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European Journal of Histochemistry

a journal of functional cytology

The European Journal of Histochemistry was founded in 1954 by Maffo Vialli and published till 1979 under the title of *Rivista di Istochimica Normale e Patologica*, from 1980 to 1990 as *Basic and Applied Histochemistry* and in 1991 as *European Journal of Basic and Applied Histochemistry*. It is now published under the auspices of the University of Pavia, Italy.

The European Journal of Histochemistry is the official organ of the Italian Society of Histochemistry and a member of the journal subcommittee of the International Federation of Societies for Histochemistry and Cytochemistry (IFSHC), and has been an influential cytology journal for over 60 years, publishing research articles on functional cytology and histology in animals and plants.

The Journal publishes Original Papers, Technical Reports, Reviews, Brief Reports, Letters to the Editor, Views and Comments, and Book Reviews concerning investigations by histochemical and immunohistochemical methods, and performed with the aid of light, super-resolution and electron microscopy, cytometry and imaging techniques; attention is also given to articles on newly developed or originally applied histochemical and microscopical techniques.

Coverage extends to:

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- cell differentiation and death;
- cell-cell interaction and molecular trafficking;
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P03**THE ROLE OF FILAMIN A IN REGULATING BREAST CANCER CELL BEHAVIOR AND TUMOR MICROENVIRONMENT INTERACTIONS**

W. Arendt¹, P. Zawadka¹, M. Hałas-Wisniewska¹, M. Gagat², M. Izdebska¹

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Filamin A (FLNa) is a multifunctional actin-binding protein involved in maintaining cell architecture, transducing mechanical signals, and regulating cytoskeletal dynamics. Its role in cancer is complex and context-dependent, particularly in breast cancer, where FLNa has been reported to act either as a tumor suppressor or a promoter of malignancy. In this study, we investigated the contribution of FLNa to key processes driving breast cancer progression, including proliferation, migration, invasion, and adhesion. FLNa expression was modulated in two breast cancer cell lines: the highly invasive triple-negative MDA-MB-231 and the luminal, less aggressive MCF-7. Efficient downregulation of FLNa was confirmed by Western blotting and quantitative fluorescence analysis. A series of functional assays -including wound healing, open-field migration, modified Boyden chamber, and adhesion assays- were conducted to assess the phenotypic consequences of FLNa depletion. Control groups consisted of cells with unaltered FLNa expression. Our findings show that reduced FLNa expression significantly alters the migratory, invasive, and adhesive properties of breast cancer cells. These results underscore the role of FLNa as a critical regulator of tumor cell behavior and support its relevance as a potential therapeutic target. However, due to the cell line-specific effects and conflicting reports in the literature, further research is required to clarify the context-dependent functions of FLNa within diverse tumor microenvironments.

P04**CYTOCHEMICAL AND BIOLOGICAL EFFECT OF BIORESONANCE IN IRRITABLE BOWEL SYNDROME**

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Technological advances in the field of instrumental therapies have allowed for progress in the creation of equipment capable of treating multiple pathologies through a bio-quantum approach^{1,2}. The opportunity of exploiting electromagnetic physical energies opens new scenarios in relation to the possibilities of conditioning in a curative way the structure and functionality of specific tissues and systems of the human body^{1,3}. In this context we can find the approach of Bioresonance Therapy (BT), which leverages the specificity of the electromagnetic signal of each biological tissue,

that could in fact be the objective and means of calibrated stimulations aimed at inducing an inversion of pathobiological processes^{1,3}. A pathology characterized by altered quality and function of tissues involved is Irritable Bowel Syndrome (IBS)^{1,4}. Therefore, we made a study on 20 patients (12 women and 8 men, mean age of 51 years) to verify the ability of BT to influence IBS through a direct action of its low-intensity magnetic fields on the biological tissues of the affected organs¹. To monitor the health of patients, we observed the values recorded, before and after the execution of a cycle of 8 BT sessions, for the Short-Form Health Survey 36, the Bristol Stool Form Scale and, above all, the Fecal Calprotectin. The results of our study suggest that BT is associated with a significant improvement in all the parameters observed and to an inversely proportional correlation between the Calprotectin values and the quality of life experienced by the patients in relation to the perceived IBS symptomatology¹. Therefore, it is possible to highlight both the efficacy of the proposed treatment and the important role of the collaboration between clinical medicine and the physical and histochemical sciences in defining the validity and the possibilities of improvement attributable to innovative therapeutic techniques such as BT.

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P05**ENTERIC NERVOUS SYSTEM NEUROPLASTICITY IN COLON OF ANIMAL MODELS OF OBESITY**

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An altered functional innervation of the intestine by the enteric nervous system (ENS) has been demonstrated in different diseases. Pathological changes in histological features confirm neuronal plasticity in metabolic syndrome and related obesity conditions. Changes in functionality and composition of gut microbiota and intestinal dysbiosis seem closely linked to the degeneration of myenteric plexus in the ENS¹. Animal models of genetic obesity (Obese Zucker's rat), Diet-induced obesity (DIO rats) and Western diet-induced obesity (Cafeteria rats)² were studied to appreciate the progression of changes in glial cells and neurons along the innervation of the gut, particularly at the level of colon. Using different markers through immunohistochemical and immunochemical techniques, the morphological and functional modulations of the heterogeneous neuronal population of the gut wall were evaluated and the histological damage, neurodegeneration, and pro-inflammatory cytokines expression were detected on the colon. Myenteric neurons showed a reduction in neuronal markers expression, suggesting a degeneration associated with obesity. An altered immunoreactivity of enteric glial cells (EGCs) was found in the obese rats, which pointed out a suffering condition of nervous tissue suggesting a degeneration related to lipotoxicity. Also, the cholinergic and nitrergic networks appear affected by dysmetabolic conditions. In conclusion, the obesity establishment

seems to induce myenteric neurodegeneration. Also, gut dysbiosis promoted by an increment of intestinal permeability and related pro-inflammatory microenvironment makes an alteration of EGCs and neurons along the innervation of the gut in animal models of dysmetabolic conditions. However, further studies are needed to understand if the use a possible supplementation, such as prebiotic, probiotic or specific food are helpful in the management of intestinal disorders and resulting in a modulation of the ENS in the obese condition.

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P06

MICROGRAVITY-INDUCED CHANGES IN CYTOSKELETON STRUCTURE OF TCAM-2 CELLS

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Cytoskeleton, a mechano-sensing apparatus of the cells, is sensitive to gravity conditions¹. In our previous study, we investigated the effects of simulated microgravity (SM) on the cytoskeleton of human male germ cells (TCam-2). We observed that SM for 24 h altered microtubule and actin organization². To determine whether cytoskeletal changes occur during the early stages of SM exposure, we analysed the effects after a short 3 h period. Quantitative analyses were performed using Reverse Phase Protein Array (RPPA). The distribution pattern of cytoskeletal filaments was studied by indirect immunofluorescence (IIF) and Confocal microscopy, using maximum projections obtained from 1µm optical sections. RPPA analyses showed an increase in total cofilin and tubulin as early as 3h of exposure. Cellular cofilin exists in two forms: cofilin and phospho-cofilin (the latter represents the inactive form), both regulating actin polymerization and dynamics in the cytoplasm. However, both forms are also present in the nucleus, where they transiently influence gene regulation in response to specific stimuli. This dual localization highlights the dynamic regulatory role of cofilin phosphorylation in cellular processes³. Indirect IIF showed that, in SM samples, total cofilin levels increased while nuclear phospho-cofilin levels decreased. Consistent with RPPA data, tubulin levels also increased according to IIF. These observations suggest a possible perturbation of actin cytoskeleton caused by SM exposure, which in turn also affects tubulin polymerization and organization confirming the already known interaction between microtubules and microfilaments⁴. Future studies are necessary to better understand the biological significance of these modifications. Nonetheless, these results indicate that cytoskeleton remodelling occurs in TCam-2 cells as early as 3 h after SM exposure, providing further insight into how TCam-2 cells adapt to SM condition.

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P07

DISRUPTED GUT-VASCULAR BARRIER AND INFLAMMATORY REMODELING IN MNGIE: A SPATIAL TRANSCRIPTOMIC AND HISTOLOGICAL STUDY

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Mitochondrial Neurogastrointestinal Encephalomyopathy (MNGIE) is a rare genetic disorder caused by loss-of-function mutations in the enzyme thymidine phosphorylase (TP). TP deficiency disrupts nucleoside metabolism, leading to progressive and fatal mitochondrial dysfunction. Although liver transplantation restores TP activity and improves survival, it does not reverse gastrointestinal damage, which includes fibrosis, hypoxia, muscle layer disorganization, and sometimes life-threatening haemorrhage. TP plays a critical role in early blood vessel development, and its absence in MNGIE is associated with impaired vascularization, marked by the preponderance of very small (<50 µm) and more fragile vessels. To study vascular alterations, we applied Visium CytAssist spatial transcriptomics to full-thickness jejunal samples from two MNGIE patients and two controls. Despite limited tissue availability, this approach revealed distinct layer-specific transcriptional profiles, highlighting signatures of aberrant vascular remodelling. Among all the analysed tissue layers, considering specifically the vascular compartment, 68 differentially expressed genes (DEGs) were identified with a $p < 0.001$. Among these, we observed increased expression of genes involved in inflammatory and bacterial responses, including IGHA1, DEFA6, REG3A, DEFA5, JCHAIN, OLFM4, IGKC, REG1A, LYZ, DMBT1, and APOC3. This observation is consistent with the quantification of mast cells in full-thickness jejunal tissue. Compared to controls, MNGIE samples showed a 68% increase in mast cell count in the mucosa ($p = 0.0115$), an 82% increase in the submucosa ($p = 0.0076$), and a 98% increase in the neuromuscular layer ($p = 0.0017$). The submucosa itself contained 48 DEGs ($p < 0.001$), mirroring the vascular signature of increased inflammatory and bacterial response. Collectively, these data, together with jejunal histology, suggest impaired gut-vascular barrier integrity and increased permeability. Ongoing analyses of circulating biomarkers and dedicated permeability assays are underway to validate the molecular evidence presented in this preliminary study.