INTRODUCING

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DC, CIHP

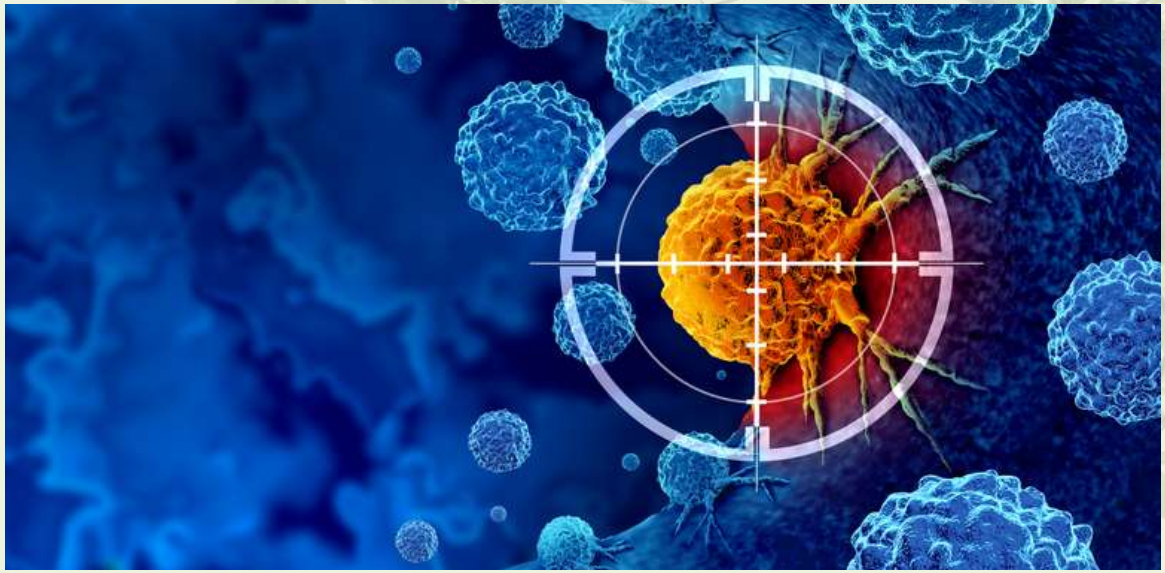
TRANSFORMATION
THE GENESIS OF GOOD HEALTH®

Enzyme Therapy Seminar

October 11-12 • Houston, TX

Proteolytic Enzymes

One of Nature's Answers to Cancer



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American Cancer Society

2024—First Year the US Expects More than 2M New Cases of Cancer

- This trend is largely affected by the aging and growth of the population and by a rise in diagnoses of 6 of the 10 most common cancers—breast, prostate, endometrial, pancreatic, kidney, and melanoma

- The other 4 top 10 cancers are lung, colon/rectum, bladder, and non-Hodgkin lymphoma

Cancer patients are getting younger

- Cancer risk increases with age, and people most likely to be diagnosed with cancer are adults aged 65 and older but this trend is beginning to change

- Especially notable is the rise in colorectal cancer diagnoses among people younger than 50.

- Cervical cancer is increasing in incidence in an even younger population—women ages 30 to 44

Burden of 30 cancers among men: Global statistics in 2022 and projections for 2050 using population-based estimates

- Incident cancer cases are projected to reach 19 million globally by 2050, an 84.3% increase from the 2022 estimate
- The number of cancer deaths is projected to reach 10.5 million by 2050, a 93.2% increase from the 2022 estimate
- Lung cancer is projected to remain the leading cancer type for both cases and deaths by 2050, with both cases and deaths increasing by greater than 87% compared with the 2022 estimate

The *Lancet* Commission on prostate cancer: planning for the surge in cases; Lancet 2024; 403: 1683–722

- We project that the number of new cases of prostate cancer annually will rise from 1.4 million in 2020 to 2.9 million by 2040
- The projected rise in prostate cancer cases cannot be prevented by lifestyle changes or public health interventions
- Correspondingly, we estimated that prostate cancer deaths will rise by 85%, from 375,000 in 2020 to close to 700,000 by 2040

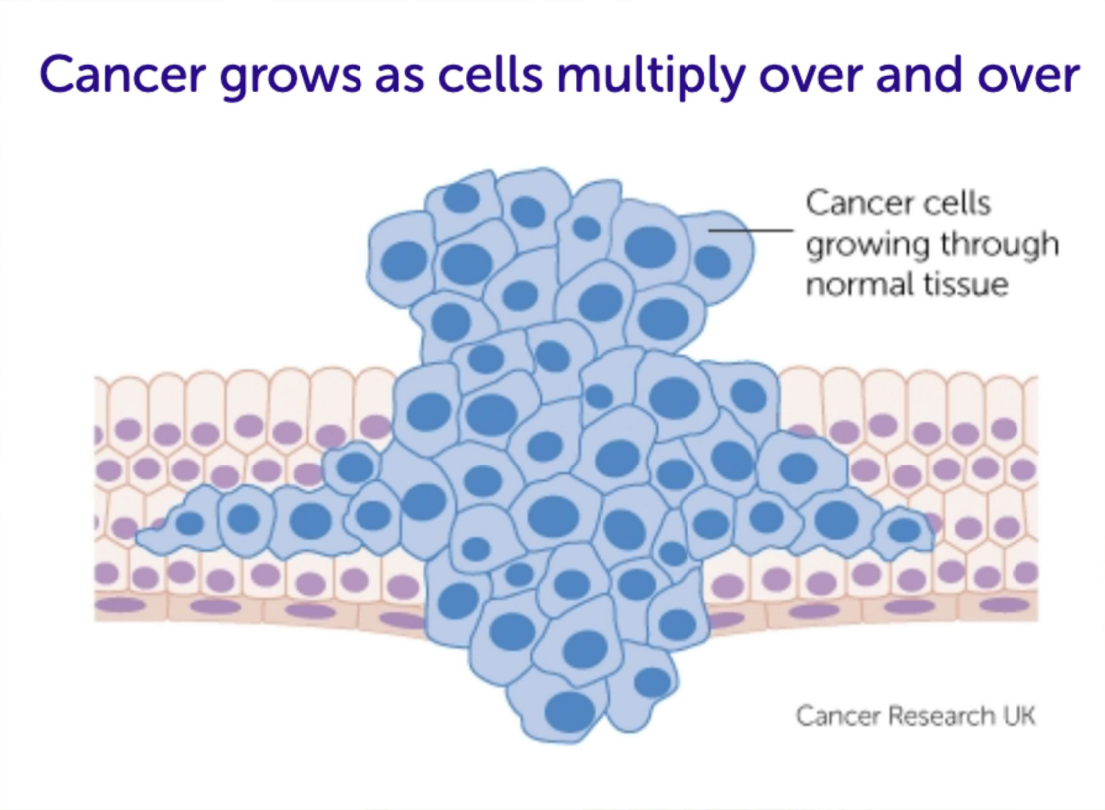
Proteolytic Enzymes

One of Nature's Answers to Cancer

Learning Objectives

- Review normal cell cycle
- Identify signaling factors that keep the cell cycle in check
- Briefly discuss the hallmarks of cancer
- Discuss tumor microenvironment and biomarkers that supports cancer cell survival
- Review proteolytic enzymes, their potential role in combating cancer, and their scientific basis
- Case Study

- Cancer occurs when abnormal cells divide in an uncontrolled way. Cancer starts when gene changes make one cell or a few cells begin to grow and multiply too much. This may cause a growth called a tumor – called a primary tumor.
- Some cancers may eventually spread into other parts of the body – this is called a secondary tumor or a metastasis.
- There are more than 200 different types of cancer. Cancer and its treatments can affect body systems such as the blood and lymph circulation, the immune systems, and hormones.



Normal Cell Cycle

M: mitotic phase is a multistep process during which the duplicated chromosomes are aligned, separated, and move into two new, identical daughter cells.

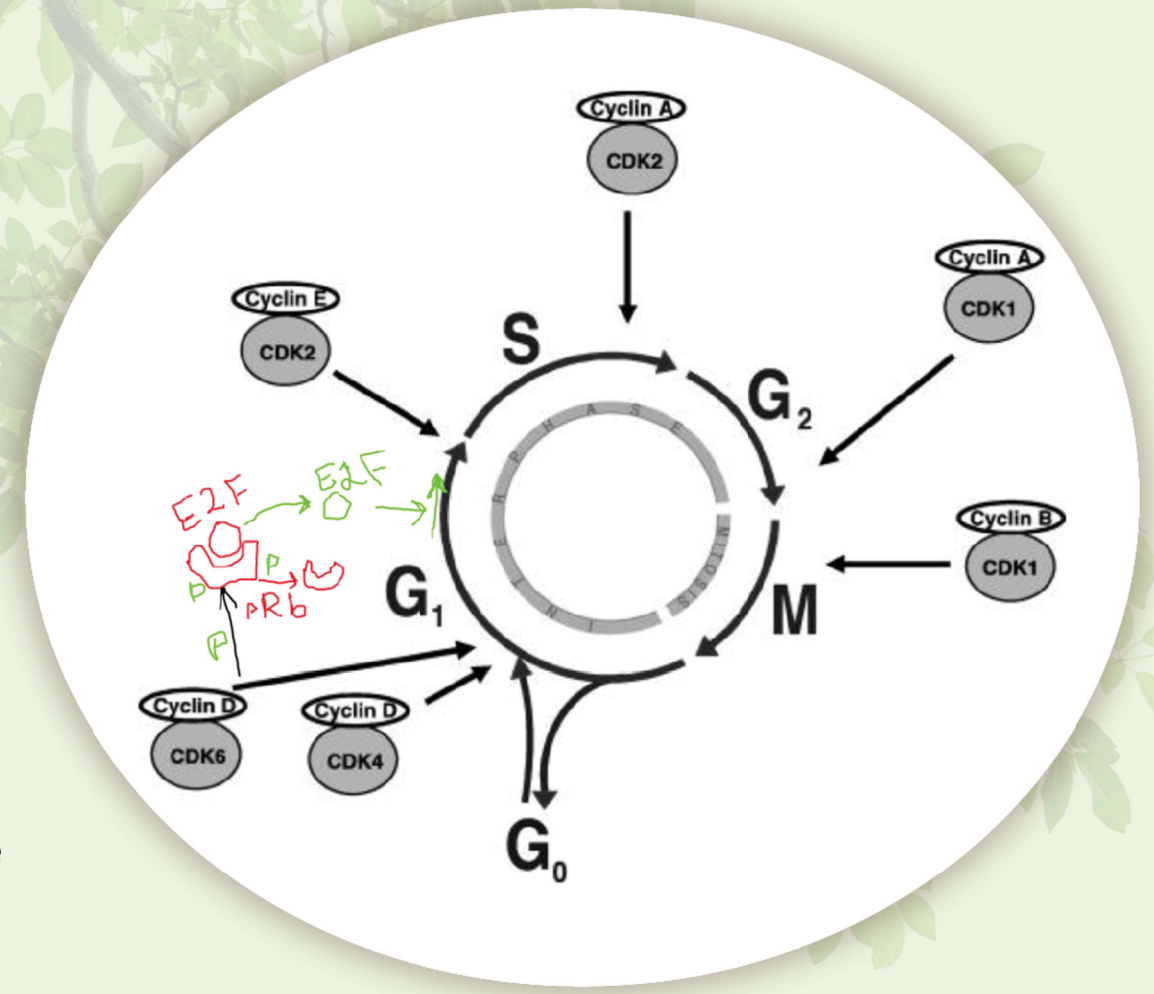
G0: Quiescent stage. Cells that never or rarely divide, such as mature cardiac muscle and nerve cells, remain in G_0 permanently.

G1: The cell is accumulating the building blocks of chromosomal DNA and the associated proteins as well as accumulating sufficient energy reserves to complete the task of replicating each chromosome in the nucleus.

S: DNA replication can proceed through the mechanisms that result in the formation of identical pairs of DNA molecules—sister chromatids. The centrosome is also duplicated during the S phase.

G2: The cell replenishes its energy stores and synthesizes proteins necessary for chromosome manipulation and movement. Some cell organelles are duplicated, and the cytoskeleton is dismantled to provide resources for the mitotic phase. There may be additional cell growth during G2.

- The transition from one cell cycle phase to another occurs in an orderly fashion and is regulated by different cellular proteins.
- Key regulatory proteins are the cyclin-dependent kinases (CDK), a family of serine/threonine protein kinases that are activated at specific points of the cell cycle during G1 (CDK4, CDK6 and CDK2), S (CDK2), G2, and M (CDK1).
- When activated, CDK induce downstream processes by phosphorylating selected proteins. Different cyclins are required at different phases of the cell cycle. The three D type cyclins (cyclin D1, cyclin D2, cyclin D3) bind to CDK4 and to CDK6 and CDK-cyclin D complexes are essential for entry in G1. Another G1 cyclin is cyclin E which associates with CDK2 to regulate progression from G1 into S phase. Cyclin A binds with CDK2 and this complex is required during S phase. In late G2 and early M, cyclin A complexes with CDK1 to promote entry into M. Mitosis is further regulated by cyclin B in complex with CDK1.



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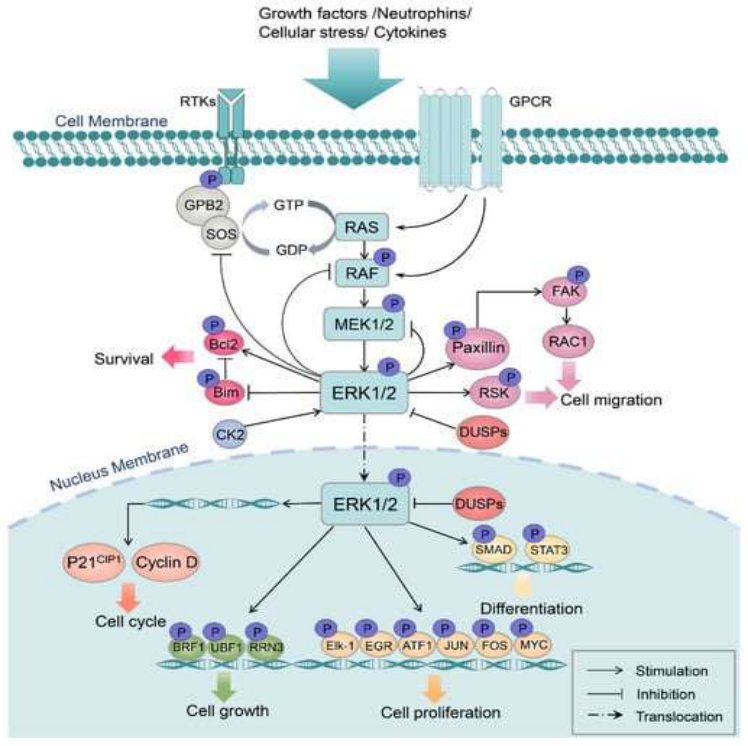
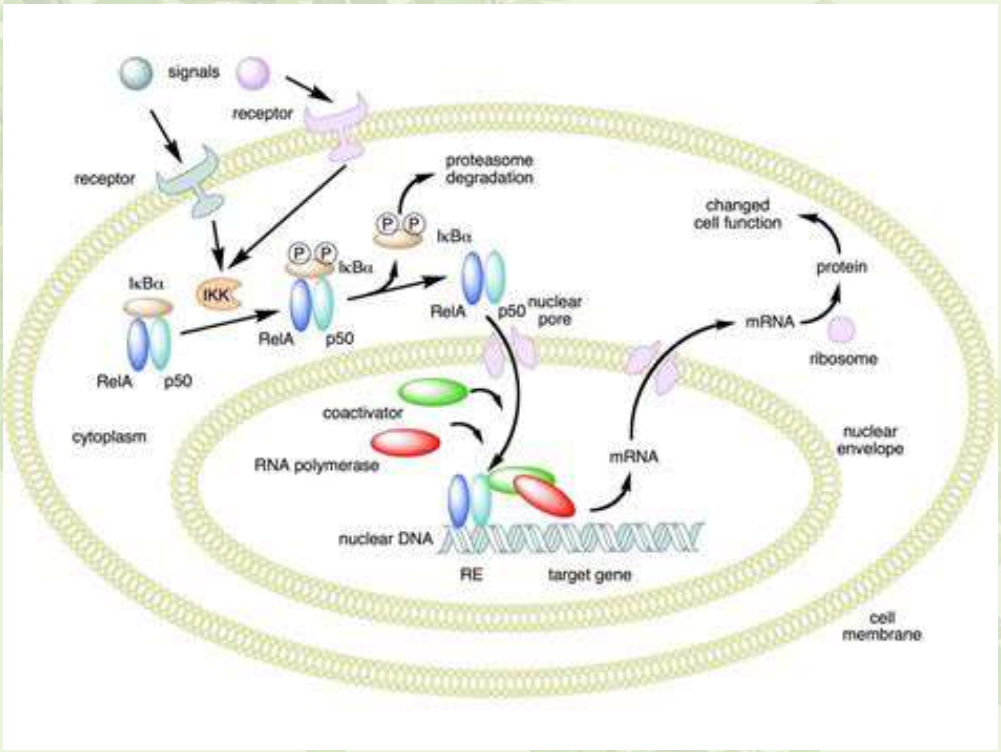
Kinases add phosphates; Phosphatases take off phosphates





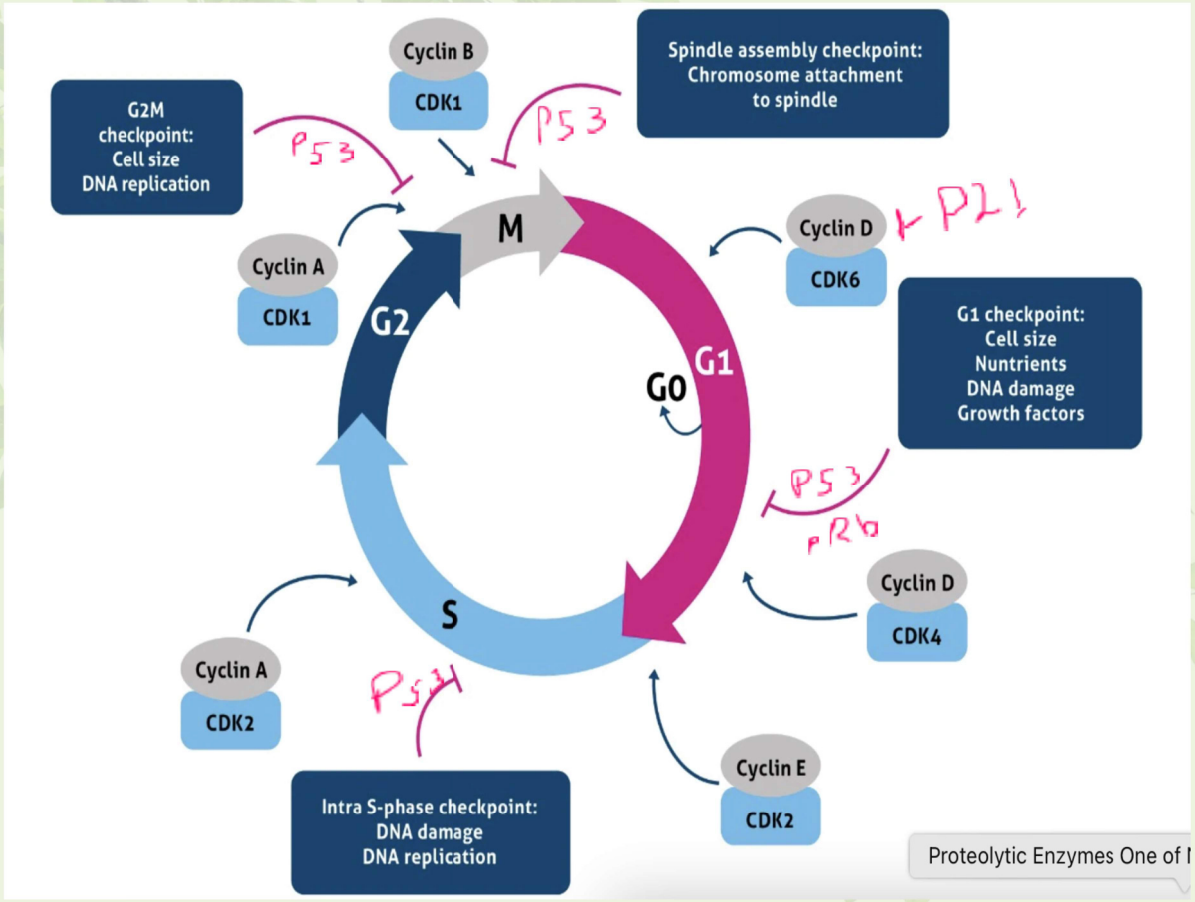
ERK: Extracellular signal-regulated kinase
MAPK: Mitogen-activated protein kinase
MEK: Ras/Raf/MAPK

Checkpoints: Regulated by Tumor Suppressors p53 and pRb

The cell cycle is based on three main checkpoints:

- Phase G1 – DNA integrity and cell size
- Phase G2 – DNA damage and chromosome duplication
- Phase M – Attachment of kinetochore and a spindle fiber

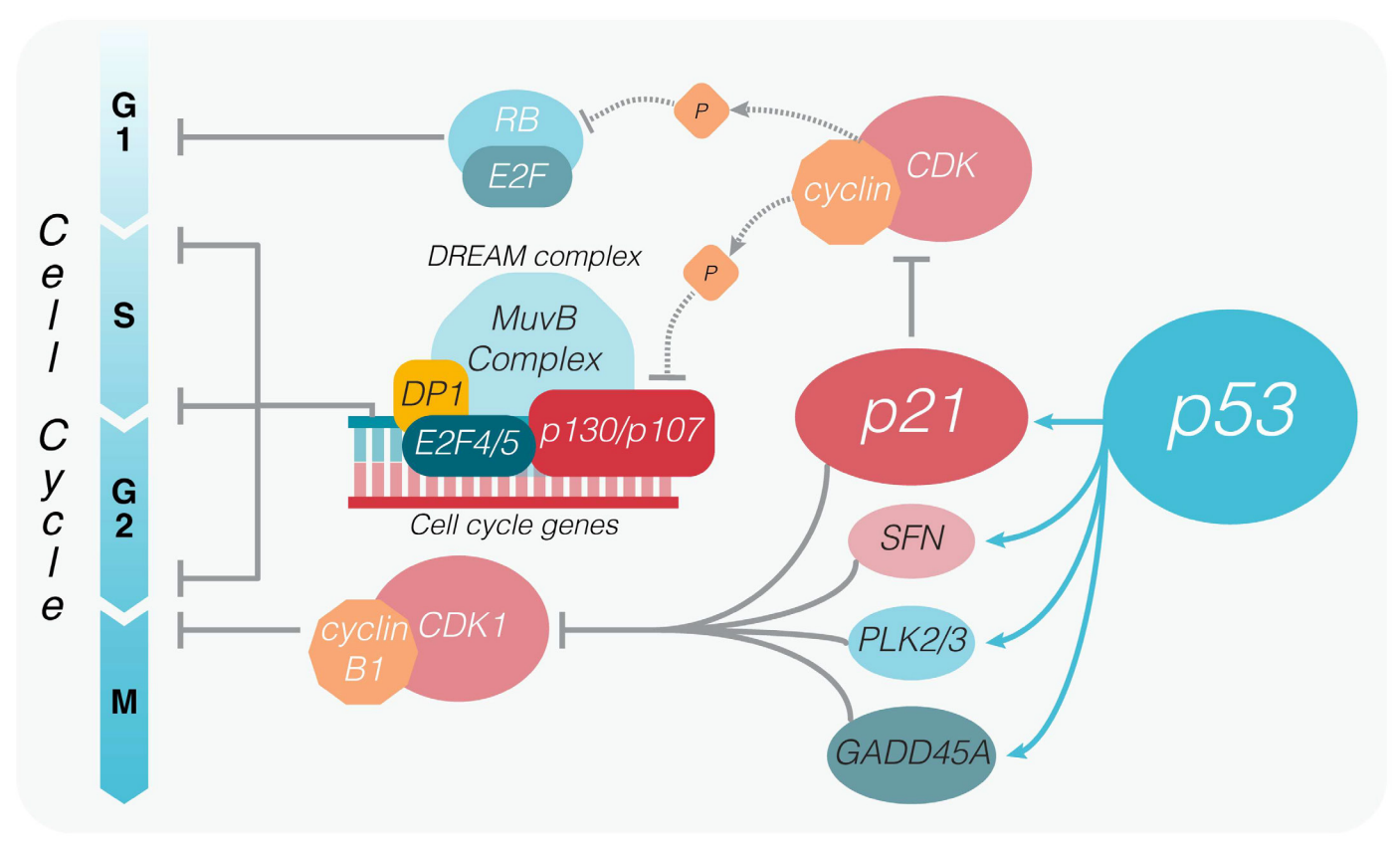
The key role of checkpoint proteins is to detect DNA damage and send a signal to delay cell cycle advance until the damaged chromosomes are repaired.



Cell Cycle Checkpoints and Signaling Factors

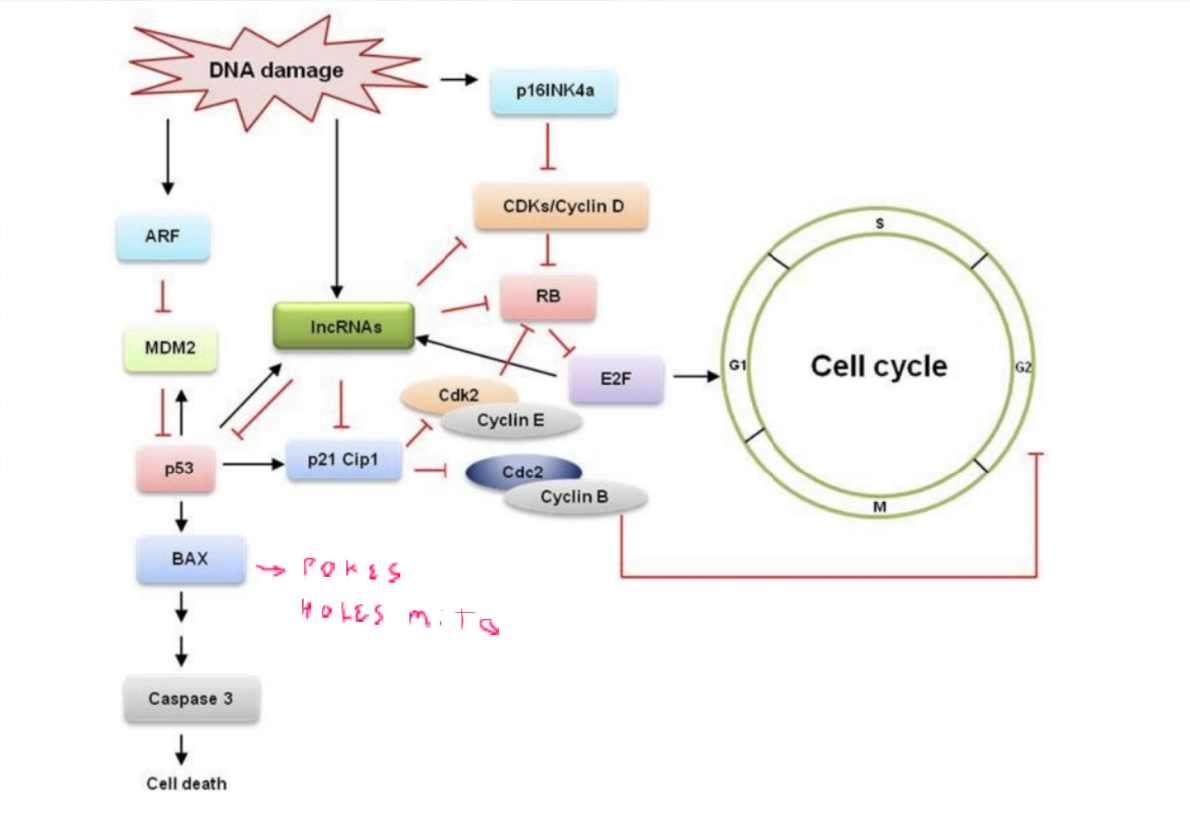
- P53- p53 has been shown to play a role of cell cycle arrest, senescence, DNA repair, and apoptosis
- pRb- Rb protein (pRb) is a master regulator of biological pathways influencing virtually every aspect of intrinsic cell fate including cell growth, cell-cycle checkpoints, differentiation, senescence, self-renewal, replication, genomic stability and apoptosis
- P21- Cyclin-dependent kinase (CDK) inhibitor p21 (also known as p21(WAF1/Cip1)) is one of these factors that promote cell cycle arrest in response to a variety of stimuli. P21 can be induced by both p53-dependent and p53-independent mechanisms. Some other important functions attributed to p21 include transcriptional regulation, modulation or inhibition of apoptosis. In addition, p21 can play a role in DNA repair by interacting with proliferating cell nuclear antigen
- BAX- When Bax protein is activated, its function is to bind and induce Mitochondrial Outer Membrane permeabilization. Such permeabilization results in mitochondrial swelling and rupture with subsequent leakage of intermembrane space proteins, specifically Cytochrome c and endonuclease G
- Bcl-2 - anti-apoptotic member that inhibits the activation and activities of Bax (pro-apoptotic)
- Caspases- Cytochrome c binds and activates cytosolic caspases, which are known to be the effector proteases of cell death

Tumor Suppressors



Tumor Suppressor Pathways

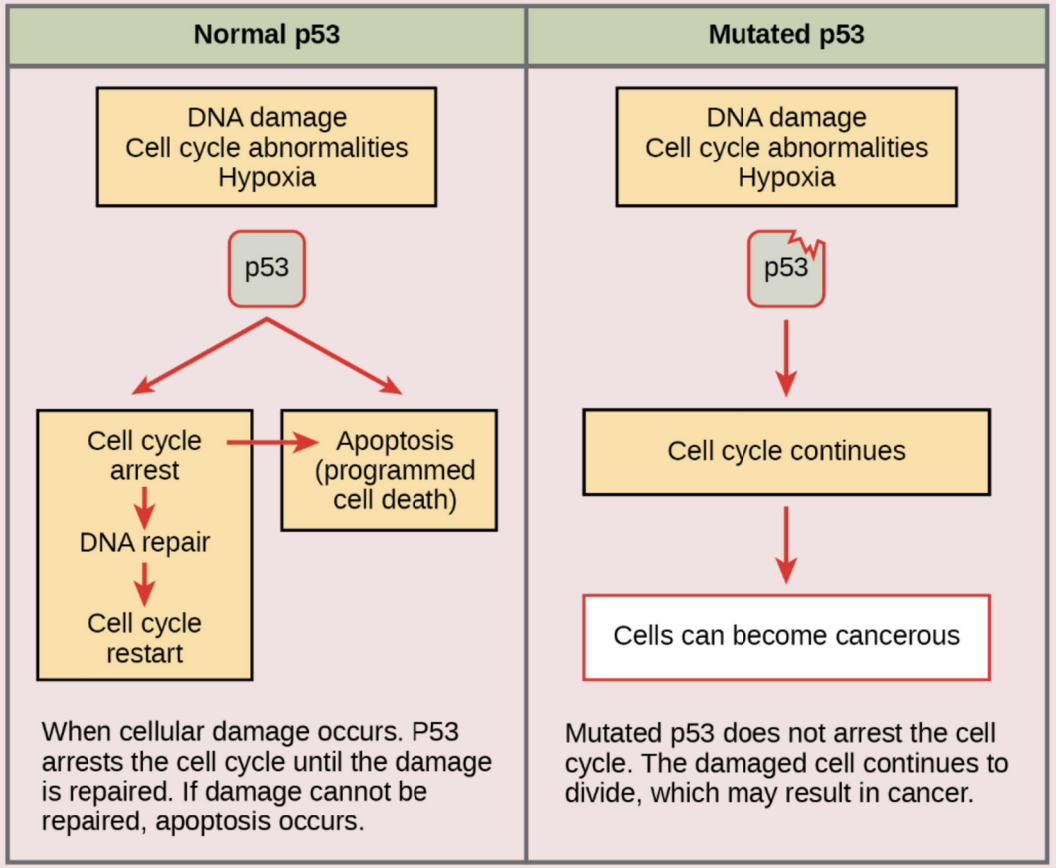
DNA damage by way of UV rays, hypoxia, certain viruses, oxidative stress, smoking, etc. turns on the tumor suppressor pathways p53 and retinoblastoma (RB) which control the DNA damage response. p16INK4a and p14ARF controls the activity of RB and p53. RB promotes cell cycle arrest in G1 and regulates entry into S phase by inhibiting the E2Fs. p53 mediates several effects, including causing G1 and G2 arrest and promoting apoptosis. Loss of p53 function also promotes genomic instability.
The *p53* gene is the most mutated gene in human cancer and regulation of p21 in response to DNA damage is lost when p53 is inactivated



Tumor Suppressor p53

The role of normal p53 is to monitor DNA and the supply of oxygen (hypoxia is a condition of reduced oxygen supply). If damage is detected, p53 triggers repair mechanisms. If repairs are unsuccessful, p53 signals apoptosis. A cell with an abnormal p53 protein cannot repair damaged DNA and thus cannot signal apoptosis. Cells with abnormal p53 can become cancerous.

(credit: modification of work by Thierry Soussi)



Example of potential p53 inhibitors

- **SARS-CoV-2 spike S2 subunit inhibits p53 activation of p21(WAF1), TRAIL Death Receptor DR5 and MDM2 proteins in cancer cells**

  "In summary, we identified the SARS-CoV-2 spike S2 subunit as a factor that interrupts p53 binding to MDM2 in cancer cells and demonstrated the suppressive effect of SARS-CoV-2 spike S2 on p53 signaling in cancer cells. As *loss of p53 function is a known driver of cancer development* and confers chemo-resistance, our study provides insight into cellular mechanisms by which SARS-CoV-2 spike S2 may be involved in reducing barriers to tumorigenesis"

- **S2 subunit of SARS-nCoV-2 interacts with tumor suppressor protein p53 and BRCA: an in-silico study**

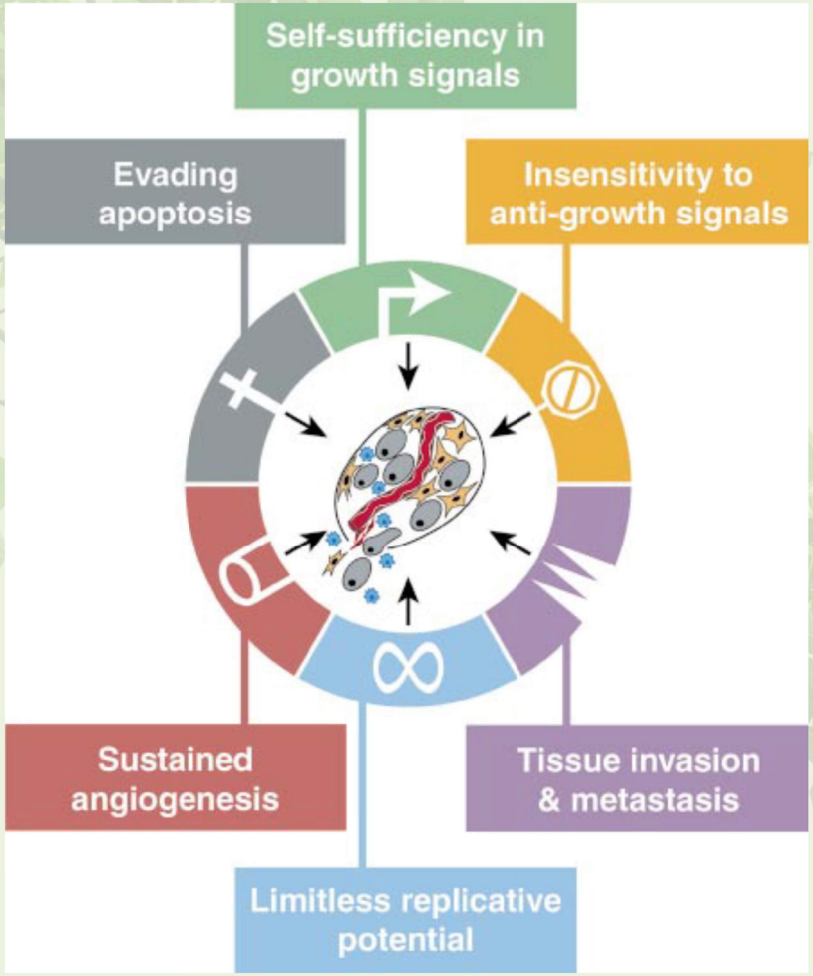
  An in-silico modeling study that concluded the S2 segment of the SARS-CoV-2 Spike protein could be anticipated to inhibit the p53 and BRCA1/2 tumor surveillance systems. In-silico is a computer simulation to study biological events and data

Hallmarks of Cancer

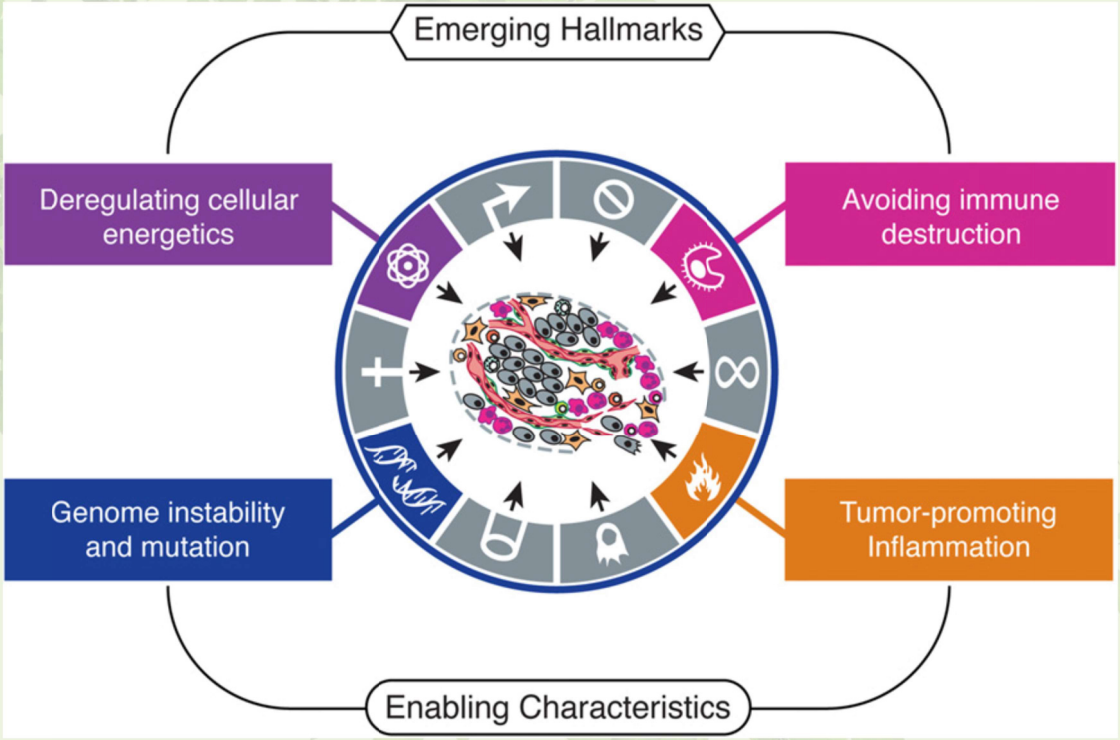
Research has revealed cancer to be a disease involving dynamic changes in the genome. The foundation has been set in the discovery of mutations that produce oncogenes with dominant gain of function and tumor suppressor genes with recessive loss of function; both classes of cancer genes have been identified through their alteration in human and animal cancer cells and by their elicitation of cancer phenotypes in experimental models (Bishop and Weinberg, 1996).

The vast catalog of cancer cell genotypes is a manifestation of six essential alterations in cell physiology that collectively dictate malignant growth: self-sufficiency in growth signals, insensitivity to growth-inhibitory (antigrowth) signals, evasion of programmed cell death (apoptosis), limitless replicative potential, sustained angiogenesis, and tissue invasion and metastasis.

Douglas Hanahan and Robert A. Weinberg 2000



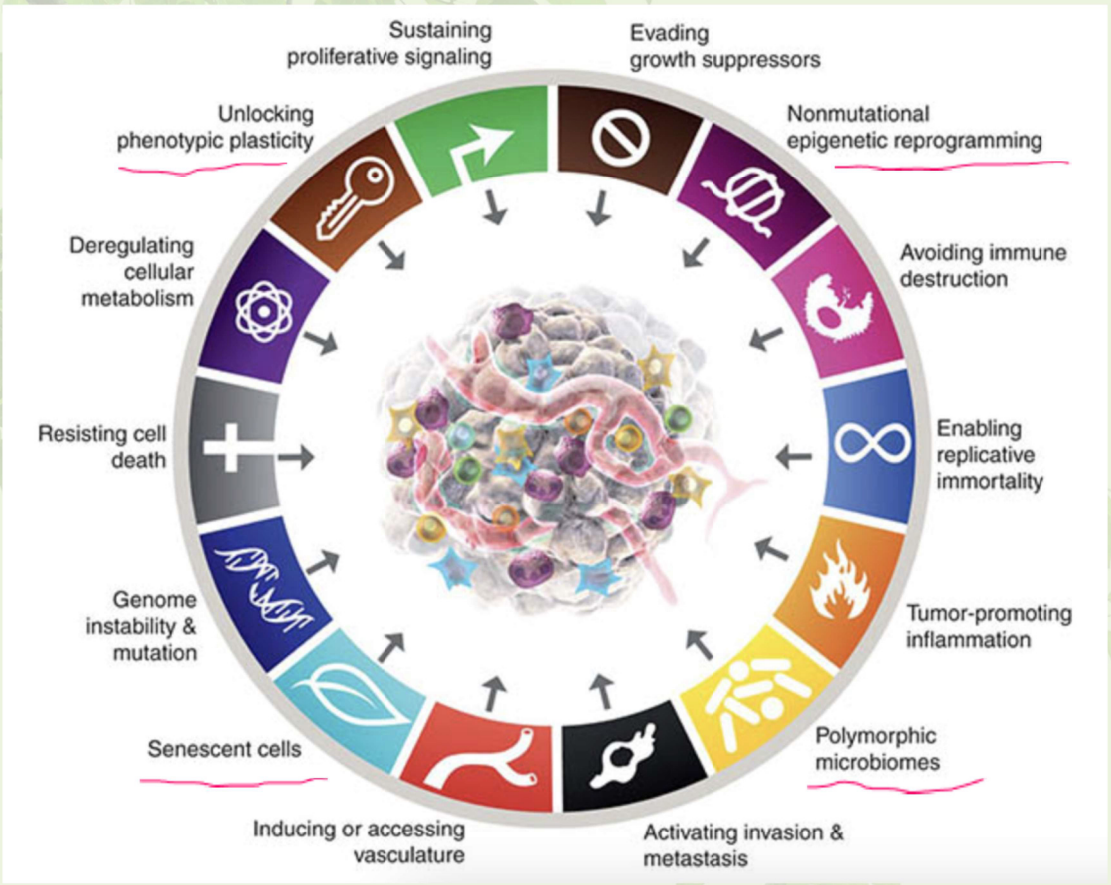
An increasing body of research suggests that two additional hallmarks of cancer are involved in the pathogenesis of some and perhaps all cancers. One involves the capability to modify, or reprogram, cellular metabolism to most effectively support neoplastic proliferation. The second allows cancer cells to evade immunological destruction, by T and B lymphocytes, macrophages, and natural killer cells. Because neither capability is yet generalized and fully validated, they are labeled as emerging hallmarks. Additionally, two consequential characteristics of neoplasia facilitate acquisition of both core and emerging hallmarks. Genomic instability and thus mutability endow cancer cells with genetic alterations that drive tumor progression. Inflammation by innate immune cells designed to fight infections and heal wounds can instead result in their inadvertent support of multiple hallmark capabilities, thereby manifesting the now widely appreciated tumor-promoting consequences of inflammatory responses.



Hallmarks of Cancer

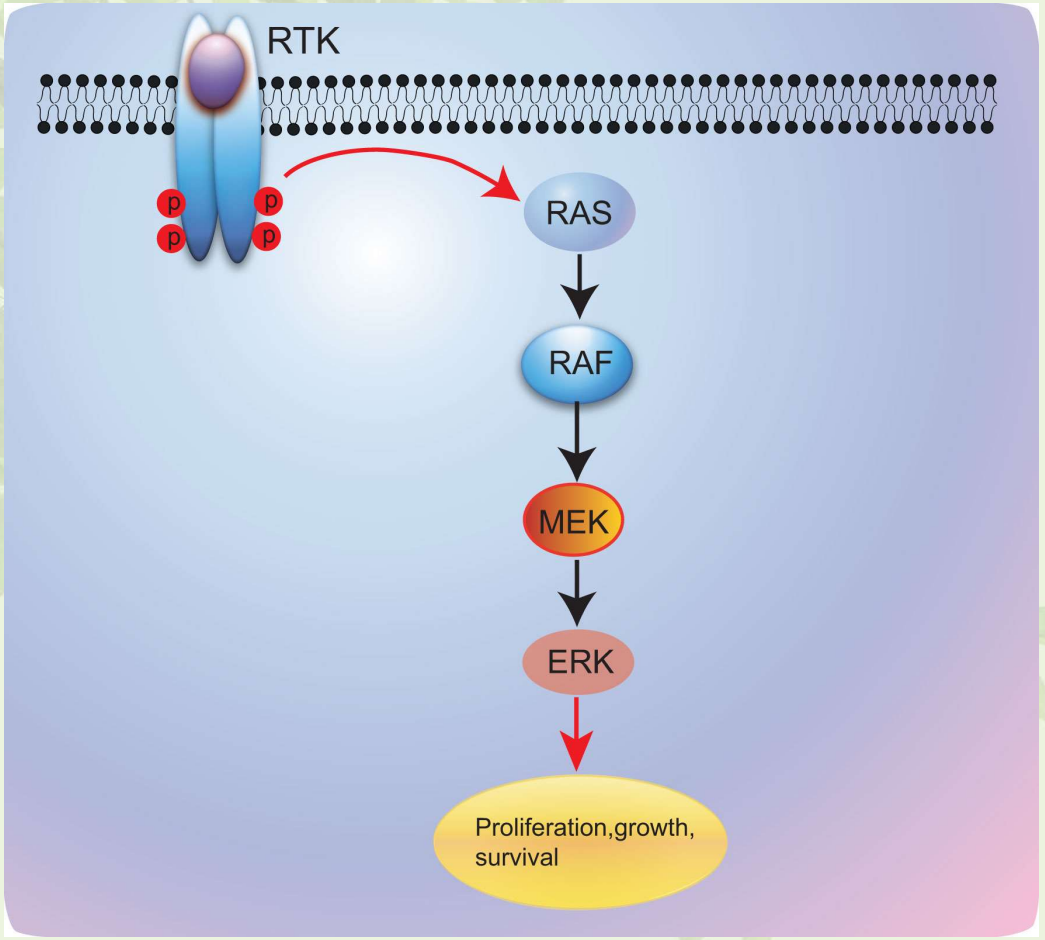
This revision incorporates additional proposed emerging hallmarks and enabling characteristics involving unlocking phenotypic plasticity, non-mutational epigenetic reprogramming, polymorphic microbiomes, and senescent cells.

The Hallmarks of Cancer: New Dimensions. Hanahan 2022



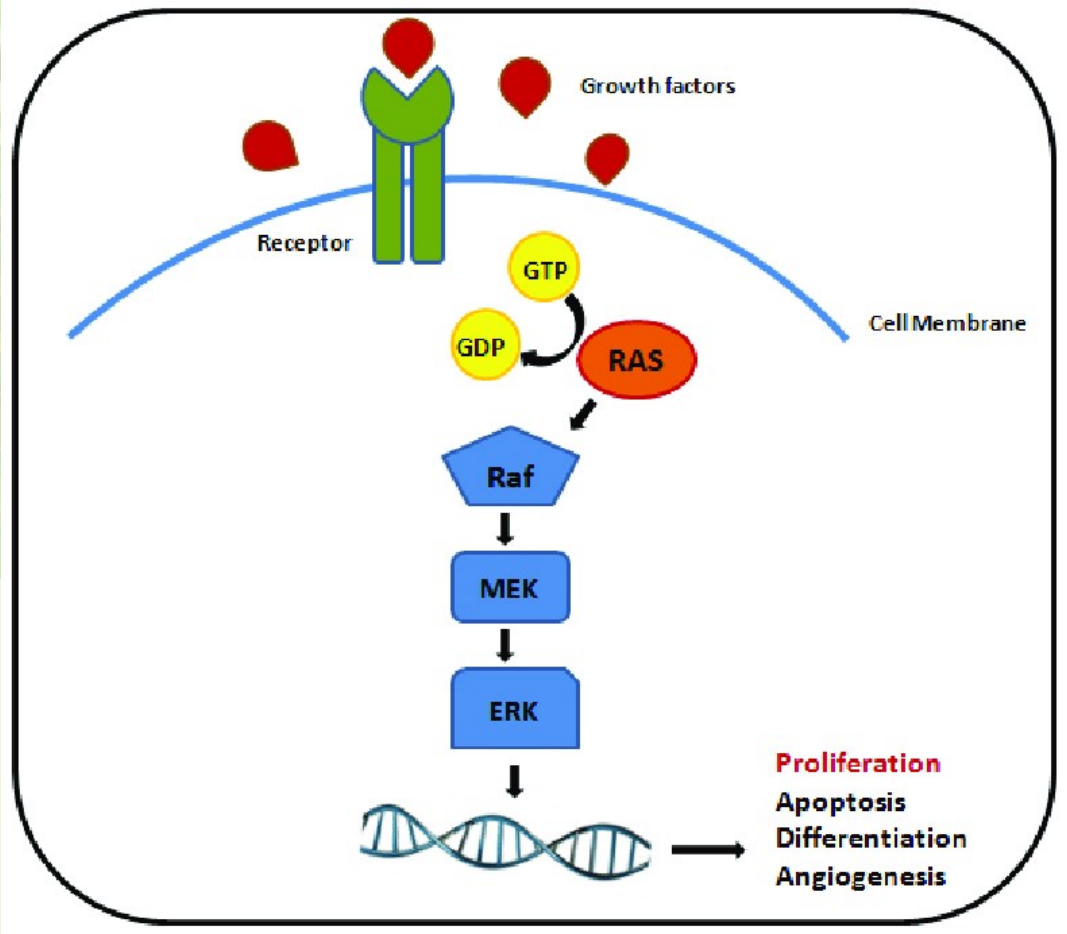
Ras-Raf-Mek-Erk signaling pathway

- Unlimited cell proliferation, dedifferentiation and a lack of apoptosis are important biological characteristics of tumors
- The activation of the ERK/MAPK signaling pathway promotes proliferation and has an anti-apoptotic effect
- Hypoxia-induced VEGF can inhibit the apoptosis of serum-starved cells by activating the ERK/MAPK signaling pathway
- Inhibiting the expression of this pathway can inhibit the proliferation of and lack of apoptosis in tumor cells and promote their differentiation.
- ERK1/2 signaling pathway is involved in cell survival following intestinal injury, and inhibition of this pathway can promote the apoptosis of intestinal injury cells
- Blocking the ERK/MAPK signaling pathway inhibited the proliferation of a diffuse large B cell lymphoma cell line and promoted cell apoptosis
- Inhibiting the expression of the ERK/MAPK signaling pathway to inhibit tumor cell proliferation may involve inhibition of the cell cycle



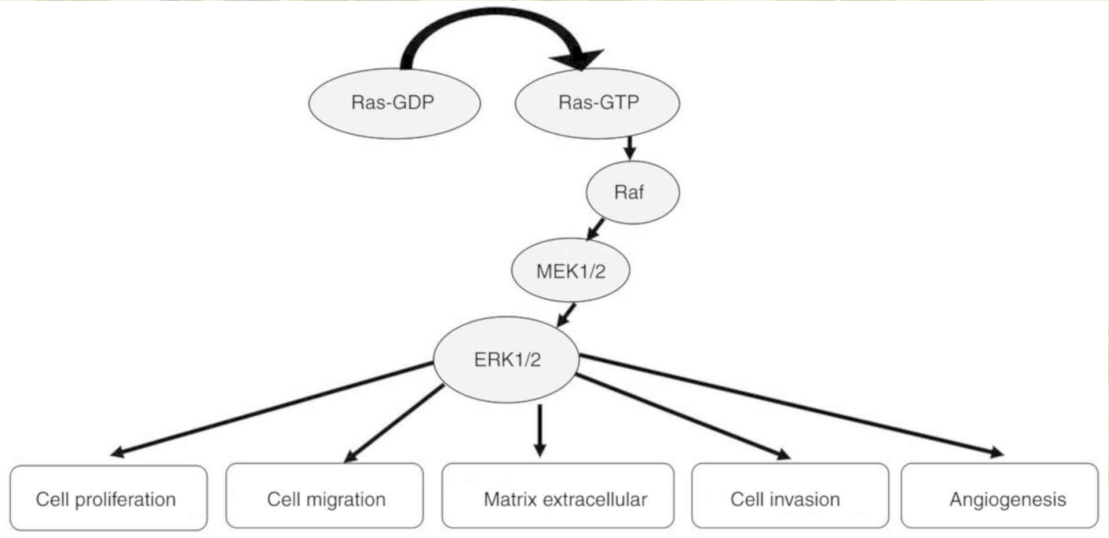
Ras-Raf-Mek-Erk signaling pathway

- The use of MEK1/2 inhibitors to inhibit ERK1/2 activity in colon cancer cells could prevent the cells from entering the S phase from the G1 phase and inhibit the growth of adherent cells
- Inhibition of the ERK/MAPK signaling pathway can reduce cell dedifferentiation and the anti-apoptosis effect
- ERK/MAPK signaling pathway promotes proliferation and inhibits apoptosis by influencing the activity of downstream cell cycle regulatory proteins, apoptosis-related proteins and other effector molecules, such as G1/S specific cyclin D1
- Gonadotropin-releasing hormone induces activation of the MAPK signaling pathway in normal and carcinoma cells of the human ovary and placenta
- SPACRC-like protein 1 (SPARCL1) is overexpressed in ovarian cancer; by inhibiting activation of the MEK/ERK signaling pathway, SPARCL1 is downregulated through the MEK/ERK pathway and inhibits the proliferation and migration of ovarian cancer cells



Raf-Mek-Erk signaling pathway

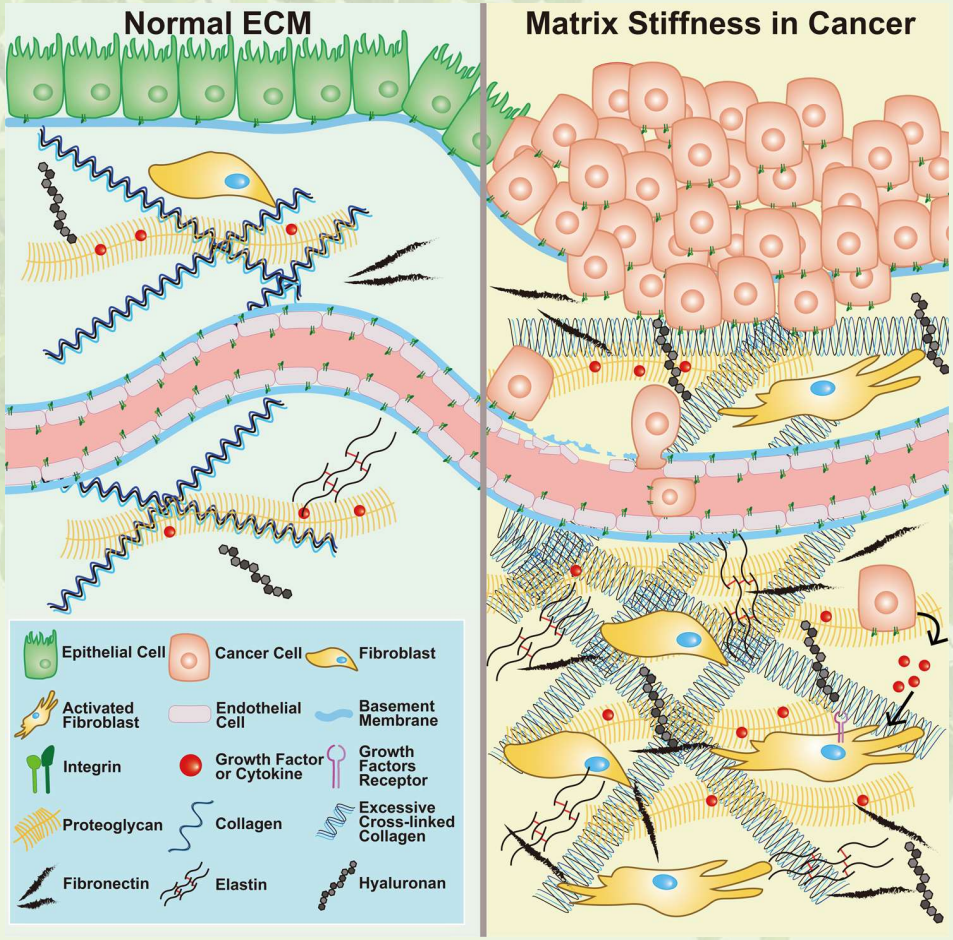
- Activation of ERK/MAPK signaling pathways activates other extracellular signaling pathways. Extracellular signals such as vascular endothelial growth factor (VEGF), platelet-derived growth factor and EGF can be activated by receptor tyrosine kinase autologous phosphorylation of the ERK/MAPK signaling pathway. Activated ERK may enter the nucleus and bind to transcription factors that induce gene expression in response to extracellular stimuli, and regulate cell proliferation, differentiation, apoptosis and transcription
- The ERK/MAPK signaling pathway is not only involved in regulating cellular biological functions, such as cell proliferation, cell differentiation, cell cycle regulation, cell apoptosis and tissue formation, but is also related to tumor formation. Elevated ERK expression has been detected in various human tumors, such as ovarian, colon, breast and lung cancer.
- The expression of MAPK phosphatase-1 (MKP-1) in normal ovarian surface epithelium and benign cystadenomas is increased compared to invasive carcinomas and low malignancy potential tumors and borderline tumors. Abnormal expression of MKP-1 and ERKs may play a role in the development of ovarian cancer.
- Continuous activation of the ERK/MAPK signaling pathway can promote the transformation of normal cells into tumor cells, while inhibition of the ERK/MAPK signaling pathway can restore tumor cells to a non-transformed state *in vitro* and can inhibit tumor growth *in vivo*
- Therefore, increased activation of the ERK/MAPK signaling pathway may be closely related to the occurrence and development of tumors



Extracellular Matrix

- Fibroblasts are regulated by many signals, including cytokines, chemicals, and environmental signals. TNF-α and interleukin (IL)-1 can induce the production of MMP-1, -3, and -9 by fibroblasts, leading to the degradation of collagen in the ECM

- Furthermore, ECM degradation contributes to the release of growth factors and cytokines. During tumorigenesis, MMP-2 and MMP-9 are upregulated in human colorectal cancer, and growth factors released from ECM cleaved by MMPs would promote tumor progression. For example, the VEGF is released when heparan sulfate is degraded, and such process promotes angiogenesis in colorectal carcinoma



Overexpressed molecules in the tumor microenvironment (ECM)

- MMPs: can release cytokines and fibroblast growth factors and vascular endothelial growth factors as well as degrade the ECM
- Transforming Growth Factor beta
- Cancer Associated Fibroblast (CAF)
- COX2 enzyme upregulation
- Interleukin 1beta upregulation
- Interleukin 6 upregulation
- CD44 adhesion molecule overexpression involved in metastasis

Proteolytic Enzymes

One of Nature's Answers to Cancer

Potential to impact:

- Cell cycle checkpoints
- Inhibit phosphorylation in cellular pathways that lead to proliferation
- Inhibit phosphorylation of Nf Kappa beta blocking COX2 stimulation
- Increase apoptotic signals
- Decrease anti-apoptotic signals
- Decrease proinflammatory cytokines
- Inhibit cell adhesion molecules that helps tumor metastasis
- Increase autophagy

PROTEASE

Transformation's most therapeutic systemic proteolytic formula

This proprietary blend of highly active, GI stable proteolytic enzymes has been combined to promote circulation, a strong healthy immune system, reduced inflammation, and timely detoxification.

Product Highlights

- Endo/exo peptidases break the inner/terminal bonds of amino acid chains for more efficient hydrolysis of proteins
- Protease blend (including bromelain plant enzymes) for reducing inflammation
- More than 400,000 HUT for the highest proteolytic activity available (600,000 PU = 51,000 HUT)
- 18 SAPU units from Protease 3.0
- Approx. 2,400 FU breaks down fibrin and clots and promotes healthy blood flow
- Calcium improves tolerance on an empty stomach

SUPPLEMENT FACTS

Serving Size 1 Capsule

Amount Per Serving		% Daily Value
Tzyme™ Protease Blend (peptidases, bromelain) (355,020 HUT + 19 SAPU) (600,000 PU)	492 mg	†

† Daily Value not established

Other Ingredients: Vegetable Capsule (Hypromellose, Water), Calcium Citrate

Clinical Applications

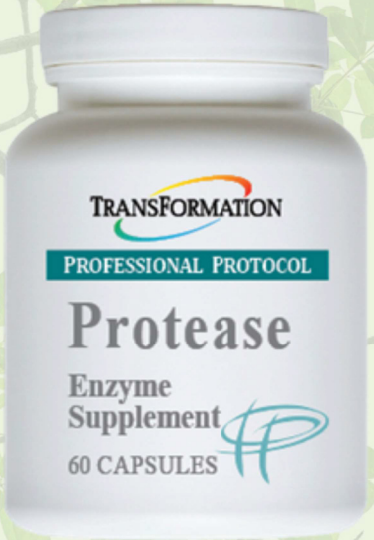
- Cancer of any kind
- Arthritis
- CVD / heart disease
- Chronic Fatigue Syndrome / Fibromyalgia
- Bacterial / Viral / Fungal Infections
- Hepatitis
- Kidney Disorders / Renal Insufficiency
- Eczema / Psoriasis
- Asthma / Emphysema
- All hormone imbalances
- Auto-immune disorders
- Autism
- Diabetes
- Muscle strains, soreness, injuries, and surgeries

For Your Information

- First choice when patient has been diagnosed with a condition (this is the "therapeutic" strength blend)
- It is better to take small doses of protease frequently throughout the day rather than large doses once or twice a day (protease has a half-life of approximately 3-4 hours; the goal is to keep the protease activity constant in the blood stream for therapeutic benefits)
- Compares with Nattokinase and Serra-peptidase
- May be given to children if condition warrants; may be given to pets
- Caution with patients on prescription blood thinning drugs (give protease formulas 3-4 hours away from Rx dose)
- May cause discomfort for individuals with stomach ulcers as protease will debride necrotic tissue and promote healing
- Discontinue taking Protease 24-48 hours prior to surgery and resume 24 hours post-surgery

Dosage

- Maintenance dose: 1 capsule 3 x day (rise, midday, rest) on an empty stomach
- General therapy dose: 2 capsules 3 x day between meals (approximately 1 hour before or 2 hours after meals)
- Therapeutic dose: 2-3 capsules 4-5 x day, or every 3 hours (goal is to keep high levels of activity in the blood stream at all times; "between meals" becomes difficult, so 30-60 minutes before or after meals is acceptable)
- **MAY BE TAKEN WITH FOOD IF UNABLE TO TOLERATE BETWEEN MEALS**
- Sometimes it is suggested 1 capsule may be taken with meals to enhance the digestion of proteins (diabetes, heartburn, acid reflux, gout, autism, high protein diet)
- For those who have difficulty swallowing pills, the capsules may be pulled apart and mixed in a small amount of tepid water or liquid and consumed immediately
- Topical application: open capsule and make a paste with a small amount of water, then apply to insect bite, fungal rash, mouth sores, inflamed gums, etc



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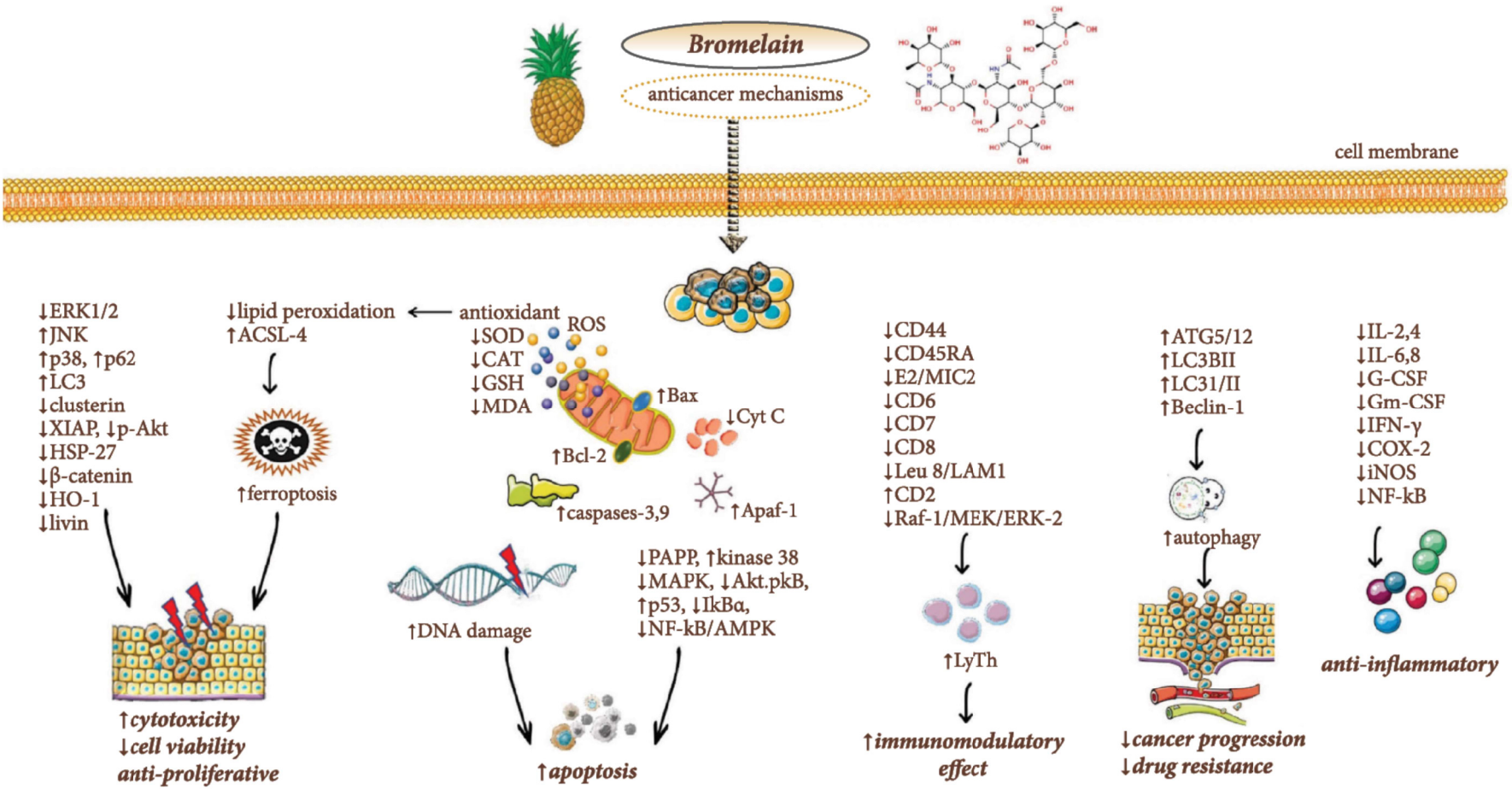
ANTICANCER MOLECULAR MECHANISMS OF BROMELAIN



Bromelain

- An enzyme from pineapple
- A mixture of different thiol endopeptidases and other components like phosphatase, glucosidase, peroxidase, cellulase, escharase, and several protease inhibitors
- In vitro and in vivo studies demonstrate that bromelain exhibits various fibrinolytic, antiedematous, antithrombotic, and anti-inflammatory activities
- Considerably absorbable in the body without losing its proteolytic activity and without producing any major side effects

Antitumor activity of Bromelain in the literature

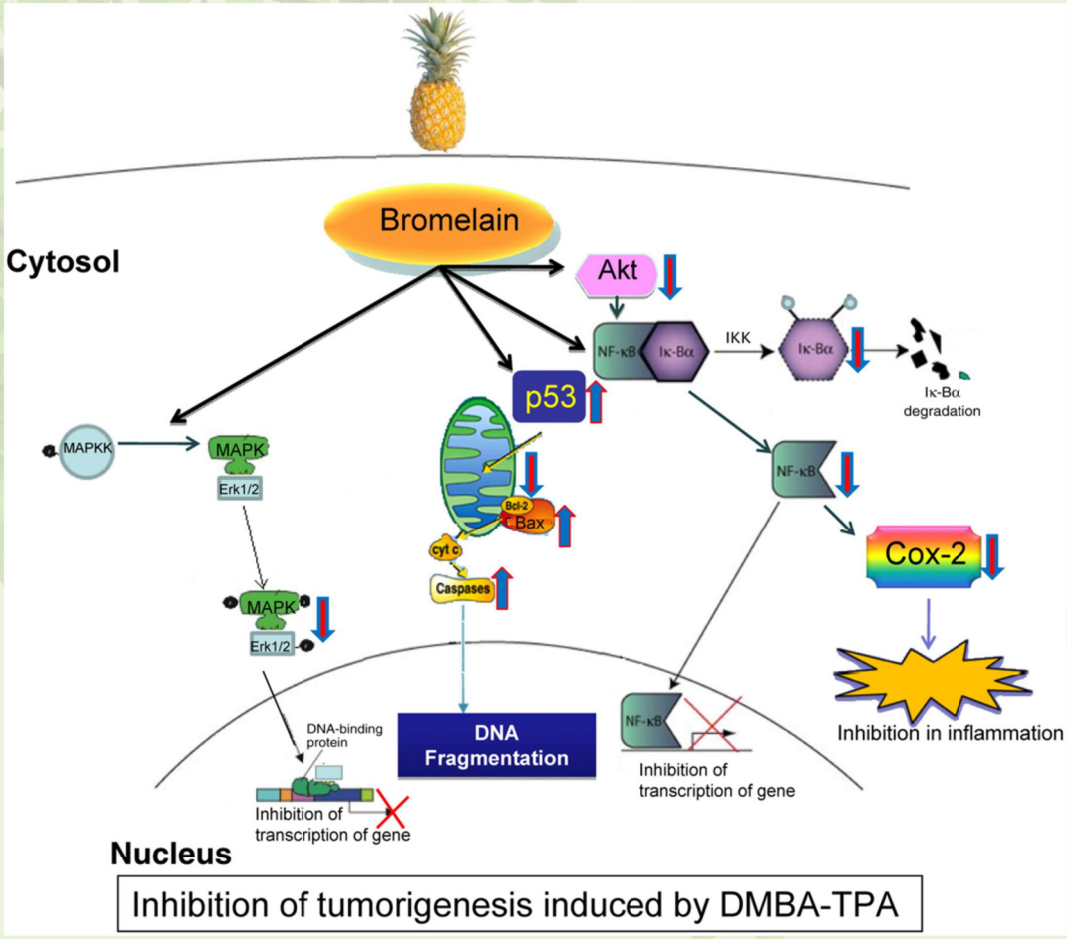
- Bromelain inhibits COX-2 expression by blocking the activation of MAPK regulated Nfkappa beta against skin tumor initiation triggering mitochondrial death pathway
- Bromelain, from Pineapple Stems, Proteolytically Blocks Activation of Extracellular Regulated Kinase-2 in T Cells
- Pineapple Bromelain induces Autophagy, facilitating apoptotic response in mammary carcinoma cells
- Regulation of p53, nuclear factor κB and cyclooxygenase-2 expression by bromelain through targeting mitogen-activated protein kinase pathway in mouse skin

Antitumor activity of Bromelain in the literature

- Bromelain treatment resulted in upregulation of p53 and Bax and subsequent activation of caspase 3 and caspase 9 with concomitant decrease in antiapoptotic protein Bcl-2 in mouse skin.
- Since persistent induction of cyclooxygenase-2 (Cox-2) is frequently implicated in tumorigenesis and is regulated by nuclear factor-kappa B (NF-κB), we also investigated the effect of bromelain on Cox-2 and NF-κB expression
- Results showed that bromelain application significantly inhibited Cox-2 and inactivated NF-κB by blocking phosphorylation and subsequent degradation of IκBα

Protease effects on cell signaling

In the present study, antitumorigenic activity of bromelain was recorded in DMBA-TPA-promoted 2-stage mouse skin model. Results showed that bromelain application delayed the onset of tumorigenesis and reduced the cumulative number of tumors, tumor volume and the average number of tumors/mouse. Bromelain treatment resulted in upregulation of p53 and Bax and subsequent activation of caspase 3 and caspase 9 with concomitant decrease in antiapoptotic protein Bcl-2 in mouse skin. Since persistent induction of cyclooxygenase-2 (Cox-2) is frequently implicated in tumorigenesis and is regulated by NF-κB, we also investigated the effect of bromelain on Cox-2 and NF-κB expression. Results showed that bromelain application significantly inhibited Cox-2 and inactivated NF-κB by blocking phosphorylation and subsequent degradation of IκBα. In addition, bromelain treatment attenuated phosphorylation of extracellular signal regulated protein kinase (ERK1/2), mitogen-activated protein kinase (MAPK) and Akt. Taken together, we conclude that bromelain induces apoptosis-related proteins along with inhibition of NF-κB-driven Cox-2 expression by blocking the MAPK and Akt/protein kinase B signaling in DMBA–TPA-induced mouse skin tumors, which may account for its anti-tumorigenic effects.



Proteolytic Enzyme | Health Benefits

- Systemic enzyme therapy has been shown to overcome the “cytokine storm” or “immunosuppression” seen in infections and cancer to salvage the host’s immune system
- Enzymes aid in destruction of cancer cells by activating death signals
- Enzymes activate alpha-2 macroglobulin, the “cytokine catcher” which usually exists in blood in an inactive form. Upon activation, this macroglobulin has high affinity for the TGF beta cytokine present in high levels in cancer
- Enzymes selectively reduce expression of CD44 adhesion molecules which encodes for metastasis
- Reduces proinflammatory cytokines such as IL6, IL1 beta, IL12, TNF alpha, NF kappa beta, and the COX2 enzyme upregulated in the tumor microenvironment

CASE STUDY

- We have a 71yr. old male with a diagnosis of Prostate Adenocarcinoma confirmed by biopsy, MRI, Lymphadenopathy, and elevated PSA
- Pt's. family history includes Prostate cancer for his father and brother. Dad died of a heart weakened by radiation received for prostate cancer in the early 90's
- Pt. denies any allergies to drugs, foods, or environmental triggers and has unremarkable past medical history. Pt. states he feels well and has no symptoms. He is willing to make lifestyle changes and comply with our recommended protocol for his condition identified as a tumor
- His immediate concerns are the tumor on his prostate and elevated PSA
- The goal of our protocol is to reduce the prostate tumor significantly, bring PSA levels to normal <4ng/mL, eliminate the large pelvic lymph nodes, and reduce the Detrusor muscle hypertrophy observed in the bladder upon MRI.

CASE STUDY

BIOPSY REPORT 12/14/22:

- Site A: No. of cores:1; Dimensions: 13 mm (left lateral base)
- Diagnosis Summary: PROSTATIC ADENOCARCINOMA; Gleason Score 7(4+3); 1 of 1 core involved; Tumor measures 7 mm in length; Microscopic Description: 90% of Gleason pattern 4.
- Site B: No. of cores: 3; Dimensions: 10,6,4 mm (left lateral mid)
- Diagnosis Summary: PROSTATIC ADENOCARCINOMA; Gleason Score 8(4+4); 3 of 3 cores involved; Tumor measures 13 mm in length; Microscopic Description: 100% of Gleason pattern 4.
- Site C: No. of cores: 2; Dimensions: 20,11mm (left lateral apex)
- Diagnosis Summary: Benign soft tissue. No prostatic glands present.
- Site D: No. of cores: 1; Dimensions: 14 mm (right lateral base)
- Diagnosis Summary: PROSTATIC ADENOCARCINOMA; Gleason Score 8(4+4); 1 of 1 core involved; Tumor measures 13 mm in length; Microscopic Description: 100% of Gleason pattern 4.
- Site E: No. of cores: 1; Dimensions: 13 mm (right lateral mid)
- Diagnosis Summary: PROSTATIC ADENOCARCINOMA; Gleason Score 8(4+4);1 of 1 core involved; Tumor measures 13 mm in length; Microscopic Description: 100% of Gleason pattern 4.
- Site F: No. of cores: 2; Dimensions: 10,8 mm (right lateral apex)
- Diagnosis Summary: PROSTATIC ADENOCARCINOMA; Gleason Score 8(4+4); 2 of 2 cores involved; Tumor measures 12 mm in length; Microscopic Description: 100% of Gleason pattern 4.
- Case Comments: Large cribriform glands present. This case has been reviewed in an Intradepartmental conference and all participating pathologists agree with the above diagnoses.

CASE STUDY

PSA and HS CRP 10/07/22:

- PSA: 336 ng/mL
- Hs CRP: >10mg/L
- MRI REPORT 11/08/22: MRI Prostate with and without contrast on high field 3.0 Tesla showed:
- Prostate volume: 51 .69 cc
- Prostate dimensions: 5.2 x 5.2 x 3.9 cm
- Transition Zone: Central gland hypertrophy and large tumor measuring 15x29x30mm (APxMLxCC) at the posterior apex and mid prostate gland. The lesion extends posteriorly and inferiorly from the prostate capsule and is in close proximity to the anterior low rectum.
- Peripheral Zone: In the right mid posterior peripheral zone there is a markedly hypointense ADC signal measuring 14x8x12mm and increase permeability.
- Seminal Vesicles: the seminal vesicles are normal and symmetrical bilaterally.
- Extracapsular extension: the bilateral neurovascular bundles are not well defined and underlying invasion is within the differential.
- Bladder: there is detrusor muscle hypertrophy.
- Lymph Nodes: Mildly enlarged oval-shaped lymph node in the right pelvic sidewall measuring 9x7mm with a few other smaller lymph nodes also identified at this location.
- Other: heterogenous hypointense lesion measuring 11x10mm at the right pubic symphysis.

CASE STUDY

- 12/7/22: Initial consultation, the patient had already been diagnosed by his Oncologist and provided lab testing and biopsy as shown above. The patient had begun taking a set of supplements when he learned of his diagnosis to start fighting his war against cancer. He was allowed to stay on several of them as I didn't see any contraindications with my protocol, and I didn't want to affect his belief in what he had started. After consent and consultation, the patient was recommended a treatment protocol of Protease IFC 2 caps between meals 3 times a day, Protease powder 1 teaspoon between meals 3 times a day (15 grams), L-Drain 1 dropper full in 8oz glass 3 times a day, Probiotic 42.5 1 cap in the morning and 1 cap at night along with a diet which excluded GMO's, gluten, dairy, and any artificial processing. Pt. initially had no adverse effects or discomfort in taking 15 grams of Protease Powder.
- 1/4/23: I recommended continuing the rest of the supplements at the same dose and only increasing the dose of the Protease Powder to 10 grams 3 times a day (30 grams) on an empty stomach until the end of the study on May 17th, 2023, which ran for a total of 23 weeks.

CASE STUDY

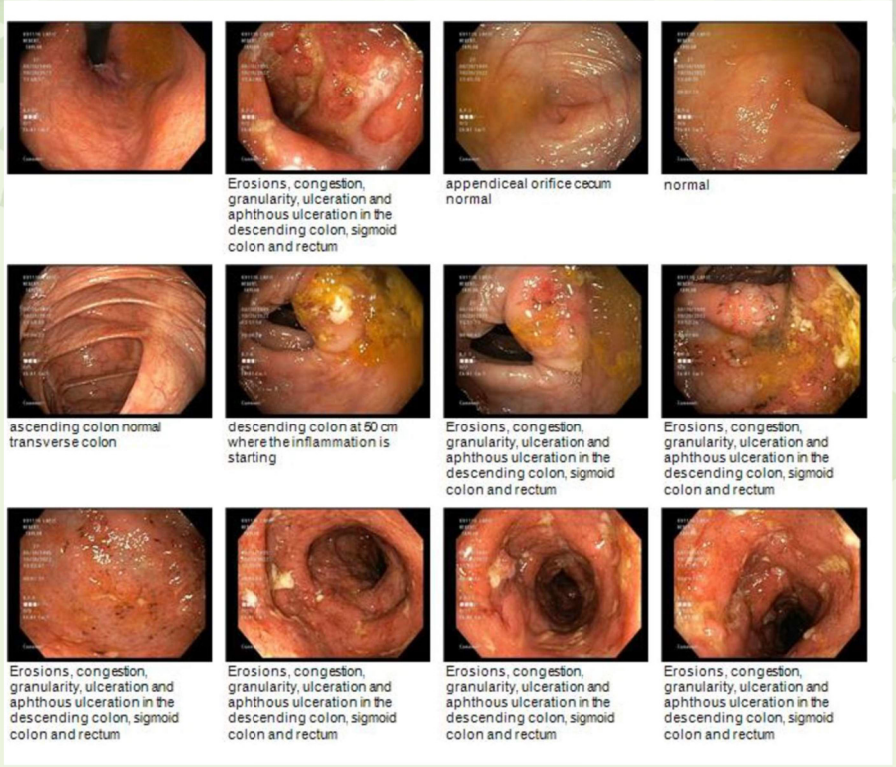
- 2/3/23: by the direction of his Oncologist the patient began Testosterone blockers with one shot of Lupron depot
- 2/16/23: started Zytiga with Prednisone
- 3/3/23: he began Orgivyx for blocking Testosterone plus Prednisone plus Zytiga and was told that with these blockers he should see PSA go down within 4 to 6 months (July to September)
- 5/15/23: PSA was already 0.6 ng/mL (2 to 4 months earlier than expected by Oncologist) which could be due to the mega dose of proteolytic enzymes the patient had been taking since December.

CASE STUDY

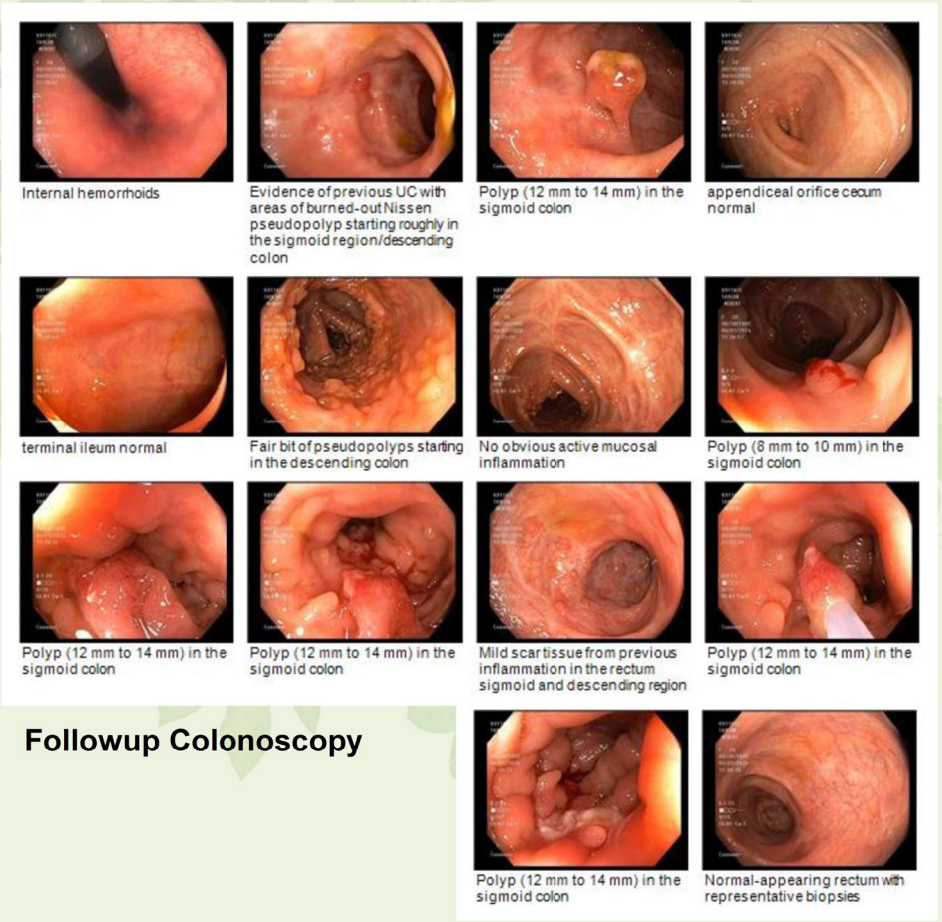
Follow up MRI and blood tests are as follows:

- MRI FINDINGS 5/15/23:Prostate volume: 15.54 cc down from 51.69 cc on previous MRI.
- Prostate dimensions: 3.4 x 3.0 x 3.0 cm down from 5.2 x 5.2 x 3.9 cm on previous MRI.
- PSA: 0.6 ng/ml. previously 336 on the 11 /8/2022 examination.
- Transition Zone: The transitional zone is low in T2 signal intensity. The central zone demonstrates low ADC signal symmetrically. If additional biopsy is indicated, the right central zone demonstrates focal moderate hypointense ADC and mild hyperintensity DWI, without increased permeability and measures greater than 1.5 cm in size (PI-RADS 3). This demonstrates no increased permeability.
- Peripheral Zone: Since the prior exam, the prostate is smaller in appearance with more diffuse decreased T2 signal, most consistent with posttreatment change. There is persistent low signal in the posterior apex on T2-weighted imaging, without associated low ADC. By strict scoring, this likely represents a PI-RAD 2 lesion, but is at the known site of prior PI-RADS 5 lesions, consistent with posttreatment response.
- Seminal vesicles: The seminal vesicles are normal and symmetrical bilaterally.
- Extracapsular extension: The prostatic capsule is preserved. The neurovascular bundles are intact. There is no evidence of tumor in the rectal prostatic angles.
- Bladder: Normal.
- Lymph nodes: The previously visualized areas of right pelvic sidewall adenopathy have resolved. No abnormally enlarged pelvic lymph nodes.
- IMPRESSION:
- PI-RADS: Category: post treatment change with visible reduction in size and restricted diffusion in the posterior apical known prostate cancer. The overall prostate size is reduced in the signal intensity is decreased nearly diffusely on T2-weighted imaging. The pelvic lymphadenopathy has also resolved. There is no focal area in the peripheral zone meeting current PIRADS 4 or 5 rating. The inferior right pubic ramus lesion appears unchanged. The central zone demonstrates restricted diffusion but appears symmetric and is indeterminant (PI-RADS 3).

CASE STUDY | Ulcerative Colitis



Initial Colonoscopy



Followup Colonoscopy

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