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Original article

Comparison of autophagic activity and flux in breast cancer cell lines: MDA-MB-231 and MCF-7 under stress condition

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Abstract

Autophagy is a fundamental cellular process responsible for recycling cytoplasmic components, playing a crucial for maintaining cellular homeostasis and influencing cancer progression. Breast cancer comprises various molecular subtypes with different levels of aggressiveness and therapeutic responses.

This study aimed to investigate differences in autophagic activity and flux between two breast cancer cell lines-MDA-MB-231, a triple-negative aggressive subtype, and MCF-7, a luminal hormone-sensitive subtype-compared to non-tumorigenic MCF-10A breast epithelial cells. We also evaluated the effects of pharmacological modulation on autophagy to understand how autophagic status may influence cancer cell behavior.

Methods. We conducted an *in vitro* study with MDA-MB-231, MCF-7, and MCF-10A cells. Autophagic activity was measured by analyzing LC3B and p62 protein levels through western blotting and immunofluorescence microscopy. Pharmacological modulation included nutrient deprivation to induce autophagy, 3-methyladenine to inhibit initiation, and Bafilomycin A1 to block degradation. Data were analyzed descriptively and comparatively to highlight differences in marker expression.

Results. Both cancer cell lines showed higher basal autophagy than MCF-10A. MCF-7 cells accumulated p62 and LC3B-II, suggesting impaired degradation. MDA-MB-231 cells showed reduced p62 and increased LC3B-II, indicating active flux. Upon pharmacological treatment, MDA-MB-231 cells responded with expected marker

changes, confirming functional autophagy. In contrast, MCF-7 cells showed further accumulation of LC3B-II and p62, reinforcing impaired degradation.
Conclusion. This study highlights distinct differences in autophagy between breast cancer subtypes. MDA-MB-231 cells maintain active autophagy, potentially supporting their aggressive behavior and adaptability. MCF-7 cells exhibit a block in degradation, which may limit their response to metabolic stress and therapy. These insights underscore the importance of considering autophagy status in breast cancer treatment strategies.

Keywords: autophagy, autophagic flux, breast cancer, triple-negative breast cancer.

1. Introduction

Autophagy is a lysosome-mediated intracellular degradation pathway that enables the recycling of dysfunctional or unnecessary cellular components, including proteins, lipids, and organelles. This evolutionarily conserved process plays a vital role in maintaining cellular homeostasis and facilitating adaptation to environmental stress. It begins with the formation of an autophagosome—a double-membraned vesicle that engulfs cytoplasmic targets—and ends with fusion to the lysosome for degradation. Dysregulation of autophagy is implicated in various diseases, including neurodegeneration, infections, and cancer [1-5].

In cancer biology, autophagy exhibits a dual and context-dependent role. During early tumorigenesis, it acts as a tumor suppressor by clearing damaged proteins and organelles, thereby maintaining genomic stability and homeostasis. However, in established tumors, autophagy supports tumor progression by promoting survival under metabolic and therapeutic stress, contributing to drug resistance and metastasis [6-11]. Thus, autophagy represents a promising yet complex therapeutic target in oncology. Among its molecular markers, LC3B is associated with autophagosome formation, whereas p62/SQSTM1 accumulates when autophagic degradation is impaired, serving as an autophagy receptor.

Breast cancer remaining the leading oncological disease among women worldwide, including in Kazakhstan. In Kazakhstan, it ranks first among cancer types, with its prevalence continuing to rise [12-13]. Efforts such as expanded access to early screening and improvements in diagnostic and treatment methods have achieved certain successes [12,14]. Breast cancer is a highly heterogeneous disease, classified into distinct subtypes based on receptor status: estrogen receptor (ER), progesterone receptor (PR), and human epidermal growth factor receptor 2 (HER2). Triple-negative breast cancer (TNBC), which lacks all three receptors, accounts for approximately 15% of cases and is particularly aggressive and resistant to hormonal therapies. Despite progress in screening and the development of targeted therapies—such as endocrine therapy, aromatase inhibitors, HER2-targeted antibodies, and CDK4/6 inhibitors—TNBC continues to present significant clinical challenges, with metastatic relapse observed in up to 40% of patients [5,15].

Recent studies suggest that autophagy contributes to breast cancer progression and therapy resistance, with regulatory patterns varying among subtypes [7]. While autophagy inhibition may sensitize some tumors to chemotherapy, it may conversely promote survival in more aggressive cancers. Therefore, a more nuanced understanding of autophagy regulation across breast cancer subtypes is essential for optimizing therapeutic strategies.

The present study aims to evaluate and compare the autophagic profiles of two breast cancer cell lines, MDA-MB-231 (TNBC) and MCF-7 (luminal A), under basal conditions and upon autophagy modulation. To this end, we applied nutrient deprivation (starvation),

early-stage autophagy inhibition with 3-methyladenine, and late-stage inhibition with bafilomycin A1. Western blot analysis of LC3B and p62 was employed to assess autophagic activity and flux. As a physiological control, MCF-10A, a non-tumorigenic mammary epithelial cell line, was included to evaluate basal level of autophagic activity without stress stimuli.

These findings aim to enhance our understanding of the dynamic behavior of autophagy in different breast cancer subtypes and may help identify potential vulnerabilities to autophagy-targeted therapies.

2. Materials and Methods

Cell culture

Human breast cancer cell lines such as MDA MB-231 and MCF-7 were obtained from the NUSOM cell repository. Characteristics of the cell lines are summarized in Table 1. Cells were cultured in Dulbecco's Modified Eagle Medium (DMEM, 11574486, Gibco, USA) containing 4.5g/mL glucose and supplemented with 10% fetal bovine serum (FBS, F9665, Sigma-Aldrich, USA), 1mM sodium pyruvate, 1% of penicillin-streptomycin antibiotic mixture and 4 mM L-glutamine. Culture were maintained as monolayers in a tissue culture incubator at 37°C in a humidified 5% CO_2 atmosphere and passaged every 1-2 days to sustain exponential growth. All cell lines were routinely tested and confirmed to be free of mycoplasma contamination.

Table 1 - Characteristics of cell lines used in the study

Cell line	Receptor Status	Subtype	Notable characteristics
MCF-7	ER+, PR+, HER2−	Luminal A	Hormone-responsive model, slow growing
MDA-MB-231	ER−, PR−, HER2−	Basal-like (TNBC)	Highly invasive, drug resistance model

Treatment options

To induce autophagy via nutrient depriviation, cells were washed twice with phosphate-buffered saline (PBS; Biological Industries, 02–023-1A) and incubated in Earle's Balanced Salt Solution (EBSS; Biological Industries, 02–011-1A). To inhibit early-stage autophagy initiation, 10 mM 3-methyladenine (3-MA) was used for 4 h. To block late-stage autophagy, 100 nM bafilomycin A1 (LC Laboratories, B-1080) was applied for 4 h to prevent autophagosome-lysosome fusion and lysosomal acidification. These pharmacological modulators, along with the starvation condition, were used to assess autophagic flux, as illustrated in Figure 1.

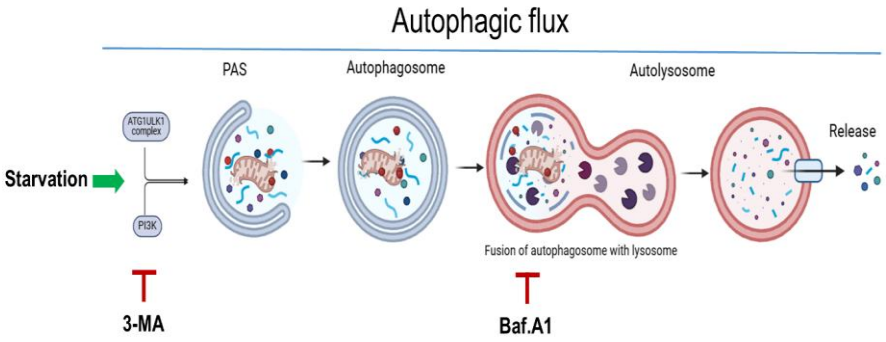

Figure 1 - Modulation of autophagic flux in MCF-7 and MDA-MB-231 cells

Note: Schematic representation of autophagy regulation under experimental conditions. Starvation (EBSS) induces autophagy by nutrient depletion. 3-MA inhibits autophagy at the early stage by blocking class III PI3K activity and autophagosome formation. Baf. A1 impairs late-stage autophagy by preventing autophagosome-lysosome fusion and lysosomal acidification. This experimental setup allows assessment of autophagic flux by comparing LC3-II and p62 levels in the presence or absence of these modulators.

Fluorescence imaging of live-cell staining

Cells were seeded in 12-well plates at a density of 1.5×10^5 cells/well and allowed to adhere for 24 h. For visualization of active mitochondria, cells were incubated with 50 nM TMRE (Tetramethylrhodamine ethyl ester) for 20 min at 37°C and then rinsed with PBS. Lysosomes were stained using 50 nM LysoTracker Deep Red DND-99 for 30 min at 37°C. Nuclei were counterstained with 1 μg/mL Hoechst in PBS for 5 min at RT, and excess dye was removed with two PBS washes. Fluorescence imaging was performed using the EVOS FL Auto Imaging System with a 20× objective. Excitation/emission settings were: TMRE with 549/573 nm (RFP filter set), LysoTracker Deep Red with 647/668 nm (RFP filter set), and Hoechst with 358/461 nm (DAPI filter set).

Western blotting

Expression of autophagy-related proteins LC3B and p62 was assessed via immunoblotting in accordance with [16-17]. Cells were lysed in RIPA buffer [0.1 M NaCl, 5 mM EDTA, 0.1 M sodium phosphate (pH 7.5), 1% Triton X-100, 0.5% sodium deoxycholate, 0.1% SDS] with protease inhibitor cocktail. Lysates were centrifuged at 13,400 × g for 15 min at 4°C, and protein concentrations were measured using the BCA Protein Assay Kit. Equal amounts of protein (20 μg/lane) were resolved by 12% SDS–PAGE and transferred to PVDF membranes. The membranes were blocked for 1 h at room temperature (RT) in Tris-buffered saline with 0.01% Tween 20 (TBS-T) with 5% nonfat dry milk, then incubated overnight with primary antibody (anti-LC3B 1:1000, or anti-p62 1:5000, GAPDH 1:5000) in PBS. After TBST washes, membranes were incubated with horseradish peroxidase-conjugated secondary antibodies (anti-rabbit or anti-mouse) for 1 h at RT. Blots were developed using ECL substrate (1–5 min exposure) and visualized on a ChemiDoc imaging system then analyzed with Image Lab software.

Statistical Analysis

All data are presented as mean ± standard deviation (SD) from at least three separate experiments. Analyses were performed using Microsoft Excel® (2016). Statistical significance was determined using Student's t-test. Significance thresholds were set as follows: $p < 0.05$ (*), $p < 0.01$ (**), $p < 0.001$ (***), and $p < 0.0001$ (****); NS indicates non-significant differences.

3. Results

Morphological differences between cell lines

The morphological characteristics of MDA-MB-231 and MCF-7 cells were assessed by differential interference contrast (DIC) microscopy. As shown in Figure 2 & 3 (DIC panels), MCF-7 cells display a cobblestone or polygonal shape typical of epithelial cells, while MDA-MB-231 cells exhibit an elongated, spindle-shaped morphology with a mesenchymal-like appearance. Images captured at both low (4×) and higher (20×) magnifications demonstrate the difference in cell shape and size between the two lines. The observed morphological differences correspond to distinct cell phenotypes and physical properties.

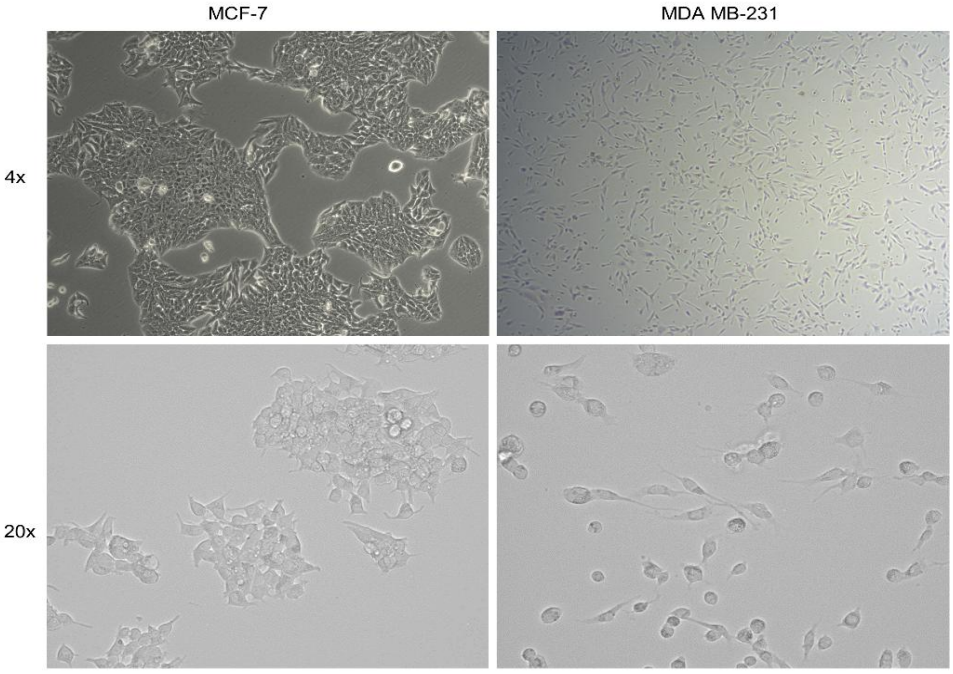

Figure 2 - Differential interference contrast (DIC) images of MCF-7 and MDA MB-231 cells. Upper and bottom panels show 4x and 20x objectives magnifications, respectively

Live-cell imaging with TMRE and Hoechst was used to assess mitochondrial membrane potential and nuclear integrity. As shown in Figure 3, TMRE staining revealed active mitochondria, appearing as red fluorescent networks throughout the cytoplasm in both cell lines. Hoechst staining showed intact, round nuclei without signs of fragmentation or condensation in either cell type. The mitochondrial network appeared more elongated shape and scattered arrangement in MDA-MB-231 cells compared to the more compact arrangement in MCF-7 cells (Figure 3).

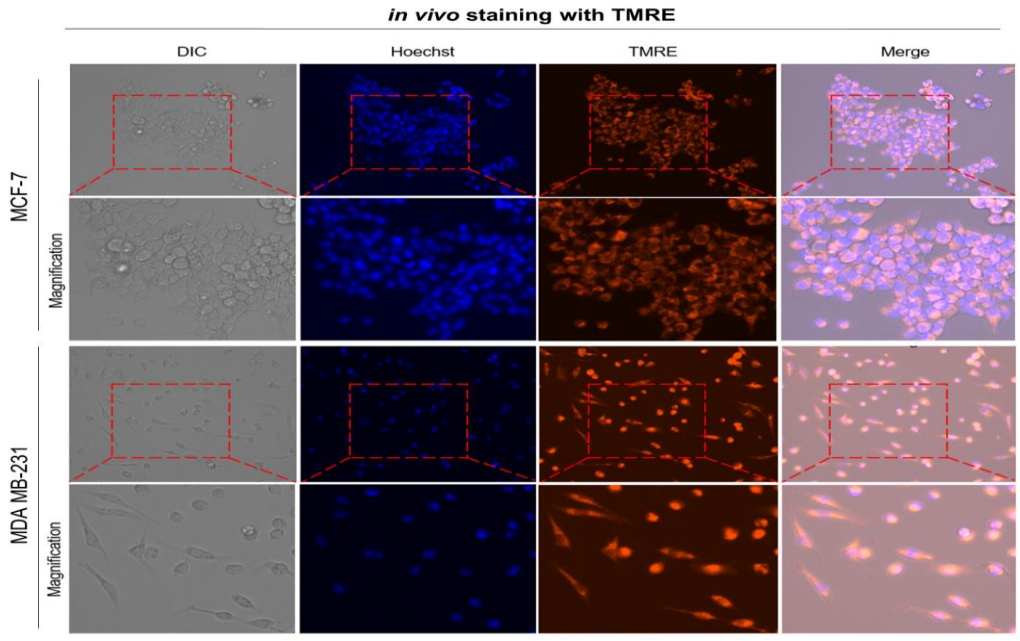

Figure 3 - Fluorescence micrographs of in vivo stained cells where mitochondria were stained with TMRE (red), and nuclei with Hoechst (blue). Upper and bottom panels show MCF-7 and MDA MB-231 cells with its magnification, as respectively

Fluorescence imaging of live MCF-7 and MDA-MB-231 cells stained with LysoTracker Deep Red DND-99 and Hoechst is presented in Figure 4. Acidic compartments predominantly lysosomal staining (red) is localized predominantly in the perinuclear region in both cell types, consistent with active lysosomal populations. MCF-7 cells formed compact colonies with tight cell-cell contact, while MDA-MB-231 cells displayed scattered and loosely associated cell clusters. This difference in colony formation reflects the inherent differences in cellular adhesion properties and organization.

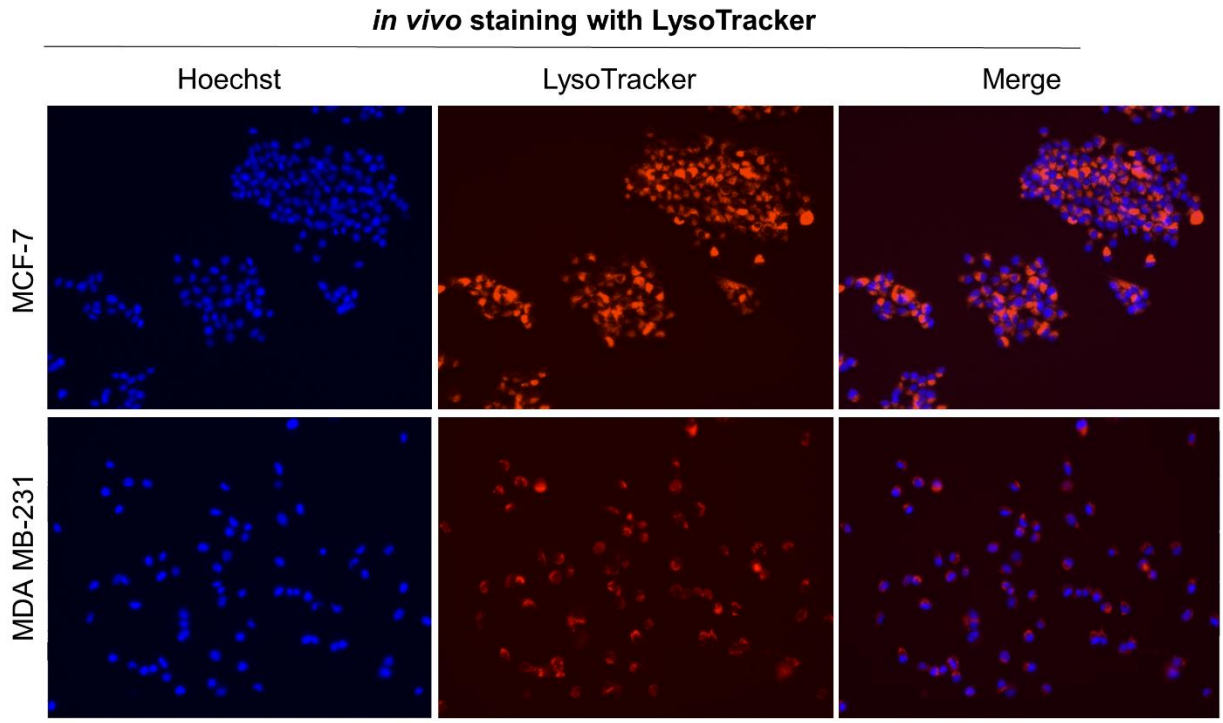

Figure 4 - Fluorescence images of in vivo stained MCF-7 cells where lysosomes were stained with LysoTracker Deep Red DND-99 (red), and nuclei with Hoechst (blue). Upper and bottom panels show MCF-7 and MDA MB-231 cells, as respectively

Basal autophagic activity in cancer vs. non-cancer cell lines

To assess the basal autophagy levels under standard culture conditions, the non-tumorigenic MCF-10A human mammary epithelial cell line was used as a control alongside breast cancer cell lines MCF-7 and MDA-MB-231. As shown in Figure 5A, differences in the expression of autophagy-related markers LC3B-II and p62 were observed among the three cell lines. Visually, p62 levels appeared slightly elevated in MCF-7 and reduced in MDA-MB-231, compared to MCF-10A. LC3B-II levels were the lowest in MCF-10A among the three cell lines, while MCF-7 cells showed the highest levels, and MDA-MB-231 cells displayed intermediate levels. Quantification of protein levels normalized to actin is presented in Figures 5b and 5c. MCF-7 cells expressed p62 at approximately 1.3 times higher than the non-tumorigenic control MCF-10A, while MDA-MB-231 cells showed a twofold decrease relative to MCF-10A (Figure 5b). LC3B-II levels were over fivefold higher in MCF-7 cells and approximately twofold higher in MDA-MB-231 cells compared to MCF-10A (Figure 5c).

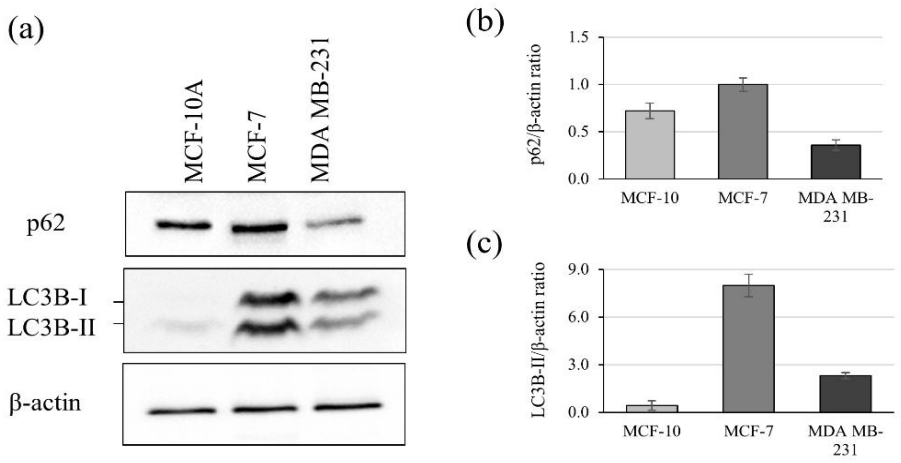

Figure 5 - Basal autophagic activity in MCF-10A (non-cancer), MCF-7, and MDA-MB-231 cells under normal growth conditions. (a) Level of autophagy-related markers LC3-II, p62/SQSTM1 as assessed by western blotting; (b) Quantification of p62 levels normalized to actin; (c) quantification of LC3B-II levels normalized to actin

Autophagic modulation and flux analysis in MDA-MB-231 and MCF-7 cells

To assess dynamic autophagic flux, MDA-MB-231 and MCF-7 cells were subjected to autophagy modulation using nutrient deprivation (Strv), Bafilomycin A1 (Baf.A), and 3-methyladenine (3-MA), with or without lysosomal inhibition (Baf.A). The protein levels of autophagy markers LC3B and p62 were examined under six conditions: Control (Con), Con + Baf.A, Starvation (Str), Str + Baf.A, 3-MA, and 3-MA + Baf.A. As shown in Figure 6a, western blot analysis of MDA-MB-231 cells demonstrates modulation-dependent changes in LC3B-II and p62 levels. Immunoblotting shows that both p62 and LC3B-II levels increase in all conditions involving Baf.A, consistent with lysosomal inhibition leading to accumulation of autophagy substrates. Starvation led to a noticeable decrease in p62 compared to control, while co-treatment with Baf.A reversed this reduction. Treatment with 3-MA alone resulted in an accumulation of LC3B-I levels, indicating inhibition at an earlier stage of autophagosome formation.

As shown in Figure 6b, western blot analysis of MCF-7 cells indicates similar trends. Baf.A treatment resulted in increased levels of both p62 and LC3B-II, reflecting blocked autophagic degradation. Starvation led to a reduction in p62 and LC3B-I levels, with restoration upon Baf.A co-treatment. Interestingly, 3-MA alone led to an accumulation of both p62 and LC3B-II, possibly due to impaired autophagosome formation and disrupted autophagic flux.

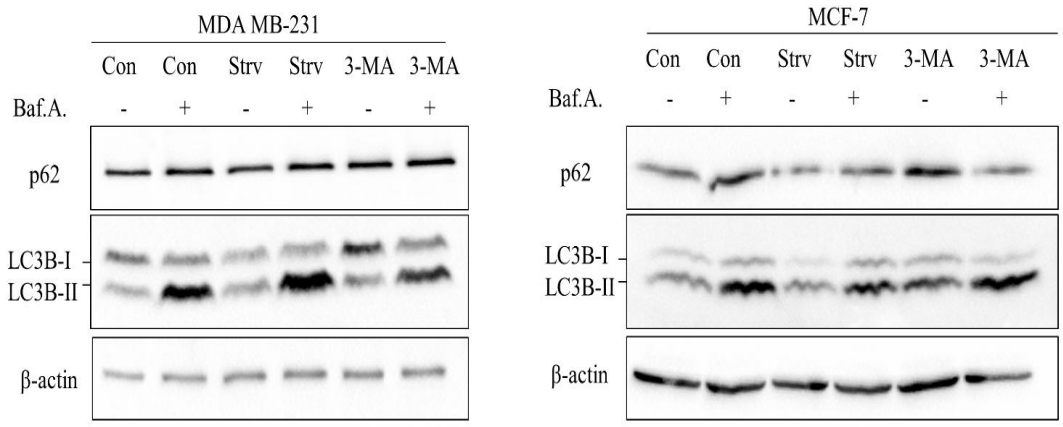

Figure 6 - Response to autophagic modulation in MDA MB-231 and MCF-7 cells. (a) Western blot showing LC3B and p62 protein levels in MDA-MB-231 cells and (b) in MCF-7 cells under six treatment conditions: Control (Con), Con + Baf.A, Starvation (Strv), Str + Baf.A, 3-MA, and 3-MA + Baf.A

4. Discussion

Our study provides insight into the differential autophagic profiles of non-cancerous and cancerous breast epithelial cell lines under basal and modulated conditions. The observed morphological differences between MCF-7 and MDA-MB-231 cells support their known biological behaviors—MCF-7 being more epithelial and indolent, and MDA-MB-231 exhibiting mesenchymal traits aligned with aggressive, metastatic potential.

We found that both MCF-7 and MDA-MB-231 cells exhibited elevated basal autophagic markers compared to non-tumorigenic MCF-10A cells. This is consistent with previous findings suggesting that cancer cells often upregulate autophagy as an adaptive mechanism to maintain cellular homeostasis and survival under stress [18-19]. It is important to distinguish between autophagic activity and autophagic flux. While autophagic activity reflects the initiation and extent of autophagosome formation, it does not necessarily indicate whether degradation is completed. For instance, elevated LC3B-II levels suggest increased autophagosome presence but may also result from impaired degradation. In contrast, autophagic flux encompasses the entire process from autophagosome formation to cargo degradation, providing a more accurate measure of autophagy efficiency [16, 20].

Among the markers analyzed, p62/SQSTM1 and LC3B-II provided key insights into autophagic flux. p62 is a multifunctional scaffold protein that plays a central role in selective autophagy by recognizing ubiquitinated cargo and linking it to the autophagosome through its LC3-interacting region (LIR). Once sequestered, p62 and its bound cargo are degraded in lysosomes [21-22]. Thus, high levels of p62 indicate impaired autophagic degradation, while low levels suggest efficient autophagic clearance.

In this context, the accumulation of p62 in MCF-7 cells, coupled with high LC3B-II levels, suggests a blockage at the autophagosome degradation stage—indicative of defective autophagic flux. This aligns with previous reports of impaired autophagy in certain luminal breast cancer subtypes [23-24]. In contrast, the decline in p62 and elevated LC3B-II in MDA-MB-231 cells point toward a more efficient and active autophagic process, which may support the metabolic flexibility and aggressiveness of this triple-negative cell line [25-26]. An increase in LC3B-II levels is suggestive of an increase in autophagosomes and an upregulated autophagic process.

However, it is important to note that LC3B-II levels

can sometimes appear low even when autophagy is elevated, due to rapid turnover of autophagosomes [27].

Autophagic modulation further confirmed these differential flux patterns. Bafilomycin A1 (Baf.A), a vacuolar H^+-ATPase inhibitor, prevents acidification of lysosomes and blocks autophagosome–lysosome fusion, thereby inhibiting the degradation of autophagic cargo. It is commonly used to distinguish between increased autophagosome formation and impaired autophagic degradation [28-29]. In our experiments, Bafilomycin A1 treatment led to an accumulation of p62 and LC3B-II in both cell lines, validating that autophagy is dynamically regulated and that lysosomal fusion was successfully inhibited. Notably, MDA-MB-231 cells showed robust increases in LC3B-II and p62 upon Baf.A treatment, confirming an intact autophagic flux and efficient autophagosome formation. Starvation led to a reduction in p62 levels, indicative of increased autophagic activity, and LC3B-II levels declined as well—likely due to enhanced degradation rather than reduced autophagosome synthesis [16]. Unexpectedly, 3-MA treatment caused accumulation of p62 and LC3B-II in MCF-7 cells, raising questions about its efficacy as an early-stage autophagy inhibitor in this context. While 3-MA is known to inhibit PI3K activity, its effectiveness can vary depending on experimental conditions, treatment duration, or reagent stability [10, 30]. In MDA-MB-231 cells, 3-MA led to only a moderate reduction in LC3B-II, suggesting partial inhibition of autophagosome formation.

Taken together, our findings support the notion that basal autophagy and autophagic flux differ significantly between MCF-7 and MDA-MB-231 cells. These differences may underlie their distinct biological behaviors and therapeutic responses. Efficient autophagy in MDA-MB-231 cells may provide survival advantages under metabolic stress, while impaired autophagy in MCF-7 cells could contribute to altered cell fate decisions or therapy resistance.

5. Conclusions

Our results demonstrate that triple-negative MDA-MB-231 cells exhibit higher autophagic flux compared to luminal MCF-7 cells, which show signs of autophagic blockage. These differences could be exploited for subtype-specific therapeutic strategies targeting the autophagic pathway.

Conflicts of Interest. The authors declare that they have no conflict of interest.

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Author Contributions. K.K. – concept and supervision of the work; E.A. – conducting the experiments; E.A. and K.K. – discussion of the research results and writing the text; K.K. – editing the text of the article. All authors have read and agreed to the published version of the manuscript.

Compliance with ethical standards. This article does not contain a description of studies performed by the authors involving people or using animals as objects.

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Аутофагиялық белсенділік пен ағыстың сүт безі қатерлі ісігінің жасушалық желілерінде салыстырмалы талдауы: MDA-MB-231 және MCF-7 стресс жағдайында

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Түйіндеме

Аутофагия - бұл жасушалық гомеостазды сақтауға және қатерлі ісіктің даму барысына әсер ететін маңызды жасушалық қайта өңдеу процесі. Сүт безі қатерлі ісігі әртүрлі агрессивтілік пен емдеуге жауап беретін молекулалық түрлерден тұрады.

Бұл зерттеудің мақсаты - MDA-MB-231 (үштік теріс агрессивті түр) және MCF-7 (люминальды гормонға тәуелді түр) сүт безі қатерлі ісігінің екі жасушалық желісіндегі аутофагиялық белсенділік пен ағынды салыстыру және оларды ісік емес MCF-10A жасушаларымен салыстыру. Сонымен қатар, фармакологиялық модуляцияның

аутофагияға әсерін зерттеп, аутофагиялық күйдің ісік жасушаларының мінез-құлқына қалай әсер ететінін анықтадық.

Әдістер. Бұл *in vitro* зерттеу MDA-MB-231, MCF-7 және MCF-10A жасушаларында жүргізілді. Аутофагиялық белсенділік LC3B және p62 ақуыздарының деңгейлерін вестерн-блотинг және иммунды-флуоресценциялық микроскопия арқылы бағаланды. Фармакологиялық модуляция ашығуды (аутофагияны индукциялау), 3-метиладенинді (инициацияны тежеу) және Bafilomycin A1-ді (деградацияны бөгеп тастау) қамтыды. Деректер сипаттамалық және салыстырмалы әдістермен талданды.

Нәтижелер. Сүт безі қатерлі ісігінің екі жасушалық желісінде MCF-10A-ға қарағанда базалық аутофагия жоғары болды. MCF-7 жасушаларында p62 мен LC3B-II жинақталып, деградацияның бұзылғанын көрсетті. MDA-MB-231 жасушаларында p62 төмен, ал LC3B-II жоғары деңгейде болды, бұл белсенді ағынды білдіреді. Фармакологиялық әсер еткенде, MDA-MB-231 жасушалары күтілетін маркерлік өзгерістер көрсетіп, функционалды аутофагияны растады. Ал MCF-7 жасушаларында LC3B-II мен p62-нің әрі қарай жинақталуы деградацияның бөгелгенін көрсетті.

Қорытынды. Зерттеу сүт безі қатерлі ісігінің әртүрлі түрлеріндегі аутофагиядағы айырмашылықтарды көрсетті. MDA-MB-231 жасушаларындағы белсенді аутофагиялық ағын олардың агрессивтілігіне және стреске бейімделуіне ықпал етуі мүмкін. MCF-7 жасушаларында деградацияның бөгелуі олардың метаболикалық стреске және емдеуге жауап беруін шектеуі мүмкін. Бұл нәтижелер сүт безі қатерлі ісігін емдеуде аутофагиялық күйді ескерудің маңыздылығын айқындайды.

Түйін сөздер: аутофагия, аутофагиялық ағын, сүт безі қатерлі ісігі, үштік теріс сүт безі қатерлі ісігі.

Сравнительный анализ аутофагической активности и потока в клеточных линиях рака молочной железы: MDA-MB-231 и MCF-7 в условиях стресса

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Резюме

Аутофагия - это фундаментальный клеточный процесс, отвечающий за переработку цитоплазматических компонентов, который играет важную роль в поддержании клеточного гомеостаза и влияет на прогрессию рака. Рак молочной железы включает различные молекулярные подтипы с разной агрессивностью и ответом на терапию.

Целью этого исследования было сравнение уровня аутофагической активности и потока у двух линий рака молочной железы - MDA-MB-231 (тройной отрицательный агрессивный подтип) и MCF-7 (люминальный гормонозависимый подтип) - в сравнении с нетуморогенными эпителиальными клетками молочной железы MCF-10A. Также мы оценили влияние фармакологической модуляции на аутофагию, чтобы понять, как аутофагический статус может влиять на поведение раковых клеток.

Методы. Проведено *in vitro* исследование с использованием клеток MDA-MB-231, MCF-7 и MCF-10A. Аутофагическая активность оценивалась по уровням белков LC3B и p62 с помощью вестерн-блоттинга и иммунофлуоресцентной микроскопии. Фармакологическая модуляция включала голодание для индукции аутофагии, 3-метиладенин для ингибирования инициации и Bafilomycin A1 для блокировки деградации. Данные анализировались описательными и сравнительными методами.

Результаты. Обе линии рака молочной железы показали более высокую базальную аутофагию, чем MCF-10A. В клетках MCF-7 наблюдалось накопление p62 и LC3B-II, что указывает на нарушение деградации. В клетках MDA-MB-231 уровень p62 был снижен, а LC3B-II повышен, что свидетельствует об активном потоке. После фармакологической модуляции клетки MDA-MB-231 показали ожидаемые изменения маркеров, подтверждающие функциональную аутофагию. В клетках MCF-7 наблюдалось дальнейшее накопление LC3B-II и p62, что указывает на блок в деградации.

Вывод. Исследование выявило чёткие различия в аутофагии между подтипами рака молочной железы. У клеток MDA-MB-231 активный аутофагический поток может способствовать их агрессивности и устойчивости к стрессу. У клеток MCF-7 блок деградации может ограничивать их реакцию на метаболический стресс и лечение. Эти данные подчеркивают важность учёта статуса аутофагии при разработке стратегий терапии рака молочной железы.

Ключевые слова: аутофагия, аутофагический поток, рак молочной железы, тройной отрицательный рак молочной железы.