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Case Number: T 0951/21 - 3.3.08

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Title of invention:
Angiopoietin-like 3 (ANGPTL3) iRNA compositions and methods of use thereof

Patent Proprietor:
Alnylam Pharmaceuticals, Inc.

Opponent:
Grünecker Patent- und Rechtsanwälte PartG mbB

Headword:
ANGPTL3 iRNA/ALNYLAM

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Inventive step - (no)

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Case Number: T 0951/21 - 3.3.08

D E C I S I O N
of Technical Board of Appeal 3.3.08
of 6 November 2023

Appellant: (Patent Proprietor)	Alnylam Pharmaceuticals, Inc. 300 Third Street, 3rd Floor Cambridge, MA 02142 (US)
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Respondent: (Opponent)	Grünecker Patent- und Rechtsanwälte PartG mbB Leopoldstrasse 4 80802 München (DE)
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 28 April 2021 revoking European patent No. 2723758 pursuant to Article 101(3)(b) EPC**

Composition of the Board:

Chair	T. Sommerfeld
Members:	A. Schmitt
	R. Winkelhofer

Summary of Facts and Submissions

I. The appeal of the patent proprietor (appellant) lies from the decision of the opposition division to revoke European patent No. 2 723 758 (the patent).

II. The patent, entitled "*Angiopoietin-like 3 (ANGPTL3) iRNA compositions and methods of use thereof*" was granted on the basis of European patent application No. 12 802 093.0, which had been filed as an international application published as WO 2012/177784 (the application).

III. The opposition proceedings were based on the grounds for opposition in Article 100(a) EPC, in relation to novelty (Article 54 EPC) and inventive step (Article 56 EPC), and those in Article 100(b) and (c) EPC.

IV. In the decision under appeal, the opposition division considered sets of claims of a main request and auxiliary requests I to XII. It found, *inter alia*, that the subject-matter of claim 1 of the main request and auxiliary requests I to XII did not involve an inventive step (Article 56 EPC) in view of the teaching in document D9.

V. With the statement of grounds of appeal, the appellant maintained the main request and auxiliary requests I to XII dealt with in the decision under appeal.

Claims 1 and 3 of the main request read as follows:

"1. A double-stranded ribonucleic acid (dsRNA) for inhibiting expression of ANGPTL3, wherein said dsRNA

comprises a sense strand, an antisense strand and a ligand, wherein the ligand is an N-acetylgalactosamine (GalNAc) derivative, wherein
(a) the antisense strand comprises a region of complementarity which comprises at least 15 contiguous nucleotides differing by no more than 3 nucleotides from any one of the following antisense sequences:
(a1) UAAAAAGACUGAUCAAAUA
(a2) AAAGACUGAUCAAAUAUGU
(a3) AUAAAAAGACUGAUCAAAU
(a4) AGACUGAUCAAAUAUGUUG
(a5) AAAAAGACUGAUCAAAUAUGUUG
(a6) AAAGACUGAUCAAAUAUGUUGAG
(a7) AAAAGACUGAUCAAAUAUGUUGA
(a8) AUAGAUCAUAAAAAGACUGAUCA
(a9) AUCAAAUAUGUUGAGUUUUUGAA
(a10) AAGACUGAUCAAAUAUGUUGAGU
(a11) UAAAAAGACUGAUCAAAUAUGUU
(a12) UAGAUCAUAAAAAGACUGAUCAA,
wherein preferably the region of complementarity consists of one of said antisense sequences, or
(b) the dsRNA comprises a sense strand consisting of a sense strand sequence selected from the following sense sequences:
(s1) UAUUUGAUCAGUCUUUUUA
(s2) ACAUAUUUGAUCAGUCUUU
(s3) AUUUGAUCAGUCUUUUUAU
(s4) CAACAUAUUUGAUCAGUCU
(s5) ACAUAUUUGAUCAGUCUUUUUx
(s6) CAACAUAUUUGAUCAGUCUUUx
(s7) AACAUAUUUGAUCAGUCUUUUx
(s8) AUCAGUCUUUUUAUGAUCUAUx
(s9) CAAAAACUCAACAUAUUUGAUx
(s10) UCAACAUAUUUGAUCAGUCUUx
(s11) CAUAUUUGAUCAGUCUUUUUAx
(s12) GAUCAGUCUUUUUAUGAUCUAx,

wherein "x" indicates that the sequence contains a GalNAc conjugate,
and an antisense strand consisting of one of said antisense sequences."

"3. The dsRNA of claim 1 or 2, wherein said dsRNA comprises at least one modified nucleotide, wherein preferably at least one of said modified nucleotides is selected from the group consisting of a 2'-O-methyl modified nucleotide, a nucleotide comprising a 5'-phosphorothioate group, a terminal nucleotide linked to a cholesteryl derivative or a dodecanoic acid bisdecylamide group, a 2'deoxy-2'-fluoro modified nucleotide, a 2'-deoxy-modified nucleotide, a locked nucleotide, an abasic nucleotide, a 2'-amino-modified nucleotide, a 2'-alkylmodified nucleotide, a morpholino nucleotide, a phosphoramidate, and a nonnatural base comprising nucleotide."

VI. Claim 1 of each of auxiliary requests I to XII differs from claim 1 of the main request in one or more of the following features, as indicated in the brackets.

The antisense strand of the dsRNA as defined in option (a) of the claim comprises a region of complementarity which comprises at least 17 contiguous nucleotides from any of the antisense sequences defined in option (a) of the claim (auxiliary requests I, V and VIII).

The antisense strand of the dsRNA as defined in option (a) of the claim comprises a region of complementarity which has no mismatches with any of the antisense sequences defined in option (a) of the claim (auxiliary requests I, V, VIII, X, XI and XII).

The antisense strand of the dsRNA as defined in option (a) of the claim comprises a region of complementarity with any mismatch(es) being restricted to the last five nucleotides from either the 5' or 3' end of the region of complementarity (auxiliary requests II, VI and IX).

The claimed dsRNA is "for use in a method of treating a subject having a disorder that would benefit from reduction in ANGPTL3 expression" (auxiliary request III).

Sequences (a1), (a2), (a3), (a4) and (s1), (s2), (s3), (s4) have been deleted from options (a) and (b) of the claim (auxiliary requests IV, V, VI and XI).

Sequences (a1), (a2), (a3), (a4), (a5), (a6), (a7) and (s1), (s2), (s3), (s4), (s5), (s6), (s7) have been deleted from options (a) and (b) of the claim (auxiliary requests VII, VIII, IX and XII).

The antisense strand of the dsRNA comprises a region of complementarity which comprises at least 19 contiguous nucleotides from any of the antisense sequences defined in option (a) of the claim (auxiliary requests X, XI and XII).

Claim 1 of auxiliary request XII hence reads as follows:

"1. A double-stranded ribonucleic acid (dsRNA) for inhibiting expression of ANGPTL3, wherein said dsRNA comprises a sense strand, an antisense strand and a ligand, wherein the ligand is an N-acetylgalactosamine (GalNAc) derivative, and wherein:

(a) the antisense strand comprises a region of complementarity which comprises at least 19 contiguous nucleotides from any one of the following antisense sequences:
(a8) AUAGAUCAUAAAAAGACUGAUCA
(a9) AUCAAAUAUGUUGAGUUUUUGAA
(a10) AAGACUGAUCAAAUAUGUUGAGU
(a11) UAAAAAGACUGAUCAAAUAUGUU
(a12) UAGAUCAUAAAAAGACUGAUCAA,
wherein preferably the region of complementarity consists of one of said antisense sequences, or
(b) the dsRNA comprises a sense strand consisting of a sense strand sequence selected from the following sense sequences:
(s8) AUCAGUCUUUUUAUGAUCUAUx
(s9) CAAAAACUCAACAUAUUUGAUx
(s10) UCAACAUAUUUGAUCAGUCUUx
(s11) CAUAUUUGAUCAGUCUUUUUAx
(s12) GAUCAGUCUUUUUAUGAUCUAx,
wherein "x" indicates that the sequence contains a GalNAc conjugate,
and an antisense strand consisting of one of said antisense sequences."

VII. The opponent (respondent) replied to the appeal.

VIII. The board summoned the parties to oral proceedings in accordance with their requests and, in a communication pursuant to Article 15(1) RPBA, expressed its preliminary opinion that, *inter alia*, the subject-matter of claim 1 of each request on file lacked an inventive step.

IX. Oral proceedings took place as scheduled.

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

D1 A. Reynolds et al., Nature Biotechnology 22(3), 2004, 326-330

D3 US 2005/0255487

D3b Excerpt from the "table-xii.txt" file of US 2005/0255487 (D3)

D6 Comparative in vitro studies provided by the opponent in the opposition proceedings

D9 WO 2011/085271 A2

D18 J. K. Watts and D. R. Corey, J Pathol, 2012, Author manuscript, 1-28

XI. The appellant provided arguments supporting their view that the subject-matter of claim 1 of the main request and each of the auxiliary requests I to XII involved an inventive step over the disclosure in documents D9 and D3. In support of their arguments, they also referred to documents D1 and D18. The experimental report D6 provided no legitimate comparative data and should not be taken into account. For the details of the appellant's arguments, reference is made to the reasons for the decision set out below.

XII. The respondent provided arguments supporting their view that the opposition division was right in that the subject-matter of claim 1 of the main request and each of auxiliary requests I to XII lacked an inventive step in view of the disclosure in document D9, irrespective of the teaching in documents D18 and D1, the latter instead supporting the respondent's case. To support their arguments, the respondent also referred to the dsRNA design algorithm and the human ANGPTL3-specific dsRNA molecules designed by this algorithm disclosed in Table XII of document D3/D3b and the experimental data

disclosed in document D6. For details of the respondent's arguments, reference is made to the reasons for the decision set out below.

XIII. The parties' requests relevant for the decision are as follows.

The appellant requests that the decision under appeal be set aside and amended such that the patent be maintained based on the set of claims of the main request submitted on 13 August 2019 or, in the alternative, the set of claims of one of auxiliary requests I to XII, all submitted on 17 December 2020.

The respondent requests that the appeal be dismissed.

Reasons for the Decision

Main request
Inventive step (Article 56 EPC) - claim 1

1. The patent discloses double-stranded ribonucleic acids (dsRNAs) for inhibiting expression of ANGPTL3 (angiopoietin-like 3), a member of the angiopoietin-like family of secreted factors that regulates lipid metabolism. Such dsRNAs may be used in the treatment of lipid metabolism disorders (paragraphs [0001] to [0003] of the patent).

Closest prior art and difference

2. In agreement with the opposition division and the parties, document D9 may be taken as the closest prior art. It discloses antisense compounds for modulating gene expression "*via antisense mechanisms of action*

such as RNaseH, RNAi and dsRNA enzymes" (see page 6, lines 10 to 12 of D9). These antisense compounds target ANGPTL3 mRNA (see e.g. lines 17 and 18 on page 6 and the claims of D9), include "*antisense oligonucleotides, and siRNAs*" (see page 32, lines 12 to 16 of D9) and are useful for the treatment of e.g. cardiovascular disease or metabolic disease (see e.g. lines 13 to 16 on page 6 and lines 5 to 7 on page 59 of D9).

3. In the experimental section of document D9, a series of ANGPTL3-specific single-stranded chimeric oligonucleotides with a length of 20 nucleotides, so-called gapmers, were tested for their ability to reduce human or mouse ANGPRTL3 mRNA levels (see Examples 1 and 2 and Tables 4 and 5 of D9). These gapmers include SEQ ID NO:50, which has the nucleobase sequence GACTGATCAAATATGTTGAG and is 100% complementary to human and mouse ANGPTL3 mRNA.

4. The claimed dsRNA targets the same region of the ANGPTL3 mRNA sequence as SEQ ID NO:50 of document D9. In fact, the nucleic acid sequence of the (a10) antisense strand recited in the claim, AAGACUGAUCAAAUAUGUUGAGU, encompasses a nucleic acid sequence (see underlined sequence) in which the nucleobases are identical to those of SEQ ID NO:50 (see point 3. above) except for the replacement of the nucleobase thymine with the RNA-specific nucleobase uracil. The same is true for the (a6) antisense strand recited in the claim.

5. The claimed subject-matter differs from the gapmer identified by SEQ ID NO:50 of document D9 in that (1) it is a dsRNA and (2) it comprises a ligand which is an N-acetylgalactosamine (GalNAc) derivative.

6. With respect to difference (2), the opposition division found that the addition of carbohydrate ligands, in particular GalNAc-derived ligands, to enhance, *inter alia*, cellular uptake of oligomers was a standard technique in the art and did not involve an inventive step (see point 3.1 of the decision under appeal). This finding was not challenged by the appellant and is therefore not a subject of the appeal proceedings. The following assessment is hence based on the technical effect of difference (1) as defined in point 5. above.

Technical effect and objective technical problem

7. According to the appellant, the claimed dsRNAs were the first ANGPTL3 dsRNAs which effectively knocked down human ANGPTL3 mRNA that had an exceptionally high silencing activity of at least 84% (Table 11 of the patent). The objective technical problem was hence the provision of further means for (highly) effectively inhibiting the expression of human ANGPTL3.

8. However, the results shown in Table 11 of the patent were achieved with dsRNAs consisting of defined sequences of 21 nucleotides and containing specifically modified nucleotides, including 2'-O-methyl- and 2'-fluoro nucleotides, at particular positions within these sequences (see Table 10 of the patent).

9. The claim encompasses many variations of these dsRNAs as it only requires that the claimed dsRNAs comprise 15 contiguous nucleotides of any of the sequences recited in the claim that may include up to three nucleotides that differ from these sequences, defining neither the length of the dsRNA nor any nucleotide modifications. Nucleotide modifications could therefore be present at any position(s) of the nucleotide

sequence in any combination. This is also clear from, for example, dependent claim 3 (see section VI. for the wording of this claim). However, minor changes in the nucleotide sequences of a dsRNA and the type of nucleotide modification in the dsRNA can affect its inhibitory activity.

10. This is evident from, for example, document D1, which discloses that "*a two-base shift in target position was sufficient to significantly alter siRNA functionality*" and that, therefore, "*functionality is determined by the siRNA-specific properties and not by the local mRNA target properties*" (see lines 10 to 13 of the left-hand column on page 326 of D1). The experimental data disclosed in Table 3 of document D6 also support the notion that minor changes in a dsRNA's nucleotide sequence can have an impact on its inhibitory activity. These data demonstrate, *inter alia,* that the inhibitory activity of the AD-45929.1 molecule ("D11") was significantly decreased when several of its 2'-O-methyl-modified nucleotides were exchanged for unmodified nucleotides comprising the same nucleobase (see e.g. the "D12" molecule).

11. The appellant asserted that since it was not known when, where and by whom the data of document D6 had been established, document D6 should not be considered. In addition, document D6 did not provide any legitimate comparative data because it did not use the same experimental conditions as the examples of the patent and should therefore not be taken into account for this reason, either. The mutations made to the dsRNAs in document D6 deliberately disregarded the design rules for functional dsRNA molecules known from document D1 (first full paragraph of the right-hand column on page 328, first full paragraph of the left-hand column

on page 329, Table 1 on page 330). The functional limitation in the claim excluded dsRNAs that did not inhibit the expression of ANGPTL3.

12. However, these arguments cannot be accepted. Document D6 was submitted in the opposition proceedings and was discussed in the decision under appeal (see the last paragraph (point 2) on page 5 and the fourth paragraph (point 3.3) on page 6). Since the EPC does not provide a legal basis for excluding, in appeal proceedings, documents, requests or evidence admitted into the opposition proceedings, especially when the impugned decision was based on them, document D6 is part of the appeal proceedings.

13. Furthermore, for the assessment of document D6's data summarised in point 10. above, no comparison of the patent's data and those of document D6 is necessary. Only the relative inhibitory activities of dsRNAs obtained from the experiment of document D6 must be and were compared. This comparison demonstrated an effect on the dsRNA's inhibitory activity not only when particular nucleobases were mutated, but also when 2'-O-methyl-modified nucleotides were exchanged for unmodified nucleotides, i.e. when modifications were made in the RNA backbone (see point 10. above). Since the dsRNA design rules of document D1 only concern the nature of the nucleobases, the appellant's argument that these design rules were not observed in some of the mutated dsRNAs analysed in document D6 are not relevant for this teaching in document D6.

14. The comparison of the inhibitory activity of the "D11" and "D12" molecules in document D6 therefore supports the notion in document D1 that minor changes in a dsRNA's nucleotide sequence can have an impact on its

inhibitory activity and that, therefore, a "highly effective" silencing activity of at least 84% inhibition, as described in Table 11 of the patent for specific modified dsRNAs, is not an inherent feature of the claimed dsRNAs. The objective technical problem is therefore the provision of further means for inhibiting the expression of human ANGPTL3.

Obviousness

15. The teaching of document D9 is not limited to gapmers but explicitly includes dsRNAs (see point 2. above; an siRNA is a dsRNA). Moreover, document D9 expressly teaches that the same nucleotide sequences which were designed and tested in document D9 as gapmers can be used to design dsRNA molecules. This is evident from Example 3 entitled "*Design and screening of duplexed oligomeric compounds targeting angiopoietin-like 3*". This example discloses that "[i]*n accordance with the invention, a series of duplexes, including dsRNA and mimetics thereof, comprising oligomeric compounds of the invention and their complements can be designed to target angiopoietin-like 3. The nucleobase sequence of the antisense strand of the duplex comprises at least a portion of an oligonucleotide targeted to angiopoietin-like 3 as disclosed herein*".

16. Document D9 therefore proposes designing dsRNAs comprising at least a portion of the disclosed oligonucleotides targeted to ANGPTL3 that include SEQ ID NO:50. Document D9 also describes design features for dsRNAs such as length and overhangs at either terminus (see lines 1 to 21 on page 68 of D9) and discloses methods to produce (see the paragraph that bridges pages 68 and 69 of D9) and evaluate (see the first full paragraph on page 69 of D9) these

dsRNAs. The design, production and evaluation of dsRNA molecules comprising at least a portion of SEQ ID NO:50, such as those recited in the claim (see point 4. above), was hence obvious to the skilled person and did not involve more than routine tasks.

17. This conclusion is furthermore supported by the fact that algorithms for the design of functional inhibitory dsRNAs were known in the art at the priority date of the patent (see e.g. the design rules described in document D3) that, according to paragraph [0319] of D3, automatically output "*the optimal siRNA*". When applying this algorithm to the ANGPTL3 mRNA sequence, *inter alia,* the 771252 (CAACAUAUUUGAUCAGUCU) sequence was obtained (see page 11307 of Table XII of D3 shown in D3b). This sequence is identical to a sequence recited in the claim ((s4)), is fully comprised within two other of these sequences ((s6) and (s10)) and shares 17 contiguous nucleotides with the reverse complement of SEQ ID NO:50 (thymine replaced by uracil). According to the ranking in D3 shown in Table XII (*supra*), it has the seventh highest score of all 125 ANGPTL3-specific sequences provided by the algorithm and is hence a promising candidate for an inhibitory ANGPTL3-specific dsRNA.

18. Document D3 therefore provides an additional indication that the region on the ANGPTL3 mRNA targeted by SEQ ID NO:50 is suitable for designing an inhibitory dsRNA molecule. The provision of dsRNA molecules according to claim 1 does not therefore involve an inventive step.

19. The appellant argued that the contribution of the patent was not merely the provision of inhibitory sequences but the first dsRNA for effectively

inhibiting the expression of human ANGPTL3. Document D9 did not actually disclose any ANGPTL3-specific dsRNAs but only that such molecules could be designed. Example 3 of document D9 was a mere hypothetical example that could not be put into practice. In view of the different mechanisms of action of gapmers and dsRNAs, it was not predictable whether a dsRNA that bound to the same target sequence as a gapmer was inhibitory. This was supported by the teaching in documents D1 and D18. Document D1 disclosed that the functionality of a dsRNA was determined by the dsRNA-specific properties and not by the local mRNA target properties (lines 10 to 13 of the left-hand column on page 326 of D1). Document D18 disclosed that "[a]*n ideal target sequence for an ASO* [antisense oligonucleotide] *is not necessarily ideal for an siRNA and vice versa*" (see the last sentence of the first paragraph on page 8). Without an impermissible hindsight approach, the skilled person did not have any motivation to use the same target sequence for a gapmer and a dsRNA.

20. This line of argument is, however, not persuasive. Document D9 explicitly proposes designing dsRNAs based on the nucleic acid sequences of each of the disclosed gapmers (see points 15. and 16. above) and therefore prompts the skilled person to design and test dsRNAs based on each of these sequences using the standard technology described in document D9. In view of this teaching, the fact that not every dsRNA "necessarily" has the same inhibitory activity as a gapmer designed for the same target sequence, as reported in document D18, or that the functionality of a dsRNA was determined by the dsRNA-specific properties as indicated in document D1, would not have discouraged the skilled person from carrying out the routine screen

proposed in document D9 with the expectation that at least some of these dsRNAs had satisfactory inhibitory activity. In the absence of any evidence that the production of functional ANGPTL3-specific dsRNAs involved specific difficulties or was not possible, it is hence irrelevant that the dsRNAs of the patent were the first ANGPTL3-specific dsRNAs that had actually been tested.

21. The appellant also argued that even if the skilled person was motivated by Example 3 of document D9 to design and test dsRNAs, they would not have considered SEQ ID NO:50 a suitable candidate sequence since the gapmer with SEQ ID NO:50 of document D9 was not particularly successful in silencing human ANGPTL3 gene expression and document D9 disclosed a number of gapmers that outperformed SEQ ID NO:50 (Table 5 of D9). The data in document D9 hence did not provide any motivation or pointer to design dsRNAs based on the target sequence of the gapmer of SEQ ID NO:50. If the skilled person would have considered, at all, designing a dsRNA based on any sequence of document D9, they would have selected one of the more successful gapmers.

22. These arguments cannot be accepted either. The gapmer of SEQ ID NO:50 is one of only a few gapmers that do not have any mismatches with the human ANGPTL3 mRNA (see Table 5). Moreover, it inhibits human ANGPTL3 expression levels by 53% (Table 4 of D9) and is therefore functional. The skilled person hence would (and not only could) have tested each of the few human ANGPTL3-specific candidate sequences proposed in document D9, including SEQ ID NO:50, when confronted with the objective technical problem of providing further means for inhibiting the expression of human ANGPTL3. The fact that the skilled person would not

have excluded SEQ ID NO:50 is furthermore supported by document D3, which identified the same ANGPTL3 mRNA region targeted by SEQ ID NO:50 as a suitable dsRNA target (see point 17. above and Table XII of D3).

23. With respect to the teaching in document D3, the appellant pointed out that none of the *in silico* designed dsRNAs proposed in Table XII of document D3 (D3b) had been tested and validated. Document D3 only provided an algorithm to predict dsRNAs which, however, were not necessarily functional, as discussed in the first sentence of the third paragraph on page 5 of document D18. Since there was no guarantee that inhibitory dsRNAs could be identified based on an algorithm alone, as evident from Example II and Figure 10 of document D3, document D3 was at best an invitation to a research programme.

24. This argument is likewise not persuasive. It is true that each candidate dsRNA proposed in document D3 must be validated and that the skilled person would not have expected that all candidate dsRNAs identified by the algorithm were equally effective. However, the identification of effective dsRNAs from these candidate dsRNAs was a matter of routine (see e.g. the last two paragraphs of Example 3 of D9 on pages 68 and 69).

25. Moreover, the teaching in document D3 only provided an additional pointer to dsRNAs targeting the same ANGPTL3 mRNA sequence as SEQ ID NO:50 of document D9. The respondent did not argue that the claimed dsRNA molecules were not inventive in view of the disclosure in document D3 alone. In view of this and since the dsRNAs assessed in Figure 10 of document D3 are not specific for human ANGPTL3 mRNA, the appellant's argument that Figure 10 of D3 demonstrated that

document D3's algorithm would not necessarily allow identifying suitable dsRNAs with sufficient inhibitory activity is irrelevant for the case at hand, irrespective of its validity.

26. In view of the above considerations, the subject-matter of claim 1 of the main request does not involve an inventive step (Article 56 EPC).

Auxiliary requests I to XII
Inventive step (Article 56 EPC) - claim 1

27. Claim 1 of each of auxiliary requests I to XII differs from claim 1 of the main request in the definition of the claimed dsRNA molecules (see section VI.). However, each of these claims still encompasses many variations of the dsRNAs tested in the patent. Even in the narrowest definition (see claim 1 of auxiliary request XII in section VI.), the claim only requires that the dsRNAs comprise 19 contiguous nucleotides of the recited sequences, defining neither the length of the dsRNA nor any nucleotide modifications.

28. The technical effect and objective technical problem as formulated by the appellant (see point 7. above) can therefore not be acknowledged for the subject-matter of claim 1 of any of the auxiliary requests I to XII for the same reasons indicated in points 8. to 14. above for claim 1 of the main request. Consequently, the considerations on obviousness for claim 1 of the main request (see points 15. to 26. above) also apply to claim 1 of each of auxiliary requests I to XII.

29. The subject-matter of claim 1 of each of auxiliary requests I to XII therefore does not involve an inventive step (Article 56 EPC) for the same reasons as

the subject-matter of claim 1 of the main request (see points 2. to 26. above).

Order

For these reasons it is decided that:

The appeal is dismissed.

The Registrar: The Chair:

L. Malécot-Grob T. Sommerfeld

Decision electronically authenticated