Whole-Person Care in Cancer: The Power of Transformation… Physical, Emotional & Spiritual HEALING

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Integrative Personalized Medicine

LONDON

June 19, 2026

The JOURNEY

The JOURNEY

The JOURNEY

The Healing Journey


ELEKTA

The Metabolic Basis for Cancer

Typical Cellular Energy Production
REVIEW

Oxidative phosphorylation
Cellular Compartment: Mitochondria
Requires O2 (aerobic)
Generates 36 molecules of ATP

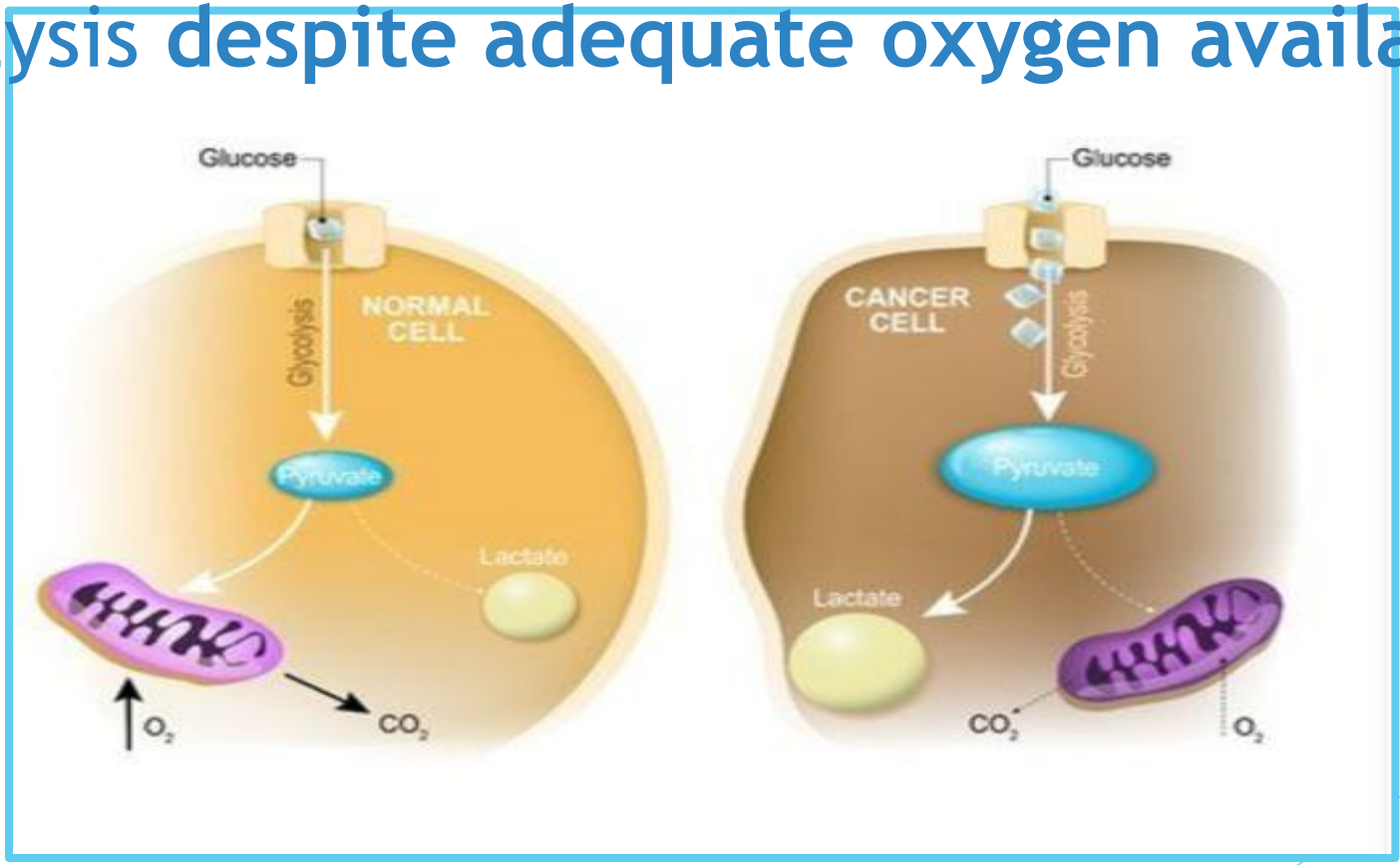
Glycolysis
Cellular Compartment: Cytoplasm
Does Not Require O2 (anaerobic)
Generates 2 molecules of ATP

In the presence of adequate oxygen, ATP is generated by oxidative phosphorylation (the more energetically efficient method).

As oxygen becomes less available, glycolysis will become the more prominent method of energy production

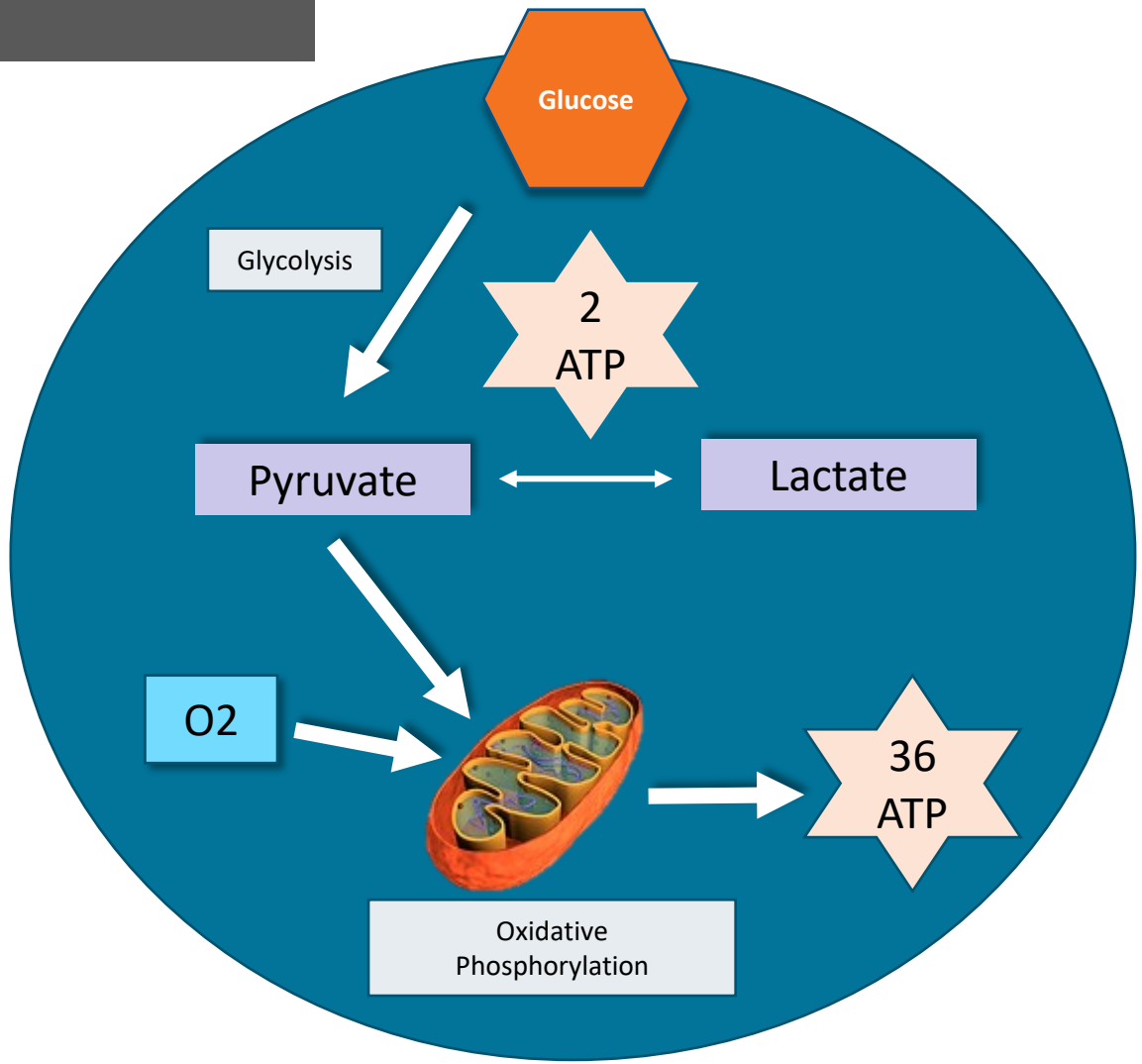
The Warburg Effect

In 1924, German physiologist Otto Warburg discovered that cancer cells exhibit increased rates of glycolysis **despite adequate oxygen availability**



Vander Heiden MG, Cantley LC, Thompson CB. Understanding the Warburg effect: the metabolic requirements of cell proliferation. *Science*. 2009;324(5930):1029–1033. doi:10.1126/science.1160809

NORMAL CELL



1. Glucose enters cell

2. Glucose is converted to pyruvate via glycolysis

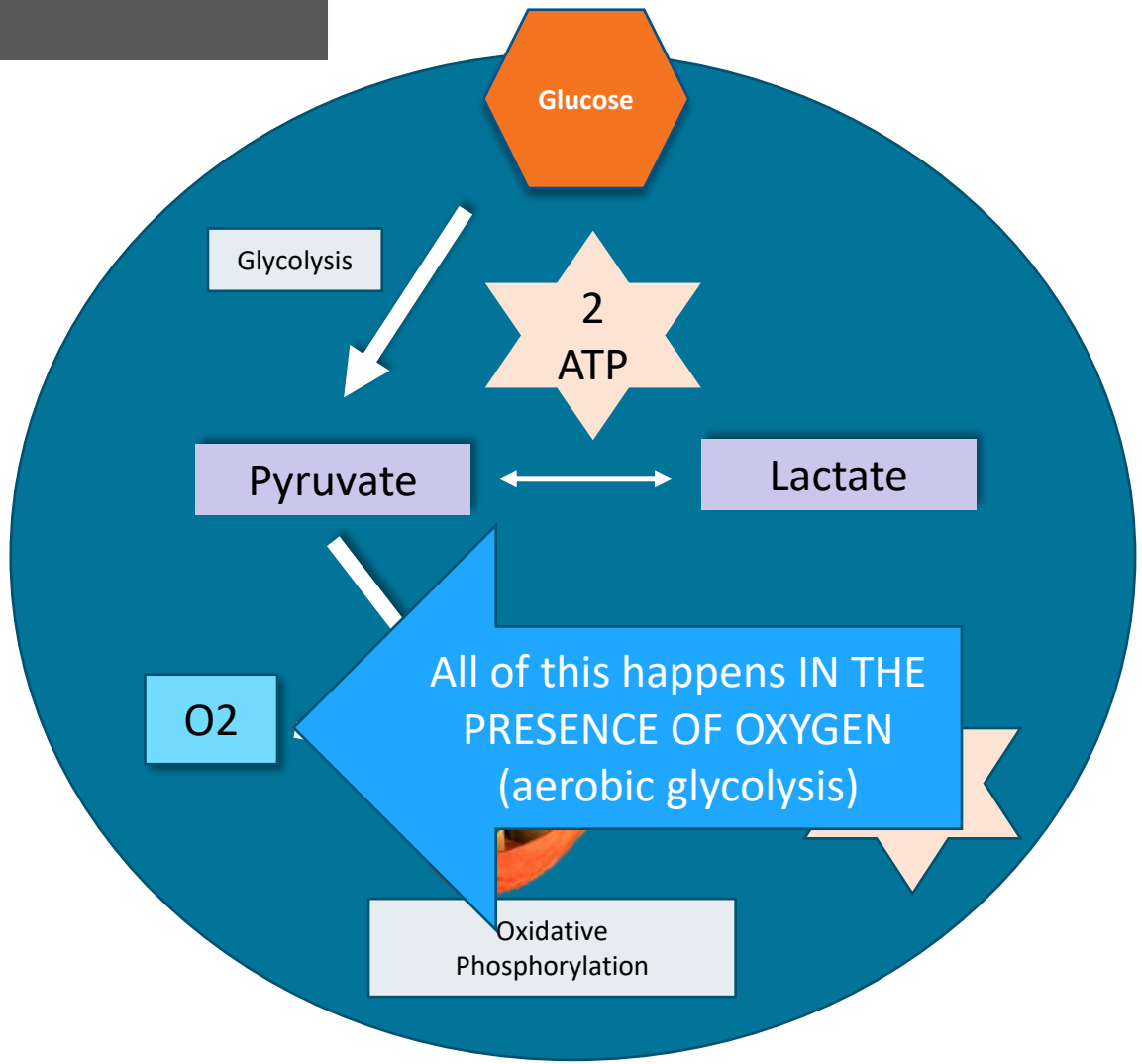
3. A very small percentage of pyruvate will be converted to lactate

4. In the presence of oxygen, pyruvate enters the mitochondria to undergo oxidative phosphorylation

5. 36 ATP are generated and exported out of the cell → very efficient energy production!

See references: Warburg animation

CANCER CELL



1. In cancer cells, energy production shifts away from oxidative phosphorylation, favoring glycolysis

2. Glycolysis alone generates 2 ATP → less ATP than ox phosphorylation BUT does not rely on mitochondria

3. Cancer cells become hungry for energy and start taking up several times more glucose than normal cells

4. Increased rate of glycolysis creates more pyruvate, which is then converted to lactate in larger concentrations

5. Lactate lowers extracellular pH (favorable for tumor proliferation) and drives angiogenesis by promoting VEGF

See references: Warburg animation

Calories, Carbohydrate, and Cancer

- Cancer cells are heavily reliant on glycolysis
- Increased glycolysis results in influx of glucose and lactate production
- Tumor cells have increased ability to resist radiation therapy in the presence of elevated glucose
- Poorer prognosis in patients with metabolic issues (ie obesity, diabetes, hyperinsulinemia, elevated IGF-1)

Klement RJ, Champ CE. Calories, carbohydrates, and cancer therapy with radiation: exploiting the five R's through dietary manipulation. Cancer Metastasis Rev. 2014 Mar;33(1):217-29. doi: 10.1007/s10555-014-9495-3.

How do we shift our cellular metabolism?

Fasting

Caloric Restriction - Definitions

- Caloric restriction is generally defined as a 30-50% reduction in energy intake, without malnutrition
- Methods for restricting calories vary:
  - Intermittent fasting
  - Water-only, short-term fasting
  - Daily energy restriction
  - Fasting Mimicking Diet

Klement RJ, Champ CE. Calories, carbohydrates, and cancer therapy with radiation: exploiting the five R's through dietary manipulation. Cancer Metastasis Rev. 2014 Mar;33(1):217-29. doi: 10.1007/s10555-014-9495-3.

Differential Stress Resistance (DSR)

- The specific response of healthy cells versus cancer cells to short term fasting is known as **differential stress resistance**

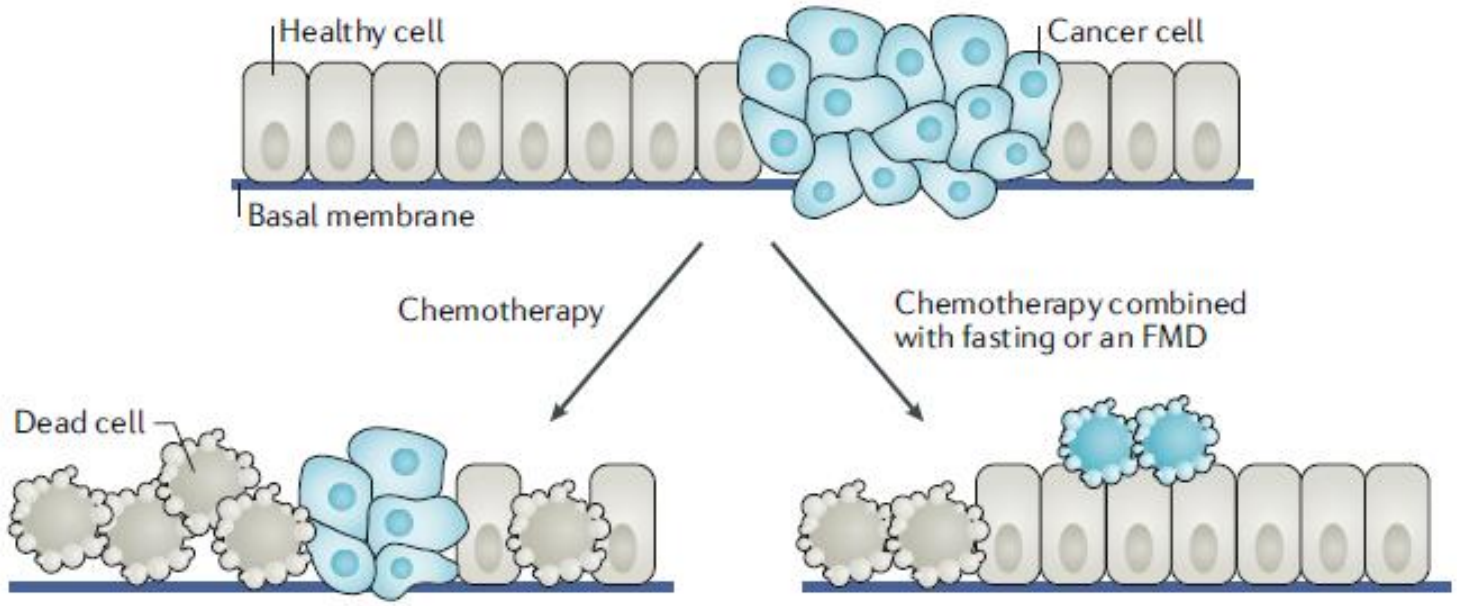
- Fasting forces healthy cells to enter a slow division process in order to re-invest energy into maintenance and repair, which is protective against the toxic effects of anticancer drugs

- Cancer cells are unable to decrease growth and metabolism due to mutations in tumor suppressor genes and are more susceptible to nutrient deprivation (particularly glucose)

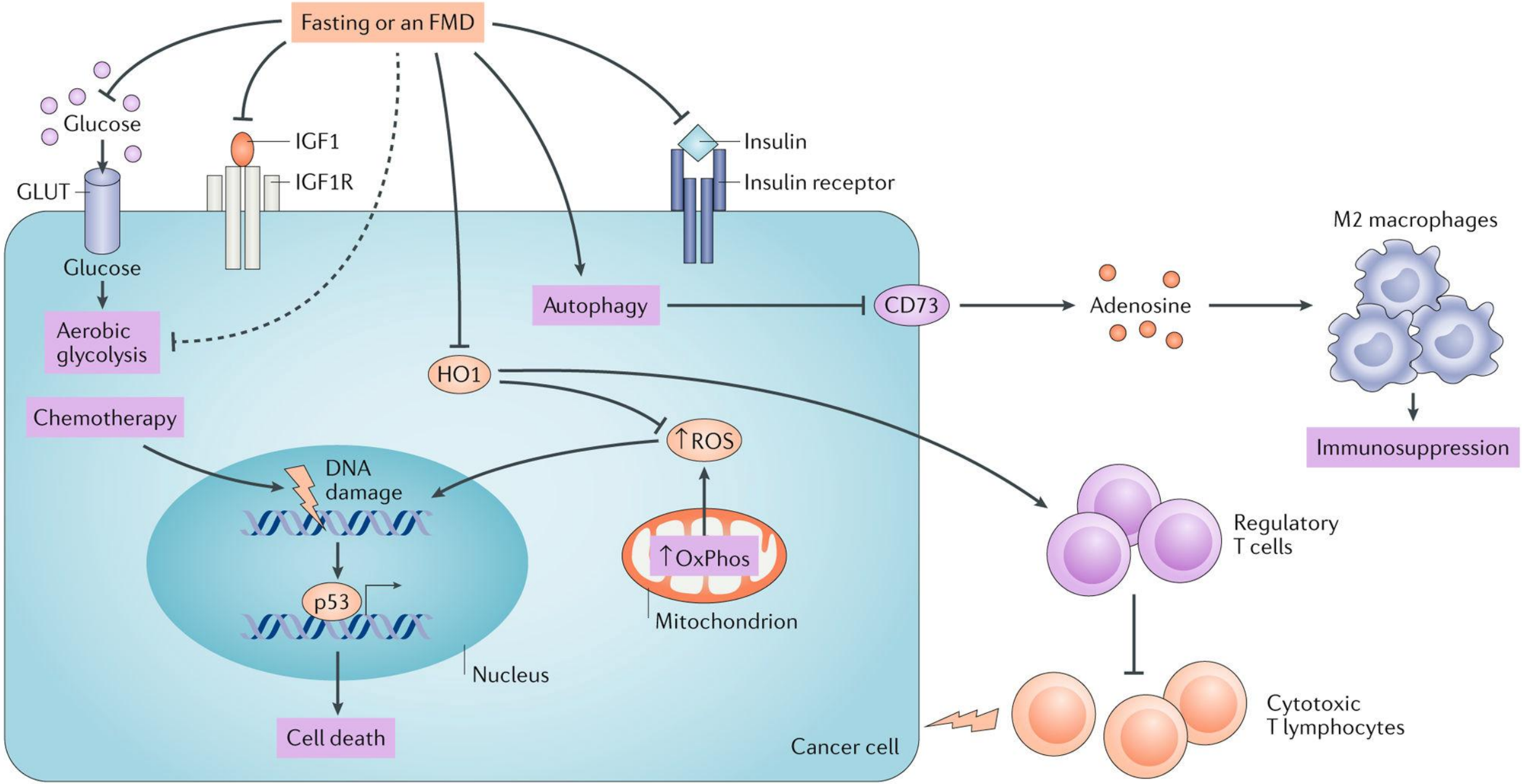
de Groot, S., Pijl, H., van der Hoeven, J.J.M. *et al.* Effects of short-term fasting on cancer treatment. *J Exp Clin Cancer Res* **38,** 209 (2019) doi:10.1186/s13046-019-1189-9

Fasting protects healthy cells from the effects of chemotherapy

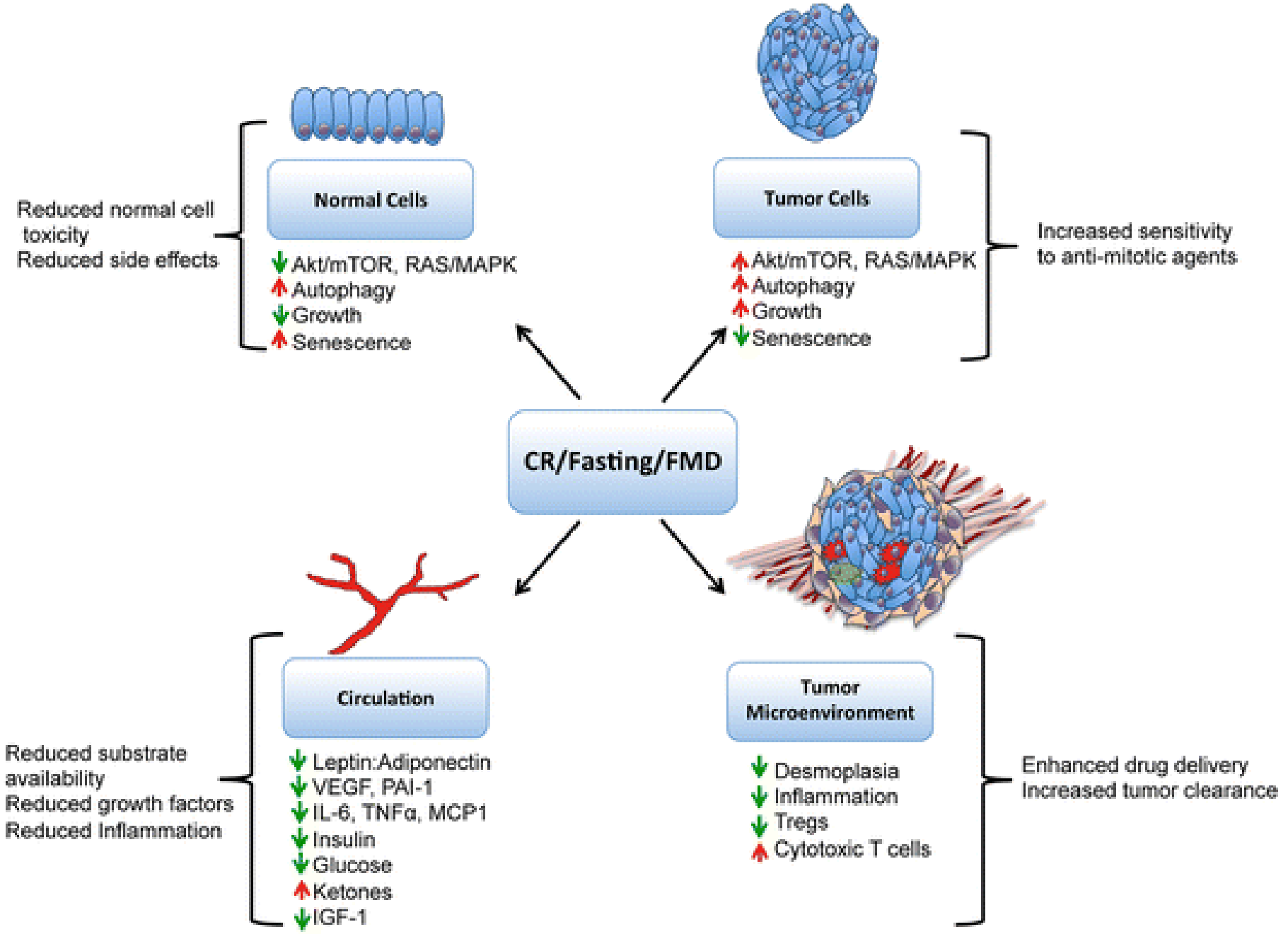
"As a consequence of these differential responses of healthy versus cancer cells to short term fasting, chemotherapy causes more DNA damage and apoptosis in tumor cells, while leaving healthy cells unharmed. Thus, short term fasting protects healthy cells against the toxic properties of chemotherapy and renders tumor cells more sensitive, a phenomenon called differential stress sensitization (DSS)."



Nencioni A, Caffa I, Cortellino S, Longo VD. Fasting and cancer: molecular mechanisms and clinical application. Nat Rev Cancer. 2018;18(11):707–719. doi:10.1038/s41568-018-0061-0



Nencioni A, Caffa I, Cortellino S, Longo VD. Fasting and cancer: molecular mechanisms and clinical application. Nat Rev Cancer. 2018;18(11):707–719. doi:10.1038/s41568-018-0061-0



O'Flanagan, C.H., Smith, L.A., McDonell, S.B. *et al.* When less may be more: calorie restriction and response to cancer therapy. *BMC Med* 15, 106 (2017) doi:10.1186/s12916-017-0873-x

Strategies for Chemo-Prep Fasting

Few published studies:

- 48hr starvation (water fast) 24hr before and 24hr after chemotherapy
  - Decreased hematological toxicity and accelerated recovery from DNA damage
  - Breast CA patients receiving multi-modal ChemoTx[1]
- 0/24/48 hours fast (<200 cal/d) before and 24hr after chemotherapy
  - Decreased neutropenia & reduced DNA damage
  - Urothelial, Ovarian & Breast CA receiving platinum-based ChemoTx[2]
- 36hr FMD (<400cal/ d) before and 24hr after chemotherapy
  - Improves quality of life and fatigue; no fasting AE reported
  - Breast and Ovarian CA patients receiving ChemoTx[3]

1. DeGroot S et al. BMC Cancer. 2015;15:652. doi:10.1186/s12885-015-1663-5.
2. Dorff TB et al. BMC Cancer. 2016;16:360.
3. Baursfield SP et al. BMC Cancer. 2018;18(1):476. doi:10.1186/s12885-018-4353-2.

Fasting during chemo decreases DNA damage

- Decreased DNA damage in white blood cells
- Decreased IGF1
- Better white blood cell counts
- Benefits are immediate
- 48 hours optimal time

Dorff TB, Groshen S, Garcia A, et al. Safety and feasibility of fasting in combination with platinum-based chemotherapy. *BMC Cancer*. 2016;16:360. Published 2016 Jun 10. doi:10.1186/s12885-016-2370-6

nature COMMUNICATIONS

ARTICLE

https://doi.org/10.1038/s41467-020-16138-3 OPEN

Fasting mimicking diet as an adjunct to neoadjuvant chemotherapy for breast cancer in the multicentre randomized phase 2 DIRECT trial

Stefanie de Groot[1], Rieneke T. Lugtenberg[1], Danielle Cohen[2], Marij J. P. Welters[1], Ilina Ehsan[1], Maaike P. G. Vreeswijk[3], Vincent T. H. B. M. Smit[2], Hiltje de Graaf[4], Joan B. Heijns[5], Johanneke E. A. Portielje[1,6], Agnes J. van de Wouw[7], Alex L. T. Imholz[8], Lonneke W. Kessels[8], Suzan Vrijaldenhoven[9], Arnold Baars[10], Elma Meershoek-Klein Kranenbarg[11], Marjolijn Duijm-de Carpentier[11], Hein Putter[12], Jacobus J. M. van der Hoeven[1], Johan W. R. Nortier[1], Valter D. Longo[13,14], Hanno Pijl[15], Judith R. Kroep[1✉] & Dutch Breast Cancer Research Group (BOOG)[16,*]

Short-term fasting protects tumor-bearing mice against the toxic effects of chemotherapy while enhancing therapeutic efficacy. We randomized 131 patients with HER2-negative stage II/III breast cancer, without diabetes and a BMI over 18 kg m^{-2}, to receive either a fasting

Ketogenesis & Cancer

“Ketogenic diet” requires drastic cut in carbohydrate AND protein intake

- Fat = 80-85%
- Protein = 10-15%
- Carbs = 5%

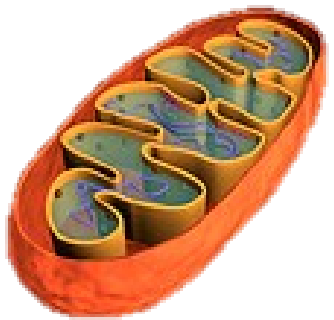
Why ketogenic for cancer?

First, let's review what happens in a "normal" scenario of CHO restriction:

Most tumor cells have mitochondrial abnormalities that prevent them from utilizing ketones for energy

When glucose levels are limited, normal cells in the brain, heart, and skeleton transition from the metabolism of glucose to the metabolism of ketone bodies for energy

Ketone bodies are produced by β-oxidation of fatty acids in the liver in order to compensate for glucose depletion



Ketone bodies are metabolized directly to acetyl CoA in mitochondria

1. Seyfried TN, Flores RE, Poff AM, D'Agostino DP. Cancer as a metabolic disease: implications for novel therapeutics. *Carcinogenesis*. 2014;35(3):515–527. doi:10.1093/carcin/bgt480

Why ketogenic for cancer?

A ketogenic diet:

- Decreases blood glucose and increases ketones
- Increases fatty acids (which can inhibit major glycolytic enzymes)
- Reduces lactate concentrations, potentially slowing tumor progression
- **Lowers insulin**

Klement RJ. Restricting carbohydrates to fight head and neck cancer-is this realistic?. *Cancer Biol Med*. 2014;11(3):145–161. doi:10.7497/j.issn.2095-3941.2014.03.001

β-OHB: fuel and signal

- Decreases inflammation
- Inhibits HDAC (induces apoptosis in cancer cells)
- Increases sirtuins
- Modulates circadian rhythm
- Increases innate immunity
- Increases seizure threshold
- Decreases insulin
- Modulates *PPAR-α, PPAR-γ…*

Insulin as a Driver of Tumor Proliferation

- There is evidence to support insulin resistance and hyperinsulinemia as independent risk factors for cancer development
- IGF-1 and insulin stimulate proliferation and growth and inhibit apoptosis in response to calorie and protein availability
- The expression of insulin receptors has been implicated as a risk factor for metastases in colorectal and endometrial cancers

- **The most common cancers associated with insulin resistance include breast, colorectal, endometrial, pancreatic, glioblastoma**

1. Tsujimoto T, Kajio H, Sugiyama T. Association between hyperinsulinemia and increased risk of cancer death in nonobese and obese people: A population-based observational study. *Int J Cancer*. 2017;141(1):102–111. doi:10.1002/ijc.30729
2. Perseghin G, Calori G, Lattuada G, Ragogna F, Dugnani E, Garancini MP,Crosignani P, Villa M, Bosi E, Ruotolo G, Piemonti L. Insulin resistance/hyperinsulinemia and cancer mortality: the Cremona study at the 15th year of follow-up. Acta Diabetol. 2012 Dec;49(6):421-8. doi: 10.1007/s00592-011-0361-2.

Enteral Feeding of Ketogenic Therapy

- High-CHO enteral formulas have been associated with increased mortality rates despite preservation of body weight
- In animal studies, ketogenic formulas suppress the progression of cancer and the accompanying systemic inflammation while preserving lean muscle, preventing cancer cachexia

1. Klement RJ. Restricting carbohydrates to fight head and neck cancer-is this realistic?. *Cancer Biol Med*. 2014;11(3):145–161. doi:10.7497/j.issn.2095-3941.2014.03.001
2. Nakamura K, Tonouchi H, Sasayama A, Ashida K. A Ketogenic Formula Prevents Tumor Progression and Cancer Cachexia by Attenuating Systemic Inflammation in Colon 26 Tumor-Bearing Mice. Nutrients. 2018 Feb 14;10(2). pii: E206. doi: 10.3390/nu10020206.

Dietary Approaches to Oncology SUMMARY

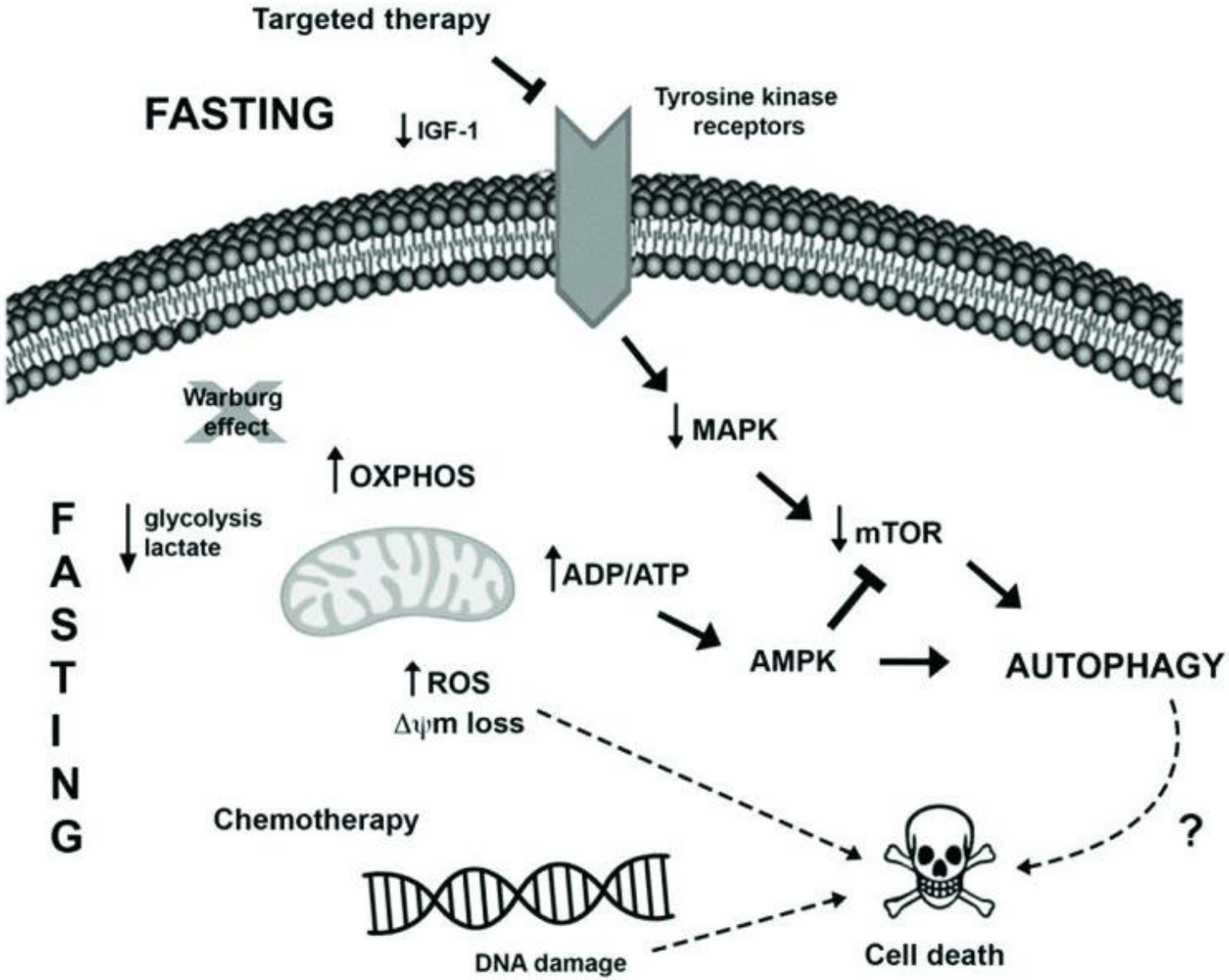
Table 1 | **Dietary approaches with proposed applications in oncology**

Type of diet	Restriction in calories	Composition	Schedule	IGF1 reduction (humans)	Glucose reduction (humans)	Ketone bodies increase (humans)	Location of pro-regenerative effects	Protection from chemotherapy toxicity
Fasting or FMD	>50%	Vegan and low-protein and low-sugar, high-plant-based fat composition, with micronutrient supplementation	Typically 2–5 consecutive days per month	Yes	Yes	Yes	Haematopoietic system, central nervous system, skeletal muscle and pancreatic β-cells (mouse data)[22,25,41,153]	Yes (mouse data and DNA damage analyses in patient leukocytes)[12,25,26,29,51–53]
Calorie restriction	20–40%	Reduction in all diet constituents except for vitamins and minerals	Chronic	Only in the presence of protein restriction[117]	No	No	Intestinal niche stem cells (mouse data)[118,119]	Yes (effect lower than that with fasting or FMDs; mouse data)[51]
Ketogenic diet	None (isocaloric)	High-fat, low-carbohydrate composition, with adequate protein content	Chronic	Yes	No	Yes	Peripheral nerves (rat data)[87]	NA

FMD, fasting-mimicking diet; NA, not available.

Nencioni A, Caffa I, Cortellino S, Longo VD. Fasting and cancer: molecular mechanisms and clinical application. *Nat Rev Cancer*. 2018;18(11):707-719. doi:10.1038/s41568-018-0061-0

Intermittent Fasting and Autophagy



Antunes F, Erustes AG, Costa AJ, et al. Autophagy and intermittent fasting: the connection for cancer therapy?. *Clinics (Sao Paulo)*. 2018;73(suppl 1):e814s. Published 2018 Dec 10. doi:10.6061/clinics/2018/e814s

The Microbiome

How does “dysbiosis” relate to carcinogenesis?

“Can we define “normal” not in terms of ecology, but in functional terms? A healthy microbiome would be commensal, immune tolerant, diverse, resistant to change, tumor suppressive. There might be lots of ecologies which would give the same result. Any departure from that you could define as *dysbiosis*.”

- Alasdair Scott, MD at the 2017 International Cancer Microbiome Consortium

Scott AJ, Alexander JL, Merrifield CA, et al. International Cancer Microbiome Consortium consensus statement on the role of the human microbiome in carcinogenesis. Gut. 2019;68(9):1624–1632. doi:10.1136/gutjnl-2019-318556

The Microbiome in Immunotherapy

- Immunotherapy uses monoclonal antibodies (MAbs) that target:
  - Programmed Cell Death protein-1 (PD-1) and
  - Cytotoxic T Lymphocyte antigen-4 (CTL-4)
- Multiple studies demonstrate a role of the gut microbiome in modulating responses to Immune Checkpoint Inhibitors (ICIs):
  - Differential microbiome ‘signatures’ differentiate responders from non-responders
  - Close relationship between decreased microbiome diversity and ICI toxicity
- Antibiotic and PPI use is associated with increased toxicity and decreased disease-free survival after treatment with ICIs.

The Microbiome in Immunotherapy

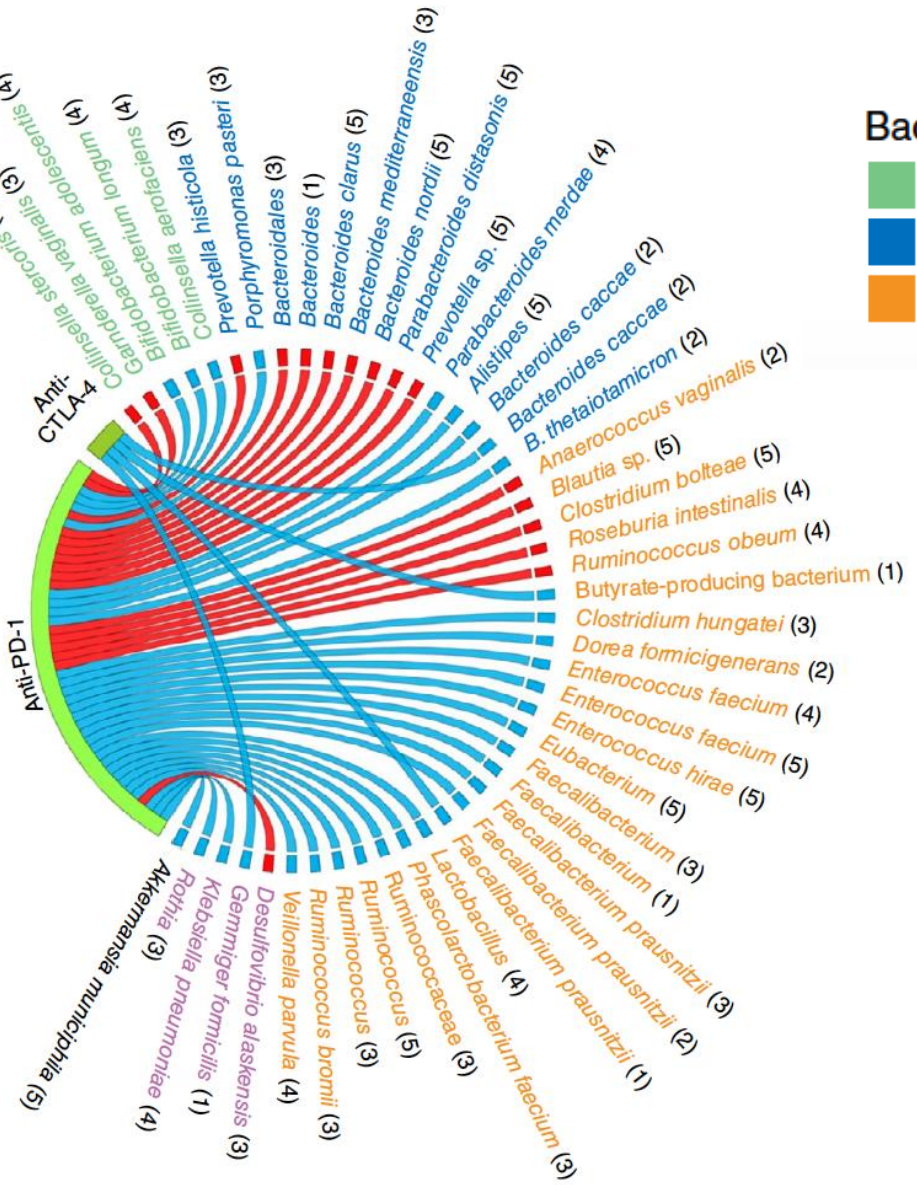
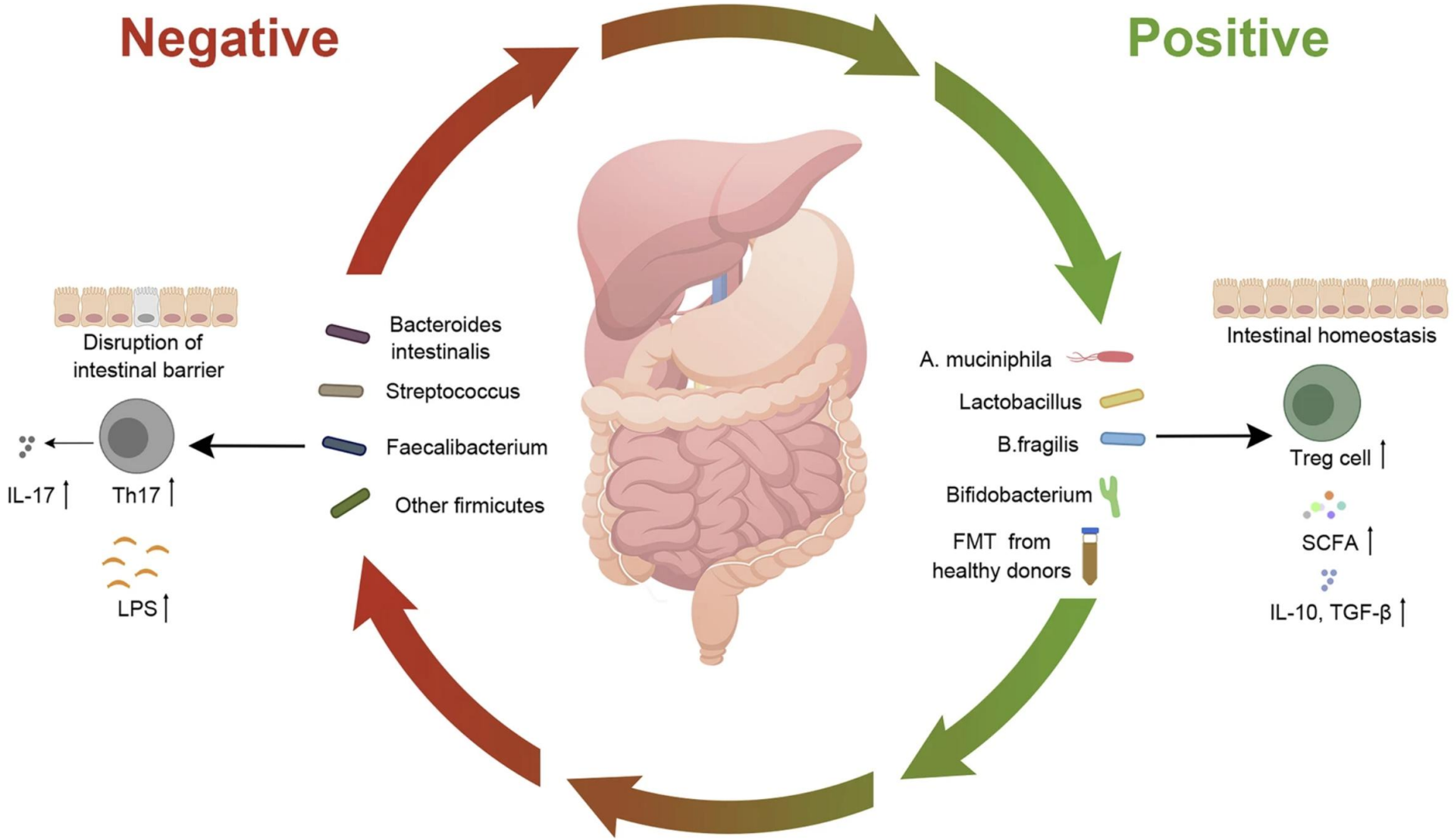


Fig. 3 | Existing evidence for the role of the gut microbiota in cancer development and treatment response. Circos plots illustrating the relationships of unique microbial taxa response to cancer immunotherapy agents (anti-CTLA-4 and/or anti-PD-1). The blue ribbons represent the microbial taxa that are enriched in the context of a beneficial therapeutic response. The red ribbons represent the microbial taxa that are enriched in nonresponders to therapy. These reported associations should be interpreted with caution as they are correlative in nature, and causality has yet to be established. Different taxa are further grouped and colored by their phyla as indicated by the legend. Numbers in parentheses indicate the references that demonstrate these relationships: (1) Chaput et al.[71]; (2) Frankel et al.[9]; (3) Gopalakrishnan et al.[12] (4) Matson et al.[11]; (5) Routy et al.[10]. Credit: Debbie Maizels/Springer Nature.

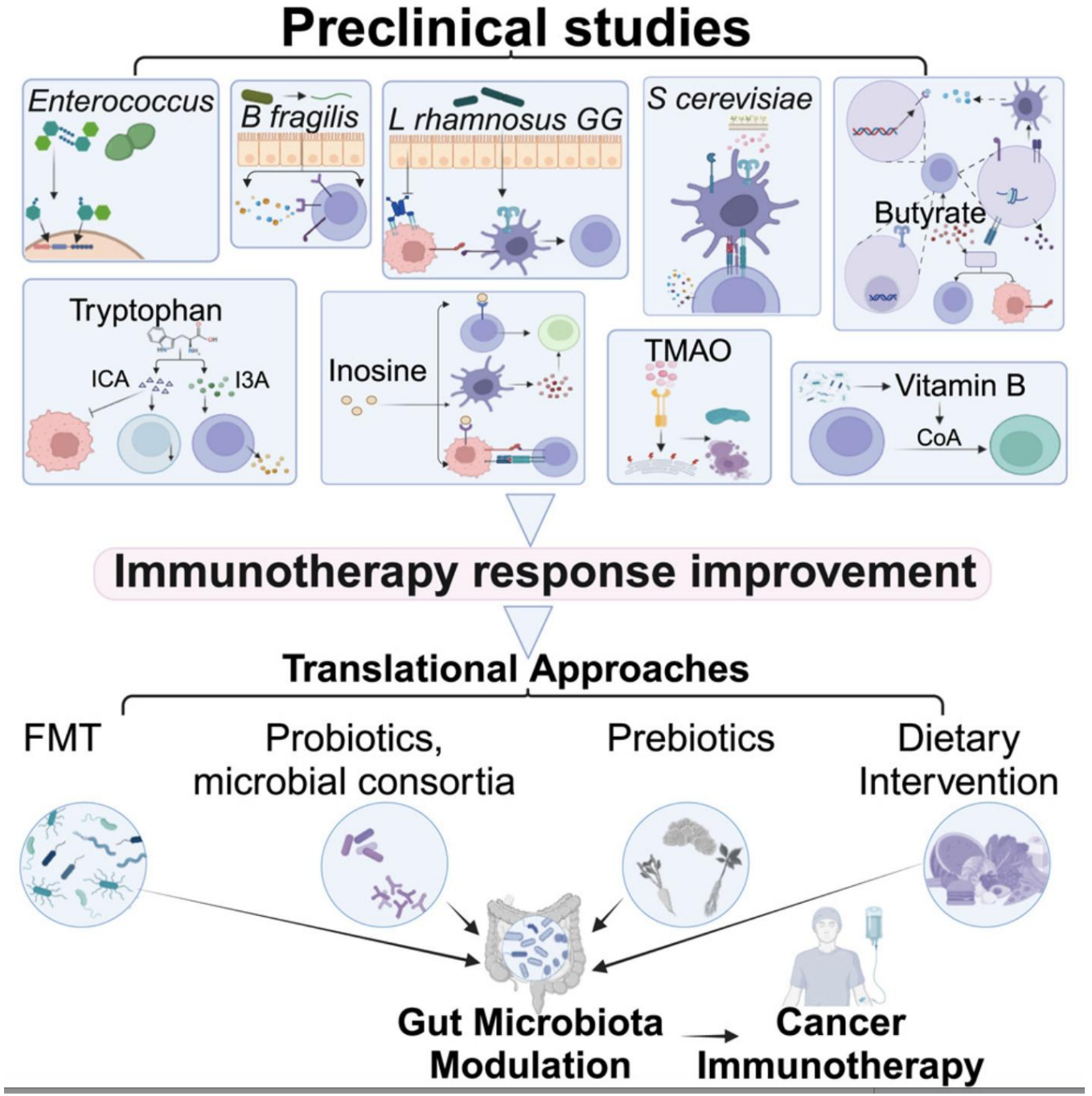
Helmink BA et al. The microbiome, cancer, and cancer therapy. Nature Medicine. 2019;35(3):377-388.

The Microbiome in Immunotherapy



Lei, W., Zhou, K., Lei, Y. *et al.* Gut microbiota shapes cancer immunotherapy responses. *npj Biofilms Microbiomes* **11**, 143 (2025). https://doi.org/10.1038/s41522-025-00786-8

The Microbiome in Immunotherapy



Kang X, Lau HC, Yu J. Modulating gut microbiome in cancer immunotherapy. Cell Rep Med. 2024 Apr 16;5(4):101478. doi: 10.1016/j.xcrm.2024.101478.

Mind – Body – Connection

Heart Rate Variability (HRV)

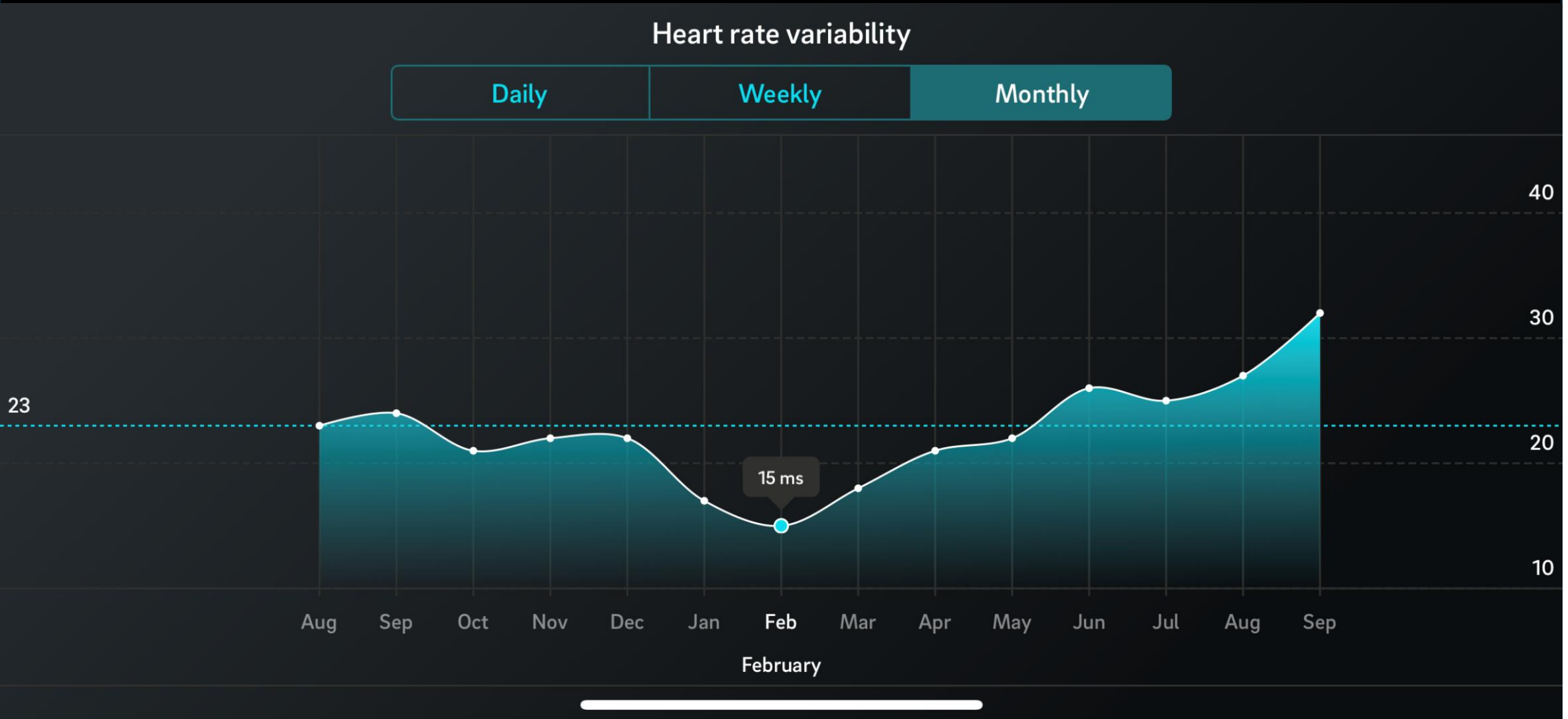
- HRV is a marker of autonomic nervous system function and represents the balance between sympathetic and parasympathetic nervous system activity
- Heart rate variability can be used clinically to assess stress coping mechanisms
  - High HRV indicates adequate coping
  - Low HRV indicated loss of resiliency

Kloter E, Barrueto K, Klein SD, Scholkmann F, Wolf U. Heart Rate Variability as a Prognostic Factor for Cancer Survival - A Systematic Review. *Front Physiol*. 2018;9:623. Published 2018 May 29. doi:10.3389/fphys.2018.00623

Heart Rate Variability & Cancer

- A lower HRV is associated with tumor growth through multiple pathways:
  - ↑ Inflammation
  - ↑ Oxidative stress
  - ↑ Sympathetic nerve activation

Kloter E, Barrueto K, Klein SD, Scholkmann F, Wolf U. Heart Rate Variability as a Prognostic Factor for Cancer Survival - A Systematic Review. *Front Physiol*. 2018;9:623. Published 2018 May 29. doi:10.3389/fphys.2018.00623


Heart rate variability
Daily
Weekly
Monthly
40
30
23
20
15 ms
10
Aug
Sep
Oct
Nov
Dec
Jan
Feb
Mar
Apr
May
Jun
Jul
Aug
Sep
February


For fast acting relief…
try slowing down

Forest Bathing and Immune Activation

- In Japan, “Shinrinyoku” (a leisurely visit to the forest) has been long regarded as a natural way to boost the immune system
- Japanese researchers have been studying this effect over the last decade. This data suggests that:
  - Forest bathing **increases NK cell activity** and **increases numbers of NK cells**
  - Decreases concentration of urinary adrenaline
  - The **increased NK activity lasts for more than 30 days**, suggesting that a forest bathing trip once a month would enable individuals to maintain a higher level of NK activity.

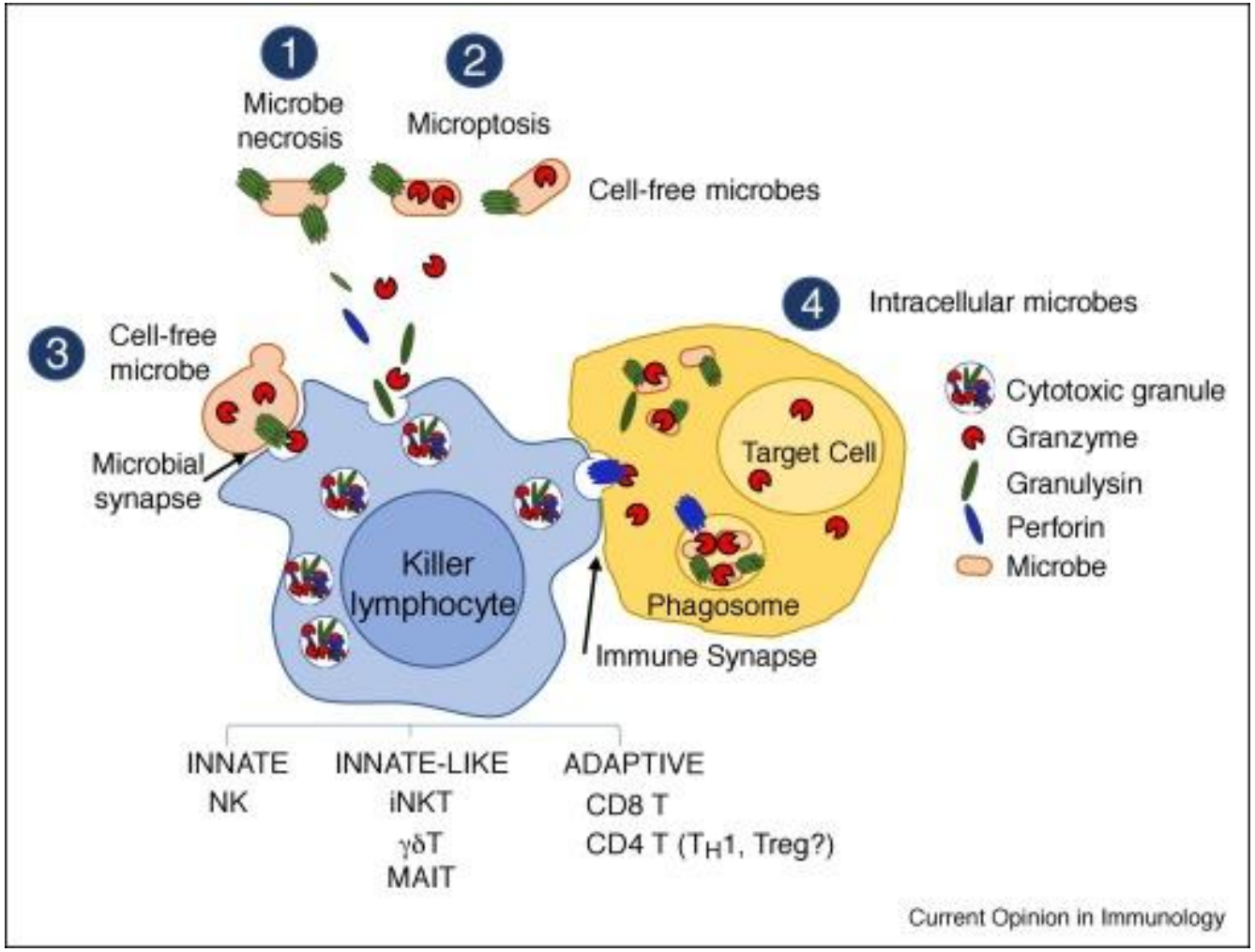
Li Q. Effect of forest bathing trips on human immune function. *Environ Health Prev Med*. 2010;15(1):9–17. doi:10.1007/s12199-008-0068-3

Forest Bathing and Anti-Cancer Proteins

Researchers found that a forest bathing trip

lasting 3 days and 2 nights

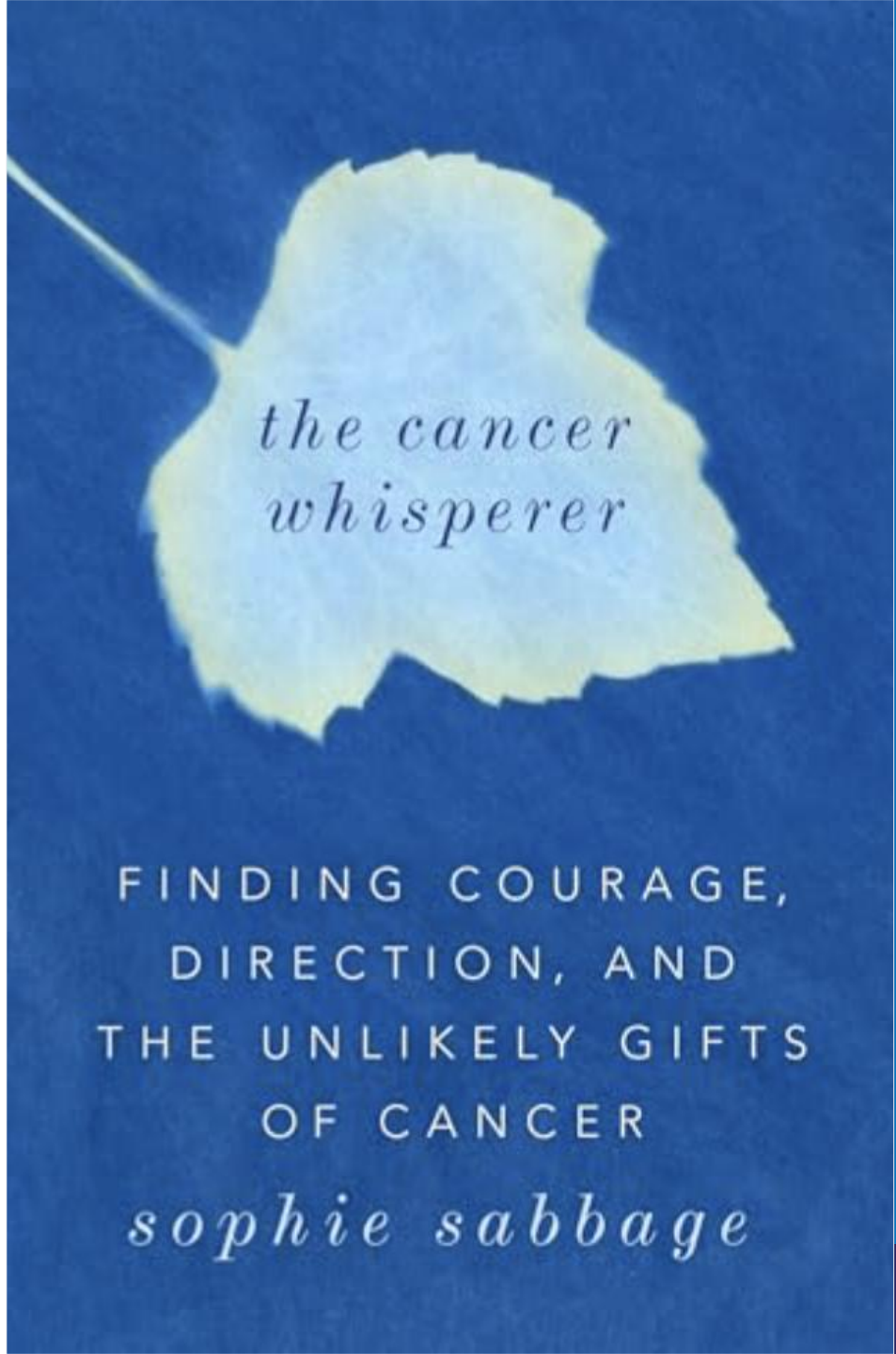
significantly **increased anti-cancer proteins**, including: granulysin-, perforin-, and granzymes A/B-expressing cells



Li Q, Morimoto K, Nakadai A, Inagaki H, et al. Forest bathing enhances human natural killer activity and expression of anti-cancer proteins. Int J Immunopathol Pharmacol. 2007 Apr-Jun;20(2 Suppl 2):3-8.

Listening to the World
. . . Listening to Cancer

- **Learning --** choosing a response
- **Transforming** cancer from enemy to teacher.
- **Emotional freedom and expression**
- **Empowerment.**
- **Finding purpose.**
  - opportunity for rebirth, to find joy and meaning in the present moment.



The power of connection

Optimal Healing Environment


RINGING OUT
Ring this bell
Three times well
Its toll to clearly say,
My treatment's done
This course is run
And I am on my way!
— Irve Le Moyne


RINGING OUT
Ring this bell
Three times well
Its toll to clearly say,
My treatment's done
This course is run
And I am on my way!

Uncertainty creeps into medical practice through every pore. Whether a physician is defining a disease, making a diagnosis, selecting a procedure, observing outcomes, assessing probabilities, assigning preferences, or putting it all together, s/he is walking a very slippery terrain. . . . and how easy it is for honest people to come to different conclusions.

David Eddy[1]

Eddy DM. Variations in physician practice: the role of uncertainty. Health Affairs. 1984 Summer;3(2):74-89.

UNCERTAINTY & COMPLEXITY

"***Complexity and uncertainty*** are built into the physician's effort to understand the particular in light of general rules…

The obstacle they encounter is the ***radical uncertainty*** of clinical practice: not just the incompleteness of medical knowledge but, more important, the imprecision of the application of even the most solid-seeming fact to a *particular patient.*"

Kathryn Montgomery. **How Doctors Think**. Oxford Univ Press. 2006

TOGETHER:

Clarifying the implications of scientific knowledge and mindful presence in the context of appalling uncertainty is where the processes of transformational change are discovered.

God grant me the SERENITY to accept the uncertainty in medicine, COURAGE to understand the knowledge that I can, and the WISDOM to know the difference.

THE JOURNEY

One day you finally knew
what you had to do, and began . . .

determined to do the
only thing you could do—

determined to save
the only life you could save.