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**Datasheet for the decision  
of 2 December 2010**

**Case Number:** T 1642/07 - 3.3.04

**Application Number:** 96934086.8

**Publication Number:** 0862445

**IPC:** A61K 35/76

**Language of the proceedings:** EN

**Title of invention:**

Methods and compositions for viral enhancement of cell killing

**Applicant:**

Arch Development Corporation, et al.

**Headword:**

Viral enhancement of cell killing/ARCH DEVELOPMENT CORPORATION  
et al.

**Relevant legal provisions:**

EPC Art. 123(2), 54, 56, 111(1)

**Relevant legal provisions (EPC 1973):**

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**Keyword:**

"Main request: added subject-matter (no)"  
"Novelty (yes)"  
"Inventive step (yes)"

**Decisions cited:**

T 1329/04

**Catchword:**

-



Case Number: T 1642/07 - 3.3.04

**DECISION**  
of the Technical Board of Appeal 3.3.04  
of 2 December 2010

**Appellant:** Arch Development Corporation  
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Chicago, IL 60637 (US)

**Representative:** Bösl, Raphael Konrad  
Isenbruck Bösl Hörschler LLP  
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**Decision under appeal:** Decision of the Examining Division of the  
European Patent Office posted 19 February 2007  
refusing European patent application  
No. 96934086.8 pursuant to Article 97(1) EPC.

**Composition of the Board:**

**Chairman:** C. Rennie-Smith  
**Members:** R. Gramaglia  
B. Claes

## Summary of Facts and Submissions

I. The appellants (applicants) lodged an appeal against the decision of the examining division on the refusal under Article 97(1) EPC 1973 of the European patent application No. 96934086.8 (published as WO-A-97/12623), having the title "Methods and compositions for viral enhancement of cell killing".

II. The decision was based on claims 1 to 20 of the main request filed during the oral proceedings on 23 January 2007 and on claims 1 to 21 of the auxiliary request filed with the letter dated 22 December 2006.

III. Independent claims 1 and 7 of the main request pending before the examining division read as follows:

"1. A combination of a herpes simplex virus (HSV) and an effective amount of a chemotherapeutic agent for simultaneous, separate or sequential use in a method of treatment of the human or animal body, wherein the HSV is not a HSV wherein the genome of the virus contains an expressible non-herpes simplex virus nucleotide sequence encoding a desired protein capable of eliciting an immune response in a subject, and is altered in the  $\gamma_1$  34.5 gene and the ribonucleotide reductase gene."

"7. Use of a herpes simplex virus (HSV) for the manufacture of a medicament for the therapy of tumors, wherein the virus is administered simultaneously, separately or sequentially in combination with an effective amount of a chemotherapeutic agent,

wherein the HSV is not a HSV wherein the genome of the virus contains an expressible non-herpes simplex virus nucleotide sequence encoding a desired protein capable of eliciting an immune response in a subject, and is altered in the  $\gamma$ 1 34.5 gene and the ribonucleotide reductase gene."

IV. Independent claims 1 and 7 of the auxiliary request pending before the examining division read as follows:

"1. A combination of a herpes simplex virus (HSV) and an effective amount of a chemotherapeutic agent for simultaneous, separate or sequential use in a method of treatment of the human or animal body."

"7. Use of a herpes simplex virus (HSV) for the manufacture of a medicament for use in the killing of a malignant cell, wherein the virus is administered simultaneously, separately or sequentially in combination with an effective amount of a chemotherapeutic agent."

V. The examining division considered that, in the light of document

D3 EP-A-0 514 603

taken as closest prior art, the technical problem underlying the present application was the provision of an alternative anticancer therapy using HSV.

VI. The examining division noted that the then claimed combination therapy (HSV + chemotherapeutic agent) resulted in an additional or synergistic effect (see

page 4, line 11 of the published WO application) and came to the following conclusions:

- (a) If an additional effect occurred, the claimed combination therapy (HSV together with a chemotherapeutic agent) was obvious over document D3 combined with the common general knowledge. Combination therapies with chemotherapeutic agents had been a standard in the treatment of cancer for many years. Therefore, the provision of additive effects was obvious.
  
- (b) If a synergistic effect occurred, this synergistic effect was not disclosed in the application as filed and all the experimental evidence concerning any synergistic effect was published well after the filing date of the present application. However, according to decision T 1329/04 of 28 June 2005, later evidence could only be taken into account when the disclosure in an application rendered plausible that its teaching indeed solved the problem it purported to solve.
  
- (c) It was highly improbable that just any HSV virus species exhibited a synergistic anticancer activity when administered in combination with any DNA damaging agent.
  
- (d) Hence, in the absence of a demonstrated synergistic effect in the application as filed, which in any case did not extend to all the possible combinations covered by claim 1 (see post-published documents E7, E8 and E13 to E15), no inventive step could be acknowledged.

VII. Together with the statement setting out the grounds of appeal, the appellant filed on 28 June 2008 a main request and auxiliary requests 1 to 7.

VIII. Oral proceedings were held on 2 December 2010, during which the appellant filed a new main request, of which independent claims 1 and 8 read as follows:

"1. A combination of a herpes simplex virus (HSV) and an effective amount of a chemotherapeutic agent which induces DNA damage for simultaneous, separate or sequential use in a method of treatment of the human or animal body."

"8. Use of a herpes simplex virus (HSV) for the manufacture of a medicament for the therapy of tumors, wherein the virus is administered simultaneously, separately or sequentially in combination with an effective amount of a chemotherapeutic agent which induces DNA damage."

Dependent claims 2 to 7 and 9 to 22 related to specific embodiments of the combination according to claim 1 or the use according to claim 8, respectively.

IX. The following documents, in addition to the one cited in paragraph V above, are referred to in the present decision:

D1 Fujiwara T. et al., Cancer Research, Vol. 54, pages 2287-2291 (1994);

- D2 Hallahan D.E. et al., *Nature Medicine*, Vol. 1, No. 8, pages 786-791 (August 1995);
- E1 Sibley G.S et al., *International Journal of Radiation Oncology Biology Physics*, Vol. 32, Supplement 1, page 173, Abstract No. 64 (September 1995);
- E4 Hallahan D.E. et al., *Proc. Natl. Acad. Sci. USA*, Vol. 88, pages 2156-2160 (1991);
- E7 Post D.E. et al., *Current Gene Therapy*, Vol. 4, pages 41-51 (2004);
- E8 Eisenberg D.P. et al., *Journal of Gastrointestinal Surgery*, Vol. 9, No. 8, pages 1068-1079 (2005);
- E10 WO-A-96/00007;
- E11 Rodrigues R.L., *Emergency Medicine Clinics of North America*, Vol. 11, No. 2; pages 431-443 (1993);
- E12 Lesser G.J. et al., *Cancer Treatment Reviews*, Vol. 19, pages 261-281 (1993);
- E13 Gutermann A. et al., *Human Gene Therapy*, Vol. 17, pages 1241-1253 (2006);
- E14 Adusumilli P.S. et al., *Cancer Biology & Therapy*, Vol. 5, No. 1, pages 48-53 (2006);
- E15 Mullerad M. et al., *The Journal of Urology*, Vol. 174, pages 741-746 (2005);

E16 Declaration of Dr. Bernard Roizman dated 26 June 2007.

X. The submissions by the appellant (applicants), insofar as they are relevant to the present decision, can be summarized as follows:

*Main Request*

*Article 123(2) EPC*

- The language "a chemotherapeutic agent which induces DNA damage" in claims 1 and 8 found a basis on page 4, lines 24-27 of the published WO application.
- Support for claims 2 and 19 could be found on page 16, line 6 of the published WO application.
- No formal objections had been raised against the remaining claims by the examining division either during the oral proceedings or in the written decision.

*Article 54 EPC*

- The limitation in independent claims 1 and 8 of the chemotherapeutic agent to "a chemotherapeutic agent which induces DNA damage" ensured that the subject-matter of the claims was novel.



*Article 56 EPC*

- Document D3 represented the closest prior art. There was no suggestion in this document that HSV could be combined with a chemotherapeutic agent which induced DNA damage.
  
- The skilled person coming across document E1 would not have considered a DNA damaging drug as an alternative to radiotherapy in combination with HSV. He/she would ascribe the increase in cell killing effect by virus R899-6 noted in document E1 to enhanced TNF- $\alpha$  production, not to the virus itself. As regards virus R3616, it was not known by which mechanism this virus exerted its cytolytic activity. Moreover, the biological mechanism underlying radiotherapy and chemotherapy were known to be different.
  
- The skilled person would expect the chemotherapeutic agent to induce DNA damage not only in the cancer cells but on the virus DNA as well.
  
- Documents E11 and E12 did not suggest that a chemotherapeutic agents inducing DNA damage could be combined with any other type of anti-tumour agent, let alone with HSV. Therefore the skilled person would not have combined document D3 with documents E11 or E12.
  
- There was no hint in the prior art that HSV and a DNA damaging drug could be effective in all tumour types.

XI. The appellants requested that the decision under appeal be set aside and that a patent be granted on the basis of either the claims of the new main request filed at the oral proceedings on 2 December 2010 or on the basis of one of auxiliary requests 1 to 7 filed on 28 June 2008.

## **Reasons for the Decision**

### *Article 123(2) EPC*

1. Independent claims 1 and 8 are respectively based on claim 1 and 8 as filed with letter dated 26 November 2004, against which the examining division did not raise any objection under Article 123(2) EPC, and neither does the board, with the wording "a DNA damaging agent" replaced with the expression "a chemotherapeutic agent which induces DNA damage". A basis for this language is on page 4, lines 24-27 and page 6, lines 13-17 of the published WO application, from which it can be derived that the agent referred to in claim 1 is a chemotherapeutic agent which induces DNA damage when applied to a cell.
2. Furthermore, the wording in former claim 8 "for use in killing an undesirable cell" has been replaced with the expression "for the therapy of tumors" in present claim 8. A basis for this language can be found on page 5, lines 29-30 of the published WO application.
3. Dependent claims 2 and 19 specify that the HSV is non-pathogenic. Support for these claims can be found on

page 16, line 6 in combination with the title "Herpes Simplex Virus" on page 15, last line of the published WO application.

4. Dependent claims 3 to 7 are based on claims 2 to 4, 6 and 7 filed with letter dated 26 November 2004. Dependent claims 9 to 22 are based on claims 9, 10, 13 to 15, 17 and 19 to 25 as filed with letter dated 26 November 2004. No formal objections have been raised against these claims by the examining division and the board also sees none.
5. In conclusion, the claims of the main request satisfy the requirements of Article 123(2) EPC.

*Article 54 EPC*

6. Document E10 (cited under Article 54(3) EPC) discloses the use of a modified herpes simplex virus (see page 7, lines 10-23) bearing alterations in both the  $\gamma$ -34.5 gene and the ribonucleotide reductase gene (see page 9, lines 5-11), in combination with chemotherapy (see page 7, lines 21-24). However, since the broad term "chemotherapy" can not be considered as being a direct and unambiguous disclosure of a "chemotherapeutic agent which induces DNA damage", document E10 is not novelty-destroying for the subject-matter of the claims at issue.
7. Document D4 (cited under Article 54(3) EPC) discloses on page 43, lines 19-21, the use of a DNA damaging agent in conjunction with a construct expressing p16 (a tumour suppressor; see page 7, lines 32-33). The expression construct may be a virus (see page 27,

line 28 to page 35, line 31). However, no reference is made to HSV. On page 41, lines 13-17, an "herpes simplex-thymidine kinase gene" is mentioned, however, this occurs in the context of the delivery of this gene to brain tumours by means of a retrovirus. Therefore, document D4 is also not novelty-destroying for the subject-matter of the claims at issue.

8. In conclusion, no prior art document presently before the board anticipates the claimed subject-matter.

*Article 56 EPC*

*Closest prior art*

*Document D3*

9. This document discloses a thymidine kinase mutant of HSV (HSV1-dlsptk) capable of killing human gliomas (see column 12, last paragraph). Fig. 2 of document D3 shows that at days 14 and 26 after inoculation, the HSV1-dlsptk-treated tumours were significantly smaller than the controls. There is no suggestion in document D3 that HSV could be combined with a chemotherapeutic agent which induces DNA damage for the purpose of killing cancer cells.

*Document E1*

10. It has not been disputed by the appellants that document E1 is a pre-published abstract by the inventors of the present application describing the same experiments as in "Study 1" of Example 1 of the present application (see page 23 of the published WO application). In particular, the treatments used and

the mean tumour volume after each treatment shown in the Table of the abstract are identical to those in Table 1 of the present application. Abstract E1 compares the tumour cell-killing effect of each of a  $\gamma$ 34.5 negative HSV-1 strain (R3616), a  $\gamma$ 34.5 negative HSV-1 strain which expresses human TNF- $\alpha$  (R899-6) and radiotherapy (RT) alone, to the tumour cell-killing effect of either virus R3616 or virus R899-6 in combination with RT. The results of the in vitro combined cell killing tests (performed with 2-9 Gy radiation) are said to be "additive" for both viruses, although no enhanced cytotoxicity with the TNF- $\alpha$ -producing virus is seen. As for the in vivo tests (performed with 20 and 25 Gy radiation), the Table of abstract E1 shows that combining either the R3616 virus or the R899-6 virus with RT leads to a greater reduction in mean tumour volume than treatment with any of the R3616 virus, the R899-6 virus and radiotherapy alone.

11. In essence, the claims of the main request relate to the combined administration of HSV and a chemotherapeutic agent inducing DNA damage for use in therapy in general according to claim 1 (e.g. killing benign prostate hyperplasia cells; see page 6, line 24 of the published WO application) or for treating cancer according to claim 8. It can be derived from page 3, line 18 to page 4, line 11 that when HSV is administered in combination with a chemotherapeutic agent inducing DNA damage, a potentiation of the therapeutic effect (i.e., an increase of the level of cell killing above that seen for a treatment modality alone) occurs. It is stated on page 4, line 11 of the published WO application that "[P]otentiation may be

additive, or it may be synergistic". Therefore the board interprets independent claims 1 and 8 of the main request in the light of the description as **implicitly** including the feature that at least an additive or in some case a synergistic effect should take place.

12. The examining division considered document D3 to be the closest state of the art (see paragraph V supra). The board does not endorse this approach. Document D3 (see point 9 supra) is concerned neither with a virus-based combination therapy, let alone with a potentiated combination therapy, unlike document E1 (see point 10 supra). In fact, the board observes that the only difference between the claimed subject-matter and document E1 lies in the use of a chemotherapeutic agent which induces DNA damage instead of radiation. Therefore, document E1 represents the closest prior art.

*Problem to be solved*

13. The examining division considered that, departing from document D3 as closest prior art, the technical problem underlying the present application was the provision of an alternative anticancer therapy using HSV (see paragraph V supra). Starting from document E1, the board arrives at a different formulation of the problem to be solved as being the provision of an alternative potentiated virus-based combination therapy for killing cells (claim 1 and dependent claims) or cancer cells (claim 8 and dependent claims), wherein "potentiated" means additive until synergistic, or in other words "at least additive" (see point 11 supra). This new formulation finds support on page 4, line 11 and

page 29, line 10, of the published WO application. The proposed solution lies in the replacement of radiation with a chemotherapeutic agent which induces DNA damage.

*Has the problem been solved?*

14. In the decision under appeal, the examining division considered that the formulated technical problem had **not** been solved.
15. However, as explained in detail below (see points 16 to 19 infra), the examining division's negative arguments summarized in paragraphs VIb to VIId supra in relation to the failed solution of the technical problem do not (or no longer) apply to the formulation of the technical problem set out by the board. This formulation is now the provision of an alternative potentiated virus-based combination therapy (wherein "potentiated" means additive until synergistic, or in other words "at least additive") (see point 13 supra).
16. One of the examining division's lines of argument (see paragraph VIb supra) was that the patent application as filed did not comprise experimental data showing the synergistic effect.
17. Firstly, the board cannot agree with the approach adopted by the examining division which unjustifiably turned its original formulation from "the provision of an alternative anticancer therapy using HSV" (see paragraph V supra) into "the provision of a synergistic anticancer combination therapy", as the arguments summarized in paragraphs VIb to VIId supra seem to suggest.

18. Secondly, it is true that the patent application contains no experimental evidence in support of the claimed combination therapy but only theoretical statements that viral therapy in combination with a chemotherapeutic agent inducing DNA damage results in an additive until synergistic killing effect on cells/cancer cells (see page 6, lines 9 to 18 and page 4, lines 3 to 11). A "prophetic" Example III(3) disclosing mitomycin C at a dose of 20 mg/m<sup>2</sup> to be used in conjunction with an adenovirus can also be found on page 29 of the published WO application. HSV viruses are dealt with on pages 16 and 17. The patent application as filed thus addresses expressis verbis the claimed subject matter and potentiation (additive until synergistic killing effect on cells/cancer cells).

However, the board observes that there is no requirement in the EPC, let alone in Article 56 EPC, that a patent application should include experimental evidence in support of patentability or a claimed technical effect. Hence, the fact that the disclosure in a patent application is merely theoretical and not supported by experimental data is in itself no bar to patentability or to the presence of a technical effect being acknowledged.

19. Further, the examining division, relying on decision T 1329/04, decided that post-published documents E7, E8 and E13 to E15 could not be taken into account for showing that the synergistic effect occurred, because the disclosure in the present application did not render plausible that its teaching indeed solved the problem of providing a synergistic anticancer



combination therapy and did not render plausible that this synergistic effect occurred for all the possible combinations covered by claim 1.

20. However, the formulation of the technical problem to be solved as set out by the board is less demanding than the examining division's, since it now only requires that the potentiating effect be additive until synergistic (in other words, "at least additive") rather than "synergistic" for any combination. Post-published document E7 (this document shows that HSV R1716 + mitomycin C = additive effect in 3/5 cells and synergistic effect in 2/5 cells and that HSV R3616 + cisplatin = additive effect), document E8 (HSV NV1066 + 5-FU or gemcitabine = synergistic effect in 3 cell lines), E13 (HSV NV1020 + 5-FU, SN38 or oxaliplatin = additive up to synergistic effect), document E14 (HSV NV1066 + cisplatin = synergistic effect in 6 cell lines) and document E15 (HSV NV1066 + mitomycin C = synergistic effect in 2 cell lines), submitted by the appellant, illustrate such an "at least additive" effect for **all** the combinations. Therefore, the dichotomy noted by the examining division between the disclosure in the patent application and the technical teaching in post-published documents E7, E8 and E13 to E15 no longer subsists. Rather, the post-published documents can be viewed as being a mere confirmation of the technical effect already announced (albeit at a theoretical level) in the application as filed.
21. The board observes that such a dichotomy arose between, on the one hand, the disclosure in the patent application underlying decision T 1329/04 (lack of the seven cystein residues with their peculiar spacing

required for a protein (in that case, GDF-9) to belong to the TGF- $\beta$  superfamily -see T 1329/04, point 7 of the reasons- and the lack of functional characterisation of GDF-9 -see ibidem, point 9 of the reasons-) and, on the other hand, the teaching in post-published document (4) that GDF-9 was indeed a growth differentiation factor (see T 1329/04, point 12 of the reasons). Hence, the then competent board concluded that there was not enough evidence in the application as filed to make it at least plausible that a solution had been found to the problem alleged to be solved.

22. In summary, the negative arguments produced by the examining division no longer apply to the less demanding problem set out in point 13 supra. The board sees also no grounds for doubting that the combined administration of HSV and a chemotherapeutic agent inducing DNA damage is able to achieve an increase of the level of cell killing above that seen for a treatment modality alone. Under these circumstances, post-published documents E7, E8 and E23 to E15 can be taken into account.

23. In view of the foregoing, the board concludes that the problem highlighted in point 13 supra has indeed been solved by the claimed subject-matter.

*Inventive step*

24. The only issue remaining to be decided is whether or not the proposed solution (replacement of radiation in document E1 with a chemotherapeutic agent which induces DNA damage) to the problem formulated in point 13 supra follows from the prior art in an obvious way.

25. Departing from document E1 (see the detailed analysis of this document made in point 10 supra), the skilled person faced with solving the problem at issue would ascribe the increase in cell killing effect by virus R899-6 to enhanced TNF- $\alpha$  production, not to the virus itself. This is because it was already known from documents D2 (see page 786, bottom of l-h column) and E4 (see page 2159, l-h column, lines 20-23) that X-rays at doses of 20-25 Gy were capable of inducing a high TNF- $\alpha$  production by virus constructs expressing said cytotoxic agent.
  
26. Therefore the skilled person would focus on the tests in document E1 relating to HSV virus R3616, which belonged to the family of oncolytic HSV viruses (see page 17, line 20 of the published application) which did not require the use of a specific insert for function (see page 16, lines 4-5 of the application). Another example of virus belonging to the latter category is the recombinant adenovirus expressing the wild type p53 gene, which upon transfer into H358 tumour cells markedly increased the cellular sensitivity of these cells to the chemotherapeutic drug cisplatin (see abstract of document D1). The results pertaining to HSV virus R3616 of the in vitro combined cell killing test (performed with 2-9 Gy radiation) is said to be "additive". As for the in vivo test, the Table of abstract E1 shows that combining the R3616 virus with RT leads to a greater reduction in mean tumour volume than treatment with any of the R3616 virus and radiotherapy alone.

27. However, the board observes that, in spite of these promising results, the mechanism by which R3616 exerted its (increased) oncolytic activity was not explained in document E1, nor was it known from other sources. In fact, researchers were still investigating this aspect even after the priority date (6 October 1995) of the present application (see document E7, page 47, paragraph bridging l-h and r-h columns).
28. Moreover, as highlighted in paragraph 10 of document E16, the biological mechanism underlying radiotherapy (breaks in ss-DNA and ds-DNA, loss of entire genes and local application) and chemotherapy (intra-strand cross-linking of DNA, point mutations, alkylation of the DNA, inhibition of nucleotide/DNA biosynthesis and systemic application) were known to be different.
29. Finally, it should be noted that the skilled person would expect the chemotherapeutic agent to induce DNA damage not only in the cancer cells but on the virus DNA as well.
30. In view of these uncertainties, in the board's judgement, the skilled person departing from the combined method to kill tumour cells using HSV and radiation described in document E1 was not motivated to replace radiation with a chemotherapeutic agent inducing DNA damage. Much less had he/she any expectation that such replacement would achieve an increase of the level of cell killing above that seen for one modality of treatment alone.
31. The examining division concluded (see paragraph VI(a) supra) that if an additional effect occurred, the

claimed combination therapy was obvious over document D3 combined with the common general knowledge. It was argued that combination therapies with chemotherapeutic agents had been a standard in the treatment of cancer for many years.

The only documents before the board dealing with the combinations of chemotherapeutic agents are documents E11 and E12. These documents show that one or more chemotherapeutic agents inducing DNA damage could be combined for the purpose of treating a specific cancer. However, no suggestion could be drawn from these documents that a chemotherapeutic agent inducing DNA damage could be combined with another type of anti-tumour agent (non-DNA damaging agent), let alone with HSV, for the same scope. Further, the fact that the combinations of chemotherapeutic agents disclosed in documents E11 and E12 were effective in killing very specific cancer cell lines only (see e.g. document E12, page 265, second full paragraph) was not encouraging for the skilled person looking for a combined therapy effective in all tumour types, like the claimed combined therapy (see page 3, line 26 to page 4, line 2 of the published WO application and point 20 supra).

32. The board also notes that although oncolytic viruses had been known since 1954 (see document E10, page 2, lines 4-21), and alkylating ("mustard") drugs since the fifties of the last century, no combination therapy as claimed had been proposed until the priority date of the present application (6 October 1995). It is true that document E7 encourages (see page 45, r-h column, third paragraph) the combination of oncolytic viruses with a chemotherapeutic agent, however, this teaching,

made available to the public in 2004, can not be made retroactive for the purpose of deciding inventive step.

33. Moreover, the examining division itself acknowledged, albeit in the context of synergy, that "the demonstration of a synergistic effect for HSV + radiotherapy cannot be extrapolated to HSV + chemotherapy due to the different mechanisms of action of radiotherapy as opposed to chemotherapy" (see page 6, point 3.3.2 (1) of the decision under appeal).
34. In conclusion, the subject-matter of independent claims 1 and 8 and dependent claims 2 to 7 and 9 to 22 satisfies the requirements of Article 56 EPC.

#### *Remittal*

35. As decided in points 5, 8 and 34 above, the claims according to the main request satisfy the requirements of Article 123(2), 54 and 56 EPC and meet the objections on which the appealed decision exclusively relies. Since the substantive issues of Art 83 EPC and adaptation of the description have not been the subject of discussion, the board considers it appropriate to exercise its discretion under Article 111(1) EPC and to remit the case to the department of first instance for further prosecution.

**Order**

**For these reasons it is decided that:**

1. The decision under appeal is set aside.
2. The case is remitted to the first instance for further prosecution.

The Registrar:

The Chairman:

P. Cremona

C. Rennie-Smith