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Cell survival regulation during receptor-mediated endocytosis of chemically-modified lipoproteins associated to the formation of an Amphiphysin 2 (Bin1)/c-Myc complex

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ABSTRACT

Amphiphysin 2 and members of the BAR-domain family of proteins participate in a wide array of cellular processes including cell cycle and endocytosis. Given that amphiphysin 2 is related to diverse cell responses as a result of metabolic stress, we investigated in macrophages whether oxidative stress originated by the internalization of oxidized low density lipoproteins (oxLDL) affect both, the expression of amphiphysin 2 and its binding partner c-Myc. Here we report that under oxidative stress, a complex formation between amphiphysin 2(Bin1) and c-Myc allows the cell to develop a novel survival equilibrium state established between cell proliferation and cell death. We propose that under conditions of oxidative stress given by the internalization of oxLDL, macrophages employ the formation of the amphiphysin 2(Bin1)/c-Myc complex as a control mechanism to initially avoid the process of cell death in an attempt to prolong cell survival.

1. Introduction

The eukaryotic cell contains a complex system of membranes that performs specialized functions such as internalization of extracellular molecules, nutrients, and ligands by clathrin-mediated endocytosis. Ligand binding to receptors in the plasma membrane triggers the formation of coated pits [1], where the formation of vesicles requires several adaptor proteins that in a coordinated sequence induce clathrin nucleation at specific internalization sites. Membrane invagination at these sites is mediated by a group of proteins that promote membrane remodeling and where the Bin1/Amphiphysin/Rvs (BAR) superfamily plays an important role.

This group of proteins participates in the organization of the cytoskeleton and processes such as programmed cell death and transcriptional control [2,3] and share two functional domains, a membrane binding BAR domain at their N-terminus and the SH3 domain for protein interactions at their C-terminus [4,5]. Two members of the BAR family, amphiphysins 1 and 2, have been identified in brain cells where they function as a dimer recruiting dynamin 1 to the plasma membrane. The GTPase activity shown by this dimer promotes fission of the endocytic vesicle neck during internalization by coupling the protein conformational change with the catalytic activity [2].

The role for amphiphysin 1 in clathrin-mediated endocytosis was first identified based on interactions with clathrin, dynamin, and the AP-2 complex, among other accessory endocytic proteins [6] as well as to cell cycle regulators such as c-Myc [7]. Further support for a role in cell proliferation has been provided by the observation that Bin1is frequently absent in breast tumors [8] and the fact that programmed cell death via a caspase-independent pathway is induced following ectopic overexpression of Bin1 in cells expressing c-Myc [9]. In skeletal muscle cells (C2C12), expression of Bin containing exons 10 and 13 has also been found to induce cellular differentiation [10,11]. However, expression of exon 10 prevents interactions between the SH3 domain of amphiphysin 2 and dynamin 1, possibly due to the possibility that dynamin

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might not be recruited to the membrane, and therefore impeding its function [3].

Since to date it has been difficult to understand the role of amphiphysin 2 in the regulation of the cell cycle coupled to clathrin-mediated endocytosis, our goal has been to extend our previous observations of the mechanisms involved in scavenger receptor internalization and to explore the subtle mechanisms regulating the equilibrium between cell proliferation and cell death during this process [12,13]. Given that amphiphysin 2 appears to be related to diverse cell physiological responses as a result of oxidative stress, it was investigated whether internalization of oxidized low density lipoproteins (oxLDL) affects both, the expression of amphiphysin 2 and the interaction carried out between amphiphysin 2 and its binding partner c-Myc, controlling a potential survival mechanism established between cell proliferation and cell death.

2. Materials and methods

2.1. Materials

All salts and reagents used in this study were purchased from Sigma-Aldrich (St. Louis, MO, USA). Reagents for cell culture, vectors for transfection, fluorescence reagents and Lipofectamine were purchased from Invitrogen (Carlsbad, CA, USA). Opti MEM was obtained from Thermo Fisher Scientific (Waltham, MA, USA). Antibodies were from Santa Cruz Biotechnology (Santa Cruz, CA, USA). PC12 rat pheochromocytome cells, mouse RAW 264.7 cells, and human vascular smooth muscle cells (VSMC) were obtained from American Type Culture Collection (Manassas, VA, USA). CHO-F7 cells were kindly provided by Dr. Robert E. Pitas. The research and experimental protocols have been approved by the Ethics and Research Committee of Instituto de Fisiología Celular, Universidad Nacional Autónoma de México. Human plasma was obtained from healthy volunteers who attend as blood donors in the blood bank of the National Medical Center “20 de Noviembre” ISSSTE, Mexico City. Each subject signed the respective informed consent following guidelines of the Declaration of Helsinki.

2.2. Cell culture

CHO-F7 cells correspond to CHO cells stably transfected with scavenger receptor I class A. CHO cells not expressing scavenger receptor A were used as control cells [12]. CHO cells were grown in 50% F12 medium and 50% DMEM added with 10% of fetal bovine serum (FBS) in the presence of 100U of gentamycin and 0.25 mg/mL of fungizone as reported before [12]. PC12 cells were grown under the same conditions used for CHO cells, except 100 mg/mL penicillin/streptomycin was included in the medium instead of gentamycin [12]. RAW 264.7 cells were grown in RPMI 1640 supplemented with 10% FBS plus penicillin and streptomycin (50 μg/mL) [14]. For experiments performed with low-density lipoproteins (LDL), RAW cells were grown in reduced serum Opti MEM without phenol red plus FBS (<0.1%) as reported before [15].

2.3. Cloning and transfection

Trizol reagent was used to extract high quality RNA from CHO, CHO-F7, and PC12 cells. For cDNA synthesis, 2 μg of RNA were amplified after a denaturation step of 5 min at 95 °C with 25 cycles of 95 °C for 30 s, 58 °C for 30 s, and 42 °C for 1.5 min, followed by an extension step at 42 °C for 5 min. The oligonucleotides used to amplify amphiphysin 2 were designed based on reported mouse (U86405), rat (Y13380) and human (AF004015) sequences. A sense primer (BIN1-1S 5′-ATGGCAGAGATGGGGAGC3′) and an antisense primer (BIN1-735A 5′ CACCCGCTCTGTGAAATTC-3′) were synthesized. For amphiphysin 2(Bin1) isoform cloning, vector pcDNA3 V5 His TOPO was used. Transfection experiments using CHO-F7 cells were performed using lipofectamine, according to the manufacturer's instructions incubating cells with the appropriate DNA/liposome mixture.

2.4. LDL modification

The isolation and acetylation of LDL (densities of 1.019–1.063 g/mL) were performed as previously described [12,16]. Labeling of LDL was carried out employing 1,1′-dioctadecyl-3,3,3′3′-tetramethylindocarbocyanine perchlorate (DiI) (Molecular Probes, Eugene, OR). Acetylation (ac) or oxidization (ox) reactions were also performed as previously described [12,17,18]. Oxidation was terminated with the addition of EDTA, and resulting oxLDL dialyzed against 150 mM NaCl containing 100 μM EDTA and stored at 4 °C [19,20].

2.5. Internalization experiments

After a starvation period of 1 h, RAW and CHO-F7 cells were incubated with DiI-nLDL, DiI-acLDL, orDiI-oxLDL at the indicated concentrations, at 37 °C under a 5% CO2/95% air atmosphere [19,20]. After internalization experiments, cell cultures were washed (5×) with 2 mg/mL albumin in PBS and fluorescence measured by flow cytometry using a FACSCalibur (BD Biosciences, San Jose, CA, USA) at 480/538 nm (excitation/emission). Parallel cultures used for internalization experiments were prepared for confocal microscopy or lysed for Western blot assays.

2.6. Cell viability assays

Cell viability was estimated using 3-(4,5-dimethylthiazol-2-yl)-2,5-diphenyltetrazolium bromide (MTT). Formazan crystals were dissolved in a lysis buffer containing 20% SDS and 50% N,N-dimethylformamide (pH 3.7) during 12 h at 37 °C and absorbance readings performed at 570 nm.

2.7. Confocal microscopy

For confocal microscopy experiments, after incubation for 4 h with DiI-acLDL, cells were rinsed with PBS and fixed in 4% paraformaldehyde/PBS. After 30 min, cells were washed with PBS (3×), mounted and then imaged using excitation/emission wavelengths of 543/595 nm in a confocal microscope Olympus FV1000 (Shinjuku-ku, Tokyo, Japan).

2.8. Western blot and immunoprecipitation assays

RAW cells or CHO-F7 transfected cells were lysed in a buffer containing 1% Triton-X-100, 0.5% NP40, 150 m MNaCl, 10 mM Tris, 1 mM EDTA (pH 8.0), 1 mM EGTA (pH 8.0), 2 mM sodium orthovanadate, 1 mM benzamidine, 1 mM PMSF (pH 7.4), 37.5 μg/mL leupeptin, and 2 μg/mL aprotinin. Cell protein was recovered by centrifugation and pellets resuspended in 0.1% SDS and stored at −20 °C. Thirty μg of total protein for each sample were separated in 12% polyacrylamide gel electrophoresis (SDS-PAGE) and transferred to PVDF membranes for Western blot analysis. Unspecific binding was blocked at 37 °C with TBS-T buffer (100 mM Tris (pH 7.5), 0.15 M NaCl, 0.1% Tween-20, and 5% fat-free milk). After 1 h, membranes were incubated at 37 °C for 1 h with primary antibodies diluted as follows: 1:200 for amphiphysin 2 (N19), 1:750 for c-Myc (M-262), 1:800 for SR-B1 (G13) and 1:500 for β-actin. Membranes were subsequently incubated with the appropriate

horseradish peroxidase-coupled secondary antibodies in TBST (1:5000) at 37 °C. After 1 h, bound antibodies were visualized using an Immobilon chemiluminescence HRP reagent (Merck Millipore; Billerica, MA).

For immunoprecipitation assays, treated RAW cells were rinsed with PBS and resuspended in a buffer containing 250 mM sucrose, 3 mM imidazole (pH 7.4) and freshly added protease/phosphatase inhibitors at 4 °C. Cell fractions were recovered by centrifugation at 3400 rpm for 15 min at 4 °C. Pellets and supernatants were incubated with protease/phosphatase inhibitors with gentle agitation at 4 °C. and centrifuged at 8000 rpm for 10 min. Both cytosolic and nuclear fractions containing 400 μg and 300 μg of total protein, respectively, were incubated with a c-Myc antibody (1:400) at 4 °C. After 2 h, the immune complexes were precipitated with G protein agarose Fast Flow (Millipore) overnight at 4 °C. After three washes with lysis buffer, the immunoprecipitated proteins were resuspended in 20 μl of Laemmli sample buffer, loaded on 10% SDS-PAGE and transferred to PVDF membranes. PVDF membranes were incubated with a Bin1 99D antibody (1:300) overnight at 4 °C, followed by incubation with the corresponding secondary antibody and visualization reagents as described above.

3. Results

3.1. Internalization of oxLDL by RAW 264.7 cells

When studied by Western blot analysis, internalization assays performed incubating RAW 264.7 cells with native LDL (nLDL) or oxLDL (10 μg/mL) show the presence of Bin1 in the cytoplasmic fraction (Fig. 1A), in contrast to the nuclear fraction where it is not identified (not shown). Nevertheless, in contrast to control cells, when incubated with oxLDL, cytoplasmic Bin1 expression shows a steady increase with time reaching its maximum expression level between 4 and 16 h (Fig. 1A). Interestingly, the increase in nuclear Bin1 expression is directly correlated to the fact that nuclear c-Myc expression under the same experimental conditions tends to decrease with time (Fig. 1B). Since there is evidence that Bin1 is able to influence the cell cycle by interacting with c-Myc promoting the initiation of apoptosis [8,9], cell viability was also evaluated during the internalization of both nLDL and oxLDL particles. Interestingly, when both types of particles are being internalized up to 16 h incubation, cell viability remained at control values (Fig. 1C). Further analysis carried out for the internalization process using Dil-oxLDL and flow cytometry measurements, shows with time an increment of particle internalization using a fixed lipoprotein concentration (Fig. 1D) in the same way as when assays increasing the lipoprotein concentration are employed (Fig. 1E). To determine whether Bin1 interacts with c-Myc in the cytoplasm when Bin1 expression increases in response to the internalization of oxLDL, c-Myc immunoprecipitation experiments were performed. An interaction between Bin1 and c-Myc was detected in the cytoplasm from cells exposed to high oxLDL concentrations (10–75 μg/mL), indicating a concentration dependent interaction (Fig. 1F). At the highest oxLDL concentration used (75 μg/mL), this interaction is apparently lost, indicating that cell viability might start to be compromised (Fig. 1F). Although it is known that c-Myc mediates cell cycle progression forming a powerful transcriptional activator through a heterodimerization with Max [21–25] implicated in differentiation, cell growth and cell proliferation [26–28], Bin1/c-Myc complexes have been also detected in actively proliferating cells suggesting that Bin1/c-Myc interactions inhibit cell proliferation [29]. In contrast, our results indicate that the formation of a Bin1/c-Myc complex in conditions of cellular oxidative stress tends to maintain viability when RAW 264.7 cells are exposed to oxLDL (Fig. 1C). We provide original evidence consistent with the fact that under a condition of oxidative stress, this type of interaction such as the one achieved between Bin1 and c-Myc provides a positive regulation of the cell cycle maintaining its progression. Our results suggest that Bin1/c-Myc interactions during oxLDL internalization constitutes a compensatory mechanism that enables macrophages to buffer the effects of reactive oxygen species (ROS) due to the internalization and presence of oxLDL, therefore maintaining cell viability as a survival mechanism despite the harmful effects of oxLDL present in the cytoplasm.

3.2. Cloning and expression of several amphiphysin2/Bin1 isoforms employing CHO-F7 and PC12 cells

In order to investigate the role of several key amphiphysin 2/Bin1 exons involved in the internalization of oxLDL during the establishment of a state of oxidative stress, four isoforms were cloned in CHO-F7 and PC-12 cells showing diverse patterns for exon deletion (Fig. 2A and B), and therefore differentiated protein products (Fig. 2C). Three amphiphysin 2 isoforms were obtained employing CHO-F7 cells, and one from PC12 cells, a cell line that internalizes molecules via the same scavenger receptor pathway used by RAW cells and then used as a good model for studying endocytosis [30]. Employing CHO-F7 cells, clones obtained are: pcBin1-6/F7 (−10, +12A, −13), pcBin1-7 (−10, −12A, −13), and pcBin1-10/F7 (−6, −10, −12A, −13), while clone pcBin1-1/PC12 (−10, +12A, +13) was obtained from PC12 cells. All clones are classified based on the presence (+) or absence (−) of the indicated exons (Fig. 2A and B). Additional information related to clone sequences and alignment is also provided (Table S1 and Fig. S1). The efficiency of transfection by the amphiphysin 2 clones was confirmed by Western blot analysis. While control CHO-F7 cells and CHO-F7 cells transfected with the *LacZ* vector do not exhibit Bin1 expression, all isoform clones were successfully translated (Fig. 2C). Although translation efficiency seems to vary among clones, the expression of c-Myc is constant in controls and all Bin1 transfected cells (Fig. 2C).

3.3. Confocal microscopy of cells expressing amphiphysin2/Bin1 isoforms during DiI-acLDL internalization

For confocal microscopy observations, DiI-acLDL (10 μg/mL) was used instead of DiI-oxLDL in order to avoid a possible interference known to exist between $-O^2$ and DiI. Visualized as areas of maximum fluorescence 4 h after the start of internalization, *LacZ* transfected CHO-F7 cells are found to show a discrete number of endocytic vesicles compared to control cells (Fig. 3A, a-c). Following the transient transfections of each *Bin1* isoform, the corresponding internalization of DiI-acLDLs is also observed (Fig. 3A, d-g). In particular, cells transfected with pcBin1-1/PC12 (Fig. 3A, d) accumulated the highest levels of internalized DiI-acLDLs. In order to directly measure the level of DiI-acLDL internalization, fluorescence quantitation was also performed by flow cytometry. Cells transfected with pcBin1-1/PC12 in comparison to the positive control and the Bin1/CHO-F7 transfected isoforms, show the highest internalization rate (Fig. 3B) that also corresponds to the optimal Bin1 isoform translation (Fig. 2C).

Considering that exon 12A forms part of the Clathrin-Associated Protein-Binding Domain (CLAP), a consensus region for motifs interacting with several scaffolding proteins during endocytosis [31], our results show that modifications in exon 12 are consistent with changes observed in internalization. Additionally, exon 13 of amphiphysin 2 that encodes for the N-terminal portion of the c-Myc Binding Domain (MBD), plays an important role in cell cycle control. In contrast to clone pcBin1-1/PC12 showing the highest rate of internalization compared to controls and CHO-F7 isoforms;

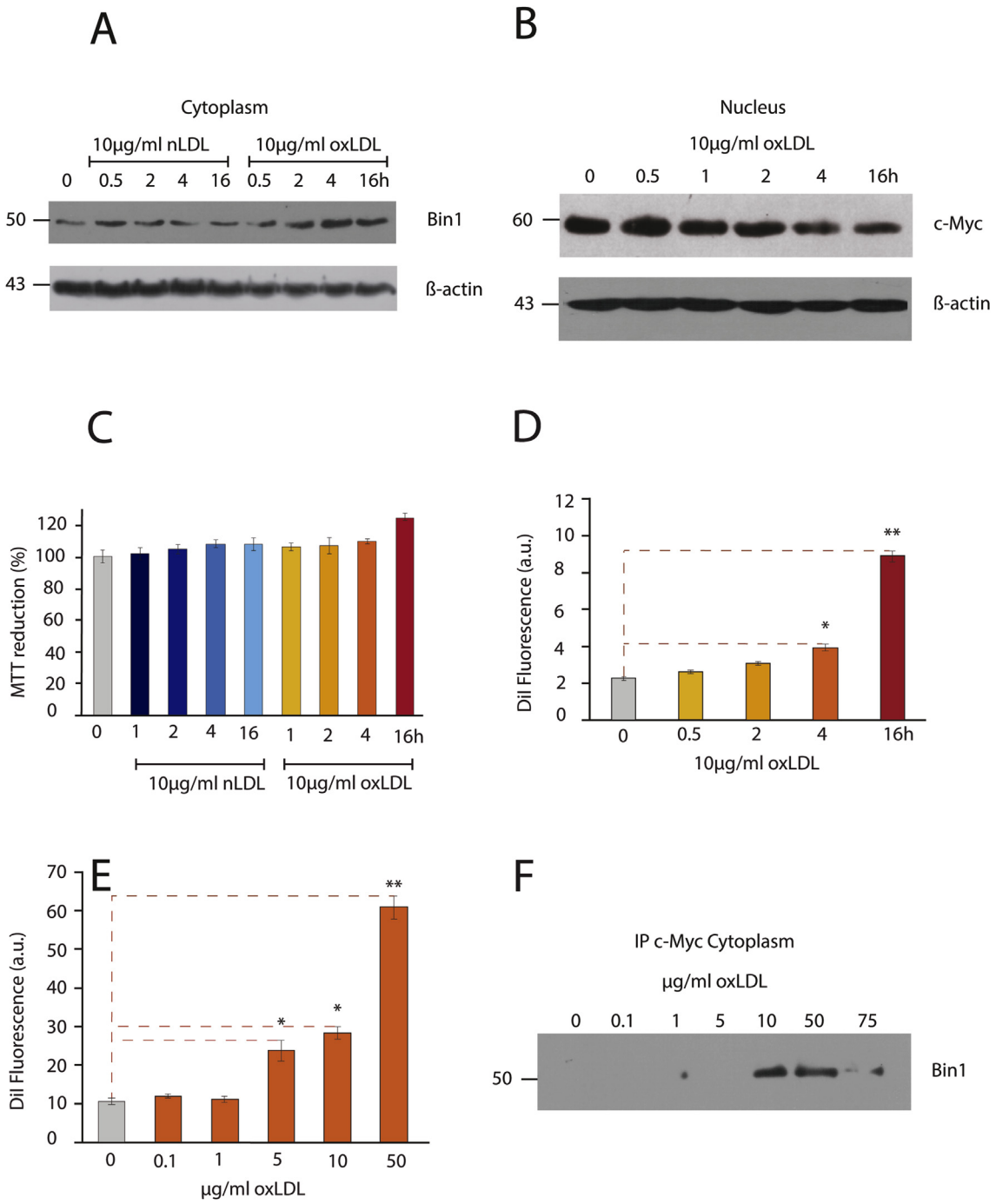


Fig. 1. Analysis of LDL internalization by native RAW cells. (A) Immunodetection of Bin1 performed during internalization of 10 µg/mL of nLDL or oxLDL by native RAW cells carried out for 16 h. (B) Immunodetection of nuclear cMyc performed during internalization of 10 µg/mL of oxLDL by native RAW cells carried out for 16 h. (C) MTT assays performed to examine cell viability following the internalization of nLDL or oxLDL (n = 3). (D) Internalization of DiL-oxLDL (10 µg/mL) during a period of 16 h (n = 3). (E) Internalization of DiI-oxLDL following exposure to increasing concentrations of oxLDL (n = 3). (F) c-Myc/Bin1 immunoprecipitation (representative assay) from the cytoplasmic fraction of RAW cells internalizing increasing concentrations of oxLDL.

clones pcBin1-6/F7, pcBin1-7/F7 and pcBin1-10/F7, show a lower rate of internalization. Considering that one of the interactions mediated by the SH3 domain of amphiphysin 2 involves dynamin [32], a protein responsible for endocytic vesicle neck cleavage [33], our results also suggest that an interaction between amphiphysin 2 and other endocytosis-effecting proteins via the SH3 domain is also a key point to properly carry out the endocytic process.

To our knowledge, this is the first study describing an amphiphysin 2 isoform (pcBin1-10/F7) that contains a BAR domain lacking exon 6 that nevertheless is able to perform endocytosis in a comparable level to that shown by control *LacZ* transfected cells (Fig. 3A and B). These results confirm the importance of the region flanked between helices encoded by exon 6 in the BAR domain that seem to provide a key connection between the membrane and the actin polymerization machinery.

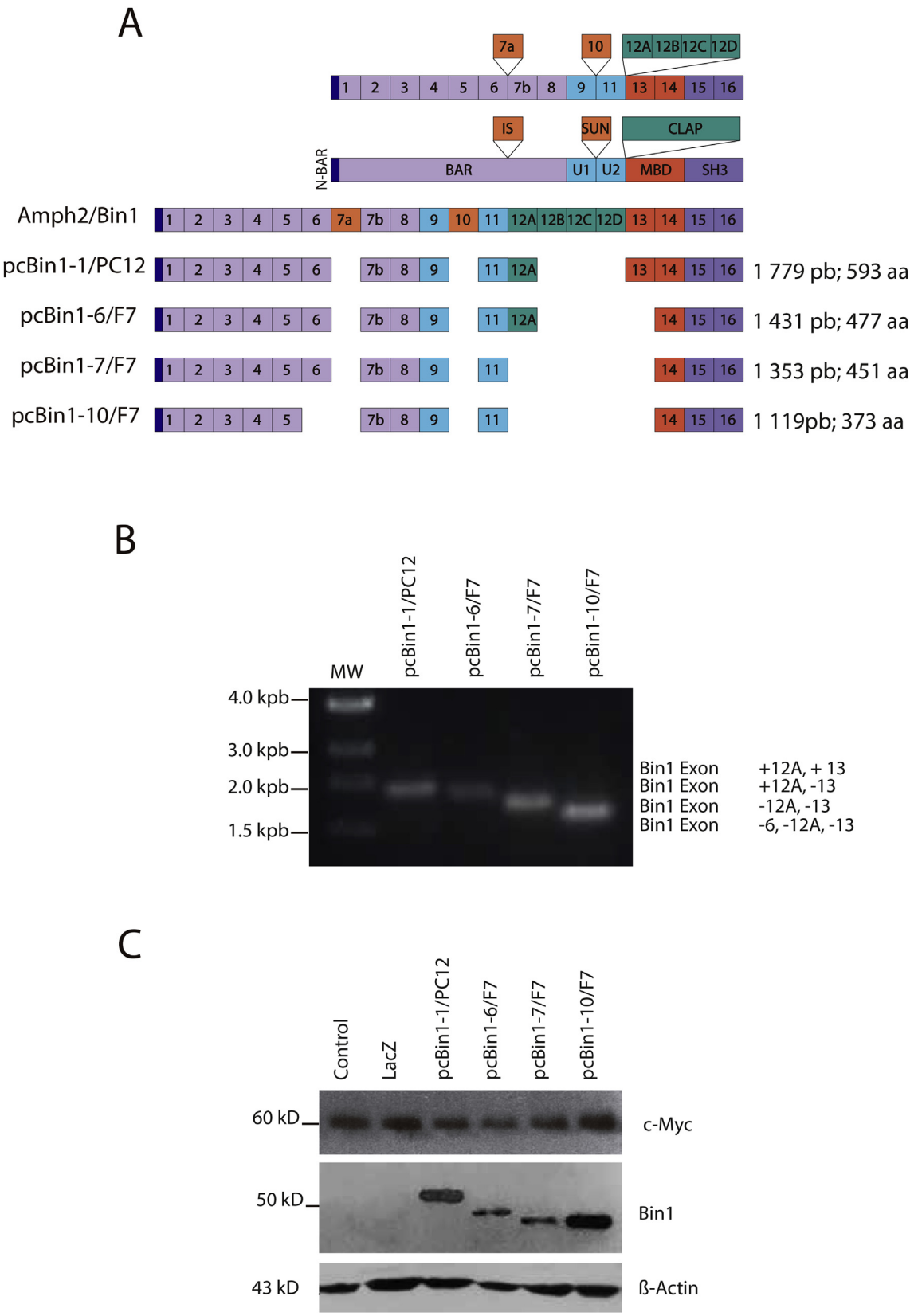
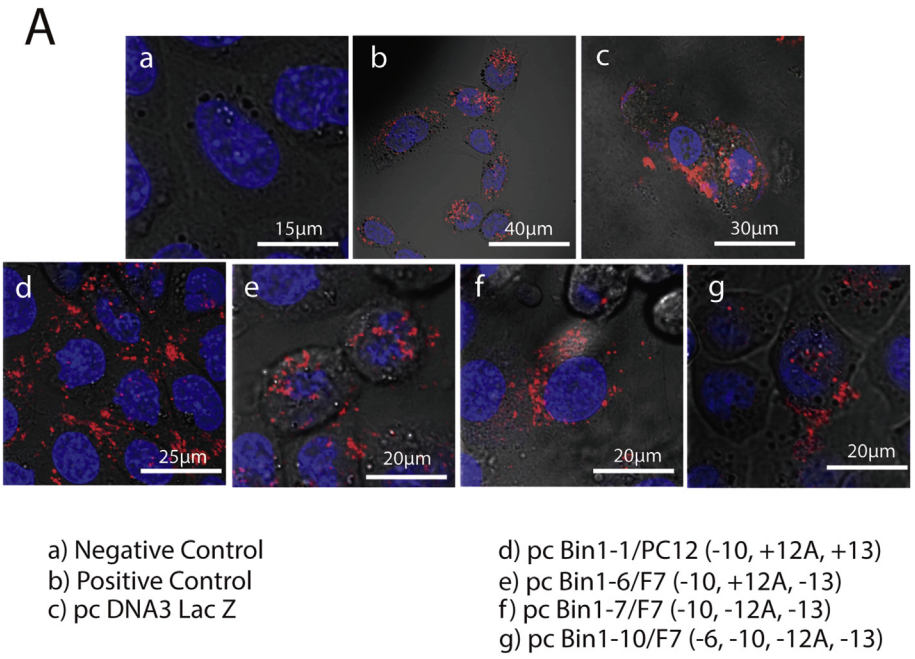


Fig. 2. Amphiphysin 2/Bin1 gene exon structure and corresponding codified domains. (A) Amphiphysin 2 gene comprises 19 exons that can be edited post-transcriptionally or by splicing as indicated. (B) Inserts cloned in vector pcDNA 3.1 V5 His TOPO and over-expressed in PC12 and CHO-F7 cells. (C) Amphiphysin 2 and c-Myc expression detected in CHO-F7 cells lysates collected 16 h after transfection with each amphiphysin 2/Bin1 clone (1 μg/lane). Two negative controls were included: non-transfected CHO-F7 cells and cells transfected with LacZ (without amphiphysin 2).



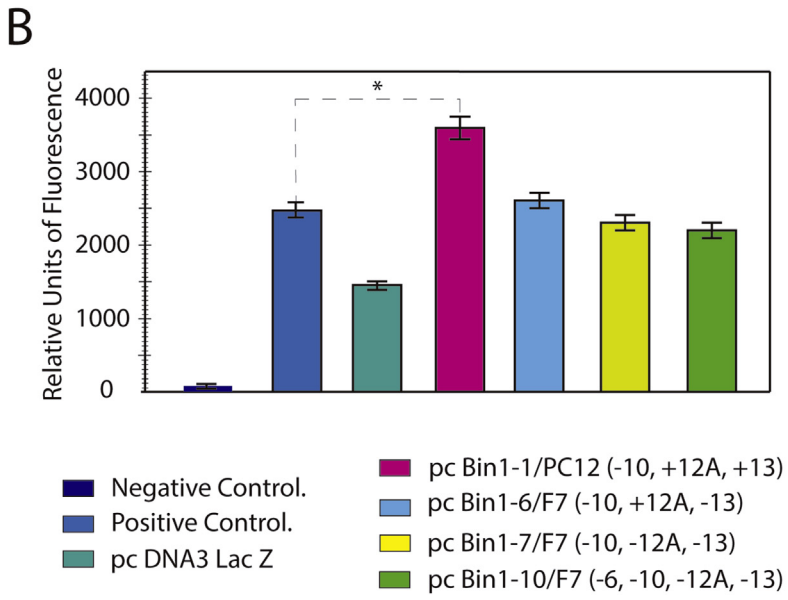


Fig. 3. Confocal microscopy of CHO-F7 cells internalizing DiI-acLDL. (A) DiI-acLDL internalization by CHO-F7 cells: (a) Native CHO-F7 cells (negative control); (b) DiI-acLDL basal internalization by native CHO-F7 cells (positive control); (c) DiI-acLDL internalization by LacZ CHO-F7 transfected cells; (d) DiI-acLDL internalization by clone pBin1-1/PC12; (e) Clone pBin1-6/F7; (f) Clone pBin1-7/F7; (g) Clone pBin1-10/F7. (B) Fluorometric quantitation of DiI-acLDL internalization carried out by the same clones.

4. Discussion

This study represents an effort to elucidate the role of Bin1/c-Myc interactions associated to oxidative stress and its repercussions on cell proliferation in cells known to be importantly involved in the process of atherogenesis. Our results showing a clear association between Bin1 and c-Myc secondary to oxidative stress induced by internalization of chemically modified LDL particles, seem to modulate the progression of the cell cycle and therefore constitute a cell survival mechanism under stressful conditions.

The observation that cells transfected with isoform Bin1-PC12 exhibit a high endocytic activity suggest that this isoform actively participate in the control of the cell cycle, activating proliferation possibly by interacting with c-Myc via the MBD domain. Interactions involving the SH3 domain can be ruled out since isoform Bin1-1/PC12 includes exon 12A (Fig. 2), able to inhibit interactions between c-Myc and Bin1 via auto-interactions with the SH3 domain [34].

Here we report a novel clone of amphiphysin 2 that lacks exon 6 (pcBin1-10/F7) known to encode part of helix 2 and the linking loop with helix 3 of the BAR domain (Fig. S2). *In vitro* experiments performed with the BAR-domain a region rich in positive charges, revealed its importance for function since mutation of these residues resulted in the incapability of this segment to bind to negatively charged lipids contained in liposomes. Clone pcBin1-6/F7 that

expresses exon 12A but lacks exon 13, reduces its participation in endocytosis compared to clone pcBin1-1/PC12 but shows no differences with respect to the control. It is known that BAR-domain-containing constructs without the dynamin-binding domain (the SH3 domain) can stimulate the GTPase activity of dynamin in the presence of liposomes [2]. Although clone pcBin1-10/F7 expresses high levels of Bin1 (Fig. 3C), it did not internalize Dil-acLDL as efficiently as clone pcBin1-1/PC12. Most probably, the lack of exons 6, 12A and 13 make this clone incapable of recruiting dynamin in the neck of the endocytic vesicle therefore hindering internalization [2].

Since the mechanisms by which c-Myc expression is modulated under conditions of oxidative stress are still not well known and following previous studies from our laboratory [35], here we studied a potential way to control cell survival through c-Myc binding to partner molecules. In this particular situation, amphiphysin seems to be a key molecule participating in the internalization of chemically modified LDL particles. We demonstrate that under oxidative stress given by the internalization of oxLDL particles, cells carry out the formation of an amphiphysin 2/c-Myc complex intended as a control mechanism to initially avoid the process of apoptosis in an attempt to prolong cell survival.

Considering classic studies showing c-Myc as an important player not only in the process of proliferation during oxidative stress in atherosclerosis, but also in the development of neoplasia [36], further investigation is needed in order to fully understand how alterations in the many signaling pathways carried out by c-Myc achieved through complexation with partner proteins, could help to explain the pathophysiology of these diseases.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.bbrc.2018.09.121.

Transparency document

Transparency document related to this article can be found online at https://doi.org/10.1016/j.bbrc.2018.09.121.

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