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Datasheet for the decision of 6 April 2022

Case Number: T 0499/18 - 3.3.04

Application Number: 08779934.2

Publication Number: 2173379

IPC: A61K39/395

Language of the proceedings: EN

Title of invention:
Compositions and methods for treating and diagnosing cancer

Patent Proprietor:
Oncomed Pharmaceuticals, Inc.

Opponent:
Strawman Limited

Headword:
Compositions and methods for treating cancer/ ONCOMED

Relevant legal provisions:
EPC Art. 83, 111(1)
EPC R. 103(4)(c)

Keyword:
Main request - sufficiency of disclosure (yes);
remittal of the case to the opposition division (yes)

Decisions cited:

Catchword:

Europäisches Patentamt
European Patent Office
Office européen des brevets

Beschwerdekammern

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Case Number: T 0499/18 - 3.3.04

D E C I S I O N
of Technical Board of Appeal 3.3.04
of 6 April 2022

Appellant: (Patent Proprietor)	Oncomed Pharmaceuticals, Inc. 800 Chesapeake Drive Redwood City, CA 94063-4748 (US)
Representative:	Miller, David James Mathys & Squire The Shard 32 London Bridge Street London SE1 9SG (GB)
Respondent: (Opponent)	Strawman Limited Orchard Lea Horns Lane Combe, Witney Oxfordshire OX29 8NH (GB)
Representative:	Grünecker Patent- und Rechtsanwälte PartG mbB Leopoldstraße 4 80802 München (DE)
Decision under appeal:	**Decision of the Opposition Division of the European Patent Office posted on 6 December 2017 revoking European patent No. 2173379 pursuant to Article 101(3)(b) EPC.**

Composition of the Board:

Chair	G. Alt
Members:	R. Morawetz
	P. de Heij

Summary of Facts and Submissions

I. The appeal of the patent proprietor (appellant) lies from the decision of the opposition division revoking European patent No. 2 173 379 ("the patent").

II. The patent, entitled "*Compositions and methods for treating and diagnosing cancer*", was granted for European patent application No. 08 779 934.2 with a filing date of 2 July 2008. It claims priority from US patent application No. 947611, filed on 2 July 2007.

Claims 1, 5 and 16 as granted read as follows:

"1. An isolated monoclonal antibody that specifically binds to a human R-spondin (RSPO) protein and inhibits growth of a solid tumor comprising solid tumor stem cells, wherein the antibody
disrupts binding of the RSPO protein to a leucine-rich repeat-containing G protein-coupled receptor (LGR) protein, wherein the LGR protein is LGR5; and/or
disrupts RSPO activation of LGR5 signaling.

5. An isolated monoclonal antibody that specifically binds to an extracellular domain of a human leucine-rich repeat-containing G protein-coupled receptor (LGR) protein, wherein the LGR protein is LGR5, and inhibits growth of a solid tumor comprising solid tumor stem cells, wherein the antibody
disrupts binding of RSPO to LGR5; and/or
disrupts RSPO activation of LGR5 signaling.

16. An agent for use in treating a cancer comprising cancer stem cells in a human or inhibiting growth of a tumor in a human, the method comprising administering a

therapeutically effective amount of an agent that
(a) disrupts the binding of a human R-spondin (RSPO) protein and a human leucine-rich repeat-containing G protein-coupled receptor (LGR), wherein the LGR protein is LGR5; and/or
(b) disrupts RSPO activation of LGR5 signalling,
wherein the agent is an antibody according to any one of claims 1 to 8 or a soluble receptor according to any one of claims 12 to 15."

III. The following documents are referred to in this decision:

D14 De Lau W. et al. (2011), Nature, vol. 476, pp. 293-298

D15 Supplementary information linked to the online version of document D14 at www.nature.com/nature

D25 WO 2005/040828 (2005)

D31 De Lau W. et al. (2014), Genes & Development, vol. 28, pp. 305-316

D43 Atwood B.K. et al. (2011), BMC Genomics, vol. 12, pp. 1-14

D44 Extract from Human Protein Atlas database regarding LGR5 (https://www.proteinatlas.org/ENSG00000139292-LGR5/cell)

D45 Alizadeh-Navaei R. et al. (2016), Biomedical Reports, vol. 5, pp. 130-132

D46 Glinka A. et al. (2011), EMBO reports, vol. 12, pp. 1055-1061 including supplementary information

D49 Declaration by A. Gurney, 2 December 2018, pp. 1-5

D60 Kendrick N. (25 September 2014), Kendrick Labs, Inc, pp. 1-8

IV. In this decision, document D14 (the article) and accompanying document D15 (the supplementary information) are referred to as document D14/D15.

V. One opposition was filed against the patent in its entirety. The patent was opposed under Article 100(a) EPC on the grounds of lack of novelty (Article 54 EPC) and lack of inventive step (Article 56 EPC) and under Article 100(b) and (c) EPC.

VI. The decision under appeal dealt with sets of claims of a main request (patent as granted) and auxiliary requests 1 to 13. Based on the evidence before it, the opposition division considered that HEK293 cells did not express LGR5. It concluded that the application's experiments could not show an interaction between RSPO and LGR5 and that therefore it was not plausible that the claimed antibodies would inhibit beta-catenin signalling and tumour growth. The invention claimed in claims 1 to 11 and 16 to 19 of the main request and in the set of claims of, *inter alia*, auxiliary request 6 was held to fail the requirements of Article 83 EPC.

VII. With the statement setting out the grounds of appeal, the appellant filed sets of claims of a main request and auxiliary requests 1 to 54 and, *inter alia*,

documents D43, D44, D45, D46 and D49. It submitted arguments to the effect that the claimed invention was sufficiently disclosed.

VIII. The main request is identical to the set of claims of auxiliary request 6 considered in the decision under appeal. Claims 1 and 5 of the main request are identical to claims 1 and 5 as granted (see section II), while claim 12 is based on claim 16 as granted and reads as follows (amendments are indicated):

"12. An agent for use in treating a cancer comprising cancer stem cells in a human or inhibiting growth of a tumor in a human, the method comprising administering a therapeutically effective amount of an agent that
(a) disrupts the binding of a human R-spondin (RSPO) protein and a human leucine-rich repeat-containing G protein-coupled receptor (LGR), wherein the LGR protein is LGR5; and/or
(b) disrupts RSPO activation of LGR5 signalling,
wherein the agent is an antibody according to any one of claims 1 to 8~~ or a soluble receptor according to any one of claims 12 to 15~~."

IX. With the reply to the statement setting out the grounds of appeal, the opponent (respondent) filed, *inter alia*, document D60 and made submissions concerning sufficiency of disclosure.

X. The board summoned the parties to oral proceedings, as requested by the parties, and issued a communication under Article 15(1) RPBA 2007 in which it indicated its preliminary opinion on sufficiency of disclosure of the invention claimed in the main request. The board informed the parties that should the appeal be found

allowable, the board intended to remit the case to the opposition division for further prosecution.

XI. In reply, the appellant withdrew its request for oral proceedings within one month of notification of the communication issued by the board in preparation for the oral proceedings. The respondent likewise withdrew its request for oral proceedings.

XII. Thus, the board cancelled the oral proceedings.

XIII. The appellant's arguments, in so far as relevant to the present decision, are summarised below.

Main request
Sufficiency of disclosure (Article 83 EPC)

The results in Example 2 of the application made it plausible to a skilled person that RSPO1 and LGR5 interacted with each other to activate beta-catenin signalling and conversely that beta-catenin activity could be inhibited by disrupting the RSPO/LGR5 interaction. It was common general knowledge that the Wnt signalling pathway was implicated in cancer. From this, it was credible that agents that disrupted the binding between RSPO1 and LGR5 would inhibit the ability of RSPO1 to increase beta-catenin activity and, accordingly, tumour growth.

The impugned decision relied on post-published document D14/D15 as the sole evidence in reaching the conclusion that the HEK293 cells used in the experiments of the application did not express LGR5 and that, as a result, these experiments did not show that RSPO and LGR5 interacted.

Firstly, document D14/D15 had to be disregarded since it was post-published.

Secondly, none of the experiments in document D14/D15 conclusively proved that LGR5 was not present in all HEK293 cells. The findings in document D14/D15 with respect to LGR5 mRNA expression were contradicted by document D25, which showed that LGR5 mRNA was present in HEK293 cells. The discrepancy could have been caused by experimental problems in document D14/D15. Furthermore, it was known that differences in expression levels appeared in cell lines with time, passage number and growth conditions as also noted in document D43 (see page 11, left-hand column, lines 24 to 28). As a result, the Northern blot results of document D14/D15 could not be interpreted to mean that all HEK293 cells did not express LGR5.

Document D25 disclosed that GPR49 (i.e. LGR5) mRNA was highly expressed in HEK293 cells (see Table 1 on page 83; page 54, lines 10 to 11 and page 56, lines 8 to 9). The opposition division incorrectly dismissed document D25. The reasonable scientific conclusion to be drawn from document D25 was that HEK293 cells shown to express high levels of LGR5 mRNA also expressed the LGR5 protein (see also document D49, paragraph 7).

Documents D43 and D44 demonstrated that LGR5 mRNA was present in HEK293 cells. Document D45 demonstrated that LGR5 protein was present in HEK293 cells, and document D46 demonstrated that functional LGR5 protein was present in HEK293T cells.

No convincing evidence had been provided to doubt that an anti-RSPO or anti-LGR5 antibody as claimed could be generated by the skilled person or that these anti-RSPO

and anti-LGR5 antibodies could be used to inhibit solid tumour growth.

Remittal

No arguments were provided why the case should be further decided by the board and not remitted to the opposition division if it was considered that the requirements of Article 83 EPC were met.

XIV. The respondent's arguments, in so far as relevant to the present decision, are summarised below.

Main request
Sufficiency of disclosure (Article 83 EPC)

Post-published evidence such as document D14/D15 could be considered when evaluating whether the skilled person was able to re-work the experiments shown in the application.

Where common general knowledge, as proven by document D14/D15, suggested failure of the experiments, there was an undue burden to do research and establish whether the teaching of the application could be put into practice. Document D14/D15 was sufficient to find that the experimental data presented in the application were based on an erroneous premise.

Document D25 only demonstrated that LGR5 mRNA was present. There was no mention in document D25 that LGR5 protein was expressed on the cell surface. This fitted with the finding in document D14/D15. There was thus no discrepancy between the finding in document D14/D15 and document D25.

Document D60, a review article from 2014, analysed work from before 2011 and stated that protein expression levels could not be extrapolated from mRNA levels. Moreover, the existence of mRNA encoding a protein did not provide any evidence of cell surface expression of the protein instead of soluble protein.

Documents D43 and D44 disclosed that LGR5 mRNA was present in HEK293 cells but provided no information on LGR5 protein expression. Document D45, which detected LGR5 protein in HEK293 cells, and document D46, which provided knock-down data of LGR5 in HEK293T cells, did not show that HEK293 cells expressed LGR5 protein on the cell surface.

The mechanism of beta-catenin signalling provided in the application was incorrect and had to be replaced with a more complicated mechanism, the canonical Wnt signal cascade (see post-published document D31).

The opposition division had been correct to conclude that if HEK293 cells did not express LGR5, it was not plausible that the claimed anti-LGR5 antibodies were able to disrupt the binding of RSPO to LGR5 and inhibit beta-catenin signalling and tumour growth. Therefore, the opposition division had correctly decided that the application did not meet the requirements of sufficiency of disclosure.

Remittal

The case should be remitted to the opposition division for further prosecution because it would be unfair for the respondent to have inventive step decided by just one instance.

XV. The appellant requested, as far as relevant to the present decision, that the decision under appeal be set aside and that the patent be maintained in amended form on the basis of the set of claims of the main request or, alternatively, that the patent be maintained in amended form on the basis of one of the set of claims of auxiliary requests 1 to 54, in each case with a description amended as appropriate. If any request on file is considered to satisfy the requirements of Article 83 EPC, it requested that the board consider and decide on the Articles 54 and 56 EPC objections without remitting the case to the opposition division. Lastly, it requested that the appeal fee be refunded in accordance with Rule 103(4)(c) EPC.

The respondent requested that the appeal be dismissed, that the case be remitted to the opposition division if the board sets aside the decision under appeal regarding lack of sufficiency of disclosure and that auxiliary requests 9 to 54 be not admitted into the appeal proceedings.

Reasons for the Decision

1. The appeal complies with Articles 106 to 108 and Rule 99 EPC and is admissible.

2. Following the parties' withdrawals of their respective requests, the oral proceedings were cancelled. The present decision is issued in writing on the basis of the requests, grounds and evidence on file and taking into account the board's preliminary opinion (Article 113 EPC and Article 12(8) RPBA).

3. The following abbreviations are used in the decision: human R-spondin protein (RSPO protein); leucine-rich

repeat-containing G protein coupled receptor protein (LGR protein); human embryonic kidney 293 cells (HEK293 cells); Wingless-related integration site (Wnt) and fragment crystallisable (Fc).

Main request

Sufficiency of disclosure (Article 83 EPC)

4. Article 83 EPC requires that the application disclose the invention in a manner sufficiently clear and complete for it to be carried out by a person skilled in the art. It is established case law of the boards that the claimed invention must be sufficiently disclosed at the effective date of the application (priority date or date of filing) based on the application as a whole including examples and taking into account the common general knowledge of the skilled person (see Case Law of the Boards of Appeal of the European Patent Office, 9th edition 2019 ("CLBA"), section II.C.1).

The claimed invention, the teaching in the application and the common general knowledge

5. The claimed invention relates to anti-RSPO and anti-LGR5 antibodies that disrupt binding of RSPO to LGR5 and the use of such antibodies in treating cancer.

6. The application discloses that LGR5 is over-expressed in a large number of tumours compared to corresponding normal tissue (see Example 1) and that HEK293 cells stably transfected with a luciferase reporter under the control of a beta-catenin responsive promoter show greater luciferase activity in response to increasing concentrations of RSPO (see Figure 4). Furthermore,

RSPO1-induced activation of luciferase activity in these reporter cells is shown to be inhibited by soluble LGR5 (see Example 2 and Figure 5).

7. The conclusions drawn in the application from these experimental results and taking into account common general knowledge as regards Wnt signalling (see e.g. paragraphs [0044] and [0045] of the application) can be summarised as follows: the two proteins RSPO1 and LGR5 interact, and this interaction results in the activation of beta-catenin signalling suggesting that functional blocking of an interaction between RSPO and LGR5, e.g. with antibodies, can inhibit tumour growth (see e.g. paragraphs [0020], [0021], [0039], [0046] and [0047] of the application).

The impugned decision

8. The opposition division did not question the experimental results summarised in point 6 above nor that an antibody which inhibits beta-catenin signalling would inhibit tumour growth. However, the opposition division accepted that document D14/D15 provided evidence that HEK293 cells do not express LGR5, as a matter of fact, while document D25, which discloses a high expression of LGR5 mRNA in HEK293 cells, was given less weight. It concluded that LGR5 could not be the receptor on the HEK293 cells interacting with RSPO in the experiments of Example 2 of the application and that the conclusions drawn in the application from Example 2 (summarised in point 7 above) could be incorrect. Accordingly, it was not plausible that the claimed antibodies would be able to disrupt binding of RSPO to LGR5 and inhibit beta-catenin signalling and tumour growth, and the requirements of Article 83 EPC

were not met.

Serious doubts substantiated by verifiable facts

9. Whether the application discloses the claimed invention in a manner sufficiently clear and complete for it to be carried out by a person skilled in the art is a question of fact that must be answered on the basis of the available evidence and on the balance of probabilities in each individual case.
In accordance with established case law of the boards, a finding of lack of sufficiency of disclosure of the claimed invention presupposes serious doubts substantiated by verifiable facts (see CLBA, sections II.C.5.3., II.C.7.1.4 and II.C.9).

10. The case at hand turns on whether it can be accepted, as a matter of fact, that all HEK293 cells do not express a functional LGR5 protein.

11. Given that the primary object of the appeal proceedings is to review the decision under appeal in a judicial manner (Article 12(2) RPBA), the board will first assess the evidence relied on in the impugned decision, i.e. document D14/D15 and document D25.

12. In a first line of defence, the appellant disputed that document D14/D15 can be relied upon at all in the assessment of sufficiency of disclosure as it is post-published.

13. In accordance with established case law of the boards, a document which is not part of the state of the art but which is submitted in substantiation of an allegation of fact does not stand or fall by its publication date (see CLBA, section III.G.4.1). The

board therefore does not concur with the appellant that, as a matter of principle, post-published evidence cannot be relied upon for the substantiation of allegations of verifiable facts that support serious doubts. The appellant's first line of defence is thus not found persuasive.

14. In a further line of argument, the appellant submitted that it could not be inferred from document D14/D15 that all HEK293 cells do not express a functional LGR5 protein.

15. Document D14/D15 reports that expression of LGR5 mRNA and protein was not detected in HEK293T cells while expression of LGR4 mRNA and protein was detected (see document D14, page 296, left-hand column, second paragraph; and Supplemental Figure 9 and Supplemental Table 2 of document D15).

16. Document D14/D15 is a scientific article on LGR5 homologues and their role in the canonical Wnt pathway, and HEK293T cells are used as a tool to search for the cognate R-spondin receptor. Document D14/D15 does not summarise the state of the art with respect to LGR5 expression in HEK293 cells, nor does it report that LGR5 has never been detected in HEK293 cells. Contrary to the submissions of the respondent, document D14/D15 therefore does not qualify as an account of the common general knowledge in the art as regards LGR5 expression in HEK293 cells. Instead, it presents an isolated finding.

17. The finding in document D14/D15 with respect to LGR5 mRNA expression in HEK293T cells is contradicted by document D25, which reports that GPR49 (i.e. LGR5; see page 3, line 19) mRNA is in fact highly expressed in

HEK293 cells (see Table 1 on page 83; page 54, lines 10 to 11 and page 56, lines 8 to 9).

18. For the issue at stake in this decision, it is of no relevance whether the likely cause of the discrepancy is i) an experimental error in document D14/D15; ii) to be attributed to the fact that differences in mRNA expression levels are known to appear in cell lines with time, passage number and growth conditions (see also document D43, page 11, left-hand column, lines 24 to 28); or iii) due to the fact that not HEK293 cells, but HEK293T cells, a variant cell line, are used in document D14/D15. The upshot in any case is that document D14/D15 cannot, in the board's view, be accepted to provide evidence that *all* HEK293 cells do not express LGR5 mRNA.

19. The next question that arises is whether the finding in document D25 that HEK293 cells do express LGR5 mRNA allows any conclusion with respect to the expression of a functional LGR5 protein in HEK293 cells. In this context, it is of importance that the skilled person is aware that proteins are synthesised using the information in mRNA as a template.

20. As for the presence of a functional LGR5 protein in HEK293 cells, the opposition division held that "*D25 did not investigate the LGR4 expression in parallel, while D14/D15 could show in a side-by-side Northern blot that the LGR4 mRNA is expressed, while LGR5 transcripts could not be detected. The OD gives much higher weight to the data in D14/D15 which show the LGR4 protein expression in said cells, while failing to detect LGR5 protein expression*".

21. The board does not find the opposition division's justification for giving a "*much higher weight*" to the data in document D14/D15 over the data in document D25 persuasive. Firstly, for the issue at stake, i.e. the expression of functional LGR5 protein in HEK293 cells, it is irrelevant that document D25, unlike document D14/D15, did not investigate LGR4 expression in parallel. Secondly, that document D14/D15 failed to detect LGR5 protein expression in HEK293T cells while detecting LGR4 protein expression is merely in line with the findings in this document on mRNA expression of LGR4 and LGR5 in HEK293T cells (see point 15 above). However, none of these findings justifies that the negative data for LGR5 mRNA and protein expression in document D14/D15 be given more weight than the positive data for LGR5 mRNA expression in document D25.

22. The respondent's assertion that there is no discrepancy between the finding in document D14/D15 and document D25 because document D25 does not mention LGR5 protein expression cannot hold either. Document D25 did not look for and fail to detect LGR5 protein expression; it is merely silent about the expression of the LGR5 protein. However, as set out in point 17 above, document D25 reports a high expression of LGR5 mRNA in HEK293 cells, and the board agrees with the appellant that the reasonable conclusion that would be drawn by the skilled person is that LGR5 protein is also likely present in these HEK293 cells.

23. Since the exact level of the LGR5 protein in HEK293 cells is not at issue, the respondent's assertion that protein levels could not be extrapolated from mRNA levels in document D25 is of no consequence. Furthermore, in the absence of any evidence that a soluble form of LGR5 exists, the respondent's concerns

with respect to a possible soluble form of the LGR5 protein in the HEK293 cells of document D25 are unfounded.

24. In view of the above considerations on the evidence on which the decision under appeal was based, the board is not persuaded that it has been established, on the balance of probabilities, that all HEK293 cells do not express functional LGR5 protein. Accordingly, no serious doubts exist as to whether the cells used in the experiments underlying Example 2 of the application express functional LGR5 protein. Therefore, the opposition division's reasoning, which was solely based on the assumption that HEK293 cells do not express LGR5 and that LGR5 could thus not be the receptor on HEK293 cells interacting with RSPO in the experiments of Example 2 (see point 8 above), cannot hold.

25. In view of the above findings, there is no need for the board to consider documents D43, D44, D45 and D46 filed by the appellant as further evidence for the expression of LGR5 on HEK293 cells.

26. The respondent's assertions that common general knowledge, as proven by document D14/D15, suggested failure of the experiments and that there was an undue burden to do research and establish whether the teaching of the application could be put into practice likewise fail (see also point 16 above).

27. The respondent's further objection of a lack of sufficiency of disclosure based on post-published document D31 is not found persuasive. The mere assertion that the canonical Wnt signal cascade turned out to be more complicated than the mechanism postulated in the application does not suffice to raise

serious doubts substantiated by verifiable facts that the claimed antibodies can be generated and used in treating cancer.

28. Although not relevant for the present decision, the board notes that document D31 actually confirms that LGR5 is a receptor for R-spondins and that the interaction between LGR5 and R-spondins ultimately leads to increased beta-catenin levels in the canonical Wnt signal cascade (see abstract, page 309, right-hand column, last paragraph; paragraph bridging pages 312 and 313; and Figure 5).

Conclusion on sufficiency of disclosure

29. The teaching of the application has been summarised in points 6 and 7 above. The board furthermore agrees with the appellant that the results in the experiments of Example 2 allow the conclusion that both the soluble LGR5-Fc and the cell-surface receptor on HEK293 cells compete for the same binding site on RSPO suggesting that the receptor on the HEK293 cells used in these experiments is in fact the LGR5 protein. For these reasons, the application renders it plausible to a skilled person that RSPO1 and LGR5 interact with each other to activate beta-catenin signalling and, conversely, that beta-catenin activity can be inhibited by disrupting the RSPO/LGR5 interaction.

30. Upon Wnt signalling, levels of free cytoplasmic beta-catenin increase resulting ultimately in the transcription of Wnt-responsive genes (the Wnt signalling pathway). The Wnt signalling pathway has long been implicated in cancer due to the presence of mutations activating the pathway in certain tumours (see paragraph [0045] of the application). Taking into

account the common general knowledge of the skilled person with respect to Wnt signalling, it is furthermore credible that agents that disrupt the binding between RSPO1 and LGR5 would inhibit the ability of RSPO1 to increase beta-catenin activity and, accordingly, tumour growth. In the board's view, the disclosure in the application thus renders it plausible that the claimed antibodies can be generated and used in treating cancer.

31. Based on the evidence on file and the arguments presented to it, the board concludes that the invention claimed in the main request fulfils the requirements of Article 83 EPC. The appeal is allowable.

32. In view of the above findings with respect to the main request, there is no need for the board to consider any of auxiliary requests 1 to 54.

Remittal of the case (Article 111(1) EPC)

33. Pursuant to Article 111(1), second sentence, EPC, the board may either exercise any power within the competence of the department responsible for the decision appealed or remit the case to that department for further prosecution.

34. The sole ground for opposition considered in the decision under appeal with respect to claims corresponding to the current set of claims is the ground in Article 100(b) EPC. The board has reviewed the decision on this ground (see points 9 to 31 above). The decision under appeal did not consider the grounds in Article 100(a) as to lack of novelty (Article 54 EPC) raised by the opponent with respect to the subject-matter of claim 5 and lack of

inventive step (Article 56 EPC) raised with respect to the subject-matter of claims 1, 5 and 16 as granted, respectively.

35. As set out in Article 12(2) RPBA, the primary object of the appeal proceedings is to review the decision under appeal in a judicial manner. This principle would not be observed if the board were to give a first decision with respect to the grounds for opposition not considered in the decision under appeal.

36. Thus, special reasons are present to remit the case to the opposition division for further prosecution (Article 11 RPBA), and the board accedes to the respondent's request for a remittal.

Reimbursement of the appeal fee (Rule 103(4)(c) EPC)

37. The appellant had initially requested oral proceedings. This request was withdrawn within one month of notification of the communication issued by the board in preparation for the oral proceedings, and no oral proceedings took place (see sections X, XI and XII above). Accordingly, the appellant is entitled to the requested reimbursement of the appeal fee in accordance with Rule 103(4)(c) EPC.

Order

For these reasons it is decided that:

1. The decision under appeal is set aside.

2. The case is remitted to the opposition division for further prosecution.

3. The appeal fee is reimbursed at 25%.

The Registrar: The Chair:

I. Aperribay G. Alt

Decision electronically authenticated