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Greetings from the New Editor-in-Chief

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The purpose of this brief editorial is twofold. As the new editor-in-chief of Journal of Clinical and Experimental Pathology I would like to introduce myself to our readers. I have been a professor of Pathology at The University of Georgia for many years. For long time I have worked in the vast area of growth factors, specifically, on the identification and characterization of Transforming Growth Factor e (TGFe), a member of the (pro)granulin family [1-3], and on characterization of avian isoforms of TGFβ. For the last 12 years I have worked on tendon diseases in domestic animals. Our laboratory has worked on enrofloxacin-induced changes in proteoglycan and glycosaminoglycans in avian and equine tendon cell cultures [4,5]. Another major project in our laboratory is biochemical characterization and elucidation of pathogenesis of an equine disease leading to pain and lameness in certain horses, so called degenerative suspensory ligament desmitis. Until recently this disorder was considered a disease of collagen affecting exclusively so call suspensory ligaments of equine legs. However, we have determined that the primary defect is in processing of proteoglycans which accumulate in tissues and organs with high content of connective tissues [6,7].

I hope that the interest of readers in Journal of Clinical and Experimental Pathology will rise and inspire them to submit their papers in the near future. We are looking for reports from all areas of pathology, but particularly, original reports on new assays and technology use in diagnosis are welcome.

Just a few comments on one paper in this issue. The paper describes the author's experience with evaluation of nuclear changes in oral leukoplakia [8]. This small study deals with an important problem of diagnosing and evaluating malignant potential of oral leukoplakias, well known precancerous lesions of the oral cavity. That their origin and prevalence is tied to smoking has been recognized for a long time [9]. A fairly high prevalence and incidence leukoplakia has been observed in HIV-infected patients as well [10], however, the relationship of leukoplakias to human papillomavirus is less clear [9]. Because of high degree of disagreement among pathologists on the degree of dysplasia and/or presence of outright malignancy and the paucity of standardized detection assays the paper by Chatterjee is a welcome addition to the Journal. The author found that determination of mitotic indices was unhelpful, however, statistical evaluation of nucleoli numbers showed a significantly and progressively increased count for nucleoli in dysplastic and malignant lesions. Interestingly, Ki67 was not found of much use [8]. If confirmed in more studies involving many more samples and patients (in this study 50 archival samples were evaluated) this assay would represent an easy and simple for evaluating an important lesion, and would enable to implement treatment and measures aimed at prevention of recurrences more rapidly and effectively. This is particularly important as up to 37% of dysplastic oral leukoplakia progress to invasive squamous cell carcinoma within 5 years [11]. The same authors found marked difficulty in recruiting patients study utilizing medical records as a screening method for leukoplakia clinical trial entry [11].

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