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The Combination of Iron Chelator Deferoxamine and Glycolytic Inhibitors, 2-Deoxy-D-Glucose and Dichloroacetate, Synergistically Suppress the Proliferation of Human Breast Carcinoma Cell Lines

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Abstract

Cancer cells preferentially use glycolysis to produce energy, even in the presence of oxygen and functional mitochondria. Iron chelator deferoxamine mesylate (DFOM) suppresses cell growth of tumors and increases tumor cell glycolysis via stabilization of hypoxia-induced factor 1. We hypothesized that iron chelator and glycolytic inhibitors synergistically inhibited tumor cell proliferation. Human breast carcinoma cell lines, MCF-7 and MDA-MB-231, were treated with DFOM and glycolytic inhibitors, 2-deoxy-d-glucose (2-DG) and dichloroacetate (DCA). Real time PCR (qPCR) and ^{3}[H]-Thymidine incorporation assays were used to measure the expression of glycolysis-associated genes and cancer cell proliferation, respectively. The combinative effect of drugs was analyzed with program CompuSyn. qPCR showed that DFOM up-regulated the expression of glycolytic genes and glucose transporters, SLC2A1 and SLC2A3. Combination of DFOM and 2-DG or DCA synergistically inhibited the proliferation of human breast carcinoma cell lines (all combination indices were less than 1). In conclusion, iron chelator may synergistically increase the anticancer effectiveness of glycolytic inhibitors.

Keywords

Iron Chelator, Human Breast Carcinoma, Glycolysis, Glycolytic Inhibitor, Deferoxamine, Drug Synergism

1. Introduction

Cancer cells markedly increase glycolysis and lactate production in the presence of oxygen without an increase in oxidative phosphorylation (OXPHOS). This phenomenon of aerobic glycolysis is known as the "Warburg effect" [1]. Increased glycolysis produces more intermediates for the synthesis of nucleotides and amino acids that are required by rapid cancer cell growth [2]. Cancer cells usually upregulate hypoxia-inducible transcription factor (HIF-1). HIF-1 stimulates the expression of most glycolytic enzymes and glucose transporters GLUT1 and GLUT3 in cancer cells [3]. Glycolytic enzymes have become the target for cancer therapeutics. 2-deoxy-d-glucose (2-DG), a competitive inhibitor of hexokinase (HK), and dichloroacetate (DCA), an inhibitor of pyruvate dehydrogenase kinase (PDK) have been shown to suppress cancer growth in cell culture, animal and human patients [4].

Iron is an essential element required for DNA synthesis and several other important cell functions. Iron chelator deferoxamine mesylate (DFOM) suppressed cancer cell proliferation via iron depletion and other mechanisms. Treatment with DFOM mimics hypoxia induction of HIF-1 which activated transcription by binding to hypoxia response elements (HRE). Increased HIF-1 further upregulates glycolytic enzymes, the targets for glycolytic inhibitors, and increases glucose uptake [5] [6]. We hypothesized that iron chelator DFOM synergistically improves the anticancer effect of glycolytic inhibitors. Gene expression of glycolytic enzymes was measured by real time PCR (qPCR). The proliferation of human breast carcinoma cells was examined by ^{3}[H]-Thymidine incorporation assay. The results showed that DFOM and glycolytic inhibitors, 2-DG and DCA, exhibited a synergistic inhibition effect on human breast carcinoma cell lines MCF-7 and MDA-MB-231 (all combination indices were less than 1).

2. Materials and Methods

2.1. Cell Culture

Breast carcinoma cell lines, MCF-7 and MDA-MB-231, were obtained from ATCC. MCF-7 is estrogen receptor (ER) and progesterone receptor (PR) positive, but MDA-MB-231 is ER and PR negative. All cancer cell lines were grown in alpha-MEM supplemented with 10% fetal bovine serum. Cells were detached from tissue culture flasks by digestion with TrypLE express (GIBCO Invitrogen, Carlsbad, CA, USA). All cell lines were maintained in the media without supplement of any antibiotics.

2.2. Real Time Polymerase Chain Reaction (qPCR)

MCF-7 and MDA-MB-231 cells were incubated in the media containing DFOM at 37˚C and 5%CO_2 for 24 hours. The experiments were repeated three times. The total RNA was isolated by PurLink RNA Kit (Invitrogen, Carlsbad, CA). We used qPCR to measure mRNA expression of genes. The detailed protocol was published previously [7]. In brief, cells were removed from plates by TrypLE express. cDNA

was synthesized by the High Capacity RNA-to-cDNA kit (Applied Biosystems, Grand Island, NY). Gene expression quantification was performed with TaqMan Gene Expression Assay, a proven 5' nuclease-based real-time PCR chemistry. Primers and probes (PrimeTime Mini qPCR assay) were synthesized by Integrated DNA Technologies (IDT, Coralville, Iowa). Sequences of primers and probe of glycolytic enzyme genes are described in Table 1. Probes contain at the 5' end the FAM (6-carboxy fluorescein) as a fluorescent reporter dye, and internal and at 3' end the ZEN™/Iowa Black FQ as fluorescent double quenchers. The expression of β-actin (ACTB) was not changed by iron chelator DFOM in the pre-experiment, thus ACTB was used as endogenous gene control to normalize PCRs for the amount of RNA added to the reverse transcription reactions. Forty µl of qPCR reaction mixture contained 20 µl of TaqMan universal PCR master mix (Applied Biosystems, Grand Island, NY), 4 µl of 10x PrimeTime Mini qPCR assay and 16 µl of cDNA (100 ng). The qPCR reaction was aliquoted in triple to wells of 384-well PCR plate. The plate was sealed, briefly centrifuged, and performed reaction with 7900HT real time PCR system (Applied Biosystems, Grand Island, NY). Standard mode ran as 2 minutes at 50˚C and 10 minutes at 95˚C, and 40 cycles (15 seconds at 95˚C and 1 minute at 60˚C). Target gene expression was determined by relative quantification (RQ). Gene expression was analyzed by RQ Manager 1.2 software (Applied Biosystems, Grand Island, NY) and calculated as the ratio of mRNA of the DFOM-treated cell lines to that of the untreated cell lines.

Table 1. List of primer and probe sequences for qPCR.

Gene	Pair of primers (FWD and REV)	Probe
ACTB	GGATCAGCAAGCAGGAGTATG; AGAAAGGGTGTAACGCAACTAA	TCGTCCACCGCAAATGCTTCTAGG
HIF1a	GTCTGCAACATGGAAGGTATTG; GCAGGTCATAGGTGGTTTCT	ACTGCACAGGCCACATTCACGTAT
ALDOC	GGGTGTACGCTCACTGATTT; GATGGAGAAACCACCACTCAA	AGAAGGATGGTGCTGACTTTGCCA
ENO1	CATGCCGATGACCACCTTAT; CTCCCAACATCCTGGAGAATAAA	AGACTGCTATTGGGAAAGCTGGCT
GAPDH	GAGTCCTTCCACGATACCAAAG; GGTGTGAACCATGAGAAGTATGA	AGATCATCAGCAATGCCTCCTGCA
GPI	GTTGATGAGCCCATTGGTAGAA; GGAAAGCAGCTGGCTAAGAA	TTGATGGCAGTGCTCAAGTGACCT
HK2	GCAGAAGGTTGACCAGTATCTC; CCAAGCCCTTTCTCCATCTC	CACATGCGCCTCTCTGATGAGACC
PFKM	GCATCCCATTTGTGGTCATTC; GTCACAGGTTGTGCAGATAGT	AATGTCCCTGGCTCAGACTTCAGC
PGK1	TTGGGACAGCAGCCTTAATC; CTGGACAAGCTGGACGTTAAA	CGACTCTCATAACGACCCGCTTCC
PKM2	CTGTGGCTGGACTACAAGAA; CTGCTTCACCTGGAGAGAAATA	AAGTGGGCAGCAAGATCTACGTGG
TMI1	CAGTCACAGAGCCTCCATAAA; CCCAGGAAGTACACGAGAAG	ACCGCATCAGAGACGTTGGACTTC
SLC2A1	CTGGGCAAGTCCTTTGAGAT; GTGACACTTCACCCACATACA	AGTACACACCGATGATGAAGCGGC
SLC2A3	AGGATGCAGGTGTTCAAGAG; GCCCTTTCCACCAGAAATAGA	CGGCGCGGGTGTGGTTAATACTAT
LDHA	AGATTCCAGTGTGCCTGTATG; ACCTCTTTCCACTGTTCCTTATC	AGTGGAATGAATGTTGCTGGTGTCTCT
PDK1	GAGGTCTTGGTGCAGTTGAATA; ACGCTGGGTAATGAGGATTTG	TGTGAAGATGAGTGACCGAGGAGGT

2.3. ^{3}[H]-Thymidine Incorporation Assay

Four thousands of MCF-7 or MDA-MB-231 cells per well in 100 µl media were plated in 96-well culture plates and incubated overnight at 37˚C. Then, the media

were aspirated and replaced by fresh media containing DFOM, glycolytic inhibitors 2-DG or DCA, or combination of DFOM and 2-DG or DCA. The dose that suppressed 90% - 100% cell growth was determined as the starting dose of single drug via pre-experiment. The combination treatment was the simultaneous addition of DFOM and 2-DG or DCA. For MCF-7 cells, the starting doses were 80 μM DFOM, 40 mM 2-DG or 100 mM DCA, and 80 μM DFOM + 40 mM 2-DG or 100 mM DCA. For MDA-MB-231, the starting doses were 160 μM DFOM, 80 mM 2-DG or 200 mM DCA, and 160 μM DFOM + 80 mM 2-DG or 200 mM DCA. The fixed 2 folds of serial dilution was used. The control was replaced by media only. The plate was continuously cultured for another 4 days. ^{3}[H]-thymidine (0.1 μCi/well, MP Biomedical, Santa Ana, CA, USA) was added to all wells for the last 16 hours of incubation. The cells were removed from the plates by trypsin-EDTA digestion, and harvested onto a glass-fiber filter (Skatron Basic 96 Harvester, Skatron, Inc., Sterling, VA, USA). The filters were placed into scintillation fluid, and the radioactivity was counted by liquid scintillation (LS 1800, Beckman Co., Fullerton, CA, USA). Cell proliferation was quantitated by ^{3}[H]-thymidine incorporation. Cell inhibition of drug was expressed as a percentage of the control. All the ^{3}[H]-thymidine incorporation experiments were done in triplicate and were repeated three times. Combination index (CI) was analyzed with CompuSyn software. The detailed method was referred to Chou TC's publication [8]. CI values were calculated for each independent experiment before averaging. Drug synergism (CI < 1), additive-effect (CI = 1) and antagonism (CI > 1) were determined [8].

3. Results

3.1. Iron Chelator DFOM Upregulated the Expression of Glycolytic Genes and Glucose Transporters

Human breast carcinoma cells MCF-7 and MDA-MB-231 were treated with DFOM for 24 hours. qPCR showed DFOM increased mRNA of HIF1a, glucose transporter genes SLC2A1 and SLC2A3, and multiple glycolytic genes (**Figure 1** and **Figure 2**). DFOM-induced gene upregulation is dose-dependent. After cells treated with 1600 μM DFOM for 24 hours, mRNA HK2 increased approximately 21- and 28-folds in MCF-7 and MDA-MB-231, respectively. The mRNA levels of PDK1 also elevated approximately 14- and 7-times in MCF-7 and MDA-MB-231, respectively. These results suggest that iron chelator stimulates aerobic glycolysis in cancer cells.

3.2. Iron Chelator DFOM Augmented the Cytotoxicity of Glycolytic Inhibitors 2-DG and DCA to Human Breast Carcinoma Cells MCF-7 and MDA-MB-231

DFOM, 2-DG and DCA suppressed the proliferation of MCF-7 and MDA-MB-231 cells in a dose dependence manner (**Figures 3-6**). DFOM synergistically increased cell suppression of glycolytic inhibitors 2-DG and DCA. The combinative indices of DFOM and 2-DG or DCA were 0.80 ± 0.08 (DFOM + 2-DG) and 0.56

± 0.13 (DFOM + DCA) in MCF-7 cells, and 0.72 ± 0.21 (DFOM + 2-DG) and 0.65 ± 0.15 (DFOM + DCA) in MDA-MB-231 cells, respectively. All drug combinative effects were synergistic (Table 2).

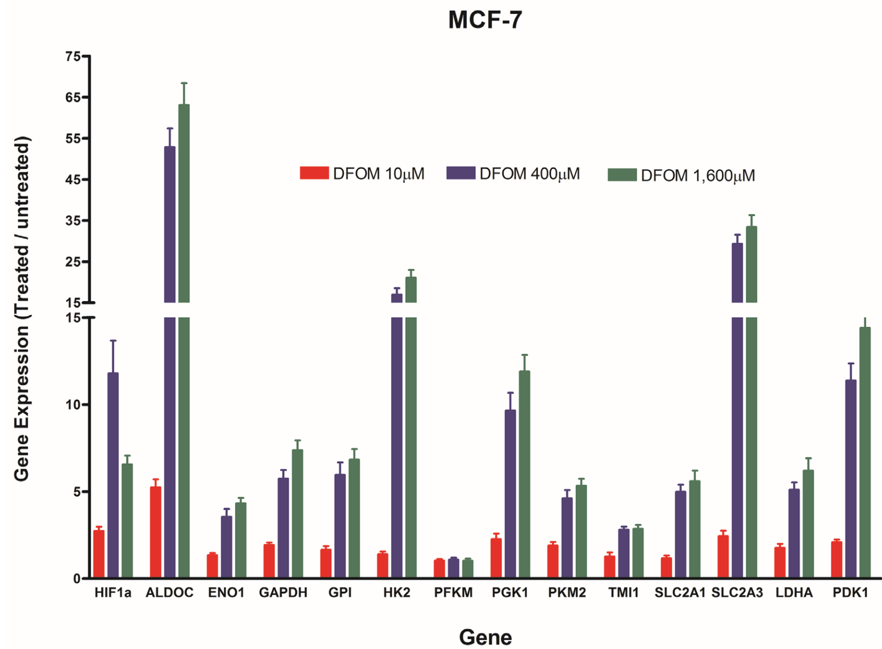

Figure 1. DFOM increases the expression of HIF1a, glycolytic enzyme and glucose transporter genes in human breast carcinoma cell line MCF-7. The cells were treated with DFOM for 24 hours and gene expression levels were measured by qPCR.

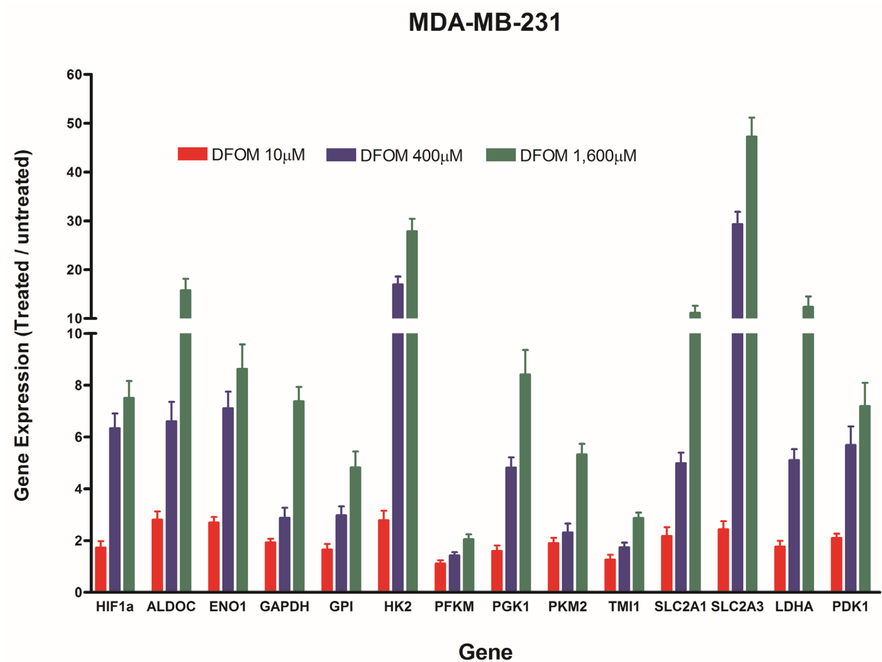

Figure 2. DFOM increases the expression of HIF1a, glycolytic enzyme and glucose transporter genes in human breast carcinoma cell line MDA-MB-231. The cells were treated with DFOM for 24 hours and gene expression levels were measured by qPCR.

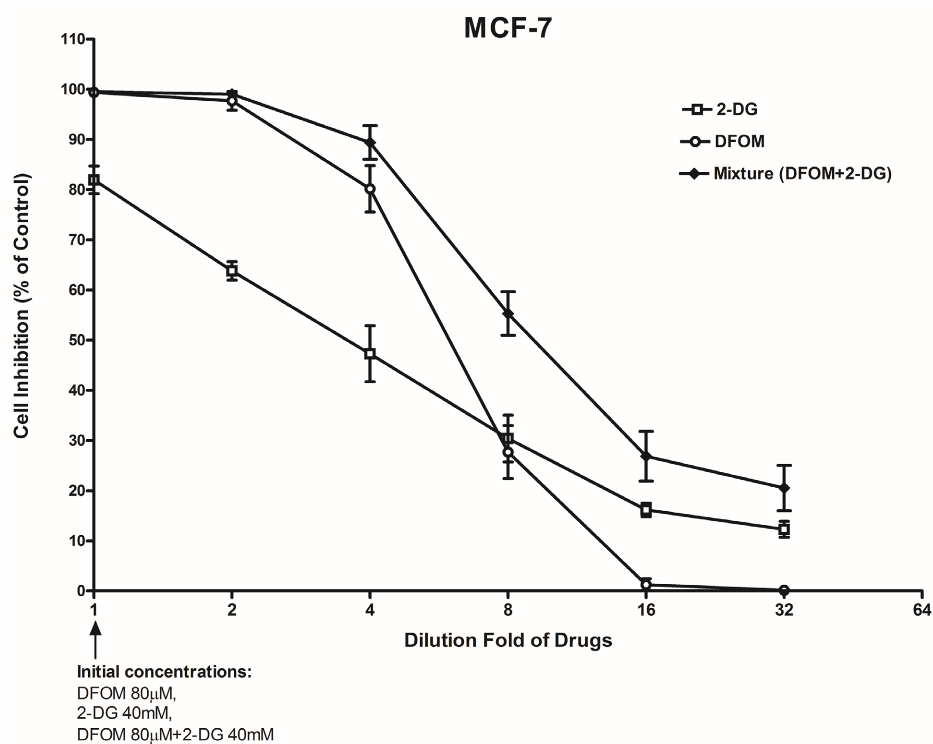

Figure 3. Iron chelator DFOM increases cell cytotoxicity of glycolytic inhibitor 2-DG on human breast carcinoma cell line MCF-7. The cells were treated with drugs for 4 days. The details were seen in the Materials and Methods.

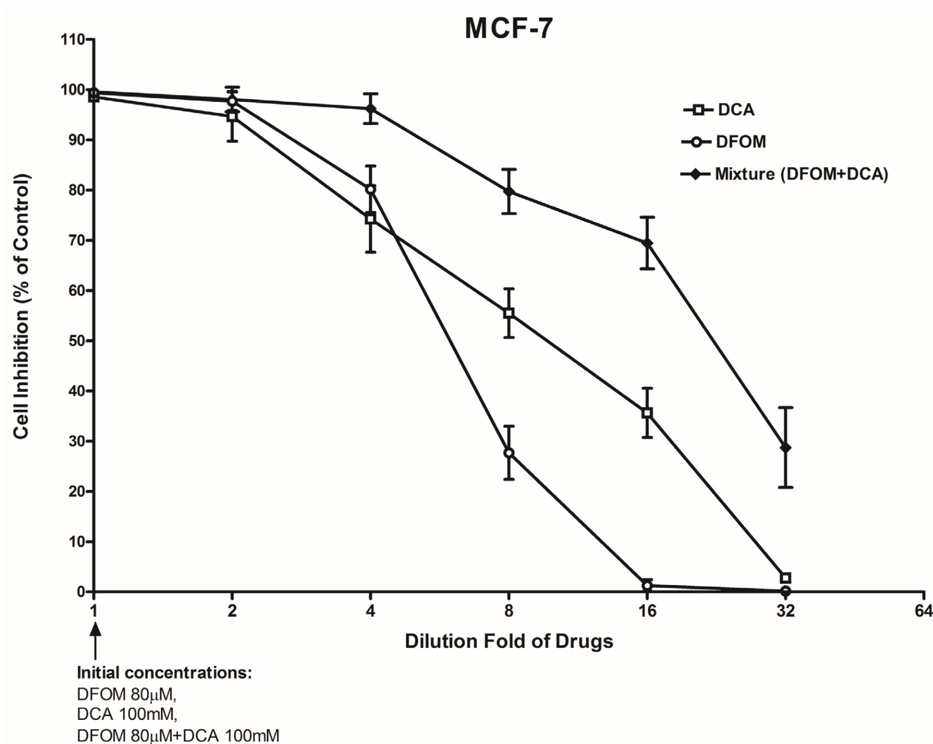

Figure 4. Iron chelator DFOM increases cell cytotoxicity of glycolytic inhibitor DCA on human breast carcinoma cell line MCF-7. The cells were treated with drugs for 4 days.

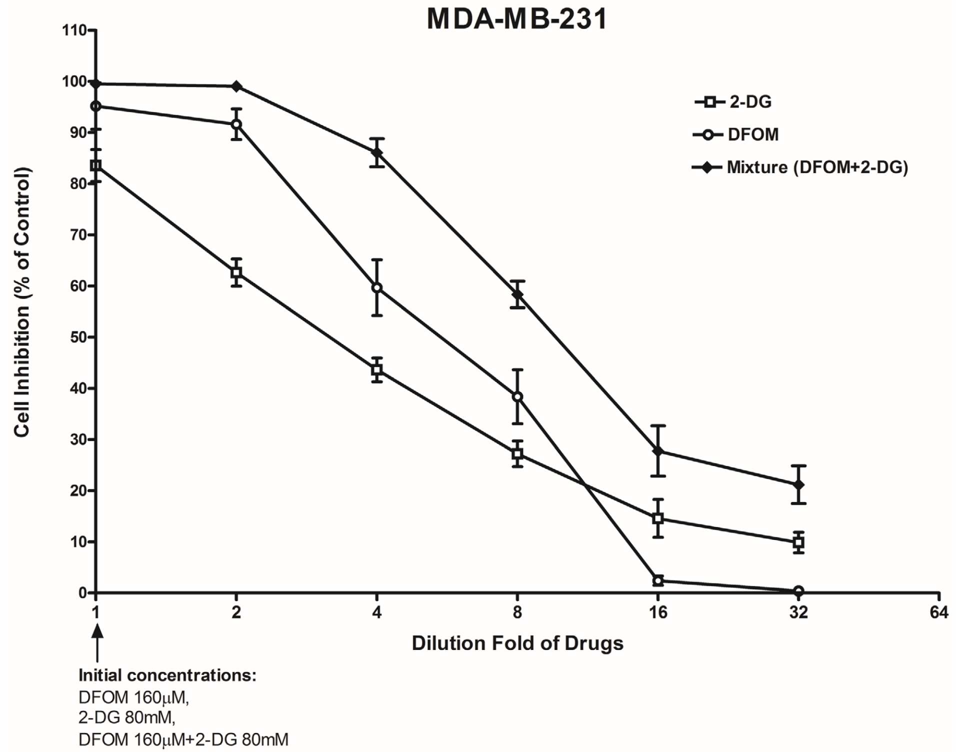

Figure 5. Iron chelator DFOM increases cell cytotoxicity of glycolytic inhibitor 2-DG on human breast carcinoma cell line MDA-MB-231. The cells were treated with drugs for 4 days.

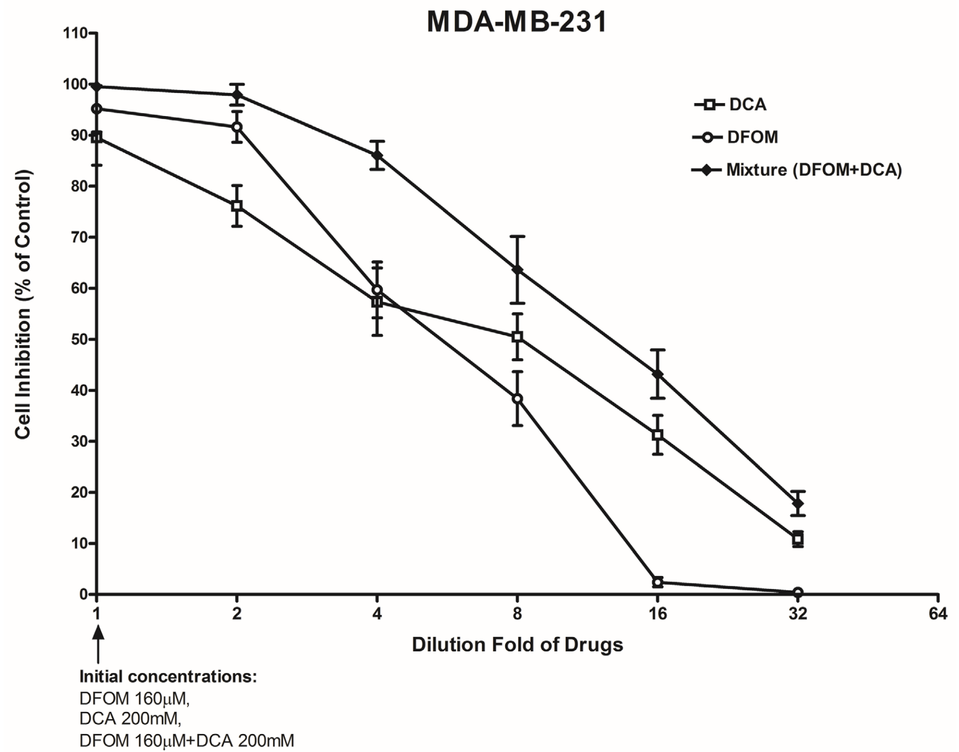

Figure 6. Iron chelator DFOM increases cell cytotoxicity of glycolytic inhibitor DCA on human breast carcinoma cell line MDA-MB-231. The cells were treated with drugs for 4 days.

Table 2. The combinative effects of iron chelator and glycolytic inhibitors on human breast carcinoma cell lines. The details were seen in the materials and methods.

Cell Line	Mixture	CI[1]	Combination Effect
MCF-7	DFOM + 2-DG	0.80 ± 0.08 (3)[2]	synergism
MCF-7	DFOM + DCA	0.56 ± 0.13 (3)	synergism
MDA-MB-231	DFOM + 2-DG	0.72 ± 0.21 (3)	synergism
MDA-MB-231	DFOM + DCA	0.65 ± 0.15 (3)	synergism

[1]Combination indices < 1， =1 and >1 indicate combinative effect synergism, addition and antagonism, respectively. [2]Mean ± standard deviation (number).

4. Discussion

Iron is an important nutrient element involved in cell replication, metabolism and growth. Physiological homeostasis of iron is strictly controlled through a series of iron-associated protein network including iron-responsive element binding proteins (IRPs), iron uptake protein transferrin receptor (TFR1), iron storage protein ferritin (FT), etc. Increased levels of TFR1, FT, and intracellular iron have been demonstrated in various cancers [9]. Iron depletion by chelators such as DFOM has been utilized in anticancer therapies [6] [10]. Moreover, iron chelator might augment the treatment of chemotherapy or radiotherapy for cancer [10]. Iron chelators also induce a similar response of hypoxia by stabilizing hypoxia-inducible factor 1 alpha (HIF-1α) [5] [11]. HIF-1α is regulated by iron-dependent enzymes called prolyl hydroxylases (PHDs). PHDs use iron as a cofactor to modify and degrade HIF-1α under normal oxygen conditions. When iron is depleted, PHDs are less active, leading to HIF-1α stabilization and activation of HIF-1. In the current study, DFOM inhibited the proliferation of human breast carcinoma cell lines MCF-7 and MDA-MB-231 in the dose dependent pattern (**Figures 3-6**). DFOM also increased HIF-1α mRNA in human breast carcinoma cell lines MCF-7 and MDA-MB-231 (**Figure 1** and **Figure 2**). HIF1 is a key regulator in cancer, promoting glycolysis by activating glycolytic enzymes, cell proliferation and cancer progression [12] [13]. After DFOM treatment, increased HIF-1α further upregulated the expression of glycolysis associated genes and glucose transporter genes in MCF-7 and MDA-MB-231 cells (**Figure 1** and **Figure 2**). These suggest that the monotherapy of iron chelation not only inhibits cancer cell proliferation, but also promotes cancer progression via increased HIF1 and aerobic glycolysis. The latter effect counteracts the anticancer effect of iron chelator.

Cancer prefers to ferment glucose to lactate for energy even when oxygen is present, which is termed as "Aerobic glycolysis" or "Warburg effect". This metabolic reprogramming provides rapid access to ATP, precursors for biosynthesis, and growth-promoting metabolic intermediates for cancer cells, although it is an inefficient energy source [14]. Aerobic glycolysis has become a target for cancer therapeutics [15]. 2-DG inhibits glycolysis by a non-metabolizable glucose analog, competing with glucose transport into cells, and accumulating as an intermediate that inhibits hexokinase (HK) [16] [17]. Another glycolytic inhibitor DCA inhib-

its pyruvate dehydrogenase kinase (PDK), forcing cancer cells back to oxidative metabolism and restoring the normal apoptosis process [18] [19]. We found that glycolytic inhibitors 2-DG and DCA suppressed the proliferation of MCF-7 and MDA-MB-231 in a dose dependent manner (**Figures 3-6**). Iron chelator DFOM increased multiple glycolytic enzymes and glucose transporters, the targets for glycolytic inhibitors (**Figure 1** and **Figure 2**). We hypothesized that combination of DFOM and 2-DG or DCA might enhance the drug's effectiveness. In the current study, the results show that combination of DFOM and 2-DG or DCA synergistically inhibits the proliferation of human breast carcinoma cell lines MCF-7 and MDA-MB-231, compared to single drug treatment (**Figures 3-6**, **Table 2**). DFOM combined with glycolytic inhibitor 2-DG or DCA enhanced cancer cell inhibition and reduced drug concentrations, compared to the treatment of single drug DFOM, 2-DG or DCA. The combination effects will be tested in more cancer cell lines. Even though the synergistic effectiveness, drug doses and side effects remain to be determined in cancer animal models and patients, the current results suggest that combination of iron chelation and glycolytic inhibition is a potential treatment for aggressive types of breast carcinoma such as triple negative breast cancer.

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Conflicts of Interest

The authors declare that they have no conflict of interest.

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Abbreviations

HIF1	hypoxia-induced factor 1
qPCR	real time polymerase chain reaction
OXPHOS	oxidative phosphorylation
DFOM	deferoxamine mesylate
2-DG	2-deoxy-d-glucose
DCA	dichloroacetate
ATP	adenosine triphosphate
SLC2A1	glucose transporter 1
SLC2A3	glucose transporter 3
ALDOC	aldolase A
ENO1	enolase 1
GAPDH	glyceraldehyde-3-phosphate dehydrogenase
GPI	glucose-6-phosphate isomerase
HK2	hexokinase 2
PFKM	phosphofructokinase-1
PGK1	phosphoglycerate kinase 1
PKM2	pyruvate kinase
TMI	triosephosphate isomerase 1
LDHA	lactate dehydrogenase A
PDK1	pyruvate dehydrogenase kinase 1