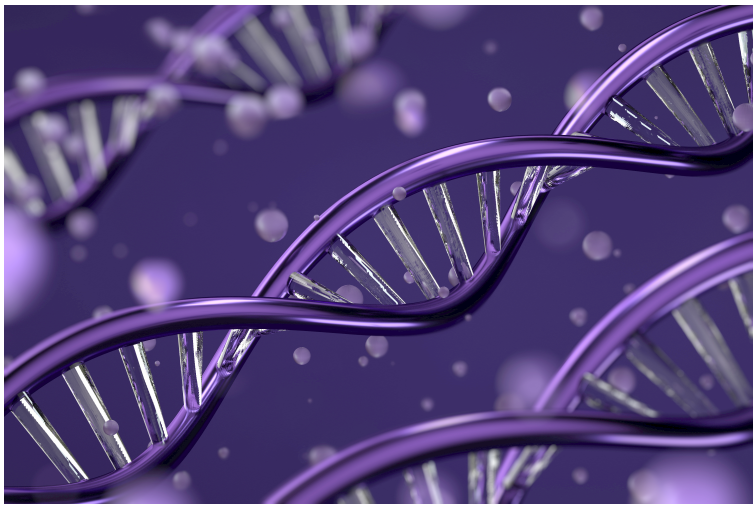
Università
degli Studi
della Campania
Luigi Vanvitelli



GMAX – LVU000001

MEASURMENT REPORT

GMAX FIELD-TECHNOLOGY-BASED MEDICINE

GMAX Aerospace – 2026
https://gmax-aerospace.com

GMAX AEROSPACE – QUANTUM FIELD DYNAMICS & SPACE RESEARCH LABORATORY
(FIELD-TECHNOLOGY-BASED MEDICINE – BIOINFORMATICS)
&
UNIVERSITÀ DEGLI STUDI DELLA CAMPANIA "LUIGI VANVITELLI"
(LUIGI VANVITELLI UNIVERSITY, CAMPANIA, NAPOLI)

HITE-BASED COMPARATIVE ANALYSIS

HITE - Hauszmann Integrated Tensor Theory

Primary laboratory indicators measured during the irradiation of A375 melanoma samples using GMAX Aerospace's quantum-coherent field technology in laboratory collaboration with the University of Campania "Luigi Vanvitelli" (LVU), as well as their comparison with A375 studies reported in the international scientific literature

similarities, differences, expected patterns, and unexpected features

Document type: monographic comparative analysis for publication preparation.

Focus: comparison of the short-term pattern of the LVU A375 laboratory measurement with the literature published on the A375 melanoma line concerning ultrasound, radiofrequency/hyperthermia, ECM adhesion, and mechanobiology.

Study number: LVU00001 (first laboratory study)

Study location: Azienda Ospedaliera Universitaria "Luigi Vanvitelli", Naples, Italy

Study date: 2026.03.18.

Applied wave type: HITE-based scalar waves

Applied technology devices: GMAX **CYGNUS** V.7.2 + GMAX **NEURA** V.6.0 + **SCHUM.ART.DNA** V.3.0 / V.3.1 **PYR**.

Source basis: HITE sources uploaded by the user and key publications from the publicly available literature from 2011–2025.

Prepared: April 17, 2026

Prepared by: Dr. János Hauszmann – GMAX Aerospace, Chief Engineer

List of experts participating in the Naples LVU laboratory measurements of CHL-1 and A375 melanoma samples

On behalf of GMAX Aerospace Ltd. (Technological Background)

	Name	Institution	Position Held (Parent Institution)	Role in the Project
	Zsolt Lengyel	GMAX Aerospace ltd.	CEO/ System Architect / Specialist / GMAX Founder	Technical Specialist & System Architect
Dr.Ph.D	János Hauszmann	GMAX Aerospace ltd.	Chief Engineer & Technology Developer / GMAX Founder	Chief Engineer & Technology Developer
Dr.	Rafael A. Pizarro	GMAX Aerospace ltd.	Clinical Chief for the Technology / GMAX Co-Founder	Clinical Chief for the Technology
Dr.	Balázs Rokonai	GMAX Aerospace ltd.	Legal Counsel / GMAX Co-Founder	Legal Oversight & Regulatory Counsel

On behalf of the University of Campania “Luigi Vanvitelli” in Naples (University Background)

	Name	Institution	Position Held (Parent Institution)	Role in the Project
Prof.	Domenico Parmeggiani	University of Campania “Luigi Vanvitelli”	Associate Professor	Clinical Chief
Dr.	Francesco Miele	University of Campania “Luigi Vanvitelli”	General Surgery Specialist	Co-investigator
Prof.	Massimo Agresti	University of Campania “Luigi Vanvitelli”	Full Professor	Co-investigator
Dr.	Alessio Cece	University of Campania "Luigi Vanvitelli"	General Surgery Resident	Co-investigator
Dr.	Pasquale Luongo	University of Campania "Luigi Vanvitelli"	General Surgery Resident	Co-investigator
Dr.	Giancarlo Moccia	University of Campania "Luigi Vanvitelli"	General Surgery Specialist	Co-investigator
Dr.	Manuela Miccio	University of Campania "Luigi Vanvitelli"	General Surgery Resident	Co-investigator
Prof.	Pasquale Sperlongano	University of Campania "Luigi Vanvitelli"	Full Professor	Co-investigator
Dr.	Rossella Sperlongano	University of Campania "Luigi Vanvitelli"	Technical scientific officer	Co-investigator
Prof.	Marina Di Domenico	University of Campania "Luigi Vanvitelli"	Full Professor	Co-investigator
Dr.	Paola Della Monica	University of Campania "Luigi Vanvitelli"	Pathology Resident	Co-investigator
Dr.	Federica Colapietra	University of Campania "Luigi Vanvitelli"	Pathology Resident	Co-investigator
Prof.	Michele Caraglia	University of Campania "Luigi Vanvitelli"	Full Professor	Co-investigator
Dr.	Alessia Cossu	University of Campania "Luigi Vanvitelli"	Researcher	Co-investigator

Università degli Studi della Campania *Luigi Vanvitelli*

GMAXO®

Table of Contents

1. Abstract
2. Objective, Research Question and Method
3. Reconstructed Pattern of the LVU Clinical A375 Measurement
   - 3.1. Raw Numerical Series and Its Normalized Description
   - 3.2. What Can We State and What Can We Not?
4. International Scientific Literature Background: What A375 Pattern Emerges?
   - 4.1. Ultrasound and Sonodynamic Approach
   - 4.2. Radiofrequency/Hyperthermia Approach
   - 4.3. ECM, Adhesion, Morphology, Migration
   - 4.4. Mechanical and Biomechanical Plasticity
5. Strict Comparative Matrix
6. Detailed Professional Interpretation
   - 6.1. Where the LVU Clinical A375 Result Fits Well with the International Literature
   - 6.1.1. A375 as a Rapidly Responsive, Plastic System
   - 6.1.2. The Response Is Not Simply a Linear Cell Death Curve
   - 6.1.3. The Relationship Between Adhesion and the Measurable Projection Is Plausible
   - 6.2. Where the Own Result Differs from the Standard A375 Literature
   - 6.2.1. Short Time Window and Narrow Endpoint Set
   - 6.2.2. The Main Signal Here Can Be Described in Terms of Compaction Rather Than Apoptosis
   - 6.2.3. Compared with the International Literature, the Motif of Metastability Is Stronger Here
7. What Did the LVU Clinical Measurement Produce That Could Be Expected in Advance According to HITE?
8. What Is the Distinctive Feature That We Did Not Necessarily Expect?
   - 8.1. Persistently Positive Compaction Regime
   - 8.2. Separation of A375 and CHL-1
   - 8.3. The Early Structural Signal May Precede the Classical Biological Endpoints
9. Limitations and Publication Caution
10. Conclusions of Publication Value
11. References

1. Abstract

The aim of the study is to determine to what extent the short-term response dynamics observed in the LVU00001 A375 melanoma measurement follow, differ from, or extend the A375 state-response patterns known in the international scientific literature. How does the short-term pattern of the uploaded LVU A375 melanoma measurement fit into the A375 studies known in the international scientific literature, particularly studies related to physical interventions – ultrasound, radiofrequency/hyperthermia stimulation –, as well as studies related to the extracellular matrix, adhesion, morphology, and mechanical state?

According to the LVU source, in the A375 cell line, the measurable cell concentration decreases from the initial value of 8.30 x 10^5 cells/mL to 5.85 x 10^5 cells/mL at 15 minutes, and then partially re-organizes to 6.60 x 10^5 cells/mL at 30 minutes. In normalized form, this course is D15 = 0.7048, D30 = 0.7952, while with the HITE compaction potential it is Xi15 = 0.3498, Xi30 = 0.2292, meaning that at 30 minutes the system remains in a projection that is more compact than the initial state.

The international scientific literature presents three robust motifs in the A375 profile: (i) A375 exhibits sensitivity and plastic responsiveness to physical stimulation, with viability, apoptotic, stress, and cell-cycle changes under ultrasound and hyperthermia; (ii) the adhesive and ECM environment of A375 strongly determines its spreading, focal adhesions, migration, and proliferation; and (iii) the cell line is mechanically and morphologically heterogeneous, capable of switching migratory modes, with its biomechanical state closely associated with cytoskeletal and EMT-related markers.

The main conclusion of the comparison is that the LVU A375 measurement directionally fits this international profile in showing an early, rapid, structurally characterized state shift, rather than a simple linear cell-death pattern.

At the same time, it differs from most published A375 studies in that here the leading measurable signal is a short-term cell-number/dispersion projection, rather than a direct annexin/PI, ROS, caspase, HSP, clonogenicity, or invasion endpoint.

The strongest distinctive feature of the measurement is that a persistently positive compaction regime remains present in A375 even at 30 minutes, which, in HITE terminology, raises the possibility of a metastable compact basin. The document provides a detailed comparative matrix, an interpretive audit, and a cautious framework of conclusions from a publication perspective.

2. Objective, Research Question and Method

The present analysis is not simply a literature review, but a fit audit. The central question is to what extent the early pattern visible in the LVU A375 laboratory measurement – a strong decrease at 15 minutes, followed by a partial reorganization at 30 minutes – follows, extends, or differs from the international scientific literature concerning A375.

The audit answers six questions:

1. What precise pattern can be read from the LVU A375 measurement?
2. What types of responses does the international scientific literature report in A375 under physical stimulation?
3. Where are the direct similarities?
4. Where are the methodological or biological differences?

5. What can be regarded as the expected form according to HITE?
6. Does the LVU00001 result contain any feature that goes beyond the standard A375 profile?

The source base consists of two parts:

· internal HITE sources: the uploaded LVU00001 melanoma working document and the HITE validation document;

· external scientific literature: publications on A375 concerning ultrasound, hyperthermia, ECM adhesion, migration, and mechanobiology.

3. Reconstructed Pattern of the LVU00001 Clinical A375 Measurement

3.1. The Raw Numerical Series and Its Normalized Description

The LVU source provides the following short-term cell concentration series for the A375 line:

$$N0 = 8.30 \times 10^5,\ N15 = 5.85 \times 10^5,\ N30 = 6.60 \times 10^5 \text{ sejt/mL.} \quad (1)$$

From this, the normalized projection is:

$$D15 = N15 / N0 = 0.7048,\ D30 = N30 / N0 = 0.7952, \quad (2)$$

and the relative deviation:

$$R15 = -29.52\%,\ R30 = -20.48\%. \quad (3)$$

The HITE-based compaction potential is expressed in the internal material in the following form:

$$Xi(t) = - \ln D(t) \quad (4)$$

from which:

$$Xi15 = 0.3498,\ Xi30 = 0.2292 \quad (5)$$

which yields. The positive sign at both time points indicates that the measurable state remains more compact than the initial projection even at 30 minutes.

LVU00001 A375 pattern in brief: a rapid and strong initial shift, followed by partial relaxation, but not complete return. In HITE terminology, this is a short-term compaction-metastable regime.

3.2. What Can We State and What Can We Not?

The uploaded HITE validation framework explicitly warns that a single channel sensitive to artifacts — for example, cell number or cell density alone — is not sufficient for formulating a strong claim of behavioral change; this requires the consistent co-movement of multiple layers.

Therefore, in its present form, the LVU clinical measurement result can primarily be interpreted as an early structural/projectional signal and cannot be regarded as complete, multi-endpoint biological evidence.

4. International Scientific Literature Background: What A375 Profile Emerges?

4.1. Ultrasound and Sonodynamic Approach

Bernard et al. investigated low-intensity therapeutic ultrasound and cisplatin in the A375 cell line; viability was monitored using an MTT assay, and they found that ultrasound alone produced a measurable effect, while in combined application with cisplatin, the treatment sequence also influenced the strength of the response.

A later A375 study by Rezaei et al. showed that a bystander effect may also occur under therapeutic ultrasound, which may be associated with apoptosis, as well as changes in p53 and HO-1.

These studies suggest that, in the A375 cell line, ultrasound stimulation is not merely a mechanical disturbance, but is capable of initiating measurable biological programs.

4.2. Radiofrequency/Hyperthermia Approach

The hyperthermia literature on A375 is strong and consistent.

Bhayani et al. found that, with radiofrequency, particle-mediated heating, the cellular effect at 43 C can also be reproduced with short RF-induced heating, and that the HSP70/HSP90 response is pronounced.Mantso et al. demonstrated in the A375 cell line that hyperthermia induces apoptosis, oxidative stress, and cell-cycle shifts.

According to Wang et al., inhibition of HSPB1 enhances hyperthermia-induced cell death in melanoma cell lines, including A375.

This line of investigation shows that the A375 cell line exhibits marked stress and fate responses to physical-thermal stimulation.

4.3. ECM, Adhesion, Morphology, Migration

The extracellular matrix (ECM)-dependent behavior of the A375 cell line is extensively documented.

Chung et al. demonstrated that keratinocyte-derived, laminin-332-rich ECM supports the adhesion, spreading, and migration of A375 cells more effectively than the cells' own ECM; in parallel, integrin alpha6 expression, FAK phosphorylation, and focal adhesion formation increase.

Balion et al. found on PEG-CLP-based hydrogels that the proliferation, shape index, and focal adhesion pattern of A375 cells sensitively follow changes in matrix ligands.

According to Mazurkiewicz et al., gelsolin plays a particularly important role in laminin-dependent A375 migration.

A 2024 review of melanoma-ECM summarizes that collagen- and LOX-driven ECM remodeling, matrix stiffness, and integrin signaling pathways are key factors in melanoma progression.

4.4. Mechanical and Biomechanical Plasticity

Lin et al. described the general mechanical phenotype of tumor cells as "cell softening and loss of stiffness sensing", meaning that cancer cells are in many cases softer and less responsive to substrate stiffness than their normal counterparts.

In the case of the A375 cell line, however, the picture is more nuanced.

Makowiecka et al. demonstrated significant biomechanical heterogeneity among A375 clones, and also showed that biomechanical properties do not correlate with a single "stemness" component.

Cykowska et al. found in an A375 YB-1 model that cells expressing YB-1 are significantly stiffer, while the cell line exhibits a high degree of migratory plasticity and is capable of switching between mesenchymal and amoeboid modes of movement.

This is important because the A375 cell line is therefore not simply a "soft melanoma", but rather a mechanically and morphologically multistate system.

5. Strict Comparative Matrix

Detailed Comparison of the LVU00001 Primary A375 Measurement and A375 Patterns in the International Scientific Literature

LVU A375 result	International Scientific Literature A375 Pattern	Similarity	Difference	HITE Interpretation	Unexpected Element
Large, close to 30% negative projectional shift from baseline at 15 minutes.	Under ultrasound and hyperthermia, rapid stress, viability, and apoptotic responses may be initiated in A375.	A375 is a rapidly responding cell line.	In the Italian data, the main signal is a cell-number/dispersion projection; in most publications, viability/apoptosis/HSP endpoints are used.	An early structural or phase-state shift is probable, which is not yet a final cell-fate reading.	The early signal is very strong considering that no direct cell-death endpoint accompanies it.
Partial reorganization at 30 minutes, but the system does not yet return to the baseline state.	The response is often not immediate complete "killing", but rather a stress and fate program that unfolds over time.	Non-monotonic, time-dependent response on both sides.	In the international literature, later time points typically extend to 12–72 hours; here, visibility extends only to 30 minutes.	Compaction → partial relaxation, but with a metastable shift.	The 30-minute residual compactness suggests stronger metastability than is typically described by the standard endpoint literature.
Positive HITE potential at both 15 and 30 minutes: Xi15 = 0.3498, Xi30 = 0.2292.	ECM, adhesion, and biomechanical states can change rapidly; focal adhesions, spreading, and migration change.	State-structural plasticity is consistent.	Publications generally describe these phenomena in terms of adhesion/migration or AFM/IF, rather than compaction potential.	The measured signal may correspond to a transient adhesion/clustering state.	The persistence of the positive signal raises the possibility of a deeper compact state basin in the short term.
Short-term projectional character: cell number/dispersion.	Use of multimodal endpoints: apoptosis, ROS, HSP, and cell-cycle response.	Methodological caution is warranted on both sides.	Apoptosis, necrosis, or mitochondrial damage do not directly follow from the own measurement.	The result is an early indicator, not a closed biological judgment.	The magnitude of the signal gives it strong hypothesis-generating value.
Coherent, unidirectional, then partially dissolving response over a short period.	ECM-dependent rapid reorganization, migratory and adhesion shifts.	Rapid restructuring is plausible.	The international literature measures specific molecular and morphological markers; the Italian measurement does not.	Rapid adhesion/aggregation shift is probable.	The signal appears in a clear two-step form purely in the cell-number series.
Stronger compaction signal in A375 than in CHL-1.	Cell-line-specific mechanical and migratory behavior is known.	Cell-line-specific response is plausible.	Direct A375 vs. CHL-1 comparison is less common in the standard literature.	Different state-space basins are activated.	The cell-line difference is stronger than suggested by the standard descriptions.
No direct ROS/HSP/apoptosis marker.	These endpoints are measured as standard.	Physical stimulation is relevant on both sides.	The direct mechanism has not been verified.	The early projection should subsequently be completed with functional endpoints.	The own data capture the structural level "earlier" than the classical endpoints.

6. Detailed Professional Interpretation

6.1. Where the LVU A375 Result Fits Well with the International Scientific Literature

6.1.1. A375 as a Rapidly Responding, Plastic System

One of the strongest common elements of the international A375 profile is plasticity.

This is manifested in the stress response to physical stimulation, ECM-dependent spreading and migration, as well as the ability to switch between mechanical and migratory states.

The LVU short-term data show the same plasticity in their own terms: the system does not remain in the initial projection, but rapidly enters a new structural regime.

6.1.2. The Response Is Not Simply a Linear Cell Death Curve

The international A375 publications on physical interventions indicate that the response cannot be interpreted as a single monotonous “alive vs. dead” variable.

Under hyperthermia, ROS response, apoptosis, cell-cycle shifts, and HSP activation develop together. In the case of ultrasound, even bystander-mediated phenomena may occur.

The LVU clinical measurement does not show a linear disappearance curve, but rather a decrease + partial reorganization pattern. This is much more indicative of a multiphase response dynamic.

6.1.3. The Relationship Between Adhesion and the Measurable Projection Is Plausible

The adhesion, ECM, and focal adhesion systems of the A375 cell line are well documented as active and dynamic.

This means that the cells may be capable of changes in spreading, aggregation, clustering, or modification of their mode of attachment to the substrate over a short period.

Therefore, the “fewer countable cells” signal visible in the own measurement is fully compatible with a structural reorganization that the classical literature describes in terms of adhesion, cytoskeletal, or migratory

6.2. Where the Own Result Differs from the Standard A375 Literature

6.2.1. Short Time Window and Narrow Endpoint Set

The greatest difference is methodological.

Most published A375 ultrasound or hyperthermia studies use 12, 24, 48, or 72-hour follow-up and also measure direct biological endpoints.

In contrast, the LVU measurement result is based on 0, 15, and 30-minute time points and primarily provides a cell-number/dispersion projection.

This should not be interpreted as a weakness, but as a different layer: the internal data carry early state-structural information, but on their own do not close the cell-fate question.

6.2.2. The Main Signal Here Can Be Described in Terms of Compaction Rather Than Apoptosis

The standard A375 literature largely speaks in terms of apoptosis, ROS, HSP, cell cycle, migration, or invasion.

The own LVU result, however, can most clearly be described in terms of compaction/redistribution.

This is not a contradiction, but rather a difference in perspective. The own measurement captures the trajectory earlier than where most publications measure an endpoint.

6.2.3. Compared with the International Literature, the Motif of Metastability Is Stronger Here

$$Xi30 > 0$$

The fact indicates that the system does not return to the baseline state even at 30 minutes.

This supports the idea of a metastable compact basin.

In the international A375 publications, plasticity and state transitions are clear, but such a clear, early, two-point compaction-metastable reading is less common. This is where the own measurement material is distinctive

7. What Did the Measurement at LVU Produce That Could Be Expected in Advance According to HITE?

According to the uploaded HITE validation document, the primary effect of the technical intervention is not necessarily direct cell death, but rather a trajectory shift between structural, phase, adhesion, and functional layers.

The LVU A375 data support precisely this logic:

1. the response is rapid,
2. the first measured signal is strong, but is not a final cell-fate signal,
3. the system partially relaxes, but does not return,
4. the overall form therefore fits a trajectory shift better than a simple decrease in cell number.

In HITE terms, this is close to the following scheme:

external excitation → internal phase/structural reorganization → projectional cell-number decrease → partial relaxation. (6)

8. What Is the Distinctive Feature That We Did Not Necessarily Expect?

8.1. Persistently Positive Compaction Regime

The most interesting feature is that in A375, the compaction potential remains positive at 30 minutes.

This means that the system did not merely “twitch” and then return to its previous state, but temporarily entered a new local structural basin.

Most standard A375 publications do not use this conceptual language, but rather descriptions in terms of stress, apoptosis, or migration. The own data therefore provide an early, distinctive HITE interpretation.

8.2. Separation of A375 and CHL-1

According to the LVU source, CHL-1 has already shifted toward a hyperdispersive direction at 30 minutes, whereas A375 remains compact.

This indicates a strong cell-line-specific state-space difference.

One of the strongest points of the own system is precisely this: it is not simply that “melanoma behaves this way”; A375 behaves this way under this stimulation, while CHL-1 behaves differently.

This is also valuable from a publication perspective, because it offers a differential state-space reading.

8.3. The Early Structural Signal May Precede the Classical Biological Endpoints

The LVU primary measurements may also imply that, with certain physical interventions, a shift in the structural projection may become visible earlier than classical annexin/PI, caspase, HSP, or clonogenicity endpoints.

If this can be confirmed by subsequent measurements, then this would be of interest not only from a HITE perspective, but also as a general methodological finding.

9. Limitations and Publication Caution

In the current state, the following statements can be maintained with professional safety:

· the LVU A375 measurement is not a trivial noise pattern, but a strong, time-dependent early shift,

· this pattern is consistent with the internationally known plasticity of A375 and its sensitivity to physical stimulation,

· the own data do not yet methodologically establish what proportions of the short-term projection are attributable specifically to aggregation, adhesion reorganization, membrane response, mitochondrial stress, or actual cell death,

· the strongest publication form at present is to position the own result as an early HITE signal, rather than as a completely established biological mechanism.

10. Publication-Value Conclusions

Overall professional assessment:

The principal pattern of the LVU clinical A375 melanoma measurement — a marked 15-minute projectional decrease followed by a partial, but not complete, 30-minute reorganization — shows good agreement with the A375 plasticity, sensitivity to physical stimulation, and capacity for adhesion/mechanobiological state transitions known from the international literature.

The most important difference is that the own data currently represent an early cell-number/dispersion projection, whereas the standard literature predominantly works with later, multimodal biological endpoints.

The most interesting distinctive feature is that in A375, the short-term compaction regime remains positive at 30 minutes, which, in HITE terms, raises the possibility of a metastable compact state basin.

This is one of the strongest, non-trivial contributions of the own measurement.

Based on the present monograph, the most defensible publication narrative is as follows:

The LVU measurement shows an early, structurally characterized A375 response. This is compatible with the international literature on A375 ultrasound/hyperthermia/ECM-mechanobiology. The distinctive feature of the signal is its short-term compaction–metastable form. The next validation step is multi-endpoint closure: membrane permeability, mitochondrial potential, annexin/PI, morphological clustering, Raman or impedance, as well as later proliferation/clonogenicity time points.

11. References

[1] Hauszmann internal source: hite_melanoma_italiano.tex. Uploaded working document from the HITE_kernel package, April 8, 2026. Internal A375/CHL-1 melanoma comparison, with 0/15/30-minute measurement series and HITE compaction indicators.

[2] Hauszmann internal source: HITE_daganatos_sejt_publikacios_valtozat.tex. Uploaded working document, April 11, 2026. HITE validation operator and multilayer interpretation framework for the characterization of behavioral changes in tumor cells following technical intervention.

[3] Hauszmann internal source: HITE_melanoma_index.txt. Uploaded project index, 2026. Logical chains and source list of the HITE melanoma branch.

[4] Bernard V, Mornstein V, Skorpikova J, Jaros J. Ultrasound and cisplatin combined treatment of human melanoma cells A375 - the study of sonodynamic therapy. Ultrasound Med Biol. 2012;38(7):1205-1211. doi:10.1016/j.ultrasmedbio.2012.02.006.

[5] Rezaei M, Samani FS, Aghaie A, et al. Bystander effect of therapeutic ultrasound in the presence of cisplatin on human melanoma cell line A375. J Biomed Phys Eng. 2023;13(5):503-514.

[6] Bhayani KR, Kale SN, Jagannathan NR. In vitro evaluation of heat shock protein response in A375 melanoma cells under radio-frequency induced Dex-LSMO mediated heating. Int J Hyperthermia. 2013;29(5):404-414. doi:10.3109/02656736.2013.807438.

[7] Mantso T, Vasileiadis S, Anestopoulos I, et al. Hyperthermia induces therapeutic effectiveness and potentiates adjuvant therapy with non-targeted and targeted drugs in an in vitro model of human malignant melanoma. PLoS One. 2018;13(8):e0201258.

[8] Wang HX, Yang X, Guo L, et al. HSPB1 deficiency sensitizes melanoma cells to hyperthermia induced cell death. Oncotarget. 2016;7(41):67449-67463. doi:10.18632/oncotarget.11894.

[9] Chung H, Suh EK, Han IO, Oh ES. Keratinocyte-derived laminin-332 promotes adhesion and migration in melanocytes and melanoma. J Biol Chem. 2011;286(15):13438-13447. doi:10.1074/jbc.M110.166751.

[10] Balion Z, Svirskis D, Vailionis A, et al. Investigation of cancer cell migration and proliferation on biomimetic extracellular matrix peptide hydrogels. Front Bioeng Biotechnol. 2020;8:773. doi:10.3389/fbioe.2020.00773.

[11] Mazurkiewicz E, Simiczyjew A, Dratkiewicz E, et al. Gelsolin contributes to the motility of A375 melanoma cells and this activity is mediated by the fibrous extracellular matrix protein laminin. Cells. 2021;10(8):1848. doi:10.3390/cells10081848.

[12] Lin HH, Lin HK, Lin IH, et al. Mechanical phenotype of cancer cells: cell softening and loss of stiffness sensing. Oncotarget. 2015;6(25):20946-20958. doi:10.18632/oncotarget.4173.

[13] Makowiecka A, Simiczyjew A, Nowak D, Mazur AJ. Changes in biomechanical properties of A375 cells due to the reduction of thymosin beta4 expression. Cells. 2021;10(4):769. doi:10.3390/cells10040769.

[14] Cykowska A, Ostrowska-Podhorodecka Z, Marciniak L, et al. Biomechanical and biochemical assessment of YB-1 expression in A375 melanoma cell line: exploratory study. Front Mol Med. 2023;3:1050487. doi:10.3389/fmmed.2023.1050487.

[15] Mayasin YP, Wangpraseurt D, et al. Extracellular matrix as a target in melanoma therapy. Int J Mol Sci. 2024;25(22):11869.

Supplementary Explanation for General Interpretation:

A375 and CHL-1 are human melanoma cell lines, that is, human cells derived from skin cancer that are maintained and studied under laboratory conditions. They do not represent the current condition of a specific patient, but rather standardized experimental models that allow different effects to be analyzed in a comparable manner.

The purpose of laboratory cell studies is to observe what changes a given effect induces in the behavior of cells – for example, in their growth, activity, or structural state. It is important that these processes take place in an isolated environment and are therefore not identical to the complex biological processes occurring in the human body.

The A375 and CHL-1 cell lines have different properties and may therefore respond differently to the same effect. The combined use of these two models makes it possible to distinguish whether an observed phenomenon is general in nature or rather cell-type-dependent.

The results presented in the present material originate from laboratory measurements and primarily serve to describe observations. These data alone cannot be regarded as clinical evidence and are not suitable for directly establishing medical conclusions or treatment decisions.

Simplified interpretive picture:

In a simplified interpretation, the LVU00001 measurement does not show that the cells "disappear" within a short period of time, but rather that they rapidly reorganize into another, temporary state. Following the initial decrease, partial reorganization can be observed; however, the system does not fully return to the baseline state. This indicates that the functioning of the cells temporarily shifts into a different form of organization. The difference between A375 and CHL-1 indicates that this response may differ by cell type: what results in a more compact, stable state in one case shifts more toward a disintegrating, dispersive direction in the other. The observed phenomenon is thus a picture of an early structural reorganization, rather than an immediate, irreversible process of cell death.