of therapy, we have carried out the following studies: Clinical-sky analysis of blood, biochemical analysis of blood, morfologische and immuno-histochemically the study of liver biopsy spec-imens). Performance evaluation of Pro-was carried out using 1, 6, 12 months after the start of therapy.

The results of the study: In the study group experienced a more rapid normalization of syndrome of cytolysis ALT, AST is characterized by the presence of Las in the first month after initiation of therapy, complete normalization of the syndrome of cytolysis and cholestasis marked by a 3-month treatment. In the control group, normalization of indicators of cytolysis and cholestasis was noted by 6 months-TSU after the start of the hepatoprotective and pathogenetic therapy.

We have found that patients in the study group, characterized by the presence of is the increase in the number of T-helper cells (CD 3+, CD 4+) relative indicators-La, also, the reduction of T-cytotoxic lymphocytes, relative and absolute measures, a significant increase in IL-8 in sponta-neous and induced activity in the serum. In the study of liver biopsy specimens revealed a reduc-tion of CD3 T-lymphocytes, CD8 T lymphocytes, and CD68, an increase in T-lymphocytes, which indicates a lowering of inflammatory histio-lymphocytic infiltration of the liver tissue. In the HS from patients, there is maintaining the same level of these indicators, which indicates the degree of inflammation in the liver tissue.

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THE CONCEPT OF CARCINOGENESIS IN VIRAL CONTAMINATION

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Introduction. Despite numerous information on the biology of the virus, ways of infection, pathogenesis, clinical manifestations of infection, HPV infection, the nature and mechanisms of the development of the growth of the structures of the

skin that have been contaminated with the virus are still unknown, and it is also not proven that it is the papillomaviruses that produce starting substances for carcinogenesis [7, 9, 12].

Morphological identity is set against the backdrop of the developing cells of prolonged human papillomavirus infection (HPVI) human blasts skin tumors cells human blood smear, a patient with leukemia [1, 13]. Morphologically and immunohistochemically cells that populate the area damaged, apply to blood stem cells. It was showed dinamics changes structures of the skin on phone HPVI, characterized by the violation of the interaction between the epidermis and the underlying connective tissue, the destruction of the basement membrane, violation of the main differentiation of keratinocytes, the lack of perlatum and granular layers. It is concluded that the identification of progenitor cells in the human skin damage associated with violation of intercellular interactions in the epithelium-mesenchymal tissue [3, 11]. Increased proliferative activity, changing lives main keratinocytes, moving the Effector antigen presenting CD68 in contiguous to skin connective tissue, are inductors factors for migration stem cells leucocytes in area destruction of the skin. Disappearing not only property restitution keratinocyte and epithelial dependent differentiation of young blood. Migration not differentiation cells is the vector of the blood – zone of alteration of epithelial cells, but not vice versa [6, 10]. As a result of insufficient or no differentiation of progenitor blood cells in the epidermis and damage surrounding tissue starts to carcinogenesis. Offered an author's model of carcinogenesis, modifying model Correa (1990). Conclusions based on the results of the work are changing perceptions about the pathogenesis of cancer and provide a new strategy towards finding the earliest diagnosis of carcinogenesis [2, 5]. Also, precise mechanisms of damage to the skin and mucous membranes of human HPV, as well as the origin of tumor cells, have not been established [4]. Cambium of its own tissues, hich has lost the ability to differentiate into mature cells, or cells with a damaged oncogene genome, are the main open questions in the study of tumor cells [8]. Given the high infection rate of the population of HPV, as well as the high risk of cancer on the background of HPV contamination, the relevance of studying the features of the alteration of the skin with prolonged HPV infection is extremely high. We have studied the features of damage to human skin structures in chronic HPV infection in the dynamics of reparative regeneration. Skin biopsies were obtained in accordance with the Helsinki Declaration, fixed according to classical protocols and embedded in paraffin. Sliches and all further processing of the material were performed on the automated equipment of the laboratory of pathomorphology of the Medical University of Niigata (Japan). Identification of immunocompetent cells was carried out according to the same scheme, in spite of different antigen localization in cellular structures: membranes, lysosomes, Golgi complex. Immunohistochemical methods were used to determine CD68 and the intensity of proliferative processes in the epithelial plate was studied by using a marker on the Ki-67 gene protein. Antibodies were used at a dilution of 1:50 and 1:100. The material was analyzed using an Olympus-Bx82 microscope and a digital camera PDx25.

Results and its discussion. We have established that in chronic HPV infection with a disease duration of more than 2 years, in the age group over 45 years, in the skin of some patients after necroticisation of part of the epidermis, both on the surface and in the depth of papillomatous growths, infiltration of blood cells around the zone damage to the tissue. Depletion of CD68 in non-inflammatory papillomas with HPV is associated with a decrease in the level of MIP-3α and E-cadherins of keratinocytes, followed by migration to the inflammation zone of various subsets of DC and cytotoxic T cells. The total apoptosis in the prickly and basal layers of the epidermis is accompanied by an increase in the proliferative activity of basal keratinocytes, and then depletion of the regenerative potential. In addition, leukocyte infiltration, which when HPV infection before 2 years is observed only within the connective tissue of the dermis, is accompanied by its spread beyond the destroyed basement membrane to the epidermis up to the shiny and horny layers, replacing the layer of cambial cells of the epidermis.

Due to the death of keratinocytes of the basal layer and in the absence of differentiation and specialization of apoptotic cells of the spiky epidermis layer, only the shiny and horny layers are preserved, sometimes granular. When an inflammatory infiltrate appears under a shiny layer in combination with the destruction of the basal membrane of the epidermis and the death of basal and prickly cells, they are replaced by blood cells unable to differentiate into effector cells.

Given the tropicity of HPV to the cambial cells of the epidermis, we can conclude that the regenerative potential of stem keratinocytes is depleted, and keratinocyte production of inducers for the maturation of incoming young undifferentiated lymphocytes is stopped.

Therefore, we concluded that, possibly, the dying cambial cells of the epidermis could signal the migration of young undifferentiated blood cells to the area of skin damage, to close the defect, but without performing the function of the destroyed HPV structures.

Differentiation of these cells in the new conditions of contact interactions is absent, at the beginning of reparative regeneration they have only high proliferation - as an adaptation to the damaging effect of HPV. It should be noted that our data are consistent with the data of Susman S, Tomuleasa C, Soritau O, et al. (2012), who found similarity between colon cancer cells and blood cells in leukemia [15]. But these authors, like Warburg (1924) [14], suggested that it was the gene mutations that caused the cambial epithelial cells of the intestinal mucosa tissue to rearrange into cancerous cells. Our findings differ fundamentally from the conclusions of Susman S. et al. (2012), since we believe that the stem cell tumor is performed by blood stem cells as a result of the antigen presentation violation, the initiation of apoptosis of keratinocytes, and then adaptation to the physiological demand of tissue in conditions of impaired reparative regeneration. The absence of an epidermal factor in the differentiation of lymphocytes (ELDIF) in connection with the death of keratinocytes leads first to the appearance of blast cells in the area of damage, and later fibroblast-like cells appear that participate in the structuring of the developing cyst towards the tumor of a more dense tissue organization.

Conclusion. According to many authors, neoplastic transformation is a multi-step process associated with progressive accumulation of damage in DNA reparants in cambial tissue cells, as well as tumor suppressors, oncogenes, growth factors, cell surface receptors, and cell adhesion molecules. It is generally accepted that genetic instability, inactivation of oncosuppressive genes, overexpression of telomerase predispose to early carcinogenesis, while oncogen activation, expression of growth factors, cytokines and angiogenic factors lead to late tumor progression and invasion [8]. In our studies, data have been obtained confirming that in the connective tissue of the human skin in HPV infection the physiological regeneration algorithm, leading to the appearance of sprouting in the form of warts and condylomas, is violated, which is associated with a violation of the antigen presentation of CD68, their migration to the underlying connective tissue against the background of their complete absence in epithelial beds. Peculiarities of the distribution of effector immunocytes CD68 in HPV indicate a violation of antigen presentation in the structures of the human skin and subsequent reduction of control over physiological and reparative regeneration as a whole, which can lead to disruption of cellular interactions in the effector immunocyte system and initiation of the oncogenesis process in the structures of the skin. One of the causes of the disorder is that keratinocytes in the postnatal ontogenesis secrete the epidermal lymphocyte differentiation factor (ELDIF), which inhibits proliferation and stimulates differentiation, specialization and maturation in whole lymphocytes [13], which agrees with our conclusions about the origin of the tumor from young blood cells, migrating to the zone of damage as a result of changes in signals from damaged epithelial cells.

The foregoing allows us to modify the model of carcinogenesis according to P. Correa (1990). The process of oncogenesis in the presumed infectious etiology can be divided into 2 stages: Stage I — changes in local local immune homeostasis — increase in proliferative activity in the case of damaging effects of any etiology (microbial, carcinogenic, hormonal) — metaplasia — infiltration of effector cells of the keratinocyte damage zone — redistribution of APC — apoptosis — death of the cambium (point of no return) — destruction of the basal membrane; II — generalized process in the body — disorders in the hematopoiesis system and immunogenesis — migration of blood stem cells into the area of skin damage — secondary immunodeficiency.

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THE EPITHELIUM BARRIER OF THE GASTROINTESTINAL TRACT IN PATHOLOGY

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Introduction. We investigated *H. pylori* infection in children patients with gastrointestinal diseases in Vladivostok, Far Eastern Russia. In this study, we further investigated the role of *Helicobacter pylori* infection in lactase deficiency pathogenesis in children. In the pediatric fields, secondary and transient lactase deficiency was seen during clinical practice of different gastrointestinal diseases. Many previous studies have shown the mucosal conditions of small intestine and duodenum in secondary lactase deficiency; however, local immune responses in gastrointestinal tract have not been examined [1–10]. Especially, conditions of gastric mucosa and epithelium in different pathogenetic variants of lactase deficiency in infants and children under 3 years have not been well studied. In this study, we investigated roles of *H.pylori* infection and immune responses of gastric mucosa and epithelium in, pathogenetic aspects of lactase deficiency in children under 3 years.

Methods. Sixty-three pediatric patients (age: 5 months to 3 years) with different loss of weight in Regional Clinical Center of Maternity, Vladivostok, Russia, were also included during 2008–2011. All patients were diagnosed as lactase deficiency. Morphological changes of gastrointestinal mucosa were examined by endoscopy and dark field microscopy. *H. pylori* in biopsy specimens was detected by immunostaining. CD4-, CD8-, CD 68-, CD163-, or CD204-positive immune cells in the specimens were detected by immunostaining.