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VON PATENTANWÄLTEN

FICPI AI Patent Drafting Masterclass

16 September 2026

Corinthia Hotel, Budapest, Hungary

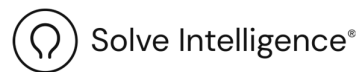
Exercise Materials: Biotechnology



120
YEARS

SINCE 1906

COURSE SPONSORS



AFTERNOON DRAFTING EXERCISE

Cancer-susceptibility gene — drafting from scratch

DELEGATE NOTES

DELEGATE NOTES

This section contains instructions for delegates.
Circulate with the client email, IDF, sequence listing and drawings.

Purpose

This exercise is about learning to use your AI drafting tool to draft a patent application for a biotechnology invention. It is not about the science, and there is no single right answer.

You will be given a client email attaching an invention disclosure form. The task is to draft a patent application — description, claims, and abstract — using your tool, and then review the result.

There is no right answer.

What you get

An email from the client, attaching the invention disclosure form.

The IDF describes: the FICPI1 gene associated with cancer susceptibility; its wild-type sequence (SEQ ID NO: 1) and encoded protein (SEQ ID NO: 2); four mutant alleles with their detection methods; and figures embedded in the disclosure.

A sequence listing (text file) containing SEQ ID NO: 1 (wild-type cDNA) and SEQ ID NO: 2 (encoded polypeptide).

Drawings — two figures showing the gene structure and the mutation detection method.

You will have 50 minutes, working with your own AI drafting tool.

What to do

Using your tool, draft a patent application from the invention disclosure. That is likely to mean:

- drafting a description: technical field, background, summary of the gene and variants, and a detailed description including the detection methods;
- drafting claims — consider isolated nucleic acid claims, polypeptide claims, detection method claims, and kit claims;
- incorporating the sequence listing appropriately — SEQ ID NO references should be used consistently in the description and claims;
- drafting an abstract.

Then review the result. Does the description support the claims? Has the tool stayed within what the disclosure actually says, or has it invented sequences, primer designs, or other detail not provided?

Then discuss

Compare notes with the others: how the tool handled the sequence-dependent claims, whether it used the sequence listing correctly, and what corrections were needed.

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Client Email — Drafting from Scratch

Biotechnology / Life Sciences

16 September 2026

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ADVANCED DELEGATES

Prepared for workshop use. Follow the circulation instructions in this document.

Friday, 15 May 2026 at 10:47:12 British Summer Time

Subject: New Patent Application — Cancer-Susceptibility Gene and Mutation Detection**Date:** Friday, 15 May 2026 at 10:47:12 British Summer Time**From:** Dr Ronelle Goldhouse <ronelle.goldhouse@cetbiosciences.ch>**To:** Dr Newton Ventive <newton.ventive@docketfielding.com>

Attachments:  FICPI_IDF_(Biotech).docx (1.6 MB)  FICPI_Sequence_Listing.txt (13 KB)

Dear Newton,

We are writing to instruct you on a new patent application arising from work in our lab here at CET Biosciences.

We have isolated a human gene — we are calling it FICPI1 — associated with inherited susceptibility to breast and ovarian cancer. We have sequenced the wild-type cDNA and the encoded polypeptide, and identified several mutant alleles found in affected families, together with reliable means of detecting them.

Attached:

1. Invention Disclosure Form (Word)
2. Sequence listing — SEQ ID NO: 1 (wild-type cDNA) and SEQ ID NO: 2 (encoded polypeptide)

We are sorry to trouble you with this now, as we know you are in Budapest at a very important conference this week, but since we need to disclose this remarkable discovery to our external partners by the end of the week, we would be most grateful if you would skip a couple of the wonderful dinners you will be attending and draft us a patent application to protect the invention ahead of the disclosure. We know you have access to some terrific AI drafting tools, so perhaps they will help you be quick enough that you only miss the starter.

Please let us know what else you need from us, and how you would like to proceed.

Kind regards,

Ronelle & Daniel



Dr Ronelle Goldhouse

Chief Scientific Officer

Dr Daniel Huntsworth

CET Biosciences AG

Hauptstrasse 42, 4052 Basel, Switzerland

FICPI AI Patent Drafting Masterclass

Invention Disclosure Form — Drafting from Scratch

Biotechnology / Life Sciences

16 September 2026
Corinthia Hotel, Budapest, Hungary

ADVANCED DELEGATES

Prepared for workshop use. Follow the circulation instructions in this document.

INVENTION DISCLOSURE FORM

Cancer-Susceptibility Gene and Mutation Detection

PROJECT TITLE	Cancer-Susceptibility Gene and Mutation Detection
INVENTORS	Dr Ronelle Goldhouse — <i>Chief Scientific Officer, CET Biosciences AG</i> Dr Daniel Huntsworth — <i>CET Biosciences AG</i>
DATE	29 July 2026
EXTERNAL DATA FILES	<i>FICPI_Sequence_Listing.txt</i> Companion WIPO ST.26 sequence listing — SEQ ID NO: 1 (wild-type cDNA, 5,914 nt) and SEQ ID NO: 2 (encoded polypeptide, 1,863 aa). Figures are included directly within this disclosure.

TITLE OF INVENTION

Cancer-susceptibility gene and mutation detection

SUMMARY OF INVENTION

Our invention is an isolated human gene, which we call FICPI1, associated with inherited susceptibility to breast and ovarian cancer, together with the mutant forms of that gene which we have found in affected families and the means of detecting them.

We have isolated and sequenced the wild-type cDNA, which is 5,914 nucleotides long (SEQ ID NO: 1). Its coding sequence begins at nucleotide 120, its sense codons end at nucleotide 5708, and a termination codon occupies nucleotides 5709 to 5711. It encodes a polypeptide of 1,863 amino acids (SEQ ID NO: 2), containing a C3HC4 zinc-finger motif near the N-terminus and a markedly acidic region towards the C-terminus. All nucleotide positions we give are numbered from nucleotide 1 of SEQ ID NO: 1. We have also seen several transcript forms, which we believe arise by alternative splicing.

In families with a strong history of breast and ovarian cancer we have so far identified four mutant

alleles. One has an additional cytosine at position 5385, shifting the reading frame and altering the encoded sequence from 108 residues before the C-terminus. One has guanine at position 5443, changing the methionine at residue 1775 to arginine. One has nucleotides 189 to 199 deleted, shifting the reading frame from the 24th residue onwards. One has a cytosine-to-thymine substitution at position 4056, converting a glutamine codon to a termination codon and predicted to give a truncated protein lacking the final 551 residues; this substitution also creates a new restriction-enzyme recognition site, so that the allele can be detected by amplification followed by digestion. In each case the mutation was present in relatives carrying the disease-associated haplotype and absent in the non-carriers we tested.

Each of the four mutations lies within a coding exon. We identified them by amplifying the coding exons and their associated splice junctions, either from genomic DNA or from cDNA prepared from lymphocyte RNA, and comparing the amplified sequences with the wild-type sequence. Candidates were then assessed for whether they were likely to disrupt the encoded protein, whether they cosegregated with the disease-associated haplotype within the family, and whether they were rare in control populations. We expect the same approach to identify further mutant alleles.

We confirmed the insertion at position 5385, and the deletion of nucleotides 189 to 199, by sequencing additional family members. We tested for the substitution at position 5443 using an allele-specific oligonucleotide, against both family members and control samples. We detect the substitution at position 4056 by amplifying the relevant region and digesting the product, since the base change creates a new restriction site.

We think susceptibility may also arise from other kinds of alteration — deletions, insertions and point mutations, in coding or in non-coding regions — including changes that introduce a premature stop codon, shift the reading frame, interfere with RNA processing, or reduce expression of the gene. We believe the wild-type protein acts as a tumour suppressor, so we would expect any alteration that impairs its function to predispose to disease.

Because the gene lies on chromosome 17q, primer pairs derived from that region can be used to amplify all or part of the gene by the polymerase chain reaction.

We also regard as part of our invention fragments of the gene of at least 15 nucleotides, primer pairs for amplifying it, methods of detecting a mutant allele by amplifying a portion of the gene and comparing it with SEQ ID NO: 1, and kits containing a primer or an allele-specific probe together with instructions, and, where useful, a restriction enzyme whose recognition site is created or abolished by the mutation, control nucleic acids, and buffers and reagents for amplification or hybridisation. Detection may be by direct sequencing, digestion with a restriction enzyme, allele-specific oligonucleotide hybridisation or single-strand conformation analysis. Germline mutations can be detected in DNA from blood, and somatic mutations in DNA from tumour tissue.

ADVANTAGES OF THE INVENTION

Until now the risk of carrying an inherited predisposition of this kind could only be assessed indirectly. A susceptibility locus had been mapped to chromosome 17q by linkage analysis, but the gene had not been isolated, so testing relied on following marker haplotypes through a family. That approach needs a large kindred with several affected and unaffected members before it yields anything useful, and it says nothing at all about an individual with few or no affected relatives.

Because we now have the isolated gene and the actual sequences of the mutations, a predisposing mutation can be detected directly in a single sample from a single individual, with no family material required. Where a mutation creates or destroys a restriction site, it can be detected simply by amplifying the relevant region and digesting the product, without any sequencing at all. Testing is therefore quicker, cheaper and applicable to far more people than the linkage-based approach it replaces.

FIGURES AND ENCLOSURES

Figure 1 shows the single-nucleotide insertion at position 5385 and its cosegregation within the family. Sequences from carriers of the disease-associated haplotype show the additional cytosine, while the sequence from a non-carrier shows the wild type.

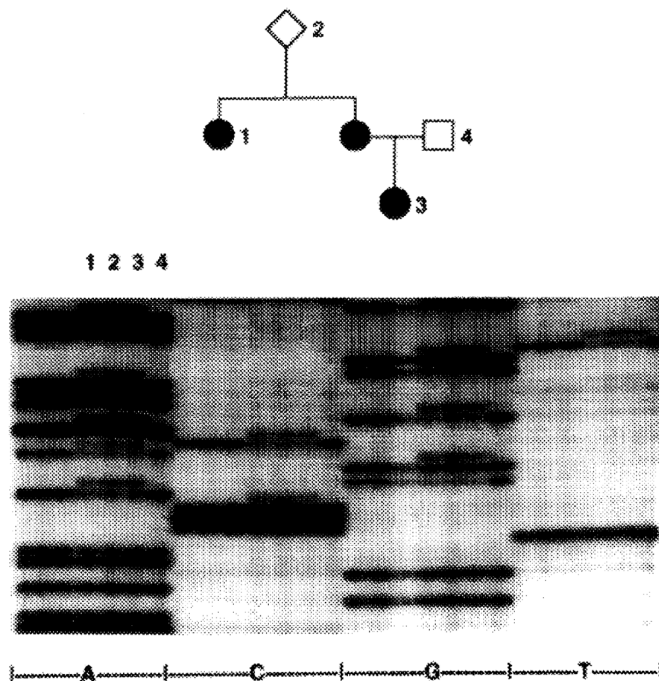


Figure 1

Figure 2 shows restriction analysis of amplification products from individual family members, alongside

molecular-weight markers. Products from those carrying the disease-associated haplotype are heterozygous for the newly created restriction site and give both cut and uncut products, whereas products from those tested who do not carry it remain uncut.

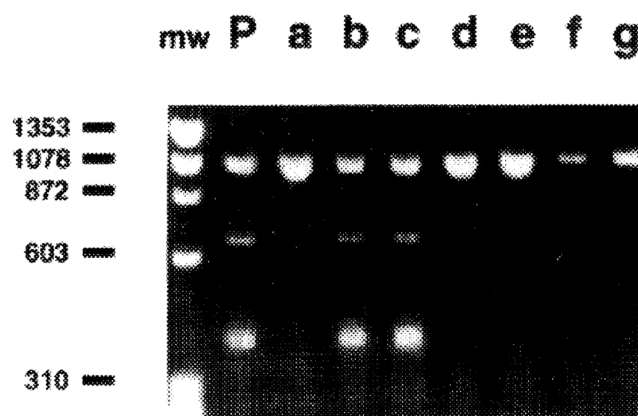


Figure 2

We also attach the sequences of the wild-type cDNA (SEQ ID NO: 1) and of the encoded polypeptide (SEQ ID NO: 2).

MOTIVATION FOR THE INVENTION

We had spent several years mapping a breast and ovarian cancer susceptibility locus to chromosome 17q, but without the gene itself we could tell families very little: risk could only be inferred from marker haplotypes in large kindreds, and individuals from small families could not be helped at all. We wanted to isolate the gene, find the mutations that actually cause the predisposition, and turn that into a test which could be run on a single blood sample.

POSSIBLE COMMERCIAL APPLICATIONS OF INVENTION

The most immediate application is a diagnostic service. Testing blood samples for the mutant alleles we have identified would allow individuals from affected families to learn whether they carry a predisposing mutation, and would allow those who do to be offered increased surveillance. We would expect to offer this as a laboratory service and to license it to other testing laboratories.

We also envisage selling detection kits containing the appropriate primers or allele-specific probes together with instructions, for use in hospital and reference laboratories. Testing tumour tissue for somatic mutations may have further value in diagnosis and prognosis.



Hauptstrasse 42, 4052 Basel, Switzerland

Beyond testing, the isolated gene and its encoded polypeptide are of interest as research reagents and as a starting point for therapeutic work, since the wild-type protein appears to act as a tumour suppressor.

FICPI AI Patent Drafting Masterclass

Sequence Listing — Drafting from Scratch

Biotechnology / Life Sciences

16 September 2026

Corinthia Hotel, Budapest, Hungary

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Sequence Listing Information:

DTD Version: V1_3
File Name: FICPI AI Drafting Masterclass (Biotech).xml
Software Name: WIPO Sequence
Software Version: 2.3.0
Production Date: 2026-07-29

General Information:

Current application / Applicant file reference: CET-G5/BIO/2026-001
Applicant name: CET Biosciences AG
Applicant name / Language: en
Inventor name: Ronelle Goldhouse
Inventor name / Language: en
Invention title: CANCER-SUSCEPTIBILITY GENE AND MUTATION DETECTION (en)
Sequence Total Quantity: 2

Sequences:

Sequence Number (ID): 1
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 > organism, Homo sapiens
- CDS, 120..5711
 > protein_id, 2
 > translation,

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5460	tcagggggct	agaaatctgt	tgctatgggc	ccttcaccaa	catgcccaca	gatcaactgg
5520	aatggatggt	acagctgtgt	ggtgcttctg	tggtgaagga	gctttcatca	ttcacccttg
5580	gcacaggtgt	ccaccaatt	gtggttgtgc	agccagatgc	ctggacagag	gacaatggct
5640	tccatgcaat	tgggcagatg	tgtgaggcac	ctgtggtgac	ccgagagtgg	gtgttggaca
5700	gtgtagcact	ctaccagtgc	caggagctgg	acacctacct	gataccccag	atccccaca
5760	gccactactg	actgcagcca	gccacaggta	cagagccaca	ggaccccaag	aatgagctta
5820	caaagtggcc	tttccaggcc	ctgggagctc	ctctcactct	tcagtccttc	tactgtcctg
5880	gctactaaat	attttatgta	catcagcctg	aaaaggactt	ctggctatgc	aagggtcctt
5914	taaagatttt	ctgcttgaag	tctcccttgg	aaat		

Sequence Number (ID): 2

Length: 1863

Molecule Type: AA

Features Location/Qualifiers:

- source, 1..1863
 > mol_type, protein
 > organism, Homo sapiens

Residues:

	MDLSALRVEE	VQNVINAMQK	ILECPICLEL	IKEPVSTKCD	HIFCKFCMLK	LLNQKKGPSQ	60
	CPLCKNDITK	RSLQESTRFS	QLVEELLKII	CAFQLDTGLE	YANSYNFAKK	ENNSPEHLKD	
120	EVSIIQSMGY	RNRAKRLQSQ	EPENPSLQET	SLSVQLSNLG	TVRTLRTKQR	IQPQKTSVYI	
180	ELGSDSSEDT	VNKATYCSVG	DQELLQITPQ	GTRDEISLDS	AKKAACEFSE	TDVTNTEHHQ	
240	PSNNDLNTTE	KRAAERHPEK	YQGSSVSNLH	VEPCGTNTHA	SSLQHENSSL	LLTKDRMNVE	
300	KAFCNKSQK	PGLARSQHNR	WAGSKETCND	RRTPSTEEKV	DLNADPLCER	KEWNKQKLPC	
360	SENPRDTEDEV	PWITLNSSIQ	KVNEWFSRSD	ELLGSDSDSHD	GESESNAKVA	DVLDVLNEVD	
420	EYSGSSEKID	LLASDPHEAL	ICKSERVHSK	SVESNIEDKI	FGKTYRKKAS	LPNLSHVTEN	
480	LIIGAFVTEP	QIIQERPLTN	KLKRKRRPTS	GLHPEDFIKK	ADLAVQKTPE	MINQGTNQTE	
540	QNGQVMNITN	SGHENKTKGD	SIQNEKNPNP	IESLEKESAF	KTKAEPISSS	ISNMELELNI	
600	HNSKAPKKNR	LRRKSSTRHI	HALELVSRN	LSPPNCTELQ	IDSCSSSEEI	KKKKYNQMPV	
660	RHSRNLQLME	GKEPATGAKK	SNKPNEQTSK	RHSDTFPEL	KLTNAPGSFT	KCSNTSELKE	
720	FVNPSLPREE	KEEKLETVKV	SNNAEDPKDL	MLSGERVLQT	ERSVESSSIS	LVPGTDYGTQ	
780	ESISLLEVST	LGKAKTEPNK	CVSQCAAFEN	PKGLIHGCSK	DNRNDTEGFK	YPLGHEVNHS	
840	RETSIEMEES	ELDAQYLQNT	FKVSKRQSFA	PFSNPGNAEE	ECATFSAHSG	SLKKQSPKVT	
900	FECEQKEENQ	GKNESNIKPV	QTVNITAGFP	VVGQKDKPVD	NAKCSIKGGS	RFCLSSQFRG	
960	NETGLITPNK	HGLLQNPYRI	PPLFPIKSFV	KTKCKKNLLE	ENFEEHSMSP	EREMGNENIP	
1020	STVSTISRNN	IRENVFKEAS	SSNINEVGSS	TNEVGSSINE	IGSSDENIQA	ELGRNRGPKL	
1080	NAMLRGLVLQ	PEVYQQLPG	SNCKHPEIKK	QEYEEVVQTV	NTDFSPYLIS	DNLEQPMGSS	
1140	HASQVCSETP	DDLDDGEIK	EDTSFAENDI	KESSAVFSKS	VQKGELSRSP	SPFTHTHLAQ	
1200	GYRRGAKKLE	SSEENLSSD	EELPCFQHLL	FGKVNNIPSQ	STRHSTVATE	CLSKNTEENL	
1260	LSLKNSLNDC	SNQVILAKAS	QEHHLSEETK	CSASLFSSQC	SELEDLTANT	NTQDPFLIGS	
1320	SKQMRHQSES	QGVGLSDKEL	VSDDEERGTG	LEENNQEEQS	MDSNLGEAAS	GCESETSVSE	
1380							

1440	DCSGLSSQSD	ILTTQQRDTM	QHNLIKLQQE	MAELEAVLEQ	HGSQPSNSYP	SIISDSSALE
1500	DLRNPEQSTS	EKAVLTSQKS	SEYPISQNPE	GLSADKFEVS	ADSSTSKNKE	PGVERSSPSK
1560	CPSLDDRWYM	HSCSGSLQNR	NYPSQEELIK	VVDVEEQQLE	ESGPHDLTET	SYLPRQDLEG
1620	TPYLESGISL	FSDDPESDPS	EDRAPESARV	GNIPSSTSAL	KVPQLKVAES	AQSPAAAHTT
1680	DTAGYNAMEE	SVSREKPELT	ASTERVNKRM	SMVVSGLTPE	EFMLVYKFAR	KHHITLTNLI
1740	TEETTHVVMK	TDAEFVCERT	LKYFLGIAGG	KWVVSYFWVT	QSIKERKMLN	EHDFEVRGDV
1800	VNGRHHQGPK	RARESQDRKI	FRGLEICCYG	PFTNMPTDQL	EWMVQLCGAS	VVKELSSFTL
1860	GTGVHPIVVV	QPDAWTEDNG	FHAIGQMCEA	PVVTREWVLD	SVALYQCQEL	DTYLIPQIPH
1863	SHY					
END						