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Case Number: T 2258/15 - 3.3.04

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Title of invention:
Human monoclonal antibodies to protein tyrosine kinase 7 (PTK7) and their use

Patent Proprietor:
E. R. Squibb & Sons, L.L.C.

Opponent:
Albrecht von Menges

Headword:
PTK7 antibodies/SQUIBB

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EPC Art. 56

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Catchword:

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Case Number: T 2258/15 - 3.3.04

D E C I S I O N
of Technical Board of Appeal 3.3.04
of 16 January 2020

Appellant I: (Patent Proprietor)	E. R. Squibb & Sons, L.L.C. Route 206 & Province Line Road Princeton, NJ 08540 (US)
Representative:	J A Kemp LLP 14 South Square Gray's Inn London WC1R 5JJ (GB)
Appellant II: (Opponent)	Albrecht von Menges Am Krähenberg 24 22587 Hamburg (DE)
Representative:	Mewburn Ellis LLP Aurora Building Counterslip Bristol BS1 6BX (GB)
Decision under appeal:	**Interlocutory decision of the Opposition Division of the European Patent Office posted on 8 October 2015 concerning maintenance of the European Patent No. 1957539 in amended form.**

Composition of the Board:

Chair	R. Morawetz
Members:	A. Chakravarty
	R. Romandini

Summary of Facts and Submissions

I. Appeals against the interlocutory decision of the opposition division that European patent No. 1 957 539 in an amended form, based on auxiliary request 6, met the requirements of the EPC, were filed both by the patent proprietor (appellant I) and by the opponent (appellant II). The patent is based on European patent application No. 06 848 508.5 and has the title "*Human monoclonal antibodies to protein tyrosine kinase 7 (PTK7) and their use*".

II. The patent was opposed on the grounds of lack of novelty (Article 100(a) EPC and Article 54 EPC), lack of an inventive step (Article 100(a) EPC and Article 56 EPC), lack of sufficient disclosure (Article 100(b) EPC) and added subject-matter (Article 100(c) EPC and Article 123(2) EPC).

III. The opposition division considered a main and six auxiliary requests. Auxiliary request 6 was held to meet the requirements of the EPC.

IV. With their statement of grounds of appeal, appellant I submitted sets of claims of a main request and of auxiliary requests 1 to 12.

V. Claim 1 of the main request (claim 1 as granted) reads:

"1. An isolated human monoclonal antibody, or an antigen-binding portion thereof, wherein the antibody:
(a) specifically binds to human PTK7; and
(b) binds to a Wilms' tumor cell line having ATCC Acc No. CRL-1441 with an EC_{50} of 4.0 nM or less,

in an assay which comprises incubating 1 x 10^5 cells with the antibody at a starting concentration of 30µg/ml and serially diluting the antibody at a 1:10 dilution; and
(c) binds to the same epitope on human PTK7 as a reference antibody comprising:
(i) a heavy chain variable region comprising the amino acid sequence of SEQ ID NO:2 and a light chain variable region comprising the amino acid sequence of SEQ ID NO:7; or
(ii) a heavy chain variable region comprising the amino acid sequence of SEQ ID NO:3 and a light chain variable region comprising the amino acid sequence of SEQ ID NO:8; or
(iii) a heavy chain variable region comprising the amino acid sequence of SEQ ID NO:4 and a light chain variable region comprising the amino acid sequence of SEQ ID NO:10".

Claim 1 of auxiliary request 2 is identical to claim 1 of the main request.

Claim 1 of auxiliary requests 1 and 4 are identical to claim 1 of the main request, except that they include the feature "*and assessing binding to the cells by flow cytometry*" at the end of part (b).

Claim 1 of auxiliary request 3 is identical to claim 1 of the main request, except that part (c)(ii) of the main request is deleted, while part (iii) is renumbered (ii).

Claim 1 of auxiliary request 5 combines the amendments made in claim 1 of auxiliary request 1 and those made in claim 1 of auxiliary request 3.

Claim 1 of auxiliary requests 6 and 7 differ from claim 1 of the main request in that part (b) contains the additional final feature "*washing the cells, and detecting binding with a PE-labelled anti-human IgG antibody using flow cytometric analysis*".

Claim 1 of auxiliary request 8 combines the features of claim 1 of auxiliary requests 6 and 7 with claim 1 of auxiliary request 3.

Claim 1 of auxiliary request 9 reads:

"1. An immunoconjugate comprising a therapeutic agent, such as a cytotoxin or a radioactive isotope, linked to a human monoclonal antibody, or an antigen binding portion thereof, wherein the antibody:
(a) specifically binds to human PTK7; and
(b) binds to a Wilms' tumor cell line having ATCC Acc No. CRL-1441 with an EC_{50} of 4.0 nM or less, in an assay which comprises incubating 1 x 10^5 cells with the antibody at a starting concentration of 30µg/ml and serially diluting the antibody at a 1:10 dilution; and
(c) binds to the same epitope on human PTK7 as a reference antibody comprising:
(i) a heavy chain variable region comprising the amino acid sequence of SEQ ID NO:2 and a light chain variable region comprising the amino acid sequence of SEQ ID NO:7; or
(ii) a heavy chain variable region comprising the amino acid sequence of SEQ ID NO:3 and a light chain variable region comprising the amino acid sequence of SEQ ID NO 8; or
(iii) a heavy chain variable region comprising the amino acid sequence of SEQ ID NO:4 and a light chain

variable region comprising the amino acid sequence of SEQ ID NO:10.

Claim 1 of auxiliary request 10 reads:

"1. An isolated human monoclonal antibody, or antigen-binding portion thereof,
which comprises:
(a) a heavy chain variable region CDR1 comprising SEQ ID NO:12,
a heavy chain variable region CDR2 comprising SEQ ID NO:16,
a heavy chain variable region CDR3 comprising SEQ ID NO:20,
a light chain variable region CDR1 comprising SEQ ID NO:25,
a light chain variable region CDR2 comprising SEQ ID NO:31 and
a light chain variable region CDR3 comprising SEQ ID NO:37; or
(b) a heavy chain variable region CDR1 comprising SEQ ID NO:14,
a heavy chain variable region CDR2 comprising SEQ ID NO:18,
a heavy chain variable region CDR3 comprising SEQ ID NO:22,
a light chain variable region CDR1 comprising SEQ ID NO:28,
a light chain variable region CDR2 comprising SEQ ID NO:34 and
a light chain variable region CDR3 comprising SEQ ID NO:40; or
(c) a heavy chain variable region CDR1 comprising SEQ ID NO:13,
a heavy chain variable region CDR2 comprising SEQ ID NO: 17,

a heavy chain variable region CDR3 comprising SEQ ID NO:21,
a light chain variable region CDRl comprising SEQ ID NO:26,
a light chain variable region CDR2 comprising SEQ ID NO:32 and
a light chain variable region CDR3 comprising SEQ ID NO:38.

Claim 1 of auxiliary request 11 is the same as claim 1 of the main request except that part (b) includes the additional feature "*washing the cells and detecting binding with a PE-labelled anti-human IgG antibody; and performing flow cytometry analysis using a FACScan flow cytometer*".

Claim 1 of auxiliary request 12 (auxiliary request 6 before the opposition division) reads:

"1. An isolated human monoclonal antibody, or an antigen-binding portion thereof, wherein the antibody:

(a) specifically binds to human PTK7; and

(b) binds to a Wilms' tumor cell line having ATCC Acc No. CRL-1441 with an EC_{50} of 4.0 nM or less, in an assay which comprises incubating 1 x 10^5 cells with the antibody at a starting concentration of 30 µg/ml and serially diluting the antibody at a 1:10 dilution; washing the cells and detecting binding with a PE-labelled anti-human IgG antibody; and performing flow cytometry analysis using a FACScan flow cytometer; and
(c) binds to the same epitope on human PTK7 as a reference antibody comprising:

(i) a heavy chain variable region comprising the amino acid sequence of SEQ ID NO:2 and a light chain variable region comprising the amino acid sequence of SEQ ID NO:7; or
(ii) a heavy chain variable region comprising the amino acid sequence of SEQ ID NO:4 and a light chain variable region comprising the amino acid sequence of SEQ ID NO: 10".

VI. In their statement of grounds of appeal appellant II *inter alia* submitted arguments with respect to lack of inventive step of the subject-matter of the claims maintained by the opposition division (identical to present auxiliary request 12).

VII. In their reply to appellant II's statement of grounds of appeal, appellant I provided argumentation with respect to the inventive step of the subject-matter of auxiliary request 12.

VIII. The board issued a communication pursuant to Article 15(1) RPBA setting out its non-binding preliminary appreciation of substantive and legal matters concerning the appeals. In that communication the board *inter alia* informed the parties that "*In the board's preliminary opinion, the rabbit anti-PTK7 polyclonal antibodies disclosed in document D2 can be taken to represent the closest prior art for the claimed invention. The difference between these antibodies and the claimed ones lies in the type of antibody. In particular, the antibodies of claim 1 are monoclonal and human, whereas those disclosed in document D2 are polyclonal and rabbit. The technical effect of this difference is that the claimed antibodies are more suitable for treatment of human patients. The board is of the preliminary view that*

good internalisation properties cannot be considered a technical feature of the claimed antibodies essentially for the reasons set out by appellant II (see statement of grounds of appeal, sections 2.3.4 to 2.5). Thus the technical problem to be solved can be formulated as the provision of anti-PTK7 antibodies suitable for the treatment of human patients" (see point 11 of that communication).

IX. In response, appellant I informed the board that they would not attend the oral proceedings.

X. Oral proceedings before the board took place on 16 January 2020. At the end of these proceedings, the chair announced the decision of the board.

XI. The following documents are referred to in this decision.

D2: WO 2005/063987
D8: WO 02/100348
D9: WO 01/09187

XII. The relevant arguments of appellant I, submitted in writing, are summarised as follows:

Inventive step - Article 56 EPC
Auxiliary request 12 - claim 1

The claim was for antibodies with a surprisingly high level of binding affinity to the target antigen PTK7, which exhibited a surprisingly high level of internalisation into the cell bearing said target antigen.

The problem to be solved was the provision of anti-PTK7 antibodies having improved functional characteristics compared to those of the prior art.

Improved internalisation of the claimed antibodies was directly demonstrated in Example 8 of the patent. Antibodies that bind to the epitope group defined by feature (c) of claim 1 are found to internalise better than antibodies binding to a different epitope group. Specifically, monoclonal antibodies 4D5, 12C6 and 7C8 internalise into Wilms' tumour cells more effectively than the monoclonal antibody 3G8, which binds a different epitope, see the sentence beginning 7 lines from the bottom of page 83 of the application as filed. In fact, the reference antibodies of claim 1 were at least 2.5 fold more effective than monoclonal antibody 3G8 at internalisation into Wilms' tumour cells when measured in the same assay (EC_{50} of 0.6437nM for 3G8 versus 0.2516nM for 4D5). Example 8 furthermore demonstrated that at least antibodies 12C6 and 7C8 also internalise well into two other tumour cell lines (A-431 and PC3).

The skilled person would have understood from the application as filed that all of the claimed antibodies exhibited this good internalisation effect, which was clearly related to the technical problem initially suggested, being a desirable improvement.

Thus "*the opposition division were correct in their conclusion that the problem could be reformulated as the provision of anti-PTK7 antibodies with good internalisation properties. The internalisation properties demonstrated in the opposed patent must be taken into account when assessing inventive step*" (see

reply to appellant II's statement of grounds of appeal, page 6, first full paragraph).

The opposition division had also been correct to consider that it was plausible to the skilled person from Example 8 that the "epitope group" of the antibodies tested in the assay is responsible for the differences in their internalisation properties. "*The only demonstrated difference between the "good" internalising antibodies (4D5, 12C6 and 7C8) and the "bad" internalising antibody (3G8), is the difference in epitope group*" (see the reply to appellant II's statement of grounds of appeal, page 6, third full paragraph). All of the antibodies were raised using the same protocols (Examples 1 and 2) and, to the extent that they were compared, their other properties appeared to be similar, see for example Figure 15. Thus it was plausible that the difference in internalisation properties was linked to the difference in epitope group. No evidence had been submitted to suggest any alternative conclusion.

XIII. The relevant arguments of appellant II, submitted in writing and at the oral proceedings, are summarised as follows:

Inventive step - Article 56 EPC
Auxiliary request 12 - claim 1

Document D2 could be considered to represent the closest prior art. It disclosed PTK7 as well as anti-PTK7 antibodies (note that PTK7 was referred to as GP153 in document D2). Human anti-PTK7 antibodies and their use for the treatment of carcinoma were explicitly mentioned.

The difference between the claimed antibodies and those disclosed in document D2 was that the former were characterised by functional features (b) and (c) of the claim.

The objective technical problem in view of this prior art was the provision of anti-PTK7 antibodies suitable for human administration.

A detailed protocol on how to obtain monoclonal antibodies against the protein GP153 or fragments thereof was given on page 53, lines 3 to 12 of document D2.

A skilled person at the relevant date would have known how to raise the fully human monoclonal antibodies using HuMab mice as disclosed in documents D8 or D9.

Starting from the disclosure in document D2, the skilled person seeking a solution to the technical problem would have combined the disclosure in document D2 with that in document D8 or D9. Indeed, at the priority date of the patent in suit, a skilled person would have known that fully human antibodies could be generated against proteins of the protein tyrosine kinase receptor family and this conclusion was explicitly confirmed in document D8.

No particular technical effect was associated with the epitope bound by the reference antibodies and the antibodies of the patent were obtained as anti-PTK 7 antibodies without particular screening or selection steps.

The affinity of the antibodies disclosed in document D2 for their target was characterised as at least

1 x 10^{-6} M, preferably up to 1 x 10^{-10} M (page 32, lines 6 to 9). The preparation of rabbit polyclonal-anti-GP153 antibodies was illustrated in Example 10. The EC_{50} value was in the range of 2 x 10^{-3} to 2 x 10^{-4} for different anti-sera (page 52, Example 10). Thus, the binding affinity was not a surprising feature.

The opposition division in the decision under appeal had accepted that antibodies 4D5 and 7C8 (i.e. the reference antibodies specified in feature group (c) of claim 1) were internalised better than other anti-PTK7 antibodies and this had been the basis for acknowledging inventive step. However, the subject-matter of claim 1 was not characterised by an internalisation feature. Internalisation was also not an inherent feature of the claimed antibodies. There was no evidence in the patent that the internalisation of the anti-PTK7 antibodies was affected by the epitope bound. The term "internalisation" was not used in the opposed patent outside of Example 8, which did not show that internalisation was due to a particular epitope bound by the antibodies and did not even allow the conclusion that the antibodies 4D5 and 7C8 were internalised particularly well. The example merely concluded that the data obtained demonstrated that "*the anti-PTK7 antibodies 3GB, 4D5, 12C7 and 7C8 internalize into cancer cells*" (page 28, line 25).

Moreover, in view of the error bars in Figure 20A, Example 8 did allow the conclusion that antibody 4D5 was better internalised than antibody 3G8. The epitope bound by the antibody 4D5 had not identified in the patent.

In summary, no evidence had been presented that the antibodies claimed internalised efficiently or better

than expected. This property therefore could not be used to support inventive step of the subject matter of any claim. The claimed antibodies were an obvious solution to the technical problem which the skilled person starting from document D2 could have arrived at using well known techniques.

The main request and auxiliary requests 1 to 11 - claim 1

The arguments for claim 1 of auxiliary request 12 applied equally to all higher ranking claim requests, since its subject-matter was comprised in claim 1 of all said requests.

XIV. Appellant I requested in writing that the decision under appeal be set aside and that the patent be maintained as granted. Alternatively, the patent should be maintained in amended form on the basis of one of the sets of claims of auxiliary requests 1 to 12.

XV. Appellant II requested that the decision under appeal be set aside and that the patent be revoked.

Reasons for the Decision

1. The appeals comply with Articles 106 to 108 and Rule 99 EPC and are admissible.

2. Appellant I informed the board in writing that they would not be present or represented at the oral proceedings. Accordingly, the oral proceedings were held in their absence, pursuant to Rule 115(2) EPC and they are treated as relying on their written case (Article 15(3) RPBA).

Inventive step - Article 56 EPC

Auxiliary request 12 - claim 1

3. This claim request was auxiliary request 6 in the proceedings before the opposition division and was considered to meet the requirements of the EPC by the opposition division (see decision under appeal, point C).

4. The claimed subject-matter is a human monoclonal antibody defined by the following three functional features: (a) the target protein human PTK7, to which it specifically binds, (b) the ability to bind to a Wilms' tumour cell line and (c) the ability to bind to the same epitope as a reference antibody defined by two pairs of heavy and light chain variable sequences; either
(i) a heavy chain variable region comprising the amino acid sequence of SEQ ID NO:2 and a light chain variable region comprising the amino acid sequence of SEQ ID NO:7; or
(ii) a heavy chain variable region comprising the amino acid sequence of SEQ ID NO:4 and a light chain variable region comprising the amino acid sequence of SEQ ID NO: 10".

5. SEQ ID NO: 2 and SEQ ID NO: 7 represent the amino acid sequences of the heavy and light chain variable regions of exemplified monoclonal antibody 4D5, while SEQ ID NO: 4 and SEQ ID NO: 10 represent the amino acid sequences of the heavy and light chain variable regions of exemplified monoclonal antibody 7C8 (see paragraph [0146] of the patent).

6. According to the patent, the target of the claimed antibodies, protein tyrosine kinase 7 (PTK7), is a member of the receptor protein tyrosine kinase family. Its mRNA is variably expressed in colon carcinoma derived cell lines but not found to be expressed in human adult colon. By immunohistochemistry, tumour specific staining of PTK7 has been observed in breast, colon, lung, pancreatic, kidney and bladder cancers (see paragraphs [0002] and [0003] of the patent).

7. In the decision under appeal, document D2 was taken as representing the closest prior art for the claimed antibodies. This view was maintained by appellant II in the appeal and was not contested by appellant I.

8. Document D2 concerns the same protein as bound by the presently claimed antibodies, i.e. PTK7, which is also referred to therein as GP153. *Inter alia*, document D2 concerns the human PTK7 protein (see page 6, line 10ff). The document discloses antibodies specifically recognising this protein in general, including human monoclonal antibodies (see paragraph bridging pages 31 and 32). More concretely, Example 10 discloses the production of rabbit polyclonal antibodies capable of binding human PTK7. It is these rabbit polyclonal antibodies disclosed in document D2 that the board considers to represent the closest prior art. These antibodies - while not tested in the assay defined in the present claim - are polyclonal and therefore exhibit a range of affinities and bind to various epitopes on PTK7.

The technical problem

9. The claimed antibodies differ from those disclosed in Example 10 of document D2 in that they are human as

opposed to rabbit and that they are monoclonal as opposed to polyclonal. Moreover, they are defined by a certain binding to a Wilms' tumour cell line and also by their binding to the same epitope on human PTK7 as a reference antibody defined in claim 1(c).

10. A technical effect of these differences is that the claimed antibodies are less likely to be immunogenic in humans, by virtue of the fact that they are human antibodies and they are also well defined, in the sense of being monoclonal antibodies, making them more suitable for clinical use.

11. Appellant I argued that the claimed antibodies had further technical effects, these being improved functional characteristics, specifically the level of binding affinity and a surprisingly high level of internalisation. They stated that "*the opposition division were correct in their conclusion that the problem could be reformulated as the provision of anti-PTK7 antibodies with good internalisation properties. The internalisation properties demonstrated in the opposed patent must be taken into account when assessing inventive step.*"

12. The board notes, however, that "*good internalisation properties*" are not a feature mentioned in the claim. It must therefore be determined whether or not "*good internalisation properties*" are an inherent feature of the claimed antibodies, in particular whether they are a result of the epitope group bound by the antibodies, as alleged by appellant I.

13. Example 8 of the patent concerns "*Internalisation of anti-PTK7 monoclonal antibody*". It describes how the antibodies 3G8, 4D5, 12C6 and 7C8 were tested for

internalisation using a Hum-Zap internalisation assay. It is stated that "*the anti-PTK7 antibodies 3G8, **4D5**, 12C6 and **7C8** showed an antibody concentration dependent decrease in 3H-thymidine incorporation in the PTK7-expressing Wilms' Tumor cancer cell line*" (emphasis added to indicate monoclonal antibodies whose variable regions are comprised in the 'reference' antibodies mentioned in the claim). Example 8 concludes that "*This data demonstrates that the anti-PTK7 antibodies 3G8, **4D5**, 12C6 and **7C8** internalize into cancer cells*" (emphasis added). However, in Example 8 it is not concluded that internalisation is due to a particular epitope bound by these antibodies and no evidence has been put forward that it was recognised in the art that a causal link existed between the epitope bound by an antibody and the antibody's internalisation properties.

14. The patent also discloses that antibodies 7C8, 12C6 are in one epitope group, whereas antibody 3G8 is in a different epitope group (see example 4). The epitope bound by antibody 4D5 was not determined in the patent nor was this antibody assigned to the same epitope group as antibodies 7C8 and 12C6. Accordingly, appellant I's argument that it is plausible from Example 8 that the "epitope group" of the antibodies tested in the assay is responsible for the differences in their internalisation properties fails because this argument is based on the proposition that antibodies 7C8, 12C6 and 4D5 belong to the same antibody group.

15. Furthermore, the board cannot identify any disclosure in the patent that would allow the skilled person to conclude that the degree of internalisation is a property of the epitope group on PTK7 bound by the antibody. Thus, Example 8 does allow the conclusion

that antibodies 4D5 and 7C8 represent antibodies which internalise particularly well, while Figure 20A, relied on by appellant I to show that antibody 3G8 is "*bad*" at being internalised, in fact shows that the internalisation behaviours of antibodies 3G8 and 4D5 are similar enough that they fall within each other's respective error margins.

16. The board is thus not persuaded that the patent demonstrates that internalisation of PTK7 antibodies is a function of the epitopes bound nor that antibodies that bind to the epitope group defined by feature (c) of claim 1 internalise better than antibodies binding to a different epitope group.

17. In view of the above, in the board's judgement "*good internalisation properties*" are not a feature that can be taken into account in formulating the technical problem.

18. Finally, the range of affinities achievable by such antibodies is given as being "*at least 1 x 10^{-6} molar (M), preferably at least 5x10^{-7}M, more preferably at least 1x10^{-7}M, with affinities and avidity is of at least 5x10^{-8}M, 5x10^{-9}M, and 1x10^{-10}M being especially useful*" (see document D2, page 32, lines 7 to 9). This range of binding affinity corresponds to that specified in the claims, as can be seen from paragraph [0040] of the patent which states that "*an antibody of the invention binds to PTK7 with high affinity, for example with a K_D of 1 x 1x10^{-7}M or less*". The binding affinity is therefore not a feature that distinguishes the claimed subject-matter from the antibodies disclosed in document D2.

19. Considering the differences between the claimed antibodies and those disclosed in document D2 and the technical effect thereof, the problem to be solved by the claimed subject-matter can be formulated as the provision of anti-PTK7 antibodies suitable for the treatment of human patients.

Obviousness of the claimed solution

20. The question to be answered in assessing obviousness is whether the skilled person seeking to solve the above formulated technical problem and starting from the polyclonal rabbit antibodies disclosed in Example 10 of document D2 would have arrived at the claimed antibodies without inventive effort.

21. The production of monoclonal antibodies against PTK7 is directly suggested in document D2 on page 53, last sentence of the first full paragraph as follows: "*fusion products from the positive wells will be further subcloned and tested to generate monoclonal hybridoma lines*". Moreover, the general section on "*Antibodies and Antibody Producing Cells*" (see pages 31 and 32) discloses that "*an antibody can be […] a fully human antibody*", and also that "*monoclonal antibodies are generally preferred*". Processes for producing such human antibodies in mice were routine before the priority date of the patent and are described for example in document D9, where human monoclonal antibodies were produced which had an high affinity for their target antigen (see document D9, page 15, first full paragraph).

22. In view of the above, the board is of the view that the claimed antibodies are among those that the skilled person starting from the disclosure in document D2

would have arrived at as a matter of routine when faced with the technical problem. Thus, the subject-matter of claim 1 was obvious for the skilled person before the relevant date of the patent. Auxiliary request 12 does not meet the requirements of Article 56 EPC.

The main request and auxiliary requests 1 to 11 - claim 1

23. Since the finding of obviousness for the subject-matter of claim 1 of auxiliary request 12 was arrived at by considering the reference antibodies 4D5 and 7C8, and claim 1 of each of the main request and auxiliary requests 1 to 11 encompasses these reference antibodies (see section IV), the finding of obviousness extends to the subject-matter of all higher ranking claim requests. Thus, no claim request is allowable.

24. In view of the above, none of the claim requests forming part of the appeal proceedings meets the requirements of Article 56 EPC. Accordingly, the patent cannot be maintained on the basis of any of these requests and, in the absence of another, allowable claim request, the patent has to be revoked.

Order

For these reasons it is decided that:

The decision under appeal is set aside.

The patent is revoked.

The Registrar: The Chair:

I. Aperribay R. Morawetz

Decision electronically authenticated