attention*** to the choice of end point, the optimal timing of the analysis, the role of the non-proportional hazard ratio, and ***of course, the role of PD-L1 expression and the sample size.***" (January 28, 2016.)
>
> "[L]et me emphasize, we've really taken great care in the design of study 026 ***in its choice of its endpoint***, the optimal timing of the analysis, which I just mentioned, the role of non-proportional hazard ratio, ***the role of PDL-1 expression***, sample size. And we've used the results of many of our published and unpublished data to look at this and essentially we remain very confident. ***We think we've got the optimum balance of speeds and design*** . . . . Let me just reiterate, ***we are very comfortable about the design of our trial. We are very comfortable about the depth of understanding that went into to the statistical plans***. . . ." (April 28, 2016.)

Defendant Namouni, Bristol-Myers' Head of Oncology Development and Development Lead for Opdivo:

> "CheckMate-026. . . ***was well-designed***, and this study ***represents all the knowledge in terms of design that was really made at Bristol-Myers Squibb from our understanding***, from a number of positive Phase 3s looking at different endpoints, and ***this helps us to design these type of studies***. . . ***[T]he study has been well-designed*** and well-powered to detect the difference in all the positives but also ***in what we define [as] the high positives***. . . ." (March 7, 2016.)

224. Likewise, in the Company's Form 10-K for fiscal years 2013 and 2014—filed February 14, 2014 and February 13, 2014, respectively—Defendants Andreotti, Bancroft, and (for 2014 only) Caforio noted that they "were focusing our efforts and resources in certain disease areas . . . . In particular, Opdivo[.]"

225. Moreover, former employees directly confirm the personal involvement of the Individual Defendants in the design and oversight of Checkmate-026, and the decision to use a 5% cutoff, falsely described as "strong" and "high." *See* ¶¶86-90.

226. The Individual Defendants' personal involvement in the design and oversight of Checkmate-026 establishes their personal knowledge concerning the PD-L1 expression cutoff and

the concealed risk inherent in the clinical trial—that the clinical trial targeted the majority (55%) of the NSCLC population with weak 5% levels of PD-L1 expression for whom the data indicated Opdivo was much less likely to be effective.

D. The Individual Defendants Consistently Spoke To Investors About Checkmate-026 And PD-L1 Expression, And Held Themselves Out As Knowledgeable About These Topics.

227. The Individual Defendants' scienter is further supported by the fact that they consistently spoke to investors about Checkmate-026 and the science underlying the design of the clinical trial, including specifically the role of PD-L1 expression in Opdivo's efficacy.

228. For example, during Bristol-Myers' first earnings call discussing Checkmate-026 after their first *ClinicalTrials.gov* disclosures, Defendant Andreotti emphasized that "PD-1 is a ***cornerstone for immunotherapy*** and we are very committed to it." On April 29, 2014, Andreotti similarly stated that Defendants "remain as confident as we've been in the potential for immuno-oncology, as evidenced by the unique scale of our program and its continued expansion." Then, in announcing Bristol-Myers' collaboration with Ono Pharmaceutical Co., Ltd (¶¶30, 39), Andreotti stated on July 23, 2014 that Defendants' "goal [is] to maximize the full potential of our immuno-oncology portfolio for patients worldwide." Later, in Bristol-Myers' March 3, 2015 press release announcing that Opdivo had received FDA approval for second-line NSCLC treatment, Andreotti stated, "Bristol-Myers Squibb is committed to patients with lung cancer . . . . Because lung cancer is one of the most commonly diagnosed cancers in the United States, with high mortality, there is a significant need for treatments that extend survival." As another example, Defendant Namouni stated during the 2014 ASCO conference that "the basis for" Checkmate-026 was "the potential of PD-L1 expression in the tumor to predict the activity of nivolumab in first line lung cancer." Likewise, in September 2015, Defendants Caforio, Bancroft, Giordano, and Namouni, as well as Bristol-Myers Head of Research Carl DeCicco, told analysts from Evercore

ISI that they "believe[] the data for Opdivo in PDL1+ tumors is strong, ***[have] confidence in the design of -026*** and [are] ***more than optimistic*** about the results." Similarly, on October 1, 2015, Giordano stated that "the 026 study, which was fully enrolled and may mature, will mature next year . . . . has as an endpoint PFS. The data from 012 and others which looks at PFS suggests that ***we are very confident that that study will be a positive study***; in particular the characterization of the survival. We have chosen a PFS endpoint because of potential cross-over[.]" Finally, during Bristol-Myers' January 28, 2016 earnings call, Defendant Cuss stated, "[A]s far as 026 is concerned, ***we have actually taken great care in the design of this study. We have paid real attention*** to the choice of end point, the optimal timing of the analysis, the role of the non-proportional hazard ratio, and ***of course, the role of PD-L1 expression and the sample size.***"

229. Moreover, before and during the Class Period, the Individual Defendants were increasingly called upon to comment on Checkmate-026 and specifically on the PD-L1 expression cutoff used in the clinical trial. For example, on January 7, 2014—before Checkmate-026 had even been publicly revealed—Andreotti was asked, with respect to PD-1, "how do we think about how this is going to play out[?]," to which he responded, "[F]irst of all, think of the evolving knowledge we have in monotherapy because I think that ***the data in lung for PD-1 alone*** was not bad. I mean ***at least it was beyond my expectations***." Then, on April 28, 2016, Defendants Caforio and Cuss had the following exchange with an analyst:

> [Analyst]: ***Obviously investors are very focused on the upcoming front-line long studies from both Merck and you guys.*** Can you compare and contrast the first – the two first line monotherapy trials of Opdivo versus Keytruda, since that's obviously what we're focused on? . . . .
>
> [Defendant Caforio]: We have, I am convinced, the right approach of investigating monotherapy, both in a broader population of PD-1-positive patients and ***in a subset of patients that express PD-1 at a higher level***. . . .
>
> [Defendant Cuss]: [L]et me emphasize, we've really taken great care in the design of study 026 ***in its choice of its endpoint***, the optimal timing of the analysis, which

> I just mentioned, the role of non-proportional hazard ratio, ***the role of PDL-1 expression***, sample size. And we've used the results of many of our published and unpublished data to look at this and essentially we remain very confident. . . .
>
> Just to be very specific, in the trial design we're looking at both ***strongly expressing patients*** and patients with any level of expression. . . .
>
> Let me just reiterate, ***we are very comfortable about the design of our trial. We are very comfortable about the depth of understanding that went into to the statistical plans***. . . .

230. Thus, the Individual Defendants were aware of and sensitive to the highly material nature of this information, and held themselves out as knowledgeable about this topic. Therefore, investors reasonably expected them to have knowledge about the truth or falsity of their statements.

E. Describing Checkmate-026's PD-L1 Expression Cutoff As "Strong" And/Or "High" Plainly Contradicted Industry Convention.

231. Defendants' scienter is further supported by the fact that their description—and refusal to correct the description—of Checkmate-026's 5% PD-L1 expression cutoff as "strongly PD-L1+ tumor expression," "high positive," or "high expression" so plainly contradicted the ordinary understanding of industry participants and observers that Defendants knew, or were reckless in not knowing, that their statements were manifestly unreasonable and misleading.

232. As an initial matter, Defendants were keenly aware of—and indeed helped define—industry consensus. First, at all relevant times the industry for PD-1 checkpoint inhibitors was new, rapidly evolving, and largely dominated by a handful of companies, including most prominently Bristol-Myers and Merck. For example, in a June 1, 2012 article, the *New York Times* principally described the results of research by Bristol-Myers into PD-1 checkpoint inhibitors, noting as other competitors only Merck, Roche, GlaxoSmithKline, and Teva. Similarly, in a June 2, 2015 article addressing the growing industry for PD-1 checkpoint inhibitors, Reuters specifically named just Bristol-Myers and Merck as the "[d]rugmakers . . . testing which patients will most benefit from

new cancer treatments based on a protein found in their tumors," before noting that "[d]rug companies are working to refine and standardize their PD-L1 testing."

233. Second, during the Class Period, Defendants directly communicated to investors they were taking the lead on harmonizing the industrywide approach to measuring PD-L1 expression. For example, at ASCO 2015 on June 1, 2015—the same day and event that Merck implicitly confirmed that, consistent with the usage by Merck and other academics for months, the "strong" expression in Keynote-24's referred to PD-L1 expression levels of 50% or greater—an analyst asked Defendant Giordano about the discussion around "trying to harmonize how [PD-L1] is done across companies" and "how you're thinking about that approach and working with the other sponsors of these PD-L1 assets to have something that's more useful for clinicians?" In response, Defendant Giordano stated, "[A]bout harmonization of PD-L1 assays . . . there are ***ongoing approaches to harmonizing the assays***, and Bristol-Myers Squibb, as a leader in the field and committed to the patient's benefit, will participate in the efforts to harmonize PD-L1 expression assays to enable helping the most patients."

234. Finally, FE-2 specifically described how Bristol-Myers maintained competitive intelligence teams that were incredibly important to the Company's work. (¶72.) FE-2 explained that, using competitor data, the Company would typically run 40-100 scenarios so Bristol-Myers' executive team could know what the competitors were doing, what Bristol-Myers was poised to do, and how those two situations would interact under various conditions. FE-2 specifically stated that Bristol-Myers would have immediately incorporated any information learned about Merck's PD-L1 threshold into new forecasting to understand Merck's position in the market and to compare to Bristol-Myers' efforts. FE-2 stated that this crucial information and resulting forecast updates would have gone to Defendant Namouni. FE-1 similarly described discussing Merck's 50% cutoff

with certain Individual Defendants (Namouni and Giordano) as part of strategy meetings before the start of the Class Period. (¶71.)

235. Accordingly, the Individual Defendants knew or were extremely reckless in not knowing of the industrywide consensus that "strong" PD-L1 expression was inconsistent with a 5% PD-L1 expression level.

1. Industry consensus was that 5% PD-L1 expression was inconsistent with "strong" expression because 5% was the most common threshold for mere positivity.

236. It was industry consensus that a 5% PD-L1 expression level was the most common ***minimum*** to establish PD-L1 positivity. This is facially inconsistent with the idea that a 5% expression was "strong." For example, an article published on March 28, 2012 in the medical journal Science Translation Medicine noted that "a 5% expression threshold was chosen to define [PD-L1] positivity, in keeping with previous studies," citing studies from 2006 and 2007. Likewise, in an article published on May 26, 2016, researchers looked at data going back to 2002 and concluded that "***[s]ince the first published data, the threshold most often chosen [for positivity] is >5% expression***."

237. Bristol-Myers' Checkmate studies established that 5% expression was the most common minimum expression for PD-L1 positivity in lung cancer, as demonstrated by a chart presented in *Journal of Thoracic Oncology* in July 2015 summarizing PD-L1 findings in Bristol-Myers and other NSCLC trials:

TABLE 1. Summary of Published Findings for PD-L1 Immunohistochemistry in Therapeutic Trials

Drug	Biomarker Antibody	Rx Line	Definition of "Positive"[a] (%)	N Positive (%)	Positive Predictive Outcome	ORR % IHC pos. Cases	ORR % IHC neg. Cases	Ref.
Nivolumab	Dako 28-8	1st	≥5 in >100 cells	59	Yes	31[b]	10	7,8[f]
Nivolumab	Dako 28-8	≥2nd	≥5, ≥1	49, 56	No	15, 13	14, 17	9,10
Nivolumab + Ipilimumab	Dako 28-8	1st	≥5 in >100 cells	42	No	19	14	11
Nivolumab	Dako 28-8	≥2nd	≥5	33[c]	Yes	24	14	12[f]
Nivolumab	5H1[d]	≥2nd	≥5, also studied TIICs	67	Yes	No data for lung	No data for lung	13
Pembrolizumab	Dako 22C3	Any	"Strong" ≥50, "Weak" 1–49	25, 70	Yes, Yes	37, 17	9	14
Pembrolizumab	Dako 22C3	1st	≥50, ≥1	?	Yes	47, 26	?	15
MPDL3280A	Roche Ventana, SP142	≥2nd	≥10,[e] ≥5, ≥1 TIICs	13, 28, 56	Yes	83, 46, 31	18, 18, 20	16–18
MEDI-4736	Roche Ventana, SP263	≥2nd	Data not available	41	Yes	25	3	19,20

[a]Expression in tumor cells unless otherwise stated.
[b]The 31% figure is for all tumors. The ORR was 37% in nonsquamous tumors and 12% in squamous cases. In PDL-1 negative cases, ORR was 14% in nonsquamous tumors and 0% in squamous tumors.
[c]This study concerned squamous cell carcinomas only.
[d]These authors also used the anti-PD-1 monoclonal M3 in their immunohistochemical analysis.
[e]IHC score 3, ≥10% TIICs positive; IHC score 2–3, ≥5% TIICs positive; IHC score 1–2–3, ≥1% TIICs positive.
[f]ORR quoted are those actually presented, as opposed to those published in the abstract
IHC, immunohistochemistry; TIICs, tumor infiltrating immune cells; ORR, overall response rate (response evaluation criteria in solid tumors).

Keith M. Kerr et al., "Programmed Death-Ligand 1 Immunohistochemistry in Lung Cancer," *Journal of Thoracic Oncology*, 2015;10: 985-989, at 986 (July 2015).

238. Defendants knew of the industry convention that 5% was the minimum for mere positivity—and therefore facially inconsistent with 5% being a "strong" expression—because Defendants had themselves participated in establishing that industry consensus, including specifically with respect to NSCLC. As the chart above and the discussion in ¶¶54-61, 120 makes clear, the industry addressing PD-L1 expression in treating NSCLC was very limited, involving just a handful of companies, of which Bristol-Myers dominated. ***In so doing, Defendants themselves had frequently used a 5% expression to define PD-L1 positivity***.

239. For example, on June 2, 2014, Defendants Giordano and Namouni presented the results of the clinical trials Checkmate-012 (studying nivolumab against NSCLC in patients with no history of chemotherapy, including as a monotherapy and in combination with other drugs) and Checkmate-003 (studying nivolumab as a monotherapy against NSCLC in patients with a history of chemotherapy), which utilized a 5% expression threshold for mere PD-L1 positivity. During this presentation, Namouni explained that detecting "good expression" was critical because "PDL-

1 is a very ubiquitous protein," and specifically referred to the 5% "cutoff that was used to this analysis" in discussing the "durable" and "interesting" responses from the studies.

240. Likewise, during the Bank of America Merrill Lynch Global Healthcare Conference on September 18, 2014, Bristol-Myers Senior Vice President John Elicker stated that Bristol-Myers was calibrating the "percent of patients with PDL1 expression in the lung cancer studies . . . we expect" "based on the data we have seen, generally speaking at a 5% cutoff."

241. Defendants continued into the Class Period to use the industry-standard 5% as the bare minimum of PD-L1 positivity. For example, in an article published in March 2015 in *Lancet Oncology* discussing Bristol-Myers's Checkmate-063 study, the clinical doctors specifically explained that they "used a cutoff of 5% to define PD-L1 positivity[] on the basis of preliminary findings" in Bristol-Myers's earlier studies. Similarly, on April 20, 2015, Defendants announced the results of Checkmate-069, which also used a 5% cutoff for mere positivity.

242. The market took note of this industry consensus and specifically of Defendants' repeated use of 5% expression as a baseline for PD-L1 positivity. For example, in a January 23, 2014 report that discussed Checkmate-026, a Leerink analyst also noted prior data from Bristol-Myers itself that "defined" "PD-L1 expressing tumors" as 5% expression.

243. Accordingly, using the word "strong" or "high" to describe an expression level that Defendants knew or should have known was frequently ***the bare minimum*** for PD-L1 expression—and which Defendants themselves had helped define as the bare minimum for PD-L1 expression—was manifestly unreasonable, and could only have been done deliberately or with reckless disregard.

2. To the extent "strong" PD-L1 expression was discussed, industry consensus was that "strong" was well above 5%.

244. Defendants' use of the word "strong" or "high" to describe the 5% PD-L1 expression cutoff in Checkmate-026 contradicted the industry's descriptions of the relative 'strength' of PD-L1 expressions. For example, an article published in *Science Translational Medicine* on March 28, 2012 used a 5% expression threshold to define positivity "in keeping with previous studies," while defining a "***high-aggregate***" PD-L1 expression as tumors demonstrating at least 20% expression.

245. By the start of and continuing throughout the Class Period, Merck—one of the most prominent players in the industry, and Bristol-Myers' primary competitor—had prominently defined "strong" PD-L1 expression to mean PD-L1 expression levels of 50% or greater. For example, on April 6, 2014, Merck issued a press release and presented on "early findings from studies exploring the relationship between tumor PD-L1 expression and clinical outcomes following monotherapy treatment with MK-3475 [Keytruda], an investigational anti-PD-1 immunotherapy, in patients with advanced melanoma and advanced non-small cell lung cancer (NSCLC)." Merck concluded that "the preliminary analysis suggests that the optimal cut-point is ≥50 percent of tumor cells" and that "[w]hen using this measurement, approximately 25 percent of advanced NSCLC patients had tumors that ***strongly expressed PD-L1***." Similarly, in the materials for Merck's June 2, 2014 presentation at annual industry convention ASCO 2014, Merck explicitly defined "strong" PD-L1 as ≥50%, with "weak" as 1-49%. During that presentation, Dr. Tanguy Seiwert, Associate Professor of Medicine for the University of Chicago, described PD-L1 expressions greater than 50% as "strongly and diffusively positive." Perhaps most notably, Merck defined 50% PD-L1 expression as "strong" for Keynote-024, its parallel monotherapy trial.

246. The industry at large adopted Merck's presentation of "strong" PD-L1 expression. For example, the European Society for Medical Oncology's 2014 Congress Meeting Report described Merck's Keynote-001 test as concerning "patients with strong PD-L1 expression (≥50% staining) [versus] in patients with weak/negative PD-L1 expression" and "patients with PD-L1 strong-positive vs. PD-L1 weak-positive/negative tumours," and further referenced Merck's ongoing Keynote-024 study. Then, during the Class Period, an article published in the peer-reviewed scientific journal *PLOS One* on March 16, 2015 noted that "[i]n NSCLC immune and tumor cells . . . a strong expression (staining ≥50%) derived greater clinical benefit than patients with weak or negative score PD-L1 expression." Similarly, in a presentation on October 8, 2015, competitor Nektar spoke about Keytruda's application for the "subset of high PD-L1 expression of more than 50%."

247. To the extent the industry spoke of "low" PD-L1 expression, it frequently referred to 5% PD-L1 expression levels. For example, during the Class Period, the industry publication *Journal of Thoracic Oncology* had described PD-L1 expressions of "1% ***or even 5%***" as "***very low staining thresholds***" that pose "a greater risk that scoring will be inconsistent and is more likely to reflect inaccurately the patient's tumor burden overall," Keith M. Kerr et al., "Programmed Death-Ligand 1 Immunohistochemistry in Lung Cancer," *Journal of Thoracic Oncology*, 2015;10: 985-989, at 986 (July 2015). The article also explicitly referred to PD-L1 expressions of 1-49% as "weak," and above 50% as "strong." *Id.* Likewise, a scholarly article published on March 3, 2016 titled "PD-L1 Expression and Survival Among Patients With Advanced Non-Small Cell Lung Cancer Treated With Chemotherapy" determined that:

> Using the clinical trial assay to identify levels of PD-L1 expression that maximally predict clinical response to pembrolizumab, ***PD-L1 weak positive*** is defined as membranous PD-L1 expression in 1% to 49% of tumor cells, and ***PD-L1 strong positive*** is defined as expression in ≥ 50% of tumor cells.

248. As purported experts in the field of immuno-oncology, Defendants knew or should have known of these descriptions of PD-L1 expression. For example, Defendants themselves also attended and presented at ASCO 2014—in which Merck's presentation expressly described "strong" PD-L1 expression as referring to greater than 50%—where Defendants stated that "the potential of ***PD-L1 expression*** in the tumor to predict the activity of nivolumab [Opdivo] in first line lung cancer" was "***the basis for***" Checkmate-026. Indeed, Bristol-Myers' Senior Vice President for Global Commercialization for Oncology at the time the Checkmate-026 trial was announced—FE-1—explained that s/he participated in strategy committee meetings that s/he co-chaired with Defendant Giordano, and in which Defendant Namouni also participated, during which there were discussions about what level of PD-L1 expression was "good" and about Merck's use of a 50% PD-L1 expression level. (¶71.)

249. Ultimately, in revealing the full results of Checkmate-026 on October 9, 2016, Bristol-Myers Vice President Nick Botwood admitted that 5% was not strong when he described Checkmate-026's 5% PD-L1 expression cutoff as ***not*** "***high levels of PD-1.***" Likewise, Defendant Namouni himself admitted in November 2016 that "[Checkmate-026] was not designed to look at the smaller subgroup of . . . ***high expression*** of PD-L1."

250. Accordingly, Defendants knew or should have known that their use of the word "strong" or "high" to describe a 5% expression level—which Defendants themselves had helped defined as the most common ***bare minimum*** for PD-L1 expression—was manifestly unreasonable, and could only have been done deliberately or with reckless disregard.

F. The Critical Importance Of The Level Of PD-L1 Expression.

251. Further supporting scienter is the critical importance of the information that Defendants misrepresented. As early as 2012, the market understood that research indicated that PD-L1 expression had an important relationship with the efficacy of PD-1 checkpoint inhibitors

such as Opdivo, when the *New York Times* wrote that "preliminary evidence [indicated] that PD-1 blockers will ***not work in people whose tumors do not make PD-L1***." Indeed, Defendant Namouni even stated during the 2014 ASCO conference that "***the basis for***" Checkmate-026 was Defendants' own "data generated with monotherapy" that showed "***the potential of PD-L1 expression in the tumor to predict the activity of nivolumab in first line lung cancer***." Similarly, during the 2015 ASCO annual conference, Defendant Giordano stated that "[t]he role of PD-L1 expression has one which Bristol-Myers Squibb has articulated a strategy" that "***relate[d] that level of expression to meaningful endpoints***."

252. Thus, the level of the PD-L1 expression cutoff in Checkmate-026 was critical to understanding the clinical trial's odds of success. As an analyst from Cowen noted in its August 5, 2016, report, it had noted on April 1, 2016 that the PD-L1 expression cutoff determined whether Checkmate-026 was a "certain" success or an almost-guaranteed failure:

> ***[A]t a 5% PD-L1 expression cut-off, [the] odds of CM-026 success [are] 0.5%.*** [If the] expression cut off [is] 10%, odds of success would [be] 67%. ***[If] BMY used the expression cut-off [of] (50%), then CM-026 success would [be] certain.***

253. However, through their deception, Defendants misled investors as to the true risk inherent in Checkmate-026's primary focus—that the clinical trial targeted the majority (55%) of the NSCLC population with weak 5% levels of PD-L1 expression for whom the data indicated Opdivo was much less likely to be effective. Defendants were directly confronted with the market's mistaken belief as to the true risk of the clinical trial. For example, on March 7, 2016, Defendant Namouni was directly asked to correct an analyst report predicting that Checkmate-026's expression cutoff was 50%—an expression cutoff ten times greater than the actual cutoff—but did not do so. As another example, on July 28, 2016, Defendant Cuss was directly told that by that time "the market assumes 10%"—an expression cutoff double the true cutoff, and according

to one analyst from Cowen ***134 times*** more likely to succeed—but expressly refused to "talk about the high cutoff at this point."

254. After Checkmate-026's failure, another analyst wrote that the now-revealed PD-L1 expression cutoff had concealed the true risk of the clinical trial, stating that they were:

> ***completely puzzled*** by Bristol's decision to evaluate … at a threshold this low, ***particularly given that the trial description indicated patients would be strongly expressing PD-L1. . . .*** [BMO] and probably most of the market, thought the threshold was at least 10%, and ***therefore expected that the trial had a reasonably high probability of success.*** . . . [We] suspect that ***the reason the trial failed is that the PD-1 threshold of at least 5% was too low.***

255. Ultimately, even Defendants themselves have since acknowledged the importance of this metric, ***admitting*** that the PD-L1 expression cutoff chosen for Checkmate-026 caused the clinical trial's failure. In December 2016, Defendants Caforio, Bancroft, Namouni and Gordon told analysts at Leerink that, "[o]verall, mgmt. believes the failure of CM-026 was ***due to it choosing the wrong endpoint***." Then, on June 2, 2017, *Forbes* published a discussion of Checkmate-026's failure with Dr. Lynch, which stated that Dr. Lynch said the reason Checkmate-026 failed was because "Bristol chose a lower cutoff [than Merck's 50%]. . . . Quite simply, Lynch says, it looks as if Merck's predictive cutoff was 'superior' to Bristol's. ***No excuses, no mysteries***."

G. The Critical Importance Of Checkmate-026 To The Financial Success Of The Company And Investors' Interest In The Company.

256. Defendants' scienter is further supported by the critical importance of Checkmate-026 to the financial success of the Company and investors' interest in the Company. Checkmate-026 was at the center of Bristol-Myers' strategy in the rapidly emerging field of immuno-oncology, which the *Wall Street Journal* called "***the next big hope for the drug sector***" in search of "blockbuster sales." Checkmate-026 targeted the most "lucrative" cancer treatment in the race for those blockbuster sales, NSCLC. Just two weeks before the start of the Class Period, *Reuters* wrote "[s]ome industry analysts expect . . . PD-1 or PD-L1 inhibitors, to generate more

than $30 billion in worldwide annual sales by 2025, ***with lung cancer seen as the most lucrative use***." At the start of the Class Period, investors believed it was particularly critical that Checkmate-026 succeed because Bristol-Myers had lost the first-mover advantage to Merck by only just receiving the first FDA approval for Opdivo—months after Merck.

257. As the Class Period continued, investors focused even more keenly on Checkmate-026. For example, *Reuters* wrote in its February 26, 2016 article that, while Bristol-Myers had by then "pulled ahead in the race to dominate the hot new cancer immunotherapy market . . . much is still to play for in a market that has grabbed the attention of investors and which some analysts believe could eventually be worth more than $40 billion a year." *Reuters* further noted that "[d]evelopments this year and next will be crucial in determining if Bristol's rivals can catch up"—namely in "first-line lung cancer."

258. Defendants themselves acknowledged the critical importance of Checkmate-026 to investors. For example, on June 1, 2015, Defendant Giordano stated when discussing Checkmate-026, "***You've been quite curious, and appropriately so, about the front-line strategy*** given our commitment to move immunotherapy increasingly into the front-line setting."

259. Indeed, Bristol-Myers' Former Vice President and Head, Clinical Pharmacology and Pharmacometrics, FE-4, who worked at Bristol-Myers from October 2004 through June 2017, confirmed that Checkmate-026 was a "critical study" for Bristol-Myers, and learned from colleagues that Checkmate-026 had to be approved at the highest level of the Company. (¶89.) Likewise, FE-3—the Company's former Head of Product Portfolio and Access Strategy for Oncology when s/he left in October 2016, and Vice President, Head of Access Strategy for Immuno-oncology from 2013-2015—explained that due to Opdivo's importance to Bristol-Myers

there was a constant dialogue about anything Opdivo with senior management, meaning C-suite level. (¶88.)

H. Several Individual Defendants Traded Over $75 Million Of Bristol-Myers Stock During The Class Period, Netting Nearly $55 Million In Profits.

260. Further supporting Defendants' scienter is the fact that certain Individual Defendants realized substantial financial benefits from insider sales of Bristol-Myers stock while the stock was artificially inflated. These sales were unusual in timing based on historical trading patterns and on the misstatements and omissions made during the time. Additionally, the Defendants' stock sales far exceed their purchases during the Class Period, which further supported scienter. For example, during the Class Period, those Defendants required to report their insider transactions—Defendants Andreotti, Bancroft, Caforio, and Cuss—collectively ***sold 1,131,467 shares*** of Bristol-Myers stock at artificially inflated prices as high as $75.35 per share for proceeds in excess of ***$75.7 million***, ***without purchasing a single share on the open market***.

261. Below is a chart based on Forms 4 filed with the SEC reflecting the reporting Defendants' insider sales and whether the trades were made pursuant to a 10b5-1 trading plan:

Name	Date	Shares Sold	Price[4]	Proceeds	10b5-1 Plan
Andreotti	01/29/15	90,246	$61.14	$5,518,001.42	No
Andreotti	08/03/15	31,200	$64.75	$2,020,293.60	No
Andreotti	08/03/15	283,800	$65.43	$18,569,034.00	No
Andreotti	09/02/15	100	$58.60	$5,860.00	Yes
Andreotti	09/02/15	11,500	$59.23	$681,145.00	Yes
Andreotti	09/03/15	5,500	$59.13	$325,215.00	Yes
Andreotti	09/03/15	6,100	$59.97	$365,817.00	Yes
Andreotti	10/05/15	9,900	$61.84	$612,216.00	Yes
Andreotti	10/05/15	1,700	$62.83	$106,811.00	Yes
Andreotti	10/06/15	3,200	$58.89	$188,448.00	Yes
Andreotti	10/06/15	6,700	$59.86	$401,062.00	Yes
Andreotti	10/06/15	1,700	$60.82	$103,394.00	Yes
Andreotti	11/19/15	11,400	$67.01	$763,914.00	Yes
Andreotti	11/19/15	200	$67.78	$13,556.00	Yes

[4] "Price" rounded to the nearest cent. "Proceeds" uses the disclosed (unrounded) price.

Name	Date	Shares Sold	Price[4]	Proceeds	10b5-1 Plan
Andreotti	11/20/15	11,600	$67.76	$786,016.00	Yes
Andreotti	12/14/15	11,300	$67.99	$768,287.00	Yes
Andreotti	12/14/15	300	$68.52	$20,556.00	Yes
Andreotti	12/15/15	11,600	$70.05	$812,580.00	Yes
Andreotti	01/04/16	11,500	$66.92	$769,580.00	Yes
Andreotti	01/04/16	100	$67.52	$6,752.00	Yes
Andreotti	01/05/16	11,000	$68.16	$749,760.00	Yes
Andreotti	01/05/16	600	$68.78	$41,268.00	Yes
Andreotti	02/11/16	11,300	$59.50	$672,350.00	Yes
Andreotti	02/11/16	300	$60.08	$18,024.00	Yes
Andreotti	02/12/16	11,100	$59.69	$662,559.00	Yes
Andreotti	02/12/16	500	$60.18	$30,090.00	Yes
Andreotti	03/08/16	11,600	$66.06	$766,296.00	Yes
Andreotti	03/09/16	11,400	$65.35	$744,990.00	Yes
Andreotti	03/09/16	200	$66.35	$13,270.00	Yes
Andreotti	04/04/16	6,900	$65.60	$452,640.00	Yes
Andreotti	04/04/16	4,700	$66.08	$310,576.00	Yes
Andreotti	04/05/16	11,400	$65.99	$752,286.00	Yes
Andreotti	04/05/16	200	$66.70	$13,340.00	Yes
Andreotti	05/02/16	100,000	$71.89	$7,188,600.00	No
Andreotti	05/02/16	134,720	$71.87	$9,682,191.68	No
Andreotti	05/04/16	11,600	$70.48	$817,568.00	No
Andreotti	05/05/16	11,600	$70.59	$818,844.00	No
Andreotti	06/28/16	10,500	$71.40	$749,700.00	Yes
Andreotti	06/28/16	1,100	$71.94	$79,134.00	Yes
Andreotti	06/28/16	1,800	$73.09	$131,562.00	Yes
Andreotti	06/29/16	9,800	$72.79	$713,342.00	Yes
Andreotti	07/07/16	11,600	$73.86	$856,776.00	Yes
Andreotti	07/08/16	5,900	$74.74	$440,966.00	Yes
Andreotti	07/08/16	5,700	$75.35	$429,495.00	Yes
Andreotti	08/24/16	3,500	$58.82	$205,870.00	Yes
Andreotti	08/24/16	8,100	$59.69	$483,489.00	Yes
Andreotti	08/25/16	11,600	$58.92	$683,472.00	Yes
Andreotti	09/07/16	10,800	$56.77	$613,116.00	Yes
Andreotti	09/08/16	10,800	$56.97	$615,276.00	Yes
	Total:	**939,966**		**$62,575,388.70**	
Bancroft	05/20/16	30,201	$70.79	$2,137,838.19	No
	Total:	**30,201**		**$2,137,838.19**	
Cuss	03/16/15	114,666	$67.81	$7,775,501.46	No
	Total:	**114,666**		**$7,775,501.46**	

Name	Date	Shares Sold	Price[4]	Proceeds	10b5-1 Plan
Caforio	02/17/16	12,040	$63.58	$765,527.28	No
Caforio	05/03/16	15,145	$71.32	$1,080,171.69	No
Caforio	05/03/16	19,449	$71.30	$1,386,713.70	No
	Total:	**46,634**		**$3,232,412.67**	
TOTALS		**1,131,467**		**$75,721,141.02**	

262. During the Class Period, while Defendants made no open market purchases of Bristol-Myers stock, they were able to convert and acquire previously granted stock options at prices that were drastically lower than the inflated prices at which they currently traded. Below is a chart based on Forms 4 filed with the SEC reflecting the net total of the reporting Defendants' shares acquired through the conversion of vested options at prices far lower than the open market prices that were inflated during the Class Period:

Filer Name	Transaction Type	Transaction Date	Number Of Shares	Price	Amount
Andreotti	Conversion	01/29/2015	112,500	$25.45	$2,863,125.00
Andreotti	Conversion	08/03/2015	200,000	$24.74	$4,948,000.00
Andreotti	Conversion	08/03/2015	115,000	$22.73	$2,613,950.00
Andreotti	Conversion	05/02/2016	134,720	$27.01	$3,638,787.20
Andreotti	Conversion	05/02/2016	100,000	$24.74	$2,474,000.00
		Total:	**662,220**	**Total:**	**$16,537,862.20**
Cuss	Conversion	03/16/2015	75,000	$24.74	$1,855,500.00
Cuss	Conversion	03/16/2015	39,010	$27.01	$1,053,660.10
		Total:	**114,010**	**Total:**	**$2,909,160.10**
Caforio	Conversion	02/17/2016	20,125	$22.89	$460,661.25
Caforio	Conversion	05/03/2016	21,615	$27.01	$583,821.15
Caforio	Conversion	05/03/2016	28,840	$23.12	$666,780.80
		Total:	**70,580**	**Total:**	**$1,711,263.20**

263. Moreover, in making these purchases at non-inflated prices, these insider Defendants were aware of the aggressive gamble that they and the other Defendants were taking

with the design of Checkmate-026, while investors that purchased Bristol-Myers stock at far higher, inflated prices during the Class Period were not aware of the study's highly aggressive design.

264. The Class Period insider stock sales referenced above resulted in ***nearly $55 million*** in net profits for Defendants Andreotti, Bancroft, Caforio, and Cuss:

Defendant	**Total Profits**
Andreotti	$46,037,526.50
Bancroft	$2,137,838.19
Caforio	$1,521,149.47
Cuss	$4,866,341.36
TOTAL PROFITS	**$54,562,855.52**

265. As indicated in the above charts, control person Defendant Andreotti sold 939,966 shares of Bristol-Myers Squibb stock during the Class Period generating proceeds of ***$62,575,388.70.*** Many of these trades appear to be calculated to maximize his personal benefit from the artificial inflation of Bristol-Myers' stock price. For example, seven months after the Class Period began, Andreotti entered into a 10b5-1 plan to sell 23,200 shares of Company stock each month beginning in September 2015 for an undisclosed duration. Nonetheless, most of Andreotti's sales during the Class Period were made ***outside*** of this 10b5-1 plan (and none of Bancroft's, Cuss' or Caforio's sales were made pursuant to a 10b5-1 plan). According to Forms 4 filed with the SEC, Andreotti sold 663,166 shares outside of any 10b5-1 trading plan during the Class Period, while selling only 276,800 shares pursuant to a plan.[5]

[5] Filings submitted with the SEC state that Andreotti's largest sales, totaling more than two-thirds of his Class Period sales and occurring on January 29, 2015, August 3, 2015, May 2, 2016, May 4, 2016 and May 5, 2016, were all made without the use of a 10b5-1 plan. That some of Andreotti's stock sales were made pursuant to a 10b5-1 trading plan does not insulate those trades from scrutiny in any way. Pursuant to SEC Rule 10b5-1(c), a 10b5-1 plan is a potential (but not an

266. Andreotti's stock sales referenced above were also significantly higher than sales for the same date range immediately prior to the start of the Class Period. For example, he sold 586,075 shares from May 15, 2013 through January 26, 2015, which equates to a 621-day period immediately before the start of the Class Period on January 27, 2015. By comparison, during the 621-day Class Period, Andreotti sold 939,966 shares, an increase of 60.1% over his pre-Class Period sales. Similarly, in the 621 days following the Class Period, between October 10, 2016 and June 23, 2018, Andreotti sold a mere 68,000 shares. None of the other reporting Defendants reported any stock sales at all in the 621-day period following the Class Period.

267. In addition to historical trading patterns, the sales made by these Individual Defendants are suspiciously and unusually timed, particularly the May 2016 sales. Indeed, only a few weeks earlier, in April 2016, Defendants were directly confronted with the market's perception that the PD-L1 expression cutoff used by Checkmate-026—which directly related to the study's odds of success—was 50%. However, while Defendants conceded at that time that the study's endpoint was less than 50%, they also preserved the artificial inflation in the stock by continuing to omit that the study's endpoint was far less, only just 5%, and thus the risk that the critical study would not succeed was far greater than the market understood. At the same time, however, Defendants assured the market that they were "increasingly comfortable about the choices [] made in terms of dosing and schedule of Opdivo, as it relates to the efficacy [they were] seeing . . . ." On this news, Bristol-Myers' share price ***increased***, prompting several Defendants to convert and

absolute) defense to accusations of insider trading only if it is entered into by an insider "before becoming aware" of inside information and was established "in good faith and not as part of a plan or scheme to evade the prohibitions" against insider trading. As noted above, Andreotti did not enter into a 10b5-1 trading plan until seven months into the Class Period. Notably, none of the Class Period sales executed by Defendants Bancroft, Caforio, or Cuss appear to have been made pursuant to a 10b5-1 plan.

sell significant amounts of stock soon after this statement was made. For example, just days later between May 2 and May 6, Andreotti converted 234,720 options and then sold those newly converted shares—***the same day***—and others from his direct holdings for gross proceeds of over $18.5 million and for a net profit of more than $10.7 million. Likewise, the following day, May 3, Caforio converted 50,455 options and then immediately sold these newly converted shares for gross proceeds of over $2.4 million and net profit in excess of $1.2 million. Then, two weeks later on May 20, 2016, Bancroft made his only Class Period sale, selling 30,201 shares for proceeds of over $2.1 million.

268. Significantly, none of these May 2016 sales—which again, took place only weeks after Defendants conveyed to the market that they were "increasingly comfortable" with Checkmate-026—were pursuant to a 10b5-1 trading plan.

269. The timing of other sales is also suspicious. Defendant Andreotti's illicit sales begin just after the Class Period begins, when Defendants updated their *ClinicalTrials.gov* submission on January 27, 2015 to falsely and misleadingly claim that Checkmate-026 is examining "strong" PD-L1 expressions. Within days, on January 29, 2015, Defendant Andreotti sold over $5.5 million of Bristol-Myers stock from open-market sales not done pursuant to a Rule 10b5-1 trading plan.

270. Likewise, on March 16, 2015, Defendant Cuss sold an astonishing $7.7 million of Bristol-Myers' stock in open-market sales—his only sale during the Class Period, which was also not pursuant to a Rule 10b5-1 plan. Earlier that same month, on March 1, 2015, the medical journal *Lancet Oncology* had explained that Bristol-Myers' Checkmate-063 trial "used a cutoff of 5% to define PD-L1 positivity[] on the basis of preliminary findings" in Bristol-Myers's earlier studies. This reaffirmed the market's understanding that 5% was the most-common bare minimum for PD-L1 positivity—indeed, in part because of Bristol-Myers' own studies. Within days, on March

5, 2015, Defendants took advantage of the market's understanding by again repeating their *ClinicalTrials.gov* submission to falsely and misleadingly claim that Checkmate-026 is examining "strong" PD-L1 expression, and by the end of the month Defendant Cuss cashed out millions of dollars in stock at the expense of the misled market.

271. Unlike Defendants Andreotti, Bancroft, Caforio, and Cuss, who are required as "officers" under Section 16 of the Exchange Act to publicly report their trading activity on Forms 4 filed with the SEC, by all accounts Bristol-Myers does not consider Defendants Namouni and Giordano to be "officers" under Section 16. Thus, their trading activity is not subject to the same SEC reporting requirements. Without further discovery, no inference should be made about Namouni and Giordano's trading patterns.

272. In sum, given that these and other Class Period sales—particularly when compared to Defendants' far fewer Class Period purchases at prices well below the inflated open market prices—were made at the same time Defendants misrepresented and concealed the true progress of Checkmate-026, they support a strong inference that Defendants knew the statements were false and misleading when made or otherwise omitted material information from investors.

273. That some of Defendants' holdings may have increased over the Class Period is not inconsistent with this inference of scienter. First, ***no*** Defendant reported ***any open-market purchases*** of Bristol-Myers stock during the Class Period. Instead, Defendants' "acquisitions" of additional shares of Bristol-Myers stock occurred only from vesting compensation incentives or the exercise of options to acquire shares of Bristol-Myers' stock at prices far lower the (artificially inflated) market price.

274. Likewise, that Defendants did not sell all or the majority of their holdings is also not inconsistent with an inference of scienter. There is no allegation that Defendants did not

earnestly ***want*** Checkmate-026 to succeed. Rather, Plaintiffs allege that Defendants concealed that they had gambled in the design of Checkmate-026 by aggressively pursuing approval for a low PD-L1 cutoff. This fraud not only prevented the market from accurately assessing the risk of Checkmate-026's failure, it also prevented the public—including competitors—from knowing just how enormous the reward would have been had the trial succeeded. Had the gamble paid off, Bristol-Myers would have obtained an incredible—possibly insurmountable—lead in the competition for the industry's next blockbuster drug, and the value of Defendants' Bristol-Myers' holdings almost certainly would have increased dramatically. *See Makor Issues & Rights, Ltd. v. Tellabs, Inc.*, 513 F.3d 702, 709-710 (7th Cir. 2008) (noting that, in ruling that false statements about "strong demand" were made with scienter, "the fact that a gamble . . . fails is not inconsistent with its having been a considered, though because of the risk a reckless, gamble.").

I. Defendants' Refusal To Answer Direct Questions About The Checkmate-026 Expression Focus.

275. Defendants' scienter is further supported by their demonstrated unwillingness to be forthcoming in the matters at issue. As noted market commentator Jim Cramer concluded on January 20, 2017, when it came to Opdivo, Defendants had been "***the exact opposite***" of "a company that gives you a very clear narrative about ***what can happen***."

276. For example, on July 28, 2016, an analyst directly confronted Defendants Cuss and Caforio with Defendants' inexplicable refusal to provide the cutoff, speculating that Defendants might attempt to change the protocol for the clinical trial. Rather than answer the analyst's question—or correct the market's assumption as to the cutoff—Cuss and Caforio instead reiterate their confidence in the study's design:

> [Defendant Cuss]: ***[A]s I have noted in the past and will note again today, I'm confident in the study design of 026. . . .*** I will say again, we're not going to talk about the high [expressive] cutoff at this point. But you, obviously, will have an opportunity to see that when we talk about the data. . . .

[Defendant Caforio]: ***[W]e are very confident in . . . the design of the clinical trial***.

Incidentally, months later, Defendants would in fact admit that their design in this very respect caused Checkmate-026's failure. (*E.g.* ¶255.)

277. As another example, as noted above, throughout the Class Period, Defendants were aware of, and had access to, the trial protocol for Checkmate-026, which contradicted their public statements and omissions concerning the "strong" PD-L1 expression cutoff. (¶¶132-33.) This trial protocol was not publicly available until June 2017, when it was published by the New England Journal of Medicine. Even then, however, the trial protocol is considerably redacted, with 15% of its 130 pages having redactions—including entire sections, certain descriptions of amendments made to the protocol since inception, and even entries in the table of contents and references.

278. As a final example, on October 9, 2016, Defendants presented for the first time the full data from Checkmate-026, which revealed that the clinical trial did not provide sufficient data under accepted statistical methodologies to find a significant difference between the performance of Opdivo and chemotherapy in subgroup analyses focusing on patients in the trial who met a higher, 50% PD-L1 threshold. Prior to its public presentation, this full data from Checkmate-026 had been available to Defendants since August 5, 2016, when Defendants announced that the clinical trial had failed to achieve its primary outcome. Yet Defendants did not disclose that data for over two months—during which time they continued to sow hope that the unreleased data would provide useful information about Opdivo's efficacy through such subgroup analyses. (¶¶134-137.)

J. Defendant Cuss's And Giordano's Conspicuous Departures.

279. Defendants' scienter is further supported by the fact that Defendant Cuss—who, as the Company's Chief Scientific Officer, had been in charge of the development of Opdivo and Checkmate-026, and the Company's principal spokesperson for both—departed from the

Company unexpectedly in March 2017, close on the heels of the market's reaction to having been misled about the true risk of Checkmate-026. Indeed, on March 9, 2017, industry website *FiercePharma* published an article titled "Bristol-Myers' R&D revamp mirrors upheaval in Opdivo's lunch cancer fortunes," which attributed Cuss' departure to Checkmate-026:

> Bristol-Myers Squibb has a new R&D chief. ***That's not much of a surprise to those who've been following the company's fall from grace, thanks to its unexpected and brutal failure in a cancer immunotherapy trial last fall, or the precipitous decline in its share price since***.
>
> With billions cut from peak sales estimates for its PD-1 checkpoint inhibitor Opdivo—which until the trial fiasco was seen as the natural market leader—and shares down to $57.40 Wednesday from more than $72 before the data went public, Chief Scientific Officer Francis Cuss made for the exit Wednesday. Board member and cancer doctor Thomas Lynch stepped into his place.

280. Defendant Giordano's unexpected departure on July 26, 2016, just days before Bristol-Myers announced the shocking failure of Checkmate-026, also raises an inference of scienter. After seventeen years at Bristol-Myers, the Company announced his immediate "retirement". Despite this purported "retirement", Giordano resumed active employment in the biopharmaceutical industry beginning in December 2017.

XII. CLAIMS BROUGHT PURSUANT TO THE EXCHANGE ACT

FIRST CLAIM FOR RELIEF
For Violations Of Section 10(b) Of The Exchange Act And Rule 10b-5
(Against Defendants Bristol-Myers, Giordano, Namouni, Cuss, Bancroft and Caforio)

281. Plaintiffs repeat and re-allege each and every allegation contained above as if fully set forth herein.

282. During the Class Period, Defendants Bristol-Myers, Giordano, Namouni, Cuss, Bancroft, and Caforio disseminated or approved the false statements specified herein, which they knew or recklessly disregarded were misleading in that they failed to disclose material facts

necessary in order to make the statements made, in light of the circumstances under which they were made, not misleading, and they contained material misrepresentations.

283. Defendants Bristol-Myers, Giordano, Namouni, Cuss, Bancroft, and Caforio violated Section 10(b) of the Exchange Act and Rule 10b-5 thereunder in that they: (i) employed devices, schemes, and artifices to defraud; (ii) made untrue statements of material facts or omitted to state material facts necessary in order to make the statements made, in light of the circumstances under which they were made, not misleading; and/or (iii) engaged in acts, practices, and a course of business that operated as a fraud or deceit upon Plaintiffs and others similarly situated in connection with their purchases of Bristol-Myers common stock during the Class Period. As detailed herein, the misrepresentations contained in, or the material facts omitted from, these Defendants' public statements, concerned, among other things: (i) the "strong" and "high positive" PD-L1 expression targeted by Checkmate-026; and (ii) the risk posed by the Checkmate-026 clinical trial design.

284. Defendants Bristol-Myers, Giordano, Namouni, Cuss, Bancroft, and Caforio, individually and in concert, directly and indirectly, by the use of means or instrumentalities of interstate commerce and/or of the mails, engaged and participated in a continuous course of conduct that operated as a fraud and deceit upon Plaintiffs and the Class; made various false and/or misleading statements of material facts and omitted to state material facts necessary in order to make the statements made, in light of the circumstances under which they were made, not misleading; made the above statements with a reckless disregard for the truth; and employed devices, schemes, and artifices to defraud in connection with the purchase and sale of securities, which were intended to, and did: (a) deceive the investing public, including Plaintiffs and the Class, regarding, among other things, (i) the "strong" and "high positive" level of PD-L1

expression targeted by Checkmate-026, and (ii) the risk posed by the Checkmate-026 clinical trial design; (b) artificially inflate and maintain the market price of Bristol-Myers common stock; and (c) cause members of the Class to purchase Bristol-Myers securities at artificially inflated prices.

285. The Individual Defendants, as top executive officers of the Company, are liable as direct participants in the wrongs complained of herein. Through their positions of control and authority as officers of the Company, each of the Individual Defendants was able to and did control the content of the public statements disseminated by Bristol-Myers. The Individual Defendants had direct involvement in the daily business of Bristol-Myers and participated in the preparation and dissemination of the false and misleading statements.

286. As described above, Defendants acted with scienter throughout the Class Period, in that they either had actual knowledge of the misrepresentations and omissions of material facts set forth herein, or acted with reckless disregard for the truth in that they failed to ascertain and to disclose the true facts, even though such facts were available to them.

287. Plaintiffs and the Class have suffered damages in that they paid artificially inflated prices for Bristol-Myers securities. Plaintiffs and the Class would not have purchased Bristol-Myers securities at the prices they paid, or at all, if they had been aware that the market price had been artificially and falsely inflated by Defendants' misleading statements.

288. As a direct and proximate result of Defendants' wrongful conduct, Plaintiffs and the Class suffered damages in connection with their purchases of Bristol-Myers securities during the Class Period.

SECOND CLAIM FOR RELIEF
For Violations Of Section 20(a) Of The Exchange Act
(Against The Individual Defendants)

289. Plaintiffs repeat and re-allege each and every allegation contained above as if fully set forth herein.

290. This count is asserted against the Individual Defendants for violations of Section 20(a) of the Exchange Act, 15 U.S.C. § 78t(a), on behalf of all members of the Class.

291. As alleged herein, the Individual Defendants caused Bristol-Myers to violate Section 10(b) and Rule 10b-5 promulgated thereunder by making material misstatements and omissions in connection with the purchase and sale of securities throughout the Class Period. This conduct was undertaken with the scienter of the Individual Defendants who knew of or recklessly disregarded the falsity of the Company's statements and the nature of its scheme during the Class Period.

292. During their tenures as officers and/or directors of Bristol-Myers, the Individual Defendants were controlling persons of Bristol-Myers within the meaning of Section 20(a) of the Exchange Act. By reason of their positions of control and authority as officers and/or directors of Bristol-Myers, the Individual Defendants had the power and authority to cause Bristol-Myers to engage in the wrongful conduct complained of herein. As set forth in detail above, the Individual Defendants named in this claim were able to and did control, directly and indirectly, and exert control over Bristol-Myers, including the content of the public statements made by Bristol-Myers during the Class Period, thereby causing the dissemination of the false and misleading statements and omissions of material facts as alleged herein.

293. In their capacities as senior corporate officers or directors of the Company, and as more fully described above, the Individual Defendants had direct involvement in the day-to-day

operations of the Company. By reason of their positions as officers or directors of Bristol-Myers, and more specifically as controlling officers—as can be seen by their corresponding ability to influence and control Bristol—each of these Individual Defendants is a "controlling person" within the meaning of Section 20(a) of the Exchange Act, and had the power and influence to direct the management and activities of the Company and its employees, and to cause the Company to engage in the unlawful conduct complained of herein. Because of their positions, these Individual Defendants had access to adverse nonpublic information about the Company and acted to conceal the same, or knowingly or recklessly authorized and approved the concealment of the same.

294. Moreover, each of the Individual Defendants also culpably participated in providing false information and certifying and/or approving the false statements disseminated by Company during the Class Period. Each of the Individual Defendants was provided with or had access to copies of the Company's submissions to *ClinicalTrials.gov*, reports, press releases, public filings, and other statements alleged by Plaintiffs to be misleading prior to and/or shortly after these statements were issued and had the ability to prevent the issuance of the statements or cause the statements to be corrected.

295. Individual Defendants Andreotti, Cuss, Caforio, and Bancroft further culpably participated in personally profiting while Bristol-Myers' securities traded at artificially inflated and distorted prices by selling 1,131,467 shares of Bristol-Myers common stock while in possession of adverse, material non-public information about Bristol-Myers, pocketing over $75.7 million in illegal insider trading proceeds, as detailed above.

296. As set forth above, the Individual Defendants caused Bristol-Myers to violate Section 10(b) of the Exchange Act by making material misstatements and omissions in connection with the purchase and sale of securities and by participating in a scheme and course of business or

conduct throughout the Class Period. This conduct was undertaken with the scienter and culpable participation of the Individual Defendants who knew of or recklessly disregarded the falsity of the Company's statements and the nature of its scheme during the Class Period.

297. As a direct and proximate result of these Individual Defendants' conduct, Plaintiffs and the Class suffered damages in connection with their purchase or acquisition of Bristol-Myers securities.

THIRD CLAIM FOR RELIEF
For Violations Of Section 20A Of The Exchange Act
(Against Individual Defendants Andreotti And Caforio)

298. Plaintiffs repeat and re-allege each and every allegation contained above as if fully set forth herein.

299. This count is asserted against Individual Defendants Andreotti and Caforio for violations of Section 20A of the Exchange Act, 15 U.S.C. § 78t-1, on behalf of all members of the Class.

300. While Bristol-Myers' securities traded at artificially inflated and distorted prices, the Individual Defendants personally profited by selling 1,131,467 shares of Bristol-Myers common stock while in possession of adverse, material non-public information about Bristol-Myers, pocketing over $75.7 million in illegal insider trading proceeds, as detailed above.

301. By contrast, for example, Plaintiffs purchased Bristol-Myers stock contemporaneously with several of these Individual Defendants' sales, as follows:

Defendants' Open Market Sales				Contemporaneous Purchases			
Defendant	**Sale Date**	**Shares Sold**	**Price**	**Lead Plaintiff**	**Purchase Date**	**Shares Purchased**	**Price**
Andreotti	11/19/15	11,400	$67.01	APERS	11/19/2015	1,117	$67.07
		200	$67.78			468	$66.98
		11,600	$67.76			498	$67.01
						417	$67.01
Andreotti	12/14/15	11,300	$67.99	APERS	12/15/2015	18,200	$70.17
		300	$68.52				
	12/15/15	11,600	$70.05				
Andreotti	01/04/16	11,500	$66.92	Louisiana Sheriffs	01/04/16	900	$67.00
		100	$67.52		01/06/16	400	$67.54
	01/05/16	11,000	$68.16				
		600	$68.78				
Andreotti	02/11/16	11,300	$59.50	Louisiana Sheriffs	02/11/16	800	$59.49
		300	$60.08				
	02/12/16	11,100	$59.69				
		500	$60.18				
Andreotti	02/12/16	11,100	$59.69	Erste AM	02/17/2016	29,000	$63.22
		500	$60.18		02/18/2016	25,000	$63.45
Caforio	02/17/16	12,040	$63.58				
Andreotti	03/08/16	11,600	$66.06	Louisiana Sheriffs	03/08/16	100	$66.02
	03/09/16	11,400	$65.35		03/10/16	1,200	$65.49
		200	$66.35		03/11/16	300	$64.86
						500	$64.78
Andreotti	04/04/16	6,900	$65.60	Louisiana Sheriffs	04/04/16	3,900	$65.77
		4,700	$66.08				

Defendants' Open Market Sales				Contemporaneous Purchases			
Defendant	**Sale Date**	**Shares Sold**	**Price**	**Lead Plaintiff**	**Purchase Date**	**Shares Purchased**	**Price**
Andreotti	05/02/16	100,000	$71.89	Louisiana Sheriffs	05/05/16	800	$70.63
		134,720	$71.87			300	$71.06
Caforio	05/03/16	15,145	$71.32		05/06/16	900	$71.08
		19,449	$71.30				
Andreotti	05/04/16	11,600	$70.48				
	05/05/16	11,600	$70.59				
Andreotti	08/25/16	11,600	$58.92	Louisiana Sheriffs	08/30/16	2,703	$57.29
						690	$57.27
						1,747	$57.37

302. Plaintiffs and all other members of the Class who purchased shares of Bristol-Myers stock contemporaneously with the sales of Bristol-Myers common stock by Individuals Defendants Andreotti and Caforio have suffered damages because:

a) In reliance on the integrity of the market, they paid artificially inflated prices as a result of the violations of §§10(b) and 20(a) of the Exchange Act as alleged herein; and

b) they would not have purchased the Bristol-Myers stock at the prices they paid, or at all, if they had been aware that the market prices had been artificially inflated by Defendants' false and misleading statements and omissions alleged herein.

303. By reason of the foregoing, Individual Defendants Andreotti and Caforio violated §20A of the Exchange Act and are liable to Plaintiffs and the other members of the Class for the substantial damages suffered in connection with their purchase of Bristol-Myers common stock during the Class Period.

XIII. CLASS ACTION ALLEGATIONS

304. Plaintiffs bring this lawsuit pursuant to Rule 23(a) and (b)(3) of the Federal Rules of Civil Procedure, on behalf of themselves and on behalf of a Class of persons who purchased or otherwise acquired Bristol-Myers common stock during the Class Period. Excluded from the Class are: Defendants; members of the immediate family of each Defendant; any person who was an officer or director of Bristol-Myers during the Class Period; any firm, trust, corporation, officer, or other entity in which any Defendant has or had a controlling interest; any person who participated in the wrongdoing alleged herein; and the legal representatives, agents, affiliates, heirs, beneficiaries, successors-in-interest, or assigns of any such excluded party.

305. The members of the Class are so numerous that joinder of all members is impracticable. The disposition of their claims in a class action will provide substantial benefits to the parties and the Court. Throughout the Class Period, Bristol-Myers' common stock was actively traded on NYSE, an efficient market. As of September 30, 2016, the Company had 1,671,229,946 shares of Bristol-Myers common stock outstanding. While the exact number of Class members is unknown to Plaintiffs at this time, and can only be ascertained through appropriate discovery, Plaintiffs believe that there are at least hundreds of thousands of members in the Class.

306. There is a well-defined community of interest in the questions of law and fact involved in this case. Questions of law and fact common to the members of the Class predominate over questions that may affect individual Class members, including:

a) Whether Defendants violated the federal securities laws;

b) Whether Defendants misrepresented material facts concerning Checkmate-026, among other things;

c) Whether Defendants' statements omitted material facts necessary to make their statements not misleading in light of the circumstances under which they were made;

d) Whether Defendants knew or recklessly disregarded that their statements were false and misleading;

e) Whether Defendants engaged in perpetrating a manipulative and deceptive device and/or scheme and/or otherwise engaged in a fraudulent course of conduct;

f) Whether the prices of Bristol-Myers common stock were artificially inflated; and

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

307. The claims of Plaintiffs are typical of those of the Class.

308. Plaintiffs will adequately protect the interests of the Class and have retained counsel experienced in class action securities litigation. Plaintiffs have no interests that conflict with those of the Class.

309. A class action is superior to other available methods for the fair and efficient adjudication of this controversy.

XIV. PRAYER FOR RELIEF

310. WHEREFORE, Plaintiffs pray for judgment individually and on behalf of the Class, as follows:

a) Declaring this action to be a proper class action pursuant to Rule 23 of the Federal Rules of Civil Procedure;

b) Awarding Plaintiffs and the Class members damages, including interest;

c) Awarding Plaintiffs reasonable costs, including attorneys' and experts' fees; and

d) Awarding such equitable/injunctive or other relief for the benefit of the Class as the court may deem just and proper.

XV. JURY DEMAND

Plaintiffs hereby demand a trial by jury on all issues.

Dated: October 29, 2019

Respectfully submitted,

/s/ Lauren A. Ormsbee

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APPENDIX A

FE Key

FE No.	Tenure	Relevant Position(s) or Role(s)
1	1989-2015	Senior Vice President, Global Commercialization for Oncology
2	2010-2013	Associate Director of Global Oncology Forecasting & Strategic Planning
3	1983-2016	Head of Product Portfolio and Access Strategy for Oncology; Vice President, Head of Access Strategy for Immuno-oncology
4	2004-2017	Vice President and Head, Clinical Pharmacology and Pharmacometrics
5	2012-2016	Group Medical Director, Immuno-oncology