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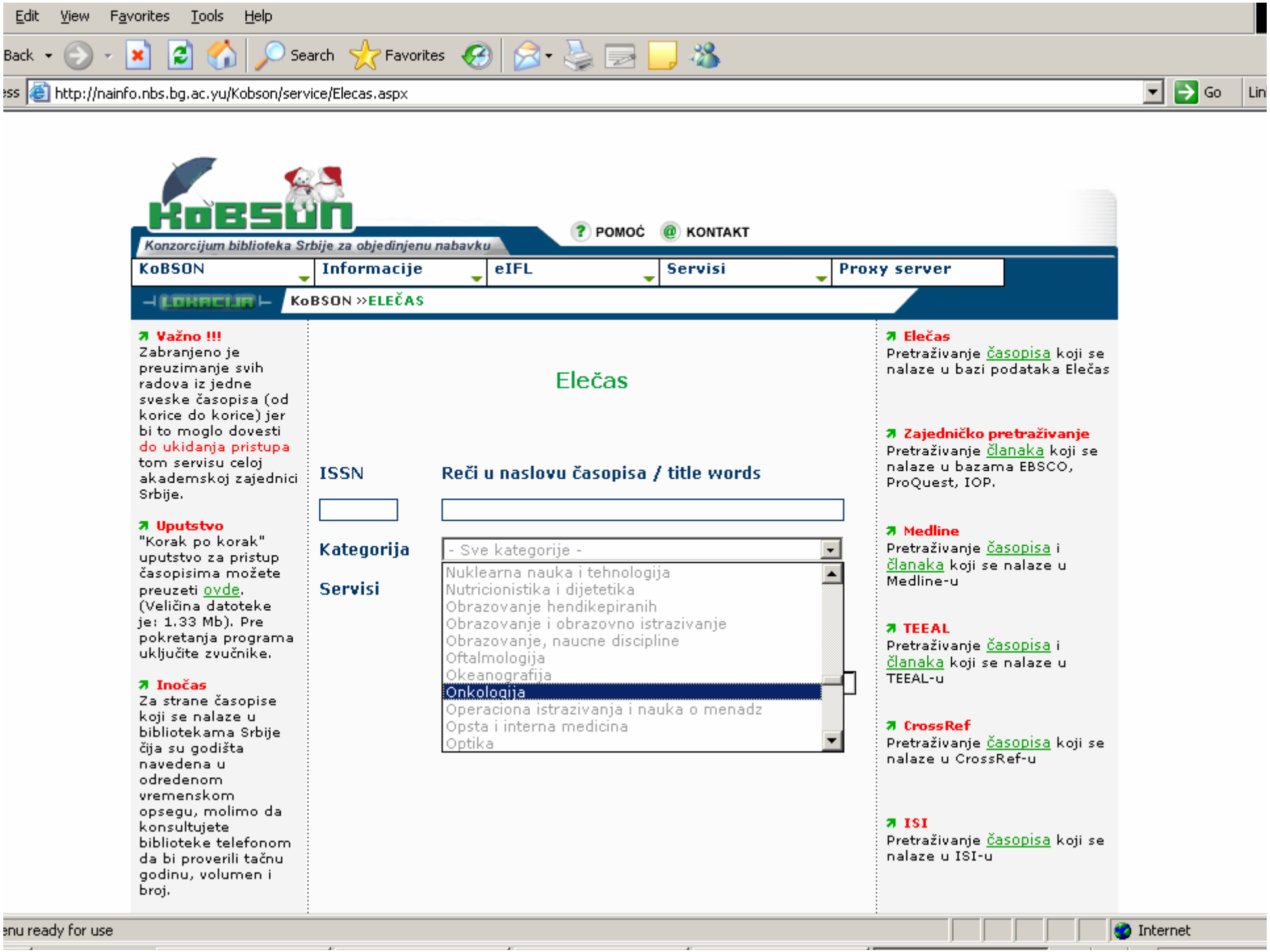
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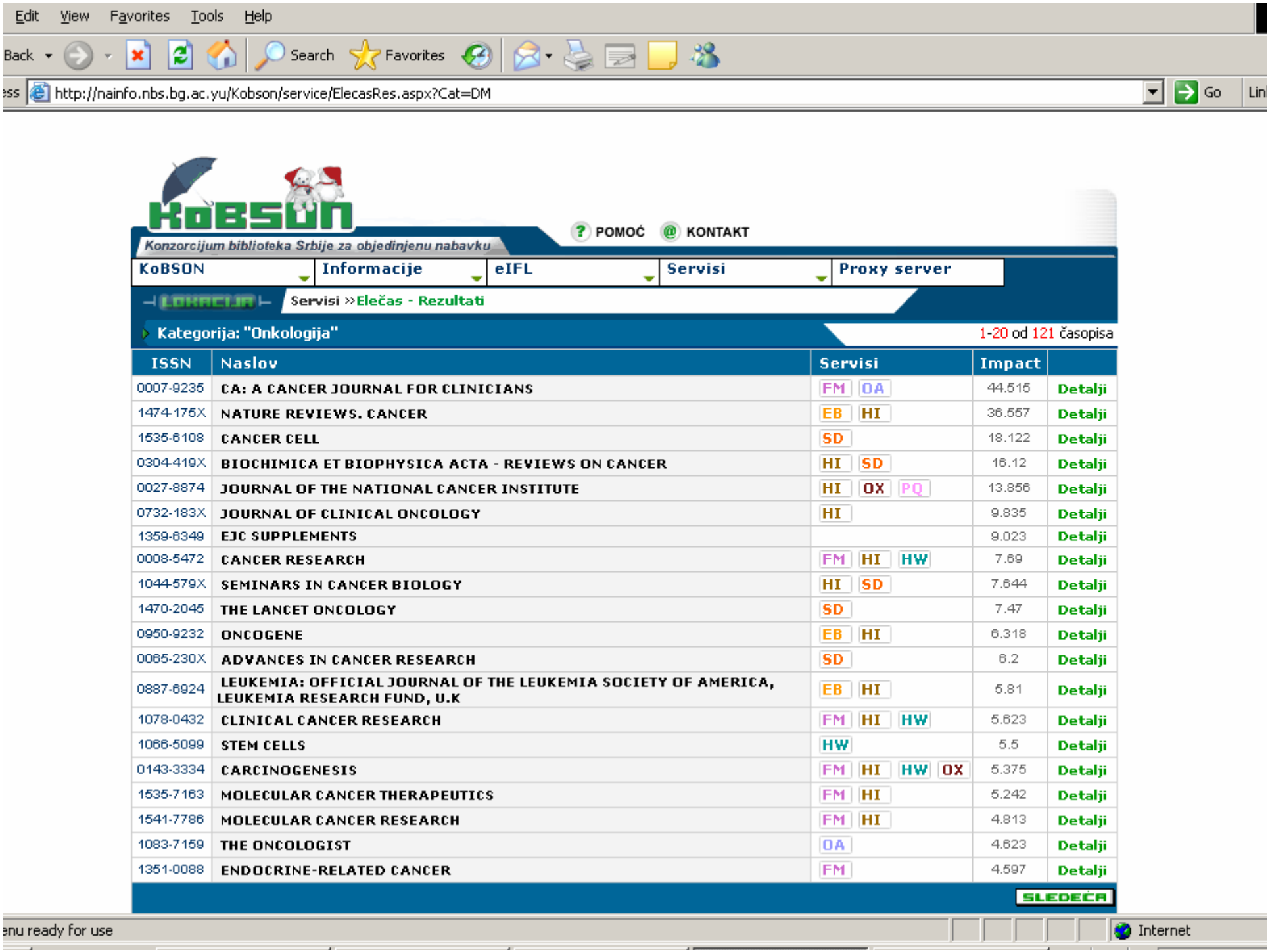
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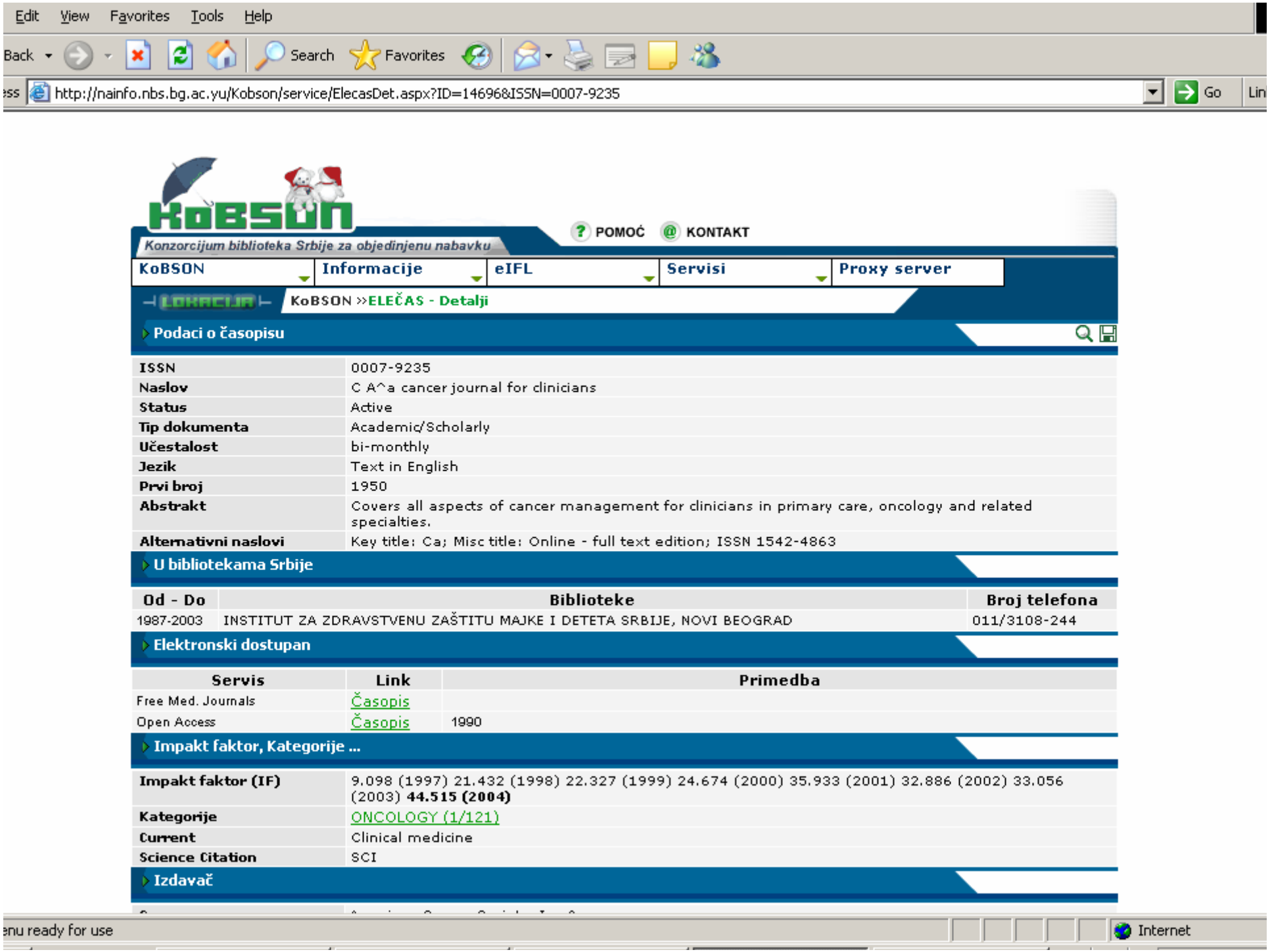
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Abstrakt Covers all aspects of cancer management for clinicians in primary care, oncology and related specialties.
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SMOKING CESSATION: TWO OUT OF "5A'S" AREN'T ENOUGH
CA Cancer J Clin 2005 55: 331-333. [FREE Full Text] [FREE PDF]

ARTICLES:

Rhonda F. Souza and Stuart J. Spechler
Concepts in the Prevention of Adenocarcinoma of the Distal Esophagus and Proximal Stomach
CA Cancer J Clin 2005 55: 334-351. [Abstract] [FREE Full Text] [FREE PDF] [FREE CME]

Gary Y. Yang, Timothy D. Wagner, Martin Fuss, and Charles R. Thomas, Jr.
Multimodality Approaches for Pancreatic Cancer
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James O. Armitage
Staging Non-Hodgkin Lymphoma
CA Cancer J Clin 2005 55: 368-376. [Abstract] [FREE Full Text] [FREE PDF] [FREE CME]

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Multimodality Approaches for Pancreatic Cancer

Gary Y. Yang, MD; Timothy D. Wagner, MD, MBA; Martin Fuss, MD; Charles R. Thomas, Jr., MD

Dr. Yang is Assistant Professor, Residency Program Director, Department of Radiation Medicine, Roswell Park Cancer Institute, Buffalo, NY.

Dr. Wagner is Chief Resident, Department of Radiation Medicine, Roswell Park Cancer Institute, Buffalo, NY.

Dr. Fuss is Associate Professor, Department of Radiation Oncology, University of Texas Health Science Center at San Antonio, San Antonio Cancer Institute, San Antonio, TX.

Dr. Thomas is Professor and Chairman, Department of Radiation Medicine, Oregon Health & Science University, Portland, OR.

This article is available online at http://CAonline.AmCancerSoc.org

ABSTRACT The role of combined-modality therapy for pancreatic cancer is evolving with the recent development and completion of major, multi-institutional clinical trials. One of the challenges for the busy clinician is to appreciate the variation in staging, surgical expertise, and application of either definitive chemoradiotherapy or adjuvant chemoradiotherapy for local and/or regionally advanced disease. Our aim is to summarize the current state-of-the-art management and future directions regarding the multimodality approach to pancreatic cancer. *(CA Cancer J Clin 2005;55:352–367.)* © *American Cancer Society, Inc., 2005.*

INTRODUCTION

Pancreatic cancer has the poorest prognosis of any common gastrointestinal malignancy, with a 5-year overall survival of less than 5%.[1] In 2005, it is estimated that 32,180 people will be diagnosed with and 31,800 deaths will be attributed to pancreatic cancer in the United States. It represents the second leading cause of death among gastrointestinal malignancies and the fourth leading cause of cancer death overall in the United States.[2] Although the incidence has stabilized nationally over the last 25 years at approximately 11 cases per 100,000 people per year, there has been very little improvement in death-to-incidence ratio, which continues to approach 0.95.[3]

Certain prognostic factors, both positive and negative, will help guide the appropriate therapy. Historically, those patients with an Eastern Cooperative Oncology Group (ECOG) performance status of 0 to 1, early (T1–T2) lesions with no preoperative evidence of lymph node involvement, or tumor encasement of the superior mesenteric vessels and portal vein have a more favorable outcome. In addition, those lesions located toward the head of the pancreas are more likely to be surgically resectable. Factors predicting a poorer outcome include the presence of gross or microscopic residual disease following an attempted curative resection, invasion of the major vessels, and perineural and/or lymphatic invasion. The presence of metastasis to local lymph nodes and distant metastasis are both negative prognostic factors for overall survival.[1,4–6]

The poor survival associated with pancreatic cancer stems from the advanced stage at diagnosis for the majority of cases. In many instances, even in patients with apparently localized resectable tumors, distant micrometastases

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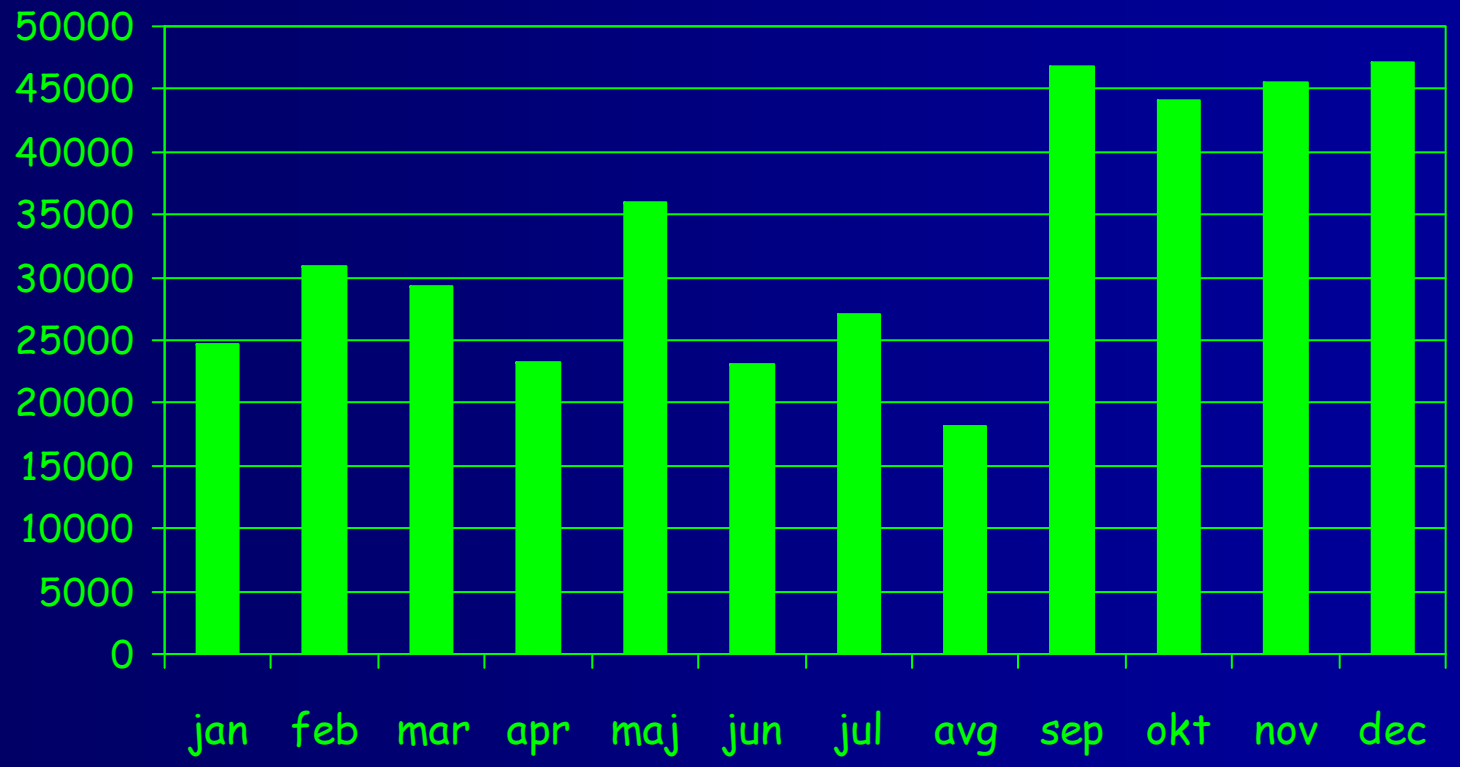

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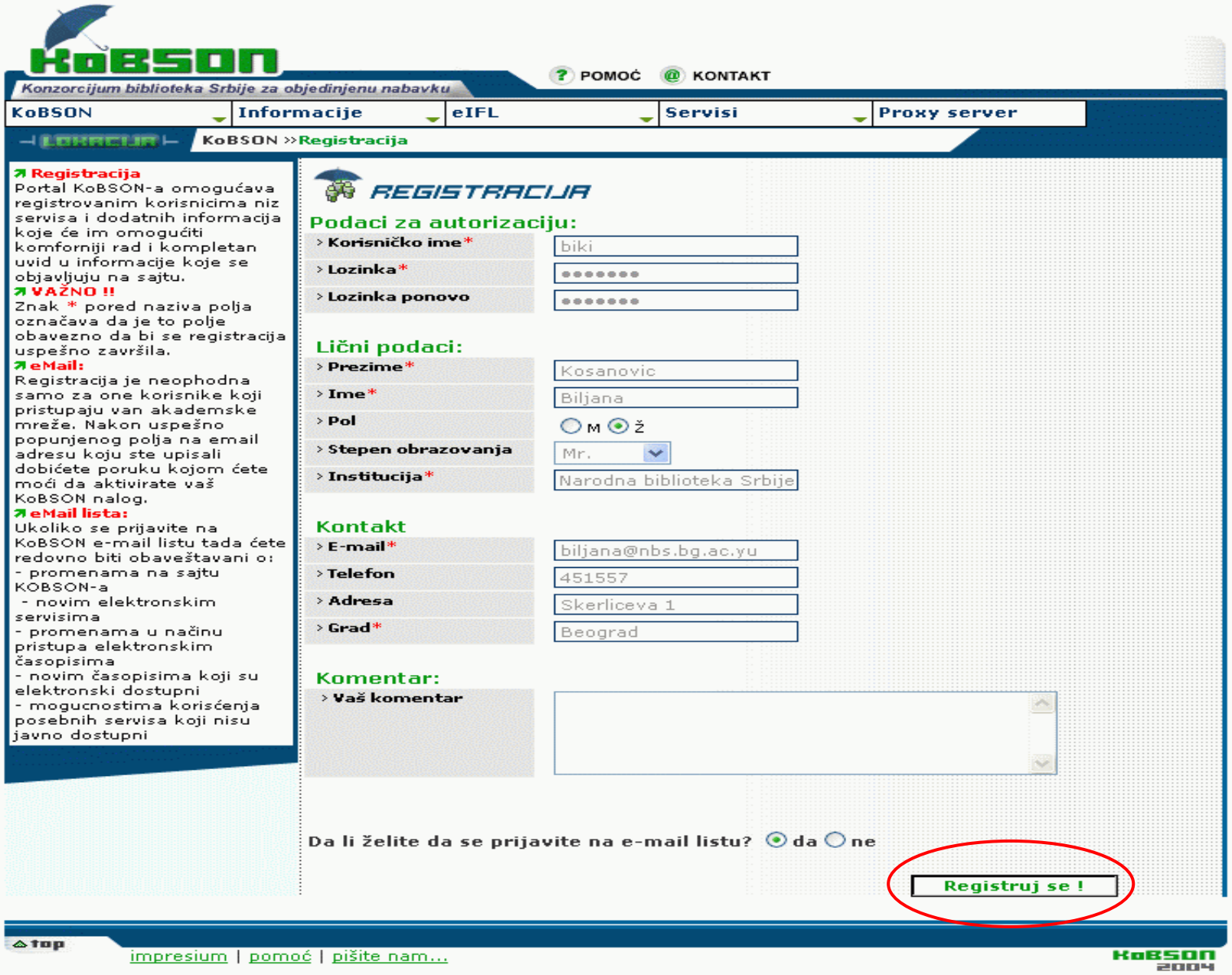

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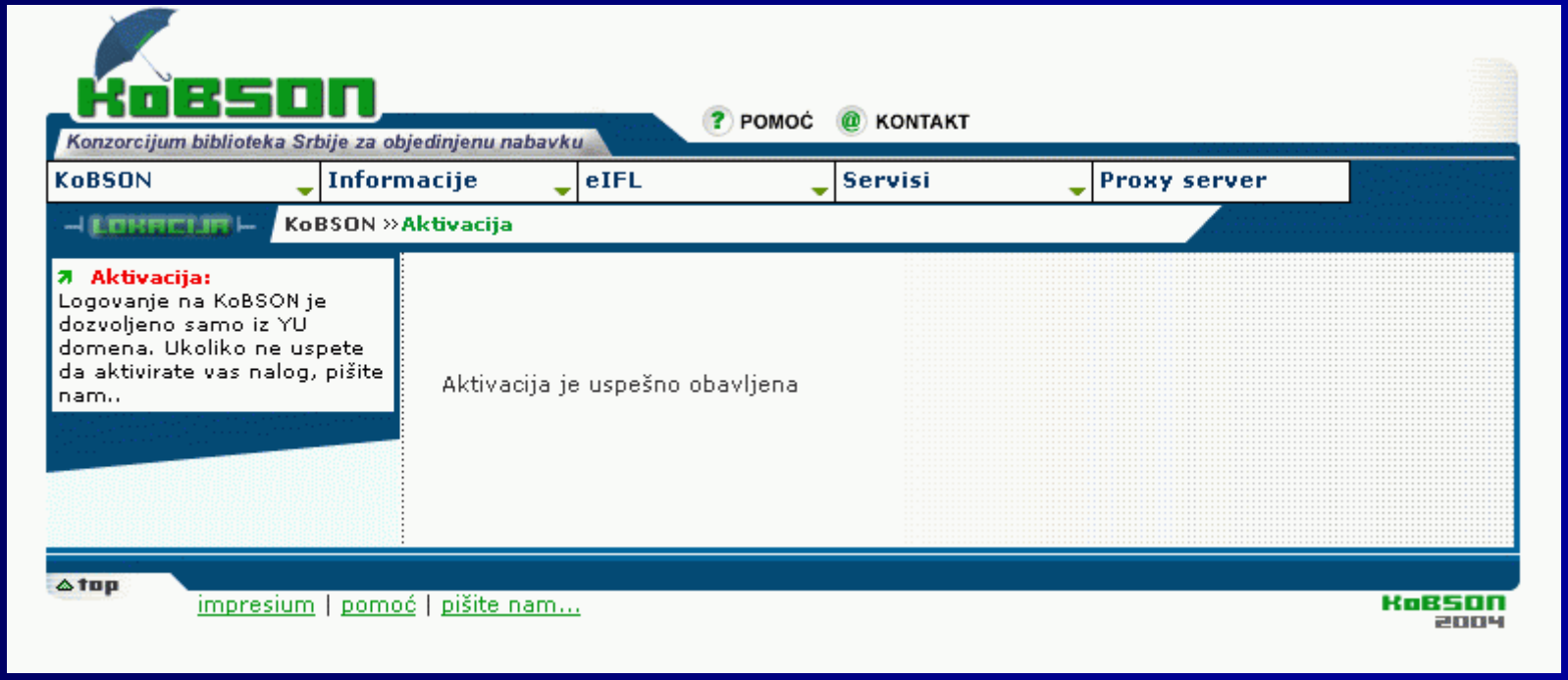

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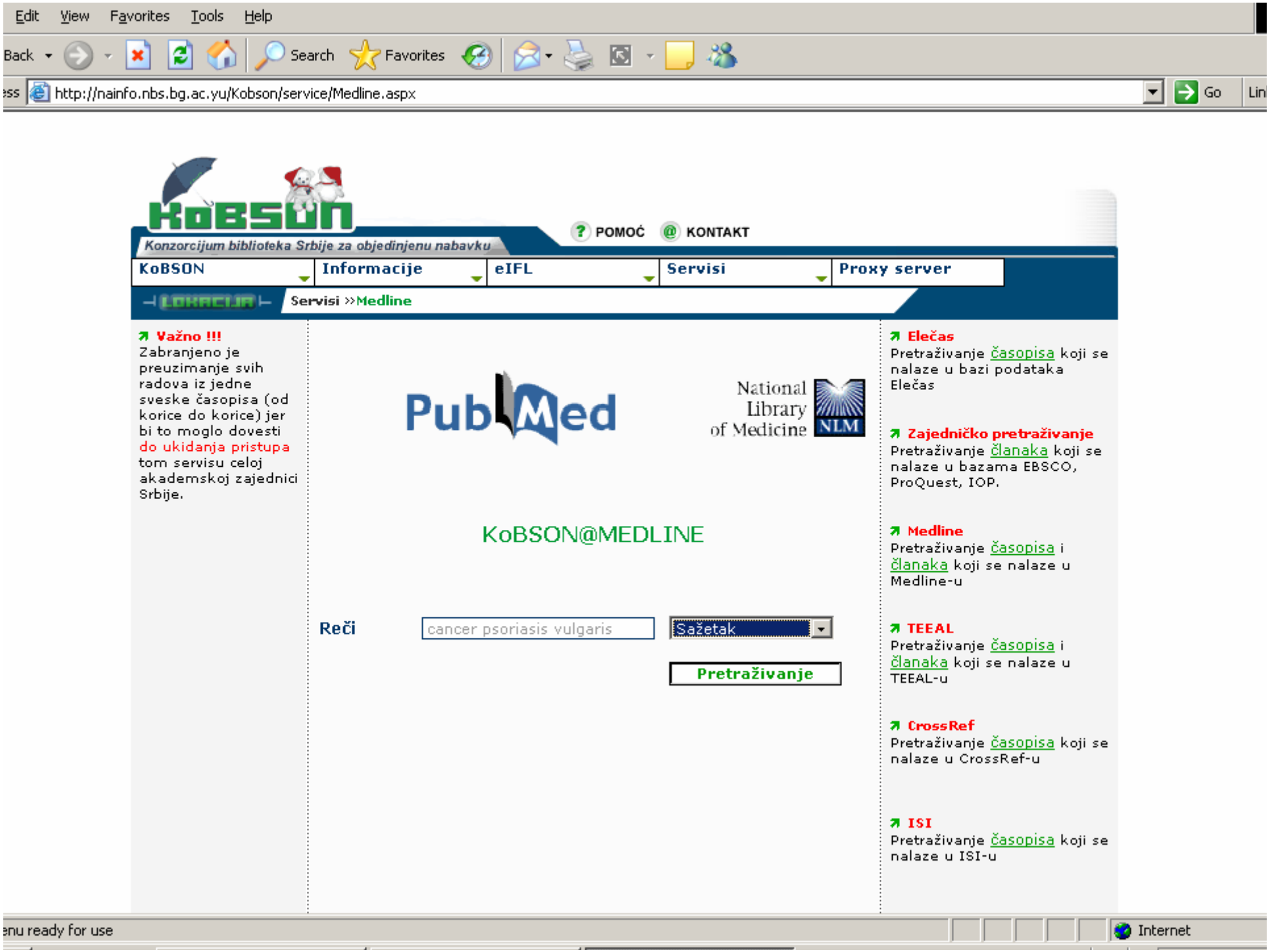
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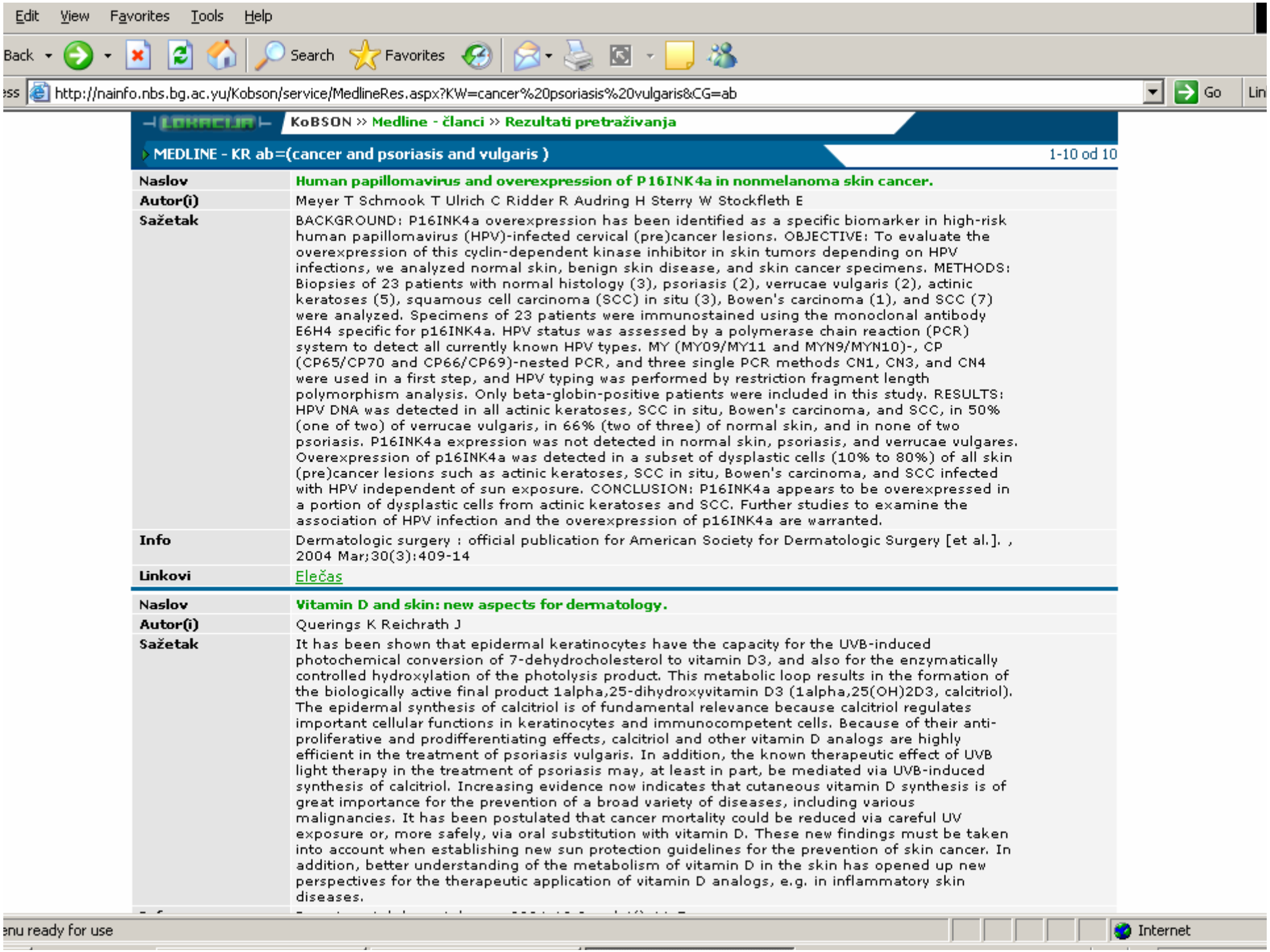
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Human papillomavirus and overexpression of P16INK4a in nonmelanoma skin cancer.
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BACKGROUND: P16INK4a overexpression has been identified as a specific biomarker in high-risk human papillomavirus (HPV)-infected cervical (pre)cancer lesions. OBJECTIVE: To evaluate the overexpression of this cyclin-dependent kinase inhibitor in skin tumors depending on HPV infections, we analyzed normal skin, benign skin disease, and skin cancer specimens. METHODS: Biopsies of 23 patients with normal histology (3), psoriasis (2), verrucae vulgaris (2), actinic keratoses (5), squamous cell carcinoma (SCC) in situ (3), Bowen's carcinoma (1), and SCC (7) were analyzed. Specimens of 23 patients were immunostained using the monoclonal antibody E6H4 specific for p16INK4a. HPV status was assessed by a polymerase chain reaction (PCR) system to detect all currently known HPV types. MY (MY09/MY11 and MYN9/MYN10)-, CP (CP65/CP70 and CP66/CP69)-nested PCR, and three single PCR methods CN1, CN3, and CN4 were used in a first step, and HPV typing was performed by restriction fragment length polymorphism analysis. Only beta-globin-positive patients were included in this study. RESULTS: HPV DNA was detected in all actinic keratoses, SCC in situ, Bowen's carcinoma, and SCC, in 50% (one of two) of verrucae vulgaris, in 66% (two of three) of normal skin, and in none of two psoriasis. P16INK4a expression was not detected in normal skin, psoriasis, and verrucae vulgares. Overexpression of p16INK4a was detected in a subset of dysplastic cells (10% to 80%) of all skin (pre)cancer lesions such as actinic keratoses, SCC in situ, Bowen's carcinoma, and SCC infected with HPV independent of sun exposure. CONCLUSION: P16INK4a appears to be overexpressed in a portion of dysplastic cells from actinic keratoses and SCC. Further studies to examine the association of HPV infection and the overexpression of p16INK4a are warranted.
Info
Dermatologic surgery : official publication for American Society for Dermatologic Surgery [et al.]. , 2004 Mar;30(3):409-14
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Vitamin D and skin: new aspects for dermatology.
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It has been shown that epidermal keratinocytes have the capacity for the UVB-induced photochemical conversion of 7-dehydrocholesterol to vitamin D3, and also for the enzymatically controlled hydroxylation of the photolysis product. This metabolic loop results in the formation of the biologically active final product 1alpha,25-dihydroxyvitamin D3 (1alpha,25(OH)2D3, calcitriol). The epidermal synthesis of calcitriol is of fundamental relevance because calcitriol regulates important cellular functions in keratinocytes and immunocompetent cells. Because of their anti-proliferative and prodifferentiating effects, calcitriol and other vitamin D analogs are highly efficient in the treatment of psoriasis vulgaris. In addition, the known therapeutic effect of UVB light therapy in the treatment of psoriasis may, at least in part, be mediated via UVB-induced synthesis of calcitriol. Increasing evidence now indicates that cutaneous vitamin D synthesis is of great importance for the prevention of a broad variety of diseases, including various malignancies. It has been postulated that cancer mortality could be reduced via careful UV exposure or, more safely, via oral substitution with vitamin D. These new findings must be taken into account when establishing new sun protection guidelines for the prevention of skin cancer. In addition, better understanding of the metabolism of vitamin D in the skin has opened up new perspectives for the therapeutic application of vitamin D analogs, e.g. in inflammatory skin diseases.
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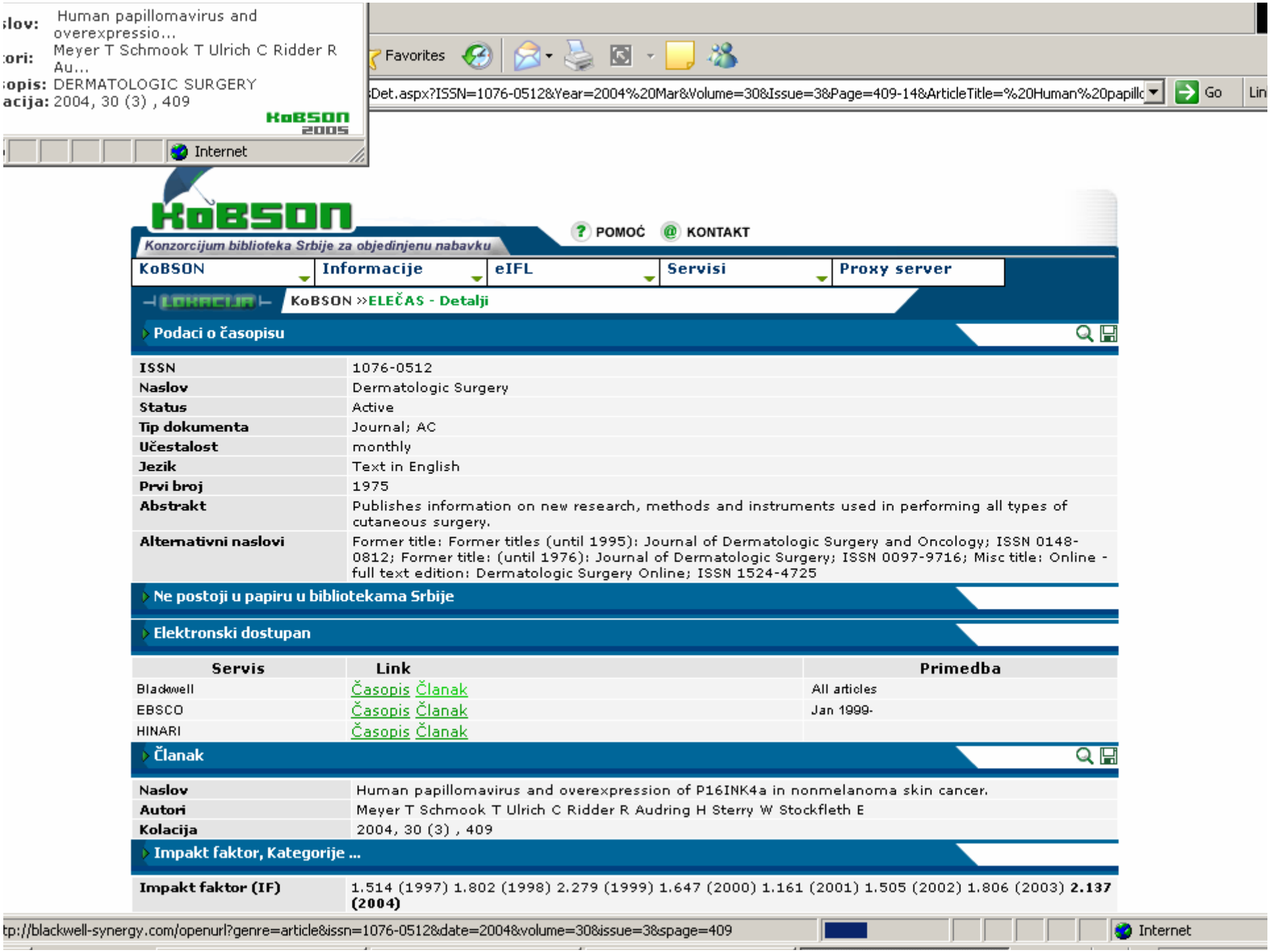
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Human Papillomavirus and Overexpression of P16^{INK4a} in Nonmelanoma Skin Cancer

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BACKGROUND. P16^{INK4a} overexpression has been identified as a specific biomarker in high-risk human papillomavirus (HPV)–infected cervical (pre)cancer lesions.

OBJECTIVE. To evaluate the overexpression of this cyclin-dependent kinase inhibitor in skin tumors depending on HPV infections, we analyzed normal skin, benign skin disease, and skin cancer specimens.

and CN4 were used in a first step, and HPV typing was performed by restriction fragment length polymorphism analysis. Only β-globin–positive patients were included in this study.

RESULTS. HPV DNA was detected in all actinic keratoses, SCC in situ, Bowen's carcinoma, and SCC, in 50% (one of two) of verrucae vulgaris, in 66% (two of three) of normal skin, and in none of two psoriasis. P16^{INK4a} expression was not detected in

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