Federal Court Cour fédérale

Date: 20240808

Docket: T-1563-22

Citation: 2024 FC 1198

Ottawa, Ontario, August 8, 2024

PRESENT: The Honourable Madam Justice Furlanetto

BETWEEN:

**BOEHRINGER INGELHEIM (CANADA) LTD.
AND BOEHRINGER INGELHEIM
INTERNATIONAL GMBH**

Plaintiffs

and

JAMP PHARMA CORPORATION

Defendant

PUBLIC JUDGMENT AND REASONS

(Confidential Judgment and Reasons issued July 26, 2024)

I. Overview

[1] This judgment arises from a patent infringement action brought under subsection 6(1) of the *Patented Medicines (Notice of Compliance Regulations)*, SOR/93-133 [*PMNOC Regulations*]. The patents at issue are Canadian Patent Nos. 2,591,083 [083 Patent] and

2,726,267 [267 Patent]. The innovative drug at issue is nintedanib, which is sold under the brand name OFEV®.

[2] Boehringer Ingelheim (Canada) Ltd. [BI] is the "first person" in accordance with the *PMNOC Regulations*. Boehringer Ingelheim International GmbH [BII] is the registered owner of the 083 Patent and the 267 Patent and is a party to the action pursuant to subsection 6(2) of the *PMNOC Regulations*.

[3] BI claims that the making, constructing, using and/or selling by the Defendant JAMP Pharma Corporation [JAMP] of its nintedanib capsules in a strength of 150 mg [JAMP Nintedanib] in accordance with JAMP's Abbreviated New Drug Submission [ANDS] Control Number 262177 will infringe, and induce infringement of, at least one of claims 1-6 of the 083 Patent [083 Asserted Claims], and at least one of claims 8 (when read through claims 7, 5, 3, 2 and 1, or claims 7, 2 and 1), 22 (when read through claims 8, 7, 5, 3, 2 and 1, or claims 8, 7, 2 and 1) and 23 (when read through claims 8, 7, 5, 3, 2 and 1, or claims 8, 7, 2 and 1) of the 267 Patent [267 Asserted Claims].

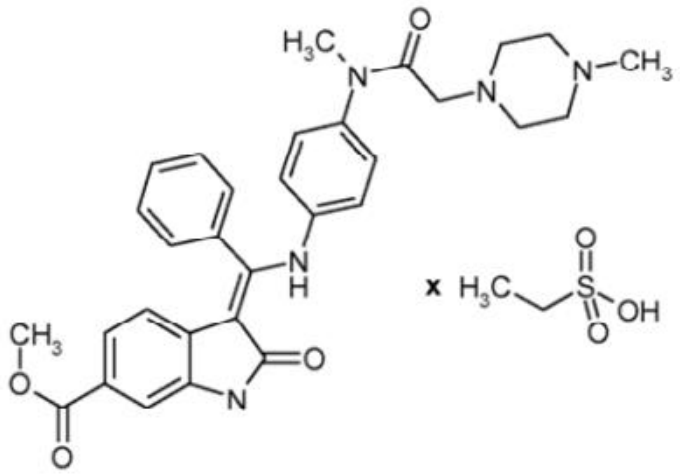
[4] JAMP denies infringement of the 267 Patent and also asserts in defence that claim 8 (when read through claims 7, 2, and 1) and claims 22 and 23 (when read through claims 8, 7, 2, and 1) of the 267 Patent are invalid for obviousness and that claim 2 is invalid for overbreadth. JAMP has stipulated that it will induce infringement of the 083 Asserted Claims, but denies that it will directly infringe the claims and also asserts in defence that the 083 Patent is invalid for

anticipation, double patenting, lack of sound prediction of utility, or in the alternative to lack of sound prediction, obviousness.

[5] As set out further below, I find that the action should be allowed in part as the 083 Patent is valid and infringed, but the 267 Patent is not infringed.

II. **Background**

[6] The 083 Patent and the 267 Patent are listed on the Patent Register in association with the medicine nintedanib esylate (also spelled esilate), which is the monoethanesulfonate salt of 3-Z-[1-(4-(N-((4-methyl-piperazin-1-yl)-methylcarbonyl)-N-methyl-amino)-anilino)-1-phenyl-methylene]-6-methoxycarbonyl-2-indolinone, also known as 3-Z-[1-(4-(N-((4-methyl-piperazin-1-yl)-methylcarbonyl)-N-methyl-amino)-anilino)-1-phenyl-methylene]-6-methoxycarbonyl-2-indolinone-monoethanesulfonate, depicted below:



[7] BI markets and sells nintedanib capsules in strengths of 100 mg and 150 mg (corresponding to 120.40 mg and 180.60 mg of nintedanib esylate, respectively) under the brand

name OFEV®. A generic version of the 100 mg dosage strength of nintedanib is the subject of a separate action in this Court (T-352-24).

[8] OFEV® is indicated, *inter alia*, for the treatment of idiopathic pulmonary fibrosis [IPF], a rare disease of the lungs. IPF is a chronic, incurable disease that typically leads to respiratory failure and death. IPF is one of seven diseases that are classified as an idiopathic interstitial pneumonia [IIP], which is a sub-category of interstitial lung disease [ILD]. It is a progressive disease in which fibrotic material deposits and accumulates, creating scarring and distorting the interstitium (the passageway that separates the air and the blood) in ways that alters gas exchange function.

[9] Nintedanib is one of only two medicines that have been approved for the treatment of IPF in Canada. BI holds Notices of Compliance [NOC] for OFEV® and began selling OFEV® in Canada in 2014.

[10] JAMP was issued a NOC for JAMP Nintedanib on September 6, 2023. JAMP Nintedanib is approved for the treatment of IPF. However, JAMP Nintedanib has not yet been offered for sale or sold in Canada. If JAMP elects to launch JAMP Nintedanib in Canada, it will be the importer of JAMP Nintedanib into Canada, it will sell JAMP Nintedanib in Canada, and it will market JAMP Nintedanib for the indications contained in the JAMP Nintedanib product monograph.

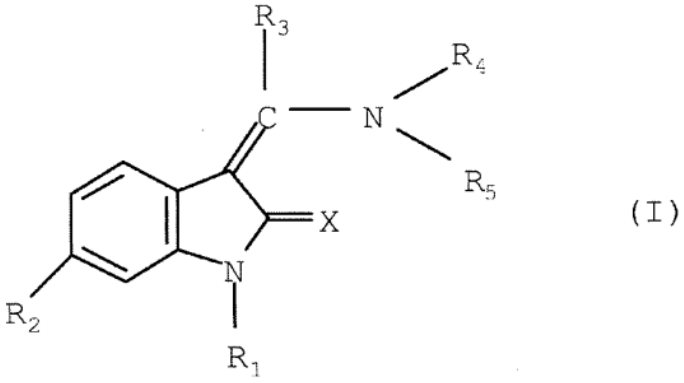
[11] For purposes of this proceeding, the Plaintiffs have renounced the 24-month stay provided by paragraph 7(1)(d) of the *PMNOC Regulations* in accordance with paragraph 7(5)(b) of the *PMNOC Regulations*.

III. **The Patents**

A. *The 083 Patent*

[12] The 083 Patent is entitled "Medicaments for the Treatment or Prevention of Fibrotic Diseases" and is the national phase entry of a Patent Co-operation Treaty [PCT] patent application. The 083 Patent has a filing date of December 21, 2005 and claims priority from European Patent Application No. 04030770.4, filed on December 24, 2004. The application for the 083 Patent was published on June 29, 2006, and issued to patent on October 1, 2013. It will expire on December 21, 2025.

[13] Page 1 of the 083 Patent identifies the technical field of the invention of the 083 Patent as relating to a new use of indolinones of general formula (I) substituted in the 6 position, the tautomers, the diastereomers, the enantiomers, the mixtures thereof and the salts thereof, particularly physiologically acceptable salts thereof (083 Patent, Ex1, 1:13-21).



(I)

[14] The Background of the 083 Patent describes the compounds of general formula (I) as previously known and as having "valuable pharmacological properties" and "an inhibiting effect on various kinases, especially receptor tyrosine kinases such as VEGFR2, PDGFRα, PDGFRβ, FGFR1, FGFR3, EGFR, HER2, IGF1R and HGFR" (083 Patent, Ex1, 2:5-9).

[15] It notes that none of the compounds of general formula (I) have previously been described for their use in the treatment or prevention of fibrotic diseases (083 Patent, Ex1, 2:19-21). The 083 Patent notes that instances where fibrosis "severely compromises the normal function(s) of the organ" can lead to severe fibrotic diseases, including "idiopathic pulmonary fibrosis (IPF), liver cirrhosis, scleroderma, or renal fibrosis" (083 Patent, Ex1, 3:5-12).

[16] The 083 Patent discusses the biochemical mechanisms that underlie fibrosis and notes that "[a] large body of literature implicates the platelet-derived growth factor (PDGF), fibroblast growth factor (FGF), vascular endothelial growth factor (VEGF), epidermal growth factor (EGF), and transforming growth factor beta (TGF-β) growth factor families in the induction or persistence of fibrosis" (083 Patent, Ex1, 3:14-24). These factors bind to receptors with tyrosine kinase activity, *i.e.*, VEGFR, for example. The 083 Patent states that experimental models reported in the literature suggest that inhibition of tyrosine kinases seems to reduce the severity of fibrosis (083 Patent, Ex1, 5: 21-23).

[17] In the Summary of the Invention, the inventors indicate that they have surprisingly found that the compounds of general formula (I) are effective in the treatment or prevention of specific

fibrotic diseases (083 Patent, Ex1, 5:28-30). The Summary provides embodiments of the invention relating to nintedanib, its esylate salt and pharmaceutical compositions thereof for use in the prevention or treatment of IPF and to the use of nintedanib and its esylate salt for the prevention or treatment of IPF.

[18] In the Detailed Description [Description], the 083 Patent discloses compounds of general formula (I) and their synthesis with reference to methods described in prior patent applications. The Description provides a list of 795 compounds that it states are illustrative of the present invention (083 Patent, Ex1, 92:19-179:16). It identifies the monoethanesulfonate salt of nintedanib as a particularly preferred compound and recognizes its prior disclosure in WO 04/13099 [WO 099] (083 Patent, Ex1, 91:14-92:1).

[19] The Description provides a lengthy list of fibrotic diseases suitable for treatment with the compounds of general formula (I), highlighting that the compounds of the invention are especially suitable for the prevention or treatment of IPF as a “preferred embodiment” (083 Patent, Ex1, 180:27-31).

[20] Under the heading “Biological Activity”, the 083 Patent provides two examples evaluating compounds in a bleomycin-induced rat lung fibrosis model following a regimen known to the person skilled in the art [PSA] as the “preventative regimen”. Example B2 refers to a study involving nintedanib (compound (u)) (083 Patent, Ex1, 188:10-14). Three groups of rats were tested in the example. The first group was exposed to bleomycin sulfate (bleomycin) on day 0, followed by daily administration of nintedanib (compound (u)) for 21 days. The second group

(which was the positive control) was exposed to bleomycin on day 0, followed by a saline vehicle for 21 days. The third group was treated with a saline vehicle (instead of bleomycin) between days 1 and 21, and served as the negative control.

[21] On day 21 of the study, the rats were sacrificed and their lungs surgically removed. One lung was used for histological analysis and the other lung was used to evaluate gene expression activity.

[22] From the histological analysis, the 083 Patent reported that "daily treatment of bleomycin-treated rats with 50 mg/kg of compound (u) [or nintedanib] showed a consistent, nearly complete reversal of lung fibrosis" (083 Patent, Ex1, 189:7-14, Figure 4). Similarly, the 083 Patent reported that the gene expression analysis demonstrated that nintedanib suppressed levels of fibrotic marker genes (083 Patent, Ex1, 190:6-10).

[23] The 083 Patent suggests that "[b]y reason of their biological properties" the compounds of the invention may be used as a monotherapy or in conjunction with other pharmacologically active compounds (083 Patent, Ex1, 190:13-206:25). It goes on to provide 10 exemplary formulations, including a tablet (Examples F1-F3), capsule (Examples F4 and F9), suppository (Example F5), suspension (Example F6), ampoule (Examples F7-F8), and solution for inhalation (Example 10) (083 Patent, Ex1, 207:16-214:12).

B. *The 267 Patent*

[24] The 267 Patent is entitled “Capsule Pharmaceutical Dosage Form Comprising a Suspension Formulation of an Indolinone Derivative” and is the national phase entry of a PCT patent application. The 267 Patent has a filing date of June 4, 2009, and claims priority from European Patent Application No. 08157748.8, filed on June 6, 2008. The application for the 267 Patent was published on December 10, 2009, and issued to patent on October 31, 2017. It will expire on June 4, 2029.

[25] The 267 Patent describes the invention of the patent as relating to a suspension formulation containing the active substance nintedanib esylate, a capsule containing the formulation, a process for preparing the formulation and capsule, and packaging material for the capsule (267 Patent, Ex2, 1:4-9). More particularly, the invention is described as relating to formulations of nintedanib esylate containing lipid suspensions of the active substance in 1 to 90 wt.% medium chain triglycerides [MCT], 1 to 30 wt.% of hard fat, and 0.1 to 10 wt.% of lecithin (267 Patent, Ex2, 1:11-15).

[26] The 267 Patent describes the “aim of the …invention” as obtaining an oral pharmaceutical dosage form of the active substance that meets chemical stability and bioavailability requirements for the desired dosage range tailored to treatment and obtaining packaging suitable for the product (267 Patent, Ex2, 4:9-13).

[27] The Description refers to the surprising finding that a soft gelatin capsule including a liquid formulation comprising a viscous suspension of nintedanib esylate in MCT, hard fat and

lecithin, meets the "adequate bioavailability requirements for the desired dosage range tailored to treatment" (267 Patent, Ex2, 8:31-9:5).

[28] The 267 Patent states that an advantage of such soft gelatin capsules containing a lipid suspension is that water uptake into the formulation is very unlikely and the present system is primarily in the capsule shell such that it does not further affect the chemical stability of the active substance (267 Patent, Ex2, 9:7-8, 15-16). The 267 Patent further states that there is no mass increase or sticking problem for the capsules when stored in tight packaging materials (267 Patent, Ex2, 9:23-25).

[29] The Description provides a list of at least 20 different compounds or mixtures thereof that can serve as suitable carriers or carrier components for the active substance of the preferred embodiment, while stating that the most preferred lipid carrier is MCT (267 Patent, Ex2, 11:12-13). It also goes on to identify at least eight different compounds that can serve as suitable thickeners to be used in a preferred embodiment of the invention, stating that the most preferred thickener is hard fat (267 Patent, Ex2, 11:26).

[30] The Description (267 Patent, Ex2, 12:1-7) describes lecithin as "a common excipient for carrier-systems in soft gelatin capsules" that is used "as a glidant of the highly concentrated suspension during encapsulation" and acts as a surfactant, "which may improve distribution of the formulation-droplets during *in-vitro* dissolution testing as well as *in-vivo* for drug resorption". It may also improve wetting of the active substance crystals. It identifies the

commercial product Topcithin® as a suitable lecithin to be used in the formulation of the invention.

[31] The Description states that for the present invention lecithin up to a certain content is "surprisingly" useful to improve dissolution behaviour of the finished capsules (267 Patent, Ex2, 12:9-10). The 267 Patent provides as Figure 2, a graphic representation of the effect of increasing amounts of lecithin in the formulation on the *in-vitro* dissolution rate.

[32] The 267 Patent describes three carrier systems (lipophilic with one surfactant, lipophilic with two surfactants, and hydrophilic) that were tested in rats for bioavailability. The 267 Patent states that all three carrier systems are suitable options for an oral dosage form of nintedanib esylate (267 Patent, Ex2, 12:33-13:4), but that "for reasons of bioavailability … [the] lipid (lipophilic) suspension formulation comprising nintedanib esilate in MCTs, hard fat and lecithin are preferred" (267 Patent, Ex2, 13:6-10).

[33] The 267 Patent provides 10 examples. Examples 1-3 disclose formulations containing nintedanib esylate in different carrier systems. Examples 4-8 disclose soft gelatin capsules having 50, 100, 125, 150 and 200 mg of nintedanib esylate. Example 9 discloses packaging materials for packaging the soft gelatin capsules of Examples 4 to 8, and Example 10 describes a manufacturing process for the preparation of a lipid formulation containing the nintedanib esylate and a process for encapsulation.

IV. **Issues**

[34] The following issues were identified by the parties as those in dispute for this action:

083 Patent

a) The construction of the 083 Asserted Claims, and specifically the meaning to be given to "treatment or prevention of [IPF]" / "prevention or treatment of [IPF]", and in particular the word "prevention" as used in these phrases, including, as applicable, any permitted alternative construction based on subsection 27(5) of the *Patent Act*, RSC, 1985, c P-4 [*Patent Act*];

b) Whether the making, constructing, using or selling of JAMP Nintedanib in accordance with ANDS Control Number 262177 will directly infringe claims 2, 3 and 4 of the 083 Patent?

c) Whether WO 2004/017948 A2 [WO 948] anticipates the subject-matter defined by the 083 Asserted Claims, contrary to paragraph 28.2 of the *Patent Act*?

d) Whether the 083 Asserted Claims are invalid for double patenting over claims 1, 2, 6 or 7 of Canadian Patent No. 2,495,350 [350 Patent];

e) Whether the use of nintedanib or nintedanib esylate in the prevention or treatment of IPF in human patients could be soundly predicted by the 083 Patent's Canadian filing date of December 21, 2005?

f) Or, in the alternative, if a sound prediction is found, whether the subject-matter defined by the 083 Asserted Claims would have been obvious on the claim date of December 24, 2004 to a PSA, contrary to section 28.3 of the *Patent Act*?

g) If the 083 Asserted Claims are determined to be valid and infringed, in addition to a declaration of infringement (and scope of same), the appropriate remedies including the scope of a permanent injunction.

267 Patent

a) The construction of the 267 Asserted Claims, and specifically:

   i. whether "lecithin" (and its amount) is a non-essential element of each of the 267 Asserted Claims; and

   ii. the extent of any permitted variability in the weight percentages relevant to claim 5.

b) Whether the making, constructing, use or sale of JAMP Nintedanib in accordance with ANDS Control Number 262177 would infringe the 267 Asserted Claims?

c) Whether claim 8 (when read through claims 7, 2, and 1) and claims 22 and 23 (when read through claims 8, 7, 2, and 1) of the 267 Patent would have been obvious on the claim date of June 6, 2008 to a PSA, contrary to section 28.3 of the *Patent Act*?

d) Whether claim 2 of the 267 Patent is broader than the invention made by the named inventors of the 267 Patent?

e) If the 267 Asserted Claims are determined to be valid and infringed, in addition to a declaration for infringement (and the scope of same), the appropriate remedies including the scope of a permanent injunction.

V. **Witnesses**

[35] Seven expert witnesses gave testimony at trial: three experts were called by BI (Dr. Roland Bodmeier, Dr. Allan Myerson, and Dr. Timothy S. Blackwell), and four experts were called by JAMP (Dr. Rampurna Prasad Gullapalli, Dr. Sem Hin Phan, Dr. Robert Strieter, and Dr. Jonathan Steed). The parties agreed to stipulations as to the expertise of all expert witnesses.

A. *BI's Experts*

[36] **Dr. Roland Bodmeier** – Dr. Bodmeier obtained his Ph.D. from the University of Texas at Austin in 1986, specializing in pharmaceutics. He is currently a Professor of Pharmaceutical Technology at the College of Pharmacy Freie Universität Berlin, Germany where he has worked since 1994. Dr. Bodmeier has experience in pharmaceutical formulation, including the formulation of oral dosage forms of poorly water-soluble drugs. His research includes the solubilisation of poorly water-soluble drugs to improve the rate of dissolution and bioavailability. Dr. Bodmeier was admitted as an expert in pharmaceutical formulation, including the design, development, and testing of oral dosage forms (for example, including capsule, tablet, solution and suspension formulations, including formulations of poorly water-soluble drugs).

[37] Dr. Bodmeier provided three expert reports relating to the 267 Patent. His first report opined on claims construction and infringement. The second report opined on the validity of the 267 Patent and responded to the expert report of JAMP's expert, Dr. Gullapalli, on that issue. The third report replied to certain comments made by Dr. Gullapalli on infringement.

[38] Dr. Bodmeier was a knowledgeable witness who provided some helpful testimony during his examination. However, his answers on cross-examination were not always direct or responsive leading to lengthy bouts of cross-examination, which at times were difficult to follow. There were also certain inconsistencies with approaches taken in this proceeding to positions taken in prior litigation in which Dr. Bodmeier was an expert. While he described the inconsistencies as a matter of context, his explanations at times fell short of providing the Court with the necessary understanding of the nuances justifying the distinct opinions given.

[39] **Dr. Allan Myerson** – Dr. Myerson is a Professor in the Department of Chemical Engineering at the Massachusetts Institute of Technology and a registered professional engineer in New York and Ohio. He has over forty years experience in the area of crystallization science and technology, including the study of pharmaceutical salts, crystalline solid forms (polymorphs, pseudopolymorphs), pharmaceutical manufacturing, and industrial applications of crystallization. Dr. Myerson was admitted as an expert in the area of crystallization science and technology, including the study of pharmaceutical salts (including techniques used in salt and polymorph screens), crystalline solid forms (polymorphs, pseudopolymorphs), pharmaceutical manufacturing, and industrial applications of crystallization. Dr. Myerson provided a single expert report, offering opinions on claims construction, obviousness and double patenting

concerning the 083 Patent in response to the expert report of JAMP's expert Dr. Steed and aspects of the expert report of JAMP's expert Dr. Phan.

[40] I found Dr. Myerson to be a very knowledgeable witness who was direct and forthright in his testimony.

[41] **Dr. Timothy S. Blackwell** − Dr. Blackwell is the Chair of the Department of Internal Medicine at the University of Michigan. He obtained his M.D. from the University of Alabama in 1988 and completed post-graduate work in the Division of Pulmonary and Critical Care Medicine at Vanderbilt University. Dr. Blackwell has significant experience treating patients with IPF and has conducted research on pulmonary diseases, including IPF for over twenty-five years. Dr. Blackwell was admitted as an expert in the diagnosis and treatment of IPF and other pulmonary diseases with expertise in preclinical studies (including animal model studies) relating to the pathobiology and/or treatment of IPF and other pulmonary diseases. In addition, he is an expert in cell biology, cancer biology and pulmonary immunology.

[42] Dr. Blackwell provided two expert reports on the 083 Patent. In his first report, he opined on claims construction and infringement. In his second report, he opined on anticipation, obviousness, double patenting and sound prediction in response to the expert reports of JAMP's experts Dr. Phan and Dr. Strieter.

[43] I found Dr. Blackwell to be a knowledgeable and straightforward witness who answered questions fully and fairly, readily conceding points even if against the Plaintiffs' interests. I have relied on his testimony and the cross-examination on his evidence heavily in my decision.

B. *JAMP's Experts*

[44] **Dr. Rampurna Prasad Gullapalli** – Dr. Gullapalli obtained his Ph.D. from the University Health Sciences Centre in Memphis, Tennessee in 1993, specializing in pharmaceutics. He is currently the Vice President of the Pharmaceutical Development Department at Iambic Therapeutics, Inc. in San Diego, California. He has thirty years combined experience working in the soft gels development and manufacturing industry as well as working at various pharmaceutical and biotechnology companies particularly on capsule, tablet, solution and suspension formulation development and product manufacturing. Dr. Gullapalli was admitted as an expert in pharmaceutical formulation with expertise in the design, development, manufacturing and testing of oral dosage forms (including for poorly water-soluble drugs), including capsule, tablet, solution and suspension formulations, and specifically soft gelatin capsule formulations.

[45] Dr. Gullapalli provided two expert reports relating to the 267 Patent. His first report opined on claims construction, obviousness and overbreadth. His second report opined on JAMP Nintedanib and responded to the expert report of Dr. Bodmeier on infringement.

[46] In general, I found Dr. Gullapalli to be a helpful witness who provided useful testimony for the Court. However, there were some aspects of his opinion that appeared to evolve through

testimony, particularly as it related to what he initially perceived as certain differences between lecithin and polyglyceryl-3 dioleate [PG3D], which raised some uncertainty as to the strength of those aspects of his opinion. There were also some positions on prior art which seemed to be inconsistent as between his opinions on substitutability and obviousness. However, as it is my view that the issue of infringement of the 267 Patent is dispositive of the issues on that patent, and turns largely on a reading of the 267 Patent itself, these concerns with Dr. Gullapalli's evidence were not significant to my overall findings.

[47] **Dr. Sem Hin Phan** – Dr. Phan obtained a Ph.D. in Biological Chemistry and M.D. from Indiana University in 1975 and 1976, respectively. He is currently a Professor in the Department of Pathology at the University of Michigan Medical School where he has worked since 1980. He has over forty years research experience in the areas of tissue injury, repair, remodeling and fibrosis. Dr. Phan was admitted as an expert in biochemistry, pathology and immunology, including expertise in lung immunopathology, tissue injury, repair, remodeling and fibrosis (including lung/pulmonary fibrosis) and pre-clinical models of lung/pulmonary fibrosis (including animal model studies) relating to the pathobiology and/or treatment of lung/pulmonary diseases (including IPF). In addition, he is an expert in cell biology and pulmonary immunology. Dr. Phan provided a single expert report on the 083 Patent, opining on claims construction, obviousness, anticipation, and double patenting.

[48] I found Dr. Phan to be a straightforward and direct witness whose evidence on scientific matters was helpful to the Court. Dr. Phan applied his personal knowledge to the reading of the

083 Patent and the prior art, but at times seemed to not fully consider the language used in the patents in context as will be discussed further below.

[49] **Dr. Robert Strieter** – Dr. Strieter is a physician-scientist (pulmonary and critical care) and currently acts as a consultant to early drug development companies/organizations with a focus on therapies for pulmonary diseases. He obtained his M.D. from Michigan State University in 1983. Prior to his consulting work, Dr. Strieter was a Professor in the Department of Internal Medicine and Division of Pulmonary and Critical Care at the University of Michigan, a Professor and Chief of the Division of Pulmonary and Critical Care Medicine at the University of California-Los Angeles, a Professor and Chairman of the Department of Medicine at the University of Virginia, and the Global Head of Translational Medicine for Respiratory Diseases at Novartis. He has extensive experience researching and treating interstitial lung diseases, including IPF.

[50] Dr. Strieter was admitted as an expert in lung/pulmonary diseases (including lung/pulmonary fibrosis), including the diagnosis and treatment of IPF and other lung/pulmonary diseases. In addition, he has expertise in pre-clinical models of lung/pulmonary fibrosis (including animal model studies) relating to the pathobiology and/or treatment of lung/pulmonary diseases (including IPF), and early drug development (preclinical and translational medicine) of medicinal candidates for the treatment of lung/pulmonary diseases. He was also admitted as an expert in cell biology, cancer biology and pulmonary immunology. In his expert report, Dr. Strieter provided opinions on claims construction and the utility of the 083 Patent.

[51] There is no doubt that Dr. Strieter is a very knowledgeable witness who had valuable insights to provide to the Court arising from his clinical experience with IPF and the treatment of patients with IPF, as well as his experience working as the head of a drug development team. He provided his evidence from his personal standpoint, which in some instances imparted a perspective that went beyond that of the unimaginative skilled technician who is the PSA. I comment on those aspects further below.

[52] **Dr. Jonathan Steed** – Dr. Steed is a Professor of Chemistry at Durham University in the United Kingdom. Over the past thirty years, his research has focussed on crystallography, crystallization, solid-state chemistry, coordination chemistry and intermolecular interactions in solids, with a particular focus on pharmaceutical solids, co-crystals and hydrates. Dr. Steed was admitted as an expert in synthetic methods, crystallography, crystallization, solid-state chemistry, coordination chemistry and intermolecular interactions in solids, supramolecular gels and crystalline solids (including polymorphs and pseudopolymorphs), particularly pharmaceutical solids, co-crystals and hydrates and techniques used in salt and polymorph screens. Dr. Steed provided a single expert report on the 083 Patent, opining on certain mandate questions relating to claims construction, obviousness, and double patenting.

[53] I found Dr. Steed to be a forthright witness. While his direct experience with salt screens was limited, I found his knowledge of the areas within his mandate to be appropriate and his evidence helpful.

C. *Fact Witnesses*

[54] Two fact witnesses gave evidence at trial on behalf of the Plaintiffs.

[55] The first fact witness, Dr. John Edward Park [Park], is one of the inventors of the 083 Patent. He is a "Highly Distinguished Research Fellow" in the Department of Cancer Immunology and Immune Modulation at Boehringer Ingelheim Pharma GmbH & Co. KG, a member of the Boehringer Ingelheim group of companies. Dr. Park was the former Senior Principal Scientist and Laboratory Head for the cross-therapeutic development work on nintedanib (then called BIBF1120), which compound was initially tested in the company's Cancer Research department. Dr. Park testified on the pre-clinical development work on nintedanib for IPF.

[56] The second fact witness, Dr. Abhya Gupta [Gupta] is an "Associate Head of Medicine of Therapeutic Area Inflammation" at BII. Dr. Gupta testified on the clinical development of nintedanib for IPF, including the design of a Phase II clinical program and on external expert meetings, which took place as a means to evaluate nintedanib as a drug candidate of interest.

[57] As discussed further below, the evidence of Dr. Park and Dr. Gupta was relevant to the issue of sound prediction of utility and in particular, consideration of the factual basis and sound line of reasoning for the prediction of nintedanib's use in the prevention or treatment of IPF.

VI. **Claims Construction**

A. *Legal Principles*

[58] I recently summarized the principles of claims construction in *Takeda Canada Inc v Apotex Inc*, 2024 FC 106 at paragraphs 69-74 [*Takeda*]. These principles, which are equally applicable here, are repeated as follows.

[59] The first task for the Court in a patent infringement action is to construe the claims in issue. Claims construction is not a results-oriented exercise. Rather, the claims are to receive one and the same interpretation for all purposes: *Whirlpool Corp v Camco Inc*, 2000 SCC 67 [*Whirlpool*] at para 49(b).

[60] The focus of claims construction is on the language of the claims: *Free World Trust v Électro Santé Inc*, 2000 SCC 66 [*Free World Trust*] at para 39. The specification describes the invention so that a PSA can understand what the invention is and, when the patent expires, put it into practice, but it is the claims that carve out the boundaries of the proprietary right granted by the patent: *Free World Trust* at para 33; *Merck & Co, Inc v Pharmascience Inc*, 2010 FC 510 at para 44.

[61] Purposive construction involves the Court trying to understand the inventor's objective intention and the particular words or phrases in the claims that describe what the inventor considered to be the essential elements of the invention: *Whirlpool* at para 45; *Free World Trust* at para 31(e); *Biogen Canada Inc v Pharmascience Inc*, 2022 FCA 143 [*Biogen*] para 74. The interpretative task of the Court is two-fold: to separate and distinguish between the essential and

non-essential elements so that legal protection is given only to the essential elements of the invention claimed (*Free World Trust* at paras 15 and 31(e); *Tearlab Corporation v I-MED Pharma Inc*, 2019 FCA 179 [*Tearlab*] at para 31); and, to ascertain what is meant and encompassed by the essential elements so that the invention as claimed is properly characterized (*Biogen* at para 74).

[62] To ascertain the inventor's objective intention, however, purposive construction involves looking at the words of the claims in context, which includes the claims individually and as a whole, and considering their purpose, as well as, the patent's description: *Biogen* at paras 71-72; *Whirlpool* at para 49(e). Although the entire patent must be considered, the disclosure should not be used "to enlarge or contract the scope of the claims as written and … understood": *Whirlpool* at para 52; *Free World Trust* at para 32. Adherence to the claim language allows the claims to be read in a way in which the inventor is presumed to have intended, thereby promoting fairness and predictability: *Biogen* at para 72; *Free World Trust* at paras 31(a), (b) and 41.

[63] The objective of the analysis is to interpret and respect the inventor's objective intention as manifested in the words of the claims used, being neither benevolent nor harsh, but rather seeking a construction that is reasonable and fair to both the patentee and the public: *Biogen* at para 73; *Consolboard Inc v MacMillan Bloedel (Sask) Ltd*, [1981] 1 SCR 504 at p 520; *Teva Canada Ltd v Pfizer Canada Inc*, 2012 SCC 60 [*Teva*] at para 50; *Tearlab* at para 33. The words of the claims are to be read in an informed way, with a mind willing to understand, and in a way that is sympathetic to accomplishment of the inventor's purpose, expressed or implicit in the text

of the claims: *Free World Trust* at paras 31(c) and 44. However, as highlighted in *Free World Trust* at paragraph 51, "...if the inventor has misspoken or otherwise created an unnecessary or troublesome limitation in the claims, it is a self-inflicted wound. The public is entitled to rely on the words used *provided* the words used are interpreted fairly and knowledgeably." [Emphasis in the original]

[64] Although the claim language must be read through the eyes of a PSA, as equipped with their common general knowledge [CGK] (*Free World Trust* at paras 44-45; *Tearlab* at para 32), a Court is entitled to differ from the construction put forward by either party in arriving at its construction as it is the task of the Court alone to construe the claims as a matter of law: *Biogen* at para 73; *Whirlpool* at para 61. The role of experts are to put the judge in the best position to do so in an informed and knowledgeable way: *Biogen* at para 73; *Whirlpool* at para 57.

[65] I will thus characterize the PSA of the 083 Patent and the PSA of the 267 Patent and their CGK before construing the claims of the patents.

B. *The 083 Patent*

(1) PSA and CGK

[66] The PSA is a hypothetical person to whom the patent is addressed. They are deemed to be unimaginative and uninventive, but at the same time to have an ordinary level of competence and knowledge, incidental to the field of the patent and to be reasonably diligent in keeping up with advances: *AstraZeneca Canada Inc v Apotex Inc*, 2014 FC 638 at para 51, aff'd 2015 FCA 158, rev'd on other grounds 2017 SCC 36. They have a mind willing to understand the

specification put before them and is trying to achieve success, rather than look for difficulties or seek failure: *Abbott Laboratories v Canada (Minister of Health)*, 2005 FC 1332 at para 43, aff'd 2007 FCA 153, citing to *Free World Trust* at para 44. This may be a single individual or a team of individuals representing different disciplines, depending on the nature of the invention. It is the person, or team of individuals, that would work the patent in a real sense: *Takeda* at para 76; *Alcon Canada Inc v Cobalt Pharmaceuticals Company*, 2014 FC 462 at para 37, aff'd 2015 FCA 191, 2015 FCA 192.

[67] The parties agree that the PSA of the 083 Patent would include a physician who treats IPF patients, such as a pulmonologist/respirologist. In addition, I agree with JAMP that the 083 Patent, which includes details on the synthesis of the compounds of the invention, their biological activity, genetic testing and analysis, formulation, pharmaceutical use and administration also includes teachings directed to other members of the drug development team, such as a chemist, biologist, molecular biologist, and formulator. Thus, although it is the perspective of the pulmonologist/respirologist on which the parties focus for the issues at play, a multi-disciplinary team would form the PSA of the 083 Patent.

[68] To properly equip myself for the claims construction exercise, I must also consider the CGK of the PSA of the 083 Patent as of June 29, 2006. CGK is the knowledge generally known by the PSA at the relevant date; however, it does not include all information in the public domain: *Apotex Inc v Sanofi-Synthelabo Canada Inc*, 2008 SCC 61 [*Sanofi*] at para 37; *Bell Helicopter Textron Canada Limitée v Eurocopter, société par actions simplifiée*, 2013 FCA 219 [*Eurocopter*] at paras 64-65.

[69] The evidence from the experts was consistent that the CGK of the PSA of the 083 Patent included at least the knowledge that:

a) IPF was a rare and serious disease of unknown etiology that was without an effective treatment (Blackwell XC, TT7, 1269:14-22, 1270:5-8; Strieter Ex25 para 69; Phan, XX, TT4, 810:17-811:3).

b) There were no accepted means to identify individuals with susceptibility of developing the disease and no biomarkers for the disease had been identified (Strieter, XX, TT5, 990:1-12; Phan, XX, TT4, 810:6-9, 811:21-812:4).

c) IPF was a progressive disease in which fibrosis (scarring) in the lungs worsened over time, both at existing sites in the interstitium and in additional areas of the lung parenchyma where fibrosis was not yet evident (Blackwell, Ex37, para 97; Strieter, Ex25, para 68).

d) IPF typically was not diagnosed until after onset of symptoms and the disease had progressed. In rare circumstances if an individual had a chest x-ray that showed the development of fibrosis, an IPF patient could be identified at an earlier stage (Strieter XX, TT5, 1038:17-1039:14; Blackwell, XC, TT7, 1275:21-1276:11).

e) As a practical matter, while physicians attempted to treat IPF, the treatments did not improve patient outcomes (Blackwell, XX, TT7, 1346:3-7).

f) Multiple strategies for treating IPF were under investigation with uncertain prospects of success. Those treating patients with IPF were desperate to find therapeutic solutions (Phan XX, TT4, 777:20-781:8, Ex20, para 332; Strieter XX, TT5, 973:1-12, 19-23, 974:8-12; Blackwell, Ex38, paras 228-229).

(2) Claims Construction 083 Patent

[70] The 083 Asserted Claims include claims 1-6 of the 083 Patent, which read as follows:

> 1. Compound [nintedanib] for use in the prevention or treatment of idiopathic pulmonary fibrosis.
>
> 2. [Nintedanib esylate], for use in the prevention or treatment of idiopathic pulmonary fibrosis.
>
> 3. A pharmaceutical composition for use in the prevention or treatment of idiopathic pulmonary fibrosis, comprising [nintedanib esylate] and a pharmaceutically acceptable carrier.
>
> 4. The pharmaceutical composition according to claim 3, in the form of a capsule.
>
> 5. Use of compound [nintedanib] in the treatment or prevention of idiopathic pulmonary fibrosis.
>
> 6. Use of [nintedanib esylate] in the treatment or prevention of idiopathic pulmonary fibrosis.

[71] The parties do not dispute, and I agree, that all elements of the 083 Asserted Claims are essential. The PSA's understanding of IPF is as set out earlier in the CGK. The only outstanding construction issue between the parties is the interpretation to be given to the words "prevention or treatment of [IPF]"/"treatment or prevention of [IPF]" as used in the claims, and in particular the word "prevention" as used in that phrase.

[72] JAMP asserts that “prevention” should be read as distinct from “treatment” and that it should be given its ordinary medical meaning, which is to hinder or stop IPF from arising.

[73] BI argues, and I agree, that “prevention or treatment”/“treatment or prevention” should be read together as synonymous terms whose interpretation is linked to the pathogenesis of IPF. It relies on the evidence of Dr. Blackwell who testified to the PSA’s understanding that the therapeutic goal relating to IPF was to slow disease progression by preventing or slowing the worsening/extension of fibrosis (Blackwell, Ex37, paras 97-98). If the clinician caught the disease early on, this could result in prevention of symptoms. Otherwise, it would result in treating those that already had symptoms by stabilizing the symptoms and avoiding disease progression (Blackwell XC, TT7, 1275:18-1276:18).

[74] There is no definition of either “prevention” or “treatment” provided in the 083 Patent. As highlighted by Dr. Blackwell, “prevention” is not used on its own in the patent, but is only used as part of the phrase “prevention or treatment” or “treatment or prevention” (Blackwell, Ex37, para 92).

[75] Both Dr. Phan and Dr. Strieter agreed that “prevention” could mean different things in different contexts. The nature of the disease is relevant. While primary prevention seeks to block disease from forming in the first place, secondary prevention is aimed at preventing a disease from progressing further (Strieter XX, TT5, 1012:21-25; Phan XX, TT4, 869:26-870:14).

[76] There is no mention of screening for IPF biomarkers in the 083 Patent and all experts agreed that in 2006, the PSA knew that primary prevention of IPF was not possible as there were no known biomarkers for the disease or means for identifying individuals that were susceptible to developing IPF (Strieter XC, TT5, 911:22-912:5, 990:1-12; Phan XX, TT4, 811:21-812:4).

[77] While Dr. Blackwell acknowledged that before 2006 the scientific literature suggested that up to 20 percent of cases of IPF might be familial and that some research was ongoing to investigate the possibility of genetic biomarkers (Blackwell XX, TT7, 1431:3-1432:22), he noted that this research was a long way off and that even today patients believed to have a familial genetic predisposition to IPF are not treated on the basis of this predisposition (Blackwell XR, TT7, 1445:5-17).

[78] There was also no dispute amongst the experts that the PSA would not view true prophylactic use of nintedanib as a practical option due to the rarity of IPF, the side effects of drug use, cost, and potential long-term risks (Blackwell XX, TT7, 1423:24-1424:14, 1427:19-1428:23; Phan XX, TT4, 810:22-811:10; Strieter XC, TT5, 911:22-912:5).

[79] As admitted by Dr. Phan, an interpretation of the phrase “prevention or treatment” as meaning secondary prevention would be consistent with the use of “or” between the words (Phan XX, TT4, 871:23-27). It is also consistent with the use of the words in an interchangeable order. The use of “or” in the phrase, instead of “and” in my view also suggests a more synonymous reading rather than one of additive, distinct terms.

[80] It also aligns with the design of the preventative regimen of the bleomycin-induced rat model, which administered compound immediately after initial exposure to bleomycin and for 21 days thereafter, rather than before exposure to bleomycin at all (Strieter, XX, TT5, 991:21-992:4).

[81] When considering the claims of the patent at issue in *Apotex Inc v Wellcome Foundation Ltd*, 2002 SCC 77 [*Wellcome Foundation*], the Supreme Court of Canada [SCC] found that there was not always a bright line distinction between prevention and treatment, and that prophylaxis could mean preventing development of the signs and symptoms associated with the disease, rather than the disease itself (at para 88).

[82] Using a mind willing to understand and appreciating the CGK of the PSA as at June 29, 2006 as it relates to IPF, in my view the PSA would understand "prevention" to mean secondary prevention – that is, prevention of the progression of the disease state and the development of symptoms arising from IPF. This meaning is closely associated with the meaning of "treatment" which would be understood to be referring to treating the existing disease state and stalling further progression after symptoms have already manifested.

[83] Given my findings on this construction issue, I need not go on to consider subsection 27(5) of the *Patent Act*.

C. *The 267 Patent*

(1) PSA and CGK

[84] The parties agree that the PSA of the 267 Patent includes a pharmaceutical formulator, with a Ph.D. in pharmaceutical sciences, or a related field, and 1-2 years of experience formulating pharmaceutical compositions including oral dosages forms, or someone with a M.Sc. in pharmaceutical sciences with additional practical experience with formulations. While JAMP asserts that the PSA would also include a full drug development team, it agrees that nothing turns on that debate and that the evidence each side has provided is solely from the perspective of the pharmaceutical formulator. I agree that a pharmaceutical formulator is the relevant PSA for the 267 Patent.

[85] The relevant date for the construction of the claims of the 267 Patent is December 10, 2009. As of this date, the CGK of the PSA included knowledge of at least:

a) The principal objectives of dosage form design, including how to profile the active ingredient and evaluate information about its physical and chemical properties, including solubility, dissolution rate, particle size, pK_a, polymorphism, chemical and physical stability, salt formation, and wettability (Gullapalli, Ex9 paras 45-55, 57-71; Bodmeier, Ex8 paras 43-46; Ex15 paras 61-62, 67-70, 72-75)

b) How the active ingredient interacts with other excipients in the formulation and how to choose formulation excipients, including the role of various excipients such as diluents/fillers, binders, lubricants, glidants, surfactants, disintegrants, and

colorants, and more specifically the role of lecithin, MCT, and hard fat (Gullapalli, Ex9 paras 72-82, 87-92, 94-99; Bodmeier Ex8 paras 47-56; Bodmeier Ex15 paras 76, 78-81)

c) How to test pharmaceutical formulations and to conduct excipient compatibility studies, stability testing, and dissolution testing (Gullapalli Ex9 paras 103-108; Bodmeier Ex15 paras 83-85)

d) Different dosage form designs, including tablets, hard gelatin capsules, soft gelatin capsules and oral suspensions, and the typical excipients used with each type of dosage form (Gullapalli, Ex9 paras 126, 132-144, 149, 154-157, 162-165; Bodmeier Ex8 paras 33-39; Bodmeier Ex15 paras 95-96, 101, 104)

e) How to manufacture hard gelatin capsules and soft gelatin capsules (Gullapalli, Ex9 paras 157-161; 166-169; Bodmeier, Ex15 para 104) and how to package pharmaceutical products in glass or plastic containers and in blister packaging (Gullapalli, Ex9 paras 174-183)

(2) Claims Construction of the 267 Patent

[86] The relevant claims of the 267 Patent are set out below, with the claims in square brackets being those that are not directly asserted:

> [1. Formulation of the active substance [nintedanib esylate] which comprises a lipid suspension of the active substance in 1 to 90 wt.% of medium chain triglycerides, 1 to 30 wt.% of hard fat and 0.1 to 10 wt.% of lecithin.]

[2. Formulation according to claim 1, which comprises a lipid suspension of the active substance in 10 to 70 wt.% of medium chain triglycerides, 10 to 30 wt.% of hard fat and 0.25 to 2.5 wt.% of lecithin.]

[3. Formulation according to claim 2 selected from the group consisting of Formulation A, B or C:]

Formulation	**A**	**B**	**C**
Ingredients	wt.%		
Active Substance	43.48	43.48	43.48
Triglycerides, Medium-Chain	28.70	37.38	38.045
Hard fat	27.39	18.26	18.26
Lecithin	0.43	0.43	0.215

[5. Formation [sic] according to claim 3, which is Formulation B.]

[7. Capsule comprising a capsule shell and a capsule formulation, wherein the capsule formulation comprises the formulation in accordance with claim [2 or 5].]

8. Capsule according to claim 7, wherein the capsule is a soft gelatin capsule.

22. Glass container or flexible/hard plastic container suitable for the packaging of capsules, containing one or more capsules according to [claim 8].

23. Plastic blister, optionally with an over-packaging of aluminum, or aluminum blister suitable for the packaging of capsules, containing one or more capsules according to [claim 8].

[87] There are four claim combinations at issue in respect of the 267 Patent, which can be recited as follows:

a) Combination #1: Claim 8, when read through claim 7, through claim 5, through claim 3, through claim 2, through claim 1.

> A soft gelatin capsule comprising a capsule shell and a capsule formulation, wherein the capsule formulation comprises a formulation

of [nintedanib esylate] which comprises a lipid suspension consisting of Formulation B: 43.48 wt.% of [nintedanib esylate], 37.83 wt.% of medium chain triglycerides, 18.26 wt.% of hard fat, and 0.43 wt.% of lecithin.

b) Claim Combination #2: Claim 22 or claim 23 when read through Claim Combination #1.

Glass container or flexible/hard plastic container suitable for the packaging of capsules, containing one or more capsules of Combination #1.

Plastic blister, optionally with an over-packaging of aluminum, or aluminum blister suitable for the packaging of capsules, containing one or more capsules according to Combination #1.

c) Claim Combination #3: Claim 8, when read through claim 7, through claim 2, through claim 1.

A soft gelatin capsule comprising a capsule shell and a capsule formulation, wherein the capsule formulation comprises a formulation of [nintedanib esylate] which comprises a lipid suspension of [nintedanib esylate] in 10 to 70 wt.% of medium chain triglycerides, 10 to 30 wt.% of hard fat, and 0.25 to 2.5 wt.% of lecithin.

d) Claim Combination #4: Claim 22 or claim 23 when read through Claim Combination #3.

Glass container or flexible/hard plastic container suitable for the packaging of capsules, containing one or more capsules of Combination #3.

Plastic blister, optionally with an over-packaging of aluminum, or aluminum blister suitable for the packaging of capsules, containing one or more capsules according to Combination #3.

[88] As set out earlier, there are two claim construction issues relating to the asserted claim combinations. First, the parties disagree as to whether lecithin (and its amount) is a non-essential element of each of the 267 Asserted Claims. Second, the parties disagree as to the extent of any permitted variability in the weight percentages relating to Formulation B of claim 3 on which claim 5 depends.

(a) *Is lecithin (and its amount) a non-essential element*

[89] Claim elements are presumed to be essential. A party alleging otherwise bears the onus of establishing non-essentiality: *Mediatube Corp v Northvu Inc*, 2017 FC 6 at para 33; *Western Oilfield Equipment Rentals Ltd v M-I LLC*, 2021 FCA 24 [*Western Oilfield*] at para 41.

[90] The SCC in *Free World Trust* at paragraph 55, set out a two-part test for determining whether a claim element is non-essential based on the analysis and questions posed by Hoffman J. in *Improver Corp v Remington*, [1990] FSR 181 [*Improver*]:

> It would be unfair to allow a patent monopoly to be breached with impunity by a copycat device that simply switched bells and whistles, to escape the literal claims of the patent. Thus the elements of the invention are identified as either essential elements (where substitution of another element or omission takes the device outside the monopoly), or non-essential elements (where substitution or omission is not necessarily fatal to an allegation of infringement). For an element to be considered non-essential and thus substitutable, it must be shown either (i) that on a purposive construction of the words of the claim it was clearly not intended to be essential, or (ii) that at the date of publication of the patent, the skilled addressees would have appreciated that a particular element could be substituted without affecting the working of the invention, i.e., had the skilled worker at that time been told of both the element specified in the claim and the variant and "asked whether the variant would obviously work in the same way", the answer would be yes: *Improver Corp. v. Remington, supra,* at p. 192. In this context, I think "work in the same way" should be

> taken for our purposes as meaning that the variant (or component) would perform substantially the same function in substantially the same way to obtain substantially the same result. In *Improver Corp. v. Remington*, Hoffmann J. attempted to reduce the essence of the *Catnic* analysis to a series of concise questions, at p. 182:
>
> > (i) Does the variant have a material effect upon the way the invention works? If yes, the variant is outside the claim. If no: –
> >
> > (ii) Would this (i.e.: that the variant had no material effect) have been obvious at the date of publication of the patent to a reader skilled in the art? If no, the variant is outside the claim. If yes: –
> >
> > (iii) Would the reader skilled in the art nevertheless have understood from the language of the claim that the patentee intended that strict compliance with the primary meaning was an essential requirement of the invention? If yes, the variant is outside the claim.

[91] Although the two part test adopted by the SCC uses the words "either" / "or", to be consistent with the *Improver* questions and the overall objectives of conducting a purposive construction otherwise taught by the SCC in *Free World Trust*, the test has been interpreted by this Court as one that should be read conjunctively. As noted by Justice Locke, then of this Court, in *Shire Canada Inc v Apotex Inc*, 2016 FC 382 [*Shire*] at paragraphs 135-138:

> [135] A careful reader will note that the series of three questions from I*mprover* which is quoted by the SCC does not appear to be entirely consistent with the two-part analysis earlier in the paragraph for determining whether or not an element is essential. The first part of the SCC's characterization of the analysis ("on a purposive construction of the words of the claim [the element] was clearly not intended to be essential") corresponds roughly to the third question in *Improver*. The second part of the SCC's characterization ("at the date of publication of the patent, the skilled addressees would have appreciated that a particular element could be substituted without affecting the working of the invention") corresponds roughly to the first and second questions in *Improver*. However, under the *Improver* test, the defendant need only be successful on one of the questions. In order for the

> patentee to establish that a claim element is not essential, it must succeed on all three questions. On the other hand, the SCC's characterization of the analysis appears to indicate that the defendant must be successful on both parts of the analysis, and that the patentee can establish that a claim element is not essential by succeeding on just one part.
>
> [136] It seems unlikely that the SCC intended this apparent difference. Its decision does not acknowledge any inconsistency between the *Improver* questions and its own test for determining essentiality. Nor does the SCC suggest any disapproval of the *Improver* questions. In fact, the SCC clearly relies in *Improver*.
>
> [137] In my view, the SCC likely intended that, in order for a patentee to establish that a claim element is non-essential, it must show both (i) that on a purposive construction of the words of the claim it was clearly not intended to be essential, and (ii) that at the date of publication of the patent, the skilled addressees would have appreciated that a particular element could be substituted without affecting the working of the invention.
>
> [138] It seems clear enough that a claim element should be considered essential if that is what the words of the claim indicate, regardless of whether skilled addressees would have appreciated that a particular element could be substituted without affecting the working of the invention. At para 51 of Free World Trust, the SCC stated as follows:
>
> > The words chosen by the inventor will be read in the sense the inventor is presumed to have intended, and in a way that is sympathetic to accomplishment of the inventor's purpose expressed or implicit in the text of the claims. However, if the inventor has misspoken or otherwise created an unnecessary or troublesome limitation in the claims, it is a self-inflicted wound. The public is entitled to rely on the words used *provided* the words used are interpreted fairly and knowledgeably.
> >
> > [Emphasis in original]

[92] This same view was reiterated by Justice Locke in *Teva Canada Limited v Janssen Inc*, 2018 FC 754 [*Janssen*] at paragraph 71, aff'd 2019 FCA 273, and was also adopted by Justice Zinn in his analysis in *Choueifaty v Canada (Attorney General)*, 2020 FC 837 [*Choueifaty*]. As

emphasized by Justice Zinn at paragraph 39 of *Choueifaty*, *Free World Trust* teaches that the inventor's intention must be considered when conducting a purposive construction:

> [39] As Justice Locke, then of this Court, noted in *Shire Canada Inc v Apotex Inc*, 2016 FC 382 at paras 134-143, in order "to establish that a claim element is non-essential, it must show both (i) that on a purposive construction of the words of the claim it was clearly not intended to be essential, and (ii) that at the date of publication of the patent, the skilled addressees would have appreciated that a particular element could be substituted without affecting the working of the invention" [emphasis in original]. The problem-solution approach to claims construction focuses only on the second aspect above, it fails to respond, as taught in *Free World Trust*, to the issue of the inventor's intention. Regarding the inventor's intention, para 51 of *Free World Trust* states: "The words chosen by the inventor will be read in the sense the inventor is presumed to have intended, and in a way that is sympathetic to accomplishment of the inventor's purpose expressed or implicit in the text of the claims."

[93] Although not dealt with directly, Justice Gauthier, when also of this Court, similarly rejected the notion that the third question of the *Improver* test could be irrelevant to the Court's analysis. As stated in *Bauer Hockey Corp, v Easton Sports Canada Inc*, 2010 FC 361 at paragraph 144, aff'd 2011 FCA 83:

> [144] In coming to this conclusion, the Court has considered Bauer's counsel's arguments based on the Supreme Court Decision in *Free World Trust v. Electro Santé Inc.*, 2000 SCC 66, [2000] 2 S.C.R. 1024, 194 D.L.R. (4th) 232 (*Free World*) at paras. 55-57, but more specifically at para. 57. At best, this passage can be construed as an agreement that there is a presumption created when a posita would know that the elements under review would make no difference at the time of publication. It cannot, in my opinion, be construed as meaning that the Supreme Court found the third question in *Improver Corp. v. Remington Consumer Products Ltd.*, [1990] F.S.R. 181 (Pat. Ct.) to be irrelevant. Even if the Court were to apply such a presumption here it would not change its conclusion on the matter. [Emphasis in the original]

[94] BI argues that I am not bound by judicial comity and horizontal *stare decisis* to follow the *Shire, Janssen and Choueifaty* decisions on this point of law, as there were subsequent decisions of this Court that interpreted the test articulated by the SCC in *Free World Trust* differently. It refers to the decisions in *Allergan Inc v Sandoz Canada Inc*, 2020 FC 1189 [*Allergan*] and *dTechs EPM Ltd v British Columbian Hydro and Power Authority*, 2021 FC 190 [*dTechs*], aff'd 2023 FCA 115 as support for its position. However, I do not view these decisions as responsive to this issue.

[95] In *Allergan*, Chief Justice Crampton expressly declined to comment on the interpretation given by the Court in *Shire*, noting only that the Court in that case had suggested that the SCC likely intended the *Free World Trust* test to be conjunctive:

> [48] I am aware that in *Shire Canada Inc v Apotex Inc*, 2016 FC 382 at para 137, the Court suggested that the Supreme Court of Canada likely intended the disjunctive test described in (i) and (ii) of the immediately preceding paragraph to be conjunctive. However, given the findings that I have made in Part VI.A.(4) below with respect to the two prongs of the test for essentiality, nothing in this decision turns on the issue of whether that test is disjunctive or conjunctive.

[96] In *dTechs*, Justice Fothergill considered whether the claim element "notifying the utility" could be omitted or substituted from the claim at issue, without changing the way in which the claimed process worked. While Justice Fothergill did not specifically comment on both parts of the *Free World Trust* test, his analysis included consideration of the purpose and intent of the inventor with respect to the claims at issue. As stated by Justice Fothergill at paragraph 155:

> [155] BC Hydro contrasts the language of claim 1 with that of claim 21. "Notifying the utility of the identified suspect consumption pattern in the primary line" does not appear in claim 21. Nor does claim 21 contemplate "the utility thereafter

> monitoring characteristics of the plurality of transformers". Mr. Shepherd expressed the view that this demonstrates a difference in the purposes and intentions of the two claims. Reading the 087 Patent as a whole, the purpose and intent of claim 1 is for an entity that is not a utility (a police officer, for example) to perform some of the steps described in the claim, and for a utility to perform other steps.

[97] Although the Federal Court of Appeal [FCA] in *dTechs* (2023 FCA 115) was not tasked with determining if the Federal Court erred in its claim construction analysis, I agree with JAMP that their comments also demonstrate consideration of the first part of the *Free World Trust* test for determining essentiality.

> [82] Although this Court is not tasked with determining if the Federal Court erred in law in its construction of the claims (that distinct ground of appeal was abandoned), there is little doubt that the words "further comprising" as used in claim 4 are meant to add an essential element to the combination of elements found to be essentials in claim 1. The Federal Court held that this construction was in line with the purpose of the invention set out in claim 1, as well as the purpose and advantages of adding the additional step disclosed in claim 4. The Court simply could not re-write the claims as proposed by Mr. LaPlace to capture what he felt would make more sense. The Federal Court rejected Mr. LaPlace's construction on the basis of its own purposive construction of the claims. This was a legal finding that could not be affected by the new evidence and its potential impact on the evidence of Mr. Shepherd.

[98] In my view, neither *Allergan* nor *dTechs* suggest that determination of the essentiality of claim elements should be conducted devoid of consideration of the patent or of the purpose intended by the inventor as expressed in the language of the claims.

[99] Indeed, it is the primacy of the claim language in determining the essential elements that the SCC has stated is the foundation of claims construction: *Eurocopter* at para 96.

[100] In *Free World Trust*, the SCC identifies as an overriding point that "the claim language will on a purposive construction show that some elements of the claimed invention are essential while others are non-essential" (see heading at p. 1053, after paragraph 50). It cautions, with reference to Justice Pratte's comments in *Eli Lilly & Co v O'Hara Manufacturing Ltd* (1989), 1989 CarswellNat 504, 26 CPR (3d) 1 (Fed CA) [*O'Hara*], that "A court must interpret the claims; it cannot redraft them. When an inventor has clearly stated in the claims that he considered a requirement as essential to his invention, a court cannot decide otherwise for the sole reason that he was mistaken." (at para 59). In view of these overarching themes, it would be entirely inconsistent for the Court to find an element of the claim not essential if the language of the claims and a reading of the patent indicates otherwise.

[101] As noted by the Quebec Court of Appeal in its recent decision *Lames Nordik (Usinage Pro-24) v Hamel*, 2023 QCCA 874 [*Hamel*] at paragraph 18:

> [18] …the court must always bear in mind the fundamental principle of forseeability underlying the *Patent Act*, which requires that the determination of essential and non-essential elements should not be made in a manner completely detached from the terms used in the claims, since the monopoly granted to a patentee is limited to what is claimed under the patent. Indeed, while the person who discloses his invention to the public has the right to expect to be granted a monopoly over what he has actually claimed, the public in turn has the right to rely on the monopoly being clearly defined, so that it can reasonably foresee which activities are likely infringe it. This explains, among other things, why courts must confine themselves to interpreting claims – not rewriting them. It would not be fair to allow the patentee to extend the scope of a claim to more or something other than what is claimed.

[102] As stated earlier, claim construction is to be conducted before consideration of the issue of infringement. Thus, the question should not be whether a specific surfactant is equivalent to

lecithin in the formulation: *Eurocopter* at paras 95-97; *Eli Lilly v Apotex*, 2020 FC 814 [*Eli Lilly*] at paras 189-190. While the Court can consider factual elements pertaining to the existence of known variants when tasked with qualifying elements as essential or non-essential, the patentee's intention remains the key factor: *Eli Lilly* at para 191.

[103] Indeed, as noted in *Hamel* at paragraph 19, it is the third *Improver* question alone that goes to construction. The overriding importance of the third *Improver* question to the claim construction exercise was called out by Hoffman J. in his reasons (reproduced below) after formulating the three questions of the *Improver* test:

> …It is worth noting that Lord Diplock's first two questions, although they cannot sensibly be answered without reference to the patent, do not primarily involve questions of construction: whether the variant would make a material difference to the way the invention worked and whether this would have been obvious to the skilled reader are questions of fact. The answers are used to provide the factual background against which the specification must be construed. It is the third question which raises the question of construction and Lord Diplock's formulation makes it clear that on this question the answers to the first two questions are not conclusive. Even a purposive construction of the language of the patent may lead to the conclusion that although the variant made no material difference and this would have been obvious at the time, the patentee for some reason was confining his claim to the primary meaning and excluding the variant. If this were not the case, there would be no point in asking the third question at all.
>
> [emphasis from *Hamel* removed]

[104] In this case, it is clear from the language of the 267 Patent claims that lecithin was intended to be an essential element of the claimed invention. There are no other options provided in the claims, or in the patent for the glidant/solubilizing agent that is lecithin. As was acknowledged by Dr. Bodmeier, the 267 Patent states that lipid suspension formulations of the monoethanesulfonate salt of nintedanib in MCT, hard fat and lecithin are preferred to other

formulations “for reasons of bioavailability” (Bodmeier, Ex8, para 80; Gullapalli, Ex10, para 107; Bodmeier XX, TT1, 220:16-22; 222:23-223:1). The inventors provide a specific study outlining the impact and importance of lecithin on the formulation (Figure 2) (Bodmeier XX, TT1, 220:3-15). As admitted by Dr. Bodmeier, the PSA could understand that the presence of lecithin, even in a small amount, has a significant effect on the dissolution of the finished capsules (Bodmeier, Ex15, para 119). Lecithin is a specific component of each of the formulations covered by the Asserted Claim Combinations (Gullapalli, Ex10, para 107; Bodmeier XX, TT1, 217:1-3). When asked on cross-examination, Dr. Bodmeier agreed that a formulation including lecithin was the solution taught by the patent (Bodmeier XX, TT1, 223:25-224:13).

[105] BI does not dispute this reading of the patent. Indeed, BI conceded in argument that they were not arguing inessentiality based on the first arm of the *Free World Trust* test (third *Improver* question) and that if evaluation of this part of the test was determined to be necessary by the Court that they would not succeed in their argument. They provided no evidence from their expert on the determination of the essential elements of the claims, and specifically instructed Dr. Bodmeier not to consider whether any of the elements were essential or inessential (Bodmeier, Ex15, para 135).

[106] Even on the second arm of the *Free World Trust* test, I am not satisfied based on the evidence before me that the PSA would have considered lecithin to be readily substitutable with another surfactant, such as PG3D, as of the date of publication of the patent.

[107] As explained by Dr. Gullapalli, lecithin is a zwitterionic surfactant that serves many roles, acting as a dispersing, emulsifying, stabilizing and solubilizing agent (Gullapalli, XC, TT2, 261:20-24). It also acts as a viscosity modifier and improves flow properties (Gullapalli, XX, TT2, 352:18-353:3).

[108] It is not simply a "plug in play" scenario, where properties such as chemical stability and bioavailability can be assumed (Gullapalli, Ex10, para 104; Gullapalli, XC, TT2, 271:15-272:2). There was no evidence on the chemical stability or bioavailability of a formulation with any variant, including PG3D, to be able to conclude that it would perform substantially the same function in substantially the same way to obtain substantially the same result (Gullapalli, Ex10, paras 102-103). To the contrary, the evidence that was provided suggested that lecithin as an ionic surfactant works by way of electrostatic stabilization while PG3D as a non-ionic surfactant functions by steric stabilization (Bodmeier, Ex14, para 38).

[109] Further, the evidence indicates that the PSA would not even view all types of lecithin to have the appropriate properties to satisfy the claimed formulation let alone a variant that was not lecithin. The evidence of Dr. Bodmeier was that the lecithin of the formulation was limited to a specific type of commercial lecithin, called Topcithin® and that if a formulation contained all of the elements of claim 1 but did not include a lecithin that was similar to Topcithin® then it would fall outside the claims (Bodmeier XX, TT1, 226:1-19). Given the specificity of lecithin contemplated by the patent, the PSA, in my view, would not have expected that lecithin in the claimed formulations could be substituted with another surfactant without affecting the working of the invention.

[110] The suggestion that lecithin is not essential is also inconsistent with positions taken by the patentee with the Canadian patent office during prosecution of the application for the 267 Patent.

[111] As provided by section 53.1 of the *Patent Act*, a "written communication" of the applicant with the Patent Office may be admitted into evidence to rebut a representation made by a patentee in an action as to the construction of a claim in a patent. As part of its evidence, JAMP tendered documents from the prosecution history of the 267 Patent in which BII argued that the claims of the 267 Patent were not anticipated or rendered obvious by prior art because that prior art did not have the composition of the claimed formulation, including the component lecithin. It further referred to Figure 2 of the 267 Patent and highlighted the advantages of having a certain amount of lecithin to improve the dissolution and bioavailability of the active substance in its response to an argument on obviousness. The response noted that "[t]he cited art has not disclosed or suggested the effect of lecithin in lipid suspensions, and not in relation to the specific active substance."

[112] While BI argues that this correspondence does not come within section 53.1 as it is not correspondence about the construction of the patent and should be inadmissible, I do not agree that such an asserted restriction applies to section 53.1. The correspondence here comes precisely within that contemplated by section 53.1 and that which has been applied in other cases (*i.e.*, *Eli Lilly* at para 172). It is my view that a fair reading of the correspondence provided indicates that representations were made by the patentee that support the view that lecithin is essential to the formulation claimed.

[113] In my view, BI has not met its burden of establishing that lecithin is a non-essential element of the claims.

(b) *Extent of any permitted variability in the weight percentages relating to Combination #1 (Formulation B)*

[114] As set out above, Formulation B of the 267 Patent claims provides for the components of the formulation in weight percentage amounts that are specified to two decimal places.

[115] BI argues that the PSA would understand that Formulation B allows for minor variability of up to 5% of the specified wt.%, namely 41.31-45.65 wt.% for the active ingredient, 35.94-39.72 wt.% for MCT, and 17.35-19.173 wt.% for hard fat. It relies on the evidence of Dr. Bodmeier that there is permissible variability in the specification for a drug product in its active ingredient and excipients (Bodmeier, Ex8, paras 103-104).

[116] However, this evidence must also be considered alongside a fair reading of the patent and the full claim set. As acknowledged by Dr. Bodmeier, where the inventors wanted to claim ranges for the components of the formulation, they knew how and did so, as for example in claims 1 and 2 of the 267 Patent (Bodmeier XX, TT1, 229:13-230:6).

[117] The inventors did not claim a weight percentage range for Formulation B. Nor did they use the word “about” to qualify the precise weight percentage amounts claimed.

[118] As admitted by Dr. Bodmeier, the PSA would understand that claiming a specific weight percentage, as opposed to a weight percentage range, was intended to communicate greater

precision (Bodmeier XX, TT1, 230:17-232:2). In my view, this is what was done for the formulations claimed in claim 3, of which Formulation B is relevant to claim 5.

[119] I agree with JAMP that a fair reading of the evidence and the patent supports an interpretation of Formulation B as being limited to the precise weight percentage amounts claimed.

VII. **Infringement**

[120] To establish that there is infringement, the Court must be satisfied that JAMP Nintedanib has all essential elements of the asserted claims: *Free World Trust* at para 31(f). The burden of proof lies with the Plaintiffs to establish infringement on a balance of probabilities.

A. *267 Patent*

[121] With respect to the 267 Patent, my findings on the construction issues above are determinative of the issue of infringement. As such, I will deal with this patent first.

[122] As JAMP Nintedanib does not include lecithin in its formulation, it does not include all essential elements of the formulations claimed and accordingly does not infringe the 267 Asserted Claims.

[123] While BI argues that the surfactant PG3D used in JAMP Nintedanib could be substituted for lecithin without affecting the functioning of the formulation, this is an improper equivalence argument when divorced from the assessment of essentiality/inessentiality of claim elements:

Eurocopter at paras 95-97. As stated in *Steelhead LNG (ASLNG) v ARC Resources Ltd*, 2024 FCA 67 at paragraph 83, citing to *Free World Trust* at paragraph 32, "[f]inding a different way to accomplish the benefit of an invention by designing around a patent does not constitute infringement since the protection of the patent 'lies not in the identification of a desirable result but in teaching one particular means to achieve it'".

[124] As there is no lecithin in JAMP Nintedanib, there is no infringement of the 267 Asserted Claims.

[125] Further, JAMP Nintedanib does not include the precise weight percentage amounts for MCT and hard fat that are required for Formulation B, which is relevant to claim 5. [redacted] These weight percentages are not the same as the weight percentages claimed for Formulation B (37.83 wt.% MCT and 18.26 wt.% hard fat).

[126] In view of my earlier finding that there is no accepted variability in the weight percentage amounts for Formulation B, JAMP Nintedanib does not infringe claim 5 or any claim that depends from claim 5 for this additional reason.

[127] My finding of non-infringement of the 267 Patent is dispositive of BI's claim on this patent. At trial, counsel for JAMP submitted that if the Court found that the 267 Patent was not

infringed it need not go on to consider its further defence of invalidity of the 267 Patent. As such, I shall not go on to consider the further invalidity defences raised by JAMP in association with the 267 Patent.

B. *083 Patent*

[128] JAMP has stipulated that the selling, offering to sell, marketing or having others market, distributing or having others distribute, and/or making available for use of JAMP Nintedanib would induce infringement of the 083 Asserted Claims, except for any claims found invalid. The outstanding issue between the parties is whether JAMP's actions would also constitute direct infringement of claims 2, 3 and 4 of the 083 Patent.

[129] JAMP argues that this case closely parallels that in *Hoffmann-La Roche Limited v Sandoz Canada Inc*, 2021 FC 384, involving the drug pirfenidone [*Pirfenidone*]. In *Pirfenidone*, Justice Manson found that direct infringement did not apply as the medicament or pharmaceutical composition was not adapted to support the novel use of the drug in question. Rather, as the claims were in effect directed to the *new* use (*i.e*., new dosing regimen), the activities of the generic were of inducement only. As stated at paragraphs 97, 103, 109 and 110 of *Pirfenidone*:

> [97] Roche's approach seeks a finding of claim *form* over *substance*. In doing so, it obscures the proper approach to claims construction. As discussed above, the claims construction exercise emphasizes a purposive construction. In this case, the 654 and 997 Asserted Claims have been properly construed as use claims, as provided above. Both experts have agreed that the POSITA would understand the dose escalation regimen provided in the 654 Patent. Dr. Kolb testified that an essential element of all of the Asserted Claims of the 654 Patent is using pirfenidone at a first oral daily dosage of 801 mg for seven days, followed by a second oral daily dosage of 1602 mg for a further seven days, followed by a third oral daily dosage of 2403 mg. As further construed above, the 997

Patent is directed at a method of providing pirfenidone therapy to IPF patients having exhibited defined ALT and/or AST elevations. The alleged invention in this case resides in the use of pirfenidone, whether in the context of the 654 or 997 Patent, and not in the manufacture or composition of pirfenidone, a known compound.

[…]

[103] The 654 and 997 Patents do not disclose a composition or other product for use in a medicament that enables the *new* use of pirfenidone in question – the dose escalation regimen. The circumstances of this case are therefore distinguishable from *Janssen Inc v Teva Canada Limited*, 2020 FC 593 [*Janssen*], where the "medicament" referenced in the Swiss-style claims referred to a medicine suitable for the depot formulation of paliperidone palmitate (*Janssen*, above at paras 161-163). The medicament was therefore adapted for administration according to the claimed dosage regimen. In *Hospira Healthcare Corporation v Kennedy Trust for Rheumatology Research*, 2018 FC 259 [*Hospira FC*], the patent in question claimed a combination of elements, leading to efficacy in duration and response (*Hospira FC*, above at paras 150, 155 rev'd on other grounds 2020 FCA 30 [*Hospira FCA*]). The Federal Court distinguished the invention in *Hospira FC* from a dosage regimen leading to increased efficacy on this basis when considering whether the patent in question claimed a method of medical treatment. For the reasons already discussed, there is nothing novel about the manufacture or composition of pirfenidone to treat IPF in this case, but only a new dosing regimen of pirfenidone for use in the treatment of IPF.

[…]

[109] Having purposively construed the Swiss-style and product for use claims as use claims and having identified the essential claim elements, Sandoz will not directly infringe the 654 and 997 Asserted Claims in that it does not use pirfenidone in the treatment of IPF, as identified by the 654 and 997 Asserted Claims

[110] Roche relies on several cases for the proposition that Courts have found direct infringement of product for use and Swiss-style claims (*Hospira FC, Janssen and AB Hassle v Canada (Minister of National Health and Welfare)*, 2001 FCT 1264 [*AB Hassle FC*], aff'd 2002 FCA 421 [*AB Hassle FCA*]). *Hospira FC* and *Janssen* are distinguishable from the current case for the reasons discussed above, where the medicament or pharmaceutical composition in those cases in fact supported the novel use of the drug in question. I do not consider the approach of the Court *in AB*

> *Hassle FC* to be applicable to the current case. As use claims, the Asserted Claims of the 654 and 997 Patents do not support a finding of direct infringement.

[130] BI asserts that evidence that a generic company proposes to make or sell its product for a patented use (even if it is only one use among others) is enough to establish direct infringement. It asserts that the FCA's decision in *Teva Canada Limited v Janssen Inc*, 2023 FCA 68 [*Paliperidone*], which was decided after *Pirfenidone*, is supportive of its position. The claims of the patent at issue in *Paliperidone* related to a pharmaceutical preparation for use in the treatment of a condition and Swiss-type product claims. As stated at paragraphs 77-79 of *Paliperidone:*

> [77] In the context of product claims like those in claims 1 to 16 of the 335 Patent (i.e., claims to a pharmaceutical preparation for use in the treatment of a condition), evidence that a generic company proposes to make or sell its product for the patented use (even if it is only one use among others) is enough to establish direct infringement in an action brought under section 6 of the PMNOC Regulations *(AB Hassle v. Canada (Minister of National Health and Welfare)*, 2001 FCT 1264, 16 C.P.R. (4th) 21 at paras. 6, 33, 35–36, aff'd 2002 FCA 421, leave to appeal to S.C.C. refused, 29533 (27 March 2003) [*AB Hassle*]; *Eli Lilly Canada Inc. v. Apotex Inc.*, 2019 FC 884, 166 C.P.R. (4th) 191 at paras. 24–33).
>
> [78] Similarly, in the context of Swiss-type product claims like those in claims 33 to 48 of the 335 Patent (*i.e.*, claims to the use of a drug for the preparation of a medicament for use in treatment of a condition), evidence that a generic company proposes to make or sell its product for the patented use (even if it is only one use among others) is enough to establish direct infringement in an action brought under section 6 of the PMNOC Regulations (*AB Hassle* (F.C.T.) at paras. 6, 33, 35–36; *Hospira Healthcare Corporation v. Kennedy Trust for Rheumatology Research*, 2018 FC 259 at paras. 152–153, 268–323, aff'd on that ground and rev'd in part in 2020 FCA 30, 316 A.C.W.S. (3d) 537 at paras. 16–18, leave to appeal to S.C.C. refused, 39099 (23 December 2020) [*Hospira*]).

> [79] In some respects, this case is similar to *Hospira*. There, the generic company was found to have both directly infringed and induced infringement of the patent that claimed a product for use in an adjunctive therapy. The generic company produced the component claimed in the patent at issue in that case. Even though the infringing adjunctive therapy was only one of several potential uses mentioned in the generic company's PM, it was found to have directly infringed and induced infringement of the patent at issue in that case. The generic company's conduct in *Hospira* is analogous to that of Teva in the case at bar.

[131] In this case, BI's allegation of direct infringement relates to claims 2, 3 and 4 of the 083 Patent, which are reproduced below:

> 2. [Nintedanib esylate], for use in the prevention or treatment of idiopathic pulmonary fibrosis.
>
> 3. A pharmaceutical composition for use in the prevention or treatment of idiopathic pulmonary fibrosis, comprising [nintedanib esylate] and a pharmaceutically acceptable carrier.
>
> 4. The pharmaceutical composition according to claim 3, in the form of a capsule.

[132] The claims are directed to the medicine (nintedanib esylate) (claim 2), and a pharmaceutical composition of the medicine (claims 3 and 4), for use in the prevention or treatment of IPF.

[133] JAMP Nintedanib contains nintedanib esylate and a pharmaceutically acceptable carrier in soft gelatin capsules (Blackwell, Ex37, para 111). As set out in the Agreed Statement of Facts, if JAMP elects to launch JAMP Nintedanib in Canada, it will be the importer of JAMP Nintedanib into Canada, it will sell JAMP Nintedanib in Canada, and it will market JAMP Nintedanib for the indications contained in the JAMP Nintedanib product monograph, which include IPF.

[134] I agree with BI, the decision in *Paliperidone* is binding and applicable. While JAMP's intended activities will not involve the actual use of nintedanib esylate, or a pharmaceutical composition containing nintedanib esylate, for the prevention or treatment of IPF (Strieter, Ex26, paras 21-23), in my view this is not relevant to claims 2-4, which under *Paliperidone* would be considered as product claims. As the capable, approved and intended use of JAMP Nintedanib includes use of nintedanib esylate for the treatment of IPF, it is my view that JAMP's import, and sale of JAMP Nintedanib is sufficient to constitute direct infringement of claims 2-4 of the 083 Patent.

VIII. **Validity of the 083 Patent**

[135] As set out earlier, JAMP raises the following grounds of invalidity as part of its defence: anticipation, double patenting, lack of sound prediction of utility and in the alternative to sound prediction, obviousness.

A. *Anticipation*

[136] The proposed invention claimed in a patent must be new to be patentable. Subsection 28.2 of the *Patent Act* sets out the requirement for novelty of a patented invention.

[137] The SCC in *Sanofi*, set out two requirements for establishing anticipation: prior disclosure and enablement. For disclosure to be satisfied, the disclosure must include clear direction so that a PSA following it would in every case and without possibility of error be led to the claimed invention; a signpost will not suffice: *Free World Trust* at para 26; *Western Oilfield* at para 82. It is not necessary that "the exact invention" be publicly disclosed. Rather, "the

requirement of prior disclosure means that the prior patent must disclose subject matter which, if performed, would necessarily result in infringement of that patent": *Sanofi* at paras 23 and 25. For enablement, the PSA must have been able to perform the invention: *Sanofi* at para 26.

[138] BI does not contest enablement. Rather, they contest whether the application, WO 948, disclosed the invention as claimed in the 083 Patent. Specifically, whether disclosure in WO 948 of compounds that include nintedanib for use in the treatment of a number of diseases, some of which give rise to lung fibrosis, would be considered by the PSA to encompass treatment of IPF. Further if so, whether WO 948 when practiced in view of its breadth would necessarily infringe the 083 Asserted Claims. As relevant to claims 2, 3, 4 and 6 of the 083 Patent, BI also contests whether WO 948 discloses the esylate salt of nintedanib by referring to a physiologically acceptable salt.

[139] As a starting point, the prior art document is to be given the same type of purposive interpretation as the patent at issue, with its teachings read as a PSA would understand it: *Shire Biochem Inc v Canada (Health)*, 2008 FC 538 at para 65; *Whirlpool* at para 49c; *Eli Lilly Canada Inc v Apotex Inc*, 2007 FC 455 at para 252, aff'd 2008 FCA 44.

[140] The WO 948 application is entitled "Use of Lck Inhibitors for Treatment of Immunologic Diseases". It describes the field of the invention on page 1 as relating to "a method of treating immunologic diseases or pathological conditions involving an immunologic component using a compound selected from compounds (A) to (AL) … and the use of [the] compounds for the manufacture of a pharmaceutical composition for the treatment of said immunologic diseases or

pathological conditions". The application discloses 38 compounds, which comprise the compounds (A) to (AL), one of which is nintedanib (Blackwell, Ex38, SchM, 3:29, 7:10-12, 17-21, para 126; Phan, Ex20, para 222).

[141] WO 948 sets out the context for its teachings in the background to the application. Here, it explains the focus on T-cell mediated immunologic diseases that are functionally regulated by the tyrosine kinase, Lck (Blackwell, Ex38, SchM, 1:22-31; para 120). WO 948 explains that certain autoimmune diseases such as inflammatory diseases are believed to be associated with inappropriate T-cell activation, and exemplifies lung fibrosis as an example (Blackwell, Ex38, SchM, 1:31-2:3; para 121). The application explains that Lck inhibitors offer an approach for treatment of these diseases (Blackwell, Ex38, SchM, 1:29-31, 2:6-9, para 120; Phan, Ex20, paras 65, 223).

[142] In the Summary of the Invention, WO 948 refers to "a clear need for compounds being active as Lck inhibitors in order to treat T-cell mediated diseases, e.g. in the treatment of immunologic diseases or pathological conditions involving an immunologic component" (Blackwell, Ex38, SchM, 3:4-7, para 125; Phan, Ex20, para 224).

[143] In the Detailed Description of the invention, the inventors state how it has been surprisingly found that the compounds of the invention are effective inhibitors of Lck and therefore, especially suitable and effective in the treatment of immunologic diseases or pathological conditions involving an immunologic component (Blackwell, Ex38, SchM, 3:29, 7:10-12, 17-21, para 126; Phan, Ex20, at paras 226-227).

[144] WO 948 defines “immunologic diseases or pathological conditions involving an immunologic component” as including, amongst other diseases, autoimmune diseases, such as inflammatory diseases that have an autoimmune component. It identifies lung fibrosis as a select autoimmune inflammatory disease (Blackwell, Ex38, SchM, 8:9-25; para 127):

> The indication “immunologic diseases or pathological conditions involving an immunologic component” should be understood in a non-limiting manner to comprise
>
> autoimmune diseases, for instance inflammatory diseases having an autoimmune component such as inflammatory diseases selected from
>
> > inflammatory bowel disease (e.g. colitis ulcerosa and Morbus Crohn), rheumatoid arthritis, glomerulonephritis and lung fibrosis,
>
> furthermore, psoriasis, psoriasis arthritis, hypersensitivity reactions of the skin, atherosclerosis, restenosis, asthma, multiple sclerosis and type 1 diabetes,
>
> and indications which need immunosuppressant therapy, for instance prevention or therapy of tissue or organ transplant rejection, e.g. acute or chronic graft-versus-host disease, allograft or xenograft rejection etc. The transplanted organ may be kidney, heart, liver, lung, bone marrow, peripheral blood stem cells, pancreas or islet cells thereof, cornea, small bowl, skin, or heart valve.

[145] WO 948 goes on to refer to a preferred subgroup of the previously mentioned conditions that deserve special mention, which comprises “morbus crohn, lung fibrosis, psoriasis arthritis, hypersensitivity reactions of the skin, graft-versus-host disease (acute and chronic), asthma, multiple sclerosis and type 1 diabetes” (Blackwell, Ex38, SchM, 9:9-12, para 130; Phan, Ex20, para 66).

[146] The claims of WO 948 track the language of the disclosure and claim "[a] method for treating immunologic diseases or pathological conditions involving an immunologic component" with the compounds of the invention, where the "immunologic disease or pathological condition involving an immunologic component" may be selected from "autoimmune diseases, for instance inflammatory diseases having an autoimmune component", which include lung fibrosis (claim 2). Claim 4 depends from claim 2 and specifies "lung fibrosis" as one of the selected "immunologic disease[s] or pathological condition[s] involving an immunologic component". Claims 20, 21 and 23 are "use" claims, with claims 21 and 23 including the same language as claims 2 and 4 as it relates to the specific "immunologic disease or pathological condition involving an immunologic component" (Blackwell, Ex38, paras 134-139).

[147] Dr. Phan asserts that as a matter of CGK, "lung fibrosis" would be understood by the PSA to be a term that captures the collection of diseases or conditions that afflict the lungs, with the hallmark being the formation of fibrotic tissue (Phan, Ex20, paras 26, 101). This would include the most common form, which is IPF (Phan, Ex20, paras 26, 103).

[148] However, the meaning of "lung fibrosis" as used in the 083 Patent must be considered in context. Although Dr. Phan suggested that his understanding of "lung fibrosis" did not change upon reading WO 948, I am not convinced from the approach taken by Dr. Phan in his report and that explained during his examination that he applied a purposive interpretation to the meaning of "lung fibrosis". Indeed, Dr. Phan admitted on cross-examination that he provided his opinion on the meaning of lung fibrosis before he reviewed WO 948, the 083 Patent, and the 350 Patent

(discussed below with reference to the issue of double patenting) and applied this definition to the rest of his mandates (Phan, XX, TT4: 756:23-758:1).

[149] Dr. Blackwell asserts, and I agree, that when read in context, "lung fibrosis", which is a pathological finding, would be understood by the PSA to be referring to lung fibrosis arising from autoimmune diseases believed to be associated with inappropriate T-cell activation (Blackwell, Ex38, paras 117, 122, 128, 131). This is clear from the definition of "immunologic disease or pathological condition involving an immunologic component" that is provided by WO 948 and from reading WO 948 as a whole. Contrary to the assertions of JAMP, I do not consider Dr. Blackwell to be proposing that IPF be carved-out of the PSA's reading of WO 948 (*Alcon Canada Inc v Apotex Inc*, 2014 FC 699). Rather, the approach taken by Dr. Blackwell is a contextual, purposive interpretation of WO 948 based on an understanding that IPF is not an autoimmune or T-cell mediated disease (Blackwell, Ex38, paras 117, 123, 185).

[150] I note that there is no reference to IPF in WO 948. Nor does Dr. Phan suggest that IPF is an autoimmune or T-cell mediated disease (Blackwell, Ex38, para 185). Significantly, both he and Dr. Strieter differentiated IPF from autoimmune lung diseases as separate lung disorders (Phan XX, TT4, 805:9-13, referring to Ex20, Sch9-17, 5), and in the case of Dr. Strieter identified this as an important point of clarification for a clinician looking to treat the patient (Strieter, XX, TT5: 947:7-948: 21).

[151] All experts agreed that by December 2004, various anti-inflammatory agents that were used in the clinic as a treatment for IPF had largely been found to be ineffective and the PSA's

understanding of IPF was shifting away from associating IPF with its inflammatory response (Phan, Ex20, para 151; Strieter, Ex25, para 70; Blackwell, Ex38, paras 123, 225; Blackwell, XX, TT7, 1354:21-26). While there remained an understanding that T-cells might play some role in IPF (Blackwell, XX, TT7, 1386:10-1387:25), the evidence did not support any established view that IPF was a T-cell mediated disease or one that would be regulated by Lck.

[152] In view of the evidence and the focus in WO 948 on Lck and T-cell mediated inhibition, I am unable to conclude that the PSA would necessarily consider the treatment of "lung fibrosis" to be encompassing the treatment of IPF. As stated in *Gilead Science Inc v Canada (Health),* 2013 FC 1270 at paragraph 30: "[i]f there is doubt about what the prior art reference includes, it cannot be taken to meet the definition of anticipation": see also *Bristol-Myers Squibb Canada Co v Teva Canada Limited*, 2016 FC 580 at para 243; aff'd 2017 FCA 76; *Sanofi* at para 21, citing *General Tire & Rubber Co v Firestone Tyre & Rubber Co*, [1972] RPC 457 at 486.

[153] Further, even if I were to accept that the PSA would associate IPF as a form of "lung fibrosis" contemplated by WO 948, the disclosure of "lung fibrosis" as one of the many disease conditions within WO 948 in my view is insufficient to constitute disclosure of the use of nintedanib to treat IPF.

[154] JAMP asserts that prior disclosure of a medicine that can treat an umbrella of diseases can constitute disclosure of the medicine for treating individual diseases within that umbrella, even if the specific disease is not explicitly identified. It relies on *Lundbeck Canada Inc v*

Ratiopharm Inc, 2009 FC 1102 [*Lundbeck*] as support for its position (see also *Apotex Inc v Shire LLC*, 2021 FCA 52 [*Shire*] at para 46).

[155] In *Lundbeck*, the patent at issue related to the drug memantine for use in the treatment of Alzheimer's disease. The anticipatory references indicated that memantine was useful to treat "organic brain syndrome", "organic psychosyndrome" and "dementia". Justice Mactavish (then of this Court) held that while the anticipatory references did not specifically recite the use of memantine for Alzheimer's disease because "organic brain syndrome" was well understood to encompass Alzheimer's disease, the listing satisfied the disclosure requirement: *Lundbeck* at paras 115-120.

[156] JAMP asserts that "lung fibrosis" is used as an umbrella term within WO 948. As IPF was the most common example of a disease or condition leading to "lung fibrosis", it asserts that the PSA reading "lung fibrosis" would understand WO 948 to be disclosing treatment of IPF.

[157] However, I do not consider *Lundbeck* to be on all fours with the present case. In *Lundbeck*, there was a prior association of memantine with Alzheimer's disease as it was already an approved drug for moderate to severe Alzheimer's disease at the time of the decision. There was also no indication as to how many diseases were encompassed within "organic brain syndrome". Further, there was direct evidence within one of the references to Alzheimer's disease being a form of dementia, which was an organic psychosyndrome.

[158] In the present case, there was no prior association of nintedanib with IPF. Further, the experts agreed that "lung fibrosis" is not a homogeneous condition and can vary in severity depending on its cause. While lung fibrosis is characteristic of IPF, not all lung fibrosis is associated with IPF. Rather, it is associated with upwards of 150 different diseases (Phan, XX, TT4, 756:15-22, 761:9-13; Blackwell, Ex38, para 79) and WO 948 discloses 38 different compounds.

[159] As was the case in *Eli Lilly Canada Inc v Apotex Inc*, 2010 FC 1065 at paragraphs 64-76, the present case requires a number of choices to be made to arrive at the invention claimed in the 083 Patent. Thus, even if "lung fibrosis" in WO 948 was understood to include IPF, I do not consider this to provide clear and unmistakable directions to the use of nintedanib for the treatment of IPF.

[160] My finding that the use of nintedanib for the treatment of IPF is not disclosed by WO 948 is sufficient to dispose of JAMP's anticipation defence and I need not go on to consider whether the esylate salt form of nintedanib was disclosed by WO 948.

[161] WO 948 does not anticipate the 083 Asserted Claims.

B. *Double Patenting*

[162] An inventor is only entitled to a single patent for an invention: *Patent Act*, section 36(1); *Whirlpool* at para 63. A patent will be invalid for double patenting if the inventor has already received a patent for the same invention (same invention double patenting) or for an invention

that is not patentably distinct (obviousness-type double patenting): *Hospira Healthcare Corporation v Kennedy Trust for Rheumatology Research*, 2020 FCA 30 [*Hospira*] at para 96.

[163] The analysis as to whether there is double patenting involves a comparison of the claims: *Whirlpool* at para 63; *Sanofi* at para 108; *Eli Lilly Canada Inc v Mylan Pharmaceuticals ULC*, 2016 FCA 119 [*Mylan*] at para 37. In "same invention" double patenting, the claims of the second patent must be identical or coterminous with those of the earlier patent: *Whirlpool* at para 65; *Sanofi* at para 108; *Mylan* at para 27. In "obviousness-type double patenting", the claims of the second patent cannot be an obvious and uninventive extension of the claims of the first patent. For this attack, the analytical approach is similar to an obviousness challenge; however, only the earlier patent can be considered, and any other prior art is only relevant as it relates to the CGK of the PSA: *Mylan* at paras 29, 36.

[164] JAMP asserts that the 083 Asserted Claims are invalid for double-patenting as they are both coterminous with, and not patentably distinct from, claims 2, 6 and 7 of the 350 Patent. I note that while JAMP also raised claim 1 of the 350 Patent in the agreed joint statement of issues, as set out further below, the comparisons made relate to claims 2, 6 and 7 of the 350 Patent and refer to claim 1 only because of claim dependency.

[165] The 350 Patent was filed on August 11, 2003, and expired on August 11, 2023. As noted earlier, the 083 Patent will not expire until December 21, 2025. It therefore provides BII with an additional 2 years and 4 months of patent protection.

[166] The 350 Patent is the Canadian national phase entry of WO 948. It is identical to WO 948, except that the claims are specifically directed to a pharmaceutical composition of nintedanib (or a physiologically acceptable salt thereof) for treating a patient suffering from lung fibrosis instead of "immunologic diseases or pathological conditions involving an immunologic component". The 350 Patent also includes two additional consistory paragraphs within the Summary of the Invention that reflect the language of the claims and are directed to the treatment of a patient suffering from lung fibrosis.

[167] The allegation of double patenting includes the following claim pairings:

<table>
<tr><th>Claims of the 350 Patent</th><th>083 Asserted Claims</th></tr>
<tr><td rowspan="2">7. Use of an effective amount of the compound [nintedanib], or a physiologically acceptable salt thereof, in the treatment of a patient suffering from lung fibrosis.</td><td>1. Compound [nintedanib] for use in the prevention or treatment of idiopathic pulmonary fibrosis.</td></tr>
<tr><td>2. [Nintedanib esylate], for use in the prevention or treatment of idiopathic pulmonary fibrosis.</td></tr>
<tr><td>2. A pharmaceutical composition according to claim 1, which comprises the compound [nintedanib], or a physiologically acceptable salt thereof.

[1. A pharmaceutical composition for the treatment of a patient suffering from lung fibrosis, which comprises: the compounds [nintedanib], or a tautomer, a stereoisomer or a physiologically acceptable salt thereof; and a pharmaceutically acceptable diluent and/or carrier]</td><td>3. A pharmaceutical composition for use in the prevention or treatment of idiopathic pulmonary fibrosis, comprising [nintedanib esylate] and a pharmaceutically acceptable carrier.</td></tr>
<tr><td>6. A pharmaceutical composition in accordance with claim 3, wherein the</td><td>4. The pharmaceutical composition according to claim 3, in the form of a capsule.</td></tr>
</table>

formulation is a capsule for oral administration [3. A pharmaceutical composition in accordance with claim 1 or 2, which is an oral formulation] [2. A pharmaceutical composition according to claim 1, which comprises the compound [nintedanib], or a physiologically acceptable salt thereof] [1. A pharmaceutical composition for the treatment of a patient suffering from lung fibrosis, which comprises the compounds [nintedanib], or a tautomer, a stereoisomer or a physiologically acceptable salt thereof; and a pharmaceutically acceptable diluent and/or carrier]	[3. A pharmaceutical composition for use in the prevention or treatment of idiopathic pulmonary fibrosis, comprising [nintedanib esylate] and a pharmaceutically acceptable carrier.]
7. Use of an effective amount of the compound [nintedanib], or a physiologically acceptable salt thereof, in the treatment of a patient suffering from lung fibrosis.	5. Use of compound [nintedanib] in the treatment or prevention of idiopathic pulmonary fibrosis.
	6. Use of [nintedanib esylate] in the treatment or prevention of idiopathic pulmonary fibrosis.

[168] As a preliminary matter, JAMP asserts that in "same invention" double patenting, a genus or umbrella term in the claims of a prior patent can be considered coterminous with a specific term in the claim of a later patent. However, I do not find support for this general proposition in *Whirlpool,* which was cited by JAMP, nor in the broader jurisprudence. While this type of double patenting does not require the existence of identical language in the two patent claims, the wording of the claims, however different, must claim the same invention: *Sanofi* at para 109. This will not generally be the case if the invention claimed in one patent is broader than the other: *Sanofi* at para 110.

[169] Here, the issue before me is two-fold: 1) whether the claims of the 083 Patent, which provide for the prevention or treatment of IPF are coterminous or an obvious extension of the claims of the 350 Patent, which provide for the treatment of a patient suffering from lung fibrosis; and 2) with respect to claims 2, 3, 4 and 6 of the 083 Patent, whether nintedanib esylate is coterminous or uninventive over claims to physiologically acceptable salts of nintedanib.

[170] JAMP places much emphasis on the new claim language and the two additional consistory paragraphs in the disclosure of the 350 Patent. It argues that this indicates that "lung fibrosis" is not limited to fibrosis arising from autoimmune diseases. However, the new claim language does not change the invention of the patent, which remains directed to inhibition of Lck and T-cell mediated diseases, and lung fibrosis arising from those disease states.

[171] As I have already concluded that "lung fibrosis" as construed in WO 948 is relating to autoimmune disorders, Lck inhibition and T-cell mediated diseases, and would not understood to be targeting IPF, the claims of the 350 Patent cannot be coterminous with the 083 Asserted Claims. Further, even if I were wrong on this point, "lung fibrosis", which is a pathological finding, would not be coterminous with IPF, which is a disease state.

[172] Further, while JAMP points to the fact that Dr. Roth was a named inventor on both the 350 and 083 Patents, and asserts that nintedanib's inhibition of Lck was discovered in the same project leading to the filing of the 083 Patent, these assertions are not borne out by the full evidence. As noted by the Plaintiffs, Dr. Roth was a medicinal chemist. Thus, his work is not relevant to the points in issue. Further, neither Drs. Park nor Chaudhary who are named inventors

on the 083 Patent were named as inventors on the 350 Patent. The different set of full inventors weighs against there being same invention double patenting.

[173] Similarly, with respect to point (b), claims 2, 6 and 7 of the 350 Patent cover nintedanib or a pharmaceutically acceptable salt thereof. A "physiologically acceptable" salt form would be understood to mean one that is non-toxic to the patient (human or non-human mammals) (Steed, Ex31, para 190). The claims do not state which, if any, of the possible salts of nintedanib would be contemplated. In my view, without this direction, the claims of the 350 Patent cannot be construed to be coterminous with claims to the esylate salt of nintedanib and same invention double patenting does not apply for this additional reason.

[174] With respect to obviousness-type double patenting, while I agree with JAMP that claims to the esylate salt of nintedanib are not patentably distinct from the claims in the 350 Patent to a physiologically acceptable salt of nintedanib, I nonetheless do not view claims to the treatment of IPF as an obvious extension over claims to the treatment of a patient suffering from lung fibrosis as construed within the 350 Patent. I will deal with each of these aspects in turn.

[175] BI argues that without any problem associated with the free base of nintedanib, there would be no motivation to develop a specific salt of nintedanib. However, this is not the proper question for double patenting. The issue is whether the claims of the 083 Patent are an obvious extension over the claims of the 350 Patent. The claims of the 350 Patent recite a physiologically acceptable salt of nintedanib. Thus, salt formation was contemplated by the 350 Patent.

[176] Further, as agreed by the experts, the PSA's first course of action would be to investigate nintedanib as a free base and obtain information about its solubility. In doing this, the PSA would have quickly learned that the free base had poor solubility and would have been motivated to increase solubility (and dissolution rate) through salt formation (Gullapalli, Ex9, Sch7-19, 7; Myerson, XX, TT6, 1231:17-1232:15).

[177] BI asserts that even if the PSA decided to conduct a salt screen, as there were a number of potential anions that could be used, there would be no motivation to select ethanesulfonic acid as a salt former to prepare the esylate salt. They assert that there would be no basis to predict which anions would crystallize and provide desirable properties (Myerson, XC, TT 6, 1210:23-26; Steed, XX, TT6, 1100:4-16, 1100:26-1101:11).

[178] However, the PSA knew that various salts had been marketed to human patients in FDA-approved products and that such salts would be considered to be physiologically acceptable salts. These included salts of hydrochloric acid, sulfuric, hydrobromic, nitric and phosphoric acids, sulfonic acid salts such as mesylate, esylate, isethionate, tosylate, napsylate and besylate, salts of carboxylic acids such as acetic, propionic, maleic, benzoic, aspartic and fumaric; salts of anionic amino acids such as glutamic and aspartic and hydroxy acids such as citric, lactic, succinic, tartaric, glycolic and malic (Steed, Ex31, para 190). Dr. Myerson agreed that the esylate salt of nintedanib as claimed in the 083 Asserted Claims would be considered as a physiologically acceptable salt of nintedanib (Myerson, Ex35, paras 43 and 178).

[179] The esylate salt was listed as the sixth most common pharmaceutical salt in one of the key prior art references (Bastin) that formed the PSA's CGK (Steed, XC, TT6, 1070:24-1071:12; Ex31, Sch10, Table 1).

[180] Further, by 2004, salt screening was routine. A PSA looking to increase water solubility of a relatively hydrophobic molecule like nintedanib, would not be looking at hydrophobic anions or aromatic groups which would rule out a number of common anions from testing (Steed, XC, TT6: 1071:21-1072:9). This would narrow the likely candidates to a smaller subset of about fifteen anions to screen, which would include the ethanesulfonic acid for making the esylate salt (Steed, XC, TT6, 1072:10-15).

[181] As argued by JAMP, all experts admitted that a PSA interested in formulating pharmaceutical compositions of nintedanib would be capable of, and motivated to conduct a preliminary search of the prior art to discover any salt forms of nintedanib that were already known and would identify WO 099, a prior art patent application that identifies nintedanib esylate as a salt of interest (Steed, Ex31, para 82; Myerson, XX, TT6: 1214:22-1215:3; Phan, Ex20, para 215). Indeed, the 083 Patent refers to the disclosure of the esylate salt of nintedanib in WO 099 within its background teachings (Steed, Ex31, paras 214, 256; Myerson, Ex35, para 144).

[182] BI argues that the teachings of WO 099 cannot be used in the double patenting analysis. The law is clear that it is not a mosaic of art that is to be considered for double patenting; rather the doctrine is limited to the prior patent at issue and the CGK (*Mylan* at paras 29, 36), of which

WO 099 would not be included. Indeed, the purpose behind the doctrine is to prohibit evergreening of patent rights arising from an applicant seeking to claim protection for an invention already claimed in a previous patent.

[183] The doctrine prohibits the same applicant from claiming subject-matter that is an obvious extension of an earlier claimed invention. While this is not the same as an obviousness attack, I nonetheless agree with JAMP that the recognition in the 083 Patent that the esylate salt of nintedanib was previously disclosed indicates that the inventors did not consider the esylate salt of nintedanib to be inventive.

[184] In my view, once it was identified that physiologically active salts of nintedanib were useful for treatment, the identification of the esylate salt of nintedanib would have been obvious to identify by routine testing. In this case, the applicant was well aware by the time of filing the 083 Patent that the esylate salt of nintedanib was already a salt form of interest.

[185] However, in my view the extension from the use of nintedanib in treating lung fibrosis to the treatment of IPF was a bigger hurdle. While JAMP asserts that there is no patentable distinction between treatment of “lung fibrosis” in the 350 Patent and treatment of IPF in the 083 Patent, I cannot agree.

[186] JAMP points to testimony from Dr. Blackwell who acknowledged that the PSA would understand that what is being treated in either set of claims is the fibrosis (Blackwell, XX, TT7, 1413:13-19). It also cites to evidence of Dr. Strieter from European Opposition proceedings

involving European Patent No. 1,830,843, a patent described by Dr. Strieter as being "similar in subject matter to the 083 Patent", where he stated:

> [I]f a skilled person would consider a drug suitable for the treatment of lung fibrosis, in general, they would also consider the drug suitable for the treatment IPF [sic], especially when treatment is not targeting the cause of the disease, but rather the pathology of fibrosis.

[187] However, this latter evidence is of limited assistance without further context. Dr. Strieter did not opine on the 350 Patent nor on double patenting. He does not provide an opinion on the mechanism of action of nintedanib.

[188] As stated earlier, it was known that "lung fibrosis" was not a homogeneous condition and could vary in severity depending on its cause. Treatment was also known to vary depending on the magnitude of fibrosis, its histopathological presentation, and the prognoses of the disease state (Strieter, XX, TT5, 961:6-962:17; Blackwell, Ex38, para 323).

[189] Similarly, while IPF was known to be a chronic progressive illness, characterized by sequential acute lung injury and subsequent scarring, the disease itself was not well understood. It was known to be a distinct disease from other IIPs that was particularly difficult to treat (Blackwell, Ex38, para 346). There was limited knowledge of the underlying biological mechanisms involved and of the etiology of the disease (Strieter, Ex25, para 27; Phan, Ex20, para 146).

[190] With the teachings of the 350 Patent alone and the CGK, in my view the totality of the evidence does not support a finding that the PSA would view the treatment of IPF as an obvious

and self-evident extension of the treatment of lung fibrosis that arises from Lck inhibition and T-cell mediated diseases.

[191] As a further factor, JAMP notes that the 350 Patent was listed on the Patent Register in association with OFEV® from June 30, 2015 until July 28, 2022. The only approved indication for OFEV® at the time of listing was for the treatment of IPF. It was only when BI commenced the current action that it delisted the 350 Patent.

[192] JAMP asserts that BI's listing of the 350 Patent constitutes an admission against interest (*i.e.*, an admission that the 350 Patent encompasses a treatment for IPF). BI contends that this is extrinsic evidence and is inadmissible. Moreover, it asserts that the *PMNOC Regulations* permit a first person to delist a patent at any time.

[193] While there are instances where a concession made in one proceeding under the *PMNOC Regulations* may be construed as binding upon the conceding party in later proceedings involving a different party (*Apotex Inc v Pfizer Canada Inc*, 2014 FCA 250 at para 104), there has been no legal finding in this case relating to the prior listing of the 350 Patent. This is not a situation where issue estoppel or *res judicata* applies. Nor does the fact of the listing establish a construction of the claims of the 350 Patent.

[194] Further, the 350 Patent was delisted before the proceeding was commenced.

[195] In my view, the prior listing of the 350 Patent is not fatal to the issue of double patenting.

C. *Utility*

[196] To determine whether a patent discloses an invention with sufficient utility, the Court must first identify the subject-matter of the invention as claimed in the patent and second ask whether that subject-matter is useful or capable of a practical purpose: *AstraZeneca Canada Inc v Apotex Inc*, 2017 SCC 36 [*AstraZeneca*] at para 54. A mere scintilla of utility is enough: *AstraZeneca* at para 55.

[197] The utility requirement in section 2 of the *Patent Act* is to be interpreted in line with its purpose; to prevent the patenting of fanciful, speculative or inoperative inventions: *AstraZeneca* at para 57; *Takeda* at para 229.

[198] To avoid granting patents prematurely, and thereby limit potentially useful research and development by others, utility must be either demonstrated or soundly predicted as of the patent's filing date. As explained in *Wellcome Foundation* at paragraph 66, the doctrine of sound prediction, "balances the public interest in early disclosure of new and useful inventions, even before their utility has been fully verified by tests …, and the public interest in avoiding cluttering the public domain with useless patents, and granting monopoly rights in exchange for speculation or misinformation".

[199] The parties agree that the practical utility of the invention as claimed in the 083 Patent is the use of nintedanib and its esylate salt in the treatment of IPF in human patients (Strieter, Ex25, para 137; Blackwell, Ex38, paras 356-357; Blackwell, XX, TT7, 1343:16-27). Utility was not

demonstrated at the time of patent filing. Instead, it was premised on a sound prediction of utility.

[200] For a sound prediction to be established, there must be a factual basis for the prediction, the inventor must have an articulable line of reasoning from which the desired result could be inferred from the factual basis, and there must be proper disclosure: *Wellcome Foundation* at para 70; *Eli Lilly Canada Inc v Novopharm Limited*, 2010 FCA 197 [*Novopharm*] at para 83.

[201] As a preliminary matter, the parties are not *ad idem* as to the perspective that applies to the various parts of the *Wellcome Foundation* test, including the assessment of the soundness of the prediction. BI argues that it is only the disclosure in the patent that must be considered. The inventors' work is irrelevant to the determination of whether a sound prediction exists, and only comes into play if there is a complete disconnect with what is in the patent. Although seemingly the focus of the debate between the parties (as discussed further below), they assert that this is not the situation here. JAMP contends that the assessment is subjective and it is the actual factual basis and line of reasoning of the inventors that is relevant. The PSA assesses the disclosure in the patent and then moves forward with its teachings. In my view, this is consistent with the patent bargain and the reasoning set out in the jurisprudence, including *Wellcome Foundation*.

[202] Indeed, to obtain a patent, it is the inventors who must have a factual basis and a sound line of reasoning to support the invention as claimed, and it is the inventors who are responsible for disclosing the foundation for their prediction of utility in the patent: *Wellcome Foundation* at paras 3, 52, 55, 62, 65, 70 and 71. While an understanding of the disclosure and the prediction as

disclosed therein is ultimately assessed from the perspective of the PSA, this does not shift the focus of the factual inquiry away from the inventors: *Teva Canada v Leo Pharma Inc*, 2017 FCA 50 [*Leo Pharma*] at paras 12, 25, 28, 37. The prediction is a question of fact and evidence must be led as to what was known or not known as of the filing date: *Wellcome Foundation* at para 71; *Eli Lilly Canada v Apotex Inc*, 2008 FC 142 at para 162 [*Raloxifene FC*], aff'd 2009 FCA 97 [*Raloxifene FCA*]. The knowledge, activities and endeavours of the inventors must be considered: *Sanofi-Aventis Canada Inc v Apotex Inc*, 2009 FC 676 at para 151, aff'd 2011 FCA 300, leave to appeal to SCC refused.

[203] In order to engage the patent bargain, the work of the inventors and the foundation for the prediction must be shared with the public. It is at the third stage of the test (*i.e.*, the requirement for disclosure) that the perspective of the PSA comes into play as they are the intended recipient of the patent disclosure. It is at this stage that the SCC in *Wellcome Foundation* differentiates between inventions based on demonstrated utility and those based on a sound prediction. As stated by the SCC in its paragraph 70, for the former it is normally "sufficient if the specification provides a full, clear and exact description of the nature of the invention and the manner in which it can be practiced … [i]t is generally not necessary for an inventor to provide a theory of *why* the invention works. Practical readers merely want to know that it does work and how to work it." However, disclosure is necessary for a sound prediction of utility as it engages "*the quid pro quo* the applicant offers for the patent monopoly". The patent must provide sufficient disclosure such that a PSA could as the inventors did, soundly predict that the invention would work once reduced to practice: *Raloxifene FCA* at para 18.

[204] While there is no statutory requirement to disclose the utility of an invention where the utility is demonstrated (*Teva* at para 40; *AstraZeneca* at para 58), if the utility arises from a sound prediction, the factual basis for the prediction must be set out in the patent disclosure to the extent it is not based on the CGK (*Eurocopter* at para 153; *Pharmascience Inc v Teva Canada Innovation*, 2022 FCA 2 [*Pharmascience*] at para 5; *Takeda* at paras 231-232).

[205] The disclosure is directed to the PSA who comes equipped with their CGK. As stated in *Apotex Inc v Allergan Inc*, 2015 FCA 137 at paragraph 9, elements that would be self-evident to the PSA and that are within their CGK need not be explicitly disclosed for the third part of *Wellcome Foundation* test to be met:

> [9] The Federal Court identified the factual basis for the prediction (the minimum inhibitory concentration values of several compounds tested against a number of bacteria species together with comparative data) and the line of reasoning that would, to the skilled reader, flow from that data. As this Court observed in *Eurocopter v. Bell Helicopter Textron Canada Ltée*, 2013 FCA 219, 449 N.R. 111, at paragraphs 152 and 153, the factual basis, line of reasoning and level of disclosure required by the doctrine of sound prediction are to be assessed as a function of both the knowledge that the skilled person would have to base that prediction on and what the skilled person would understand as a logical line of reasoning leading to the utility of the invention. Those elements of the doctrine of sound prediction that would be self-evident to the skilled person need not be explicitly disclosed in the patent.

[206] In this case, the invention is the recognition that nintedanib/nintedanib esylate, which was already known, can be *used* for the prevention or treatment of IPF in humans. Disclosure of the underlying facts and line of reasoning for the prediction of nintedanib's utility was the *quid pro quo* for the valuable propriety rights that BII obtained for the 083 Patent.

[207] I agree that if it was not contested that the full factual basis and line of reasoning of the inventors was disclosed in the patent then the focus could shift entirely to the PSA to determine whether this foundational work was sufficient to ground a sound prediction (*i.e.*, as was done in *Leo Pharma* at paras 35-38). However here, JAMP takes direct issue with the fulfillment of the disclosure requirement under *Wellcome Foundation*. Their assertion is that the inventors did not disclose foundational work in the patent thereby not meeting the *quid pro quo*. Thus, the analysis must also focus on the inventors' work and what actually made the prediction sound.

[208] Below is a summary of the inventors work as of the filing date of December 21, 2005, as introduced into evidence by the Plaintiffs.

(1) The Inventors' Work

[209] Park began working on potential therapeutic targets for IPF in 2002. Initially he identified transforming growth factor-beta [TGF-β] as a potential target for inhibition, which was thought to be responsible for transforming fibroblasts to myofibroblasts, the cells responsible for increasing the synthesis of the extracellular matrix proteins (*e.g.*, collagen) involved in the formation of scar tissue leading to fibrosis (Park XC, TT8, 1458:24-1459:3). However, in 2003, there were reports in the literature suggesting that direct inhibition of TGF-β signalling upstream could induce the formation of cancer. In view of these potential side effects, Park shifted his work away from TGF-β and began exploring other signalling pathways as potential therapeutic targets for IPF (Park, XC, TT8, 1461:2-18). This involved consideration of molecules under parallel investigation at the company, including combined inhibitors of VEGFR, PDGFR and FGFR (triple kinase inhibitor).

[210] It was at this time that Park became aware of BIBF1120 (now known as nintedanib), an internal triple kinase inhibitor and front-runner compound for cancer research. Park knew that BIBF1120 was undergoing clinical investigation, and that another compound BIBF1000 had a similar inhibition profile to BIBF1120 on the three kinases and on TGF-β. He began tests on BIBF1000 as a surrogate for BIBF1120 (Park, XC, TT8, 1464:15-23).

[211] Park's laboratory conducted an initial set of experiments using the rat bleomycin model, which they had established for their work on TGF-β inhibition (Park, Ex43, para 31). The testing involved intratracheal bleomycin administration on day 0, followed by oral administration of test compound starting on day 1 for a duration of 21 days (Park, XC, TT8, 1465:12-28). The results of the study showed significantly reduced expression of pro-fibrotic markers and decreased collagen deposition, however, it was unclear if the decrease in fibrosis was due to anti-inflammatory or anti-fibrotic mechanisms (or a combination of the two) (Park, Ex43, para 32). Results from further investigation on the time course of the development of inflammation and fibrosis suggested a switch between inflammation and fibrosis between days 9 and 14 (Park, Ex43, para 33). This prompted Park to modify the existing bleomycin model (the "preventative regimen") to a delayed treatment regimen (the "therapeutic regimen"), where test compounds were administered at day 10 for a duration of 12 days, rather than administered starting on day 1 (Park, Ex43, para 36, XC, TT8, 1470:1-20).

[212] Park's team discussed their preliminary work with colleagues and upper management, prompting release of BIBF1120 for formal testing (Park, XC, TT8, 1473:4-15), which started in October 2004.

[213] Park's team first examined the effect of BIBF1120 using the preventative regimen in the bleomycin model by testing three different doses of BIBF1120. The results showed a dose-dependent response, with the highest dose (50 mg/kg) completely reversing the fibrosis induced by bleomycin (Park, Ex43, paras 44-48, XC, TT8, 1473:16-1475:13). The effects were corroborated by gene expression analyses, which showed dose-dependent inhibition of profibrotic markers TGF-β1 and pro-collagen I (Park, Ex43, para 49).

[214] To examine whether BIBF1120 acted through anti-fibrotic processes or inhibition of the initial inflammation, Park's team then went on to test 50 mg/kg of BIBF1120 in the therapeutic regimen. The results showed that delaying treatment until day 10 post-bleomycin administration in the fibrotic phase also led to consistent, nearly complete attenuation of fibrosis (Park, Ex43, para 50, XC, TT8, 1476:20-1477:1). The effects were further corroborated by gene expression analyses.

[215] To investigate the effect of BIBF1120 on TGF-β mediated myofibroblast differentiation Park's team set up an *ex vivo* assay using primary human fibroblasts, which showed a reduction of alpha smooth muscle actin (αSMA) expression, a hallmark for myofibroblasts (Park, Ex43, paras 51-54, XC, TT8, 1478:19-1479:20). Next, the Park team examined whether the activities of BIBF1120 observed in the *in vivo* animal models and *ex vivo* model were attributable to direct inhibition of the TGF-β signaling pathway using a series of assays, which showed that BIBF1120 exerted its anti-fibrotic effects by indirectly interfering with some of the signaling pathways downstream of TGF-β. Lack of direct inhibition supported continued development of BIBF1120 for the treatment of IPF (Park, Ex43, paras 55-56, XC, TT8, 1480:16-1481:4).

[216] Following these positive results, BII established an internal evaluation team within the company to do an in-depth evaluation of the compound in order to make a recommendation to upper management. As the medical member of the team, Gupta's role was to assess the suitability of BIBF1120 for IPF patients based on available preclinical data and design and to create a clinical development plan for a possible Phase II program. The approach was to build on the safety backbone of the oncology clinical program and design IPF trials informed by patient experience with the existing dosage form (Gupta, Ex46, para 16).

[217] In May 2005, Gupta met with three IPF experts to discuss whether the safety profile for BIBF1120 emerging from BII's oncology program was compatible with the planned investigation in IPF patients (Gupta, Ex46, para 18). Gupta presented the external experts with: results of the bleomycin model therapeutic regimen; toxicology data for nintedanib [redacted]; and exposure, pharmacokinetics and adverse events data from human studies involving nintedanib in the oncology program (Gupta, XX, TT9, 1545:11-1546:21). While the experts suggested that BII should consider longer-term animal models of fibrosis (Gupta, XX, TT9, 1547:4-26; JAMP read-ins, Ex33, V2:56), the overall profile of BIBF1120 was considered acceptable for initiation of clinical investigations in IPF (Gupta, Ex46, para 20).

[218] Following the meeting, Gupta drafted a clinical development plan proposing two development scenarios: 1) a Phase IIa bridging study to address the possibility of unexpected adverse events not previously encountered in oncology trials; or 2) a direct entry to a Phase IIb study (a larger and longer trial focused on exploring efficacy and dosing requirements, in

addition to adverse events). The options were discussed internally in early July 2005 (Gupta, Ex46, para 22).

[219] Gupta also met with additional experts who endorsed the proposal for a Phase II clinical program in IPF patients, suggesting that BII proceed directly to the Phase IIb study (Gupta, Ex46, para 25).

[220] As part of the final approvals a [redacted] was prepared setting out the rationale for the project (Gupta, Ex46, para 30). The data in the [redacted] included (Gupta, XC, TT9, 1550:26-1553:7):

(a) the *in-vitro* tyrosine kinase inhibition results for nintedanib with respect to VEGFR, FGFR and PDGFR;

(b) *in-vivo* studies using nintedanib in both the preventative and therapeutic regimens of the bleomycin-induced rat model;

(c) *ex-vivo* studies of the inhibition of nintedanib in transformation of fibroblasts to myofibroblasts;

(d) an estimate of the human efficacious exposure based upon tumour, xenograft models and exposure during the oncology clinical trials;

(e) drug metabolism and pharmacokinetics data for nintedanib; and

(f) toxicology, drug exposure and adverse events data from the oncology work on nintedanib.

[221] Gupta along with others presented the work package to BII's senior development committee who recommended direct entry of BIBF1120 into the Phase IIb clinical trial (Gupta,

Ex46, para 31). BIBF1120 entered Phase II clinical development for IPF in 2007 (Gupta, XC, TT9, 1534:27-28).

(2) The Parties' Arguments

[222] In its written submissions, JAMP contends that the actual factual basis for the prediction of nintedanib/nintedanib esylate's use for the prevention or treatment of IPF in humans was the information included in the [redacted], as well as the animal toxicology studies that were presented to the external experts, all of which had been completed by December 2005, namely:

(a) *in vitro* tyrosine kinase inhibition results for nintedanib with respect to VEGFR, FGFR, and PDGFR;

(b) an *in vivo* evaluation of nintedanib in the bleomycin-induced rat lung fibrosis model using the "preventative" regimen (*i.e.*, treating on days 1 to 21);

(c) an *in vivo* evaluation of nintedanib in the bleomycin-induced rat lung fibrosis model using the "therapeutic" regimen (*i.e.*, treating on days 11 to 21 alone);

(d) *ex vivo* studies of nintedanib on fibroblast to myofibroblast transformation;

(e) an evaluation of nintedanib's ability to directly inhibit TGFβ Receptor I (and the conclusion it would not);

(f) animal toxicology data for nintedanib;

(g) safety/tolerability/toxicity/adverse event data for nintedanib derived from clinical trials that were part of the oncology program; and

(h) pharmacokinetic data for nintedanib derived from clinical trials that were part of the oncology program.

[223] It contends that the inventors' line of reasoning for the prediction that nintedanib and its esylate salt would be useful to treat IPF in human patients included the following:

(a) nintedanib is a potent *in vitro* inhibitor of tyrosine kinases (VEGFR, FGFR and PDGFR) implicated in the processes that lead to fibrosis. Therefore, inhibition of these kinases will lead to a reduction in fibrosis, which is a hallmark symptom of IPF;

(b) nintedanib lowers the formation of fibrosis in the *in vivo* bleomycin-induced rat lung fibrosis model using the therapeutic regimen (*i.e.*, treating on days 11 to 21 alone) and *ex vivo* studies on fibroblast to myofibroblast transformation, which is evidence from a mammal that nintedanib is interfering in the processes that lead to fibrosis, and that this would likewise lead to a reduction in fibrosis in human IPF patients;

(c) nintedanib does not directly inhibit TGFβ Receptor I and therefore is unlikely to cause side effects in humans, including the creation of tumours;

(d) pharmacokinetic data from clinical trials that were part of the nintedanib oncology program, coupled with the efficacy data from the *in vivo* bleomycin-induced rat lung fibrosis model, can be used to provide estimates of suitable doses for treating IPF in humans; and

(e) safety data from the same clinical trials from the oncology program offer assurance that nintedanib would be suitably tolerated when used in the treatment of patients with IPF.

[224] In argument, JAMP's counsel narrowed its assertion of the inventors' actual asserted factual basis to the tyrosine kinase inhibition results, the results from the bleomycin-induced rat lung fibrosis model using the therapeutic regimen, the *ex vivo* studies, and the subsequent evaluation of TGF-β inhibition (items (a), (c) through (e) of paragraph 222 above), and asserted

that the line of reasoning identified in paragraphs (a) and (b) of paragraph 223 above is what made the prediction sound.

[225] It relies on a statement made by Park that the bleomycin model preventative regimen "was not predictive of whether a molecule would work clinically in a human being to treat IPF" (Park, XX, TT8, 1488:11-16), and asserts that this supports its contention that the preventive regimen did not form part of the inventors' actual factual basis for the prediction.

[226] During cross-examination, Park acknowledged that the reason for his statement was that "compounds were working well in the preventative bleomycin model, but … not working well in humans" (Park XX, TT8, 1488:17-23). These realities led to further work by the inventors, including the development of the therapeutic regimen as reported in Park's 2006 scientific paper, *American Journal of Respiratory and Critical Care Medicine* (Park, XX, TT8, 1491:19-1495:10, Ex43, SchN).

[227] BI does not dispute that all work conducted by the inventors as of December 21, 2005 was not disclosed in the 083 Patent. The 083 Patent disclosed the *in vitro* tyrosine kinase inhibition results for nintedanib with respect to VEGFR, FGFR and PDGFR, and the *in vivo* evaluation of nintedanib in the preventative regimen only. It does not disclose the additional work reported in the [redacted] that the inventors had completed by December 2005, including, *inter alia*, the results from the bleomycin model therapeutic regimen, the *ex vivo* studies, and the evaluation as to TGF-β inhibition.

[228] However, BI asserts that this additional experimentation was corroborative rather than grounding. It argues that there is no requirement in law to disclose all studies as long as the PSA would have a sufficient factual basis and line of reasoning to support the prediction. It refers to comments made by Justice Snider in *Teva Canada Limited v Novartis* AG, 2013 FC 141 relating to the patent listed in association with the drug imatinib [*Imatinib*], paragraphs 318-323 which are reproduced below:

> [318] The Plaintiffs complain that the sound prediction – if, indeed, one could be made – needed the disclosure of Ciba-Geigy's test data which were not included in the '203 Patent.
>
> [319] In making this argument, the Plaintiffs rely heavily on the decision of my colleague, Justice Hughes, *in Eli Lilly Canada Inc v Apotex Inc*, 2008 FC 142, 63 CPR (4th) 406 [*Raloxifene (FC)*], aff'd 2009 FCA 97, 78 CPR (4th) 388 [*Raloxifene (FCA)*] to support their position that important data and test results should have been disclosed in the '203 Patent. I do not understand *Raloxifene (FC)*, or the subsequent affirmation of Justice Hughes by the Court of Appeal in *Raloxifene (FCA)*, to extend to the extreme position taken by the Plaintiffs.
>
> [320] In *Raloxifene (FC)*, Justice Hughes concluded that the disclosure requirement for sound prediction had not been met. In that case, Eli Lilly had not disclosed, in the patent, the results of a study – referred to as the Hong Kong Study – which had been carried out by Eli Lilly prior to the Canadian filing date. The Hong Kong Study involved direct testing of raloxifene and, as found by Justice Hughes, was sufficient to turn the prediction that raloxifene would be effective in treating osteoporosis and bone loss into a sound prediction (*Raloxifene (FC)*, above at para 156). In other words, without the Hong Kong Study, Eli Lilly did not have a sound prediction that raloxifene would work as claimed.
>
> [321] The singular importance of the Hong Kong Study was emphasized by the Court of Appeal, where it stated that (*Raloxifene (FCA)*, above at para 15):
>
> > In my respectful view, the Federal Court Judge proceeded on proper principle when he held, relying on *AZT*, that when a patent is based on a sound prediction, the disclosure must include the prediction. As the prediction was made sound by

> the Hong Kong study, this study had to be disclosed. [Emphasis added]

[322] In *Lovastatin (FC),* I was faced with very similar arguments to those now before me. In that case, there was no question that some information known to the inventors was not disclosed in the patent. At paragraphs 527-528:

> I agree that the cross-examination of Mr. Alberts resulted in a list of facts and information that the inventors of the '380 Patent knew as of the Canadian filing date. One of the areas of interest relates to the dog studies carried out in 1979. Apotex pounces on this information as something that ought to have been disclosed in the '380 Patent in order to justify the sound prediction. However, I do not understand the jurisprudence to teach that the patent specification must disclose absolutely everything that the inventor knew up to the relevant date. In [*Raloxifene (FC),* above], without disclosure of the Hong Kong study, a skilled person would not have had sufficient information to understand the justification for the prediction. We must examine the specification to determine whether, with the information disclosed (even if there was more information available and undisclosed), a skilled person could have soundly predicted that the invention would work once reduced to practice.
>
> In the case of the '380 Patent, the question is whether sufficient information was disclosed to allow the skilled person to soundly predict that the compounds of the invention would be "useful as antihypercholesteremic agents for the treatment of atherosclerosis, hyperlipemia and like diseases in humans".

[323] The fact that all of the Ciba-Geigy tests are not described in the patent is not, in my view, fatal to Novartis's case. Applying the relevant jurisprudence to the '203 Patent now before me, the question is whether sufficient information was disclosed to allow the person of ordinary skill in the art to soundly predict that the compound of Claim 29 (imatinib) would be useful for the chemotherapy of tumours. I emphasize that the person to whom this question is relevant is a skilled person who would come to this

> exercise with the common general knowledge that is discussed above.
>
> [Emphasis in the original]

(3) Was there a Sound Prediction

[229] Consistent with *Wellcome Foundation* and *Raloxifene*, on which *Imatinib* relied, a patent need not disclose all test results obtained by the inventors, but must disclose the information that made the prediction sound.

[230] To be sound, the prediction must be more than "a lucky guess", "mere speculation", belief, or a hypothesis that might later prove useful: *Wellcome Foundation* at paras 25, 69, 83, 84. The public is entitled to a teaching that is solid, accurate and meaningful (*Wellcome Foundation* at paras 69, 83), and one that is based on exact science (*Wellcome Foundation* at para 84). A regulatory standard and certainty is too much: *Wellcome Foundation* at paras 62, 63, 77. The threshold is not high: *Sandoz Canada Inc v Janssen Inc*, 2023 FCA 221 [*Sandoz*] at para 24. The patent may be sustained provided a *prima facie* reasonable inference of utility exists: *Novopharm* at para 85; *Sandoz* at para 24.

[231] As stated earlier, Park testified to modifying the preventative regimen to make it suitable to discern between compounds that were active in the fibrotic phase, and to treat the animals in a way that would be consistent with treating the fibrotic phase of the disease in humans (Park, XX, TT8, 1489:21-24, 1491:19-1492:5). He adapted the preventative regimen to the therapeutic regimen where drug was not administered until day 10, and was then administered for twelve consecutive days, which in his view more closely resembled the clinical setting physicians faced

with patients with fibrosis presenting long after the resolution of inflammation (Park, XX, TT8, 1496:17-1497:5).

[232] Park reported that the findings from the therapeutic regimen "might offer a preclinical model to ultimately more accurately judge the therapeutic potential of …compounds for the treatment of IPF" and that "[t]he use of different treatment regimes in the bleomycin model may prove a valuable method by which drugs with true anti-fibrotic potential can be identified and investigated" (Park, XX, TT8, 1494:24-1495:10, 1498:8-20). He further testified that one of the reasons for his confidence that nintedanib would be successful in treating humans with IPF was the *ex vivo* studies that showed that nintedanib inhibited fibroblast to myofibroblast transformation (Park, XX, TT8, 1501:12-17) and his subsequent confirmation that nintedanib did not have a direct inhibitory effect on TGF-β (Park, XX, TT8, 1503:7-1504:4).

[233] BI asserts that this evidence from Park is not relevant to a determination of whether the prediction of nintedanib's utility to treat IPF was sound. It relies on the comments made in *Western Oilfield* at paragraph 126 that, "utility is assessed from the point of view of the skilled person, not the inventor" and that "[d]oubts that the inventor expressed about the utility… would not necessarily lead to a conclusion of inutility."

[234] However, these comments must be viewed in context. The latter comment made in *Western Oilfield* related to doubts of the inventor as to a specific embodiment of the invention only, not as to the system as a whole (para 126). They do not suggest, in my view, that evidence from the inventors is irrelevant, particularly where the issue here is one of disclosure.

[235] I accept that when making his comments Park was not evaluating the work of the inventors in a legal context. Therefore, Park's comments are not dispositive of the legal issues. However, the evidence was clear that both Park and Gupta considered these additional results important to include in the [redacted] and prioritized the bleomycin model therapeutic regimen results when reporting the findings from the bleomycin model to the external experts. It was never explained why BII did not disclose the results from testing in the therapeutic regimen, the *ex vivo* studies, and the subsequent evaluation of TGF-β inhibition in the 083 Patent despite having these results well in advance of its filing date.

[236] While BI argues that it is not relevant why this was not included, it begs the question whether the prediction is sound without this disclosure and whether this additional disclosure would have advanced the PSA's knowledge moving forward. JAMP asserts that BII should not be entitled to withhold information that it knew would be helpful to the PSA. It argues that the practical consequence of the failure to disclose the missing information was two-fold. First, by failing to disclose key information that would have led the PSA to view nintedanib as being superior to other candidates, the PSA would not have considered nintedanib to be any more promising as a treatment for IPF than any other drug tested before it, and would be inclined to believe that a significant number of other tyrosine kinase inhibitors found in the prior art would be more attractive candidates for development in the treatment of IPF over nintedanib. Second, by failing to disclose an evaluation of TGF-β inhibition, the CGK and disclosure of the 083 Patent would have led the PSA to believe that direct inhibition of TGF-β was favourable for a drug being developed to treat IPF.

[237] However, I do not find either of these arguments compelling. On the first point, the 083 Patent is not a selection patent. As such, there is no obligation on the patentee to establish that nintedanib has superior properties over other compounds. The PSA would not be looking in different directions because of the 083 Patent disclosure as it was pointing to nintedanib. On the second point, the 083 Patent does not discuss the potential carcinogenic effects of direct inhibition of TGF-β Receptor I. Therefore, there would be no basis to conclude one way or the other whether nintedanib had these properties and, as it did not, no need to disclose a negative finding.

[238] BI contends that at least the latter argument is also misplaced and should have been raised as an argument under section 53 of the *Patent Act*, which provides that a patent may be invalidated through an omission made to the specification where it has been "wilfully made for the purpose of misleading".

[239] However, the argument here is not that BII intentionally withheld this information from the 083 Patent for the purpose of misleading the PSA. Rather, the argument is simply that in failing to disclose the results from the bleomycin model therapeutic regimen, *ex vivo* studies, and evaluation as to TGF-β inhibition, BII did not satisfy the *quid pro quo* required for BII's patent rights (*i.e.*, the third part of the *Wellcome Foundation* test). In failing to do so, JAMP asserts that the PSA was not put in the same successful position as the patentee; nor were they given the same level of certainty available to the inventors as to their findings.

[240] As set out earlier, it is undisputed that the only elements of the factual basis provided by the inventors in the 083 Patent were: a) a general discussion of the *in vitro* tyrosine kinase inhibition results for nintedanib with respect to VEGFR, FGFR and PDGFR; and b) an evaluation of nintedanib in the *in vivo* bleomycin-induced rat model, using the preventative regimen, followed by histologic and gene expression analyses.

[241] The evidence from the experts was consistent; the general discussion of the *in vitro* tyrosine kinase inhibition results for nintedanib with respect to VEGFR, FGFR and PDGFR while informative, was insufficient on its own to support a sound prediction of clinical efficacy of nintedanib in humans. The PSA did not fully understand the role of different growth factors and the interplay between tyrosine kinase signalling pathways in the pathogenesis of the disease (Blackwell, XX, TT7, 1347:4-10; Strieter, Ex25, paras 192-193). While some in the field had proposed that the inhibition of tyrosine kinases that were implicated in the biological pathways of fibrosis might be a viable therapeutic approach, this perspective was not universally held (Strieter, Ex25, para 193). There were no known treatments of this type (or of any type) for IPF in existence, nor had any been validated in clinical trials (Blackwell, XX, TT7, 1345:11-20). The PSA understood that kinase inhibition was an untested and unproven treatment strategy and were experimenting with a variety of other options (Blackwell, XX, TT7, 1346:19-25, 1347:11-15; Strieter, Ex25, para 192).

[242] Thus, *in vivo* results were necessary. However, the bleomycin model included known limitations. First, it was known that chemical injury to the rat lungs by a single administration of bleomycin did not align with how IPF actually developed in patients (Strieter, Ex25, para 200).

Second, although a small number of compounds had shown positive results from the bleomycin model preventative regimen, this had not translated to efficacious results in the clinic nor a successful treatment in humans for IPF (Blackwell, XX, TT7, 1352:28-1353:6; Strieter, Ex25, para 201). Indeed, no animal model had predicted clinical efficacy in IPF (Strieter, XX, TT5, 1035:14-17). As explained by Dr. Phan the bleomycin model was used as an initial screening tool to identify those compounds that had promise to move on to the next stage of analysis (Phan, RX, TT4, 872:11-873:4). It was not viewed as being confirmatory of clinical results. The results presented in the 083 Patent indicated only a low probability of success of nintedanib being useful for treating IPF in humans (Blackwell, XX, TT7, 1367:8-18; Strieter, Ex25, para 201).

[243] Dr. Strieter also opined that the *in vivo* testing in the 083 Patent was "inconclusive" of nintedanib having an anti-fibrotic effect. He asserted that a PSA reviewing the *in vivo* results in the 083 Patent would have been unable to discern if nintedanib was limited to acting as an anti-inflammatory agent, rather than acting in an anti-fibrotic manner (Strieter, Ex25, paras 179-181, 202). As admitted by Dr. Blackwell, by December 2005, the PSA understood that "the preventative model was problematic because drug administration before or simultaneously with initial injury did not simulate the clinical scenario in which IPF patients present most often which is after fibrosis is well established" (Blackwell, XX, TT7, 1357:22-1358:3).

[244] As noted earlier, in December 2005, there was still controversy as to the importance of the inflammatory response in the pathogenesis of IPF and whether a successful therapy for IPF would require treatments directed at both inflammation and fibrosis (Phan, Ex20, para 37; Phan, XX, TT4, 784:19-785:24). A number of types of anti-inflammatory agents had been evaluated as

potential treatments for IPF, with disappointing results. While the theory was that inflammation was the precipitating event that ultimately triggered fibrosis, no anti-inflammatory agent at that time had yet been found to be an effective treatment for IPF (Strieter, Ex25, para 203; Strieter, XC, TT5, 905:18-906:3).

[245] However, while there were these known limitations and criticisms of the bleomycin model, in December 2005 it was still regarded as the "gold standard" (Phan, Ex20, paras 154, 326, 334) and best available option for evaluating compounds as a potential treatment for IPF (Blackwell, Ex38, para 410; Strieter, Ex25, at para 199; Phan, Ex38, paras 37, 334). Indeed, others in the literature were evaluating whether compounds might have therapeutic value in humans based on test results from the bleomycin model preventative regimen (Phan, XX, TT4, 802:6-11, 803:1-3, 804:8-25, referring to Ex20, Sch9-17, 806:1-807:21, referring to Ex20, Sch9-11).

[246] While studies using a delayed treatment version of the bleomycin model had been reported in the literature for other compounds, allowing others to conclude that positive results indicated a reduced fibrotic response (Blackwell, XX, TT7, 1355:25-1357:10, Ex38, SchE, 416, 418, 419), a delayed treatment regimen was not yet widely in use prior to December 2005 (Blackwell, XX, TT7, 1354:27-1355:5; Phan, XX, TT4, 808:22-809:20). Indeed, Dr. Strieter referred to the preventative regimen as the predominant model and one that had been used by his research group on a number of occasions before 2005, while acknowledging that they had never used a delayed treatment regimen (Strieter, XX, TT5, 1014:1-9).

[247] Even today, the therapeutic regimen has not replaced the preventative regimen. Rather, it acts as a supplement to the preventative regimen, which is still used first to determine whether a proposed compound has any effect, followed by the therapeutic regimen to investigate whether the effect is occurring during the fibrotic phase (Blackwell, XC, TT7, 1293:24-1294:24).

[248] In *Raloxifene FC,* Justice Hughes found that the patent disclosure did not give the PSA any more than they would have already had based on the prior art and that it was only the Hong Kong study that made the prediction that already existed from the prior art, a sound prediction. As summarized in *Raloxifene FCA*:

> [9] In assessing the evidence, the Federal Court Judge noted that the parallels between the prior art, specifically Dr. Jordan's paper entitled "*Effects of Anti-Estrogens on Bone in Castrated and Intact Female Rats*", and the disclosure of the '356 Patent are readily apparent. He held that both studies show that raloxifene used in Sprague Dawley ovariectomized rats demonstrates positive effects in respect of bone loss and uterine weight. He noted that the Jordan paper concluded that a long term study on post-menopausal women was warranted. The disclosure in the '356 Patent suggests that such a study on women was underway and that certain results were expected with a long term study to follow (the results of the study are not part of the disclosure of the '356 Patent). The Federal Court Judge therefore, held that the Jordan paper and the '356 Patent disclosure were at the same point, the rat studies were positive, and human studies were warranted. He noted that the '356 Patent simply claimed that raloxifene is an appropriate medicine for humans without any further supporting disclosure (Reasons, paras 105 and 106).
>
> […]
>
> [17] In this respect, the appellant properly accepted that the Hong Kong study was required in order to make the prediction underlying the '356 Patent sound. After taking all of the relevant evidence into consideration, it was open to the Federal Court Judge to find that as of the priority date the prior art Jordan article and the disclosure of the '356 Patent were at the same point given that both studies demonstrated positive effects in respect of bone loss in rats and both concluded that human studies were warranted. In

> particular, the ‘356 Patent did not disclose any more than the Jordan article did, and as such, the person skilled in the art was given, by way of disclosure, no more than such a person already had available in the prior art.

[249] In this case, it cannot be said that the PSA was given nothing by the 083 Patent disclosure. By being given the positive results of testing using the preventative regimen, they were given the data from the leading *in vivo* model, indicating that nintedanib had an inhibitory effect on fibrosis and therefore establishing nintedanib as a compound of interest.

[250] Identifying a compound that was successful in the preventative regimen was not a given; it was known that many compounds had failed to show positive results (Phan, XX, TT4, 801:7-12, 803:10-804:7, discussing Ex20, Sch 9-17:9, 807:22-808:14, discussing Ex20, Sch9-11:811; Strieter, XX, TT5, 1017:13-1018:6, 1019:1-10, discussing Ex28, p. 4697).

[251] Dr. Blackwell described the information obtained from the 083 Patent as providing “a starting point” that held “promise” for “a potential to have some effectiveness in humans”. It was a “potential treatment that … needed a lot of additional information and studies to get even close to being a treatment for humans” (Blackwell, XX, TT7, 1367:26-1369:8).

[252] Dr. Strieter referred to the disclosure as providing “a hypothesis that need[ed] to have a bigger story” and be tested in a human being (Strieter, XC, TT5, 915:10-916:9). He refers in his report to the results as supporting “a hypothesis that needed to be evaluated in further experimental models tied to IPF and early-phase clinical trials in IPF patients” (Strieter, Ex25,

para 41). However, proof of clinical efficacy is not the standard for sound prediction. It is understood that further work may need to be done: *Wellcome Foundation* at para 77.

[253] While Dr. Strieter described the undisclosed information as "add[ing] a level of increased enthusiasm" to the "hypothesis" to take the compound forward, he nonetheless did not consider even the additional work to be sufficient (Strieter, XX, TT5, 1022:4-20). Dr. Strieter was simply of the view that all of the information from the [redacted] should have been disclosed in the 083 Patent (i.e., items (a) to (h) referred to above); a position even JAMP's counsel backed away from by the time of the closing submissions at trial. Dr. Strieter's vantage point was based on his experience at Novartis and the types of disclosures that would be submitted to receive funding and approval to take a drug into the clinic (Strieter, Ex25, paras 205-206; Strieter, XX, TT5, 1033:5-15).

[254] In *Raloxifene*, the additional work that was known and not included in the patent were clinical trial results which significantly advanced the understanding of the efficacy of the drug in question. It was a study that everyone admitted made the prediction sound. Here, the additional work was cumulative assay work that was corroborative in nature. It did not move the needle closer to a prediction of efficacy in humans. The therapeutic regimen results provided further results from the same bleomycin model, with the same underlying limitations. While it provided greater support that results occurred in the fibrotic phase, the relationship between inflammation and fibrosis at the time was not yet fully reconciled.

[255] In his report, Dr. Strieter opined that with the disclosure in the 083 Patent, the inventors' line of reasoning for their prediction that nintedanib would be useful in the treatment of IPF was the following (Strieter, Ex25, para 183):

> (a) Nintedanib is a potent *in vitro* inhibitor of tyrosine kinases (VEGFR, FGFR, and PDFR) that are implicated in the processes that lead to fibrosis. Therefore, inhibition of these kinases will lead to a reduction in fibrosis, which is a hallmark symptom of IPF; and
>
> (b) Nintedanib was observed to lower the formation of fibrosis in the *in vivo* bleomycin-induced rat lung fibrosis model, using the preventative regimen (i.e., treating on days 1 to 21), which is evidence in a mammal that nintedanib is interfering in the processes that lead to fibrosis, and that this would likewise lead to a reduction in fibrosis in human IPF patients.

[256] Taking into consideration the CGK of the PSA in December 2005, including the small number of compounds that had shown positive results in the bleomycin model, and the uncertainty that was inherent for those treating patients with IPF, in my view the disclosure in the 083 Patent and its line of reasoning is sufficient. It provided the PSA with a sound prediction of utility of nintedanib in the treatment of IPF that was more than mere speculation or hypothesis, but one grounded in the accepted *in vivo* model for evaluating potential efficacy.

[257] As such, it is my view that the 083 Patent is not invalid for lack of sound prediction of utility.

D. *Obviousness*

[258] JAMP argues in the alternative that if a lack of sound prediction is not found then the 083 Patent is invalid for obviousness. Section 28.3 of the *Patent Act* requires that the subject-matter

defined by a claim must not have been obvious on the claim date to a PSA having regard to the state of the art.

[259] As a preliminary matter, I note that there is no inherent inconsistency with a finding of sound prediction and lack of obviousness. As recently stated in *Pharmascience* at paragraph 38:

> [38] … There is no necessary inconsistency between the finding, on the one hand, that an idea is sufficiently described in the patent disclosure and the common general knowledge to support a sound prediction that it will be useful (a *prima facie* reasonable inference of utility, per *Eli Lilly Canada Inc. v. Novopharm Limited*, 2010 FCA 197, 85 C.P.R. (4th) 413 at para. 85) and, on the other, that the idea is not sufficiently known in the prior art (including but not limited to the common general knowledge) to lead the PSA directly and without difficulty to the solution taught in the patent.

[260] Indeed, obviousness is a difficult test to satisfy because it necessitates showing that the PSA would have come directly and without difficulty to the invention, without the benefit of hindsight: *Bridgeview Manufacturing Inc v 931409 Alberta Ltd (Central Alberta Hay Centre)*, 2010 FCA 188 [*Bridgeview*] at para 50.

[261] The Supreme Court of Canada in *Sanofi* set out a four-step approach to an analysis of obviousness, as follows (para 67):

> (1) (a) Identify the notional "person skilled in the art";
>
> (b) Identify the relevant common general knowledge of that person;
>
> (2) Identify the inventive concept of the claim in question or if that cannot readily be done, construe it;
>
> (3) Identify what, if any, differences exist between the matter cited as forming part of the "state of the art" and the inventive concept of the claim or the claim as construed;

> (4) Viewed without any knowledge of the alleged invention as claimed, do those differences constitute steps which would have been obvious to the person skilled in the art or do they require any degree of invention?

[262] For areas of endeavour where advances are often won by experimentation, an "obvious to try" analysis may be appropriate to take into consideration at the fourth step of the obviousness inquiry. The critical question is whether it "was more or less self-evident to try to obtain the invention" having regard to the following non-exhaustive factors, while noting that "[m]ere possibility that something might turn up is not enough" (*Sanofi* at paras 66, 68-69):

> 1. Is it more or less self-evident that what is being tried ought to work? Are there a finite number of identified predictable solutions known to persons skilled in the art?
>
> 2. What is the extent, nature and amount of effort required to achieve the invention? Are routine trials carried out or is the experimentation prolonged and arduous, such that the trials would not be considered routine?
>
> 3. Is there a motive provided in the prior art to find the solution the patent addresses?

[263] The Court must be cautious when approaching the obvious to try analysis as it remains as only one factor amongst many that may assist in the obviousness inquiry: *Bristol Myers Squibb Canada Co v Teva Canada Limited*, 2017 FCA 76 [*Atazanavir*] at para 38; *Sanofi* at para 64. The Court favours "an expansive and flexible approach that would include 'any secondary considerations that [will] prove instructive'": *Sanofi* at para 63; *Atazanavir* at para 61. The analysis is to be flexible, contextual, expansive and fact-driven (*Apotex Inc v Pfizer Canada Inc*, 2019 FCA 16 at para 39).

[264] In this case, the PSA and CGK were discussed previously.

[265] The parties agree that the inventive concept that pervades the claims *(Shire* at paras 87, 88) is the use of nintedanib, its esylate salt, or a pharmaceutical composition of the esylate salt of nintedanib, in the prevention or treatment of IPF.

[266] The parties spent only a short amount of time on obviousness during final argument. Dr. Phan opined that there were no differences between the state of the art and the inventive concept. The state of the art relied upon by Dr. Phan for his opinion included:

a) WO 948 and U.S. Patent No. 6,762,180 [US 180], which taught classes of compounds that were useful to treat lung fibrosis (WO 948) and fibrotic diseases (US 180);

b) research that was acknowledged within the disclosure of the 083 Patent, evaluating the inhibition of profibrotic cytokines (*e.g.* TGF-β and PDGF) as a way to suppress the formation of IPF, and the disclosure of nintedanib and its esylate salt, in WO 04/096224 [WO 224] and WO 099, as an inhibitor of tyrosine kinase with *in vitro* tyrosine kinase inhibition results; and

c) knowledge that imatinib (GLEEVEC), a known tyrosine kinase inhibitor, had shown positive results in the bleomycin induced model using the preventative regimen and was being evaluated in a clinical trial for IPF (Phan, Ex20, Sch8, doc35, “Daniels”).

[267] Dr. Phan opined that it was reasonable to infer that nintedanib would be useful to treat IPF from WO 948 and US 180, and the PSA’s knowledge regarding nintedanib’s tyrosine kinase

inhibitory activity. Based on the example of imatinib's testing, he opined that it would have been obvious to try nintedanib in the bleomycin preventative regimen, which was admittedly not very complicated (Park, XX, TT8, 1506:22-1507:6, 1507:27-1509:7), and that there would be a high probability of success.

[268] However, as set out previously, it is my view that the reference to "lung fibrosis" in WO 948 refers to lung fibrosis arising from autoimmune and T-cell mediated diseases, which was not understood to include IPF. Further, even if IPF were considered as encompassed under this umbrella term, in view of the breadth of the teaching in WO 948, the number of diseases that can lead to lung fibrosis, and the diversity of its pathology, the PSA would not have been directed to the use of nintedanib to treat IPF.

[269] I similarly find that US 180, which is even broader than WO 948, would not direct the PSA to the use of nintedanib for the treatment of IPF. US 180 discloses a large class of compounds for treating many hundreds of diseases that span across multiple organ systems (Blackwell, Ex38, paras 195, 272). Only one of those many hundred disease groups is "fibrotic diseases", which would be understood by the PSA as not even restricted to the lung, let alone a specific disease affecting the lung such as IPF.

[270] IPF was poorly understood and many different types of treatment strategies were under investigation with uncertain prospects of success (Blackwell, Ex38, paras 228-229; Strieter, Ex25, para 191). There was no specific motivation for the PSA to turn to nintedanib as a

compound of interest and without recognizing nintedanib as a compound of interest, there would have been no reason for the PSA to look to WO 099 and WO 224.

[271] As highlighted by Dr. Blackwell, no one other than BII and its related companies had pursued nintedanib between the publication of Canadian Patent Application No. 2,387,013 in April 2001 (an application that disclosed nintedanib amongst hundreds of other tyrosine kinase inhibitors) and the 083 Patent, including even after WO 948 was published (Blackwell, Ex38, para 241).

[272] The PSA was looking in different directions. Multiple different kinase signalling pathways had been implicated in fibrosis in animal studies (Blackwell, Ex38, paras 230). Imatinib was the first tyrosine kinase inhibitor to show positive results in the bleomycin preventative regimen and move on to clinical studies, however it had a different kinase inhibition profile to nintedanib (Blackwell, Ex38, paras 233, 243, 400). There was no basis to suggest that there was a class effect (Blackwell, Ex38, para 236). Even Dr. Phan acknowledged that he was looking at other compounds and had not tested any kinase inhibitor in the bleomycin preventative regimen, despite routinely using this bleomycin model to study IPF (Phan, XX, TT4, 860:26-861:9).

[273] There was also significant uncertainty associated with testing potential candidates in the bleomycin model and what results would be obtained as there were many compounds that had failed to reduce bleomycin-induced lung fibrosis when tested in the preventative regimen (Phan,

XX, TT4, 801:7-12, 803:10-804:7, discussing Ex20, Sch 9-17:9, 807:22-808:14, discussing Ex20, Sch9-11:811; Strieter, XX, TT5, 1017:13-1018:6, 1019:1-10, discussing Ex28, p. 4697).

[274] Even if a compound seemed interesting, the evidence indicated that efficacy would not have been self-evident, particularly for a compound with a novel kinase inhibition profile like nintedanib (Blackwell, Ex38, para 240, 242).

[275] The actual course of conduct of the inventors demonstrated that the identification of nintedanib as a compound for use in the treatment of IPF was spurred by decisions made by Park that were not grounded in the state of the art, but rather his specific circumstances and prior work experiences. Indeed, Park only turned to nintedanib because of his specific experiences in the oncology group.

[276] While a general motive existed in 2004 for those working in the field to find a therapeutic treatment for IPF, there was no specific motive to study nintedanib.

[277] Even today, nintedanib remains as one of only two drugs approved for the treatment of IPF (Blackwell, Ex38 para 248).

[278] The FDA designated nintedanib as a breakthrough therapy and orphan drug. The approval was described by Dr. Blackwell as constituting a major shift in clinical practice that affected patient recruitment for clinical trials for other drugs (Blackwell, XC, TT7, 1284:5-1285:4).

[279] In my view, without the benefit of hindsight, it would not have been obvious to the PSA to use nintedanib (or to try nintedanib) as a treatment for IPF. This finding is dispositive of each of the claims. It is my view that the 083 Asserted Claims are not obvious.

IX. **Conclusion and Remedies**

[280] For all of these reasons, I find that the 083 Patent is valid and infringed, but that the 267 Patent is not infringed. As such, the action is allowed in part.

[281] An injunction shall form part of my Judgment as it relates to the 083 Patent only. For the reasons already stated, the injunction shall cover acts relating to the direct infringement of claims 2, 3 and 4 of the 083 Patent, as well as inducement of all of the 083 Asserted Claims.

X. **Costs**

[282] The parties provided preliminary submissions on costs and agreed that costs should be awarded as a lump sum based on a default percentage of 37.5% of reasonable legal fees and 100% of reasonable and necessary disbursements. However, the parties were not aligned on how costs should be awarded if success was divided and requested the opportunity to make submissions on the appropriate allocation of costs in that case, on the basis of an agreed to timetable. As the outcome is divided, the schedule proposed by the parties to make further submissions as to the allocation of costs shall be adopted.

XI. ***Official Languages Act***

[283] While the Plaintiffs have renounced the 24-month stay provided by paragraph 7(1)(d) of the *PMNOC Regulations* in accordance with paragraph 7(5)(b) of the *PMNOC Regulations*, as noted earlier there is another proceeding involving these parties and the same patents relating to 100 mg nintedanib under T-352-24 that is under the 24-month stay to which this decision will be of interest. Similarly, there are also other proceedings under the *PMNOC Regulations* involving the same patents and additional generics, which are pending under T-1336-23 and T-1358-24. In view of these various proceedings, it is my view that the timing of this decision is relevant. To the extent that paragraph 20(1)(a.1) of the *Official Languages Act* R.S.C., 1985, c 31 (4th Supp) [OLA] applies to this decision, it is my view that paragraph 20(2)(b) of the OLA is invoked, justifying immediate release of this decision in English (the language in which the matter was argued) with the translation into French being released at the earliest possible time thereafter.

JUDGMENT IN T-1563-22

THIS COURT'S JUDGMENT is that:

1. The action is granted with respect to the 083 Patent, but dismissed with respect to the 267 Patent.

2. The making, constructing, using and/or selling of JAMP Nintedanib in a strength of 150 mg (of nintedanib) for oral administration in accordance with ANDS No. 262177 would directly infringe claims 2, 3 and 4 of the 083 Patent and induce infringement of claims 1, 2, 3, 4, 5 and 6 of the 083 Patent.

3. JAMP and those acting on behalf or under the authority of JAMP are hereby enjoined from the following acts until December 21, 2025, the date when the 083 Patent expires:

    a) making, constructing, using or selling JAMP Nintedanib in Canada;

    b) offering for sale, marketing or having JAMP Nintedanib marketed in Canada;

    c) importing, exporting, distributing or having JAMP Nintedanib distributed in Canada; and

    d) otherwise infringing or inducing infringement of the 083 Patent.

4. The parties shall provide brief submissions as to the appropriate allocation of costs in accordance with the following schedule:

   a) Within fourteen (14) days of the date of this decision, the Plaintiffs shall make written submissions on costs not to exceed five (5) pages;

   b) Within fourteen (14) days of receipt of the above submissions, the Defendant shall make written responding submissions not to exceed five (5) pages; and,

   c) Within seven (7) days of receipt of the responding submissions, the Plaintiffs shall make written reply submissions not to exceed two (2) pages.

"Angela Furlanetto"
Judge

FEDERAL COURT

SOLICITORS OF RECORD

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