**Can deficiencies in nutrition (i.e., micronutrients) cause metabolic disease and damaged DNA be corrected via "Metabolic Correction"?**

Citation: Gonzalez, M.J., and Massari, J.R. (2012). Metabolic Correction: A Functional Explanation of Orthomolecular medicine. Journal of Othomolecular Medicine, 27(1).

Direct link to article: [http://wakeupsense.com/Gonzalez-Metabolic-Correction.pdf](https://www.researchgate.net/go.Deref.html?url=http%3A%2F%2Fwakeupsense.com%2FGonzalez-Metabolic-Correction.pdf)

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In the above cited review, Michael J. Gonzalez and Jorge R. Massari review the theoretical framework of what they call “Metabolic Correction” derived from the works and concepts of Roger J. Williams (Genetrophic Disease), Linus C. Pauling (Orthomolecular Medicine), Jeffrey S. Bland (Functional Medicine), and Bruce N. Ames (Triage Theory) . They state, “The key idea of orthomolecular medicine is that genetic factors affect not only the physical characteristics of individuals, but also their biochemical milieu. Biochemical pathways of the body have significant genetic variability, and diseases such as atherosclerosis, cancer, schizophrenia or depression are associated with specific biochemical abnormalities, which are causal or contributing factors of the illness.”
After an historical review of the concepts and explanation of their practical application, they conclude that: “We need to abandon outdated paradigms of nutrient intake merely to prevent deficiencies and [instead] expand them to prevent chronic diseases and achieve optimal health with metabolic correction.”
Our colleagues on Research Gate come from so many varied perspectives in their work, from nutrition to genetics, biochemistry to medicine; it would be interesting to discuss the merits of the concept of Metabolic Correction from these varying perspectives. Thank you.

Sep 12, 2013 · Modified Sep 12, 2013 by the commenter.

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[**Max Stanley Chartrand**](https://www.researchgate.net/profile/Max_Chartrand) · [88.60](https://www.researchgate.net/profile/Max_Chartrand/reputation) · [4.82](https://www.researchgate.net/profile/Max_Chartrand/publications) · [Northcentral University](https://www.researchgate.net/institution/Northcentral_University3)

William, I agree the system is flexible, but with limits. The system is designed for organisms and not inert elements; for instance, calcium in ionic form not in free (inert) form. But even with free calcium our system can convert some of it into ionic form, but not overwhelmingly. The differences in the dead, sterile modern diet distorts both genetic orchestration and metabolic processes. I know what you mean relative to the advertising quality aspect as I feel there is a huge difference between what nature calls a nutrient (each with its own universe of co-factors and metabolic features)--i.e, Vitamin C is NOT ascorbic acid, but an immensely complex element that humans cannot manufacture as other mammals can. Too much of ascorbic acid causes our bodies to go into acisosis and all the chronic conditions that go with it. In organic form, however, (and this applies essentially to all nutrients), the body can regulate what is needed and what is not needed, making acidosis as a result of eating, say, too many oranges an impractical rather than health threatening matter. I am among those who considers both synthetic nutrition and natural crystalline nutrition as far inferior to organically bioavailable forms of nutrition. Hence, over-reliance of synthetic and natural crystalline in the short term may suffice in a nutrition crisis, but in the long term both variably will render consequences that a less than optimal to the body. Food, then, should be our main nutrition for optimal outcomes--and there there is much lattitude. In many cultures and climes diet can vary significantly and still be optimal. My contention in these times is that the US food supply has become less than optimal in micro and organic nutrients, and therefore, some supplementation may be needed. That supplementation will vary from person to person and is purely adjunctive to the person's diet, hydration, sleep, lifestyle, enviornmental challenges, mental-emtional, and physical exercise habits. So there is no misunderstanding, though, I feel certain that Michael Gonzeles and his associates have not conflict of interest in their endorsement of nutrition, and therefore are providing an hypothesis that is both observational and theoretical.

 13 / 0 · Mar 23, 2014

[**Constantine Kaniklidis**](https://www.researchgate.net/profile/Constantine_Kaniklidis) · [66.74](https://www.researchgate.net/profile/Constantine_Kaniklidis/reputation) · No Surrender Breast Cancer Foundation (NSBCF)

Let me here try to (1) put a bit more precision in (a least a brief) statement of the metabolic correction hypothesis of Michael Gonzalez et al. to show that it is by no means trivial or transparent, and also to suggest some of the challenges it now does and will confront, closing nonetheless with an appreciation of the authors' contributions: METABOLIC CORRECTION 101 Simplifying greatly, metabolic correction posits that metabolic harmony is requisite to optimal health, and that vitamins, minerals and nutrients in this context function as cofactors in the orchestration of enzymatically-driven biochemical reactions, and can be harnessed towards the improvement of cellular biochemistry to ultimately achieve metabolic/physiological optimization, primarily via the fact that increased concentrations of these agents fundamentally improve enzymatic/metabolic efficiency, which in turn may repair genetic and hereditary defects and disorders consequent to defective enzymes, allowing for: (1) reduction of drug dosing/scheduling requirements, (2) reduction of adverse events, and (3) improvement of patient outcome. This is intended only as a concise distillation, and readers would be well advised to pursue the full articles for detail. CHALLENGES Now, I will here present just four of several issues associated with the metabolic correction hypothesis, both because they reflect my concerns in the critical appraisal of any viable hypotheses, and also the stimulate further discussion: 1. Given that VM/nutrient concentrations, metabolism, and associated effects themselves exhibit wide individual variability as well as critical dependencies on multiple relatively uncontrollable internal and external factors and influences, including other coadministered nutraceuticals and individual dietary patterns - which to their credit the authors acknowledge - the consistency and sustainability of positive effects is not assured, and must be independently demonstrated rigorously, and in addition we do not currently have sufficient evidence and knowledge to wholly determine the achievement of the optimized metabolic efficiency state and its harmonic dynamics (see also below). 2. The metabolic correction and optimization needed and exerted for addressing one target condition may be in conflict with, or induce, potential collateral damage on other conditions that the same nutraceuticals can also influence. There is for instance some data that folate supplementation may be associated with increased mortality risk, although a dispositive conclusions awaits critical appraisal of the underlying studies, not yet systematically undertaken, made difficult by the fact that correlations between dietary folate intake and blood folate concentration vary from weak to moderate at best. In addition, the latest systematic review and meta-analysis (the Oslo SR/MA, Wien et al., BMJ Open 2012) of 10 RCTs on folate supplementation in the oncology context, finds for the six RCTs reporting prostate cancer incidence, an RR for prostate cancer of 1.24 for those receiving folic acid compared to controls, a clinically and statistically significant increase in prostate cancer after folic acid supplementation. This would represent unintended consequences if for instance we were using folate supplementation (alone or in the "Metanx" combination regimen) in the service of benefiting diabetic neuropathy (as the authors advocate in an associated article on metabolic correction for that condition target (these negative long-term findings are confirmative of the ProtecT Trial (Collin et al, Cancer Epidemiol Biomarkers Prev. 2010), as also suggested in the analysis of the Janus Cohort (de Vogel et al. Int. J. Epidemiol. 2013)). 3. Furthermore, long-term safety and exposure dynamics may be radically different, and adverse, from the main short-term effects examined in almost all studies. Consider the New Zealand meta-analysis (Fife et al., Colorectal Dis, 2011; independently critically appraised by me elsewhere) on folic acid supplementation on colorectal cancer risk: although there was no effect, positive or negative, up to 3-years, colonic surveillance beyond 3 years found increased risk of colorectal adenoma - and unfortunately, especially advanced adenoma - among the folate group, challenging contrary but weaker epidemiological findings claiming folate status is inversely related to the risk of developing colorectal cancer. Similarly, I have argued independently based on robust data that that high omega-3 fatty acid consumption may exert clinically relevant adverse pharmacokinetic sequelae on the anti-HER2 biological agent's, lapatinib (Tykerb), metabolism in the oncology context (along with many other sobering cases). 3. Nutraceuticals may exert benefits beyond those exerted through enzymatic modulation and its consequences: there is data to suggest non-metabolic effects via both (1) inhibition or activation or modulation of underlying molecular pathways, as well as (2) epigenetic, not genetic, mechanisms, and these may more robustly be positively influenced by CAM agents outside the nutraceutical borders such as curcuminoids or AKBA/boswellic acids or natural HDAC/DNMT inhibitors. IN GUARDED BUT REAL APPRECIATION Nonetheless, there is much to be credited to Michael Gonzalez and co-investigators efforts: (1) who have begun to systematically put the original perhaps overbroad precepts of orthomolecular medicine that too often argued beyond the boundaries of the evidence, onto a methodologically sounder foundation, accompanied with particular testable sub-hypotheses, and (2) who have put forth in fact considerable plausible data - including I note, human clinical, not just preclinical, data - to support new precepts of the metabolic correction hypothesis at a more evidence-based level, and (3) who by their highly articulated and often quite plausible and data-driven analyses are bound to stimulate pursuance of new directions in the scientific, not just anecdotal or speculative, study of the complex matrix of relationships between nutraceuticals - and their balance - and genetic factors and disorders long felt - incorrectly - to be the purview only of traditional pharmaceuticals, with a peripheral, cure-the-deficiency role assigned to vitamins, minerals and nutrients.

* [**Olga Shnaybel**](https://www.researchgate.net/profile/Olga_Shnaybel) · [4.74](https://www.researchgate.net/profile/Olga_Shnaybel/reputation) · International Center of Revitalization and Rejuvenation LLC “La Lik

Dear Mr Max! You raised an interesting and important topic.
I'm not familiar with the works of Dr..Michael Gonzalez,.but I think that ортомолекулярная medicine in combination with the traditional methods of treatment, was very useful. Most importantly, finding a competent specialist who will be able to choose the right elements. I think everybody knows that free radicals destroy the lipid membrane cells and causes damage to the DNA molecule.
In Russia and some other countries have developed methods of prophylaxis and treatment, which, probably, can also be attributed to orthomolecular medicine. Is the use of calcium phosphate drugs to prevent the disease Kashin-Beck , the iodization of products in areas where goiter, dental caries prevention substitution of sugar on the other sweet foods and water fluoridation. Not only vitamin C, but many other vitamins are considered from the point of view of orthomolecular medicine. First of all vitamins A, B1, B6, B12, E, and some others.
Biologically active substance, determine the atherogenic or antiatherogenic nature of the action diets. Оrthomolecular medicine should also include the use of plant sterols, fatty acids, multivitamin preparations, antioxidants and other substances for the prevention of atherosclerosis and increasing life expectancy. Of great importance antioxidants, especially natural, possessing a wide range of therapeutic and preventive actions
Since the beginning of 50-s N. Century Lazarev developed a direction for the use in medicine of purine and pyrimidine derivatives of a specific chemical structure, making related them with normal nitrogenous bases of DNA and RNA. Stimulating the synthesis of nucleic acids and proteins, these connections increased physiological and reparative regeneration, biological reactions and some other functions. The most known pyrimidine derivatives - methyluracil and pentauxil, which are applied for treatment of various forms of performance in leukopenia, wounds, burns; methyluracil is used for the treatment of stomach ulcer and duodenal ulcer.
There are various options for molecular interventions to improve the adaptive reactions of the organism in heart failure, and some other disorders. All they are connected with the influence on the synthesis of protein, nucleic acids and provision of energy of these processes. For these purposes it is supposed to use folic and orotovoy acids, vitamin B12, ATF, methionine, cysteine, glutathione and some other substances.
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 9 / 0 · Sep 18, 2013 · Flag

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[**Rob Weijers**](https://www.researchgate.net/profile/Rob_Weijers) · [26.36](https://www.researchgate.net/profile/Rob_Weijers/reputation) · [91.33](https://www.researchgate.net/profile/Rob_Weijers/publications) · [Onze Lieve Vrouwe Gasthuis](https://www.researchgate.net/institution/Onze_Lieve_Vrouwe_Gasthuis)

Dear Max,
I am no professional in the area of orthomolecular medicine. If I have an opinion, it is based on careful study of the related literature and mostly accompanied with self-performed experimental work.
In the field of type 2 diabetes, I promote "diet-lifestyle-exercise" to enhance the correct gene expressions and to minimize medical interventions.

 4 / 0 · May 6, 2014

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Excellent, Rob. Working from the standpoint of gene expression and mitochondrial stimulation is where it's at as far as I am concerned. And in essence that is the orthomolecular medicine approach, also. But in mainstream medical practice what we find is drug theraphy as if it is the end-all and a half-hearted stab at sterile diet that does not approach the richness and detail we would like to see in helping patients overcome their diabetis type 2 condition. Our experience is that about 90% of current sufferers can be reversed with the changes we recommend.

 1 / 0 · May 6, 2014

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[**Jacqueline Feltrin Quintana**](https://www.researchgate.net/profile/Jacqueline_Quintana2) · [2.22](https://www.researchgate.net/profile/Jacqueline_Quintana2/reputation) · [Fundação Universitária de Cardiologia (FUC)](https://www.researchgate.net/institution/Fundacao_Universitaria_de_Cardiologia_FUC)

This Brazilian study found some points that answers your question :
Among the main sources of human exposure to mutagens
and / or carcinogenic can cite the diet , either for the food composition itself or its preparation, use of flavoring , coloring or contaminants therein. Furthermore, the use of certain drugs have potentially harmful , such as the habit of smoking , intake of alcohol , consumption of illicit drugs and pharmaceuticals .Food intake is a major route of human exposure to different compounds , as a complex mixture of chemicals is found in the diet . Some of the substances in certain foods can induce mutations in DNA and may favor the development of tumors Fats present in food , for example , stand out in the carcinogenic process, since
that there are relationships between the intake of foods rich in fats and breast, colon and prostate.Studies by Mohr et al. (17) and Ito et al. ( 18 ) demonstrated that caffeine present in tea and coffee , affects the restorative action of DNA during cell division , alters the apoptotic response and alters the functioning of the p53 gene , which encodes a protein that regulates the cell cycle. Other substance added to food is the acidulant phosphoric acid , which showed , in children , the risk of developing hypocalcemia , a disease that involves a low concentration of calcium in the blood , may cause tetany , subnormal intelligence, depression, problems
cardiovascular diseases, among others.Roberts ( 48 ) reports numerous reactions , before the use of products containing Aspartame , known artificial sweetener present in thousands of products such as headache , seizures , depression , acceleration of Alzheimer's disease , as well as brain cancer , has warned that time , in 1997 , for the removal of such products from public use .

Elisângela Düsman , Alessandra Paim , Berti1Lilian , Capelari Smith , Veronica Elisa , pepper Vicentini Sages . Rev. Biol Health , v.7 , n.2 , p.66 -81 , May / Aug 2012 .

 3 / 0 · May 7, 2014

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Jacqueline, thank you for such a circumspect and sobering reminder on these studies. Hippocrates had it right when he said, "Let thy food by thy medicine, and thy medicine be thy food" (i.e., eat that which is good for us), and, also his dictum, "First, do no harm". In a field that virtually ignores the food connection and which routinely does inherent harm to its patients, we've got our work cut out for us. Thank you, again.

 4 / 0 · May 7, 2014

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[**Diana Rodríguez Hurtado**](https://www.researchgate.net/profile/Diana_Rodriguez_Hurtado) · [12.55](https://www.researchgate.net/profile/Diana_Rodriguez_Hurtado/reputation) · [41.25](https://www.researchgate.net/profile/Diana_Rodriguez_Hurtado/publications) · [Universidad Peruana Cayetano Heredia](https://www.researchgate.net/institution/Universidad_Peruana_Cayetano_Heredia)

Dear Max:
I think this topic is very interesting.
I was reading and I found these articles .
1.- M. Raghunat, L. Veme, I.J.N Padmavathi, Y.D Kishore, M. Ganeshan, K. Anand kumar, P.B. Sainath & K.R Kao
Modulation of macronutrients metabolism in the offspring by maternal micro nutrient deficiency in experimental animals.
Indian J Med Res 130, November 2009, pp 655-665.
2.- Davis Kibirige and Raymund Mwebaze
Vitamin B12 deficiency among patients with diabetes mellitus: is routine screening and suplementation justified?
Journal of Diabetes & Metabolic Disorders 2013, 12:17.
Sincerely.
Diana from Perú.

 3 / 0 · 24 days ago

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Excellent work, Diana. One of the weak spots in screenings like this is that I do not find the testing protocols used for most nutrients to be very reliable. We keep assuming that the vitamins and trace elements we are looking for are identical to their synthetic cousins. Vitamin D, for instance, of the type the body synthesizes is totally different from the D2 form looked for in the lab. A miniscule amount of the "super D" the body makes on its own is immensely richer and more bioavailable than the toxic quantities that people are advised to take in the prescriptive D2 form. The same with the other vitamins taken in their organic, bioavailable form and synthetic forms. It is so difficult to duplicate the immense complexicity and bioavailability of naturally occuring nutrients into a testable form. We know we have advanced in nutrition when we can tell these two vastly differing forms apart in routine blood tests.

 5 / 0 · 24 days ago

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Thank You.

 3 / 0 · 20 days ago

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[**Michael J Gonzalez**](https://www.researchgate.net/profile/Michael_Gonzalez3) · [33.48](https://www.researchgate.net/profile/Michael_Gonzalez3/reputation) · [56.35](https://www.researchgate.net/profile/Michael_Gonzalez3/publications) · [University of Puerto Rico, Medical Sciences Campus](https://www.researchgate.net/institution/University_of_Puerto_Rico_Medical_Sciences_Campus)

Great comments! I feel humble and honored by the excellent, in -depth discussion of our article on metabolic correction...more to come!

3 / 0 · 19 days ago

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[**Arif Aliovsad Mekhtiev**](https://www.researchgate.net/profile/Arif_Mekhtiev) · [16.28](https://www.researchgate.net/profile/Arif_Mekhtiev/reputation) · [12.64](https://www.researchgate.net/profile/Arif_Mekhtiev/publications) · A.I. Karaev Institute of Physiology, Azerbaijan National Academy of Sciences

From my standpoint, intrinsic milieu may as well exert harmful effects on genetic apparatus as external adverse conditions do. As food ingredients significantly participate in formation of internal milieu of the organism, so they may, on one hand, bring to serious pathology and, on the other hand, be a powerful tool in getting back pathological process and recover to original healthy state.

 2 / 0 · 10 days ago

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Good points, Arif. Nutrition is best when it supports genetic needs. In our work, for instance, we have found some genetics and cultures need more omega-3 (complete EPA echeisonoid families and DHA fatty acid families both) than others. Others may need a lower Omega 3 to 6 ratio for optimal genetic needs. But the foundation seems always go to strengthening the telomeres of the DNA and giving the mitochondria the elements needed ATP used for homeostasis and repair. That is one of the reasons a single food pyramid for the entire population is doomed to fail, bringing with it (as it has in our country) escalating numbers of chronic disease sufferers. The milieu as you say varies from individual to individual and from genetic group to another. The blind eye to these variations in allopathy are testament to its miserable failure at bringing wellness. Thank you for your kind contribution.