EPMC 22.04 release review

April 25, 2022

[34]:
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from pyspark.sql import DataFrame, SparkSession, Window
from pyspark.sql.types import DoubleType, IntegerType, StringType, StructType,␣
 ↪StructField
import pyspark.sql.functions as F

spark = (SparkSession.builder
         .master('local[*]')
         .config("spark.driver.memory", "15g")
         .appName('spark')
         .getOrCreate())
```

[35]:
```
new_cooc_path = 'gs://open-targets-pre-data-releases/22.04/output/
 ↪literature-etl/parquet/cooccurrences'
old_cooc_path = 'gs://open-targets-pre-data-releases/22.02.4/output/literature/
 ↪parquet/cooccurrences'
```

[20]:
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new_cooc_df = (
    spark.read.parquet(new_cooc_path)
    .filter(F.col('type') == 'GP-DS')
    .select('pmid', 'year', 'month', 'section', 'text', F.col('label1').
 ↪alias('target'), F.col('label2').alias('disease'))
)
new_cooc_df = new_cooc_df.select([F.col(c).alias("new_" + c) for c in␣
 ↪new_cooc_df.columns])
old_cooc_df = (
    spark.read.parquet(old_cooc_path)
    .filter(F.col('type') == 'GP-DS')
    .select('pmid', 'year', 'month', 'section', 'text', F.col('label1').
 ↪alias('target'), F.col('label2').alias('disease'))
)
old_cooc_df = old_cooc_df.select([F.col(c).alias("old_" + c) for c in␣
 ↪old_cooc_df.columns])

print(new_cooc_df.first())
new_cooc_df.printSchema()
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Row(new_pmid='10066760', new_year=1999, new_month=3, new_section='abstract',
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new_text='Snake venom and mammalian secreted phospholipases A2 (sPLA2s) have
been associated with toxic (neurotoxicity, myotoxicity, etc.), pathological
(inflammation, cancer, etc.), and physiological (proliferation, contraction,
secretion, etc.)processes.', new_target='sPLA2s', new_disease='cancer')
root
 |-- new_pmid: string (nullable = true)
 |-- new_year: integer (nullable = true)
 |-- new_month: integer (nullable = true)
 |-- new_section: string (nullable = true)
 |-- new_text: string (nullable = true)
 |-- new_target: string (nullable = true)
 |-- new_disease: string (nullable = true)
```

[ ]:
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comparison_df = new_cooc_df.join(old_cooc_df, new_cooc_df['new_pmid'] ==␣
↪old_cooc_df['old_pmid'], how='inner').distinct()

print(comparison_df.first())
comparison_df.printSchema()
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Row(new_pmid='22883923', new_year=2012, new_month=1, new_section='abbr',
new_text="A : beta-amyloid peptide; AD: Alzheimer's disease; CSF: cerebrospinal
fluid; ISF: interstitial fluid.", new_target='beta-amyloid peptide',
new_disease='AD', old_pmid='22883923', old_year=2012, old_month=1,
old_section='intro', old_text="Autosomal-dominant, early-onset Alzheimer's
disease (AD) is due to mutations in the processing of the amyloid precursor
protein (APP), resulting in excess production of the 42-amino acid form of the
beta-amyloid peptide (A 1-42).", old_target='amyloid precursor protein',
old_disease="Alzheimer's disease")
root
 |-- new_pmid: string (nullable = true)
 |-- new_year: integer (nullable = true)
 |-- new_month: integer (nullable = true)
 |-- new_section: string (nullable = true)
 |-- new_text: string (nullable = true)
 |-- new_target: string (nullable = true)
 |-- new_disease: string (nullable = true)
 |-- old_pmid: string (nullable = true)
 |-- old_year: integer (nullable = true)
 |-- old_month: integer (nullable = true)
 |-- old_section: string (nullable = true)
 |-- old_text: string (nullable = true)
 |-- old_target: string (nullable = true)
 |-- old_disease: string (nullable = true)
```

[26]:
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comparison_w_section_df = (
    new_cooc_df.withColumnRenamed('new_pmid', 'pmid').
 ↪withColumnRenamed('new_section', 'section')
    .join(
        old_cooc_df.withColumnRenamed('old_pmid', 'pmid').
 ↪withColumnRenamed('old_section', 'section'),
        on=['pmid', 'section'], how='inner')
    .distinct()
)

comparison_w_section_df.first()
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[26]:
```
Row(pmid='10774451', section='abstract', new_year=2000, new_month=1,
new_text='The clinical gene therapy trials for adenosine deaminase (ADA)
deficiency have defined both the potential benefits and the present limitations
of gene therapy with hematopoietic stem cells (HSC).', new_target='adenosine
deaminase', new_disease='(ADA) deficiency', old_year=2000, old_month=1,
old_text='The clinical gene therapy trials for adenosine deaminase (ADA)
deficiency have defined both the potential benefits and the present limitations
of gene therapy with hematopoietic stem cells (HSC).', old_target='adenosine
deaminase', old_disease='(ADA) deficiency')
```

[28]:
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tmp = (
    comparison_w_section_df
    .withColumn('new_assoc', F.concat(F.col('new_target'), F.lit('|'), F.
 ↪col('new_disease')))
    .withColumn('old_assoc', F.concat(F.col('old_target'), F.lit('|'), F.
 ↪col('old_disease')))
    .groupBy('pmid', 'section')
    .agg(
        F.collect_set('new_assoc'),
        F.collect_set('old_assoc'),
        F.collect_set('new_text'),
        F.collect_set('old_text'),
    )
)
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[31]:
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tmp = (
    tmp.withColumn('novel_assocs', F.array_except('collect_set(new_assoc)',␣
 ↪'collect_set(old_assoc)'))
    .withColumn('missed_assocs', F.array_except('collect_set(old_assoc)',␣
 ↪'collect_set(new_assoc)'))
)
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[38]:
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# Ten examples of publications with missing associations and how their text␣
 ↪looks like
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```python
tmp.filter(F.size('missed_assocs') > 0).show(10, False, True)
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 pmid                   | 10723113
 section                | abstract
 collect_set(new_assoc) | [PiMS|FMD, alpha 1-antitrypsin|FMD]
 collect_set(old_assoc) | [PiMS|FMD, alpha 1-AT|FMD, alpha 1-antitrypsin|FMD]
 collect_set(new_text)  | [However, despite FMD being three times less common in
males than females, and carotid artery dissection being a rare occurrence, a
male with PiMS deficiency phenotype presented with internal carotid artery
dissection and had bilateral renal artery FMD., A rare association between FMD
and alpha 1-antitrypsin (alpha 1-AT) deficiency has been reported.]
 collect_set(old_text)  | [However, despite FMD being three times less common in
males than females, and carotid artery dissection being a rare occurrence, a
male with PiMS deficiency phenotype presented with internal carotid artery
dissection and had bilateral renal artery FMD., A rare association between FMD
and alpha 1-antitrypsin (alpha 1-AT) deficiency has been reported., We compared
the alpha 1-AT phenotype distribution in 83 patients with renal arterial FMD
with those published for Australian populations. alpha 1-AT phenotyping was
performed by isoelectric focusing between pH 4.2 and pH 4.9 on polyacrylamide
gels with PiM1M2, PiFM (non-deficiency alleles), PiMS and PiMZ (deficiency
alleles) markers.]
 novel_assocs           | []
 missed_assocs          | [alpha 1-AT|FMD]
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pmid | 10751347
section | discuss
collect_set(new_assoc) | [fgl2|viral hepatitis, thrombin receptor|hepatitis, fgl2|hepatitis, prothrombinase|viral hepatitis]
collect_set(old_assoc) | [TNF|hepatic failure, fgl2|viral hepatitis, fgl2|hepatic failure, thrombin receptor|hepatitis, fgl2|hepatitis, TNF- |hepatic failure, prothrombinase|hepatitis, tumor necrosis factor- |hepatic failure, prothrombinase|viral hepatitis]
collect_set(new_text) | [In favor of this, is the recent report demonstrating increased expression of the thrombin receptor in patients with fulminant hepatitis.28, Fulminant murine hepatitis and fgl2 gene expression were shown to be genetically linked and it is likely that host genetic factors are also important in human fulminant hepatitis.19, Having established this relationship, the provocative and exciting findings of successful abrogation of the lethal effects of the fgl2 gene in murine fulminant viral hepatitis offers the potential for developing methods for attenuating the disease in man., The second is the co-localization of the HFGL2 prothrombinase gene expression in macrophages and fibrin deposition in sinusoids with microvascular thrombosis and hepatocellular necrosis in the most hepatitic lesions in fulminant viral hepatitis., The murine fgl2 gene has been cloned, sequenced, and characterized in this laboratory.2, 4, 14, 15 Recent sequential studies showed that the development of fulminant viral hepatitis always followed the same pattern: initiation by viral-induced up-regulation of the fgl2 prothrombinase gene with focal deposits of fibrin in sinusoids and accumulation of inflammatory cells with a predominance of neutrophils and macrophages and focal individual liver cell necrosis.]
collect_set(old_text) | [Progression ensued by further fibrin deposition and

arrest of sinusoidal blood flow leading to the rapid development of confluent multicellular hepatic necrosis resulting in fulminant hepatic failure and death in 4 days.4 There is strong evidence for implicating fgl2 prothrombinase as pivotal in the pathogenesis of this disease in the mouse model: levels of this prothrombinase activity correlate with the severity of the disease;16, 17 and there is concordance between expression of fgl2 prothrombinase in the liver with fibrin deposition; and neutralizing antibodies attenuate the pathological and clinical manifestations.5 Indeed, it is not the viral load that determines the occurrence of massive necrosis but the viral induced up-regulation of the fgl2 gene which initiates the pathological process.5, 18 Although, treatment with neutralizing antibodies to the fgl2 gene abrogates the disease, a high viral load persists.5 Fulminant murine hepatitis and fgl2 gene expression were shown to be genetically linked and it is likely that host genetic factors are also important in human fulminant hepatitis.19, In favor of this, is the recent report demonstrating increased expression of the thrombin receptor in patients with fulminant hepatitis.28, Having established this relationship, the provocative and exciting findings of successful abrogation of the lethal effects of the fgl2 gene in murine fulminant viral hepatitis offers the potential for developing methods for attenuating the disease in man., Of particular relevance to this study, are recent studies of murine viral hepatitis caused by mouse Coronavirus (murine hepatitis virus type 3, MHV-3) in susceptible strains which is an excellent animal model for studying the pathogenesis of fulminant viral hepatitis.3 Especially important is that MHV-3 infection in susceptible BALB/cJ mice causes the de novo synthesis of a unique procoagulant fgl2 prothrombinase by macrophages.2 The murine fgl2 gene has been cloned, sequenced, and characterized in this laboratory.2, 4, 14, 15 Recent sequential studies showed that the development of fulminant viral hepatitis always followed the same pattern: initiation by viral-induced up-regulation of the fgl2 prothrombinase gene with focal deposits of fibrin in sinusoids and accumulation of inflammatory cells with a predominance of neutrophils and macrophages and focal individual liver cell necrosis., The second is the co-localization of the HFGL2 prothrombinase gene expression in macrophages and fibrin deposition in sinusoids with microvascular thrombosis and hepatocellular necrosis in the most hepatitic lesions in fulminant viral hepatitis., Other inflammatory mediators including tumor necrosis factor- (TNF-), interleukin-1 (IL-1), and reactive oxygen intermediates have also been implicated in the pathogenesis of organ failure.29, 30 Treatment of rats with recombinant neutralizing antibody to TNF- has been shown to protect against hypotension, hypothermia, and mortality of Gram-negative sepsis.31, 32 Furthermore, the mortality associated with endotoxin shock was reduced with an IL-1 receptor antagonist.33 We have previously reported that IL-1 and TNF- induce endothelial cell transcription of fgl2 linking cytokines and induction of coagulation.18 However, antibodies to TNF or IL-1 were unable to prevent the hepatic necrosis caused by MHV-3.3 The role of the immune coagulation system in fulminant hepatic failure is controversial.33 Mori et al34 have reported that fibrin is a classical feature but this has not been substantiated by others.]

novel_assocs	[]
missed_assocs	[TNF|hepatic failure, fgl2|hepatic failure,

TNF- |hepatic failure, prothrombinase|hepatitis, tumor necrosis factor- |hepatic failure]

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 pmid                   | 10937811
 section                | abstract
 collect_set(new_assoc) | [prothrombin|ulcerative colitis, factor V
Leiden|Crohn's disease, factor V Leiden|thrombophilia, prothrombin|UC, factor V
Leiden|UC, prothrombin|Crohn's disease, factor V Leiden|ulcerative colitis]
 collect_set(old_assoc) | [prothrombin|ulcerative colitis, factor V Leiden|IBD,
factor V Leiden|Crohn's disease, prothrombin|IBD, factor V Leiden|thrombophilia,
prothrombin|UC, factor V Leiden|UC, prothrombin|Crohn's disease, factor V
Leiden|ulcerative colitis, Factor V Leiden|IBD]
 collect_set(new_text)  | [The aim of the present paper was to study the
prevalence of the two most important causes of inherited thrombophilia: factor V
Leiden and the G20210A prothrombin-gene mutation in patients with Crohn's
disease (CD) and ulcerative colitis (UC).]
 collect_set(old_text)  | [One out of 52 IBD patients (1.9%) and three out of
156 control subjects (1.9%) were heterozygous for factor V Leiden., In the
subgroup of IBD patients with previous thrombotic events, only one patient was
heterozygous for the prothrombin-gene mutation., One IBD patient (1.9%) and four
healthy controls (2.6%) were heterozygous for the prothrombin-gene mutation.,
Factor V Leiden and the G20210A prothrombin-gene mutation do not seem to play a
major role in the pathogenesis of IBD or be associated with an increased
incidence of thrombotic complications, but with limited data., The aim of the
present paper was to study the prevalence of the two most important causes of
inherited thrombophilia: factor V Leiden and the G20210A prothrombin-gene
mutation in patients with Crohn's disease (CD) and ulcerative colitis (UC).]
 novel_assocs           | []
 missed_assocs          | [factor V Leiden|IBD, prothrombin|IBD, Factor V
Leiden|IBD]
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 pmid                   | 11589380
 section                | abstract
 collect_set(new_assoc) | [CD26|Crohn disease, CD26|UC, CD26|ulcerative colitis]
 collect_set(old_assoc) | [CD26|IBD, CD26|Crohn disease, CD26|UC,
CD26|inflammatory bowel disease, CD26|ulcerative colitis]
 collect_set(new_text)  | [Serum DP IV activity and CD26 (DP IV)-positive
peripheral blood lymphocytes were measured in 110 patients with IBD (Crohn
disease (CD): n = 63, ulcerative colitis (UC): n = 47)., Furthermore, patients
with IBD had higher numbers of CD26-positive cells coexpressing CD25 and a
higher surface expression of CD26 (DP IV) (mean fluorescence intensity, mean
57.1 (CD) and 59.8 (UC) versus 29.9 (HC), P < 0.001).]
 collect_set(old_text)  | [We hypothesized that the activity of DP IV in serum
and expression of CD26/DP IV in lymphocytes may be altered in patients with
inflammatory bowel disease (IBD)., Serum DP IV activity and CD26 (DP
IV)-positive peripheral blood lymphocytes were measured in 110 patients with IBD
(Crohn disease (CD): n = 63, ulcerative colitis (UC): n = 47)., Furthermore,
patients with IBD had higher numbers of CD26-positive cells coexpressing CD25
and a higher surface expression of CD26 (DP IV) (mean fluorescence intensity,
mean 57.1 (CD) and 59.8 (UC) versus 29.9 (HC), P < 0.001).]
 novel_assocs           | []
 missed_assocs          | [CD26|IBD, CD26|inflammatory bowel disease]
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 pmid                   | 11805148
 section                | other
 collect_set(new_assoc) | [C1s|systemic lupus erythematosus,
IgA|antiphospholipid syndrome, C1r|systemic lupus erythematosus,
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IgM|antiphospholipid syndrome]
 collect_set(old_assoc) | [aPL|APS, aPL|antiphospholipid syndrome]
 collect_set(new_text)  | [Hypocomplementemia with low C1s-C1r inhibitor complex
in systemic lupus erythematosus., Induction of thrombosis in a mouse model by
IgG, IgM and IgA immunoglobulins from patients with antiphospholipid syndrome.]
 collect_set(old_text)  | [Abbreviations used in this paper: aCL,
anticardiolipin; aPL, antiphospholipid; aPL-IgG, human IgG containing aPL
antibody; APS, antiphospholipid syndrome;  2GPI,  2-glycoprotein I; Crry,
complement receptor 1-related gene/protein y; MAC, membrane attach complex.]
 novel_assocs           | [C1s|systemic lupus erythematosus,
IgA|antiphospholipid syndrome, C1r|systemic lupus erythematosus,
IgM|antiphospholipid syndrome]
 missed_assocs          | [aPL|APS, aPL|antiphospholipid syndrome]
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 pmid                   | 12486099
 section                | discuss
 collect_set(new_assoc) | [CD8|Crohn's disease]
 collect_set(old_assoc) | [CD8|IBD, CD8|Crohn's disease, CD4|IBD, TNF|IBD,
Tpl2|IBD, Tnf|IBD, JNK2|IBD]
 collect_set(new_text)  | [Interestingly, enhanced peripheral blood T cell
cytotoxicity attributed to CD8+ lymphocytes has been detected in patients with
Crohn's disease (37, 38).]
 collect_set(old_text)  | [The Cellular Interactions Governing TNF-mediated
IBD., Signaling Requirements in TNF-driven IBD., In molecular terms, genetic
inactivation experiments revealed that at least two kinases, Tpl2 and JNK2,
promote, whereas a third one, MK2, opposes the induction of IBD, indicating that
regardless of pleiotropy of cellular processes that these signals modulate, they
are likely candidates for pharmaceutical targeting., In this study, analysis of
the impact of genetic ablation of MK2, JNK2, or Tpl2 kinases in the IBD
pathology developing in the Tnf Δ AREmice led to differential results on the
development of intestinal inflammation., In the absence of p38/MK2 signaling,
TNF-dependent IBD is exacerbated., Interestingly, enhanced peripheral blood T
cell cytotoxicity attributed to CD8+ lymphocytes has been detected in patients
with Crohn's disease (37, 38)., In contrast, the aberrant TNF load in our model
shapes up a hyperactive CD8+ lymphocytic response, which is detectable in the
periphery of Tnf Δ AREmice even before the onset of IBD as suggested by our MLR
assays., Regardless of the aetiopathogenic event(s) that initiate IBD, aberrant
TNF production and function appear to be centrally involved in the pathogenesis
of both the human disease and its animal models., Development of IBD in the
absence of JNK2 was significantly attenuated., The absence of the ARE from the
Tnf gene in the Tnf Δ AREmice provided us with the opportunity to examine the
involvement of specific effector signals delivered by TNF in IBD in the absence
of a parallel impact of such signals on TNF biosynthesis itself., Our data
provide new mechanistic insights into the pathophysiology of IBD and identify
Tpl2 and JNK2 kinases as potential targets for therapy., The observation that
effector TNF signaling on bone marrow-derived cells is fully sufficient to drive
the development of pathology supports the hemopoietic origin of TNF targets in
IBD., Most importantly, our data demonstrate a dominant role for Th1-driven CD8+
T cells as IBD effectors in Tnf ΔARE mice., From our data it is clear that T
lymphocyte-derived TNF is also sufficient, albeit less efficacious, to drive the
pathogenic events leading to IBD., The action of TNF in IBD, as exemplified by
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our studies, does not seem to be solely of an innate proinflammatory character
but may extend in the modulation of a specific lymphocytic response., The data
presented in this report suggest that, in cellular terms, a critical element in
TNF-induced Crohn's-like IBD is the activation of a Th1-driven pathogenic CD8+ T
cell response., An additional interesting observation in this study is that
deficiency in the CD4+ T cell compartment exacerbates IBD in Tnf ΔARE mice,
suggesting that CD4+ T cells may regulate pathogenic CD8+ T cell responses in
this model.]
 novel_assocs           | []
 missed_assocs          | [CD8|IBD, CD4|IBD, TNF|IBD, Tpl2|IBD, Tnf|IBD,
JNK2|IBD]
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 pmid                   | 12847137
 section                | other
 collect_set(new_assoc) | [PD-L1|tumor, NOD|Cancer, PD-1|experimental autoimmune
encephalomyelitis, CD8|diabetes, NOD|diabetes, NOD|AIDS, B7-H1|Tumor,
NOD|Diabetes, B7-2|diabetes, B7-1|diabetes]
 collect_set(old_assoc) | [CTLA-4|EAE, CTLA-4|experimental autoimmune
encephalomyelitis]
 collect_set(new_text)  | [Tumor-associated B7-H1 promotes T-cell apoptosis: a
potential mechanism of immune evasion., Critical role of the programmed death-1
(PD-1) pathway in regulation of experimental autoimmune encephalomyelitis., CD8
T cell clones from young nonobese diabetic (NOD) islets can transfer rapid onset
of diabetes in NOD mice in the absence of CD4 cells., NOD Mice and Related
Strains: Research Applications in Diabetes, AIDS, Cancer and Other Diseases.,
Involvement of PD-L1 on tumor cells in the escape from host immune system and
tumor immunotherapy by PD-L1 blockade., Early expression of antiinsulin
autoantibodies of humans and the NOD mouse: evidence for early determination of
subsequent diabetes., Differential effects of anti-B7-1 and anti-B7-2 monoclonal
antibody treatment on the development of diabetes in the nonobese diabetic
mouse.]
 collect_set(old_text)  | [Abbreviations used in this paper: CTLA-4, cytotoxic T
lymphocyte-associated antigen 4; EAE, experimental autoimmune encephalomyelitis;
NOD, nonobese diabetic; PD-1, programmed death-1.]
 novel_assocs           | [PD-L1|tumor, NOD|Cancer, PD-1|experimental autoimmune
encephalomyelitis, CD8|diabetes, NOD|diabetes, NOD|AIDS, B7-H1|Tumor,
NOD|Diabetes, B7-2|diabetes, B7-1|diabetes]
 missed_assocs          | [CTLA-4|EAE, CTLA-4|experimental autoimmune
encephalomyelitis]
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 pmid | 12915891
 section | results
 collect_set(new_assoc) | [VEGF|ALL]
 collect_set(old_assoc) | [BCP|ALL, caspase-3|ALL, VEGF|ALL]
 collect_set(new_text) | [Modulation of VEGF secretion by Aplidin on ALL cell lines, Therefore, we evaluated whether a block in VEGF secretion in ALL-PO cells occurs as previously seen in Molt-4 cells (Broggini et al, 2003)., By using ELISA assay, we tested the level of VEGF secretion in the four leukaemia cell lines used and found that only ALL-PO secrete detectable amounts of the growth factor.]
 collect_set(old_text) | [ND=not determined., the cytotoxic effect of Aplidin on BCP-ALL cells was dose dependent., Data reported in Figure 7Figure 7Autoradiography of RNAse protection assay on ALL-PO cells treated with 20 nM Aplidin performed at different time intervals after drug-washout. show that

after different times from drug-washout, Aplidin downregulated the level
expression of VEGF mRNA in ALL-PO cells., Modulation of VEGF secretion by
Aplidin on ALL cell lines, Therefore, we evaluated whether a block in VEGF
secretion in ALL-PO cells occurs as previously seen in Molt-4 cells (Broggini et
al, 2003)., As previously reported on other cell type (Garcia-Fernandez et al,
2002), Aplidin was found to induce apoptosis in a caspase-3-dependent manner
(Figure 5Figure 5Detection of active caspase-3 in ALL cells by flow cytometric
analysis., By using ELISA assay, we tested the level of VEGF secretion in the
four leukaemia cell lines used and found that only ALL-PO secrete detectable
amounts of the growth factor.]
 novel_assocs | []
 missed_assocs | [BCP|ALL, caspase-3|ALL]
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 pmid                  | 14583772
 section               | results
 collect_set(new_assoc) | [IGFBP-1|cancer]
 collect_set(old_assoc) | [IGFBP-1|diabetes mellitus, IGF1|diabetes mellitus,
IGFBP-1|cancer, IGF-I|diabetes mellitus, IGFBP-3|diabetes mellitus]
 collect_set(new_text)  | [Overall, IGFBP-1 levels were higher among the cancer
cases than among control women.]
 collect_set(old_text)  | [We found no clear evidence of an interaction between
the use of oral contraceptives levels of IGF-I, IGFBPs, and insulin (Table 7
Table 7Serum levels of IGF-I, IGFBP-1, IGFBP-3, insulin, and risk of endometrial
cancer, according to use of oral contraceptives (OC, ever ,or never used during
lifetime) OR (95 CI%)a  1234P trendQuartiles and IGF-I (P interaction
IGF1/OC=0.17)Ever used OC Cases/controls: 70/110  Age (years)1.00.30
(0.11-0.81)0.33 (0.13-0.86)0.58 (0.23-1.49)0.58  Age, diabetes mellitus, HRT
types, BMI1.00.22 (0.07-0.68)0.28 (0.10-0.82)0.40 (0.14-1.18)0.63 Never used OC
Cases/controls: 204/203  Age (years)1.00.78 (0.45-1.35)1.18 (0.68-2.05)1.07
(0.63-1.80)0.58  Age, diabetes mellitus, HRT types, BMI1.00.70 (0.39-1.25)1.09
(0.61-1.94)0.86 (0.49-1.52)0.74 IGFBP-1 (P interaction BP1/OC=0.08)Ever used OC
Cases/controls: 70/110  Age (years)1.00.98 (0.41-2.39)1.79 (0.76-4.20)3.26
(1.39-7.68)0.004  Age, diabetes mellitus, HRT types, BMI1.00.97 (0.35-2.74)1.80
(0.67-4.84)5.24 (1.90-14.48)0.99 Never used OC Cases/controls: 204/203  Age
(years)1.00.86 (0.47-1.60)1.03 (0.58-1.85)0.98 (0.55-1.73)0.89  Age, diabetes
mellitus, HRT types, BMI1.01.06 (0.55-2.04)1.38 (0.74-2.56)1.50 (0.78-2.88)0.43
IGFBP-3 (P interaction BP3/OC=0.19)Ever used OC Cases/controls: 65/99  Age
(years)1.00.54 (0.21-1.39)0.52 (0.21-1.27)0.40 (0.15-1.01)0.06  Age, diabetes
mellitus, HRT types, BMI1.00.49 (0.17-1.43)0.44 (0.16-1.22)0.20 (0.06-0.62)0.99
Never used OC Cases/controls: 192/187  Age (years)1.01.08 (0.62-1.86)0.88
(0.49-1.59)1.24 (0.70-2.22)0.62  Age, diabetes mellitus, HRT types, BMI1.00.93
(0.52-1.66)0.79 (0.42-1.46)1.26 (0.69-2.30)0.53 Insulin (P interaction
ins/OC=0.82)Ever used OC Cases/controls: 66/102  Age (years)1.00.94
(0.37-2.37)1.02 (0.40-2.62)0.68 (0.26-1.79)0.47  Age, diabetes mellitus, HRT-
types, BMI1.00.87 (0.31-2.49)0.97 (0.32-2.91)0.38 (0.12-1.22)0.16 Never used OC
Cases/controls: 194/194  Age (years)1.01.32 (0.72-2.39)1.12 (0.60-2.08)1.27
(0.68-2.36)0.67  Age, diabetes mellitus, HRT-types, BMI1.01.12 (0.58-2.16)0.76
(0.37-1.56)0.76 (0.36-1.57)0.17HRT=hormone replacement therapy.aOdds ratios and
95% confidence intervals for models including age only, and age, history of
diabetes mellitus, use of different HRT types and body mass index (BMI).)., The
correlation coefficients between the different endogenous hormones studied did
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not differ substantially from those calculated separately for cancer patients and controls (Table 2 Table 2Pearson's correlation coefficients between the various hormone levels in the entire study population (endometrial cancer cases and control women)a INSULIN (ng ml-1)IGFBP-1 (ng ml-1)IGF-I (ng ml-1)IGFBP-1 (ng ml-1)-0.221.0 IGF-I (ng ml-1)0.04-0.091.0IGFBP-3 (ng ml-1)0.09-0.180.35aThe correlation coefficients calculated separately for endometrial cancer cases and control women were not substantially different from the coefficients presented above (cases and controls together).)., Overall, IGFBP-1 levels were higher among the cancer cases than among control women.]
 novel_assocs | []
 missed_assocs | [IGFBP-1|diabetes mellitus, IGF1|diabetes mellitus, IGF-I|diabetes mellitus, IGFBP-3|diabetes mellitus]
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 pmid                  | 14680511
 section               | discuss
 collect_set(new_assoc) | [CD4|RA, CD4|arthritis, Mmp9|arthritis,
Bst1|arthritis, Mcpt1|arthritis, Mmp9|experimental autoimmune encephalomyelitis,
p53|arthritis, Bst1|RA, Ccng1|arthritis, MHC|RA]
 collect_set(old_assoc) | [CD4|arthritis, MHC|experimental autoimmune
encephalomyelitis, Mmp9|arthritis, Mmp9|experimental autoimmune
encephalomyelitis, Mcpt1|arthritis, mitogen-activated protein
kinases|experimental autoimmune encephalomyelitis, mitogen-activated protein
kinases|arthritis, p53|arthritis, Ccng1|arthritis, MHC|arthritis]
 collect_set(new_text)  | [Ccng1 has been suggested as a negative feedback
regulator of p53 [38], and therefore lower expression of Ccng1 in the DA rat
would lead to more apoptosis in the lymph nodes just before arthritis onset.,
The disappearing difference in CD4+   T cells is interesting because these
cells are believed to play an important role in both adjuvant arthritis and RA
[20-22]., In addition, a higher expression of Bst1 has been associated with
severe RA in patients [29]., Mmp9 has the potential to promote arthritis in many
ways and its role in early autoimmune disease is supported by Mmp9 knockout
mice, which show less susceptibility to experimental autoimmune
encephalomyelitis [33]., In addition, MHC haplotypes associated with RA are more
densely expressed on the cell surface than haplotypes that are not associated
[26]., The exact role played by Ccng1 in the cell cycle and various phases of
arthritis must therefore be further investigated., The lower expression of Mcpt1
[35] supports recent observations that mast cells are important for arthritis.,
The Bst1 molecule may be involved in the same mechanism as the recently cloned
arthritis controlling gene Ncf1 [27] because it supports NADPH oxidase catalyzed
superoxide generation [28], among several other interesting functions.,
Interestingly, at least two subgroups of mast cells with different roles in
arthritis have been identified, and these subgroups are distinguished by their
differential Mcpt1 expression [36].]
 collect_set(old_text)  | [In addition to a direct association with the immune
system, a differential regulation of apoptosis between the DA and E3 rat is
indicated because Ccng1 is differentially expressed. Ccng1 has been suggested as
a negative feedback regulator of p53 [38], and therefore lower expression of
Ccng1 in the DA rat would lead to more apoptosis in the lymph nodes just before
arthritis onset., The disappearing difference in CD4+   T cells is interesting
because these cells are believed to play an important role in both adjuvant
arthritis and RA [20-22]., Two genes, in addition to MHC, were expressed at a
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higher level in the pristane-treated DA rats, Mmp9 and Ptpn16. Mmp9 is a
granulocyte-secreted type IV collagenase and Ptpn16 is induced by oxidative
stress and inflammation [30,31] to act as a negative feedback regulator of
mitogen-activated protein kinases [32]. Mmp9 has the potential to promote
arthritis in many ways and its role in early autoimmune disease is supported by
Mmp9 knockout mice, which show less susceptibility to experimental autoimmune
encephalomyelitis [33]., The exact role played by Ccng1 in the cell cycle and
various phases of arthritis must therefore be further investigated., The lower
expression of Mcpt1 [35] supports recent observations that mast cells are
important for arthritis., Interestingly, at least two subgroups of mast cells
with different roles in arthritis have been identified, and these subgroups are
distinguished by their differential Mcpt1 expression [36].]
 novel_assocs           | [CD4|RA, Bst1|arthritis, Bst1|RA, MHC|RA]
 missed_assocs          | [MHC|experimental autoimmune encephalomyelitis,
mitogen-activated protein kinases|experimental autoimmune encephalomyelitis,
mitogen-activated protein kinases|arthritis, MHC|arthritis]
only showing top 10 rows
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[39]:
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# Ten examples of publications with novel associations and how their text looks␣
↪like

tmp.filter(F.size('novel_assocs') > 0).show(10, False, True)
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 pmid                    | 10770794
 section                 | other
 collect_set(new_assoc) | [CD8|hepatitis B virus infection, HBe|HBV infection,
CD8|infection, CD8|HBV infection]
 collect_set(old_assoc) | [HBe|HBV infection, CD8|infection, CD8|HBV infection]
 collect_set(new_text)  | [This mechanism plays an important role in the early
phase of acute HBV infection because downregulation of HBV replication precedes
massive infiltration of CD8+ T cells and manifestation of liver disease 7.,
Maini Maini M.K. M.K. Boni Boni C. C. Lee Lee C.K. C.K. Larrubia Larrubia J.R.
J.R. Reignat Reignat S. S. Ogg Ogg G.S. G.S. King King A.S. A.S. Herberg Herberg
J. J. Gilson Gilson R. R. Alisa Alisa A. A. Williams Williams R. R. Vergani
Vergani D. D. Naoumov Naoumov N.V. N.V. Ferrari Ferrari C. C. Bertoletti
Bertoletti A. A. The role of virus-specific CD8 + +  cells in liver damage and
viral control during persistent hepatitis B virus infection J. Exp., Thus, the
results that Maini et al. obtained in persistently infected, HBeAg- patients
with low viral load 6, and additional studies by other investigators 7, 8 are
leading to a new appreciation of the function of HBV-specific CD8+ T cells
favoring a protective rather than a pathogenic role in HBV infection.,
Conceivably, therefore, virus-specific T cells may persist not only in the
peripheral blood, but also in the liver, as recently reported for intrahepatic
hepatitis C virus (HCV)-specific CD8+ T cells after recovery from acute, self-
limited HCV infection 14., Maini Maini M.K. M.K. Boni Boni C. C. Ogg Ogg G.S.
G.S. King King A.S. A.S. Reignat Reignat S. S. Lee Lee C.K. C.K. Larrubia
Larrubia J.R. J.R. Webster Webster G.J. G.J. McMichael McMichael A.J. A.J.
Ferrari Ferrari C. C. Direct ex vivo analysis of hepatitis B virus-specific CD8
+ +  T cells associated with the control of infection Gastroenterology
Gastroenterology 117 117 1999 1999 1386 1386 1396 1396 supplied-pmid 10579980?,
Individuals with acute, self-limited HBV infection characteristically mount a
vigorous, polyclonal, and multispecific Th and CTL response to epitopes within
the HBV envelope (HBe), nucleocapsid, and polymerase proteins that is readily
detectable in the peripheral blood.]
 collect_set(old_text)  | [This mechanism plays an important role in the early
phase of acute HBV infection because downregulation of HBV replication precedes
massive infiltration of CD8+ T cells and manifestation of liver disease 7.,
Conceivably, therefore, virus-specific T cells may persist not only in the
peripheral blood, but also in the liver, as recently reported for intrahepatic
hepatitis C virus (HCV)-specific CD8+ T cells after recovery from acute, self-
limited HCV infection 14., Individuals with acute, self-limited HBV infection
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characteristically mount a vigorous, polyclonal, and multispecific Th and CTL response to epitopes within the HBV envelope (HBe), nucleocapsid, and polymerase proteins that is readily detectable in the peripheral blood., Thus, the results that Maini et al. obtained in persistently infected, HBeAg- patients with low viral load 6, and additional studies by other investigators 7 8 are leading to a new appreciation of the function of HBV-specific CD8+ T cells favoring a protective rather than a pathogenic role in HBV infection.]
 novel_assocs | [CD8|hepatitis B virus infection]
 missed_assocs | []
-RECORD 1---

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 pmid | 11178262
 section | other
 collect_set(new_assoc) | [IGF2|genetic disorder, Igf2|cancer, IGF2|Beckwith-Wiedemann syndrome, Igf2|BWS, IGF2|BWS, IGF2|childhood tumors, IGF2|Beckwith Wiedemann syndrome]

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 collect_set(old_assoc) | [IGF2|genetic disorder, Igf2|cancer, IGF2|childhood
tumors, Igf2|BWS, IGF2|BWS, IGF2|Beckwith Wiedemann syndrome]
 collect_set(new_text)  | [This is of interest in relation to the loss of
imprinting of IGF2 that occurs in the human genetic disorder Beckwith Wiedemann
syndrome (BWS), which is associated with fetal overgrowth and predisposition to
childhood tumors., Enlargement of the tongue is the most consistent feature of
BWS, a feature that might correspond to the strong reactivation of Igf2, a
potent growth factor, in the mouse tongue following deletion of the muscle-
specific silencer., Brown Brown KW KW Villar Villar AJ AJ Bickmore Bickmore W W
Clayton-Smith Clayton-Smith J J Catchpoole Catchpoole D D Maher Maher ER ER Reik
Reik W W Imprinting mutation in the Beckwith-Wiedemann syndrome leads to
biallelic  IGF2 IGF2  expression through an  H19 H19  independent pathway., The
situation in mice may be pertinent to that in humans, where loss of imprinting
of IGF2 can occur without altering the imprinting of H19 in hepatoblastoma and
in many patients with BWS [20,21,22]., The Igf2 silencers identified in mice add
to the ever-increasing number of elements controlling the imprinting of Igf2;
they provide additional targets for mutations that can lead to disruption of
imprinting, and to diseases including cancer., Joyce Joyce JA JA Lam Lam WK WK
Catchpoole Catchpoole DJ DJ Jenks Jenks P P Reik Reik W W Maher Maher ER ER
Schofield Schofield PN PN Imprinting of  IGF2 IGF2  and  H19 H19 : lack of
reciprocity in sporadic Beckwith-Wiedemann syndrome.]
 collect_set(old_text)  | [This is of interest in relation to the loss of
imprinting of IGF2 that occurs in the human genetic disorder Beckwith Wiedemann
syndrome (BWS), which is associated with fetal overgrowth and predisposition to
childhood tumors., Enlargement of the tongue is the most consistent feature of
BWS, a feature that might correspond to the strong reactivation of Igf2, a
potent growth factor, in the mouse tongue following deletion of the muscle-
specific silencer., The situation in mice may be pertinent to that in humans,
where loss of imprinting of IGF2 can occur without altering the imprinting of
H19 in hepatoblastoma and in many patients with BWS [20,21,22]., The Igf2
silencers identified in mice add to the ever-increasing number of elements
controlling the imprinting of Igf2; they provide additional targets for
mutations that can lead to disruption of imprinting, and to diseases including
cancer.]
 novel_assocs           | [IGF2|Beckwith-Wiedemann syndrome]
 missed_assocs          | []
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 pmid                   | 11355941
 section                | abstract
 collect_set(new_assoc) | [MMP-7|colorectal carcinomas, Matrilysin|colorectal
carcinomas, matrilysin|colorectal carcinomas]
 collect_set(old_assoc) | [MMP-7|colorectal carcinomas, Matrilysin|colorectal
carcinomas]
 collect_set(new_text)  | [Matrilysin (MMP-7) has been shown to correlate with
nodal or distant metastasis in colorectal carcinomas; however, its implication
in early invasive colorectal carcinomas has not been determined., Tumour budding
at the invasive margin and matrilysin expression are more useful in identifying
high-risk groups for adverse outcome in patients with early invasive colorectal
carcinomas.]
 collect_set(old_text)  | [Matrilysin (MMP-7) has been shown to correlate with
nodal or distant metastasis in colorectal carcinomas; however, its implication
in early invasive colorectal carcinomas has not been determined.]
 novel_assocs           | [matrilysin|colorectal carcinomas]
 missed_assocs          | []
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 pmid                   | 11424125
 section                | abstract
 collect_set(new_assoc) | [HBs|HBV infection, HBc|HBV infection, HBs|chronic
hepatitis]
 collect_set(old_assoc) | [HBs|HBV infection, HBs|chronic hepatitis]
 collect_set(new_text)  | [To evaluate whether HCV genotype and a "silent" HBV
infection may be related to a more severe clinical presentation of liver
disease, 205 anti-HCV/HCV-RNA positive, HBsAg/anti-HBs negative patients with
chronic hepatitis (113 males and 92 females; median age 55 years, range 18-77),
were studied on presentation at the Liver Unit from January 1993 to December
1997., Presence of serum anti-HBc, in the absence of HBsAg and anti-HBs, was
considered a marker of "silent" HBV infection., Instead, the 88 patients with
"silent" HBV infection showed a higher percentage of severe liver disease than
the 97 anti-HBc negative patients (72.7% vs. 46.4%, respectively: P < 0.0005).]
 collect_set(old_text)  | [To evaluate whether HCV genotype and a "silent" HBV
infection may be related to a more severe clinical presentation of liver
disease, 205 anti-HCV/HCV-RNA positive, HBsAg/anti-HBs negative patients with
chronic hepatitis (113 males and 92 females; median age 55 years, range 18-77),
were studied on presentation at the Liver Unit from January 1993 to December
1997., Presence of serum anti-HBc, in the absence of HBsAg and anti-HBs, was
considered a marker of "silent" HBV infection.]
 novel_assocs           | [HBc|HBV infection]
 missed_assocs          | []
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 pmid                   | 11448986
 section                | other
 collect_set(new_assoc) | [telethonin|Limb-girdle muscular dystrophy type 2G,
Telethonin|limb-girdle muscular dystrophy type 2G, Myotilin|limb-girdle muscular
dystrophy, Myotilin|muscular dystrophy, T-CAP|limb-girdle muscular dystrophy
type 2G]
 collect_set(old_assoc) | [Telethonin|limb-girdle muscular dystrophy type 2G,
T-CAP|limb-girdle muscular dystrophy type 2G]
 collect_set(new_text)  | [Limb-girdle muscular dystrophy type 2G is caused by
mutations in the gene encoding the sarcomeric protein telethonin., Myotilin is
mutated in limb girdle muscular dystrophy 1A., Telethonin/T-CAP has taken on
added importance due to its causal role in limb-girdle muscular dystrophy type
2G (Moreira et al., 2000)., Myotilin, a novel sarcomeric protein with two Ig-
like domains, is encoded by a candidate gene for two limb-girdle muscular
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dystrophy.]
 collect_set(old_text)  | [Telethonin/T-CAP has taken on added importance due to
its causal role in limb-girdle muscular dystrophy type 2G (Moreira et al.,
2000).]
 novel_assocs           | [telethonin|Limb-girdle muscular dystrophy type 2G,
Myotilin|limb-girdle muscular dystrophy, Myotilin|muscular dystrophy]
 missed_assocs          | []
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 pmid                   | 11675938
 section                | abstract
 collect_set(new_assoc) | [CYP11B1|aldosteronoma, CYP11B2|aldosteronoma,
CYP11B1|hyperaldosteronism, aldosterone synthase|primary aldosteronism,
CYP11B2|primary aldosteronism, renin|APA, CYP11B2|hyperaldosteronism,
CYP11B1|APA, CYP11B2|APA]
 collect_set(old_assoc) | [CYP11B1|hyperaldosteronism, aldosterone
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synthase|primary aldosteronism, CYP11B2|primary aldosteronism,
CYP11B2|hyperaldosteronism, renin|APA, CYP11B1|APA, CYP11B2|APA]
 collect_set(new_text)  | [In the present study, we investigated the genetic
analysis of aldosterone synthase gene, CYP11B2 in patients with primary
aldosteronism and review the recent studies., It has been reported that renin
suppression and aldosterone levels are lower and hypokalemia milder in patients
with IHA than in patients with APA., The chimeric CYP11B1/CYP11B2 gene, which is
a candidate gene for glucocorticoid-remediable hyperaldosteronism, was not found
in either the DNA from aldosteronoma or in the genomic DNA from patients with
APA or IHA., The level of CYP11B2 messenger RNA (mRNA) was much higher in the
aldosteronoma portion than in nonadenomatous portion.]
 collect_set(old_text)  | [In the present study, we investigated the genetic
analysis of aldosterone synthase gene, CYP11B2 in patients with primary
aldosteronism and review the recent studies., It has been reported that renin
suppression and aldosterone levels are lower and hypokalemia milder in patients
with IHA than in patients with APA., The chimeric CYP11B1/CYP11B2 gene, which is
a candidate gene for glucocorticoid-remediable hyperaldosteronism, was not found
in either the DNA from aldosteronoma or in the genomic DNA from patients with
APA or IHA.]
 novel_assocs           | [CYP11B1|aldosteronoma, CYP11B2|aldosteronoma]
 missed_assocs          | []
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 pmid                   | 11782869
 section                | title
 collect_set(new_assoc) | [growth hormone-releasing hormone|growth hormone
deficiency, growth hormone-releasing hormone|pituitary adenomas]
 collect_set(old_assoc) | [growth hormone-releasing hormone|pituitary adenomas]
 collect_set(new_text)  | [The use of the pyridostigmine growth hormone-
releasing hormone stimulation test to detect growth hormone deficiency in
patients with pituitary adenomas.]
 collect_set(old_text)  | [The use of the pyridostigmine growth hormone-
releasing hormone stimulation test to detect growth hormone deficiency in
patients with pituitary adenomas.]
 novel_assocs           | [growth hormone-releasing hormone|growth hormone
deficiency]
 missed_assocs          | []
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 pmid                  | 11805148
 section               | other
 collect_set(new_assoc) | [C1s|systemic lupus erythematosus,
IgA|antiphospholipid syndrome, C1r|systemic lupus erythematosus,
IgM|antiphospholipid syndrome]
 collect_set(old_assoc) | [aPL|APS, aPL|antiphospholipid syndrome]
 collect_set(new_text)  | [Hypocomplementemia with low C1s-C1r inhibitor complex
in systemic lupus erythematosus., Induction of thrombosis in a mouse model by
IgG, IgM and IgA immunoglobulins from patients with antiphospholipid syndrome.]
 collect_set(old_text)  | [Abbreviations used in this paper: aCL,
anticardiolipin; aPL, antiphospholipid; aPL-IgG, human IgG containing aPL
antibody; APS, antiphospholipid syndrome;  2GPI,  2-glycoprotein I; Crry,
complement receptor 1-related gene/protein y; MAC, membrane attach complex.]
 novel_assocs          | [C1s|systemic lupus erythematosus,
IgA|antiphospholipid syndrome, C1r|systemic lupus erythematosus,
IgM|antiphospholipid syndrome]
 missed_assocs         | [aPL|APS, aPL|antiphospholipid syndrome]
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 pmid                    | 11875709
 section                 | other
 collect_set(new_assoc) | [vascular endothelial growth factor|colorectal
carcinoma, vascular endothelial growth factor|Cancer, VEGF|CRC, VEGF|cancer,
vascular endothelial growth factor|cancer, VEGF|benign breast tumor]
 collect_set(old_assoc) | [VEGF|CRC]
 collect_set(new_text)  | [The aims of the present study were to compare the
prognostic significance of matched preoperative plasma and serum VEGF
concentrations in patients with CRC and to evaluate whether serum or plasma was
the best predictor of overall survival., In a previous study including 614
patients, it was shown that preoperative serum VEGF concentration, independent
of Dukes stage, was a strong predictor of overall survival of patients with
colorectal cancer (CRC) (Werther et al, 2000)., Banks Banks RE RE Forbes Forbes
MA MA Kinsey Kinsey SE SE Stanley Stanley A A Ingham Ingham E E Walters Walters
C C Selby Selby PJ PJ Release of the cytokine vascular endothelial growth factor
(VEGF) from platelets: significance for VEGF measurements and cancer biology
Release of the cytokine vascular endothelial growth factor (VEGF) from
platelets: significance for VEGF measurements and cancer biology Br J Cancer Br
J Cancer 1998 1998 77 77 956 956 964 964 [see comments] PubMed citation query:
'Br J Cancer||77|956||bib1|' 9528841 9528841, Salven Salven P P Orpana Orpana A
A Joensuu Joensuu H H Leukocytes and platelets of patients with cancer contain
high levels of vascular endothelial growth factor Leukocytes and platelets of
patients with cancer contain high levels of vascular endothelial growth factor
Clin Cancer Res Clin Cancer Res 1999b 1999b 5 5 487 487 491 491 PubMed citation
query: 'Clin Cancer Res||5|487||bib9|' 10100697 10100697, Lee Lee JK JK Hong
Hong YJ YJ Han Han CJ CJ Hwang Hwang DY DY Hong Hong SI SI Clinical usefulness
of serum and plasma vascular endothelial growth factor in cancer patients: which
is the optimal specimen?, Clinical usefulness of serum and plasma vascular
endothelial growth factor in cancer patients: which is the optimal specimen?,
Salven Salven P P Perhoniemi Perhoniemi V V Tykka Tykka H H Maenpaa Maenpaa H H
Joensuu Joensuu H H Serum VEGF levels in women with a benign breast tumor or
breast cancer Serum VEGF levels in women with a benign breast tumor or breast
cancer Breast Cancer Res Treat Breast Cancer Res Treat 1999a 1999a 53 53 161 161
166 166 PubMed citation query: 'Breast Cancer Res Treat||53|161||bib10|'
10326793 10326793, Werther Werther K K Christensen Christensen IJ IJ Brunner
Brunner N N Nielsen Nielsen HJ HJ Danish Danish RANX RANX Soluble vascular
endothelial growth factor levels in patients with primary colorectal carcinoma
[In Process Citation] Soluble vascular endothelial growth factor levels in
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patients with primary colorectal carcinoma [In Process Citation] Eur J Surg
Oncol Eur J Surg Oncol 2000 2000 26 26 657 657 662 662 PubMed citation query:
'Eur J Surg Oncol||26|657||bib12|' 11078612 11078612]
 collect_set(old_text)  | [The aims of the present study were to compare the
prognostic significance of matched preoperative plasma and serum VEGF
concentrations in patients with CRC and to evaluate whether serum or plasma was
the best predictor of overall survival., In a previous study including 614
patients, it was shown that preoperative serum VEGF concentration, independent
of Dukes stage, was a strong predictor of overall survival of patients with
colorectal cancer (CRC) (Werther et al, 2000).]
 novel_assocs           | [vascular endothelial growth factor|colorectal
carcinoma, vascular endothelial growth factor|Cancer, VEGF|cancer, vascular
endothelial growth factor|cancer, VEGF|benign breast tumor]
 missed_assocs          | []
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 pmid                   | 12014995
 section                | other
 collect_set(new_assoc) | [Lactase|metabolic bone disease,
lactase|nephrocalcinosis, HRPT1|multiple endocrine neoplasia Types I,
lactase|bone disease, PTH|neoplasia, RET|multiple endocrine neoplasia type 2B,
RET|medullary thyroid carcinoma, elastin|Williams syndrome]
 collect_set(old_assoc) | [Lactase|metabolic bone disease, HRPT1|multiple
endocrine neoplasia Types I, PTH|neoplasia]
 collect_set(new_text)  | [Lactase deficiency may give rise to metabolic bone
disease [31]., While neoplasia with osseous metastasis is a potential cause of
hypercalcemia, tumors may also secrete materials such as PTH-rp or other
osteoclast-activating factors, cytokines, or other growth factors that modulate
bone remodeling leading to calcium adsorption from bone., Familial
hyperparathyroidism may be confined to parathyroid adenoma (HRPT1 #145000) or
may involve the broader endocrine disturbances of multiple endocrine neoplasia
Types I (#131100) and IIA (#171400)., Ewart Ewart AK AK Morris Morris CA CA
Atkinson Atkinson D D Jin Jin W W Sternes Sternes K K Spallone Spallone P P
Stock Stock AD AD Leppert Leppert M M Keating Keating MT MT Hemizygosity at the
elastin locus in a developmental disorder, Williams syndrome., A mutation in the
RET proto-oncogene associated with multiple endocrine neoplasia type 2B and
sporadic medullary thyroid carcinoma., Hypercalcemia and nephrocalcinosis in
patients with congenital lactase deficiency., Metabolic bone disease as a result
of lactase deficiency., Hemizygosity at the elastin locus in a developmental
disorder, Williams syndrome., Hofstra Hofstra RM RM Landsvater Landsvater RM RM
Ceccherini Ceccherini I I Stulp Stulp RP RP Stelwagen Stelwagen T T Luo Luo Y Y
Pasini Pasini B B Hoppener Hoppener JW JW van Amstel van Amstel HK HK Romeo
Romeo G G A mutation in the RET proto-oncogene associated with multiple
endocrine neoplasia type 2B and sporadic medullary thyroid carcinoma.]
 collect_set(old_text)  | [Lactase deficiency may give rise to metabolic bone
disease [31]., While neoplasia with osseous metastasis is a potential cause of
hypercalcemia, tumors may also secrete materials such as PTH-rp or other
osteoclast-activating factors, cytokines, or other growth factors that modulate
bone remodeling leading to calcium adsorption from bone., Familial
hyperparathyroidism may be confined to parathyroid adenoma (HRPT1 #145000) or
may involve the broader endocrine disturbances of multiple endocrine neoplasia
Types I (#131100) and IIA (#171400).]
 novel_assocs           | [lactase|nephrocalcinosis, lactase|bone disease,
RET|multiple endocrine neoplasia type 2B, RET|medullary thyroid carcinoma,
elastin|Williams syndrome]
 missed_assocs          | []
only showing top 10 rows
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