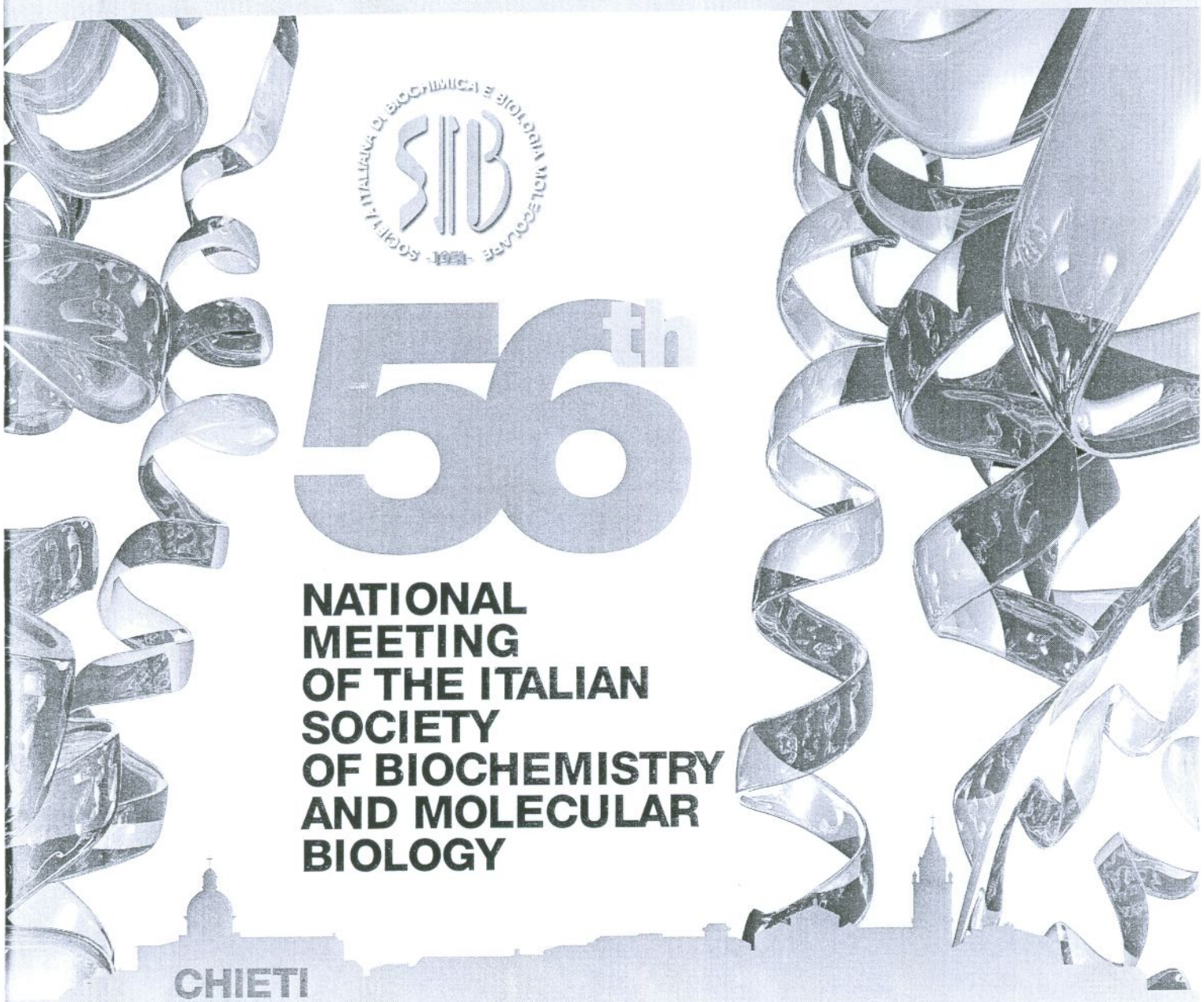

56th NATIONAL MEETING OF THE ITALIAN SOCIETY OF BIOCHEMISTRY AND MOLECULAR BIOLOGY

CHIETI
26th-29th SEPTEMBER 2012
Venue: New Rectorate Auditorium

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A comparison between the role of SPARC in osteogenic differentiation of mesenchymal stem cells and in WIN/TRAIL-induced apoptosis in osteosarcoma cells

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Mesenchymal stem cells are well known to possess multipotential differentiative ability and represent a good tool for clinical research.
In our study, after induction of osteogenic differentiation of bone marrow mesenchymal stem cells (BM-MSC) by using conditioned media, we focused our attention on SPARC, a regulatory protein which affects cell differentiation, proliferation, and survival. During osteogenic differentiation SPARC levels raised in time-dependent manner and were functional to the increase of other osteogenic markers, such as osterix and osteopontin. The effects of SPARC downregulation were investigated through the use of a specific small-interfering RNA, demonstrating a reduction in the levels of osteogenic markers in siSPARC cells.
Since SPARC, besides to its important role in osteogenic differentiation, can modulate cell-cell and cell-matrix interactions and act either as a tumor suppressor or a tumor promoter, we were interested to investigate its levels in osteosarcoma MG63 cells, a malignant osteoblastic-like cell line. In these cells the basal levels of SPARC resulted very high.
It is well known that MG63 cells are resistant to TRAIL (tumor necrosis factor apoptosis inducing ligand) although they can be sensitised to TRAIL-induced apoptosis by different compounds. Treatment of these cells with a combination of the synthetic cannabinoid WIN and TRAIL induced marked cytotoxic effects. Since treatment with WIN/TRAIL combination further enhanced the levels of SPARC, we sought to evaluate the possible role of SPARC in WIN/TRAIL-mediated apoptotic pathway. To this purpose, we induced SPARC down-regulation by means of gene silencing and observed a decrease in the apoptotic features induced by the combined treatment.
In conclusion, in osteosarcoma cells SPARC seems to behave like a tumor suppressor protein which is able to actively participate to apoptotic cell death induced by WIN/TRAIL combined treatment.