TheGMS
The Gazette of Medical Sciences
https://www.thegms.co ISSN 2692-4374 DOI https://www.doi.org/10.46766/thegms

Oncology | Review

Energy Dynamics in Breast Cancer: Fields, Hormones & the Menstrual Cycle

K. E. Thorp

MD, Department of Radiology, University of Michigan Health-Sparrow, Lansing, MI.

Submitted: 11 November 2024
Approved: 08 December 2024
Published: 09 December 2024

Address for correspondence: k.t@earthlink.net

How to cite this article: Thorp KE. Energy Dynamics in Breast Cancer: Fields, Hormones & the Menstrual Cycle. G Med Sci. 2024; 5(1):169-249. https://www.doi.org/10.46766/thegms.oncol.24111101

INTRODUCTION

Since the late 19th century medical scientists have conceived breast cancer as a local disease originating at the cellular level despite a large and growing body of evidence to the contrary. This collective belief has decisively influenced how breast cancer is diagnosed and treated.

The radical mastectomy, introduced by American surgeon William Halsted in the 1880s, was regarded as the therapeutic gold standard for nearly a century until it was shown to have no impact on outcomes [1-3]. Surgical removal of cancerous breast tissue nonetheless remains a key part of therapy even though risk/benefit questions continue to surface [4-6] and studies document adverse outcomes such as 'surgical wounding' in which excision of potentially curable tumors at the wrong time of the menstrual cycle triggers explosive disease [7-15].

In 1896 English physician George Beatson discovered that oophorectomy in women with breast cancer (BC) led to disease regression. In the ensuing decades BC increasingly became conceived as a hormonally-mediated disease [16-18]. Many studies confirm that estrogen levels are increased in BC. Molecular therapies targeting estrogen now form the cornerstone of treatment [19-26]. While they induce remission in a high percentage of women, not uncommonly disease recurs months, years, even decades later and is often refractory to treatment.

Mammography, introduced in the 1960s, is routinely used to diagnose BC but its effectiveness, which hinges on early detection, is hotly debated [27-31]. Its public health impact in terms of reducing mortality is marginal [32, 33] **(Figure 1)**. One large meta-analysis found that mortality was 'generally reduced' but effects were not statistically significant and magnitudes small [34]. Overdiagnosis of early

cancers, which is said to occur in the 15-50% range, is a major shortcoming [35, 36]. One study examining usage patterns found that adherence to recommended guidelines was 'disappointingly low' [37]. And whether diagnosed early or late in the course of disease a woman's only options remain surgery and hormonal therapies.

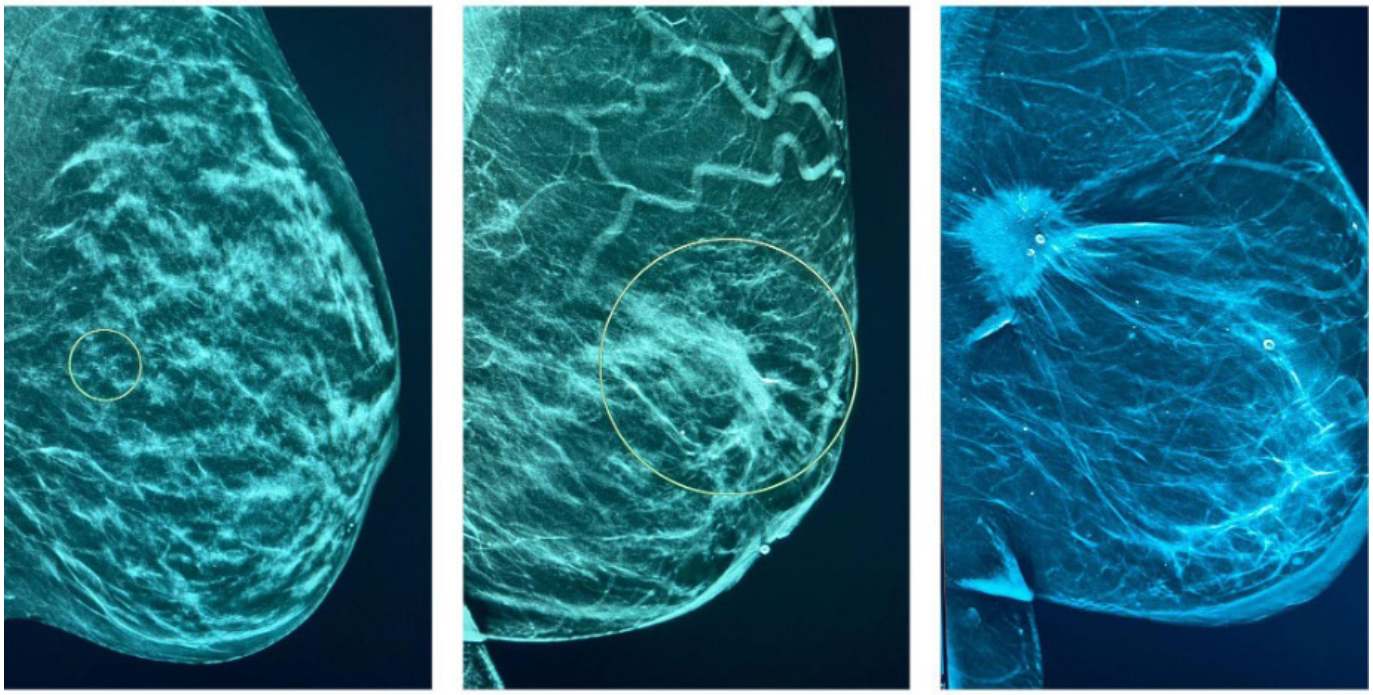

Figure 1. Mammographic images of breast cancer: ductal carcinoma in situ (DCIS) (left); invasive ductal carcinoma (middle & right).

Genetic screening for BC susceptibility genes, the so-called BRCA1 and BRCA2 variants, was introduced in the 1990s to detect the pre-cancer state but this too hasn't gone quite according to plan. Less than 10% of women diagnosed with BC have these hereditary markers while 80-85% of women have no familial history [38-40]. In ensuing decades no fewer than two dozen other BC susceptibility genes ranging from high- to low-penetrance have been found. Researchers continue to investigate their role in BC oncogenesis [41-46]. Once such genes are detected a woman's options are vigilant mammographic screening, bilateral mastectomy, or chemoprevention with anti-estrogens [47].

Evidence indicates that from the onset BC is not an isolated cellular event but a systemic disorder. A 1987 Danish study of 110 medicolegal autopsies of young and middle-aged women found BC in 20%. Nearly half had multicentric or bilateral lesions [48]. Women with BC are at risk for secondary tumors: the incidence of synchronous bilateral BC is ~1-2% while metachronous BC occurs in ~5-6% [49, 50] **(Figure 2).** Multiple studies support the notion of a pre-neoplastic spectrum in BC [51, 55]. Women with mammographically dense breasts are not only at increased risk for BC but ovarian cancer as well [56-61]. Same applies to women with BRCA-positive status [62-66]. BC survivors have increased risk for second primary cancers including uterine, lung, colon, pancreas, melanoma, leukemia, lymphoma and others [67-76].

Thorp KE. Energy Dynamics in Breast Cancer: Fields, Hormones & the Menstrual Cycle. G Med Sci. 2024; 5(1):169-249.
https://www.doi.org/10.46766/thegms.oncol.24111101

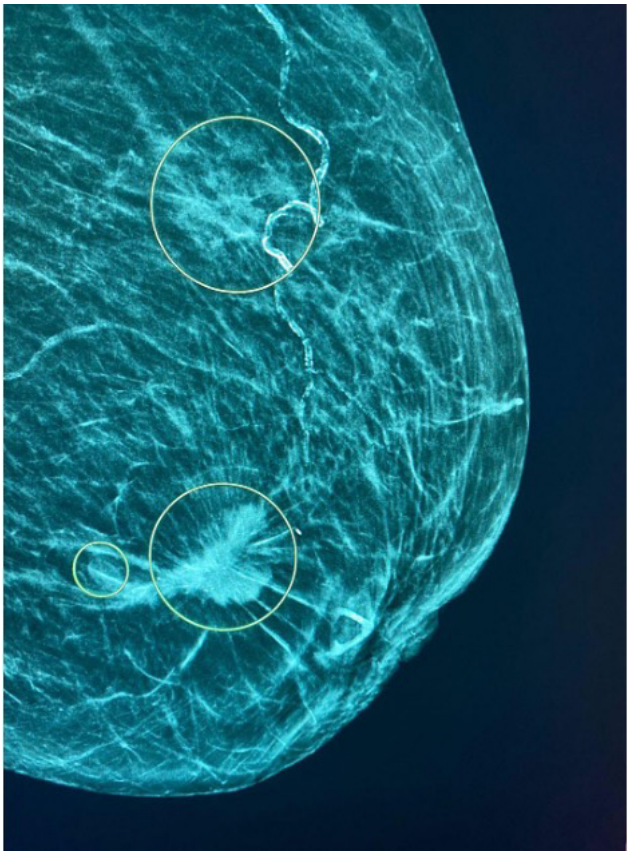

Figure 2. Multicentric breast cancer.

The prevalence of BC has increased steadily in recent decades and it is now the most commonly diagnosed cancer in the world [77-81]. In 2020 there were about 2.3 million new cases globally. It is the leading cause of cancer mortality in women. Cumulative risk is 12-13%, meaning that about 1 in 8 women will develop BC in their lifetimes. Large global variations exist with rates ranging from $<40/10^5$ women in various Asian and African locales to $>80/10^5$ in North America, Australia and Europe [82]. BC rates are expected to climb with up to 3 million new cases/year and 1 million deaths/year by 2040.

BC has consistently been linked to western lifestyles, reproductive habits and environmental factors [83-85]. One study found a six-fold gradient in BC risk related to migration patterns. Asian women born in the US had a 60% higher risk than those born in Asia. Those who emigrated from urban areas in Asia had a 30% higher risk than those who came from rural areas; migrants who had lived in the US for over a decade had an 80% greater risk than more recent émigrés [86].

Provocative evidence implicates a relationship between BC and light-at-night exposure [87-91]. Nocturnal light exposure inhibits secretion of the pineal hormone melatonin which plays a role in replenishing the metabolism. This led scientist Richard Stevens to advance the 'melatonin hypothesis' imputing that nocturnal light exposure promotes BC development [92, 93]. Once again, hormonal interactions seem to play a mediating role in BC oncogenesis.

But other lifestyle factors come into play. Research consistently finds links between obesity and BC [94,

Thorp KE. Energy Dynamics in Breast Cancer: Fields, Hormones & the Menstrual Cycle. G Med Sci. 2024; 5(1):169-249.
https://www.doi.org/10.46766/thegms.oncol.24111101

95]. Studies also show associations between BC and the metabolic syndrome, a cluster of alterations including obesity, insulin resistance, dyslipidemia, hypertension, and hyperglycemia [96-103]. The metabolic syndrome is associated with higher incidence of BC, more aggressive tumor subtypes and poorer outcomes. Such findings suggest that metabolic factors play a key role in BC origination.

Breast thermography has been in use since the 1960s to diagnose BC but due to its purported low specificity was largely abandoned in favor of mammography. But its ability to detect tumors, on the other hand, is rather high [104-106]. One study found that only 28% of noncancerous breasts had thermographic abnormalities versus 65-88% of cancerous breasts [107]. Another found that in women with questionable thermal abnormalities one-third developed BC within five years [108]. Women with BC have characteristic disturbances in their menstrual cycle heat rhythms and some researchers have suggested using such deviations to detect the pre-cancerous state [109-114]. An early step in the neoplastic transformation of breast tissue is acquisition of blood supply once again tying BC into the metabolism [115-118].

BC was described in the 2nd century AD by Roman physician Galen who called it *karkinos*, meaning 'crab', from which the term carcinoma derives. Galen was the first to describe the existence of a blood-borne energy field generated by the motions of the heart that formed the basis of his humoral system of medicine. His doctrine *omnia incipit in sanguine* held that all bodily functions originate in the blood. Galen regarded BC as a metabolic disorder arising from an excess of the humor known as black bile, which translates to our modern concept of acid. He cautioned against surgical excision of early breast tumors noting that 'any attempt at removal will irritate the cancer still more and kill the patient faster' [119].

This article explores the dynamics of BC which, in the end, originates in the deterioration of the blood-borne energy field in conjunction with a well-chronicled nest of hormonal disturbances related to the menstrual cycle. These imbalances, in turn, trigger excessive proliferation of cells in the face of insufficient energy to effect their transformation into functional breast elements. BC is not a disease of cells but of disordered transformation of cells into tissues. And its causes are neither cellular nor genetic but 'epigenetic' [120-122], a causal layer increasingly invoked by scientists which, as we will see, is nothing other than the organizing field that directs organismic growth and development.

CANCER HALLMARKS

In 2000 Hanrahan and Weinberg published their acclaimed 'Hallmarks of Cancer' in which they attempt to integrate the nest of known cellular and systemic disturbances into a coherent framework that encompasses the core functional attributes of all cancers [123]. Their 'hallmarks' provide a set of principles around which further attempts to understand cancer dynamics now constellate. The original list of six hallmarks continues to expand and evolve [124-129]. We will use it as a means by which to conceptualize BC oncogenesis with the proviso that, while a laudatory work-in-progress, it is more descriptive than explanatory. In turn new hallmarks will be introduced into the mix **(Figure 3).**

Thorp KE. Energy Dynamics in Breast Cancer: Fields, Hormones & the Menstrual Cycle. G Med Sci. 2024; 5(1):169-249.
https://www.doi.org/10.46766/thegms.oncol.24111101

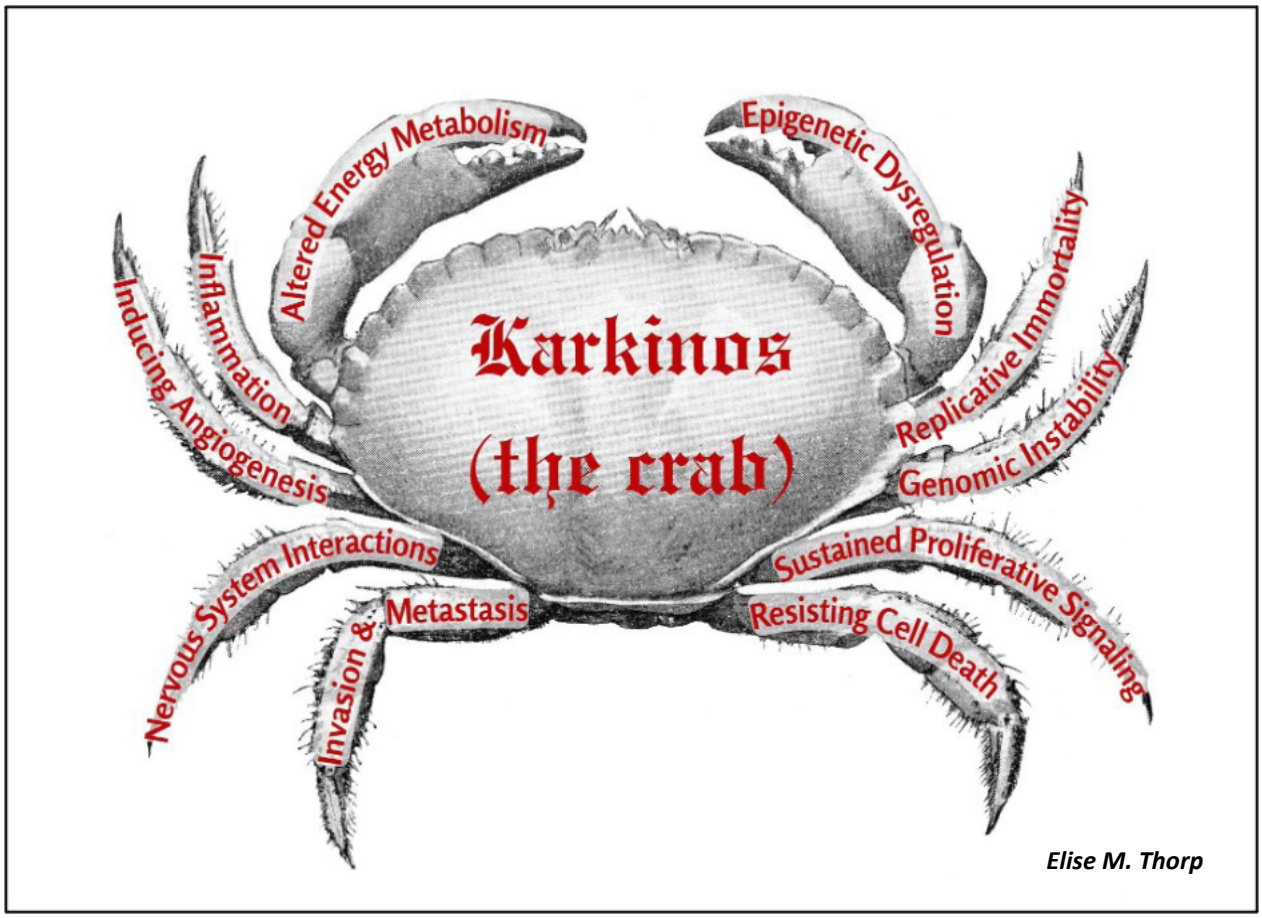

Figure 3. Hallmarks of Cancer

Implicit in their formulation is that cancer is more than just proliferation of cells but, rather, a complex set of interactions between various cell lines that compose cancerous tissue. They place emphasis on extracellular dynamics and the 'tumor microenvironment' [130, 131] which is recognized to play a key role in BC [132-139]. As this nexus of mutually sustaining interactions evolves toward the cancer state, tissues acquire a series of hallmark attributes which allow them to establish and maintain a unique functional niche and express typical neoplastic behaviors.

The most conspicuous attribute of cancer cells is their ability to continuously replicate and proliferate. A myriad of branching molecular pathways modulates cell mitotic cycles and growth through 'mitogenic signaling'. Such pathways are well-documented in BC [140-146]. Not only are pro-mitotic signals in the tumor microenvironment enhanced but there is active suppression of mechanisms that would counteract cellular proliferation.

In addition to increased proliferation, processes involving organized cell death, i.e., apoptosis, are disrupted. The monthly menstrual flow, for example, is an orchestrated functional event that resets the clock, so to speak, in order for a new cycle of cellular generation to occur. Programmed cell death is an integral part of the menstrual cycle [147-152]**.** Such apoptotic cycles are hormonally regulated [153-156]. Disturbances in apoptosis are a hallmark of BC [157-159]. One of the most common genetic defects in BC is silencing of the tumor suppressor p53 gene which orchestrates cellular apoptosis, DNA repair, cell-cycle arrest and angiogenesis [160-166].

Thorp KE. Energy Dynamics in Breast Cancer: Fields, Hormones & the Menstrual Cycle. G Med Sci. 2024; 5(1):169-249.
https://www.doi.org/10.46766/thegms.oncol.24111101

Another hallmark is 'replicative immortality'. During the course of oncogenesis cancer cells acquire the potential to propagate in an almost unrestricted fashion. This capacity contrasts starkly with normal cells which undergo only a limited number of propagational cycles. Such 'planned obsolescence' is enforced at the gene level by an enzyme known as DNA telomerase which adds telomere repeat sequences onto the terminal segments of DNA so that it can be accurately read during transcription [167, 168]. DNA telomerase levels normally diminish during both human and cellular aging but, paradoxically, are elevated in cancer states leading to sustained replication and so-called immortalization [169-172]. Telomerase levels are higher in women with BC compared to those without malignancy [173-178].

But increased proliferation and replicative immortality would not be possible without a more fundamental element, cancer stem cells (CSCs), the sine qua non of malignancy which are the drivers of BC [179-185]. CSCs are highly plastic cells which, through asymmetric division, replicate not only themselves and their 'stemness' but produce daughter cells which differentiate into all the various lineages that compose cancerous tissue. CSCs, *uber*-plastic, not only give rise to primary tumors but are behind tumor recurrence, invasion, metastasis, and chemo-resistance [186-189]. Their adaptability accounts for cancer cell heterogeneity as well as the hallmark of 'genetic instability'. By the same token CSCs may enter the dormant state and remain quiescent for extended periods of time [190-194].

It is apparent to many that genetic causal theories cannot explain such cancer-related behaviors and thus researchers increasingly invoke epigenetic causes, which implies a distinct causal layer 'above' the gene ensemble [195-203]. Epigenetic dysregulation is central to BC oncogenesis [204-208]. The altered gene functions that characterize the various hallmarks are clearly adaptive responses to extant cellular or systemic conditions. But what regulates such adaptations?

The most compelling evidence in favor of a distinct epigenetic causal layer concerns DNA methylation, a reversible modification of chromosomal architecture that determines which gene functions are expressed and which are silenced. DNA methylation, a genome-wide regulatory process, is the basis for preservation of genomic stability [209-218]. For much of the 20th century cancer was believed to be the result of mutations in gene structure but disruption of DNA methylation is now recognized to account for most cases – including in BC [219-230]. Inactivation of tumor-suppressor genes or induction of 'oncogenes' is related to hyper- and/or hypomethylation of various gene regions. Once again, we are led to question the nature of this regulatory process. To gain better perspective we must briefly examine how the science of genetics came into being.

What we now call genetics was originally subsumed under the discipline of embryology, the 'queen of the sciences' according to Aristotle, and now generally known as developmental biology. During the closing decades of the 19th century there was awakening interest in embryology undoubtedly spawned by Darwin's Theory of Evolution and it became subject to experimental investigation.

German embryologist Wilhelm Roux argued that during cell division there was asymmetric parceling of genetic material to daughter cells and on this basis advanced his 'mosaic theory' holding that each cell in the early embryo was pre-programmed to differentiate along certain lines. With successive divisions each daughter cell received a different allotment of genetic material. Growth and development thus were little more than differentiation. It is now known that all cells receive the same chromosomal allotment and that different groups of genes are turned ‘on’ and ‘off’ depending

Thorp KE. Energy Dynamics in Breast Cancer: Fields, Hormones & the Menstrual Cycle. G Med Sci. 2024; 5(1):169-249.
https://www.doi.org/10.46766/thegms.oncol.24111101

on the intended function of various cell lines. Once again this imputes some kind of organized gene-regulatory function.

In the 1890s, Hans Driesch separated fertilized sea-urchin eggs at the four-cell stage and upon development found that each cell had formed a complete, albeit smaller, organism which refuted Roux's mosaic theory. Driesch, observing that embryogenesis was predetermined and directed toward a fixed end or *telos*, argued for the existence of an organizing entity distinct from the cellular layer. In early development all cells possess the same undifferentiated potential, what Driesch called 'potency', and undergo stepwise transformation as the organismic blueprint seats itself into the proliferating mass of cells.

In the early decades of the 20th century German embryologist Hans Spemann performed experiments, recounted in *Embryonic Development and Induction* (1936) which added to Driesch's work. When he transplanted undifferentiated embryonic cells from one region to another the translocated cells differentiated according to the new area into which they were placed. Spemann thus concluded that the cells were influenced by the surrounding milieu and that morphogenesis must be orchestrated from outside the cell. From this he deduced the existence of an 'induction field'.

Within years of Spemann's induction field, two other embryologists, Russian Alexander Gurwitsch and Austrian Paul Weiss, advanced similar ideas. Gurwitsch, who published an embryologic atlas in 1907, began using the field concept as early as 1912 and articulated a mature field theory in the 1940s. Development became conceived as a coming-into-being governed by a meta-cellular organizing principle operating through the embryonic field. In 1923 Weiss advanced the concept of a 'determination field', which he popularized in his textbook *Principles of Development* (1939).

These developments are in accord with the writings of Aristotle, first biologist and embryologist, who argued that in order to explain morphogenesis a minimum of three distinct causal layers must be invoked: material causes, corresponding to the gene level, which determine whether an individual is a blue-eyed woman or a brown-eyed man; efficient causes, the layer of flowing fluids which provide the energetic means by which such transformations occur; and formal causes, the life principle, aka soul, responsible for the functional attributes peculiar to all members of a given species.

Against this background, American embryologist Thomas Morgan, now regarded as the father of modern genetics, forcefully inveighed against developmental field theories in his 1934 work *Embryology and Genetics*. Most biologists, he claimed, 'are not so much impressed by the idea that there is a principle of life as they are by the great variety of phenomena shown by living things'. It would seem 'premature and pretentious', he went on, 'to discuss some imaginary ideal property of life' **(Figure 4).**

Figure 4. Thomas Hunt Morgan: early proponent of the primacy of genetics in development.

https://www.biografiasyvidas.com/biografia/m/fotos/morgan_thomas.jpg

In one of the most circular propositions ever advanced in science Morgan argued that 'acceptance

Thorp KE. Energy Dynamics in Breast Cancer: Fields, Hormones & the Menstrual Cycle. G Med Sci. 2024; 5(1):169-249.
https://www.doi.org/10.46766/thegms.oncol.24111101

of such a principle would seem to make it hardly worthwhile to use the experimental method to study development'. Referring to such ideas as 'mysticism' he cautioned that 'it would seem better to table these metaphysical questions'. Genetics, he wrote, 'has become so interwoven with that of experimental embryology that the two can now to some extent be told as a single story'. This marks the origins of the scientific discipline we know as genetics.

After Watson and Crick's 1953 discovery of the structure of DNA and Crick's 1957 articulation of his 'Central Dogma', imputing the primacy of DNA and nuclear mechanisms, we observe emergence of a strict genetic determinism at which point genetics became less science than ideology. Geneticists augured a future replete with genetic engineering and elimination of disease which, to date, has yet to transpire. In *The Century of the Gene* (2000) physicist Evelyn Fox Keller remarks on the 'ever-widening gaps' between geneticists' assumptions and the actual data that emerged from experiments. 'It seems evident', she writes, 'that the primacy of the gene as the core explanatory concept of biological structure and function is more a feature of the twentieth century than it will be of the twenty-first'. Modern genetics, it seems, is theoretically threadbare.

In 1981 English biologist Rupert Sheldrake published *A New Science of Life: The Hypothesis of Formative Causation,* a thorough exposition of evidence and set of arguments in support of the field concept. Sheldrake was excoriated, vilified, and ridiculed by the science community. The mainstream science journal *Nature* called his book 'the best candidate for burning there has been in many years'. Fifteen years later biologists Gerry Webster and Brian Goodwin advanced similar ideas in *Form and Transformation: Generative and Relational Principles in Biology* (1996) which was also brushed aside. In each case the science community failed to meet the primary criterion that drives all scientific knowledge formation: falsification of hypotheses. Denial and ridicule do not equate to refutation.

Scientists seemed undeterred even by admonitions from within their own circle. In *Genes, Dreams And Realities* (1971) Nobel laureate Macfarlane Burnet attempts to quell the misguided fervor claiming that 'too much sensational material was being written about the future significance for medicine of discoveries in molecular biology'. He predicted this endeavor was destined to fail. A half-century on his comments seems prophetic.

The recourse by scientists to epigenetics increasingly points toward the primacy of metabolic factors in determining gene expression [231-240]. A great deal of attention has been focused on the role of mitochondria in the epigenetic orchestration of the genome [241-251]. And equally, metabolic factors are now recognized to play a key role in shaping the tumor microenvironment [252-260].

This makes sense from Aristotle's 'three-tier' perspective: energy metabolism forms the efficient means by which the 'upper' formal causal layer integrates into 'lower' cell processes. Sheldrake claims this occurs on the basis of 'morphic resonance', which implies the presence of a resonance field. Sheldrake's surmise is spot on: the field arises in the blood in accordance with Galen's humoral thesis and is generated by hormonal fluctuations thereby creating a complex oscillatory structure which, definitionally, constitutes a resonance field. BC is a 'mosaic' not in the sense of Roux's theory of gene inheritance but because affected tissues have fallen out of relationship with the developmental field.

ENERGY DYNAMICS

One of the most compelling (and overlooked) arguments in favor of an organized energy field in the body was advanced by Swedish radiologist Björn

Thorp KE. Energy Dynamics in Breast Cancer: Fields, Hormones & the Menstrual Cycle. G Med Sci. 2024; 5(1):169-249.
https://www.doi.org/10.46766/thegms.oncol.24111101

Nordenström in his pioneering work *Biologically Closed Electric Circuits: Clinical, Experimental and Theoretical Evidence for an Additional Circulatory System* (1983). In the 40 years since its publication clinical and laboratory evidence roundly supports his assertions.

During biopsy procedures Nordenström measured electrical potentials and found voltage differences between tumors and surrounding tissues that were often related to the degree of differentiation or invasiveness of the tumor. Curiously, at the outer margins of tumors he frequently measured electropositive potentials which, upon advancing the needle more deeply, reverted to electronegativity. Potentials fluctuated in accordance internal conditions such as necrosis and vascularity.

Nordenström deduced that metabolic activity involves voltage fluctuations, 'waves of slowly changing potential', which likely account for electrical variances between normal and cancerous tissues. Tumor metabolism generates 'redox potentials' resulting in charge accumulations that alter relations between cancers and their surrounding environment. Such polarizations, he intuited, account for the structural modifications of cancer tissue suggesting that such fields possess a 'morphogenetic capacity'. Nordenström advanced the concept of a second circulatory system composed of the flow of electrical currents, i.e., a 'biologically closed electric circuit', but left unaddressed its origins.

One of the most significant and unanticipated turn of events in 20th century experimental medicine occurred in the 1980s around the time Nordenström's work was published. For most of the 20th century scientists conceived the heart to function in the manner of a mechanical pump, with blood propelled forward through the arteries during the systolic phase of the cardiac cycle. The diastolic phase of the cycle, on the other hand, was said to represent a period of passive relaxation.

In the early 1980s physiologists discovered negative intraventricular pressures, i.e., a suction force, in the early diastolic phase indicating that diastole was not a time of passive relaxation but, instead, a period during which blood was actively drawn forward through the veins into the ventricular chamber [261-264]. A handful of studies later found the presence of spiral flow currents in arteries and veins which can only be explained on the basis of a suctional force [265-267].

In an earlier paper I show that in its cycles of contraction and dilation the heart generates an electromagnetic field which gives rise to both diastolic expansion and the suctional force [268]. In a series of later papers I show that deterioration of this field is the primary cause of diseases like chronic heart failure and chronic kidney disease [269], neurodegenerative disorders like Alzheimer's [270], autoimmune disease [271], seizure disorders [272], and the metabolic syndrome [273]. For over a century it has been recognized that the heart and blood contain large iron stores and, while iron's role in various chemical reactions has been extensively detailed, there has been little consideration as to whether it might play a more fundamental role in the body's energy economy.

Equally the question arises as to the function served by nerves that course over the surface of the heart. Cardiologists claim these nerves cause the heart to contract but is this correct? Galen observed in animal experiments that when the heart was cut out and placed in a fluid bath it continued to dilate and contract, what is now called cardiac automaticity. Transplanted hearts continue to function even as nerve conduction has been interrupted.

What happens during systolic contraction of the ventricle is identical to what happens during the induction of an external magnetic field by electrification of ferrous objects. As the ventricle contracts and iron stores are brought into closer

Thorp KE. Energy Dynamics in Breast Cancer: Fields, Hormones & the Menstrual Cycle. G Med Sci. 2024; 5(1):169-249.
https://www.doi.org/10.46766/thegms.oncol.24111101

apposition iron nuclei in heart muscle align and precess synchronously on the basis of field interactions. Electrical potentials in the nerves induce formation of a three-dimensional magnetic field in the heart muscle leading to ventricular expansion, which occurs on the basis of repulsion. By the same token, all nerves, i.e., Nordenström's 'second circulation', derive their flow currents from the extracellular fluid (ECF) space in accordance with Galen's doctrine of *omnia incipit in sanguine*. One need only recall that in cardiac arrest all nervous system functions immediately cease.

Nordenström's 'double circulation' explains two more cancer hallmarks: tumor angiogenesis and invasion of nerves. Like other tissues, cancers require a nutrient and energetic source. A sentinel event in their development is acquisition of blood supply, effected by various molecular signaling factors and known as the 'angiogenic switch' [274-283]. Consistent with defective morphogenesis, tumor blood vessels display erratic capillary growth, tortuous, irregularly branching vessels with fluctuating diameters resulting in dysregulated blood flow, leaky vessels, microhemorrhages and enhanced tendency for tumor necrosis [284].

In recent decades the contribution of nerves to the progression of cancers has become widely recognized. Cancers release various 'neurotrophic factors' that induce growth of nerves into the tumor microenvironment while, at the same time, nerve currents promote cancer cell proliferation and dissemination [285-293]. Over a century ago embryologists, observing that nerves were vital for development of embryonic structures as well as limb regeneration and healing, coined the term 'trophic nerves' implicating them in morphogenesis [294-297]. High nerve density in BC tumors and/or perineural invasion are associated with poor outcomes and high recurrence rates [298-300] **(Figure 5).**

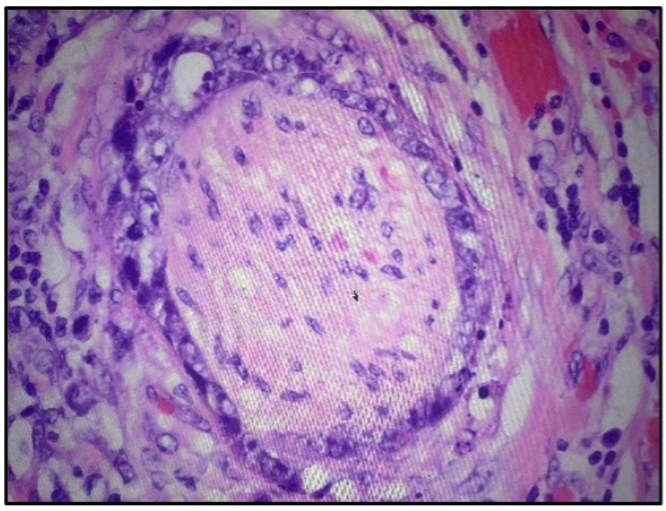

Figure 5. H & E stained image of perineural invasion in invasive ductal BC. A ring of cuboidal epithelial cells with intensely dark blue (basophilic) nuclei encircle a peripheral nerve with faintly pink (eosinophilic) cytoplasm.

(https://instagram.com/pathweb/p/CwUINzERqak/?img_index=1)

In one experiment intended to demonstrate the effects of electric fields in water Nordenström packed a U-shaped glass tube with cotton wool in its lower curved portion to simulate capillary resistance **(Figure 6).** Both limbs of the tube were filled with water and metallic electrodes connected to a DC power source were placed on each side. After a variable period, depending on the strength of the voltage currents, he observed differential water levels in the two limbs of the tube with the left (cathodic) side higher than the right (anodic) pole. What is at play here?

Thorp KE. Energy Dynamics in Breast Cancer: Fields, Hormones & the Menstrual Cycle. G Med Sci. 2024; 5(1):169-249.
https://www.doi.org/10.46766/thegms.oncol.24111101

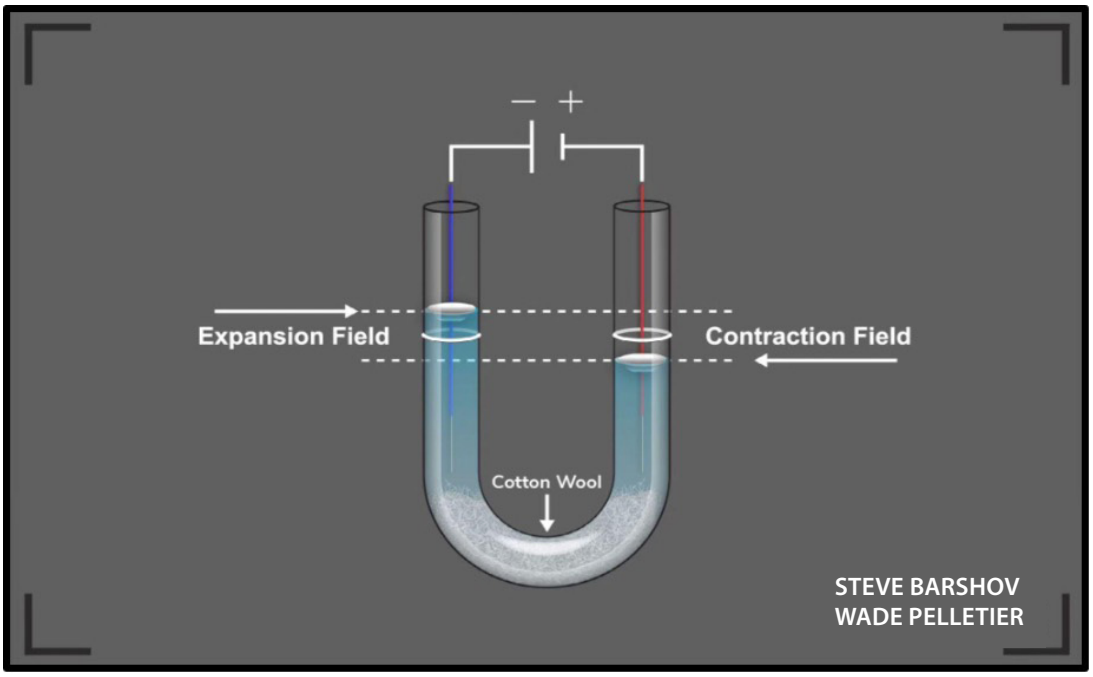

Figure 6. Nordenström's electrolytic decomposition of water.

At the cathodic terminal hydrogen gas is released into the air leaving an excess of negatively-charged (alkaline) hydroxyl ions (OH^-) in the fluid medium while at the anode oxygen is released resulting in an excess of positively-charged (acidic) hydronium ions (H_3O^+). Water's ability to undergo electrolytic decomposition and dissociate into ionic species with opposing charge is related to its 'dielectric polarizability' but this does not explain the fluid levels. Differential movements of water between the two poles are equally untenable: OH^- at the cathode would theoretically be drawn toward the positively-charged anode while H_3O^+ at the anode would migrate toward the cathode. At some point in between the two species would recombine resulting in no net movement of water.

The spatial reorganization of water is a field-mediated effect. Electrical currents streaming through the electrode at the cathode generate a magnetic field around the wire causing expansion of water effected by negatively-charged ions. Conversely, currents drawn out the water at the anodic pole induce contraction of the surrounding fluid medium thereby causing its level in the tube to drop. When one says that water is polarizable, it means that electricity causes the expansionary magnetic and contractionary fields to separate and repel each other. This same field effect can be observed in the periodic waxing and waning of the oceanic tides.

Since the time of Newton scientists have attempted to explain tidal fluctuations on the basis of gravity but as evidence accumulates this hypothesis seems less likely. High tides occur when the moon is directly overhead (0°) and at 180° on the opposite side of the globe while low tides occur simultaneously at 90° and 270°. Clearly two forces, one expansionary and the other contractionary, are necessary to explain tidal phenomena.

If gravity were the sole determinant, then why don't large freshwater bodies like the Great Lakes exhibit tidal movements? How does gravity explain the synchronous tidally-induced fluctuations in voltage potentials in rivers upstream to large saltwater bodies, or tide-related variations in tree stem

Thorp KE. Energy Dynamics in Breast Cancer: Fields, Hormones & the Menstrual Cycle. G Med Sci. 2024; 5(1):169-249.
https://www.doi.org/10.46766/thegms.oncol.24111101

diameters, leaf movements and root growth [301-313]? The gravity hypothesis is wrong. These diverse phenomena are explainable only on the basis of distortions in the geomagnetic field induced by overhead passage of the moon (which itself is laden with iron). Such considerations will be developed in greater depth and detail in a subsequent piece on the nature of tidal phenomena **(Figure 7).**

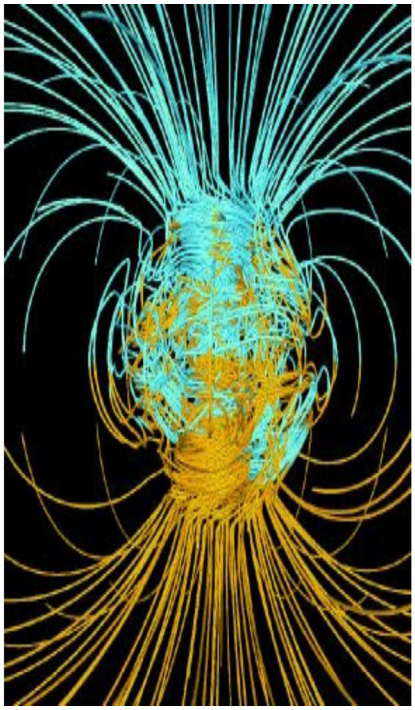

Figure 7. Glatzmaier-Roberts simulation of the earth's magnetic field.

https://www.sdsc.edu/pub/envision/v16.1/geo.html

If one accepts the existence of an organized electromagnetic field in the human body and, by the same token, recognizes the primacy of the geomagnetic field in organizing global tidal dynamics, it is not a great leap to recognize an interrelation between the two: whenever two magnets come into apposition the two fields unite and become one. Just as compasses spontaneously align themselves to the surrounding field anywhere across the globe so too must iron in living bodies be similarly affected. It cannot be otherwise.

The same can be said concerning the developmental field: given that it confers upon individuals' functional attributes that define the species, so too must it be globally distributed and, in some manner, inhere within the geomagnetic field, which amounts to the interpenetration of Aristotle's formal and efficient causal layers. In the ancient world this collective entity was referred to as the 'world soul', and on this basis the planet was regarded as a living entity [314]. This concept, aka the 'great chain of being', held sway for over 2,000 years until it was arbitrarily discarded by scientists in the 18th century as they sought to reinterpret natural phenomena from a 'rational' point-of-view [315].

Concerning the nature of water, we are led to reconsider ancient views. In the Earth, Water, Air, Fire (EWAF) cosmology, advanced by Empedocles around 450 BC, energetic interactions between the Four Elements transpire in top-down fashion: Fire (sun), gives rise to light and heat; Air, aka *pneuma* meaning 'energy', corresponds to the geomagnetic field and possesses attributes we now ascribe to a complex resonance field. Resonance seems to be subsumed under ancient notions of 'sympathy'. Excitations induced in Air by Fire are directly transmitted to Water, the energetic intermediary between Air and Earth. Such vertical causality forms the basis for magnetic resonance imaging (MRI): radiofrequency pulses induce field deformations in tissue water which, upon pulse termination, reverts to its original state. MR images are made possible by only water's ability to undergo resonance **(Figure 8).**

Thorp KE. Energy Dynamics in Breast Cancer: Fields, Hormones & the Menstrual Cycle. G Med Sci. 2024; 5(1):169-249.
https://www.doi.org/10.46766/thegms.oncol.24111101

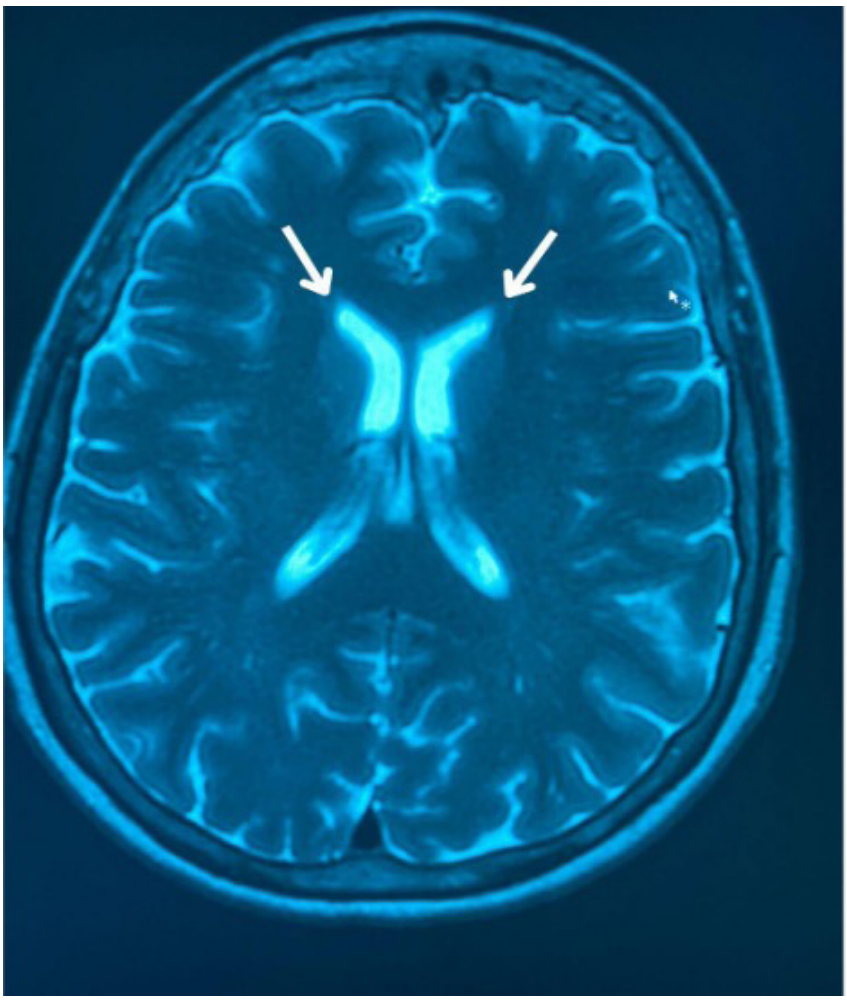

Figure 8. T2-weighted MR image of the brain demonstrating water's ability to undergo resonance. Bright signal (arrows)represents cerebrospinal fluid in the lateral ventricles.

Since the early 1800s scientists have sought to define water on a chemical basis despite recognition of dozens of anomalous properties that defy explanation. Recent works like Gerald Pollack's *The Fourth Phase of Water* (2013) and Hasok Chang's *Is Water H_2O?* (2012) raise serious doubts regarding this proposition and suggest a fundamental reappraisal is needed. Is water a distinct element? As we will see, its ability to undergo energetic transformations plays a key role not just in BC but in all cancers. Given similarities in ionic composition between seawater and water in the ECF space it might be better conceived as the 'ocean within'.

WARBURG METABOLISM

We now consider what is increasingly recognized as a primary cancer hallmark: altered energy metabolism. Most scientists regard it as a cellular disturbance due to reprogramming of genes or mitochondrial dysfunction [316-324], while others ascribe it to epigenetic factors [325-330]. All these answers are correct but not for stated reasons. Oncogenesis begins 'upstream' in the ECF space with insufficient energy delivery to cells which impairs mitochondrial metabolism thereby altering gene function. The dynamics are similar to Nordenström's experimental induction of field effects in the test

Thorp KE. Energy Dynamics in Breast Cancer: Fields, Hormones & the Menstrual Cycle. G Med Sci. 2024; 5(1):169-249.
https://www.doi.org/10.46766/thegms.oncol.24111101

tube. Recognizing water's central role in such events necessitates reconsideration of ideas concerning the tumor microenvironment.

In the 1920s, German scientist Otto Warburg found altered glucose metabolism in cancer cells characterized by increased lactic acid production, aka 'aerobic glycolysis' [331, 332]. Even in the presence of sufficient oxygen cancer cells seem prone to use such alternate pathways despite up to 19-fold lower ATP production when compared to mitochondrial oxidative phosphorylation. This metabolic switch is associated with host of well-described cancer-related abnormalities and forms the basis for fludeoxyglucose-18 (FDG-18) positron emission tomography (PET) widely used in cancer staging [333-335] **(Figure 9).**

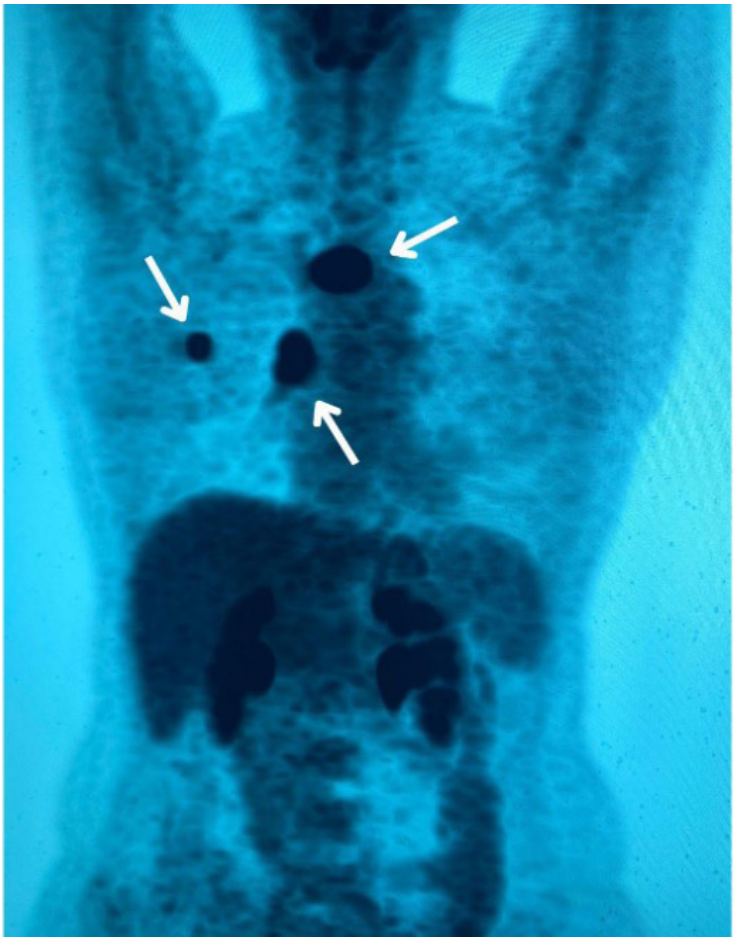

Figure 9. Warburg metabolism in breast cancer. FDG-18 PET image with avid uptake of radiolabeled glucose by primary breast tumor (left arrow). Lower arrow represents metastatic spread to right hilum while upper arrow represents sternal metastasis. (Courtesy of Cynthia M. Wheeler, MD)

Thorp KE. Energy Dynamics in Breast Cancer: Fields, Hormones & the Menstrual Cycle. G Med Sci. 2024; 5(1):169-249.
https://www.doi.org/10.46766/thegms.oncol.24111101

Not only does tumor metabolism generate acid in the microenvironment [336-340], explaining Nordenström's finding of positive voltage potentials at the outer margins of tumors, but is at play in other cancer phenomena such as impaired iron metabolism [341-347], cellular crowding due to loss of magnetic field strength, i.e., zeta potentials, at the outer cell membrane [348-359], inflammation [360-370], and, most significantly, cellular dedifferentiation with emergence of cancer stem cells [371-380]. Warburg metabolism is a primary feature of embryonic stem cells suggesting that as organisms evolve from unicellular to multicellular forms, i.e., from purely proliferative to possession of higher adaptive functions, cells change their metabolic repertoire – once again implicating the developmental field.

The 'severely polarized extracellular acidity' in the tumor microenvironment, i.e., the black bile state, is recognized to be a primary driver of oncogenesis [381, 382]. Acidic ECF is said to influence expression of up to 150 different genes associated with functions like angiogenesis, cell proliferation and tissue remodeling [383]. In addition to enhancing tumor cell motility, invasiveness, and metastasis, the acidic milieu inhibits activation and proliferation of immune cells thus engendering the hallmark of 'evading immune destruction' [384-396]. Based on water's electrical polarizability we would situate such events at the anodic pole which contains an excess of H_3O^+ and deficient O_2. This can only be secondary to impairment of the magnetic field which implicates iron metabolism.

Iron plays a central role inside cells based on its capacity to accept and donate electrons and thereby influence cellular energetic pathways. Iron, concentrated in mitochondria, effects synthesis of substances that mediate enzyme function and redox reactions [397-399]. Although the chemical basis of iron metabolism is well described there has been little discussion as to its involvement in generation of an intracellular magnetic field. When mitochondria function optimally their pH is in the 7-8 range corresponding to the cathodic pole of the 'biologically closed electric circuit'. Coincident to this is generation of an outwardly-directed (electronegative) field, aka the zeta potential, by which cells repel one another and thereby maintain circulation through the ECF space. Such expansion fields are mediated via water.

As energy flow through the ECF space diminishes and mitochondrial metabolism shifts toward the anodic pole, iron is oxidized, cells become less electronegative and the field contracts. This, in turn, leads to loss of zeta potentials, compromise of the ECF space and cellular crowding, another characteristic histologic feature of cancer **(Figure 10).** Impaired iron flux leads to intracellular accumulation of free radicals, i.e., oxidative stress, that degrade organelles and biomolecules including DNA [400, 401]. Many papers detail the role of iron in tumorigenesis, including BC, but it has yet to be cited as a hallmark [402-420]. Defective iron-directed energy metabolism and loss of zeta potentials must now be added to the ever-growing list. Disruption of tissue architecture related to deterioration of the magnetic field underscores its intertwined relation with the developmental field, i.e., the conjoined nature of Aristotle's efficient and formal causal layers.

Thorp KE. Energy Dynamics in Breast Cancer: Fields, Hormones & the Menstrual Cycle. G Med Sci. 2024; 5(1):169-249.
https://www.doi.org/10.46766/thegms.oncol.24111101

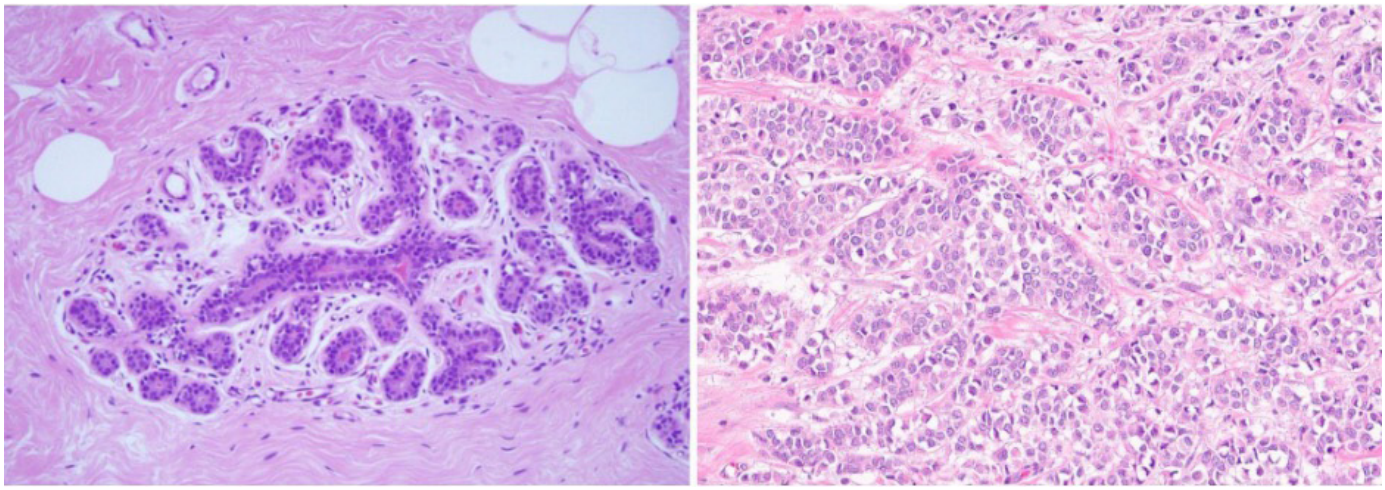

Figure 10. Image on left depicts normal lobular breast anatomy. Image on right showing cellular crowding and loss of normal lobular architecture in breast cancer related to Warburg metabolism, acidification of the ECF compartment and loss of magnetic field strength.

Normal-- https://www.ncbi.nlm.nih.gov/books/NBK567766/figure/article-35923.image.f1/

Carcinoma-- www.pathologyoutlines.com

Altered mitochondrial metabolism is responsible for yet another cancer hallmark: inflammation [421-424]. The relation between chronic 'smoldering' inflammation, oxidative stress, and cancer is well recognized [425]. In the 2nd century Galen advanced the doctrine of *functio laesa* stating that inflammation is associated with loss of normal physiologic function [426]. Cancer-related inflammation triggers cellular proliferation and angiogenesis while impairing immune function and promoting resistance to drug therapies [427-443]. Inflammation is a hallmark feature of BC [444-451]. But cancer-related inflammation is no different than that associated with any other chronic disease and is secondary to insufficient energy generation in the blood [452-455].

Such derangements set the stage for what must be regarded as the 'main event' of oncogenesis: emergence of cancer stem cells. Stem cells mark a reversion to a primitive ontogenetic state defined not only by Warburg metabolism but absence of higher adaptive functions beyond proliferation. Stem cells can be regarded as having fallen out of relationship with the developmental field and set free to pursue their own random telos at the expense of the organism. In an Aristotelian sense the ensemble of functions expressed by normal cells must, as Spemann's experiments attest, originate in the 'external' field and be secondarily imposed on otherwise amorphous aggregates of cells.

In the case of BC, stem cells possess an unlimited capacity to respond to evolving conditions in the microenvironment and adapt their program accordingly. They propagate, develop signaling pathways in support of clonal expansion or, when conditions are not optimal, enter a variable dormant phase [456-466]. BC tissues are heterogenous, comprising multiple genetically diverse cell lines that form an integrated ecosystem [467]. Tumors often possess up to 20 different clonal subtypes organized into a smaller number of 'super-clones' all of which derive from a reservoir of stem cell precursors [468-478] **(Figure 11).** This constitutes an 'insurgency of clones' which gives rise to a ceaselessly evolving

Thorp KE. Energy Dynamics in Breast Cancer: Fields, Hormones & the Menstrual Cycle. G Med Sci. 2024; 5(1):169-249.
https://www.doi.org/10.46766/thegms.oncol.24111101

mosaic [479, 480]. The genetic signatures of metastatic lesions, for example, are usually quite distinct from that of primary tumors [481-485]. All of this supports the primacy of the developmental field and its dominion over all cellular process. We must therefore inquire more deeply into the nature of such field dynamics.

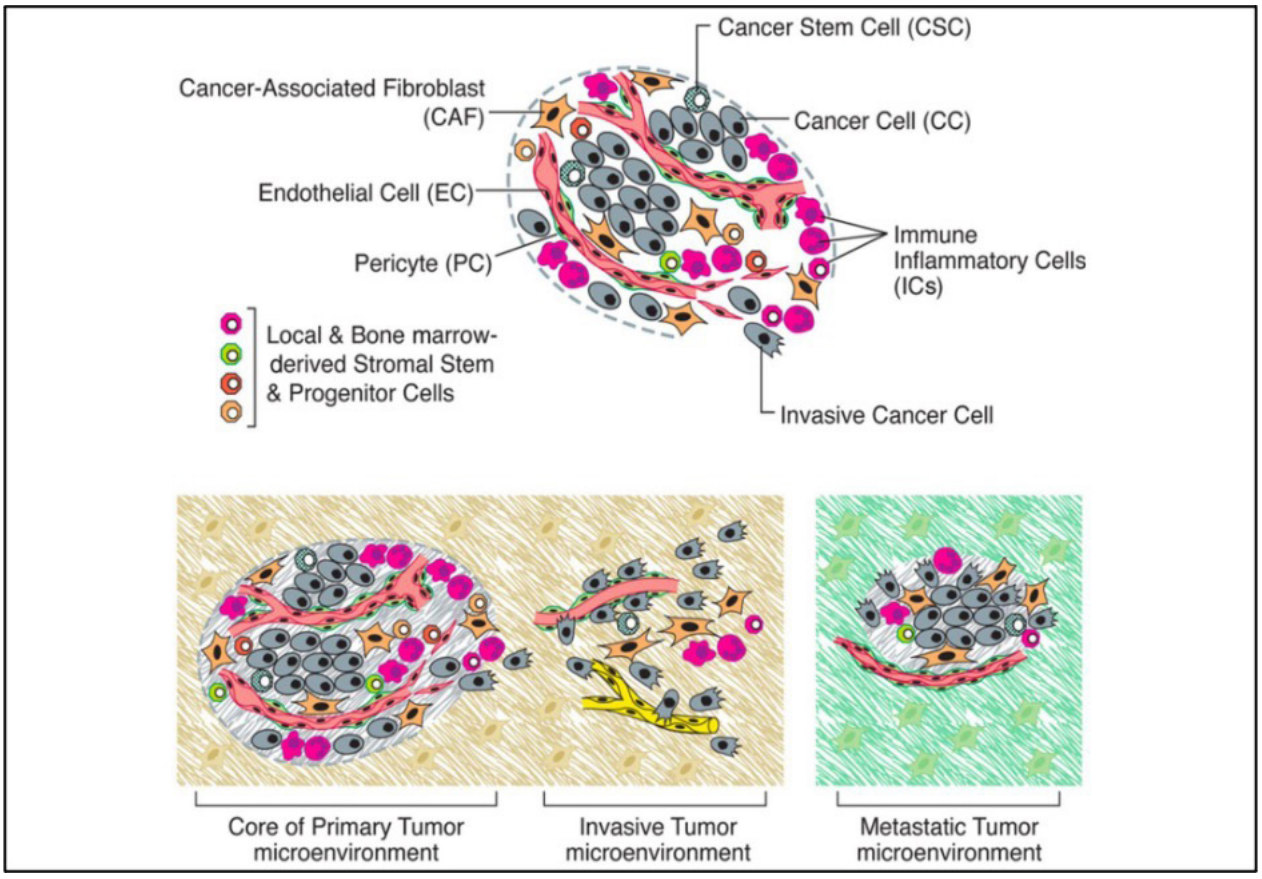

Figure 11. Cancer as a complex set of interactions between genetically distinct cell lines that establish their own self-sustaining niche based on the Warburg state.

(From: Hallmarks of Cancer: the next generation. Hanrahan D, Weinberg RA)

THREEFOLD TURNING CYCLES

For over a half-century chronobiologists have studied relationships between BC, the menstrual cycle, and conditions in the natural environment related to daily and seasonal fluctuations of light, heat and dark induced by the Earth's daily rotation about its axis, precessional movements in its annual orbit around the sun, and exposure to light in the external environment induced by artificial illumination. Not only do such dynamics influence both the sleep/wake and menstrual cycles but also play into the developmental origins of BC.

Regarding the menstrual cycle there is general consensus that the circalunar rhythm is the primary driver of reproductive functions in a wide spectrum of terrestrial and marine organisms but debate continues as to whether lunar or tidal influences dominate [486-506]. Given the conjoined

Thorp KE. Energy Dynamics in Breast Cancer: Fields, Hormones & the Menstrual Cycle. G Med Sci. 2024; 5(1):169-249.
https://www.doi.org/10.46766/thegms.oncol.24111101

geomagnetic and developmental field and its relationship to both lunar and tidal oscillations the answer is both and neither: menstrual functions occur synchronously in relation to fluctuations in the field. The lunar and tidal periodicities are clearly less impactful than the metabolic and endocrine interactions that compose the cycle.

We have established that BC is associated with disturbances in the relationship between cells and the developmental field. We are less concerned with a detailed step-by-step account of the menstrual cycle or the role played by the various hormones that mediate this special function but, rather, simply to highlight key relationships in the field and the metabolic consequences that ensue when its orderly progression is disrupted. By the same token, given that up to two-thirds of women with BC are postmenopausal we must examine its energetic basis which, ultimately, takes origin in the disturbed circadian hormonal milieu. Before we attempt this, we must revisit Empedocles' EWAF framework and examine the nature of resonance and energy transmission, i.e., sympathy.

Conventional wisdom holds that light originates in the sun and 'travels' through space to illuminate earth. Based on similar logic we assume that fire is composed of heat and light. But it is more accurate to say that heat and light arise from an interaction *between* Fire Air or, more precisely, *between* radiofrequency (RF) waves and the geomagnetic field. This forms the very basis of resonance. In the case of heat the relationship is obvious: during daylight hours as the atmosphere is 'excited' heat is released thus raising ambient temperatures. As the resonant state of water in a pan on the stove increases once again heat is emitted. The relationship between the resonance field and light however is not so intuitive as it is generally assumed that light travels through space but is this accurate? The basis for this belief lies in the fact that as we gaze into the sky, we observe the sun aglow and thereby conclude that light is transmitted from one point to another. But equally one could argue that longitudinally directed RF waves passing through space generate light along their path via resonance. And upon reaching earth they set the atmosphere aglow on the same basis. In images of the earth taken from space it is immersed in complete darkness **(Figure 12).** If light actually traveled from the sun to the earth, it should illuminate the entire solar system like a grand stadium. Similarly, solar storm activity generates RF waves that, upon reaching earth at night, give rise to the Northern Lights **(Figure 13).** How to explain such celestial occurrences? We must assume that light and heat are emission phenomena. This concept will be addressed in an upcoming article on the nature of light.

Figure 12. Illuminated earth against the dark background of open space. Suggests light as an atmospheric emission phenomenon arising from an interaction between solar RF waves and the geomagnetic field.

https://eoimages.gsfc.nasa.gov/images/imagerecords/0/885/modis_wonderglobe_lrg.jpg

Thorp KE. Energy Dynamics in Breast Cancer: Fields, Hormones & the Menstrual Cycle. G Med Sci. 2024; 5(1):169-249.
https://www.doi.org/10.46766/thegms.oncol.24111101

Elise M. Thorp

Figure 13. Northern Lights (*Aurora Borealis*) October 10, 2024, Williamston, Michigan. Images reveal pulsatile oscillatory patterns with shifting color displays substantiating light as an emission phenomenon originating in the geomagnetic resonance field.

Based on resonance dynamics involving the flow and transmission of energy we identify three primary oscillational states, i.e., light, heat, and dark, which, from a temporal perspective, constitute what we will call the *threefold turning cycles.* Ancient Chinese cosmology immortalized the light/dark dynamic in the yin-yang (*taijitu*) symbol as distinct oscillational states that ransition back and forth on the basis of planetary rotation **(Figure 14).**

A third oscillator, heat, observed in both daily and seasonal rhythms, must be added to the mix. Like light and dark, heat fluctuates between higher, i.e., 'hot,' and lower, 'cold,' energy states. In both daily and seasonal rhythms light and heat dissipate during the dark phase. Far from being simply the absence of light or heat the dark phase is a distinct oscillational state that constitutes a period of equilibration and restoration **(Figure 15).**

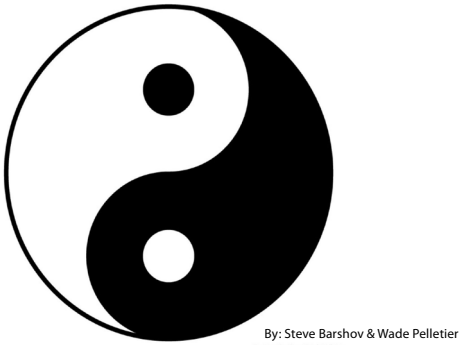

Figure 14. Chinese taijitu symbol depicting the primary oscillational dynamics between light and dark.

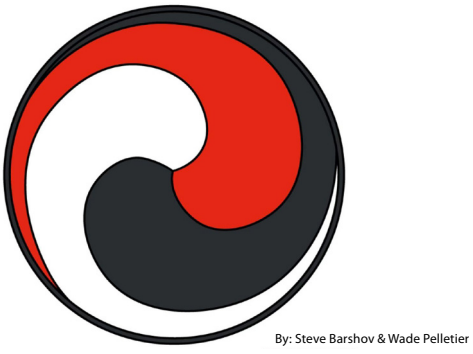

Figure 15. Threefold turning cycles as oscillational interactions between light, heat and dark which represent periods of energy release followed by obligatory repletion phase.

Thorp KE. Energy Dynamics in Breast Cancer: Fields, Hormones & the Menstrual Cycle. G Med Sci. 2024; 5(1):169-249.
https://www.doi.org/10.46766/thegms.oncol.24111101

Energy dynamics in the body are driven entirely by the threefold turning cycles. We may regard the daily light and heat phases, which occur during the waking state, as periods of active energy release while the dark-driven sleep phase is a recovery period. The menstrual cycle is organized along similar lines but extends over the lunar month. In each case the dark recovery phase is governed by a single hormone, melatonin, which mediates the transition in large measure by dissipation of body heat. We can conceive melatonin as the dark/cool principle in the body. In BC and other chronic diseases disruption of the nightly melatonin rhythm impairs the blood-borne metabolic field thus giving rise to a condition the ancients called 'insufficient cooling' [507].

The threefold turning cycles originate in interactions between the brain and cardiovascular system. The brain mediates light/dark relationships between the external environment and body. It is said to possess a 'biological clock' in the hypothalamus which governs sleep/wake cycles. Given that such cycles are highly variable from person to person it is obvious that this 'clock' is not strictly entrained to the environment but generates its own internal rhythms. But neither is it entirely independent: appropriately timed external light pulses alter daily sleep/wake patterns as well as the length of the menstrual cycle [508-521]. Nocturnal light pulses affect core body temperature by impairing heat dissipation thereby delaying or 'phase-shifting' sleep onset [522-529]. Such circadian effects are mediated by stimulating or inhibiting release of cortisol and melatonin from the pituitary and pineal glands [530-538].

According to chronobiologists, upon awakening light enters through the eyes and travels along nerve pathways to the hypothalamus which, in turn, suppresses secretion of melatonin by the pineal gland and relays signals to the pituitary gland triggering release of cortisol by the adrenal glands [539-543]. The hypothalamus also induces release of thyroid stimulating hormone by the pituitary which causes secretion of thyroid hormone (TH) initiating the heat phase of the cycle [544-548]. TH acts by enhancing cardiac function and increasing thermogenesis [549-553] which in large measure forms the basis of the daily body heat rhythm [554-558]. In the ancient system of natural correspondences, the heart was regarded as the 'sun of the microcosm' as William Harvey succinctly describes it in *On the Motions of the Heart* (1628) [559].

The daily threefold turning cycle is completed around darkfall as light recedes and melatonin is released into the circulation by the pineal gland. Discovered in 1958 melatonin is ubiquitous in animals, plants and even bacteria indicating its primordial role in mediating biological energy dynamics. Synthesized in virtually all cells of the body it circulates in the blood and ECF compartments. The amount of melatonin released by the pineal is but a fraction of that produced elsewhere in the body [560-572].

One of its prime effects at the organismic level is induction of vasodilation and heat dissipation which plays a pivotal role in sleep onset [573-588]. Beyond this it is endowed with a virtual litany of functions: acting in 'non-enzymatic' manner it possesses broad anti-inflammatory and anti-oxidant properties, acts as a free radical scavenger, improves mitochondrial function, induces gene expression via DNA methylation, enhances DNA repair, and the beat goes on and on [589-606].

But how does all of this transpire without increasing energy flow into cells? Based on Nordenström's test tube model this amounts to shifting cell physiology from the anode back toward the cathode. And given that this happens in organisms without beating hearts one can only assume that melatonin alters the resonance structure of cell water thereby

Thorp KE. Energy Dynamics in Breast Cancer: Fields, Hormones & the Menstrual Cycle. G Med Sci. 2024; 5(1):169-249.
https://www.doi.org/10.46766/thegms.oncol.24111101

importing energy from the geomagnetic field into the fluid compartment. What other mechanism suffices?

Wakefulness is highly energy intensive. During the transition from sleep there is widespread cortical activation as EEG frequencies shift from low to high ranges which seems to be modulated by the catabolic 'stress' hormone cortisol. The morning cortisol surge is associated with heightened perceptual discrimination and frontal executive functions which appear to occur on the basis of regional alterations in EEG activity and energy disposition in the CNS [607-611]. In conjunction with this resting heart rate often increases by 10-20 beats/minute to provide heightened current flow into the nervous system [612-615].

In a series of insightful studies Van Cauter shows that sleep disruption adversely impacts glucose metabolism and insulin resistance inducing a 'pre-diabetic state' that promotes weight gain and obesity [616-623]. These conditions, in turn, are secondary to impaired mitochondrial function [624-629]. And herein lies the link between the metabolic syndrome (MetS), postmenopausal BC, and deterioration of the dark phase of the threefold turning cycles.

In a recent piece I show that every single aspect of MetS is a consequence of deterioration of the blood-borne energy field [630]. Hormonal alterations in MetS include elevated cortisol levels [631-636], resistance to the actions of thyroid hormone [637-643], and deficient melatonin secretion [644-650] which, collectively, implicate the threefold turning cycles. We should also take stock of similarities between MetS and BC: both are defined by inflammation, mitochondrial dysfunction, and oxidative stress. Similarly, studies describe links between BC and impaired thyroid function [651-655], Given such shared attributes it is hardly surprising that BC is more common among women w/ MetS [656-666].

Melatonin supplementation in MetS improves glucose metabolism and insulin resistance [667-674], mitochondrial function [675-688], inflammatory markers [689-699], as well as associated conditions like non-alcoholic fatty liver [700-703] and polycystic ovary syndrome [704-707]. Once again, all of these energy-dependent actions must shift cellular metabolism toward the cathodic pole. From where else would this energy transfer originate *other* than the fluid compartment? And by what means *other* than resonance?

At the cellular level cancer begins with alterations between opposing light- and dark-mediated physiologic pathways. Such imbalances, in turn, drive the evolution of two hallmarks: sustained proliferation and replicative immortality. Through the aegis of the hypothalamic-pituitary axis, estrogen production is stimulated in breast tissue triggering cell mitosis [708-714]. This in turn plays into the well-recognized association between mammographically dense breasts and BC [715-722]. A recent large epidemiological study suggests that breast density and BMI interact 'synergistically' to increase postmenopausal BC risk [723] **(Figure 16).**

Thorp KE. Energy Dynamics in Breast Cancer: Fields, Hormones & the Menstrual Cycle. G Med Sci. 2024; 5(1):169-249.
https://www.doi.org/10.46766/thegms.oncol.24111101

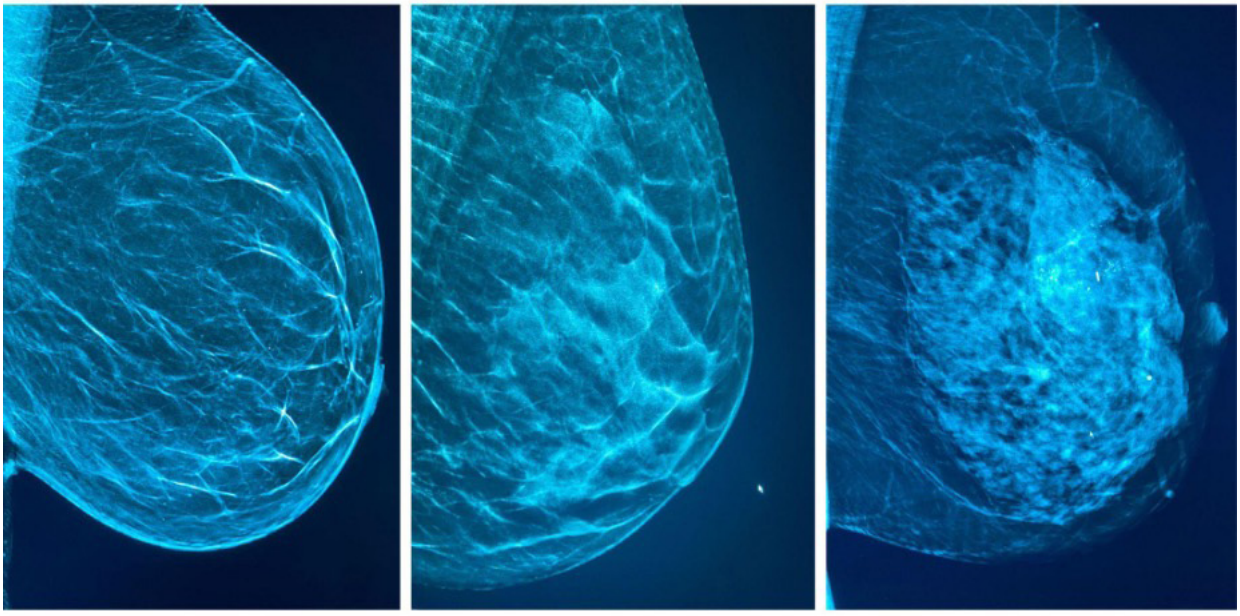

Figure 16. Variations in mammographic breast density reflect the hormonal milieu of the monthly menstrual cycle with highly dense breasts (far right) related to heightened estrogenic stimulation.

Elevated blood estrogen levels in postmenopausal women are associated with heightened BC risk [724-735]. Inside cells estrogen levels may be 15-25X higher than in the blood [736-739]. In breast tissue estrogen has pro-oxidative effects related to its impact on cellular replication which is an energy-dependent process [740-745]. Sustained cellular division promotes mitochondrial dysfunction, a shift toward the anodic pole and acid-generating pathways which, ultimately, culminates in the 'metabolic switch' and reversion to Warburg metabolism.

Increased estrogenic activity leads to down-regulation of uncoupling proteins (UCPs), functional elements along the inner mitochondrial membrane, a primary source of body heat [746]. Thermogenesis augments metabolic efficiency as well as reducing oxidative stress and generation of reactive oxygen species [747-752]. Estrogenic modulation of UCPs translates functionally into impairment of the heat field and resistance to TH [753-759]. UCP dysfunction and blunted thermogenesis, characteristic of obesity and MetS, are associated with increased inflammation-promoting white adipose tissue (WAT) and diminished mitochondria-laden brown adipose tissue (BAT) [760-769]. WAT inflammation is also a feature of BC [770-773]. Melatonin stimulates conversion of WAT to BAT enhancing thermogenesis [774-777].

Disturbed UCP function has been linked with transition to the Warburg state and tumor aggressiveness [778-780]. A key juncture appears to involve the cortisol receptor on cell membranes which induces what is known as the epithelial-to-mesenchymal transition (EMT) [781, 782]. EMT is a normal part of morphogenesis and wound healing. In cancer, EMT initiates dedifferentiation toward more primitive stem cell lines which, in turn, engender hallmarks like intratumoral heterogeneity, invasiveness, and metastasis [783-790]. It is associated with loss of function of the BRCA1 gene [791-794]. EMT seems to demarcate the point at which functional breast tissue falls out of relationship with the developmental field and begins to assert its own gene-driven survival agenda.

Thorp KE. Energy Dynamics in Breast Cancer: Fields, Hormones & the Menstrual Cycle. G Med Sci. 2024; 5(1):169-249.
https://www.doi.org/10.46766/thegms.oncol.24111101

Melatonin, the dark/cool principle, opposes the estrogen- and cortisol-mediated effects and shifts metabolism toward the cathodic pole. Melatonin's 'oncostatic' properties lie in its ability to decrease cellular proliferation and induce apoptosis in tumor cell lines [795-802]. It directly reduces hypothalamic-pituitary activity [803-807] and down-regulates aromatase and sulfatase, two key enzymes involved in estrogen synthesis [808-816]. As anti-estrogenic drugs like the selective estrogen receptor modulators also induce apoptosis in BC cells it is likely that melatonin, via enzymatic inhibition, triggers apoptosis by shifting current flow away from estrogen-mediated pathways [817-821]. It seems intuitively inescapable that such energetic transformations originate in the water compartment and involve the developmental field. Any other mechanism invokes Einstein's 'spooky action at a distance'.

Postmenopausal BC and MetS share another light-dark imbalance: not only is melatonin release impaired but such women are often vitamin D deficient [822-829]. Up to two-thirds of women with BC have low blood vitamin D levels and tumors tend to be more aggressive with worse overall survival compared to women with normal vitamin D levels [830-835]. Women with normal vitamin D levels also have decreased breast density [836-840]. Vitamin D, like melatonin, blocks estrogen synthesis and promotes tumor cell apoptosis [841, 842]. Thus, as others point out, women with BC not only tend to have excessive nocturnal light exposure but insufficient sunlight exposure during the daylight period [843-845]. It would seem that what the wakeful state draws from the blood in terms of energy is offset by influx of energy through the skin in the form of vitamin D. Such vertically-mediated energy transmission must involve the ECF compartment beneath the skin as Empedocles' EWAF framework would imply.

BREAST CANCER & THE MENSTRUAL CYCLE

In the 1990s chronobiology emerged as a central factor in BC oncogenesis after studies found an increased incidence of BC in airline cabin attendants [846-857]. Rapid air travel across multiple time zones induced alterations in cortisol and melatonin rhythms, sleep-wake patterns, and desynchronization of body heat rhythms which persisted for up to 5 days after women returned home [858-860]. Earlier studies found that menstrual cycle abnormalities were common among stewardesses [861, 862]. Various studies also documented heightened BC risk in women engaged in night shift work [863-870].

Night shift work has been linked to weight gain, diabetes, heart disease, stroke, and various cancers [871-880]. These effects involve the developmental field and play into two other hallmarks: epigenetic dysregulation and genetic instability. Disruption of the circadian time structure is associated with altered gene expression in cells throughout the body including DNA repair genes [881-884]. Once again disturbances in the cortisol-melatonin axis, reproductive hormones and daily heat rhythms loom large [885-894]. Individuals who develop intolerance to shiftwork often have diminished amplitude of their daily heat rhythm which may undergo internal desynchronization with the sleep-wake cycle or even become free-floating [895-898]. All of this ties into the ubiquitous light-at-night issue.

It is now firmly established that artificial light-at-night is a major contributor to the expanding global burden of chronic disease. It is associated with MetS, diabetes, obesity, as well as breast, prostate, lung and colorectal cancers [899-912]. Given that up to 80% of the world population is exposed to artificial light-at-night on a regular basis it is a serious public health concern [913-915]. Artificial light-at-night, like smoking, is potentially carcinogenic. The fundamental disturbance, increasingly, seems

Thorp KE. Energy Dynamics in Breast Cancer: Fields, Hormones & the Menstrual Cycle. G Med Sci. 2024; 5(1):169-249.
https://www.doi.org/10.46766/thegms.oncol.24111101

to hinge on disruption of the nocturnal melatonin rhythm. Richard Stevens' melatonin hypothesis is affirmed. But how does this impact the relationship with the developmental field?

The orthodox account of hormonal interactions between the brain and its 'target' organs is based on a linear sequential mechanism: alterations in environmental light/dark patterns cause the hypothalamus to release factors that stimulate the pituitary gland to secrete messenger substances which then induce organs like the adrenals, thyroid, ovaries, or breasts to synthesize and secrete hormones such as cortisol, TH, estrogen, progesterone etc. But this is not quite accurate.

All the hormonal principals mediating circadian and menstrual cycle functions, including melatonin, are released in pulsatile fashion and it seems that oscillatory patterns determine how effects are mediated. Researchers describe a complex 'temporal endocrine structure' that fluctuates based on 'feedback loops' between a host of 'internal and external stimuli' implicating an elaborate resonance structure [916-926]. Gonadotropin releasing hormone (GnRH), for example, triggers release of either LH or FSH by the pituitary solely on the basis of pulse frequency and amplitude and this plays into various disorders of the menstrual cycle [927-935]. A given hormone may not elicit the same effects at different times of the cycle [936].

In one study transition from dim to bright light in the morning induced an 'immediate' elevation of cortisol levels [937]. Another found that morning exposure to light resulted in an 'immediate' secretion of LH and FSH into the blood along with fluctuations in pulse frequency and amplitude [938]. Yet another found that in normally cycling women nocturnal light exposure triggered simultaneous changes in blood LH, FSH and melatonin levels [939]. And as melatonin levels decrease upon nocturnal light exposure cortisol levels rise as one would expect based on the taijitu dynamic [940].

It is often overlooked that melatonin, GnRH, the various pituitary intermediaries, and every circadian and menstrual cycle hormone are present in the blood *all the time* and their plasma concentrations are constantly changing with respect to one another. This impossibly complex oscillatory structure represents the resonant medium through which interactions with the developmental field take place. This substantiates the primacy of the blood-borne energy field as well as Galen's *omnia incipit in sanguine* doctrine. So how does melatonin impact the menstrual cycle?

Melatonin regulates reproductive cycles in a wide range of species including humans [941-948]. Acting on the ovary it stimulates granulosa cell function which induces estrogen synthesis and the development of eggs in follicles [949-953]. After ovulation it promotes progesterone synthesis by the corpus luteum and maturation of oocytes [954-960]. It delays oöcyte aging and prolongs the fertile window [961-963]. It supports angiogenesis throughout the cycle [964-973]. But melatonin *isn't* the menstrual cycle. It is only the darkness of the theater through which the performance unfolds. It acts by enhancing mitochondrial function, reducing oxidative stress, and shifting metabolism back toward the cathodic pole [974-982]. Plain and simple.

Women living in the far northern reaches of the globe often stop menstruating during the dark phase of the yearly cycle coincident with surging melatonin levels only to have their cycles return in the spring as light and estrogen levels increase [983, 984]. Clearly two principles – light and dark – are involved and must be present in some prescribed balance. How could such effects transpire other than by altering the resonance structure of the

Thorp KE. Energy Dynamics in Breast Cancer: Fields, Hormones & the Menstrual Cycle. G Med Sci. 2024; 5(1):169-249.
https://www.doi.org/10.46766/thegms.oncol.24111101

blood and ECF compartment? No spooky action at a distance is needed.

The biphasic menstrual cycle body temperature rhythm, first described in the late 19th century, has been used for over a century to predict ovulation [985]. During the follicular (estrogenic) phase core body temperature slowly rises until shortly before ovulation at which time it dips by up to 0.5°F. Afterward progesterone secretion by the corpus luteum increases sharply and basal body temperature rises by up to 1°F or more until menstruation at which time it again drops **(Figure 17).**

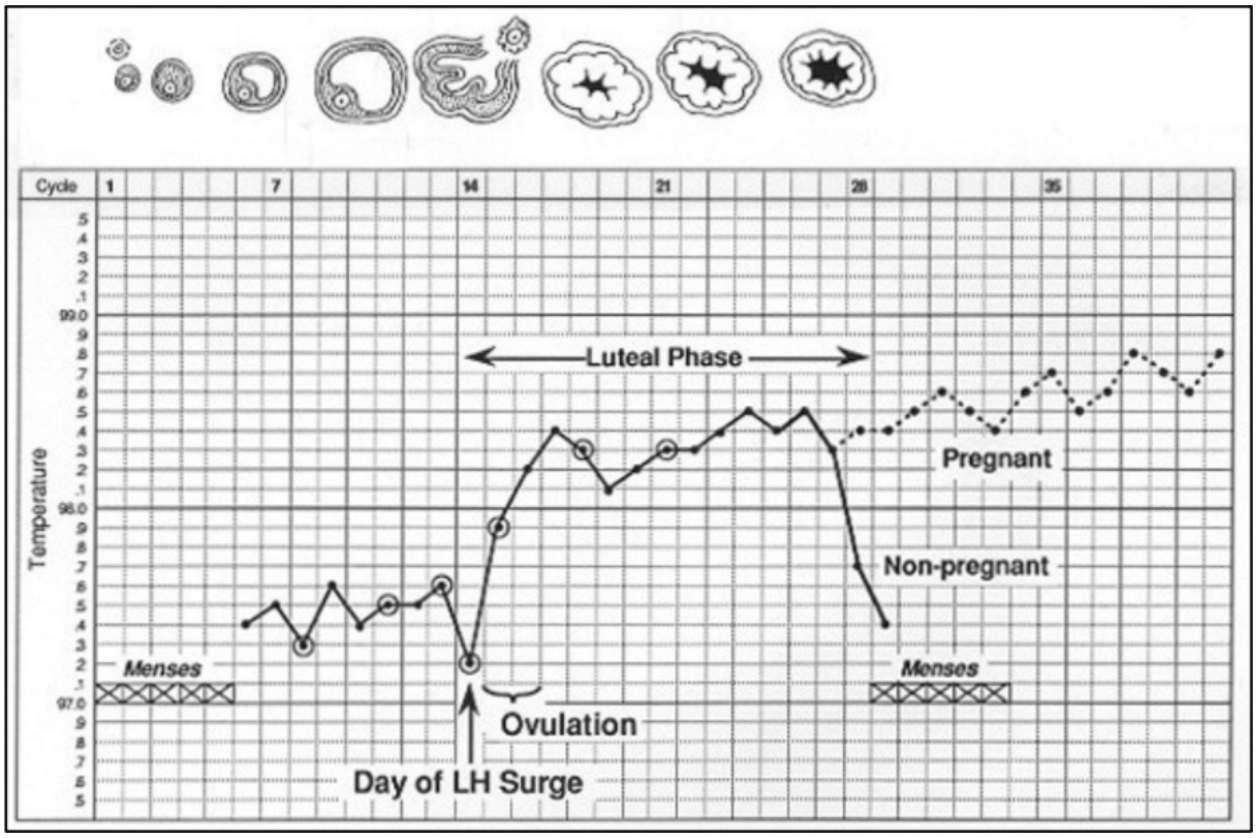

Figure 17. The monthly biphasic menstrual cycle body heat rhythm.

https://www.uwmedicine.org/sites/stevie/files/2018-11/Basal-Body-Temperature-Chart.pdf

Research over the past 5 decades indicates that impaired corpus luteal function plays into BC oncogenesis [986-996]. A 1974 study raised concerns that 'persistent estrogenic stimulation, in the absence of an adequate progestational phase, may provide a hormonal setting in the mammary gland favorable to the development of carcinoma' [997]. One study tracking infertile women over three decades found a 5-fold increase in BC in those with low progesterone levels [998]. Since then luteal phase insufficiency has been a recurrent motif in BC research circles though scientists remain divided as to its basis.

In the 1990s scientists began tracking breast skin temperatures with a wearable 'chronobra' they described as an 'internal bioassay' of breast vascularity [999-1001]. Over 20 years of monitoring in a range of women including BC survivors and non-survivors, they found 'striking'

Thorp KE. Energy Dynamics in Breast Cancer: Fields, Hormones & the Menstrual Cycle. G Med Sci. 2024; 5(1):169-249.
https://www.doi.org/10.46766/thegms.oncol.24111101

heat rhythm variations during the luteal phase. Instead of biphasic cycles women with BC had monophasic monthly heat patterns with higher average temperatures and lower oscillational amplitude. BC non-survivors had higher breast skin temperatures consistent with insufficient cooling and an abnormal progesterone-associated metabolic state [1002-1009]. Researchers claim this method has 90% sensitivity. Given this why would a woman even consider mammography?

Such findings are in accord with other lines of evidence. It is said that estrogen has pro-oxidative effects while progesterone reduces oxidative stress but this is hard to reconcile with its known proliferative effects on breast tissue [1010-1014]. Is it really progesterone that is anti-oxidative [1015-1018]? As progesterone levels surge following ovulation there is a corresponding rise in melatonin prompting questions as to the origins of luteal phase insufficiency [1019-1022]. Increased melatonin should stimulate thermogenesis and likely induces the post-ovulatory rise in basal body temperature. Decreased melatonin would explain both defective thermogenesis and deficient progesterone. Similar dynamics seem to play into hormone receptor status in BC.

For years oncologists have used the presence or absence of hormonal receptors on the surface of BC cells not only as a means to gauge prognosis but in directing therapy [1023-1027]. Tumors with estrogen (ER+) and progesterone (PR+) receptors, usually more differentiated and less aggressive, are responsive to hormonal therapies and carry a better prognosis. Tumors lacking receptors, i.e., ER- and PR-, on the other hand, tend to grow more rapidly and are resistant to hormonal therapies. Triple-negative BC, even more aggressive and with worse outcomes, is both ER- and PR- and lacks the HER2 receptor [1028, 1029]. Receptor status likely represents an approximate indicator of when during the menstrual cycle tumor cells fell out of relationship with the developmental field.

Serum melatonin concentrations and/or the presence of melatonin receptors are higher in tumors with more favorable prognosis, i.e., ER+ and PR+, and decrease in 'stage-dependent' fashion as they become more aggressive and trend toward receptor negativity with lowest levels in triple-negative tumors [1030-1037]. Melatonin levels correlate with tumor size [1038]. And black women with TNBC, who are 30% more likely to die from the disease than white women with the same diagnosis, not only have lower vitamin D levels but are more likely to have decreased blood melatonin and receptor levels [1039].

Regarding its effects vis à vis the cancer hallmarks, as others point out, we find that melatonin plays a key role in each and every case [1040]. And increasingly its actions are thought to be mediated at the epigenetic level [1041]. Calling it a 'full-service anti-cancer agent', Russell Reiter, grand guru of melatonin research with hundreds of published articles, argues that it regulates epigenetic influences in BC and points to effects on DNA methylation, gene induction, cell cycle modulation, and circadian rhythm regulation. Such diverse actions 'suggest that what is being observed are merely epiphenomena of an underlying more fundamental action of melatonin that remains to be disclosed' [1042, 1043].

It would seem that melatonin, as primary mediator of energy metabolism and efficient causes in living bodies, represents the means by which the 'epigenetic landscape' is biologically translated, finally offering a comprehensive explanation for experimental phenomena described by early 20th century embryologists. This, in turn, substantiates the developmental/morphogenic field and Aristotle's layer of formal causes. Breast cancer is not a local cellular or molecular disorder as our 20th century scientists long believed.

Thorp KE. Energy Dynamics in Breast Cancer: Fields, Hormones & the Menstrual Cycle. G Med Sci. 2024; 5(1):169-249.
https://www.doi.org/10.46766/thegms.oncol.24111101

EPILOGUE

Our deliberations affirm Galen's designation of BC as a metabolic disorder arising from a disturbed nexus of relations in the blood-borne energy field. Scientists spent the last century teasing apart and analyzing the complex maze of molecular pathways, chasing elusive genetic phantoms, and erecting a monumental edifice of fact-based structural knowledge which, we now realize, corresponds to that layer of relations Aristotle classified as material causes. And none of their mainstay therapies directed toward BC meaningfully impact energy dynamics in the tumor microenvironment, i.e., the ECF compartment. BC survivors remain at heightened risk for recurrence as well as a multitude of second primary cancers.

Ultimately scientists' failure to clarify what 'metabolism' actually represents, to have recognized the primacy of the energetic field, or to have integrated decades of research regarding the epigenetic determinants of BC, i.e., the efficient and formal causal layers, led to the present grave epistemic crisis, one which science historian Thomas Kuhn had earlier referred to as a 'paradigm collapse'. BC will forever be remembered as the disease that killed the cellular and molecular science paradigm.

The very ideas that would have enabled scientists to escape their conceptual labyrinth, namely Aristotle's hierarchical causal account of the life principle, Galen's omnia incipit in sanguine doctrine, Empedocles' EWAF framework, and the notion of the world soul, had been summarily rejected by early scientists at the onset of their centuries-long odyssey. We may regard these ancient concepts as the ultimate context in which the multitude of orphan facts, gleaned through decades of tedious piecemeal experimental research, can now finally find repose.

Given that the genetic hypothesis has failed to deliver on its promises, and that during the 20th century the developmental field concept continued to resurface and seems to hold the answer to many lingering questions, it is now incumbent upon our scientists to explain why it doesn't suit their fancy and to provide a credible refutation of evidence presented herein.

REFERENCES

1. History of mastectomy before and after Halsted. Ghossain A, Ghossain MA. *J Med Librar.* 2009 Apr; 57(2):65-71.

2. History of surgery for breast cancer: Radical to the sublime. Cotlar AM, Dubose JJ, Rose DM. *Curr Surg.* 2003 May; 60(3):329-37.

3. Evolution of radical mastectomy for breast cancer. Plesca M, Bordea C, El Houcheimi B, et al. *J Med Life.* 2016 Apr; 9(2):183-86.

4. Ductal carcinoma in situ of the breast: Finding the balance between overtreatment and undertreatment. Delaloge S, Khan SA, Wesseling J, et al. *Lancet.* 2024 Jun; 403(10445):2734-46.

5. Finding the balance between over- and under-treatment of ductal carcinoma in situ (DCIS). Groen EJ, Elshof LE, Visser LL, et al. *Breast.* 2017 Feb; 31:274-83.

6. Treatment of ductal carcinoma in situ: An uncertain harm-benefit balance. [No authors listed]. *Prescrire Int.* 2013 Dec; 22(144):298-303.

7. Menstrual influence on surgical cure of breast cancer. Hrushesky WJ, Bluming AZ, Gruber SA, et al. *Lancet.* 1989 Oct; 2(8669):949-52.

Thorp KE. Energy Dynamics in Breast Cancer: Fields, Hormones & the Menstrual Cycle. G Med Sci. 2024; 5(1):169-249.
https://www.doi.org/10.46766/thegms.oncol.24111101

8. The relationship between surgery and phase of the menstrual cycle affects survival in breast cancer. Kucuk AI, Atalay C. *Breast Cancer.* 2012 Dec; 15(4):434-40.

9. Timing of surgery during the menstrual cycle and survival of premenopausal women with operable breast cancer. Badwe RA, Gregory WM, Chaudary MA, et al. 1991 May; 337(8752):1261-64.

10. Timing of surgery in relation to the menstrual cycle in premenopausal women with operable breast cancer. Saad Z, Bramwell V, Duff J, et al. *Br J Surg.* 1994 Feb; 81(2):217-20.

11. Timing of breast cancer excision during the menstrual cycle influences duration of disease-free survival. Senie RT, Rosen PP, Rhodes P, et al. *Ann Int Med.* 1991 Sep; 115(5):337-42.

12. Fertility cycle influence on surgical breast cancer cure. Bove K, Lincoln DW, Wood PA, et al. *Breast Cancer Res Treat.* 2002 Sep; 75(1):65-72.

13. The effects of surgery on tumor growth: A century of investigations. Demicheli R, Retsky MW, Hrushesky WJ, et al. *Ann Oncol.* 2008 Nov; 19(11):1821-28.

14. Is surgical trauma prometastatic? Lejeune FJ. *Anticancer Res.* 2012 Mar; 32(3):947-51.

15. Potential impact of invasive surgical procedures on primary tumor growth and metastasis. Alieva M, van Rheenen J, Broekman MLD. *Clin Exp Metastasis.* 2018 Apr; 35(4):319-31.

16. Role of hormones in mammary cancer initiation and progression. Russo IH, Russo J. *J Mammary Gland Biolo Neoplasia.* 1998 Jan; 3(1):49-61.

17. Unveiling the role of hormonal imbalance in breast cancer development: A comprehensive review. Satpathi S, Gaurkar SS, Potdukhe A, et al. *Cureus.* 2023 Jul; 15(7):e41737.

18. Hormone and breast cancer. Gompel A. *Press Med.* 2019 Oct; 8(10):1085-91.

19. Hormone therapy in breast cancer. Drăgănescu M, Carmocan C. *Chirurgia (Bucur).* 2017 Jul; 112(4):413-17.

20. Pharmacogenomics and Endocrine Therapy in Breast Cancer. Hayes DF, Rae JM. *J Clin Oncol.* 2020 Feb; 38(6):525-28.

21. Molecular mechanisms of anti-estrogen therapy resistance and novel targeted therapies. Ozyurt R, Ozpolat B. *Cancers (Basel).* 2022 Oct; 14(21):5206.

22. The estrogen receptor: A model for molecular medicine. Jensen EV, Jordan VC. *Cancer Res.* 2003 Jun; 9(6):1980-89.

23. Aromatase inhibitors: role in postmenopausal breast cancer. Kharb R, Haider K, Neha K, et al. *Arch Pharm (Weinheim).* 2020 Aug; 353(8):e2000081.

24. Breast cancer, estrogen receptor and ligands. Bai Z, Gust R. *Arch Pharm (Weinheim).* 2009 Mar; 342(3):133-49.

25. Ovarian function suppression in pre-menopausal women with early-stage breast cancer. Lambertini M, Del Mastro L, Viglietti G, et al. *Curr Treat Options Oncol.* 2017 Jan; 18(1):4.

26. Role of ovarian function suppression in premenopausal women with early breast cancer. Park WC. *J Breast Cancer.* 2016 Dec; 19(4):341-48.

27. Mammographic screening: A major issue in medicine. Autier P, Boniol M. *Eur J Cancer.* 2018 Feb; 90:34-62.

28. The benefits and harms of breast cancer screening: An independent review. Independent UK Panel on Breast Cancer Screening. *Lancet.* 2012 Nov; 380(9855):1778-86.

Thorp KE. Energy Dynamics in Breast Cancer: Fields, Hormones & the Menstrual Cycle. G Med Sci. 2024; 5(1):169-249.
https://www.doi.org/10.46766/thegms.oncol.24111101

29. Breast cancer screening. Jatoi I. *Am J Surg.* 1999 Jun; 177(6):518-24.

30. Screening for breast cancer: Worries about its effectiveness. Autier P. *Rev Prat.* 2013 Dec; 63(10):1369-77.

31. Mammographic screening of women aged 40-49 years: Is it justified? Feig SA. *Obstet Gynecol Clin North Am.* 1994 Dec; 21(4):587-606.

32. Effect of mammographic screening from age 40 on breast cancer mortality at 10 years' follow-up: A randomized controlled trial. Moss SM, Cuckle H, Evans A, et al. *Lancet.* 2006 Dec; 368(9552):2053-60.

33. Breast cancer screening: A summary of the evidence for the US Preventive Services Task Force. Humphrey LL, Helfand M, Chan BK, et al. *Ann Intern Med.* 2002 Sep; 137(5 Pt1):347-60.

34. Effectiveness of breast cancer screening: Systematic review and meta-analysis to update the 2009 US Preventive Services Task Force Recommendation. Nelson HD, Fu R, Cantor A, et al. *Ann Intern Med.* 2016 Feb; 164(4):244-55.

35. Overdiagnosis and risks of breast cancer screening. Neal CH, Helvie MA. *Radiol Clin North Am.* 2021 Jan; 59(1):19-27.

36. Benefits and harms from mammographic screening. Løberg M, Lousdal ML, Bretthauer M, et al. *Breast Cancer Res.* 2015 May; 17(1):3.

37. Mammographic screening: Patterns of use and estimated impact on breast cancer survival. Blanchard K, Colbert JA, Puri D, et al. *Cancer.* 2004 Aug; 101(3):495-507.

38. The BRCA1 and BRCA2 genes in early-onset breast cancer patients. Saleem M, Ghazali MB, Wahab MAMA, et al. *Exp Med Biol.* 2020; 1292:1-12.

39. Meta-analysis of BRCA1 and BRCA2 penetrance. Chen S, Parmigiani G. *J Clin Oncol.* 2007 Apr; 25(11):1329-33.

40. Hereditary breast cancer: Genetic penetrance and current status with BRCA. Mahdavi M, Nassiri M, Kooshyar MM, et al. *J Cell Physiol.* 2019 May; 234(5):5741-50.

41. Breast Cancer Risk Genes - Association Analysis in More than 113,000 Women. Breast Cancer Association Consortium. Dorling L, Carvalho S, Allen J, et al. *NEJM.* 2021 Feb; 384(5):428-39.

42. How many more breast cancer predisposition genes are there? Easton DF. *Breast Cancer Res.* 1999; 1(1):14-17.

43. The spectrum of genetic mutations in breast cancer. Sheikh A, Hussain SA, Ghori Q, et al. *Asian Pac J Cancer Prev.* 2015; 16(6):2177-85.

44. Breast cancer genes: Beyond BRCA1 and BRCA2. Filippini SE, Vega A. *Front Biosci.* 2013 Jun; 18(4):1358-72.

45. Genomic variants and worldwide epidemiology of breast cancer: A genome-wide association studies correlation analysis. da Costa Nunes GG, de Freitas LM, Monte N, et al. *Genes (Basel).* 2024 Jan; 15(2):145.

46. The genetics of breast cancer risk in the post-genome era: Thoughts on study design to move past BRCA and towards clinical relevance. Skol AD, Sasaki MM, Onel K. *Breast Cancer Res.* 2016 Oct; 18(1):99.

47. BRCA Gene Mutations: Cancer Risk and Genetic Testing. *National Cancer Institute.* https://www.cancer.gov/about-cancer/causes-prevention/genetics/brca-fact-sheet#:~:text=and%20become%20cancer.-,How%20much%20does%20an%20inherited%20harmful%20variant%20in%20BRCA1%20or,age%20(2%E2%80%934)

Thorp KE. Energy Dynamics in Breast Cancer: Fields, Hormones & the Menstrual Cycle. G Med Sci. 2024; 5(1):169-249.
https://www.doi.org/10.46766/thegms.oncol.24111101

48. Breast cancer and atypia among young and middle-aged women: A study of 110 medicolegal autopsies. Nielsen M, Thomsen JL, Primdahl S, et al. *Br J Cancer.* 1987 Dec; 56(6):814-19.

49. Bilateral breast cancer. Donovan AJ. *Surg Clin North Am.* 1990 Oct; 70(5):1141-49.

50. Bilateral breast cancer. Fracchia AA, Borgen PI. *Semin Surg Oncol.* 1991 Sep; 7(5): 300-05.

51. The prevalence of carcinoma in situ in normal and cancer-associated breasts. Alpers CE, Wellings SR. *Human Pathol.* 1985 Aug; 16(8):796-807.

52. Preneoplastic lesions in the human breast. Jensen HM, Rice JR, Wellings SR. *Science.* 1976 Jan; 191(4224):295-97.

53. Biology of breast preneoplasia. Cardiff RD, Wellings SR, Faulkin L. *J Cancer.* 1977 Jun; 39(6 Suppl):2734-46.

54. Persistent and atypical lobules in the human breast may be precancerous. Wellings SR, Jensen HM, DeVault MR. *Experientia.* 1976 Nov; 32(11):1463-65.

55. Considerations on the preneoplastic lesions of the mammary gland. Gullino PM. *J Pathol.* 1977 Nov; 89(2):413-30.

56. Association of breast density with breast cancer risk among women aged 65 years or older by age group and body mass index. Advani SM, Zhu W, Demb J, et al. *JAMA Netw Open.* 2021 Aug; 4(8):e2122810.

57. Mammographic breast density and the risk of breast cancer: A systematic review and meta-analysis. Bodewes FTH, van Asselt AA, Dorrius MD, et al. *Breast.* 2022 Dec; 66:62-68.

58. Association of the interaction between mammographic breast density, body mass index, and menopausal status with breast cancer risk among Korean women. Tran TXM, Moon SG, Kim S, et al. *JAMA Netw Open.* 2021 Dec; 4(12):e2139161.

59. Mammographic breast density and survival in women with invasive breast cancer. Pizzato M, Carioli G, Rosso S, et al. *Cancer Causes Control.* 2022 Sep; 33(9):1207-13.

60. Investigation of mammographic breast density as a risk factor for ovarian cancer. Henderson LM, Buist DS. *J Natl Cancer Inst.* 2014 Jan; 106(1):djt341.

61. Mammographic breast density and risk of ovarian cancer in Korean Women. Tran TXM, Kim S, Park B, et al. *Cancer Epidemiol Biomarkers Prev.* 2023 Dec; 32(12):1690-98.

62. High prevalence of deleterious BRCA1 and BRCA2 germline mutations in Arab breast and ovarian cancer patients. Alhuqail AJ, Alzahrani A, Almubarak H, et al. *Breast Cancer Res Treat.* 2018 Apr; 168(3):695-702.

63. The role of BRCA1/2 in hereditary and familial breast and ovarian cancers. Hawsawi YM, Al-Numair NS, Sobahy TM, et al. *Mol Genet Genomic Med.* 2019 Sep; 7(9):e879.

64. Spectrum of germline BRCA1 and BRCA2 variants identified in 2351 ovarian and breast cancer patients referring to a reference cancer center in Rome. Santonocito C, Rizza R, Paris I, et al. *Cancers (Basel).* 2020 May; 12(5):1286.

65. BRCA1 and BRCA2 breast/ovarian cancer susceptibility gene products and participants in DNA double-strand break repair. O'Donovan PJ, Livingston DM. *Carcinogenesis.* 2010 Jun; 31(6):961-67.

Thorp KE. Energy Dynamics in Breast Cancer: Fields, Hormones & the Menstrual Cycle. G Med Sci. 2024; 5(1):169-249.
https://www.doi.org/10.46766/thegms.oncol.24111101

66. Homologous recombination deficiency in ovarian, breast, colorectal, pancreatic, non-small cell lung and prostate cancers, and the mechanism of resistance to PARP inhibitors. Mekonnen N, Yang H, Shin YK. *Front Oncol.* 2022 Jun; 12:880643.

67. Risks of subsequent primary cancers among breast cancer survivors according to hormone receptor status. Sung H, Freedman RA, Siegel RL, et al. *Cancer* 2021 Sep; 127(18):3310-24.

68. Elevated risks of subsegmental endometrial cancer development among breast cancer survivors with different hormone receptor status: A SEER analysis. Liu J, Jiang W, Mao K, et al. *Breast Cancer Res Treat.* 2015 Apr; 150(2):439-45.

69. Uterine cancer in breast cancer survivors: A systematic review. Wijayabahu AT, Egan KM, Yaghjyan L. *Breast Cancer Res Treat.* 2020 Feb; 180(1):1-19.

70. Breast cancer risk factors and second primary malignancies among women with breast cancer. Trentham-Dietz A, Newcomb PA, Nichols HB, et al. *Breast Cancer Res Treat.* 2007 Oct; 105(2):195-207.

71. Incidence of second primary malignancy after breast cancer and related risk: Is breast-conserving surgery safe? A nested case-control study. Li Z, Wang K, Shi Y, et al. *Int J Cancer.* 2020 Jan; 146(2):352-62.

72. Risk of second primary cancer among women in the Kaiser Permanante Breast Cancer survivors cohort. Ramin C, Veiga LHS, Vo JB, et al. *Breast Cancer Res.* 2023 May; 25(1):50.

73. Risk factors for developing both primary breast and ovarian cancer: A systematic review. Ferris JS, Morgan DA, Tseng AS, et al. *Crit Rev Oncol Hematol.* 2023 Oct; 190:104081.

74. Risk of second primary female genital malignancies in women with breast cancer: A SEER analysis. Li Z, Wu Q, Song J, et al. *Horm Cancer.* 2018 Jun; 9(3):197-204.

75. Multiple primary cancers of the breast, endometrium and ovary. Ewertz M, Storm HH. *Eur J Cancer Clin Oncol.* 1989 Dec; 25(12):1927-32.

76. Common multiple primary cancers associated with breast and gynecologic cancers and their risk factors, pathogenesis, treatment and prognosis: A review. Ge S, Wang B, Wang Z, et al. *Front Oncol.* 2022 Jun; 12:840431.

77. Global trend of breast cancer mortality rate: A 25-year study. Azamjah N, Soltan-Zadeh Y, Zayeri F. *Asian Pac J Cancer Prev.* 2019 Jul; 20(7):2015-20.

78. Global incidence and mortality of breast cancer: A trend analysis. Huang J, Chan PS, Lok V, et al. *Aging (Albany NY).* 2021 Feb; 13(4):5748-5803.

79. Incidence and mortality and epidemiology of breast cancer in the world. *Asian Pac J Cancer Prev.* 2016; 17(S3):43-46.

80. Global challenges and policy solutions in breast cancer control. Trapani D, Ginsburg O, Fadelu T, et al. *Cancer Treat Rev.* 2022 Mar; 104:1023.

81. Understanding breast cancer as a global health concern. Wilkinson L, Gathani T. *Br J Radiol.* 2022 Feb; 95(1130):20211033.

82. Current and future burden of breast cancer: Global statistics for 2020 and 2040. Arnold M, Morgan E, Rumgay H, et al. *Breast.* 2022 Dec; 66:15-23.

83. Breast cancer epidemiology and risk factors. Rojas K, Stuckey A. *Clin Obstet Gynecol.* 2016 Dec; 59(4):651-72.

Thorp KE. Energy Dynamics in Breast Cancer: Fields, Hormones & the Menstrual Cycle. G Med Sci. 2024; 5(1):169-249.
https://www.doi.org/10.46766/thegms.oncol.24111101

84. Population attributable risk of modifiable and nonmodifiable breast cancer risk factors in postmenopausal breast cancer. Tamimi RM, Spiegelman D, Smith-Warner SA, et al. *Am J Epidemiol.* 2016 Dec; 184(12):884-93.

85. Population attributable risks for modifiable lifestyle factors and breast cancer in New Zealand women. Hayes J, Richardson A, Frampton C. *Intern Med J.* 2013 Nov; 43(11):1198-204.

86. Migration patterns and breast cancer risk in Asian-American women. Ziegler RG, Hoover RN, Pike MC, et al. *J Nat Cancer Inst.* 1993 Nov; 85(22):1819-27.

87. Light exposure at night, sleep duration, melatonin, and breast cancer: A dose-response analysis of observational studies. Yang WS, Deng Q, Fan WY. *Eut J Cancer Prev.* 2014 Jul; 23(4):269-76.

88. Light at night exposure and risk of breast cancer: A meta-analysis of observational studies. Luo Z, Liu Z, Chen H, et al. *Front Public Health.* 2023 Dec; 11:1276290.

89. Light at night and risk of breast cancer: A systematic review and dose-response meta-analysis. Urbano T, Vinceti M, Wise LA. *Int J Health Geogr.* 2021 Oct; 200(1):44.

90. The effect of light exposure at night (LAN) on carcinogenesis via decreased nocturnal melatonin synthesis. Giudice A, Crispo A, Grimaldi M, et al. *Molecules.* 2018 May; 23(6):1308.

91. Light during darkness, melatonin suppression and cancer progression. Blask DE, Dauchy RT, Sauer LA, et al. *Neuro Endocrinol Lett.* 2002 Jul; 23(Suppl 2):52-56.

92. Artificial lighting in the industrialized world: Circadian disruption and breast cancer. Stevens RG. *Cancer Causes Control.* 2006 May; 17(4):501-07.

93. Electric light causes cancer? Surely you're joking, Mr. Stevens. Stevens RG. *Mutat Res.* 2009 Jul; 682(1):1-6.

94. The impact of obesity on breast cancer diagnosis and treatment. Lee K, Kruper L, Dieli-Conwright CM, et al. *Curr Oncol Rep.* 2019 Mar; 21(5):41.

95. The impact of obesity on breast cancer. Argolo DF, Hudis CA, Iyengar NM. *Curr Oncol Rep.* 2018 Apr; 20(6):47.

96. Metabolic syndrome and breast cancer: Prevalence, treatment response, and prognosis. Dong S, Wang Z, Shen K, et al. *Front Oncol.* 2021 Mar; 11:629666.

97. Relationship between Metabolic syndrome and postmenopausal breast cancer. Ekinci O, Eren T, Kurtoglu Yakici M, et al. *Cir Esp (Engl Ed).* 2020 Nov; 98(9):540-46.

98. Metabolic syndrome affects breast cancer risk in postmenopausal women: National Cancer Institute of Naples experience. Capasso I, Esposito E, Pentimalli F, et al. *Cancer Biol Ther.* 2010 Dec; 10(12):1240-43.

99. Metabolic syndrome, central obesity and insulin resistance are associated with adverse pathological features in postmenopausal breast cancer. Healy LA, Ryan AM, Carroll P, et al. *Clin Oncol (R Coll Radiol).* 2010 May; 22(4):281-88.

100. Insulin resistance and the development of breast cancer in premenopausal women: The Kangbuk Samsung Health Study. Lee J, Chang Y, Kim Y, et al. *Breast Cancer Res Treat.* 2022 Apr; 192(2):401-09.

101. Metabolic syndrome and breast cancer risk. Mohammed AM, Hamed HB, Noaman MK, et al. *J Egypt Natl Cancer Inst.* 2023 Dec; 35(1):42.

102. The association of metabolic syndrome and triple-negative breast cancer. Maiti B, Kundranda MN, Spiro TP, et al. *Breast Cancer Res Treat.* 2010 Jun; 121(2):479-83.

Thorp KE. Energy Dynamics in Breast Cancer: Fields, Hormones & the Menstrual Cycle. G Med Sci. 2024; 5(1):169-249.
https://www.doi.org/10.46766/thegms.oncol.24111101

103. Metabolic syndrome: Does this influence breast cancer outcomes in the triple-negative population? Kennard K, Buckley ME, Sizer LM, et al. *Breast Cancer Res Treat.* 2021 Feb; 186(1):53-63.

104. Thermography in breast diagnosis. Sterns EE, Curtis AC, Miller S, et al. *Cancer.* 1982 Jul; 50(2):323-25.

105. Thermography as a screening and diagnostic tool: A systematic review. Fitzgerald A, Berentson-Shaw J. *NZ J Med.* 2012 Mar; 125(1351):80-91.

106. A comparative review of thermography as a breast cancer screening technique. Kennedy DA, Lee T, Seely D. *Integr Cancer Ther.* 2009 Mar; 8(1):9-16.

107. Breast thermography is a noninvasive prognostic procedure that predicts tumor growth rate in breast cancer patients. Head JF, Wang F, Elliott RL. *Ann NY Acad Sci.* 1993 Nov; 698:153-58.

108. Breast thermography and cancer risk prediction. Gautherie M, Gros CM. *Cancer.* 1980 Jan; 45(1):51-56.

109. A breast pre-cancer test? Preliminary results based on a breast temperature rhythm abnormality during the menstrual cycle. Simpson HW. *Breast Cancer Res Treat.* 1990 Jul; 16(1):51-55.

110. Loss of the progesterone-associated metabolic rhythm in cancer-associated breast tissue. Simpson HW, Pauson AW, McArdle C, et al. *Prog Clin Biol Res.* 1990;341A:47-53.

111. Circadian breast skin temperature rhythms: Overt and occult benign and occult primary malignant breast disease. Wilson DW, George D, Mansel RE, et al. *Chronobiol Int.* 1984; 1(2):167-72.

112. A non-invasive test for the pre-cancerous breast. Simpson HW, McArdle C, Pauson AW, et al. *Eur J Cancer.* 1995 Oct; 31A(11):1768-72.

113. The chronobra identifies prevailing mammary vascularity as a candidate variable in breast cancer post-operative outcome prediction. Simpson HW, George D, Sothern RB, et al. *Springerplus.* 2013 May; 2(1):241.

114. The luteal heat cycle of the breast in health. Simpson HW, Griffiths K, McArdle C, et al. *Breast Cancer Res Treat.* 1993 Sep; 27(3):239-45.

115. Angiogenesis: A marker for neoplastic transormation of mammary papillary hyperplasia. Brem SS, Gullino PM, Medina D. 1977 Mar; 195(4281):880-82.

116. Angiogenesis in breast cancer. Weidner N. *Cancer Treat Res.* 1996; 83:265-301.

117. Angiogenesis and breast cancer. Folkman J. *J Clin Oncol.* 1994 Mar; 12(3):441-43.

118. Angiogenesis induction in breast cancer: A paracrine paradigm. Badodekar N, Sharma A, Patil V, et al. *Cell Biochem Function.* 2021 Oct; 39(7):860-73.

119. *Galen's System of Physiology and Medicine.* Rudolph E. Siegel publ Karger (Basel). 1968, pp. 285-287.

120. Cancer Epigenetics: Past, present and future. Lee JE, Kim MY. *Semin Cancer Biol.* 2022 Aug; 83:4-14.

121. Metabolic interactions in cancer: Cellular metabolism at the interface between the microenvironment, the cancer cell phenotype and the epigenetic landscape. Rinaldi G, Rossi M, Fendt SM. *Wiley Interdiscip Rev Syst Biol Med.* 2018 Jan; 10(1).

122. Epigenetic control of cancer cell dormancy and awakening in endocrine therapy resistance. Llinas-Bertran A, Bellet-Ezquerra M, Seoane JA. *Cancer Discov.* 2024 May; 14(5):704-06.

123. The hallmarks of cancer. Hanrahan D, Weinberg RA. *Cell.* 2000 Jan; 100(1):57-70.

124. Hallmarks of cancer: The next generation. Hanrahan D, Weinberg RA. *Cell.* 2011 Mar; 144(5):646-74.

Thorp KE. Energy Dynamics in Breast Cancer: Fields, Hormones & the Menstrual Cycle. G Med Sci. 2024; 5(1):169-249.
https://www.doi.org/10.46766/thegms.oncol.24111101

125. Hallmarks of cancer: New dimensions. Hanrahan D. *Cancer Discov.* 2022 Jan; 12(1):31-46.

126. Cancer hallmarks intersect with neuroscience in the tumor microenvironment. Hanrahan D, Monie M. *Cancer Cell.* 2023 Mar; 41(3):573-80.

127. Hallmarks of cancer – the new testament. Senga SS, Grose RP. *Open Biol.* 2021 Jan; 11(1):200358.

128. The emerging hallmarks of cancer metabolism. Pavlova NN, Thompson CB. *Cell Metab.* 2016 Jan; 23(1):27-47.

129. The hallmarks of cancer metabolism: Still emerging. Pavlova NN, Zhu J, Thompson CB. *Cell Metab.* 2022 May; 34(3):355-77.

130. Tumor microenvironment. Ameth B. *Medicina (Kaunas).* 2019 Dec; 56(1):15.

131. Tumor microenvironment – Selective pressures boosting cancer progression. Nunes SC. *Adv Exp Med Biol.* 2020; 1219:35-49.

132. Role of the tumor microenvironment in breast cancer. Soysal SD, Tzankov A, Muenst SE. *Pathobiology.* 2015 Sep; 82(3-4):142-52.

133. Microenvironment in breast tumorigenesis: Friend or foe? 2019 Jan; 34(1):13-24.

134. Tumor-stroma crosstalk: Targeting stroma in breast cancer. Criscitiello C, Esposito A, Curigliano G. *Curr Opin Oncol.* 2014 Nov; 26(6):551-55.

135. Tumor microenvironment: Challenges and opportunities in targeting metastasis of triple negative breast cancer. Deepak KGK, Vempati R, Nagaraju GP, et al. *Pharmacol Res.* 2020 Mar; 153:104683.

136. Role of tumor microenvironment in triple-negative breast cancer and its prognostic significance. Yu T, Di G. *Chin J Cancer Res.* 2017 Jun; 29(3):237-52.

137. Endocrine disruptors and the tumor microenvironment: A new paradigm in breast cancer biology. Burks H, Pashos N, Martin E, et al. *Mol Cell Endocrinol.* 2017 Dec; 457:13-19.

138. Metabolic crosstalk in the breast cancer microenvironment. Dias AS, Almeida CR, Helguero LA, et al. *Eur J Cancer.* 2019 Nov; 121:154-71.

139. Metabolism shapes the tumor microenvironment. Reina-Campos M, Moscat J, Diaz-Meco M. *Curr Opin Cell Biol.* 2017 Oct; 48:47-53.

140. Mitogenic signal transduction in human breast cancer cells. Strobl JS, Wonderlin WF, Flynn DC. *Gen Pharmacol.* 1995 Dec; 26(8):1643-49.

141. The HER2 signaling network in breast cancer – Like a spider in its web. Dittrich A, Gautrey H, Browell D, et al. *J Mammary Gland Biol Neoplasia.* 2014 Dec; 19(3-4):253-70.

142. Estrogen receptor signaling as a target for novel breast cancer therapeutics. Renoir JM, Marsaud V, Lazennec G. *Biochem Pharmacol.* 2013 Feb; 85(4):449-65.

143. Mechanistic pathways of malignancy in breast cancer stem cells. Yousefnia S, Seyed Forootan F, Seyed Forootan S, et al. *Front Oncol.* 2020 Apr; 10:4552.

144. Molecular pathways: MERTK signaling in cancer. Cummings CT, Deryckere D, Earp HS, et al. *Clin Cancer Res.* 2013 Oct; 19(19):5275-80.

145. Signaling pathways in cancer-associated fibroblasts: Recent advances and future perspectives. Cummings CT, Deryckere D, Earp HS, et al. *Cancer Commun (London).* 2023 Jan; 43(1):3-41.

Thorp KE. Energy Dynamics in Breast Cancer: Fields, Hormones & the Menstrual Cycle. G Med Sci. 2024; 5(1):169-249.
https://www.doi.org/10.46766/thegms.oncol.24111101

146. MAP kinase signaling pathways in cancer. Dhillon AS, Hagan S, Rath O. *Oncogene.* 2007 May; 26(22):3279-90.

147. Morphological evaluation of cell turnover in relation to the menstrual cycle in the "resting" human breast. Ferguson DJ, Anderson TJ. *Br J Cancer.* 1981 Aug; 44(2):177-81.

148. Apoptosis in the human endometrium during the menstrual cycle. Kokawa K, Shikone T, Nakano R, et al. *J Clin Endocrinol Metab.* 1996 Nov; 81(11):4144-47.

149. Apoptosis of human ovary and uterine endometrium during the menstrual cycle. Shikone T, Kokawa K, Yamoto M, et al. *Horm Res.* 1997; 48 (Suppl 3):27-34.

150. Relevance of apoptosis in the female reproductive system. Meresman G. *Invest Clin.* 2011 Sep; 52(3):274-90.

151. Apoptosis as a basic mechanism in the ovarian cycle: Follicular atresia and luteal regression. Palumbo A, Yeh J. *J Soc Gynecol Invest.* 1995 May; 2(3):565-73.

152. A matter of new life and cell death: programmed cell death in the mammalian ovary. Chesnokov MS, Mamedova AR, Zhivotovsky B, et al. *J Biomed Sci.* 2024 Mar; 31(1):31.

153. Hormonal regulation of apoptosis in breast cells and tissues. Gompel A, Somaï S, Chaouat M, et al. *Steroids.* 2000 Oct; 65(10-11):593-98.

154. The neuroendocrine regulation of the human ovarian cycle. Buffet NC, Bouchard P. *Chronobiol Int.* 2001 Nov; 18(6):893-919.

155. Proapoptotic effects of antiestrogens, progestins, and androgen in breast cancer cells. Kandouz M, Lombet A, Perrot JY, et al. *J Steroid Biochem Mol Biol.* 1999 Apr; 69(1-6):463-71.

156. Antiestrogens are pro-apoptotic in normal human breast epithelial cells. Somaï S, Chaouat M, Jacob D, et al. *Int J Cancer.* 2003 Jul; 105(5):607-12.

157. Apoptosis in normal and neoplastic mammary gland development. Strange R, Metcalfe T, Thackray L. *Microsci Res Tech.* 2001 Jan; 52(2):171-81.

158. Studies of apoptosis in breast cancer. Parton M, Dowsett M, Smith I. *BMJ.* 2001 Jun; 322(7301):1528-32.

159. Apoptosis in breast cancer and its relationship to clinicopathological characteristics and prognosis. Nishimura R, Nagao K, Miyayama H, et al. *J Surg Gynecol.* 1999 Aug; 71(4):226-34.

160. Role of p53 in breast cancer: Focus on mutation spectrum and therapeutic strategies. Kaur RP, Vasudeva K, Kumar R, et al. *Curr Pharm Des.* 2018; 24(30):3566-75.

161. How does p53 induce apoptosis and how does this relate to p53-mediated tumour suppression? Aubrey BJ, Kelly GL, Janic A, et al. *Cell Death Differ.* 2018 Jan; 25(1):104-113.

162. Tumour suppression by p53: The importance of apoptosis and cellular senescence. Zuckerman V, Wolyniec K, Sionov RV, et al. *J Pathol.* 2009 Sep; 219(1):3-15.

163. The role of p53 in apoptosis. Amaral JD, Xavier JM, Steer CJ, et al. *Discov Med.* 2010 Feb; 9(45):145-52.

164. Triple-negative breast tumors are dependent on mutant p53 for growth and survival. Dibra D, Moyer SM, El-Naggar AK, et al. *Proc Natl Acad Sci USA.* 2023 Aug; 120(34):e2308807120.

165. P53, apoptosis, and breast cancer. *J Mammary Gland Biol Neoplasia.* 1996 Apr; 1(2):163-75.

Thorp KE. Energy Dynamics in Breast Cancer: Fields, Hormones & the Menstrual Cycle. G Med Sci. 2024; 5(1):169-249.
https://www.doi.org/10.46766/thegms.oncol.24111101

166. p53 and breast cancer, an update. Lacroix M, Toillon RA, Leclercq G. *Endocrine Relat Cancer.* 2006 Jun; 13(2):293-325.

167. Telomerase: Structure, functions, and activity regulation. Zvereva MI, Shcherbakova DM, Dontsova OA. *Biochemistry (Mosc).* 2010 Dec; 75(12):1563-83.

168. Role of telomerase in normal and cancer cells. Meyerson M. *J Clin Oncol.* 2000 Jul; 18(13):2626-34.

169. Telomerase at the intersection of cancer and aging. Bernardes de Jesus B, Blasco MA. *Trends Genet.* 2013 Sep; 29(5):513-20.

170. Telomere biology: Cancer firewall or aging clock? Mitteldorf JJ. *Biochemistry (Mosc).* 2013 Sep; 78(9):1054-60.

171. Telomeres, aging, and cancer: The big picture. Lansdorp PM. *Blood.* 2022 Feb; 139(6): 813-21.

172. Telomerase and cancer. Testorelli C. *Exp Clin Cancer Res.* 2003 Jun; 22(2):165-69.

173. Telomerase and breast cancer. Herbert BS, Wright WE, Shay JW. *Breast Cancer Res.* 2001; 3(3):146-49.

174. Telomerase in breast cancer: A critical evaluation. Baykal A, Rosen D, Zhou C, et al. *Adv Anat Pathol.* 2004 Sep; 11(5):262-68.

175. The association of the telomerase reverse transcriptase rs10069690 polymorphisms with the risk, age at onset and prognosis of triple negative breast cancer. Zins K, Peka E, Miedl H, et al. *Int J Mol Sci.* 2023 Jan; 24(3):1825.

176. Regulation and clinical potential of telomerase transcriptase (TERT/hTERT) in breast cancer. Yang R, Han Y, Guan X, et al. *Cell Commun Signal.* 2023 Aug; 21(1):218.

177. Accuracy of telomerase in estimating breast cancer risk: A systematic review and meta-analysis. Winnikow EP, Medeiros LR, Edelweiss MI, et al. *Breast.* 2012 Feb; 21(1):1-7.

178. Telomerase activity and telomere length in women with breast cancer or without malignancy: A systematic review and meta-analysis. Benites-Zapata VA, Ulloque-Badaracco JR, Alarcón-Braga EA, et al. *Maturitas.* 2024 Feb; 180:107882.

179. Stem cells in normal mammary gland and breast cancer. Luo J, Yin X, Ma T, et al. *Am J Med Sci.* 2010 Apr; 339(4):366-70.

180. Breast cancer stem cells: Current advances and clinical implications. Luo M, Clouthier SG, Deol Y, et al. *Methods Mol Biol.* 2015; 1293:1-49.

181. Breast cancer stem cell: The roles and therapeutic implications. Yang F, Xu J, Tang L, et al. *Cell Mol Life Sci.* 2017 Mar; 74(6):951-66.

182. Breast cancer and stem cells: A mini-review. Filipova A, Seifrtova M, Mokry J, et al. *Tumori.* 2014 Jul; 100(4):363-69.

183. Breast cancers, mammary stem cells, and cancer stem cells, characteristics, and hypotheses. Taurin S, Alkhalifa H. *Neoplasia.* 2020 Dec; 22(12):663-78.

184. Tumour stem cells in breast cancer. Ibragimova M, Tsyganov M, Litviakov N. *Int J Mol Sci.* 2022 May; 23(9):5058.

185. Cancer stem cells in TNBC. Hua Z, White J, Zhou J. *Semin Cancer Biol.* 2022 Jul; 82:26-34.

Thorp KE. Energy Dynamics in Breast Cancer: Fields, Hormones & the Menstrual Cycle. G Med Sci. 2024; 5(1):169-249.
https://www.doi.org/10.46766/thegms.oncol.24111101

186. Breast cancer stem cells as drivers of tumor chemoresistance, dormancy and relapse: New challenges and therapeutic opportunities. De Angelis ML, Francescangeli F, Zeuner A. *Cancers (Basel).* 2019 Oct; 11(10):1569.

187. Cancer stem cell in breast cancer therapeutic resistance. Bai X, Ni J, Beretov J, et al. *Cancer Treat Rev.* 2018 Sep; 69:152-63.

188. Breast cancer stem cells, heterogeneity, targeting therapies and therapeutic implications. Zeng X, Liu C, Yao J, et al. *Pharmacol Res.* 2021 Jan; 163:105320.

189. Single-cell analysis reveals a stem-cell program in human metastatic breast cancer cells. Lawson DA, Bhakta NR, Kessenbrock K, et al. *Nature.* 2015 Oct; 526(7571:131-35.

190. Dormancy in breast cancer. Dalla E, Sreekumar A, Aguirre-Ghiso JA, et al. *Cold Harb Perspect Med.* 2023 Nov; 13(11):a041331.

191. Cellular dormancy in minimal residual disease following targeted therapy. Ruth JR, Pant DK, Pan TC, et al. *Breast Cancer Res.* 2021 Jun; 23(1):63.

192. Metastasis dormancy in estrogen receptor-positive breast cancer. Zhang XH, Giuliano M, Trivedi MV, et al. *Clin Cancer Res.* 2013 Dec; 19(23):6389-97.

193. How can we integrated the biology of breast cancer cell dormancy into clinical practice? Elkholi IE, Rose AAN, Aguirre-Ghiso JA, et al. *Cancer Cell.* 2024 Jul; 42(7):1147-51.

194. Epigenetic dynamics in cancer stem cell dormancy. Ferrer AI, Trinidad JR, Sandiford O, et al. *Cancer Metastasis Rev.* 2020 Sep; 39(3):721-38.

195. The epigenetic progenitor origin of human cancer. Feinberg AP, Ohlsson R, Henikoff S. *Nat Rev Genet.* 2006 Jan; 7(1):21-33.

196. Epigenetic determinants of cancer. Baylin SB, Jones PA. *Cold Spring Harb Perspect Biol.* 2016 Sep; 8(9):a019505.

197. Molecular and cellular changes during cancer progression resulting from genetic and epigenetic alterations. Pruitt K. *Prog Mol Biol Transl Sci.* 2016; 144:3-48.

198. Epigenetic changes in cancer. Grønbaek K, Hother C, Jones PA. *APMIS.* 2007 Oct; 115(10):1039-59.

199. The fundamental role of epigenetic events in cancer. Jones PA, Baylin SB. *Nature Rev Genet.* 2002 Jun; 3(6):415-28.

200. Epigenetic gene silencing in cancer – a mechanism for early oncogenic pathway addiction? Baylin SB, Ohm JE. *Nat Rev Cancer.* 2006 Feb; 6(2):107-16.

201. Epigenetics: Unraveling the cancer code. Brower V. *Nature.* 2011 Mar; 471(7339):S12-13.

202. Epigenetic plasticity and the hallmarks of cancer. Flavahan WA, Gaskell E, Bernstein BE. *Science.* 2017 Jul; 357(6348):eaal2380.

203. Cancer epigenetics: Past, present and future. Lee JE, Kim MY. *Semin Cancer Biol.* 2022 Aug; 83:4-14.

204. Epigenetic regulation in breast cancer. Nam HJ, Baek SH. *Adv Exp Med Biol.* 2021; 1187:103-19.

205. The epigenetics of breast cancer. Jovanovic J, Rønneberg JA, Tost J. *Med Oncol.* 2010 Jun; 4(3):242-54.

206. Epigenetics of breast cancer: Clinical status of epi-drugs and phytochemicals. Shukla S, Penta D, Mondal P, et al. *Adv Exp Med Biol.* 2019; 1152:293-310.

Thorp KE. Energy Dynamics in Breast Cancer: Fields, Hormones & the Menstrual Cycle. G Med Sci. 2024; 5(1):169-249.
https://www.doi.org/10.46766/thegms.oncol.24111101

207. Epigenetic alterations in triple-negative breast cancer – The critical role of extracellular matrix. Zolota V, Tzelepi V, Piperigkou Z, et al. *Cancers (Basel).* 2021 Feb; 13(4):713.

208. Epigenetic inactivation of DNA repair in breast cancer. Nowsheen S, Aziz K, Tran PT, et al. *Cancer Lett.* 2014 Jan; 342(2):213-22.

209. DNA methylation and cancer. Momparler RL, Bovenzi V. *J Cell Physiol.* 2000 May; 183(2):145-54.

210. DNA methylation in cancer: A gene silencing mechanism and the clinical potential of its biomarkers. Fukushige S, Horii A. *Tohoku J Exp Med.* 2013 Mar; 229(3):173-85.

211. DNA methylation and cancer. Kulis M, Esteller M. *Adv Genet.* 2010; 70:27-56.

212. Navigating the DNA methylation landscape of cancer. Nishiyama A, Nakanishi M. *Trends Genet.* 2021 Nov; 37(11):1012-27.

213. The DNA methylation landscape in cancer. Skvortsova K, Stirzaker C, Taberlay P. *Essays Biochem.* 2019 Dec; 63(6):797-811.

214. Aberrant DNA methylation of ABC transporters in cancer. Zappe K, Cichna-Markl M. *Cells.* 2020 Oct; 9(10):2281.

215. DNA methylation profiles in cancer diagnosis and therapeutics. Pan Y, Liu G, Zhou F, et al. *Clin Exp Med.* 2018 Feb; 18(1):1-14.

216. The role of epigenetic transcription repression and DNA methyltransferases in cancer. Daniel FI, Cherubini K, Yurgel LS, et al. *Cancer.* 2011 Feb; 117(4):677-87.

217. A decade of exploring the cancer epigenome – biological and translational implications. Baylin SB, Jones PA. *Nature Rev Cancer.* 2011 Sep; 11(10):726-34.

218. The language of chromatin modification in human cancers. Zhao S, Allis CD, Wang GG. *Nat Rev Cancer.* 2021 Jul; 21(7):413-30.

219. DNA methylation and breast cancer-associated variants. Ho PJ, Dorajoo R, Ivanković I, et al. *Breast Cancer Res Treat.* 2021 Aug; 188(3):713-27.

220. The mechanism of DNA methylation and mRNA in breast cancer. Ma L, Li C, Yin H, et al. *Int J Mol Sci.* 2023 May; 24(11):9360.

221. DNA methylation contributes to deregulation of 12 cancer-associated microRNAs and breast cancer progression. Pronina IV, Loginov VI, Burdennyy AM, et al. *Gene.* 2017 Mar; 604:1-8.

222. Genome-wide analysis of DNA methylation and expression of microRNAs in breast cancer cells. Morita S, Takahashi RU, Yamashita R, et al. *Int J Mol Sci.* 2012; 13(7):8259-72.

223. DNA methylation as a biomarker in breast cancer. Martens JW, Margossian AL, Schmitt M, et al. *Future Oncol.* 2009 Oct; 5(8):1245-56.

224. DNA methylation markers predict outcome in node-positive, estrogen receptor-positive breast cancer with adjuvant anthracycline-based chemotherapy. Hartmann O, Spyratos F, Harbeck N, et al. *Clin Cancer Res.* 2009 Jan; 15(1):315-23.

225. DNA methylation markers for breast cancer detection in the developing world. Downs BM, Mercado-Rodriguez C, Cimino-Mathews A, et al. *Clin Cancer Res.* 2019 Nov; 25(21):6357-67.

226. DNA methylation profile of triple-negative breast cancer-specific genes comparing lymph node positive patients to lymph node negative patients. Mathe A, Wong-Brown M, Locke WJ, et al. *Sci Rep.* 2016 Sep; 6:33435.

Thorp KE. Energy Dynamics in Breast Cancer: Fields, Hormones & the Menstrual Cycle. G Med Sci. 2024; 5(1):169-249.
https://www.doi.org/10.46766/thegms.oncol.24111101

227. Identification of DNA methylation biomarkers with potential to predict response to neoadjuvant chemotherapy in triple-negative breast cancer. Meyer B, Clifton S, Locke W, et al. *Clin Epigenetics.* 2021 Dec; 13(1):226.

228. A DNA methylation-based gene signature can predict triple-negative breast cancer diagnosis. Mendaza S, Guerrero-Setas D, Monreal-Santesteban I, et al. *Biomedicines.* 2021 Oct; 9(10):1394.

229. The altered transcriptome and DNA methylation profiles of docetaxel-resistant resistance in breast cancer PDX models. Gómez-Miragaya J, Morán S, Calleja-Cervantes ME, et al. *Mol Cancer Res.* 2019 Oct; 17(10):2063-76.

230. Aberrant methylation and silencing of ARHI, an imprinted tumor suppressor gene in which the function is lost in breast cancers. Yuan J, Luo RZ, Fujii S, et al. *Cancer Res.* 2003 Jul; 63(14):4174-80.

231. Cancer cell metabolism connects epigenetic modifications to transcriptional regulation. Morrison AJ. *FEBS J.* 2022 Mar; 289(5):1302-14.

232. Connections between metabolism and epigenetics in cancers. Thakur C, Chen F. *Semin Cancer Biol.* 2019 Aug; 57:52-58.

233. Metabolic recoding of epigenetics in cancer. Wang YP, Lei QY. *Cancer Commun (Lond).* 2018 May: 38(1):25.

234. Metabolism and epigenetics: A link cancer cells exploit. Carrer A, Wellen KE. *Curr Opin Biotechnol.* 2015 Aug; 34:23-29.

235. Epigenetics and cancer metabolism. Johnson C, Warmoes MO, Shen X, et al. *Cancer Lett.* 2015 Jan; 356(2 Pt A):309-14.

236. Research advances on epigenetics and cancer metabolism. Han H, Feng F, Li H. *Zhejiang Da Xue Xue Bao Yi Xue Ban.* 2021 Feb; 50(1):1-16.

237. Metabolic signaling to the nucleus in cancer. Campbell SL, Wellen KE. *Mol Cell.* 2018 Aug; 71(3):398-408.

238. Metabolic signaling into chromatin modifications in the regulation of gene expression. Gao T, Díaz-Hirashi Z, Verdeguer F. *Int J Mol Sci.* 2018 Dec; 19(12):4108.

239. Molecular pathways: Metabolic control of histone methylation and gene expression in cancer. Tran TQ, Lowman XH, Kong M. *Clin Cancer Res.* 2017 Aug; 23(15):4004-09.

240. Metabolic reprogramming and epigenetic modifications in cancer: From the impacts and mechanisms to the treatment potential. Xu X, Peng Q, Jiang X, et al. *Exp Mol Med.* 2023 Jul; 55(7): 1357-70.

241. New challenge: Mitochondrial epigenetics? Stimpfel M, Jancar N, Virant-Klun I. *Stem Cell Rev Rep.* 2018 Feb; 14(1):13-26.

242. Mitochondrial DNA: Epigenetics and environment. Sharma N, Pasala MS, Prakash A. *Environ Mol Mutagen.* 2019 Oct; 60(8):668-82.

243. The mitochondrial side of epigenetics. Castegna A, Iacobazzi V, Infantino V. *Physiol Genomics.* 2015 Aug; 47(8):299-307.

244. Mitochonfrial DNA methylation and human diseases. Stoccoro A, Coppedè F. *Int J Mol Sci.* 2021 Apr; 22(9):4594.

Thorp KE. Energy Dynamics in Breast Cancer: Fields, Hormones & the Menstrual Cycle. G Med Sci. 2024; 5(1):169-249.
https://www.doi.org/10.46766/thegms.oncol.24111101

245. From powerhouse to regulator: The role of mitoepigenetics in mitochondrion-related cellular functions and human diseases. Donato L, Mordà D, Scimone C, et al. *Free Radic Biol Med.* 2024 Jun; 218:105-19.

246. Regulation of mitochondrial gene expression, the epigenetic enigma. Mposhi A, Van der Wijst MG, Faber KN, et al. *Front Biosci (Landmark Ed.).* 2017 Mar; 22(7):1099-1113.

247. Progress in mitochondrial epigenetics. Manev H, Dzitoyeva S. *Biomol Concepts.* 2013 Aug; 4(4):381-89.

248. Mitochondrial epigenetics: An overlooked layer of regulation? van der Wijst MG, Rots MG. *Trends Genet.* 2015 Jul; 31(7):353-56.

249. Mitochondria and epigenetics: Crosstalk in homeostasis and stress. Matilainen O, Quirós PM, Auwerx J. *Trends Cell Biol.* 2017 Jun; 27(6):453-63.

250. An epigenetic role of mitochondria in cancer. Liu Y, Chen C, Wang X, et al. *Cells.* 2022 Aug; 11(16):2518.

251. Mitochondrial DNA: Epigenetics and environment. Sharma N, Pasala MS, Prakash A. *Environ Mol Mutagen.* 2019 Oct; 60(8):668-82.

252. Metabolic interactions in the tumor microenvironment. Lyssiotis CA, Kimmelman AC. *Trends Cell Biol.* 2017 Nov; 27(11):863-75.

253. Metabolism shapes the tumor microenvironment. Reina-Campos M, Moscat J, Diaz-Meco M. *Curr Opin Cell Biol.* 2017 Oct; 48:47-53.

254. Metabolic alterations in the tumor microenvironment and their role in oncogenesis. Eisenberg L, Eisenberg-Bord M, Eisenberg-Lerner A, et al. *Cancer Lett.* 2020 Aug; 484:65-71.

255. Hypoxia in the tumor microenvironment. Li Y, Zhao L, Li XF. *Technol Cancer Res Treat.* 2021 Jan; 20:15330338211036304.

256. Epigenetic regulation in the tumor microenvironment: Molecular mechanisms and therapeutic targets. Yang J, Xu J, Wang W, et al. *Signal Transduct Target Ther.* 2023 May; 8(1):210.

257. Beggars banquet: Metabolism in the tumor immune microenvironment and cancer therapy. Zou W, Green DR. *Cell Metab.* 2023 Jul; 35(7):1101-13.

258. Dietary fat and lipid metabolism in the tumor microenvironment. Goswami S, Zhang Q, Celik CE, et al. *Biochim Biophys Acta Rev Cancer.* 2023 Nov; 1878(6):188984.

259. Metabolism within the tumor microenvironment and its implication on cancer progression: An ongoing therapeutic target. Ocaña MC, Martínez-Poveda B, Quesada AR, et al. *Med Res Rev.* 2019 Jan; 39(1):70-113.

260. Metabolic codependencies in the tumor microenvironment. Dey P, Kimmelman AC, DePinho RA. *Cancer Discov.* 2021 May; 11(5):1067-81.

261. Negative intraventricular diastolic pressure in patients with mitral stenosis: Evidence of left ventricular diastolic suction. Sabbah HN, Anbe DT, Stein PD. *Am J Cardiol.* 1980; 45(3):pp 562-66.

262. Left ventricular diastolic suction as a mechanism of ventricular filling. Hori M, Yellin EL, Sonnenblick EH. *Jpn Circ J.* 1982 Jan; 46(1):pp 124-129.

263. The heart as a suction pump. Robinson TF, Factor SM, Sonnenblick EH. *Scientific American.* 1986; 254(6):pp 84-91.

Thorp KE. Energy Dynamics in Breast Cancer: Fields, Hormones & the Menstrual Cycle. G Med Sci. 2024; 5(1):169-249.
https://www.doi.org/10.46766/thegms.oncol.24111101

264. The heart is not a pump: A refutation of the pressure propulsion premise of heart function. Marinelli R, Fürst B, van der Zee H, McGinn H, Marinelli W. *Frontier Perspectives.* 5(1):pp 15-24; Fall-Winter 1995.

265. Spiral laminar flow in arteries? Stonebridge PA, Brophy CM. *Lancet.* 1991; 338(8779):pp 1360-61.

266. Spiral laminar flow in vivo. Stonebridge PA, Hoskins PR, Allan PL, Belck JF. *Clin Sci (Lond).* 1996; 91(1):pp 17-21.

267. Helical and retrograde secondary flow patterns in the aortic arch studied by three-directional magnetic resonance velocity mapping. Kilner PJ, Yang GZ, Mohiaddin RH, Firmin DN, Longmore DB. *Circulation.* 1993; 88(5): pp 2235-47.

268. Aether, fields & energy dynamics in living bodies - Part II. Thorp KE, Thorp JA, Walker PR. *G Med Sci.* 2021; 2(6):001-020.

269. Energy dynamics in chronic heart failure, chronic kidney disease & the cardiorenal syndrome: A new causal paradigm. Thorp KE, Thorp JA, Northrup C, et al. *G Med Sci.* 2023; 4(1):290-347.

270. Energy dynamics in dementia & the neurodegenerative diseases: A new causal paradigm. Thorp KE, Thorp JA, Northrup C, et al. *G Med Sci.* 2023; 4(1):348-405.

271. Energy dynamics in the autoimmune disorders: The unity of immunity. Thorp KE, Thorp JA, Northrup C, et al. *G Med Sci.* 2023; 4(1):406-470.

272. Energy dynamics in seizure disorder: Reexamining the sacred disease. Thorp KE, Thorp JA, Northrup C, et al. *G Med Sci.* 2024; 5(1):1-49.

273. Energy dynamics in the metabolic syndrome: Underpinnings of an evolving global catastrophe. Thorp KE, Thorp EM. *G Med Sci.* 2024; 5(1):61-146.

274. Patterns and emerging mechanisms of the angiogenic switch during tumorigenesis. Hanrahan D, Folkman J. *Cell.* 1996 Aug; 86(3):353-64.

275. Tumor angiogenesis: Causes, consequences, challenges and opportunities. Lugano R, Ramachandran M, Dimberg A. *Cell Mol Life Sci.* 2020 May; 77(9):1745-70.

276. The angiogenic switch in solid tumors: Clinical implications. Tosetti F, Benelli R, Albini A. *Suppl Tumori.* 2002 Nov; 1(6):S9-11.

277. Angiogenesis in pre-malignant conditions. Raica M, Cimpean AM, Ribatti D. *Eur J Cancer.* 2009 Jul; 45(11):1924-34.

278. Angiogenesis in pre-malignant conditions. Menakuru SR, Brown NJ, Staton CA, et al. *Br J Cancer.* 2008 Dec; 99(12):1961-66.

279. Tumorigenesis and the angiogenic switch. Bergers G, Benjamin LE. *Nat Rev Cancer.* 2003 Jun; 3(6):401-10.

280. Angiogenesis and oncogenesis. Gullino PM. *J Nat Cancer Inst.* 1978 Sep; 61(3):639-43.

281. Angiogenesis induction in breast cancer: A paracrine paradigm. Badodekar N, Sharma A, Patil V, et al. *Cell Biochem Func.* 2021 Oct; 39(7):860-73.

282. Angiogenesis: A marker for neoplastic transformation of mammary papillary hyperplasia. Brem SS, Gullino PM, Medina D. *Science.* 1977 Mar; 195(4281):880-82.

283. HIFs, angiogenesis, and metabolism: Elusive enemies in breast cancer. de Heer EC, Jalving M, Harris AL. *J Clin Invest.* 2020 Oct; 130(10):5074-87.

284. Hallmarks of Cancer: The next generation. Hanrahan D, Weinberg RA. *Cell.* 2011 Mar; 144(5):646-74.

Thorp KE. Energy Dynamics in Breast Cancer: Fields, Hormones & the Menstrual Cycle. G Med Sci. 2024; 5(1):169-249.
https://www.doi.org/10.46766/thegms.oncol.24111101

285. Neural regulation of cancer: Cancer-induced remodeling of the central nervous system. Krishna S, Hervey-Jumper SL. *Adv Biol (Weinh).* 2022 Sep; 6(9):e2200047.

286. Insights and opportunities at the crossroads of cancer and neuroscience. Pan C, Winkler F. *Nat Cell Biol.* 2022 Oct; 24(10):1454-60.

287. Roadmap for the emerging field of cancer neuroscience. Monje M, Borniger JC, D'Silva NJ, et al. *Cell.* 2020 Apr; 181(2):219-22.

288. Cancer neuroscience: State of the field, emerging directions. Winkler F, Venkatesh HS, Amit M, et al. *Cell.* 2023 Apr; 186(8):1689-1707.

289. Cancer-associated neurogenesis and nerve-cancer cross-talk. Silverman DA, Martinez VK, Dougherty PM, et al. *Cancer Res.* 2021 Mar; 81(6):1431-40.

290. Tumor neurobiology and the war of nerves in cancer. Faulkner S, Jobling P, March B, et al. *Cancer Discov.* 2019 Jun; 9(6):702-10.

291. Nerves in cancer. Zahalka AH, Frenette PS. *Nat Rev Cancer.* 2020 Mar; 20(3):143-57.

292. Neural signaling in cancer. Keough MB, Monie M. *Annu Rev Neurosci.* 2022 Jul; 45:199-221.

293. Pathology of perineural spread. Brown IS. *Neurol Surg B Skull Base.* 2016 Apr; 77(2):124-30.

294. Do you have the nerves to regenerate? The importance of neural signaling in the regeneration process. Pirotte N, Leynen N, Artois T, et al. *Dev Biol.* 2016 Jan; 409(1):4-15.

295. Nerve dependence in tissue, organ, and appendage regeneration. Kumar A, Brockes JP. *Trends Neurosci.* 2012 Nov; 35(11):691-99.

296. The role of peripheral nerves in urodele limb regeneration. Stocum DL. *Eur J Neurosci.* 2011 Sep; 34(6):908-16.

297. The concept of trophic units in the central nervous system. Agnati LF, Cortelli P, Pettersson R, et al. *Prog Neurobiol.* 1995 Aug; 46(6):561-74.

298. Perineural invasion predicts unfavorable prognosis in patients with invasive breast cancer. Hosoya K, Wakahara M, Ikeda K, et al. *Cancer Diagn Progn.* 2023 Mar; 3(2):208-14.

299. Perineural invasion as a risk factor for locoregional recurrence of invasive breast cancer. Narayan P, Flynn J, Zhang Z, et al. *Sci Rep.* 2021 Jun; 11(1):12781.

300. Prevalence and significance of perineural invasion in invasive breast cancers. Karak SG, Quatrano N, Buckley J, et al. *Conn Med.* 2010 Jan; 74(1):17-21.

301. Diurnal, semidiurnal, and fortnightly tidal components in orthotidal proglacial rivers. Briciu AE. *Environ Monit Assess.* 2018 Feb; 190(3):160.

302. Orthotidal signal in the electrical conductivity of an inland river. Briciu AE, Mihăilă D, Oprea DI, et al. *Environ Monit Assess.* 2018 Feb; 190(5):282.

303. Earth-tide-induced fluctuations in the salinity of an inland river, New South Wales, Australia: A short-term study. Jasonsmith JF, Macdonald BC, White I. *Environ Monit Assess.* 2017 Apr; 189(4):188.

304. Tree stem diameters fluctuate with tide. Zürcher E, Cantiani MG, Sorbetti-Guerri, et al. *Nature.* 1998; 392:665-66.

305. Tree-stem diameter fluctuates with the lunar tides and perhaps with geomagnetic activity. Barlow PW, Mikulecky M, Strestik, et al. *Protoplasma.* 2010 Nov; 247(1-2):25-43.

Thorp KE. Energy Dynamics in Breast Cancer: Fields, Hormones & the Menstrual Cycle. G Med Sci. 2024; 5(1):169-249.
https://www.doi.org/10.46766/thegms.oncol.24111101

306. Are there tides within trees? Fisahn J. *Ann Bot.* 2018 Nov; 122(5):735-39.

307. Are cyclic plant and animal behaviours driven by gravimetric mechanical forces? de Mello Gallep C, Robert D. *Exp Botan.* 2022 Feb; 73(4):1093-1103.

308. Leaf movements and their relation with the lunisolar gravitational force. Barlow PW. *Ann Bot.* 2015 Aug; 116(2):149-87.

309. The effect of lunisolar tidal acceleration on stem elongation growth, nutations and leaf movements in peppermint (Mentha x piperita L). Zajączkowska U, Barlow PW. *Plant Biol (Stuttg).* 2017 Jul; 19(4):630-42.

310. Arabidopsis thaliana root growth kinetics and lunisolar tidal acceleration. Fisahn J, Yazdanbakhsh N, Klingele E, et al. *New Phytol.* 2012 Jul; 195(2):346-55.

311. Lunisolar tidal force and the growth of plant roots, and some other of its effects on plant movements. Barlow PW, Fisahn J. *Ann Bot.* 2012 Jul; 110(2):301-18.

312. A proposal to explain how the circatidal rhythm of the Arabidopsis thaliana root elongation root rate could be mediated by the lunisolar gravitational force: A quantum physical approach. Fisahn J, Barlow P, Dorda G. *Ann Bot.* 2018 Nov; 122(5):725-33.

313. Arabidopsis thaliana root elongation growth is sensitive to lunisolar tidal acceleration and may also be weakly correlated with geomagnetic variations. Barlow PW, Fisahn J, Yazdanbakhsh N. *Ann Bot.* 2013 May; 111(5):859-72.

314. *World Soul: A History.* James Wilberding (ed.). Oxford University Press, 2021.

315. *The Great Chain of Being.* Arthur O. Lovejoy. Harvard University Press, 1936.

316. Cancer metabolism: new insights into classic characteristics. Kato Y, Maeda T, Suzuki A, et al. *Jpn Dent Sci Rev.* 2018 Feb; 54(1):8-21.

317. Metabolic interactions in cancer: cellular metabolism at the interface between the microenvironment, the cancer cell phenotype and the epigenetic landscape. Rinaldi G, Rossi M, Fendt SM. *Wiley Interdiscip Rev Syst Biol Med.* 2018 Jan; 10(1).

318. Metabolism in cancer metastasis: Bioenergetics, biosynthesis, and beyond. Teoh ST, Lunt SY. *Wiley Interdiscip Rev Syst Biol Med.* 2018 Jan; 10(2).

319. Metabolism shapes the tumor microenvironment. Reina-Campos M, Moscat J, Diaz-Meco M. *Curr Opin Cell Biol.* 2017 Oct; 48:47-53.

320. Metabolic alterations in the tumor microenvironment and their role in oncogenesis. Eisenberg L, Eisenberg-Bord M, Eisenberg-Lerner A, et al. *Cancer Lett.* 2020 Aug; 484:65-71.

321. Metabolic alterations in the tumor microenvironment. Lyssiotis CA, Kimmelman AC. *Trends Cell Biol.* 2017 Nov; 27(11):863-75.

322. Metabolic reprogramming and epigenetic modifications on the path to cancer. Sun L, Zhang H, Gao P. *Protein Cell.* 2022 Dec; 13(12):877-919.

323. The biology of cancer: Metabolic reprogramming fuels cell growth and proliferation. DeBerardinis RJ, Lum JJ, Hatzivassiliou G, et al. *Cell Metab.* 2008 Jan; 7(1):11-20.

324. Metabolism and its sequelae in cancer evolution and therapy. Gillies RJ, Gatenby RA. 2015 Mar;21(2):88-96.

325. Connections between metabolism and epigenetics in cancers. Thakur C, Chen F. *Semin Cancer Biol.* 2019 Aug; 57:52-58.

Thorp KE. Energy Dynamics in Breast Cancer: Fields, Hormones & the Menstrual Cycle. G Med Sci. 2024; 5(1):169-249.
https://www.doi.org/10.46766/thegms.oncol.24111101

326. Metabolic recoding of epigenetics in cancer. Wang YP, Lei QY. *Cancer Commun (Lond).* 2018 May; 38(1):25.

327. Metabolism and epigenetics: A link cancer cells exploit. Carrer A, Wellen KE. *Curr Opin Biotechnol.* 2015 Aug; 34:23-29.

328. Cancer cell metabolism connects epigenetic modifications to transcriptional regulation. Morrison AJ. *FEBS J.* 2022 Mar; 289(5):1302-14.

329. An epigenetic role of mitochondria in cancer. Liu Y, Chen C, Wang X, et al. *Cells.* 2022 Aug; 11(16):2518.

330. Epigenetics and cancer metabolism. Johnson C, Warmoes MO, Shen X, et al. *Cancer Lett.* 2015 Jan; 356(2 Pt A):309-14.

331. The metabolism of tumours in the body. Warburg O, Wind F, Negelein E. *J Gen Physiol.* 1927 Mar; 8(6):519-30.

332. On the origin of cancer cells. Warburg O. *Science.* 1956 Feb; 123(3191):309-14.

333. The metabolic switch and its regulation in cancer cells. Zhou S, Huang C, Wei Y. *Sci China Life Sci.* 2010 Aug; 53(8):942-58.

334. Cancer cell metabolism: Warburg and beyond. Hsu PP, Sabatini DM. 2008 Sep; 134(5):703-07.

335. The Warburg effect: Essential part of metabolic reprogramming and central contributor to cancer progression. Vaupel P, Schmidberger H, Mayer A. *Int J Radiation Biol.* 2019 Jul; 95(7):912-19.

336. The acidic tumor microenvironment as a driver of cancer. Boedtkjer E, Pedersen SF. *Annu Rev Physiol.* 2020 Feb; 82:103-26.

337. Extracellular acidity, a 'reappreciated' trait of tumor environment driving malignancy: Perspectives in diagnosis and therapy. Peppicelli S, Bianchini F, Calorini L. *Canc Metastasis Rev.* 2014 Sep; 33(2-3):823-32.

338. Acidic extracellular microenvironment and cancer. Kato Y, Ozawa S, Miyamoto C, et al. *Cancer Cell Int.* 2013 Sep; 13(1):89.

339. Acidity generated by the tumor microenvironment drives local invasion. Estrella V, Chen T, Lloyd M, et al. *J Cancer Res.* 2013 Mar; 73(5):1524-35.

340. How protons pave the way to aggressive cancers. Swietach P, Boedtkjer E, Pedersen SF. *Nat Rev Cancer.* 2023 Dec; 23(12):825-41.

341. Iron in the tumor microenvironment. Vela D. *Adv Exp Med Biol.* 2020; 1259:39-51.

342. Role of iron in carcinogenesis: Cancer as a ferrotoxic disease. Toyokuni S. *Cancer Sci.* 2009 Jan; 100(1):9-16.

343. Iron and cancer: Recent insights. Manz DH, Blanchette NL, Paul BT, et al. *Ann NY Acad Sci.* 2016 Mar; 1368(1):149-61.

344. The iron metabolism of neoplastic cells: Alterations that facilitate proliferation? Kwok JC, Richardson DR. *Crit Rev Oncol Hematol.* 2002 Apr; 42(1):65-78.

345. Iron metabolism in cancer. Wang Y, Yu L, Ding J, et al. *Int J Mol Sci.* 2018 Dec; 20(1):95.

346. Iron metabolism and drug resistance in cancer. Kazan HH, Urfali-Mamatoglu C, Gunduz U. *Biometals.* 2017 Oct; 30(5):629-41.

Thorp KE. Energy Dynamics in Breast Cancer: Fields, Hormones & the Menstrual Cycle. G Med Sci. 2024; 5(1):169-249.
https://www.doi.org/10.46766/thegms.oncol.24111101

347. Iron metabolism in cancer progression. Forciniti S, Greco L, Grizzi F, et al. *Int J Mol Sci.* 2020 Mar; 21(6):2257.

348. Cells electric charge analyses define specific properties for cancer cells activity. Moleón Baca JA, Ontiveros Ortega A, Aránega Jiménez A, et al. *Bioelectrochemistry.* 2022 Apr; 144:108028.

349. Zeta potentials, van der Waals forces, and hemagglutination. van Oss CJ, Absolom DR. *Vox Sang.* 1983 Mar; 44(3):183-90.

350. Zeta potential: A surface electrical characteristic to probe the interaction of nanoparticles with normal and cancer human breast epithelial cells. Zhang Y, Yang M, Portney NG, et al. *Biomed Microdevices.* 2008 Apr; 10(2):321-28.

351. Dynamic characterization of human breast cancer cells using a piezoresistive microcantilever. Shim S, Kim MG, Jo K, et al. *J Biomech Eng.* 2010 Oct; 132(10):104501.

352. Zeta potential: A surface electrical characteristic to probe the interaction of nanoparticles with normal and cancer human breast epithelial cells. Zhang Y, Yang M, Portney NG, et al. *Biomed Microdevices.* 2008 Apr; 10(2):321-28.

353. Monitoring the zeta potential of human cells upon reduction in their viability and interaction with polymers. Bondar OV, Saifullina DV, Shakhmaeva II, et al. *Acta Naturae.* 2012 Jan; 4(1):78-81.

354. Zeta potential: A case study of cationic, anionic, and neutral liposomes. Smith MC, Crist RM, Clogston JD, et al. *Anal Bioanal Chem.* 2017 Sep; 409(24):5779-87.

355. Zeta potential measurement. Clogston JD, Patri AK. *Methods Mol Biol.* 2011; 697:63-70.

356. Nonlinear dependence (on ionic strength, pH) of surface charge density and zeta potential in microchannel electrokinetic flow. Chen D, Arancibia-Miranda N, Escudey M, et al. *Heliyon.* 2023 Oct; 9(10):e20888.

357. Measurement of the size and concentration and zeta potential of extracellular vesicles using nanoparticle tracking analyzer. Dissanayake K, Midekessa G, Lättekivi F, et al. *Methods Mol Biol.* 2021; 2273:207-18.

358. Extracellular vesicles and their zeta potential as future markers associated with nutrition and molecular biomarkers in breast cancer. Mendivil-Alvarado H, Limon-Miro AT, Carvajal-Millan E, et al. *Int J Mol Sci.* 2023 Apr; 24(7):6810.

359. Bioelectric potential gradients may initiate cell cycling: ELF and zeta potential gradients may mimic this effect. Beech JA. *Bioelectromagnetics.* 1997; 18(5):341-48.

360. Inflammation and cancer: Breast cancer as a prototype. Mantovani A, Marchesi F, Porta C, et al. *Breast.* 2007 Dec; 16(Suppl 2):S27-33.

361. Inflammation and cancer. Eiró N, Vizoso FJ. *J Gastroenterol Surg.* 2012 Mar;4(3):62-72.

362. Inflammation and cancer. Singh N, Baby D, Rajguru JP, et al. *Ann Afr Med.* 2019 Jul; 18(3):121-26.

363. Cancer-related inflammation, the seventh hallmark of cancer: Links to genetic instability. Colotta F, Allavena P, Sica A, et al. 2009 Jul; 30(7):1073-81.

364. Inflammation, a key event in cancer development. Lu H, Ouyang W, Huang C. *Mol Cancer Res.* 2006 Apr; 4(4):221-33.

365. Cancer-related inflammation. Mantovani A, Allavena P, Sica A, et al. *Nature.* 2008 Jul; 454(7203):436-44.

Thorp KE. Energy Dynamics in Breast Cancer: Fields, Hormones & the Menstrual Cycle. G Med Sci. 2024; 5(1):169-249.
https://www.doi.org/10.46766/thegms.oncol.24111101

366. Cytokines as a key component of cancer-related inflammation. Germano G, Allavena P, Mantovani A. *Cytokine.* 2008 Sep; 43(3):374-79.

367. Inflammatiion and cancer: Friend or foe? Turizo-Smith AD, Córdoba-Hernandez S, Mejía-Guarnizo LV, et al. *Front Pharmacol.* 2024 May; 15(1385479.

368. A blueprint for cancer-related inflammation and host innate immunity. García-López L, Adrados I, Ferres-Marco D, et al. *Cells.* 2021 Nov; 10(11):3211.

369. DNA damage in inflammation-related carcinogenesis and cancer stem cells. Ohnishi S, Ma N, Thanan R, et al. *Oxid Med Cell Longev.* 2013; 2013:387014.

370. Cancer-related inflammation. Candido J, Hagemann T. *J Clin Immunol.* 2013 Jan; 33 Suppl:S79-84.

371. The War burg effect: Persistence of stem-cell metabolism in cancers as a failure of differentiation. Riester M, Xu Q, Moreira A, et al. *Ann Oncol.* 2018 Jan; 29(1):264-70.

372. Human embryonic stem cells and embryonal carcinoma cells have overlapping and distinct metabolic signatures. Abu Dawud R, Schreiber K, Schomburg D, et al. *PLoS One.* 2012; 7(6):e39896.

373. Stem metabolism: Insights from oncometabolism and vice versa. Rodrigues AS, Pereira SL, Ramalho-Santos J, et al. *Biochim Biophys Acta Mol Basis Dis.* 2020 Jul; 1866(7):165760.

374. Metabolism in embryonic and cancer stemness. Jang H, Yang J, Lee E. *Arch Pharm Res.* 2015 Mar; 38(3):381-88.

375. Cancer etiology: A metabolic disease originating from life's major evolutionary transition? Poljsak B, Kovac V, Dahmane R, et al. *Oxid Med Cell Longev.* 2019 Oct; 2019:7831952.

376. The role of the mitochondria in the evolution of stem cells, including MUSE stem cells and their biology. Trosko JE. *Adv Exp Med Biol.* 2018; 1103-131-52.

377. Core pluripotency factors directly regulate metabolism in embryonic stem cell to maintain pluripotency. Kim H, Jang H, Kim TW, et al. *Stem Cells.* 2015 Sep; 33(9):2699-711.

378. Hallmarks of cancer stem cell metabolism. Sancho P, Barneda D, Heeschen C *Br J Cancer* 2016 Jun;114(12)1305-12

379. The Emerging Facets of Non-Cancerous Warburg Effect. Abdel-Haleem AM, Lewis NE, Jamshidi N, et al. *Front Endocrinol (Lausanne).* 2017 Oct; 8:279.

380. A role for the Warburg effect in preimplantation embryo development: Metabolic modification to support rapid cell proliferation. Krisher RL, Prather RS. *Mol Reprod Dev.* 2012 May; 79(5):311-20.

381. Severely polarized extracellular acidity around tumour cells. Feng Q, Bennett Z, Grichuk A, et al. *Nat Biomed Engin.* 2024 Jun; 8(6):787-99.

382. Acidic extracellular microenvironment and cancer. Kato Y, Ozawa S, Miyamoto C, et al. *Cancer Cell Int.* 2013 Sep; 13(1):89.

383. Impact of the acidic microenvironment on gene expression and functional parameters of tumors in vitro and in vivo. Rauschner M, Lange L, Hüsing T, et al. *J Exp Clin Cancer Res.* 2021 Jan; 40(1):10.

384. Association between pH regulation of the tumor microenvironment and immunological state. Hosonuma M, Yoshimura K. *Front Oncol.* 2023 Jul; 13:1175563.

385. Acidic extracellular environment affects mRNA expression in tumors in vitro and in vivo. Riemann A, Reime S, Thews O. *Int J Cancer.* 2019 Apr; 144(7):1609-18.

Thorp KE. Energy Dynamics in Breast Cancer: Fields, Hormones & the Menstrual Cycle. G Med Sci. 2024; 5(1):169-249.
https://www.doi.org/10.46766/thegms.oncol.24111101

386. Lactic acid and an acidic tumor microenvironment suppress anticancer immunity. Wang JX, Choi SYC, Niu X, et al. *Int J Mol Sci.* 2020 Nov; 21(21):8363.

387. Role of acidosis-sensitive microRNAs in gene expression and functional parameters of tumors in vitro and in vivo. Rauschner M, Hüsing T, Lange L, et al. *Neoplasia.* 2021 Dec; 23(12):1275-88.

388. Functional impact of acidosis-regulated microRNAs on the migration and adhesion of tumor cells. Hüsing T, Lange L, Rauschner M, et al. *Adv Exp Med Biol.* 2012; 1269:151-55.

389. Tumor-derived lactate creates a favorable niche for tumor via supplying energy source for tumor and modulating the tumor microenvironment. Jin M, Cao W, Chen B, et al. *Front Cell Dev Biol.* 2022 May; 10:808859.

390. Lactate-lactylation hands between metabolic reprogramming and immunosuppression. Chen L, Huang L, Gu Y, et al. *Int J Mol Sci.* 2022 Oct; 23(19):11943.

391. Lactate in the tumor microenvironment: An essential molecule in cancer progression and treatment. Pérez-Tomás R, Pérez-Guillén I. *Cancers (Basel).* 2020 Nov; 12(11):3244.

392. Lactate enhances motility of tumor cells and inhibits monocyte migration and cytokine release. Goetze K, Walenta S, Ksiazkiewicz M, et al. *Int J Oncol.* 2011 Aug; 39(2):453-63.

393. Inhibitory effect of tumor cell-derived lactic acid on human T-cells. Fischer K, Hoffmann P, Voelkl S, et al. *Blood.* 2007 May; 109(9):3812-19.

394. Acidity generated by the local tumor microenvironment drives local invasion. Estrella V, Chen T, Lloyd M, et al. *Cancer Res.* 2013 Mar; 73(5):1524-35.

395. Extracellular acidity as a favouring factor of tumour progression and metastatic dissemination. Calorini L, Peppicelli S, Bianchini F. *Exp Oncol.* 2012 Jul; 34(2):79-84.

396. Breast cancer and the immune system. Standish LJ, Sweet ES, Novack J, et al. *J Soc Integr Oncol.* 2008 Fall; 6(4):158-68.

397. Iron metabolism: State of the art in hypoxic cancer cell biology. Liu S, Cao X, Wang D, et al. *Arch Biochem Biophys.* 2022 Jul; 723:109199.

398. Cancer: The role of iron and ferroptosis. Rishi G, Huang G, Subramaniam VN. *Int J Biochem Cell Biol.* 2021 Dec; 141:106194.

399. Mitochondria and iron: Current questions. Paul BT, Manz DH, Torti FM, et al. *Exp Rev Hematol.* 2017 Jan; 10(1):65-79.

400. Iron metabolism, free radicals, and oxidative injury. Emerit J, Beaumont C, Trivin F. *Biomed Pharmacother.* 2001 Jul; 55(6):333-39.

401. Iron, free radicals, and oxidative injury. McCord JM. *Semin Hematol.* 1998 Jan; 35(1):5-12.

402. Iron: An essential element of cancer metabolism. Rishi G, Huang G, Subramaniam VN, et al. *Cells.* 2020 Dec; 9(12):2591.

403. Iron metabolism in cancer progression. Forciniti S, Greco L, Grizzi F, et al. *Int J Mol Sci.* 2020 Mar; 21(6):2257.

404. Therapy-resistant nature of cancer stem cells in view of iron metabolism. Wang W, Tabu K, Aimaitijiang A, et al. *Inflamm Regen.* 2022 Nov; 42(1):34.

405. Ferroptosis: Cancer stem cells rely on iron until 'to die for' it. Cosialls E, El Hage R, Dos Santos L, et al. *Cells.* 2021 Nov; 10(11):2981.

Thorp KE. Energy Dynamics in Breast Cancer: Fields, Hormones & the Menstrual Cycle. G Med Sci. 2024; 5(1):169-249.
https://www.doi.org/10.46766/thegms.oncol.24111101

406. Ferroptosis in cancer stem cells. Xu T, Liu Y, Zhao Z, et al. *Pathol Res Pract.* 2023 May; 245:154492.

407. Regulation of iron metabolism and ferroptosis in cancer stem cells. Wang H, Zhang Z, Ruan S, et al. *Front Oncol.* 2023 Sep; 13:1251561.

408. Targeting ferroptosis as a vulnerability in cancer. Lei G, Zhuang L, Gan B, et al. *Nat Rev Cancer.* 2022 Jul; 22(7):381-96.

409. Iron metabolism: Backfire of cancer cell stemness and therapeutic modalities. Yu R, Hang Y, Tsai HI, et al. *Cancer Cell Int.* 2024 May; 24(1):157.

410. Targeting iron metabolism in cancer therapy. Morales M, Xue X. *Theranostics.* 2021 Jul; 11(17):8412-29.

411. Iron metabolism and drug resistance in cancer. Kazan HH, Urfali-Mamatoglu C, Gunduz U. *Biometals.* 2017 Oct; 30(5):629-41.

412. Ferroptosis in cancer progression. Zhang R, Chen J, Wang S, et al. *Cells.* 2023 Jul; 12(14):1820.

413. Iron hemostasis in breast cancer. Marques O, da Silva BM, Porto G, et al. *Cancer Lett.* 2014 May; 347(1):1-14.

414. Does excess iron play a role in breast carcinogenesis? An unresolved hypothesis. Kabat GC, Rohan TE. *Cancer Causes Control.* 2007 Dec; 18(10):1047-53.

415. Cellular iron metabolism in prognosis and therapy of breast cancer. Torti SV, Torti FM. *Crit Rev Oncog.* 2013; 18(5):435-48.

416. Iron metabolism in breast cancer. Durigova A, Jacot W, Pouderoux S, et al. *Ann Biol Clin (Paris).* 2012 Jul; 70(4):387-96.

417. Role of iron in estrogen-induced cancer. Liehr JG, Jones JS. *Curr Med Chem.* 2001 Jun; 8(7):839-49.

418. Dysregulation of iron metabolism in cancer stem cells. Recalcati S, Gammella E, Cairo G. *Free Radic Biol Med.* 2019 Mar; 133:216-20.

419. Regulation of iron metabolism and ferroptosis in cancer stem cells. Wang H, Zhang Z, Ruan S, et al. *Front Oncol.* 2023 Sep; 13:1251561.

420. Puzzling out iron complications in cancer drug resistance. Abedi M, Rahgozar S. *Crit Rev Oncol Hematol.* 2022 Oct; 178:103772.

421. Cancer-related inflammation, the seventh hallmark of cancer: links to genetic instability. Colotta F, Allavena P, Sica A, et al. *Carcinogenesis.* 2009 Jul; 30(7):1073-81.

422. Inflammation, a key event in cancer development. Lu H, Ouyang W, Huang C. *Mol Cancer Res.* 2006 Apr; 4(4):221-33.

423. Cancer-related inflammation. Mantovani A, Allavena P, Sica A, et al. *Nature.* 2008 Jul; 454(7203):436-44.

424. Cancer-related inflammation. Candido J, Hagemann T. *J Clin Immunol.* 2013 Jan; 33 (Suppl 1):S79-84.

425. Oxidative stress, inflammation and cancer: How are they linked? Reuter S, Gupta SC, Chaturvedi MM, et al. *Free Radic Biol Med.* 2010 Dec; 49(11):1603-16.

426. Disturbance of function (function laesa): The legendary fifth cardinal sign of inflammation, added by Galen to the four cardinal signs of Celsius. Rather LJ. *Bull NY Acad Med.* 1971 Mar; 47(3): 303-22.

Thorp KE. Energy Dynamics in Breast Cancer: Fields, Hormones & the Menstrual Cycle. G Med Sci. 2024; 5(1):169-249.
https://www.doi.org/10.46766/thegms.oncol.24111101

427. Inflammation and cancer: Triggers, mechanisms, and consequences. Greten FR, Grivennikov SI. *Immunity.* 2019 Jul; 51(1):27-41.

428. Inflammation and cancer. Coussens LM, Werb Z. *Nature.* 2002 Dec; 420(6917):860-67.

429. Molecular pathways in cancer-related inflammation. Del Prete A, Allavena P, Santoro G, et al. *Biochem Med (Zagreb).* 2011; 21(3):264-75.

430. Chronic inflammation and cancer: The role of mitochondria. Kamp DW, Shacter E, Weitzman SA. *Oncology (Williston Park).* 2011 Apr; 25(5):400-10.

431. Inflammation and oxidative stress in angiogenesis and vascular disease. Kim YW, West XZ, Byzova TV, et al. *J Mol Med (Berlin).* 2013 Mar; 91(3):323-28.

432. Pathological angiogenesis and inflammation in tissues. Jeong JH, Ojha U, Lee YM. *Arch Pharm Res.* 2021 Jan; 44(1):1-15.

433. Leukocytes, inflammation, and angiogenesis in cancer: Fatal attractions. Ruegg C. *J Leukoc Biol.* 2006 Oct; 80(4):682-84.

434. Cellular and molecular mechanisms of inflammation-induced angiogenesis. Szade A, Grochot-Przeczek A, Florczyk U, et al. *IUBMB Life.* 2015 Mar; 67(3):145-59.

435. Angiopoietin-like proteins in angiogenesis, inflammation and cancer. Carbone C, Piro G, Merz V, et al. *Int J Mol Sci.* 2018 Feb; 19(2):431.

436. Inflammation-mediated promotion of invasion and metastasis. Solinas G, Marchesi F, Garlanda C, et al. *Cancer Metastasis Rev.* 2010 Jun; 29(2):243-48.

437. Role of inflammatory cells and mediators in tumor invasion and metastasis. Mantovani A. *Cancer Metastasis Rev.* 2010 Jun; 29(2):241.

438. Role of interleukin-6 in cancer progression and therapeutic resistance. Kumari N, Dwarakanath BS, Das A, et al. *Tumour Biol.* 2016 Sep; 37(9):11553-72.

439. Functio Laesa: Cancer inflammation and therapeutic resistance. Liu M, Kalbasi A, Beatty GL. *J Oncol Pract.* 2017 Mar; 13(3):173-80.

440. DNA damage in inflammation-related carcinogenesis and cancer stem cells. Ohnishi S, Ma N, Thanan R, et al. *Oxid Med Cell Longev.* 2013; 2013:387014.

441. Chemoresistance and cancer-related inflammation: Two hallmarks of cancer connected by an atypical link, PKCzeta. Rimessi A, Patergnani S, Ioannidi E, et al. *Front Oncol.* 2013 Sep; 3:232.

442. A blueprint for cancer-related inflammation and host immunity. García-López L, Adrados I, Ferres-Marco D, et al. *Cells.* 2021 Nov; 10(11):3211.

443. Cytokines as a key component of cancer-related inflammation. Germano G, Allavena P, Mantovani A. *Cytokine.* 2008 Sep; 43(3):374-79.

444. Inflammation and cancer: Breast cancer as a prototype. Mantovani A, Marchesi F, Porta C, et al. *Breast.* 2007 Dec; 16 Suppl 2:S27-32.

445. The role of inflammation and its related microRNAs in breast cancer. A narrative review. Bahiraee A, Ebrahimi R, Halabian R, et al. *J Cell Physiol.* 2019 Nov; 234(11):19480-93.

446. The role of inflammation in inflammatory breast cancer. Fouad TM, Kogawa T, Reuben JM, et al. *Adv Exp Med Biol.* 2014; 816:53-73.

Thorp KE. Energy Dynamics in Breast Cancer: Fields, Hormones & the Menstrual Cycle. G Med Sci. 2024; 5(1):169-249.
https://www.doi.org/10.46766/thegms.oncol.24111101

447. The immune system and inflammation in breast cancer. Jiang X, Shapiro DJ. *Mol Cell Endocrinol.* 2014 Jan; 382(1):673-82.

448. Role of inflammation in obesity-related breast cancer. Crespi E, Bottai G, Santarpia L. *Curr Opin Pharmacol.* 2016 Dec; 31:114-22.

449. Inflammation, dysregulated metabolism and aromatase in obesity and breast cancer. Zahid H, Simpson ER, Brown KA. *Curr Opin Pharmacol.* 2016 Dec; 31:90-96.

450. Obesity-associated inflammation promotes angiogenesis and breast cancer via angopoietin-like-4. Kolb R, Kluz P, Tan ZW, et al. *Oncogene.* 2019 Mar; 38(13):2351-63.

451. Association of angiogenesis and inflammation-related gene functional polymorphisms with early-stage breast cancer prognosis. Korobeinikova E, Ugenskiene R, Insodaite R, et al. *Oncol Lett.* 2020 Jun; 19(6):3687-3700.

452. Energy dynamics in chronic heart failure, chronic kidney disease & the cardiorenal syndrome: A new causal paradigm. Thorp KE, Thorp JA, Northrup C, et al. *G Med Sci.* 2023; 4(1):290-347.

453. Energy dynamics in dementia & the neurodegenerative diseases: A new causal paradigm. Thorp KE, Thorp JA, Northrup C, et al. *G Med Sci.* 2023; 4(1):348-405.

454. Energy dynamics in the autoimmune disorders: The unity of immunity. Thorp KE, Thorp JA, Northrup C, et al. *G Med Sci.* 2023; 4(1):406-470.

455. Energy dynamics in the metabolic Syndrome: Underpinnings of an Evolving Global Catastrophe. Thorp KE, Thorp EM. *G Med Sci.* 2024; 5(1):61-146.

456. Breast cancers, mammary stem cells, and cancer stem cells, characteristics and hypotheses. Taurin S, Alkhalifa H. *Neoplasia.* 2020 Dec; 22(12):663-78.

457. Breast cancer stem cells as drivers of tumor chemoresistance, dormancy and relapse: New challenges and therapeutic opportunities. De Angelis ML, Francescangeli F, Zeuner A. *Cancers (Basel).* 2019 Oct; 11(10):1569.

458. Signaling pathways governing breast cancer stem cells behavior. Song K, Farzaneh M. *Stem Cell Res Ther.* 2021 Apr; 12(1):245.

459. Breast cancer stem cells: Biology and therapeutic implications. Butti R, Gunasekaran VP, Kumar TVS, et al. *Int J Biochem Cell Biol.* 2019 Feb; 107:38-52.

460. Breast cancer stem cells. Velasco-Velázquez MA, Homsi N, De La Fuente M, et al. *Int J Biochem Cell Biol.* 2012 Apr; 44(4):573-77.

461. Developmental signaling pathways regulating mammary stem cells and contributing to the etiology of triple-negative breast cancer. Rangel MC, Bertolette D, Castro NP, et al. *Breast Cancer Res Treat.* 2016 Apr; 156(2):211-26.

462. Dormancy and cancer stem cells: An enigma for therapeutic targeting. Talukdar S, Bhoopathi P, Emdad L, et al. *Adv Cancer Res.* 2019; 141:43-84.

463. Tumor dormancy and cancer stem cells: Two sides of the same coin? Kleffel S, Schatton T. *Adv Exp Med Biol.* 2013; 734:145-79.

464. Metastatic dormancy: A complex network between cancer stem cells and their microenvironment. Bleau AM, Agliano A, Larzabal L. *Histol Histopathol.* 2014 Dec; 29(12):1499-1510.

465. Tumor dormancy and slow-cycling cancer cells. Davis JE Jr, Kirk J, Ji Y, et al. *Adv Exp Med Biol.* 2019; 1164:199-206.

Thorp KE. Energy Dynamics in Breast Cancer: Fields, Hormones & the Menstrual Cycle. G Med Sci. 2024; 5(1):169-249.
https://www.doi.org/10.46766/thegms.oncol.24111101

466. Slow-cycling (dormant) cancer cells in therapy resistance, cancer relapse, and metastasis. Basu S, Dong Y, Kumar R, et al. *Semin Cancer Biol.* 2022 Jan; 78:90-103.

467. Breast cancers as ecosystems: A metabolic perspective. Martino F, Lupi M, Giraudo E, et al. *Cell Mol Life Sci.* 2023 Aug; 80(9):244.

468. Breast cancer stem cells programs: Enter the (non)-code. Salvador MA, Birnbaum D, Charafe-Jauffret E, et al. *Brief Funct Genomics.* 2016 May; 15(3):186-99.

469. Breast-cancer stem cells: Beyond semantics. Badve S, Nakshatri H. *Lancet Oncol.* 2012 Jan; 13(1):e43-48.

470. Breast cancer stem cells and intrinsic subtypes: Controversies rage on. Nakshatri H, Srour EF, Badve S. *Curr Stem Cell Res Ther.* 2009 Jan; 4(1):50-60.

471. Genetic heterogeneity, tumor microenvironment and immunotherapy in triple-negative breast cancer. Kudelova E, Smolar M, Holubekova V, et al. 2022 Nov; 23(23):14937.

472. The genomic and transcriptomic architecture of 2,000 breast tumors reveals novel subgroups. Curtis C, Shah SP, Chin SF, et al. *Nature.* 2012 Apr; 486(7403):346-52.

473. The landscape of cancer genes and mutational processes in breast cancer. Stephens PJ, Tarpey PS, Davies H, et al. *Nature.* 2012 May; 486(7403):400-04.

474. Sequence analysis of mutations and translocations across breast cancer subtypes. Banerji S, Cibulskis K, Rangel-Escareno C, et al. *Nature.* 2012 Jun; 486(7403):405-09.

475. Breast tumours maintain a reservoir of subclonal diversity during expansion. Minussi DC, Nicholson MD, Ye H, et al. *Nature.* 2021 Apr; 592(7853):302-308.

476. Unravelling subclonal heterogeneity and aggressive disease states in TNBC through single-cell RNA-seq. Karaayvaz M, Cristea S, Gillespie SM, et al. *Nat Commun.* 2018 Sep; 9(1):3588.

477. The causes and consequences of genetic heterogeneity in cancer evolution. Burrell RA, McGranahan N, Bartek J, et al. *Nature.* 2013 Sep; 501(7467):338-45.

478. Pervasive chromosomal instability and karyotype order in tumour evolution. Watkins TBK, Lim EL, Petkovic M, et al. *Nature.* 2020 Nov; 587(7832):126-32.

479. Cancer: An insurgency of clones. Gisselsson D, Egnell R. *Trends Cancer.* 2017 Feb; 3(2):73-75.

480. Clonal evolution in cancer. Greaves M, Maley CC. *Nature.* 2012 Jan; 481(7381):306-13.

481. Genomic characterization of *de novo* metastatic breast cancer. Garrido-Castro AC, Spurr LF, Hughes ME et al *Clin Cancer Res* 2021 Feb;27(4):1105-18

482. Genetic heterogeneity in therapy-naïve synchronous primary breast cancers and their metastases. Ng CKY, Bidard FC, Piscuoglio S, et al. *Clin Cancer Res.* 2017 Aug; 23(15):4402-15.

483. Genomic characterization of metastatic breast cancers. Bertucci F, Ng CKY, Patsouris A, et al. *Nature.* 2019 May; 569(7757):560-64.

484. Genomic characterization of de novo metastatic breast cancer. Mullangi S, Vasan N. *Clin Breast Cancer.* 2022 Feb; 22(2):98-102.

485. Transcriptional diversity and bioenergetic shift in human breast cancer metastasis revealed by single-cell RNA sequencing. Davis RT, Blake K, Ma D, et al. *Nat Cell Biol.* 2020 Mar; 22(3):310-20.

486. The still dark side of the moon: molecular mechanisms of lunar-controlled rhythms and clocks. Andreatta G, Tessmar-Raible K. *J Mol Biol.* 2020 May; 432(12):3525-46.

Thorp KE. Energy Dynamics in Breast Cancer: Fields, Hormones & the Menstrual Cycle. G Med Sci. 2024; 5(1):169-249.
https://www.doi.org/10.46766/thegms.oncol.24111101

487. An overview of monthly rhythms and clocks. Raible F, Takekata H, Tessmar-Raible K. *Front Neurol.* 2017 May; 8:189.

488. Circalunar clocks: Old experiments for a new era. Kaiser TS, Neumann J. *Bioessays.* 2021 Aug; 43(8):e2100074.

489. Lunar periodicity in human reproduction: A likely unit of biological time. Menaker W, Menaker A. *Am J Obstet Gynecol.* 1959 Apr; 77(4):905-14.

490. Common 30-day multiple in gestation time of terrestrial placentals. Brown FM. *Chronobiol Int.* 1988; 5(3):195-210.

491. A lunar effect on fertility. Criss TB, Marcum JP. *Soc Biol.* 1981 Spring; 28(1-2):75-80.

492. Women temporarily synchronize their menstrual cycles with the luminance and gravimetric cycles of the Moon. Helfrich-Förster C, Monecke S, Spiousas I, et al. *Sci Adv.* 2021 Jan; 7(5):eabe1358.

493. Circadian components of semilunar and lunar timing mechanisms. Neumann D. *J Biol Rhythms.* 1989 Summer; 4(2):285-94.

494. Menstrual and lunar cycles. Friedmann E. *Am J Obstet Gynecol.* 1981 Jun; 140(3):350.

495. The regulation of the menstrual cycle and its relation to the moon. Law SP. *Acta Obstet Gynecol Scand.* 1986; 65(1):45-48.

496. The menstrual cycle is influenced by weekly and lunar rhythms. Ecochard R, Leiva R, Bouchard TP, et al. *Fertil Steril.* 2024 Apr; 121(4):651-59.

497. Dueling hypotheses: Circatidal versus circalunidian battle basics. Palmer JD. *Chronobiol Int.* 1997 Jul; 14(4):337-46.

498. Towards an understanding of circatidal clocks. Rock A, Wilcockson D, Last KS. *Front Physiol.* 2022 Feb; 13:830107.

499. Lunar rhythms in the deep sea: Evidence from the reproductive periodicity of several marine invertebrates. Mercier A, Sun Z, Baillon S, et al. *J Biol Rhythms.* 2011 Feb; 26(1):82-86.

500. Circatidal rhythm and the veiled clockwork. Goto SG, Takekata H. *Opin Insect Sci.* 2015 Feb; 7:92-97.

501. Biological clocks: Riding the tides. de la Iglesia HO, Johnson CH. *Curr Opin.* 2013 Oct; 23(20):R921-23.

502. Biological clocks and rhythms in intertidal crustaceans. de la Iglesia HO, Hsu YW. *Front Biosci (Elite Ed).* 2010 Jun; 2(4):1394-404.

503. Bivalve mollusc circadian clock genes can run at tidal frequency. Tran D, Perrigault M, Ciret P, et al. *Proc Biol Sci.* 2020 Jan; 287(1918):20192440.

504. Births, fertility, rhythms and lunar cycle. A statistical study of 5,927,978 births. Guillon P, Guillon D, Lansac J, et al. *J Gynecol Obstet Biol Reprod (Paris).* 1986; 15(3):265-71.

505. Tidal and lunar rhythms. Neumann D. *Arzneimittelforschung.* 1978; 28(10a):1842-49.

506. Tidally rhythmic behavior of marine animals. Naylor E. *Symp Soc Exp Biol.* 1985; 39:63-93.

507. *Heat and Life: The Development of the Theory of Animal Heat.* Everett Mendelsohn. Harvard University Press. 1964.

508. Sensitivity of the human circadian pacemaker to moderately bright light. Boivin DB, Duffy JF, Kronauer RE, et al. *J Biol Rhythms.* 1994 Winter; 9(3-4):315-31.

Thorp KE. Energy Dynamics in Breast Cancer: Fields, Hormones & the Menstrual Cycle. G Med Sci. 2024; 5(1):169-249.
https://www.doi.org/10.46766/thegms.oncol.24111101

509. The effects of light at night on circadian clocks and metabolism. Fonken LK, Nelson RJ. *Endocr Rev.* 2014 Aug; 35(4):648-70.

510. The effect of light on the human circadian pacemaker. Czeisler CA. *Ciba Found Symp.* 1995; 183:290-302.

511. A phase response curve to single bright light pulses in human subjects. Khalsa SB, Jewett ME, Cajochen C, et al. *J Physiol.* 2003 Jun; 549(Pt 3):945-52.

512. Bright light resets the human circadian pacemaker independent of the timing of the sleep-wake cycle. Czeisler CA, Allan JS, Strogatz SH, et al. *Science.* 1986 Aug; 233(4764):667-71.

513. Phase resetting of the human circadian pacemaker with use of a single pulse of bright light. Dawson D, Lack L, Morris M. *Chronobiol Int.* 1993 Apr; 10(2):94-102.

514. Phase-amplitude resetting of the human circadian pacemaker via bright light: A further analysis. Jewett ME, Kronauer RE, Czeisler CA. *J Biol Rhythms.* 1994 Winter; 9(3-4):295-314.

515. Dose-response relationships for resetting of human circadian clock by light. Boivin DB, Duffy JF, Kronauer RE, et al. *Nature.* 1996 Feb; 379(6565):540-42.

516. Effect of photic stimulation on the human menstrual cycle. Dewan EM, Menkin MF, Rock J. *Photochem Photobiol.* 1978 May; 27(5):581-85.

517. Night light alters menstrual cycles. Lin MC, Kripke DF, Parry BL, et al. *Psychiatry Res.* 1990 Aug; 33(2):135-38.

518. Nocturnal light effects on menstrual cycle length. Rex KM, Kripke DF, Cole RJ. *J Altern Complemetary Med.* 1997 Winter; 3(4):387-90.

519. Early morning bright light phase advances the human circadian pacemaker within one day. Buresová M, Dvoráková M, Zvolský P, et al. *Neurosci Lett.* 1991 Jan; 121(1-2):47-50.

520. Efficacy of a single sequence of intermittent bright light pulses for delaying circadian phase in humans. Gronfier C, Wright KP Jr, Kronauer RE, et al. *Am J Physiol Endocrinol Metab.* 2004 Jul; 287(1):E174-81.

521. Circadian phase resetting by a single short-duration light exposure. Rahman SA, St Hilaire MA, Chang AM, et al. *JCI Insight.* 2017 Apr; 2(7):e89494.

522. Bright light can delay human temperature rhythm independent of sleep. Drennan M, Kripke DF, Gillin JC. *Am J Physiol.* 1989 Jul; 257(1 Pt2):R136-41.

523. The effects of evening light on body temperature. Kubota T, Uchiyama M, Hirokawa G, et al. *Psychiatry Clin Neurosci.* 1998 Apr; 52(2):248-49.

524. The influence of light on thermal responses. te Kulve M, Schellen L, Schlangen LJ, et al. *Acta Physiol (Oxf).* 2016 Feb; 216(2):163-85.

525. Thermoregulatory effect of suppressed endogenous melatonin by pre-sleep bright-light exposure in a cold environment. Ishibashi K, Arikura S, Kozaki T, et al. *Chronobiol Int.* 2010 Jun; 27(4):782-806.

526. Light exposure induces equivalent phase shifts of the endogenous circadian rhythms of circulating plasma melatonin and core body temperature in men. Shanahan TL, Czeisler CA. *J Clin Endocrinol Metab.* 1991 Aug; 73(2):227-35.

527. The relationship between insomnia and body temperatures. Lack LC, Gradisar M, Van Someren EJ, et al. *Sleep Med Rev.* 2008 Aug; 12(4):307-17.

Thorp KE. Energy Dynamics in Breast Cancer: Fields, Hormones & the Menstrual Cycle. G Med Sci. 2024; 5(1):169-249.
https://www.doi.org/10.46766/thegms.oncol.24111101

528. Effect of bright light at night on core temperature, subjective alertness and performance as a function of exposure time. Foret J, Daurat A, Tirilly G. *Scand J Work Environ Health.* 1998; 24 Suppl 3;115-20.

529. Objective measures of immediate 'energizing' effect of light: Studies review and data analysis. Danilenko KV. *Clocks Sleep.* 2022 Sep; 4(4):475-96.

530. Characterizing the temporal dynamics of melatonin and cortisol changes in response to nocturnal light exposure. Rahman SA, Wright KP Jr, Lockley SW, et al. *Sci Rep.* 2019 Dec; 9(1):19720.

531. Circadian and sleep-dependent regulation of hormone release in humans. Czeisler CA, Klerman EB. *Recent Prog Horm Res.* 1999; 54:97-130-32.

532. Effects of diurnal sleep on secretion of cortisol, luteinizing hormone, and growth hormone in man. Pietrowsky R, Meyrer R, Kern W, et al. *J Clin Endocrinol Metab.* 1994 Mar; 78(3):683-87.

533. Resetting of circadian melatonin and cortisol rhythms in humans by ordinary room light. Boivin DB, Czeisler CA. *Neuroreport.* 1998 Mar; 9(5):779-82.

534. Inhibition of melatonin secretion onset by low levels of illumination. der J, Armstrong SM, O'Brien C, et al. *J Sleep Res.* 1996 Jun; 5(2):77-82.

535. Melatonin-induced decrease in body temperature in women: A threshold event. Cagnacci A, Soldani R, Romagnolo C, et al. *Neuroendocrinology.* 1994 Nov; 60(5):549-52.

536. The effect of light on core body temperature is mediated by melatonin in women. Cagnacci A, Soldani R, Yen SS. *J Clin Endocrinol Metab.* 1993 Apr; 76(4):1036-68.

537. Effects of nocturnal bright light on saliva melatonin, core body temperature and sleep propensity rhythms in human subjects. Kubota T, Uchiyama M, Suzuki H, et al. *Neurosci Res.* 2002 Feb; 42(2):115-22.

538. Sensitivity of the human circadian pacemaker to nocturnal light: Melatonin phase resetting and suppression. Zeitzer JM, Dijk DJ, Kronauer R, et al. *J Physiol.* 2000 Aug; 526 (Pt 3):695-702.

539. Transition from dim to bright light in the morning induces an immediate elevation in cortisol levels. Leproult R, Colecchia EF, L'Hermite-Balériaux M, et al. *J Clin Endocrinol Metab.* 2001 Jan; 86(1):151-57.

540. The circadian system modulates the cortisol awakening response in humans. Bowles NP, Thosar SS, Butler MP, et al. *Front Neurosci.* 2022 Nov; 16:995452.

541. The cortisol awakening response: More than a measure of HPA axis function. Clow A, Hucklebridge F, Stalder T, et al. *Neurosci Biobehav Rev.* 2010 Sep; 35(1):97-103.

542. The cortisol awakening response in context. Clow A, Hucklebridge F, Thorn L. *Int Rev Neurobiol.* 2010; 93:153-75.

543. The influence of light wavelength on human HPA axis rhythms: A systematic review. Robertson-Dixon I, Murphy MJ, Crewther SG, et al. *Life (Basel).* 2023 Sep; 13(10):1968.

544. Hypothalamus-pituitary-thyroid axis. Ortiga-Carvalho TM, Chiamolera MI, Pazos-Moura CC, et al. *Compr Physiol.* 2016 Jun; 6(3):1387-428.

545. 60 years of neuroendocrinology: TRH, the first hypophysiotropic releasing hormone isolated: Control of the pituitary-thyroid axis. Joseph-Bravo P, Jaimes-Hoy L, Uribe RM, et al. *J Endocrinol.* 2015 Aug; 226(2):T85-100.

546. The hypothalamic-pituitary-thyroid axis. Carlson HE, Hershman JM. *Med Clin North Am.* 1975 Sep; 59(5):1045-53.

Thorp KE. Energy Dynamics in Breast Cancer: Fields, Hormones & the Menstrual Cycle. G Med Sci. 2024; 5(1):169-249.
https://www.doi.org/10.46766/thegms.oncol.24111101

547. Dominant role of thyrotropin-releasing hormone in the hypothalamic-pituitary-thyroid axis. Nikrodhanond AA, Ortiga-Carvalho TM, Shibusawa N, et al. *J Biol Chem.* 2006 Feb; 281(8):5000-07.

548. General background on the hypothalamic-pituitary-thyroid (HPT) axis. Zoeller RT, Tan SW, Tyl RW. *Crit Rev Toxicol.* 2007 Jan; 37(1-2):11-53.

549. The thyroid and the heart. Polikar R, Burger AG, Scherrer U, et al. *Circulation.* 1993 May; 87(5):1435-41.

550. Effects of thyroid hormones on the heart. Vargas-Uricoechea H, Bonelo-Perdomo A, Sierra-Torres CH. *Clin Invest Arterioscler.* 2014 Nov; 26(6):296-309.

551. Cardiac oxidative metabolism, function, and metabolic performance in mild hyperthyroidism: A non-invasive study using positron-emission tomography and magnetic resonance imaging. Bengel FM, Lehnert J, Ibrahim T, et al. *Thyroid.* 2003 May; 13(5):471-77.

552. Effects of thyroid hormone on the cardiovascular system. Fazio S, Palmieri EA, Lombardi G. *Recent Prog Horm Res.* 59:31-50.

553. Thyroid hormone and the cardiovascular system. Klein I, Ojamaa K. *NEJM.* 2001 Feb; 344(7): 501-09.

554. Non-exercise activity thermogenesis (NEAT): A component of total daily energy expenditure. Chung N, Park MY, Kim J, et al. *J Exerc Nutr Biochem.* 2018 Jun; 22(2):23-30.

555. Nonexercise activity thermogenesis—liberating the life-force. Levine JA. *J Intern Med.* 2007 Sep; 262(3):273-87.

556. Nonexercise activity thermogenesis in obesity management. Villablanca PA, Alegria JR, Mookadam F, et al. *Mayo Clin Proc.* 2015 Apr; 90(4):509-19.

557. Role of nonexercise activity thermogenesis (NEAT) in obesity. Kotz CM, Levine JM. *Minn Med.* 2005 Sep; 88(9):54-57.

558. Variability in energy expenditure and its components. Donahoo WT, Levine JA, Melanson EL. *Curr Opin Clin Nutr Metab Care.* 2004 Nov; 7(6):599-605.

559. *Anatomical Studies on the Motion of the Heart and Blood (The Leake Translation).* William Harvey publ. Charles C. Thomas, 1928.

560. Melatonin, hormone of darkness and more: occurrence, control mechanisms, actions and bioactive metabolites. Hardeland R. *Cell Mol Life Sci.* 2008 Jul; 65(13):2001-18.

561. Melatonin as a regulator of metabolism. Dzherieva IS, Volkova NI, Rapoport SI. *Kiln Med (Mosk).* 2012; 90(10):27-30.

562. Melatonin: Physiological effects in humans. Claustrat B, Leston J. *Neurochirurgie.* 2015 Apr; 61(2-3):77-84.

563. Circadian rhythm dysregulation and restoration: The role of melatonin. Vasey C, McBride J, Penta K. *Nutrients.* 2021 Sep; 13(10):3480.

564. Melatonin as a hormone: New physiological and clinical insights. Cipolla-Neto J, Amaral FGD. *Endocr Rev.* 2018 Dec; 39(6):990-1028.

565. Melatonin as a chronobiotic. Arendt J, Skene DJ. *Sleep Med Rev.* 2005 Feb; 9(1): 25-39.

566. Melatonin synthesis and function: Evolutionary history in animals and plants. Zhao D, Yu Y, Shen Y, et al. *Front Endocrinol (Lausanne).* 2019 Apr; 10:249.

Thorp KE. Energy Dynamics in Breast Cancer: Fields, Hormones & the Menstrual Cycle. G Med Sci. 2024; 5(1):169-249.
https://www.doi.org/10.46766/thegms.oncol.24111101

567. Melatonin in plants and other phototrophs: Advances and gaps concerning the diversity of functions. Hardeland R. *J Exp Bot.* 2015 Feb; 66(3):627-46.

568. Melatonin and its relation to plant hormones. Arnao MB, Hernández-Ruiz J. *Ann Bot.* 2018 Feb; 121(2):195-207.

569. Melatonin: A master regulator of plant development and stress responses. Sun C, Liu L, Wang L, et al. *Integr Plant Biol.* 2021 Jan; 63(1):126-45.

570. Melatonin-mediated temperature stress tolerance in plants. Raza A, Charagh S, García-Caparrós P, et al. *GM Crops Food.* 2022 Dec; 13(1):196-217.

571. Melatonin from microorganisms, algae, and plants as possible alternatives to synthetic melatonin. Arnao MB, Giraldo-Acosta M, Castejón-Castillejo A, et al. *Metabolites.* 2023 Jan; 13(1): 72.

572. Microorganisms: Producers of melatonin in fermented foods and beverages. Que Z, Ma T, Shang Y, et al. *Agric Food Chem.* 2020 Apr; 68(17):4799-811.

573. The vascular reactivity of melatonin. Monroe KK, Watts SW. *Gen Pharmacol.* 1998 Jan; 30(1):31-35.

574. Vasorelaxant effects of the chronic treatment with melatonin on mesenteric artery and aorta of spontaneously hypertensive rats. Girouard H, Chulak C, Lejossec M, et al. *J Hypertens.* 2001 Aug; 19(8):1369-77.

575. Vasorelaxing action of melatonin in rat isolated aorta; Possible endothelium dependent relaxation. Satake N, Oe H, Shibata S. *Gen Pharmacol.* 1991; 22(6):1127-33.

576. Melatonin mediates vasodilation through both direct and indirect activation of BK(Ca) channels. Zhao T, Zhang H, Jin C, et al. *J Mol Endocrinol.* 2017 Oct; 59(3):219-33.

577. Melatonin suppresses sympathetic vasomotor tone through enhancing $GABA_A$ receptor activity in the hypothalamus. Yu Q, Guo Q, Jin S, et al. *Front Physiol.* 2023 Mar; 14:1166246.

578. Exogenous melatonin administration modifies cutaneous vasoconstrictor response to whole body skin cooling in humans. Aoki K, Zhao K, Yamazaki F, et al. *J Pineal Res.* 2008 Mar; 44(2):141-48.

579. Vasorelaxing action of melatonin in rabbit basilar artery. Shibata S, Satake N, Takagi T, et al. *Gen Pharmacol.* 1989; 20(5):677-80.

580. Modification of cutaneous vasodilator response to heat stress by daytime exogenous melatonin administration. Aoki K, Stephens DP, Zhao K, et al. *Am J Physiol Regul Integr Comp Physiol.* 2006 Sep; 29(1):R619-24.

581. Role of melatonin in blood pressure regulation: An adjunct anti-hypertensive agent. Baker J, Kimpinski K. *Clin Exp Pharmacol Physiol.* 2018 Aug; 45(8):755-66.

582. Melatonin-induced decrease of body temperature in women: A threshold event. Cagnacci A, Soldani R, Romagnolo C, et al. *Neuroendocrinology.* 1994 Nov; 60(5):549-52.

583. Homeostatic versus circadian effects of melatonin on core body temperature in humans. Cagnacci A, Kräuchi K, Wirz-Justice A, et al. *J Biol Rhythms.* 1997 Dec; 12(6):509-17.

584. The effect of light on core body temperature is mediated by melatonin in women. Cagnacci A, Soldani R, Yen SS. *J Clin Endocrinol Metab.* 1993 Apr; 76(4):1036-38.

585. Hypothermic effect of melatonin and nocturnal core body temperature decline are reduced in aged women. Cagnacci A, Soldani R, Yen SS. *J Appl Physiol.* 1995 Jan; 78(1):314-17.

Thorp KE. Energy Dynamics in Breast Cancer: Fields, Hormones & the Menstrual Cycle. G Med Sci. 2024; 5(1):169-249.
https://www.doi.org/10.46766/thegms.oncol.24111101

586. Thermoregulatory effects of melatonin in relation to sleepiness. Kräuchi K, Cajochen C, Pache M, et al. *Chronobiol Int.* 2006; 23(1-2):475-84.

587. Core body temperature changes before sleep are associated with nocturnal heart rate variability. Bigalke JA, Cleveland EL, Barkstrom E, et al. *J Appl Physiol.* 2023 Jul; 135(1):136-45.

588. Melatonin: A major regulator of the circadian rhythm of core temperature in humans. Cagnacci A, Elliott JA, Yen SS. *J Clin Endocrinol Metab.* 1992 Aug; 75(2):447-52.

589. One molecule, many derivatives: A never-ending interaction of melatonin with reactive oxygen and nitrogen species? Tan DX, Manchester LC, Terron MP, et al. *J Pineal Res.* 2007 Jan; 42(1):28-42.

590. A review of the molecular aspects of melatonin's anti-inflammatory actions: Recent insights and new perspectives. Mauriz JL, Collado PS, Veneroso C, et al. *J Pineal Res.* 2013 Jan; 54(1):1-14.

591. The chemistry of melatonin's interaction with reactive species. Allegra M, Reiter RJ, Tan DX, et al. *J Pineal Res.* 2003 Jan; 34(1):1-10.

592. Melatonin: Detoxification of oxygen and nitrogen-based toxic reactants. Reiter RJ, Tan DX, Manchester LC, et al. *Adv Exp Med Biol.* 2003; 527:539-48.

593. Melatonin's impact on antioxidative and anti-inflammatory reprogramming in homeostasis and disease. Chitimus DM, Popescu MR, Voiculescu SE, et al. *Biomolecules.* 2020 Aug; 10(9):1211.

594. Melatonin as an antioxidant: Under promises but over delivers. Reiter RJ, Mayo JC, Tan DX, et al. *J Pineal Res.* 2016 Oct; 61(3):253-78.

595. Melatonin: Exceeding expectations. Reiter RJ, Tan DX, Galano A. *Physiology (Bethesda).* 2014 Sep; 29(5):325-33.

596. Melatonin as an antioxidant: Biochemical mechanisms and pathophysiological implications in humans. Reiter RJ, Tan DX, Mayo JC, et al. *Acta Biochim Pol.* 2003; 50(4):1129-46.

597. Melatonin and mitochondrial function. Leon J, Acuña-Castroviejo D, Sainz RM, et al. *J Life Sci.* 2004 Jul; 75(7):765-90.

598. Melatonin as a free radical scavenger in the ovarian follicle. Tamura H, Takasaki A, Taketani T, et al. *Endocrinol J.* 2013; 60(1):1-13.

599. The role of melatonin as an antioxidant in the follicle. Tamura H, Takasaki A, Taketani T, et al. *J Ovarian Res.* 2012 Jan; 5:5.

600. Essential actions of melatonin in protecting the ovary from oxidative damage. Cruz MH, Leal CL, Cruz JF, et al. *Theriogenology.* 2014 Oct; 82(7):925-32.

601. Role of melatonin in the epigenetic regulation of breast cancer. Korkmaz A, Sanchez-Barcelo EJ, Tan DX, et al. *Breast Cancer Res Treat.* 2009 May; 115(1):13-27.

602. Apoptotic effect of melatonin on ER-positive breast cancer cell lines: ADGRL4 gene expression and promoter methylation. Rajabi A, Safaralizadeh R, Saber A, et al. *Naunyn Schmiedebergs Arch Pharmacol.* 2024 Aug 23.

603. Melatonin and cancer suppression: Insights into its effects on DNA methylation. Davoodvandi A, Nikfar B, Reiter RJ, et al. *Cell Mol Biol Lett.* 2022 Sep; 27(1):73.

604. Melatonin enhances the development of porcine cloned embryos by improving DNA methylation reprogramming. Qu J, Sun M, Wang X, et al. *Cell Reprogram.* 2020 Jun; 22(3):156-66.

Thorp KE. Energy Dynamics in Breast Cancer: Fields, Hormones & the Menstrual Cycle. G Med Sci. 2024; 5(1):169-249.
https://www.doi.org/10.46766/thegms.oncol.24111101

605. Melatonin: A pleiotropic molecule that modulates DNA damage response and repair pathways. Majidinia M, Sadeghpour A, Mehrzadi S, et al. *J Pineal Res.* 2017 Aug; 63(1).

606. Melatonin: A smart molecule in the DNA repair system. Mir SM, Aliarab A, Goodarzi G, et al. *Cell Biochem Funct.* 2022 Jan; 40(1):4-16.

607. The cortisol awakening response predicts same morning executive function: Results from a 50-day case study. Law R, Evans P, Thorn L, Hucklebridge F, et al. *Stress.* 2015; 18(6):616-21.

608. The cortisol awakening response is related to executive function in older age. Evans P, Hucklebridge F, Loveday C, et al. *Int J Psychophysiol.* 2012 May; 84(2):201-04.

609. Circadian fluctuations in glucocorticoid level predict perceptual discrimination sensitivity. Obleser J, Kreitewolf J, Vielhauer R, et al. *Science.* 2021 Mar; 24(4):102345.

610. Associations between cortisol awakening response and resting electroencephalograph asymmetry. Duan H, Fang H, Zhang Y, et al. *Peer J.* 2019 Jun; 7:e7059.

611. The cortisol awakening response is associated with performance of a serial sequence reaction time task. Hodyl NA, Schneider L, Vallence AM, et al. *Int J Psychophysiol.* 2016 Feb; 100:12-18.

612. Associations between the cortisol awakening response and heart rate variability. Stalder T, Evans P, Hucklebridge F, et al. *Psychoneuroendocrinology.* 2011 May; 36(4):454-62.

613. A circadian rhythm in heart rate variability contributes to the increased cardiac sympathovagal response to awakening in the morning. Boudreau P, Yeh WH, Dumont GA, et al. *Chronobiol Int.* 2012 Jul; 29(6):757-68.

614. Autonomic changes during wake-sleep transition: A heart rate variability based approach. Shinar Z, Akselrod S, Dagan Y, et al. *Auton Neurosci.* 2006 Dec; 130(1-2):17-27.

615. Diurnal salivary cortisol in relation to body composition and heart rate variability in young adults. Cvijetic S, Keser I, Jurasović J, et al. *Front Endocrinol (Lausanne).* 2022 Mar; 13:831831.

616. Impact of sleep debt on metabolic and endocrine function. Spiegel K, Leproult R, Van Cauter E. *Lancet.* 1999 Oct; 354(9188):1435-39.

617. Sleep disturbances and insulin resistance. Van Cauter E. *Diabet Med.* 2011 Dec; 28(12):1455-62.

618. Sleep influences on obesity, insulin resistance, and the risk of type 2 diabetes. Reutrakul S, Van Cauter E. *Metabolism.* 2018 Jul; 84:56-66.

619. Metabolic consequences of sleep and sleep loss. Van Cauter E, Spiegel K, Tasali E, et al. *Sleep Med.* 2008 Sep; 9(Suppl 1):S23-28.

620. Sleep and the epidemic of obesity in children and adults. Van Cauter E, Knutson KL. *Eur J Endocrinol.* 2008 Dec; 159(Suppl 1):S59-66.

621. Disturbances of sleep and circadian rhythms: Novel risk factors for obesity. Broussard JL, Van Cauter E. *Curr Opin Endocrinol Diabetes Obes.* 2016 Oct; 23(5):353-59.

622. Roles of circadian rhythmicity and sleep in human glucose regulation. Van Cauter E, Polonsky KS, Scheen AJ. *Endocrinol Rev.* 1997 Oct; 18(5):716-38.

623. Association between sleep loss and increased risk of obesity and diabetes. Knutson KL, Van Cauter E. *Ann NY Acad Sci.* 2008; 1129:287-304.

624. Mitochondrial dysfunction in obesity. de Mello AH, Costa AB, Engel JDG, et al. *Life Sci.* 2018 Jan; 192:26-32.

Thorp KE. Energy Dynamics in Breast Cancer: Fields, Hormones & the Menstrual Cycle. G Med Sci. 2024; 5(1):169-249.
https://www.doi.org/10.46766/thegms.oncol.24111101

625. Mitochondrial dysfunction in the metabolic syndrome. Prasun P. *Biochim Biophys Acta Mol Basis Dis.* 2020 Oct; 1866(10):165838.

626. Mitochondrial dysfunction in obesity and reproduction. Das M, Sauceda C, Webster NJG. *Endocrinology.* 2021 Jan; 162(1):bqaa158.

627. Mitochondrial dysfunction, insulin resistance, and potential genetic implications. Sangwung P, Petersen KF, Shulman GI, et al. *Endocrinology.* 2020 Apr; 161(4):bqaa017.

628. Mitochondrial dysfunction and oxidative stress in insulin resistance. Rocha M, Rovira-Llopis S, Bañuls C, et al. *Curr Pharm Des.* 2013; 19(32):5730-41.

629. Mitochondrial dysfunction, insulin resistance, ad type 2 diabetes mellitus. Abdul-Ghani MA, DeFronzo RA. *Curr Diab Rep.* 2008 Jun; 8(3):173-78.

630. Energy dynamics in the metabolic syndrome: Underpinnings of an evolving global catastrophe. Thorp KE, Thorp EM. *G Med Sci.* 2024; 5(1):61-146.

631. Association of cortisol and the metabolic syndrome in Korean men and women. Park SB, Blumenthal JA, Lee SY, et al. *J Korean Med Sci.* 2011 Jul; 26(7):914-18.

632. Glucocorticoids associate with cardiometabolic risk factors in black South Africans. Dlamini SN, Lombard Z, Micklesfield LK, et al. *Endocrinol Connect.* 2021 Aug; 10(8):873-84.

633. Cortisol and the metabolic syndrome in South Asians. Ward AM, Fall CH, Stein CE, et al. *Clin Endocrinol (Oxf).* 2003 Apr; 58(4):500-05.

634. Elevated fasting plasma cortisol is associated with ischemic heart disease and its risk factors in people with type 2 diabetes: The Edinburgh type 2 diabetes study. Reynolds RM, Labad J, Strachan MW, et al. *J Clin Endocrinol Metab.* 2010 Apr; 95(4):1602-08.

635. The hypothalamic-pituitary-adrenal axis activity as a predictor of cardiovascular disease, type 2 diabetes and stroke. Rosmond R, Björntorp P. *J Intern Med.* 2000 Feb; 247(2):188-97.

636. Elevated plasma cortisol concentrations: A link between low birth weight and the insulin resistance syndrome? Phillips DI, Barker DJ, Fall CH, et al. *J Clin Endocrinol Metab.* 1998 Mar; 83(3): 757-60.

637. Impaired sensitivity to thyroid hormones is associated with diabetes and metabolic syndrome. Laclaustra M, Moreno-Franco B, Lou-Bonafonte JM, et al. *Diabetes Care.* 2019 Feb; 42(2):303-10.

638. Reduced sensitivity to thyroid hormone is associated with diabetes and hypertension. Mehran L, Delbari N, Amouzegar A, et al. *J Clin Endocrinol Metab.* 2022 Jan; 107(1):167-76.

639. The role of thyroid hormone in metabolism and metabolic syndrome. Teixeira PFDS, Dos Santos PB, Pazos-Moura CC. *Ther Adv Endocrinol Metab.* 2020 May; 11:2042018820917869.

640. Reduced sensitivity to thyroid hormone is associated with diabetes and hypertension. Mehran L, Delbari N, Amouzegar A, et al. 2022 Jan; 107(1):167-76.

641. Impaired sensitivity to thyroid hormones is associated with hyperuricemia, obesity, and cardiovascular disease risk in subjects with subclinical hypothyroidism. Sun Y, Teng D, Zhao L, et al. *Thyroid.* 2022 Apr; 32(4):376-84.

642. Impaired sensitivity to thyroid hormones is associated with central obesity in euthyroid type 2 diabetes mellitus patients with overweight and obesity. Chen F, Chen R, Zhou J, et al. *Diabetes Metab Syndr Obes.* 2024 Sep; 17:3379-96.

643. Correlation between thyroid hormone sensitivity and diabetic peripheral neuropathy in euthyroid patients with type 2 diabetes mellitus. Sun M, Yu L, Zhao X, et al. *Sci Rep.* 2024 Aug; 14(1):19603.

644. Obesity and metabolic syndrome: Association with chrono disruption, sleep deprivation, and melatonin suppression. Reiter RJ, Tan DX, Korkmaz A, et al. *Ann Med.* 2012 Sep; 44(6):564-77.

645. Melatonin, mitochondria, and the metabolic syndrome. Cardinali DP, Vigo DE. *Cell Mol Life Sci.* 2017 Nov; 74(21):3941-54.

646. Impaired melatonin secretion, oxidative stress and metabolic syndrome in night shift work. Hohor S, Mandanach C, Maftei A, et al. *Antioxidants (Basel).* 2023 Apr; 12(4):959.

647. Inflammaging, metabolic syndrome and melatonin: A call for treatment studies. Cardinali DP, Hardeland R. *Neuroendocrinology.* 2017; 104(4):382-97.

648. Melatonin: Manager of psychosomatic and metabolic disorders in polymorbid cardiovascular pathology. Partsernyak AS, Polyakova VO, Trufanov AG, et al. *Front Neurosci.* 2022 Sep; 16:989497.

649. Salivary melatonin levels and sleep-wake rhythms in pregnant women with hypertensive and glucose metabolic disorders: A prospective analysis. Shimada M, Seki H, Samejima M, et al. *Biosci Trends.* 2016 Feb; 10(1):34-41.

650. Melatonin and the metabolic syndrome: Physiopathologic and therapeutical implications. Cardinali DP, Cano P, Jiménez-Ortega V, et al. *Neuroendocrinology.* 2011; 93(3):133-42.

651. The effect of thyroid dysfunction on breast cancer risk: an updated meta-analysis. Tran TV, Kitahara CM, Leenhardt L, et al. *Endocr Relat Cancer.* 2022 Dec; 30(1):e220155.

652. Thyroid dysfunction and breast cancer risk among women in the UK Biobank cohort. Tran TV, Maringe C, Benitez Majano S, et al. *Cancer Med.* 2021 Jul; 10(13):4604-14.

653. Hormonal crosstalk between thyroid and breast cancer. Halada S, Casado-Medrano V, Baran JA, et al. *Endocrinology.* 2022 Jul; 163(7):bqac075.

654. Hypothyroidism and hyperthyroidism and breast cancer risk: A nationwide cohort study. Søgaard M, Farkas DK, Ehrenstein V, et al. *Eur J Endocrinol.* 2016 Apr; 174(4):409-14.

655. Thyroid diseases and breast cancer. Baldini E, Lauro A, Tripodi D, et al. *Pers Med.* 2022 Jan; 12(2):156.

656. Metabolic syndrome and breast cancer: Prevalence, treatment response, and prognosis. Dong S, Wang Z, Shen K, et al. *Front Oncol.* 2021 Mar; 11:629666.

657. Metabolic syndrome and postmenopausal breast cancer in the ORDET cohort: A nested case-control study. Agnoli C, Berrino F, Abagnato CA, et al. *Nutr Metab Cardiovasc Dis.* 2010 Jan; 20(1):41-48.

658. Relationship between metabolic syndrome and postmenopausal breast cancer. Ekinci O, Eren T, Kurtoglu Yakici M, et al. *Cir Esp (Engl Ed).* 2020 Nov; 98(9):540-46.

659. Metabolic syndrome and breast cancer risk: A case-cohort study nested in a multicenter Italian cohort. Agnoli C, Grioni S, Sieri S, et al. *PLoS One.* 2015 Jun; 10(6):e0128891.

660. Metabolic syndrome affects breast cancer risk in postmenopausal women. Capasso I, Esposito E, Pentimalli F, et al. *Cancer Biol Ther.* 2010 Dec; 10(12):1240-43.

661. Association between metabolic syndrome risk factors and immunohistochemical profile in women with breast cancer. Danesh H, Anbiaei R, Ziamajidi N, et al. *Iran J Med Sci.* 2023 Sep; 48(5): 456-64.

662. Risk of metabolic syndrome in postmenopausal breast cancer survivors. Buttros Dde A, Nahas EA, Vespoli Hde L, et al. *Menopause.* 2013 Apr; 20(4):448-54.

663. The role of obesity in breast cancer pathogenesis. Glassman I, Le N, Asif A, et al. *Cells.* 2023 Aug; 12(16):2061.

Thorp KE. Energy Dynamics in Breast Cancer: Fields, Hormones & the Menstrual Cycle. G Med Sci. 2024; 5(1):169-249.
https://www.doi.org/10.46766/thegms.oncol.24111101

664. Obesity and breast cancer. Stoll BA. *Int J Obes Relat Metab Disord.* 1996 May; 20(5):389-92.

665. Upper abdominal obesity, insulin resistance and breast cancer risk. Stoll BA. *Int J Obes Relat Metab Disord.* 2002 Jun; 26(6):747-53.

666. Effect of obesity on breast cancer development. Cleary MP, Grossmann ME, Ray A. *Vet Pathol.* 2010 Mar; 47(2):202-13.

667. Effects of melatonin supplementation on insulin levels and insulin resistance: A systematic review and insulin resistance: A systematic review and meta-analysis of randomized controlled trials. Li Y, Xu Z. *Horm Metab Res.* 2021 Sep; 53(9):616-24.

668. Exploring effects of melatonin supplementation on insulin resistance: An updated systematic review of animal and human studies. Fayazi F, Kheirouri S, Alizadeh M. *Diabetes Metab Syndr.* 2024 Jul; 18(7):103073.

669. Effects of daily administration of melatonin before bedtime on fasting insulin, glucose and insulin sensitivity in healthy adults and patients with metabolic diseases. A systematic review and analysis. Lauritzen ES, Kampmann U, Smedegaard SB, et al. *Clin Endocrinol (Oxf).* 2021 Nov; 95(5):691-701.

670. Melatonin alleviates insulin resistance through the PI3K/AKT signaling pathway in ovary granulosa cells of polycystic ovary syndrome. Guo R, Zheng H, Li Q, et al. *Reprod Biol.* 2022 May; 22(1):100594.

671. Role of melatonin in mitigation of insulin resistance and ensuing diabetic cardiomyopathy. Nath A, Ghosh S, Bandyopadhyay D. *Life Sci.* 2024 Oct; 355:122993.

672. Melatonin effects on glucose metabolism: Time to unlock the controversy. Garaulet M, Qian J, Florez JC, et al. *Trends Endocrinol Metab.* 2020 Mar; 31(3):192-204.

673. Beneficial effect of melatonin treatment on age-related insulin resistance and on the development of type 2 diabetes. Tresguerres JA, Cuesta S, Kireev RA, et al. *Horm Mol Biol Clin Invest.* 2013 Dec; 16(2):47-54.

674. Absence of melatonin induces night-time hepatic insulin resistance and increased gluconeogenesis due to stimulation of the nocturnal unfolded protein response. Nogueira TC, Lellis-Santos C, Jesus DS, et al. *Endocrinology.* 2011 Apr; 152(4):1253-63.

675. Melatonin, mitochondria, and the metabolic syndrome. Cardinali DP, Vigo DE. *Cell Mol Life Sci.* 2017 Nov; 74(21):3941-54.

676. Metabolic syndrome, its pathophysiology and the role of melatonin. Srinivasan V, Ohta Y, Espino J, et al. *Recent Pat Endocr Metab Immune Drug Discov.* 2013 Jan; 7(1):11-25.

677. Melatonin in mitochondria: mitigating clear and present dangers. Reiter RJ, Ma Q, Sharma R. *Physiology (Bethesda).* 2020 Mar; 35(2):86-95.

678. Melatonin, energy metabolism, and obesity: A review. Cipolla-Neto J, Amaral FG, Afeche SC, et al. *J Pineal Res.* 2014 May; 56(4):371-81.

679. Protective effects of melatonin and mitochondria-targeted antioxidants against oxidative stress: A review. Ramis MR, Esteban S, Miralles A, et al. *Curr Med Chem.* 2015; 22(22):2690-711.

680. The chemistry of melatonin's interaction with reactive species. Allegra M, Reiter RJ, Tan DX, et al. *J Pineal Res.* 2003 Jan; 34(1):1-10.

681. Melatonin and mitochondrial function. Leon J, Acuña-Castroviejo D, Sainz RM, et al. *Life Sci.* 2004 Jul; 75(7):765-90.

Thorp KE. Energy Dynamics in Breast Cancer: Fields, Hormones & the Menstrual Cycle. G Med Sci. 2024; 5(1):169-249.
https://www.doi.org/10.46766/thegms.oncol.24111101

682. Melatonin mitigates mitochondrial malfunction. León J, Acuña-Castroviejo D, Escames G, et al. *J Pineal Res.* 2005 Jan; 38(1):1-9.

683. Melatonin, mitochondria and hypertension. Baltatu OC, Amaral FG, Campos LA, et al. *Cell Mol Life Sci.* 2017 Nov; 74(21):3955-64.

684. Melatonin as a mitochondrial protector in the neurodegenerative diseases. Wongprayoon P, Govitrapong P. *Cell Mol Life Sci.* 2017 Nov; 74(21):3999-4014.

685. Melatonin role in mitochondrial function. Acuna-Castroviejo D, Escames G, Rodriguez MI, et al. *Front Biosci.* 2007 Jan; 12:947-63.

686. Protective role of melatonin in mitochondrial dysfunction and related disorders. Paradies G, Paradies V, Ruggiero FM, et al. *Arch Toxicol.* 2015 Jun; 89(6):923-39.

687. Melatonin-mitochondria interplay in health and disease. Acuña Castroviejo D, López LC, Escames G, et al. *Curr Top Med Chem.* 2011; 11(2):221-40.

688. Melatonin as a mitochondria-targeted anti-oxidant: one of evolution's best ideas. Reiter RJ, Rosales-Corral S, Tan DX, et al. *Cell Life Mol Sci.* 2017 Nov; 74(21):3863-81.

689. The effects of melatonin supplementation on inflammatory markers among patients with metabolic syndrome or related disorders: A systematic review and meta-analysis of randomized controlled trials. Akbari M, Ostadmohammadi V, Tabrizi R, et al. *Inflammopharmacology.* 2018 Aug; 26(4):899-907.

690. Melatonin administration lowers biomarkers of oxidative stress and cardio-metabolic risk in type 2 diabetic patients with coronary heart disease: A randomized, double-blind, placebo-controlled trial. Raygan F, Ostadmohammadi V, Bahmani F, et al. *Clin Nutr.* 2019 Feb; 38(1):191-96.

691. Anti-inflammatory effects of melatonin in obesity and hypertension. Prado NJ, Ferder L, Manucha W, et al. *Curr Hypertens Rep.* 2018 May; 20(5):45.

692. Metabolic and anti-inflammatory response to melatonin administration in patients with diabetic nephropathy. Satari M, Bahmani F, Reiner Z, et al. *Iran J Kidney Dis.* 2021 Jan; 1(1):22-30.

693. Melatonin's impact on antioxidative and anti-inflammatory reprogramming in homeostasis and disease. Chitimus DM, Popescu MR, Voiculescu SE, et al. *Biomolecules.* 2020 Aug; 10(9):1211.

694. Melatonin and its relation to the immune system and inflammation. Reiter RJ, Calvo JR, Karbownik M, et al. *Ann NY Acad Sci.* 2000; 917:376-86.

695. Melatonin: A pleiotropic molecule regulating inflammation. Radogna F, Diederich M, Ghibelli L. *Biochem Pharmacol.* 2010 Dec; 80(12):1844-52.

696. Melatonin and inflammation: Story of a double-edged sword. Hardeland R. *J Pineal Res.* 2018 Nov; 65(4):e12525.

697. Anti-inflammatory effects of melatonin: A mechanistic review. Nabavi SM, Nabavi SF, Sureda A, et al. *Crit Rev Food Sci Nutr.* 2019; 59(Suppl 1):S4-16.

698. A review of the molecular aspects of melatonin's anti-inflammatory actions: Recent insights and new perspectives. Mauriz JL, Collado PS, Veneroso C, et al. *J Pineal Res.* 2013 Jan; 54(1): 1-14.

699. Melatonin as a master regulator of cell death and inflammation: Molecular mechanisms and clinical implications for newborn care. Tarocco A, Caroccia N, Morciano G, et al. *Cell Death Dis.* 2019 Apr; 10(4):317.

700. Melatonin effects on non-alcoholic fatty liver disease are related to microRNA-34a-5p/Srt1 axis and autophagy. Stacchiotti A, Grossi I, García-Gómez R, et al. *Cells.* 2019 Sep; 8(9):1053.

Thorp KE. Energy Dynamics in Breast Cancer: Fields, Hormones & the Menstrual Cycle. G Med Sci. 2024; 5(1):169-249.
https://www.doi.org/10.46766/thegms.oncol.24111101

701. Melatonin prevents alcohol- and metabolic dysfunction-associated steatotic liver disease by mitigating gut dysbiosis, intestinal barrier dysfunction and endotoxemia. LeFort KR, Rungratanawanich W, Song BJ. *Antioxidants (Basel).* 2023 Dec; 13(1):43.

702. Melatonin: A potential intervention for hepatic steatosis. Sun H, Huang FF, Qu S. *Lipids Health Dis.* 2015 Jul; 14:75.

703. Melatonin improves insulin resistance and hepatic steatosis through attenuation of alpha-2-HS-glycoprotein. Heo JI, Yoon DW, Yu JH, et al. *J Pineal Res.* 2018 Sep; 65(2):e12493.

704. The role of melatonin in polycystic ovary syndrome: A review. Mojaverrostami S, Asghari N, Khamisabadi M, et al. *Int J Reprod Biol.* 2019 Dec; 17(12):865-82.

705. Melatonin effective to reduce the microscopic symptoms of polycystic ovary syndrome-related infertility: An experimental study. Arık GN, Kaplanoğlu GT, Sağlam ASY, et al. *Tissue Cell.* 2023 Apr; 81:102015.

706. Effect of melatonin supplementation on cardiometabolic risk factors, oxidative stress and hormonal profile in PCOS patients: A systematic review and meta-analysis of randomized clinical trials. Ziaei S, Hasani M, Malekahmadi M, et al. *J Ovarian Res.* 2024 Jul; 17(1):138.

707. Melatonin treatment may be able to restore menstrual cyclicity in women with PCOS: A pilot study. Tagliaferri V, Romualdi D, Scarinci E, et al. *Reprod Sci.* 2018 Feb; 25(2):269-75.

708. The biology of gonadotropin-releasing hormone and its analogs. Glode LM. *Urology.* 1986 Jan; 27(Suppl 1):16-20.

709. Effect of estradiol on nonmalignant human mammary cells in primary culture. Chambon M, Cavalie-Barthez G, Veith F, et al. *Cancer Res.* 1984 Dec; 44(12 Pt 1):5733-43.

710. Effects of sex steroids on proliferation in normal mammary tissue. Söderqvist G. *Ann Med.* 1998 Dec; 30(6):511-24.

711. The action of estrogens and progestogens in the young female breast. Zolfaroli I, Tarín JJ, Cano A. *Eur J Obstet Gynecol Reprod Biol.* 2018 Nov; 230:204-07.

712. Estrogens, progestogens, normal breast cell proliferation, and breast cancer risk. Pike MC, Spicer DV, Dahmoush L, et al. *Epidemiol Rev.* 1993; 15(1):17-35.

713. Growth control and differentiation in mammary epithelial cells. Borellini F, Oka T. *Environ Health Perspect.* 1989 Mar; 80:85-99.

714. William L. McGuire Memorial Symposium: Estrogen and progesterone effects in human breast carcinogenesis. King RJ. *Breast Cancer Res Treat.* 1993; 27(1-2):3-15.

715. Mammographic breast density and the risk of breast cancer: A systematic review and meta-analysis. Bodewes FTH, van Asselt AA, Dorrius MD, et al. *Breast.* 2022 Dec; 66:62-68.

716. The influence of mammogram acquisition on the mammographic density and breast cancer association in the Mayo Mammography Health Study cohort. Olson JE, Sellers TA, Scott CG, et al. *Breast Cancer Res.* 2012 Nov; 14(6):R147.

717. Breast Cancer Risk and mammographic density assessed with semiautomated and fully automated methods and BI-RADS. Jeffers AM, Sieh W, Lipson JA, et al. *Radiology.* 2017 Feb; 282(2):348-55.

718. Endogenous sex hormones, prolactin and mammographic density in postmenopausal Norwegian women. Bremnes Y, Ursin G, Bjurstam N, et al. *Int J Cancer.* 2007 Dec; 121(11):2506-11.

Thorp KE. Energy Dynamics in Breast Cancer: Fields, Hormones & the Menstrual Cycle. G Med Sci. 2024; 5(1):169-249.
https://www.doi.org/10.46766/thegms.oncol.24111101

719. Effect of baseline breast density on breast cancer incidence, stage, mortality, and screening parameters: A 25-year follow-up of a Swedish mammographic screening. Chiu SY, Duffy S, Yen AM, et al. *Cancer Epidemiol Biomarkers Prev.* 2010 May; 19(5):1219-26.

720. Endogenous hormone levels, mammographic density, and subsequent risk of breast cancer in postmenopausal women. Tamimi RM, Byrne C, Colditz GA, et al. *J Natl Cancer Inst.* 2007 Aug; 99(15):1178-87.

721. The association of endogenous sex steroids and sex steroid binding proteins with mammographic density: Results from the Postmenopausal Estrogen/Prgestin Interventions Mammographic Density Study. Greendale GA, Palla SL, Ursin G, et al. *Am J Epidemiol.* 2005 Nov; 162(9):826-34.

722. Relationships between circulating hormone levels, mammographic percent density and breast cancer risk factors in postmenopausal women. Johansson H, Gandini S, Bonanni B, et al. *Breast Cancer Res Treat.* 2008 Mar; 108(1):57-67.

723. Association of the interaction between mammographic breast density, body mass index, and menopausal status with breast cancer risk among Korean women. Tran TXM, Moon SG, Kim S, et al. *JAMA Netw Open.* 2021 Dec; 4(12):e2139161.

724. Postmenopausal plasma sex hormone levels and breast cancer risk over 20 years of follow-up. Zhang X, Tworoger SS, Eliassen AH, et al. *Breast Cancer Res Treat.* 2013 Feb; 137(3): 883-92.

725. Association of serum estrone levels with estrogen receptor-positive breast cancer risk in postmenopausal Japanese women. Miyoshi Y, Tanji Y, Taguchi T, et al. *Clin Cancer Res.* 2003 Jun; 9(6):2229-33.

726. Endogenous hormones and risk of breast cancer in postmenopausal women. Hankinson SE. *Breast Dis.* 2005; 24:3-15.

727. Circulating estrogen metabolites and risk of breast cancer in postmenopausal women. Arslan AA, Koenig KL, Lenner P, et al. *Cancer Epidemics Biomarkers.* 2014 Jul; 23(7):1290-97.

728. Serum oestrogen levels in postmenopausal women: comparison of American whites and Japanese in Japan. Shimzu H, Ross RK, Bernstein L, et al. *Br J Cancer.* 1990 Sep; 62(3):451-53.

729. Estrogen and cancer. Liang J, Shang Y. *Annu Rev Physiol.* 2013; 75:225-40.

730. Sex hormones and breast cancer risk and prognosis. Folkerd E, Dowsett M. *Breast.* 2013 Aug; 22 (Suppl 2):S38-43.

731. Estrogen carcinogenesis in breast cancer. Yager JD, Davidson NE. *NEJM.* 2006 Jan; 354(3):270-82.

732. Oestrogen directly stimulates growth factor signal transduction pathways in human breast cancer cells. van der Burg B, de Groot RP, Isbrücker L, et al. *J Steroid Biochem Mol Biol.* 1991; 40(1-3):215-21.

733. Endogenous hormones as a major factor in human cancer. Henderson BE, Ross RK, Pike MC, et al. *Cancer Res.* 1982 Aug; 42(8):3232-39.

734. Effects of estrogen on breast cancer development: Role of estrogen receptor independent mechanisms. Yue W, Wang JP, Li Y, et al. *Int J Cancer.* 2010 Oct; 127(8):1748-57.

735. Endogenous estrogens: breast cancer and chemoprevention. Starek-Świechowicz B, Budziszewska B, Starek A. *Pharmacol Rep.* 2021 Dec; 73(6):1497-1512.

736. Endogenous concentration and subcellular distribution of estrogens in normal and malignant human breast tissue. van Landeghem AA, Poortman J, Nabuurs M, et al. *Cancer Res.* 1985 Jun; 45(6):2900-06.

Thorp KE. Energy Dynamics in Breast Cancer: Fields, Hormones & the Menstrual Cycle. G Med Sci. 2024; 5(1):169-249.
https://www.doi.org/10.46766/thegms.oncol.24111101

737. Control and expression of estrone sulphatase activities in human breast cancer. Pasqualini JR, Chetrite G, Nestour EL. *J Endocrinol.* 1996 Sep; 150 Suppl:S99-105.

738. Determinants of tissue oestradiol levels in human breast cancer. Santen RJ. *Cancer Surv.* 1986; 5(3):597-616.

739. Recent insight on the control of enzymes involved in estrogen formation and transformation in human breast cancer. Pasqualini JR, Chetrite GS. *J Steroid Biochem Mol Biol.* 2005 Feb; 93(2):221-36.

740. Role of inflammation and oxidative stress in post-menopausal oestrogen-dependent breast cancer. Madeddu C, Gramignano G, Floris C, et al. *J Cell Mol Med.* 2014 Dec; 18(12):2519-29.

741. Critical role of oxidative stress in estrogen-induced carcinogenesis. Bhat HK, Calaf G, Hei TK, et al. *Proc Natl Acad Sci USA.* 2003 Apr; 100(7):3913-18.

742. Estrogen-induced generation of reactive oxygen and nitrogen species, gene damage, and estrogen-dependent cancers. Roy D, Cai Q, Felty Q, et al. *Toxicol Environ Health B Crit Rev.* 2007 Jun; 10(4):235-57.

743. Oxidative stress in breast cancer: A biochemical map of reactive oxygen species production. Bel'skaya LV, Dyachenko EI. *Curr Issues Mol Biol.* 2024; 46(5):4646-87.

744. Role of estrogen receptors in pro-oxidative and anti-oxidative actions of estrogenes: A perspective. Kumar S, Lata K, Mukhopadhyay S, et al. *Biochim Biophys Acta.* 2010 Oct; 1800(10):1127-35.

745. Influence of breast cancer risk factors on proliferation and and DNA damage in human breast glandular tissues: Role of intracellular estrogen levels, oxidative stress and estrogen biotransformation. Wunder J, Pemp D, Cecil A, et al. *Arch Toxicol.* 2022 Feb; 96(2):673-87.

746. Estrogen down-regulates uncoupling proteins and increases oxidative stress in breast cancer. Sastre-Serra J, Valle A, Company MM, et al. *Free Radic Biol Med.* 2010 Feb; 48(4):506-12.

747. Mitochondrial uncoupling: A key controller of biological processes in physiology and diseases. Demine S, Renard P, Arnould T. *Cells.* 2019 Jul; 8(8):795.

748. Mitochondrial electron transport chain, ROS generation and uncoupling (Review). Zhao RZ, Jiang S, Zhang L, et al. *Int J Mol Med.* 2019 Jul; 44(1):3-15.

749. New advances in adaptive thermogenesis: UCP1 and beyond. Chouchani ET, Kazak L, Spiegelman BM. *Cell Metab.* 2019 Jan; 29(1):27-37.

750. The role of uncoupling proteins in the regulation of metabolism. Erlanson-Albertsson C. *Acta Physiol Scand.* 2003 Aug; 178(4):405-12.

751. Uncoupling proteins: Their roles in adaptive thermogenesis and substrate metabolism reconsidered. Dulloo AG, Samec S. *Br J Nutr.* 2001 Aug; 86(2):123-39.

752. Mitochondrial uncoupling proteins in human physiology and disease. Hagen T, Vidal-Puig A. *Minerva Med.* 2002 Feb; 93(1):41-57.

753. Uncoupling proteins and the molecular mechanisms of thyroid thermogenesis. Solmonson A, Mills EM. *Endocrinology.* 2016 Feb; 157(2):455-62.

754. Thyroid hormone and uncoupling proteins. Lanni A, Moreno M, Lombardi A, et al. *FEBS Lett.* 2003 May; 543(1-3):5-10.

755. Thyroid hormone and other regulators of uncoupling proteins. Reitman ML, He Y, Gong DW. *Int J Obes Relat Metab Disord.* 1999 Jun; 23 (Suppl 6):S56-59.

Thorp KE. Energy Dynamics in Breast Cancer: Fields, Hormones & the Menstrual Cycle. G Med Sci. 2024; 5(1):169-249.
https://www.doi.org/10.46766/thegms.oncol.24111101

756. Potential involvement of mammalian and avian uncoupling proteins in the thermogenic effect of thyroid hormones. Collin A, Cassy S, Buyse J, et al. *Domest Anim Endocrinol.* 2005 Jul; 29(1):78-87.

757. Thyroid hormone regulation of metabolism. Mullur R, Liu YY, Brent GA. *Physiol Rev.* 2014 Apr; 94(2):355-82.

758. Thermogenesis in adipose tissue activated by thyroid hormone. Yau WW, Yen PM. *Int J Mol Sci.* 2020 Apr; 21(8):3020.

759. Thyroid hormones in the regulation of brown adipose tissue thermogenesis. Sentis SC, Oelkrug R, Mittag J. *Endocrinol Connect.* 2021 Feb; 10(2):R106-R115.

760. Human uncoupling proteins and obesity. Schrauwen P, Walder K, Ravussin E. *Obes Res.* 1999 Jan; 7(1):97-105.

761. Uncoupling proteins: Functional characteristics and role in the pathogenesis of obesity and type II diabetes. Dalgaard LT, Pedersen O. *Diabetologia.* 2001 Aug; 44(8):946-65.

762. Obesity is associated with a decrease in expression but not with the hypermethylation of thermogenesis-related genes in adipose tissue. Kurylowicz A, Jonas M, Lisik W, et al. *J Transl Med.* 2015 Jan; 13:31.

763. Defective thermogenesis contributes to metabolic syndrome and liver steatosis in obese mice. Poekes L, Legry V, Schakman O, et al. *Clin Sci (Lond).* 2017 Feb; 131(4):285-96.

764. The role of uncoupling protein 1 (UCP1) on the development of obesity and type 2 diabetes mellitus. Brondani LA, Assmann TS, Duarte GC, et al. *Arq Bras Endocrinol Metab.* 2012 Jun; 56(4):215-25.

765. Mitochondrial thermogenesis and obesity. Gambert S, Ricquier D. *Curr Opin Clin Nutr Metab Care.* 2007 Nov; 10(6):664-70.

766. Human brown adipose tissue and metabolic health: Potential for therapeutic avenues. Singh R, Barrios A, Dirakvand G, et al. *Cells.* 2021 Nov; 10(11):3030.

767. Brown adipose tissue, thermogenesis, angiogenesis: Pathophysiological aspects. Honek J, Lim S, Fischer C, et al. *Horm Mol Biol Clin Investig.* 2014 Jul; 19(1):5-11.

768. Mitochondrial energy metabolism in the regulation of thermogenic brown fats and human metabolic diseases. Takeda Y, Harada Y, Yoshikawa T, et al. *Int J Mol Sci.* 2023 Jan; 24(2):1352.

769. Molecular pathways linking metabolic inflammation and thermogenesis. Solinas G. *Obes Rev.* 2012 Dec; 13 (Suppl 2):69-82.

770. Systemic correlates of white adipose tissue inflammation in early-stage breast cancer. Iyengar NM, Zhou XK, Gucalp A. *Clin Cancer Res.* 2016 May; 22(9):2283-89.

771. Obesity and inflammation: New insights into breast cancer development and progression. Iyengar NM, Hudis CA, Dannenberg AJ. *Am Soc Clin Oncol Educ Book.* 2013; 33:46-51.

772. The relationship between white adipose tissue inflammation and overweight/obesity in Chinese female breast cancer: A retrospective study. Zhao YX, Sun YL, Ye JH, et al. *Adv Ther.* 2020 Jun; 37(6):2734-47.

773. Metabolic pathways in obesity-related breast cancer. Brown KA. *Nat Rev Endocrinol.* 2021 Jun; 17(6):350-53.

774. Melatonin increases brown adipose tissue volume and activity in patients with melatonin deficiency: A proof-of-concept study. Halpern B, Mancini MC, Bueno C, et al. 2019 May; 68(5):947-52.

Thorp KE. Energy Dynamics in Breast Cancer: Fields, Hormones & the Menstrual Cycle. G Med Sci. 2024; 5(1):169-249.
https://www.doi.org/10.46766/thegms.oncol.24111101

775. Melatonin multiple effects on brown adipose tissue molecular machinery. de Souza CAP, Gallo CC, de Camargo LS, de Carvalho PVV, et al. *J Pineal Res.* 2019 Mar; 66(2):e12549.

776. Melatonin increases brown adipose tissue mass and function in Zucker diabetic fatty rats: Implications for obesity control. Fernández Vázquez G, Reiter RJ, et al. *J Pineal Res.* 2018 May; 64(4):e12472.

777. Significance and application of melatonin in the regulation of brown adipose tissue metabolism: Relation to obesity. Tan DX, Manchester LC, Fuentes-Broto L, et al. *Obes Rev.* 2011 Mar; 12(3):167-88.

778. Cellular model of Warburg effect identifies tumor promoting function of UCP2 in breast cancer and its suppression by genipin. Ayyasamy V, Owens KM, Desouki MM, et al. *PLoS One.* 2011; 6(9):e24792.

779. Negative regulation of UCP2 by TGFβ signaling characterizes low and intermediate-grade primary breast cancer. Sayeed A, Meng Z, Luciani G, et al. *Cell Death Dis.* 2010 Jul; 1(7):e53.

780. Uncoupling protein overexpression in metabolic disease and the risk of uncontrolled cell proliferation and tumorigenesis. Ruiz-Ramirez A, Lopez-Acosta O, Barrios-Maya M, et al. *Curr Mol Med.* 2017; 17(9):598-607.

781. Glucocorticoid receptor: A harmonizer of cellular plasticity in breast cancer-directs the road towards therapy resistance, metastasis progression and recurrence. Thakur D, Sengupta D, Mahapatra E, et al. *Cancer Metastasis Rev.* 2024 Mar; 43(1):481-99.

782. Glucocorticoid receptor-IRS-1 axis controls EMT and the metastasis of breast cancers. Shi W, Wang D, Yuan X, et al. *J Mol Cell.* 2019 Dec; 11(12);1042-55.

783. Breast cancer epithelial-to-mesenchymal transition: Examining the functional consequences of plasticity. Drasin DJ, Robin TP, Ford HL. *Breast Cancer Res.* 2011; 13(6):226.

784. Breast cancer cells: Focus on the consequences of epithelial-to-mesenchymal transition. Bong AHL, Monteith GR. *J Biochem Cell Biol.* 2017 Jun; 87:23-26.

785. Epithelial-to-mesenchymal transition and cancer stem cells contribute to breast cancer heterogeneity. Hong D, Fritz AJ, Zaidi SK, et al. *J Cell Physiol.* 2018 Dec; 233(12):9136-44.

786. Revisiting epithelial-mesenchymal transition in cancer metastasis: The connection between epithelial plasticity and stemness. Liao TT, Yang MH. *Mol Oncol.* 2017 Jul; 792-804.

787. Epithelial-mesenchymal plasticity in cancer progression and metastasis. Lu W, Kang Y. *Dev Cell.* 2019 May; 49(3):361-74.

788. EMT, cell plasticity and metastasis. Chaffer CL, San Juan BP, Lim E, et al. *Cancer Metastasis Rev.* 2016 Dec; 645-54.

789. Review article epithelial to mesenchymal transition-associated micro-RNAs in breast cancer. Javdani H, Mollaei H, Karimi F, et al. *Mol Biol Rep.* 2022 Oct; 49(1):9963-73.

790. Epithelial-mesenchymal transition and cancer stem cells: A dangerously dynamic duo in breast cancer progression. May CD, Sphyris N, Evans KW, et al. *Breast Cancer Res.* 2011 Feb; 13(1):202.

791. Loss of function of BRCA1 promotes EMT in mammary tumors through activation of TGFβR2 signaling pathway. Bai F, Wang C, Liu X, et al. *Cell Death Dis.* 2022 Mar; 13(3):195.

792. BRCA1 suppresses epithelial-to-mesenchymal transition and stem cell dedifferentiation during mammary and tumor development. Bai F, Chan HL, Scott A, et al. *Cancer Res.* 2014 Nov; 74(21): 616-72.

793. Regulation of epithelial to mesenchymal transition by BRCA1 in breast cancer. Sengodan SK, K H S, Nadhan R, et al. *Crit Rev Oncol Hematol.* 2018 Mar; 123:74-82.

Thorp KE. Energy Dynamics in Breast Cancer: Fields, Hormones & the Menstrual Cycle. G Med Sci. 2024; 5(1):169-249.
https://www.doi.org/10.46766/thegms.oncol.24111101

794. Cancer-associated stemness and epithelial-to-mesenchymal transition signatures related breast invasive carcinoma prognostic. Groza IM, Braicu C, Jurj A, et al. *Cancers (Basel).* 2020 Oct; 12(10):3053.

795. Oncostatic activities of melatonin: Roles in cell cycle, apoptosis, and autophagy. Targhazeh N, Reiter RJ, Rahimi M, et al. *Biochimie.* 2022 Nov; 202:34-48.

796. Melatonin, an inhibitory agent in breast cancer. Nooshinfar E, Safaroghli-Azar A, Bashash D, et al. *Breast Cancer.* 2017 Jan; 24(1):42-51.

797. Inhibitory effects of melatonin on breast cancer. Jiang Z, Ma F. *Zhong Nan Da Xue Xue Bao Yi Xue Ban.* 2017 Mar; 42(3):346-51.

798. Melatonin and mammary pathological growth. Cos S, Sánchez-Barceló EJ. *Front Neuroendocrinol.* 2000 Apr; 21(2):133-70.

799. Melatonin: A potential therapeutic option for breast cancer. Kong X, Gao R, Wang Z, et al. *Trends Endocrinol Metab.* 2020 Nov; 31(11):859-71.

800. Melatonin: An inhibitor of breast cancer. Hill SM, Belancio VP, Dauchy RT, et al. *Endocrinol Relat Cancer.* 2015 Jun; 22(3):R183-204.

801. Melatonin: An anti-tumor agent in hormone-dependent cancers. Menéndez-Menéndez J, Martínez-Campa C. *Int J Endocrinol.* 2018 Oct; 2018;3271948.

802. Melatonin-estrogen interactions in breast cancer. Sánchez-Barceló EJ, Cos S, Mediavilla D, et al. *J Pineal Res.* 2005 May; 38(4):217-22.

803. Interactions between pineal, hypothalamus, and pituitary involving melaton in, MSH release-inhibiting factor and MSH. Kastin AJ, Viosca S, Nair RM, et al. *Endocrinology.* 1972 Nov; 91(5):1323-28.

804. The control of reproductive physiology and behavior by gonadotropin-inhibitory hormone. Ubuka T, McGuire NL, Calisi RM, et al. *Integr Comp Biol.* 2008 Nov; 48(5):560-69.

805. Melatonin stimulates the release of gonadotropin-inhibitory hormone by the avian hypothalamus. Chowdhury VS, Yamamoto K, Ubuka T, et al. *Endocrinology.* 2010 Jan; 151(1):271-80.

806. Melatonin and the human gonadotropin-releasing hormone pulse generator. Silman R. *J Endocrinol.* 1991 Jan; 128(1):7-11.

807. Melatonin inhibits hypothalamic gonadotropin-releasing hormone release and reduces biliary hyperplasia and fibrosis in cholestatic rats. McMillin M, DeMorrow S, Glaser S, et al. *Am J Physiol Gastrointest Liver Physiol.* 2017 Nov; 313(5):G410-418.

808. Melatonin as an oncostatic molecule based on its anti-aromatase role in breast cancer. Jin Y, Choi YJ, Heo K, et al. *Int J Mol Sci.* 2021 Jan; 22(1):438.

809. Melatonin as a selective estrogen enzyme modulator. Cos S, González A, Martínez-Campa C, et al. *Curr Cancer Drug Targets.* 2008 Dec; 8(8):691-702.

810. Selective estrogen enzyme modulator actions of melatonin in human breast cancer cells. Gonzalez A, Cos S, Martinez-Campa C, et al. *J Pineal Res.* 2008 Aug; 45(1):86-92.

811. In vivo inhibition of the estrogen sulfatase enzyme and growth of DMBA-induced mammary tumors by melatonin. González A, Alvarez-García V, Martínez-Campa C, et al. *Curr Cancer Drug Targets.* 2010 May; 10(3):279-86.

812. Modulation of estrogen receptor mRNA expression by melatonin in MCF-7 human breast cancer cells. Molis TM, Spriggs LL, Hill SM. *Mol Endocrinol.* 1994 Dec; 8(12):1681-90.

Thorp KE. Energy Dynamics in Breast Cancer: Fields, Hormones & the Menstrual Cycle. G Med Sci. 2024; 5(1):169-249.
https://www.doi.org/10.46766/thegms.oncol.24111101

813. Melatonin modulates aromatase activity in MCF-7 human breast cancer cells. Cos S, Martínez-Campa C, Mediavilla MD, et al. *J Pineal Res.* 2005 Mar; 38(2):136-42.

814. Melatonin suppresses aromatase expression and activity in breast cancer associated fibroblasts. Knower KC, To SQ, Takagi K, et al. *Breast Cancer Res Treat.* 2012 Apr; 132(2): 765-71.

815. Melatonin modulation of estrogen-regulated proteins, growth factors, and proto-oncogenes In human breast cancer. Molis TM, Spriggs LL, Jupiter Y, et al. *J Pineal Res.* 1995 Mar; 18(2):93-103.

816. Estrogen-signaling pathway: A link between breast cancer and melatonin oncostatic actions. Cos S, González A, Martínez-Campa C, et al. *Cancer Detect Prevent.* 2006; 30(2):118-28.

817. Active cell death induced by the anti-estrogens tamoxifen and ICI 164 384 in human mammary carcinoma cells (MCF-7) in culture: The role of autophagy. Bursch W, Ellinger A, Kienzl H, et al. *Carcinogenesis.* 1996 Aug; 17(8):1595-607.

818. Influence of estrogen, antiestrogen and UV-light on the balance between proliferation and apoptosis in MCF-7 breast adenocarcinoma cells culture. Swiatecka J, Dziecioł J, Anchim T, et al. *Neoplasma.* 2000; 47(1):15-24.

819. Antiestrogens, aromatase inhibitors, and apoptosis in breast cancer. Riggins RB, Bouton AH, Liu MC, et al. *Vitam Horm.* 2005; 71;201-37.

820. Antiestrogens are pro-apoptotic in human breast epithelial cells. Somaï S, Chaouat M, Jacob D, Perrot JY, et al. *Int J Cancer.* 2003 Jul; 105(5):607-12.

821. The pure antiestrogen ICI 182780 is more effective in the induction of apoptosis and down regulation of BCL-2 than tamoxifen in MCF-7 cells. Diel P, Smolnikar K, Michna H. *Breast Cancer Res Treat.* 1999 Nov; 58(2):87-97.

822. Plasma vitamin D levels, menopause, and risk of breast cancer: Dose-response meta-analysis of protective studies. Bauer SR, Hankinson SE, Bertone-Johnson ER, et al. *Medicine (Baltimore).* 2013 May; 92(3):123-31.

823. High prevalence of vitamin D deficiency in women with breast cancer: The first Chilean study. Acevedo F, Pérez V, Pérez-Sepúlveda A, et al. *Breast.* 2016 Oct; 29:39-43.

824. Association between plasma 25-hydroxyvitamin D and breast cancer risk. Crew KD, Gammon MD, Steck SE, et al. *Cancer Prev Res (Phila).* 2009 Jun; 2(6):598-604.

825. Vitamin D baseline levels at diagnosis of breast cancer: A systematic review and meta-analysis. Voutsadakis IA. *Hematol Oncol Stem Cell Ther.* 2021; 14(1):15-26.

826. The role of vitamin D and its molecular bases in insulin resistance, diabetes, metabolic syndrome, and cardiovascular disease: State of the art. Argano C, Mirarchi L, Amodeo S, et al. *Int J Mol Sci.* 2023 Oct; 24(20):15485.

827. Associations of vitamin D with insulin resistance, obesity, type 2 diabetes, and metabolic syndrome. Wimalawansa SJ. *J Steroid Biochem Mol.* 2018 Jan; 175:177-89.

828. The interplay between vitamin D status, subclinical inflammation, and prediabetes. Arabi A, Nasrallah D, Mohsen S. *Heliyon.* 2024 Aug; 10(15):e35764.

829. Mechanisms involved in the relationship between vitamin D and insulin resistance; impact on clinical practice. Contreras-Bolívar V, García-Fontana B, García-Fontana C, et al. *Nutrients.* 2021 Oct; 13(10):3491.

830. Vitamin D levels in newly diagnosed breast cancer patients according to tumor sub-types. Rosso C, Fera N, Murugan NJ, et al. *J Diet Suppl.* 2023:20(6):926-38

Thorp KE. Energy Dynamics in Breast Cancer: Fields, Hormones & the Menstrual Cycle. G Med Sci. 2024; 5(1):169-249.
https://www.doi.org/10.46766/thegms.oncol.24111101

831. The vitamin D receptor as a prognostic marker in breast cancer: A cohort study. Huss L, Gulz-Haake I, Nilsson E, et al. *Nutrients.* 2024 Mar; 16(7):931.

832. Vitamin D deficiency is associated with poor breast cancer prognostic features in postmenopausal women. de Sousa Almeida-Filho B, De Luca Vespoli H, Pessoa EC, et al. *J Steroid Biochem Mol Biol.* 2017 Nov; 174:284-89.

833. Vitamin D baseline levels at diagnosis of breast cancer: A systematic review and meta-analysis. Voutsadakis IA. *Hematol Oncol Stem Cell Ther.* 2021 Mar; 14(1):16-26.

834. Association between plasma 25-hydroxyvitamin D and breast cancer risk. Crew KD, Gammon MD, Steck SE, et al. *Cancer Prev Res (Phila).* 2009 Jun; 2(6):598-604.

835. Plasma vitamin D levels, menopause, and risk of breast cancer: Dose-response meta-analysis of prospective studies. Bauer SR, Hankinson SE, Bertone-Johnson ER, et al. Medicine (Baltimore). 2013 May; 92(3):123-131.

836. Mammographic density and vitamin D levels: A cross-sectional study. Straub L, Riedel J, Luppa PB, et al. *Gebertshilfe Frauenheilkd.* 2017 Mar; 77(3):257-67.

837. Relationship of vitamin D deficiency with mammographic breast density and triple-negative breast cancer: A cross-sectional study. Singh CK, Thomas S, Goswami B, et al. *Natl Med J India.* 2021 Sep; 34(5):271-75.

838. Premenopausal plasma 25-hydroxyvitamin D, mammographic density, and risk of breast cancer. Bertrand KA, Rosner B, Eliassen AH, et al. *Breast Cancer Res Treat.* 2015 Jan; 149(2):479-87.

839. Serum 25-hydroxyvitamin D and mammographic density in premenopausal Spanish women. Lope V, Toribio MJ, Pérez-Gómez B, et al. *J Steroid Biochem Mol Biol.* 2019 May; 189:101-07.

840. Synchronized seasonal variations of mammographic breast density and plasma 25-hydroxyvitamin D. Brisson J, Bérubé S, Diorio C, et al. *Cancer Epidemiol Biomarkers Prev.* 2007 May; 16(5):929-33.

841. Vitamin D and breast cancer: Inhibition of estrogen synthesis and signaling. Krishnan AV, Swami S, Feldman D. *J Steroid Biochem Mol Biol.* 2010 Jul; 121(1-2):343-48.

842. The role of vitamin D3 and antiestrogens in modulating apoptosis of breast cancer cells and tumors. Welsh J, VanWeelden K, Flanagan L, et al. *Subcell Biochem.* 1998; 30:245-70.

843. Nocturnal light pollution and underexposure to daytime sunlight: Complementary mechanisms of circadian disruption and related diseases. Smolensky MH, Sackett-Lundeen LL, Portaluppi F. *Chronobiol Int.* 2015; 32(8):1029-48.

844. The endocrinology of sunlight and darkness. Complementary roles of vitamin D and pineal hormones. Stumpf WE. *Naturwissenschaften.* 1988 May; 75(5):247-51.

845. Is Melatonin the "Next Vitamin D"?: A Review of Emerging Science, Clinical Uses, Safety, and Dietary Supplements. Minich DM, Henning M, Darley C, et al. *Nutrients.* 2022 Sep 22; 14(19):3934.

846. Incidence of cancer among Finnish airline cabin attendants, 1967-92. Pukkala E, Auvinen A, Wahlberg G. *BMJ.* 1995 Sep; 311(7006):649-52.

847. Risk of breast cancer is also increased among Danish female airline cabin attendants. Lynge E. *BMJ.* 1996 Jan; 312(7025):253.

848. Risk of breast cancer is also increased among retired US female airline cabin attendants. Wartenberg D, Stapleton CP. *BMJ.* 1998 Jun; 316(7148):1902.

Thorp KE. Energy Dynamics in Breast Cancer: Fields, Hormones & the Menstrual Cycle. G Med Sci. 2024; 5(1):169-249.
https://www.doi.org/10.46766/thegms.oncol.24111101

849. Risk of breast cancer in female flight attendants: A population-based study (Iceland). Rafnsson V, Tulinius H, Jónasson JG, Hrafnkelsson J. *Cancer Causes Control.* 2001 Feb; 12(2):95-101.

850. Breast cancer risk among Finnish cabin attendants: A nested case-control study. Kojo K, Pukkala E, Auvinen A. *Occup Environ Med.* 2005 Jul; 62(7):488-93.

851. Breast cancer risk in flight attendants: An update. Salhab M, Mokbel K. *Int J Fertil Womens Med.* 2006 Sep-Oct; 51(5):205-7.

852. Breast cancer in female flight attendants. Mawson AR. *Lancet.* 1998 Aug; 352(9128):626.

853. Flight attendants, breast cancer, and melatonin. Barker ME, Stookey JD. *Lancet.* 1998 Oct; 352(9137):1389.

854. Flight attendants, breast cancer, and melatonin. Gurwitz D. *Lancet.* 1998 Oct; 352(9137):1389-90.

855. Risk of breast cancer among female airline cabin attendants. Large European studies are now being carried out. Auvinen A, Pukkala E, Blettner M. *BMJ.* 1999 Jan; 318(7176):125-26.

856. Breast cancer risk in airline cabin attendants: A nested case-control study in Iceland. Rafnsson V, Sulem P, Tulinius H, et al. *Occup Environ Med.* 2003 Nov; 60(11):807-9.

857. Breast cancer incidence in a cohort of U.S. flight attendants. Schubauer-Berigan MK, Anderson JL, Hein MJ, et al. *Am J Ind Med.* 2015 Mar; 58(3):252-66.

858. The effect of four-day round trip flights over 10 time zones on the circadian variation of salivary melatonin and cortisol in airline flight attendants. Härmä M, Laitinen J, Partinen M, et al. *Ergonomics.* 1994 Sep; 37(9):1479-89.

859. The effect of four-day round trip flights over 10 time zones on the sleep-wakefulness patterns of airline flight attendants. Härmä M, Suvanto S, Partinen M. *Ergonomics* 1994 Sep; 37(9):1461-78.

860. Effects of 10 h time zone changes on female flight attendants' circadian rhythms of body temperature, alertness, and visual search. Suvanto S, Härmä M, Ilmarinen J, et al. *Ergonomics.* 1993 Jun; 36(6):613-25.

861. Effect of flying on the menstrual function of air hostesses. Cameron RG. *Aerospace Med.* 1969 Sep; 40(9):1020-23.

862. Disorders of the menstrual cycle in airline stewardesses. Iglesias R, Terrés A, Chavarria A. *Aviat Space Environmental Med.* 1980 May; 51(5):518-20.

863. Incidence of breast cancer in Norwegian female radio and telegraph operators. Tynes T, Hannevik M, Andersen A, et al. *Cancer Causes Control.* 1996 Mar; 7(2):197-204.

864. Rotating night shifts and risk of breast cancer in women participating in the nurses' health study. Schernhammer ES, Laden F, Speizer FE, et al. *J Natl Cancer Institute.* 2001 Oct; 93(20):1563-68.

865. Shift Work and Breast Cancer. Gehlert S, Clanton M, On Behalf Of The Shift Work And Breast Cancer Strategic Advisory Group. *Int J Environ Res Public Health.* 2020 Dec 20; 17(24):9544.

866. Night Shift Work and Risk of Breast Cancer. Hansen J. *Curr Environ Health Rep.* 2017 Sep; 4(3):325-39.

867. Rotating Night-Shift Work and the Risk of Breast Cancer in the Nurses' Health Studies. Wegrzyn LR, Tamimi RM, Rosner BA, et al. *Am J Epidemiol.* 2017 Sep 1; 186(5):532-540.

868. Night-shift work, breast cancer incidence, and all-cause mortality: An updated meta-analysis of prospective cohort studies. Wei F, Chen W, Lin X. *Sleep Breath.* 2022 Dec; 26(4):1509-1526.

Thorp KE. Energy Dynamics in Breast Cancer: Fields, Hormones & the Menstrual Cycle. G Med Sci. 2024; 5(1):169-249.
https://www.doi.org/10.46766/thegms.oncol.24111101

869. Night-shift work increases morbidity of breast cancer and all-cause mortality: A meta-analysis of 16 prospective cohort studies. Lin X, Chen W, Wei F, et al. *Sleep Med.* 2015 Nov; 16(11):1381-1387.

870. Night-shift work duration and breast cancer risk: An updated systematic review and meta-analysis. Manouchehri E, Taghipour A, Ghavami V, et al. *BMC Womens Health.* 2021 Mar; 21(1):89.

871. Shift work and chronic disease: The epidemiological evidence. Wang XS, Armstrong ME, Cairns BJ, et al. *Occup Med (Lond).* 2011 Mar; 61(2):78-89.

872. Health consequences of shift work and insufficient sleep. Kecklund G, Axelsson J. *BMJ.* 2016 Nov; 355:5210.

873. Shift work and cancer risk: Potential mechanistic roles of circadian disruption, light at night, and sleep deprivation. Haus EL, Smolensky MH. *Sleep Med Rev.* 2013 Aug; 17(4):273-84.

874. Shift work and the risk of cardiovascular disease. A systematic review and meta-analysis including dose-response relationship. Torquati L, Mielke GI, Brown WJ, et al. Scand J Work Environ Health. 2018 May; 44(3):229-38.

875. Circadian disruption, shift work and the risk of cancer: A summary of the evidence and studies in Seattle. Davis S, Mirick DK. *Cancer Causes Control.* 2006 May; 17(4):539-45.

876. NTP Cancer Hazard Assessment Report on Night Shift Work and Light at Night. National Toxicology Program. Research Triangle Park (NC): National Toxicology Program; 2021 Apr.

877. Night Shift Work Associates with All-Cause and Cause-Specific Mortality: A Large Prospective Cohort Study. Chang Q, Zhu Y, Liang H, et al. *J Gen Intern Med.* 2024 Sep 10.

878. Long-term night shift work is associated with the risk of atrial fibrillation and coronary heart disease. Wang N, Sun Y, Zhang H, et al. *Eur Heart J.* 2021 Oct 21; 42(40):4180-88.

879. Night shift work and myocardial infarction in the UK Biobank. Yang MJ, Jia ZW, Wang E, et al. *Occup Med (Lond).* 2024 Sep 23; 74(6):409-416.

880. Poor sleep and shift work associate with increased blood pressure and inflammation in UK Biobank participants. Kanki M, Nath AP, Xiang R, et al. Nat Commun. 2023 Nov 4; 14(1):7096.

881. Disruption of daily rhythms in gene expression: The importance of being synchronised. Hughes AT, Piggins HD. *Bioessays.* 2014 Jul; 36(7):644-48.

882. Sleep interruption associated with house staff work schedules alters circadian gene expression. Fang MZ, Ohman-Strickland P, Kelly-McNeil K, et al. *Sleep Med.* 2015 Nov; 16(11):1388-94.

883. Shift Work Disrupts Circadian Regulation of the Transcriptome in Hospital Nurses. Resuehr D, Wu G, Johnson RL, et al. *J Biol Rhythms.* 2019 Apr; 34(2):167-177.

884. Night shift schedule causes circadian dysregulation of DNA repair genes and elevated DNA damage in humans. Koritala BSC, Porter KI, Arshad OA, Gajula RP, et al. *J Pineal Res.* 2021 Apr; 70(3):e12726.

885. Twenty-four-hour melatonin and core body temperature rhythms: Their adaptation in night workers. Weibel L, Spiegel K, Gronfier C, et al. *Am J Physiol.* 1997 Mar; 272(3 Pt 2):R948-54.

886. Different patterns of light exposure in relation to melatonin and cortisol rhythms and sleep of night workers. Koller M, Härma M, Laitinen JT, et al. *J Pineal Res.* 1994 Apr; 16(3):127-35.

887. Disruption of central and peripheral circadian clocks and circadian controlled estrogen receptor rhythms in night shift nurses in working environments. Yang Z, Black K, Ohman-Strickland P, et al. *FASEB J.* 2024 Jun; 38(11):e23719.

Thorp KE. Energy Dynamics in Breast Cancer: Fields, Hormones & the Menstrual Cycle. G Med Sci. 2024; 5(1):169-249.
https://www.doi.org/10.46766/thegms.oncol.24111101

888. Disruption of central and peripheral circadian clocks in police officers working at night. Koshy A, Cuesta M, Boudreau P, et al. *FASEB J.* 2019 Jun; 33(6):6789-6800.

889. Disturbance of the Circadian System in Shift Work and Its Health Impact. Boivin DB, Boudreau P, Kosmadopoulos AJ. *Biol Rhythms.* 2022 Feb; 37(1):3-28.

890. Circadian Disruption as a Risk Factor for Development of Cardiovascular and Metabolic Disorders - From Animal Models to Human Population. Sumová A, Sládek M. *Physiol Res.* 2024 Apr; 73(Suppl 1):S321-34.

891. Circadian rhythm disorganization produces profound cardiovascular and renal disease in hamsters. Martino TA, Oudit GY, Herzenberg AM, et al. *Am J Physiol Regul Integr Comp Physiol.* 2008 May; 294(5):R1675-83.

892. Effects of night shift on plasma concentrations of melatonin, LH, FSH and prolactin, and menstrual irregularity. Miyauchi F, Nanjo K, Otsuka K. *Sangyo Igaku.* 1992 Nov; 34(6):545-50.

893. Bright-light exposure during daytime sleeping affects nocturnal melatonin secretion after simulated night work. Nagashima S, Osawa M, Matsuyama H, et al. *Chronobiol Int.* 2018 Feb; 35(2):229-39.

894. Internal desynchronization of circadian rhythms and tolerance to shift work. Reinberg A, Ashkenazi I. *Chronobiol Int.* 2008 Jul; 25(4):625-43.

895. Amplitude of the oral temperature circadian rhythm and the tolerance to shift-work. Andlauer P, Reinberg A, Fourré L, et al. *J Physiol (Paris).* 1979; 75(5):507-12.

896. Desynchronization of the oral temperature circadian rhythm and intolerance to shift work. Reinberg A, Andlauer P, De Prins J, et al. *Nature.* 1984 Mar; 308(5956):272-74.

897. Alteration of period and amplitude of circadian rhythms in shift workers. With special reference to temperature, right and left hand grip strength. Reinberg A, Motohashi Y, Bourdeleau P, et al. *Eur J Appl Physiol Occup Physiol.* 1988; 57(1):15-25.

898. Misaligned core body temperature rhythms impact cognitive performance of hospital shift work nurses. Molzof HE, Prapanjaroensin A, Patel VH, et al. *Neurobiol Learn Mem.* 2019 Apr; 160:151-59.

899. Association between light at night, melatonin secretion, sleep deprivation, and the internal clock: Health impacts and mechanisms of circadian disruption. Touitou Y, Reinberg A, Touitou D. *Life Sci.* 2017 Mar; 173:94-106.

900. Artificial Light at Night and Type 2 Diabetes Mellitus. Baek JH, Zhu Y, Jackson CL, et al. *Diabetes Metab J.* 2024 Sep; 48(5):847-63.

901. Artificial Light at Night and Cancer: Global Study. Al-Naggar RA, Anil SH. *Asian Pac J Cancer Prev.* 2016 Oct; 17(10):4661-64.

902. Evaluating the Association between Artificial Light-at-Night Exposure and Breast and Prostate Cancer Risk in Spain (MCC-Spain Study). Garcia-Saenz A, Sánchez de Miguel A, Espinosa A, et al. *Environ Health Perspect.* 2018 Apr 23; 126(4):047011.

903. Exposure to Light at Night and Risk of Cancer: A Systematic Review, Meta-Analysis, and Data Synthesis. Ma S, Alsabawi Y, El-Serag HB, et al. *Cancers (Basel).* 2024 Jul 26; 16(15):2653.

904. Artificial light at night: Melatonin as a mediator between the environment and epigenome. Haim A, Zubidat AE. *Philos Trans R Soc Lond B Biol Sci.* 2015 May; 370(1667):20140121.

905. Exposure to artificial light at night: A common link for obesity and cancer? Muscogiuri G, Poggiogalle E, Barrea L, et al. *Eur J Cancer.* 2022 Sep; 173:263-75.

Thorp KE. Energy Dynamics in Breast Cancer: Fields, Hormones & the Menstrual Cycle. G Med Sci. 2024; 5(1):169-249.
https://www.doi.org/10.46766/thegms.oncol.24111101

906. Light at night and breast cancer risk among California teachers. Hurley S, Goldberg D, Nelson D, et al. *Epidemiology.* 2014 Sep; 25(5):697-706.

907. Light at night and lung cancer risk: A worldwide interdisciplinary and time-series study. Wang R, Wang Q, Li J. *Chin Med J Pulm Crit Care Med.* 2024 Mar 15; 2(1):56-62.

908. Light at night disrupts biological clocks, calendars, and immune function. Walker WH, Bumgarner JR, Becker-Krail. *Semin Immunopathol.* 2022 Mar; 44(2):165-73.

909. Light Pollution and Cancer. Walker WH, Bumgarner JR, Walton JC, et al. *Int J Mol Sci.* 2020 Dec 8; 21(24):9360.

910. Light pollution as a factor in breast and prostate cancer. Lamphar H, Kocifaj M, Limón-Romero J, et al. *Sci Total Environ.* 2022 Feb 1; 806(Pt 4):150918.

911. Artificial light-at-night - a novel lifestyle risk factor for metabolic disorder and cancer morbidity. Zubidat AE, Haim AJ. *Basic Clin Physiol Pharmacol.* 2017 Jul 26; 28(4):295-313.

912. Association Between Outdoor Light-at-night Exposure and Colorectal Cancer in Spain. Garcia-Saenz A, de Miguel AS, Espinosa A, *et al. Epidemiology.* 2020 Sep; 31(5):718-27.

913. Light at night pollution of the internal clock, a public health issue. Touitou Y. *Bull Acad Natl Med.* 2015 Oct; 199(7):1081-1098.

914. Lighting for the human circadian clock: Recent research indicates that lighting has become a public health issue. Pauley SM. *Med Hypotheses.* 2004; 63(4):588-96.

915. Exposure to Artificial Light at Night and the Consequences for Flora, Fauna, and Ecosystems. Falcón J, Torriglia A, Attia D, et al. *Front Neurosci.* 2020 Nov; 14:602796.

916. Physiological significance of the rhythmic secretion of hypothalamic and pituitary hormones. Gan EH, Quinton R. *Prog Brain Res.* 2010; 181:111-26.

917. Modeling robust oscillatory behavior of the hypothalamic-pituitary-adrenal axis. Kyrylov V, Severyanova LA, Vieira A. *IEEE Trans Biomed Eng.* 2005 Dec; 52(12):1977-83.

918. Regulation of complex pulsatile and rhythmic neuroendocrine systems: The male gonadal axis as a prototype. Veldhuis JD, Keenan DM, Pincus SM. Prog Brain Res. 2010; 181:79-110.

919. The aging male hypothalamic-pituitary-gonadal axis: Pulsatility and feedback. Veldhuis JD, Keenan DM, Liu PY, et al. *Mol Cell Endocrinol.* 2009 Feb 5; 299(1):14-22.

920. Chronobiology in endocrinology. Sensi S, Pace Palitti V, et al. *Ann Ist Super Sanita.* 1993; 29(4):613-31.

921. Pulsatility of Hypothalamo-Pituitary Hormones: A Challenge in Quantification. Keenan DM, Veldhuis JD. *Physiology (Bethesda).* 2016 Jan; 31(1):34-50.

922. Chronobiology in the endocrine system. Haus E. *Adv Drug Deliv Rev.* 2007 Aug; 59(910):985-1014.

923. Pulsatile secretion of LH, FSH, prolactin, oestradiol and progesterone during the human menstrual cycle. Bäckström CT, McNeilly AS, Leask RM, et al. Clin Endocrinol (Oxf). 1982 Jul; 17(1): 29-42.

924. The circadian pattern of melatonin and its positive relationship with progesterone in women. Webley GE, Leidenberger F. *J Clin Endocrinol Metab.* 1986 Aug; 63(2):323-28.

925. Episodic nyctohemeral secretion of melatonin in adult humans: Lack of relation with LH pulsatile pattern. de Leiva A, Tortosa F, Peinado MA, et al. *Acta Endocrinol (Copenh).* 1990 Jan; 122(1):76-82.

Thorp KE. Energy Dynamics in Breast Cancer: Fields, Hormones & the Menstrual Cycle. G Med Sci. 2024; 5(1):169-249.
https://www.doi.org/10.46766/thegms.oncol.24111101

926. Amplification of pulsatile LH secretion by exogenous melatonin in women. Cagnacci A, Elliott JA, Yen SS. *J Clin Endocrinol Metab.* 1991 Jul; 73(1):210-12.

927. Gonadotropin regulation by pulsatile GnRH: Signaling and gene expression. Stamatiades GA, Kaiser UB. Mol Cell Endocrinol. 2018 Mar; 463:131-41.

928. The Gonadotropin-Releasing Hormone Pulse Generator. Herbison AE. Endocrinology. 2018 Nov; 159(11):3723-36.

929. Biologically active luteinizing hormone is secreted in episodic pulsations that vary in relation to stage of the menstrual cycle. Veldhuis JD, Beitins IZ, Johnson ML, et al. *J Clin Endocrinol Metab.* 1984; 58:1050–1058.

930. Pulsatile GnRH stimulates normal cyclic ovarian function in amenorrheic lactating postpartum women. Zinaman MJ, Cartledge T, Tomai T, et al. *J Clin Endocrinol Metab.* 1995 Jul; 80(7):2088-93.

931. Longitudinal evaluation of the luteal phase and its transition into the follicular phase. Vermesh M, Kletzky OA. *J Clin Endocrinol Metab.* 1987 Oct; 65(4):653-58.

932. Mathematical modeling of gonadotropin-releasing hormone signaling. Pratap A, Garner KL, Voliotis M, et al. *Mol Cell Endocrinol.* 2017 Jul; 449:42-55.

933. Altered luteinizing hormone pulse frequency in early follicular phase of the menstrual cycle with luteal phase defect patients in women. Suh BY, Betz G.Fertil Steril. 1993 Nov; 60(5):800-5.

934. Abnormal patterns of pulsatile luteinizing hormone in women with luteal phase deficiency. Soules MR, Steiner RA, Clifton DK, et al. *Obstet Gynecol.* 1984 May; 63(5):626-29.

935. Reduction of gonadotropin-releasing hormone pulse frequency is associated with subsequent selective follicle-stimulating hormone secretion in women with polycystic ovarian disease. Christman GM, Randolph JF, Kelch RP, et al. *J Clin Endocrinol Metab.* 1991 Jun; 72(6):1278-85.

936. Melatonin enhances the luteinizing hormone and follicle-stimulating hormone responses to gonadotropin-releasing hormone in the follicular, but not in the luteal, menstrual phase. Cagnacci A, Paoletti AM, Soldani R, et al. J Clin Endocrinol Metab. 1995 Apr; 80(4):1095-99.

937. Transition from dim to bright light in the morning induces an immediate elevation of cortisol levels. Leproult R, Colecchia EF, L'Hermite-Balériaux M, et al. J Clin Endocrinol Metab. 2001 Jan; 86(1):151-7.

938. Immediate effect of blue-enhanced light on reproductive hormones in women. Danilenko KV, Sergeeva OY. *Neuro Endocrinol Lett.* 2015; 36(1):84-90.

939. The effects of light exposure on plasma concentrations of melatonin, LH, FSH and prolactin in women. Miyauchi F, Nanjo K, Kato H, et al. *Nihon Naibunpi Gakkai Zasshi.* 1990 Aug; 66(8):737-46.

940. Characterizing the temporal Dynamics of Melatonin and Cortisol Changes in Response to Nocturnal Light Exposure. Rahman SA, Wright KP Jr, Lockley SW, et al. *Sci Rep* 2019 Dec; 9(1):19720.

941. Modulation of human ovarian function by melatonin. Rai S, Ghosh H. *Front Biosci (Elite Ed).* 2021 Jan 1; 13(1):140-57.

942. Influence of melatonin and photoperiod on animal and human reproduction. Cagnacci A, Volpe A. J Endocrinol Invest. 1996 Jun; 19(6):382-411.

943. Melatonin regulates ovarian function: An update. Zhang L, Liang YJ. *Zhonghua Nan Ke Xue.* 2014 Jun; 20(6):548-53.

944. From Implantation to Birth: Insight into Molecular Melatonin Functions. Carlomagno G, Minini M, Tilotta M, et al. *Int J Mol Sci.* 2018 Sep; 19(9):2802.

Thorp KE. Energy Dynamics in Breast Cancer: Fields, Hormones & the Menstrual Cycle. G Med Sci. 2024; 5(1):169-249.
https://www.doi.org/10.46766/thegms.oncol.24111101

945. Melatonin and Female Reproduction: An Expanding Universe. Olcese JM. *Front Endocrinol (Lausanne).* 2020 Mar 6; 11:85.

946. Role of Melatonin in Ovarian Function. Basini G, Grasselli F. *Animals (Basel).* 2024 Feb; 14(4):644.

947. Roles of melatonin in thee teleost ovary: A review of the current status. Takahashi T, Ogiwara K. *Comp Biochem Physiol A Mol Integr Physiol.* 2021 Apr; 254:110907.

948. Melatonin and the ovary: Physiological and pathophysiological implications. Tamura H, Nakamura Y, Korkmaz A, et al. *Fertil Steril.* 2009 Jul; 92(1):328-43.

949. Effects of melatonin on development and hormone secretion of sheep theca cells in vitro. Ma J, Wang J, Hu S, et al. *Theriogenology.* 2023 Mar; 198:172-182.

950. Effects of melatonin on the in vitro growth of early antral follicles and maturation of ovine oocytes. Barros VRP, Monte APO, Santos JMS, et al. *Domest Anim Endocrinol.* 2020 Apr; 71:106386.

951. Melatonin effects on ovarian follicular cells: A systematic review. Minguini IP, Luquetti CM, Baracat MCP, et al. *Rev Assoc Med Bras.* 2019 Sep; 65(8):1122-27.

952. Melatonin regulates the ovarian function and enhances follicle growth in aging laying hens via activating the mammalian target of rapamycin pathway. Hao EY, Chen H, Wang DH, et al. *Poult Sci.* 2020 Apr; 99(4):2185-95.

953. Effects of melatonin on morphology and development of primordial follicles during in vitroculture of bovine ovarian tissue. Cavalcante BN, Matos-Brito BG, Paulino LRFM, et al. *Reprod Domest Anim.* 2019 Dec; 54(12):1567-1573.

954. "Hormone of darkness" and human reproductive process: Direct regulatory role ofmelatonin in human corpus luteum. Scarinci E, Tropea A, Notaristefano G, et al. *J Endocrinol Invest.* 2019 Oct; 42(10):1191-97.

955. Melatonin stimulates the secretion of progesterone along with the expression of cholesterol side-chain cleavage enzyme (P450scc) and steroidogenic acute regulatory protein (StAR) in corpus luteum of pregnant sows. Zhang W, Wang Z, Zhang L, et al. *Theriogenology.* 2018 Mar; 108:297-305.

956) Protective role of melatonin in progesterone production by human luteal cells. Taketani T, Tamura H, Takasaki A, et al. *J Pineal Res.* 2011 Sep; 51(2):207-13.

957. Melatonin deprival modifies follicular and corpus luteal growth dynamics in a sheep model. Manca ME, Manunta ML, Spezzigu A, et al. *Reproduction.* 2014 Jun; 147(6):885-95.

958. Melatonin Stimulates STAR Expression and Progesterone Production via Activation of the PI3K/AKT Pathway in Bovine Theca Cells. Wang X, Meng K, He Y, et al. *Int J Biol Sci.* 2019 Jan; 15(2):404-15.

959. Melatonin directly stimulates the secretion of progesterone by human and bovine granulosa cells in vitro. Webley GE, Luck MR. *J Reprod Fertil.* 1986 Nov; 78(2):711-17.

960. Melatonin suppresses apoptosis and stimulates progesterone production by bovine granulosa cells via its receptors (MT1 and MT2). Wang SJ, Liu WJ, et al. *Theriogenology.* 2012 Oct; 78(7):1517-26.

961. Long-term melatonin treatment delays ovarian aging. Tamura H, Kawamoto M, Sato S, et al. *J Pineal Res.* 2017 Mar; 62(2).

962. Melatonin prevents postovulatory oocyte aging and promotes subsequent embryonic development in the pig. Wang T, Gao YY, Chen L, et al. *Aging.* 2017 Jun; 9(6):1552-64.

963. Melatonin prevents postovulatory oocyte aging in the mouse and extends the window for optimal fertilization in vitro. Lord T, Nixon B, Jones KT, et al. *Biol Reprod.* 2013 Mar; 88(3):67.

964. Endometrial angiogenesis throughout the human menstrual cycle. Maas JW, Groothuis PG, Dunselman GA, et al. *Hum Reprod.* 2001 Aug; 16(8):1557-61.

965. Epigenetic regulation of endometrium during the menstrual cycle. Munro SK, Farquhar CM, Mitchell MD, et al. *Mol Hum Reprod.* 2010 May; 16(5):297-310.

966. Recent advances in endometrial angiogenesis research. Girling JE, Rogers PA. *Angiogenesis.* 2005; 8(2):89-99.

967. Vasculogenesis and angiogenesis in the endometrium during menstrual cycle and implantation. Demir R, Yaba A, Huppertz B. *Acta Histochem.* 2010 May; 112(3):203-14.

968. Is the effect of melatonin on vascular endothelial growth factor receptor-2 associated with angiogenesis in the rat ovary? Kandemir YB, Konuk E, Katırcı E, et al. *Clinics (Sao Paulo).* 2019 Mar;74:e658.

969. Effect of Exogenous Melatonin on the Development of Mice Ovarian Follicles and Follicular Angiogenesis. Tao J, Zhang L, Zhang X, et al. *Int J Mol Sci.* 2021 Oct 19; 22(20):11262.

970. Melatonin: An atypical hormone with major functions in the regulation of angiogenesis. Mirza-Aghazadeh-Attari M, Reiter RJ, Rikhtegar R. *IUBMB Life.* 2020 Aug; 72(8):1560-84.

971. Role of melatonin in controlling angiogenesis under physiological and pathological conditions. Ma Q, Reiter RJ, Chen Y. *Angiogenesis.* 2020 May; 23(2):91-104.

972. Effect of Exogenous Melatonin on the Development of Mice Ovarian Follicles and Follicular Angiogenesis. Tao J, Zhang L, Zhang X, et al. *Int J Mol Sci.* 2021 Oct; 22(20):11262.

973. Melatonin potentially acts directly on swine ovary by modulating granulosa cell function and angiogenesis. Basini G, Bussolati S, Ciccimarra R, et al. *Reprod Fertil Dev.* 2017 Nov; 29(12):2305-12.

974. Melatonin acts through different mechanisms to control oxidative stress and primordial follicle activation and survival during in vitro culture of bovine ovarian tissue. Silva BR, Costa FC, De Lima Neto MF, et al. *Domest Anim Endocrinol.* 2024 Jan; 86:106824.

975. The Role of Melatonin as a Hormone and an Antioxidant in the Control of Fish Reproduction. Maitra SK, Hasan KN. *Front Endocrinol (Lausanne).* 2016 May 4; 7:38.

976. Melatonin attenuates postovulatory oocyte dysfunction by regulating SIRT1 expression. Yang Q, Dai S, Luo X, et al. *Reproduction.* 2018 Jul; 156(1):81-92.

977. Oxidative stress impairs oocyte quality and melatonin protects oocytes from free radical damage and improves fertilization rate. Tamura H, Takasaki A, Miwa I, et al. *J Pineal Res.* 2008 Apr; 44(3):280-87.

978. Essential actions of melatonin in protecting the ovary from oxidative damage. Cruz MH, Leal CL, Cruz JF, et al. *Theriogenology.* 2014 Oct; 82(7):925-32.

979. Melatonin as a free radical scavenger in the ovarian follicle. Tamura H, Takasaki A, Taketani T, et al. *Endocr J.* 2013; 60(1):1-13.

980. The role of melatonin as an antioxidant in the follicle. Tamura H, Takasaki A, Taketani T, et al. *J Ovarian Res.* 2012 Jan 26; 5:5.

981. Melatonin Attenuates Oxidative Stress-Induced Apoptosis of Bovine Ovarian Granulosa Cells by Promoting Mitophagy via SIRT1/FoxO1 Signaling Pathway. Xu G, Dong Y, Wang Z, et al. *Int J Mol Sci.* 2023 Aug 16; 24(16):12854.

982. Melatonin, Its Beneficial Effects on Embryogenesis from Mitigating Oxidative Stress to Regulating Gene Expression. Ivanov D, Mazzoccoli G, Anderson G, et al. *Int J Mol Sci.* 2021 May; 22(11):5885.

Thorp KE. Energy Dynamics in Breast Cancer: Fields, Hormones & the Menstrual Cycle. G Med Sci. 2024; 5(1):169-249.
https://www.doi.org/10.46766/thegms.oncol.24111101

983. Inverse seasonal relationship between melatonin and ovarian activity in humans in a region with a strong seasonal contrast in luminosity. Kauppila A, Kivela A, Pakarinen A, et al. *J Clin Endocrinol Metab.* 1987 Nov; 65(5):823–28.

984. The effect of season on the circulating concentrations of anterior pituitary, ovarian and adrenal cortex hormones and hormone binding proteins in the subarctic area; Evidence of increased activity of the pituitary-ovarian axis in spring. Kauppila A, Pakarinen A, Kirkinen P, et al. *Gynecol Endocrinol.* 1987 Jun; 1(2):137-50.

985. Temperature regulation in women: Effects of the menstrual cycle. Baker FC, Siboza F, Fuller A. *Temperature (Austin).* 2020 Mar; 7(3):226-62.

986. Progesterone resistance in women who have had breast cancer. Simpson HW, McArdle CS, Griffiths K, et al. *Br J Obstet Gynaecol.* 1998 Mar; 105(3):345-51.

987. Progestogen deficiency and endometrial cancer risk. Schindler AE. *Maturitas.* 2009 Apr 20; 62(4):334-37.

988. Impaired ovulation and breast cancer risk. Stoll BA. *Eur J Cancer.* 1997 Sep; 33(10):1532-35.

989. Luteal phase deficiency: abnormal gonadotropin and progesterone secretion patterns. Soules MR, Clifton DK, Cohen NL, et al. *J Clin Endocrinol Metab.* 1989 Oct; 69(4):813-20.

990. Luteal phase deficiency: Characterization of reproductive hormones over the menstrual cycle. Soules MR, McLachlan RI, Ek M, et al. *J Clin Endocrinol Metab.* 1989 Oct; 69(4):804-12.

991. Corpus luteum dysfunction and the epidemiology of breast cancer: A reconsideration. Sherman BM, Wallace RB, Korenman SG. *Breast Cancer Res Treat.* 1981; 1(4):287-96.

992. In Defense of Progesterone: A Review of the Literature. Lieberman A, Curtis L. *Altern Ther Health Med.* 2017 Nov; 23(6):24-32.

993. Estradiol/progesterone interaction in normal and pathologic breast cells. Mauvais-Jarvis P, Kuttenn F, Gompel A. *Ann NY Acad Sci.* 1986; 464:152-67.

994. Progesterone and breast. Lamb CA, Fabris VT, Lanari C. *Best Pract Res Clin Obstet Gynaecol.* 2020 Nov; 69:85-94.

995. Progesterone and Breast Cancer. Trabert B, Sherman ME, Kannan N, et al. *Endocr Rev.* 2019 May; 41(2):320-44.

996. Progesterone and Breast Cancer: An NCI Workshop Report. Sathyamoorthy N, Lange CA. *Horm Cancer.* 2020 Feb; 11(1):1-12.

997. Inadequate corpus luteum function: A pathophysiological interpretation of human breast cancer epidemiology. Sherman BM, Korenman SG. *Cancer.* 1974 May; 33(5):1306-12.

998. Breast cancer incidence in women with a history of progesterone deficiency. Cowan LD, Gordis L, Tonascia JA, et al. *Am J Epidemiol.* 1981 Aug; 114(2):209-17.

999. A non-invasive test for the pre-cancerous breast. Simpson HW, McArdle C, Pauson AW, et al. *Eur J Cancer.* 1995 Oct; 31A(11):1768-72.

1000. The diagnosis of breast pre-cancer by the chronobra--I. Background review. Simpson HW, Griffiths K. *Chronobiol Int.* 1989; 6(4):355-69.

1001. The diagnosis of breast pre-cancer by the chronobra--II. The breast pre-cancer test. Simpson HW, Griffiths K. *Chronobiol Int.* 1989; 6(4):371-93.

Thorp KE. Energy Dynamics in Breast Cancer: Fields, Hormones & the Menstrual Cycle. G Med Sci. 2024; 5(1):169-249.
https://www.doi.org/10.46766/thegms.oncol.24111101

1002. Circadian breast skin temperature rhythms: Overt and occult benign and occult primary malignant breast disease. Wilson DW, George D, Mansel RE, et al. *Chronobiol Int.* 1984; 1(2):167-72.

1003. The luteal heat cycle of the breast in disease. Simpson HW, Griffiths K, McArdle C, et al. *Breast Cancer Res Treat.* 1996; 37(2):169-78.

1004. Loss of the progesterone-associated metabolic rhythm in cancer-associated breast tissue. Simpson HW, Pauson AW, McArdle C, et al. *Prog Clin Biol Res.* 1990; 341A:47-53.

1005. Circadian breast skin temperature rhythms: Overt and occult benign and occult primary malignant breast disease. Wilson DW, George D, Mansel RE, et al. *Chronobiol Int.* 1984; 1(2):167-72.

1006. The chronopathology of breast pre-cancer. Simpson HW, Pauson A, Cornélissen G. *Chronobiologia.* 1989 Oct-Dec; 16(4):365-72.

1007. The chronobra identifies prevailing mammary vascularity as a candidate variable in breast cancer post-operative outcome prediction. Simpson HW, George D, Sothern RB, et al. *Springerplus.* 2013 May; 2(1):241.

1008. The luteal heat cycle of the breast in health. Simpson HW, Griffiths K, McArdle C, et al. *Breast Cancer Res Treat.* 1993 Sep; 27(3):239-45.

1009. Circadian rhythm chaos: A new breast cancer marker. Keith LG, Oleszczuk JJ, Laguens M. *Int J Fertil Womens Med.* 2001 Sep; 46(5):238-47.

1010. Progesterone induces adult mammary stem cell expansion. Joshi PA, Jackson HW, Beristain AG, et al. *Nature.* 2010 Jun; 465(7299):803-07.

1011. RANKL expression in normal and malignant breast tissue responds to progesterone and is up-regulated during the luteal phase. Hu H, Wang J, Gupta A, et al. *Breast Cancer Res Treat.* 2014 Aug; 146(3):515-23.

1012. DNA replication licensing and progenitor numbers are increased by progesterone in normal human breast. Graham JD, Mote PA, Salagame U, et al. *Endocrinology.* 2009 Jul; 150(7):3318-26.

1013. Effects of sex steroids on proliferation in normal mammary tissue. Söderqvist G. *Ann Med.* 1998 Dec; 30(6):511-24.

1014. Control of mammary stem cell function by steroid hormone signalling. Asselin-Labat ML, Vaillant F, Sheridan JM, et al. *Nature.* 2010 Jun; 465(7299):798-802.

1015. Oxidative Stress: The Role of Estrogen and Progesterone. Cagnacci A, Gazzo I, Stigliani S, et al. *J Clin Med.* 2023 Nov 25; 12(23):7304.

1016. Progesterone decreases apnoea and reduces oxidative stress induced by chronic intermittent hypoxia in ovariectomized female rats. Joseph V, Laouafa S, Marcouiller F, et al. *Exp Physiol.* 2020 Jun; 105(6):1025-34.

1017. Progesterone prevents oxidative stress and respiratory dysfunctions in female rats exposed to intermittent hypoxia. Bairam A, Laouafa S, Marcouiller F, et al. *FASEB J.* 2020 Apr; 34(Suppl 1):1.

1018. Progesterone alleviates acute brain injury via reducing apoptosis and oxidative stress in a rat experimental subarachnoid hemorrhage model. Cai J, Cao S, Chen J, et al. Neurosci Lett. 2015 Jul 23; 600:238-43.

1019. Melatonin Patterns and Levels During the Human Menstrual Cycle and After Menopause. Greendale GA, Witt-Enderby P, Karlamangla AS, et al. J Endocr Soc. 2020 Aug; 4(11):bvaa115.

Thorp KE. Energy Dynamics in Breast Cancer: Fields, Hormones & the Menstrual Cycle. G Med Sci. 2024; 5(1):169-249.
https://www.doi.org/10.46766/thegms.oncol.24111101

1020. The circadian pattern of melatonin and its positive relationship with progesterone in women. Webley GE, Leidenberger F. *J Clin Endocrinol Metab.* 1986 Aug; 63(2):323-28.

1021. Human serum melatonin changes during the menstrual cycle. Wetterberg L, Arendt J, Paunier L, et al. *J Clin Endocrinol Metab.* 1976 Jan; 42(1):185-88.

1022. Melatonin and the ovulatory luteinizing hormone surge. Zimmermann RC, Schröder S, Baars S, et al. *Fertil Steril.* 1990 Oct; 54(4):612-18.

1023. Hormone receptors in breast cancer: More than estrogen. Lamb CA, Vanzulli SI, Lanari C. *Medicina (B Aires).* 2019; 79(S 6/1):540-45.

1024. Estrogen and progesterone receptors in breast cancer: Relationships to tumour histopathology and survival of patients. Blanco G, Alavaikko M, Ojala A, et al. Anticancer Res. 1984 Nov; 4(6):383-89.

1025. Estrogen receptor, progesterone receptor, and glucocorticoid receptor expression in normal breast tissue, breast in situ carcinoma, and invasive breast cancer. Buxant F, Engohan-Aloghe C, Noël JC, et al. *Appl Immunohistochem Mol Morphol.* 2010 May; 18(3):254-57.

1026. The Role of Progesterone Receptors in Breast Cancer. Li Z, Wei H, Li S, et al. *Drug Des Devel Ther.* 2022 Jan 26; 16:305-14.

1027. Progesterone receptors in normal breast development and breast cancer. Giulianelli S,Lamb CA, Lanari C. *Essays Biochem.* 2021 Dec; 65(6):951-69.

1028. Triple-Negative Breast Cancer: Histopathologic Features, Genomics, and Treatment. Adrada BE, Moseley TW, Kapoor MM, et al. *Radiographics.* 2023 Oct; 43(10):e230034.

1029. An overview of triple-negative breast cancer. Kumar P, Aggarwal R. *Arch Gynecol Obstet.* 2016 Feb; 293(2):247-69.

1030. The pineal gland and breast cancer: Serum levels of melatonin in patients with mammary tumors and their relation to clinical characteristics. Barni S, Lissoni P, Sormani A, et al. *Int J Biol Markers.* 1989 Jul; 4(3):157-62.

1031. Stage-dependent depression of melatonin in patients with primary breast cancer. Correlation with prolactin, thyroid stimulating hormone, and steroid receptors. Bartsch C, Bartsch H, Fuchs U, et al. *Cancer.* 1989 Jul; 64(2):426-33.

1032. Decreased nocturnal plasma melatonin peak in patients with estrogen receptor positive breast cancer. Tamarkin L, Danforth D, Lichter A, et al. *Science.* 1982 May; 216(4549):1003-05.

1033. Plasma melatonin and the hormone-dependency of human breast cancer. Danforth DN Jr, Tamarkin L, Mulvihill JJ, et al. *J Clin Oncol.* 1985 Jul; 3(7):941-48.

1034. Role of Melatonin in Breast Carcinoma: Correlation of Expression Patterns of Melatonin-1 Receptor With Estrogen, Progesterone, and HER2 Receptors. Goyal R, Gupta T, Bal A, et al. *Appl Immunohistochem Mol Morphol.* 2020 Aug; 28(7):518-23.

1035. Alteration of the MT1 melatonin receptor gene and its expression in primary human breast tumors and breast cancer cell lines. Lai L, Yuan L, Cheng Q, et al. *Breast Cancer Res Treat.* 2009 Nov; 118(2):293-305.

1036. Expression of melatonin receptor MT1 in cells of human invasive ductal breast carcinoma. Jablonska K, Pula B, Zemla A, et al. *J Pineal Res.* 2013 Apr; 54(3):334-45.

1037. Melatonin increases oestrogen receptor binding activity of human breast cancer cells. Danforth DN, Tamarkin L, Lippman ME. *Nature.* 1983 Sep; 305(5932):323-25.

Thorp KE. Energy Dynamics in Breast Cancer: Fields, Hormones & the Menstrual Cycle. G Med Sci. 2024; 5(1):169-249.
https://www.doi.org/10.46766/thegms.oncol.24111101

1038. Nocturnal urinary 6-sulphatoxymelatonin excretion is decreased in primary breast cancer patients compared to age-matched controls and shows negative correlation with tumor-size. Bartsch C, Bartsch H, Karenovics A, et al. *J Pineal Res.* 1997 Sep; 23(2):53-58.

1039. Expression of melatonin receptors in triple negative breast cancer (TNBC) in African American and Caucasian women: Relation to survival. Oprea-Ilies G, Haus E, Sackett-Lundeen L, et al. *Breast Cancer Res Treat.* 2013 Feb; 137(3):677-87.

1040. Melatonin and Cancer Hallmarks. Talib WH. *Molecules.* 2018 Feb; 23(3):518.

1041. Melatonin seems to be a mediator that transfers the environmental stimuli to oocytes for inheritance of adaptive changes through epigenetic inheritance system. Irmak MK, Topal T, Oter S. *Med Hypotheses.* 2005; 64(6):1138-43.

1042. Melatonin, a Full Service Anti-Cancer Agent: Inhibition of Initiation, Progression and Metastasis. Reiter RJ, Rosales-Corral SA, Tan DX, et al. *Int J Mol Sci.* 2017 Apr; 18(4):843.

1043. Role of melatonin in the epigenetic regulation of breast cancer. Korkmaz A, Sanchez-Barcelo EJ, Tan DX, Reiter RJ. Breast Cancer Res Treat. 2009 May; 115(1):13-27.

Thorp KE. Energy Dynamics in Breast Cancer: Fields, Hormones & the Menstrual Cycle. G Med Sci. 2024; 5(1):169-249.
https://www.doi.org/10.46766/thegms.oncol.24111101