Original Article

Personalized Food Avoidance Dietary Approach for Psoriasis and Hypertension: A Case Study and Pilot Trial

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ABSTRACT

Background: The comorbidity, coheritability and common immune pathways of the inflammatory 'chronic' (non-communicable) diseases, suggest a shared aetiopathogenic mechanism, with phenotypic localization dependent on genetic predisposition, for example, the arteries in hypertension and the skin in psoriasis. Lifelong observation of psoriasis suggests genetically predisposed toxicity of amphiphilic fats, flavor enhancers, and nonsugar sweeteners. **Aim:** To conduct trials of personalized dietary intervention to abate the phenotypic expression of psoriasis and hypertension. **Materials and Methods:** The interventional case study of psoriasis was conducted by means of repetitive dietary challenge and avoidance testing. We then conducted an open trial of personalized nutrition on nine consenting recruits with uncomplicated essential hypertension. They were counseled on which foods to avoid or to take based on the experience gained in psoriasis. Study participants with 'GOOD' or 'FAIR/POOR' dietary compliance were compared with regard to blood pressure (BP) control, antihypertensive drug treatment requirement (ADTR), and anthropometry. **Results:** Data from four FAIR/POOR diet compliers and three GOOD compliers, as at 29 weeks of dietary intervention, showed mean systolic home B*P* values of 128.1 (±6.74) mmHg and 122.3 (±2.03) mmHg, respectively; the mean systolic automated office B*P* values were 139.8 (±8.80) mmHg and 108.3 (±5.55) mmHg, respectively; as at 39 weeks of dietary intervention, the mean ADTR scores were 4.2 (±2.12) and 1.03 (±0.57), respectively. Using data at baseline and from all available timepoints after dietary intervention, two-way ANOVA confirmed highly significant improvement of BP control ($P < 0.0001$) and reduction of ADTR score ($P = 0.0008$) in GOOD compliers. GOOD compliers exhibited significantly more reductions in BMI, abdominal circumference, and triceps skinfold thickness than FAIR/POOR compliers (two-way ANOVA and linear regression: $P < 0.05$). **Conclusion:** These results support the case for adverse dietary exposure avoidance to abate the phenotypic expression of chronic disease.

Keywords: *Anthropometry, dietary intervention, hypertension, personalized nutrition, psoriasis,*

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Introduction

The pandemic of noncommunicable diseases[1-3] is attributable to human lifestyle changes, which accompany increasing urbanization and industrialization.[4] Further research to elucidate the etiopathogenic mechanism would help control the pandemic.

Various observations suggest that noncommunicable diseases share a common epigenetic etiologic mechanism, in which the gene–environment interaction promotes proinflammatory immune dysfunction and phenotypic expression of chronic disease, with tissue localization according to genetic predisposition. First, epidemiological studies show that chronic diseases are often associated: hence, for example, the entity referred to as 'metabolic syndrome', and the 'metabolic syndrome' – associated immune – mediated inflammatory diseases (MSA-IMIDs).[5-7] Second, studies of population dietary habits show that the benefit of 'healthy' foods, for example, the traditional Mediterranean diet, extends across the broad spectrum of noncommunicable diseases.[8-10] Myles has reviewed the adverse impact of Western diet on immune function.[11] Third, there is striking pharmacotherapeutic overlap between some of the chronic diseases, for example, the efficacy of statins in rheumatoid arthritis and psoriasis[12,13] and that of lisinopril in male infertility.[14] This treatment overlap also supports the case for a common etiopathogenic mechanism. Finally, different MSA-IMIDs have been shown to share similar T-lymphocyte-mediated immunocytopathogenic pathways, as in the case of psoriasis and hypertension, for example.[15,16]

Psoriasis is notable, among the MSA-IMIDs, for enabling direct observation of the chronic disease process expressing in the skin. This inspired life-long observations on the dietary dependence of the phenotypic expression of psoriasis (with eschewal of suppressive medications). Egregious dietary ingredients consistently engender relapse of psoriasis.[17,18] The slow onset of this relapse (1–3 days) and the slower remission (1–3 weeks) upon strict avoidance of the culprit ingredients are consistent with an epigenetic mechanism.

Further serendipitous observation indicated side benefits of the antipsoriasis personalized food avoidance dietary approach not only on associated features such as nail changes and arthropathy but also on blood pressure (BP) and other associated metabolic parameters.[17-20] This supports a nutritoxi-epigenetic etiopathogenic mechanism for the MSA-IMIDs. The Dietary Intervention Research Group was established to investigate this hypothesis.

Methods

Two decades of textual and photographic diary records in psoriasis form the basis of an inductive model of the diet–genome interaction which incubates immune dysfunction and chronic disease. The interventional case study of psoriasis was conducted by the first author on himself, mainly between 2004 and 2014. The open trial of personalized dietary intervention for essential hypertension was a prospective cohort study conducted between 2015 and 2018. There were retrospective controls, as determined by *post hoc* Dietary Compliance scores. Thus study participants with FAIR/ POOR compliance were deemed to be controls and compared with GOOD compliers. The settings for the open trial include the Teaching Hospital for recruitment (from medical outpatient clinic), exercise assessments, pharmacy assessment and echocardiography. There is a separate clinical research centre, incorporated as a not-for-profit non-governmental organisation. Blood and urine samples were collected at the old hospital site and processed in a private laboratory.

Sample size calculation was based on the pilot case studies in psoriasis and hypertension,[17-20] which demonstrated reversibility of the chronic disease process with good dietary compliance. Say, for example, that the study participants have a mean blood pressure of 150 mmHg (SD 10 mmHg) at recruitment. Then, to detect a 20 mmHg reduction in 'GOOD' diet compliers compared to 'POOR' compliers, as being statistically significant, would require five subjects in each group, assuming that single measurements of blood pressure and antihypertensive drug treatment requirement are undertaken in each study participant before and after the dietary intervention (clincalc.com/stats/samplesize.aspx).[21,22] Repeated measures of these parameters (as was the case in our study) would correspondingly reduce the number of participants required.

Patients attending the medical outpatients clinic were sequentially referred as prospective recruits for the Personalized Food Avoidance Dietary Approach to Stop Hypertension (PFADASH) trials. Those included had an average automated office blood pressure (BP) above 160/100mm Hg (140/90 if additional vascular risk*), regardless of treatment or drug adherence, OR they were on two antihypertensive drugs (good adherence) with

average BP above 140/90 (130/80 if additional vascular risk*), OR they were on three or more antihypertensives (good adherence), regardless of whether BP controlled.

*e.g. diabetes, chronic kidney disease, sickle cell disease, history of heart attack, stroke, TIA, heart failure.

Further inclusion criteria included adulthood (age 18 and above, not pregnant), being sufficiently literate to follow detailed dietary instructions (both verbal and textual), freely given full informed consent, and ability to undertake their normal daily activities and attend the outpatient clinic or clinical research centre without difficulty.

Nine eligible consenting adults (baseline demographics in Suppl Table 1 [see Additional file 19]) with uncomplicated essential hypertension were thus recruited sequentially for the pilot open trial of the Personalized Food Avoidance Dietary Approach to Stop Hypertension (PFADASH), being a 'phase 6' clinical trial, the main intervention being withdrawal from putatively egregious dietary exposures. The study participants attended the clinical research centre for baseline and monthly follow-up assessments which include blood pressure measurement (automated office blood pressure, AOBP, with Omron HEM907, home BP monitoring with various devices e.g. Omron M2, Foracare AG Suisse, validated against the AOBP monitor; manual office BP using Accoson mercury column sphygmomanometer), anthropometry (body mass index, abdominal and mid-upper arm circumferences, triceps skin fold thickness using Harpenden's skinfold callipers), routine clinical follow-up (for adverse events or side-benefits of antihypertensive dietary intervention), and recording of all drug and toxin exposures, including antihypertensive drug treatment requirement (ADTR).

For ethical reasons, study participants were kept on whatever antihypertensive drug treatment they required, while they embarked upon the personalized food avoidance dietary intervention. This would reduce the utility of blood pressure (BP) control as an outcome parameter, since BP control may be good on drug treatment ab initio, while later on BP control remains good on dietary intervention with a reduced need for antihypertensive medication. Hence antihypertensive drug treatment requirement (ADTR) shall also be scored as an outcome parameter, initially by adding up unitary daily dosages defined as follows: hydrochlorthiazide 25mg, amlodipine 5mg, nifedipine 20mg, atenolol 25mg, prazosin 1mg, lisinopril 5mg, enalapril 5mg, ramipril 2.5mg, losartan 25mg, telmisartan 20mg, frusemide 20mg, methyldopa 250mg, valsartan 80mg. diuretic max = 2 ('ceiling' effect). Adherence shall be measured as number of doses taken (determined by asking the SP and confirmatory capsule count) divided by number of doses prescribed over the relevant time period. Actual antihypertensive drug treatment (AdhRx score) is then number of unitary dosages of antihypertensive times adherence. Antihypertensive drug treatment requirement (ADTR score) is finally arrived at by adding 0.1 to AdhRx score for every mmHg that average systolic AOBP or monthly average home BP (whichever is higher) exceeds 120 mmHg. Conversely, 0.1 should be subtracted for each mmHg that average systolic AOBP or home BP (whichever is lower) is below 100 mmHg. Using the Shapiro - Wilk Statistic (in SPSS), both the AdhRx and ADTR scores showed more than 80% likelihood of being normally distributed at 5% significance level.

The following assessments were undertaken at baseline and repeated every 3 months, or as soon thereafter as logistically feasible i.e. at least every 6 months. Clinical assessment includes a comprehensive clinical history and physical examination, with a focus on adverse clinical outcomes in those dropping out of the clinical trial. Nutritional characterisation of study participants aimed to qualify and quantify macronutrients and micronutrients, with special attention to method / temperature of cooking, nature of fats / oils used, flavourant and sweetener additives. Pharmaceutic trimestrial assessments record medication intake in detail, including dosages, adverse effects, traditional / herbal medicines, nutraceutical supplements, with independent validation of the ADTR score. Exercise assessments include filling the International Physical Activity Questionnaire [http://www.sdp.univ.fvg.it/sites/default/files/IPAQ_English_self-admin_long.pdf] to quantify usual exercise habit. This was followed by a treadmill test to measure maximal exercise ability (HK- 2008 3.0 HP electrical treadmill, manufactured by Shandong Huikang Sports Equipment Co. Ltd in 2008). Sociology assessments address the quality of life of study participants, focusing particularly on indices of socio-economic status, social isolation and sleep deprivation. Echocardiography was undertaken with calculation of left ventricular ejection fraction, interventricular septal thickness, left ventricular diastolic function (E/E' ratio), right ventricular diastolic function (E/A ratio). Laboratory studies were undertaken at baseline and repeated at 6 - 12 months intervals. These include fasting serum lipids and blood sugar, uric acid, albuminuria, inflammatory markers. Study participants with significant abnormalities in their clinical or laboratory assessments were advised to see their physician.

Regular 'training of trainers' sessions were conducted by the Principal Investigator at the Teaching Hospital old site, training PFA-DASH dietary counsellors to whom study participants will be primarily and

secondarily assigned for regular counselling and follow-up. The training was based on the lifelong personal observational experience described above.[17-20] In summary, the dietary intervention emphasises the need to carefully differentiate between harmful and beneficial oils and fats. Trans-fats, hydrogenated / oxidised fats, adulterated oils, emulsifiers should be strictly avoided. A simplified strategy would be to avoid oil unless one is certain of the source, method/ temperature of extraction i.e. it should be cold-pressed oil, not subjected to high temperature (frying, grilling, baking etc.) Glutamatergic flavour enhancers and non-sugar (aspartatergic) sweeteners should also be avoided, albeit on a personalized basis, in view of tolerance variability between individuals. The Dietary Counselling and Compliance Assessment and Scoring (DCCAS) guidelines are Bimonthly DCCAS meetings were held at the hospital old site to reinforce the whole process and encourage dietary compliance.

Study participants (SPs) with 'GOOD' dietary compliance were compared with 'FAIR / POOR' diet compliers (controls) as regards:

Blood pressure control (AOBP, home BP)

Antihypertensive drug treatment requirement (ADTR score)

Exercise habit and ability

Anthropometric indices (BMI, abdominal circumference, triceps skinfold thickness)

Echocardiographic indices (listed above)

Quality of life indices (Socio-economic status, Social isolation and sleep deprivation)

Laboratory parameters (urine analysis, electrolytes, albumin, creatinine; full blood count, renal and liver function tests, serum urate and fasting lipids, fasting blood sugar)

Data confidentiality was ensured by keeping the study participants' file jackets in a locked room at the clinical research centre. Access to the files was strictly limited to requirements for running the research programme, and for data computerization.

Data will be computerized using Microsoft Excel spreadsheets, and then later plotted and analysed using GraphPad Prism v5 and Statistical Package for Social Sciences (SPSS) v19. Statistical techniques used included 2-way analysis of variance (with repeated measures, as appropriate) and linear regression, for the comparison of treatment outcome of the various groups, as the data are normally distributed. The conventional significance level of $P < 0.05$ (95% confidence) will be used in the statistical significance testing between the two dietary compliance groups.

Ethics approval and clinical trial registration

This clinical trial proposal was approved by the University of Nigeria Teaching Hospital Health Research Ethics Committee (certificate no. NHREC/05/01/2008B-FWA00002458-IRB00002323, dated 28th July, 2012, renewed on 28th July, 2015 and 23rd January, 2017) and was registered at www.clinicaltrials.gov (Trial Registration Number: NCT02136264).

Results

Diary records on generalized psoriasis, as from 2002, include the realization that diet has a marked impact on the phenotypic expression of the disease (personal diary observations: see Additional file 2). A total of 101 photographs of truncal and elbow psoriasis (see Photographic Series, Additional File 3) show a time lapse sequence spanning 14 years. They are an extract from occasional skin surveys which portray insidious aggravation and ensuing abatement of generalized psoriasis by dint of repeated dietary challenge and avoidance experimentation, with strict eschewal of suppressive drug treatments. Side benefits of the antipsoriatic food avoidance dietary approach on BP and associated parameters are shown in Suppl Figures 1 and 2 [see Additional Files 4 and 5].

Figure 1 demonstrates the efficacy of a personalized food avoidance dietary approach in a 45-year-old man

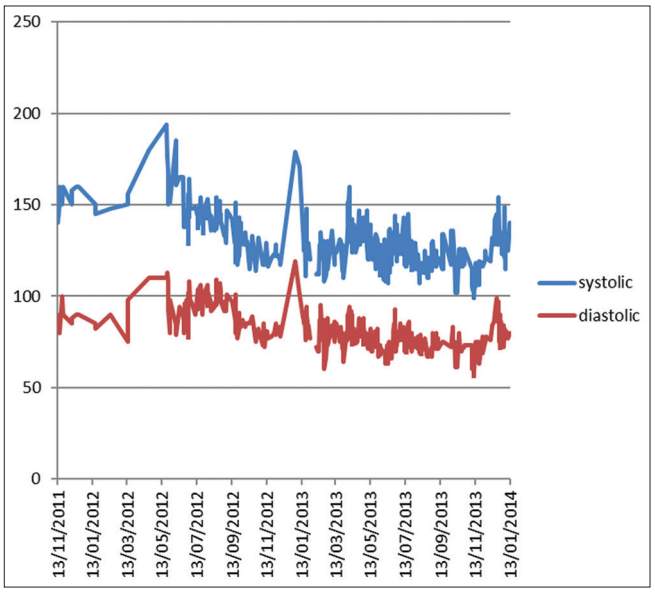

Figure 1: PFADASH in a propositus intolerant of antihypertensive medication. 168 blood pressures were recorded over 2 years with an Omron M2 device. A paired samples *t*-test shows a statistically significant difference between the first 6 months of 2012 and the last 6 months of 2013 for both the systolic ($P = 0.004$) and diastolic ($P = 0.017$) pressures. (PFADASH: Personalized Food Avoidance Dietary Approach to Stop Hypertension)

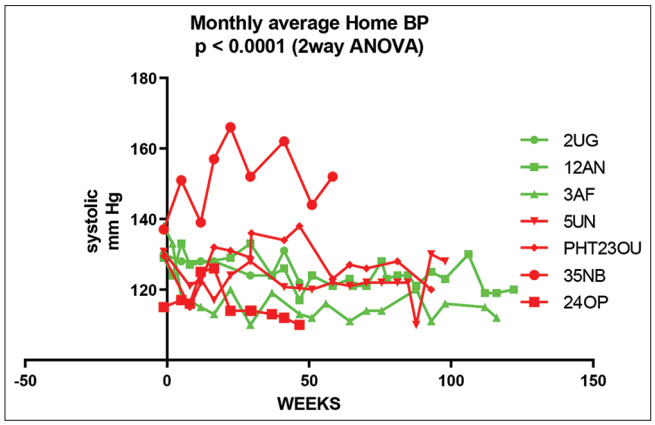

Figure 2: Monthly average systolic home blood pressure in GOOD diet compliers (green) compared to FAIR/POOR compliers (red). 2-ways ANOVA: $P < 0.0001$. (BP: blood pressure; ANOVA: analysis of variance)

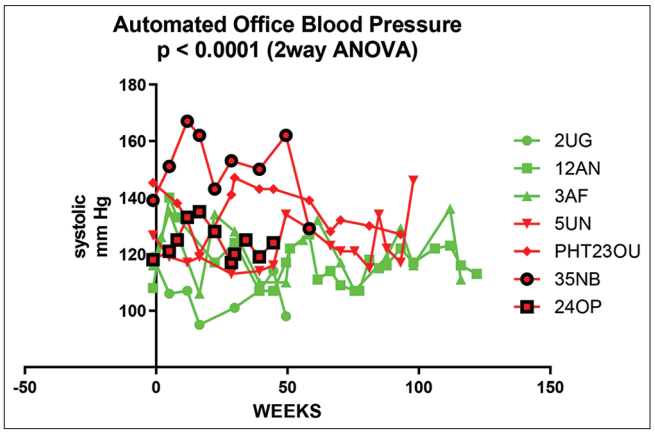

Figure 3: Comparison of average automated office blood pressure measurements from GOOD diet compliers and FAIR/POOR diet compliers. Two-way ANOVA: $P < 0.0001$. (BP: blood pressure; ANOVA: analysis of variance)

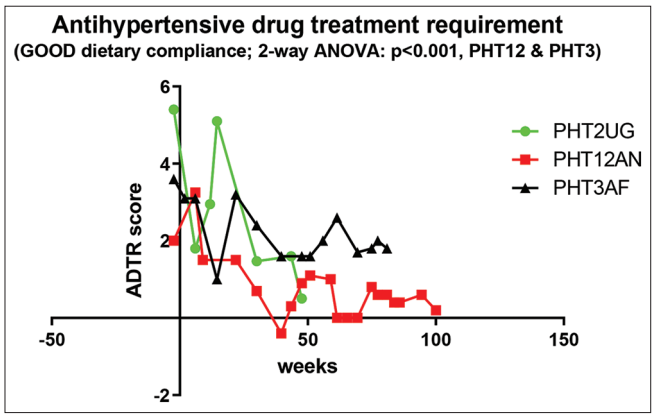

Figure 4: Antihypertensive drug treatment requirement score in three study participants with GOOD dietary compliance score. Two-way ANOVA shows a significant decline during the study period in two participants (BP: blood pressure; ANOVA: analysis of variance)

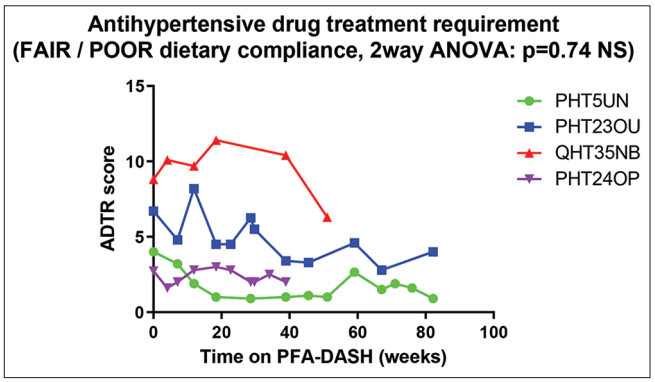

Figure 5: Antihypertensive drug treatment requirement score in four study participants with FAIR/POOR dietary compliance score. Two-way ANOVA shows no significant decline with time (BP: blood pressure; ANOVA: analysis of variance)

with hypertension. He was strongly motivated toward a dietary solution, being intolerant of antihypertensive medication on account of adverse effects.

Nine recruits for the pilot open trial were on antihypertensive drugs. Two dropped out within 2 months (overseas travel, complete heart block). Hence, data for analysis were available in seven study participants, as displayed in Figures 2-5 and Suppl Figures 3-15 [see Additional Files 6-18]. These figures show statistically significant differences between 'GOOD' and 'FAIR/POOR' diet compliers as regards blood pressure control, antihypertensive drug requirement, anthropometric features, usual exercise habit, maximal exercise ability, and left ventricular systolic function.

Available laboratory data from second morning urine, whole blood, plasma, and serum samples of study participants are tabulated [see Suppl Appendix 2, Additional File 20]. These data are from before PFADASH dietary intervention started and 14–29 months after dietary intervention started.

Sociological quality of life parameters for study participants [shown in Suppl Table 2, Additional File 21] did not change during the course of the clinical trial

For their 2–3 years of PFADASH intervention so far, no adverse clinical outcomes have been reported in the seven ongoing study participants.

Discussion

Lifelong observational study of psoriasis inspired an inductive model of the diet – genome interaction which incubates the chronic disease process. This model postulates that amphiphilic fats potentiate glutamatergic and aspartatergic (i.e. excitotoxic amino-acid – mediated) adverse gene expression, with secondary intolerances to foods with early-life immune unfamiliarity. In this regard, we have demonstrated chronic toxicity of low dose glutamate in rats.[23] Toxicant-induced loss of tolerance was originally postulated by Miller.[24]

In the ongoing live case study of essential hypertension [Figure 1], it took *ab initio* 6 months

for the propositus to effectively comply with dietary counseling. There have been significant decline of BP with time, attainment of normal BP, relapses attributable to dietary indiscretion, and remissions with restoration of dietary compliance. The consistency and reproducibility of the dietary intervention response are consistent with the postulated nutritoxi-epigenetic etiopathogenesis of hypertension, that is, genetically predisposed, diet-mediated causation.

This pilot open trial of PFADASH is a 'phase 6' trial of withdrawal from putatively toxic chronic dietary exposures. We compared study participants exhibiting 'GOOD' dietary compliance with 'FAIR/POOR' diet compliers (as *post-hoc* controls). For most parameters studied, there was a significantly better response in 'GOOD', diet-compliant study participants, as compared to 'FAIR/POOR' compliers. Automated office BP and home BP control were significantly better in 'good' compliers. Antihypertensive drug treatment requirement was significantly reduced in 'good' compliers, but this was not the case for 'poor' compliers. There was greater improvement of anthropometric parameters in 'good' diet compliers. Usual exercise habit and maximal exercise ability improved significantly over a year in two 'good' and one 'fair' diet complier. Out of the echocardiographic systolic and diastolic function indices, only left ventricular ejection fraction showed significant improvement over 18 months, in three 'good' compliers and one 'poor' complier. These results are consistent with reversibility of systemic hypertension in 'good' diet compliers, in view of the improved blood pressure control, the remarkable reduction in antihypertensive drug treatment requirement, and the improvement in anthropometric parameters, exercise ability and systolic function.

The limited range of laboratory results in six study participants showed macroalbuminuria in two with 'POOR' dietary compliance and microalbuminuria in four SPs with 'GOOD'/'FAIR' compliance. Serum urate was raised in one 'GOOD' complier and in one 'POOR' complier. Raised fasting serum LDL in PHT12AN normalized after the PFADASH intervention. In five study participants, serum LDL was 3.2 mM (123.7 mg/dl) or less after the dietary intervention.

The observations and analyses above are in accordance with the findings of epidemiological surveys on diet and health, including hypertension and cardiovascular outcomes.[8-10,25-28] The PURE studies[9,10] showed that total fat intake and types of fat were not associated with mortality or adverse cardiovascular outcome, while fruit, vegetable, and legume consumption was beneficial. The EPIC Spain study[25] showed that egg consumption (up to one a day) is not associated with mortality in a large Mediterranean population. The E3N EPICN study[26] shows an independent association between chronic consumption of artificial sweeteners and type 2 diabetes risk. Meta-analyses have shown that the purported association between red meat and colorectal cancer applies to processed meats rather than fresh red meat.[27] Our dietary recommendations concur with these epidemiological findings, except for the need to strictly avoid trans /hydrogenated/oxidized fats, which were not specifically addressed in the PURE study.[9] Had the PURE study[9] differentiated between 'good' and 'bad' fats, then a beneficial effect of the former may have been apparent. A Cochrane review[28] found that regular omega 3 fish oil supplements were of no benefit on mortality and cardiovascular events. Our dietary recommendation differs with this finding in that as regards psoriasis and occasional (say weekly) rotational consumption of MUFA/PUFA as a macronutrient was beneficial, while regular micronutrient supplementation with fish oils and so on was counterproductive and even sustained relapse of psoriasis (Chijioke CP, personal observations and web log). Occasional rotational consumption of immune unfamiliar MUFA/PUFA/SFA reduces the likelihood of secondary intolerances (which would account for the paradoxical inefficacy of regular micronutrient supplementation, at least insofar as the intuitive model of the diet–genome interaction is concerned).

Apart from correcting the proinflammatory immune dysregulation (measurable as the visceral adiposity index[29]) which mediates chronic noncommunicable disease, the PFADASH, by curbing immune dysfunction, may also improve immune responsiveness to communicable diseases and antineoplastic immunosurveillance. For instance, the severity of Coronavirus Disease 2019 and its mortality are worse with increasing age and chronic disease comorbidity.[30] These are harbingers of proinflammatory immune dysfunction, which is, hypothetically, opportunistically exacerbated by SARS-CoV-2. Indeed, the importance of strategies to boost immunity has recently been emphasized.[31] Even with vaccination against SARS-CoV-2, personalized diet mediated immune optimization strategies would still be helpful to enable, promote, and prolong the efficacy of active immunization, especially with the advent of mutant strains which are more contagious and more virulent and for which the vaccines may be less effective.

Hence, we recommend clinical trials and community studies of the efficacy and safety of the PFADASH intervention strategy versus both communicable and noncommunicable diseases.

The chronic diseases are polygenic: Hence, there is little hope for benefit from genome-wide association studies[32] and genetic engineering. In any case, these genes may confer a survival advantage in times of famine and deprivation ('thrifty gene' hypothesis) such that tinkering with our genetic make up would pose ethical dilemmas. Undue emphasis on the genetic aspect of the gene–environment interaction encourages phenotypic ablation manoeuvres such as mastectomies or prostatectomy for cancer prevention and renal sympathetic denervation for refractory systemic hypertension.

It is noteworthy that such procedures are less appealing than addressing the environmental factor in the causative mechanism of disease. Our proposed approach to addressing the environmental factor of the gene–environment interaction in the etiopathogenesis of immune dysregulation and chronic disease/cancer offers the prospect of preventative health benefits for humanity in the foreseeable future. This 'phase 6' food safety clinical trial, although focused as a 'proof-of-concept' study in essential hypertension, potentially addresses the worldwide waxing swathe of chronic diseases, infectious diseases, and cancers.

Ancient wisdom presages awareness of nutritoxi-epigenetics, being enshrined in well-known aphorisms, such as "You are what you eat.", "Let food be thy medicine and medicine be thy food." (Hippocrates c. 460–377 BC), and "Quod ali cibus est aliis fuat acre venenum: what is food to one man may be fierce poison to others." (Lucretius c. 99–55 BC: De Rerum Natura. iv. 637.)

Study limitations

This pilot trial for hypertension was non-randomized with retrospective controls. Although a randomized controlled trial provides better evidence, random assignation of study participants to a modern fast-food diet would be ethically questionable. However, a randomized controlled trial could compare interventional PFA-DASH with conventional DASH (including scoring of dietary compliance in both groups).

Efforts were made to ensure that potential confounder variables (e.g. sociological factors, exercise, exposure to tobacco, alcohol and native / herbal medicines) remained constant for the duration of the study. However the data gathered for potential confounder variables were too few to allow conclusive analysis.

The number of study participants was too small, and the duration of follow-up too short to enable meaningful assessment of hard clinical endpoints such as cardiovascular morbidity and mortality. However the seven recruits have remained well for the duration of the trial, with general health improvement, particularly in those with 'GOOD' dietary compliance. For the soft endpoints (blood pressure control, antihypertensive drug requirement, anthropometry), the multiplicity of measurements conferred adequate power for meaningful statistical analysis.

There were funding constraints which limited our ability to recruit and follow-up SPs, in particular as regards laboratory studies.

Conclusions

We have reported our pioneer studies of personalized nutrition to address immune dysfunction and chronic disease, comprising the lifelong documented personal observational case study of psoriasis and a pioneer 'phase 6' clinical trial of withdrawal from harmful dietary exposures in hypertension. We are not aware of previous such reports or publications.

The results of our observational studies and clinical trial support a nutritoxi-epigenetic (diet-mediated) cause of immune dysfunction and chronic disease viz psoriasis and hypertension. Study participants with 'GOOD' compliance show marked reduction in ADTR, with improved blood pressure control, an increase in exercise ability, and improved cardiac systolic function.

As the PFADASH intervention excludes some aspects of modern dietary trends, it may not be of therapeutic value in patients lacking motivation. However, our data do support a diet–genome interaction mechanism in the etiopathogenesis of chronic disease. Further confirmatory studies would pave the way for advocacy, regulation, or legislation on harmful ingredients to help stem the increasing tide of noncommunicable and communicable diseases worldwide.

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labour and generator costs for routine serum, plasma and urine laboratory analyses.
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7. Dept of Pharmacology and Therapeutics, College of Medicine, University of Nigeria: laboratory facilities (including cold centrifuge) were used free of charge.
8. DIRG (Dietary Intervention Research Group): investigators made sacrifices of their time, effort and finances, in particular the PI (Prof. Chijioke), the research group coordinator (Dr MT Okafor), the laboratory coordinator (Ms IC Onah), the PFADASH counseling coordinator (Mrs NI Nubila), the PFADASH study coordinator (Ms BA Nwokolo), and the Head, Dept Pharmacology and Therapeutics (Can Dr CA Anusiem)
9. CIMRO (Chiolive International Medical Research Organisation) made its premises available free of charge for running the dietary intervention clinical trials.
10. Statistical test for normality of ADTR score was undertaken by Mr N. Ugwu.
11. The diagnosis and topical treatment of index case psoriasis by Professor A.N. Okoro, Professor G. Ozoh and other dermatologists for about 35 years
12. We appreciate the enthusiasm and cooperation of our study participants who sacrifice time and effort for the project, and also family members and caterers who accede to dietary requests.

Ethics committee approval and consent to participate

Ethics approval was obtained for the clinical trial of dietary intervention for hypertension, including written informed consent by all study participants.

The clinical trial proposal was approved by the University of Nigeria Teaching Hospital Health Research Ethics Committee (certificate no. NHREC /05/01/2008B-FWA00002458-IRB00002323, issued 28th June, 2012, renewed on 28th July, 2015 and 23rd January, 2017).

List of abbreviations

ADTR: Antihypertensive Drug Treatment Requirement
BP: Blood pressure
COVID-19: Coronavirus disease 2019
DASH: Dietary Approach to Stop Hypertension
DCCAS: Dietary counselling and dietary compliance assessment and scoring
MSA-IMIDs: 'metabolic syndrome' – associated immune-mediated inflammatory diseases
PFADASH: Personalized Food Avoidance Dietary Approach to Stop Hypertension
SPs: Study participants.

Financial support and sponsorship

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Foracare AG Suisse donated 40 home BP monitors. Study investigators sacrificed time, effort, space and funds to support this work.

Conflicts of interest

There are no conflicts of interest.

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Suppl Appendix 1: (extract from study protocol): Dietary Counseling and Compliance Assessment and Scoring (DCCAS) guidelines

It is important to note that the dietary guidelines below are based on the personalized dietary requirements of the corresponding author. These guidelines need to be personally adjusted for each study participant, bearing in mind the general rules that amphiphilic fats and oils must be avoided, while glutamatergic flavour enhancers and aspartatergic sweeteners should be minimized. The degree of minimization required would vary between individuals, based on nutrigenetic differences, affecting perhaps the nutrikinetics of flavourants and sweeteners, for example. Hence the dietary guidelines below should not be misconstrued as a general prescription or proscription of the foods and ingredients mentioned. They should rather be taken as a guide and subjected to modification (personalization) according to individual variability in response to the foods, drinks and ingredients concerned.

Plain food diet: no dairy (no milk, no cheese, no yoghurt), no oil (unless cold-pressed oil, not subjected to high temperature e.g. frying or baking), NO FRIED or GRILLED FOOD, no flavour enhancers, NO MSG, no sweeteners*, no emulsifiers, no hot spices, plain boiled vegetables (no butter, no margarine). Salt is allowed, according to daily requirements (maximum 5g per day). Certain additives / spices e.g. ginger may be of benefit, especially if there is early life / immune familiarity on the part of the study participant. Food should be boiled, steamed, microwave or poach (to cook food), or bake at carefully controlled temperature: no burning, no charring, no frying, no grilling, NO OIL, NO GREASE (subject to provisos above). With the above conditionalities, a wide range of vegetables, salad items, fruit, unrefined starch, protein sources (meat, pulses, poultry, some fish) is permitted. There are a few exceptions, for which personal experience (cpcpsoriasis.blogspot.com) suggests egregious ingredients e.g. broad beans, sour-sop, mangoes, banana, ripe plantain, sweet grapes, papaya, wines, vinegar, champagne. Detailed food classification with further such details is available from the corresponding author upon request.

*i.e. no non-sugar sweeteners, whether artificial or naturally occurring, as in certain very sweet fruit. Ordinary sugar (sucrose, less so, fructose) e.g. demerara sugar is permissible, with strict moderation, tailored to calorie requirements. Complex carbohydrates are preferable of course.

Summary

No trans-/hydrogenated/oxidized fats. Avoid oil unless KNOWN source and method/ temperature of extraction i.e. cold-pressed, not fried, grilled or baked. Plain food diet: no dairy, no additives (unless approved), no fried/grilled/baked oil, NO FRIED FOOD, no flavour enhancers, NO MSG, no sweeteners, no emulsifiers, no hot spices, plain boiled vegetables (no butter, no margarine). Little salt. Boil, steam, microwave or poach (to cook food), or bake at LOW oven temperature: no burning, no charring, no frying, no grilling of OIL, NO GREASE.

Based on these dietary guidelines, a Personalized Categorical Food Avoidance Dietary Approach to Stop Hypertension (PCFA-DASH) chart is constructed for each study participant, considering 3 categories of foods:

A) Primary culprits to be avoided:

Amphiphilic fats e.g. hydrogenated fats, oxidized fats, trans-fats, emulsifiers

Glutamatergic/ aspartatergic agonists

B) Secondary (facultative) culprits [early life – unfamiliar, food dislikes, autacoids, flavourant, sweetener content (e.g. glutamatergic/ aspartatergic partial agonists), preservatives e.g. ethanol, nitrites, sulphites, unduly frequent/ high dose consumption of normally well tolerated foods], **processed foods**

Occasional, low-dose, infrequent short-term consumption may be safe and may encourage immunotolerance. Unduly frequent and repetitive consumption of foods promotes immune intolerance, especially if there is early-life unfamiliarity with the food concerned. NB: this accounts for paradoxical persistence or aggravation of the chronic disease process due to unduly frequent and repetitive consumption of recommended 'good' foods or 'superfoods', **especially early-life unfamiliar proteinaceous foods**.

Occasional rest from such unduly frequent consumption encourages immunotolerance. Tolerance of 'unfamiliar' molecules in a food would promote beneficial effect of other molecules in the same food e.g. onubu ('bitter leaf'), 'good' oils.

C) Safe foods and drinks

Well tolerated, early life familiarity (especially in utero), beneficial effects

Compliance with the PFA-DASH dietary guidelines is obviously a crucial determinant of response to the dietary intervention. To promote the objectivity of Dietary Counselling and Compliance Assessment and Scoring (DCCAS) by the dietary counsellors, up to three counsellors were assigned to each study participant (SP) i.e. one counsellor primarily assigned and two secondarily assigned. Each counsellor employed three different methods of evaluating PFA-DASH Dietary Compliance:

1) From the comprehensive dietetic assessment pro formas (baseline and trimestrial follow-up) with qualitative and quantitative nutritional characterisation of study participants.
2) From the dietary blog records of SPs (dietary experimentation away from the strictly agreed MENU and PCFA CHART compiled for each SP i.e. beneficial adventures and harmful misadventures)
3) By direct interview and discussion with the SP, guided by the agreed MENU and PCFA CHART, as well as by the dietetic assessment pro formas and the dietary blog records.

Three independent Dietary Compliance scores (based on the above) are then discussed and reconciled into a final agreed score by the respective counsellors

DCCAS proceeds on a continual basis for periods of two months punctuated by bimonthly meetings for further training, and reconciliation of Dietary Compliance scores.

DIETARY COMPLIANCE SCORING METHOD (PFADASH Personalized Food Avoidance Dietary Approach to Stop Hypertension)

NB: very good compliance with **occasional / rotational** consumption of so-called 'good' fats /oils would favourably adjust borderline scores. Frequent, repetitive consumption however, may be counter-productive. Discuss with PI / PFADASH coordinator if need be.

Agreed scoring method (simplified version)

GOOD dietary compliance means that there is established dietary indiscretion less than once a month

FAIR dietary compliance means that there is established dietary indiscretion less than once a fortnight

POOR dietary compliance means that there is established dietary indiscretion once a fortnight or more frequently

Agreed scoring method (full version)

GOOD dietary compliance means that there is established MAJOR (category A) dietary indiscretion less than once a month [OR minor (category B) indiscretion less than once a fortnight].

FAIR dietary compliance means that there is established MAJOR dietary indiscretion less than once a fortnight [OR minor indiscretion less than once a week].

POOR dietary compliance means that there is established MAJOR dietary indiscretion once a fortnight or more frequently [OR minor indiscretion once a week or more frequently].

Category A and Category B refer to the categories of the PCFA chart for major dietary culprits causing primary intolerance (category A) and minor culprits causing secondary intolerance (category B).

METHOD FOR DASH DIETARY COMPLIANCE SCORING (Conventional Dietary Approach to Stop Hypertension)

Definitions

'Little' salt: one level teaspoonful = 5g NaCl (2g sodium); 'added salt' means added during preparation, processing, cooking or at the dining table (any of these)

One alcoholic 'drink' corresponds to one unit of alcohol

One 'serving' of fruit or vegetables corresponds to one portion

GOOD (A) = VERY GOOD

Consumes little added salt (or less than little) per day (max 5g NaCl per day)

Consumes vegetables (about 3-6 servings, or more) daily

Takes fruits (about 4-6 servings, or more) daily

PULSES (Nuts/Seeds/Legumes) about 1 serving per day (or more)

Alcohol: at most 2 drinks (2 units) per month (or less than 2 drinks per month)

GOOD (B) = GOOD

Consumes at most 2 level teaspf (10g) salt per day (more than 5g salt per day)

Consumes vegetables (up to 3 servings) daily

Takes fruits (up to 4 servings) daily

Nuts/Seeds/Legumes about 3 servings per week, or more (less than 1 serving per day)

Alcohol: more than 2 drinks per month (less than 2 drinks per week)

FAIR

Consumes up to a tablespoonful (15g) of salt daily (more than 10g salt daily)

Consumes at least about 2 servings vegetables per week

Takes fruits (up to or less than 2 servings) per day

Nuts/Seeds/Legumes less than 3 servings per week, more than 1 serving per week

Alcohol: 2 or more drinks per week

POOR

Consumes more than a tablespf (15g) of salt daily

Consumes vegetables about 1 serving per week (or less)

Takes fruits, at most one serving per day, or less

Nuts/Seeds/Legumes: less than one serving per week

Alcohol: 2 or more drinks (units) per day

END OF PROTOCOL APPENDIX 1

Suppl Appendix 2: Blood and urine laboratory results of open trial study participants before and after personalized food avoidance dietary intervention

Second morning urines: dipstix analysis, sodium, potassium, albumin: creatinine ratio

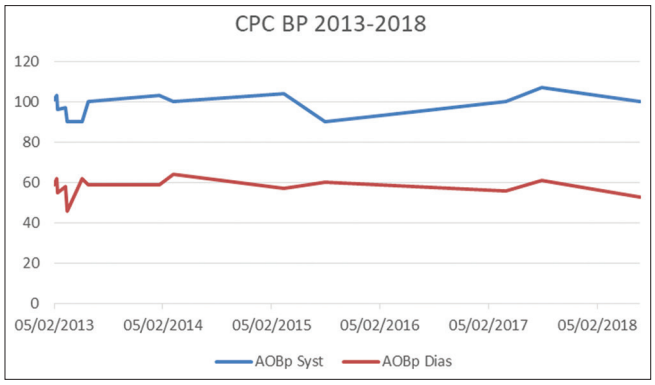
Code number Start date	Date of assessment	Albumin (dipstix)	Blood (dipstix)	WBC (/hpf)	RBC (/hpf)	Urine Na+ (mmo/l)	Urine K+ (mmo/l)	Urine Albumin (mg/l)	Urine Creatinine (g/l)	Urine Albumin/ Creatinine Ratio (mg/g)
PHT12AN	02-Aug-10	nil	nil	1-2	nil					
PHT12AN	24-May-18	nil	nil	Nil	nil	120	13.5	182	0.59	309
17-Dec-15	7-Mar-19	nil	nil	nil	Nil	122	5.4	170	0.21	810
PHT24OP	22-Mar-14	trace		0-1	2-5					
29-Mar-17	7-Mar-19	nil	nil	0-2	nil	133	9.1	68	0.29	235
PHT3AF	07-Sep-16	Nil		0-1	nil					
PHT3AF	24-May-18	nil	nil	nil	nil	130	41.7	303	1.36	223
19-Feb-16	7-Mar-19	nil	nil	0-1	nil	120	6.1	155	0.39	397
QHT35NB	08-May-17	++		0-1	Nil					
QHT35NB	24-May-18	nil	++	nil	20-22	131	12.9	303	0.33	918
15-Feb-17	7-Mar-19			0-1	nil	132	11.6	330	1.13	292
PHT5UN	24-May-18	nil	nil	nil	nil	118	13.2	242	0.96	252
17-Dec-15	7-Mar-19	nil	nil	nil	Nil	121	3.6	61	0.22	277
QHT23OU	24-May-18	++	nil	nil	nil	126	28.5	818	1.21	676
15-Mar-16										

Code number Start date	Date of assessment	Haemoglobin (g/dl)	PCV (v/v)	Total WBC ($x10^9$/l)	Neutrophils (%)	Lymphocyte (%)	Eosinophils (%)	Mono-cytes (%)	Platelets ($x10^9$/l)
PHT12AN	13-Jun-12	13.3	41.3	7.1					244
PHT12AN	24-May-18	12.7	39	5.5	30	69		01	216
17-Dec-15	7-Mar-19	13.5	41	5.0	39	59	02		238
PHT24OP									
29-Mar-17	7-Mar-19	11.1	34	6.1	28	70	02		264
PHT3AF	08-Jul-13	12.6		4.5					293
PHT3AF	24-May-18	12.3	38	4.1	25	74	01		189
19-Feb-16	7-Mar-19	10.8	33	3.2	28	71	01		268
QHT35NB	13-Oct-15	11.4	34	4.8	53	45	02		303
QHT35NB	24-May-18	12.5	38	6.8	56	42	02		205
15-Feb-17	7-Mar-19	13.0	39	4.9	47	51	02		239
PHT5UN	24-May-18	13.2	41	3.3	35	62	02	01	193
17-Dec-15	7-Mar-19	13.8	42	5.2	50	49	01		214
QHT23OU	24-May-18	10.7	33	4.8	5.1	48	01		166
15-Mar-16									

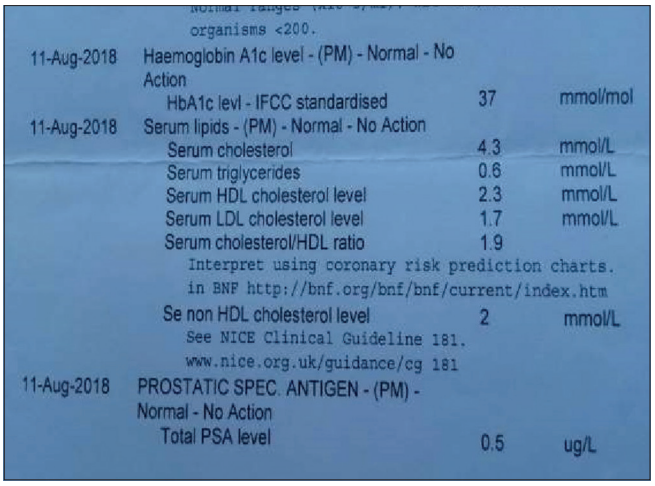
Code number Start date	Date of assessment	Fasting blood glucose (mmol/l)	Total Cholesterol (Mmol/l)	VLDL (Mmol/l)	HDL (Mmol/l)	LDL (Mmol/l)	Triglycerides (Mmol/l)
PHT12AN	21-May-09	3.7	5.7	0.3	1.2	4.2	0.7
PHT12AN	24-May-18	4.8	4.6	0.5	1.2	2.9	1.1
17-Dec-15	7-Mar-19	4.6	5.4	0.5	2.0	2.9	1.0
PHT24OP	31-Mar-14	3.6	6.3	0.6	1.3	4.4	1.4
29-Mar-17	7-Mar-19	5.2	6.5	0.8	0.9	4.8	1.8
PHT3AF	24-May-18	4.9	5.3	0.6	1.5	3.2	1.3
19-Feb-16	7-Mar-19	5.1	5.6	0.3	1.7	3.6	0.7
QHT35NB	01-Jun-15	4.7	5.8	0.8	2.4	2.6	1.8
QHT35NB	24-May-18	5.1	5.0	0.7	1.3	3.0	1.5
15-Feb-17	7-Mar-19	7.8	4.8	1.1	0.9	2.8	2.5
PHT5UN	24-May-18	4.7	4.2	0.4	1.1	2.7	0.9
17-Dec-15	7-Mar-19	4.6	3.3	0.3	1.5	1.5	0.6
QHT23OU	24-May-18	4.7	5.2	1.0	1.3	2.9	2.1
15-Mar-16							

Code number Start date	Date of assessment	Total bilirubin (umol/l)	Conjugated bilirubin (umol/l)	Alkaline phosphatise (iu/l)	Alaninne Transaminase (iu/l)	Aspartate transaminase (iu/l)	Serum Total protein (g/l)	Serum Albumin (g/l)	Serum globulin (g/l)
PHT12AN	24-May-18	10.3	6.1	71	6	10	65	41	24
17-Dec-15	7-Mar-19	12.7	5.3	35	5	10	70	46	24
PHT24OP									
29-Mar-17	7-Mar-19	10.8	5.8	29	8	10	68	42	26
PHT3AF	01-Jun-15								
PHT3AF	24-May-18	16.4	8.8	68	3	5	71	46	25
19-Feb-16	7-Mar-19	12.7	7.9	35	6	8	73	40	33
QHT35NB	01-Jun-15								
QHT35NB	24-May-18	13.4	6.8	60	3	10	66	45	21
15-Feb-17	7-Mar-19	9.5	3.8	28	15	17	61	41	20
PHT5UN	24-May-18	13.7	6.8	82	4	11	69	46	23
17-Dec-15	7-Mar-19	14.8	8.5	32	7	10	73	41	32
QHT23OU	24-May-18	7.5	4.4	60	3	7	62	42	20
15-Mar-16									

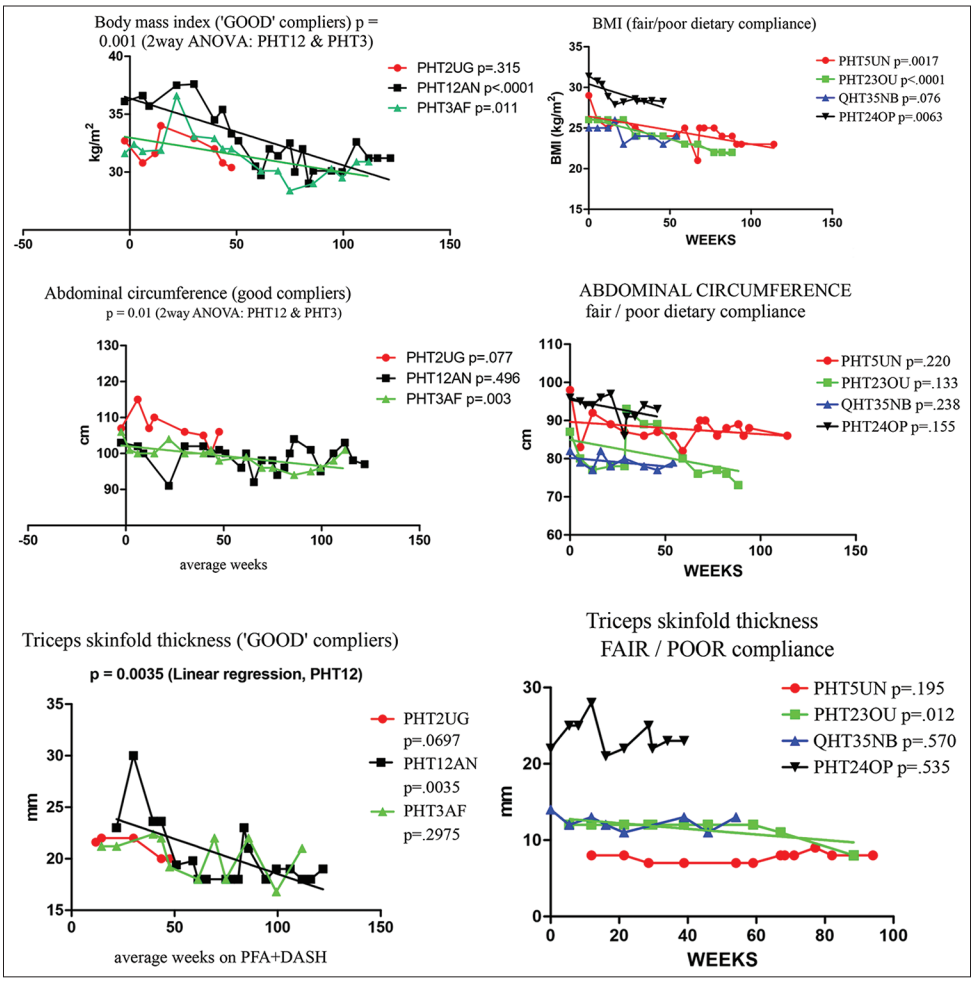
Code number Start date	Date of assessment	Serum Na+ (mmol/l)	Serum k+ (mmol/l)	Serum CI- (mmol/l)	Serum HCO3- (mmol/l)	Blood urea (mmol/l)	Serum creatinine (mmol/l)	Serum uric acid (umol/l)	eGFR (ml/min)	Code number
PHT12AN	21-May-09	140	4.2	91	23	2.8	89			PHT12AN
PHT12AN	24-May-18	139	4.0	103	27	3.2	78	391	68	PHT12AN
17-Dec-15	7-Mar-19	138	3.9	102	27	4.0	91	258	65	
PHT24OP	31-Mar-14	141	4.5	103	26	2.9	75		-	PHT24OP
29-Mar-17	7-Mar-19	138	3.8	103	28	3.8	88	247	76	
PHT3AF	05-Jul-13	137	4.4	24	97	4.3	67			PHT3AF
PHT3AF	24-May-18	138	3.8	100	25	3.7	73	492	80	PHT3AF
19-Feb-16	7-Mar-19	137	3.5	100	29	3.7	78	385	86	
QHT35NB	23-Sep-16	140	2.0	97	25	3.2	106			QHT35NB
QHT35NB	24-May-18	141	3.0	103	30	4.1	85	503	80	QHT35NB
15-Feb-17	7-Mar-19	140	3.6	99	26	6.6	135	427	50	
PHT5UN	24-May-18	139	3.7	100	26	3.2	93	224	91	PHT5UN
17-Dec-15	7-Mar-19	140	3.9	102	30	3.9	87	301	101	
QHT23OU	24-May-18	138	3.5	104	25	4.o	68	336	101	QHT23OU
15-Mar-16										



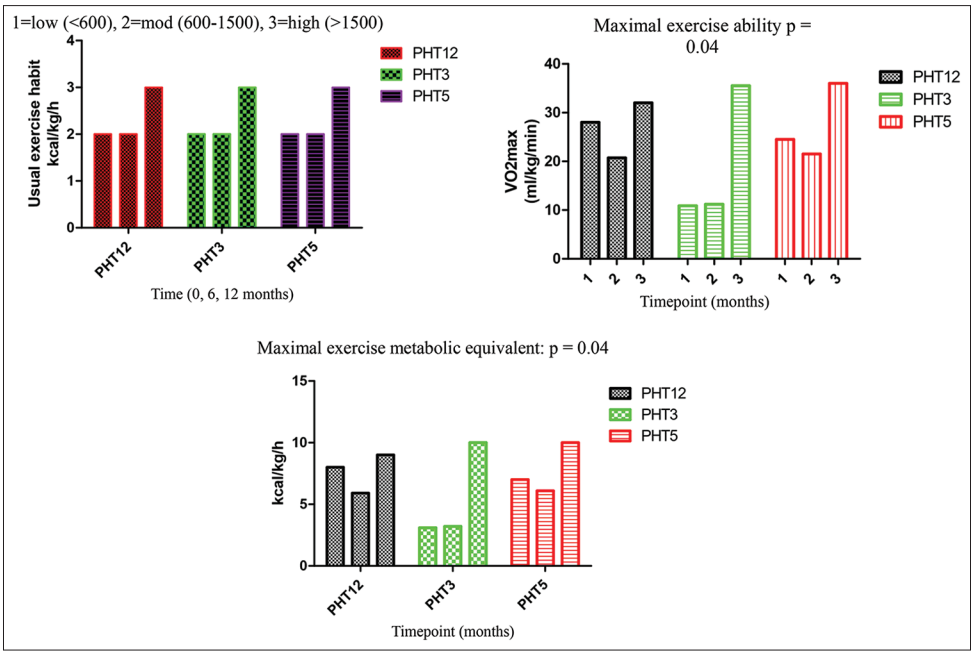
Suppl Figure 1: Side benefit on blood pressure of personalized food avoidance dietary approach to abate severe generalized psoriasis. The propositus' age was 60 years in 2016. He also has a family history of hypertension. (AOBP: automated office blood pressure, systolic and diastolic)



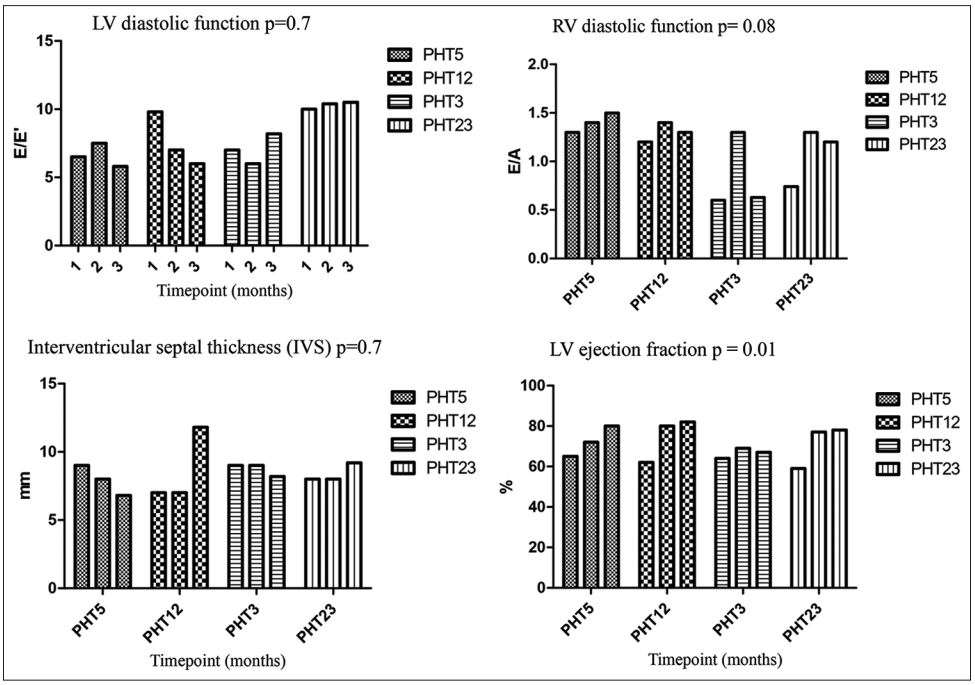
Suppl Figure 2: Side benefit (on 'metabolic syndrome' – associated parameters) of personalized food avoidance dietary approach to abate severe generalized psoriasis. The propositus' age was 62 years. He also has a family history of hypertension



Suppl Figures 3 – 8: Show anthropometric indices (BMI: Body Mass Index, abdominal circumference, triceps skinfold thickness) in 'GOOD' and 'FAIR/POOR' compliers respectively. The *P* values for non-zero significance of linear regression slopes have been added next to the study participant codes. Significant improvement in these parameters is more evident in the 'GOOD' compliers, whether significance is tested by 2-way ANOVA or by linear regression. (PHT12AN, PHT3AF, PHT2UG refer to 'GOOD' compliers, while PHT5UN, PHT23OU, QHT35NB, PHT24OP refer to 'FAIR/POOR' diet compliers. ANOVA: analysis of variance; PFADASH: personalized food avoidance dietary approach to stop hypertension)



Suppl Figures 9 – 11: Show the usual exercise habit and maximal exercise ability in three study participants for whom sufficient data were available. The exercise parameters increased significantly after the dietary intervention. (PHT12, PHT3, PHT5 refer to three study participants at timepoints 3, 6 and 12 months after the start of dietary intervention.)



Suppl Figures 12 – 15: Show salient echocardiographic parameters from four study participants. There was no significant change in diastolic function parameters or in interventricular septum thickness. However there was a significant increase in left ventricular ejection fraction. (PHT12, PHT3, PHT5, PHT23 refer to four study participants at timepoints 3, 6 and 12 months after the start of dietary intervention.)

Suppl Table 1: Baseline demographics of study participants. The age in years refers to when the study participant began the personalized dietary intervention

Code	Sex	DOB	Age yrs	Occupation	Education	Religion	Residence	Tribe
PHT2UG	F	27FEB1950	65	Teacher (nursing school)	3 (NCE)	Anglican	ENUGU	IGBO
PHT5UN	M	16SEP1965	50	Civil servant	4 (Masters degree)	Roman catholic	ENUGU	IGBO
PHT24OP	F	4DEC1967	48	Civil servant	4 (Masters degree)	Roman catholic	ENUGU	IGBO
PHT3AF	F	18APR1964	51	Civil servant	3 (HND)	Roman catholic	ENUGU	IGBO
PHT8EE	F	31APRI1941	75	Retired teacher	3 (Graduate)	Roman catholic	ENUGU	IGBO
PHT15OE	M	2JULY1963	51	Na	4 (Masters degree)	Roman catholic	ENUGU	IGBO
PHT12AN	F	10JUNE1951	64	Retired civil servant	1 (Primary school)	Methodist	ENUGU	IGBO
QHT35NB	F	20AUG1980	34	Civil servant	2 (Secondary)	Anglican	ENUGU	IGBO
PHT23OU	F	8DEC1985	30	Business woman	2 (NECO)	Roman catholic	ENUGU	IGBO
PHTJEU	M	15JUN1968	44	Builder	3 (NCE)	Roman catholic	ENUGU	IGBO

Suppl Table 2: Each parameter for Social Isolation index (SII) was scored 0, 0.5 or 1 based on five parameters: age, having children, having a job, having a faith and belonging to an association. A score of 1 or less than 1 indicate low SII, 2 to 3 indicate intermediate SII while greater than 3 indicate high SII. Sleep deprivation index (SDI) was scored based on hours of sleep. 0 SDI indicate greater than 7 hours sleep per night, 1 SDI indicate 6 to 7 hours sleep while 2 SDI indicate less than 6 hours sleep per night

Study participant code	Social isolation index	Sleep deprivation index	Socio-economic status
PHT2UG	1	0	Middle class
PHT3AF	0.5	1	Upper class
PHT5UN	0.5	0	Upper class
PHT23OU	0.5	0	Middle class
PHT35NB	1	1	Middle class
PHT24OP	0.5	0	Middle class
PHT12AN	1	0	Middle class

Supplementary Materials

Additional File 1. PFADASH PROTOCOL APPENDIX.: details of Dietary Counseling and Compliance Assessment and Scoring (DCCAS) guidelines

Additional File 2. (http:\\cpcpsoriasis.blogspot.com) Online case diary of psoriasis: dietary challenge and avoidance effects.

Additional File 3. (Links to time lapse photographs of psoriasis)

https://drive.google.com/drive/folders/1zA9tHBiwqC1ix-9fEfY8xjcUZG3WXq0T?usp=sharing

https://drive.google.com/drive/folders/1CTCcLOhVu9oSENZsbeVtXi8aRJ3nNvV-?usp=sharing

14 years of serial time lapse photographs of abdomen and elbow in generalized psoriasis, showing aggravation and remission in response to dietary challenge and avoidance testing.

Additional File 4. Suppl Fig1 CPC BP.doc

Side benefit on blood pressure of personalized food avoidance dietary approach to abate severe generalized psoriasis. The propositus' age was 60 years in 2016. He also has a family history of hypertension.

Additional File 5. Suppl Fig2 CPC MS PSA.pdf

Side benefit (on 'metabolic syndrome' – associated parameters) of personalized food avoidance dietary approach to abate severe generalized psoriasis. The propositus' age was 62 years. He also has a family history of hypertension.

Additional File 6. Suppl Fig3 BMI for GOOD PHT compliers.pdf

Additional File 7. Suppl Fig4 BMI for FAIR POOR PHT compliers.pdf

Additional File 8. Suppl Fig5 ABDO for GOOD PHT compliers.pdf

Additional File 9. Suppl Fig6 ABDO for FAIR POOR PHT compliers.pdf

Additional File 10. Suppl Fig7 TSF for GOOD PHT compliers.pdf

Additional File 11. Suppl Fig8 TSF for FAIR POOR PHT compliers.pdf

Legend for Additional Files 6 – 11:

Anthropometric indices (BMI, abdominal circumference, triceps skinfold thickness) in 'GOOD' and 'FAIR/ POOR' compliers respectively. The p-values for non-zero significance of linear regression slopes have been added next to the study participant codes. Significant improvement in these parameters is more evident in the 'GOOD' compliers, whether significance is tested by 2-way ANOVA or by linear regression.

Additional File 12. Suppl Fig9 Usual exercise habit.pdf

Additional File 13. Suppl Fig10 maximal exercise VO2.pdf

Additional File 14. Suppl Fig11 maximal exercise MET.pdf

Legend for Additional Files 12-14:

Usual exercise habit and maximal exercise ability in three study participants. Sufficient data were available in these participants to show a significant increase in these parameters after the dietary intervention.

Additional File 15. Suppl Fig12 ECHO LV diastolic.pdf

Additional File 16. Suppl Fig13 ECHO RV diastolic.pdf

Additional File 17. Suppl Fig14 ECHO IVS.pdf

Additional File 18. Suppl Fig15 ECHO LVEF.pdf

Legend for Additional Files 15-18:

Salient echocardiographic parameters from four study participants. There was no significant change in diastolic function parameters or in interventricular septum thickness. However there was a significant increase in left ventricular ejection fraction.

Additional File 19. SUPPL TABLE 1.docx

Legend for Additional File 19:

Suppl Table 1: Baseline demographics of study participants. The age in years refers to when the study participant began the personalized dietary intervention. One study participant is on a life-long case study of PFADASH, being intolerant of antihypertensive medications in general. The other nine were recruited for the open trial of PFADASH.

Additional File 20. Appendix PHT LABS PRE & POST DIET urinalysis.docx

Legend for Additional File 20:

Laboratory results of study participants before and after PFADASH intervention.

Additional File 21. Suppl Table 2: Social Isolation index (SII) was scored 0, 0.5 or 1 based on five criteria: age, having children, having a job, having a faith and belonging to an association. A score of 1 or less than 1 indicate low SII, 2 to 3 indicate intermediate SII while greater than 3 indicate high SII. Sleep deprivation index (SDI) was scored based on hours of sleep. 0 SDI indicate greater than 7 hours sleep per night, 1 SDI indicate 6 to 7 hours sleep while 2 SDI indicate less than 6 hours sleep per night