Aloe Vera, in various forms, has been applied to the treatment of diabetes of animals and humans in a few small scale trials and one larger trial. The results indicate that Aloe has a hypoglycaemic effect (i.e. a blood sugar lowering effect), and other effects, sufficient to make it extremely interesting for possible wide-scale use in the treatment of diabetic conditions. The nature and implications of these findings are discussed in this newsletter.
**ALOE VERA AND DIABETES**

The diabetes being referred to in this Newsletter is Diabetes Mellitus, otherwise known as “sugar diabetes”, because an inevitable aspect of it is a blood sugar level far higher than normal, with glucose appearing in the urine. The name “Mellitus” refers to the sweet taste of the patient’s urine on account of its glucose content. It has nothing to do with Diabetes Insipidus, which is a disorder of the Posterior Pituitary Gland. Diabetes Mellitus is due either to a failure of insulin production in the Islets of Langerhans in the pancreas or to a condition of the tissue cells known as “insulin resistance”. In some cases there is a combination of these two conditions.

There are two rather distinct versions of Diabetes Mellitus, differing in respect of their patterns of inheritance, insulin responses and origins. These distinctions were drawn up by the National Diabetes Data Group of the National Institutes of Health in the USA and are called “Insulin Dependent Diabetes Mellitus”, which used to be called “Juvenile Onset Diabetes Mellitus” and “Noninsulin Dependent Diabetes Mellitus”, which used to be referred to as “Adult Onset Diabetes Mellitus. The nature of these two conditions and the distinctions between them are discussed below.

**The Normal Action of Insulin**

Insulin is a hormone produced from the “Endocrine Pancreas”, i.e. that part of the pancreas which is concerned with hormone production (the Islets of Langerhans, Figure 1 & 2) as opposed to the part of the pancreas which produces digestive juices. Its chemical nature is a polypeptide / small protein molecule containing 51 amino acid residues. Its formula is given in Figure 3. To understand this structure it is necessary to know that each of the amino acid residues is referred to in an abbreviated form consisting of the first three letters of the name of the corresponding amino acid.

It is most important to appreciate the actions exerted by insulin in the normal human body. These are to stimulate:

- Transmembrane transport of glucose and amino acids (transport into cells - increased rates of glucose oxidation usually result)
- Glycogen formation in liver and skeletal muscles
- Glucose conversion to triglycerides
- Nucleic acid synthesis, promoting growth and differentiation
- Protein synthesis

These actions make insulin “a major anabolic hormone”. The term “anabolic” means favouring the building up of cellular constituents from simpler substances. Any substance which encourages the reverse process is referred to as being “catabolic”.

The combined effects upon blood glucose level of insulin secretion is to reduce it. Injection of excess insulin is therefore capable of producing dangerously low blood sugar levels. The normal human body depends upon a “trickle” of insulin coming from the pancreas to control the level of glucose in the blood, but larger quantities are produced in response to eating sugar or sugary foods. The effect upon body glucose of a pulse of insulin released from the pancreas is to strongly encourage the clearing of glucose out of the blood by tissue oxidation, by conversion to stored

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Figure 1  A microscopic view of a section of pancreas showing the islets of Langerhans clearly demarcated. These contain the beta cells which produce insulin.
glycogen and by conversion to fat. At the same time the formation of “new glucose” from protein (a process referred to as “gluconeogenesis”) is inhibited, an action which also tends to reduce the blood sugar level. In the normal body insulin in the blood is balanced by another hormone from the pancreas, called “glucagon”, which exerts a largely opposite action. The normal balance of body glucose is therefore achieved by the appropriate regulation of these two hormones.

The actions of insulin (and related substances called insulin-like growth factors) are exerted most particularly upon muscle cells, including heart muscle cells, fibroblasts and fat cells, which represent collectively about two thirds of the entire body weight. The effects of a lack of insulin are best seen in the symptoms of Insulin Dependent Diabetes Mellitus, described below.

The Deep Philosophical Divide over the Treatment Approach to Diabetes

Ever since the discovery of the hormone Insulin by Banting and Best in 1922 there has been a polarization of views about the best ways to treat Diabetes Mellitus between the orthodoxy and Alternative Practitioners. The immediacy of the need for treatment of diabetic patients at risk of their lives, as well as the power of orthodoxy, ensured that the orthodox view, which has always consisted in the administration of insulin, held sway. Naturally once the hormone was known and means were available for its production, it was an easy and obvious way to overcome the immediate symptoms of diabetes to give the patient regular doses of the hormone they were known to be lacking.
Yet it is well known that if one administers a hormone to the body the organ whose normal role it is to produce that hormone will atrophy - in other words become dysfunctional and even shrivel. Hence the administration of insulin to diabetics, whilst solving an immediate problem - turns them into hormonal cripples for the rest of their lives. By contrast, the Alternative Medicine approach is to seek to encourage the production of insulin once again in the patients' own pancreas. Only in this way can true normality ever be regained. This gives rise to a deep philosophical divide which does not just concern diabetes, but which runs throughout the entire field of medicine. It is drug medicine as opposed to a natural nurturing of a sick body back to health. Maximum interference as opposed to gentle persuasion. It is taking over the situation as opposed to leaving the patient in control of themselves. These orthodox views have often been imposed on the public, using threats of dire things that would happen and even invoking the Law to ensure that diabetic patients would certainly have insulin administered to them.

There is little room for compatibility between these two differing views of Medicine - though it is easy to acknowledge that a diabetic who has got into emergency difficulties, through failing to take steps soon enough, needs insulin. But in the alternative view he or she certainly should be spared from life-long intervention of this kind. Wherever possible - and it is more often possible than not - the patient should be carefully nurtured back towards independence from insulin. The reader will find (below) that the Alternative view is the one that must be taken to appreciate the case for the use of Aloe vera in diabetes - and that view is also the one which brings Nutritional Medicine Practice into its own in diabetic treatment.

Another important aspect of orthodox treatment of Diabetes Mellitus is the use of hypoglycaemic drugs. These are usually drugs of the sulphonylurea type, specifically carbutamide, tolbutamide, acetohexamide, tolazamide, chlorpropamide, glipizide and glyburide. It has been reported that these drugs exert their hypoglycaemic effect by both stimulating insulin release from the beta cells and by increasing insulin sensitivity at the level of the target cells. They are effective only in some cases of non-insulin dependent Diabetes Mellitus. Not enough is known in detail about the mechanism of action of Aloe in diabetes to say whether this action is, in itself, more "natural" or more desirable than that of the sulphonylurea drugs. More research is needed to truly define just how Aloe exerts the anti-diabetic effects which are to be described in detail below. However, the main known difference is that Aloe, particularly Aloe vera Gel, or the de-aloinized Whole Leaf Extract, is much more likely to exert its action through active principles which leave no residue afterwards. For example, this would be completely true of Aloe glucomannan, which is degradable to simple sugars which then enter the normal carbohydrate pathways of the cell. Sulphonylureas, on the other hand, are non-biological molecules foreign to the human intracellular environment. Whatever way one looks at these drugs, they have to be regarded as chemical toxins. There is really all the difference in the world between these two approaches. One cannot help being reminded of a now famous quote from the Nei Ching of ancient China.

"There are three categories of drugs; the lowest one of which is poisonous, the second one is a little poisonous and the highest one is no poison."

The anti-diabetic effect of sulphonylureas is available to be used. But who would choose to use potentially toxic nonbiological drugs compared with a natural substance that can afterwards be metabolized away? In particular, if one's orthodox prognosis is to use sulphonylureas for the rest of one's life, is there not an alternative option? Most Alternative Practitioners think there most certainly is. Moreover, a combination of Aloe with the foods and nutrients most needed to support the diabetic subject, has the potential to enable him or her in time to regain a normality which is free from the need for continuous treatment of any kind. Suddenly, the gulf between the potential for drug treatment compared to herbal / nutritional treatment appears extremely wide.

Insulin Dependent Diabetes Mellitus (IDDM).

This is also now known as "Type I Diabetes" and accounts for some 10-20% of all cases of Diabetes Mellitus. This results from a severe, absolute lack of insulin caused by a reduction in the numbers and activity of the beta-cells in the Islets of Langerhans in the pancreas. It usually develops during childhood, becoming manifest and severe at puberty. The classic symptom picture of diabetes tends to appear, with glycosuria (glucose in the urine), polyuria (high urine output with a profound loss of water and electrolytes with depletion of intracellular water), polydipsia (intense thirst), polyphagia (increased appetite). Without insulin administration they develop acute metabolic complications, such as acidosis (high acid levels in the blood, tissues and urine) and ketosis (high levels of the "ketone bodies" acetone and acetoacetic acid in the blood, tissues and urine, which may result in coma. Patients depend upon extraneous insulin for survival.
Much has now been discovered about the causes of Type I Diabetes. Three interlocking mechanisms are known to be responsible for the Islet cell destruction. These are genetic susceptibility, autoimmunity and environmental insult. It is thought that there is genetic susceptibility to the development of autoimmunity to the Islet beta cells. The genes responsible for this genetic susceptibility have been identified and their position in the human genome has been found, associated with a gene group known as the “Class II Major Histocompatibility Complex”. The autoimmunity can either develop spontaneously or, more likely, it can be triggered by an environmental agent such as a virus, a chemical or an unknown toxin. The evidence for the inheritance of genetic susceptibility is overwhelming. Up to 90% of patients with Type I diabetes have antibodies against Islet cells in the blood. Subjects with no diabetic symptoms who have a higher than normal risk of developing Type I diabetes develop Islet cell antibodies months to years before the clinical onset of diabetes.

The autoimmune processes result in “Insulitis”, or an inflammation of the Islets of Langerhans and, consequently, damage to beta cells. As a result of the damage to beta cells, some of the proteins from these cells are released into the blood, starting an autoimmune reaction directly against the beta cells. This causes further and extensive beta cell injury. Eventually most of the beta cells are destroyed, and overt diabetes appears.

Among the viruses, mumps, measles, Coxsackie B and cytomegalovirus have all been implicated as triggering factors for Type I diabetes in susceptible individuals. The triggering effect of chemicals and environmental toxins is obviously of particular interest to naturopathically minded Practitioners. This finding of orthodox medical science really is entirely confirmatory of what naturopaths have always maintained about the role of toxicity in the causation of diabetes. Specific chemicals implicated include alloxan, pentamidine, and streptozotocin. However, the most viable thesis is that a much wider range of drugs and more common environmental substances will be found to have this triggering action in various degrees, contributing to a multi-pronged environmental attack upon all susceptible individuals. Incidentally, the scientific findings now available make a mockery of past (and even present) orthodox attempts to belittle the naturopathic approach to Type I diabetes on the over-simplistic grounds that the condition is “hormonal” or “genetic”. The orthodox implication that conditions which involve either hormones or genes cannot be touched therapeutically by the hand of the naturopath has been made to look, itself, ridiculous by the discovery that the pathogenesis (fundamental causes of) Type I diabetes is multi-factorial and includes a strong element of environmental toxicity. The fact that this is so makes detoxification an essential component of any logical approach to the treatment of Type I diabetes, especially during its developmental stages.

Developmental stages of Type I Diabetes are summarized in Figure 4.

Figure 4 A diagrammatical illustration of the developmental stages of Type I Diabetes.

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Noninsulin Dependent Diabetes Mellitus (NIDDM)

This has not always been referred to as being “Noninsulin Dependent” and in the past a great deal of insulin has been administered unnecessarily to such patients. This form is also known as Type II Diabetes. Much less is known within the orthodox medical sciences about the fundamental causes of this form of diabetes, which, nonetheless, is by far the most common type. By contrast, the Alternative field of Medicine, particularly that of Wholistic Nutritional Medicine, has developed a far better understanding of it and can offer programmes of treatment which are mostly dependable and effective.

Here, we look first at what the medical sciences recognise about the causes of Type II diabetes as of 1996.

Genetic factors are of even greater importance than in Type I diabetes and, among identical twins, the concordance rate is over 90%. However, the mechanism of these genetic factors is not understood and the “Class II Major Histocompatibility Complex” is not involved. Nor is there any evidence that autoimmunity is involved (except in specific rare cases where there is autoimmunity to the insulin receptors at the cell surface - see below). Obesity and overeating play important roles in Type II diabetes. There is also a correlation between incidence of the disease and increasing age, as is the case with many chronic diseases in which increasing nutritional imbalance, accumulating toxicity and the accrual of the resulting cellular damage play major roles.

Two major metabolic defects characterize Type II diabetes. The first is a derangement of insulin secretion that is delayed or that is insufficient relative to the glucose load. The second is an inability of the peripheral tissues to respond to insulin - this is the condition known as insulin resistance. There has often been disagreement among orthodox researchers as to which of these two aspects of Type II diabetes is the more important.

The frequency with which Type II diabetes occurs in company with obesity is also a complicating factor, particularly so since insulin resistance, accompanied by elevated levels of insulin in the blood, often also occurs in subjects who are obese without suffering from diabetes.

To appreciate the position one has to compare obese Type II diabetics with weight matched nondiabetics. One then sees that although the obese diabetics have elevated blood insulin levels, it is rather less elevated than it is in the nondiabetic controls. Hence the Type II diabetics have a degree of insulin deficiency compared to nondiabetics of the same weight. The insulin deficiency of Type II diabetics becomes a marked and absolute one when one looks at patients who have the disease to a fairly severe degree, having a fasting plasma glucose level of 200 to 300 mg/100ml. It is therefore possible to conclude that most patients with Type II diabetes have a relative or absolute deficiency of insulin. However, this deficiency is much milder than in Type I diabetes.

One orthodox medical view is that Type II diabetes represents a progressive, age-related loss of the function of the beta cells of the Islets of Langerhans of the pancreas, which is accelerated in subjects with a genetic susceptibility. This view is entirely compatible with the naturopathic view already expressed above, that Type II diabetes is principally triggered by increasing nutritional imbalance, accumulating toxicity and the accrual of the resulting cellular damage. Naturally, increasing age allows more time for the nutritional imbalances to worsen, for the chronic toxic load to reach greater heights, and for the extent of resulting cellular damage to accrue to serious proportions. If it happens to be the Islets of Langerhans that are affected, then it is entirely understandable that diabetes will result. It will also be understandable to the naturopathic practitioner, and most especially to the Iridologist, that wherever the inherited constitution compromises the pancreas specifically, then, in these subjects, diabetes will result from the generalized cellular damage more often than other possible chronic conditions. That is to say, that when generalized body chronicity occurs, it is the genetically compromised organs which tend to give way first. Once again, medical orthodoxy and the alternatives need not see themselves as taking up opposing positions at all here (at least about the causes of the disease - though they may differ fundamentally over treatment) so long as each of them examines their own position carefully and fully.

However, there is also strong evidence that the phenomenon of insulin resistance, mentioned above, is also important in the causation of Type II diabetes. This can be substantiated because even nonobese patients with Type II diabetes show insulin resistance. The nature of the phenomenon of insulin resistance is important - and it is multifactorial. The different factors involved are as follows.

1. The normal way in which insulin interacts with its target cells around the body takes place through the attachment of the insulin molecule to a specific “receptor” substance attached to the cell surface, known simply as the “insulin receptor”. The concept of cell surface receptors to which biologically active molecules (like Aloe glucomannan) become attached, was presented in Newsletter No4 on “The Healing Properties of Aloe vera”. When
the biologically active molecule (in this case insulin), attaches to the cell surface receptor, this brings about consequences within the cell by way of a response. For example, one of the main consequences of insulin molecules combining with insulin receptors at the cell surface is to bring about the transportation of glucose into the cell. In Type II diabetes the numbers of insulin receptors at the cell surface is reduced, and therefore, the transportation of glucose into the cell is reduced irrespective of elevated insulin concentrations on the outside. Clearly the number of insulin receptors at the cell surface is controlled from within the cell. A cell may reduce the insulin receptors at its surface by drawing them inside the cell, where they may be either stored for future use or destroyed.

2 Alternatively, the insulin receptors may remain at the cell surface in usual numbers but may undergo alteration so that they no longer bind with insulin in accord with their normal function. This is just as effective as (1) above in reducing the cell's responsiveness to insulin.

3 Alternatively again, the cells may develop a different kind of defect in which the insulin receptors are normal, and insulin binds to the receptors normally, but other changes within the cell block the usual post-receptor response to insulin binding. The cells contain specialized molecules known as "glucose transport units" which normally carry glucose molecules to appropriate sites within the cell. If these are reduced then the cell's ability to respond to insulin is again partially blocked because insulin then binds normally to its receptors but the normal transportation of glucose cannot occur. Processes contributing to Type II Diabetes are set out in Figure 5.

A Naturopathic View of Type II Diabetes
A naturopathic view has already been expressed above, relating Type II diabetes to nutritional imbalance, toxicity and cellular damage. However, another aspect also presents itself in relation to the findings on insulin receptors. It appears, from all that has been explained above, as if the cell is "putting up its shutters" so far as glucose is concerned, i.e. taking steps to halt the inrush of glucose into the cell. One may well theorize that this could be a defence mechanism. When cells go into a partially chronic condition through toxicity their ability to metabolize bulk nutrients through the citric acid cycle and the respiratory chain pathways inside the cell is compromised. Hence, if the cell is

Figure 5  The physiological processes known to contribute towards the development of Type II Diabetes.
unable to oxidize glucose at an adequate rate, it may well be under pressure to limit the ingress of glucose to avoid becoming flooded with high glucose concentrations which it would be unable to handle. This would also make sense in connection with the common occurrence of obesity and overeating along with Type II diabetes. Tissue cells which are compromised in their oxidation capacity and yet are at the same time being faced with a surfeit of incoming nutrients through over consumption of food may well be under double pressure to "put up the shutters" to avoid being flooded with bulk nutrients they cannot use - a situation which may well threaten their control systems and their survival. In this way the excessive glucose is kept in the blood, where at least it is rather more safe.

If this naturopathic view of Type II diabetes is correct, then the answer to this disease should be to provide conditions in which the tissue cells can better handle the glucose that is coming into the body. This will involve providing the cells with catalysts and enzyme co-factors - vitamins and minerals - to re activate their respiratory pathways, encourage the elimination of toxicity and the repair of cell damage that is likely to have occurred to the DNA and membranes. The provision of nutrients and the active encouragement of cleansing are, of course, the main routes by which naturopathic treatments proceed. This is the core of the approach which has now long proved so markedly successful in the hands of Practitioners of Nutritional Medicine and exemplified by the book by W. K. Philpott & D.K. Kalita, "Victory over Diabetes - A BioEcological Triumph" (1983) Published by Keats Publishing Inc., Connecticut, USA.

How the Action of Aloe impinges upon Hyperglycaemia in Type II Diabetes

All the reported studies on the use of Aloe in diabetes relate to Type II Diabetes.

A paper from Ghannam et al (1986) in Riyadh, Saudi Arabia reports the effects of Aloe on human diabetics. Their abstract makes the interesting statement that "The dried sap of the Aloe plant (aloes) is one of several traditional remedies used for diabetes in the Arabian peninsular." The study concerned five patients only, all of whom had non-insulin-dependent diabetes (NIDD). Two of these patients yielded two separate sets of experimental data, making seven records in all. Although the sample of patients is so small, the results obtained were most impressive. The fasting serum glucose levels were reduced from a mean of 273mg/dl before treatment to a mean of 151mg/dl after treatment. The insulin levels of these patients were unchanged. Interestingly, as has been stated, insulin levels in the blood of patients with NIDD commonly do not exhibit absolute insulin deficiency and it appears that their problem has much to do with a relative insensitivity of the body tissues towards being influenced insulin. The inference here is that treatment with aloes in some manner not yet understood, improves the responsiveness of the body tissues towards insulin, making the insulin which is already circulating in the blood, more effective. In four out of the seven records, the patient's serum glucose fell to normal (80-100mg/dl) or just above that range, while in three other cases, although serum glucose was dramatically reduced, the level continued to hover at or just slightly above the renal threshold for glucose excretion, of 180mg/dl.

This paper is enormously encouraging towards the idea that aloes are an effective remedy against NIDD. Without a doubt more trials, and, in particular, larger trials, preferably organised on a double-blind basis, are needed to clearly establish aloes as an effective remedy. This matter should not be left in abeyance because of its potential importance. The extent of the reduction in the blood sugar levels of these patients is both great and significant. Indeed, with the blood sugar reduced to 151mg/dl, the level has been reduced below the threshold at which sugar is obligatorily excreted into the urine. In that sense, these patients, after treatment, were not really diabetic at all, even though they still had a degree of hyperglycaemia. However, some surprise arises because the source of Aloe material being used was "bitter aloes", otherwise known as "drug aloes", and these names represent the aloin fraction. Therefore, if the Aloe material used is, indeed, effective against NIDD, then which subfraction of the aloin fraction is responsible? We do not know. Indeed, one cannot be completely sure that the effect is caused by an anthraquinone or phenolic component at all, since the sap of the plant - otherwise called the exudate - whilst it is a concentrate of anthraquinones and phenolics, also contains some of the same components which are in the gel or de-aloinized whole leaf extract.

Some confusion exists in the above paper about dose levels. A Pharmacopoeia reference dose for producing a purgative effect is given as 100-300mg/ day. The dose given in the study to the patients concerned was "half a teaspoon daily", which is much more than the 100-300mg. However, later the dose used is quoted as being "too small to produce diarrhoea". It may be that the investigators were employing an Aloe preparation far less concentrated that the reference Pharmacopoeia material, but this point is not explained. Future trials will therefore have to proceed with some care with
Ghannam et al (1986) also studied the effect of Aloe treatment upon diabetic mice and reported an improvement (hypoglycaemic effect) of approximately 43% in their plasma glucose levels after 7 days of treatment. Another significant paper was published the year before (1985) by O.P. Agarwal, entitled “The Prevention of Atheromatous Heart Disease”. There were 5000 patients in the study in all, aged 35 to 65 years, with follow-up over a five year period. The study is of interest here because over 60% of these patients with heart disease also had diabetes. Over 94% of these diabetic patients experienced improvements in their blood sugar levels during the trial. This trial was also of interest because the Aloe material used was fresh Aloe Gel (100g / day, given along with 20g / day Husk of Isabgol. This was quite different from the drug Aloes used by Ghannam et al (1986).

The work of Ghannam et al (1986) also provides the result that Aloe exerts its hypoglycaemic effect by reducing the body’s production of blood glucose by the breakdown of protein (termed “gluconeogenesis”), whereas the positive action of myrrh in diabetes was achieved by increasing the tissue oxidation of glucose. The obvious inference from this is that Aloe, in a form which contains both main fractions, and myrrh, should be especially effective in combination for the treatment of diabetes.

The remaining publications concerning Aloe and diabetes also deal with the treatment of animals. Some such publications also come from the Middle East, such as those of Al-Awadi (1985) and Farida et al. (1987) of Kuwait. They concluded that both Aloe, and another plant which they studied, myrrh, were significantly hypoglycaemic. Blood sugar was reduced in diabetic mice in one set of experiments by 6% and in another by 26%. By comparing the 1987 study with their results from previous studies, also concluded that “different parts of the Aloe plant may lower blood glucose by different mechanisms”. This holds the likelihood that an optimum Aloe product for the treatment of diabetes might need to contain, in addition to the material of the gel or whole leaf extract, some components from the aloin fraction. Therefore, the evidence is mounting that an optimum Aloe product for the treatment of diabetes might need to contain, in addition to the material of the gel or whole leaf extract, some components from the aloin fraction. Therefore, the evidence is mounting that an optimum Aloe product for the treatment of diabetes might need to contain, in addition to the material of the gel or whole leaf extract, some components from the aloin fraction.

Further work was done by Ajabnoor (1990). This work, again used the exudate compounds of Aloe (drug Aloes), confirming a powerful hypoglycaemic effect in diabetic mice, producing a lowering of blood sugar by up to 53%. In this case the conclusion was reached that the Aloe exerted its effect through stimulation if insulin secretion and that it was more effective in this respect than the drug tolbutamide.

It is fair to conclude from all the above work that Aloe vera can be an impressively potent medicine in the treatment of Type II Diabetes and that its widespread use should be strongly encouraged throughout the alternative field of medicine. The medical orthodoxy will find it hard to refute the evidence that exists in the literature. They just need the motivation to examine it carefully and to conduct whatever further trials on Aloe that they might require for their own purposes.

The Prospects for the Use of Aloe in Type I Diabetes

Type I Diabetes usually arises in circumstances which make it hard to perform trials of a herbal remedy. This is because the processes leading up to the full-blown disease take place insidiously over a few years and are likely to go unsuspected until the full-blown disease appears. At that point the patient’s situation is already critical and orthodox treatment, using insulin, is usually applied under the pressures of an emergency need.

However, there are very good reasons to believe that Aloe, and other alternative approaches, could help the Type I diabetic patient very considerably and that they may well be capable of reversing the condition if applied in time. In this context, the genetic susceptibility factor has to be recognised. The aim of alternative therapy has to be to keep the condition in a form in which it remains no more than a susceptibility. The factors which trigger the overt disease need to be kept at bay or reversed.

The reasons why one should expect Aloe to help significantly in fending off Type I diabetes are:
- That, at root, Type I Diabetes is an autoimmune condition and its origins therefore lie in a disorder of the immune system. The power of Aloe to support the immune system has been demonstrated clearly in Newsletter No.1. This power to normalize the immune system bears promise of possibly reversing a major triggering factor in the disease.
- That the attack upon the beta cells of the islets in Type I Diabetes, from its onset, gives rise to a great deal of inflammation. Indeed, the autoimmune condition, once initiated, is increased and taken on to a more profound
stage precisely because the early, normally unseen stages, have already given rise to some degree of inflammatory damage to beta cells. The moderation and control of that inflammatory damage, using the anti-inflammatory properties of Aloe, would be expected to moderate the progress and at least delay the onset of Type I diabetes.

- The role that Aloe has in encouraging tissue cleansing, through its action upon the immune system, can be expected to remove cellular toxins that play a key role in promoting Type I diabetes in susceptible individuals. This power to encourage cleansing of tissue cells throughout the body bears promise of possibly reversing another major triggering factor in the disease.

It is not possible to report any trials at all with Aloe and Type I Diabetes. None have been found. However, the above observations provide the strongest possible reasons for conducting such trials in the future.

The Nature of the Secondary Complications of Diabetes

The principal complications of diabetes mellitus are:

- **Microangiopathy** - which means deterioration of the very small blood vessels in many different parts of the body. The organs most affected are the capillaries of the skin, skeletal muscles, retina, the glomeruli of the kidney, and the medulla of the kidney. However, peripheral nerves can be affected and also, where applicable, the placenta. The deterioration takes the form of a diffuse thickening of the basement membrane of the vessels. Despite this thickening, the vessels are much more leaky than normal towards the plasma proteins. The condition has far-reaching implications, inducing serious lesions in the kidneys, the retina and possibly also the nervous system (Figure 6).

- **Atherosclerosis** - which is the principal form of arterial disease. Less than 5% of nondiabetics as opposed to 75% of diabetics below the age of 40 have moderate to severe atherosclerosis. In diabetics this leads to the narrowing and occlusion of arteries, ulceration, calcification, aneurysms (areas of severe weakness in walls of large arteries) and much concomitant ischaemic injury to organs (injury which occurs through oxygen lack). All this, especially conditions of the larger vessels, commonly leads to gangrene of the lower extremities, which is 100 times more common in diabetics than nondiabetics.

- **Retinopathy** - which is pathological change in the retina of the eye, apparently due to the small blood vessel deterioration in that area. It commonly leads to visual impairment and is the fourth most frequent cause of legal blindness in the USA. It may be accompanied by cataract or glaucoma.

- **Nephropathy** - which is pathological change in the kidneys, apparently due mainly to the small blood vessel deterioration in those organs. The kidneys are usually the most severely damaged organs in the diabetic.

- **Neuropathy** - peripheral nerves, brain and spinal cord may all be damaged in long-standing diabetes. Very commonly, both motor and sensory nerves to the lower extremities are most affected, so both movement and feeling may be lost to various degrees.

Both of the mechanisms by which these adverse changes occur result directly from the hyperglycaemia of diabetes. These are:

- **The glycosylation of tissue proteins.** This consists of the non-enzymic combination between glucose - at the excessive concentrations which exist in the tissues, and tissue proteins.
non-enzymic, these reactions are not under metabolic control. These spontaneous combinations of protein with carbohydrate result in corrupted forms of tissue proteins which then get in the way of normal metabolism and which may also alter cellular and extracellular structures. The protein collagen particularly suffers from alteration in this way. Such derivatives can be fairly regarded as toxins.

The accumulation of excess glucose, in those cell types which do not require insulin for glucose transport, leads to metabolic derangements. These include high internal concentrations of the carbohydrates sorbitol and fructose, which in turn, upset the concentration and metabolism of Inositol, resulting in much loss of control over cell processes.

How Aloe impinges upon the Secondary Complications of Diabetes

There is no evidence to suggest that Aloe can either inhibit the glycosylation of proteins or reduce the intracellular concentrations of sorbitol or fructose. Consequently it is most probably not able to intervene in the secondary complications directly, but only by contributing, in the ways already described, to eliminating the factors which triggered the diabetic condition in the first place. It is true however, that at times these complications result in infections and / or inflammations. Where these occur there is reason to think that Aloe will assist, since it clearly is anti-inflammatory and also helps the body to fight infection. However, by the time that stage has been reached, the damage caused by the complications will have become well established, thus limiting the amount of real help that can be given. The secondary complications of diabetes are very dependent upon the extent to which the hyperglycaemia gets out of control and the length of time during which is allowed to remain out of control. Therefore, the best way to fend off these complications is to use Aloe, in conjunction with full use of Nutritional Medicine methods, to reduce the severity of the diabetes, to limit the duration of the diabetic condition and, wherever possible to actually reverse the triggering factors which set up the condition in the beginning.

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The information contained in this newsletter is derived mainly from scientific and medical literature and is intended to inform the reader. None of what is contained here is to be interpreted as a medical consultation. Sufferers from Diabetes who are potential patients are responsible themselves for any action taken in self medication but are advised to see a Practitioner. In fact the process of coming off insulin or reducing insulin dose in conjunction with herbal remedies and nutrition calls for careful monitoring of blood sugar and other factors by a Practitioner. The Author is not responsible for clinical steps based upon the newsletter.
stage precisely because the early, normally unseen stages, have already given rise to some degree of inflammatory damage to beta cells. The moderation and control of that inflammatory damage, using the anti-inflammatory properties of Aloe, would be expected to moderate the progress and at least delay the onset of Type I diabetes.

The role that Aloe has in encouraging tissue cleansing, through its action upon the immune system, can be expected to remove cellular toxins that play a key role in promoting Type I diabetes in susceptible individuals. This power to encourage cleansing of tissue cells throughout the body bears promise of possibly reversing another major triggering factor in the disease.

It is not possible to report any trials at all with Aloe and Type I Diabetes. None have been found. However, the above observations provide the strongest possible reasons for conducting such trials in the future.

The Nature of the Secondary Complications of Diabetes

The principal complications of diabetes mellitus are:

Microangiopathy - which means deterioration of the very small blood vessels in many different parts of the body. The organs most affected are the capillaries of the skin, skeletal muscles, retina, the glomeruli of the kidney, and the medulla of the kidney. However, peripheral nerves can be affected and also, where applicable, the placenta. The deterioration takes the form of a diffuse thickening of the basement membrane of the vessels. Despite this thickening, the vessels are much more leaky than normal towards the plasma proteins. The condition has far-reaching implications, inducing serious lesions in the kidneys, the retina and possibly also the nervous system (Figure 6).

Atherosclerosis - which is the principal form of arterial disease. Less than 5% of nondiabetics as opposed to 75% of diabetics below the age of 40 have moderate to severe atherosclerosis. In diabetics this leads to the narrowing and occlusion of arteries, ulceration, calcification, aneurysms (areas of severe weakness in walls of large arteries) and much concomitant ischaemic injury to organs (injury which occurs through oxygen lack). All this, especially conditions of the larger vessels, commonly leads to gangrene of the lower extremities, which is 100 times more common in diabetics than nondiabetics.

Retinopathy - which is pathological change in the retina of the eye, apparently due to the small blood vessel deterioration in that area. It commonly leads to visual impairment and is the fourth most frequent cause of legal blindness in the USA. It may be accompanied by cataract or glaucoma.

Nephropathy - which is pathological change in the kidneys, apparently due mainly to the small blood vessel deterioration in those organs. The kidneys are usually the most severely damaged organs in the diabetic.

Neuropathy - peripheral nerves, brain and spinal cord may all be damaged in long-standing diabetes. Very commonly, both motor and sensory nerves to the lower extremities are most affected, so both movement and feeling may be lost to various degrees.

Both of the mechanisms by which these adverse changes occur result directly from the hyperglycaemia of diabetes. These are;

The glycosylation of tissue proteins. This consists of the non-enzymic combination between glucose - at the excessive concentrations which exist in the tissues, and tissue proteins. Being
non-enzymic, these reactions are not under metabolic control. These spontaneous combinations of protein with carbohydrate result in corrupted forms of tissue proteins which then get in the way of normal metabolism and which may also alter cellular and extracellular structures. The protein collagen particularly suffers from alteration in this way. Such derivatives can be fairly regarded as toxins.

The accumulation of excess glucose, in those cell types which do not require insulin for glucose transport, leads to metabolic derangements. These include high internal concentrations of the carbohydrates sorbitol and fructose, which in turn, upset the concentration and metabolism of Inositol, resulting in much loss of control over cell processes.

How Aloe impinges upon the Secondary Complications of Diabetes

There is no evidence to suggest that Aloe can either inhibit the glycosylation of proteins or reduce the intracellular concentrations of sorbitol or fructose. Consequently it is most probably not able to intervene in the secondary complications directly, but only by contributing, in the ways already described, to eliminating the factors which triggered the diabetic condition in the first place. It is true however, that at times these complications result in infections and / or inflammations. Where these occur there is reason to think that Aloe will assist, since it clearly is anti-inflammatory and also helps the body to fight infection. However, by the time that stage has been reached, the damage caused by the complications will have become well established, thus limiting the amount of real help that can be given. The secondary complications of diabetes are very dependent upon the extent to which the hyperglycaemia gets out of control and the length of time during which is allowed to remain out of control. Therefore, the best way to fend off these complications is to use Aloe, in conjunction with full use of Nutritional Medicine methods, to reduce the severity of the diabetes, to limit the duration of the diabetic condition and, wherever possible to actually reverse the triggering factors which set up the condition in the beginning.

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