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UNITED STATES PATENT AND TRADEMARK OFFICE

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BEFORE THE PATENT TRIAL AND APPEAL BOARD

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Ex parte YOICHIRO IWAKURA, NORIYUKI FUJIKADO, and GUANGYU MA

_____

Appeal 2019-002524
Application 13/580,689[1]
Technology Center 1600

_____

Before ERIC B. GRIMES, FRANCISCO C. PRATS, and RACHEL H. TOWNSEND, *Administrative Patent Judges*.

TOWNSEND, *Administrative Patent Judge*.

DECISION ON APPEAL

This is an appeal under 35 U.S.C. § 134 involving claims to a method of treating a disease accompanied by abnormal bone metabolism, which have been rejected as failing to comply with the written description requirement and as being obvious. We have jurisdiction under 35 U.S.C. § 6(b).

We affirm.

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[1] We use the word "Appellant" to refer to "Applicant" as defined in 37 C.F.R. § 1.42. Appellant identifies the real party in interest as the University of Tokyo. (Appeal Br. 2.)

STATEMENT OF THE CASE

"Dendritic cells (DCs) play a central role in immune system regulation." (Spec. ¶ 2.) DCIR (also called LLIR) is a receptor expressed on dendritic cells. (*Id.* ¶¶ 2, 3.) "[T]he mouse DCIR is suggested to act as an inhibitory receptor and to control the dendritic cell function." (*Id.* ¶ 3.) DCIR has been reported to be involved in development of arthritis and rheumatoid arthritis ("RA"). (*Id.*)

Claims 17, 19, 21–24, 26–29, 37, and 38 are on appeal. Claims 17 and 38 are representative and read as follows:

> 17. A method of treating a disease accompanied by abnormal bone metabolism selected from the group consisting of osteoporosis, Paget's disease, osteitis deformans, and rheumatoid arthritis in a subject in need thereof, comprising administering to the subject an antibody or a Fab fragment thereof wherein the antibody or Fab fragment specifically binds to human dendritic immunoreceptor (LLIR) or mouse dendritic cell immunoreceptor Clec4a2, acts as an agonist of a dendritic cell immunoreceptor and treats the disease, wherein the antibody or the Fab fragment thereof is not modified with another molecule or is optionally chemically linked to only polyethylene glycol (PEG).
>
> 38. A method of suppressing osteoclast production in a subject in need thereof, the method comprising administering an antibody or a Fab fragment thereof to the subject, wherein the antibody or Fab fragment specifically binds to human dendritic immunoreceptor (LLIR) or mouse dendritic cell immunoreceptor Clec4a2, acts as an agonist of a dendritic cell immunoreceptor, and inhibits GM-CSF action to suppress osteoclast production, wherein the antibody or the Fab fragment thereof is not modified with another molecule or is optionally chemically linked to only polyethylene glycol (PEG).

(Appeal Br. 2, 4.)

The following grounds of rejection by the Examiner are before us on review:

Claims 17, 19, 21–24, 26–29, 37, and 38 under 35 U.S.C. § 112, first paragraph as failing to comply with the written description requirement.

Claims 17, 19, 21–23, 26–28, 37, and 38 under 35 U.S.C. § 103 as unpatentable over Iwakura,[2] Valladeau,[3] Meyer-Wentrup,[4] and Vasanthi,[5]

Claims 24 and 29 under 35 U.S.C. § 103 as unpatentable over Iwakura, Valladeau, Meyer-Wentrup, Vasanthi, and Chapman.[6]

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[2] Iwakura et al., US 2009/0202440 A1, published Aug. 13, 2009. The Examiner refers to this as the '440 publication. We, however, refer to it as Iwakura herein.

[3] Valladeau et al., US 6,277,959 B1, issued Aug. 21, 2001. The Examiner refers to this as the '959 patent. We, however, refer to it as Valladeau herein. The Examiner also relies on GenBankAccession # AAH96565, available at https://www.ncbi.nlm.nih.gov/protein/AAH96565.1, to demonstrate that SEQ ID NO:8 of this reference is mouse Clec4a2. (Non-Final Action 6.)

[4] Meyer-Wentrup, F., et al., *DCIR is endocytosed into human dendritic cells and inhibits TLR8-mediated cytokine production*, 85 J. Leukocyte Biology, 518–25 (2009). The Examiner also relies on an R&D Systems product information sheet for the Murine anti-human DCIR mAb clone 216110 from R&D systems described in this reference, which information sheet indicates the antibody is unconjugated. This R&D Systems information sheet further references the immunogen used to obtain the antibody is a recombinant human DCIR Accession #Q9UMR7. The Examiner cites to the UniProt Accession #Q9UMR7, available at https://www.ncbi.nlm.nih.gov/protin/59797977?sat=11&satkey=7804620, but does not indicate for what purpose it is being relied upon in the rejection. (Non-Final Action 5–7.)

[5] Vasanthi, P., et al., *Role of tumor necrosis factor-alpha in rheumatoid arthritis: a review*, 10 APLAR J of Rheumatology, 270–74 (2007).

[6] Chapman, A.P., et al. *Therapeutic antibody fragments with prolonged in vivo half-lives*, 17 Nature Biotechnology, 780–83 (1999).

DISCUSSION

Lack of Adequate Written Description

The Examiner notes that the claims are "drawn to a method employing genus of antibodies that function to bind to human dendritic cell immunoreceptor LLIR or mouse Clec4a2, and also function as agonist of the dendritic cell immunoreceptor, inhibit GM-CSF action, and suppression of osteoclast production and/or treatment of disease." (Non-Final Action[7] 3.) The Examiner finds that the claims encompass a large genus of structurally and functionally distinct antibodies. (*Id.*) In particular, the Examiner finds that the antibodies can be from human, rat, or mouse, can have different VH/VL and CDR sequences, can bind to human or mouse LLIR/Clec4a2 at different epitopes with different functions such as inhibiting GM-CSF action or suppressing osteoclasts. (*Id.*) The Examiner finds that while three particular mouse monoclonal antibodies that bind to mouse dendritic cell immunoreceptor Clec4a2 and inhibit osteoclast differentiation and suppress TNF-alpha production are disclosed in the Specification, neither the structure nor the amino acid sequence of those antibodies is disclosed. (*Id.*)

Moreover, the Examiner finds that the Specification fails to disclose a correlation between the structure of one of those antibodies, or any other, and the functions claimed, i.e., "suppressing osteoclasts, inhibiting GM-CSF action, or treating diseases." (*Id.*) The Examiner further explains that "[t]he mechanistic studies cited by Appellant do not provide any information as to the actual structures of the antibodies that would function as claimed." (Ans. 9.) The Examiner concludes that in light of the scant disclosure in

[7] The Non-Final Action is dated March 27, 2018.

Appellant's Specification that "one of skill in the art would conclude that Applicant was not in possession of the structural attributes of a representative number of species possessed by the members of the genus of dendritic cell immunoreceptor antibodies with the claimed specificity and functional attributes, broadly encompassed by the claimed invention." (*Id.* at 5.)

We agree with the Examiner's factual findings and conclusion that the claimed methods are not adequately described. Appellant does not argue the claims separately. We address claim 17 as representative.

A description adequate to satisfy 35 U.S.C. § 112, first paragraph, must "'clearly allow persons of ordinary skill in the art to recognize that [the inventor] invented what is claimed.'" *Ariad Pharms., Inc. v. Eli Lilly & Co.*, 598 F.3d 1336, 1351 (Fed. Cir. 2010) (en banc). A patent disclosure is sufficient if it "reasonably conveys to those skilled in the art that the inventor had possession of the claimed subject matter as of the filing date," based on an "objective inquiry into the four corners of the specification." *Id.* A "sufficient description of a genus . . . requires the disclosure of either a representative number of species falling within the scope of the genus or structural features common to the members of the genus so that one of skill in the art can 'visualize or recognize' the members of the genus" and not just "a generic statement of an invention's boundaries." *Id.* at 1349–1350 (citing *Regents of the University of California v. Eli Lilly & Co.*, 119 F.3d 1559, 1568–69 (Fed. Cir. 1997)); *Amgen Inc. v. Sanofi*, 872 F.3d 1367, 1373 (Fed. Cir. 2017).

We agree with the Examiner that the number of antibodies encompassed by the claim for use in the method of treatment is a large

genus. Claim 17 sets forth three functional requirements for the antibodies that are within the scope of the claim. The first is that it "specifically binds to human dendritic immunoreceptor (LLIR) or mouse dendritic cell immunoreceptor Clec4a2." The second is that the antibody "acts as an agonist of a dendritic cell immunoreceptor." And the third function is that the antibody treats "a disease accompanied by abnormal bone metabolism selected from the group consisting of osteoporosis, Paget's disease, osteitis deformans, and rheumatoid arthritis." Appellant's Specification states that antibodies for use in the claimed invention, include monoclonal, polyclonal and antibody mutants and derivatives. (Spec. ¶ 27.) And, as the Examiner pointed out in the Answer, and Appellant does not dispute, "antibodies is intended to encompass mouse antibodies, human antibodies, rat antibodies, camel antibodies and bird antibodies as well as chimeric or humanized antibodies." (Ans. 10; *see* Spec. ¶ 28.)

The Specification describes production of antibodies to DCIR in Example 5. (Spec. ¶¶ 62–66.) Some of the antibodies produced were determined to have a higher DCIR binding ability than that of keratan sulfate II (KS-II), a native ligand for DCIR and to more highly suppress TNF-alpha production (Spec. ¶¶ 8, 67 (identifying hybridoma strains "3, 4, and 5 . . . in Figure 12")), and others promoted TNF-alpha compared to KS-II (*id.* ¶ 67 (identifying hybridoma strains "1 and 2 . . . in Figure 12"). The Specification states that the former "are useful as DCIR agonists that enhance DCIR activity," where the latter "are useful as DCIR antagonists that suppress DCIR activity." (*Id.*) The Specification further notes that the "[t]he hybridoma supernatants (DCIR agonist (3, 4, 5 strains in Figure 13)) showing higher DCIR binding abilities than that of keratan sulfate in

Example 5 suppressed osteoclast differentiation, compared with keratan sulfate" whereas the supernatants of 1 and 2 strains "promoted osteoclast differentiation, compared with keratan sulfate." (*Id.* ¶ 68.)

Thus, Appellant's Specification describes producing mouse antibodies to mouse Clec4a2 and evaluating the antibodies from monoclonal hybridoma culture supernatant for DCIR-binding ability and assessing agonistic as compared to antagonistic activity by measuring TNF-alpha production (Spec. 46–54.) However, it does not disclose the structure or amino acid sequence of any antibodies produced. Nor does it name those antibodies or indicate that the antibodies were deposited in a public depository. *Enzo Biochem, Inc. v. Gen-Probe Inc.*, 323 F.3d 956, 965 (Fed. Cir. 2002) ("[R]eference in the specification to a deposit in a public depository, which makes its contents accessible . . . constitutes an adequate [written] description . . .").

Indeed, Appellant does not dispute that no structure of the antibodies for use in the claimed method is disclosed. Instead, Appellant contends only that the Specification provides sufficient description that the disclosed agonist antibodies function to suppress osteoclast differentiation. (Appeal Br. 7–8 (referring in part to Example 7).) While the Federal Circuit has recognized that "the written description requirement can in some cases be satisfied by functional description," it has made clear that "such functional description can be sufficient only if there is also a structure-function relationship known to those of ordinary skill in the art." *In re Wallach*, 378 F.3d 1330, 1335 (Fed. Cir. 2004); *see also Enzo Biochem*, 323 F.3d at 964 (holding that the written description requirement would be satisfied "if the functional characteristic of preferential binding . . . were coupled with a

disclosed correlation between that function and a structure that is sufficiently known or disclosed"); *Amgen*, 872 F.3d at 1378 (holding that an "adequate written description must contain enough information about the actual makeup of the claimed products").

Such is not the case here, as Appellant's arguments in the Appeal Brief make clear. (*See, e.g.*, Appeal Br. 7–10.) Appellant argues that "there are many antibodies which can be linked to the specific receptor (DCIR) to induce an agonistic signal[]" (Appeal Br. 8) and that "[a] skilled person in the art would easily identify the claimed agonistic antibodies by differentiating osteoclasts by using bone marrow cells derived from DCIR-deficient mouse or derived from WT mouse" (*id.* at 9). However, as the en banc decision in *Ariad* made clear, to satisfy the statutory requirement of a description of the invention, it is not enough for the specification to show how to make and use the invention, i.e., to enable it. *Ariad*, 598 F.3d at 1345–46, 1347–48.

Appellant does not identify a structure-function relationship for the antibodies disclosed as agonists and which can function in the additional manner recited by the claim. And, as the Examiner notes, Appellant argues that "certain species of DCIR antibodies were known in the art" but that those "antibodies do not possess the same function as the claimed antibodies." (Ans. 9; Appeal Br. 11–12.) Thus, it cannot be disputed, as the Examiner pointed out, that there is not a well-known correlation between structure and function to support the claimed genus of antibodies. (Ans. 9.)

Appellant's argument that the claim recites sufficient specificity to describe the genus of antibodies that may be used in the claimed method (Appeal Br. 8) misunderstands the written description rejection, which is

that the Specification fails to reasonably convey to one of ordinary skill in the art that the inventors had possession of the broad genus of antibodies embraced by the claim. The argument, however, does confirm the Examiner's position that the genus of antibodies being claimed is vast.

Claim 17 and Appellant's Specification here is similar to the claims and disclosure in *University of Rochester v. G.D. Searle & Co.*, 358 F.3d 916 (Fed. Cir. 2004), where the claims were found to be inadequately described. In that case, claims directed to a method of selectively inhibiting the COX-2 enzyme by administering a non-steroidal compound that selectively inhibits the COX-2 enzyme, were found inadequately described where the patent Specification at issue there did not describe any specific compound capable of performing the claimed method and the skilled artisan would not be able to identify any such compound based on the Specification's functional description. *Id.* at 927–28. As the Federal Circuit in *Ariad* summed up, the claims in *University of Rochester* "merely recite a description of the problem to be solved while claiming all solutions to it and . . . cover any compound later actually invented and determined to fall within the claim's functional boundaries—leaving it to the pharmaceutical industry to complete an unfinished invention." *Ariad*, 598 F.3d at 1353. In the Application before us, claim 17 recites a number of diseases for treatment by an antibody that specifically binds a human or mouse dendritic cell immunoreceptor and is agonistic thereof, but the Specification does not provide a structural description of even a single specific antibody that performs that disease-treating function, much less a representative number of species falling within the scope of the genus, or structural features common to the members of the genus so that one of skill in the art could

visualize or recognize *a priori* (i.e., without carrying out experimentation to determine whether the antibody in hand functioned as claimed) antibodies within the genus. Accordingly, we agree with the Examiner that the instant disclosure is insufficient to satisfy the written description requirement.

II

Obviousness

According to the Examiner, the claims "broadly encompass any DCIR agonist antibody that functions to treat rheumatoid arthritis [RA]." (Non-Final Action 8.) The Examiner explains that internalization is a type of agonist activity for DCIR because it induces endocytosis of bound ligands. (*Id.* (citing Meyer-Wentrup ("MW")).) The Examiner further notes that according to the Specification "DCIR agonist antibodies . . . also function to inhibit TNF-alpha production (see paragraphs 67 and 20–23, in particular)." (*Id.*)

With this understanding of the claims, the Examiner finds that Iwakura teaches that it was known that DCIR (also known as LLIR) can serve as an inhibitory receptor to regulate dendritic cell function, and that lack of DCIR is linked to exacerbation of RA. (Non-Final Action 5 (citing Iwakura 1–2, 4, and 9).) The Examiner further notes that Iwakura teaches that "a substance that activates DCIR (i.e., agonists) can be used as a remedy for autoimmune arthritis." (*Id.* at 5–6.)

The Examiner further states that Iwakura teaches that "DCIR plays a role in regulation of differentiation of osteoclast cells and that a lack of DCIR protein activity is linked to osteoporosis and bone loss." (*Id.* at 6.) Additionally, according to the Examiner, Iwakura teaches that DCIR signaling negatively controls cytokine signal transduction through GM-CSF

and that "GM-CSF may exacerbate arthritis by stimulating proliferation of dendritic cells." (*Id.*)

The Examiner recognizes that Iwakura does not teach DCIR agonist antibodies but finds that such antibodies were known in the art and that the use of such antibodies in the method of treatment of RA taught by Iwakura would have been obvious in light of MW and Valladeau. (*Id.* at 6–7.) The Examiner explains that MW teaches agonist antibodies (monoclonal antibody clone 216110) that trigger DCIR which induce receptor internalization and inhibit the production of inflammatory cytokines such as TNF-alpha by dendritic cells. (*Id.* at 6.) The Examiner notes that MW teaches that this activity "points to an important role for DCIR in limiting DC mediated immune activation, and that manipulation of DCIR could be of use [to] treat immune mediated diseases." (*Id.*)

The Examiner finds that Valladeau teaches producing antibodies to mouse Clec4a2. (*Id.*) The Examiner further notes that Valladeau teaches administering the agonist antibodies "to a subject for modulating antigen presentation and immunological responses." (*Id.*)

As with the written description rejection discussed above, Appellant does not separately address the claims. However, we find that separate issues are presented by the various independent claims. Claim 17 for example, in contrast to claim 38, only requires that the antibody administered to treat RA, a disease accompanied by abnormal bone metabolism, specifically binds to human or mouse dendritic immunoreceptor and acts as an agonist thereof. Claim 38, on the other hand, requires a method of suppressing osteoclast production where the antibody

administered not only binds to a human or mouse dendritic immunoreceptor and acts as an agonist thereof, but also suppresses osteoclast production.[8]

Appellant argues that Iwakura "only suggest[s] that DCIR relates to bone metabolism" and that it would not have been expected from that publication "that the agonistic anti-DCIR antibodies can inhibit osteoclast differentiation directly," i.e., "in a TNF-independent manner." (Appeal Br. 14–15.) We do not find this argument persuasive as to claim 17 since that claim does not require inhibition of osteoclast differentiation, much less that such inhibition be in a TNF-independent manner. Claim 17 requires that the disease to be treated by an antibody or Fab fragment thereof that specifically binds to human or mouse LLIR and is an agonist thereof is one that is accompanied by abnormal bone metabolism, and that the disease is treated with the antibody or fragment thereof. The claim includes RA as one such disease. There is no stated limitation regarding osteoclasts in this claim. Thus, it is irrelevant to claim 17 and the Examiner's rejection thereof whether Iwakura teaches that the agonist antibody inhibits osteoclast differentiation.

Nevertheless, we find that the Examiner has not established a prima facie case of obviousness of claim 17 because we conclude the cited prior art would not have provided one of ordinary skill in the art with a reasonable expectation of treating RA with an agonist antibody that specifically binds to human or mouse dendritic immunoreceptor. "[T]he examiner bears the

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[8] Independent Claim 37 is similarly directed to a method of treating a disease such as RA and does not require the administered antibody suppress osteoclast production. Independent claim 19 is similar to claim 38 in requiring suppression of osteoclast production by the administered antibody.

initial burden, on review of the prior art . . . , of presenting a *prima facie* case of unpatentability." *In re Oetiker*, 977 F.2d 1443, 1445 (Fed. Cir. 1992).

While Iwakura teaches that "DCIR is involved in development or aggravation of autoimmune diseases" (Iwakura ¶ 66), it does not disclose, contrary to the Examiner's position (Non-Final Action 5–6), that any DCIR agonist can be used as a remedy for autoimmune arthritis. Rather, Iwakura discloses that one

> can screen a substance that facilitates activity of DCIR protein, that is, a preventive/remedy for autoimmune diseases . . . by co-culturing the peptide [having an amino acid sequence identical or substantially identical to DCIR protein], the partial peptide or the salt thereof with a candidate substance, and comparing the activity of DCIR protein under presence or absence of the candidate substance.

(Iwakura ¶ 64; *see also id.* ¶ 59.) Iwakura also states, in conjunction with the fact that "[t]he inventors clarified for the first time that DCIR is involved in development or aggravation of autoimmune diseases," and knowing that "DCIR is one of inhibitory regulators in immune system, playing a role to suppress immune response under physiological condition," that:

> Therefore, by administering a peptide comprising an amino acid sequence identical or substantially identical to DCIR protein (soluble extracellular protein moiety, in particular), or a partial peptide or salt thereof, inhibitory signals in immune system may be inhibited, activating immune system. Therefore, the peptide, the partial peptide or salt thereof can be used as a remedy for various infections caused by various viruses, bacteria, fungi or protozoa.

(*Id.* ¶ 66.) The ultimate goal of Iwakura might be stated as:

> The purposes of the invention are to provide a DCIR defective (KO) mouse (DCIR-/- mouse) to elucidate the role of DCIR in

> development of autoimmune diseases including arthritis, to demonstrate the usefulness of the DCIR-/- mouse as a disease model animal, to provide a method to screen a preventive/remedy for autoimmune diseases using the DCIR-/- mouse, and to provide a preventive/remedy for the autoimmune diseases using the screening method.

(*Id.* ¶ 9.) Nonetheless, there is no disclosure of using the DCIR defective mouse model to "develop [an] effective method and drug for treatment and diagnosis" of bone and cartilage disease such as RA (*id.* ¶ 15). Iwakura discloses at most a method for screening for substances that are agonists that may in turn be tested for the ability to prevent or remedy autoimmune diseases (*id.* ¶ 59), though it does not disclose a method for testing for the ability to treat autoimmune disease.

In light of the foregoing, we disagree with the Examiner that Iwakura would have motivated one of ordinary skill in the art with a reasonable expectation of success in choosing agonist antibodies disclosed in MW or Valladeau as therapeutics for autoimmune arthritis, such as RA.

As to claim 38, contrary to the Examiner's position (Non-Final Action 6), Iwakura does not teach that DCIR definitively plays a role in regulation of differentiation of osteoclast cells. Instead, based on observations made in male DCIR-/- mouse of the heel joint showing increased calcification compared to wild type mouse and showing that cartilage was replaced with bone as well as observing that femur bone mass was decreased, it was posited that "DCIR *may* play a role in bone metabolism, regulation of differentiation and proliferation of cartilage cells, osteoblast and osteoclast cells." (Iwakura ¶ 130.) Based on this observation, Iwakura states that the "DCIR-/- mouse can serve as a good model mouse of osteoporosis." (*Id.*) In short, Iwakura indicates that

> since the DCIR protein defective animal develops autoimmune diseases, a gene coding DCIR protein or a partial sequence thereof is effective as a diagnostic agent of autoimmune diseases or osteoporosis. The invention provides a diagnostic agent of autoimmune diseases or osteoporosis[.]

(*Id.* ¶ 63.) While it is true that Iwakura teaches that lack of DCIR protein activity where DCIR is defective is linked to osteoporosis, we agree with Appellant that at best Iwakura suggests that DCIR relates to bone metabolism (Appeal Br. 14). We conclude that Iwakura does not provide a reasonable expectation of success that an agonist of DCIR protein, much less an agonist antibody, will suppress osteoclast production simply because it was observed that DCIR may play a role in regulation of differentiation and proliferation of cartilage cells, osteoblast and osteoclast cells. Thus, we disagree with the Examiner that Iwakura would have motivated one of ordinary skill in the art with a reasonable expectation of success in choosing agonist antibodies disclosed in MW or Valladeau as agents for suppressing osteoclast production in patients in need thereof.

In view of the foregoing, we reverse the Examiner's rejection of claims 17, 19, 21–23, 26–28, 37, and 38 as being obvious.

Moreover, because the Examiner's rejection for obviousness of claims 24 and 29 does not remedy the deficiencies noted above, we also reverse the Examiner's obviousness rejection of those claims.

SUMMARY

In summary:

Claims Rejected	Basis	Affirmed	Reversed
17, 19, 21–24, 26–29, 37, and 38	§ 112, first paragraph (written description)	17, 19, 21–24, 26–29, 37, and 38	
17, 19, 21–23, 26–28, 37, and 38	§ 103 Iwakura, Valladeau, Meyer-Wentrup, Vasanthi		17, 19, 21–23, 26–28, 37, and 38
24 and 29	§ 103 Iwakura, Valladeau, Meyer-Wentrup, Vasanthi, and Chapman		24 and 29
Overall Outcome		17, 19, 21–24, 26–29, 37, and 38	

TIME PERIOD FOR RESPONSE

No time period for taking any subsequent action in connection with this appeal may be extended under 37 C.F.R. § 1.136(a).

AFFIRMED