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Radiation Therapy*



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BIOGRAPHY

He is now an instructor in Department of Radiation Oncology, Beth Israel Deaconess Medical Center (BIDMC) /Harvard Medical School. He completed his PhD program in Cellular and Developmental biology in 2006 at University of Fribourg, Switzerland, and identified the differentially expressed downstream targets of human nucleosome remodelling Mi-2 orthologue LET-418 and explored the influence of TOR kinase on lifespan in *C. elegans* using genetics. Then he carried out his postdoctoral training at Institute of Ageing and Pittsburgh Institute of neurodegenerative diseases, University of Pittsburgh. In 2008, he moved to Dept of Radiation Oncology/BIDMC first as research fellow and was promoted to current position in August, 2010. He have authored and coauthored 15 publications in journals, such as Nature, Developmental biology, BMC developmental biology, PLOS Genetics, PLOS one, J. Vis Exp, Cancer informatics, Gene regulation and Systems biology. He served as a reviewer in some journals, including Cellular and Molecular Life Science, Transgenic Research, Knowledge-based Systems, and the La –Press journals.

Recent Publications

Cancers with Stem-Like Attractors and “Loss Of Differentiation” Novel Hallmark: Does the “Cyto-Education” with Stem Cell Therapy Help?

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The Potential “Core” of Vitamin D Receptor and Vitamin D Hypothesis: Synthesis of Common Basis of Some Autoimmune Diseases and Associated Cancers via Autophagy

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Emerging Vitamin D Receptor-Centered Patterns of Genetic Overlap across Some Autoimmune Diseases and Associated Cancers

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Synthetic and Systems Biology: Toward Achieving Impossible Missions and Deciphering Human Complex Disease Genetics

Yue Zhang

Authorship: an Engine for Research, and a Guarantee of Quality of Publication and Currency for Career Development

Yue Zhang

Ideal Open Access (OA2) and the Lineage of Reproducibility

Yue Zhang

Cancer Embryonic Stem Cell-like Attractors alongside Deficiency of Regulatory Restraints of Cell-Division and Cell-Cycle

Zhang Yue

Research Interests

- Genetics and Epigenetics, Systems biology, SLAC (Stem cell biology, longevity /Ageing and cancer biology) with a focus on chromatin remodelers w/o the stressors, particularly radiation, heat shock, and nutrient deprivation by using both C.elegans model organisms and mammalian systems.

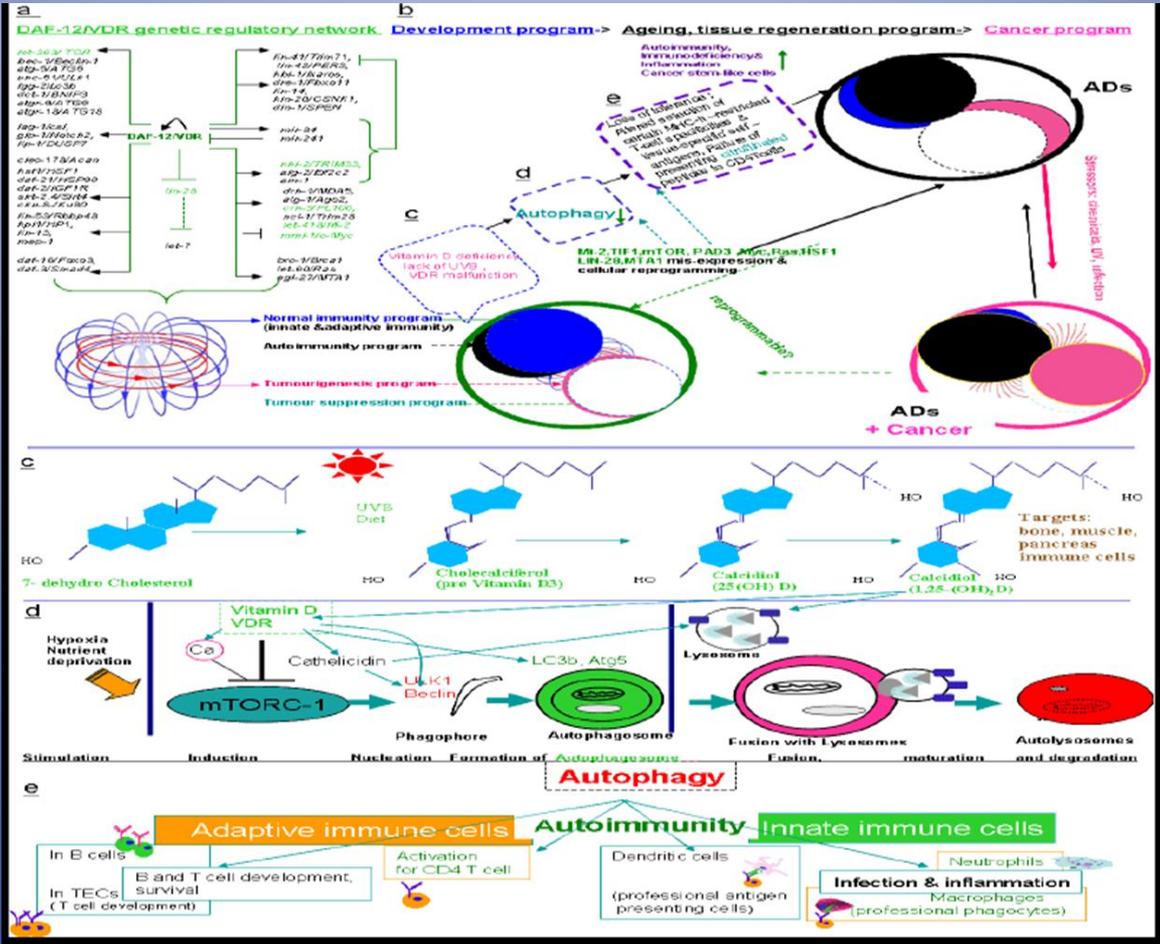
Cancers with Stem-Like Attractors and “Loss Of Differentiation” Novel Hallmark: Does the “Cyto-Education” with Stem Cell Therapy Help?

- Currently, cancer, with its unrestricted cellular growth, remains as one most significant medical and socio-economic problem. Because of the chaos and dynamics of genome-wide gene expression, cells in multicellular organisms heavily rely on master regulators for chromatin remodelling and /or cell cycle to make different fate decisions, such as proliferation or differentiation into specialized cells which eventually lead to normal tissue specification and /or organ formation. Such cell fate determination should include cell fate induction and the proper execution of the terminally differentiated cell fates originated at stem-/ progenitor- /precursor cells. Previous theoretical studies of genomewide gene regulatory networks (GRN) suggest that GRN needs to ensure cell fate trajectory on a right track with a manifestation of its ordered (stable) dynamics . Having checked their genome-wide gene expression profiling data by gene expression dynamics inspector (GEDI), we can show that cell attractors converge to a common metastable stem-like state along with aberrant expression of such masters, which, including *Mi-2 β*, *Rb*, *EZH2*, *MTA1* and *I(3)mbt*, are involved in carcinogenesis and/or cancer metastasis . This supports ‘loss of differentiation’ as a novel hallmark of cancer hereby via incorporating elements from systems biology.
- <http://omicsonline.org/open-access/cancers-with-stemlike-attractors-and-loss-of-differentiation-novel-hallmark-does-the-cytoeducation-with-stem-cell-therapy-help-2332-0737.1000e104.php?aid=28663>

The Potential “Core” of Vitamin D Receptor and Vitamin D Hypothesis: Synthesis of Common Basis of Some Autoimmune Diseases and Associated Cancers via Autophagy

- Nutrigenomics may tell us how various nutrients interact with the genome and potentially cause alteration of gene expressions. One nutrient of particular interest is vitamin D, deficiency of which may lead to diseases in various human organ and muscle systems. Decoding the genetics of complex diseases such as autoimmune diseases and/or cancers associated with the aging process is vital to understand the controversies the beneficial effects of vitamin D supplementation on these diseases. Based on comparative advantages of different model system and recent ChIPseq/ ChIP-chip studies, we previously proposed one novel insightful hypothesis: the genetic regulatory network of Vitamin D Receptor (VDR, homologue of DAF-12 in *Caenorhabditis elegans*) may act as a common basis in preventing some autoimmune diseases and associated cancers. Further, such diseases may burst up with polygenic genetic mutations and/or variations in that deficiency of vitamin D and lacking of UVB lead to the mal-functional DAF-12/ VDR and lose its buffering potential as a capacitor. The aberrance of environmental factor-induced DAF-12/VDR may counter-intuitively lead to in situ dys-regulation of the expression of an array of its target genes and locally induced autoimmunity because of the citrullination of in situ dys-regulated genes, which may be mediated by the VDR-orchestrated autophagy process. Being consistent with the “hygiene hypothesis” and the “danger signals” theory, some VDR/DAF-12 targets may be directly involved in these processes. Several testable predictions will be briefly discussed.

Model of autophagy-mediated DAF-12/VDR prevention from some ADs and cancers



Autoimmune Diseases and Associated Cancers

- **GRN of VDR may be Consider as Common Basis for ADs and Associated Cancers?**
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- Although further testing is awaiting for, one GWAS reveals that one allelic VDR variant may link to clinical autoimmune antibodies including anti-p150 (TRIM33/ TIF-1 γ)/p140(TRIM24/TIF-1 α) whose natural self-antigens may correlate with protein products encoded by TIF-1 γ/α , whose homologues flt-1 and nhl-2 are direct targets of DAF-12/VDR [8,9]. DAF-12/VDR target genes from our CHIP-chip screening showed many overlaps with validated homologues identified in human VDR studies and significantly enriched near genes that are pathologically associated with ADs and cancer [8]. But it is necessary to experimentally test these overlaps in human VDR.
- <http://omicsonline.org/emerging-vitamin-d-receptorcentered-patterns-of-genetic-overlap-across-some-autoimmune-diseases-and-associated-cancers-2157-7412.1000e123.php?aid=21837>

Synthetic and Systems Biology

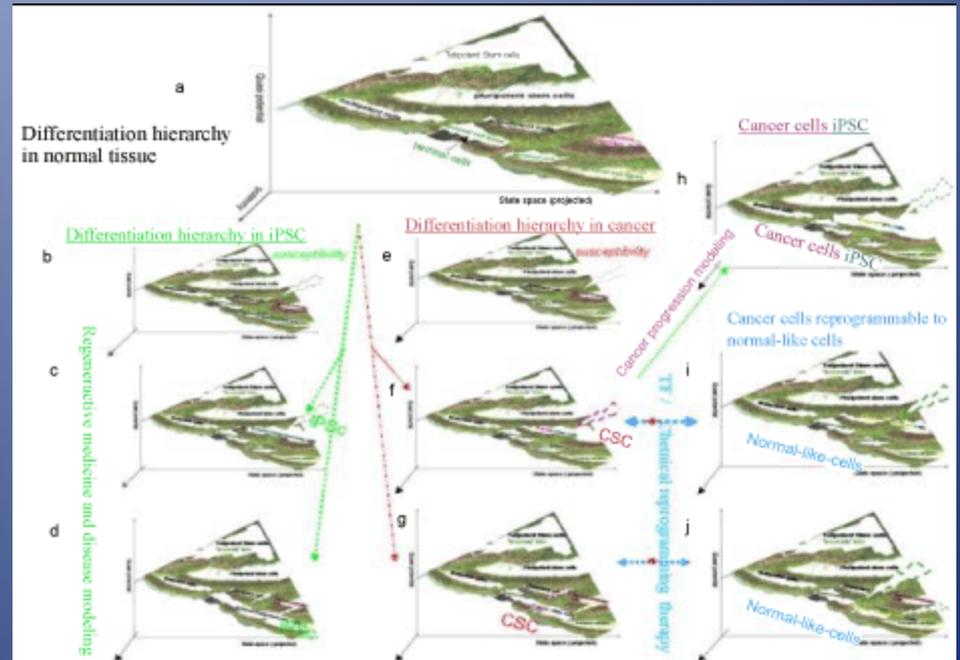
- Many pioneering works have inspired researchers to stay up-to-date on synthetic and system biology. Several cases that were originally thought to be exceptionally difficult, if not impossible, have been carried out successfully, such as Craig Venter's creation of the world's first synthetic life form. At a system level, nucleic reprogramming succeeded in frog half a century ago (reviewed in); but doubts about whether or not this was impossible lingered until 40 years later, when a cocktail of four transcriptional factors systematically reprogrammed the somatic cells to stem cells . Other cases include that telomerase reactivation may lead to the reversal of tissue degeneration in aged telomerase-deficient mice and muscle-derived stem/progenitor cell dysfunction acts as a healthspan and lifespan limiting factor for murine progeria reversal.
- <http://omicsonline.org/synthetic-and-systems-biology-achieving-impossible-missions-deciphering-human-complex-disease-genetics-2332-0737.1000e102.php?aid=20353>

Research Reproducibility

- Medical science is troubled by questions of economics, and we see a trend towards hidden conflicts of interest (COI), and/or irreproducibility. Traditional subscription journals represent the standard and have better COI control, but the sooner they move into OA, the better. If our heritage of **reproducibility** risks to end, we will need stronger systems to maintain this heritage, with a need for formal external incentives and regulations. The policy of copyright law and intellectual property crediting system should align with OA. Law has a debt to science. Authors have to abide by them to ensure high quality, ethics and scientific rigor for primary research publications. Funding agencies may make this mandatory, or a “sting” could be carried out at the discretion of reviewers or editors. Obviously, extended experiment verification is expensive.
- <http://esciencecentral.org/journals/open-access-oa-and-the-heritage-of-research-reproducibility-2329-8847.1000e104.php?aid=18678>

Cellular Reprogramming

- Here is a concise historical review and some perspectives on the spotlighted nuclear transplantation (cloning) and induced pluripotent stem cells (iPSC). Besides, we propose that iPSC may be hypothetically considered one exceptional case among “cancerous” cells. The argument between stochastic and elite model of iPSC is briefly discussed.



Engineered Genes, Genome Editing, and /or Transgenics

- To date, the luciferase in reporter assays and the gateway toolkit are among our routinely-used reagents, and the latter also facilitates the large-scale gene cloning so that we can switch to pick up ones of interest from the genome-wide ready- to- use collections However, it was particularly a small number of enzymes that probably have advanced huge successful stories in research of biology and medicine. They actually help us perform the special-pediment DNA cloning, genome editing and transgenics. This has greatly enlightened us. Interestingly, the toolkit equipped with one of them, e.g. Cre/lox (Flp /FRT) recombinase system for gene targeting has already been awarded the Nobel Prize.
- <http://omicsgroup.org/journals/less-is-more-enzymes-inspiring-the-breakthroughs-alongside-shining-on-engineered-genes-genome-editing-and-or-transgenics-2329-6674.1000e101.php?aid=4257>

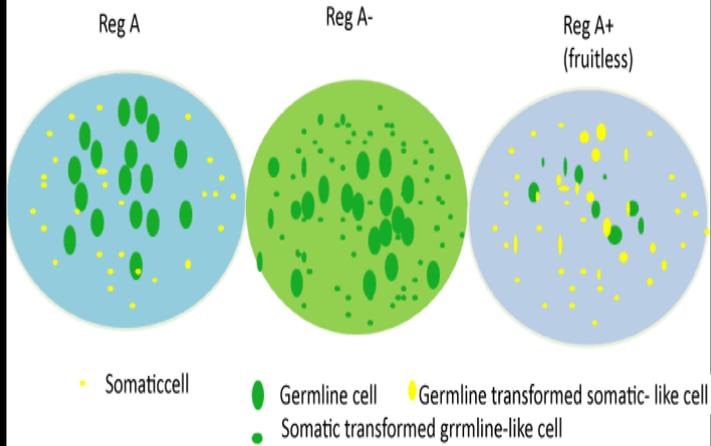
New Concepts of Germline Gene –Reactivated Cancer

- The mechanism that ectopic expression of germline genes result in somatic tumors such as melanoma and brain tumors remains a big challenge . A century of unproductive concentration on the cancer- is -a- cell-based -disease. Somatic Mutation Theory of carcinogenesis (SMT) , the paradigmatic instability is coming to eyes, for instance, the tissue level [e.g. the Tissue Organization Field Theory of carcinogenesis (TOFT)] has been repeatedly suggested as one competitive strategy. Most recently, at the genome regulatory network level (GRN), the cancer attractors hypothesis naturally explains tumorigenesis , but such a new network-based intellectual framework is still quite abstract and also remains incompletely understood what its evolutionary origin is and what causes normal somatic cells be entrapped in.
- <http://omicsonline.org/new-concepts-of-germline-gene-reactivated-cancer-2161-0436.1000e101.php?aid=2013>

New Frontiers of Aging Reversal and Aging-Related Diseases Reprogramming

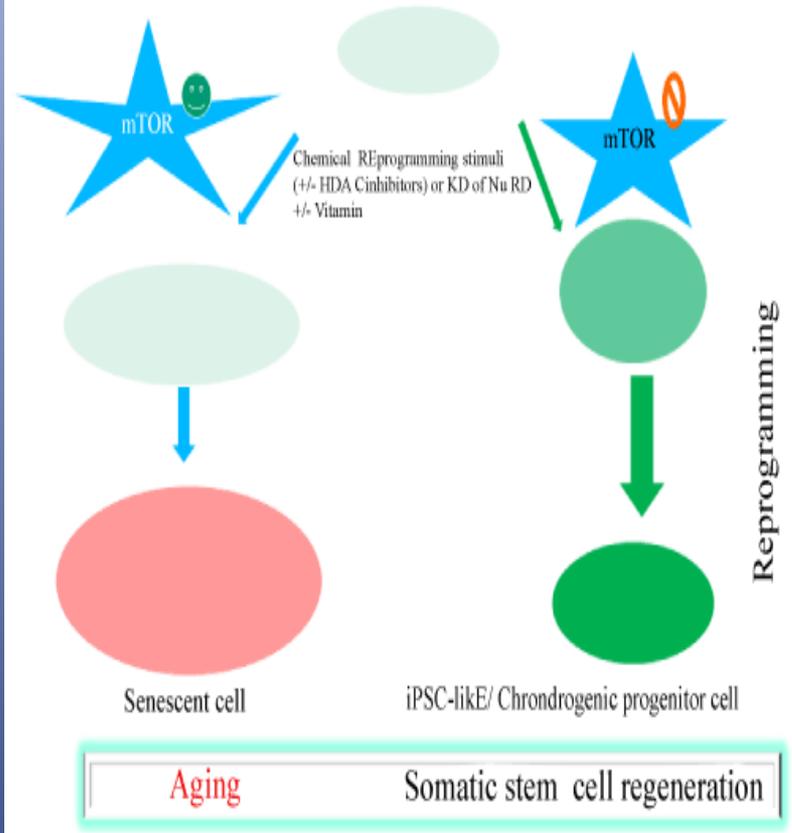
- Cancer is certainly the leading ageing –related lethal disease. Through Mi-2/NuRD chromatin remodeling –related cancer attractors theory, we could understand better the carcinogenesis, especially for germline gene - reactivated cancers, and hence develop strategy to reprogram the cancerous diseased cells to normal like cells. Hereby we focus on another ageing -related disease. It is well-known that cartilage makes the movement of joints smooth and the fading- away and breakdown of articular cartilage by injury or age-related “wear and tear” causes osteoarthritis (OA), the most common degenerative chronic disease, which is further characterized by synovial inflammation, pain, subchondral bone alterations, loss of tissue cellularity and extracellular matrix (ECM) damages, a major cause of decreased quality of life in adults in the world. Yet therapy remains a challenge because cartilage has minimal ability to repair and renew itself. Alongside studies for decades, in clinical trials in patients with established or advanced OA, candidate disease-modifying drugs have failed to show efficacy, as the case in cancer research.
- <http://omicsgroup.org/journals/new-frontiers-of-aging-reversal-and-aging-related-diseases-reprogramming-2169-0111.1000e101.php?aid=4093>

Multicellularity

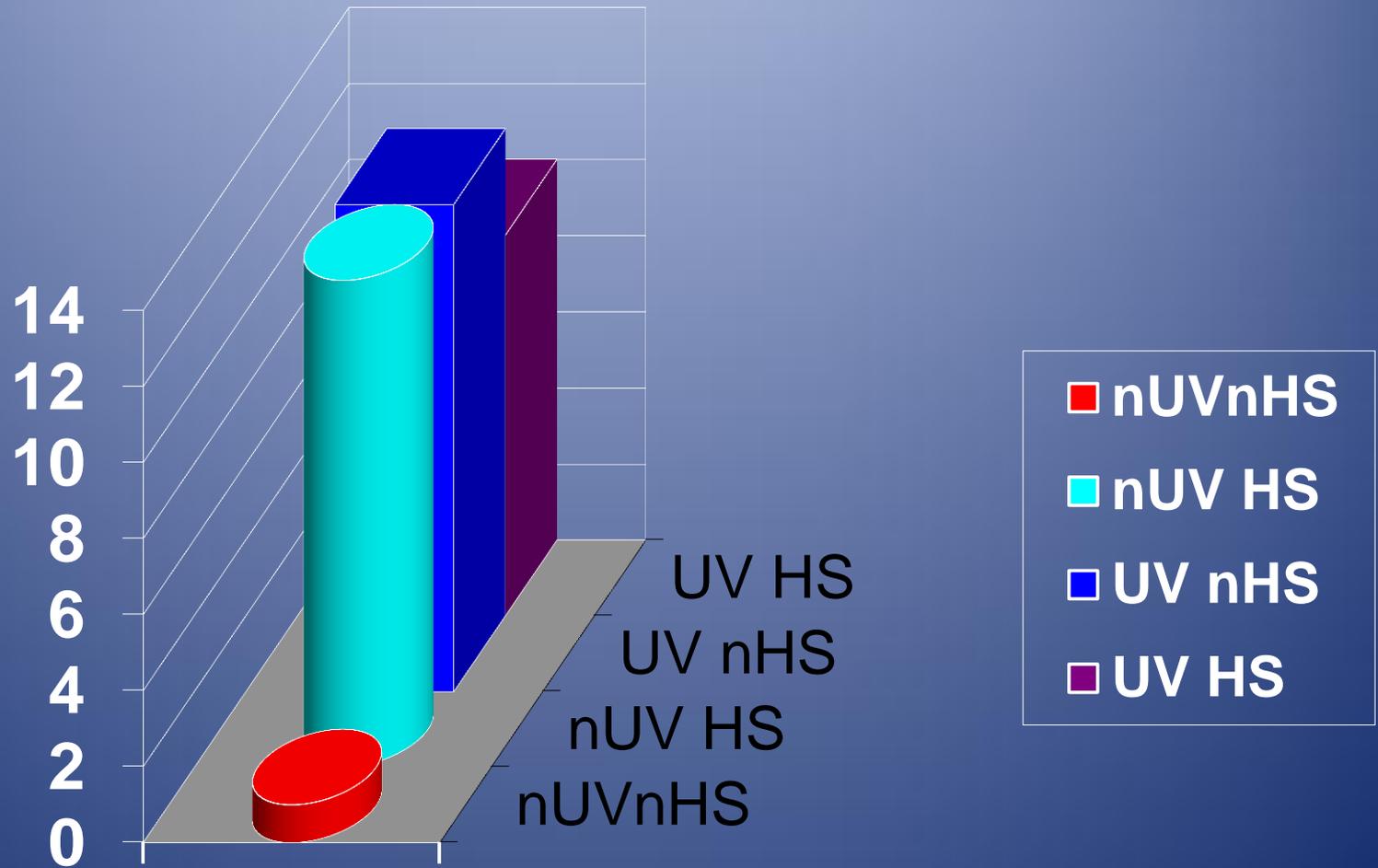


Somatic cell
RegA is the functional equivalent of Mi-2/ Nu RD complex in green algae.

Chondrocyte



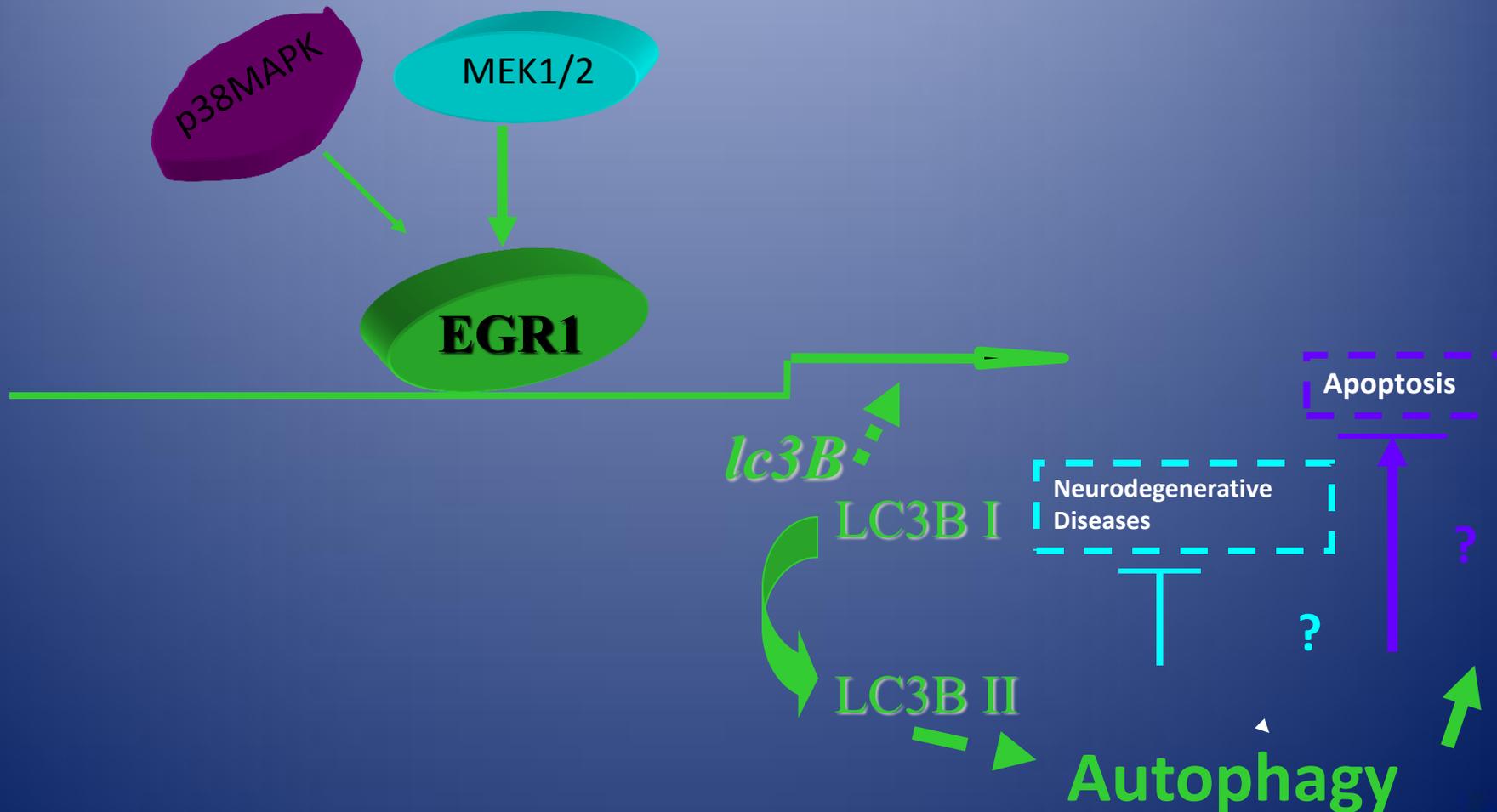
Up-regulation of endogenous *egr1* in UV treated and heat-shocked Hela cells (qRT-PCR assays)



Normalized

Model of EGR1 -mediated stress-induced autophagy

Heat shock, UV



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