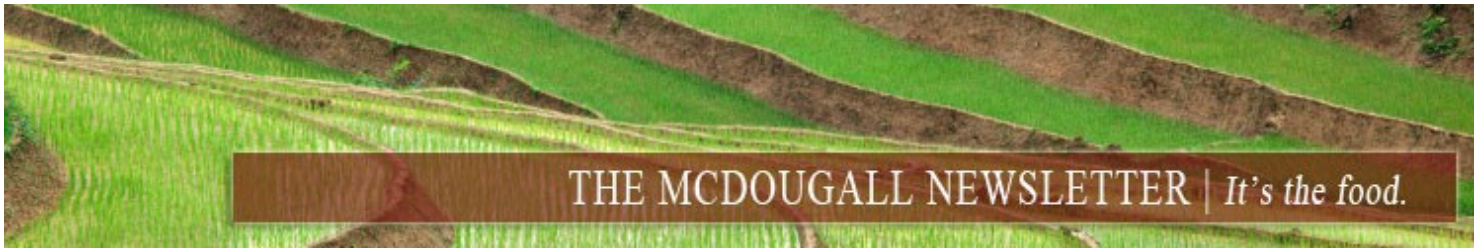

Favorite Five Articles from Recent Medical Journals

Diabetic Treatments Fail to Help Eyes and Kidneys

Effect of intensive treatment of hyperglycaemia on microvascular outcomes in type 2 diabetes: an analysis of the ACCORD randomised trial by Faramarz Ismail-Beigi, published in the August 7, 2010 issue of the *Lancet* reported, "We recorded no significant effect of intensive glycaemia therapy on the two prespecified composite microvascular outcomes—1) advanced renal or eye complications, or 2) these two outcomes or peripheral neuropathy."[1]

The ACCORD (Action to Control Cardiovascular Risk in Diabetes) study was a randomized trial done in 77 clinical sites in North America. People with type-2 diabetes, high HbA(1c) concentrations (>7.5%), and cardiovascular disease were randomly assigned to intensive (target haemoglobin A(1c) of <6.0%) or standard (7.0-7.9%) glycaemic therapy. On February 6, 2008 the National Heart, Lung, and Blood Institute (NHLBI) stopped the ACCORD study when results showed that intensive treatment of diabetics increased their risk of dying compared to those patients treated less aggressively. However, research, such as the study discussed here, continues to be published on outcomes of these 10,251 participants with type-2 diabetes

Comment: All six major studies published over the past 14 years show that attempts by physicians to make the patients' blood sugars and hemoglobin A(1c) levels look more "normal" with medications harm the patients. Patients in the intensive treatment groups oftentimes are taking four shots of insulin and three pills daily, and checking their blood-sugar levels four times a day—their entire lives are focused on this diseaase. Compared to people treated less aggressively, they have a greater risk of death, heart attacks, and hypoglycemia reactions, as well as gaining about twice as much body weight. Based on these consistent results, what excuses do doctors use to justify treating their patients aggressively?

The primary excuse is that such extraordinary efforts will reduce damage to the eyes (retinopathy), kidneys (nephropathy), and nerves (neuropathy). These problems are referred to as *microvascular complications*, because they involve small blood vessels rather than the larger blood vessels of the heart and brain (macrovascular). *However, the research that supports benefits for reducing microvascular complications is insufficient to justify the proven harms from aggressive treatment for people with type-2 diabetes.*[2]

Summary of Results for Microvascular Complications

Study	# of Participants	Benefit Found?
Diabetes Control and Complications Trial[3]	1441	Yes - type 1
Stockholm Diabetes Intervention Study[4]	91	Yes - type 1
UK Prospective Diabetes Study (UKPDS) Group[5]	753	Yes – type 2
Japanese Study[6]	110	Yes – type 2
ADVANCE[7]	11,140	Yes – type 2
ACCORD Study[1]	10,251	No – type 2
Veterans (VADT) study[8]	1,791	No – type 2

Evidence for the benefits of tightly controlling blood sugars to low levels (near normal based on a hemoglobin A(1c) of 6%) was first seen with patients with type-1 diabetes (formerly called childhood diabetes and is due to severe damage to the pancreas). One large study and one small study have shown small benefits on microvascular disease occurence from treatment of patients with type-1 diabetes with intensive insulin therapy.[3,4]

For type-2 diabetes studies, results are more contradictory and overall indicate little or no reduction in microvascular complications with intensive therapy. One large, one medium, and one small study showed small benefits of oral agents and/or insulin in treating type-2 diabetics. However, two very large studies recently published, the ACCORD and the Veterans Study, did not show benefits from intensive therapy for microvascular disease. To put these findings in a clinically relevant prespective, the benefits reported for microvascular disease in all of these five studies of patients with type-2 diabetes were limited to a reduction of protein in the urine (microalbuminuria).[1,5-8] Reductions in serious kidney (nephropathy, kidney failure) and eye (retinopathy, visual deterioration) disease were not found with intensive treatment in any of these studies.[2]

Consistent with my conclusions, researchers published an analysis of 13 randomized studies involving 34,533 patients in the July 27, 2011 issue of the *British Medical Journal* and found that intensive glucose treatment did not significantly affect all-cause mortality or cardiovascular death.[2] There was a 15% reduction in the risk of non-fatal heart attacks and a 10% reduction in protein in the urine (microalbuminuria), but more than a two-fold increase in the risk of severe hypoglycemia (dangerously low blood glucose levels) with intensive treatment.[2] They concluded: "The benefit:risk ratio of intensive glucose lowering treatment in the prevention of macrovascular and microvascular events remains uncertain. The harm associated with severe hypoglycemia might counterbalance the potential benefit of intensive glucose lowering treatment…Intensive glucose lowering treatment of type 2 diabetes should be considered with caution and therapeutic escalation should be limited."

My overall conclusions are: Based upon the overall benefits, costs, and risks, people with type-2 diabetes should be treated conservatively, with little or no medications to lower blood sugar. (Type-1 diabetics always require insulin.) A healthy starch-based, low-fat diet should be the foundation of their care. This diet along with exercise, followed by substantial weight-loss, will cure almost all people with type-2 diabetes. Furthermore, a healthy low-protein diet (especially low in animal protein) will substantially reduce the amout of protein in the urine (microalbuminuria) of patients with diabetes.[9-11] A low-fat, starch-based diet has also been shown to reverse some of the eye damage caused by diabetes.[12,13] This is the same low-fat, low-protein, high-starch diet (for example, the McDougall Diet) that dramatically reduces the risk of macrovascular diseases (strokes and heart attacks), without adverse effects or costs.

I do occasionally prescribe long-acting insulin for people with type-2 diabetes under specific circumstances. In the past I have made efforts to require by law that people in the state of California be told that medications used to treat type-2 diabetes will substantially increase their risk of death and bodily damage. This year I plan to further my efforts with the state legislators.

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2) Boussageon R, Bejan-Angoulvant T, Saadatian-Elahi M, Lafont S, Bergeonneau C, Kassai B, et al. Effect of intensive glucose lowering treatment on all cause mortality, cardiovascular death, and microvascular events in type 2 diabetes: meta-analysis of randomised controlled trials. *BMJ* 2011;343:d4169.

3) The effect of intensive treatment of diabetes on the development and progression of long-term complications in insulin-dependent diabetes mellitus. The Diabetes Control and Complications Trial Research Group. *N Engl J Med.* 1993 Sep 30;329 (14):977-86.

4) Reichard P, Pihl M, Rosenqvist U, Sule J. Complications in IDDM are caused by elevated blood glucose level: the Stockholm Diabetes Intervention Study (SDIS) at 10-year follow up. *Diabetologia.* 1996 Dec;39(12):1483-8.

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7) ADVANCE Collaborative Group, Patel A, MacMahon S, Chalmers J, Neal B, Billot L, Woodward M, Marre M, Cooper M, Glasziou P, Grobbee D, Hamet P, Harrap S, Heller S, Liu L, Mancia G, Mogensen CE, Pan C, Poulter N, Rodgers A, Williams B, Bompoint S, de Galan BE, Joshi R, Travert F. Intensive blood glucose control and vascular outcomes in patients with type 2 diabetes. *N Engl J Med.* 2008 Jun 12;358(24):2560-72.

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11) Brouhard BH, LaGrone L. Effect of dietary protein restriction on functional renal reserve in diabetic nephropathy. *Am J Med.* 1990 Oct;89(4):427-31.

12 Van Eck WF.) The effect of a low fat diet on the serum lipids in diabetes and its significance in diabetic retinopathy. *Am J Med.* 1959 Aug;27:196-211.

13) Kempner W. Radical dietary treatment of hypertensive and arteriosclerotic vascular disease, heart and kidney disease, and vascular retinopathy. *GP.* 1954 Mar;9(3):71-92.

Cardiologists Acting Criminally

Impact of National Clinical Guideline Recommendations for Revascularization of Persistently Occluded Infarct-Related Arteries on Clinical Practice in the United States by Marc W. Deyell in the July 11, 2011 issue of the *Archives of Internal Medicine* reported, "… no change in the adjusted rate of PCI for total occlusions identified at least 24 hours after MI following the publication of the OAT or the revision of the major guidelines."[1] They further commented: "The results of this study are a cause for concern on two levels. First, they imply that many stable patients with recent MI and persistent infarct artery occlusion continue to undergo a costly and ineffective procedure. Second, a large public, scientific, and human patient investment in the generation of robust clinical evidence has yet to broadly influence US practice."

Comment: Angioplasty (PCI) has been shown to be of benefit for patients who have suffered from a heart attack when the procedure is performed within the first few hours of the occlusion of the heart artery by a blood clot. However, within as little time as 90 minutes the damage that has been done to the heart muscle becomes permanent. Therefore, opening of the occluded artery with angioplasty (PCI) 24 hours or more after a heart attack has been proven beyond any doubt to provide no benefits and is actually harmful and costly**.**

The Occluded Artery Trial (OAT) was a large, randomized controlled trial funded by the National Heart, Lung, and Blood Institute that showed no reduction in death, reinfarction (repeat heart attacks), or class IV heart failure when angioplasty was performed more than 24 hours after a heart attack.[2] These findings are in addition to all other studies done, which consistently show that performing angioplasty, with or without stents, for people with chronic coronary artery disease does not save lives. Following this research, guidelines for cardiologists provided by the American College of Cardiology and the American Heart Association have called for a change in practice behaviors. This study by Deyell demonstrates that cardiologists are ignoring the research and recommendations to stop these harmful and costly procedures.

An accompanying editorial suggested the reasons for this malpractice are tied to money: "In addition, in a fee-for-service health system and in an environment in which more and more physicians are being compensated on the basis of relative value unit productivity, it remains to be determined whether personal financial gain might play a role in continuing old practices and in performing procedures shown to be of no benefit."[3] The editorial also commented that widespread publicity of physician misconduct, with the potential for lawsuits brought by patients and their families, could quickly change medical practice behaviors.

People who take your money and cause you harm are called criminals, and they should be punished. Somehow doctors have been relegated to a god-like position, higher than the Wall Street and sub-prime mortgage investors are, and as a result, their questioned professional behaviors have not attracted much public attention, so far. I have made efforts to require by law that people in the state of California be told that heart surgery for chronic coronary artery disease does not save lives, and is costly and harmful. This year I plan to further my efforts with the state legislators.

1) Deyell MW, Buller CE, Miller LH, Wang TY, Dai D, Lamas GA, Srinivas VS, Hochman JS. Impact of National Clinical Guideline Recommendations for Revascularization of Persistently Occluded Infarct-Related Arteries on Clinical Practice in the United States. *Arch Intern Med*. 2011 Jul 11.

2) Hochman JS, Lamas GA, Buller CE, Dzavik V, Reynolds HR, Abramsky SJ, Forman S, Ruzyllo W, Maggioni AP, White H, Sadowski Z, Carvalho AC, Rankin JM, Renkin JP, Steg PG, Mascette AM, Sopko G, Pfisterer ME, Leor J, Fridrich V, Mark DB, Knatterud GL; Occluded Artery Trial Investigators. Coronary intervention for persistent occlusion after myocardial infarction. *N Engl J Med*. 2006 Dec 7;355(23):2395-407.

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Diet Causes Diverticular Disease

Diet and risk of diverticular disease in Oxford cohort of European Prospective Investigation into Cancer and Nutrition (EPIC): prospective study of British vegetarians and non-vegetarians by Francesca L. Crowe, published in the July 19, 2011 issue of the *British Medical Journal,* found, "Consuming a vegetarian diet and a high intake of dietary fibre were both associated with a lower risk of admission to hospital or death from diverticular disease."[1] This study was of 47,033 men and women living in England or Scotland, of whom 15,459 (33%) reported consuming a vegetarian diet.

Comment: This recent study found that people with a higher fiber diet (greater than 25 grams daily, compared to an average of less than 10 grams consumed by most Westerners and close to 100 grams on the McDougall Diet) had a lower risk of being admitted to a hospital with or dying from diverticular disease. Vegans (who consume no animal foods) had an even lower risk. Dietary fiber is only present in plant foods. Refining, such as in making white flour, removes much of this beneficial fiber. Animal foods, including meat, poultry, fish, and dairy products contain no fiber.

Dr. Denis Burkitt, known as "The Fiber Man," served as a missionary doctor in Uganda and was appointed senior consultant surgeon to the Ugandan Ministry of Health in 1961. He observed that the diseases he had been trained to treat in Scotland were absent among rural Africans. He saw almost no cases of type-2 diabetes, obesity, appendicitis, diverticular disease, hemorrhoids, dental caries, varicose veins, pulmonary embolism, inflammatory bowel diseases (Crohn's disease and ulcerative colitis), or hiatus hernia, and only one case of gallstones, in 20 years of practice. The reason he saw no diverticular disease in the general population of rural Africans was because of their primarily vegetarian (actually vegan), high-fiber diet based on starches.[2]

A high-fiber diet results in large stools that are easy to pass. If there is minimal fiber in the diet then the stool is hard to pass and movement requires contractions of the bowel to occur at very high pressures. Years of elevated pressures produce ruptures in the walls of the intestine (balloon-like bulges) called diverticula. Half of the people who have followed the Western diet for more than 50 years have diverticular disease.

When the diverticula become irritated by the unhealthful remnants of digested food, the openings in them can close up, allowing the fluids to become stagnant and infected—a condition known as diverticulitis. Switching to a high-fiber diet will greatly reduce the risk of future bleeding and infection; however, the diverticula do not disappear with a change in diet. The commonly held notion that nuts and seeds get caught in diverticula and cause diverticulitis is unsupported by any scientific research and is untrue.

You can learn much more about the role of the Western diet in the cause of common intestinal diseases by reading my book, *Dr. McDougall's Digestive Tune-up*.

1) Crowe FL, Appleby PN, Allen NE, Key TJ. Diet and risk of diverticular disease in Oxford cohort of European Prospective Investigation into Cancer and Nutrition (EPIC): prospective study of British vegetarians and non-vegetarians. *BMJ.* 2011 Jul 19;343:d4131.

doi: 10.1136/bmj.d4131.

2) Trowell HC, Burkitt DP. Diverticular disease in urban Kenyans. *Br Med J.* 1979 Jun 30;1(6180):1795.

Niacin Combined with a Statin Is Dangerous

Trial of niacin alongside statin is stopped early by Jacqui Wise in the May 31, 2011 issue of the *British Medical Journa*l reported in a short letter that, "The US National Institutes of Health has stopped a clinical trial studying a combination of niacin (nicotinic acid) and a statin 18 months earlier than planned because of poor results."[1]

The AIM-HIGH* trial found that adding high dose, extended release niacin to a statin in patients with heart and vascular disease did not reduce the risk of cardiovascular disease and stroke. In fact there was a small and unexplained increase in ischaemic stroke rates among those receiving the combination treatment." There were 28 strokes (1.6%) in those taking the niacin compared with 12 strokes (0.7%) in the control group.

Comment: The AIM-HIGH trial, a multicenter clinical trial with about 90 sites in the United States and Canada, randomized 3,414 patients to either gradually increasing doses up to 2000 mg a day of niacin (Niaspan) or placebo. They were also prescribed simvistatin (Zocor) and, if needed, ezetimibe (Zetia) to lower the patients' blood cholesterol levels even further.

Cholesterol-lowering medications are not without hazards. A combination of these two medications (Zocor and Zetia), called Vytorin, was almost removed from the market as a result of research published on January 14, 2008 showing that Vytorin dramatically lowered cholesterol levels, without improvement in survival, compared to Zocor alone; and it doubled the thickness of the patients' arteries (intimal-media thickness).[2] This thickening is associated with an increased risk of stroke and heart attacks. On June 8, 2011, the Food and Drug Administration recommended that the use of drugs containing 80 mg of simvastatin be sharply curtailed because of the high risk of muscle injury (rhabdomyolysis). The addition of niacin adds another layer of harm to patients treated with commonly prescribed cholesterol-lowering medications.

I do prescribe statins to my patients with a high risk for artery disease. To be specific, if a patient has a history of a heart attack, heart surgery, a transient ischemic attack (TIA), or stroke then I will usually prescribe a statin medication with the goal of lowering their cholesterol level below 150 mg/dL. However, for people with a lower risk of an impending heart tragedy (the average American with simply an elevated blood cholesterol level), cholesterol-lowering medications are so ineffective that benefits cannot be detected. Plus any benefits present from prescribing these medications may be overshadowed by the harms caused by these drugs. Even though I do not prescribe cholesterol-lowering medications to otherwise healthy people, I also prescribe the McDougall Diet to everyone, which lowers cholesterol an average of 32 mg/dL within seven days.

When I must prescribe cholesterol-lowering medications then I usually choose the generic brand pravastatin (Pravachol). In one review this formula showed a greater reduction of cardiac events (heart attacks, angioplasty, bypass surgery, sudden death, and overall mortality) than did other kinds of statins.[3]

*AIM-HIGH stands for: Atherothrombosis Intervention in Metabolic Syndrome with Low HDL/High Triglyceride and Impact on Global Health Outcomes.

1) Wise J. *BMJ.* 2011 May 31;342:d3400. doi: 10.1136/bmj.d3400.

2) Lenzer J. Unreported cholesterol drug data released by company. *BMJ.* 2008 Jan 26;336(7637):180-1.

3) Ichihara K, Satoh K. Disparity between angiographic regression and clinical event rates with hydrophobic statins. *Lancet.* 2002 Jun 22;359(9324):2195-8.

Cow's Milk Damages Kidneys and Sickens Children

Early-Childhood Membranous Nephropathy Due to Cationic Bovine Serum Albumin by Hanna Debiec, published in the June 2, 2011 issue of the *New England Journal of Medicine* found, "Some patients with childhood membranous nephropathy have both

circulating cationic bovine serum albumin and anti–bovine serum albumin antibodies."[1]

These researchers found a distinct form of membranous nephropathy in children 5 months to 2.3 years of age. Human exposure to bovine serum albumin (beef and cow's milk protein) is common through the diet, and small amounts of dietary proteins may be absorbed in an undigested or partially digested form from the gastrointestinal tract in healthy persons. Antibodies to cow proteins are present in virtually all infants exposed to cow's milk. They further suggested that if cow protein is detected, eliminating it from the diet could be beneficial. Apparently all four children with circulating bovine serum albumin-related membranous nephropathy underwent a complete or partial remission when they stopped consuming the cow proteins. (Cow protein comes from all forms of dairy products: milk, skim milk, cheese, ice cream, yogurt, etc. and beef products.)

Comment: Membranous nephropathy is the most common cause of a form of kidney disease referred to as the *nephrotic syndrome* (a nonspecific condition in which the kidneys are damaged and they then lose large amounts of protein into the urine). The outcome can lead to complete kidney failure and death. In this condition antibody deposits can be found on the blood vessels of the kidneys and these antibodies damage the kidneys. This research identifies that the antibodies that attack the kidneys are formed in response to beef and cow's milk protein that enters the person's blood stream after consuming these animal foods. Other animal proteins, including those from pork, have been found to cause the same kidney problems.[2]

Thirty-five years ago (1977) it was reported in the medical journal, the *Lancet,* that cow's milk caused nephrotic syndrome in children.[3] Most importantly, when cow's milk was stopped, all six children who were studied were cured. As little as one ounce of cow's milk was enough to cause reactions in all children. Imagine the avoidable suffering that has happened over the past three and a half decades. As shocking is the almost certainty based on past performances that this report in the June 2011 *New England Journal of Medicin*e will change nothing in terms of medical advice, because of the widespread ignornace and inertia of physicians.

How many doctors give advice to their patients to stop consuming cow's milk and beef? Almost none. Eliminating animal foods and eating a healthy starch-based diet also benefits adults with nephrotic syndrome and is the fundamental treatment for all forms of chronic kidney disease.

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2) McCrory WW, Becker CG, Cunningham-Rundles C, Klein RF, Mouradian J, Reisman L. Immune complex glomerulopathy in a child with food hypersensitivity. *Kidney Int.* 1986 Oct;30(4):592-8.

3) Sandberg DH, Bernstein CW, McIntosh RM, Carr R, Strauss J. Severe steroid-responsive nephrosis associated with hypersensitivity. *Lancet.* 1977 Feb 19; 1(8008):388-91.

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