# Ten Herbs for Glycemic Control in Type 2 Diabetes

*Richard Karel*

Type 2 diabetes mellitus is a serious, noncongenital condition that is growing to epidemic proportions in the United States. Chronic diabetic hyperglycemia is associated with damage to the cardiovascular system eyes, kidneys, and nerves (Diagnosis and classification of diabetes mellitus, 1994). According to the Centers for Disease Control (CDC), as of 2007, nearly 24 million people in the United States (about 8 percent) were diagnosed with diabetes, an increase of more than 3 million over two years. Another 57 million are pre-diabetic, putting them at risk of developing diabetes (CDC, 2008). The vast majority of diabetics (90 to 95 percent) have Type 2 diabetes, or non-insulin dependent diabetes mellitus (National Diabetes Information Clearinghouse, 2007). The focus of this paper is the use of herbs in glycemic control.

## Goals for managing Type 2 diabetes

The primary goals for managing Type 2 diabetes across the age spectrum are: achieving and maintaining glycemic control, which may include improving insulin sensitivity and secretion, identifying and treating comorbidities, and preventing vascular complications (Bloomgarden 2007, Laffel & Svoren 2007).

While conventional medications, including insulin, may allow individuals with Type 2 diabetes to live relatively normal lives, it is frequently possible to reverse the condition entirely with appropriate dietary and lifestyle modifications and adjunctive use of medicinal herbs and supplements provided the condition has not progressed to permanent pancreatic beta-cell dysfunction (Anderson & Ward 1979, Barnard et al. 1994). As part of a comprehensive approach to managing glycemic issues, the herbalist should be familiar with the fundamental dietary and lifestyle variables that contribute to the development of Type 2 diabetes.

## Dietary and lifestyle modification

Modification of diet and lifestyle may be enough to move an individual with Type 2 diabetes into a metabolically normal state. Where complete reversal is not accomplished, it may still be possible to significantly enhance insulin sensitivity and glycemic control with concomitant reduction in the need for insulin or antiglycemic agents (Anderson et al. 1991, Barnard et al. 2006, Jenkins et al. 2003).

Much of what we do as clinical herbalists is based upon counseling and guiding the client toward a healthier dietary intake and generally more balanced lifestyle, so this approach is consistent with our scope of practice.

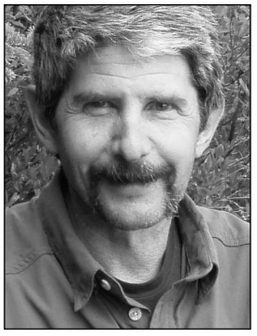
## Basic dietary and lifestyle approaches

Type 2 diabetes involves both genetic and lifestyle components, and some individuals are simply much more likely to develop the condition than others. It is important to bear this in mind when working with diabetic clients, and to avoid a moralistic or judgmental stance (Unger 2007).

While the approach must be tailored to the client, it is reasonable to aim for intake of high quality protein and high fiber carbohydrates, while decreasing the intake of refined carbohydrates and saturated fats. The challenge this poses should not be underestimated and the client should be praised for success, not criticized for nonadherence.

## Herbs for glycemic control

A successful approach to managing Type 2 diabetes requires an engaged and motivated client willing to



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consider dietary and lifestyle modifications. That said, there are many herbs (and non-herbal dietary supplements) that may be helpful in enhancing insulin sensitivity and glycemic control. In addition to herbs specifically focused on glycemic control, herbs including vascular tonics, antioxidants, adaptogens, and circulatory stimulants may play a major role in decreasing some of the complications.

In Western, Chinese, and Ayurvedic medicine, a large variety of herbs have been employed alone or in combination to help regulate blood sugar. It is beyond the scope of this paper to address this vast body of work (Bensky et al. 2004, Caldecott 2006, Hoffman 2003).

Although there are relatively few rigorous clinical studies on use of herbs for glycemic control, it is fair to say that they are generally safe when used as indicated (Yeh et al. 2003). According to a systematic review of herbs and dietary supplements for glycemic control published in 2003, acceptable clinical trials have been conducted for *Coccinia indica, Panax quinquefolius, Gymnema sylvestre,* and *Momordica charantia*. *Gymnema* and *Momordica* have been studied only in nonrandomized controlled trials (Yeh et al. 2003).

Since 2003 there has been promising research on *Panax ginseng* and *Cinnamomum cassia*, which requires further clarification (Babu et al. 2006, *Cassia cinnamon* n.d., *Cinnamon in diabetes mellitus* 2007, Harvard Heart Letter 2006, Khan et al. 2003, Kim et al. 2005, Mang et al. 2006, Pham et al. 2007, Shane-McWhorter 2005, Vanschoonbeek et al. 2006, Vuskan et al. 2008). Blond psyllium (*Plantago ovata*) and fenugreek (*Trigonella foenum-graecum*) have also undergone some clinical testing and have good safety profiles (Bradley et al. 2007, Dey et al. 2002, McWhorter 2005, 2001).

## Ten herbs for glycemic control

***Coccinia indica:*** This traditional Ayurvedic herb, also called ivy gourd, is widely and safely used in India as a traditional treatment for diabetes (Khan et al. 1980, Kuriyan et al. 2008). It was found effective in a two-armed trial assessing efficacy and safety versus both placebo and an oral antiglycemic. No adverse events were reported. Other clinical trials support efficacy, including a trial of 60 non-medication using individuals newly diagnosed with Type 2 diabetes published earlier this year (Kuriyan et al. 2008). *Coccinia's* therapeutic benefits are similar in magnitude to those of conventional oral antiglycemics (Kamble et al. 1998, Yeh et al. 2003). A literature search revealed no specific reports of drug-herb interactions.

In a recent clinical trial by Kuriyan et al. (2008), subjects were given a hydroethanolic extract equivalent to 15 grams per day of dried herb in the form of 1 mL/day of a 15:1 concentrate which may provide a guideline for clinical use. Further research surrounding both efficacy and optimal dosage is required.

Mechanisms of action are poorly understood but appear to involve insulin-mimetic properties. Kuriyan et al. (2008) note that *Coccinia* administered to diabetic rats decreased blood glucose by suppressing its synthesis via inhibition of key gluconeogenic enzymes and by enhancing glucose oxidation, both actions characteristic of insulin. They reference reports that a triterpene-containing toluene extract of *Coccinia* reduced alloxan-induced beta-cell damage in rats and thus enhanced insulin secretion. Whether or not an insulin-mimetic herb could lead to insulin resistance is unknown and speculative, and must be weighed against a long history of safe use in Indian traditional medicine.[1]

***Panax ginseng:*** Evidence suggests that *Panax ginseng* improves glucose transport, facilitating the movement of glucose across apical membranes into cells, and potentiates the effects of endogenous or administered insulin via insulin-sparing activity. Insulin-sparing results in a reduced need for exogenous insulin and may permit reduced dosing of conventional secretagogues. Ginsenosides may act as ligands for peroxisome proliferator-activated receptors (PPAR), which regulate the expression of genes involved in glucose and lipid metabolism. PPAR ligands are potent insulin sensitizers (Kyu et al. 2006).

In one recently published trial on safety and efficacy in a cohort of healthy individuals with controlled Type 2 diabetes (using oral medications and a diet designed to stabilize blood sugar), adjunctive ginseng improved plasma glucose and plasma insulin levels. There was no reduction, however, in hemoglobin A1c (hbA1c), a primary measure of average plasma glucose concentration over long periods of time. Safety was equivalent to placebo with no major adverse events. (Vuskan et al. 2008). Treatment dose was 6 grams per day. Other research backs the notion that *P. ginseng* is a

useful adjunctive antiglycemic which tends to normalize high blood sugar and contribute to overall metabolic health (Braun & Cohen, 2007).

Ginseng is contraindicated in hypertension and acute asthma. Higher doses may be overstimulating and cause headache and insomnia. It is considered safe in pregnancy and lactation. It is contraindicated for use with monoamine oxidase inhibitors (Bone 1996, Mills & Bone 2005).

***Panax quinquefolius:*** Also known as American ginseng, two clinical trials found decreases in fasting blood glucose and HbA1c, while three other trials similarly found postprandial glucose declines. In one study, a daily dose of 3 grams powdered herb was administered, which, according to the authors, is substantially higher (twice as much) as doses used in other human ginseng studies (Vuskan et al. 2000). The trials were small and of relatively short duration, and more extensive clinical testing would be helpful (Yeh et al. 2003).

*In vitro* research suggests *P. quinquefolius* acts to increase insulin production and decrease its breakdown via pancreatic beta cells by preventing beta cell apoptosis (Luo & Luo 2006, Wu et al. 2007). Despite differences in constituents, both *P. ginseng* and *P. quinquefolius* contain ginsenosides, which appear to increase insulin sensitivity by acting as ligands for PPAR receptors (Kyu et al. 2006). Preparations standardized for ginsenosides may be advisable for use in serum glucose modulation (*Possibly effective*, ginseng, n.d.). Safety profile is highly favorable and traditional use suggests that *P. quinquefolius* may provide a less stimulating alternative to *P. ginseng* for herbal therapy (Predy et al. 2005, Tierra 1988, Vuskan et al. 2001).

There is one documented report of interaction between *P. quinquefolius* and Warfarin (Yuan et al. 2004), and the herbalist should consider having clients monitor INR if starting or increasing use of *P. quinquefolius* concomitant with Warfarin.


*Panax ginseng*

***Cinnamomum cassia:*** Although cinnamon has received growing recognition for its antiglycemic properties, clinical trials in glycemic control are equivocal.

A number of clinical trials have found antiglycemic efficacy (Chase & McQueen 2007, Khan et al. 2003, Mang et al. 2006, Pham et al. 2007, Shane-McWhorter 2005). A study of cinnamon supplementation in 25 postmenopausal type 2 diabetics, however, found no efficacy for glycemic control (Vanschoonbeek et al. 2006). The basis of the discrepancy is unclear; it may be related to different baseline values and lack of nutritional standardization (Braun & Cohen 2007). Doses ranged from 1 gram to 6 grams per day.

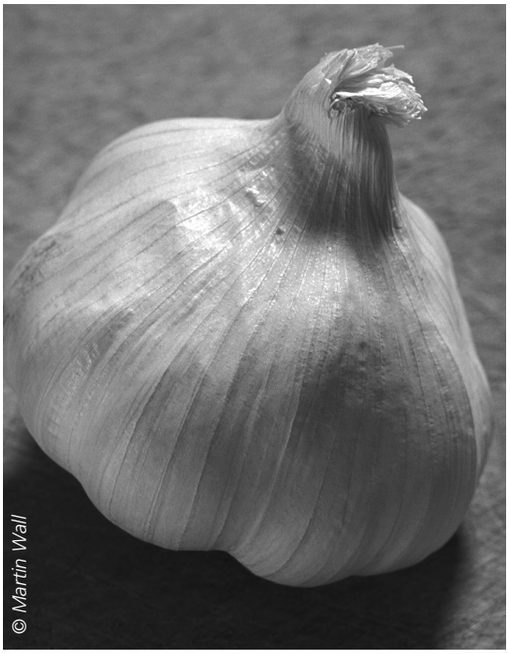
The precise mechanisms by which cinnamon may exert antiglycemic effects is imprecisely understood. Kim et al .(2006) speculate that cinnamon may exert antiglycemic activity either by potentiating insulin or by increasing either pancreatic secretion of insulin from beta cells or triggering conversion from bound to free form. Khan et al. (2003) note that cinnamon extracts have been shown to activate glycogen synthase, increase glucose uptake, activate insulin receptor kinase, and inhibit dephosphorylation of the insulin receptor, all of which enhance insulin sensitivity.

Cinnamon is well tolerated and extremely safe; there are occasional allergic reactions to cinnamic aldehyde and those allergic to Peruvian balsam (*Myroxylon pereirae*) may show cross reactivity to cinnamon (Bone 2003). It is

considered safe in pregnancy and lactation although German Commission E cites lack of clinical safety data as cause to avoid it in pregnancy and lactation (Blumenthal et al 2000, Bone 2003).

***Allium sativum:*** Garlic has a long history of use for a variety of conditions. There is some evidence that efficacy is influenced by whether garlic is used fresh or in other forms. A key practical concern is tolerability, and many individuals are simply unable to tolerate high oral intake of fresh, raw garlic (ESCOP 2000). There have been concerns surrounding constituent content of garlic extracts, which should contain allinase in order to release therapeutic levels of allicin, according to Braun & Cohen (2007). The clinical herbalist needs to do his or her own product research if choosing to recommend a garlic extract. Garlic may be dosed at 2 to 5 grams per day of fresh bulb or the equivalent (Braun & Cohen 2007).

A number of animal trials found that garlic administration resulted in dramatic decreases in blood glucose levels while other trials indicated no antiglycemic activity (Eidi et al. 2006, Swanston-Flatt et al. 1990). Similarly conflicting results have occurred in human clinical trials (see discussion by Liu et al. 2007, Yeh et al 2003). Conflicting results may involve differences in dosage, type of garlic preparation, and experimental design. As Liu et al. (2007) comment, chemical composition of garlic depends not only on species but also on harvesting season and processing conditions, and although there is general agreement that active agents are sulfur-containing compounds, there is no consensus regarding precisely which constituents may be responsible for hypoglycemic effects.




*Allium sativum*

A recently published human clinical trial in 60 type 2 diabetic patients over four weeks resulted in significant reductions in serum glucose and triglycerides (Sobenin et al. 2008). The diabetic cohort received 300 mg/day of a time-release garlic powder.[2]

If, indeed, garlic is an effective clinical antiglycemic, it may exert its effects in part by stimulating beta-cell insulin secretion, thereby resulting in peripheral insulin-like activity (Liu et al. 2007). Sobenin et al. (2008), referencing animal trials, state that garlic's sulfur-containing amino acids exert direct hypoglycemic effects, potentiate serum insulin, and increase hepatic glycogen synthesis. They note that S-allyl cysteine sulfoxide, a garlic compound, has been shown to exert insulin-stimulating effects on healthy rat beta cells *in vitro*.

Dyspepsia may occur with higher doses, and garlic breath hardly needs mention (Shane-McWhorter 2001). According to Mills & Bone (2005), consumption of doses equivalent to more than 5 grams per day of fresh garlic are contraindicated with use of Warfarin due to possible potentiation of anticoagulant action. For the same reason they advise discontinuing use prior to and immediately after surgery.

***Plantago ovata:*** Psyllium seed, which is a common ingredient in over-the-counter bulk-forming laxatives, has been shown in both animal models and human trials to significantly lower postprandial blood glucose and insulin levels in type 2 diabetes (Hannan et al. 2006, Sierra et al. 2002). Optimal efficacy is dose-dependent and requires administering psyllium prior to meals (Rodriguez-Moran et al. 1998). Efficacy as an antiglycemic agent has been proven at doses of 10 to 15 grams daily taken either three times per day before meals or twice per day before breakfast and dinner. (Rodriguez-Moran el. 1998, Ziai et al. 2005). Ziai et al. (2005)

found significant reductions in both serum glucose and hemoglobin A1c, a key measure of antiglycemic efficacy.

Psyllium, unlike the other antiglycemic herbs discussed here, exerts it effects by increasing viscosity of intestinal contents, which inhibits intestinal absorption, and by increasing gastric motility. The antiglycemic effects are a result of both mechanisms' decreased absorption and increased gastric emptying attendant upon greater bowel motility (Brennan 2005, Hannan et al. 2006, Rodriguez-Moran et al. 1998, Ziai et al. 2005).

There is conflicting evidence about whether psyllium can reduce absorption of iron and other minerals, so it is best for clients to take other supplements at least a half hour before or one hour after psyllium ingestion (Blumenthal et al. 2000, ESCOP 2003, Fernandez 1982a, 1982b, Sierra et al. 2002). Psyllium must be consumed with adequate water in order to prevent choking or intestinal obstruction. In some people it may cause flatulence. There are rare allergies to psyllium. It is considered safe in pregnancy and lactation (ESCOP 2003).

***Trigonella foenum graecum:*** Fenugreek is another kitchen cabinet herb with utility in type 2 diabetes (Duke 1997). References for use an antiglycemic agent date back to early Greek pharmacopoeias (Yeh et al. 2003).

Fenugreek has a relatively high antiglycemic effect when compared to some other traditional Indian medicinal plants in clinical trials and has proven an effective antiglycemic agent alone and in combination with other Indian medicinal plants (Kar et al. 2003, Kochar & Nagi 2005). Fenugreek is generally well tolerated, although some individuals may experience flatulence and diarrhea. It is contraindicated for people allergic to chickpeas (*Cicer arietinum*) as there are case reports of cross reactivity (Basch et al. 2003).

Clinical trials indicate fenugreek is able to significantly improve glycemic control and insulin sensitivity (Bradley et al. 2007, Dey et al. 2002, Gupta et al. 2001). Other trials have reinforced these findings (Madar & Stark 2002). Mechanisms include possible upregulation of GLUT4 and other glucose transporting membrane proteins and improvement in insulin signaling pathways (Bradley et al. 2007).[3] Safety data is encouraging, with no major adverse effects reported in clinical trials (Yeh et al. 2003).

Therapeutic doses for antiglycemic effect have ranged from 5 grams to 25 grams of seed and 25 grams to 100 grams of defatted seed, according to Bone (2003).

A toxicologic evaluation of 60 diabetic patients who consumed high doses (25 grams daily) of fenugreek for six months found no hepatic or renal toxicity and no hematologic abnormalities (Basch et al. 2003). It is theoretically possible that this herb's high mucilaginous fiber content may impair the absorption of drugs or iron taken concomitantly, so clinicians are advised to have clients take prescription drugs separately and supplemental iron an hour before or after meals (Braun and Cohen 2007, ESCOP 2003).

Fenugreek has traditionally been used as a galactagogue, and is safe in lactation (Mills & Bone 2005). Mills & Bone suggest that it may be contraindicated in people with celiac disease and upper digestive tract irritation, and, due to high content of mucilaginous fiber, for people suffering from fat malabsorption or deficiencies of fat-soluble vitamins.

***Gymnema sylvestre:*** This Ayurvedic herb has only recently gained a place in Western herbalism based on its traditional use in India to reduce sugar cravings (Yeh et al. 2003). The herb works both systemically and by altering taste perception, the latter to neutralize the sweet taste (Shane-McWhorter 2005). Clinical trials, old and new, are extremely encouraging, with robust improvements in glycemic control following *Gymnema* administration (Baskaran et al. 1990, Shanmugasundaram et al. 1990a). Research continues (Bradley et al. 2007, Dey et al. 2002, Yeh et al. 2003).

*Gymnema* appears to increase the production of endogenous insulin by pancreatic beta cells and may stimulate regeneration of remaining beta cells in individuals who have suffered beta cell loss (Bradley et al. 2007, Shanmugasundaram et al. 1990b). Extract has been successfully employed in modulating blood sugar in insulin-dependent diabetes as well as in type 2 diabetes, and clinical trials with Type 1 diabetes patients found significant declines in both fasting blood glucose and hemoglobin A1c (Shanmugasundaram et al. 1990a). *In vivo* research on streptozotocin-induced diabetic rats found a doubling in the number of both islets of Langerhans and pancreatic beta cells

(Shanmugasundaram et al. 1990b).

As Bradley et al. (2007) note, although the data on *Gymnema* are intriguing, a few scientists have conducted most of the research, so that it is too early to state conclusively that the herb is capable of regenerating pancreatic tissue in a clinically relevant manner.

The primary safety issue with *Gymnema* is possible induction of hypoglycemia when combined with insulin or oral diabetic agents (McWhorter 2005, Mills & Bone 2005). This is another way of stating that it is, in fact, an effective and useful antiglycemic. A literature search conducted in early 2008 revealed no evidence of reported side effects or interactions. Mills & Bone (2005) advise that any saponin-rich herb, including *Gymnema*, may potentially irritate gastric mucosa or be problematic in celiac disease. The herbal clinician must weigh the proven record of safety and efficacy against theoretical, speculative, and poorly defined risks.

***Momordica charantia:*** This herb, also called bitter melon, has a wide geographic distribution and has gained credence as a folk remedy for diabetes. Bitter melon in various forms has been used in Chinese, Ayurvedic, and Western herbal medicine (Dey et al. 2002). Antiglycemic activity of bitter melon has been found in animal and clinical trials; further randomized controlled trials are desirable (Dey et al. 2002, Yeh et al. 2003). A variety of compounds appear to be responsible for this herb's antiglycemic activity, including the steroidal glycosides momordin and charantin. Encouraging results have been reported with both oral and subcutaneously injected extracts (McWhorter 2005). Animal models suggest an insulin-mimetic mechanism, and there is evidence from animal models that *Momordica* may stimulate regeneration of pancreatic beta cells in diabetics (Singh & Gupta 2007).

An experimental evaluation published in 2007 (Fernandes et al.) concluded that *Momordica* appears to have several mechanisms accounting for antiglycemic and antilipidemic activity, including enhancing insulin secretion in islets of Langerhans, reducing glycogenesis in the liver, enhancing peripheral glucose utilization and increasing serum protein levels. A study that employed both cultured cells and mice found that compounds from *Momordica* stimulated GLUT4 translocation to the cell membrane in both fat and muscle cells, and enhanced fatty acid oxidation and glucose disposal in both insulin-sensitive and insulin-resistant mice (Tan et al. 2008). This action was associated with upregulation of AMPK (adenosine monophosphate-activated protein kinase), a key mediator of glucose uptake and fatty acid oxidation. The finding of AMPK activation by *Momordica* constituents was reinforced in a paper published in July 2008 in the *Journal of Agricultural and Food Chemistry* (Cheng et al.).

The primary safety concern is that the antiglycemic activity of bitter melon may be additive with conventional antiglycemic medications and precipitate hypoglycemia (McWhorter 2005). *Momordica* is contraindicated in pregnancy due to possible abortifacient effects (Chan et al. 1984). The most frequent adverse event in one recent clinical trial involved gastric discomfort; there was no evidence of any increase in potassium depletion, which has occasionally been voiced as a safety issue when *Momordica* is used with potassium-depleting agents (Dans et al. 2007, McWhorter 2001). In animal models, *Momordica* extracts show no evidence of hepatotoxicity or nephrotoxicity and there is some evidence of hepatoprotective activity (Abd El Sattar et al. 2006, Semiz & Sen 2007, Virdi et al. 2003).

***Ocimum sanctum,*** also called Tulsi or Holy Basil, is an important herb in Indian medicine and is increasingly employed by Western herbalists (Prakash & Gupta 2005). There has been only one published controlled clinical trial, which evaluated the impact of using a local preparation of fresh leaf powder for four weeks in 40 type 2 diabetics. There were significant declines in both fasting and postprandial glucose and no adverse events (Agrawal et al. 1996).

There are, however, significant data from both animal and *in vitro* research pointing to a therapeutic role for Tulsi in diabetes generally and for glycemic control in particular (Grover et al. 2002, Kar et al. 2003, Modak et al. 2007). Vats et al. (2003) found a 26 percent reduction in plasma glucose after 30 days of administering 200 mg/kg of ethanolic extract to streptozotocin-induced diabetic rats. Rai et al. (1997) performed a two-armed animal trial in which ground Tulsi leaves were fed to both normal and diabetic rats at a level comprising 1 percent of total dietary intake. The

diabetic rats exhibited a significant reduction in fasting serum glucose in both serum lipids and tissue lipids. Serum glucose remained stable in normal rats. Two trials by Chattopadhyay (1993, 1999) found that ethanolic Tulsi extract resulted in significant reductions in serum glucose in normal glucose-fed hyperglycemic rats, and streptozotocin-induced diabetic rats in a dose-dependent manner. Activity compared favorably to tolbutamide, a conventional antiglycemic.

A variety of mechanisms may be responsible for Tulsi's pharmacological activity. In a biochemical evaluation of plant activity, Narendhirakannan et al. (2006) enumerated possible mechanisms of Tulsi (and other plants) used in Indian medicine for diabetes treatment. Based on their own and prior research they cited increased glucose utilization by upregulated glycogen synthesis in the liver, decreased conversion of glycogen to glucose and decreased gluconeogenesis. Specifically, ethanolic *Ocimum* extract was shown to increase liver glycogen levels by decreasing glycogen phophorylase activity and increasing glycogen synthase activity. Another mechanism for Tulsi's antiglycemic action may be insulin potentiation via increased secretion from beta cells in Islets of Langerhans, release of insulin from bound to free form, or increased insulin sensitivity. Narendhirakann et al. (2006) note that Tulsi-extract treated diabetic rats exhibited an insulin-binding pattern very similar to that of non-diabetic controls.

In the one controlled human trial 2.5 grams of powdered leaf daily was employed (Agarwal et al. 1996). Given the dearth of human clinical data, this may be the best guideline for the practicing herbalist. Tulsi has a long history of safe use. As with other potentially antiglycemic herbs, there is a theoretical possibility of interaction with conventional antiglycemic medication.

## Conclusion

Management of type 2 diabetes in the herbal clinic is a collaborative process that optimally involves a client willing to move toward enhancement of diet and lifestyle. Even with a less willing client, however, there is a role for focused use of phytotherapy.

As is the case with many medicinal herbs, much of the evidence has not been formally evaluated in double-blind placebo trials and relies instead on traditional use and clinical experience. All of the ten herbs described have a long history of use and an exemplary safety record. The clinical herbalist can, with the few caveats mentioned, feel confident when employing these medicines as part of a program of glycemic control.

In terms of safety, a few areas require close attention. In general, any herbal medicine that is an effective antiglycemic agent is theoretically capable of inducing hypoglycemia, and this is particularly relevant when the client is concomitantly using prescription antiglycemic medications.

In terms of specific herbs, there is generally little published evidence of adverse or additive interactions. Even where there are more extensive published references surrounding the safety of a specific herbal agent, the overall picture is far from definitive.

**Footnotes**

1 James Snow, personal communication, July 2008.

2 There was no reference to standardization of marker compounds in methodology.

3 GLUT4 is a key member of a family of proteins responsible for distribution and utilization of glucose and is found in both fat cells and striated muscle. In the presence of insulin it is redistributed from intracellular storage to the plasma membrane (Watson et al. 2004).

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