

IN THE UNITED STATES PATENT AND TRADEMARK OFFICE

BEFORE THE PATENT TRIAL AND APPEAL BOARD

CSPC MEGALITH BIOPHARMACEUTICAL CO., LTD.,

Petitioner,

v.

SHANGHAI MIRACOGEN INC.,

Patent Owner.

Case No. IPR2025-00685

U.S. Patent No. 10,792,370

DECLARATION OF DJORDJE ATANACKOVIC

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I, Djordje Atanackovic, declare as follows:

1. My name is Djordje Atanackovic. I am a Professor of Medicine in the Department of Internal Medicine at the University of Maryland. A summary of my background and qualifications can be found in Section X below. My full Curriculum Vitae is attached to this declaration as Appendix 1.

2. I have prepared this declaration as an expert witness retained by Shanghai Miracogen Inc. This declaration contains statements of my opinions formed to date and the basis and reasons for those opinions. I may offer additional opinions based on further review of materials in this case, including opinions and/or testimony of other expert witnesses. I make this declaration based upon my own personal knowledge and, if called upon to testify, would testify competently to the matters contained herein.

I. Overview of the Technology

3. The subject patent under the inter partes review (“IPR”), U.S. Patent No. 10,792,370, relates to antibody-drug conjugates (“ADCs”). An ADC typically includes an antibody and one or more cytotoxic payloads each of which is linked to the antibody through a linker.

4. Each of the components of an ADC plays an important role. The antibody is primarily responsible for targeting the ADC to a target cell, e.g., a tumor cell, that expresses the corresponding antigen. Once the antibody binds the

antigen on the target cell, it may be internalized by the target cell, thereby delivering the conjugated cytotoxic payload into the target cell. The cytotoxic payload is primarily responsible for killing the target cell with its cytotoxic activity.

5. The linker provides a means for attaching the payload to the antibody. Moreover, the linker impacts how the payload is released to exert its cytotoxic activity in a target cell, and also how the payload stays attached to the antibody prior to such release, to prevent undesired toxicity prior to reaching the target cell, e.g., at normal tissues.

6. The linker can be generally categorized as cleavable linkers and non-cleavable ones. The former is designed to be cleaved at the target environment, e.g., tumor microenvironment, or within the target cell. A payload of an ADC with a non-cleavable linker, by contrast, only becomes activated when the ADC is destructed in the lysosomes within the target cell. Ex-1009, 6-7.

7. Cytotoxic payloads are typically anticancer agents on their own for ADCs intended for cancer treatments. There were multiple candidate cytotoxic payloads that can be used in ADCs, including monomethyl auristatin E (MMAE), monomethyl auristatin F (MMAF), DM-1, DM-4 (Ex-1009, 14) and camptothecin analogues (Ex-2025).

8. A suitable payload needs to have acceptable safety and stability so that it stays inactive while attached to the antibody in systemic circulation. Also important, it needs to have acceptable potency to kill the target cell once released in the target cell. Nevertheless, there were multiple known payloads, such as MMAE, MMAF, DM-1, DM-4, and camptothecin analogues.

9. In particular, these payloads, belonging to the classes of tubulin inhibitors and topoisomerase inhibitors, were known to be highly potent. Therefore, a primary challenge of designing a successful ADC was the identification of one that has manageable toxicity without compromising on the efficacy. In other words, a successful ADC needs to have an acceptable **“therapeutic window.”** The term “therapeutic window,” as well as the related terms “therapeutic index” and “safety window,” generally refer to dose ranges of a drug between boundaries of efficacy and toxicity. A drug candidate with a narrow therapeutic window (i.e., small difference between therapeutic and toxic doses) is not as desirable as one with a wide therapeutic window, as the former is more likely to cause toxicities at close-to-efficacious doses.

10. For some drugs, their toxicity may be caused by a different biological mechanism from the mechanism by which they are efficacious. Different from such off-target toxicity issues, the toxicities of ADCs are mainly on-target. That is, when the payload is released in a tumor cell expressing the antigen, it kills the

tumor cell; if a non-tumor cell also expresses the antigen, it can be bound by the antibody and killed by the payload of the ADC as well, leading to toxicity in the normal tissue.

11. Therefore, the therapeutic window of an ADC, as readily recognized in conventional knowledge, primarily depends on whether the ADC can reduce binding to or payload release in normal tissues, while at the same time maintaining binding to and payload release in tumor tissues.

12. Epithelial growth factor receptor (EGFR) is an attractive target for the development for an anti-tumor therapy “because of the antigen’s expression by many tumors and its rapid internalization.” Ex-1009, 2. However, “because EGFR is also expressed by skin tissues, EGFR-targeting agents, such as the antibodies cetuximab and panitumumab, also show levels of skin toxicities that either demand dose reduction or in some cases are so severe as to warrant discontinuation of treatment.” *Id.*

13. EGFR’s relatively high expression in normal tissues differentiates it from other antigens. For example, while both EGFR and CD30 are highly expressed in tumor cells, CD30’s expression levels in normal tissues are minimal and considerably lower than that of EGFR. EGFR’s relatively high expression in normal tissues creates unique challenges faced by therapies targeting EGFR with respect to toxicity. The toxicities associated with the use of anti-EGFR antibodies

cetuximab and panitumumab, as Tikhomirov noted, are due to the antibodies' recognition of the EGFR protein expressed on skin tissues. In other words, not only do these antibodies bind the EGFR protein on tumor cells, they also bind the EGFR proteins in non-tumor cells (e.g., skin cells). It follows that these antibodies' binding to non-tumor cells would cause toxicities when they are incorporated into ADCs. An ADC that includes an anti-EGFR antibody, therefore, is significantly more likely to cause toxicity than an ADC that targets a different antigen, such as CD30. This is exactly what Tikhomirov observed with the "Cetux 2C9-MMAE" ADC which included a cytotoxic payload MMAE conjugated to cetuximab (Cetux 2C9) through a valine-citrulline (VC) linker. In Example 4, Tikhomirov showed that Cetux 2C9-MMAE potentiated toxicity against normal cells along with its anti-tumor activities. Ex-1009, 10-11. This is likely why, more than 20 years since cetuximab was approved as an anti-cancer drug, still no anti-EGFR ADC was approved anywhere in the world when the Petition was filed.

14. Consistent with the conventional knowledge I outlined above, Tikhomirov disclosed the main strategies of addressing the toxicity issues associated with anti-EGFR ADCs.

15. Tikhomirov's main focus was on the selection of linkers. Tikhomirov refers to both cetuximab and panitumumab as "full antagonist" anti-EGFR antibodies. According to Tikhomirov, a full antagonist antibody is one "that blocks

completely or nearly so the transmission of a signal that is stimulated, in the normal course, by the EGF ligand through wtEGFR [(wild type human EGFR)] to the wtEGFR-coupled tyrosine kinase.” Ex-1009, 11, Moreover, “EGFR antibodies that are full antagonists are particularly EGFR antibodies that bind directly to EGFR domain III.” *Id.* Therefore, antibodies derived from a full antagonist antibody like cetuximab while retaining its binding specificity should also be full antagonist antibodies. By contrast, a “partial antagonist” is one “that allows transmission of some EGF-mediated signal.” *Id.* at 12. Given the toxicity issue observed with Cetux 2C9-MMAE, an ADC with a full antagonist antibody and a cleavable linker, Tikhomirov suggested that an ADC that incorporates a full antagonist antibody should use a non-cleavable linker. *Id.* at 31.

16. With respect to the choice of antibodies, Tikhomirov provides a summary of four conventional strategies for addressing these safety concerns. Strategy one entails the use of anti-EGFR antibodies that “target a mutated but naturally occurring version of EGFR, known as EGFRvIII, or on conformational forms of the EGFR, both of which predominate on tumour cells and not on skin cells.” Ex-1009, 3. An example antibody of this nature is “MAb806”. *Id.* As discussed in further details below, a humanized version of MAb806 was the antibody of focus in Leanna, “Antibody 1.”

17. Strategy two entails the use of anti-EGFR antibodies “that are preferentially activated in the tumor microenvironment.” Ex-1009, 3. To this end, Tikhomirov referred to examples in PCT Application WO 2009/025846, which include masking peptides which are cleaved off in the tumor. I consider that this category of antibodies also include those that are preferentially activated in the tumor microenvironment’s acidic pH conditions, i.e., pH-selective antibodies.

18. Strategy three entails the use of anti-EGFR antibodies “with partial antagonist activity against EGFR [which have] reduced activity against keratinocytes.” Ex-1009, 3.

19. Strategy four entails the use of anti-EGFR “antibodies with medium affinity that preferentially accumulate in the tumor and not normal tissues.” Ex-1009, 3-4. To this end, Tikhomirov refers to examples in PCT Application WO 2012/100346 (“Tikhomirov-346,” Ex-2011), which will be discussed in further details below.

20. I summarize all of these strategies in the table below:

Strategies for addressing anti-EGFR ADCs' safety concerns

| | Strategy |
|-----------------|--|
| Linker | |
| | A. Use non-cleavable linker instead of cleavable linker for full-antagonist antibodies like cetuximab and derivatives |
| Antibody | |
| | B1. Use antibodies that target EGFR epitope found in tumors only |
| | B2. Use antibodies that are preferentially activated in tumor microenvironment |
| | B3. Use antibodies with reduced antagonist activity in normal cells |
| | B4. Use antibodies with reduced affinity |

21. Strategies B1 and B2 aim at preferentially increasing the binding or activity of the ADC in tumor tissues or preferentially decreasing the binding or activity of the ADC in normal tissues. Such strategies, therefore, can increase the therapeutic window of the ADC. Strategy B3 aims to preferentially reducing the antibody's antagonist activity in normal cells to reduce toxicity to those cells. This strategy, therefore, can also increase the therapeutic window of the ADC.

22. Under strategy B4, when the affinity of an antibody is reduced, the reduction can impact how the antibody acts on both the tumor and normal tissues.

To this end, I note that it was well established that such reduction has a significantly more pronounced effect on cells having lower expression of the antigen (e.g., normal cells) than on cells having higher expression of the antibody (e.g., tumor cells). In other words, relative to a high-affinity anti-EGFR antibody such as cetuximab, an anti-EGFR antibody having lower affinity would have been expected to have a wider therapeutic window.

23. For instance, Crombet observed that an anti-EGFR antibody, h-R3, “elicited similar response in advanced SCCHN compared with what is published for other anti-EGFR antibodies, but at lower doses and without skin toxicity.” Ex-2022, 000005. h-R3, Crombet notes, “has less affinity ($K_D = 10^{-9}$ M) than IMC-C225 ($K_D = 10^{-10}$ M) for the EGFR” (*id.*). IMC-C225 is cetuximab. Based on such experimental data and mathematical modeling, Crombet concluded that “intermediate affinity mAbs (10^{-8} and 10^{-9} M) will have the maximum effect (high tumor uptake and low uptake in normal tissues), while lower affinity antibodies would have little tumor uptake and higher affinity mAbs would induce a rapid uptake by normal tissues reducing again the therapeutic index (Fig 3).” *Id.*

24. According to Crombet, therefore, anti-EGFR antibodies having an affinity that is at least 10-fold (10-fold to 100-fold) lower than cetuximab would have the maximal capability in distinguishing normal cells from tumor cells. This

is illustrated in Fig. 3, which is partially reproduced below, with annotations added.

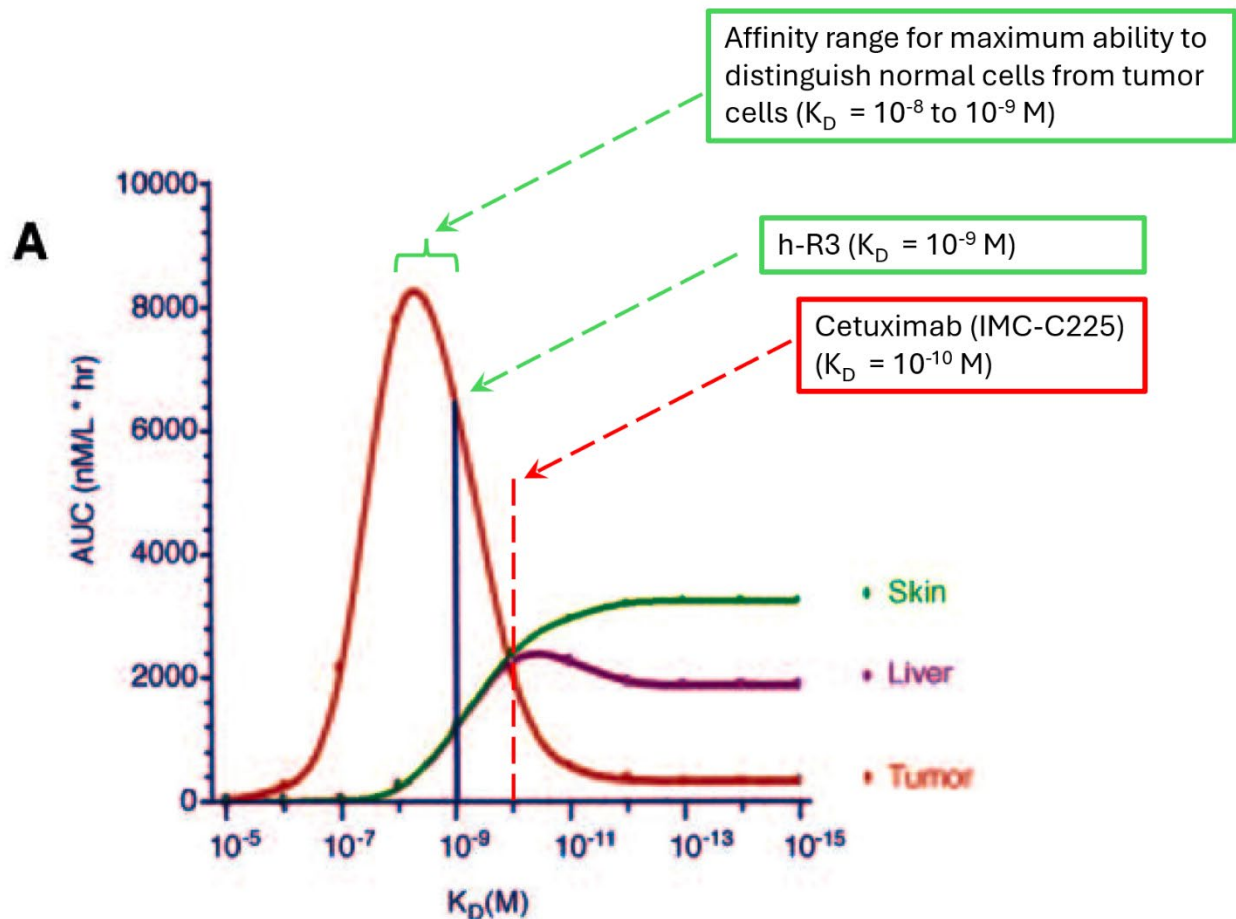


FIG. 3. Figure 3A of Crombet, with annotations added (dotted lines and texts in rectangular boxes). Ex-2022, 000007.

25. As can be easily seen from the figure above, cetuximab's binding affinity to EGFR is too high, and has poor ability in distinguishing normal cells from tumor cells. An ideal antibody should have a binding affinity that is about 10 times lower than cetuximab (with a K_D in the range of 10⁻⁸ to 10⁻⁹ M). It can also

be readily predicted that anti-EGFR antibodies with a higher binding affinity than cetuximab (i.e., with a $K_D < 10^{-10}$ M) would have poorer or even no ability in selectively binding to tumor cells without binding to normal cells.

26. Crombet's model had been validated by multiple examples since its publication in 2004. For example, in Tikhomirov-346, example antibodies had "a binding affinity for EGFR that is about 10-fold or more weaker than the EGFR binding affinity of cetuximab." Ex-2011, 5:22-30. Such reduction of the affinity "substantially eliminates binding to [normal] cells presenting EGFR at a normal EGFR density, and retains effective binding at targeted disease cells that present EGFR at a greater density relative to normal cell EGFR density." *Id.*, 3:12-16.

27. Another anti-EGFR antibody that has a binding affinity that is 10-fold or more weaker than cetuximab is nimotuzumab. Nimotuzumab has been approved in multiple countries for cancer treatment and it "has a ten-fold lower affinity for the extracellular domain of EGFR than the Fab of cetuximab." Ex-2023, 000002 ("Garrido"). It was not surprising at all, therefore, nimotuzumab was "reported to have lesser or no skin toxicity" (Ex-2024, 000003) and "has demonstrated a unique safety clinical profile, where antitumor activity was observed with a very low toxicity profile" (Ex-2023, 000002).

28. Nimotuzumab provided an explanation on why the reduced affinity of nimotuzumab improved its ability to distinguish normal cells from tumor cells.

With such low affinity, its stable binding to EGFR requires bivalent (i.e., both arms of the antibody are bound to EGFR) interaction. Such low-affinity bivalent binding, Garrido suggested, “is transient” on cells on which “EGFR density is low, such as on normal tissues.” *Id.*, 000007. By contrast, “cetuximab continues to interact strongly with [EGFR]” on such normal tissues. *Id.* This explanation is illustrated in Figure 6 of Garrido, which is reproduced below.

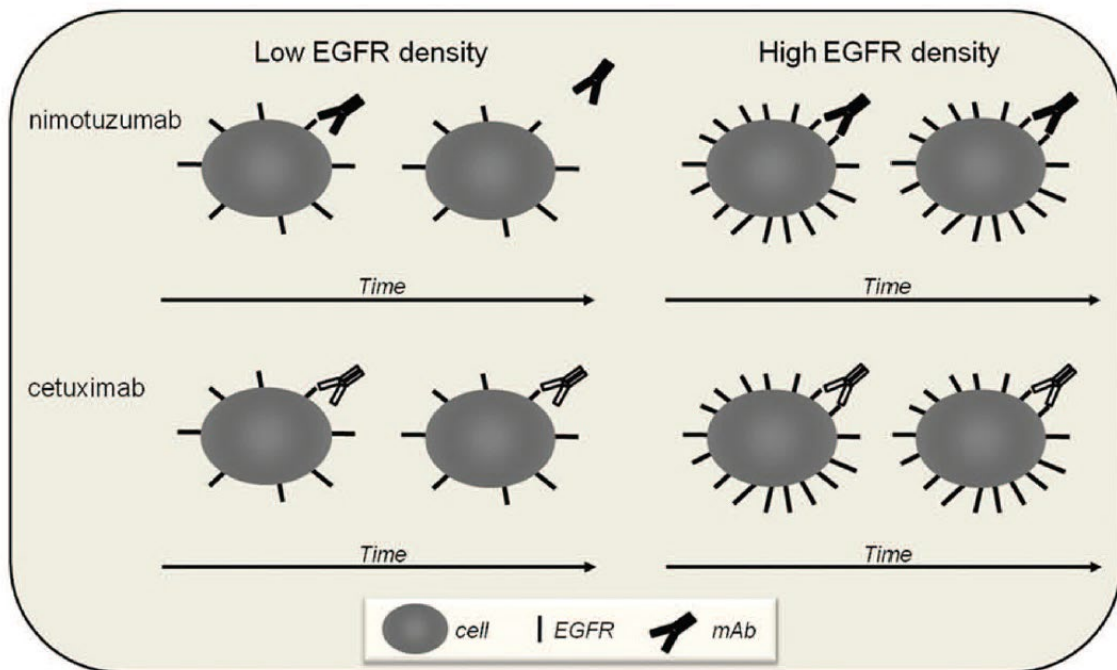


FIG. 4 Figure 6 of Garrido. Ex-2023, 000008.

29. In other words, Garrido reasoned that anti-EGFR antibodies having at least 10-fold lower affinity than cetuximab require a minimum EGFR density on normal cells to keep stable binding. As normal cells do not have such a minimum density, they are spared from binding by these low-affinity antibodies and thus are free from toxicity.

30. On this background, I provide a summary of my analysis of the main prior art references cited during prosecution of the '370 Patent, in the Petition, and in Patent Owner's Preliminary Response.

31. Tikhomirov (Ex-1009) is the primary prior art reference cited during prosecution of the '370 Patent. As noted above, the approach suggested in Tikhomirov was to use a non-cleavable linker, instead of a cleavable linker, in an anti-EGFR ADC with a full antagonist anti-EGFR as the antibody. According to Tikhomirov, cetuximab is a full antagonist, so are antibodies that bind to the same epitope on EGFR as cetuximab, including those that are derived from cetuximab.

32. Wei (Ex-1005) is cited as a primary prior art reference in the Petition. Considering toxicity concerns associated with existing anti-EGFR antibodies (such as cetuximab), Wei's objective was "to provide improved anti-EGFR antibodies that exhibit increased EGFR binding activity in a tumor microenvironment compared to in a non-tumor environment." Ex-1005, ¶9. Pursuant to such an objective, Wei found that mutations at Y104 of cetuximab rendered the antibody activity dependent upon pH. These Y104 mutants, Wei describes, "exhibit[ed] greater binding activity under acidic pH conditions and/or elevated lactate levels (e.g., present in a tumor microenvironment) than under neutral pH conditions/normal lactate levels" (Ex-1005, ¶476) and thus are tumor-specific. Humanized versions of these mutants (Y104D and Y104E) were also prepared and



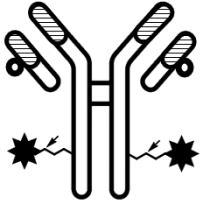
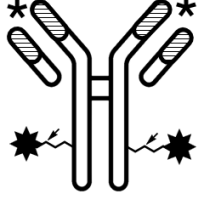
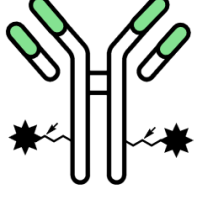
tested, including huY104D and huY104E. Ex-1005, ¶1116. The modified cetuximab antibodies in Wei “exhibit greater activity (binding affinity) under conditions of acidic pH, such as is present in a tumor microenvironment, than under conditions of neutral pH, such as exists in non-tumor tissue.” *Id.*, ¶10. Such antibodies, therefore, are preferentially activated in the tumor microenvironment and have greatly improved therapeutic index as compared to cetuximab, and are therefore consistent with Strategy B2 outlined above.

33. Leanna (Ex-1006) is another primary prior art reference cited in the Petition. In Leanna, the antibody in the anti-EGFR ADC is Antibody 1, which is a humanized version of the antibody MAb806 mentioned in Tikhomirov. MAb806 targets a mutant version of the wild-type EGFR that is only expressed in tumor cells. Antibody 1 “recognizes de2-7 EGFR and amplified EGFR, but does not recognize normal, wild-type EGFR.” Ex-1006, 24:19-24. Since only “the tumor expresses the truncated version of the EGFR de2-7” (*id.*, 12:25-13:12), Antibody 1 does not bind normal cells, and thus avoids the safety concerns associated with cetuximab. The antibody of Leanna, therefore, is consistent with Strategy B1 outlined above.

34. Finally, Patent Owner cited Tikhomirov-346 (Ex-2011) in its Preliminary Response which is the same reference discussed in Tikhomirov’s Strategy B4. More specifically, the antibodies of Tikhomirov-346 had “a binding

affinity for EGFR that is about 10-fold or more weaker than the EGFR binding affinity of cetuximab.” Ex-2011, 5:22-30. Such reduction of the affinity “substantially eliminates binding to [normal] cells presenting EGFR at a normal EGFR density, and retains effective binding at targeted disease cells that present EGFR at a greater density relative to normal cell EGFR density.” *Id.*, 3:12-16.

35. In summary, prior to filing of the '370 Patent, there was well-established strategies for developing anti-EGFR ADC that could potentially address the safety concerns associated with known anti-EGFR ADC candidates such as Cetux 2C9-MMAE. Examples described in each of the references cited by the USPTO, the Petition, and the Patent Owner are all consistent with such strategies, substantiating the validity and acceptance of these strategies. I summarize the examples in the below table:

| Benchmark ADC | Strategy | Resulting ADC | Example Antibodies/ADC |
|---|---|--|--|
|  (cetuximab-vc-MMAE) | A – non-cleavable linker for full antagonist antibody |  | - Cetux 2C9-DM1 (Tikhomirov) |
| | B2 – tumor-specific (pH-selective) antibody |  | - Y104 antibodies (Wei) |
| | B1 – tumor-specific (mutant-specific) antibody |  | - Antibody 1 (Leanna) |
| | B4 – antibody with >10-fold lower affinity than cetuximab |  | - h-R3 (Crombet) - Antibodies (Tikhomirov-346) - Nimotuzumab (Garrido) |
| Legend: Arrow (↔) – linker is cleavable Circle (○) – pH-dependency of antigen-binding fragment Asteroid (*) – mutant EGFR recognized by antibody Shaded antigen-binding fragment (◐) – EGFR-binding affinity similar to cetuximab Green antigen-binding fragment (◑) – EGFR-binding affinity lower than cetuximab | | | |

36. By the same token, anti-EGFR ADC molecules with properties contradicting such strategies would have been expected to have even elevated safety concerns. For instance, in strategy B4, using Cetux 2C9-MMAE as a reference, an ADC with an anti-EGFR antibody having a higher binding affinity

would have been expected to have a narrower or worse therapeutic window than Cetux 2C9-MMAE.

II. The '370 Patent

37. The '370 patent has a single independent claim, claim 1, which recites an antibody-drug conjugate or a pharmaceutically acceptable salt thereof, comprising an anti-epidermal growth factor receptor antibody covalently linked to a cytotoxic agent via a cleavable linker, wherein the anti-epidermal growth factor receptor antibody comprises a heavy chain and a light chain, wherein the heavy chain has a variable region comprising CDR1, CDR2 and CDR3 having sequences as shown in SEQ ID NOs: 5 to 7, and the light chain has a variable region comprising CDR1, CDR2 and CDR3 having sequences as shown in SEQ ID NOs: 12 to 14.

38. The six CDR sequences recited in claim 1 are those of antibody BA03. As noted in the application, BA03 was previously described in Liu, as a humanized version of cetuximab having increased EGFR-binding and inhibitory activity (Ex-1008, ¶140) and reduced immunogenicity (*id.*, ¶152).

A. Prosecution History

39. The '370 patent issued from U.S. Non-Provisional Patent Application No. 15/550,995 (“the '995 Application”), filed on February 16, 2016, which is a national stage application of International Application No. PCT/CN2016/073844,

filed on February 16, 2016, which claims the benefit of Chinese Application No. 201510085038.8, filed on February 17, 2015 (“the ’038.8 Provisional”).

40. The ’370 patent was allowed after three Office Actions. Each Office Action included a §103 rejection based on the combination of Tikhomirov, Liu, and other references. The Examiner cited Tikhomirov’s teachings of an “EGFR antibody in a form conjugated with an anti-microtubule toxin,” including an embodiment with “conjugation of cetuximab to MMAE anti-microtubule payload by a cleavable linker.” Ex-1004, 153-54, 244. The Examiner cited Liu for its teaching of the BA03 antibody and argued that “[i]t would have been prima facie obvious as of the effective filing date to use the BA03 antibody in place of cetuximab in the conjugates and methods of using the conjugates of Tikhomirov.” Ex-1004, 156, 247.

41. To overcome the Examiner’s §103 rejections, the Applicant explained that a POSA would not have been motivated to use Liu’s BA03 antibody as part of Tikhomirov’s ADC that has a cleavable linker, especially given that Tikhomirov teaches away from such a modification. For example, in the response to Office Action dated March 26, 2020, the Applicant explained that “one skilled in the art would not have been motivated ... to use a cleavable linker to arrive at the ADC ... as claimed.” Ex-1004, 299. The Applicant pointed out that “Figure 13 [of Tikhomirov] shows that conjugation of cetuximab to MMAE by a cleavable linker

(valine-citrulline) potentiates its toxicity against normal cells and MDA-MB-468 cancer cells, whereas conjugation via non-cleavable linker (SMCC) potentiates anti-cancer activity.” *Id.* The Applicant noted that Tikhomirov teaches away from the Examiner’s combination by stating “a safe anti-EGFR ADC should incorporate a strongly antagonistic anti-EGFR antibody linked to an anti-microtubule payload by a non-cleavable linker.” Ex-1004, 299-300. Based on Tikhomirov’s discussion of its Figure 13, the Applicant explained that “one of ordinary skill in the art would be discouraged from using a cleavable linker in incorporating a strongly antagonistic anti-EGFR antibody into an ADC due to potentiated toxicity against normal cells of such ADCs.” Ex-1004, 299-300. The Examiner issued a Notice of Allowance thereafter. Ex-1004, 306-313.

B. Priority Date

42. Dr. Bournazos opines that “at least claims 1-12 and 14-23 of the ’370 patent are not entitled to the filing date of February 17, 2015 of the ’038.8 Provisional.” Ex-1002, ¶176. I understand that the priority date issue is immaterial for determining whether the references of Grounds 1 and 2 constitute prior art and do not provide an opinion on the priority date issue in this declaration. I reserve the right, however, to supplement my opinions in the future regarding this issue.

III. Level of Ordinary Skill in the Art

43. Petitioner proposes that a person of ordinary skill in the art at the time of the invention “would typically have had a Ph.D. in immunology, molecular biology, cellular biology, or a similar field, or an MD with similar experience. A POSA would typically have had at least five years of experience with antibodies and antibody engineering, or access to other individuals with that knowledge and experience.” Pet., 32. I do not dispute Petitioner’s proposed definition for POSA. Applying Petitioner’s definition, I possessed the qualifications of a POSA as of 2015.

IV. Claim Construction

44. For purposes of this inter partes review, I have considered the claim language, specification, and portions of the prosecution history, to determine the meaning of the claim language as it would have been understood by a person of ordinary skill in the art at the time of the invention. The “plain and ordinary meaning” or *Phillips* standard has traditionally been applied in district court litigation, where a claim term is given its plain and ordinary meaning in view of the specification from the view point of a person of ordinary skill in the art. I have applied the *Phillips* standard in my analysis. Unless otherwise stated, I have applied the plain and ordinary meaning to claim terms.

V. For Ground 1, a POSA would not have found it obvious to modify Wei's ADC by replacing its antibody with that of Liu.

A. Dr. Bournazos's testimony that Liu's BA03 antibody is substantially identical to Wei's huY104D antibody is incorrect.

45. Dr. Bournazos contends that a POSA would have found it obvious to replace the antibodies in Wei's ADCs with Liu's BA03 antibody because they are substantially identical. Such an allegation is misplaced as it contradicts the specific teachings of Wei and Liu, and its error is even more pronounced in the context of ADC design strategies as I outlined above.

46. Dr. Bournazos's assertion that these antibodies are substantially identical is centrally on the ground that they are all derived from cetuximab. Such reasoning, however, clearly contradicts Wei's own teaching. As noted in Wei, a significant limitation of cetuximab is its binding to healthy cells and tissues and thus the primary objective of its invention was to "provide improved anti-EGFR antibodies that exhibit increased EGFR binding activity in a tumor microenvironment compared to in a non-tumor environment." Ex-1005, ¶9 (emphasis added). To this end, Wei identified pH-selective cetuximab derivatives that met these requirements. Examples of such pH-selective cetuximab derivatives include Y104D, which has a single mutation as compared to cetuximab. Therefore, Wei describes a cetuximab derivative, Y104D, that has a single mutation and yet has improved tumor selectivity than (hence different from) cetuximab. Wei's

teaching, therefore, directly belies Dr. Bournazos's allegation that just because two antibodies are both derived from a common parental antibody, cetuximab, they must be substantially identical.

B. The differences between Liu's BA03 antibody and Wei's Y104D/huY104D antibody would have discouraged a POSA from replacing the latter in Wei's ADC with the former.

47. Indeed, there are many significant differences between Liu's BA03 antibody and Wei's Y104D and huY104D antibodies. Contrary to Dr. Bournazos's allegations, such differences would actually have discouraged a POSA from replacing the antibody in Wei's ADC with the BA03 antibody.

1. Tikhomirov demonstrates that a POSA would have known that an ADC with cetuximab, the vc linker, and the MMAE payload was too toxic.

48. The safety concerns associated with cetuximab were well documented prior to filing of the '370 patent. For instance, Wei remarked that as anti-EGFR antibodies like cetuximab "can bind to EGFR in healthy cells and tissue [they] exhibit limitations when administered to patients." Ex-1005, ¶9.

49. Since ADCs exacerbate the toxicities of the antibodies (Ex-1009, 2), the safety concerns with cetuximab-incorporating ADCs were even more concerning. This is vividly displayed in Figure 13 of Tikhomirov which shows that Cetux 2C9-MMAE had significantly higher toxicity against skin cells than all other molecules tested. Ex-1009, Fig. 13. It cannot be denied that Cetux 2C9-MMAE has

similar toxic concentrations for both skin and tumor cells (compare the panel on the left to that on the right in Figure 13). This clearly shows that Cetux 2C9-MMAE has a unduly narrow therapeutic window.

50. Cetuximab was first approved in the US in 2004. Almost 20 years later, to my knowledge, there has been no anti-EGFR ADC candidate even in clinical trial that incorporates cetuximab conjugated to a cytotoxic payload. This fact further substantiates the unsuitability of cetuximab for ADC design.

2. The BA03 antibody does not have pH-selectivity, a hallmark property of Wei's antibodies.

51. As explained above, Wei's Y104D/huY104D antibodies were designed as improved derivatives of cetuximab with favorable properties. BA03, however, does not have such favorable properties.

52. The Y104 mutants developed by Wei have greater activity at the acidic pH of the tumor microenvironment than at a neutral pH, which is common in normal tissues. As observed by Wei, "at pH 7.4, the wild-type cetuximab antibody exhibited a slightly higher EC₅₀ than at pH 6.5 or pH 6.0. In contrast, for the Y104 mutants and the FDP-h3 control, binding was substantially weaker at pH 7.4 than at pH 6.5 or pH 6.0, as evidenced by a higher EC₅₀ under the neutral pH tested conditions than the acidic pH tested conditions. Thus, each of the mutants exhibit a greater ratio of binding at acidic pH 6.0 or 6.5 than at pH 7.4." Ex-1005, ¶10994.

53. The therapeutic index of an ADC incorporating the Y104D mutant (Y104D-MMAE), relative to that with the parental cetuximab antibody (Cetuximab-MMAE), was also measured in Example 21 of Wei.

54. The tumor cell inhibition efficacy “was virtually identical between” the two ADCs. *Id.*, ¶1130. Their toxicities against normal cells of these ADCs, however, were drastically different. The toxicities were measured in terms of rates of cell growth inhibition (CGI) against non-tumor keratinocytes. “Cetuximab-MMAE exhibited approximately 50% CGI at a concentration of approximately **0.1 µg/mL**, whereas to achieve 50% CGI, **1 µg/mL** Y104D-MMAE was required.” *Id.*, ¶1131 (emphasis added). This translates into a 10-fold difference in therapeutic index. The experimental data in Wei, therefore, demonstrates that the Y104D antibody it developed as a pH-selective derivative of cetuximab was able to increase the therapeutic index of ADCs with cetuximab by about **10-fold**. Wei additionally observes that ADCs with huY104D “exhibit[s] greater pH-dependent activity” and further “reduced growth inhibition of non-tumor keratinocytes at pH 7.4.” Ex-1005, ¶1139.

55. A POSA would have recognized the Y104D/huY104D antibody as having improved ability to distinguish tumor cells from normal cells, as compared to the parental antibody, cetuximab. Such tumor-targeting pH-selectivity of Wei’s antibodies is not a property that is possessed by a typical antibody. An antibody

that is generated through animal immunization is typically active at physiological pH conditions (e.g., 7.0-7.5) and can be the same or less active at acidic pH.

56. pH-selective antibodies are typically obtained by substituting an amino acid residue at an antigen-binding site, i.e., in the CDRs, with a histidine residue or one of the negatively-charged residues, aspartic acid or glutamic acid. The Y104D mutation is such an example, where the tyrosine (Y) residue within heavy chain CDR3 is replaced with aspartic acid (D).

57. The BA03 antibody is a humanized version of cetuximab. In a humanization process, many of the residues in the framework regions of animal origin are replaced with human counterparts. Typically, few changes are made to the CDR regions to avoid interfering with the parental antibody's binding activity. Therefore, unless specifically designed or screened for, a typical humanization process like that employed in Liu would not have conveyed tumor-targeting pH-selectivity to a parental antibody. Accordingly, there is no basis to believe that the BA03 antibody has the pH-selectivity that Wei's antibodies have.

58. Petitioner's own experiments also confirmed that BA03 does not have pH selectivity. BA03 and BA03-containing ADCs were tested in an international patent application filed by Petitioner, WO2023/088382 ("the '382 application," filed on November 17, 2022), whose counterpart Europe Application No. EP4434549A1 (Ex-2008) is in English. The '382 application describes ADC

molecules that incorporated each of four anti-EGFR antibodies, SWY2110, SWY2111, SWY2112, and SWY2113. The light chain and heavy chain sequences of SWY2110 (*see* Ex-2008, 000019) are identical to those of the BA03 antibody and thus they are the same antibody. SWY2110 (BA03) was used as the parental antibody to produce three pH-selective (also referred to as “pH-dependent” in Ex-2008) derivatives. *Id.*, 000034. The pH-selectivity of all four antibodies was measured. As shown in Table 1 (reproduced below), SWY2110 (BA03) exhibited almost identical binding affinity to EGFR at neutral (pH 7.4) and acidic (pH 6.0) conditions, and thus does not have pH-selectivity. For comparison, all three derivatives of SWY2110 (BA03) had considerably lower binding at the neutral pH and thus had pH-selectivity. *Id.*, 000035. Moreover, consistent with Wei’s teaching, Petitioner further observed that ADCs incorporating their pH-selective antibodies “had reduced toxicity to normal tissues” as compared to one with the BA03 antibody (SWY2110-JSSW-001). *Id.*, 000065. This suggests that, compared to its pH-selective derivatives, SWY2110 (BA03) has poorer ability to distinguish tumor cells from normal cells. Therefore, more than five years after the ’370 Patent was filed, Petitioner’s own data still showed that the BA03 antibody was an inferior choice as compared to its pH-selective counterparts.

Table 1 (of Ex-2008): Result of Antibody Affinity Determination

| FACS(EC ₅₀) | SWY2110 | SWY2111 | SWY2112 | SWY2113 |
|-------------------------|---------|---------|---------|---------|
| pH7.4 (nM) | 0.49 | 1.45 | 2.35 | 4.76 |
| pH6.0 (nM) | 0.51 | 0.60 | 0.67 | 3.39 |

59. Given that Wei specifically promoted its antibodies as improved versions of cetuximab with favorable properties with respect to safety and that the BA03 antibody does not have such favorable properties, it is my opinion that a POSA would certainly not have been motivated to replace the antibodies in Wei's ADC with the BA03 antibody. In particular, a POSA would have expected the replacement to increase the toxicity and harm the therapeutic window of the resulting ADC and would have avoided doing so.

3. The BA03 antibody has properties that would have been disfavored by conventional ADC design strategies.

60. As explained earlier, an important design strategy for anti-EGFR ADCs, strategy B4, involves the use of antibodies with a lower binding affinity, more specifically ones having at least 10-fold lower binding affinity than cetuximab. *Supra* §I. In particular, Crombet has demonstrated a negative correlation between an anti-EGFR antibody's affinity and its ability to distinguish tumor cells from normal cells. According to Crombet, an antibody having over 10-fold lower affinity than cetuximab would be expected to have the maximal ability in distinguishing tumor cells from normal cells; meanwhile, an antibody

having a higher affinity than cetuximab would likely have no such distinguishing ability at all. *Supra* §I.

61. As disclosed in Liu and stressed by Dr. Bournazos, the BA03 antibody has a higher binding affinity than cetuximab. Ex-1008, ¶140; Ex-1002, ¶87. According to the conventional knowledge and Crombet, therefore, a POSA would have expected the BA03 antibody to have poor or even no ability in distinguishing tumor cells from normal cells. *Supra* §I. Such an antibody, as well as its ADCs, therefore, would be expected to have no or a very narrow therapeutic window. In other words, such ADCs would be expected to be unable to avoid safety issues and be unsuccessful. A POSA would have expected that the higher binding affinity of BA03 causes the BA03-vc-MMAE ADC to have a worse therapeutic window than and be inferior to the cetuximab-vc-MMAE ADC. The cetuximab-vc-MMAE ADC was known to have an unduly narrow therapeutic window for having potentiated toxicity against normal cells, as Tikhomirov teaches. Ex-1009, 31 (“[A] safe anti-EGFR ADC should incorporate a strongly antagonistic anti-EGFR antibody linked to an anti-microtubule payload by a non-cleavable linker.”). Figure 13 of Tikhomirov shows that Cetux 2C9-MMAE had significantly higher toxicity against skin cells than all other molecules tested. Ex-1009, Fig. 13, 10-11. Because a POSA would have expected the proposed BA03-vc-MMAE ADC to have a therapeutic window that is even narrower than the

cetuximab-vc-MMAE ADC, the POSA would have had significant safety concerns about BA03-vc-MMAE and expected it to be unsuccessful. This would have discouraged a POSA from creating the BA03-vc-MMAE ADC. At a bottom line, a POSA would not have been motivated to replace the antibodies in Wei's ADC with the BA03 antibody, which would have been believed to bring about safety concerns that Wei set out to resolve.

4. Dr. Bournazos does not establish any benefit of the BA03 antibody as compared to Wei's Y104D or huY104D antibody.

62. Dr. Bournazos argues that "BA03 has several advantages over cetuximab, including stronger EGFR phosphorylation inhibition, higher ADCC activity, and significantly lower immunogenicity" and that would have motivated a POSA to create an ADC based on the BA03 antibody. Ex-1002, ¶187. I consider these arguments to lack merit. As an initial matter, these benefits are irrelevant to the proposed modification of Wei because they are benefits BA03 as compared to cetuximab, but the relied-upon ADC of Wei uses Y104D/huY104D rather than cetuximab.

63. Regardless of their relevance, the benefits would not have motivated a POSA to use the BA03 antibody in an ADC. EGFR phosphorylation inhibition is part of the antagonist activity of an anti-EGFR antibody arising from its binding to the EGFR and blocking the interaction between EGFR and its ligand EGF. To the

extent the increased EGFR phosphorylation inhibition activity of the BA03 antibody could increase the ADC's killing of tumor cells, when the ADC binds to a normal cell, however, such increased EGFR phosphorylation inhibition activity would also translate into higher killing of the normal cells, leading to toxicity. The parallel changes of the ADC's killing activity against tumor and normal cells, therefore, would mean no change to the ADC's therapeutic window. Accordingly, such an alleged benefit of the BA03 antibody would not have been considered a benefit in the context of ADC design, as it cannot at all address the fundamental concern associated with anti-EGFR ADCs, i.e., narrow therapeutic window.

64. Likewise, Dr. Bournazos alleges that the BA03 antibody has increased ADCC activity as compared to cetuximab which could lead to increased killing of tumor cells. Such increased ADCC would have been expected by the POSA to translate into increased killing of normal cells as well. Accordingly, this would not change the ADC's therapeutic window either. Therefore, the alleged increase of ADCC activity of the BA03 antibody would not have been considered by the POSA as a benefit in the context of ADC design either.

65. Finally, while the lower immunogenicity of the BA03 antibody is an advantage over the parental antibody cetuximab, I don't consider it a benefit over the antibodies of Wei. In fact, Wei's huY104D antibody is a humanized antibody. Wei has already disclosed an abundance of humanized versions of cetuximab

variations with significantly lower immunogenicity. Compared to such antibodies of Wei, I don't see an apparent benefit of replacing them with the BA03 antibody for purpose of reducing immunogenicity. Moreover, the humanized antibodies of Wei have the added benefit of enhanced tumor-targeting pH-selectivity even as compared to their own chimeric counterparts. The BA03 antibody, by contrast, does not have tumor-targeting pH-selectivity, much less at an enhanced level that is comparable to huY104D.

66. Therefore, a POSA would not have been motivated to replace the antibodies in Wei's ADC with the BA03 antibody.

VI. For Ground 2, a POSA would not have found it obvious to modify Leanna's ADC by replacing its antibody with that of Liu.

A. Leanna's antibody in the ADC recognizes tumor cells only.

67. As explained in Leanna, the antibody used in the ADC is "Antibody 1." Leanna incorporates U.S. Patent Application Publication No. 2011/0076232 by Old et al. ("Old-232") for its teachings regarding Antibody 1. Ex-1006, 24:14-17 ("The sequences and characteristics of Antibody 1 are described below (see also WO 2011/041319 and US20110076232 (see, e.g., antibody sequence of Figure 55), incorporated by reference in its entirety herein)."). Old-232 refers to Antibody 1 (shown in its Figures 55A and 55B) as the "hu806" antibody, which is a humanized version of the mouse antibody "mAb806." Ex-2015 (Old-232), ¶177 ("FIGS. 55A and 55B show the hu806 translated amino acid sequences ..."),

¶¶244-245 (explaining mouse, chimeric, and humanized versions of the 806 antibody—mAb806, ch806, and hu806). Tikhomirov uses MAb806 as an example for their strategy B1, as it “recognizes de2-7 EGFR and amplified EGFR [on tumor cells], but does not recognize normal, wild-type EGFR” on normal cells. Ex-1006 at 24. Leanna’s disclosure, therefore, is consistent with Tikhomirov’s strategy B1 that I outlined.

68. Similar to Wei’s Y104D/huY104D antibody, Leanna’s Antibody 1 (i.e., hu806) was developed to address the problem that the use of anti-EGFR antibodies was constrained by their toxicity to normal cells. *See* Ex-2015, ¶5 (“The use of [anti-EGFR] antibodies, however, may be limited by uptake in organs that have high endogenous levels of EGFR such as the liver and skin.”). A POSA would have found toxicity to liver and skin tissues undesirable and is elevated in the context of ADCs, which include highly toxic payloads. Antibody 1 solves this problem because it specifically targets an EGFR epitope that is expressed only on tumor cells and not on normal cells. Ex-2015, ¶15. The unique property of Antibody 1, therefore, is that it selectively recognizes an EGFR epitope on tumor cells and does not bind to normal cells. An ADC with such a tumor-specific antibody, therefore, would have been expected to provide an acceptable therapeutic window. A POSA would have been led by Leanna’s teachings to use antibodies that can distinguish tumor and normal cells, such as by targeting an EGFR epitope

that is only expressed on tumors, in ADCs. By the same token, a POSA reading Leanna would have had no motivation to replace the tumor-specific antibody of Leanna's ADC with one that does not have such tumor specificity.

B. BA03's lack of the tumor-specificity of Leanna's Antibody 1 would have discouraged a POSA from replacing the latter in Leanna's ADC with the former.

69. Different from Antibody 1 of Leanna, which specifically recognizes an EGFR epitope on tumor cells, the BA03 antibody does not have such specificity.

70. The BA03 antibody is a humanized derivative of cetuximab. Cetuximab has a well-recognized ability to also bind to EGFR expressed on normal cells, leading to toxicity. The BA03 antibody, therefore, would have been expected to have the same binding activity to tumor and normal cells. In other words, the BA03 antibody lacks the ability of Antibody 1 of Leanna to recognize an EGFR epitope that is only present in tumor cells.

71. The tumor specificity of Antibody 1 of Leanna, as explained in Tikhomirov, is a strategy for reducing toxicity concerns associated with anti-EGFR ADCs. Accordingly, the POSA would have had no motivation to replace Antibody 1 in Leanna's ADC with the BA03 antibody, which would contradict the teaching of these references and conventional wisdom. This is because the POSA would have expected the proposed modification of Leanna's ADC based on Liu to

significantly increase the ADC's toxicity toward normal cells at the same level of efficacy, harm the therapeutic window, and make the modified ADC substantially inferior to Leanna's ADC.

C. Dr. Bournazos does not establish any benefit of the BA03 antibody as compared to Leanna's Antibody 1.

72. Dr. Bournazos argues that BA03's advantages over cetuximab would have motivated a POSA to replace Leanna's antibody with the BA03 antibody. Ex-1002, ¶¶281-282. Similar to my discussion of Ground 1, such an argument is flawed as cetuximab is not used in Leanna's ADC and the alleged benefits are actually not benefits in the context of ADC design.

73. With respect to improved EGFR phosphorylation inhibition and ADCC, as I explained above, even if such alleged properties of the BA03 antibody were true, they are activities against both tumor cells and normal cells. Therefore, such properties would have not helped to improve the therapeutic window of an ADC that incorporates cetuximab. A POSA would have expected an ADC that incorporates the BA03 antibody to have a narrower therapeutic window than Leanna's ADC.

74. With respect to the lower immunogenicity of the BA03 antibody due to its humanization, I note that Antibody 1 is also a humanized antibody. Therefore, there is no evidence that the BA03 antibody has lower immunogenicity as compared to Antibody 1.

75. Therefore, a POSA would not have been motivated to replace the antibodies in Leanna's ADC with the BA03 antibody.

VII. A POSA would not have found it obvious to try Liu's BA03 antibody in an ADC or have had a reasonable expectation of success in combining Wei/Leanna and Liu.

A. None of the cited references provide a reason for a POSA to select Liu's BA03 antibody over all the other anti-EGFR antibodies.

76. A POSA would have understood that EGFR is different from many other antigens in terms of its presence on both normal and tumor cells. In their Preliminary Response, the Patent Owner compares the expression levels of EGFR and CD30 among different normal tissues. POPR, 5-6. The Patent Owner estimated that there is a 30-fold difference between EGFR and CD30 overall in the normal tissues. I agree that Patent Owner's estimate is correct and that this comparison underscores the particular challenge faced with anti-EGFR antibodies and ADCs. Therefore, a primary objective for designing an anti-EGFR ADC was to identify one that causes less toxicity to the normal cells. With respect to antibody selection, as I summarized above, the primary criterion is to have increased ability to distinguish tumor cells from normal cells, in order to broaden the therapeutic windows of the ADC.

77. Given these considerations, the BA03 antibody does not stand out from the hundreds of candidate antibodies as being suitable for making an ADC. In

fact, as explained above, based on known properties of BA03, a POSA would have found it a poor fit for making an ADC.

B. There were hundreds of known anti-EGFR antibodies and millions of ways to create variants of cetuximab.

78. Numerous anti-EGFR antibodies were already disclosed in the prior art references, including Tikhomirov, Tikhomirov-346, Wei, Leanna, and Liu. It was also readily appreciated by the POSA that generating new antibodies or mutated version of known antibodies takes only a routine procedure.

79. For instance, Wei has screened for hundreds or even thousands of mutant forms of cetuximab (*see, e.g.*, Tables 32-34) and even more humanized ones.

80. From such a large number of anti-EGFR antibodies that were known or could have been readily produced, it was not obvious to select the BA03 antibody for inclusion in an ADC, in particular given its properties (e.g., lack of tumor selectivity, high binding affinity) that would have been viewed unfavorably by the POSA.

C. Dr. Bournazos does not establish a reasonable expectation of success.

81. I was asked to opine on whether Dr. Bournazos has established that a POSA would have had a reasonable expectation of success combining Wei and Liu (Ground 1) and Leanna, Liu, and Wei (Ground 2) in the manner suggested by Dr.

Bournazos. I reviewed Dr. Bournazos's declaration and did not find any explicit discussion on the reasonable expectation of success issue for Ground 1. For Ground 2, Dr. Bournazos argues that a POSA would have had a reasonable expectation of success for the reason that "Leanna teaches detailed processes for preparing the Antibody 1-vc-MMAE ADC..." Ex-1002, ¶282. I note that Leanna's teaching is directed to its unmodified ADC. Given that Liu's BA03 is different from Leanna's Antibody 1, it is unclear based on Dr. Bournazos's declaration how Leanna's process for preparing the Antibody 1-vc-MMAE ADC is relevant to whether there was a reasonable expectation of success for the BA03-vc-MMAE ADC proposed under Ground 2.

82. Designing an ADC is a sophisticated process. In determining whether an ADC is expected to be successful, a POSA would have analyzed a series of factors. The '370 patent provides a good discussion of factors that a POSA would have considered in forming an expectation as to whether an ADC would have been successful:

1) characteristics of target: whether a target antigen can be internalized or not, the expression level of the target antigen, differentials of expression level of target antigen between cancer cells and normal cells, and whether the target antigen has an extracellular domain (ECD) that is soluble in blood;

- 2) characteristics of monoclonal antibody: specificity of the monoclonal antibody to the target antigen (preferably no cross-reaction with other proteins), stability of the monoclonal antibody, and whether the complex of the monoclonal antibody and the target can be endocytosed into the cell;
- 3) characteristics of small molecule drug: potency of the cytotoxicity of the small molecule drug, stability thereof in blood, and toxicity of the *in vivo* metabolites of the ADC containing the small molecule drug;
- 4) characteristics of linker: whether the linker is cleavable or non-cleavable, and stability of the linker in blood;
- 5) ADC characteristics: whether the complex of ADC and target antigen can be internalized or not, stability of ADC in blood, linker used and number of chemical drugs conjugated to the ADC, the balance between cancer killing activity of ADC and its toxicity.

Ex-1001, 3:10-37.

83. I observe that Dr. Bournazos's declaration does not discuss the vast majority of these considerations, especially with respect to the question of whether there would have been a reasonable expectation of success.

84. Considering the factors "differentials of expression level of target antigen between cancer cells and normal cells" and "the balance between cancer killing activity of ADC and its toxicity," a POSA would have realized that the targeted EGFR antigen has relatively high expression on both cancer and normal cells. The difference in expression level of EGFR between cancer and normal cells

is much lower than many other antigens, such as the CD30 antigen. EGFR's low differential of expression level between cancer and normal cells would have made it particularly challenging to balance cancer killing activity and toxicity of a potential ADC incorporating an anti-EGFR antibody. A POSA would have expected such a balance to be impossible for the proposed BA03-vc-MMAE ADC because (1) the BA03 antibody has no ability to distinguish tumor and normal cells and (2) BA03's higher-than-cetuximab binding affinity would have caused the BA03-vc-MMAE ADC to have an even narrower therapeutic window than the cetuximab-vc-MMAE ADC, which was known to be unviable.

VIII. The claimed invention addresses a long-felt and unresolved need for a clinically viable anti-EGFR ADC.

85. EGFR is frequently overexpressed on tumor cells and thus is an attractive target for anti-tumor therapy. There are currently at least five EGFR-targeting antibodies approved worldwide, including cetuximab (Erbix[®]), panitumumab (Vectibix[®]), nimotuzumab (BIOMAB-EGFR[®]), necitumumab (Portrazza[®]) and amivantamab (Rybrevant[®]). Cetuximab was first approved for medical use in the United States in 2004. At least as early as in 2012, clinical trials with anti-EGFR ADC were submitted. For example, clinical trial NCT01741727 was first submitted on September 19, 2012 (*see* "First Submitted" date on Ex-2028, 00008). This clinical trial proposes to study ABT-414, an anti-EGFR ADC, in patients with solid tumors. "ABT-806 is a humanized form for the monoclonal

antibody mAb 806.” Ex-2029, 000001. ABT-806, therefore, corresponds to Antibody 1 of Leanna. However, “enrollment in [a phase II/III study] has been halted since 2019 due to lack of survival benefit for patients receiving ABT-414.” Ex-2030, 000004.

86. Subsequently, more anti-EGFR ADCs entered into clinical trials and were terminated. The following table summarizes a few important clinical candidates and their statuses.

| Sponsor | ADC Name | EGFR antibody | Status |
|----------------|-------------------------------------|---------------------------------------|---------------|
| AbbVie | ABT-414 | EGFRvIII-specific | Terminated |
| AbbVie | ABBV-221 (losatuxizumab vedotin) | EGFRvIII-specific | Terminated |
| AbbVie | ABBV-321 (serclutamab talirine) | EGFRvIII-specific | Terminated |
| Halozyme | HTI-1511 | Tumor microenvironment (TME)-specific | Terminated |

87. AbbVie’s drug candidate ABBV-221 (losatuxizumab vedotin) is also an anti-EGFR ADC. The antibody in ABBV-221, AM1, is “an affinity-matured [anti-EGFR antibody] ABT-806” and binds EGFRvIII. Ex-2013, 000004. After the report in Ex-2013 that “ABBV-221 has advanced to a phase I clinical trial” as of 2018 (id., 000001), there has been no report of phase I results or phase 2 or 3 testing. In fact, the Phase I study of ABB-221 has reportedly been terminated. Ex-2016, 000001.

88. Another ADC drug candidate by AbbVie is ABBV-321 (serclutamab talirine). This ADC includes the same AM-1 antibody as ABBV-221. Ex-2014, 000002. AbbVie reported phase 1 results in Ex-2014 in 2022. Nevertheless, no further phase 2 or 3 testing has been reported, suggesting that its development has been terminated.

89. Halozyme reported the preclinical testing of HTI-1511 in 2016. *See* Ex-2012. HTI-1511 is an anti-EGFR ADC that includes an antibody which “was engineered with increased tumor microenvironment (TME) specificity for EGFR” (Ex-2012, 000001), suggesting that it is a pH-selective antibody. Like the pH-selective antibodies of Wei, the antibody in HTI-1511 “demonstrated undetectable *in vivo* binding to human donor foreskins grafted onto nude mice, while binding to human A431 tumor xenografts with similar intensity to cetuximab.” *Id.* Since 2016, however, there has been no report of clinical testing for HTI-1511, suggesting that its development has been terminated.

90. The fact that EGFR is an attractive target for anti-tumor therapy and the industry has had a strong interest in developing anti-EGFR ADCs shows that there has been a long-felt and unresolved need for a clinically viable anti-EGFR ADC. The failures by others in moving candidate anti-EGFR ADCs through preclinical or clinical development demonstrate that the claimed ADC was non-obvious.

IX. The clinical success of MRG003 underscores the non-obviousness of claim 13.

91. Claim 13 further specifies the cleavable linker as the vc linker and the cytotoxic payload as MMAE. Such a specific ADC was tested in the experimental examples of the '370 patent, referred to as MYK-3. Against colorectal cancer cells, MYK-3 exhibited an inhibitory activity “much higher than those of monoclonal antibody BA03 itself and BA03 plus free vcMMAE.” Ex-1001, 19. More importantly, in an *in vivo* safety testing of Example 3, “MYK-3 showed no significant change [of body weight] as compared with the control group (*see* FIG. 8), indicating that MYK-3 had no[] toxic effect of reducing body weight of mice.” *Id.*

92. Such an excellent efficacy-safety profile of MYK-3 (now known as MRG003 or becotatug vedotin) is further substantiated with its clinical success. On October 30, 2025, Patent Owner received approval for its MRG003 in China for the treatment of certain nasopharyngeal carcinoma. Ex-2021, 000001. As the first regulatory approved anti-EGFR ADC worldwide after over 20 years since the approval of the anti-EGFR antibody drug cetuximab, the success of MRG003 as prescribed in claim 13 would have been viewed by a POSA as unexpected. Such a success, therefore, underscores the non-obviousness of the claimed invention.

X. Background and Qualifications

93. This section contains a summary of my educational background, career history, publications, and other relevant qualifications. My full curriculum vitae is attached as Appendix 1 to this declaration.

94. I received my medical degree (M.D.) from the Free University of Berlin. I completed postgraduate clinical training in internal medicine, hematology, oncology, and stem cell transplantation at the University Medical Center Hamburg-Eppendorf in Germany. I also completed a research fellowship in tumor immunology at the Ludwig Institute for Cancer Research at Memorial Sloan-Kettering Cancer Center in New York. I am board-certified in Internal Medicine and in Hematology and Oncology.

95. I am currently a Professor of Medicine in the Department of Internal Medicine at the University of Maryland School of Medicine and a member of the University of Maryland Marlene and Stewart Greenebaum Comprehensive Cancer Center. I serve as Director of Cancer Immunotherapy and as Director of the Fannie Angelos Cellular Therapeutics GMP Laboratory. Prior to my current appointment, I held faculty and leadership positions at the University of Utah Huntsman Cancer Institute and at the University Medical Center Hamburg-Eppendorf. My academic and translational work has focused on antibody-based therapeutics, antibody-drug

conjugates, and cellular immunotherapies, and I have been continuously engaged in this field well before 2015.

96. I have received multiple research and translational science awards recognizing contributions to cancer immunology and therapeutic development, including national and international awards for work in antibody-based and cellular immunotherapies. These include competitive investigator awards, foundation-supported translational research awards, and institutional honors for scientific and clinical contributions.

97. As a faculty member at academic medical centers, I have participated in the education and training of medical students, residents, fellows, and graduate students in internal medicine, hematology/oncology, cancer immunology, and translational research. My teaching has included formal lectures, small-group instruction, and mentorship in laboratory-based and clinical research settings relevant to biologic and antibody-based therapeutics.

98. I have authored or co-authored over 150 peer-reviewed scientific publications in the fields of cancer immunology, antibody therapeutics, and translational oncology. My publications include original research articles, reviews, and translational studies addressing antibody structure and function, antigen targeting, therapeutic index, and safety considerations. Based on my education, training, and experience, including my work prior to and as of 2015, I possessed

the qualifications of a person of ordinary skill in the art as that term is defined in this proceeding.

A. Compensation

99. For my efforts in connection with the preparation of this declaration I have been compensated at my standard rate for this type of consulting activity. My compensation is in no way contingent on the results of these or any other proceedings relating to the above-captioned patent.

B. Materials and Other Information Considered

100. I have considered information from various sources in forming my opinions. I have reviewed and considered each of the exhibits listed in the attached Appendix 2 (Materials Considered in the Preparation of This Declaration) in forming my opinions.

XI. Understanding of the Law

101. I am not an attorney. In forming my opinions in this Declaration, I applied the relevant legal principles provided to me by counsel, which are summarized in Appendix 3.

XII. Reservation of Rights

102. My opinions are based upon the information that I have considered to date. I reserve the right, however, to supplement my opinions in the future to respond to any arguments raised by Petitioner and to take into account new information that becomes available to me.

103. I declare that all statements made herein of my knowledge are true, and that all statements made on information and belief are believed to be true, and that these statements were made with the knowledge that willful false statements and the like so made are punishable by fine or imprisonment, or both, under Section 1001 of Title 18 of the United States Code.

Executed on January 21, 2026.

By:



Djordje Atanackovic

APPENDIX 1: CURRICULUM VITAE OF DJORDJE ATANACKOVIC

CURRICULUM VITAE

NAME: Djordje Atanackovic

DATE: 01/20/2026

PRESENT POSITION AND ADDRESS

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II. EDUCATION AND TRAINING

Undergraduate and Graduate

04/1993 – 11/1998 Free University Berlin, Germany
Medicine
Degree: M.D.

06/1998-09/1998 Internship / Surgery, Hotel Dieu, Paris, France
04/1998-05/1998 Internship / Internal Medicine, University of California San Diego
02/1998-04/1998 Internship / Internal Medicine, University of Chicago
04/1992 – 02/1993 University of Hannover, Germany
Medicine
04/1994 – 11/1998 Free University Berlin, Germany
Philosophy, Psychology

Postgraduate

07/2003 – 03/2009 Clinical Fellowship, Internal Medicine and Oncology / Hematology / Stem Cell Transplantation, University Medical Center Hamburg-Eppendorf, Hamburg, Germany
10/2000 – 06/2003 Research Fellowship, Tumor Immunology, Ludwig Institute for Cancer Research at Memorial Sloan-Kettering Cancer Center, New York City
01/1999 – 09/2000 Residency, Internal Medicine and Oncology / Hematology / Stem Cell Transplantation, University Medical Center Hamburg-Eppendorf, Hamburg, Germany

Certifications

Curriculum Vitae (Djordje Atanackovic, M.D.)

| | |
|---------|--|
| 03/2019 | ECFMG Certification |
| 04/2011 | Habilitation in Internal Medicine (title of thesis: „The role of cancer-testis antigens in the immune control of solid tumors and hematologic malignancies”), University of Hamburg, Germany |
| 08/2010 | Board Certification in Oncology/Hematology, Hamburg, Germany |
| 04/2009 | Board Certification in Internal Medicine, Hamburg, Germany |

United States Medical Licensing Examinations

| | |
|---------|-----------------|
| 07/2019 | USMLE Step 3 |
| 02/2019 | USMLE Step 2 CK |
| 08/2018 | USMLE Step 1 |
| 04/2018 | USMLE Step 2 CS |

Licensures

| | |
|-------------------|---|
| 10/2020 – 09/2026 | State of Maryland Medical License (D0089361) |
| 09/2019 – 06/2028 | Maryland Controlled Substance License (FA8792574) |
| 07/2019 – 01/2028 | State of Utah Medical License (8702756-1205) |
| 10/2013 – 09/2018 | State of Utah Medical License (8702756-1252) |
| 10/2019 – 01/2028 | Utah Controlled Substance License (8702756-8905) |
| 10/2013 – 09/2018 | Utah Controlled Substance License (8702756-8952) |
| 07/2000 – present | State Medical License, Berlin, Germany |

III. PROFESSIONAL EXPERIENCE

Academic Appointments

| | |
|-------------------|--|
| 08/2020 – present | Professor of Medicine, University of Maryland / Greenebaum Comprehensive Cancer Center |
| 01/2014 – 08/2020 | Associate Professor of Medicine, University of Utah / Huntsman Cancer Institute |
| 01/2018 – present | Adjunct Associate Professor, Department of Pathology/Division of Microbiology and Immunology, University of Utah |
| 04/2011 – 12/2013 | Assistant Professor of Medicine, University Medical Center Hamburg-Eppendorf |

Administrative Appointments (Clinical)

| | |
|-------------------|--|
| 08/2020 – present | Director of Cancer Immunotherapy, University of Maryland Greenebaum Comprehensive Cancer Center |
| 08/2020 – present | Director of the Fannie Angelos Cellular Therapeutics GMP Laboratory, University of Maryland Greenebaum Comprehensive Cancer Center |
| 08/2020 – present | Member, Multiple Myeloma Program, Bone Marrow Transplant and Cellular Therapy (TCT) Program, University of Maryland Greenebaum Comprehensive Cancer Center |
| 01/2014 – 08/2020 | Director of the Multiple Myeloma Program, Hematology and Hematologic Malignancies, University of Utah / Huntsman Cancer Institute |

Curriculum Vitae (Djordje Atanackovic, M.D.)

| | |
|-------------------|---|
| 01/2014 – 08/2020 | Director of Cancer Immunotherapy, University of Utah / Huntsman Cancer Institute |
| 04/2011 – 12/2013 | Assistant Professor of Medicine, Oncology / Hematology / Stem Cell Transplantation, University Medical Center Hamburg-Eppendorf |
| 07/2010 – 12/2013 | Attending Physician, Oncology / Hematology / Stem Cell Transplantation, University Medical Center Hamburg-Eppendorf |
| 01/2013 – 12/2013 | Head of Inpatient Unit, Oncology / Hematology / Stem Cell Transplantation, University Medical Center Hamburg-Eppendorf |
| 01/2011 – 05/2012 | Head of outpatient clinic, Oncology / Hematology / Stem Cell Transplantation, University Medical Center Hamburg-Eppendorf |

Administrative Appointments (Research)

| | |
|-------------------|---|
| 08/2020 – present | Head of the Laboratory for Cancer Immunotherapy, University of Maryland Greenebaum Comprehensive Cancer Center |
| 08/2020 – present | Member, Tumor Immunology and Immunotherapy (TII) Program, University of Maryland Greenebaum Comprehensive Cancer Center |
| 01/2014 – 08/2020 | Head of the Laboratory for Cancer Immunotherapy, Hematology and Hematologic Malignancies, University of Utah / Huntsman Cancer Institute |
| 07/2003 – 12/2013 | Head of the Laboratory for Tumor Immunology, Oncology / Hematology / Stem Cell Transplantation, University Medical Center Hamburg-Eppendorf |

IV. RESEARCH AND CLINICAL INTERESTS

Research Interests

- Discovery and validation of novel antigen targets for immunotherapy in hematologic malignancies (esp. multiple myeloma, B-cell lymphomas, T-cell leukemias) and selected solid tumors (e.g., prostate cancer, lung cancer, hepatocellular carcinoma).
- Translational development of next-generation cellular immunotherapies, including first-in-human CAR T cell trials, trispecific CAR platforms, and innovative constructs (e.g., CD229, SLAMF3, TGF β RII-DN, ROR1).
- Integration of immunomonitoring, biomarker development, and mechanistic correlative science into clinical trials to guide therapy optimization and overcome resistance mechanisms.
- Advancement of immunotherapy technologies in GMP settings, with emphasis on closed-system manufacturing, quality assurance, and scalable translation from bench to bedside.

Clinical Interests

- Comprehensive care of patients with plasma cell dyscrasias (multiple myeloma, amyloidosis) and other hematologic malignancies, with focus on cellular immunotherapies (CAR T, TCR-T, NK

cells), autologous and allogeneic stem cell transplantation, and novel antibody/immunomodulatory regimens.

- Development of multidisciplinary clinical programs that integrate early-phase clinical trials, translational science, and patient-centered care pathways.
- Mentorship of trainees and faculty in hematology/oncology with a commitment to building sustainable academic programs in cellular therapy and cancer immunology.

V. HONORS AND AWARDS

Stipends

| | |
|-----------|--|
| 2006-2007 | Stipend of the University of Hamburg for Excellent Young Researchers. |
| 2002-2003 | Stipend of the Cancer Research Institute for a postdoctoral fellowship at the New York branch of the Ludwig Institute for Cancer Research. |
| 2000-2002 | Stipend of Deutsche Krebshilfe (German Cancer Aid) for a postdoctoral fellowship at the New York branch of the Ludwig Institute for Cancer Research. |

Awards

| | |
|---------|---|
| 09/2025 | Bob Bast Translational Research Award of the V Foundation for the research project "CD229-targeting CAR T cells as a novel cellular immunotherapy for T cell leukemias". |
| 07/2018 | Huntsman Translational Scholar Award |
| 12/2016 | Brian D. Novis Senior Research Award of the International Myeloma Foundation (IMF) for the research project "Anti-CD229 chimeric antigen receptor T Cells for the treatment of multiple myeloma". |
| 07/2015 | PPH/CCTS Award of the University of Utah for the research project "Development of a combination cellular immunotherapy for multiple myeloma". |
| 11/2012 | Science Award of the Hamburg Cancer Society (Hamburger Krebsgesellschaft) for the study „Role of interleukin 16 in multiple myeloma". |
| 09/2012 | Clinical Science Award of the German Society for Immune and Targeted Therapy for the study "Surface molecule CD229 as a novel target for the diagnosis and treatment of multiple myeloma". |
| 09/2009 | Research Award of the Hubertus Wald Foundation |
| 11/2007 | Research Award of the Foundation for Research on Head and Neck Cancer for studies on the expression of cancer-testis antigens in patients with head and neck cancer. |
| 02/2007 | Investigator Award of the Cancer Research Institute for studies on cancer-testis antigen-specific immunity in patients with multiple myeloma. |
| 06/2007 | Dr. Martini Award for the publication "Cancer-testis antigens are commonly expressed in multiple myeloma and induce systemic immunity following allogeneic stem cell transplantation". |
| 06/2004 | Poster prize of the 25th European Conference on Psychosomatic Research for the study "Acute psychological stress causes a redistribution of memory/effector T cell subsets in humans". |

VI. SERVICE

Intramural

| | |
|-------------------|---|
| 09/2025 – present | Member, COE Internal Advisory Committee (IAC), University of Maryland Marlene and Greenebaum Comprehensive Cancer Center |
| 03/2025 – present | Member, GMP Advisory Council, University of Maryland Marlene and Greenebaum Comprehensive Cancer Center |
| 10/2024 – present | Member, Data Safety Monitoring Committee (DSMC) of the University of Maryland Marlene and Greenebaum Comprehensive Cancer Center |
| 02/2022 – present | Member, Admissions Committee, Nathan Schnaper Intern Program in Translational Cancer Research (NSIP), University of Maryland Marlene and Greenebaum Comprehensive Cancer Center |
| 06/2024 – present | Reviewer, MMI Program Graduate Student Symposium “June Presentations”, University of Maryland School of Medicine |
| 02/2022 – present | Member, Admissions Committee, Nathan Schnaper Intern Program in Translational Cancer Research (NSIP), University of Maryland Marlene and Greenebaum Comprehensive Cancer Center |
| 11/2022 – present | Judge, Annual Medical Student Research Day Poster Presentations, University of Maryland School of Medicine |
| 10/2021 – present | Interviewer, Internal Medicine Residency Program, University of Maryland School of Medicine |
| 10/2021 – present | Interviewer, Hematology/Oncology Fellowship Program, University of Maryland and Greenebaum Comprehensive Cancer Center |
| 08/2020 – present | Member, Tumor Immunology and Immunotherapy (TII) Program, University of Maryland Marlene and Greenebaum Comprehensive Cancer Center / University of Maryland School of Medicine |
| 01/2019 – 07/2020 | Member, Syn-Tegration Committee, Huntsman Cancer Institute |
| 07/2018 – 07/2020 | Member, Society of Huntsman Translational Scholars, Huntsman Cancer Institute |
| 07/2018 – 07/2020 | Interviewer, Molecular Biology and Biological Chemistry Programs, University of Utah |
| 01/2017 – 07/2020 | Member, Hematology Division Leadership Council, Huntsman Cancer Institute |
| 07/2016 – 07/2020 | Co-chair, Cancer Immunotherapy Group of the University of Utah’s Inflammation, Immunity, and Infection (III) Initiative |
| 07/2016 – 07/2020 | Co-leader, Cancer Immunotherapy Working Group of HCI’s Strategic Planning Group |
| 07/2016 – 07/2020 | Member, Spinal Oncology Treatment Planning Conference at Huntsman Cancer Institute |
| 03/2016 – 07/2020 | Member, Chimeric Antigen Receptor (CAR) T cell Therapy Task Force |
| 09/2014 – 07/2020 | Member, Disease-Oriented Research Team (DOT) “Hematologic Malignancies” at Huntsman Cancer Institute |
| 03/2014 – 07/2020 | Member, Experimental Therapeutics (ET) Program at Huntsman Cancer Institute |

Curriculum Vitae (Djordje Atanackovic, M.D.)

01/2014 – 07/2020 Member, Treatment-Planning Conference (TPC) of the Department of Hematology and Hematologic Malignancies at Huntsman Cancer Institute

Extramural

08/2022 Abstract Reviewer, 2022 Annual Meeting of the Society for Immunotherapy of Cancer (SITC)

12/2020 – present Member, Society for Immunotherapy of Cancer (SITC) Biomarkers Committee

11/2019 – present Member, American Association for Cancer Research (AACR) Steering Committee of the Cancer Immunology Working Group (CIMM)

01/2014 – 07/2017 Representative of Huntsman Cancer Institute on the NCCN Multiple Myeloma Panel

07/2003 – present Member, Cancer Vaccine Collaborative (CVC) of the Cancer Research Institute (CRI)

Editorial Boards

07/2019 – present Editor, *Cancers*

02/2016 – 06/2019 Associate Editor, *Journal for Immunotherapy of Cancer (JITC)*

08/2012 – present Associate Editor, *BMC Cancer*

Journal Reviewer

09/2007 – present *Blood*

05/2017 – present *Journal of Clinical Investigation*

07/2023 – present *Nature Communications*

04/2014 – present *Lancet Oncology*

03/2019 – present *Lancet Haematology*

05/2018 – present *Leukemia*

11/2009 – present *Cancer Research*

10/2008 – present *Clinical Cancer Research*

11/2009 – present *Haematologica*

09/2018 – present *Journal for Immunotherapy of Cancer*

02/2022 – present *Blood Advances*

01/2018 – present *Journal of Hematology & Oncology*

08/2008 – present *British Journal of Cancer*

10/2021 – present *Clinical Infectious Diseases*

05/2019 – present *Translational Oncology*

05/2008 – present *International Journal of Cancer*

08/2004 – present *American Journal of Hematology*

10/2011 – present *Leukemia Research*

12/2014 – present *Hematology*

09/2019 – present *Hematology Reports*

02/2013 – present *Leukemia and Lymphoma*

08/2011 – present *BMC Cancer*

Curriculum Vitae (Djordje Atanackovic, M.D.)

| | |
|-------------------|--|
| 01/2013 – present | PLOS ONE |
| 03/2017 – present | Cancer Letters |
| 02/2016 – present | Expert Review of Hematology |
| 12/2019 – present | Expert Review of Molecular Diagnostics |
| 01/2016 – present | Oncotarget |
| 10/2017 – present | Oncoimmunology |
| 10/2008 – present | Immunotherapy |
| 09/2019 – present | International Immunopharmacology |
| 11/2019 – present | Immunologic Research |
| 12/2014 – present | Critical Reviews in Oncology/Hematology |
| 05/2010 – present | Journal of Translational Medicine |
| 06/2009 – present | Journal of Leukocyte Biology |
| 03/2006 – present | Cancer Immunology Immunotherapy |
| 12/2018 – present | Cellular and Molecular Immunology |
| 11/2019 – present | Cells |
| 12/2005 – present | Tumor Biology |
| 10/2017 – present | JCI INsight |
| 10/2013 – present | Molecular Cancer |
| 11/2019 – present | OncoTargets and Therapy |
| 04/2016 – present | JAMA Oncology |
| 11/2019 – present | Military Medical Research |
| 02/2007 – present | Journal of Psychosomatic Medicine |
| 12/2005 – present | Psychoneuroendocrinology |
| 09/2010 – present | Stress |
| 07/2008 – present | Journal of Neuroimmunology |
| 06/2010 – present | Brain, Behavior, and Immunity |
| 11/2014 – present | Expert Opinion on Therapeutic Targets |
| 11/2013 – present | Expert Opinion on Biological Therapy |
| 03/2009 – present | Transplant Immunology |
| 12/2014 – present | Acta Haematologica |
| 07/2005 | American Journal of Hypertension |
| 03/2010 | American Journal of Respiratory and Critical Care Medicine |
| 09/2007 – 2011 | Cancer Immunity |
| 08/2007 – present | Cancer Science |
| 02/2009 | Cellular Therapy and Transplantation |
| 03/2013 – present | Clinical and Developmental Immunology |
| 07/2008 | Clinical Medicine |
| 01/2014 – present | Hematological Oncology |
| 09/2011 – present | Lung |
| 04/2014 – present | Oncology Research and Treatment |

Grant Reviewer

| | |
|-------------------|--|
| 01/2026 – present | V Foundation Translational Cancer Research Review Panel |
| 07/2025 – present | NCI P50 Research Center Grants for Specialized Programs of Research Excellence (SPORE) Study Section of the NIH/NCI; Translational Cancer Research SPORE |
| 06/2024 – present | Cellular Immunotherapy of Cancer (CIC) Study Section of the NIH/NCI |
| 03/2021 – present | American Association for Cancer Research (AACR), Immunooncology Research Grant |
| 07/2019 – present | Multiple Myeloma Research Foundation (MMRF), Fellowship Program |
| 07/2019 – present | Multiple Myeloma Research Foundation (MMRF), Myeloma Immune Translational Research Accelerator Program |
| 11/2016 – present | Department of Defense Congressionally Directed Medical Research Programs (CDMRP) |
| 01/2006 – present | European Commission (6th and 7th Framework Programs, Horizon 2020 Program, Horizon Europe Program) |
| 03/2019 – present | European Hematology Association (EHA) |
| 09/2019 – present | Worldwide Cancer Research |
| 01/2022 – present | Sarcoma UK |
| 01/2022 – present | Blood Cancer UK |
| 10/2021 – 09/2022 | Little Warrior Foundation |
| 09/2012 – present | Leukemia & Lymphoma Research U.K. |
| 11/2011 – present | Cancer Research UK |
| 04/2015 | Italian Ministry of Health |
| 01/2012 – present | Research Foundation Flanders, Belgium |
| 05/2012 | Catalan Agency for Health Information, Spain |
| 08/2016 | Israeli Ministry of Science and Technology |
| 10/2016 | National Science Center, Poland |
| 09/2013 – present | Flemish League against Cancer |
| 10/2015 – 07/2020 | University of Utah (Funding Incentive Seed Grant Program) |
| 03/2017 | Dr. Werner Jackstädt Foundation, Germany |
| 09/2008 | FWF Research Foundation, Austria |

Memberships in Professional Societies

- American Society of Hematology (ASH)
- American Society of Clinical Oncology (ASCO)
- American Association for Cancer Research (AACR)
- European Society for Blood and Marrow Transplantation (EBMT)
- Society for Immunotherapy of Cancer (SITC)
- European Hematology Association (EHA)

Pharmaceutical Board Memberships

| | |
|-------------------|---|
| 07/2019 – 07/2020 | Advisory Board, Member, Isatuximab in Multiple Myeloma, Sanofi |
| 10/2016 – 07/2020 | Advisory Board, Member, Hematology Summit Advisory Board, Amgen |

Curriculum Vitae (Djordje Atanackovic, M.D.)

| | |
|-------------------|--|
| 08/2017 – 07/2020 | Advisory Board, Member, Multiple Myeloma Clinical Advisory Board, Juno Therapeutics |
| 08/2016 – 07/2020 | Advisory Board, Member, Multiple Myeloma Transplanter Advisory Board, Celgene |
| 02/2016 – 07/2020 | Advisory Board, Member, Best Practice Strategy for the Use of Gene Expression Profiling in Multiple Myeloma, Signal Genetics Inc. |
| 09/2015 – 07/2020 | Advisory Board, Member, Ixazomib in Multiple Myeloma, Millennium Pharmaceuticals Inc. |
| 12/2014 – 07/2020 | Advisory Board, Member, Carfilzomib in Multiple Myeloma, Amgen |
| 03/2012 – 12/2013 | Advisory Board, Member, Pemetrexed for Non-Small Cell Lung Cancer (NSCLC), Lilly |
| 10/2010 – 12/2013 | Advisory Board, Member, Afatinib for Non-Small Cell Lung Cancer (NSCLC), Boehringer Ingelheim Pharma GmbH & Co. |
| 12/2011– 07/2013 | Advisory Board, Member, MAGE3-AS15-NSC-003 clinical trial “A double-blind, randomized, placebo-controlled Phase III study to assess the efficacy of recMAGE-A3 + AS15 Antigen Specific Cancer Immunotherapeutic as adjuvant therapy in patients with resectable MAGE-A3-positive Non-Small Cell Lung Cancer” (MAGRIT), GlaxoSmithKline Biologicals |
| 08/2014 – 07/2017 | Data Monitoring Committee, Member, WT1-AS01B-AML-001 clinical trial “A Phase I study to assess the safety and immunogenicity of WT1-A10 + AS01B Antigen-Specific Cancer Immunotherapeutic as post-consolidation therapy in adult patients with WT1-positive Acute Myeloid Leukemia in complete remission”, GlaxoSmithKline Biologicals |
| 08/2014 – 07/2017 | Data Monitoring Committee, Member, WT1-AS01B-AML-002 clinical trial “A Phase I study to assess the safety and clinical activity of WT1-A10 + AS01B Antigen-Specific Cancer Immunotherapeutic in adult patients with WT1-positive Acute Myeloid Leukemia in partial remission or complete remission with incomplete blood count recovery post-induction therapy”, GlaxoSmithKline Biologicals |
| 08/2008 – 11/2014 | Global Steering Committee, Member, MAGE3-AS15-NSC-003 clinical trial “A double-blind, randomized, placebo-controlled Phase III study to assess the efficacy of recMAGE-A3 + AS15 Antigen Specific Cancer Immunotherapeutic as adjuvant therapy in patients with resectable MAGE-A3-positive Non-Small Cell Lung Cancer” (MAGRIT), GlaxoSmithKline Biologicals |
| 01/2012 – 04/2014 | Independent Data Monitoring Committee, Member, 1122P1811 clinical trial “A Phase 1/2 Study to Evaluate the Safety, Tolerability, Immune Response, and Clinical Efficacy of Cancer Peptide Vaccine S-488210 in Patients with Unresectable Locoregionally Recurrent and/or Metastatic Head and Neck Squamous Cell Carcinoma (HNSCC)”, Shionogi & Co., Ltd. |
| 09/2009 – 09/2011 | Data Safety Monitoring Board, Member, CV-9201-003 clinical trial “Safety and efficacy phase I/IIa trial of an RNActive®-derived cancer vaccine in stage IIIB/IV non small cell lung cancer (NSCLC)”, CureVac GmbH |

VII. CLINICAL STUDIES

Principal Investigator

| | |
|-----------------|---|
| 04/2024-present | 2327GCCC: A Phase Ia open-label Study of Tri-specific autologous CAR19.20.22 Chimeric Antigen Receptor (CAR) T-cells for Patients with Relapsed/Refractory B-Cell Lymphomas |
| 04/2022-present | 2043GCCC: Tissue Sampling and Biorepository to improve Cancer Cell Therapy |
| 2021 – 2022 | 2123GCCC: Monitoring of immune responses to the COVID-19 vaccine in patients with cancer or organ transplant |
| 2018 – 2020 | A Phase 1b Study of SAR650984 (isatuximab) in Combination with Pomalidomide and Dexamethasone for the Treatment of Relapsed/Refractory Multiple Myeloma |
| 2018 – 2020 | A Phase III Study Evaluating the Safety and Efficacy of BL-8040 and G-CSF compared with Placebo and G-CSF for the Mobilization of Hematopoietic Stem Cells for Autologous Transplantation in Patients with Multiple Myeloma - The GENESIS Study |
| 2017 – 2018 | A phase 1/2, open-label safety, pharmacokinetic, and efficacy study of TAS4464 in patients with Multiple Myeloma or Lymphoma |
| 2017 – 2018 | GO29695: A Phase Ib Study of the Safety and Pharmacokinetics of Atezolizumab alone or in Combination with IMid +/- Daratumumab in patients with Multiple Myeloma (Relapsed/Refractory and Post Autologous Stem Cell Transplantation) |
| 2016 – 2020 | Solitary Plasmacytoma of Bone: Randomized Phase III trial to evaluate treatment with adjuvant systemic treatment and zoledronic acid versus zoledronic acid after definite radiation therapy. |
| 2015 – 2017 | A phase III study of Pomalidomide and low dose Dexamethasone with or without Pembrolizumab (MK3475) in refractory or relapsed and refractory Multiple Myeloma (rrMM) (KEYNOTE 183). |
| 2015 – 2017 | A phase III study of Lenalidomide and low-dose Dexamethasone with or without Pembrolizumab (MK3475) in newly diagnosed and treatment naïve Multiple Myeloma (KEYNOTE 185). |
| 2015 – 2018 | Open-Label, Dose-Escalation and Multicenter Study to Evaluate the Safety and Pharmacokinetics of SAR650984 in Patients with Relapsed/Refractory Multiple Myeloma (TED14154). |
| 2015 – 2016 | A Phase 1b Multicenter, Open-Label, Dose-Escalation Study to Determine the Maximum Tolerated Dose, Safety, and Antitumor Activity of an Alternative Liquid Formulation of ACY-1215 (Ricolinostat) in Combination with Pomalidomide and Low-dose Dexamethasone in Patients with Relapsed or Relapsed-and-Refractory Multiple Myeloma.. |
| 2014 – 2017 | A Phase 1/2 Dose Escalation Safety, Pharmacokinetic and Efficacy Study of Multiple Intravenous Administrations of a Humanized Monoclonal Antibody (SAR650984) Against CD38 In Patients with Selected CD38+ Hematological Malignancies (TED10893). |
| 2015 | An Expanded Access Program for Elotuzumab in Combination with Lenalidomide plus Dexamethasone in Patients with Relapsed or Refractory Multiple Myeloma. |
| 2015 | Randomized, Phase III Study Comparing Conventional Dose Treatment using a Combination of Lenalidomide, Bortezomib, and Dexamethasone (RVD) to High-Dose Treatment with Peripheral Stem Cell Transplant in the Initial Management of Myeloma in Patients up to 65 Years of Age. |

Curriculum Vitae (Djordje Atanackovic, M.D.)

- 2014 – 2015 A Multicenter Phase 2 Study of Single-agent Filanesib (ARRY-520) in Patients With Advanced Multiple Myeloma.
- 2013 A Phase IIb/III randomized, double-blind, placebo-controlled study comparing first-line therapy with or without TG4010 immunotherapy product in patients with stage IV non-small cell lung cancer (NSCLC).
- 2013 Randomized, multicenter, double-blind, phase 3 trial comparing the efficacy of ipilimumab plus etoposide/platinum versus etoposide/platinum in subjects with newly diagnosed extensive-stage disease small cell lung cancer (ED-SCLC).
- 2012 – 2013 LUX-Lung 8: A randomized, open-label phase III trial of afatinib versus erlotinib in patients with advanced squamous cell carcinoma of the lung as second-line therapy following first-line platinum-based chemotherapy Non-Small Cell Lung Cancer (NSCLC) who have progressed on at least one prior chemotherapy.
- 2011 – 2013 Open label, randomized multi-center phase II study evaluating the efficacy and safety of Paclitaxel/Carboplatin with and without Cetuximab as first-line treatment of adeno- and undifferentiated CUP (PACET-CUP).
- 2011 – 2012 Randomized, placebo-controlled, double-blind phase 1b/2 study of U3-1287 (AMG 888) in combination with erlotinib in EGFR treatment naïve subjects with advanced Non-Small Cell Lung Cancer (NSCLC) who have progressed on at least one prior chemotherapy.
- 2011 – 2012 Open-label, randomized, controlled, multicenter Phase II study investigating cilengitide in combination with cetuximab and platinum-based chemotherapy (cisplatin/vinorelbine or cisplatin/gemcitabine) compared to cetuximab and platinum-based chemotherapy alone as first-line treatment for patients with advanced NSCLC (CERTO).
- 2010 – 2011 A randomised, open-label, phase III study of BIBW 2992 versus chemotherapy as first-line treatment for patients with stage IIIB or IV adenocarcinoma of the lung harbouring an EGFR activating mutation (LUX-Lung 3).
- 2010 – 2011 An NIS Registry for the epidemiological and scientific evaluation of EGFR mutation status in patients with newly diagnosed locally advanced or metastatic stage IIIB/IV non-small lung cancer (REASON).
- 2010 – 2013 Double-blind, placebo-controlled, randomized phase II study investigating the efficacy of Bevacizumab for symptom-control in patients with malignant ascites due to advanced-stage-gastrointestinal cancers (AIO-SUP-0108).
- 2009 – 2011 Safety and efficacy phase I/IIa trial of an RNA-derived cancer vaccine in stage IIIB/IV non small cell lung cancer (CV-9201-003 NSCLC).
- 2009 – 2011 An Open-label, single-arm, Phase II study to evaluate the efficacy and the feasibility of bevacizumab (Avastin®) based on a FOLFOXIRI regimen until progression in patients with previously untreated metastatic colorectal carcinoma (OPAL).
- 2009 – 2010 A randomized phase III trial of afatinib (BIBW2992) + best supportive care (BSC) versus placebo + BCG in patients failing 1-2 lines of chemotherapy and erlotinib or gefitinib (LUX-Lung 1).
- 2009 – 2013 An open-label, randomized, controlled, multi-center, Phase II trial investigating 2 EMD 525797 doses in combination with cetuximab+irinotecan versus cetuximab+irinotecan alone, as second-line treatment for subjects with k-ras wild type (WT) metastatic colorectal cancer (POSEIDON).

Curriculum Vitae (Djordje Atanackovic, M.D.)

- 2008 – 2010 A phase 3, double-blind, placebo-controlled study of maintenance pemetrexed plus best supportive care versus best supportive care immediately following induction treatment with Pemetrexed+Cisplatin for advanced nonsquamous non-small cell lung cancer (Alimta H3E-EW-S124).
- 2008 – 2009 Analysis of the expression of a specific set of genes and tumor antigens in patients with non-small cell lung cancer or melanoma (ONCO RD-001).
- 2008 – 2009 Phase II, randomized, open label, multicenter study of intradermal IMA901 plus GM-CSF with or without low dose cyclophosphamide pre-treatment in advanced renal cell carcinoma patients with measurable disease (IMA901-202).
- 2007 – 2012 A double-blind, randomized, placebo-controlled Phase III study to assess the efficacy of recMAGE-A3+AS15 Antigen-Specific Cancer Immunotherapeutic as adjuvant therapy in patients with respectable MAGE-A3-positive Non-Small Cell Lung Cancer (MAGRIT).
- 2007 – 2013 A Phase I/II study to assess the safety and immunogenicity of recMAGE-A3+AS15 cancer immunotherapeutic given as adjuvant therapy, with or without adjuvant chemo(-radio)therapy, to patients with MAGE-A3-positive Non-Small Cell Lung cancer (stage IB, II, or III)" (MAGE-A3-AS15-NSC-001).
- 2004 Sole immunization with NY-ESO-1 protein in comparison to immunization with NY-ESO-1 protein in combination with sodium urate crystals as an adjuvant in patients with head and neck cancer after R0 resection: An early, randomized phase II study.

Co-Investigator

- 2021 – present 2462GCCC: A First in Human Trial of T-cell Receptor (TCR) T-cells Targeting the DNAJB1-PRKACA Fusion Protein in a Single-Patient with Fibrolamellar carcinoma (FLC)
- 2021 – present 2128GCCC:EAP for Idecabtagene Vicleucel Nonconforming for Commercial Release
- 2021 – present Viralym-M (ALVR105) Compared to Placebo for Preventing AdV, BKV, CMV, EBV, HHV-6, and JCV Infection
- 2020 – present 2071GCCC: Open label, multicenter, long-term follow-up study for subjects treated with P-BCMA-101
- 2020 – present Belantamab Mafodotin, Cyclophosphamide and Dexamethasone in Relapsed/Refractory Multiple Myeloma
- 2020 – present 2058GCCC: Open label study of Tiragolumab, Dara, Rituximab in MM and NHL
- 2021 – present 2050GCCC: Phase II study evaluating DALY II USA/MB-CART2019.1 for DLBCL
- 2020 – present 2028GCC: A Study of Venetoclax & Dex Compared with Pomalidomide & Dex in Subjects w R/R MM
- 2020 – present A PHASE 1, MULTICENTER, OPEN-LABEL STUDY OF CC-95266 IN SUBJECTS WITH RRMM
- 2020 – 2024 1961GCCC: Dara & Len Vs Len as Maintenance Tx in Newly Diagnosed MM MRD-Pos After Auto-SCT
- 2020 – present 1948GCCC: P-BCMA-101 Tscm CAR-T Cells in the Treatment of Patients With Multiple Myeloma (MM)
- 2020 – present 19106GCCC: Phase 1 Study of STRO-001 in Patients with Advanced B-Cell Malignancies

Curriculum Vitae (Djordje Atanackovic, M.D.)

| | |
|----------------|---|
| 2020 – present | 1821GCCC: Phase IB/2A of CC-220 and in combo with Other Treatments for MM |
| 2020 – 2021 | 18107GCCC: PHASE 3 STUDY OF BB2121 VERSUS STANDARD REGIMENS IN RRMM (KarMMa-3) |
| 2020 – 2021 | 17119GCCC: ASCENT Black Swan SMM Protocol |
| 2020 – present | Protocol for a Research Sample Repository for Allogeneic Unrelated Hematopoietic Stem Cell Transplant |
| 2018 – 2020 | Pilot Trial Evaluating Phase II, Open-Label, Randomized, Two-Arm Study to Investigate the Efficacy and Safety of Two Doses of the Antibody Drug Conjugate GSK2857916 in Participants with Multiple Myeloma who had Three or More Prior Lines of Treatment, are Refractory to a Proteasome Inhibitor and an Immunomodulatory Agent, and have Failed an Anti-CD38 Antibody (DREAMM 2) |
| 2018 – 2020 | Pilot Trial Evaluating the Combination of Reolysin and Carfilzomib in Multiple Myeloma |
| 2018 – 2020 | Allogeneic Stem Cell Transplantation for Multiple Myeloma and Myelofibrosis |
| 2018 – 2020 | Assessment of Allogeneic Hematopoietic Cell Transplantation in Medicare Beneficiaries with Multiple Myeloma: A Study to Develop Evidence of Effectiveness for the Centers for Medicare and Medicaid Services (CMS) |
| 2018 – 2020 | Genetic Epidemiology of Hematological Malignancies |
| 2017 – 2018 | Assessment Phase IB Study Evaluating the Safety and Pharmacology of Atezolizumab (Anti-PD-L1 Antibody) Administered in Combination with Immunomodulatory Agents in Patients with Acute Myeloid Leukemia |
| 2017 – 2018 | Phase II, Open-Label, Multi-Center Study of Venetoclax in Combination with Carfilzomib and Dexamethasone in Subjects with Relapsed or Refractory Multiple Myeloma |
| 2017 – 2020 | Randomized Phase III Study to Evaluate the Efficacy and Safety of Daratumumab in Combination with Cyclophosphamide, Bortezomib, and Dexamethasone (CyBorD) Compared with CyBorD Alone in Newly Diagnosed Systemic AL Amyloidosis |
| 2017 – 2018 | Phase II, Randomized, Open-Label Study Comparing Daratumumab, Lenalidomide, Bortezomib, and Dexamethasone (D-RVd) versus Lenalidomide, Bortezomib, and Dexamethasone (RVd) in Subjects with Newly Diagnosed Multiple Myeloma Eligible for High-Dose Chemotherapy and Autologous Stem Cell Transplantation |
| 2016 – 2020 | Pilot Study for the Treatment of Steroid-Refractory Sclerodermatous Chronic Graft-Versus-Host Disease (GVHD) with GDC-0449 |
| 2015 – 2018 | A Phase 3, Randomized, Multicenter, Double-Blind, Placebo-Controlled, 2-Arm, Efficacy and Safety Study of NIOD001 Plus Standard of Care vs. Placebo Plus Standard of Care in Subjects with Light Chain (AL) Amyloidosis. |
| 2015 – 2018 | Phase 1b Study of Carfilzomib Administered Once Weekly in Combination with Lenalidomide and Dexamethasone in Subjects with Multiple Myeloma. |
| 2015 – 2020 | Multicenter Biologic Assignment Trial Comparing Reduced Intensity Allogeneic Hematopoietic Cell Transplant to Hypomethylating Therapy or Best Supportive Care in Patients Aged 50-75 with Intermediate-2 and High-Risk Myelodysplastic Syndrome. |
| 2014 – 2016 | Multicenter Phase II Trial Randomizing Novel Approaches for Graft-Versus-Host Disease Prevention Compared to Contemporary Controls. |

- 2014 – 2015 Randomized, Double-Blind, Placebo-Controlled, Parallel-Group, Multicenter, Phase III Study of the Safety, Tolerability, and Efficacy of CMX001 for the Prevention of Cytomegalovirus (CMV) Infection in CMV-Seropositive (R+) Hematopoietic Stem Cell Transplant Recipients.
- 2014 Phase III, Multicenter, Randomized, Open-Label Study to Compare the Efficacy and Safety of Pomalidomide (POM), Bortezomib (BTZ) and Low-Dose Dexamethasone (LD-DEX) versus Bortezomib and Low-Dose Dexamethasone in Subjects with Relapsed or Refractory Multiple Myeloma (MM).
- 2014 Randomized, Open-Label, Phase III Study of Carfilzomib plus Dexamethasone versus Bortezomib plus Dexamethasone in Patients with Relapsed Multiple Myeloma.
- 2011 – 2013 A multicenter, randomized phase II / III trial with 5-FU, leucovorin, oxaliplatin and docetaxel (FLOT) versus epirubicin, cisplatin and 5-FU (ECF) in patients with locally advanced, resectable adenocarcinoma of the esophageal junction and the stomach (FLOT4).
- 2011 – 2012 A clinical phase I trial of panitumumab in combination with sorafenib in patients with KRAS wildtype metastatic colorectal cancer (PASO-1).
- 2010 – 2013 Lenalidomide for optimizing treatments with dexamethasone and high-dose melphalan in patients with multiple myeloma (DSMM XIII).
- 2010 – 2013 Treatment optimization trial in the first-line treatment of early stage Hodgkin Lymphoma; Treatment stratification by means of FDG-PET (HD16).
- 2008 – 2013 An open label, multi-center dose escalating phase I study to investigate the safety and tolerability of a continuous infusion of the bispecific T-cell engager (BiTE) MT110, in patients with locally advanced, recurrent, or metastatic solid tumors which commonly express EpCAM and are not amenable to curative treatment.
- 2008 – 2013 Autologous-Allogeneic Tandem Stem Cell Transplantation and Maintenance Therapy with Thalidomide / DLI for patients with Multiple Myeloma (MM) and age < 55 years: A phase II-study.
- 2007 – 2009 Multicenter, open-label phase II study to evaluate the safety and efficacy of the tri-functional bi-specific antibody catumaxomab (anti-EpCAM x anti-CD3) in patients with gastric adenocarcinoma after neoadjuvant chemotherapy and intended curative resection (GC-03).
- 2007 – 2009 Prospective, multicenter study with 5-FU, leucovorin, oxaliplatin and docetaxel (FLOT) for patients with locally advanced, limited or diffusely metastatic adenocarcinoma of the stomach and the esophagogastric junction (FLOT3).
- 2003 – 2004 Prognostic significance of the "cyto-capacity test" after application of rHu-G-CSF in patients with lymphoproliferative disorders and high-dose chemotherapy.

VIII. GRANT SUPPORT

Ongoing Research Grants

- 09/2025 – 09/2029 "First-in-human phase I clinical trial using tri-specific (CD19/CD20/CD22) CAR T cells for the treatment of relapsed/refractory B cell lymphoma", DOD - US Department of Defense, Role: PI, \$4,553,504.
- 07/2025 – 07/2029 "CD229-targeting CAR T cells as a novel cellular immunotherapy for T cell leukemias", The V Foundation, Role: PI, \$800,000.

Curriculum Vitae (Djordje Atanackovic, M.D.)

- 08/2025 – 08/2030 “Production of a novel CD229 CAR T cell treatment for patients with T cell leukemia”, Neil Kishter Foundation, Role: PI, \$247,500.
- 08/2025 – 08/2030 “Production of a novel “armored” CAR T cell treatment targeting ROR1 for patients with solid tumors”, Neil Kishter Foundation, Role: PI, \$299,100.
- 11/2024 – 10/2025 “Community-informed development of ROR1-specific CAR T cells for the treatment of prostate cancer”, UMGCCC Pilot Grant, Community-informed Research Award, Role: PI, \$49,870.
- 10/2022 – 09/2026 “A Phase Ia Study of LT2950 Tri-specific CAR T-cells for Patients with Relapsed/Refractory B-Cell Lymphomas”, Maryland Stem Cell Research Fund (MSCRF), Role: PI, \$996,846.

Past Grants

- 06/2022 – 05/2023 “Development of Cytokine-Activated NK cells for Treatment of HCC”, Marco Chacon Foundation, Role: PI, \$50,000.
- 09/2022 – 08/2023 “Immunomonitoring of patients receiving the monoclonal anti-CD38 antibody isatuximab with or without an immune checkpoint inhibitor”, Sanofi SA Contract, Role: PI, \$287,291.
- 11/2021 – 10/2022 “Monitoring of immune responses to the COVID-19 “booster” vaccine in patients with hematologic malignancies and solid organ transplants”, Kahlert Foundation, Role: PI, \$175,000.
- 07/2021 – 06/2022 “Analysis of in vitro and in vivo activity of the Enantiomer of Edelfosine CP 201”, RDP Pharma Contract, Role: PI, \$75,000.
- 01/2021 – 12/2021 “Characterization of immune responses against COVID19 in cancer patients”, Kahlert Foundation, Role: PI, \$175,000.
- 06/2020 – 03/2021 “Identifying B cell/ T cell epitopes of virus SARS-CoV-2 to develop tests for screening and response evaluation in patients with a COVID-19 infection”, Clinical Science Research and Development (CSR D) Award of the Department of Veterans Affairs, Role: PI, \$84,700.
- 06/2020 – 03/2021 “Identifying B cell/ T cell epitopes of virus SARS-CoV-2 to develop tests for screening and response evaluation in patients with a COVID-19 infection”, Clinical Science Research and Development (CSR D) Award of the Department of Veterans Affairs, Role: PI, \$84,700.
- 08/2018 – 08/2020 “Development of CD229 CAR T cells for the treatment of Multiple Myeloma”, Translational Scholar Grant, University of Utah, Huntsman Cancer Institute, Role: PI, \$150,000.
- 07/2018 – 06/2020 “CD229 chimeric antigen receptor T cells for the treatment of Multiple Myeloma”, National Comprehensive Cancer Network award for Tim Luetkens, \$150,000.
- 07/2019 – 06/2020 Extension of “Improving T cell therapy by targeting immune escape in neuroblastoma”, St. Baldrick’s Foundation Fellowship award for Fiorella Iglesias, \$179,572.
- 05/2019 – 04/2020 “CD229 chimeric antigen receptor T cells for the treatment of B cell lymphoma”, Research Pilot Award of the Center of Excellence in

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| | Hematological Malignancies and Hematology, University of Utah, Huntsman Cancer Institute, \$35,000. |
| 03/2019 – 02/2020 | “Develop a cellular therapy to correct HLA loss in solid malignancies to augment T cell-mediated tumor cell killing”, Pilot Project Award of the Center for Clinical & Translational Science (CCTS) for Tim Luetkens, \$30,000. |
| 01/2019 – 12/2019 | “Development of a high affinity antibody against CD229 for the treatment of Multiple Myeloma”, American Cancer Society (ACS) Institutional Research Grant for Sabari Radhakrishnan, \$30,000. |
| 10/2017 – 10/2019 | Analysis of antibody responses and T-cell responses of patient samples collected in Sanofi’s TED10893 study (Phase 2 Stage 2), including sample management and data transfer, Sanofi, Role: PI, \$761,229. |
| 11/2014 – 11/2019 | Translational analysis of immune responses within the framework of the clinical study “An open-label, dose-escalation and multi-center study to evaluate the safety and pharmacokinetics of SAR650984 in patients with relapsed/refractory multiple myeloma” (TED14154)”, Sanofi, Role: PI, \$181,135. |
| 08/2018 – 08/2019 | “CD229 CAR T cells, Specificity Studies”, University of Utah, Center for Technology & Venture Commercialization: \$40,000. |
| 08/2017 – 07/2019 | “Chimeric antigen receptor (CAR) T cell therapy for multiple myeloma”, AACR-Pfizer Fellowship in Immuno-oncology Research for Sabari Radhakrishnan: \$110,000. |
| 07/2018 – 06/2019 | “Determine the Safety of CD229-directed Immunotherapies” Catalyst Grant, University of Utah, Role: PI, \$50,000. |
| 07/2017 – 06/2019 | “Improving T cell therapy by targeting immune escape in neuroblastoma”, St. Baldrick’s Foundation Fellowship award for Fiorella Iglesias: \$179,572. |
| 11/2017 – 10/2018 | “Image-guided Delivery of T Cell Receptor (TCR)-Transgenic T Cells Targeting Hepatocellular Carcinoma “, Immunology, Inflammation, and Infectious Diseases (III) Initiative Seed Grant: \$49,772. |
| 07/2017 – 06/2018 | “Development of an Anti-IL-16 Immunotherapy for Multiple Myeloma“, Experimental Therapeutics Program: \$40,000. |
| 01/2014 – 12/2017 | “Development of translational immunotherapy program for Multiple Myeloma“, University of Utah / Huntsman Cancer Institute, Start-up Grant: \$750,000. |
| 01/2017 – 12/2017 | “Anti-CD229 Chimeric Antigen Receptor T Cells for the Treatment of Multiple Myeloma”, International Myeloma Foundation 2017 Senior Research Award, Role: PI, \$80,000. |
| 01/2017 – 12/2017 | “Generation and validation of a monoclonal antibody against VISTA for the immunomodulatory therapy of Multiple Myeloma”, International Myeloma Foundation 2017 Junior Research Award for Dr. Neelam Bhardwaj: \$50,000. |
| 07/2016 – 06/2017 | “Generation of a monoclonal antibody against VISTA for the immunomodulatory therapy of Multiple Myeloma”, American Association for Cancer Research (AACR) Fellowship in Multiple Myeloma Research for Dr. Neelam Bhardwaj: \$55,000. |

Curriculum Vitae (Djordje Atanackovic, M.D.)

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| 08/2015 – 07/2016 | “Using MHC class I tetramer-coated nanoparticles to overcome autoimmune side effect and improve the efficacy of anti-programmed death-1 antibody (aPD-1)”, co-investigator with Dr. Mingnan Chen, Huntsman Cancer Institute, Experimental Therapeutics Program Grant: \$25,000. |
| 08/2015 – 07/2016 | “Development of a combinatorial cellular immunotherapy for multiple myeloma”, University of Utah School of Medicine, Program in Personalized Health CCTS Pilot Award: \$30,000. |
| 07/2015 – 10/2016 | “Targeting multiple myeloma propagating cell using CD229-specific CAR T cells“, Huntsman Cancer Institute, Experimental Therapeutics Program Grant: \$40,000. |
| 01/2015 – 06/2016 | “Analysis of cancer-testis antigen-specific immune responses in Multiple Myeloma“, Cancer Research Institute, CVC Grant: \$45,000. |
| 01/2014 – 06/2016 | “Chimeric antigen receptors for the treatment of hematologic malignancies“, Huntsman Cancer Institute, DOT Seed Grant: \$25,000. |
| 2014-2015 | “Engineering CAR T Cells That Target Mutant CALR as a Novel Therapeutic for Myeloproliferative Neoplasms“, Leukemia & Lymphoma Society, MPN Challenge Grant: \$100,000. |
| 2013-2014 | “Cytokine IL-16 as a target structure for the immunotherapy of multiple myeloma“ Hamburger Krebsgesellschaft e.V.: € 55.350 (\$62,000). |
| 2012-2013 | “Identification of mechanisms for the processing of Interleukin-16 in Multiple Myeloma“ Erich und Gertrud Roggenbuck-Stiftung: € 40.800 (\$46,000). |
| 2013 | “The biological function of cancer-testis antigen MAGE-C2/CT10 in multiple myeloma“ Eppendorfer Krebs- und Leukämiehilfe: € 20.000 (\$23,000). |
| 2011-2013 | “Sperm-specific lysozyme-like protein 1 (SLLP1) as a potential therapeutic target in Multiple Myeloma“. Else Kröner-Fresenius-Stiftung: € 155.000 (\$180,000). |
| 2010-2013 | “The biological function of cancer-testis antigen MAGE-A3 in human lung cancer“. GlaxoSmithKline: € 77.000 (\$86,000). |
| 2009-2013 | “Generation of autologous MAGEC2/CT10-specific CD4 ⁺ and CD8 ⁺ T cell clones using gene transduction for the adoptive immunotherapy of Multiple Myeloma“. Deutsche Krebshilfe: € 331.600 (\$370,000). |
| 2009-2013 | “Translational analyses on the mechanisms of action of Bevacizumab in patients with malignant ascites“. Roche: € 79.200 (\$89,000). |
| 2010-2012 | “Evaluation of surface antigen CD229 as a novel therapeutic target in multiple myeloma“. Wilhelm Sander-Stiftung: € 180.000 (\$200,000). |
| 2007-2012 | “Cancer-testis antigens as targets for graft-versus-myeloma effects and antigen-specific immunotherapy in patients with multiple myeloma“. Cancer Research Institute: \$ 200.000. |
| 2012 | “Molecular Function of MAGE-A3 in Non Small Cell Lung Cancer“ Eppendorfer Krebs- und Leukämiehilfe: € 24.000 (\$27,000). |
| 2007-2012 | “Immune responses induced by the trifunctional bispecific antibody catumaxomab“. Fresenius Biotech: € 86.724 (\$97,000). |

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| 2010-2012 | “Function of Interleukin-16 in multiple myeloma“. Erich und Gertrud Roggenbuck-Stiftung: € 67.600 (\$76,000). |
| 2010-2011 | “Immunomonitoring of Cancer Vaccine Collaborative trials“. Cancer Research Institute: \$ 40.000. |
| 2009-2010 | “Evaluation of surface antigen CD229 as a therapeutic target in multiple myeloma“. Eppendorfer Krebs- und Leukämiehilfe: € 24.000 (\$27,000). |
| 2009-2010 | “Functional analysis of tumor antigen PRAME in acute myeloid leukemia“. Hamburger Krebsgesellschaft: € 19.500 (\$22,000). |
| 2007-2010 | “Analyses of transplantation-induced immune responses against cancer-testis antigens in patients with Multiple Myeloma“. Deutsche José Carreras Leukämie-Stiftung: € 199.500 (\$222,000). |
| 2009-2010 | “‘Stem cell regulator’ SOX2 as a potential target for an active immunotherapy of multiple myeloma“. Erich und Gertrud Roggenbuck-Stiftung: € 50.000 (\$56,000). |
| 2009-2010 | “Immunomonitoring of the Cancer Vaccine Collaborative MAGE-A3 trial“. Cancer Research Institute: \$ 40.000. |
| 2008-2009 | “Longitudinal analysis of B cell-mediated immune responses against cancer-testis antigens in patients with multiple myeloma treated with allogeneic stem cell transplantation“. Cancer Research Institute: \$ 40.000. |
| 2008-2009 | “Cancer-testis antigen MAGE-C1/CT7: Analyses of the immunogenicity of a potential target antigen for tumor vaccination in patients with Multiple Myeloma“. Erich und Gertrud Roggenbuck-Stiftung: € 60.000 (\$67,000). |
| 2008-2009 | “The role of T helper 17 cells in the pathogenesis and immune control of multiple myeloma“. Eppendorfer Krebs- und Leukämiehilfe: € 15.000 (\$17,000). |
| 2008 | “Serological immune responses against cancer-testis antigens in multiple myeloma“. Aktion „Kampf dem Krebs“ e.V. der Deutschen Krebsgesellschaft: € 8.354 (\$10,000). |
| 2007-2008 | “The role of cancer-testis antigens as targets for graft-versus-myeloma effects in patients with multiple myeloma“. Cancer Research Institute: \$ 40.000. |
| 2007-2008 | “Cancer-testis antigens as potential targets for immunotherapeutic approaches in patients with chronic myeloid leukemia“. Eppendorfer Krebs- und Leukämiehilfe: € 25.000 (\$28,000). |
| 2007-2008 | “Analyses of the immunologic relevance of cancer-testis antigen MAGE-C1/CT7 as a potential target structure for tumor vaccination in patients with Multiple Myeloma“. Hamburger Stiftung zur Förderung der Krebsbekämpfung: € 15.000 (\$17,000). |
| 2006-2008 | “Expression pattern and immunogenicity of cancer-testis antigen SSX-2 as a potential target for tumor vaccination approaches in Multiple Myeloma patients following allogeneic stem cell transplantation“. Erich und Gertrud Roggenbuck-Stiftung: € 120.000 (\$134,000). |
| 2006-2007 | “The relevance of T regulatory cells for transplantation-induced immune responses against cancer-testis antigens in patients with Multiple Myeloma“. Werner Otto Stiftung: € 50.000 (\$56,000). |

Curriculum Vitae (Djordje Atanackovic, M.D.)

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| 2006-2007 | “Cancer-testis antigens as target structures for active immunotherapy in Multiple Myeloma“. Eppendorfer Krebs- und Leukämiehilfe: € 10.000 (\$12,000). |
| 2005-2007 | “Immunomonitoring of a phase II trial of immunization with proteinD MAGE-3/His and immunological adjuvant AS02B in patients with non-small cell lung cancer“. Cancer Research Institute: \$ 80.000. |
| 2005-2007 | “Expression pattern and T cell epitopes of tumor antigen NY-CO-58“. Deutsche Krebshilfe: € 181.400 (\$202,000). |
| 2005-2006 | “Analysis of T regulatory cells and the local chemokine production by tumor cells in effusions caused by solid tumors“. Hamburger Stiftung zur Förderung der Krebsbekämpfung: € 20.000 (\$23,000). |
| 2004-2005 | “The role of tumor antigen NY-CO-58 in a variety of human cancers“. Cancer Research Institute: \$ 40.000. |
| 2004-2005 | “Analysis of the local chemokine milieu for the immune control of head and neck cancers “. Werner Otto Stiftung: € 57.000 (\$64,000). |
| 2004-2005 | “Analysis of the chemokine and cytokine milieu in the malignant effusions caused by solid tumors“. Hamburger Stiftung zur Förderung der Krebsbekämpfung: € 11.000 (\$13,000). |

Pending Research Grants

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| 07/2026 – 6/2030 | “ROR1-targeted armored CAR T cells for the treatment of prostate cancer“, DOD - US Department of Defense, Idea Development Award, Role: PI, \$1,697,318. |
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IX. EDUCATIONAL ACTIVITIES

Lectures

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| 11/2023 | Speaker, “Novel Cellular Immunotherapies for Cancer“, Noon Conference, Internal Medicine Residency Program, University of Maryland School of Medicine |
| 10/2023 | Speaker, “Cancer Immunology“, Molecules to Medicine Course for MD/PhD students, University of Maryland School of Medicine |
| 07/2023 | Speaker, Nathan Schnaper Intern Program (NSIP), Bench-to-Bedside Seminar |
| 01/2022 | Speaker, “Developing novel cellular cancer immunotherapies in times of COVID-19“, Department of Medicine Grand Rounds, University of Maryland School of Medicine |
| 2018 – 2020 | Speaker, Clinical Cancer Biology course, “Cancer Immunotherapy“ lecture for PhD students at the University of Utah |
| 2014 – 2020 | Speaker, Updates on Multiple Myeloma, Annual Hematology Review at the University of Utah / Huntsman Cancer Institute |
| 2014 – 2020 | “Fellows’ Noon Conference“, Hematology and Hematologic Malignancies, University of Utah / Huntsman Cancer Institute |
| 2014 – 2020 | Hematology Training for Medical Students, Hematology and Hematologic Malignancies, University of Utah / Huntsman Cancer Institute |
| 2014 – 2020 | “Clinical Fellows’ Journal Club“, Hematology and Hematologic Malignancies, University of Utah / Huntsman Cancer Institute |

Curriculum Vitae (Djordje Atanackovic, M.D.)

- 2010 "Gastric carcinoma" for medical students, Department of Oncology/Hematology/Stem Cell Transplantation, University Medical Center Hamburg-Eppendorf
- 2007 – 2013 "Differential diagnosis in Oncology/Hematology" for medical students, Department of Oncology/Hematology/Stem Cell Transplantation, University Medical Center Hamburg-Eppendorf
- 2007 – 2013 "Bedside Teaching" for medical students, Department of Oncology/Hematology/Stem Cell Transplantation, University Medical Center Hamburg-Eppendorf
- 2006 – 2013 "Experimental Oncology/Hematology" seminar for medical students and PhD students, Department of Oncology/Hematology/Stem Cell Transplantation, University Medical Center Hamburg-Eppendorf
- 2006 – 2013 "Journal Club" seminar for clinical staff, Department of Oncology/Hematology/Stem Cell Transplantation, University Medical Center Hamburg-Eppendorf
- 2003 – 2004 "Routine Physical Exams" course for medical students, Department of Oncology/Hematology/Stem Cell Transplantation, University Medical Center Hamburg-Eppendorf

Exams

- 2018 – 2020 Examiner, Capstone Exams for Molecular Biology PhD students at the University of Utah

Research Mentoring

Graduate Students (PhD)

- 2018 – 2021 Michael Lee Olson, Design of CD229-specific CAR T cells for the treatment of B cell lymphoma, Microbiology and Immunology, University of Utah / Huntsman Cancer Institute (graduated Spring 2022, 2.5 hours/week)
- 2018 – present Erica Vander Mause, Optimization of anti-CD229 approaches for the treatment of Multiple Myeloma, University of Utah / Huntsman Cancer Institute (2.5 hours/week)
- 2010 – 2013 Julia Templin, Characterization of Interleukin-16 as a target structure for the diagnosis and therapy of Multiple Myeloma, University Medical Center Hamburg-Eppendorf
- 2012 – 2013 Stefanie Spöck, Generation of autologous MAGE-C2/CT10-specific CD4+ und CD8+ T cell clones using gene transduction for the adoptive immunotherapy of Multiple Myeloma, University Medical Center Hamburg-Eppendorf
- 2010 – 2013 Yanran Cao, Humoral immune reconstitution in patients with Multiple myeloma post allogeneic stem cell transplantation, University Medical Center Hamburg-Eppendorf
- 2010 – 2013 Nesrine Lajmi, Cancer-testis antigens as targets for graft-versus-myeloma effects and antigen-specific immunotherapy in patients with Multiple Myeloma, University Medical Center Hamburg-Eppendorf

Undergraduate Students

- 2023 – present Daniel Yamoah, Immunomonitoring of antibody-induced anti-tumor humoral immune responses and functional assessment of CAR T cells,

- daily meetings/lab supervision (2.0 hours/week), University of Maryland Comprehensive Cancer Center
- 2023 – present Rediet Mulatu, Immunomonitoring of patients receiving CAR T cell treatments and assessment of antiviral T cell responses in cancer patients, daily meetings/lab supervision (2.0 hours/week), University of Maryland Comprehensive Cancer Center,
- 2021 – 2023 Destiny Omili, Immunomonitoring of anti-SARS-CoV-2 and anti-tumor T cell responses in patients with multiple myeloma/lymphoma, daily meetings/lab supervision (2.0 hours/week), University of Maryland Comprehensive Cancer Center
- 2021 – 2023 Thierry Iraguha, Analysis of serological anti-tumor and antiviral immune responses and functional analysis of CAR T cells, daily meetings/lab supervision (2.0 hours/week), University of Maryland Greenebaum Comprehensive Cancer Center

Interns

- 2025 Sophie Bredar, Nathan Schnaper Intern Program in Translational Cancer Research (NSIP), Clinical Shadowing Program, University of Maryland Greenebaum Comprehensive Cancer Center
- 2025 Ashley Gelin, Bridges to the Doctorate (B2D) Intern Program, University of Maryland Greenebaum Comprehensive Cancer Center
- 2023 Brian Schattle, Nathan Schnaper Intern Program in Translational Cancer Research (NSIP), University of Maryland Greenebaum Comprehensive Cancer Center
- 2022 AyoOluwakiitan Oluwafemi, Nathan Schnaper Intern Program in Translational Cancer Research (NSIP), University of Maryland Greenebaum Comprehensive Cancer Center

Medical Students

- Johanna Heise: Expression and immunogenicity of the cancer-testis antigen SLLP1 in Multiple Myeloma, University Medical Center Hamburg-Eppendorf
- Stephanie Weißleder: T cell responses against cancer-testis antigen SLLP1 in Multiple Myeloma, University Medical Center Hamburg-Eppendorf
- Ronak Mirzaei: EpCAM- and cancer-testis antigen-specific immune responses in patients with gastric cancer following adjuvant treatment with the trifunctional and bispecific antibody Catumaxomab (anti-EpCAM / anti-CD3), University Medical Center Hamburg-Eppendorf
- Christiane Keller: The relevance of cytokine Interleukin-16 for the biology of Multiple Myeloma, University Medical Center Hamburg-Eppendorf
- Maja Gordic: Development of an ELISPOT assay for the quantification of tetanus toxoid-specific B cells as a model for the immunomonitoring of patients with Multiple Myeloma, University Medical Center Hamburg-Eppendorf
- Marina Ristic: Measurement of NY-ESO-1- and MAGE-A3-specific antibodies in patients with Multiple Myeloma, University Medical Center Hamburg-Eppendorf
- Sinje Tams: Measurement of PRAME- and SOX2-specific antibodies in patients with Multiple Myeloma, University Medical Center Hamburg-Eppendorf

Curriculum Vitae (Djordje Atanackovic, M.D.)

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| Corinna Eberhardt: | Measurement of SSX2- and SSX4-specific antibodies in patients with Multiple Myeloma, University Medical Center Hamburg-Eppendorf |
| Britta Bartels: | Measurement of Tetanus Toxoid- and Influenza Virus-specific antibodies in patients with Multiple Myeloma, University Medical Center Hamburg-Eppendorf |
| Benjamin Kloth: | Analysis of the expression of cancer-testis antigens in the peripheral blood and bone marrow of patients with acute myeloid leukemia, University Medical Center Hamburg-Eppendorf |
| Gregor Fuchs: | Analysis of the expression of cancer-testis antigens in cell lines derived from patients with acute myeloid leukemia, University Medical Center Hamburg-Eppendorf |
| Ruken Ablukak: | Analysis of cancer-testis antigen-specific antibody responses in patients with chronic myeloid leukemia, University Medical Center Hamburg-Eppendorf |
| Tim Stasche: | Expression of cancer-testis antigens in cell lines derived from patients with chronic myeloid leukemia, University Medical Center Hamburg-Eppendorf |
| Frederike Uhlich: | Expression of cancer-testis antigens in the peripheral blood and bone marrow of patients with chronic myeloid leukemia, University Medical Center Hamburg-Eppendorf |
| Inga Blum: | Expression of cancer-testis antigens in head and neck cancer: possible targets for antigen-specific immunotherapies, University Medical Center Hamburg-Eppendorf |
| Tim Luetkens: | Longitudinal analysis of the expression of cancer-testis antigens in the bone marrow of patients with Multiple Myeloma, University Medical Center Hamburg-Eppendorf |
| Julia Arfsten: | Analysis of the expression pattern of cancer-testis antigens in the bone marrow of patients with Multiple Myeloma, University Medical Center Hamburg-Eppendorf |
| Katrin Holz: | Effects of acute psychological stress on T regulatory cells in healthy subjects, University Medical Center Hamburg-Eppendorf |
| Nojan Sanatgar: | The role of chemokines in the local T cell-mediated anti-tumor immune response in head and neck cancer, University Medical Center Hamburg-Eppendorf |
| Christina Nölkers: | Expression of tumor antigen NY-CO-58 in different solid tumors, University Medical Center Hamburg-Eppendorf |
| Ji-Won Kim: | Analysis of the local cytokine and chemokine milieu in malignant effusions, University Medical Center Hamburg-Eppendorf |
| Kristina Pollok: | Effect of 41.8 °C whole-body hyperthermia on cellular immune parameters in patients with different solid tumors, University Medical Center Hamburg-Eppendorf |

Postdoctoral Fellows

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| 2014 – 2018 | Neelam Bhardwaj, University of Utah / Huntsman Cancer Institute |
| 2012 – 2017 | Sara Yousef, University Medical Center Hamburg-Eppendorf and University of Utah / Huntsman Cancer Institute |
| 2014 – 2015 | Julia Templin, University of Utah / Huntsman Cancer Institute |

Clinical Fellows

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| 2025 – present | Samuel Weeks, University of Maryland School of Medicine / University of Maryland Greenebaum Comprehensive Cancer Center |
| 2018 – 2020 | Maria Carter, University of Utah / Huntsman Cancer Institute |
| 2018 – 2020 | Erin Morales, University of Utah / Huntsman Cancer Institute |
| 2016 – 2020 | Fiorella Iglesias, University of Utah / Huntsman Cancer Institute |
| 2015 – 2016 | Daisuke Yunaiyama, University of Utah / Huntsman Cancer Institute |
| 2015 – 2017 | Sabari Radhakrishnan, University of Utah / Huntsman Cancer Institute |

Faculty

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| 2022 – present | Member, Research Mentoring Committee, Sarah Sunshine, M.D., University of Maryland Comprehensive Cancer Center (1 hour/month) |
| 2017 – 2020 | Sabari Radhakrishnan, University of Utah / Huntsman Cancer Institute |
| 2014 – 2020 | Tim Luetkens, University of Utah / Huntsman Cancer Institute |

PhD Thesis Supervisory Committees

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| 2021 – present | Jillian Baker, Molecular Microbiology and Immunology, University of Maryland (1 hour/month) |
| 2018 – 2020 | Kyle Isaacson, College of Pharmacy, University of Utah |
| 2018 – 2021 | Michael Lee Olson, Microbiology and Immunology, University of Utah |

Other Educational Activities

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| 2010 – 2013 | “Mentoring Program for Excellent Medical Students”, Department of Oncology/Hematology/Stem Cell Transplantation, University Medical Center Hamburg-Eppendorf |
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X. PRESENTATIONS

Symposium/Conference Chair/Coordinator

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| 2022 | Chair, 2022 SITC Annual Meeting, Cellular Immunotherapies and Bispecifics Session |
| 2018 | Chair, 2018 BMT Tandem Meetings, Lymphoma/Multiple Myeloma/Stem Cell Biology/Hematopoiesis Session |
| 2016 – 2020 | Chair, Annual Multiple Myeloma Symposium at the University of Utah / Huntsman Cancer Institute |
| 2015 – 2020 | Chair, Annual Cancer Immunotherapy Conference at the University of Utah / Huntsman Cancer Institute |
| 2011 | Chair and Coordinator, Eppendorf Afternoon – 4th Update on the Interdisciplinary Diagnostics and Therapy of Bronchial Carcinoma, University Medical Center Hamburg-Eppendorf, Hamburg, Germany |

Invited Oral Presentations (extramural) - Selection

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| 10/2025 | “First-in-Human Trispecific CAR T Cells Targeting CD19/CD20/CD22 in B-Cell Lymphoma - Transforming CAR T Therapy Through Academic Innovation” presented at <i>Tedco’s Entrepreneur Expo</i> in College Park, MD |
| 03/2024 | “Development of Novel Cellular Immunotherapies at UMCCCC” presented at the <i>2nd Miltenyi Round Table – Prostate Cancer</i> in Gaithersburg, MD |

Curriculum Vitae (Djordje Atanackovic, M.D.)

- 11/2023 “Establishing tri-specific CAR T cells for B cell lymphoma patients at UMGCC” presented at the *Cell and Gene Therapy in the DMV Symposium* in Washington, DC
- 06/2023 “Establishing tri-specific CAR T cells for B cell lymphoma patients at UMGCC” presented at the *International Society for Stem Cell Research (ISSCR) Annual Meeting* in Boston, MA
- 03/2023 “Development of Novel Cellular Immunotherapies at UMGCCC” presented at the *1st Miltenyi Round Table – HCC* in Gaithersburg, MD
- 01/2023 “Development of novel anti-MM immunotherapies at UMGCCC” presented at the *2023 Sanofi Research Meeting*, virtual meeting
- 08/2022 “A Phase Ia Study of LT2950 Tri-specific CAR T-cells for Patients with Relapsed/Refractory B-Cell Lymphomas” presented at the *Maryland Maryland Stem Cell Research Fund (MSCRF) Board Meeting* in Columbia, MD
- 04/2022 “Immune response to COVID-19 vaccination in patients with B-cell malignancies after CD19 CART cell therapy” presented at the *Annual Meeting of the American Association for Cancer Research (AACR)* in New Orleans, LA
- 11/2020 “Novel anti-tumor cellular immunotherapies for cancer” presented at the *KITE Research Meeting*, virtual meeting
- 09/2020 “Outcomes of Anti-BCMA CAR T Cell Therapy for Relapsed/refractory Multiple Myeloma: A Meta-Analysis” presented at the *Annual Meeting of European Group for Blood and Bone Marrow Transplantation (EBMT)*, virtual meeting
- 06/2020 “Receptor-transgenic T cells for the Immunotherapy of Multiple Myeloma” presented at the *XXIIIth German Wilsede Meeting* in Wilsede, Germany
- 05/2020 “Engineered T Cells enhance anti-tumor immunity by restoring HLA expression in neuroblastoma” presented at the *Annual Meeting of the American Society for Pediatric Hematology and Oncology (ASPHO)*, virtual meeting
- 03/2020 “CD229 CAR T Cells eliminate multiple myeloma and tumor propagating cells but show limited targeting of normal T Cells” presented at the *Annual Meeting of the National Cancer Center Network (NCCN)*, virtual meeting
- 03/2020 “Developing anti-tumor immunotherapies in the times of COVID-19” presented at the *Institute of Human Virology (IHV) – Special Seminar*, Institute of Human Virology (IHV) in Baltimore, MD
- 10/2019 “Rational Design of Cellular Cancer Immunotherapies” presented at the *Medical University of South Carolina (MUSC)* in Charleston, SC
- 04/2019 “Rational Design of Cellular Cancer Immunotherapies” presented at the *University of Maryland School of Medicine / University of Maryland Greenebaum Comprehensive Cancer Center* in Baltimore, MD
- 01/2019 “Development of Antibody-based Immunotherapies for Multiple Myeloma and other Hematologic Malignancies” presented at the *KITE Pharma Research Meeting* in Santa Monica, CA
- 10/2018 “Development of Cellular Immunotherapies for Hematologic Malignancies” presented at the *Research Meeting of the Tisch Cancer Institute at Mount Sinai* in New York, NY
- 09/2018 “Chimeric Antigen Receptor (CAR) T cells for Hematologic Malignancies and Solid Tumors” presented at the *SUMO Fall Meeting* in Park City, UT

Curriculum Vitae (Djordje Atanackovic, M.D.)

- 09/2018 "Development of Cellular Immunotherapies for Hematologic Malignancies" presented at *Johnson & Johnson Myeloma Research Group Meeting*, Philadelphia, PA
- 06/2018 "Final results of a phase Ib study of isatuximab plus pomalidomide and dexamethasone in relapsed/refractory multiple myeloma" presented at the *23rd Congress of the European Hematology Association (EHA)* in Stockholm, Sweden
- 03/2018 "CD229 CAR T cells are an effective treatment for Multiple Myeloma" presented at the *Annual Meeting of European Group for Blood and Bone Marrow Transplantation (EBMT)* in Lisbon, Portugal
- 03/2017 "Rapid Process for the Generation of Functional Chimeric Antigen Receptors (CARs) Against Novel Targets" presented at the *Annual Meeting of European Group for Blood and Bone Marrow Transplantation (EBMT)* in Marseille, France
- 01/2017 "Development of Cancer-Testis Antigen-specific Immunotherapies for Multiple Myeloma" presented at *Roswell Park Comprehensive Cancer Center* in Buffalo, NY
- 06/2016 "Receptor-transgenic T cells for the Immunotherapy of Multiple Myeloma" presented at the *XXIth German Wilsede Meeting on Modern Trends in Human Leukemia and Cancer* in Wilsede, Germany
- 05/2016 "Post-Transplant Immunotherapy: Tumor-specific T cells" presented at the *Juno Therapeutics Research Meeting* in Seattle, WA
- 05/2016 "Monoclonal Antibodies and Immunotherapy" presented at the *Myeloma Crowd Round Table on High-Risk Disease* in Salt Lake City, UT
- 10/2015 "Tumor-specific T cells as a post-transplant immunotherapy for Multiple Myeloma" presented at the *3rd International Congress on Controversies in Stem Cell Transplantation and Cellular Therapies (COSTEM)* in Berlin, Germany
- 07/2014 "Treatment Options for Relapsed / Refractory Myeloma" presented at the *Regional Community Workshop "Living Successfully with Multiple Myeloma" by the International Myeloma Foundation (IMF)* in Salt Lake City, UT
- 09/2013 "MAGE-A3 in NSCLC: Function and Immunogenicity" presented at *GlaxoSmithKline Research Meeting* in Rixensart, Belgium
- 06/2013 "Lung cancer" presented at the *9th Hamburg Review of the ASCO* in Hamburg, Germany
- 11/2012 "Immunotherapies for solid tumors" presented at the *Fall Congress of the Medical Oncology Working Group of the German Cancer Society (AIO)* in Berlin, Germany
- 09/2012 "Lung cancer" presented at the *Hamburg specialist course in oncology/hematology* in Hamburg, Germany
- 10/2012 "Role of Interleukin-16 in Multiple Myeloma" presented at the *Annual International Cancer Immunotherapy Symposium of the Cancer Research Institute (CRI)* in New York, NY
- 08/2012 "Immunotherapeutic approaches for cancer – from vision to clinical reality" presented at the *Medac Meeting* in Wedel, Germany
- 06/2012 "Lung cancer" presented at the *8th Hamburg Review of the ASCO* in Hamburg, Germany
- 05/2012 "Development of multimodal immunotherapy strategies for human tumors" presented at the *University of Basel* in Basel, Switzerland

Curriculum Vitae (Djordje Atanackovic, M.D.)

- 03/2012 "Pathways to the immunotherapy of multiple myeloma" presented at the *Hematology Seminar*, University of Utah/Huntsman Cancer Institute
- 03/2012 "Development of tumor-specific immunotherapies" presented at the *Seminar for Biological-Technical Assistants at the School of Life Science*, Hamburg, Germany
- 11/2011 "Cancer-testis antigens as promising targets for the immunotherapy of hematologic malignancies and solid tumors" presented at *GlaxoSmithKline Research Meeting* in Rixensart, Belgium
- 09/2011 "The trifunctional α -EpCAM mAb Catumaxomab (Removab®) promotes the development of tumor-specific immune responses in patients with gastric adenocarcinoma" presented at the *Fresenius Research Meeting*, Homburg, Germany
- 06/2011 "Lung Cancer" presented at the 7th "PostASCO" Meeting in Hamburg, Germany
- 03/2011 "Antigen-specific approaches for the immunotherapy of multiple myeloma" presented at the *Seminar for Biological-Technical Assistants at the School of Life Science*, Hamburg, Germany
- 01/2011 "Cancer testis antigens as targets for the immunotherapy of multiple myeloma" presented at the *University of Heidelberg* in Heidelberg, Germany
- 10/2010 "Interleukin-16 in the pathogenesis of multiple myeloma" presented at the *Annual Meeting of the European Society for Medical Oncology (ESMO)* in Milan, Italy
- 06/2010 "Lung Cancer" presented at the 6th "PostASCO" Meeting in Hamburg, Germany
- 05/2010 "Case of an NSCLC patient receiving the mRNA vaccine" presented at the *Curevac Investigator Meeting* in Frankfurt, Germany
- 03/2010 "New and old targets for the immunotherapy of multiple myeloma" presented at the *Lab Meeting of the New York Branch of the Ludwig Institute for Cancer Research (LICR)* in New York, NY
- 02/2010 "Identification of an immunodominant region of SSX-2 preferentially targeted by spontaneous antibodies in multiple myeloma patients" presented at the *Annual German Cancer Congress (Deutscher Krebskongress)* in Berlin, Germany
- 02/2010 "A myeloma-related cytokine/chemokine milieu in the bone marrow environment of patients before and after allogeneic stem cell transplantation" presented at the *Annual German Cancer Congress (Deutscher Krebskongress)* in Berlin, Germany
- 04/2009 "The role of cancer-testis antigens in hematologic neoplasms" presented at the *Lab Meeting of the Department of Medical Oncology*, National Center for Tumor Diseases, University Hospital Heidelberg in Heidelberg, Germany
- 03/2009 "The role of cancer-testis antigens in hematologic neoplasms" presented at the *Lab Meeting of the Department of Hematology*, University Hospital Essen in Essen, Germany
- 06/2008 "Developing Immunotherapeutic Strategies for Multiple Myeloma" presented at the *Board meeting of the Cancer Research Institute*, New York, NY
- 02/2008 "Fighting cancer – research for humanity: Tumor immunology" presented at the *Day of Health Research* in Hamburg, Germany
- 02/2008 "Immunotherapy in multiple myeloma" presented at the *Symposium for Multiple Myeloma and Amyloidosis – New Options and Perspectives* in Hamburg, Germany
- 11/2007 "Tumor Immunology" presented at the *Research Meeting of the Eppendorf Association* in Hamburg, Germany

Curriculum Vitae (Djordje Atanackovic, M.D.)

- 10/2007 "Vaccination of myeloma patients with minimal residual disease after autologous or allogeneic transplantation using recombinant MAGEA3 protein + adjuvant AS15" presented at *GlaxoSmithKline Research Meeting* in Rixensart, Belgium
- 06/2007 "Booster vaccination of non-small cell lung cancer (NSCLC) patients with MAGEA3 protein and AS02B adjuvant" presented at the *Annual Meeting of the American Society of Clinical Oncology (ASCO)* in Chicago, IL
- 11/2006 "Expression of cancer-testis antigens and anti-tumor immunity in patients with multiple myeloma following allogeneic stem cell transplantation" presented at *GlaxoSmithKline Research Meeting* in Rixensart, Belgium
- 11/2006 "Expression of cancer-testis antigens and anti-tumor immunity in patients with multiple myeloma following allogeneic stem cell transplantation" presented at *Lab Meeting of the Ludwig Institute for Cancer Research at Memorial Sloan-Kettering Cancer Center* in New York, NY
- 10/2006 "Effects of acute psychological stress on the T cell-mediated immune system" presented at the *12th Steglitz Colloquium on Psychophysiology at Benjamin Franklin University Hospital* in Berlin, Germany
- 10/2006 "Increased frequency of bone marrow-residing FOXP3+CD4+CD25+ regulatory T cells in patients with Multiple Myeloma treated with allogeneic stem cell transplantation" presented at the *Annual Meeting of the European Society for Medical Oncology (ESMO)* in Istanbul, Turkey
- 06/2006 "Spontaneous and immunotherapy-induced immune responses against cancer-testis antigens in patients with malignant diseases" presented at the *Laboratory Training of the German Rheumatism Research Center* in Berlin, Germany
- 03/2006 "Effects of acute psychological stress on virus-specific and skin-homing T cells" presented at the *Annual Meeting of the German College of Psychosomatic Medicine (DKPM)* in Magdeburg, Germany
- 01/2005 "Preliminary results: T cell responses in patients with NSCLC following two cycles of immunization with MAGE-3 protein" presented at the *Meeting of the Cancer Vaccine Collaborative (CVC)* in New York, NY
- 12/2004 "The role of the immune system in the surveillance of cancer" presented at the *Research Meeting of the Eppendorf Association* in Hamburg, Germany
- 10/2004 "Danger signals and the development of new cancer vaccine strategies" presented at the *Annual International Cancer Immunotherapy Symposium of the Cancer Research Institute (CRI)* in New York, NY
- 04/2003 "NY-CO-58: Spontaneous CD4+ T cell responses and correlation with serum antibodies" presented at the *Meeting of the Cancer Vaccine Collaborative (CVC)* in New York, NY
- 12/2001 "Effects of hyperthermia and psychological stress on the adaptive immune system" presented at the *Psychophysiological Research Meeting*, Benjamin Franklin University Hospital in Berlin, Germany
- 09/2000 "Tumor Immunology" presented at *Onko Lunch*, University Medical Center Hamburg-Eppendorf

Invited Oral Presentations (intramural) - Selection

- 05/2025 "Chimeric antigen receptor (CAR) T cells" presented at *Community Research Forum Meeting*, University of Maryland School of Medicine / University of Maryland Greenebaum Comprehensive Cancer Center

Curriculum Vitae (Djordje Atanackovic, M.D.)

- 03/2025 "CD19/CD20/CD22 Tri-specific CAR" presented at the *GMP Advisory Council Kickoff Meeting*, University of Maryland School of Medicine / University of Maryland Greenebaum Comprehensive Cancer Center
- 01/2024 "Establishing tri-specific CAR T cells for B cell lymphoma patients at UMGCCC" presented at the *Clinical Faculty Meeting*, University of Maryland School of Medicine / University of Maryland Greenebaum Comprehensive Cancer Center
- 11/2023 "Tumor Immunology and Immunotherapy Program (TII)" presented at *UMGCCC Leadership Retreat*, Stevensville, MD
- 10/2023 "Immunomonitoring in CAR T cell patients" presented at the *2023 TII Annual Retreat*, University of Maryland School of Medicine / University of Maryland Greenebaum Comprehensive Cancer Center
- 10/2023 "Novel Cellular Immunotherapies for Cancer" presented at *Noon Conference of the Internal Medicine Residency Program*, University of Maryland School of Medicine
- 10/2023 "Incorporating Community Outreach into Research (Proposals)" presented at the *7th Annual Cancer Research Day*, University of Maryland School of Medicine / University of Maryland Greenebaum Comprehensive Cancer Center
- 05/2023 "Cellular immunotherapies for hepatocellular carcinoma" presented at the *Annual Research Matters Conference of the Maryland Cigarette Restitution Fund (CRF)*, University of Maryland School of Medicine / University of Maryland Greenebaum Comprehensive Cancer Center
- 12/2022 "Adoptive Cellular Immunotherapies for HCC" presented at the *2022 Liver Cancer Therapies*, University of Maryland School of Medicine / University of Maryland Greenebaum Comprehensive Cancer Center
- 09/2022 "Assessment of CAR T cell numbers and function in patients with hematologic malignancies" presented at the *Annual Retreat of Tumor the Immunology and Immunotherapy (TII) Program*, University of Maryland School of Medicine / University of Maryland Greenebaum Comprehensive Cancer Center
- 05/2022 "Development of Novel Cellular Immunotherapies at UMGCCC" presented at the *Annual Research Matters Conference of the Maryland Cigarette Restitution Fund (CRF)*, University of Maryland School of Medicine / University of Maryland Greenebaum Comprehensive Cancer Center
- 05/2022 "Cancer Immunotherapies" presented at the *Community Advisory Board*, University of Maryland School of Medicine / University of Maryland Greenebaum Comprehensive Cancer Center
- 12/2021 "Development of novel cancer immunotherapies at UMGCC" presented at the *School of Medicine Council Meeting*, University of Maryland School of Medicine / University of Maryland Greenebaum Comprehensive Cancer Center
- 12/2021 "Adoptive Cellular Immunotherapies for HCC" presented at the *2021 Liver Cancer Conference*, University of Maryland School of Medicine / University of Maryland Greenebaum Comprehensive Cancer Center
- 09/2021 "Design of novel cellular immunotherapies at UMGCCC" presented at the *Annual Retreat of Tumor the Immunology and Immunotherapy (TII) Program*, University of Maryland School of Medicine / University of Maryland Greenebaum Comprehensive Cancer Center
- 06/2021 "Development of Novel Cellular Immunotherapies at UMGCC" presented at the *Cigarette Restitution Fund Program - 2021 Cancer Research Grant Program Site*

- Visit, University of Maryland School of Medicine / University of Maryland Greenebaum Comprehensive Cancer Center
- 01/2021 “Developing anti-tumor immunotherapies in the times of COVID-19” presented at the *School of Medicine Grand Rounds*, University of Maryland School of Medicine / University of Maryland Greenebaum Comprehensive Cancer Center
- 09/2020 “Design of cellular immunotherapies in the times of COVID-19” presented at the *Annual Retreat of Tumor the Immunology and Immunotherapy (TII) Program*, University of Maryland School of Medicine / University of Maryland Greenebaum Comprehensive Cancer Center
- 03/2019 “Development of Novel Immunotherapies for Hematologic Malignancies and Solid Tumors” presented at *Syn-Tegration Night 2019*, University of Utah/Huntsman Cancer Institute
- 11/2018 “Development of CTA-specific Cellular Immunotherapies for Multiple Myeloma” presented at the *Hematology Faculty Conference*, University of Utah/Huntsman Cancer Institute
- 10/2018 “Chimeric Antigen Receptor (CAR) T cells for Hematologic Malignancies and Solid Tumors” presented at *Faculty Research Interest Session (FRIS)*, University of Utah
- 06/2018 “Development of T cell receptor-transduced T cells for Multiple Myeloma” presented at the *Research Retreat of the Division of Hematology*, University of Utah/Huntsman Cancer Institute
- 03/2018 “Induction of Anti-Tumor Immune Responses by Isatuximab” presented at *Sanofi Study Meeting*, University of Utah/Huntsman Cancer Institute
- 03/2018 “Development of cellular immunotherapies for human cancers” presented at *Research Leadership Council Meeting*, University of Utah/Huntsman Cancer Institute
- 01/2018 “Development of Multimodal Immunotherapies for Human Cancers” presented at *Johnson & Johnson Myeloma Working Group Meeting*, University of Utah/Huntsman Cancer Institute
- 05/2017 “Approaching a Cure: Update on the Diagnosis and Treatment of Multiple Myeloma” presented at *Geriatric Grand Rounds*, University of Utah
- 04/2017 “Development of Cellular Immunotherapies for Multiple Myeloma” presented at the *Hematology/Oncology Fellows Conference*, University of Utah/Huntsman Cancer Institute
- 04/2017 “Approaching a Cure: Update on the Diagnosis and Treatment of Multiple Myeloma” presented at *Internal Medicine Grand Rounds*, University of Utah/Huntsman Cancer Institute
- 11/2016 “Multiple Myeloma” presented at the *Hemepathology Conference*, University of Utah/Huntsman Cancer Institute
- 11/2016 “Immunotherapies for Multiple Myeloma” presented at the *Hematology/Oncology Fellows Conference*, University of Utah/Huntsman Cancer Institute
- 02/2016 “Multiple Myeloma” presented at the *Sixth Annual Hematology Review*, University of Utah/Huntsman Cancer Institute
- 11/2015 “Development of a Combinational Cellular Immunotherapy for Multiple Myeloma” presented at the *PPH and CCTS Pilot Award Symposium*, University of Utah

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- 11/2015 "The Development of Multimodal Immunotherapies for Multiple Myeloma" presented at the *Research Meeting of the Department of Pharmaceutical Chemistry*, University of Utah
- 07/2015 "Multiple Myeloma - Active Clinical Trials at HCI / University of Utah" presented at the *Novartis Research Meeting*, University of Utah/Huntsman Cancer Institute
- 03/2015 "Siltuximab for multicentric Castleman's disease: a randomised, double-blind, placebo-controlled trial" presented at the *Hematology/Oncology Fellows Journal Club*, University of Utah/Huntsman Cancer Institute
- 02/2015 "Multiple Myeloma" presented at the *Fifth Annual Hematology Review*, University of Utah/Huntsman Cancer Institute
- 02/2014 "Multiple Myeloma" presented at the *Fourth Annual Hematology Review*, University of Utah/Huntsman Cancer Institute
- 11/2013 "MAGE-A3 in NSCLC: Function and Immunogenicity" presented at the *TOPCARE Symposium*, University Medical Center Hamburg-Eppendorf, Hamburg, Germany
- 09/2013 "The daily dilemma – High-performance oncological medicine against the backdrop of rising costs" presented at the *Symposium for Ethics in Oncology*, University Medical Center Hamburg-Eppendorf, Hamburg, Germany
- 08/2013 "New drug therapies for non-small cell lung cancer" presented at the *Pulmonology Day*, University Medical Center Hamburg-Eppendorf, Hamburg, Germany
- 04/2013 "Chimeric Antigen Receptor–Modified T Cells for Acute Lymphoid Leukemia" presented at *Journal Club of the Division of Hematology/Oncology*, University Medical Center Hamburg-Eppendorf
- 02/2013 "The oncology patient in the intensive care unit" presented at *ICU Beginner Training*, University Medical Center Hamburg-Eppendorf
- 12/2012 "Oncologic approach to a thoracic nodule" presented at *Advanced training in oncology nursing*, University Medical Center Hamburg-Eppendorf
- 09/2012 "Immunotherapeutic approaches for GI cancers" presented at *Merck Colorectal Cancer Forum*, University Medical Center Hamburg-Eppendorf
- 02/2012 "Hemophagocytosis syndrome" presented at *Cytology Advanced Training*, University Medical Center Hamburg-Eppendorf
- 02/2012 "The oncology patient in the intensive care unit" presented at *ICU Beginner Training*, University Medical Center Hamburg-Eppendorf
- 02/2012 "Randomized Placebo-Controlled Phase II Trial of Perifosine Plus Capecitabine As Second- or Third-Line Therapy in Patients With Metastatic Colorectal Cancer" presented at *Journal Club of the Division of Hematology/Oncology*, University Medical Center Hamburg-Eppendorf
- 02/2012 "First-line therapy for EGFR-mutated patients with NSCLC" presented at *Onko Lunch*, University Medical Center Hamburg-Eppendorf
- 01/2012 "Cancer testis antigens as targets for immunotherapy of hematologic malignancies" presented at *MDS Subcommittee Meeting*, University Medical Center Hamburg-Eppendorf
- 08/2011 "Cancer testis antigens as targets for immunotherapy of multiple myeloma" presented at *University Cancer Center Hamburg (UCCH) Research Retreat*, University Medical Center Hamburg-Eppendorf
- 05/2011 "Tumor regression in patients with metastatic synovial cell sarcoma and melanoma using genetically engineered lymphocytes reactive with NY-ESO-1"

- presented at *Journal Club of the Division of Hematology/Oncology*, University Medical Center Hamburg-Eppendorf
- 05/2011 “Lung Cancer” presented at *Onco Workshop 2011*, University Medical Center Hamburg-Eppendorf, Hamburg, Germany
- 04/2011 “Current projects of the tumor immunology group” presented at *University Cancer Center Hamburg (UCCH) Research Retreat*, University Medical Center Hamburg-Eppendorf
- 02/2011 “Antigen-specific approaches for the immunotherapy of multiple myeloma” presented at *Onko Lunch*, University Medical Center Hamburg-Eppendorf
- 02/2011 “Medical therapy of lung cancer” presented at *Meeting of the Lung Cancer Task Force*, University Medical Center Hamburg-Eppendorf
- 02/2011 “Personalized therapy of NSCLC” presented at the *Bronchoscopy Course*, University Medical Center Hamburg-Eppendorf, Hamburg, Germany
- 12/2010 “ALK inhibitor crizotinib - The new star in the sky of NSCLC therapy?” presented at *Pulmonology Update*, University Medical Center Hamburg-Eppendorf, Hamburg, Germany
- 10/2010 “Immunotherapeutic strategies in MM” presented at the *Symposium for interdisciplinary treatment strategies for multiple myeloma*, University Medical Center Hamburg-Eppendorf, Hamburg, Germany
- 10/2010 “Colorectal liver metastases - Current oncological treatment concepts” presented at *Onko Lunch*, University Medical Center Hamburg-Eppendorf
- 09/2010 “Finally a breakthrough? News on T-cell-mediated immunotherapy for cancer” presented at *Mentoring program for excellent students - Retreat 2010*, University Medical Center Hamburg-Eppendorf
- 08/2010 “Sipuleucel-T Immunotherapy for Castration-Resistant Prostate Cancer” presented at *Journal Club of the Division of Hematology/Oncology*, University Medical Center Hamburg-Eppendorf
- 06/2010 “Colorectal liver metastases - Current oncological treatment concepts” presented at *Tumors of the liver and bile ducts: Current trends in diagnostics and therapy*, University Medical Center Hamburg-Eppendorf, Hamburg, Germany
- 06/2010 “Colorectal carcinoma” presented at *Onco Workshop 2010*, University Medical Center Hamburg-Eppendorf, Hamburg, Germany
- 06/2010 “Cytokine interleukin-16 is an important growth-promoting factor for multiple myeloma” presented at *University Cancer Center Hamburg (UCCH) Research Retreat*, University Medical Center Hamburg-Eppendorf
- 03/2010 “Study IP-CAT-GC-03 - Results of the immunomonitoring” at the *Catumaxomab Research Meeting*, University Medical Center Hamburg-Eppendorf
- 12/2009 “Phase III randomized comparison of gemcitabine versus gemcitabine plus capecitabine in patients with advanced pancreatic cancer” presented at *Journal Club of the Division of Hematology/Oncology*, University Medical Center Hamburg-Eppendorf
- 11/2009 “Vaccination against lung cancer - a meaningful therapeutic strategy?” presented at the *Eppendorf Afternoon – 3rd Update on the Interdisciplinary Diagnostics and Therapy of Bronchial Carcinoma*, University Medical Center Hamburg-Eppendorf, Hamburg, Germany

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- 01/2009 "Mortality in randomized trials of antioxidant supplements for primary and secondary prevention" presented at *Journal Club of the Division of Hematology/Oncology*, University Medical Center Hamburg-Eppendorf
- 04/2008 "Immunotherapy of Multiple Myeloma" presented at *Psychosocial Oncology*, University Medical Center Hamburg-Eppendorf
- 02/2008 "Tumor Immunology" presented at *Health Research Day "Fighting Cancer – Research for Humanity"*, University Medical Center Hamburg-Eppendorf
- 02/2008 "Cisplatin, Fluorouracil, and Docetaxel in Unresectable Head and Neck Cancer" presented at *Journal Club of the Division of Hematology/Oncology*, University Medical Center Hamburg-Eppendorf
- 02/2008 "The role of cancer testis antigens in the immunosurveillance of solid and hematological neoplasms" presented at *Research Retreat of the Division of Hematology/Oncology*, University Medical Center Hamburg-Eppendorf
- 01/2008 "The role of cancer testis antigens in immune surveillance and pathogenesis of hematological diseases" presented at the *Lab Research Update*, University Medical Center Hamburg-Eppendorf, Hamburg, Germany
- 07/2007 "Expression of cancer-testis antigens and anti-tumor immunity in patients with multiple myeloma following allogeneic stem cell transplantation" presented at *BMT Research Seminar*, University Medical Center Hamburg-Eppendorf
- 10/2006 "Overview on tumor immunology" presented at *Onko Lunch*, University Medical Center Hamburg-Eppendorf
- 07/2006 "Expression of the tumor antigen NY-CO-58 in various solid tumors" presented at the *Research Meeting of the Department of General Surgery*, University Medical Center Hamburg-Eppendorf, Hamburg, Germany
- 07/2006 "Enterocolitis in Patients With Cancer After Antibody Blockade of Cytotoxic T-Lymphocyte-Associated Antigen 4" presented at *Journal Club of the Division of Hematology/Oncology*, University Medical Center Hamburg-Eppendorf
- 03/2006 "Expression of CT antigens and anti-tumor immunity after allogeneic bone marrow transplantation in patients with multiple myeloma" presented at the *Lab Research Update*, University Medical Center Hamburg-Eppendorf, Hamburg, Germany
- 02/2006 "Monitoring of anti-tumor immune responses" presented at the *Clinical Lab of the Division of Hematology/Oncology Update*, University Medical Center Hamburg-Eppendorf, Hamburg, Germany
- 12/2005 "Tumor immunology: diagnostics, prognosis, therapy" presented at *Research Day of the Center for Internal Medicine*, University Medical Center Hamburg-Eppendorf
- 08/2005 "Design of T-cell-based immunotherapies" presented at *Research Retreat of the Division of Hematology/Oncology*, University Medical Center Hamburg-Eppendorf
- 04/2005 "Design of T-cell-based immunotherapies: Antigen discovery, clinical application, immunomonitoring" presented at *Novartis Research Meeting*, University Medical Center Hamburg-Eppendorf
- 01/2005 "T-cell-mediated immunity in solid tumors - Immunomonitoring following tumor vaccination" presented at *BMT Research Seminar*, University Medical Center Hamburg-Eppendorf
- 12/2004 "Post-transplantation lymphomas" presented at *Friday Seminars*, University Medical Center Hamburg-Eppendorf

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- 11/2004 "Post-transplantation lymphomas" presented at *Onko Lunch*, University Medical Center Hamburg-Eppendorf
- 08/2003 "Joint projects of the tumor immunology laboratory of the Medical Clinic II with the Surgical Department" presented at *Research Meeting of the Department of General Surgery*, University Medical Center Hamburg-Eppendorf
- 10/2001 "Monitoring of immunological effects of whole-body hyperthermia (WBH)" presented at *Meeting of Dr. Lloyd Old's Lab at the New York branch of the Ludwig Institute for Cancer Research*, Memorial Sloan-Kettering Cancer Center
- 06/2001 "Treatment of Insomnia" presented at *Onko Lunch*, University Medical Center Hamburg-Eppendorf

Invited Poster Presentations (extramural) - Selection

- 04/2025 "Distinct expansion, phenotype, function, and toxicity of cilta-cel vs. ide-cel CAR T cells in the real world" presented at the *Annual Meeting of the American Association for Cancer Research (AACR)* in Chicago, IL
- 04/2025 "Distinct expansion, phenotype, function, and toxicity of cilta-cel vs. ide-cel CAR T cells in the real world" presented at the *Annual Meeting of European Group for Blood and Bone Marrow Transplantation (EBMT)* in Florence, Italy
- 12/2024 "Racial and Ethnic Differences in Outcomes of Multiple Myeloma Patients Treated with Idecabtagene Vicleucel or Ciltacabtagene Autoleucel" presented at the *Annual Meeting of the American Society of Hematology (ASH)* in San Diego, CA
- 04/2024 "Targeting trogocytosis through cathepsin B inhibition enhances CAR T cell persistence" presented at the *Annual Meeting of the American Association for Cancer Research (AACR)* in San Diego, CA
- 04/2024 "In vivo expansion patterns of anti-BCMA CAR T-cell in relapsed/refractory multiple myeloma: Comparative immunomonitoring of idecabtagene vicleucel and ciltacabtagene autoleucel" presented at the *Annual Meeting of the American Association for Cancer Research (AACR)* in San Diego, CA
- 12/2023 "In vivo anti-BCMA CAR T-Cell expansion kinetics correlate with early IMWG response in RRMM: A single institution study with comparative analysis of Idecabtagene Vicleucel and Ciltacabtagene Autoleucel" presented at the *Annual Meeting of the American Society of Hematology (ASH)* in San Diego, CA
- 12/2023 "Trogocytosis-resistant CAR T Cells exhibit increased persistence and prevent antigen escape" presented at the *Annual Meeting of the American Society of Hematology (ASH)* in San Diego, CA
- 12/2023 "Engineering anti-CD229 CAR T cell selectivity for multiple myeloma" presented at the *Antibody Engineering and Therapeutics Annual Meeting* in San Diego, CA
- 04/2023 "A novel multicolor fluorescent spot assay for the functional assessment of CAR T cell products" presented at the *Annual Meeting of European Group for Blood and Bone Marrow Transplantation (EBMT)* in Paris, France
- 12/2022 "Clinical outcomes of CD19 CAR-T-Cell therapy with Axi-Cel in relapsed or refractory Diffuse Large B-Cell Lymphoma (DLBCL) with and without prior autologous stem cell transplant" presented at the *Annual Meeting of the American Society of Hematology (ASH)* in New Orleans, LA
- 12/2021 "Impaired mRNA based COVID-19 vaccine response in patients with B-cell malignancies after CD19 directed CAR-T cell therapy" presented at the *Annual Meeting of the American Society of Hematology (ASH)* in Atlanta, GA

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- 12/2021 “Imaging biomarkers to predict outcomes in patients with Large B-Cell Lymphoma with a day 28 partial response by PET/CT imaging following CAR-T therapy” presented at the *Annual Meeting of the American Society of Hematology (ASH)* in Atlanta, GA
- 12/2021 “CD229 CAR T cell therapy for the treatment of relapsed B cell lymphoma” presented at the *Annual Meeting of the American Society of Hematology (ASH)* in Atlanta, GA
- 12/2021 “Eight-day point of care CAR T-cell manufacturing on Clinimacs Prodigy from healthy donors as a proof-of-concept study” presented at the *Annual Meeting of the American Society of Hematology (ASH)* in Atlanta, GA
- 05/2021 “Engineering anti-CD229 CAR T cell selectivity for multiple myeloma” presented at the *Annual Meeting of the American Association of Immunologists (AAI)*, virtual meeting
- 05/2021 “CD19 loss by CAR T cell mediated trogocytosis is regulated by CAR affinity” presented at the *Annual Meeting of the American Association of Immunologists (AAI)*, virtual meeting
- 06/2020 “Isatuximab plus pomalidomide and dexamethasone in patients with relapsed/refractory multiple myeloma” presented at the *25th Congress of the European Hematology Association (EHA)*, virtual meeting
- 04/2020 “CD229-targeted CAR T cell therapy for the treatment of B cell lymphoma” presented at the *Annual Meeting of the American Association for Cancer Research (AACR)* in Philadelphia, PA
- 04/2020 “Engineering CD229 CAR T Cell Selectivity for Multiple Myeloma Using a High-Throughput Approach” presented at the *Annual Meeting of the American Association for Cancer Research (AACR)* in Philadelphia, PA
- 04/2020 “Enhancement of anti-tumor immunity by the adoptive transfer of T Cells engineered to restore HLA expression” presented at the *Annual Meeting of the American Association for Cancer Research (AACR)* in Philadelphia, PA
- 10/2019 “CD229 CAR T cells eliminate Multiple Myeloma and tumor propagating cells without fratricide” presented at the *5th International Congress on Controversies in Stem Cell Transplantation and Cellular Therapies (COSTEM)* in Berlin, Germany
- 12/2017 “Chimeric Antigen Receptor (CAR) T Cells Specific for CD229: A Potentially Curative Approach for Multiple Myeloma” presented at the *Annual Meeting of the American Society of Hematology (ASH)* in Atlanta, GA
- 12/2017 “‘In Vivo Vaccination’ Effect in Clinical Responders to Anti-Myeloma Monoclonal Antibody Isatuximab” presented at the *Annual Meeting of the American Society of Hematology (ASH)* in Atlanta, GA
- 04/2017 “A high-throughput process for the development and characterization of chimeric antigen receptors (CARs)” presented at the *Annual Meeting of the American Association for Cancer Research (AACR)* in Washington, DC
- 06/2016 “Updated data from a phase II dose finding trial of single agent isatuximab (SAR650984, anti-CD38 mAb) in relapsed/refractory multiple myeloma (RRMM)” presented at the *Annual Meeting of the American Society of Clinical Oncology (ASCO)* in Chicago, IL

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- 12/2015 "A dose finding phase II trial of Isatuximab (SAR650984, Anti-CD38 mAb) as a single agent in relapsed/refractory Multiple Myeloma" presented at the *Annual Meeting of the American Society of Hematology (ASH)* in Orlando, FL
- 12/2015 "Exome sequencing in myeloma pedigrees implicates RAS1 and NOTCH signaling are involved in inherited myeloma risk" presented at the *Annual Meeting of the American Society of Hematology (ASH)* in Orlando, FL
- 12/2015 "A genome-wide association study of myeloma survival in Utah uncovers germline variants that may influence survival of Multiple Myeloma patients" presented at the *Annual Meeting of the American Society of Hematology (ASH)* in Orlando, FL
- 12/2015 "Elevated rates of monoclonal gammopathy in high-risk chronic lymphocytic leukemia pedigrees" presented at the *Annual Meeting of the American Society of Hematology (ASH)* in Orlando, FL
- 06/2015 "Cancer-testis antigen expression in multiple myeloma" presented at the *20th Congress of the European Hematology Association (EHA)* in Vienna, Austria
- 10/2014 "Intraperitoneal Bevacizumab (Bev) for Control of Malignant Ascites Due to Advanced-Stage Gastrointestinal Cancers (AIO SUP-0108)" presented at the *Annual Meeting of the European Society for Medical Oncology (ESMO)* in Madrid, Spain
- 04/2013 "MAGE-C2 promotes proliferation and enhances resistance to p53 DNA damage-mediated apoptosis in multiple myeloma" presented at the *Annual Meeting of the American Association for Cancer Research (AACR)* in Washington, DC
- 12/2012 "Cancer-Testis antigen MAGE-C2/CT10 promotes proliferation and enhances resistance to p53 mediated apoptosis in multiple myeloma" presented at the *Annual Meeting of the American Society of Hematology (ASH)* in Atlanta, GA
- 12/2012 "Identification of atypical plasma cells in patients with MGUS or multiple myeloma through expression of immunoreceptor CD229" presented at the *Annual Meeting of the American Society of Hematology (ASH)* in Atlanta, GA
- 12/2012 "Impact of the expression of genes related to T Regulatory Cells on the survival of patients with multiple myeloma" presented at the *Annual Meeting of the American Society of Hematology (ASH)* in Atlanta, GA
- 10/2012 "2-Year Follow-Up of a Phase II Study on catumaxomab as part of a multimodal Approach in primarily resectable gastric cancer" presented at the *Annual Meeting of the European Society for Medical Oncology (ESMO)* in Vienna, Austria
- 06/2012 "Messenger RNA vaccination and B-cell responses in NSCLC patients" presented at the *Annual Meeting of the American Society of Clinical Oncology (ASCO)* in Chicago, IL
- 04/2012 "Generation of MAGE-C2/CT10-specific T cell clones for immunotherapy of multiple myeloma" presented at the *Annual Meeting of the American Association for Cancer Research (AACR)* in Chicago, IL
- 04/2012 "Molecular remission after autologous-allogeneic tandem transplantation in patients with multiple myeloma" presented at the *Annual Meeting of European Group for Blood and Bone Marrow Transplantation (EBMT)* in Geneva, Switzerland
- 04/2012 "Introduction of differential KIR activation mediated patterns of NK cell cytotoxicity against myeloma" presented at the *Annual Meeting of European Group for Blood and Bone Marrow Transplantation (EBMT)* in Paris, France

- 12/2011 “Achievement of sustained molecular remission induces long-term freedom from disease after autologous-allogeneic tandem transplantation in patients with multiple myeloma” presented at the *Annual Meeting of the American Society of Hematology (ASH)* in San Diego, CA
- 12/2011 “Overexpression of CTLA-4 in the bone marrow of patients with multiple myeloma as a sign of local accumulation of immunosuppressive Tregs – perspectives for novel treatment strategies” presented at the *Annual Meeting of the American Society of Hematology (ASH)* in San Diego, CA
- 12/2011 “Lenalidomide maintenance therapy after toxicity-reduced myeloablative allograft as salvage therapy for refractory/relapsed myeloma patients” presented at the *Annual Meeting of the American Society of Hematology (ASH)* in San Diego, CA
- 12/2011 “Analysis of spontaneous vs. vaccine-induced antibody responses against cancer-testis antigen MAGE-A3 in cancer patients” presented at the *Annual Meeting of the American Society of Hematology (ASH)* in San Diego, CA
- 10/2011 “1-year follow-up results of a phase II study with catumaxomab as part of a multimodal approach in patients with primary resectable gastric cancer” presented at the *Annual Meeting of the German Society for Hematology and Medical Oncology (DGHO)* in Basel, Switzerland
- 06/2011 “Messenger RNA vaccination in NSCLC: Findings from a phase I/IIa clinical trial” presented at the *Annual Meeting of the American Society of Clinical Oncology (ASCO)* in Chicago, IL
- 06/2011 “The effect of the trifunctional anti-EpCAM antibody catumaxomab on the development of tumor-specific immune responses in patients with gastric cancer” presented at the *Annual Meeting of the American Society of Clinical Oncology (ASCO)* in Chicago, IL
- 04/2011 “Cytokine interleukin-16 is an important growth-promoting factor for multiple myeloma” presented at the *Annual Meeting of the American Association for Cancer Research (AACR)* in Orlando, FL
- 12/2010 “Introduction of differential KIR activation mediated patterns of NK Cell cytotoxicity against myeloma” presented at the *Annual Meeting of the American Society of Hematology (ASH)* in Orlando, FL
- 12/2010 “Strong antibody responses against a unique region of CT-antigen SSX-2 are induced after allogeneic stem cell transplantation and correlate with clinical remission in patients with multiple myeloma” presented at the *Annual Meeting of the American Society of Hematology (ASH)* in Orlando, FL
- 12/2010 “MAGE-C1/CT7 inhibition increases myeloma cell line sensitivity to bortezomib-induced apoptosis: new target for myeloma therapy?” presented at the *Annual Meeting of the American Society of Hematology (ASH)* in Orlando, FL
- 12/2010 “Donor KIR-haplotype B improved relapse-free survival after allogeneic stem cell transplantation for multiple myeloma” presented at the *Annual Meeting of the American Society of Hematology (ASH)* in Orlando, FL
- 12/2010 “Interleukin-16 is an important growth-promoting factor and a novel diagnostic and therapeutic target for multiple myeloma” presented at the *Annual Meeting of the American Society of Hematology (ASH)* in Orlando, FL
- 10/2010 “Interleukin-16 is an important growth-promoting factor and a novel diagnostic and therapeutic target for multiple myeloma” presented at the *Annual International*

- Cancer Immunotherapy Symposium of the Cancer Research Institute (CRI) in New York, NY*
- 10/2010 “Cancer-Testis antigen FMR1NB/NY-SAR-35 is expressed on the surface of leukemic blasts with increased proliferative activity” presented at the *Annual International Cancer Immunotherapy Symposium of the Cancer Research Institute (CRI) in New York, NY*
- 10/2010 “Safety and tolerability of catumaxomab as part of a multimodal therapy approach in patients with primary resectable gastric cancer” presented at the *Annual Meeting of the German Society for Hematology and Medical Oncology (DGHO) in Berlin, Germany*
- 04/2010 “Surface molecule CD229 represents a possible target for the treatment of multiple myeloma” presented at the *Annual Meeting of the American Association for Cancer Research (AACR) in Washington, DC*
- 03/2010 “Surface molecule CD229 represents a target for the treatment of multiple myeloma” presented at the *Annual Meeting of European Group for Blood and Bone Marrow Transplantation (EBMT) in Vienna, Austria*
- 02/2010 “Identification of an immunodominant region of SSX-2 preferentially targeted by spontaneous antibodies in multiple myeloma patients” presented at the *Annual German Cancer Congress (Deutscher Krebskongress) in Berlin, Germany*
- 02/2010 “A myeloma-related cytokine/chemokine milieu in the bone marrow environment of patients before and after allogeneic stem cell transplantation” presented at the *Annual German Cancer Congress (Deutscher Krebskongress) in Berlin, Germany*
- 12/2009 “Longitudinal and Functional Analysis of Spontaneous NY-ESO-1-Specific Antibody Responses in Multiple Myeloma Patients” presented at the *Annual Meeting of the American Society of Hematology (ASH) in New Orleans, LA*
- 12/2009 “Related Vs Unrelated Donors After Auto-Allo Tandem Stem Cell Transplantation for Newly Diagnosed Patients with Multiple Myeloma” presented at the *Annual Meeting of the American Society of Hematology (ASH) in New Orleans, LA*
- 12/2009 “Allogeneic Hematopoietic Stem-Cell Transplantation with Reduced-Intensity Conditioning in Patients with Refractory and Relapsing Multiple Myeloma: Long-Term Follow-up” presented at the *Annual Meeting of the American Society of Hematology (ASH) in New Orleans, LA*
- 10/2009 “Identification of an immunodominant region of SSX-2 preferentially targeted by spontaneous antibodies in multiple myeloma patients” presented at the *Annual International Cancer Immunotherapy Symposium of the Cancer Research Institute (CRI) in New York, NY*
- 10/2009 “An optimized memory B cell assay for the monitoring of CT antigen-specific B cells in myeloma patients” presented at the *Annual Cancer Vaccines Meeting of the Cancer Research Institute (CRI) in New York, NY*
- 10/2009 “Allogeneic stem cell transplantation induces high-titered antibody responses to cancer testis antigens in multiple myeloma patients” presented at the *Annual Meeting of the German Society for Hematology and Medical Oncology (DGHO) in Heidelberg, Germany*
- 10/2009 “Expression and immunogenicity of cancer-testis antigens in acute myeloid leukemia” presented at the *Annual Meeting of the German Society for Hematology and Medical Oncology (DGHO) in Heidelberg, Germany*

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- 10/2009 "Preliminary results of MAGE-A3 expression and baseline demographic data from MAGRIT, a large phase III trial of MAGE-A3 ASCI (Antigen-Specific Cancer Immunotherapeutic) in adjuvant NSCLC" presented at the *Annual Meeting of the European Society for Medical Oncology (ESMO)* in Berlin, Germany
- 10/2009 "Allogeneic stem cell transplantation induces high-titered antibody responses to cancer testis antigens in multiple myeloma patients" presented at the *Annual Meeting of the European Society for Medical Oncology (ESMO)* in Berlin, Germany
- 10/2009 "An optimized memory B cell assay revealing CT antigen-specific B cells in myeloma patients" presented at the *Annual Meeting of the European Society for Medical Oncology (ESMO)* in Berlin, Germany
- 10/2009 "Expression and immunogenicity of cancer-testis antigens in acute myeloid leukaemia" presented at the *Annual Meeting of the European Society for Medical Oncology (ESMO)* in Berlin, Germany
- 09/2009 "Cancer-testis antigen expression and immunogenicity in chronic myeloid leukaemia" presented at the *European Congress of Immunology (ECI)* in Berlin, Germany
- 09/2009 "Expression of cancer-Testis antigens is increased following epigenetic treatment of Chronic Myeloid Leukemia cell lines but does not lead to specific humoral immune responses in patients with CML" presented at the *European Congress of Immunology (ECI)* in Berlin, Germany
- 09/2009 "A longitudinal analysis of antibody responses against cancer-testis antigens in multiple myeloma patients" presented at the *European Congress of Immunology (ECI)* in Berlin, Germany
- 09/2009 "The human tonsil represents a reservoir for long-lived tetanus toxoid-specific memory B cells" presented at the *European Congress of Immunology (ECI)* in Berlin, Germany
- 06/2009 "Antibody responses to cancer-testis antigens in multiple myeloma patients" presented at the *Congress of the European Hematology Association (EHA)* in Berlin, Germany
- 04/2009 "Expression, epigenetic regulation, and immunogenicity of cancer-testis antigens in chronic myeloid leukemia (CML)" presented at the *Annual Meeting of the American Association for Cancer Research (AACR)* in Denver, CO
- 03/2009 "Cancer-testis antigen expression and immunogenicity in chronic myeloid leukaemia" presented at the *Annual Meeting of European Group for Blood and Bone Marrow Transplantation (EBMT)* in Gothenburg, Sweden
- 12/2008 "Expression of cancer-testis antigens MAGE-C1/CT7 and MAGE-A3 is central to the survival of myeloma cells and their resistance to chemotherapy" presented at the *Annual Meeting of the American Society of Hematology (ASH)* in San Francisco, CA
- 12/2008 "Lenalidomide as salvage therapy after allogeneic stem cell transplantation in multiple myeloma" presented at the *Annual Meeting of the American Society of Hematology (ASH)* in San Francisco, CA
- 12/2007 "Anti-thymocyte globulin induces higher response rates and less graft-versus-host disease in multiple myeloma patients undergoing allogeneic stem cell transplantation" presented at the *Annual Meeting of the American Society of Hematology (ASH)* in San Francisco, CA

Curriculum Vitae (Djordje Atanackovic, M.D.)

- 10/2007 "Cancer-testis antigen expression in multiple myeloma over the course of the disease" presented at the *Annual International Cancer Immunotherapy Symposium of the Cancer Research Institute (CRI)* in New York, NY
- 06/2007 "CD4+CD25+FOXP3+ T regulatory cells reconstitute and accumulate in the bone marrow of patients with multiple myeloma following allogeneic stem cell transplantation" presented at the *XIth International Myeloma Workshop* on Kos, Greece
- 06/2007 "Analysis of bone marrow-residing CD4+CD25+FOXP3+ T regulatory cells in patients with multiple myeloma treated with allogeneic stem cell transplantation" presented at the *12th Congress of the European Hematology Association (EHA)* in Vienna, Austria
- 10/2006 "Frequent expression of cancer-testis in multiple myeloma results in the induction of systemic immunity following allogeneic stem cell transplantation" presented at the *Annual International Cancer Immunotherapy Symposium of the Cancer Research Institute (CRI)* in New York, NY
- 09/2006 "Cancer-testis antigens as possible targets for graft-versus-leukemia effects following allogeneic stem cell transplantation in patients with multiple myeloma" presented at the *European Congress of Immunology (ECI)* in Paris, France
- 06/2006 "Cancer-testis antigens are commonly expressed in multiple myeloma and induce systemic immunity following allogeneic stem cell transplantation" presented at the *Annual Meeting of the European Hematology Association (EHA)* in Amsterdam, Netherlands
- 03/2006 "Cancer-testis antigens are commonly expressed in multiple myeloma and induce systemic immunity following allogeneic stem cell transplantation" presented at the *Annual Meeting of European Group for Blood and Bone Marrow Transplantation (EBMT)* in Hamburg, Germany
- 02/2006 "Comprehensive analysis of the expression of cancer-testis antigens as possible targets for antigen-specific immunotherapy in patients with head and neck squamous cell carcinoma" presented at the *Annual MidWinter Research Meeting of the Association for Research in Otolaryngology (ARO)* in Baltimore, MD
- 12/2005 "Expression of cancer/testis antigens CT10 and SSX in multiple myeloma: promising targets for antigen-specific immunotherapy" presented at the *Annual Meeting of the American Society of Hematology (ASH)* in Atlanta, GA
- 12/2005 "Allogeneic stem cell transplantation from unrelated donors within the seventh decade of life" presented at the *Annual Meeting of the American Society of Hematology (ASH)* in Atlanta, GA
- 12/2005 "Early bortezomib following allogeneic stem cell transplantation (SCT) for multiple myeloma to enhance or maintain remission status" presented at the *Annual Meeting of the American Society of Hematology (ASH)* in Atlanta, GA
- 09/2005 "Frequent expression of Cancer-Testis antigens MAGE3, MAGEC1, CT10, BAGE and SSX genes in head and neck cancer: Possible targets for antigen-specific immunotherapy" presented at the *Annual Meeting of the German Society for Immunology (DGfI)* in Kiel, Germany
- 05/2005 "A local enrichment of regulatory T cells within the tumor tissue might suppress an effective anti-tumor T cell response in patients with head-and-neck cancer" presented at the *Annual Meeting of the American Society of Clinical Oncology (ASCO)* in Chicago, IL

- 04/2005 "Analysis of cytokine and chemokine concentrations within malignant effusions" presented at the *Annual Meeting of the American Association for Cancer Research (AACR)* in Anaheim, CA
- 06/2004 "Acute psychological stress causes a redistribution of memory/effector T cell subsets in humans" presented at the *25th European Conference on Psychosomatic Research (ECPR)* in Berlin, Germany
- 03/2004 "Vaccine-induced CD4+ T cell responses to MAGE-3 protein in lung cancer patients" presented at the *Annual Meeting of the American Association for Cancer Research (AACR)* in Orlando, FL
- 07/2003 "Deficient homing of effector type cytotoxic T cells to the tumor site" presented at the *Annual Meeting of the American Association for Cancer Research (AACR)* in Washington, DC
- 06/2003 "Effects of 41.8 °C whole body hyperthermia (WBH) on T cell homing molecules in patients with advanced malignancies" presented at the *Annual Meeting of the American Society of Clinical Oncology (ASCO)* in Chicago, IL
- 06/2003 "Whole-body hyperthermia combined with chemotherapy (WBH-CT): a single institution experience" presented at the *Annual Meeting of the European Society for Hyperthermic Oncology (ESHO)* in Munich, Germany
- 06/2001 "Whole body hyperthermia (WBH, 41.8°) combined with Oxaliplatin/5-Fluorouracil/Folinic acid (L-OHP/5-FU/FA) in heavily pretreated colorectal cancer: a phase II study" presented at the *Annual Meeting of the American Society of Clinical Oncology (ASCO)* in San Francisco, CA
- 12/2000 "Whole body hyperthermia induces expression of the activation antigen CD69 on peripheral lymphocytes of cancer patients" presented at the *Annual Meeting of the American Society of Hematology (ASH)* in San Francisco, CA
- 10/2000 "Whole body hyperthermia (WBH, 41.8 °C) as an adjunct to oxaliplatin (OXA)/5-FU/FA in high-dose (HD)-5-FU/FA or HD-5-FU/FA and CPT-11 refractory/relapsed advanced colorectal cancer" presented at the *Annual Meeting of the European Society for Medical Oncology (ESMO)* in Hamburg, Germany
- 11/1999 "CD4+/CD8+-Ratio bei Patienten mit Angststörungen bzw. Depression unter stationärer Psychotherapie" presented at the *Annual Meeting of the German College of Psychosomatic Medicine (DKPM)* in Berlin, Germany
- 09/1999 "Effects of acute experimental stress on hormones, lymphocyte subsets and phagocytic function in healthy persons" presented at the *Annual Meeting of the German-Endocrine-Brain-Immune-Network (GEBIN)* in Essen, Germany

XI. PUBLICATIONS

Original papers

1. Hardy, N.M., Yared, J.A., Luetkens, T., Ahmad, H., Mulatu, R., Yamoah, D., Fan, X., Weeks, S., Bredar, S.A., Gelin, A., Baker, J.M., Dietze, K.A., Matsangos, A., Fromowitz, A., Theruvath, J., Wirthlin, L., Kaleta, R., Wijatyk, A., Koka, R., Kallen, M.E., Hankey, K.G., Rapoport, A.P., **Atanackovic, D.** (2025) CNS infiltration by zamtocabtagene autoleucel tandem CD20/CD19 CAR T cells leading to complete remission in a patient with primary CNS lymphoma. *Haematologica* (online ahead of print)
2. **Atanackovic, D.**, Luetkens, T., Schneider, D., Hu, P., Wang, X., Shetty, A.C., Tallon, L., Patel, I., Singh, R., Gebru, E., Mulatu, R., Omili, D., Yamoah, D., Fan, X., Matsangos, A., Lesho, P., 1, Dietze, K.A., Fromowitz, A.A., Hankey, K.G., Dahiya, S., Yared, J.A., Hardy, N.M., Koka, R., Kallen, M.E., Badros, A., Rapoport, A.P., Kocoglu, M.H. (2025) Immune correlates of anti-BCMA

- CAR-T products idecabtagene vicleucel and ciltacabtagene autoleucel in a real-world cohort of patients with multiple myeloma. Nature Communications 16: 6154
3. Williams, M., **Atanackovic, D.**, Mulatu, R., Matsangos, A. Yamoah, D., Badros, A. (2025) Ciltacel CAR T cells as an effective and well tolerated treatment for POEMS: a case study. Haematologica 110: 2859-2863
 4. Olson, M.L., Luetkens, T., Wada, D.A., Miles, R.R., Yu, X., Iglesias, F., Radhakrishnan, S.V., Law, J., Avila, S.A., Vander Mause, E.R., Morales, E., Camp, N.J., Rapoport, A.P., Dahiya, S., Brody, J.D., Welm, A.L., **Atanackovic, D.** (2025) CD229 CAR T cells efficiently target B cell lymphomas with reduced trogocytosis. Nature Communications (under revision)
 5. Wake, L.M., Koka, R., Fromowitz, A.A., Hausner, P.F., Rapoport, A.P., **Atanackovic, D.**, Kallen, M.E. (2025) Lifileucel (AMTAGVI) tumor-infiltrating lymphocyte morphology in CellaVision peripheral smear reviews. Journal of Hematopathology 18: 10
 6. Banerjee, R., Richards, A., Midha, S., Afrough, A., Anwer, F., **Atanackovic, D.**, Atrash, S., Bachanova, V., Beitinjaneh, A.M., Bhurtel, E., Castaneda Puglianini, O., Chhabra, S., Cicero, K.I., Davis, J.A., Dhakal, B., Dima, D., Ferreri, C.J., Forsberg, P.A., Freeman, C.L., Herr, M.M., Jain, T., Janakiram, M., Khouri, J., Kocoglu, M.H., Kumar, A., Liu, Y., Locke, F., McGuirk, J.P., Mikkilineni, L., Nadeem, O., Parrondo, R.D., Pasvolsky, O., Peres, L.C., Purvey, S., Raza, S., Reshef, R., Richard, S., Rossi, A.C., Sborov, D.W., Shune, L., Wagner, C.B., Zanwar, S.S., Sidana, S., Patel, K.K., Hansen, D.K., Kumar, S.K., Lin, Y., Martin, T.G., Voorhees, P.M., Anderson, L.D. Jr, Cowan, A.J., Kaur, G. (2025) Universal driving restrictions beyond 4 weeks appear unnecessary following CAR-T therapy in multiple myeloma. Blood Advances 9: 2336-2340
 7. Hansen, D.K., Peres, L.C., Dima, D., Richards, A., Shune, L., Afrough, A., Midha, S., Dhakal, B., Kocoglu, M.H., Atrash, S., Ferreri, C., Castaneda, O., Davis, J.A., Bhurtel, E., McGuirk, J., Wagner, C., Bansal, R., Costello, P., Smith, K., Lieberman-Cribbin, A., De Avila, G., Purvey, S., Hosoya, H., Mikkilineni, L., Oswald, L.B., Kaur, .G., Pasvolsky, O., Gaballa, M., Herr, M.M., Forsberg, P., Janakiram, M., Htut, M., Asoori Maringanti, S., Kalariya, N., Hashmi, H., Reshef, R., Sborov, D.W., Nadeem, O., Anwer, F., Khouri, J., Raza, S., **Atanackovic, D.**, Alsina, M., Freeman, C.L., Locke, F.L., Voorhees, P., Anderson, L.D. Jr, Richard, S., Martin, T., Lin, Y., Patel, K.K., Sidana, S. (2025) Comparison of Standard-of-Care Idecabtagene Vicleucel and Ciltacabtagene Autoleucel in Relapsed/Refractory Multiple Myeloma. Journal of Clinical Oncology 43: 1597-1609
 8. Chen, D.T., Goloubeva, O., Rapoport, A.P., Dahiya, S., **Atanackovic, D.**, Hardy, N., Kocoglu, M., Lutfi, F., Alkhaldi, H., Claiborne, J.P., Lee, S.T., Kline, K., Law, J.Y., Yared, J.A. (2025) CD19 CAR-T With Axicabtagene Ciloleucel in R/R Large B-Cell Lymphoma With/Without Prior Autologous Stem Cell Transplant. Clinical Lymphoma, Myeloma & Leukemia 25: 432-439
 9. de Miguel-Perez, D., Arroyo-Hernandez, M., La Salvia, S., Gunasekaran, M., Pickering, E.M., Avila, S., Gebru, E., Becerril-Vargas, E., Monraz-Perez, S., Saharia, K., Grazioli, A., McCurdy, M.T., Frieman, M., Miorin, L., Russo, A., Cardona, A.F., García-Sastre, A., Kaushal, S., Hirsch, F.R., **Atanackovic, D.**, Sahoo, S., Arrieta, O., Rolfo, C. (2024) Extracellular vesicles containing SARS-CoV-2 proteins are associated with multi-organ dysfunction and worse outcomes in patients with severe COVID-19. Journal of Extracellular Vesicles 13: e70001
 10. Sidana, S., Patel, K.K., Peres, L.C., Bansal, R., Kocoglu, M.H., Shune, L., Atrash, S., Smith, K., Midha, S., Ferreri, C.J., Dhakal, B., Dima, D., Costello, P., Wagner, C., Reshef, R., Hosoya, H., Mikkilineni, L., **Atanackovic, D.**, Chhabra, S., Parrondo, R.D., Nadeem, O., Mann, H., Kalariya, N., Hovanky, V., DeAvila, G., Freeman, C.L., Locke, F.L., Alsina, M., Wong, S., Herr, M.M., Htut, M., McGuirk, J.P., Sborov, D.W., Khouri, J., Martin, T., Janakiram, M., Lin, Y., Hansen, D.K. (2024) Safety and Efficacy of Standard of Care Ciltacabtagene Autoleucel for Relapsed/Refractory Multiple Myeloma. Blood 145: 85-97
 11. Kocoglu, M.H., Luetkens, T., Bork, J.T., Baddley, J.W., Omili, D., Gebru, E., Mulatu, R., Yamoah, D., Iraguha, T., Fan, X., Lesho, P., Yousaf, M., Baker, J.M., Dietze, K.A., Hankey, K.G., Badros, A., Yared, J.A., Rapoport, A.P., Hardy, N.M., **Atanackovic, D.** (2024) Coordinated antiviral immune response in a myeloma patient with systemic adenovirus infection post BCMA CAR T cells. Blood Advances 8: 5880-5884

12. Li, A.Y., Kamangar, F., Holtzman, N.G., Rapoport, A.P., Kocoglu, M.H., **Atanackovic, D.**, Badros, A.Z. (2024) A Clinical Perspective on Plasma Cell Leukemia: A Single-Center Experience. Cancers (Basel) 16: 2149
13. Kallen, M.E., Koka, R., **Atanackovic, D.** (2024) Carvykti CAR T-cell morphology in cellvision peripheral smear reviews. Journal of Hematopathology 17: 231-233
14. Kocoglu, M.H., Richardson, P.G., Mo, C.C., Rapoport, A.P., **Atanackovic, D.** (2024) Defibrotide improves COVID-19-related acute respiratory distress syndrome in myeloma patients after chimeric antigen receptor T-cell treatment without compromising virus-specific and anti-myeloma T-cell responses. Haematologica 109: 2372-2377
15. Kline, K., Luetkens, T., Koka, R., Kallen, M.E., Chen, W., Ahmad, H., Omili, D., Iraguha, T., Gebru, E., Fan, X., Miller, A., Dishanthan, N., Baker, J.M., Dietze, K.A., Hankey, K.G., Yared, J.A., Hardy, N.M., Rapoport, A.P., Dahiya, S., **Atanackovic, D.** (2024) Treatment of secondary CNS lymphoma using CD19-targeted chimeric antigen receptor (CAR) T cells. Cancer Immunology Immunotherapy 73: 45
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Review Articles

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APPENDIX 2: MATERIALS CONSIDERED IN THE PREPARATION OF THIS DECLARATION

| Document No. | Description |
|---------------------|---|
| Paper 1 | Petition for Inter Partes Review of Claims 1-23 of U.S. Patent No. 10,792,370 |
| Paper 8 | Patent Owner’s Preliminary Response |
| Ex-1001 | U.S. Patent No. 10,792,370 to Hu |
| Ex-1002 | Declaration of Stylianos Bournazos, Ph.D. |
| Ex-1003 | File History of U.S. Patent No. 10,792,370 Part 1 |
| Ex-1004 | File History of U.S. Patent No. 10,792,370 Part 2 |
| Ex-1005 | U.S. Patent Application Publication No. US 2015-071923 (“Wei”) |
| Ex-1006 | International Publication No. WO 2014-152199 (“Leanna”) |
| Ex-1008 | Certified English Translation of Chinese Patent App. Pub. No. CN103772504 (“Liu”) |
| Ex-1009 | International Publication No. WO 2015-000062(“Tikhomirov”) |
| Ex-2006 | EGFR expression in normal human tissues (HPA RNA-seq normal tissues) - https://www.ncbi.nlm.nih.gov/gene/1956 |
| Ex-2007 | CD30 expression in normal human tissues (HPA RNA-seq normal tissues) - https://www.ncbi.nlm.nih.gov/gene/943 |
| Ex-2008 | European Patent Application No. EP4434549A1 |
| Ex-2011 | International Publication No. WO2012/100346A1 |
| Ex-2012 | Huang L. et al., “Abstract 1217: Preclinical evaluation of a next-generation, EGFR targeting ADC that promotes regression in KRAS or BRAF mutant tumors,” <i>Cancer Res.</i> (2016) 76 (14 Supplement): 1217 |
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| Ex-2014 | Carneiro B. et al., “Phase I study of anti-epidermal growth factor receptor antibody-drug conjugate serclutamab talirine: Safety, pharmacokinetics, and antitumor activity in advanced glioblastoma,” <i>Neuro-Oncology Advances</i> 5(1), 1–12, 2022 |
| Ex-2015 | U.S. Patent Application Publication No. 2011/0076232 (“Old-232”) |
| Ex-2016 | AbbVie, A Study Evaluating Safety and Pharmacokinetics of ABB-221 in Subjects with Advanced Solid Tumor Types Likely |

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| | to Exhibit Elevated Levels of Epidermal Growth Factor Receptor. ClinicalTrials.gov Identifier NCT02365662 (March 30, 2018) |
| Ex-2021 | Lepu Biopharma Co. Ltd. 2024 Inside Information Announcement |
| Ex-2022 | Crombet T., et al., “Use of the Humanized Anti-Epidermal Growth Factor Receptor Monoclonal Antibody h-R3 in Combination with Radiotherapy in the Treatment of Locally Advanced Head and Neck Cancer Patients,” <i>Journal of Clinical Oncology</i> 22(9):1646-1654, 2004 |
| Ex-2023 | Garrido G. et al., “Bivalent binding by intermediate affinity of nimotuzumab, A contribution to explain antibody clinical profile,” <i>Cancer Biology & Therapy</i> 11:4, 373-382; 2011 (Garrido) |
| Ex-2024 | Abdullah S. et al., “Dermatologic Toxicities from Monoclonal Antibodies and Tyrosine Kinase Inhibitors against EGFR: Pathophysiology and Management,” <i>Chemother Res Pract.</i> 2012 Sep 11; 2012:351210 |
| Ex-2025 | Burke P. et al., “Design, Synthesis, and Biological Evaluation of Antibody–Drug Conjugates Comprised of Potent Camptothecin Analogues,” <i>Bioconjug Chem</i> , 2009 Jun;20(6):1242-50 |
| Ex-2028 | ClinicalTrials.gov webpage for clinical trial NCT01741727 (downloaded from https://clinicaltrials.gov/study/NCT01741727 on January 19, 2026) |
| Ex-2029 | Phillips A.C., et al., “ABT-414, an Antibody–Drug Conjugate Targeting a Tumor-Selective EGFR Epitope,” <i>Mol Cancer Ther</i> (2016) 15 (4): 661–669 |
| Ex-2030 | Yu J. et al., “Antibody-Drug Conjugates Targeting the Human Epidermal Growth Factor Receptor Family in Cancers,” <i>Front. Mol. Biosci.</i> , 27 February 2022;9:847835. doi: 10.3389/fmolb.2022.847835 |

APPENDIX 3: UNDERSTANDING OF THE LAW

I have applied the following legal principles provided to me by counsel in arriving at the opinions set forth in this report.

Legal Standard for Prior Art

I am not an attorney. I have been informed by attorneys of the relevant legal principles and have applied them to arrive at the opinions set forth in this declaration.

I understand that the petitioner for inter partes review may request the cancelation of one or more claims of a patent based on grounds available under 35 U.S.C. § 102 and 35 U.S.C. § 103 using prior art that consists of patents and printed publications.

Anticipation and Prior Art

I understand that § 102 specifies when a challenged claim is invalid for lacking novelty over the prior art, and that this concept is also known as “anticipation.” I understand that a prior art reference anticipates a challenged claim, and thus renders it invalid by anticipation, if all elements of the challenged claim are disclosed in the prior art reference. I understand the disclosure in the prior art reference can be either explicit or inherent, meaning it is necessarily present or implied. I understand that the prior art reference does not have to use the same words as the challenged claim, but all of the requirements of the claim must

be disclosed so that a person of ordinary skill in the art could make and use the claimed subject-matter.

In addition, I understand that § 102 also defines what is available for use as a prior art reference to a challenged claim.

Under § 102(a)(1), a challenged claim is anticipated if it was patented, described in a printed publication, or in public use, on sale, or otherwise available to the public before the effective filing date of the challenged claimed invention.

Under § 102(a)(2), a challenged claim is anticipated if it was described in an issued patent, or in an application for patent that was published by the USPTO, in which the patent or application, as the case may be, names another inventor and was effectively filed before the effective filing date of the challenged claimed invention.

I understand that the filing date of a patent is generally the filing date of the application filed in the United States that issued as the patent. However, I understand that a patent may be granted an earlier effective filing date if the patent owner properly claimed priority to an earlier patent application.

I understand that when a challenged claim covers several structures, either generically or as alternatives, the claim is deemed anticipated if any of the structures within the scope of the claim is found in the prior art reference.

I understand that when a challenged claim requires selection of an element from a list of alternatives, the prior art teaches the element if one of the alternatives is taught by the prior art.

Legal Standard for Obviousness

I understand that even if a challenged claim is not anticipated, it is still invalid if the differences between the claimed subject matter and the prior art are such that the claimed subject matter would have been obvious to a person of ordinary skill in the pertinent art at the time the alleged invention.

I understand that obviousness must be determined with respect to the challenged claim as a whole.

I understand that one cannot rely on hindsight in deciding whether a claim is obvious.

I also understand that an obviousness analysis includes the consideration of factors such as (1) the scope and content of the prior art, (2) the differences between the prior art and the challenged claim, (3) the level of ordinary skill in the pertinent art, and (4) “secondary” or “objective” evidence of non-obviousness.

Secondary or objective evidence of non-obviousness includes evidence of: (1) a long felt but unmet need in the prior art that was satisfied by the claimed invention; (2) commercial success or the lack of commercial success of the claimed invention; (3) unexpected results achieved by the claimed invention; (4)

praise of the claimed invention by others skilled in the art; (5) taking of licenses under the patent by others; (6) deliberate copying of the claimed invention; and (7) contemporaneous and independent invention by others. However, I understand that there must be a relationship between any secondary evidence of non-obviousness and the claimed invention.

I understand that a challenged claim can be invalid for obviousness over a combination of prior art references if a reason existed (at the time of the alleged invention) that would have prompted a person of ordinary skill in the art to combine elements of the prior art in the manner required by the challenged claim. I understand that this requirement is also referred to as a “motivation to combine,” “suggestion to combine,” or “reason to combine,” and that there are several rationales that meet this requirement.

I understand that the prior art references themselves may provide a motivation to combine, but other times simple common sense can link two or more prior art references. I further understand that obviousness analysis recognizes that market demand, rather than scientific literature, often drives innovation, and that a motivation to combine references may come from market forces.

I understand obviousness to include, for instance, scenarios where known techniques are simply applied to other devices, systems, or processes to improve them in an expected or known way. I also understand that practical and common-

sense considerations should be applied in a proper obviousness analysis. For instance, familiar items may have obvious uses beyond their primary purposes.

I understand that the combination of familiar elements according to known methods is obvious when it yields predictable results. For instance, obviousness bars patentability of a predictable variation of a technique even if the technique originated in another field of endeavor. This is because design incentives and other market forces can prompt variations of it, and predictable variations are not the product of innovation, but rather ordinary skill and common sense.

I understand that a particular combination may be obvious if it was obvious to try the combination. For example, when there is a design need or market pressure to solve a problem and there are a finite number of identified, predictable solutions, a person of ordinary skill may have good reason to pursue the known options within his or her technical grasp. This would result in something obvious because the result is the product not of innovation but of ordinary skill and common sense. However, I understand that it may not be obvious to try a combination when it involves unpredictable technologies or requires trying each of numerous possible choices unreduced by direction of the prior art.

It is further my understanding that a proper obviousness analysis focuses on what was known or obvious to a person of ordinary skill in the art, not just the patentee. Accordingly, I understand that any need or problem known in the field of

endeavor at the time of invention and addressed by the patent can provide a reason for combining the elements in the manner claimed.

It is my understanding that the Manual of Patent Examining Procedure §2143 sets forth the following as exemplary rationales that support a conclusion of obviousness:

- Combining prior art elements according to known methods to yield predictable results;
- Simple substitution of one known element for another to obtain predictable results;
- Use of known technique to improve similar devices (methods, or products) in the same way;
- Applying a known technique to a known device (method, or product) ready for improvement to yield predictable results;
- Choosing from a finite number of identified, predictable solutions, with a reasonable expectation of success;
- Known work in one field of endeavor may prompt variations of it for use in either the same field or a different one based on design incentives or other market forces if the variations are predictable to one of ordinary skill in the art;

- Some teaching, suggestion, or motivation in the prior art that would have led one of ordinary skill to modify the prior art reference or to combine prior art reference teachings to arrive at the claimed invention.

A person of ordinary skill in the art looking to overcome a problem will often use the teachings of multiple publications together like pieces of a puzzle, even though the prior art does not necessarily fit perfectly together. Therefore, I understand that references for obviousness need not fit perfectly together like puzzle pieces. Instead, I understand that obviousness analysis takes into account inferences, creative steps, common sense, and practical logic and applications that a person of ordinary skill in the art would employ under the circumstances.

I understand that a claim can be obvious in light of a single reference, if the elements of the challenged claim that are not explicitly or inherently disclosed in the reference can be supplied by the common sense of one of skill in the art.

In order for a claim to be found invalid based upon a modification or combination of the prior art, there must be reasonable expectation that a person of ordinary skill would have successfully modified or combined the prior art to arrive at the claimed arrangement. This does not mean that it must be certain that a person of ordinary skill would have been successful – the law only requires that the

person of ordinary skill in the art would have perceived a reasonable expectation of success in modifying or combining the prior art to arrive at the claimed invention.

I understand that obviousness may be defeated if the prior art indicates that the invention would not have worked for its intended purpose or otherwise teaches away from the invention. A reference teaches away when a person of ordinary skill, upon reading the reference, would be discouraged from following the path set out in the reference, or would be led in a direction divergent from the path that was taken in the claim. A reference that merely expresses a general preference for an alternative invention but does not criticize, discredit, or otherwise discourage investigation into the claimed invention does not teach away.

In sum, my understanding is that obviousness invalidates claims that merely recite combinations of, or obvious variations of, prior art teachings using understanding and knowledge of one of skill in the art at the time and motivated by the general problem facing the inventor at the time. Under this analysis, the prior art references themselves, or any need or problem known in the field of endeavor at the time of the invention, can provide a reason for combining the elements of or attempting obvious variations on prior art references in the claimed manner.

Legal Standard for Claim Construction

I understand that before any invalidity analysis can be properly performed, the scope and meaning of the challenged claims must be determined by claim construction.

I understand that a patent may include two types of claims, independent claims and dependent claims. I understand that an independent claim stands alone and includes only the limitations it recites. I understand that a dependent claim depends from an independent claim or another dependent claim. I understand that a dependent claim includes all the limitations that it recites in addition to the limitations recited in the claim (or claims) from which it depends.

In comparing the challenged claims to the prior art, I have carefully considered the patent and its file history in light of the understanding of a person of skill at the time of the alleged invention.

I understand that to determine how a person of ordinary skill would have understood a claim term, one should look to sources available at the time of the alleged invention that show what a person of skill in the art would have understood disputed claim language to mean. It is my understanding that this may include what is called “intrinsic” evidence as well as “extrinsic” evidence.

I understand that, in construing a claim term, one should primarily rely on intrinsic patent evidence, which includes the words of the claims themselves, the

remainder of the patent specification, and the prosecution history. I understand that extrinsic evidence, which is evidence external to the patent and the prosecution history, may also be useful in interpreting patent claims when the intrinsic evidence itself is insufficient. I understand that extrinsic evidence may include principles, concepts, terms, and other resources available to those of skill in the art at the time of the invention.

I understand that words or terms should be given their ordinary and accepted meaning unless it appears that the inventors were using them to mean something else or something more specific. I understand that to determine whether a term has special meaning, the claims, the patent specification, and the prosecution history are particularly important, and may show that the inventor gave a term a particular definition or intentionally disclaimed, disavowed, or surrendered claim scope.

I understand that the claims of a patent define the scope of the rights conferred by the patent. I understand that because the claims point out and distinctly claim the subject matter which the inventors regard as their invention, claim construction analysis must begin with and is focused on the claim language itself. I understand that the context of the term within the claim as well as other claims of the patent can inform the meaning of a claim term. For example, because claim terms are normally used consistently throughout the patent, how a term is used in one claim can often inform the meaning of the same term in other claims.

Differences among claims or claim terms can also be a useful guide in understanding the meaning of particular claim terms.

I understand that a claim term should be construed not only in the context of the particular claim in which the disputed term appears, but in the context of the entire patent, including the entire specification. I understand that because the specification is a primary basis for construing the claims, a correct construction must align with the specification.

I understand that the prosecution history of the patent as well as art incorporated by reference or otherwise cited during the prosecution history are also highly relevant in construing claim terms. For instance, art cited by or incorporated by reference may indicate how the inventor and others of skill in the art at the time of the invention understood certain terms and concepts. Additionally, the prosecution history may show that the inventors disclaimed or disavowed claim scope, or further explained the meaning of a claim term.

With regard to extrinsic evidence, I understand that all evidence external to the patent and prosecution history, including expert and inventor testimony, dictionaries, and learned treatises, can also be considered. For example, technical dictionaries may indicate how one of skill in the art used or understood the claim terms. However, I understand that extrinsic evidence is considered to be less

reliable than intrinsic evidence, and for that reason is generally given less weight than intrinsic evidence.

I understand that in general, a term or phrase found in the introductory words or preamble of the claim, should be construed as a limitation if it recites essential structure or steps, or is necessary to give meaning to the claim. For instance, I understand preamble language may limit claim scope: (i) if dependence on a preamble phrase for antecedent basis indicates a reliance on both the preamble and claim body to define the claimed invention; (ii) if reference to the preamble is necessary to understand limitations or terms in the claim body; or (iii) if the preamble recites additional structure or steps that the specification identifies as important.

On the other hand, I understand that a preamble term or phrase is not limiting where a challenged claim defines a structurally complete invention in the claim body and uses the preamble only to state a purpose or intended use for the invention. I understand that to make this determination, one should review the entire patent to gain an understanding of what the inventors claim they invented and intended to encompass in the claims.

I understand that 35 U.S.C. § 112(f) created an exception to the general rule of claim construction called a “means plus function” limitation. These types of terms and limitations should be interpreted to cover only the corresponding

structure described in the specification, and equivalents thereof. I also understand that a limitation is presumed to be a means plus function limitation if (a) the claim limitation uses the phrase “means for”; (b) the “means for” is modified by functional language; and (c) the phrase “means for” is not modified by sufficient structure for achieving the specified function.

I understand that a structure is considered structurally equivalent to the corresponding structure identified in the specification only if the differences between them are insubstantial. For instance, if the structure performs the same function in substantially the same way to achieve substantially the same result. I further understand that a structural equivalent must have been available at the time of the issuance of the claim.