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BCL-2 Family Proteins Hints for Oxford; containing ... Hints to Freshmen ... On studies and reading for the Schools, etc. [By John Campbell.] "A Monument of Christian Munificence;" Or, An Account of the Brotherhood of the Holy Cross, and of the Hospital of Christ of Abingdon The Christian Precept 'Whatsoever Ye Would that Men Should Do to You, Do Ye Even So to Them' Not Applicable to the Irish Church Question BCL?2 Protein Family Some Account of the Life and Character of the Late Very Rev. Thomas Garnier, B.C.L., Dean of Lincoln; to which is Subjoined a Sermon, Being the Last He Delivered from the Cathedral Pulpit ... Memory, and other poems Four Pills for Social Ills. An Address The Life and Speeches of the Very Reverend J. H. Cotton, B.C.L., Dean of Bangor, and Rector of Llanllechyd Molecular Mechanisms of Programmed Cell Death The Parker Society...: Works of John Philpot, B.C.L, archdeacon of Winchester Numerical Control BCL Standards Association The Examinations and Writings of John Philpot, B.C.L. Archdeacon of Winchester, Martyr, 1555 Bcl-2 Conformational Change as an Indicator of Chemotherapy Response Divergent Phenotypes Induced by Expression of BCL-XS The Church: the True Mutual Aid Society. A Sermon [on Galat. Vi. 8], Etc Genotypic and Phenotypic Characteristics of P53, HER/neu and Bcl-2 in Chinese Primary Breast Cancer The Role of the BCL-X Protein in Wound Neovascularization Role of Bcl-2 Family Proteins in Mitochondrial Pathway of Apoptosis Programmed Cell Death A Guide to Qualitative Organic Chemical Analysis Report on the Leaching of Substances from Packaging Materials Into Food Structural Biology of Antiapoptotic Proteins of BCL-2 Family Apoptotic Pathways as Targets for Novel Therapies in Cancer and Other Diseases Tonbridge Castle to the Year 1322: a Paper Read Before the Kent Archaeological Society on July 28, 1865 A Pastoral Address to the Members of the Congregation of St. Paul's Church, Leeds An Unfinished Revolution? Mechanism of Regulation of Apoptosis by the Bcl-2 Proteins Bcl-XL and Bcl-XS Bcl-xL Deamidation in Oncogenic Tyrosine Kinase Signalling Observations "Salvation, Now." More Plain Words from Their Absent Minister, to the Parishioners of Shirley, Derbyshire Bcl-2 Regulates Chondrocyte Phenotype Through MEK-ERK1/2 Pathway Characterization of Pro- and Anti-apoptotic BCL-2 Proteins Regulating Leukemogenesis in Juvenile Myelomonocytic Leukemia (JMML) Bcl-xL as Prognostic Marker and Potential Therapeutic Target in Cholangiocarcinoma Woolwich and Sandhurst entrance examinations The Roles of Elevated Bcl-2 in Ovarian Cancer Notes on the History and Antiquities of Liverpool Bcl-2 Family Members Regulate the Sensitivity to 2-deoxy-D-glucose in Lymphomas Bcl-2 Expression in Prostatic Adenocarcinoma Notes on the History and Antiquities of Liverpool ... Third Impression, with a New Appendix. [With Plates.].

Prostate cancer is one of the most common cancers among men and is the leading cause of cancer deaths. As a result of effective PSA screening programmes in the West, most patients are diagnosed at an early favorable stage. Unfortunately, such screening programmes are not popular in India. Majority of the patients with prostatic carcinoma present in an advanced stage in our country, when treatment options are limited. Bcl-2, one of the targets for therapy, is an anti-apoptotic mediator that has been found to be involved in the molecular biology of a wide range of human cancers including prostatic cancer. Overexpression of this protein was found to be associated with progression of prostate cancer to a metastatic and hormone insensitive state characterized by poor responses to various forms of therapy. Hence, therapy against this anti-apoptotic protein could provide a treatment option for such patients. This study was undertaken to highlight the possible role of Bcl-2 in the pathogenesis of prostatic carcinoma and its progression with the hope of developing targeted therapy to combat this disease. Programmed cell death, which is termed as apoptosis, is essential for normal development and maintenance of tissue homeostasis in multicellular organisms. Aberrant regulation of this pathway is linked to multiple human diseases, including cancer, autoimmunity, neurodegenerative disorders and diabetes. Apoptosis is pursued by two pathways; extrinsic and intrinsic pathway. The intrinsic pathway of apoptosis mainly relies on mitochondria where Bcl-2 family proteins serve as the master regulators. Mitochondria mainly execute their function through mitochondrial outer membrane permeabilization (MOMP). MOMP leads to the release of several apoptogenic factors from mitochondrial intermembrane space, such as cytochrome c and Smac/Diablo, into the cytosol that activate downstream caspase and promote cell death. Bcl-2 family proteins play key role by regulating MOMP. The complex interaction among pro-and anti-apoptotic Bcl-2 family members determine the possibility of MOMP and thereby determine the cellular commitment to apoptosis. This review focuses on the mitochondrial pathway of apoptosis, mechanism of MOMP and its regulation by Bcl-2 family proteins. Bcl-2 family members are important regulators of apoptosis, and their tampered expression is often involved in oncogenesis. Of particular importance are the levels of Bcl-2 family members in forming lymphomas. We studied two groups of murine thymic T cell lymphomas derived from either Bcl-2 or Bax overexpression in order to predict their sensitivity and resistance to treatments. While the growth rate and histological characteristics were similar for both lymphoma groups, Bax-derived lymphomas failed to undergo cell cycle arrest following radiation treatment and had frequent p53 mutations. In contrast, Bcl-2-derived lymphomas often halted proliferation following radiation delivery and rarely had p53 mutations. Bax-derived lymphomas were uniformly sensitive to treatment with 2-deoxy-D-glucose (2DG) while all Bcl-2-derived lymphomas were resistant. This led us to hypothesize that the Bcl-2 family is involved in 2DG-induced cell death. Focusing on the mechanism of 2DG toxicity in Bax-derived lymphomas, our studies demonstrate the following: cell death involved the activation of proapoptotic Bax, was effectively blocked by anti-apoptotic Bcl-2, and was mediated, at least in part, by the BH3-only family member Bim. Based on these results, we explored whether a BH3 mimetic (ABT-737) could sensitize lymphomas to 2DG killing. Indeed, a combination of ABT-737 with 2DG enhanced killing in Bax-derived lymphomas and resensitized Bcl-2-overexpressing lymphomas to 2DG. Since both 2DG and BH3 mimetics are currently in clinical trials, understanding their killing mechanisms and optimal combinations are of potential clinical significance. The work in this dissertation demonstrates a novel role of Bcl-2 family member proteins in regulating 2DG toxicity and may predict response to 2DG treatment. The information found presents a new strategy of combining 2DG with BH3 mimetics to improve existing lymphoma therapies. Most anticancer agents ultimately kill cancer cells primarily by inducing apoptosis a programmed cell death. We propose that Bcl-2 undergoes a conformational change in response to chemotherapeutic agents in breast cancer cells in vitro and in vivo and that a Bcl-2-conformation-sensitive antibody can be used to better predict and evaluate the responses of breast cancer cells to therapies. We have characterized an epitope-specific anti-Bcl-2 antibody that specific recognizes pro-apoptotic Bcl-2 conformation. By using this antibody, we show that a number of chemotherapeutic agents, including paclitaxel, retinoid-related molecules, and nonsteroidal anti-inflammatory drugs, induce breast cancer cell apoptosis by modulating Bcl-2 conformation. Induction of Bcl-2 conformational change involves Bcl-2 phosphorylation. Moreover, apoptosis induction of breast tumor grown in animal by a Nur77-derived peptide correlates with Bcl-2 conformational change. Together, we have developed a novel epitope-specific anti-Bcl-2 antibody that can be used to predict and evaluate the response of breast cancer cells to certain chemotherapeutic agents in vitro and in animal. The 2002 Nobel Prize in Physiology or Medicine was awarded to Sydney Brenner, H. Robert Horvitz, and John E. Sulston for their seminal discoveries concerning "genetic regulation of organ development and programmed cell death." This clearly marked the prime importance of understanding the molecular mechanisms controlling cell death. The 1 st International Symposium on Programmed Cell Death was held in the Shanghai Science Center of the Chinese Academy of Sciences on September 8-12, 1996. A number of key issues in apoptosis were discussed at the meeting, and progress in major areas of apopto sis research was summarized by expert participants at the meeting and published by Plenum Publishing Corporation as a book entitled Programmed Cell Death. In the last six years, we have witnessed a real explosion in our knowledge on how cells undergo apoptosis, thereby participating in various developmental and pathophysiological processes. At this ever exciting time, we organized the 2nd International Symposium on Programmed Cell Death. I have been interested in the molecular mechanisms of Haematopoietic malignant diseases such as leukaemia and lymphoma, especially those involving oncogenic tyrosine kinases. About 30 of the 90 tyrosine kinases in the human genome have been implicated in cancer (Blume-Jensen P, 2001). The oncogenic tyrosine kinases (OTKs), such as Bcr-Abl (product of chromosomal translocations of two genes bcr and abl) in Chronic Myelogenous Leukaemia, and Erythroblastic leukaemia viral oncogene homolog 2(Erb-B2) in mammary and other cancers, mediate their transforming effects via a diverse array of signalling pathways involved in DNA damage, cell survival and cell cycle regulation (Deutsch E, 2001; Skorski T, 2002; Kumar R, 1996). My work has been centred around the analysis of a mouse cancer model that is driven by an oncogenic tyrosine kinase - p56 Lck-F505 expressed on CD45 knock- out background (Baker M, 2000). The investigation of this mouse model has revealed that oncogenic inhibition of deamidation of the Bcl-xL survival protein plays a critical role in protecting thymocytes from DNA-damage induced apoptosis. Cells that would normally be eliminated due to accumulating DNA damage are instead preserved with an increasing load of double-stranded breaks, leading to genomic instability, chromosomal abnormalities and transformation. This work was published in Cancer Cell (An oncogenic tyrosine kinase inhibits DNA repair and DNA-damage-induced BclxL deamidation in T cell transformation. Zhao R, 2004). Following that I have tried to elucidate the different roles of the two deamidated species of Bcl-xL in apoptosis, and also the molecular mechanisms of DNA damage- induced Bcl-xL deamidation in order to understand the inhibition of Bcl-xL deamidation by oncogenic tyrosine kinases. Recently I have shown that Bcl-xL deamidation, whereby two critical Asn residues are converted to iso-Asp, cripples the ability of the protein to sequester pro-apoptotic BH3-only proteins such as Bim and p53-upregulated modulator of apoptosis (PUMA), thereby explaining its loss of pro-survival functionality. In vivo, DNA damage causes intracellular alkalinisation that is both necessary and sufficient to deamidate Bcl-xL, promoting apoptosis: no enzyme is necessary for this process. In pre-tumourigenic thymocytes alkalinisation is blocked, so preserving Bcl-xL in its pro-survival mode. Furthermore murine tumours are protected from genotoxic attack by native Bcl-xL, but enforced alkalinisation and consequent Bcl-xL deamidation promotes apoptosis. This part of work was published in Plos Biology (DNA damage-induced Bcl-xL deamidation is mediated by NHE-1 antiport regulated intracellular pH. Zhao R, 2007). Through collaboration with Prof AR Green's research group at the Department of Haematology of the University of Cambridge, I have also analysed the Bcl-xL deamidation pathway in human myeloproliferative disorders, e.g. Polycythemia vera(PV) and Chronic Myelogenous Leukaemia (CML). We found that the oncogenic tyrosine kinases involved in these disorders, i.e. Jak2V617F and Bcr-Abl also inhibit the Bcl-xL deamidation pathway in DNA damage responses. These findings shed light on potential therapeutic application of the Bcl-xL deamidation pathway in human malignancies. This piece of work was recently published in the New England Journal of Medicine (Inhibition of the Bcl-xL deamidation pathway in myeloproliferative disorders. Zhao R, 2008). Overall the cited work has led to several important new insights into the molecular mechanisms involved in oncogenesis: first, that Bcl-xL deamidation is important in the cascade of events leading from DNA damage to apoptosis; second, that oncogenic tyrosine kinases inhibit these events in both the murine and human context; third, that up-regulation of the NHE-1 antiport and consequent intracellular alkalinisation are critical events in this DNA damage-induced cascade leading to apoptosis. In the process I have demonstrated the first in vivo mechanism for the deamidation of an internal protein Asn. Essentially, a completely new and unexpected signalling pathway has been uncovered that seems to pertain to all murine and human haematopoietic cell lineages that have been investigated so far. Abstract: Intrahepatic, perihilar, and distal cholangiocarcinoma (iCCA, pCCA, dCCA) are highly malignant tumours with increasing mortality rates due to therapy resistances. Among the mechanisms mediating resistance, overexpression of anti-apoptotic Bcl-2 proteins (Bcl-2, Bcl-xL, Mcl-1) is particularly important. In this study, we investigated whether antiapoptotic protein patterns are prognostically relevant and potential therapeutic targets in CCA. Bcl-2 proteins were analysed in a pan-cancer cohort from the NCT/DKFZ/DKTK MASTER registry trial (n = 1140, CCA n = 72) via RNA-sequencing and transcriptome-based protein activity interference revealing high ranks of CCA for Bcl-xL and Mcl-1. Expression of Bcl-xL, Mcl-1, and Bcl-2 was assessed in human CCA tissue and cell lines compared with cholangiocytes by immunohistochemistry, immunoblotting, and quantitative-RT-PCR. Immunohistochemistry confirmed the upregulation of Bcl-xL and Mcl-1 in iCCA tissues. Cell death of CCA cell lines upon treatment with specific small molecule inhibitors of Bcl-xL (Wehi-539), of Mcl-1 (S63845), and Bcl-2 (ABT-199), either alone, in combination with each other or together with chemotherapeutics was assessed by flow cytometry. Targeting Bcl-xL induced cell death and augmented the effect of chemotherapy in CCA cells. Combined inhibition of Bcl-xL and Mcl-1 led to a synergistic increase in cell death in CCA cell lines. Correlation between Bcl-2 protein expression and survival was analysed within three independent patient cohorts from cancer centers in Germany comprising 656 CCA cases indicating a prognostic value of Bcl-xL in CCA depending on the CCA subtype. Collectively, these observations identify Bcl-xL as a key protein in cell death resistance of CCA and may pave the way for clinical application LPA also increases secreted levels of Bcl-2. In vitro human umbilical vein endothelial cell (HUVEC) tube formation assays show that OC-derived Bcl-2 or recombinant human (rh) Bcl-2 promotes aberrant formation of tube-like structures. Though extracellular Bcl-2 does not affect HUVEC cell viability, it may cause aberrant tube formation by inhibiting HUVEC migration. Finally, Bcl-2 ELISA reveals that urinary Bcl-2 levels in OC patients are higher than those in normal individuals and patients with benign gynecologic disease. Urinary Bcl-2 also complements serum CA125 when the two are compared in parallel samples. Furthermore, urinary Bcl-2 decreases following cytoreductive surgery. Altogether, the results suggest that Bcl-2 is important in OC tumorigenesis and angiogenesis. Additionally, urinary Bcl-2 may be a valuable non-invasive biomarker for OC diagnosis and/or screening. Consequently, further elucidation of mechanisms of Bcl-2 overexpression and its extra-apoptotic functions could lead to improved treatment and diagnostic strategies for OC patients. Apoptosis is very critical for the maintenance of homeostasis, tissue differentiation, and removal of damaged/infected cells, which serves as a defense strategy against emergence of diseases such as cancer. Dysregulation of apoptotic pathways have been implicated in many diseases such as cancer and neurodegeneration. Therefore regulation of apoptosis to maintain a balance between apoptosis and cell proliferation is critical for the well being of all living organisms. The Bcl-2 family of proteins is one of the major regulators of apoptotic pathways. They consist of the anti-apoptotic subgroup, including Bcl-XL, and proapoptotic subgroup, including Bcl-XS. Previous studies have shown that mouse embryos that are deficient in the Bcl-X gene, a gene

producing both Bcl-XL and Bcl-XS proteins, die before birth. Both Bcl-XL and Bcl-XS have been found to regulate cellular responses to apoptotic stimuli. The exact molecular and biochemical mechanism still remains unclear and controversial. We therefore hypothesize that Bcl-XL and Bcl-XS regulate apoptosis by differentially interacting with other members of the Bcl-2 protein family . Specific aims to test this hypothesis include (1) Investigate how endogenous Bcl-XL regulates apoptosis, and (2) Determine the molecular mechanisms by which Bcl-XS regulates apoptosis. To investigate how endogenous Bcl-XL regulates apoptosis, we developed mouse embryonic fibroblasts (MEFs) with (wild-type, WT) or without Bcl-X gene expression (Bd-X-KO). Both WT and Bcl-X-KO MEFs were treated with various apoptotic stimuli, including chemotherapeutic drugs and a panel of BH3-only Bcl-2 proteins, and cell viability was measured over time. Preliminary results showed that Bcl-X-KO MEFs were more sensitive to chemotherapeutic drugs than WT MEFs indicating that endogenous Bcl-XL plays a more prominent role in modulating cellular responses to these chemotherapeutic drugs. Furthermore, based on their effects on the viability of Bcl-X-KO and WT MEFs, the BH3-only Bcl-2 proteins were categorized into three groups: (a) neither Bcl-XKO nor WT MEFs underwent apoptosis upon Bad, Bnip3, Nix, Bim, or Bik treatment, implicating that these proteins do not directly interact with Bcl-XL, (b) both Bcl-X-KO and WT MEFs died at the same rate upon Tbid, Bmf, Hrk, or Puma treatment, showing that Bcl-XL was not able to prevent cell death initiated by these proteins, and (c) Noxa treatment induced significant cell death in Bcl-XKO but not in WT MEFs at all, indicating that endogenous Bcl-XL is able to completely inhibit the pro-apoptotic activities of Noxa. Therefore, endogenous Bcl-XL mediates cellular responses to BH3-only proteins expression by differentially antagonizing different BH3-only Bcl-2 proteins. Future study will involve identifying BH3-only pro-apoptotic Bcl-2 proteins that interact with Bcl-XL and characterizing the interaction between endogenous Bcl-XL and the BH3-only Bcl-2 protein(s). To determine the molecular mechanism by which Bcl-XS regulates apoptosis, Bcl-XS was re-expressed in both WT and Bcl-X-KO MEFs at a level similar to endogenous Bcl-XL level. All MEFs were treated with different apoptotic stimuli and cell viability was measured. Preliminary results showed that Bcl-XS expression had no effect on cell viability upon vincristine, etoposide, or doxorubicin treatment, but upon actinomycin D treatment, Bcl-XS protected cells from apoptosis. Future study involves examining the effects of Bcl-XS on apoptosis initiated by the different BH3-only Bcl-2 proteins, the activities of Bcl- XS in vitro, and the role of the intracellular localization of Bcl-XS during apoptosis. This volume explores numerous techniques for the genetic, molecular, biochemical, and structural examination of BCL-2 family proteins and their interactions. The chapters in this book cover topics such as the relevance of BCL-2 proteins in health and disease; evaluating cellular dependencies to specific BCL-2 family proteins; flow-cytometry-based methods for measuring BCL-2 proteins and mitochondrial-based cell death; measuring activity and interactions of BCL-2 family proteins in the presence of mitochondria, artificial membranes or yeast; conformational activation and oligomerization of pro-apoptotic proteins BAX and BAK leading to cytochrome c release and apoptosis; structural and biophysical studies in solution and lipid vesicles using nuclear magnetic resonance, cryo-electron microscopy, fluorescence microscopy and electron paramagnetic resonance. Written in the highly successful Methods in Molecular Biology series format, chapters include introductions to their respective topics, lists of the necessary materials and reagents, step-by-step, readily reproducible laboratory protocols, and tips on troubleshooting and avoiding known pitfalls. Cutting-edge and thorough, BCL-2 Family Proteins: Methods and Protocols is a valuable resource to inspire and encourage novice and established scientists to further their research and make new discoveries in this exciting field. As our understanding of apoptotic pathway expands, we are coming to realize the great potential of utilizing this pathway to treat diseases such as cancer. The book attempts to review, summarize, and speculate on the apoptotic pathways, how are they regulated and how targeted therapies are being used to treat a wide variety of diseases. Special emphasis is placed on cancer since new treatments either being developed or currently in the clinical setting are showing great promise to increase survival rates for cancer patients. Chapters will address the biology behind regulating the apoptotic pathways and what goes wrong in disease states whereas other chapters will concentrate on new therapies targeting apoptotic pathways. The reader by the end of the book should have greater insight into the understanding and utilization of apoptotic pathways to fight diseases such as cancer. In this book, scientists pioneering the field have compiled a series of focused chapters to highlight the relevance of the BCL?2 family of proteins in apoptosis, physiology and disease. An important focus of this volume is considering the potential TH ERA PEUT IC benefits of targeting apoptosis pathways in the context of human disease. Readers interested in understanding how a cell handles stress and the consequences of dysregulation of this process for human disease will find this book very valuable. It attempts to describe a fascinating area of research where physiology and biomedicine converge at different levels, revealing a trip from the molecular regulation of apoptosis to the impact of this process to the physiology of a whole organism. The BCL-2 family proteins are crucial regulators of the apoptotic process. This book covers recent structure-based insights into the mechanisms of action of antiapoptotic BCL-2 family proteins as well as into the interactions between antiapoptotic proteins and their ligands. While the main focus is on the apoptotic processes in mammals, the great part of the book is devoted to viral homologues of Bcl-2 family antiapoptotic proteins. Bcl-2 is an anti-apoptotic protein that has recently been shown to regulate other cellular functions. We previously reported the novel function of Bcl-2 that regulates chondrocyte matrix gene expression, independent of its anti-apoptotic function. The first hypothesis was that Bcl-2 regulates chondrocyte phenotype through the specific pathways. The role of three intracellular signaling pathways likely to be associated with Bcl-2 function, namely, NFkappaB, PKCalpha, and ERK1/2, was examined. The NFkappaB and PKCalpha signaling pathways were not involved in Bcl-2 regulated matrix expression, even thought these are known to regulate Sox9. The ERK1/2 signaling pathway was activated in Bcl-2 deficient cells that lost the chondrocyte phenotype by decreasing chondrocyte matrix protein expression and increasing fibroblastic collagen expression. The inhibition of phopspho-ERK1/2 reversed cells to have chondrocyte phenotype. Moreover, the MEK-ERK1/2 pathway limits the gene expression of matrix protein in wild type chondrocytes. These data indicate that Bcl-2 regulates chondrocyte phenotype through the MEK-ERK1/2 pathway. The second hypothesis was that Bcl-2 regulates chondrocyte phenotype in vitro as well as in vivo specifically, in human osteoarthritis. Osteoarthritis (OA) is an age-related degenerative cartilage disease and is known that the chondrocyte phenotype is altered. However, the significance of the altered phenotype in OA is unclear due to the use of non-age match samples, different source of samples, and lack of precise determination of the stage of OA progression. We developed an intrajoint comparison model using human OA samples to control for patient age and genetic background effects. The advanced OA cartilage was taken from within 1cm of overt lesions. In contrast, minimal OA cartilage was taken from areas without any obvious lesions. The chondrocyte matrix protein and Bcl-2 mRNA expression was decreased in advanced OA cartilage compared with minimal OA cartilage in most of patients studied. In contrast, osteopontin mRNA expression was up-regulated in advanced OA cartilage compared with minimal OA cartilage. A correlation was observed between the steady state mRNA coding for aggrecan and Bcl-2, and Bcl-2 and Sox9. These results support the hypothesis that Bcl-2 regulates chondrocyte phenotype in vivo as well as in vitro. Please note that the content of this book primarily consists of articles available from Wikipedia or other free sources online. Pages: 54. Chapters: Apoptosis, Cytochrome c, P53, NF- B, Bcl-2, DNA damage theory of aging, Apoptosome, XIAP, ASK1, FADD, Bcl-2-associated death promoter, IKK2, Fas receptor, Fas ligand, Bcl-2-associated X protein, Autophagy, APAF1, Neurotrophin, CARD domain, Poly ADP ribose polymerase, BH3 interacting domain death agonist, Caspase, History and highlights in apoptosis research, Noxa, TrkC receptor, Perforin, PAC-1, Apoptosis-inducing factor, The Proteolysis Map, Paracaspase, 14-3-3 protein, Mitochondrial apoptosis-induced channel, Anoikis, Bleb, TUNEL assay, Autolysis, Granzyme, Necrobiology, Metacaspase, DNA laddering, Phenoptosis, P53 upregulated modulator of apoptosis, Pyknosis, Karyolysis, Autophagy database, Autoschizis, Pyroptosis, Suicide gene, Bcl-2 homologous antagonist killer, Karyorrhexis, Mitotic catastrophe, Death-inducing signaling complex, UVB-induced apoptosis.