ISSN 2520-5110

INTERNATIONAL ANNALS of Medicine

JIFACTOR 3.12

November, 2017 2017; Vol1; Issue10 http://iamresearcher.online

Is Silicone Breast Implant Toxicity an Extreme Form of a More Generalized Toxicity Adversely Affecting the Population as a Whole?

Arthur E. Brawer

Department of Medicine, Division of Rheumatology, Monmouth Medical Center, New Jersey, USA

Corresponding Author: Arthur E. Brawer

arthurbrawer@optimum.net

ABSTRACT

In humans the element silicon is essential for normal growth and development. It is extensively biointegrated into a wide variety of matrix macromolecules that display endless variations of complex overlapping interactions. When living organisms are confronted with artificial man-made organosilicon and organosiloxane compounds there is a finite limit of adaptive mechanisms by which normal cells and tissues can dispose of these molecules. Exposure to silicone gel-filled breast implants is particularly harmful due to biochemical chaos caused by in vivo degradation of an exceedingly excessive polymer presence. The worldwide proliferation of 60,000 other man-made silicon-containing compounds permeates all aspects of everyday living and has created generalized environmental exposure via inhalation, dermal absorption, and ingestion. These exposures are producing heightened public health concerns because prior assertions that organosiloxanes are chemically and biologically inert are no longer tenable. It is proposed that vague syndromes, such as fibromyalgia and chronic fatigue syndrome, are disorders caused by insidious slow-paced toxicity mechanisms similar to the more rapid and profound biochemical disruptions occurring in sick breast implant recipients.

Keywords: Silicone; Breast implants; Toxicity; Organosiloxanes; Organosilicones; Environmental exposure

1. INTRODUCTION

Over the past eighty years disorders that were once considered vague and rare have become vague and common. Two classic examples of this are fibromyalgia and chronic fatigue syndrome, which share numerous overlapping clinical features. During this same time interval more than 60,000 organosiloxane (organosilicon) compounds have been synthesized by industry, all of which are based on the artificial silicon-carbon bond[1,2,15]. They are extraordinarily pervasive in all aspects of everyday life worldwide, and they contaminate every environmental compartment[1,2,15]. Organosilicon molecules are typically present in food packaging, cleansers, furniture polish, cookware, insect repellants, insecticides, pesticides, fungicides, herbicides, anti-perspirants, deodorants, shampoos, hair conditioners, flame retardants, sealants, adhesives, antacids, plastics, pacemakers, transdermal drug delivery, antibiotics, sunscreens, artificial lenses, hair brushes, detergents, fabric softeners, anti-wrinkle clothing, perfumes, paint, lacquer, varnish, floor waxes, spatulas, ice cube trays, baking molds, emulsifiers, lubricants, rubbers, oral care pharmaceuticals and dental products, chewing gum, nursing nipples, electronics, natural health products, anti-foaming agents in beer

To Cite This Article: Arthur E. Brawer. Is Silicone Breast Implant Toxicity an Extreme Form of a More Generalized Toxicity Adversely Affecting the Population as a Whole?. *International Annals of Medicine. 2017;1(10).* https://doi.org/10.24087/IAM.2017.1.10.347

and other beverages, skin creams, moisturizers, lotions, acne treatments, anti-aging creams and wrinkle removers, facial cleansers, cosmetics and make-up, tanning agents, resins for paper impregnation, water repellants, insulation materials, spices, powdered sugar, dried eggs, preventatives of fruit bruising, psychotropic drugs, anti-convulsants, wound and burn ointments, alopecia preparations, intravenous tubing, contact lenses, syringe lubrication, joint prostheses, heart valves, and breast implants. For decades physical chemists have professed that the strong bonds of these molecules render them chemically and biologically inert, but recent evidence is proving otherwise[1,2,3,4,5,9,15,16].

Exposures to organosiloxanes occur from inhalation, dermal absorption, ingestion, and implantation into the body[1,2,3,4,5,6,15,16]. Regarding inhalation exposure, published studies on the adverse effects of volatile organosiloxanes parallels evidence that smoking is a predisposing risk factor for rheumatoid arthritis[2,7]. Regarding dermal absorption, the lipophilic nature of these compounds can be expected to promote biointegration into the subcutaneous fat, and is paralleled by research indicating that lipocytes (adipocytes) produce a variety of cytokines capable of initiating and/or augmenting inflammatory events[3,8]. Regarding ingestion, safety advocates have generally ignored evidence from bioremediation and geomicrobiology research explaining how bacteria can break down chemicals they have never seen before and then use the carbon fragments as building blocks for new molecules[1]. The anabolism is not the reverse of the original catabolism, and absorption of unknown species from the intestinal tract (and their ultimate fate in the body) have not been adequately addressed. The increased concentration of organosiloxanes found in reptiles, birds, turtles and seals coexists with the deleterious effects of these compounds on bees, earthworms and tadpoles, and occurs from all of the above exposure routes (excluding implantation)[6,9]. Adverse effects on microbial soil organisms have also been reported[2], raising questions regarding toxicity to the human intestinal microbiome.

An example of in vivo exposure to organosiloxanes occurs following implantation of silicone gel-filled breast implants. In the past five years these devices have been implicated by many researchers as the cause of a genuinely novel illness[3,4,10,11,12]. Identical causation claims in the early 1990's created a great deal of controversy back then because of faulty autoimmune theories, one of many factors contributing to the inevitable current repetition of this avoidable public health debacle[5,10]. Some investigators have proposed that breast implant illness is nothing more than spontaneous fibromyalgia, and others have proposed that this is an autoinflammatory disorder[12]. Both of these concepts are a gross oversimplification of what is clearly a much more complicated process[1,3]. The diverse clinical manifestations of silicone-induced toxicity have been underreported because of:

a) Truncated data bases used to record the ailments of recipients; and
b) The failure to appreciate that this disorder evolves chronologically in a manner simulating a dose-response curve[3,13].

Researchers have also failed to recognize that silicone-induced toxicity is mediated by at least two dozen disruptions in the body's biochemistry, virtually none of which have anything to do with autoimmunity[1,3]. These disruptions cause a multitude of clinical manifestations in ailing implant recipients including (but not limited to): fatigue, joint pain and swelling, dry eyes and mouth, cognitive dysfunction, chest pain, protracted AM stiffness, myalgias, weakness, hair loss, skin rashes, paresthesias, headaches, skin pigment changes, telangiectasias, itching, night sweats, dizziness, nausea, abdominal pain, metallic taste, chills, photosensitivity, recurrent infections, tinnitus, and loose stools alternating with constipation[3,5,11,12,13,14,21].

2. PECULIAR OBSERVATIONS

A common and perplexing symptom experienced by ailing breast implant recipients is odor and smell hypersensitivity. Prior to implantation these patients manifested no adverse reactions to perfumes, room fresheners, deodorants, hairsprays, cleaning agents, cigarette smoke, exhaust fumes, carpeting, fabric dyes, adhesives, caulking, glues, stain removers, detergents, dry cleaning products, paints, lacquers, insecticides, pesticides, and printing resins. After their systemic illness became established, they subsequently began to experience nausea, dizziness, and headaches on exposure to nearly all of the above[3]. These intolerances were also frequently accompanied by the development of food and skin sensitivities, the latter occurring from routine use of cosmetics, skin softeners, moisturizers, wrinkle removers, shampoos, lotions, and beauty salon products[3]. These other exposures not only caused headaches, dizziness, and nausea, but also substantially worsened many if not all

of the other established and ongoing silicone-induced ailments (a circuitous observation also reproduced by the odor and smell culprits). Once explanted, improvement and/or resolution of systemic ailments was often accompanied by improvement and/or resolution of both the caustic odor and smell phenomena and the adverse dermatologic intolerances[3,14]. One should be aware, however, that silicone-induced toxicity has been reported to be a double-edged sword: the sicker patients became from exposure to gel-filled devices, the less chance they had of achieving improvement following their removal[14]. Stated another way, many patients reached a "point of no return" if their devices were left in place too long. In addition, the chronological development of silicone-induced disease is not dependent on ruptured devices (the reader is referred to references 5,13, and 14 regarding the effects of rupture). The Ethics Committee at Monmouth Medical Center did not require permission for these studies.

3. WHERE IS THE LINKAGE TO THE GENERAL POPULATION?

Now consider patients I have seen over the past 42 years where odor and smell hypersensitivity developed in non-implant scenarios. Typical examples included individuals working in "toxic" schools where a confined office environment was compromised by many different adverse exposures. For nine months during each school year such workers would experience numerous multisystem ailments that could not be attributed to any well-defined textbook condition. In the summer months, when employment was interrupted, these patients noted resolution of all complaints such as fatigue, arthralgias, cognitive dysfunction, headaches, nausea, paresthesias, itching, etc. (which is only a partial list of innumerable ailments). Multisystem complaints typically recurred during subsequent school semesters, again followed by resolution in the summer months. But after ten years or more of this cyclical scenario the summer recess did not yield cessation of these phenomena, whereupon chronicity was then accompanied by the development of odor and smell hypersensitivity virtually identical to the observations in ailing breast implant recipients. At this juncture even retirement did not guarantee future improvement. Thus, this scenario and the breast implant observations emphasize two points:

1) When an individual becomes chronically ill from one or more environmental exposures, small amounts of toxicity emanating from elsewhere are often poorly tolerated; and
2) There can be a "point of no return" whereby such an illness is capable of persisting indefinitely. Even repetitive household exposures, punctuated by interspersed vacations, have produced identical scenarios in my rheumatology practice.

4. EXPANDING THESE OBSERVATIONS TO FIBROMYALGIA

Eighty years ago fibromyalgia was vague and rare, but today it is vague and common with an estimated ten million sufferering in the USA and 3-6% of the world population afflicted[18]. The nucleus of fibromyalgia's cardinal manifestations encompass four items: wide-spread generalized pain (muscles and joints), fatigue, non-restorative sleep, and tender points. Many other manifestations can exist under this umbrella, including (but not limited to): cognitive dysfunction, abdominal complaints (nausea, loose stools, cramps, etc.), dry eyes, AM stiffness, headaches, palpitations, chest pain, paresthesias, skin rashes, dizziness, weight gain, anxiety and depression. There are three questions I have always considered relevant to my own understanding of fibromyalgia, namely:

1) if a healthcare provider did not historically elicit the four cardinal items, but instead first inquired about all the other manifestations, what would the diagnosis be?
2) why do fibromyalgia patients demonstrate marginal help and enormous intolerance to regular and/or sub-pediatric doses of anti-inflammatory medications? and
3) do fibromyalgia patients commonly experience odor and smell hypersensitivity?

The answer to the last question is a resounding "yes" in virtually all such patients encountered in my own private practice. The answers to the first two questions are, in my opinion, quite straightforward, namely: fibromyalgia patients are behaving clinically as if they are suffering from environmental toxicity. And with regard to pharmaceutical sensitivity, all drugs are potential toxins, it just depends on the dosage - hence, the same phenomenon as outlined above, namely "poor tolerance to small amounts of toxicity emanating from elsewhere." The original causation theory of fibromyalgia rested on the notion that the filters of the afferent nervous system malfunctioned,

thereby creating an increase in nociception. These events are likely to be operative, but I believe they occur as a result of the illness rather than being the cause, thereby becoming circuitously augmenting. Over the past two decades a variety of other observations have been recognized in fibromyalgia patients including, but not limited to, muscle exhaustion, impaired muscle metabolism, impaired brain metabolism, microcirculatory abnormalities, endothelial dysfunction, and dysautonomia[22,24]. In the aggregate these findings suggest a much more attractive and unifying causation hypothesis, namely that repetitive environmental exposures to a variety of organosiloxanes (and/or their degradation molecules) simultaneously cause malfunction of matrix macromolecules along with widespread biochemical disruptions, both of which occur with exponential severity in ailing breast implant recipients[1,3]. Consider the fact that endothelial cells sit on a basement membrane composed of several proteoglycans. Or that silicic acid (one of the many degradation molecules) can cross the blood brain barrier and chelate neurotransmitters (e.g., dopamine). Or that silanols (another degradation molecule) can bind to proteins (including enzymes), with obvious repercussions (especially in mitochondria).

5. THE PARALLEL RESEARCH OF TILT

Dr. Claudia Miller has long pioneered the concept that a chronic illness initiated by one or more toxic environmental exposures is capable of evolving into a more expansive illness encompassing adverse reactions to multiple other unrelated exposures, the latter of which did not precipitate the toxic illness in the first place[17]. She has named this phenomenon TILT, which stands for "toxicant induced loss of tolerance." I freely admit that I was unaware of Dr. Miller's research until five years ago, but her research has profound similarity to four decades of my own observations. This has naturally spawned three more questions:

1) what are the mechanisms responsible for these genuinely real phenomena?
2) are the observations inherent to TILT truly caused by supposedly "unrelated" agonists? and
3) if not, then what are the unifying environmental culprits?

It is clear that silicone breast implant illness, workplace and household related ailments, fibromyalgia, and TILT are neither autoimmune nor psychosomatic disorders. Etiologic clues are being promulgated by recent publications reporting on seemingly unrelated topics. These research manuscripts encompass diverse issues ranging from (but not limited to):

1) the decimation of honeybees caused by organosilicon surfactants;
2) elevated levels of volatile organosiloxanes in household inhabitants;
3) organosilicon presence in virtually all dermatology products;
4) sophisticated chemical assays providing reliable measurements of previously difficult-to-measure organosiloxane compounds;
5) the microbiome;
6) other animal studies;
7) reassessment of the biocompatibility of supposedly "inert" biomaterials;
8) new insights into hydrolysis of oganosiloxanes; and
9) illness caused by silicone gel-filled breast implants[1,2,4,5,6,9,11,12,13,14,15].

The unifying cornerstone of these reports appears to be the research diversity itself, all of which is connected in one way or another to the extraordinary pervasiveness of organosiloxane (organosilicon) compounds in the global environment. Regardless of how these molecules enter the body, it is proposed that mechanisms causing illness in fibromyalgia, TILT cohorts, and other vague syndromes will, in all probability, be likely to mimic dysfunction of matrix macromolecules and disruptions of the body's biochemistry as described in two publications on silicone breast implant toxicity[1,3].
Severity of illness in fibromyalgia and allied disorders may depend, in part, on

a) the intensity, variety, and duration of organosiloxane exposures, and
b) the constitutional fortitude of each individual.

With regard to the latter, although silicone-induced toxicity is no longer a vague syndrome, I have seen a few women who remain perfectly healthy despite having prolonged implantation with ruptured breast implants over a span of many years[3].

6. A DIFFERENT PERSPECTIVE FOR RHEUMATOLOGISTS

Academic rheumatologists and immunologists might alter their theories if they consulted with colleagues who have walked through the medical vineyards and have tasted the wine. Stated another way, although observations by clinicians toiling on the front lines are not usually the source of double blind placebo controlled randomized clinical trials, nonetheless the experiences of such colleagues often provide valuable insights into mechanisms of disease causation. As an example, when rheumatoid arthritis is initiated by physical trauma, the orchestration of continuing inflammation in injured joints is quite different from the spontaneous evolution of inflammatory and autoimmune responses described by immunologists[7]. In addition, immunologists have not yet deduced why the risk of developing rheumatoid arthritis and systemic lupus erythematosus is statistically higher if one is chronically exposed to pesticides and insecticides[7]. One-third of individuals who became systemically ill after 25 years of water usage contaminated with trichloroethylene developed a positive antinuclear antibody (ANA) test[19]. And one-third of patients suffering from silicone breast implant illness have a positive ANA test[13]. Terminology and immunology make strange bedfellows. In patients with autoimmune retinopathy there is an absence of inflammation in pathological specimens of deteriorating retinal components despite the serological presence of anti-retinal antibodies[20]. Perhaps the presence of antibodies in these and other conditions, such as multiple sclerosis, is, in part, a reflection of damaged tissues rather than the cause (i.e., the antibodies are epiphenomena). The complexity of nature far transcends man's ingenuity, and assessing the behavior of made-made chemicals in the human body is both difficult to predict and extraordinarily complex. As an example, following the in vivo degradation of organosiloxanes, residual silicon-containing molecules may:

a) rearrange themselves and combine with one another, and
b) form polymerized species of unknown crystal forms (i.e., silicates) by interacting with calcium, magnesium, and phosphorus[1,2].

Biology can energize systems, and silicates bound to sugars can become catalytically active, taking on the properties of enzymes[1]. This phenomenon has direct relevance to the reported observation that the sequential evolution of systemic illness caused by silicone gel-filled breast implants proceeds in an exponential manner analogous to a reactor catalysis mechanism[13]. Alternatively, the binding of silicates to the sugars of matrix macromolecules can have profound and virtually unlimited pathophysiologic consequences[1]. As an example, in ailing breast implant recipients the documented presence of dry eyes and dry mouth has been attributed to malfunction of the proteoglycan receptors for acetylcholine in salivary and lacrimal tissues[3]. Considering the endless variations of complex overlapping interactions of matrix macromolecules in humans, an exhausting list of questions can ensue, with one being particularly relevant: is dysautonomia (dysfunction of the autonomic nervous system) caused by organosiloxane-derived degradation molecules that then interfere with cholinergic and/or adrenergic receptor function? Dysautonomia has been documented to exist in many acquired disorders such as fibromyalgia and systemic lupus erythematosus (SLE)[22,23]. When present in SLE or any other systemic connective tissue disease, the symptoms of dysautonomia do not usually correlate with overall disease activity and/or remission[23]. Indeed, remission of inflammatory phenomena is often accompanied by vague residual complaints (especially fatigue and weakness), which in turn are often diagnosed as comorbid fibromyalgia. But what causes these residual complaints? Phosphorus in energy systems is metal-ion bound, but the element silicon behaves like a metal at times[1,3]. Therefore one cannot accumulate silicon molecules next to phosphorus molecules and expect energy production and energy utilization to proceed normally[1,3]. So what exactly is going on here? Does the presence of an established connective tissue disease predispose one to be more susceptible to the adverse effects of organosiloxanes? Or do organosiloxane-derived degradation molecules add to the problem by also altering muscle membrane permeability[1]? And last but not least, is primary fibromyalgia itself a toxic consequence of the environmental pervasiveness of organosiloxanes?

7. BIOPHYSICS CONSIDERATIONS

Each and every atom (e.g., carbon, oxygen, silicon) creates its own electromagnetic field depending on the number of protons and electrons that are present[25]. Combining individual atoms into molecules expands this phenomenon. The communications network of cellular molecules that interact with each other and produce life-sustaining processes (e.g., proteins, enzymes, DNA, RNA, matrix components, etc.) are highly dependent on ordered states of electrical and

magnetic forces[25]. Since silicon-carbon bonds do not normally occur in nature, living organisms have previously never had to contend with them. But since silicon behaves like a metal, the biointegration of organosilicon molecules into life-sustaining molecules can be expected to create erratic disturbances in normal electromagnetic fields. Such disturbances, in turn, can be expected to functionally alter cellular metabolism which, in turn, will then disturb the homeostasis of the entire organism. Ask yourself one simple question: what would happen to energy transmission if the electron transfer system of mitochondria malfunctioned? The routine manner in which the human body incorporates silicon for normal bone and matrix synthesis[26], and the routine physiology of silicon deficiency or excess[26], are vastly different from the body's "impossible" task of figuring out what to do with molecules that contain an artificial silicon-carbon bond. Stated another way, in the absence of any direct silicon-carbon bonding there is a natural and healthy utilization of silicon in the human body which is devoid of any pathologic biointegration of silicon into essential life-sustaining cellular molecules. Biochemical chaos caused by organosiloxanes also extends to the emerging discipline of epigenotoxicology, which studies adverse effects of environmental toxicants on gene expression and disruption of cell functions via changes in DNA methylation, alterations of histones, and changes to micro RNA[27]. Biophysics research is now capable of circuitously uniting:

a) the odor and smell hypersensitivity observed in my breast implant recipients and fibromyalgia patients with
b) the TILT observations of Dr. Claudia Miller.

This is achieved by coordinating reports on the harmful biological effects of electromagnetic fields (which includes odor intolerance)[28,29] with investigations that have deciphered the cellular mechanisms of olfaction[30]. Once organosiloxanes create disturbances in the electromagnetic fields of transcription factors, this leads to activation of odorant receptor genes located in the olfactory epithelium of the olfactory bulb. Subsequently, a cascade of events ensues whereby G protein coupled odorant receptors are synthesized which, in turn, activate adenylyl cyclase to increase the cellular concentration of cyclic AMP which, in turn, leads to activation of olfactory sensory neurons.

8. CONCLUSIONS

It is no longer conjecture that organosiloxanes are extraordinarily pervasive environmental contaminants, and it is no longer conjecture that these compounds are capable of causing adverse toxic effects on living organisms, including humans. With the recent development of reliable assays for in vivo detection of these molecules and their breakdown products, the knowledge gleaned from the reality of silicone breast implant toxicity, and the exponential advances in biophysics, the opportunity exists to study vague syndromes and well-defined rheumatologic disorders in a different light. Such studies can confirm or refute the question that I asked twenty years ago, namely: is silicone breast implant toxicity an extreme form of a more generalized toxicity adversely affecting the population as a whole?[1].

REFERENCES

1. Brawer AE. Silicon and matrix macromolecules: new research opportunities for old diseases from analysis of potential mechanisms of breast implant toxicity. Medical Hypotheses 1998; 51:27-35.
2. Rucker C, Kummerer K. Environmental chemistry of organosiloxanes. Chemical Reviews 2015; 115:466-524.
3. Brawer, AE. Mechanisms of breast implant toxicity: will the real ringmaster please stand up. Internat Annals of Medicine 2017; 1 (9): doi.org/10.24087/IAM.2017.1.9.249.
4. Williams DF. Biocompatibility pathways: biomaterials-induced sterile inflammation, mechanotransduction, and principles of biocompatibility control. ACS Biomater Sci Eng 2017; 3:2-35.
5. Brawer AE. Destiny rides again: the reappearance of silicone gel-filled breast implant toxicity. LUPUS 2017; 26:1060-1063
6. Wang DG, deSolla SR, Lebeuf M, et al. Determination of linear and cyclic volatile methylsiloxanes in blood of turtles, cormorants, and seals from Canada. Science of the Total Environment 2017; 574:1254-1260.
7. Brawer AE, Goel N. The onset of rheumatoid arthritis following trauma. Open Access Rheumatol: Research and Reviews 2016; 8:77-80.
8. Tilg H, Moschen AR. Adipocytokines: mediators linking adipose tissue, inflammation, and immunity. Nature Reviews Immunology 2006; 6:772-783.

9. Chen J, Fine JD, Mullin CA. Are organosilicon surfactants safe for bees or humans? Science of the total environment 2018; 612:415-421.
10. Brawer AE. Bones, groans, and silicone. LUPUS 2012; 21:1155-1157.
11. Maijers MC, deBlok CJM, Niessen FB et al. Women with silicone breast implants and un-explained systemic symptoms: A descriptive cohort study. Netherlands J Med 2013; 71:534-540.
12. Colaris MJL, deBoer M, vander Hulst RR, Tervaert JWC. Two hundred cases of ASIA following silicone implants: a comparative study of 30 years and a review of current literature. Immunol Res 2017; 65:120-128.
13. Brawer AE. Chronology of systemic disease development in 300 symptomatic recipients of silicone gel-filled breast implants. J Clean Technol, Environmental Toxicol, & Occupat Med 1996; 5:223-233.
14. Brawer, AE. Amelioration of systemic disease following removal of silicone gel-filled breast implants. J Nutritional Environmental Med 2000; 10:125-132.
15. Hirner AV, Flassbeck D, Gruemping R. Organosilicon compounds in the environment, in Peter J Craig, editor, Organometallic compounds in the environment, 2nd edition 2003; chapter 8:305-351. John Wiley and Sons, West Sussex, England.
16. Bodin F, Jung C, Dieval F, et al. Aging of retrieved gel breast implants: a comparison between two product generations. J Mechan Behav Biomed Materials 2015; 46:11-22.
17. Miller CS. Toxicant-induced loss of tolerance. J Nutritional Environmental Med 2001; 11:181-204.
18. The National Fibromyalgia Association (NFA), 3857 Birch Street, Suite 312, Newport Beach, CA 92660 (email: nfa@fmaware.org)
19. Brawer AE. Unpublished observations of illness in South Tucson residents caused by 25 years of exposure to water contaminated solely with trichloroethylene (1956 to 1981).
20. Brawer AE. Corticosteroids: the knee jerk response. Current Medical Research & Opinion 2016; 32:1935-1936.
21. Borenstein D. Siliconosis: a spectrum of illness. Semin Arthritis Rheum 1994; 24:1-7.
22. Kingsley JD. Autonomic dysfunction in women with fibromyalgia. Arthritis Research Therapy 2012; 14:103.
23. Haghighat S, Fatemi A, et al. The autonomic dysfunction in patients with lupus disease: an electrophysiological study. Adv Biomed Res 2016; 5:102-109.
24. Lund E, Kendall SA, et al. Muscle metabolism in fibromyalgia studied by P-31 magnetic resonance spectroscopy during aerobic and anaerobic exercise. Scand J Rheumatol 2003; 32:138-145.
25. Jacobson J, Sherlag B. Aging and magnetism: Presenting a possible new holistic paradigm for ameliorating the aging process and the effects thereof, through externally applied physiologic PicoTesla magnetic fields. Medical Hypotheses 2015; 85:276-286.
26. Price CT, Koval KJ, Langford JR. Silicon: a review of its potential role in the prevention and treatment of postmenopausal osteoporosis. International Journal of Endocrinology 2013; dx.doi.org/10.1155/2013/316783.
27. Hodjat M, Rahmani S, et al. Environmental toxicants, incidence of degenerative diseases, and therapies from the epigenetic point of view. Arch Toxicol 2017; 91:2577-2597.
28. Nordin S, Neely G, et al. Odor and noise intolerance in persons with self-reported electro-magnetic hypersensitivity. International Journal Environmental Research Public Health 2014; 11:8794-8805
29. Kaszuba-Zwoinska J, Gremba J, et al. Electromagnetic field induced biological effects in humans. Przegl Lek 2015; 72:636-641.
30. Malnic B, Dansela C et al, in Menini A, editor - The Neurobiology of Olfaction: Chapter 7 – Odorant Receptors. CRC Press/Taylor Francis 2010, Boca Raton.