

ALLERGIES AND THEIR MODERN THERAPIES

A SPEECH TO THE RHEUMATOID DISEASE FOUNDATION
BIRMINGHAM, ALABAMA, JULY 20, 1985

COPYRIGHT © 1985 WILLIAM E. CATTERALL. ALL RIGHTS RESERVED.

LET US FIRST TALK ABOUT ARTHRITIS AS AN ALLERGY. THE FIRST PUBLICATION ON FOOD ALLERGY IN RHEUMATOID ARTHRITIS CAME IN 1949 FROM MICHAEL ZELLER, A CHICAGO ALLERGIST¹. HE WAS USING A POOR FOOD ALLERGY TEST BUT HE GOT COMPLETE RESOLUTION OF THE DISEASE IN FOUR PATIENTS BY ELIMINATING ALL TEST-POSITIVE FOODS. HE NOTED STRIKING IMPROVEMENT IN JOINTS PREVIOUSLY CONSIDERED IRREVERSIBLY DAMAGED. AT THIS SAME TIME, PHILIP HENCH AT THE MAYO CLINIC WAS DEMONSTRATING THE VALUE OF CORTISONE IN RHEUMATOID ARTHRITIS. A COLLEAGUE OF ZELLER'S, TED RANDOLPH, BEING ONE OF THE FIRST TO KNOW THAT THIS DISEASE IS AN ALLERGY, WAS AMONG THE FIRST TO TRY CORTISONE IN OTHER ALLERGIES SUCH AS ASTHMA. HENCH GOT A NOBEL PRIZE FOR HIS WILLINGNESS TO SPECULATE ABOUT CORTISONE IN ARTHRITIS; EVERYONE ELSE MERELY WANTED TO TRY CORTISONE IN ADRENAL INSUFFICIENCY WHICH DID NOT APPLY IN ARTHRITIS. HENCH KNEW THAT UNDER SOME CONDITIONS THE BODY ALONE COULD CONQUER RHEUMATOID ARTHRITIS². HE KNEW THAT PREGNANCY OFTEN RELIEVED ARTHRITIS AS DID JAUNDICE. HE KNEW THAT FASTING OFTEN RELIEVED SYMPTOMS BUT HE WAS UNABLE TO CONNECT FASTING TO THE ABSENCE OF FOOD ANTIGENS.

FASTING IS THE BASIS FOR TODAY'S DEFINITIVE FOOD ALLERGY TEST³. IF A GROUP OF ARTHRITICS WERE FASTED FOR SIX DAYS, ABOUT HALF OF THEM WOULD BE FREE OF ACUTE SYMPTOMS. THE REST WOULD CONTINUE TO REACT TO CHEMICALS OF ALL KINDS INCLUDING CHLORINATED WATER, PESTICIDES, AND HEATING GAS. FOR BEST ACCURACY IN TESTING, IT IS ESSENTIAL TO ELIMINATE ALL ANTIGENS AT ONCE. THIS MAKES THE RESPONSES CLEARER AND ALSO MAKES THEM HYPERACUTE.

RANDOLPH SUMMARIZED HIS EXPERIENCE WITH 200 CASES OF RHEUMATOID ARTHRITIS IN 1976³. AN ILLUSTRATIVE CASE WAS A 58-YEAR OLD MAN WITH A SWOLLEN, PAINFUL KNEE WHO COULD WALK ONLY WITH CRUTCHES. HE WAS FASTED ON PURE SPRING WATER IN A HIGHLY PURIFIED HOSPITAL ENVIRONMENT. INDIVIDUAL FOOD CHALLENGES USING ORGANIC FOODS GAVE THE RESULTS SHOWN IN TABLE I. DESPITE HYPERACUTE RESPONSIVENESS, THE REACTIONS WERE DELAYED, AND THIS MAKES IT DIFFICULT FOR THE PATIENT IN ORDINARY LIVING TO CONNECT SYMPTOMS TO SPECIFIC FOODS. FIVE OF THE SIX FOODS EACH INDUCED ARTHRITIC PAINS, AND ALLERGY TO THE SIXTH FOOD, WHEAT, WAS CLEAR. THE PATIENT WAS THEN TESTED WITH A MIXTURE OF SAFE FOODS BUT IN NON-ORGANIC FORM, WHICH ALSO PRODUCED SYMPTOMS. THE PATIENT WAS SENT HOME WITH INSTRUCTIONS TO DRINK NON-CHLORINATED WATER, TO AVOID TEST-POSITIVE FOODS, AND TO EAT ALL OTHER FOODS IN ORGANIC FORM AND ONLY AT FOUR-DAY INTERVALS. THIS FOUR-DAY INTERVAL IS USUALLY THE MOST ONEROUS OF THE ALLERGY DIET RESTRICTIONS BUT IS NECESSARY BECAUSE NEW ALLERGIES CAN DEVELOP IN FOODS EATEN FREQUENTLY. THE ARTHRITIS RETURNED AFTER TWO DAYS AT HOME, PROMPTING A TEST WITH SYNTHETIC ETHYL ALCOHOL WHICH OFTEN INDICATES SENSITIVITY TO PETROLEUM HYDROCARBONS. PAINS AGAIN WERE INDUCED, SO THE PATIENT HAD HIS GAS WATER HEATER AND GAS-FIRED HEATING SYSTEM REPLACED WITH ELECTRIC SYSTEMS. HIS SYMPTOMS CLEARED AND HE HAS REMAINED WELL ON THIS PROGRAM FOR SEVEN YEARS, RUNNING DAILY AND PLAYING GOLF AND

TENNIS. HE STILL HAS LITTLE OR NO TOLERANCE FOR HIS ALLERGENIC FOODS, AND HE STILL MUST USE ORGANIC FOODS.

IN THIS SUMMARY OF 200 PATIENTS, RANDOLPH STATED THAT ARTHRITIS RESPOND TO THIS ALLERGY THERAPY "WITH BUT RARE EXCEPTIONS". IN A BOOK WRITTEN FOR LAYMEN IN 1980, RANDOLPH STATED THAT BY THEN HE HAD TREATED OVER 1000 CASES, AND THAT OSTEOARTHRITIS RESPONDS AS WELL⁴. INFLAMMATION IN OSTEOARTHRITIS IS BEING INCREASINGLY RECOGNIZED. RANDOLPH ALSO LISTS A FEW FACILITIES WHERE OTHER PHYSICIANS ARE USING HIS METHODS.

THE MEDICAL ESTABLISHMENT IS MISLED BY THE FACT THAT ARTHRITIS HAS AN AUTOIMMUNE COMPONENT IN WHICH THE BODY REGARDS ITS OWN TISSUE AS AN ANTIGEN. TO THEM, AUTOIMMUNITY FULLY EXPLAINS THE ALLERGIC CHARACTER OF THE DISEASE AND FOOD ALLERGY IS VIGOROUSLY DENIED. HOWEVER, THE AUTOIMMUNE ATTACK MUST STOP WHEN EXTERNAL ANTIGENS OR INFECTIONS ARE CLEARED, SO THAT AUTOIMMUNITY IS A SECONDARY PROCESS. FORMS OF AUTOIMMUNITY OFTEN SEEN IN ASSOCIATION WITH ARTHRITIS AND OTHER ALLERGIES ARE ATROPHIC GASTRITIS AND THYROIDITIS.

IT IS CLEAR THAT ACCURATE DETECTION AND AVOIDANCE OF ALLERGENS IS QUITE EFFECTIVE IN ARTHRITIS, AND BASED ON NUMEROUS REFERENCES THIS IS SIMILARLY TRUE IN A LONG LIST OF ALLERGIC DISEASES AND CONDITIONS PRESENTED IN TABLE II. THERE IS TIME TO DISCUSS ONLY A FEW OF THESE.

HYPOTHYROIDISM IS ABOUT UNIVERSAL IN ALLERGY SINCE 500 PATIENTS WITH BRONCHIAL ASTHMA, RHINITIS, OR ECZEMA SHOWED 90% HYPOTHYROIDISM BASED ON CAREFUL METABOLISM TESTS⁵. THIS EFFECT SEEMS TO BE BASED ON SEVERAL FACTORS. ALL ALLERGY INVOLVES A METABOLIC BIAS KNOWN AS BETA BLOCKADE IN WHICH THE PATIENT ACTS AS THOUGH HE WERE ON A BETA-BLOCKING DRUG. SUCH DRUGS INDUCE HYPOTHYROIDISM BOTH BY REDUCING THE CONVERSION OF T₄ TO T₃ AND BY REDUCING THE PERIPHERAL RESPONSE TO THYROID HORMONES⁶. IN TIME, AUTOIMMUNE THYROIDITIS CAN DEVELOP. THE HYPOTHYROIDISM TENDS TO WORSEN THE ALLERGY, SO ITS TREATMENT IS AN IMPORTANT PART OF ALLERGY THERAPY⁷ ALTHOUGH IT MUST BE RECOGNIZED THAT ABOLISHING THE ALLERGY MIGHT REVERSE THE NEED FOR THYROID THERAPY. THYROID MANAGEMENT REQUIRES BETTER TOOLS AS WILL BE DISCUSSED.

ANOTHER CONDITION THAT IS ALMOST UNIVERSAL IN ALLERGY IS FATIGUE. ALLERGIC FATIGUE IS NOT RELIEVED BY REST AND IS OFTEN WORST UPON ARISING AS WITH MORNING STIFFNESS. FATIGUE IS GENERALLY DUE TO LIMITED OXYGEN SUPPLY TO THE MUSCLES AS READILY SEEN IN HEAVY EXERCISE, IN EFFORT AT HIGH ALTITUDE, AND IN ANEMIA. ALLERGICS TEND TO BE ANEMIC FOR TWO REASONS. THERE IS THE USUAL HYPOTHYROIDISM WHICH CAUSES ANEMIA, AND AS IN ANY INFLAMMATION IRON IS SEQUESTERED OUT OF THE BLOOD (ANEMIA OF INFECTION). THE ACUTE CAUSE OF FATIGUE APPEARS TO BE RESTRICTION OF CIRCULATION BY ALLERGIC EDEMA WHICH MUST CONTRIBUTE TO THE STIFFNESS. THE CONDITION MIGHT BE WORSENED BY PLATELET AGGREGATION AND BY ALLERGIC BLOOD SLUDGING COMMONLY SEEN IN ARTHRITIS AND DIABETES.

DEPRESSION IS ANOTHER COMMON ALLERGIC CONDITION. BOTH FATIGUE AND DEPRESSION WERE IMPORTANT IN PERRY CHAPDELAINÉ'S ARTHRITIS HISTORY. FATIGUE OR DEPRESSION CAN BE HELPFUL IN ALLERGY DIAGNOSIS, FOR EXAMPLE IN HEADACHE. MIGRAINE IS ALMOST ALWAYS AN ALLERGY, WHEREAS OTHER FORMS OF HEADACHE HAVE MANY CAUSES. IN A STUDY BY A LONDON NEUROLOGIST ON 60 MIGRAINE PATIENTS, AVOIDING ALLERGENIC FOODS REDUCED MIGRAINES OVER 98%⁸. WHEN KNOWN CAUSES OF OTHER HEADACHES ARE RULED OUT, ALLERGY WILL ACCOUNT FOR MOST OF THE REMAINDER, WITH FATIGUE OR DEPRESSION CONFIRMATORY. WITH ACCURATE ALLERGEN DETECTION, RANDOLPH CLAIMS NEVER TO HAVE SEEN A PSYCHOGENIC HEADACHE, AND HE FEELS THAT TENSION HEADACHE IS A MISNOMER SINCE THE CAUSE IS USUALLY ALLERGY⁴.

AMONG THE 60 MIGRAINE PATIENTS IN THE LONDON STUDY WERE 15 WITH HYPERTENSION, ALL WITH DIASTOLIC PRESSURE OF 100 OR HIGHER. AFTER AVOIDING ALLERGENIC FOODS, IN EACH OF THE 15 CASES THE DIASTOLIC PRESSURE WAS 90 OR LOWER, INDICATING RELIEF FROM TREATABLE HYPERTENSION. SOME FOOD ALLERGISTS SIMPLY CONSIDER HYPERTENSION AS ANOTHER SIGN OF ALLERGY. THE USUAL INCREASE IN BLOOD PRESSURE WITH AGE IS NOT SEEN IN MANY PRIMITIVE POPULATIONS ON HYPOALLERGENIC DIETS LOW IN FAT, SUGAR, AND SALT, AND HIGH IN MAGNESIUM. THESE DIETARY FACTORS WILL BE DISCUSSED LATER.

GALLBLADDER SURGERY IS COMMON ALTHOUGH AUTOPSIES SHOW THAT MANY MORE PERSONS HAVE GALLSTONES WITHOUT SYMPTOMS. ALLERGEN-FREE DIETS HAVE BEEN VERY SUCCESSFUL IN TREATING GALLSTONES AND HAVE BEEN MORE POPULAR THAN STANDARD GALLSTONE DIETS⁹. ACUTE DISTRESS IS CREATED BY ALLERGENIC FOODS, AND APPARENTLY ALLERGIC INFLAMMATION IS PRESENT. IT APPEARS THAT THE METABOLIC BIAS OF ALLERGY AND THE HYPOTHYROIDISM REDUCE THE SECRETION OF BILE ACIDS AND INCREASE CHOLESTEROL, MAKING IT EASY FOR AN ALLERGIC TO HAVE SUPERSATURATED BILE CONDUCTIVE TO STONES.

WE HAVE HEARD ABOUT MS FROM RON DAVIS. THERE ARE DATA, MOSTLY UNPUBLISHED, SHOWING THAT MS READILY RESPONDS TO ALLERGY THERAPY IF IRREVERSIBLE DAMAGE HAS NOT DEVELOPED¹⁰. WYBURN-MASON TRIED ANTIAMEBA DRUGS ON SEVERAL MS PATIENTS WITHOUT CORTICOSTEROID PROTECTION AND PRODUCED IRREVERSIBLE NERVE DAMAGE. THIS WAS UNFORTUNATE BUT SEEMED TO SHOW THAT ANTIGENS FROM AMEBA CAN CAUSE MYELIN DAMAGE.

HYPERACTIVITY WAS BEING TREATED SUCCESSFULLY BY FOOD ALLERGISTS LONG BEFORE FEINGOLD PROPOSED HIS ADDITIVE-FREE DIET IN 1975¹¹. THESE ALLERGISTS FEEL THAT FEINGOLD FOUND ONLY PART OF THE PROBLEM SINCE ORDINARY FOODS OFTEN CAUSE HYPERACTIVITY. ATTEMPTS BY NON-ALLERGISTS TO VALIDATE THE FEINGOLD DIET SEEMED UNSUCCESSFUL AND LED TO SENSATIONAL DENIALS IN THE PRESS OF FEINGOLD'S CLAIMS. THE FACTS ARE THAT FEINGOLD'S EXPERIENCE THAT ABOUT 50% RESPOND TO THE DIET HAS BEEN WELL SUBSTANTIATED¹². THE PROBLEM HAS BEEN INABILITY TO GET RESPONSES TO CHALLENGE WITH ADDITIVES HIDDEN IN STANDARDIZED COOKIES. SOME CHILDREN APPARENTLY REACTED TO THE CHOCOLATE, WHEAT, EGG, SUGAR, ETC. IN THE PLACEBO COOKIES, GREATLY DISTURBING THE STATISTICS, BUT THE FATAL FLAW WAS THAT THE AMOUNT OF ADDITIVES WAS FAR TOO SMALL, BEING BASED ON THE AVERAGE ADDITIVE CONSUMPTION RATHER THAN THE CONSUMPTION OF THE PATIENTS, MANY OF WHOM CONSUME AT LEAST TEN TIMES THE AVERAGE IN SUCH PRODUCTS AS CANDY, SODA, AND ESPECIALLY POWDER-MIX BEVERAGES.

IT DOES NOT SEEM DIFFICULT TO ACCEPT THE IDEA THAT FOOD ALLERGY COULD CAUSE INFLAMMATORY BOWEL DISEASE SINCE THERE IS DIRECT CONTACT BETWEEN FOODS AND BOWEL, BUT AS USUAL MEDICINE STRONGLY RESISTS THE IDEA. THIS IS TRAGIC BECAUSE MILK IS ESPECIALLY PROMINENT IN THIS ALLERGY AND ONE STUDY HAS SHOWN THAT MERELY AVOIDING MILK IN ALL FORMS CURED 25% OF THE CASES¹³. PERSONALLY I HAVE A FORM OF IRRITABLE BOWEL IN WHICH DIARRHEA IS QUICKLY PRODUCED BY PENICILLIUM MOLD WHICH OCCURS IN CHEESE, CITRUS, AND POSSIBLY OTHER FOODS. THIS RESPONSE IS READILY BLOCKED BY HIGH-DOSE ASPIRIN, SHOWING MEDIATION BY PROSTAGLANDINS WHICH CAUSE INFLAMMATION AND INCREASE INTESTINAL MOTILITY AND OUTPOURING OF FLUIDS INTO THE BOWEL.

SYSTEMIC LUPUS SEEMS TO BE THE MOST ALLERGIC OF ALL THESE DISEASES, APPARENTLY BECAUSE IMMUNE COMPLEXES FORM AND LEAD TO POSSIBLE ATTACK ON MANY BODY SYSTEMS. KIDNEY DAMAGE CAN BE FATAL. THERE IS LITTLE PUBLISHED ABOUT ALLERGY IN LUPUS BUT I KNOW OF SOME HIGHLY DRAMATIC CASES. WE WILL BE TALKING ABOUT HEART DISEASE AS AN ALLERGY, AND IT IS

INTERESTING THAT YOUNG WOMEN WITH LUPUS CAN DEVELOP ATHEROSCLEROSIS AND HEART ATTACKS WITHOUT RECOGNIZED HEART DISEASE RISK FACTORS¹⁴. THE WELL-KNOWN DRUG-INDUCED LUPUS ACTS LIKE DRUG ALLERGY SINCE THE DISEASE COURSE PARALLELS THE LEVEL OF DRUG ANTIBODIES. SOMETIMES THE DISEASE PERSISTS AFTER DRUG WITHDRAWAL, SUGGESTING THAT FOOD ALLERGY HAS DEVELOPED. THIS IS A COMMON SITUATION IN ALLERGY, WHERE SENSITIVITY PROPOGATES TO MORE AND MORE ANTIGENS AS TIME PROGRESSES. A FORM OF THIS TENDENCY IS THAT INFECTIONS, ESPECIALLY INFLUENZA, MUMPS, MEASLES, AND WHOOPING COUGH, SOMETIMES EVOLVE INTO PERMANENT FOOD ALLERGY.

NOW LET US CONSIDER FORMS OF ALLERGY THERAPY. THE TREATMENT IS ESSENTIALLY THE SAME REGARDLESS OF THE NATURE OF THE ALLERGY. RANDOLPH'S HOSPITALIZED FASTING IS THE ULTIMATE BUT REQUIRES ABOUT THREE WEEKS IN THE HOSPITAL AND PERHAPS LONG WAITING TIME. THERE MAY BE INSURANCE QUESTIONS SINCE THE HOSPITAL PROCEDURE IS IN A SENSE DIAGNOSTIC. ELIMINATION DIETS ARE USEFUL ALTERNATIVES TO FASTING; THESE ARE BASED ON EATING UNFAMLIAR AND THEREFORE NON-ALLERGENIC FOODS FOR A FEW DAYS PRIOR TO FOOD CHALLENGE. THE CYTOTCXIC TEST DETERMINING ALLERGIC REACTIONS ON A BLOOD SAMPLE IS HELPFUL WHEN ITS LIMITATIONS ARE RECOGNIZED, BUT THERE HAS BEEN NON-MEDICAL EXPLOITATION OF THE TEST WITH POOR QUALITY CONTROL. THE RAST TEST AND ORDINARY SKIN PRICK TESTS MEASURE THE IGE ANTIBODY RESPONSE AND NEITHER IS VERY USEFUL FOR FOODS. ALL OF THESE METHODS LEAD TO ONEROUS DIETS. THE MAINSTAY OF FOOD ALLERGY TESTING TODAY IS THE SO-CALLED PROVOCATION TEST IN WHICH ANTIGEN IS APPLIED EITHER UNDER THE SKIN OR UNDER THE TONGUE, HOPEFULLY TO OBTAIN A MILD SYSTEMIC REACTION¹⁵. DESENSITIZATION BY EITHER ROUTE BECOMES THE THERAPY, AND EXCESSIVE DIETARY RESTRICTIONS ARE AVOIDED. RESULTS ARE GOOD AT TIMES BUT A GOOD STATISTICAL PRESENTATION IS NOT AVAILABLE. THE MAIN REPOSITORY OF SKILL IN FOOD AND CHEMICAL ALLERGY IS THE 400 MEMBERS OF THE AMERICAN ACADEMY OF ENVIRONMENTAL MEDICINE (FORMERLY SOCIETY FOR CLINICAL ECOLOGY). ORDINARY INHALANT ALLERGENS ALSO MAY BE INVOLVED. CONVENTIONAL ALLERGISTS GENERALLY HAVE LITTLE SKILL IN FOOD ALLERGY AND EVEN TEND TO RESIST THE IDEA.

MY GENERAL RECOMMENDATION IS THAT PRIOR TO DIRECT ALLERGY THERAPY ONE SHOULD TRY ANTI-INFECTIVE TREATMENT DIRECTED AT AMEBA OR CANDIDA YEAST, ALONG WITH NUTRITIONAL INTERVENTION. SOME ALLERGISTS HAVE FOUND THAT CANDIDA TREATMENT RESOLVES A HIGH FRACTION OF THEIR CASES, JUST AS AMEBA TREATMENT HANDLES A HIGH FRACTION OF ARTHRITIS CASES. ANTI-INFECTIVE THERAPY IS VERY SIMPLE COMPARED TO ALLERGY THERAPY, AND ITS RATE OF SUCCESS APPEARS ENCOURAGING.

NATURALLY ONE MUST WONDER WHY ALLERGEN AVOIDANCE AND AMEBA TREATMENT CAN BE EFFECTIVE IN THE SAME DISEASE. AT LEAST PART OF THE ANSWER MAY LIE IN ESSENTIAL FATTY ACID (EFA) PROCESSING WHICH UNDERLIES INFLAMMATION AS OUTLINED IN FIGURE 1. THE BASIC SOURCE OF EFA IS LINOLEIC ACID FOUND MAINLY IN OILS FROM SEEDS, GRAINS, AND NUTS. THE C18:2W6 DESIGNATION FOR LINOLEIC ACID INDICATES THAT THERE ARE 18 CARBON ATOMS, WITH 2 DOUBLE BONDS LOCATED 6 CARBONS FROM THE END OF THE CHAIN. THE BODY CONVERTS THIS MOLECULE BY A SUCCESSION OF DESATURATION AND ELONGATION REACTIONS AS SHOWN TO ARACHIDONIC ACID, C20:4W6, WHICH IS THE RAW MATERIAL FOR MOST OF THE INFLAMMATION MEDIATORS. ANIMAL PRODUCTS IN THE DIET ALSO DIRECTLY SUPPLY ARACHIDONIC ACID. THERE IS CRITICAL COMPETITION AT THE DGLA OR C20:3W6 POINT SINCE PART OF THE DGLA IS DIVERTED TO PROSTAGLANDIN PGE1 WHICH HELPS TO CONTROL CAMP (CYCLIC ADENOSINE MONOPHOSPHATE). CAMP IS AN INTRACELLULAR MESSENGER CONTROLLING MANY BODY FUNCTIONS INCLUDING RESIS-

TANCE TO ALLERGIC RESPONSE. ALLERGENS ARE UNIVERSALLY LOW IN CAMP, WITH LOW PGE1 APPARENTLY AS THE PRINCIPAL CAUSE.

AMONG POSSIBLE CAUSES FOR LOW PGE1, THERE MAY BE INSUFFICIENT DIETARY LINOLEIC ACID AS SHOWN BY RESPONSE TO LINOLEIC ACID TREATMENT. OFTEN THE FIRST DESATURATION ENZYME, THE DELTA-6 DESATURASE, IS DEFICIENT FOR GENETIC OR NUTRITIONAL REASONS WHICH WE WILL DISCUSS SHORTLY. A FUNDAMENTAL PROBLEM IS THE PRESENCE OF INFLAMMATION WHICH SIMPLY OVERLOADS THIS SYNTHESIS CHAIN, WITH RESULTING DEPLETION OF DGLA, REDUCING FEEDSTOCK AVAILABILITY FOR PGE1. THE LOWERED LEVEL OF PGE1 (AND CAMP) INCREASES ALLERGIC RESPONSE. THIS CREATES A VICIOUS CYCLE WITH ALLERGIC INFLAMMATION PERPETUATING THE BASIC PGE1 DEFICIENCY. I SUSPECT THAT AN AMEBA INFECTION CAN ACT BY CAUSING CONTINUOUS INFLAMMATION WHICH INDUCES A PGE1 DEFICIENCY AND MAKES THE HOST ALLERGIC. ELIMINATING THE AMEBA INFECTION COULD REDUCE THE DGLA DRAIN AND RENDER THE PATIENT NON-ALLERGIC. A CANDIDA YEAST INFECTION CAN PRODUCE ALLERGIES MUCH THE SAME AS AMEBA, PROBABLY BY A SIMILAR MECHANISM. AS MENTIONED, OTHER INFECTIONS SUCH AS INFLUENZA, MEASLES, MUMPS, AND WHOOPING COUGH CAN LEAD TO PERMANENT FOOD ALLERGY, PRESUMABLY BECAUSE THE TEMPORARY DRAIN ON DGLA AND PGE1 BECOMES PERMANENT AS ALLERGIC INFLAMMATION SUPERSEDES INFECTIOUS INFLAMMATION.

NOW, WHERE IS THE LOCATION OF THIS ALLEGED INFECTION? I SUSPECT THE LARGE LYMPHOID TISSUE IN THE INTESTINE WHERE THE INFECTION MIGHT BE SILENT. DRUGS SUCH AS COPPER, BILE ACIDS, AND NYSTATIN PROBABLY ACT ONLY IN THE INTESTINE BUT CAN CLEAR INFECTIONS UNDERLYING ALLERGY. THUS THE INFECTION DOES NOT NECESSARILY HAVE TO BE IN THE JOINTS; ACCORDING TO THIS PICTURE THE INFECTION SERVES PRIMARILY TO SUSTAIN THE ALLERGY, AND IT IS THE FOOD ANTIGENS THAT ACT ON THE JOINTS. OF COURSE, LOCAL INFECTION COULD WORSEN JOINT CONDITIONS. ANOTHER IMPLICATION OF THIS PICTURE IF TRUE IS THAT FAILURE OF ANTIAMEBA THERAPY DOES NOT NECESSARILY MEAN THAT AMEBA ARE STILL PRESENT. THE ALLERGY MERELY MAY HAVE BECOME SELF-SUSTAINING INDEPENDENT OF INFECTION, IN WHICH CASE INFECTION TREATMENT MIGHT BE USELESS. ALLERGY OR NUTRITION THERAPY COULD BE INDICATED, UNLESS A DIFFERENT INFECTANT IS SUSPECTED.

IN FIGURE 1 THE LOGIC OF CERTAIN INTERVENTIONS IS EVIDENT. CORTICOSTEROIDS BLOCK THE RELEASE OF ARACHIDONIC ACID FROM STORAGE, THUS SHUTTING OFF THE RAW MATERIAL FOR INFLAMMATION. REMISSIONS CAN BE INDUCED BY THESE STEROIDS SINCE THE DRAIN ON DGLA IS HALTED. HOWEVER MUCH OF OUR RESISTANCE TO INFECTION COMES FROM THE INFLAMMATORY RESPONSE, SO THAT INFECTION RESISTANCE SUFFERS. ASPIRIN AND SIMILAR DRUGS PROBABLY DO NOT REDUCE DGLA DEPLETION AT ALL. THESE DRUGS REDUCE THE FORMATION OF PROSTAGLANDINS, BUT SINCE THESE REACTIONS ARE LIMITED BY THE AVAILABILITY OF ARACHIDONIC ACID, THIS ACID MUST BE DIVERTED TO MAKE MORE LEUKOTRIENES WHICH ARE QUITE DAMAGING BUT PERHAPS LESS PAINFUL. EVENING PRIMROSE OIL CONTAINS ABOUT 8% GLA, THE INTERMEDIATE THAT BYPASSES THE FIRST DESATURATION STEP. MANY TRIALS IN VARIOUS ALLERGIES HAVE BEEN MADE WITH ONLY MODEST BENEFITS. TRIALS TO DATE IN ARTHRITIS HAVE BEEN NEGATIVE BUT HAVE BEEN SHORT WITH PERHAPS INADEQUATE DOSAGE; BETTER TRIALS ARE IN PROGRESS. THERE HAVE BEEN PROBLEMS WITH COUNTERFEIT PRODUCTS ON THE MARKET.

FATTY ACID DERIVATIVES BASED ON THE W3 STRUCTURE SUCH AS LINSEED OIL AND FISH OILS HAVE RECEIVED PUBLICITY RECENTLY, LARGELY BECAUSE ESKIMOS WHOSE DIET IS RICH IN FISH OILS HAVE LITTLE HEART DISEASE. LINSEED OIL (LARGELY C18:3W3) GENERATES FISH OILS IN THE BODY BY CHEMISTRY ANALOGOUS TO THE W6 SERIES SHOWN IN FIGURE 1. THE W3 SERIES ACTS A COMPETITIVE INHIBITOR FOR THE W6 SERIES AND THEREFORE

INFLAMMATION IS SUPPRESSED. IT REMAINS TO BE SEEN HOW BENEFICIAL THE W3 SERIES PRODUCTS ARE SINCE CORTISONE-LIKE SIDE-EFFECTS ARE POSSIBLE.

ANTIOXIDANTS ARE BEING PROMOTED BASICALLY TO SUPPRESS INFLAMMATION, ESPECIALLY VITAMINS C AND E, SELENIUM, AND CYSTEINE. THE OXIDATION OF ARACHIDONIC ACID, WHICH IS THE BASIC MECHANISM OF INFLAMMATION, CAN BE SUPPRESSED BY ANTIOXIDANTS AT HIGH DOSES. THERE ARE TWO SIDE EFFECTS NOT GENERALLY APPRECIATED, THE REDUCED INFECTION RESISTANCE AND THE PARALLEL SUPPRESSION OF THE OXIDATION OF DGLA TO PGE1. REDUCED PGE1 MAKES THE UNDERLYING ALLERGY WORSE AND APPARENTLY INDUCES HYPOTHYROIDISM IN THE FORM OF LOW T3 AND LOW PERIPHERAL RESPONSE. SELENIUM APPARENTLY HAS A UNIQUE EFFECT OF BLOCKING CHEMICAL ALLERGY, POSSIBLY BY PREVENTING THE OXIDATIVE CONVERSION OF LOW MOLECULAR WEIGHT CHEMICALS TO HIGHER MOLECULAR WEIGHT ACTIVE ANTIGENS. HIGH DOSE VITAMIN C IS EFFECTIVE AND MAY HAVE ITS OWN INFECTION-FIGHTING ABILITY OFFSETTING THE LOSS OF ARACHIDONIC ACID. THE MAXIMUM POSSIBLE DAILY DIVIDED DOSE THAT AVOIDS DIARRHEA IS USED, CALLED THE BOWEL-TOLERANCE DOSE¹⁶. ONE PATIENT WITH MIXED OSTEO/RHEUMATOID ARTHRITIS DOES WELL WITH 30G/DAY VITAMIN C, BUT UP TO 100G/DAY HAS BEEN USED IN ARTHRITIS. THESE HIGH DOSES ARE TOLERABLE BECAUSE IN ARTHRITIS VITAMIN C IS BEING CONSUMED AT A VERY HIGH RATE ABOUT 1000 TIMES NORMAL. THIS PATIENT IS CERTAINLY VERY HYPOTHYROID FROM HIS BODY TEMPERATURE BUT IS HAVING DIFFICULTY GETTING HIS DOCTORS TO ACCEPT THE IDEA.

TABLE III OUTLINES SOME FACTORS CONTROLLING THE ACTIVITY OF THE DESATURASE WHICH SOMETIMES IS THE BOTTLENECK. THIS IS BASED LARGELY ON RAT STUDIES BUT WITH SOME HUMAN CONFIRMATION^{17,18}. THIS ENZYME IS LABILE AND SUBJECT TO MANY INFLUENCES. ACTIVITY IS INCREASED BY SEVERAL NUTRIENTS, ZINC, MAGNESIUM, AND VITAMINS B3, B6, AND C, AND MOST OF THESE TEND TO BE DEFICIENT IN ARTHRITIS AND OTHER ALLERGICS AS METABOLIC CONSEQUENCES OF THE ALLERGY IN ADDITION TO POSSIBLE DIETARY DEFICIENCIES. THERE IS A METABOLIC BLOCK IN ALLERGICS WHICH MAKES B6 DEFICIENT DESPITE ADEQUATE STORES. ZINC DOES NOT HAVE NORMAL ACTIVITY SINCE LIKE IRON IT IS SEQUESTERED AS PART OF THE INFLAMMATORY RESPONSE. LIKE IRON IN THIS SITUATION, ORDINARY ZINC SUPPLEMENTS ARE USELESS. A NEW FORM, ZINC PICOLINATE, IS NOW AVAILABLE FROM GENERAL NUTRITION THROUGH THE INFLUENCE OF MEDICAL NUTRITIONIST JONATHAN WRIGHT. INCREASED ABSORPTION AND AVAILABILITY IN ALLERGY IS CLAIMED. MAGNESIUM DEFICIENCY IN ALLERGY IS INDUCED BY EXCESS URINARY EXCRETION AS WILL BE DISCUSSED SHORTLY. MANY U.S. DIETS ARE MARGINAL IN MAGNESIUM. FACTORS THAT REDUCE ENZYME ACTIVITY ARE SATURATED FATS, WITH A HIGH CARBOHYDRATE DIET DOING THE SAME THING SINCE SOME OF THE CARBOHYDRATE IS IMMEDIATELY CONVERTED TO SATURATED FAT IN THE BODY. PARTIALLY HYDROGENATED FATS HAVE BEEN INTRODUCED INTO MODERN DIETS TO IMPROVE FAT STABILITY WITHOUT ADDITIVES, AND THEIR DISADVANTAGE IS NOT FULLY APPRECIATED.

THIS CHEMISTRY OF THE CONTROL OF PGE1 FORMATION IS DIRECTLY CONFIRMED IN HUMANS BY STUDIES ON PLATELET AGGREGATION WHICH IS CONTROLLED BY cAMP AND THEREFORE BY PGE1¹⁹. DIETS WITH ADDED LINOLEIC ACID OR PRIMROSE OIL REDUCE PLATELET AGGREGATION, INDICATING THAT cAMP AND PGE1 ARE INCREASED²⁰. SATURATED FATS CONVERSELY INCREASE PLATELET AGGREGATION AS EXPECTED FROM THE PROPOSED MECHANISM. NOW CONSIDER THE WELL-KNOWN METHOD TO REDUCE SERUM CHOLESTEROL WHICH IS TO EAT LESS SATURATED FAT AND MORE LINOLEIC ACID. ALTHOUGH THE MEDICAL LITERATURE IS SILENT ON THE MECHANISM, THE ESSENTIAL FATTY ACID CHEMISTRY WE HAVE BEEN DISCUSSING APPEARS AS THE EXPLANATION. PRIMROSE OIL IS OVER 100 TIMES MORE POTENT THAN LINOLEIC ACID IN LOWERING CHOLESTEROL, SHOWING THAT THE FINAL ACTION IS DOWNSTREAM FROM LINOLEIC ACID AND PGE1 APPEARS TO BE THE CONTROL POINT. INSULIN ACTIVATES THE CONTROLLING ENZYME IN CHOLESTEROL SYNTHESIS IN THE LIVER, AND INSULIN CAN ACT BY LOWERING

PGE1. HIGH CHOLESTEROL WHICH IS CHARACTERISTIC OF ALLERGY CAN NOW BE SEEN AS A BAROMETER OF EXCESSIVE PLATELET AGGREGATION AND PROBABLY LOW CAMP AND PGE1, FACTORS WHICH MAY BE MORE IMPORTANT IN HEART DISEASE THAN CHOLESTEROL PER SE.

VEGETARIAN DIETS SUCH AS THE PRITIKIN DIET AND OTHERS HAVE DEMONSTRATED EFFECTIVENESS IN ARTHRITIS, HEART DISEASE, HYPERTENSION, DIABETES, AND OTHER ALLERGIES²¹. THESE DIETS ARE LOW IN SATURATED FAT AND THEREBY AVOID INHIBITING THE DESATURASE, SO THAT PGE1 IS ELEVATED WHICH IS PROBABLY THE FUNDAMENTAL BENEFIT OF THE DIET. THESE DIETS ALSO ARE HIGH IN MAGNESIUM AND POTASSIUM AND LOW IN DIETARY ARACHIDONIC ACID. ONE MODERN ATTEMPT TO EVALUATE VEGETARIAN DIETS IN ARTHRITIS WAS COMPLETELY NEGATIVE²². UNFORTUNATELY THE PATIENTS WERE ALLOWED TO CONTINUE THEIR ASPIRIN-TYPE DRUGS; THESE DRUGS PROBABLY BLOCKED THE FORMATION OF PGE1 FROM DGLA AND THEREBY COUNTERACTED THE PRIMARY PURPOSE OF THE DIET.

THE LIMITED CLINICAL BENEFITS OF PRIMROSE OIL IN ALLERGIC DISEASES HAVE BEEN PUZZLING IN VIEW OF THE WELL-DEMONSTRATED BENEFITS ON CHOLESTEROL AND PLATELET AGGREGATION. FACTORS OTHER THAN CAMP MUST BE IMPORTANT IN CONTROLLING ALLERGIC RESPONSE, AND IT IS CLEAR THAT DEFICIENCIES OF CALCIUM AND MAGNESIUM ARE HIGHLY SIGNIFICANT. FIGURE 2 IS A HIGHLY OVERSIMPLIFIED VIEW OF ALLERGY CONTROL AT THE TARGET CELL, USUALLY SOME FORM OF WHITE CELL. THIS IS ALSO THE SECRETION CONTROL SYSTEM FOR SOME OTHER TYPES OF CELL. IN THE CASE OF THE VASCULAR MUSCLES, INCREASED CALCIUM INFLUX (OR TRANSLOCATION) CAUSES VASOCONSTRICTION, AND INCREASED CAMP WHICH REDUCES CALCIUM INFLUX CAUSES VASODILATION. YOU MUST BELIEVE THAT CALCIUM INFLUX CAUSES VASOCONSTRICTION SINCE VERY BEAT OF YOUR HEART IS CAUSED BY A PULSE OF CALCIUM INFLUX WHICH CONTRACTS YOUR HEART MUSCLES. IN THE CASE OF ALLERGY TARGET CELLS, EXCESSIVE CALCIUM INFLUX PROMOTED BY ANTIGEN WITH OR WITHOUT ANTIBODY IS THE TRIGGER FOR ALLERGIC RESPONSE. IT IS WELL KNOWN THAT EVEN A SEVERE ALLERGIC REACTION CAN BE CONTROLLED BY AN INJECTION OF EPINEPHRINE (ADRENALINE) WHICH STRONGLY ELEVATES CAMP.

WHEN SERUM CALCIUM IS ON THE LOW SIDE OF NORMAL, IT IS PARATHYROID HORMONE WHICH IS SECRETED TO BRING THE CALCIUM BACK TOWARD NORMAL IF POSSIBLE. THIS HORMONE, CALLED PTH, ACTS BY CAUSING CALCIUM INFLUX. THERE IS A TREMENDOUS AMOUNT OF CLINICAL EXPERIENCE SHOWING THAT ALLERGIES AND ARTHRITIS BENEFIT FROM RAISING LOW SERUM CALCIUM SO AS TO SUPPRESS PTH, ESPECIALLY BY MEANS OF VITAMIN D. A STUDY AT HARVARD FOUND THAT EXACERBATIONS OF RHEUMATOID ARTHRITIS WERE LOWER IN SUMMER WHEN INCREASED SUNLIGHT EXPOSURE GENERATES MORE VITAMIN D WHICH INCREASES CALCIUM ABSORPTION²³. IN THE CLIMATE OF OAKLAND, CALIFORNIA, FOOD ALLERGY OFTEN DISAPPEARS IN THE SUMMER ALTHOUGH INHALANT ALLERGY MAY PERSIST DUE TO HIGH SEASONAL ANTIGEN LOAD²⁴. RELOCATION FROM A CLOUDY, SEACOAST AREA TO A SUNNY INLAND AREA OFTEN CLEARED UP ALLERGY. IN BRISBANE, AUSTRALIA, WHICH IS TROPICAL, THERE IS ALMOST NO SUMMER ASTHMA²⁵. AT THE DEAD SEA, CONDITIONS ARE UNUSUAL WITH NEGATIVE ALTITUDE AND MISTY ATMOSPHERE SO THAT ALL-DAY SUN BATHING IS SAFE. WITH PROLONGED SUN BATHING, PSORIASIS TENDS TO DISAPPEAR OR IMPROVE, AS DOES THE ARTHRITIS THAT SOMETIMES ACCOMPANIES PSORIASIS. BACK AT HOME, THE DISEASE RECURS IN ABOUT A MONTH AS THE VITAMIN D DISSIPATES²⁶. LETTERS TO HEALTH MAGAZINES DESCRIBING MIRACULOUS CURES COULD BE DESCRIBING WELL-KNOWN SPONTANEOUS REMISSIONS AND HAVE LITTLE VALUE. HOWEVER, THE EDITORS OF PREVENTION REMARKED ABOUT THE VOLUME OF READERS FAVORABLY COMMENTING ON CALCIUM IN ARTHRITIS. LONG BEFORE THE MODERN DRUG ERA, IT WAS CONCLUDED THAT RHEUMATOID ARTHRITIS WAS HELPED BY COD LIVER OIL AND BY MOVING IN THE TROPICAL DIRECTION^{27,28}. CALCIUM THERAPY IN ALLERGY HAS BEEN FORMALLY DESCRIBED^{29,30}. CALCIUM SUPPLEMENTS ALONE REDUCE HIGH CHOLESTEROL ABOUT 10%³¹. IT IS CLEAR THAT SUPPRESSION OF PTH WITH

VITAMIN D AND/OR CALCIUM IS A KEYSTONE OF NUTRITIONAL ALLERGY THERAPY.

POTASSIUM AND MAGNESIUM ARE SHOWN AS ACTING ALONG WITH CAMP IN RESISTING CALCIUM INFLUX AND ALLERGIC RESPONSE. THESE TWO INTRACELLULAR IONS TEND TO MOVE TOGETHER, SO THAT IF MAGNESIUM IS DEFICIENT, NO AMOUNT OF ADDED POTASSIUM CORRECTS THE SITUATION. IT IS WELL KNOWN THAT REDUCING SALT INTAKE LOWERS BLOOD PRESSURE IN SOME BUT PARADOXICALLY IT RAISES BLOOD PRESSURE IN OTHERS. THE MECHANISM OF THE SALT EFFECT HAS NOT BEEN FULLY EXPLAINED; SALT AFFECTS BLOOD VOLUME AND MAY DISPLACE INTRACELLULAR POTASSIUM. SALTY FOOD CAN TRIGGER MIGRAINE ATTACKS, AND PRIMITIVE AFRICAN TRIBES SHOWED NO ALLERGY UNTIL THE WHITE MAN BROUGHT THEM SALT. THEREAFTER THE NATIVES BEGAN TO DEVELOP HYPERTENSION AND DIABETES³². A LOW-SALT DIET APPEARS TO BE ANTI-ALLERGIC AND I AM ON SUCH A REGIME MYSELF. IT MAY BE HELPFUL TO KNOW THAT IN BLIND TESTING, ONLY 10% OF SUBJECTS COULD TELL THE DIFFERENCE BETWEEN ORDINARY SALT AND AN EQUAL MIXTURE OF SODIUM AND POTASSIUM CHLORIDES WHICH IS SOLD COMMERCIALY. POTASSIUM DIRECTIONALLY IS HELPFUL IN HYPERTENSION BUT THE EFFECT IS SMALL.

NOREPINEPHRINE HAS AS ITS BASIC ACTION MOVING CALCIUM INWARDLY JUST LIKE PTH, WHEREAS EPINEPHRINE RAISES CAMP AND THUS OPPOSES NOREPINEPHRINE. HOWEVER, IN ALLERGY WITH ITS WELL KNOWN "BETA BLOCKADE" THE MAIN ACTION OF EPINEPHRINE IS BLUNTED, EXAGGERATING ITS SECONDARY ACTION WHICH CAUSES CALCIUM INFLUX. IT IS WELL KNOWN THAT FEAR OR ANGER CAN TRIGGER AN ALLERGY ATTACK, PRESUMABLY BY PROMOTING CALCIUM INFLUX CAUSED MAINLY BY NOREPINEPHRINE. THIS EFFECT DOES NOT OCCUR IN NORMALS; APPARENTLY THERE MUST BE ANTIGEN SENSITIZATION AND LOW CAMP WAITING FOR A TRIGGERING EFFECT. NOREPINEPHRINE IS THE ONLY HORMONE INCREASING SUBSTANTIALLY DURING EXERCISE, AND THIS ELEVATION PROBABLY ACCOUNTS FOR EXERCISE ASTHMA. THE SAME EFFECT MAY OCCUR AS EXERCISE-INDUCED CARDIAC ARRHYTHMIA, CONFIRMING THE ALLERGIC PICTURE OF THESE ARRHYTHMIAS. PROSTAGLANDIN PGF_{2A} IS JUST ONE OF THE POWERFUL ALLERGY MEDIATORS WHOSE EFFECT IS VASOCONSTRICTIVE. IT HAS BEEN SHOWN IN VITRO THAT WHEN MAGNESIUM IS DEFICIENT, THIS VASOCONSTRICTION IS GREATLY EXAGGERATED AS EXPECTED FROM FIGURE 2 AND POSSIBLY COULD PRODUCE SEVERE SPASM³³. CORONARY ARTERY SPASM IS INCREASINGLY SUSPECTED IN HEART ATTACKS, AND MAGNESIUM DEFICIENCY APPEARS IMPLICATED IN MANY SUCH ATTACKS, ESPECIALLY IN SUDDEN DEATH³⁴.

NOW LET US EXAMINE CLINICAL FACTORS AFFECTING THE FIGURE 2 CONTROL SYSTEM. TWO FACTORS IN ALLERGY WORSEN CONDITIONS DRASTICALLY AND THESE HAVE BEEN LITTLE APPRECIATED. FIRST, GASTRIC ACIDITY IS LOW IN ALLERGICS^{35,36}. PROBABLY AS A RESULT OF THE LOW CAMP WHICH APPEARS TO CONTROL PART OF THE ACID SECRETION³⁷. FURTHERMORE, WITH AGE FULL ACHLORHYDRIA DEVELOPS, PRESUMABLY AS AUTOIMMUNE GASTRITIS, WHICH BASICALLY IS ALLERGIC, DESTROYS THE ACID-PRODUCING CELLS. BY AGE 60, ABOUT 25% OF THE TOTAL POPULATION HAS NO STOMACH ACID³⁸. I SUSPECT THAT MOST OF THESE ARE ALLERGICS. WITH REDUCED STOMACH ACID, THE ABSORPTION OF ALL MINERALS IS IMPEDED, AND THE MOST WIDELY USED CALCIUM SUPPLEMENT, CALCIUM CARBONATE, CAN GIVE LITTLE OR NO ABSORPTION³⁹. ADDED TO POOR ABSORPTION IS THE SECOND FACTOR, EXCESS URINARY EXCRETION OF MINERALS, ALSO CAUSED BY THE ALLERGY. IN THE GLUCOSE TOLERANCE TEST IN NORMALS, THERE IS EXCESS EXCRETION OF CALCIUM AND MAGNESIUM DURING THE FIRST THREE HOURS, CAUSED BY THE HIGH GLUCOSE AND/OR HIGH INSULIN DURING THIS PERIOD⁴⁰. ALLERGICS TYPICALLY SHOW GLUCOSE INTOLERANCE, ALWAYS WITH EXCESS INSULIN AND OFTEN WITH ABNORMALLY HIGH GLUCOSE⁴¹, SO THAT MINERAL DIURESIS IS WORSE IN ALLERGICS^{42,43}. ALLERGICS ARE TYPICALLY LOW IN SERUM CALCIUM⁴¹. A VICIOUS CYCLE DEVELOPS BECAUSE THE ABNORMAL LOSS OF CALCIUM CAN INCREASE PTH AND ABNORMAL MAGNESIUM LOSS CAN LOWER INTRACELLULAR MAGNESIUM, AND BOTH OF THESE FACTORS CAN WORSEN THE ALLERGY AND INCREASE THE LOSSES. THE DIURESIS ALSO IS WORSENED BY A DIET HIGH IN REFINED CARBOHYDRATES. SO-CALLED HYPOGLYCEMIA DIETS LOW IN REFINED CARBOHYDRATES HAVE BEEN VERY

EFFECTIVE IN ALLERGY, AND A MAJOR, UNRECOGNIZED REASON FOR THIS IS THE REDUCTION IN MINERAL DIURESIS.

THIS EXCESS CALCIUM EXCRETION ENCOURAGES KIDNEY STONES SO THAT ALLERGY IS THE UNKNOWN CAUSE OF KIDNEY STONES. KIDNEY STONES INVOLVE A HIGH FRACTION OF PATIENTS WHO EXCRETE HIGH NORMAL OR ABNORMAL LEVELS OF CALCIUM, AND IT IS CLEAR TO SOME THAT EXCESS KIDNEY LOSS IS THE PROBLEM. ONE UROLOGIST WARNS HIS STONE PATIENTS AGAINST EATING THE AMOUNT OF CARBOHYDRATE IN FOUR DONUTS. MANY UROLOGISTS UNDERSTAND THAT EXCESS CALCIUM EXCRETION IS ASSOCIATED ONLY WITH EATING, BUT THEY HAVE NOT MADE THE ALLERGY CONNECTION.

THE URINARY EXCRETION OF OTHER MINERALS NO DOUBT IS INCREASED BY ALLERGIC GLUCOSE INTOLERANCE. ZINC EXCRETION CERTAINLY IS INCREASED BUT THE URINARY ROUTE IS MINOR SO THAT THE RELATIVE EFFECT ON ZINC BALANCE IS MODERATED BUT IT IS NOT NEGLIGIBLE.

THE TWIN HANDICAPS OF REDUCED MINERAL ABSORPTION AND INCREASED EXCRETION PLACE HEAVY PRESSURE ON MINERAL BALANCE. HYPERTENSIVES ARE ALLERGIC, AND IN A RECENT STUDY A GROUP OF HYPERTENSIVES SHOWED URINARY CALCIUM EXCRETION OF 207 MG/DAY VERSUS 127 MG/DAY FOR CONTROLS⁴⁴. IN COMPANION STUDIES, PTH WAS HIGHER⁴⁵ AND SERUM IONIZED CALCIUM WAS LOWER IN HYPERTENSIVES⁴⁶. HYPERTENSIVES ALSO TEND TOWARD LOWER CALCIUM INTAKE WHICH MAKES MATTERS WORSE^{47,48,49}. THE CORRECT WAY TO PUT IT MAY BE THAT LOWER CALCIUM INTAKE IS CONDUCIVE TO HYPERTENSION. EVEN NORMAL AMERICANS HAVE DIFFICULTY WITH CALCIUM BALANCE, AS ILLUSTRATED IN TABLE IV WHICH PRESENTS RECENT SEASONAL SERUM CALCIUM LEVELS IN MARYLAND⁵⁰. THE MINIMUM CALCIUM LEVEL NO DOUBT OCCURRED BEFORE JULY, AFTER THE LONG WINTER SEASON WITH MINIMUM SUNLIGHT. HIGH PTH WOULD OCCUR AROUND THE LOW CALCIUM POINT. BY OCTOBER THE CALCIUM IS ABOVE ANYBODY'S NORMAL RANGE AS A RESULT OF THE SUMMER VITAMIN D WHICH CAN ACT ONLY WITH PTH. THESE DATA ARE NOT FROM ONE PERSON BUT REPRESENT THE AVERAGE OF 34 PERSONS. SUPPLEMENTARY VITAMIN D, ESPECIALLY NECESSARY IN WINTER, WOULD LEVEL OUT THE PEAK PTH AND COUNTERACT THE ADVERSE EFFECT ON ALLERGY AT THE TIME. THE BASIS FOR THE VITAMIN D RDA OF 200 IU/DAY IS ADMITTEDLY ALMOST ABSENT.

THE BENEFICIAL EFFECTS OF MAGNESIUM SUPPLEMENTS WHICH WOULD EXPOSE THE PROBLEMS OF MAGNESIUM DEFICIENCY ARE LESS WELL DOCUMENTED THAN THE EFFECTS OF CALCIUM AND VITAMIN D, BUT ARE NONETHELESS IMPRESSIVE. IN AN UNPUBLISHED STUDY FROM EAST TENNESSEE STATE, A MAGNESIUM SUPPLEMENT OF 200 MG/DAY ELIMINATED MIGRAINE COMPLETELY IN 65% OF 500 PATIENTS AND ANOTHER 20% WERE IMPROVED. HYPERTENSIVES AS EXPECTED ARE LOW IN MAGNESIUM⁵¹. THE EFFECTS OF MAGNESIUM ARE EVIDENT FROM THE ACTION OF AGENTS WHICH RAISE THE INTRACELLULAR LEVEL OF MAGNESIUM, ESPECIALLY VITAMIN B6⁵² AND LITHIUM⁵³. HOWEVER, THERE IS CONFUSION SINCE B6 ALSO HELPS THE SYNTHESIS OF PGE1 AS NOTED EARLIER, AND LITHIUM MAY HAVE OTHER EFFECTS ALSO. IN A RECENT BOOK, ALAN GABY⁵⁴ HAS SUMMARIZED THE USE OF B6 IN A LIST OF ALLERGIES INCLUDING ASTHMA, DEPRESSION, DIABETES, HYPERACTIVITY, PREMENSTRUAL SYNDROME, AND FORMS OF ARTHRITIS AS PUBLICIZED BY JOHN ELLIS. A COMBINATION OF B6 AND MAGNESIUM PREFERABLY IS USED, SINCE MAGNESIUM IS NEEDED AT LEAST TEMPORARILY TO REPLACE THE RELATIVELY LARGE AMOUNT MOVED OUT OF THE SERUM INTO THE CELLS. AN INTERESTING APPARENT DEMONSTRATION OF THE EFFECT OF B6 IN ALLERGY CAME IN AN EXPERIMENT WITH MONKEYS AT THE UNIVERSITY OF OREGON⁵⁵. THE MONKEYS WERE FED AN UNUSUAL DIET CONTAINING ALFALFA SEED MEAL (45%) WHICH CONTAINS A B6 ANTAGONIST WHICH TENDS TO CREATE A B6 DEFICIENCY. IN TIME THE MONKEYS DEVELOPED TWO KINDS OF ALLERGY, OBVIOUS DEPRESSION PLUS SYSTEMIC LUPUS.

HYPERTENSION IS A USEFUL MODEL FOR EXAMINING THE EFFECTS OF THESE

NUTRITIONAL VARIABLES ON CALCIUM INFLUX SINCE CALCIUM INFLUX HELPS TO CONTROL THE PERIPHERAL RESISTANCE COMPONENT OF BLOOD PRESSURE. A VERY RECENT REVIEW CONCLUDES THAT THE BASIC DEFECT IN HYPERTENSION IS ELEVATED INTRACELLULAR CALCIUM⁵⁶. AS EXPECTED, HYPERTENSION IS REDUCED BY LINOLEIC ACID⁵⁷, BY RESTRICTING SATURATED FATS⁵⁸, AND BY CALCIUM AND MAGNESIUM. THE PRITIKIN DIET REDUCES TOTAL FATS TO 10% OF CALORIES VERSUS THE USUAL 40%, AND IT ALSO REDUCES SALT. AFTER 26 DAYS, 83% OF HYPERTENSIVES SHOWED NORMAL BLOOD PRESSURE WITHOUT DRUGS⁵⁹. TO DETERMINE THE EFFECT OF DIETARY CALCIUM ALONE, HYPERTENSIVES WERE GIVEN 1000 MG/DAY OF CALCIUM, PROBABLY AS CARBONATE WHICH IS NOT A FAVORABLE FORM FOR ALLERGICS⁶⁰. A FRACTION OF THE GROUP (46%) HAD A STRONG RESPONSE WITH BLOOD PRESSURE DROPPING FROM 152/95 TO 131/88.

THE EFFECT OF MAGNESIUM HAS BEEN SHOWN IN PATIENTS ALREADY ON DIURETICS WHICH HAVE THE UNFORTUNATE EFFECT OF CAUSING MAGNESIUM DIURESIS⁶¹. DIURETICS WITHOUT POTASSIUM SPARING ARE OFFICIALLY THE FIRST CHOICE DRUGS FOR MILD HYPERTENSION, BUT IN SOME CASES SUCH DRUGS ACTUALLY INCREASE PATIENT MORTALITY, APPARENTLY BY CREATING OR WORSENING MAGNESIUM DEFICIENCY⁶². POTASSIUM SPARING IS MAGNESIUM SPARING WHICH CORRECTS THE PROBLEM. DESPITE THE OFFICIAL POLICY, DYAZIDE WITH ITS POTASSIUM AND MAGNESIUM SPARING IS TODAY'S LARGEST SELLING PRESCRIPTION DRUG. TO SHOW THE POOR STATUS OF PATIENTS ON SIMPLE DIURETICS, MAGNESIUM AT 365 MG/DAY WAS GIVEN TO 20 HYPERTENSIVES WHO WERE ON AN ASSORTMENT OF DIURETICS⁶³. THE BLOOD PRESSURE REDUCTION WAS 12/8, A LARGE EFFECT FOR PATIENTS ALREADY ON DIURETICS.

ANOTHER INSTANCE WHERE ALLERGY WORSENS THE UNDERLYING SITUATION OCCURS WITH THYROID FUNCTION. ALLERGICS TEND TO BECOME HYPOTHYROID, AND THIS MAKES MATTERS WORSE SINCE NUMEROUS STUDIES SHOW THAT CORRECTING THIS CONDITION TENDS TO BENEFIT THE ALLERGY. CORRECTING HYPOTHYROIDISM IS ALSO BENEFICIAL IN HEART DISEASE⁶⁴. EVIDENCE ON CHOLESTEROL COMES FROM BRODA BARNES WHO WAS ONE OF THE EARLIEST THYROID EXPERTS⁶⁵. BARNES TOOK 80 CONSECUTIVE PATIENTS WHOSE CHOLESTEROL WAS OVER 200 AND FOUND ALL OF THEM TO HAVE LOW TEMPERATURE BEFORE ARISING BASED ON THE SIMPLE TEST HE HAD PIONEERED. ALL WERE GIVEN THYROID EXTRACT WITH DOSAGE MONITORED BY THE TEMPERATURE, AND THE CHOLESTEROL FAILED TO DROP BELOW 200 IN ONLY 5 OF THE 80, EACH OF WHOM HAD STARTED OVER 300⁶⁶. THE MEDICAL ESTABLISHMENT DOESN'T WANT TO HEAR ABOUT THYROID IN CONNECTION WITH HEART DISEASE BECAUSE THEY MADE A MAJOR GOOF IN THE CORONARY DRUG PROJECT WHICH TESTED SEVERAL CHOLESTEROL-REDUCING AGENTS IN HEART PATIENTS. IT WAS ERRONEOUSLY CONCLUDED THAT DEXTRO-THYROXINE, THE UNNATURAL FORM OF T₄, WOULD LOWER CHOLESTEROL WITHOUT CAUSING HYPERTHYROIDISM WHICH WAS KNOWN TO BE DANGEROUS IN HEART PATIENTS. A UNIFORM DOSE OF 6 MG/DAY WAS USED, WHEREAS 4 MG/DAY GIVES FULL THYROID REPLACEMENT⁶. MANY PATIENTS MUST HAVE BECOME HYPERTHYROID; MORTALITY INCREASED COMPARED TO CONTROLS AND THE TEST HAD TO BE STOPPED PREMATURELY.

DESPITE ITS DESIRABILITY, GETTING ALLERGIC HYPOTHYROIDISM CORRECTED HAS BEEN A TREMENDOUS HURDLE SINCE THE FORMS OF HYPOTHYROIDISM ARE UNFAMILIAR; LOW T₃ AND LOW PERIPHERAL RESPONSE. THE USUAL THYROID SCREEN DOES NOT INCLUDE T₃ AND THE PHYSICIAN MAY BE MISLED BY NORMAL T₄ AND TSH. THERE IS NO ACCEPTED TEST FOR PERIPHERAL RESPONSE AND IN MY CASE THIS APPEARED TO BE THE ONLY ABNORMALITY. AFTER AN UNSUCCESSFUL BATTLE WITH MY INTERNIST, I WENT TO A WORLD-LEADING THYROID EXPERT WITH THREE COMPLAINTS, DRY SKIN, CONSTIPATION, AND LOW TEMPERATURE. SINCE NUMEROUS BLOOD TESTS WERE NORMAL, THIS EXPERT CONCLUDED THAT I COULD NOT BE HYPOTHYROID. AFTER ANOTHER ROUND WITH MY INTERNIST, HE RAISED MY T₄ DOSE AND ALL PROBLEMS RESOLVED. MY CONCLUSION FROM LONG STUDY IS THAT THE BARNES BASAL TEMPERATURE TEST IS BY FAR THE BEST TEST FOR DETECTING ALLERGIC HYPOTHYROIDISM IN RESPONSIBLE PATIENTS AND FOR MONITORING THERAPY.

I HAVE SUGGESTED THAT HEART DISEASE IS AN ALLERGY. NOTHING IS EASILY PROVED IN HEART DISEASE, BUT MY CONCLUSION IS BASED ON AN AMAZING AMOUNT OF CIRCUMSTANTIAL EVIDENCE. THIS EVIDENCE SHOWS THAT ALLERGY IS INVOLVED IN AND EVEN MAY DOMINATE ESSENTIALLY EVERY HEART DISEASE RISK FACTOR. THAT THIS SHOULD OCCUR CAN HARDLY BE A COINCIDENCE. TABLE V LISTS THE RISK FACTORS WHICH ARE DISCUSSED BRIEFLY BELOW.

HYPERTENSION WHICH USUALLY IS AN ALLERGY IS THE MOST POWERFUL AND UNEQUIVOCAL RISK FACTOR. HIGH CHOLESTEROL IS REDUCED BY ALLERGY THERAPY AND APPEARS TO BE A CONSEQUENCE OF THE LOW CAMP AND/OR HIGH INSULIN OF ALLERGY PLUS HYPOTHYROIDISM WHICH IS OFTEN ALLERGIC. TRIGLYCERIDES ARE OFTEN THE RESULT OF HIGH INSULIN. REPEATED EFFORTS TO SHOW AN ASSOCIATION BETWEEN HEART DISEASE AND NON-DIABETIC GLUCOSE INTOLERANCE HAVE FINALLY ESTABLISHED A CLEAR ASSOCIATION WITH HIGH INSULIN WHICH IS THE UNIVERSAL FORM OF ALLERGIC GLUCOSE INTOLERANCE⁶⁷. DIABETES MELLITUS ITSELF IS AN ALLERGY AND A POWERFUL RISK FACTOR. PLATELET AGGREGATION IS ENCOURAGED BY THE LOW CAMP OF ALLERGY AND IS DESCRIBED AS AN ALLERGIC RESPONSE. INTIMAL INJURY IS REGARDED BY MANY AS THE FIRST STAGE OF ATHEROSCLEROSIS, AND ALLERGIC VASCULITIS COULD BE THE MECHANISM⁶⁸. BLOOD SLUDGING AND HIGH BLOOD VISCOSITY ARE SIGNS OF INFLAMMATION COMMONLY SEEN IN ALLERGIES SUCH AS RHEUMATOID ARTHRITIS AND DIABETES AND MAY CONTRIBUTE TO CORONARY CIRCULATION PROBLEMS. CARDIAC ARRHYTHMIAS CAN BE TRIGGERED BY ALLERGY ATTACKS⁶⁹. CORONARY ARTERY SPASM CAN BE ALLERGIC AS SEEN ACCOMPANYING BEE STINGS OR AN ATTACK OF HIVES, OR SIMULATED BY HISTAMINE INFUSION. AS MENTIONED EARLIER, MAGNESIUM DEFICIENCY CAN MAGNIFY SPASM.

RECENT OBSERVATIONS HAVE SHOWN THAT FATIGUE WHICH IS SO COMMON IN ALLERGY IS ALSO SO FREQUENT IN THE PRELUDE TO HEART ATTACKS THAT IT CAN BE EVEN MORE COMMON THAN CHEST PAIN. IN ONE OF SEVERAL STUDIES, IN THE TWO WEEKS PRIOR TO SUDDEN DEATH OR MYOCARDIAL INFARCTION, 60% OF THE PATIENTS SHOWED UNEXPLAINED FATIGUE WHICH ALMOST CERTAINLY WAS ALLERGIC⁷⁰. SUCH A DIRECT, STRONG ASSOCIATION BETWEEN ALLERGY AND HEART ATTACKS IS COMPELLING. THE FATIGUE SIGNALS OXYGEN STARVATION IN THE MUSCLES GENERALLY, AND THE SAME MECHANISM MAY BE AFFECTING THE HEART.

TOBACCO SMOKING PROVIDES FREQUENT EXPOSURE TO ANTIGENS WHICH CAN CONTRIBUTE TO ANY ALLERGY. TOBACCO HAS BEEN SHOWN TO CONTRIBUTE TO PERIPHERAL VASCULAR DISEASE IN THE FORM OF BUERGER'S DISEASE. IN THE U.S., TOBACCO SMOKING INCREASES THE RISK OF HEART DISEASE 2- OR 3-FOLD, BUT IN JAPAN AND PUERTO RICO, TOBACCO ADDS NO INCREASED RISK^{71,72}. MY INTERPRETATION IS THAT BOTH OF THESE POPULATIONS ARE RELATIVELY NON-ALLERGIC. THIS IS SHOWN IN JAPAN BY THE LOW INCIDENCE OF MS. JAPAN MAY BE PROTECTED FROM ALLERGY BY ITS DIET WHICH IS LOW IN SATURATED FAT AND HIGH IN FISH OIL, AND BY A LOW RATE OF INFANT BOTTLE FEEDING WHICH IS AN IMPORTANT ALLERGY SENSITIZER IN INFANTS AND PROBABLY EVENTUALLY IN ADULTS. PUERTO RICO PROBABLY HAS LOW BOTTLE FEEDING AND ALSO HAS THE PROTECTION OF A TROPICAL CLIMATE AS DISCUSSED EARLIER. SMOKERS HAVE A HIGHER LEVEL OF ANTINUCLEAR ANTIBODIES, INDICATING A HIGHER LEVEL OF AUTOIMMUNE TISSUE ATTACK WHICH FUNDAMENTALLY IS ALLERGIC IN ORIGIN⁷³. ALL THIS EVIDENCE TENDS TO SHOW THAT THE MAJOR EFFECT OF SMOKING IS ALLERGIC RATHER THAN CHEMICAL AS COMMONLY SUPPOSED.

MANY STUDIES HAVE CONFIRMED THE PICTURE OF MEYER AND ROSENMAN THAT TYPE A BEHAVIOR DOUBLES THE RISK OF HEART DISEASE INDEPENDENT OF OTHER RISK FACTORS⁷⁴. TYPE A BEHAVIOR INVOLVES A COMPULSIVE DRIVE FOR ACCOMPLISHMENT REGARDLESS OF TIME LIMITATIONS PLUS HOSTILITY BEST DETERMINED BY INTERVIEW. THE COMPULSIVE DRIVE IS SEEN IN HYPOMANIA WHICH IS ALLERGIC, AND ALLERGIC HOSTILITY OR IRRITABILITY IS COMMON. TYPE A SHOWS A HIGH INCIDENCE OF GLUCOSE INTOLERANCE AS IN ANY ALLERGY⁷⁵. SEPARATE

STUDIES UNRELATED TO TYPE A MOSTLY SHOW AN ASSOCIATION BETWEEN HEART DISEASE AND DEPRESSION OR ANXIETY, BOTH COMMON ALLERGIES⁷⁶. THESE ARE REGARDED AS PSYCHOLOGIC OR SOCIAL RISK FACTORS, BUT THEIR MAIN IMPLICATION MAY BE AS SIGNS OF ALLERGY.

IT IS INCREASINGLY BEING RECOGNIZED THAT MAGNESIUM DEFICIENCY IS CONDUCTIVE TO CARDIAC PROBLEMS³⁴. THIS DEFICIENCY COULD RESULT FROM SOFT WATER OR DIETARY INSUFFICIENCY BUT THESE FACTORS WOULD BE MAGNIFIED IF COMBINED WITH ALLERGIC DIURESIS. OBESITY OFTEN IS CAUSED BY ALLERGY, HELPING TO ESTABLISH ITS ASSOCIATION WITH HYPERTENSION. THIS ASSOCIATION IS NOT EASILY SEPARATED IN EPIDEMIOLOGICAL STUDIES. AUTOPSIES ON KOREAN WAR SOLDIERS SHOWED A REMARKABLE INCIDENCE OF ATHEROSCLEROSIS DESPITE THE YOUTH. SIMILAR STUDIES IN FRANCE ON YOUNG ACCIDENT VICTIMS SHOWED THAT THOSE WITH EXTENSIVE ATHEROSCLEROSIS MAINLY HAD RECEIVED EARLY INFANT BOTTLE FEEDING⁷⁷, AN ALLERGY SENSITIZER.

BRIEF EXAMPLES OF EPIDEMIOLOGY MAY EMPHASIZE THE ROLES OF CALCIUM AND MAGNESIUM IN HEART DISEASE. THERE IS A LITTLE KNOWN U.S. HEART DISEASE RISK FACTOR CONSISTING OF LIVING EAST OF THE MISSISSIPPI RIVER AND SOUTH OF THE MASON-DIXON LINE⁷⁸. THE OBVIOUS DIFFERENCES ARE THAT THE SOUTHEASTERN STATES TEND TO BE LOW IN SELENIUM, HIGH IN CADMIUM, AND TO HAVE SOFT WATER. SELENIUM IS NOT WELL UNDERSTOOD BUT IT IS POSSIBLE THAT AT LEAST A SEVERE DEFICIENCY ENCOURAGES HEART DISEASE. THE ADVERSE EFFECT OF SOFT WATER, ESPECIALLY THE LOW MAGNESIUM COMPONENT, HAS BEEN CONFIRMED IN MANY BUT NOT ALL STUDIES AROUND THE WORLD. GEORGIA AND THE CAROLINAS HAVE VERY SOFT WATER AND ABOUT THE HIGHEST U.S. INCIDENCE OF TOTAL AND HYPERTENSIVE HEART DISEASE. THE CORONARY DEATH RATE IN SOUTH CAROLINA IS ABOUT 50% HIGHER THAN MY STATE, ARIZONA, WHICH HAS VERY HARD WATER. THE SOUTHEASTERN STATES ALSO HAVE THE HIGHEST U.S. INCIDENCE OF KIDNEY STONES⁷⁹, DESPITE REDUCED CALCIUM INTAKE FROM WATER. EXCESS URINARY CALCIUM IS A DOMINANT FACTOR IN KIDNEY STONES, AND IS GENERALLY CAUSED BY POOR KIDNEY RETENTION. THIS SITUATION CAN BE A SIGN OF ALLERGY AS DISCUSSED EARLIER. MY PICTURE IS THAT THE LOW CALCIUM AND MAGNESIUM INTAKE TENDS TO BIAS THE CELLS TOWARD ALLERGIC RESPONSE SINCE INITIALLY TWO OF THE THREE CRITICAL CELL CONTROL FACTORS ARE ADVERSE. ONLY A MODEST DISTURBANCE IN THE THIRD FACTOR, CAMP, MIGHT THEN ALLOW ALLERGY TO DEVELOP, PRODUCING ELEVATED URINE CALCIUM AND TENDENCY TOWARD KIDNEY STONES. I SUSPECT THAT THIS HIGH INCIDENCE OF ALLERGY ACCOUNTS PRIMARILY FOR THE HIGH RATE OF HEART DISEASE. THE KIDNEY STONE ASSOCIATION WITH HEART DISEASE ALSO HAS BEEN SHOWN IN NORWAY, WITH A KIDNEY STONE INCIDENCE IN HEART PATIENTS OF 18% VERSUS 5% IN CONTROLS⁸⁰. KIDNEY STONE PATIENTS ALSO HAVE HIGHER CHOLESTEROL⁸¹. THE HIGH CADMIUM IN THE SOUTHEASTERN STATES CONTRIBUTES TO HYPERTENSION AND THEREFORE IS AN ADDED ADVERSE FACTOR. THE CADMIUM COMES LARGELY FROM CORROSION DUE TO THE SOFT, ACID WATER.

THE TWIN CITIES OF KANSAS CITY, MISSOURI, AND KANSAS CITY, KANSAS, OFFER AN IDEAL SITUATION FOR STUDYING WATER HARDNESS⁸². BOTH CITIES USE MISSOURI RIVER WATER, BUT MISSOURI USES SOFTENING TO REDUCE HARDNESS 50%, A LEVEL STILL MUCH HARDER THAN IN THE SOUTHEAST. THE INCIDENCE OF HEART DISEASE IS HIGHER ON THE KANSAS, HARDER WATER SIDE. THE CONFUSING FACTOR IS THAT THE SOFTENING REMOVES CADMIUM SO THAT KANSAS HAS A HIGHER CADMIUM LEVEL WHICH APPARENTLY MORE THAN OFFSETS ANY BENEFIT OF THE HIGHER HARDNESS. CONSISTENT WITH THE HIGHER CADMIUM, BLOOD PRESSURE IS HIGHER ON THE KANSAS SIDE.

CONSIDERING THE POSSIBILITY THAT HEART DISEASE MAY BE AN ALLERGY, WHAT SHOULD THE PRUDENT PERSON DO ABOUT IT? CLEARING SILENT INFECTIONS SUCH AS WITH AMEBA MIGHT BE VERY BENEFICIAL. MY FEELING IS THAT TODAY'S DIETARY RECOMMENDATIONS DO NOT GO VERY FAR TOWARD SOLVING THE PROBLEM ALTHOUGH I BELIEVE THAT REDUCING SATURATED FATS AND SUGAR AND INCREASING POLYUNSATURATES ARE MAINLY ANTI-ALLERGIC. THERE ARE POTENTIALLY FRUITFUL

POSSIBILITIES SHORT OF A VEGETARIAN REGIME. TABLE VI GIVES RECOMMENDATIONS FOR A NUTRITIONAL SUPPLEMENT PROGRAM FOR BOTH ALLERGY AND HEART DISEASE CORRECTION AND PREVENTION, TO BE CARRIED OUT ONLY UNDER MEDICAL SUPERVISION. THIS RANGE OF VITAMIN D SHOULD BE NON-TOXIC FOR MOST ALTHOUGH THERE MAY BE A SLIGHT ADDED RISK OF KIDNEY STONES AND SOME PERSONS ARE HIGHLY SENSITIVE TO VITAMIN D. AMERICAN DIETS TYPICALLY CONTAIN 250-300 MG/DAY CALCIUM EXCLUDING DAIRY PRODUCTS. A GLASS OF MILK OR 1½ OUNCES OF CHEESE ADDS 300 MG CALCIUM. I WOULD AVOID CALCIUM CARBONATE IN ALL FORMS AND WOULD NOT TAKE MAGNESIUM OXIDE WITH MEALS. A 15-MG BETA-CAROTENE CAPSULE IS LABELLED 25,000 IU VITAMIN A WHICH IS FRIGHTENING SINCE THIS IS THE SAFE UPPER LIMIT OF VITAMIN A INTAKE. IN HUMANS, THE EQUIVALENCE IS ONLY ABOUT 8,000-10,000 IU SO THAT ONE DAILY CAPSULE ALLOWS ROOM FOR VITAMIN A AND PROVITAMIN A FROM FOODS.

VITAMIN C SOMETIMES LOWERS CHOLESTEROL, AND IN HEART DISEASE IT WILL CORRECT THE USUALLY LOW LEVELS OF VITAMIN C WHICH MAY BE INDICATING THE PRESENCE OF INFLAMMATION. IN EXTENSIVE INFLAMMATION, MUCH MORE VITAMIN C COULD BE TAKEN. TOGETHER WITH ZINC, THE VITAMIN C WILL SLOWLY PURGE EXCESS CADMIUM OUT OF THE BODY. SELENIUM SHOULD BE SAFE AT THIS LEVEL. A PREFERABLE FORM IS PROBABLY YEAST GROWN WITH SELENIUM (NUTRITION 21) RATHER THAN SELENIUM "ORGANICALLY BOUND TO YEAST". THE SALAD OIL TO PROVIDE LINOLEIC ACID IS PREFERABLY SAFFLOWER WHICH HAS THE HIGHEST LEVEL OF LINOLEIC ACID, BEST PURCHASED AS COLD-PRESSED OIL AS AT HEALTH STORES. THE USE OF LINSEED, FISH, OR PRIMROSE OIL IS INSUFFICIENTLY DEVELOPED FOR GENERAL RECOMMENDATIONS AT THIS TIME. UNFORTUNATELY THERE ARE NO DATA ON THE VALUE OF THIS INTEGRATED PROGRAM BEYOND THE DATA ALREADY DISCUSSED. DIRECTIONALLY, THE SELENIUM AND BETA-CAROTENE AND POSSIBLY OTHER COMPONENTS SHOULD REDUCE CANCER INCIDENCE.

TO THIS PROGRAM I WOULD ADD SOPHISTICATED ATTENTION TO THYROID STATUS. IN ADDITION, RELAXATION TRAINING IS PROMISING SINCE STRESS TENDS TO PROVOKE ALLERGY ATTACKS. ONLY SHORT TERM RESULTS ON RELAXATION TRAINING ARE AVAILABLE BUT THESE ARE ENCOURAGING.

William E. Cottrell, Sr. D.

1. ZELLER M, RHEUMATOID ARTHRITIS - FOOD ALLERGY AS A FACTOR, ANN ALLERG 7:200-205, 1949.
2. HENCH PS, THE POTENTIAL REVERSIBILITY OF RHEUMATOID ARTHRITIS, ANN RHEUM Dis 8:90-96, 1949.
3. RANDOLPH TG, ECOLOGICALLY ORIENTED RHEUMATOID ARTHRITIS, IN DICKEY LD (ED), CLINICAL ECOLOGY, SPRINGFIELD:THOMAS, 1976, PP. 201-212.
4. RANDOLPH TG, MOSS RW, AN ALTERNATIVE APPROACH TO ALLERGIES, NEW YORK: LIPPINCOTT & CROWELL, 1980; BANTAM, 1982.
5. GODLOWSKI ZZ, ALLERGY AND ANAPHYLAXIS AS METABOLIC ERROR, VOL. 2, CHICAGO:IMMUNOMETABOLIC PRESS, 1965, P. 590.
6. DEGROOT LJ ET AL, THE THYROID AND ITS DISEASES, 4TH ED., NEW YORK: WILEY, 1984.
7. RANDOLPH TG, DYNAMICS, DIAGNOSIS, AND TREATMENT OF FOOD ALLERGY, OTOL CLIN N AMER 7:617-635, 1974.
8. GRANT EEC, FOOD ALLERGIES AND MIGRAINE, LANCET 1:966-969, 1979

9. BRENNEMAN JC, ALLERGY ELIMINATION DIET AS THE MOST EFFECTIVE GALL BLADDER DIET, ANN ALLERG 26:83-87, 1968.
10. SOLL RW, GRENOBLE PB, MS--SOMETHING CAN BE DONE AND YOU CAN DO IT, CHICAGO:CONTEMPORARY, 1984.
11. FEINGOLD BF, WHY YOUR CHILD IS HYPERACTIVE, NEW YORK:RANDOM HOUSE, 1975.
12. CONNORS CK, FOOD ADDITIVES AND HYPERACTIVE CHILDREN, NEW YORK:PLENUM, 1980.
13. GUTMANN-HÖYER E, JARNUM S, INCIDENCE AND CLINICAL SIGNIFICANCE OF LACTOSE MALABSORPTION IN ULCERATIVE COLITIS AND CROHN'S DISEASE, GUT 11:338-343, 1970.
14. TSAKRACLIDES VG ET AL, CORONARY ATHEROSCLEROSIS AND MYOCARDIAL INFARCTION ASSOCIATED WITH SYSTEMIC LUPUS ERYTHEMATOSUS, AMER HEART J 87:637-641, 1974.
15. MILLER JB, FOOD ALLERGY: PROVOCATIVE TESTING AND INJECTION THERAPY, SPRINGFIELD:THOMAS, 1972.
16. CATHCART RF, VITAMIN C - TITRATING TO BOWEL TOLERANCE, MED HYPOTH 7:1359-1376, 1981.
17. HORROBIN DF, THE NUTRITIONAL REGULATION OF T-LYMPHOCYTE FUNCTION, MED HYPOTH 5:969-985, 1979.
18. BRENNER RR, NUTRITIONAL AND HORMONAL FACTORS INFLUENCING DESATURATION OF ESSENTIAL FATTY ACIDS, PROG LIP RES 20:41047, 1981.
19. HIRSH PD ET AL, PROSTAGLANDINS AND ISCHEMIC HEART DISEASE, AMER J MED 71:1009-1026, 1981.
20. HORROBIN DF, CLINICAL USES OF ESSENTIAL FATTY ACIDS, MONTREAL:EDEN, 1982, PP. 3-36, 89-96.
21. PRITIKIN N, THE PRITIKIN PROMISE: 28 DAYS TO A LONGER, HEALTHIER LIFE, NEW YORK:SIMON AND SCHUSTER, 1983.
22. SKÖLDSTAM L ET AL, EFFECTS OF FASTING AND LACTOVEGETARIAN DIET ON RHEUMATOID ARTHRITIS, SCAND J RHEUM 8:249-255, 1979.
23. SHORT CL ET AL, RHEUMATOID ARTHRITIS, CAMBRIDGE:HARVARD, 1957.
24. ROWE A, FOOD ALLERGY - ITS MANIFESTATIONS AND CONTROL, SPRINGFIELD:THOMAS, 1972.
25. WRIGHT GLT, PEDIATRIC ALLERGY IN AUSTRALIA, IN SPEER F, DOCKHORN RJ (EDS), ALLERGY AND IMMUNOLOGY IN CHILDREN, SPRINGFIELD:THOMAS, 1973, PP. 697-705.
26. AURACH WW, CLIMATOTHERAPY AT THE DEAD SEA, IN FARBER EM, COX AJ (EDS), PSORIASIS, PALO ALTO:STANFORD, 1971.
27. JORDAN EP, PRIMER ON ARTHRITIS, JAMA 119:1089-1104, 1942.
28. BRUSCH CA, JOHNSON ET, A NEW DIETARY REGIME FOR ARTHRITIS: VALUE OF COD LIVER OIL ON A FASTING STOMACH, J NATL MED ASSN 51:266-270, 1959.

29. BERNHEIM AR, A CALCIUM REGIMEN IN ALLERGY, ANN ALLERG 22:449-459, 1964.
30. REICH CJ, THE VITAMIN THERAPY OF ASTHMA, J ASTH RES 9:99-102, 1971.
31. YACOWITZ H ET AL, EFFECTS OF ORAL CALCIUM UPON SERUM LIPIDS IN MAN, BRIT MED J 1:1352-1354, 1965.
32. TROWELL HC, BURKITT DP, WESTERN DISEASES: THEIR EMERGENCE AND PREVENTION, CAMBRIDGE:HARVARD, 1981, PP. 3-32.
33. ALTURA BM ET AL, MAGNESIUM DEFICIENCY-INDUCED SPASM OF UMBILICAL VESSELS; RELATION TO PREECLAMPSIA, HYPERTENSION, GROWTH RETARDATION, SCIENCE 221:376-378, 1983.
34. SEELIG MS, MAGNESIUM DEFICIENCY IN THE PATHOGENESIS OF DISEASE, NEW YORK:PLENUM, 1980, PP. 141-264.
35. OLHAGEN B, INTESTINAL CLOSTRIDIA PERFRINGENS IN ARTHRITIS AND ALLIED CONDITIONS, IN DUMONDE DC (ED), INFECTION AND IMMUNITY IN RHEUMATIC DISEASES, OXFORD:BLACKWELL, 1976, PP. 141-145.
36. BRAY GW, THE HYPOCHLORHYDRIA OF ASTHMA IN CHILDHOOD, Q J MED 24:181, 1931.
37. RASMUSSEN H, CALCIUM AND CAMP AS SYNARCHIC MESSENGERS, NEW YORK:WILEY, PP. 217-219.
38. BARROWS CH, KOKKONEN GC, NUTRITION AND AGING, IN DRAPER HH (ED), ADVANCES IN NUTRITION RESEARCH, VOL.1, NEW YORK PLENUM, 1977, PP. 253-298.
39. IVANOVICH P ET AL, THE ABSORPTION OF CALCIUM CARBONATE, ANN INT MED 66:917-923, 1967.
40. LINDEMAN RD ET AL, INFLUENCE OF VARIOUS NUTRIENTS ON URINARY DIVALENT CATION EXCRETION, J LAB CLIN MED 70:236-245, 1967.
41. ABRAHAMSON EM, ASTHMA, DIABETES MELLITUS, AND HYPERINSULINISM, J CLIN ENDO 1:402-406, 1941.
42. LEMANN J ET AL, POSSIBLE ROLE OF CARBOHYDRATE-INDUCED CALCIURIA IN CALCIUM OXALATE KIDNEY-STONE FORMATION, NEW ENGL J MED 280:232-237, 1969.
43. BARILLA DE ET AL, AN EXAGGERATED AUGMENTATION OF RENAL CALCIUM EXCRETION FOLLOWING ORAL GLUCOSE INGESTION IN PATIENTS WITH RENAL HYPERCALCIURIA, INVEST UROL 15:486-488, 1978.
44. MORRIS CD ET AL, DISCORDANCE OF HYPERTENSIVE DIETARY CALCIUM INTAKE AND URINARY CALCIUM EXCRETION, CLIN RES 32:57A, 1984 (ABST.).
45. MCCARRON DA ET AL, ENHANCED PARATHYROID FUNCTION IN ESSENTIAL HYPERTENSION: A HOMEOSTATIC RESPONSE TO A URINARY CALCIUM LEAK, HYPERTENSION 2:162-168, 1980.
46. MCCARRON DA, LOW SERUM CONCENTRATION OF IONIZED CALCIUM IN PATIENTS WITH HYPERTENSION, NEW ENGL J MED 307:226-228, 1982.
47. MCCARRON DA, CALCIUM AND MAGNESIUM INTERACTION IN HUMAN HYPERTENSION, ANN INT MED 98:800-805, 1983.
48. GARCIA-PALMIERI MR ET AL, MILK CONSUMPTION, CALCIUM INTAKE, AND DECREASED HYPERTENSION IN PUERTO RICO, HYPERTENSION 6:322-328, 1984.

49. ACKLEY S ET AL, DAIRY PRODUCTS, CALCIUM, AND BLOOD PRESSURE, AMER J CLIN NUTR 38:457-461, 1983.
50. LAKSHMANAN FL ET AL, CALCIUM AND PHOSPHORUS INTAKES, BALANCES, AND BLOOD LEVELS OF ADULTS CONSUMING SELF-SELECTED DIETS, AMER J CLIN NUTR 40:1368-1379, 1984.
51. ALBERT DG ET AL, SERUM MAGNESIUM AND PLASMA SODIUM LEVELS IN ESSENTIAL HYPERTENSION, CIRC 17:761-764, 1958.
52. AIKAWA JK, EFFECT OF PYRIDOXINE AND DESOXYPYRIDOXINE ON MAGNESIUM IN THE RABBIT, PROC SOC EXP BIOL MED 104:461-463, 1960.
53. PLENGE P, RAFAELSEN OJ, LITHIUM EFFECTS ON CALCIUM, MAGNESIUM, AND PHOSPHATE IN MAN, ACTA PSYCHIAT 66:361-373, 1982.
54. GABY A, THE DOCTOR'S GUIDE TO VITAMIN B6, EMMAUS:RODALE, 1984.
55. BARDANA EJ, DIET-INDUCED SYSTEMIC LUPUS ERYTHEMATOSUS IN PRIMATES, AMER J KID DIS 1:345-352, 1982.
56. RESNICK LM, DIVALENT CATIONS IN ESSENTIAL HYPERTENSION, NEW ENGL J MED 309:888-891, 1983.
57. FLEISCHMAN AI, EFFECT OF INCREASED DIETARY LINOLEATE UPON BLOOD PRESSURE, PREV MED 8:163, 1979.
58. PUSKA P ET AL, CONTROLLED, RANDOMIZED TRIAL OF THE EFFECT OF DIETARY FAT ON BLOOD PRESSURE, LANCET 1:1-5, 1983.
59. BARNARD RJ, EFFECTS OF AN INTENSIVE EXERCISE AND NUTRITION PROGRAM ON PATIENTS WITH CORONARY ARTERY DISEASE, J CARD REHAB 3:183-190, 1983.
60. MCCARRON DA, MORRIS C, CLIN RES 32:335A, 1984 (ABST.).
61. REYES AJ, LEARY WP, MAGNESIUM DEFICIENCY PROVOKED BY DIURETICS, SO AFR MED J 63:410-412, 1983.
62. MORGAN TO, FAILURE OF THERAPY TO IMPROVE PROGNOSIS IN ELDERLY MALES WITH HYPERTENSION, MED J AUST 2:27-31, 1980.
63. DYCKNER T, WESTER PO, EFFECT OF MAGNESIUM ON BLOOD PRESSURE, BRIT MED J 286:1847-1849, 1983.
64. WREN JC, SYMPTOMATIC ATHEROSCLEROSIS: PREVENTION AND MODIFICATION BY TREATMENT WITH DESICCATED THYROID, J AMER GER SOC 19:7-22, 1971.
65. BARNES BO, GALTON L, HYPOTHYROIDISM: THE UNSUSPECTED ILLNESS, NEW YORK:CROWELL, 1976.
66. BARNES BO, PROPHYLAXIS OF ISCHEMIC HEART DISEASE BY THYROID EXTRACT, LANCET 2:149-152, 1959.
67. PYÖRÄLÄ K, RELATIONSHIP OF GLUCOSE TOLERANCE AND PLASMA INSULIN TO THE INCIDENCE OF CORONARY HEART DISEASE: RESULTS FROM TWO POPULATION STUDIES IN FINLAND, DIAB CARE 2:131-141, 1979.
68. BECKER CG, MINICK CR, THE ROLE OF IMMUNOLOGIC INJURY IN THE PATHOGENESIS OF ATHEROSCLEROSIS, IN ZABRISKIE JB ET AL (EDS), CLINICAL IMMUNOLOGY OF THE HEART, NEW YORK:WILEY, 1981.

69. REA WJ, ENVIRONMENTALLY TRIGGERED CARDIAC DISEASE, ANN ALLERG 40:243-251, 1978.
70. KULLER LH, PRODRROMATA OF SUDDEN DEATH, ADV CARD 25:61-72, 1978.
71. KEYS ET AL, THE SEVEN COUNTRIES STUDY: 2,289 DEATHS IN 15 YEARS, PREV MED 13:141-154, 1984.
72. GORDON T ET AL, DIFFERENCES IN CORONARY HEART DISEASE IN FRAMINGHAM, HONOLULU, AND PUERTO RICO, J CHR Dis 27:329-344, 1974.
73. MATTHEWS JD, ASSOCIATION OF AUTOANTIBODIES WITH SMOKING, CARDIOVASCULAR MORBIDITY AND DEATH IN THE BUSSELTON POPULATION, LANCET 2:754-759, 1973.
74. HAYNES SG ET AL, THE RELATIONSHIP OF PSYCHOSOCIAL FACTORS TO CORONARY HEART DISEASE IN THE FRAMINGHAM STUDY - III: EIGHT-YEAR INCIDENCE OF CORONARY HEART DISEASE, AMER J EPID 111:37-58, 1980.
75. FRIEDMAN M ET AL, CORONARY-PRONE INDIVIDUALS (TYPE A BEHAVIOR PATTERN) - SOME BIOCHEMICAL CHARACTERISTICS, JAMA 212:1030-1037, 1970.
76. JENKINS CD, RECENT EVIDENCE SUPPORTING PSYCHOLOGIC AND SOCIAL RISK FACTORS FOR CORONARY DISEASE, NEW ENGL J MED 294:987-994, 1976.
77. OSBORN GR, RELATIONSHIP OF HYPERTENSION AND INFANT FEEDING TO THE ETIOLOGY OF CORONARY DISEASE, COLLOQUES INT CENT NAT RECH SCI 169: 93, 1967.
78. McMILLAN GC, CARDIOVASCULAR DISEASE, IN GEOCHEMISTRY AND THE ENVIRONMENT, VOL 3, WASHINGTON:NATIONAL ACADEMY OF SCIENCE, 1978, pp. 114-132.
79. LANDES RR ET AL, AN INQUIRY INTO THE RELATION BETWEEN WATER HARDNESS AND THE FREQUENCY OF UROLITHIASIS, IN SEELIG MS (ED), NUTRITIONAL IMBALANCES IN INFANT AND ADULT DISEASES, NEW YORK:SPECTRUM, 1977, pp. 9-21.
80. LINDEN V, CORRELATION OF VITAMIN D INTAKE TO ISCHEMIC HEART DISEASE, HYPERCHOLESTEROLEMIA, AND RENAL CALCINOSIS, IN SEELIG (REF 79), pp. 23-42.
81. WESTLUND K, UROLITHIASIS AND CORONARY HEART DISEASE, AMER J EPID 97:167-172, 1973.
82. BIERENBAUM ML ET AL, POSSIBLE TOXIC WATER FACTORS IN CORONARY HEART DISEASE, LANCET 1:1008-1010, 1975.

TABLE I

INDIVIDUAL FOOD CHALLENGES IN A RHEUMATOID ARTHRITIS PATIENT

MILK: 4 HOURS, NASAL STUFFINESS; 12 HOURS, ARTHRITIS PAINS AND HUSKY VOICE

BEEF: 6 HOURS, SLIGHT ARTHRITIC PAINS; 18 HOURS, AWAKENED WITH ARTHRITIS

CORN: 9 HOURS, RECURRENT JOINT PAINS

RICE: 12 HOURS, ARTHRITIC PAINS

CANE SUGAR: 12 HOURS, SLIGHTLY PAINFUL JOINTS.

WHEAT: 12 HOURS, AWAKENED COLD AND PERSPIRING WITH FATIGUE AND SORE THROAT

TABLE II
COMMON FORMS OF FOOD ALLERGY

AGGRESSION	HYPERTENSION
ALCOHOLISM	HYPOTHYROIDISM
ANXIETY	IRRITABLE BOWEL
ARTHRITIS/GOUT	IRRITABILITY
ATOPIC DERMATITIS (ECZEMA)	KIDNEY STONES
BEDWETTING (ENURESIS)	LEARNING DISORDERS
BRONCHIAL ASTHMA	LUPUS
CARDIAC ARRHYTHMIAS	MENIERE'S DISEASE
CELIAC DISEASE	MULTIPLE SCLEROSIS (MS)
COLITIS/INFLAMMATORY BOWEL	MYASTHENIA GRAVIS
COLLAGEN VASCULAR DISEASES	NEPHRITIS/NEPHROSIS
CONFUSION	OBESITY
DEPRESSION/MANIA	OTITIS
DIABETES/HYPOGLYCEMIA	PHLEBITIS
EPILEPSY	PREMENSTRUAL SYNDROME
FATIGUE	PSORIASIS
GALLBLADDER PROBLEMS	RHINITIS/SINUSITIS
GASTRIC ULCER	SCHIZOPHRENIA
HEADACHE/MIGRAINE	URTICARIA (HIVES)
HYPERACTIVITY	

IN MANY CASES, NON-ALLERGY CAUSES MAY EXIST

FIGURE 1
ESSENTIAL FATTY ACID PROCESSING IN THE BODY

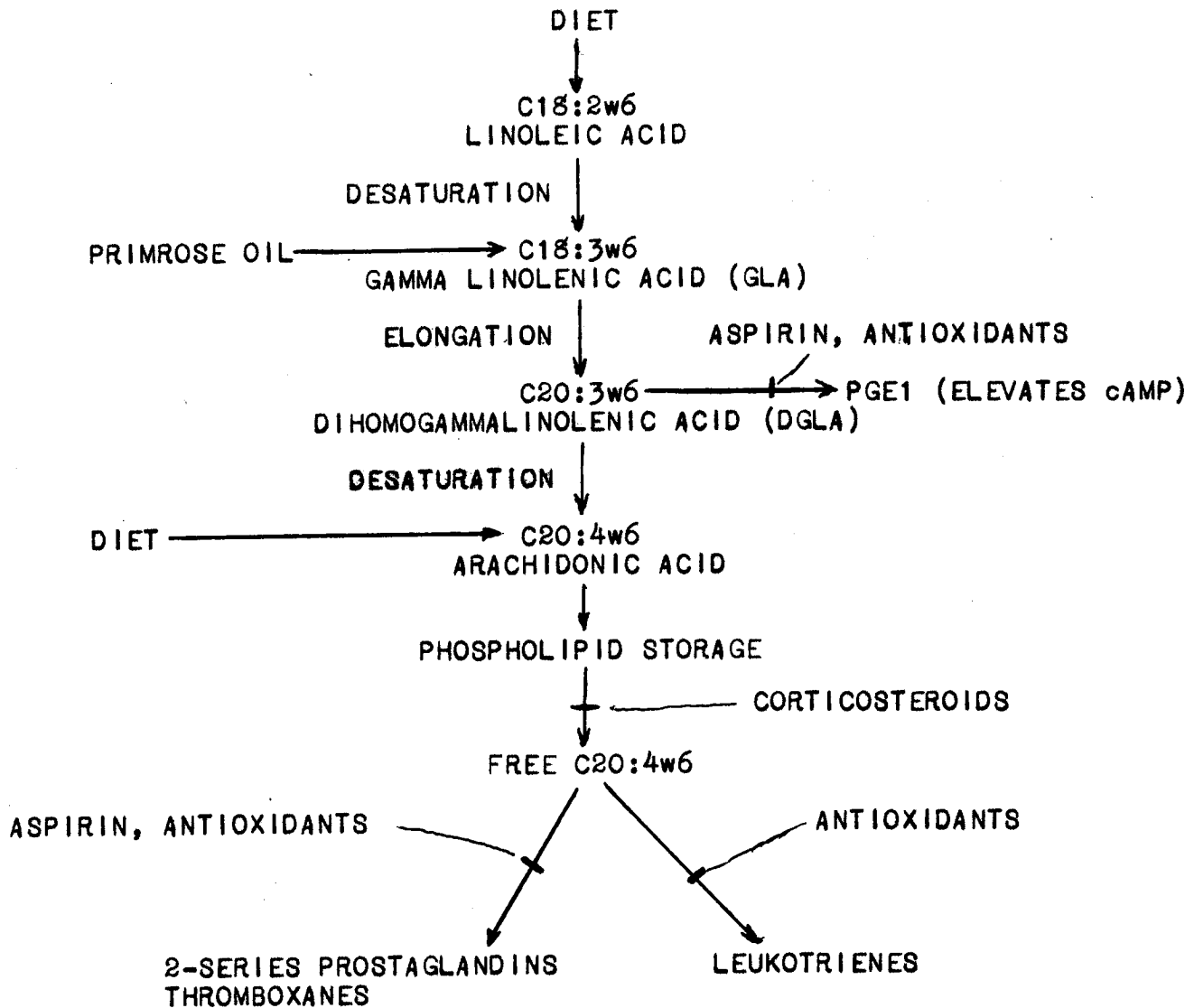


TABLE III

SOME FACTORS CONTROLLING DELTA-6 DESATURASE ENZYME

FACTORS INCREASING ACTIVITY

ZINC
VITAMIN B3
VITAMIN B6
VITAMIN C
MAGNESIUM
INSULIN

FACTORS DECREASING ACTIVITY

SATURATED FATS
HIGH CARBOHYDRATE DIET
PARTIALLY HYDROGENATED FATS
AGING

FIGURE 2
SIMPLIFIED CELL CALCIUM CONTROL

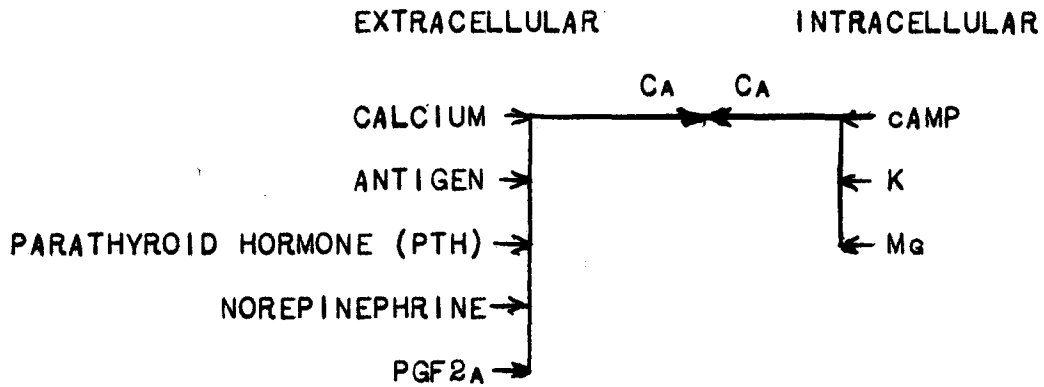


TABLE IV
SERUM CALCIUM IN 34 NORMAL MARYLAND ADULTS

APRIL 1981	9.81 mg%
JULY 1981	9.60
OCTOBER 1981	11.51
JANUARY 1982	10.84
APRIL 1982	10.10

TABLE V

HEART DISEASE RISK FACTORS TEND TO BE ALLERGY FACTORS

1. HYPERTENSION
2. ELEVATED CHOLESTEROL AND TRIGLYCERIDE
3. HYPERINSULINEMIA
4. DIABETES MELLITUS
5. PLATELET AGGREGATION IS ALLERGIC RESPONSE
6. INTIMAL INJURY BY ALLERGIC VASCULITIS
7. BLOOD SLUDGING AND HIGH VISCOSITY
8. CARDIAC ARRHYTHMIAS
9. CORONARY ARTERY SPASM
10. FATIGUE PRODROMAL IN HEART ATTACKS
11. TOBACCO IS MAJOR ALLERGEN BENIGN IN LOW-ALLERGY COUNTRIES
12. TYPE A BEHAVIOR IS LIKE ALLERGIC HYPOMANIA AND IRRITABILITY
13. MAGNESIUM DEFICIENCY OFTEN INDUCED BY ALLERGY
14. OBESITY OFTEN ALLERGIC

TABLE VI
ADULT SUPPLEMENTS FOR ALLERGY TREATMENT AND PREVENTION
(TO BE TAKEN UNDER MEDICAL SUPERVISION)

	<u>DAILY DOSE</u>
VITAMIN D	400-1000 IU TOTAL DIET
CALCIUM	1000 MG TOTAL DIET
MAGNESIUM	400 MG
B VITAMINS	ONE B-50 COMPLEX TABLET
ADDED VITAMIN B6	0 TO 400 MG
ZINC (AS PICOLINATE)	30 MG
BETA-CAROTENE	15 MG
VITAMIN C	2000 MG DIVIDED
VITAMIN E	100-200 IU
SELENIUM	100 MCG
SALAD OIL	ONE TBSP

IN ADDITION, SATURATED FATS, HYDROGENATED FATS, AND SALT PREFERABLY SHOULD BE MINIMIZED