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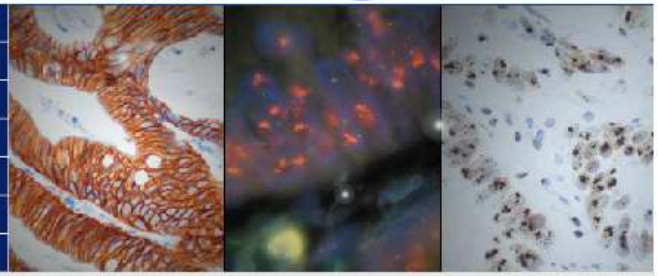

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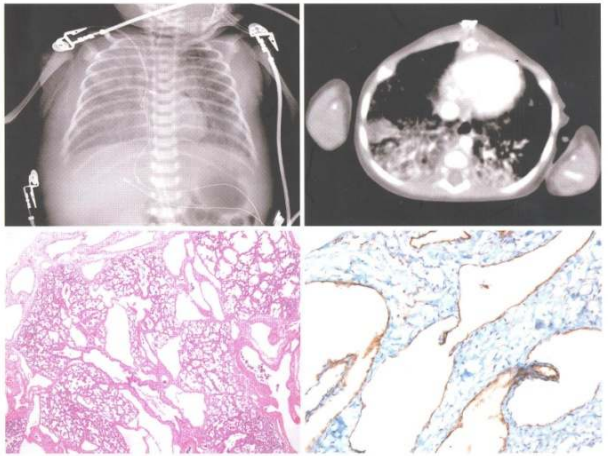

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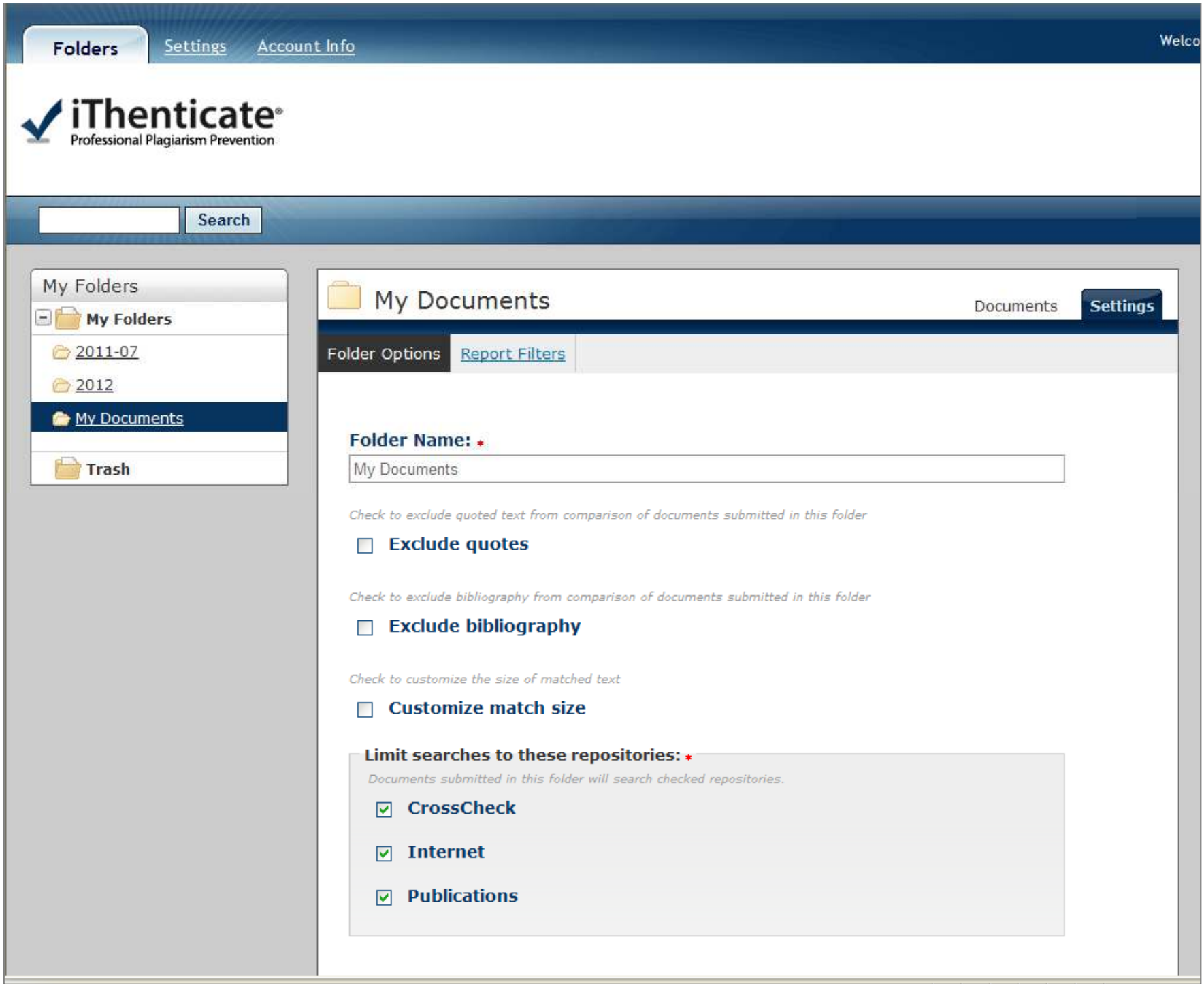
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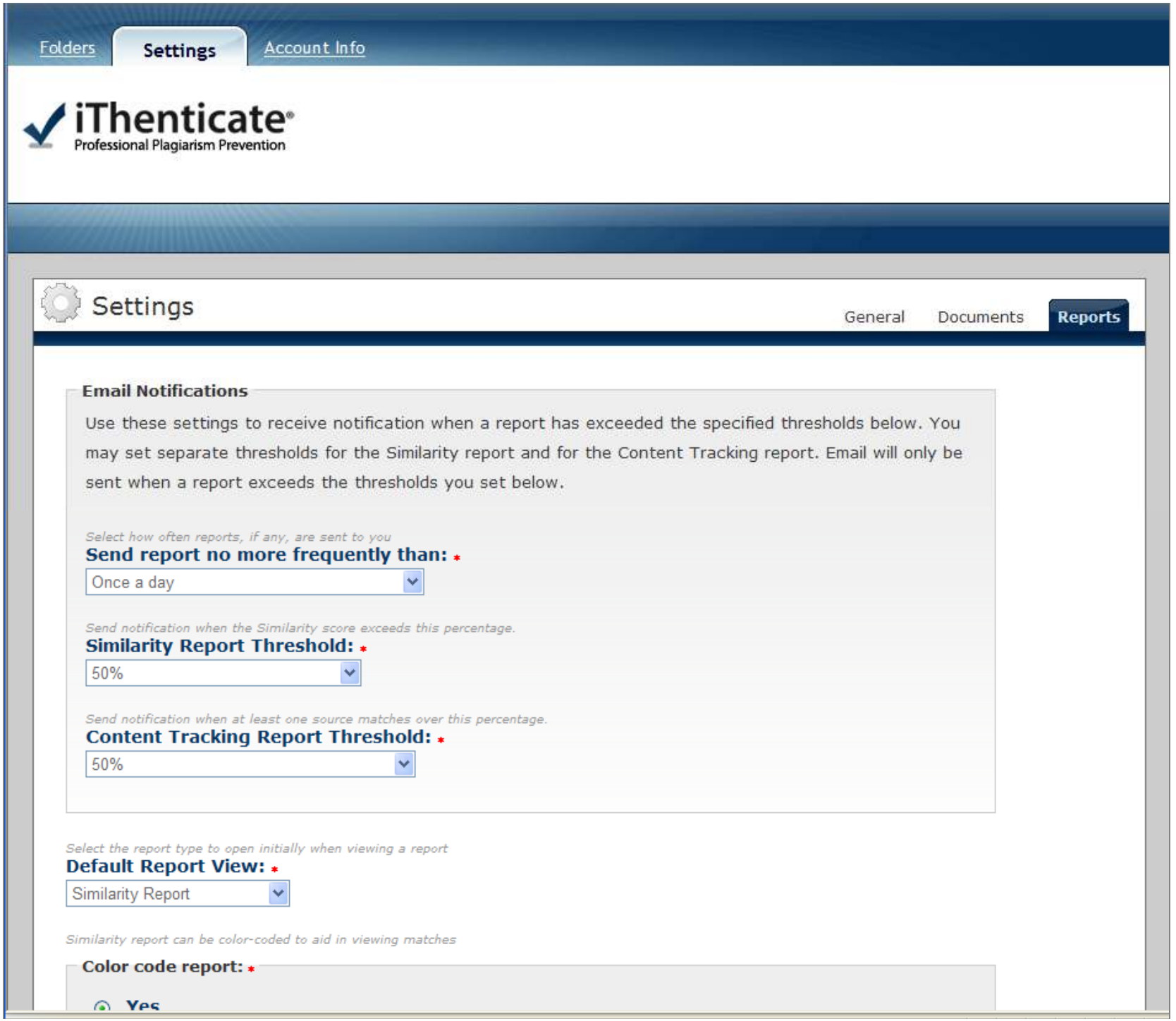
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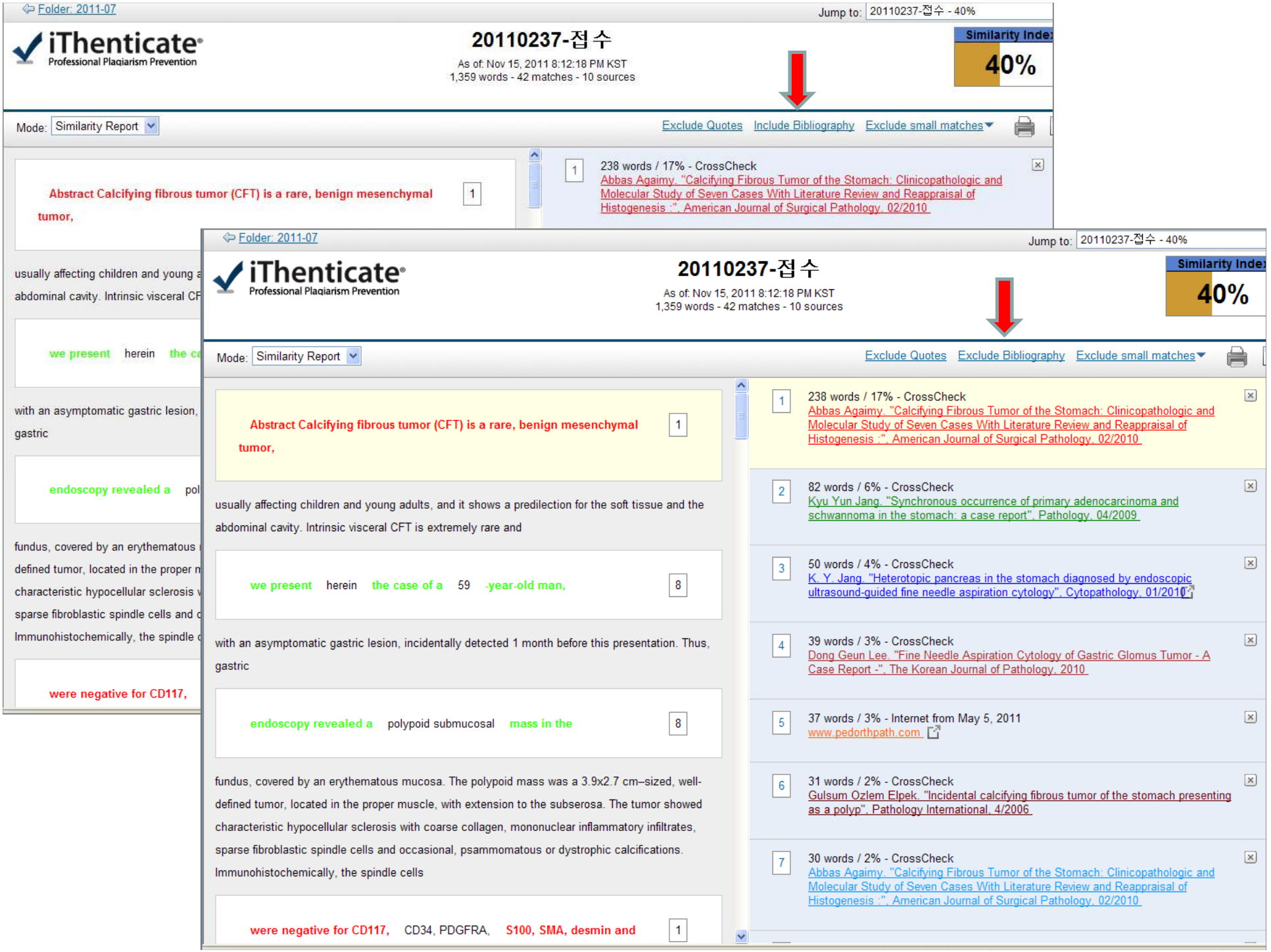
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we present herein the case of a 59 -year-old man,
with an asymptomatic gastric lesion, incidentally detected 1 month before this presentation. Thus, gastric
endoscopy revealed a polypoid submucosal mass in the
fundus, covered by an erythematous mucosa. The polypoid mass was a 3.9x2.7 cm–sized, well-defined tumor, located in the proper muscle, with extension to the subserosa. The tumor showed characteristic hypocellular sclerosis with coarse collagen, mononuclear inflammatory infiltrates, sparse fibroblastic spindle cells and occasional, psammomatous or dystrophic calcifications. Immunohistochemically, the spindle cells
were negative for CD117, CD34, PDGFRA, S100, SMA, desmin and
1 238 words / 17% - CrossCheck
Abbas Agaimy, "Calcifying Fibrous Tumor of the Stomach: Clinicopathologic and Molecular Study of Seven Cases With Literature Review and Reappraisal of Histogenesis :", American Journal of Surgical Pathology, 02/2010
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Dong Geun Lee, "Fine Needle Aspiration Cytology of Gastric Glomus Tumor - A Case Report -", The Korean Journal of Pathology, 2010
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www.pedorthpath.com
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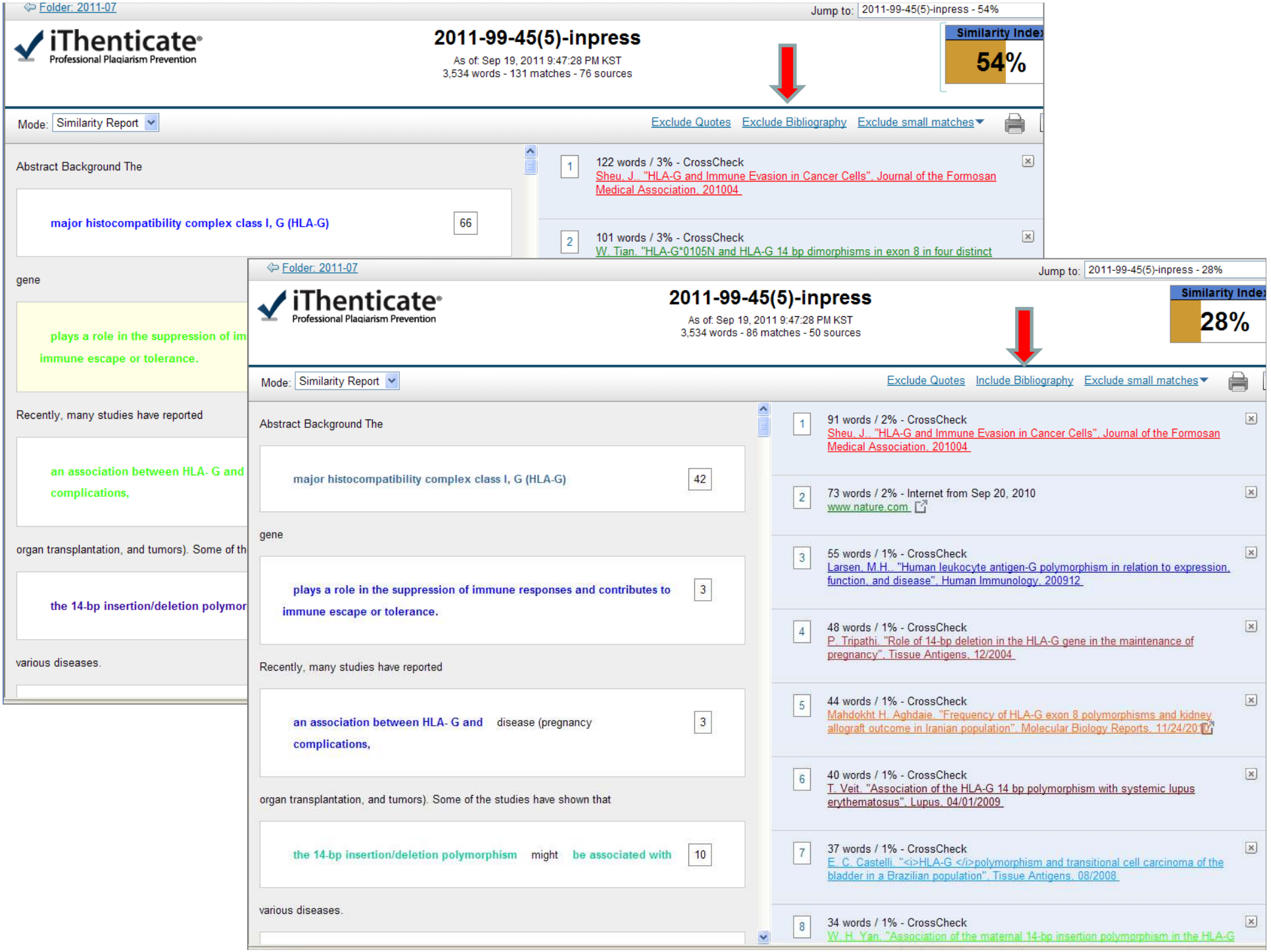
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Recently, many studies have reported
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complications,
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Sheu, J., "HLA-G and Immune Evasion in Cancer Cells", Journal of the Formosan Medical Association, 201004
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W. Tian, "HLA-G*0105N and HLA-G 14 bp dimorphisms in exon 8 in four distinct
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organ transplantation, and tumors). Some of the studies have shown that
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Sheu, J., "HLA-G and Immune Evasion in Cancer Cells", Journal of the Formosan Medical Association, 201004
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Larsen, M.H., "Human leukocyte antigen-G polymorphism in relation to expression, function, and disease", Human Immunology, 200912
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P. Tripathi, "Role of 14-bp deletion in the HLA-G gene in the maintenance of pregnancy", Tissue Antigens, 12/2004
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Mahdokht H. Aghdaie, "Frequency of HLA-G exon 8 polymorphisms and kidney allograft outcome in Iranian population", Molecular Biology Reports, 11/24/201
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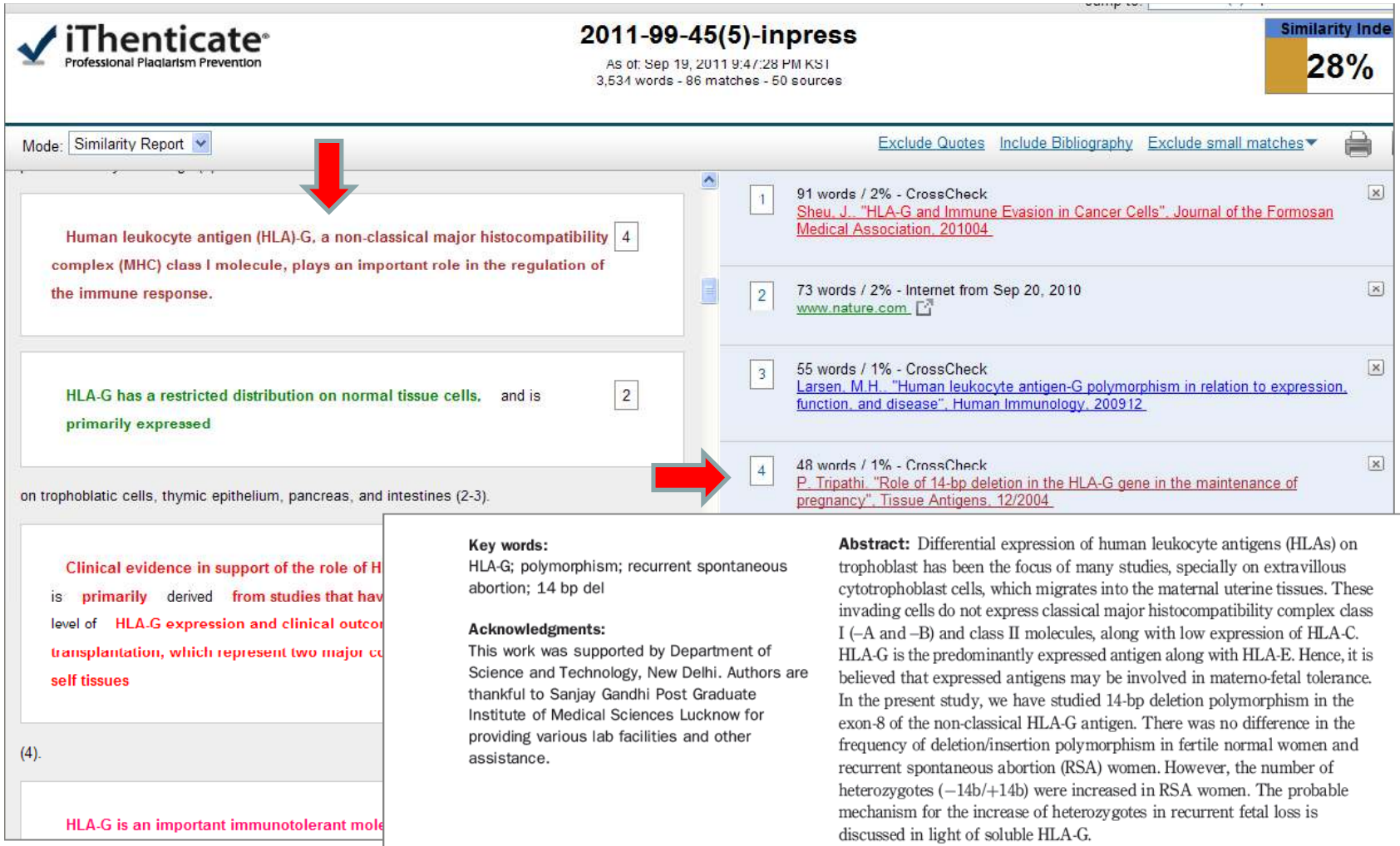

Key words:
HLA-G; polymorphism; recurrent spontaneous abortion; 14 bp del

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Abstract: Differential expression of human leukocyte antigens (HLAs) on trophoblast has been the focus of many studies, specially on extravillous cytotrophoblast cells, which migrates into the maternal uterine tissues. These invading cells do not express classical major histocompatibility complex class I (–A and –B) and class II molecules, along with low expression of HLA-C. HLA-G is the predominantly expressed antigen along with HLA-E. Hence, it is believed that expressed antigens may be involved in materno-fetal tolerance. In the present study, we have studied 14-bp deletion polymorphism in the exon-8 of the non-classical HLA-G antigen. There was no difference in the frequency of deletion/insertion polymorphism in fertile normal women and recurrent spontaneous abortion (RSA) women. However, the number of heterozygotes (−14b/+14b) were increased in RSA women. The probable mechanism for the increase of heterozygotes in recurrent fetal loss is discussed in light of soluble HLA-G.

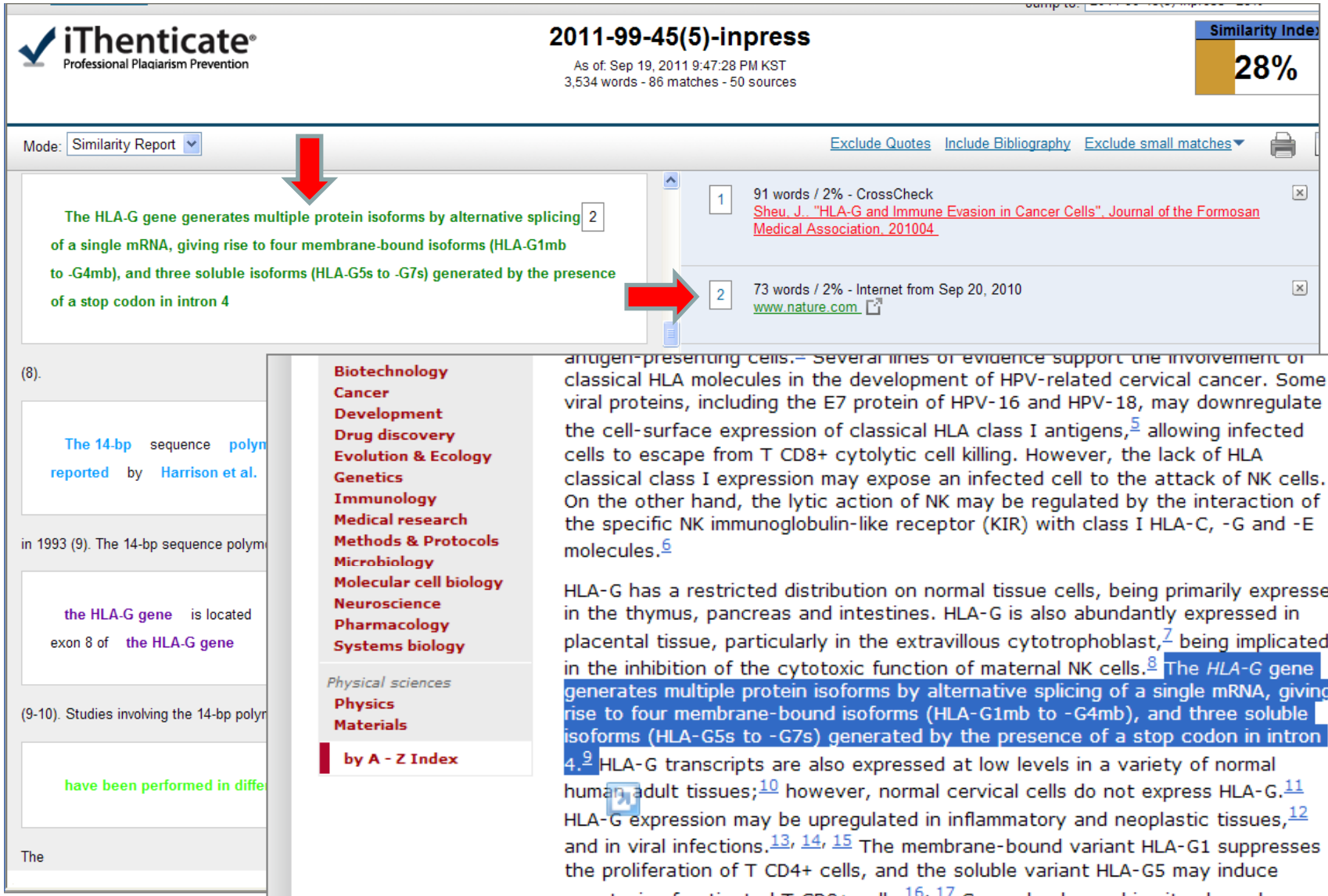
Authors' affiliation:

P. Tripathi,
A. Abbas,
S. Naik,
S. Agrawal

Department of Medical Genetics, Sanjay Gandhi Post Graduate Institute of Medical Sciences, Raebareli Road, Lucknow, UP, India

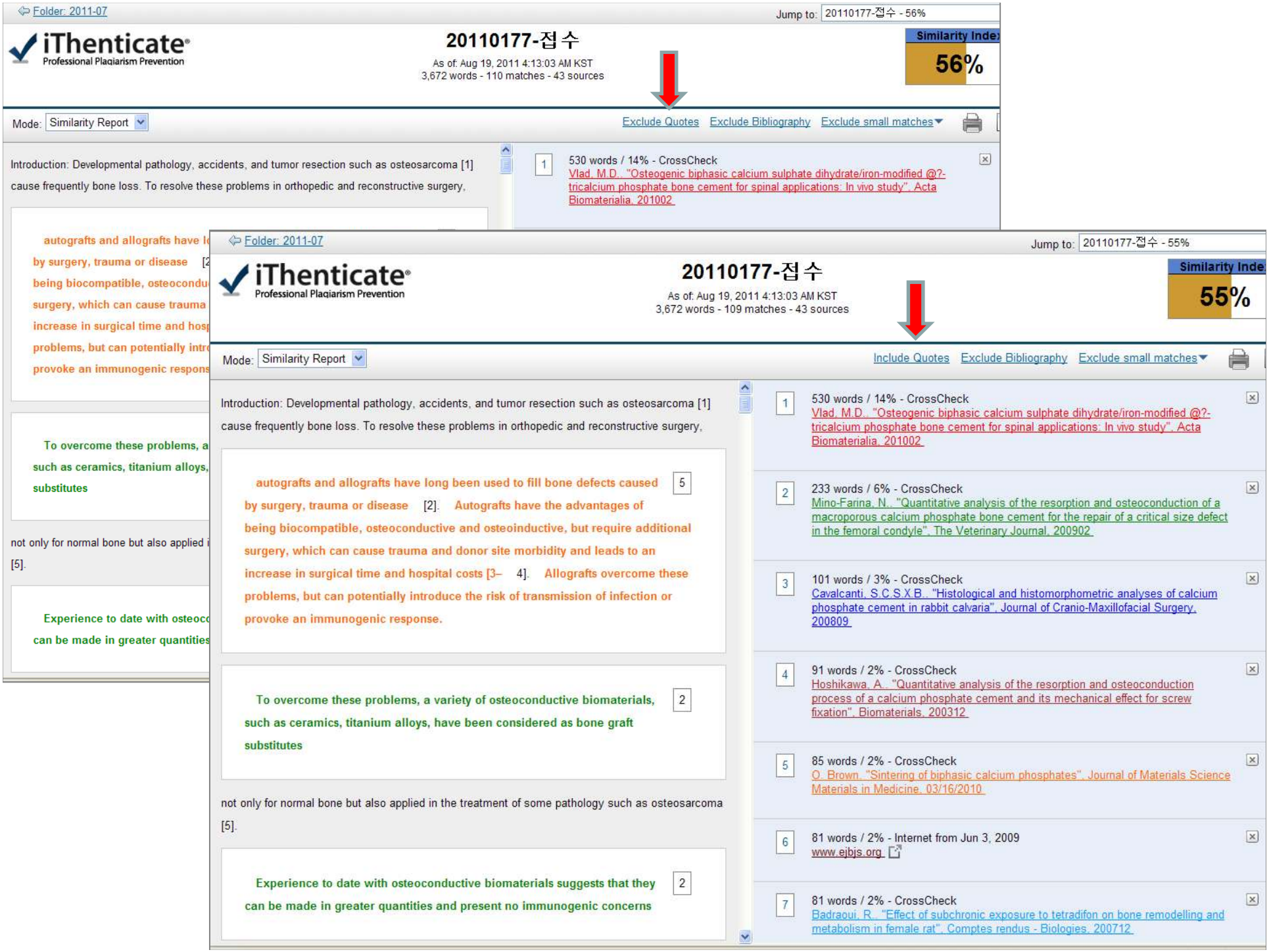
Correspondence to:
Suraksha Agrawal
Department of Medical Genetics
Sanjay Gandhi Post Graduate Institute of Medical Sciences
Raebareli Road
Lucknow
UP
India
Tel.: +91-522-2668004-8x2338
Fax: +91-522-2668973
e-mail:
suraksha@sgpgi.ac.in

Human leukocyte antigen (HLA)-G, a non-classical major histocompatibility complex (MHC) class I molecule, plays an important role in the regulation of the immune response. Although it is structurally similar to the classical HLA class I gene products (1), HLA-G also exhibits some unique features. There are seven protein isoforms of HLA-G, and it exhibits limited polymorphism. HLA-G expression varies in different pathological conditions (2–5). The mRNA profile and protein show differences (6). This may be due to regulated gene



antigen-presenting cells.[4] Several lines of evidence support the involvement of classical HLA molecules in the development of HPV-related cervical cancer. Some viral proteins, including the E7 protein of HPV-16 and HPV-18, may downregulate the cell-surface expression of classical HLA class I antigens,[5] allowing infected cells to escape from T CD8+ cytolytic cell killing. However, the lack of HLA classical class I expression may expose an infected cell to the attack of NK cells. On the other hand, the lytic action of NK may be regulated by the interaction of the specific NK immunoglobulin-like receptor (KIR) with class I HLA-C, -G and -E molecules.[6]

HLA-G has a restricted distribution on normal tissue cells, being primarily expressed in the thymus, pancreas and intestines. HLA-G is also abundantly expressed in placental tissue, particularly in the extravillous cytotrophoblast,[7] being implicated in the inhibition of the cytotoxic function of maternal NK cells.[8] The *HLA-G* gene generates multiple protein isoforms by alternative splicing of a single mRNA, giving rise to four membrane-bound isoforms (HLA-G1mb to -G4mb), and three soluble isoforms (HLA-G5s to -G7s) generated by the presence of a stop codon in intron 4.[9] HLA-G transcripts are also expressed at low levels in a variety of normal human adult tissues;[10] however, normal cervical cells do not express HLA-G.[11] HLA-G expression may be upregulated in inflammatory and neoplastic tissues,[12] and in viral infections.[13, 14, 15] The membrane-bound variant HLA-G1 suppresses the proliferation of T CD4+ cells, and the soluble variant HLA-G5 may induce apoptosis of activated T CD8+ cells.[16, 17] Several polymorphic sites have been described for the *HLA-G* locus. Nucleotide substitutions mainly in exons 2, 3 and 4 may discriminate 42 confirmed alleles clustered into nine distinct allele groups, and generating only 15 different proteins due to the presence of several synonymous substitutions among *HLA-G* alleles (Anthony Nolan Research Institute, February 2009).[18] *HLA-G* alleles may influence the plasma levels of soluble HLA-G (sHLA-G).[19] In addition, a 14bp insertion/deletion polymorphism has been reported in the 3′-untranslated region (UTR) of exon 8.[20] *HLA-G* alleles exhibiting the 14bp insertion (+14bp) undergo an alternative splicing that removes 92 bases from 3′ UTR,[21] influencing the stability of HLA-G mRNA.[22] Although removal of the 92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Vlad, M.D., "Osteogenic biphasic calcium sulphate dihydrate/iron-modified @?-tricalcium phosphate bone cement for spinal applications: In vivo study", Acta Biomaterialia, 201002
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Introduction: Developmental pathology, accidents, and tumor resection such as osteosarcoma [1] cause frequently bone loss. To resolve these problems in orthopedic and reconstructive surgery,
autografts and allografts have long been used to fill bone defects caused by surgery, trauma or disease [2]. Autografts have the advantages of being biocompatible, osteoconductive and osteoinductive, but require additional surgery, which can cause trauma and donor site morbidity and leads to an increase in surgical time and hospital costs [3– 4]. Allografts overcome these problems, but can potentially introduce the risk of transmission of infection or provoke an immunogenic response.
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To overcome these problems, a variety of osteoconductive biomaterials, such as ceramics, titanium alloys, have been considered as bone graft substitutes
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not only for normal bone but also applied in the treatment of some pathology such as osteosarcoma [5].
Experience to date with osteoconductive biomaterials suggests that they can be made in greater quantities and present no immunogenic concerns
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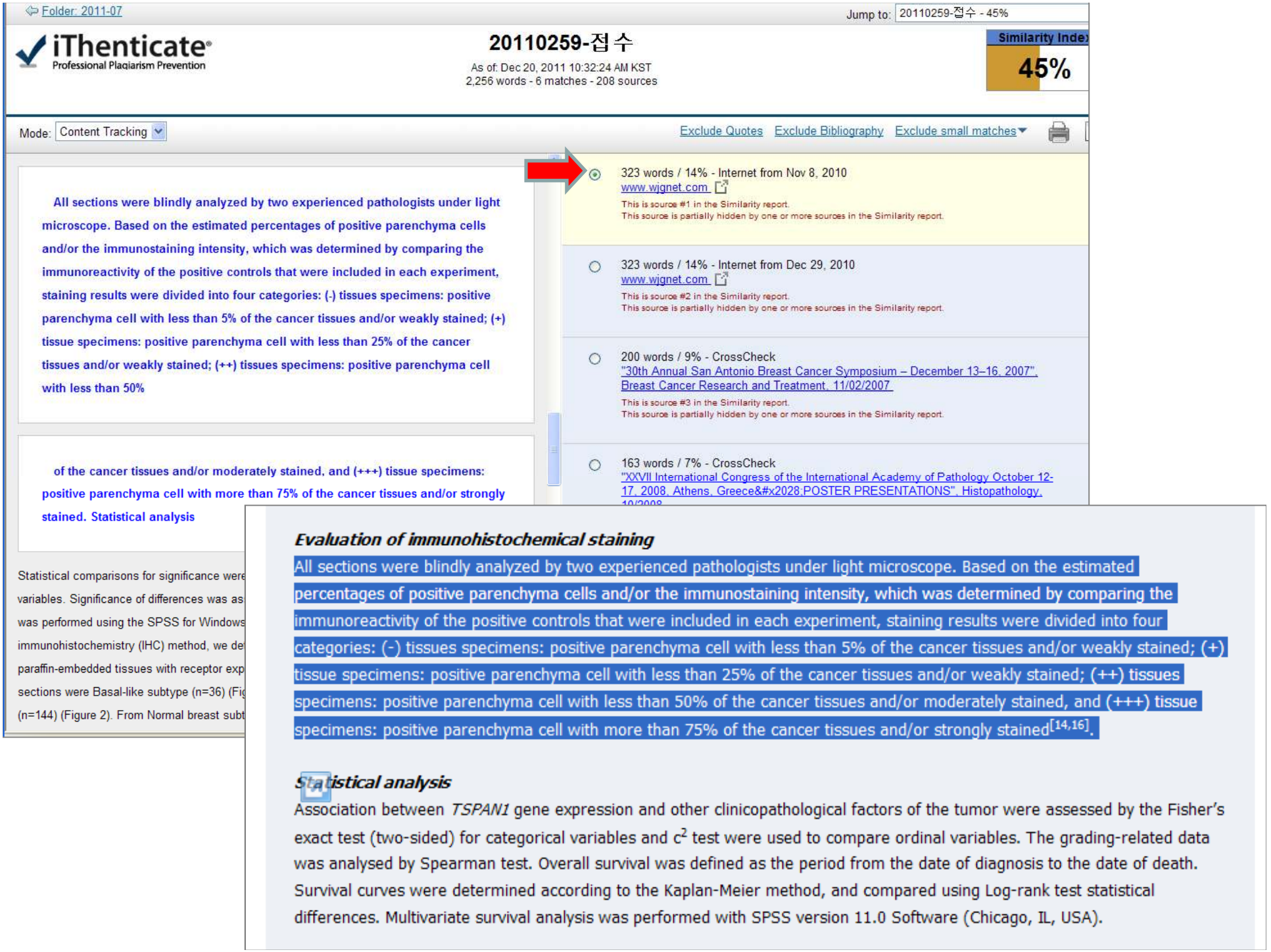
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All sections were blindly analyzed by two experienced pathologists under light microscope. Based on the estimated percentages of positive parenchyma cells and/or the immunostaining intensity, which was determined by comparing the immunoreactivity of the positive controls that were included in each experiment, staining results were divided into four categories: (-) tissues specimens: positive parenchyma cell with less than 5% of the cancer tissues and/or weakly stained; (+) tissue specimens: positive parenchyma cell with less than 25% of the cancer tissues and/or weakly stained; (++) tissues specimens: positive parenchyma cell with less than 50%
of the cancer tissues and/or moderately stained, and (+++) tissue specimens: positive parenchyma cell with more than 75% of the cancer tissues and/or strongly stained. Statistical analysis
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"XXVII International Congress of the International Academy of Pathology October 12-17, 2008, Athens, Greece POSTER PRESENTATIONS", Histopathology,
Statistical comparisons for significance were
variables. Significance of differences was as
was performed using the SPSS for Windows
immunohistochemistry (IHC) method, we de
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sections were Basal-like subtype (n=36) (Fig
(n=144) (Figure 2). From Normal breast subt
Evaluation of immunohistochemical staining
All sections were blindly analyzed by two experienced pathologists under light microscope. Based on the estimated percentages of positive parenchyma cells and/or the immunostaining intensity, which was determined by comparing the immunoreactivity of the positive controls that were included in each experiment, staining results were divided into four categories: (-) tissues specimens: positive parenchyma cell with less than 5% of the cancer tissues and/or weakly stained; (+) tissue specimens: positive parenchyma cell with less than 25% of the cancer tissues and/or weakly stained; (++) tissues specimens: positive parenchyma cell with less than 50% of the cancer tissues and/or moderately stained, and (+++) tissue specimens: positive parenchyma cell with more than 75% of the cancer tissues and/or strongly stained[14,16].
Statistical analysis
Association between TSPAN1 gene expression and other clinicopathological factors of the tumor were assessed by the Fisher's exact test (two-sided) for categorical variables and c2 test were used to compare ordinal variables. The grading-related data was analysed by Spearman test. Overall survival was defined as the period from the date of diagnosis to the date of death. Survival curves were determined according to the Kaplan-Meier method, and compared using Log-rank test statistical differences. Multivariate survival analysis was performed with SPSS version 11.0 Software (Chicago, IL, USA).

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