Below is a summary of the purported clinical benefits of curcumin. Note, however, that most of these benefits are demonstrated in animal studies and cell culture studies (with the curcumin administered by injection in the animal studies). There are only two or perhaps three curcumin products that are absorbed significantly well enough to have any desired clinical benefits. --- Most curcumin bought at the health food store has no more benefit than seasoning food with the spice turmeric.

So --- your main take-away from this write-up is that all the extraordinary benefits of curcumin demonstrated in cell cultures are on cell cultures, not live critters, and, all the extraordinary benefits in laboratory animals involve curcumin administrated by injection, not taken orally. The few studies looking at oral supplementation of both animals and human beings do not demonstrate the same extraordinary effects.

But an even more significant point is that even if curcumin does 100% of what its health food myth peddlers like to claim, it does absolutely nothing that the adaptogens in your ADAPTO-MAX (Diphasic A.M.) and OXY-MAX (Diphasic P.M.) do not do even better. --- And those adaptogens have demonstrated their benefits on human beings with no doubt about oral absorption and no doubt about biological activity.

[As regards effects on NUTRI-SPEC testing --- curcumin is somewhat anti-Anaerobic at the tissue level and anti-Dysaerobic at the systemic level. The effects it would have on your patients’ test results would be minimal, so ignore when testing --- then, tell those patients to quit throwing their money away on health food mythology. ]

- Curcumin is diferuloylmethane, the bioactive component of the spice turmeric.
- Curcumin is fat soluble and very poorly absorbed. One study shows that after ingesting 10 grams orally, absolutely none is detected in the blood.
- One reason for the poor bioavailability of curcumin is its rapid plasma clearance and conjugation. This rapid elimination of curcumin may be a greater factor in its limited bioavailability than its poor intestinal absorption. Most of the curcumin is eliminated via efficient first-pass metabolism, and some curcumin undergoes intestinal metabolism, particularly glucuronidation and sulfation.
Reconstituting curcumin with the non-curcuminoid essential oil of turmeric increases its bioavailability substantially. A patented formulation BCM-95 (R) CG increases blood levels after oral supplementation 6.93 times that achieved by plain curcumin and 6.3 times compared to curcumin-lecithin-piperine (a combination that enhances bioavailability over plain curcumin). Fasting volunteers consumed 2000 mg of Biocurcumax and blood was drawn every hour for 8 hours after dosing. BCM-95 (R) CG trades as Biocurcumax. Biocurcumax is supplied by Arjuna Natural Extracts Limited (Kerala, India) in 500 mg capsules. BCM-95 softgels are sold as CircuGel, a trademark of Tishcon. BCM-95 and Bio-Curcumin are trademarks of Dolcas-Biotech.

BCM-95 softgel CurcuGel is a trademark of Tishcon (one of the companies that manufactures NUTRI-SPEC products under contract).

Cyclodextrin-complexed curcumin has superior bioavailability compared to ordinary curcumin. The cyclodextrin-complexed curcumin demonstrates greater cellular uptake and longer half-life in the cells. [----- So, the bioavailability of polyphenolic herbal drugs is enhanced by cyclodextrin. There is no advantage at all to cyclodextrin added to the vitamins, minerals, trace minerals, amino acids, and adaptogens in NUTRI-SPEC products since our supplements are already in well-absorbed bio-available forms.]

Liposomal curcumin formulated with phosphatidylcholine increases the oral bioavailability 5-fold over ordinary curcumin. Peak plasma level, duration of plasma elevation, and liver concentration are higher. The trade name for the phosphatidylcholine-curcumin formulation is Meriva.

The product named Cucumin is a combination of curcumin and the amino acid, DL-phenylalanine, plus two other herbal drugs. [DL-phenylalanine (a combination of l-phenylalanine as you get from NUTRI-SPEC, plus d-phenylalanine) was highly promoted as an anti-inflammatory and arthritis “cure” back in the 80s. Its anti-inflammatory effects were so small as to not even begin to justify the cost, so the hype disappeared. Apparently, someone is trying to revive it now.]

BDMC = demethylated curcuminoids produced from ordinary C95 = increased neuroprotective and anti-inflammatory effects, and antagonizes the effects of TNF-alpha on endothelial cells --- but --- plain curcumin is better than BDMC in the antagonism of TNF-alpha = demonstrates the critical role of methoxy groups on the phenyl ring.

We present these tidbits on several Curcumin-derived products, not as in an endorsement, but merely to alert you that there may be a few Curcumin products that are slightly better than worthless. Another way to look at this information is that any products that is _not_ one of the few mentioned above is
entirely a waste of your patient’s money. And as you read through the next few pages of purported benefits of Curcumin, keep in mind that these are merely factoids, and almost all based upon either cell culture studies or animal studies by injection. And further keep in mind that these same benefits can be derived much more effectively from your NUTRI-SPEC adaptogen supplements such as ADAPTO-MAX, OXY-MAX, TAURINE, and of course, IMMUNOSYNBITOICS.

- Curcumin penetrates the blood brain barrier, and protects the brain from lipid peroxidation.

- Curcumin is reported to have beneficial anti-inflammatory and anti-cancer properties. In colitis, curcumin inhibits NF-kappa B. The Peroxisome Proliferator-Activated Receptor gamma (PPAR gamma) is a nuclear receptor with anti-tumor and anti-inflammatory effects, and its activation may inhibit NF-kappa B. PPAR gamma has shown therapeutic benefits in colitis. Curcumin (30 mg/kg/d) administered intraperitoneally just after colitis is induced in rats, improves long-term survival, promotes rat body weight recovery, and decreases macroscopic scores of the colitis. The expression of PPAR gamma, Prostaglandins PGJ2 & PGE2 (anti-inflammatory in the gut), are all increased by curcumin injection, but the expression of COX-2 is inhibited, just as in treatment with dexamethasone. However, dexamethasone also inhibits expression of PGJ2 and PGE2.

- Curcumin decreases NF-kappa B, TNF-α, IFN-γ, NOS, IL-1, IL-6, COX2, COX1, and 5-lipoxygenase. In IBS, curcumin suppresses colitis by changing the cytokine profile from pro-inflammatory Th1 to anti-inflammatory Th2.

- Curcumin decreases pro-inflammatory cytokines including TNF-alpha, IL-1, IL-2, IL-6, IL-8, and IL-12 --- but --- at low doses curcumin also enhances antibody responses.

- Curcumin modulates activation of T cells, B cells, macrophages, neutrophils, natural killer cells, and dendritic cells.

- Curcumin is an oxygen radical and hydroxyl radical scavenger and hydrogen donor, and exhibits both pro-oxidant and antioxidant activity.

- Curcumin is a cyclooxygenase 2 (Cox-2) inhibitor = decreases production of Prostaglandins, both good (PGE1, PGI2) and “bad” (PGD2, PGE2, Thromboxane)

- Curcumin binds metals, particularly iron and copper, and can function as an iron chelator.
Curcumin’s surprisingly wide range of beneficial properties, including anti-inflammatory, antioxidant, chemo preventative, and chemotherapeutic activity, derive from its complex chemistry as well as its ability to influence multiple signaling pathways— including survival pathways such as those regulated by NF-kappaB and growth factors, cytoprotective pathways dependent on Nrf2, and metastatic and angiogenic pathways.

Curcumin is purported to be clinically effective in: allergy, asthma, atherosclerosis, heart disease, diabetes, cancer, Alzheimer’s disease, Crohn’s disease.

In Fungal Exposure Endocrinopathy, curcumin inhibits proliferation of folliculostellate cells, and stimulates apoptosis. Immune-like functions of FS cells were impaired since curcumin down-regulates toll-like receptor 4, decreases NF-kappa B, and decreases bacterial endotoxin-induced IL-6.

Curcumin, S. boulardii, and melatonin are all immunosuppressive— particularly suppressing the portions of the immune system that defend against cancer. [Note the conflicting evidence in the literature regarding Curcumin’s effect on cancer.]

One major concern with therapeutic agents like curcumin is that their anti-inflammatory effects actually inhibit the Th1 cytokines that cancer patients so desperately need. Since curcumin can suppress Tumor Necrosis Factor-α and Interferon-γ and Natural Killer Cells, it may put cancer patients at an extreme disadvantage.

Another problem with Curcumin is that it quite effectively blocks the liver enzyme cytochrome P450. Cytochrome P450 has thousands of functions, and blocking it can create major problems.

Curcumin is anti-inflammatory; curcumin is immunosuppressive. The two mean the same thing. Neutrophils, eosinophils, mast cells, basophils, macrophages, dendritic cells, and monocytes all have inflammatory actions as part of their defense mechanism. Their release of pro-inflammatory cytokines is part of many healing processes as well as many anti-microbial defense processes. --- But --- in most patients, the inflammatory effects of the immune activity are either excessive, or out of time with need. Thus the symptomatic improvements from anti-inflammatory immunosuppressive drugs such as curcumin (diferuloylmethane).

Determining whether a particular herbal drug has estrogenic activity can pretty well be done just by looking at its molecular structure and seeing the multiple phenol cyclic structure with OH groups in appropriate locations. But really there is no reason to even take an educated guess since the medical literature clearly defines the estrogenic activity of most herbal
drugs. They have all been studied and written up extensively. (Almost anything that falls into either the flavonoid or flavanol family has some estrogen activity.)

- Just what does it mean when the literature designates a particular herbal drug as estrogenic? It does not necessarily mean that the drug performs in the body like estradiol, estriol, or estrone. It means that the molecular structure is such that the molecule binds to estrogen sites on cell membranes. What happens from there is variable to an extreme. Sometimes the binding of the estrogenic drug elicits a second messenger response within the cell; sometimes just the opposite occurs. The presence of the herb on the estrogen receptor does nothing but occupy space, thus preventing that binding site from receiving a physiological estrogen. In other words, some herbs that have an estrogen structure activate estrogen-type effects, and some of them actually can inhibit estrogen effects.

We see the research on curcumin the same way we see research on Resveratrol (which we wrote up in an Article on your NUTRI-SPEC website, “The Truth About the Resveratrol Hoax”). The effects of curcumin in cell cultures and in animals and in humans is all over the place, and often contradictory, and most of all has no unifying mechanism to explain any of its purported effects.

RED DERMOGRAPHICS === MAST CELLS = HYPERSENSITIVE (not excessive in number) By far the most effective mast cell inhibitor is Quercitin. Also somewhat effective, but with undesirable side effects, are: luteolin, chondroitin sulfate, curcumin, ketotifen, daidzein, genistein (daidzein & genistein are extremely estrogenic = breast tumors), Gastrocrom, dehydroleocodine, chrysin, fisetin, acacetin, epigallocatechin gallate. ----- Mast Cell hypersensitivity is associated with:

- Parasympathetic Imbalance
- Tissue Acidosis (Anaerobic Imbalance)
- Excess Corticotrophin Releasing Hormone (CRH)
- Alkalosis Imbalance
- Lipopolysaccharide (LPS, endotoxin)

- Curcumin: Curcumin inhibits antigen-mediated activation of mast cells. It suppresses mast cell degranulation and secretion of TNF-α and IL-4. Curcumin thus suppresses IgE-mediated allergies.
- However --- isoflavones such as Curcumin have estrogenic activity and may not be desirable in certain clinical settings.
- NUTRI-SPEC Metabolic Balancing, restoring Vital Reserves with the DNP and other Metabolic Support Systems, along with IMMUNO-SYNBIOTIC, are the keys to mast cell control.