

# SCIENTIFIC

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## PHYSICAL THERAPY

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### **Patient with Left Knee Pain**

Wesley Wlodarski, PT  
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#### **History:**

The patient is a 34 year old male who presented with complaints of left knee pain. He sustained a left knee injury during skiing approximately six months ago. He was diagnosed with a comminuted, depressed fracture of the left lateral tibial plateau. Initially the patient received knee brace and crutches and was scheduled for follow up visit after one month. The patient decided to have a second medical opinion, which took place approximately two weeks post injury. The next day he underwent a surgical procedure of open reduction and internal fixation of the lateral tibial plateau, lateral meniscal repair of the left knee.

Following surgery, the patient was using a continuous passive motion machine for approximately four weeks with gradually tapering frequency. Initially he had been using crutches, then weaned himself to a cane and for the past three weeks has been walking without assistive device.

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Didrik J Sople, PhD., L.Ac.

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Two months after surgery, the patient started physical therapy in which he continues to take part.

The patient reports being in good general health. Up until the accident, he was very active in sports such as tennis, skiing, biking, and running. He is self-employed and spends much of his day driving a car.

### **Current Symptoms:**

The patient's primary complaint is pain with closed chain daily activity, especially after walking or during stair climbing. He localizes his pain to the anterior medial and lateral aspect of the left knee. He also reports stiffness of the left knee during first steps in the morning or after prolonged driving activity. Recently the patient has been reporting right knee pain, which started since he has stopped using crutches. He also relates left and right knee pain secondary to changes in the weather. The left knee has a tendency to swell up after walking. Occasionally his left knee "gives out."

The patient informed me that about five years ago during squatting he felt a crack in his right knee then for about 10

days his knee was very painful. Since then he has been experiencing occasional right knee pain after sports activity like playing tennis.

### **Inspection**

Mild swelling is present in the left lower extremity. Mild vastus medialis atrophy is noted compared to the right. The left patella is positioned slightly superior to the right. A longitudinal scar is present over the anterior aspect of the left knee. Slight genu valgus is evident bilaterally. Calacaneal valgus is prominent on the left side. The patient is apprehensive with weight-bearing and favors his right lower extremity.

### **Function**

Gait analysis revealed that the patient is avoiding heel strike on the left side. When correcting his gait pattern he is reporting pain on the anterior inferior aspect of the left knee, distal to the patella. He is also exhibiting increased pronation on the left side. There was reduced balance on his left lower extremity as compared to his right. Pain is also present in the infrapatellar region with step-up and step-downs.

### **Active Range of Motion**

Flexion and extension are within normal limits bilaterally. During knee flexion on the left, the patient reports pain and tightness. He is reporting grinding sensation under the patella bilaterally. Slight limitation was noted towards external and internal tibial rotation.

### **Passive Range of Motion**

Slightly limited left knee flexion due to pain superior lateral aspect of the patella. Also limited internal and external rotation in the left knee. The end feel is elastic with flexion and firm with extension. Extension over pressure caused pain in the anterior knee region.

### **Resistive Tests**

Pain provoked with extension through all ranges of tension along the anterior medial aspect of the left patella. There was also pain with external tibial rotation in the mid range over the anterior lateral aspect of the left knee.

### **Palpation**

Tenderness was elicited along the lateral and medial inferior border of the left patella. Tightness and subcutaneous restriction was evident along the surgical



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incision. Decreased tone was present in the left quadriceps. Tension and tenderness was evident in the left IT band, gastrocnemius and tensor fascia lata.

### **Neurology**

Slight decrease sensation to sharp touch in the region of the lateral sural nerve distribution. Myotomes and reflexes were intact and symmetric.

### **Special Tests**

Anterior drawer, Lachman's, valgus stress tests were all positive (+) and grade I. Stutter test was (+) bilaterally at 30°. Ober's test and patella

compression test were both (+).

### **Joint Mobility Testing**

Grade 2 restriction of patellar glide in medial, lateral and inferior directions was evident. Grade 2 mobility was present with posterior tibial glide and external tibial rotation at the knee joint and lateral glide of the left subtalar joint and anterior glide of the proximal tibiofibular joint, the latter with creptius.

### **Impression**

The primary tissue in lesion: left patellar cartilage. Other problems which need to be addressed are decreased

left patellar mobility, decreased left subtalar joint mobility, shortness of the left iliotibial band, gastrocnemius, and decreased proprioception.

To address the above problem list, the patient was placed on a treatment program including:

- Soft tissue mobilization to the IT band, gastrocnemius and quadriceps muscles
- Joint mobilization including distraction and glides to restore mobility of the left knee, ankle and patellofemoral joint

- S.T.E.P. principle: To begin with hamstring exercise to promote gliding in the patellofemoral joint with minimal retro-patellar compression. Advance to closed-kinetic chain for hamstring and quad work with gradually increasing resistance, then open kinetic chain. Functional activities, balance and coordination exercises introduced as well.
- Taping of the left foot and ankle to facilitate receptor activity and to improve muscular control of left foot and ankle alignment.
- Instruction in proper movement awareness for activities of daily living as well as home exercise program to enhance gains made in the clinic.

**Goals**

1. Decrease subjective complaints of pain
2. Decrease local tenderness
3. Decrease swelling
4. Increase soft tissue mobility
5. Correct muscle imbalance
6. Improve gait pattern
7. Increase tissue tolerance to daily activity
8. Improve proprioception
9. Make the patient independent in home management program.

**Response to Treatment**

The patient responded extremely well to physical therapy intervention. He met his expected outcomes within six weeks at a frequency of one to two times per week in the clinic. He was able to return to his desired level of activity without pain and was discharged from formal physical therapy with a home program.

Studies exploring the relationship between sacralization and spondylolisthesis include Kin and Suk 1997 (6). They compared radiographs of 21 sacralized and 12 lumbarized cases with 149 controls. Sacralized cases with isthmic defects at L4-5, showed greater anterior slippage than lumbarized cases with defects; both cases showed greater slippage than controls. Sacralized cases with defects at L5-S1 showed less slippage than lumbarized; and both showed less slippage than controls. Cinotti 1997 (7) compared flexion and extension stress x-rays between 27 cases of spondylolisthesis, and 27 cases without spondylolisthesis. Facet joint orientation

(more sagittal), and movement at the affected level, was the only significant factors associated with the presence of spondylolisthesis. Sacralization, lumbar-sacral angle and intercrestal line were not significant.

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# The importance of optimal cellular metabolism as it relates to orthopedics

Didrik J Sople, PhD., L.Ac.

Cellular metabolism affects treatment outcomes.

Patients who have near optimal cellular metabolism will respond to an injury with a normal inflammatory response, followed by tissue healing within a reasonable amount of time.

Patients who have dysfunctional cellular metabolism might, instead respond with an increased inflammatory response and slow healing.

Some might even when exposed to an injury, a viral infection, psychological or chemical stress develop chronic fatigue and fibromyalgia syndrome.

Depending on the dysfunction of the cellular metabolism the type of chronic condition might vary. Other examples are chronic bursitis, tendonitis or degenerative conditions like arthritis.

With impaired cellular metabolism tissue tolerance to stress will decrease, resulting in higher

susceptibility to repetitive stress disorders.

This does not only pertain to the general patient population but also to athletes who train hard to reach their highest level of performance. Athletes who have good cellular metabolism will perform to their optimal level. The ones who have compromised cellular metabolism will experience decrease strength and endurance as compared to their optimal capacity. They may also be more prone to injuries.

Chronic conditions in general from inflammatory conditions to degeneration are commonly blamed on genetics or the aging process.

Genetic predisposition can certainly make us more susceptible to disease, and while we cannot at this time change the patient's genes, it may, however, be possible to alter the genetic expression.

More and more research is now showing how disease outcomes can be altered by supporting for example a specific metabolic pathway, which genetically was impaired, with specific nutrients. By using specific nutritional metabolites to compensate for a genetic error, disease outcomes can be altered and certain chronic conditions may be prevented.

Starting with an acute injury, this article will present physiological reactions as it relates to cellular metabolism. Suggestions on how to nutritionally support the tissue for more optimal function on a cellular level will also be discussed.

This article will not cover all the specifics of the inflammatory cascade and all the different ways to help inhibit inflammation, although it will cover some aspects of inflammation.

When injury occurs it produces a sympathetic response, leading to a release of epinephrine and cortisol<sup>1</sup>.

Epinephrine will mobilize glucose and fat, while cortisol will stimulate gluconeogenesis (mobilization of amino acids) and the mobilization of fatty acids (increases free fatty acids in the plasma). After the amino acids have been mobilized, they are deaminated in the liver to glucose.

These reactions are the body's attempt to provide the cells with fuel to prevent cell death. In the case of injury the calorie need increases as well as the requirement for protein. The more severe the injury is, the higher the calorie need will be and it should be compensated for with increased intake of complex carbohydrates.

To avoid a negative nitrogen balance, it is recommended to increase the protein intake according to the severity of the injury <sup>2</sup>.

Elective surgery:

1.5 g/kg body weight

Multiple traumas:

1.5-2 g/kg body weight

Burns:

2.5 g/kg body weight

The protein can be supplied as part of the regular meals or as an added supplement. In many cases depending on the individual's dietary habits and ability to increase protein by increasing their food intake it maybe easier to add a protein supplement. A high quality supplement would be whey protein prepared by ultra filtration at controlled temperatures and pH to prevent it from denaturing (becoming inactive). The highest quality whey protein would also provide bioactive immunoglobulins. Branched chain amino acids in the amount of 3.3 g can also be used to help maintain normal nitrogen balance <sup>3,4</sup>.

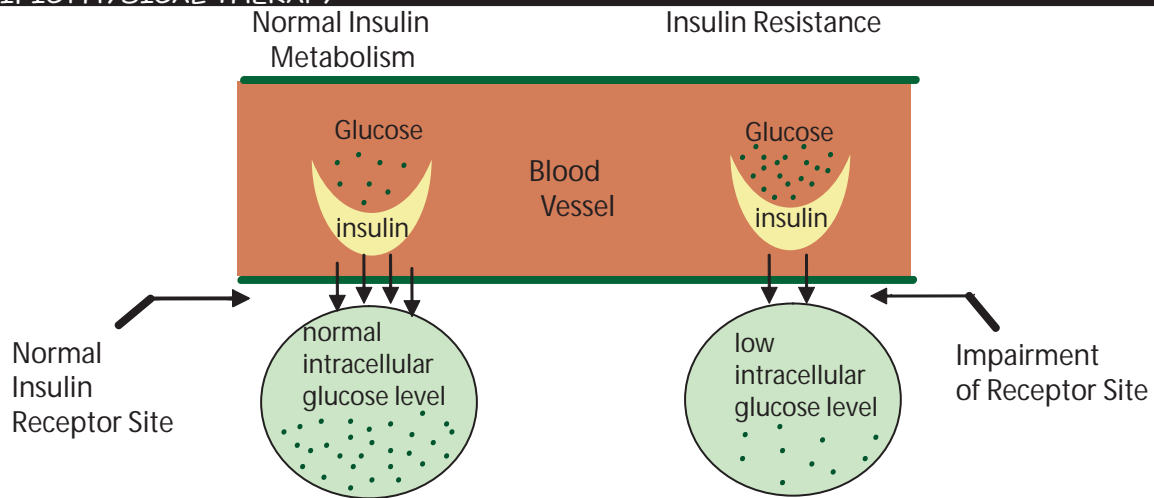
The patients who develop a chronic condition instead of recovering have a different predisposition; the injury was only the triggering factor. The triggering factor for fibromyalgia or chronic fatigue syndrome could even be a viral infection instead of a physical injury. Any stressor exceeding the physiological limit could, depending on the predisposition trigger a chronic condition.

Why some individuals have such a predisposition may

not always be easy to answer. Genetics can always be blamed, but research has started to show that genetic predispositions do not necessarily mean we have to develop certain conditions.

Let us look at some of the contributing factors to chronic conditions. The factors presented here can be modified by changing dietary habits and supplying the tissue with the right nutritional support, thereby altering the outcome. One basic, but very important factor is to pay attention to the glycemic index of the food we eat. Intake of high glycemic index foods if eaten consistently will in time result in insulin resistance <sup>5</sup>. Depending on genetic predisposition, some individuals, will end up insulin resistance faster than others. Anybody who regularly eat foods that elevate the blood sugar high can, however, develop this condition.

Glucose is a very important metabolite for the energy production and insulin resistance will lead to decreased energy as well as higher levels of inflammatory cytokines.



It may also in some individuals lead to adult onset diabetes if the pancreas is not able to increase the output of insulin in an attempt to compensate.

Looking at the mechanism of glucose transfer into the cell, it is easy to see why insulin resistance would reduce the cells ability to produce energy. In a normal functioning cell the insulin will transfer glucose from the blood into the cell by attaching to the insulin receptor site on the cell. When insulin resistance has developed, impairment of the receptor site on the cell decreases the amount of glucose transfer by the insulin into the cell. The result is decreased intracellular glucose levels and decreased energy production.

As the insulin resistance worsens, blood glucose levels will be raised. This can be noticed in a fasting blood glucose test. The pancreas will compensate with increased production of insulin in an attempt to transfer the glucose into the cells. It is when the pancreas is no longer able to do that adult onset diabetes occurs.

Even if diabetes never develops, blood insulin levels will be elevated. This can be verified by blood analysis. Still with elevated insulin levels, cell glucose levels will be low and blood glucose levels higher than it should be resulting in a starving cell. A patient with this condition will for that reason have less energy than he or she used to have. Another sign is increased waist to hip ratio.

Glucose not used for energy or stored as glycogen in the muscles and the liver will be converted to and stored as fat to be used as energy at a later occasion, when starving. Since few people in our society have a problem with starvation, that later occurrence never happens and fat will therefore continue to accumulate.

It is also common to see elevated triglyceride and cholesterol levels in individuals with insulin resistance.

Another common sign in these individuals are inflammatory conditions. That is one of the reasons their ability to heal can be impaired when they are exposed to an injury of the musculoskeletal system.

Their tissue tolerance for repetitive stress will also decrease and they may develop tendonitis and bursitis easier.

Recovery from such a condition will likely be slower and it may just be blamed on getting older.

What is the mechanism by which insulin resistance can create inflammation?

Patients who have insulin resistance will accumulate glycosylated protein which is the reaction of glucose with protein amino groups. Increased oxidative stress reactions occur and we will see a shift toward inflammation<sup>6</sup>. The increased blood levels of insulin as seen in insulin resistant individuals will raise the levels of pro-inflammatory cytokines like interleukin 1 and 6, activating the arachidonic acid cascade and production of prostaglandin E2<sup>7</sup>. This type of biochemistry may also aggravate degenerative conditions like arthritis.

While regular exercise is one of the things which can help to prevent insulin resistance, it does not mean that someone who is exer

cising is immune to insulin resistance. If their diet consists of many simple carbohydrates they can still be at risk. It is becoming more common to use high glycemic index carbohydrate drinks now as these products are promoted to be the solution to high performance. If these products are used incorrectly as a meal replacement instead of when exercising, they can create problems after a while.

The solution for prevention of insulin resistance and to help reverse it if it is already present is to adapt a low glycemic index diet. A low glycemic index diet is a diet which does not result in high blood sugar, but instead helps to stabilize the blood glucose at healthy levels.

General guidelines would be to avoid unhealthy foods like candy, cookies and donuts. By including fiber-rich foods like vegetables, beans or lentils in a meal, the absorption of glucose found in bread, rice, potatoes and pasta will be slowed down. The total meal will therefore have a lower glycemic index and help stabilize the blood sugar.

By decreasing the amount of food like bread, potatoes, rice and pasta, increasing the amount of vegetables and including enough protein to fill the need, a significant improvement to the diet is accomplished. Essential fatty acids are also very important to include daily.

Helping the patient realize the importance of a low glycemic index diet would benefit them for the rest of their lives.

In the last few years specific B vitamins have received a lot of attention. These are vitamin B<sub>6</sub>, B<sub>12</sub> and folic acid. The reason is that they are involved in a biochemical reaction called methylation. Other nutrients involved in methylation are vitamin B<sub>2</sub>, B<sub>3</sub>, betaine and magnesium.

Methylation is important for several reasons. It is involved in detoxification of a variety of toxins including the conversion of hormonal estrogens to safer derivatives<sup>8</sup>. It is involved in the formation of phosphatidylcholine important for the structure and function of the nervous system. It is involved in the conversion of norepinephrine

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to epinephrine and the biotransformation of serotonin.

Folic acid is one of the most important vitamins involved in methylation. Approximately 15-25% of the population have genetic difficulty converting folic acid to 5-methyltetrahydrofolate and 5-formyltetrahydrofolate the metabolites of folic acid<sup>9</sup>. This will lead to lowered efficiency of their methylation reactions. There are also individuals who do not get adequate amount of nutrients in their diet involved in the methylation process. **Individuals with decreased methylation capacity will be more susceptible to what is considered age related diseases like inflammatory conditions, arthritis, osteoporosis, heart disease and even cancer<sup>10</sup>.**

These are some of the steps in the methylation process: folic acid is metabolized to 5-formyltetrahydrofolate and 5-methyltetrahydrofolate. 5-methyltetrahydrofolate converts homocysteine to methionine and then to S-adenosylmethionine [SAM]. SAM is the main substance involved in the methylation process.

A high homocysteine level is recognized as a risk factor for not only cardiovascular disease, but inflammatory and degenerative conditions as well as certain neurological diseases.

A high homocysteine level can effectively be decreased by the intake of folic acid, B<sub>12</sub>, B<sub>6</sub> and betaine. Homocysteine is therefore a good test to verify a deficiency of these B vitamins. It is better than serum levels of these vitamins which sometimes can be normal even with high homocysteine and a deficiency present in other tissues. High homocysteine levels may however not be an accurate indicator of the whole methylation process since homocysteine may be normal even when methylation is not occurring properly<sup>11</sup>. The best assurance for a normal methylation process is to supply a mixture of folic acid, 5-formyltetrahydrofolate and 5-methyltetrahydrofolates, vitamins B<sub>12</sub>, B<sub>6</sub>, B<sub>3</sub> and B<sub>2</sub>, betaine and magnesium<sup>12</sup>.

This can be accomplished by taking an up to date B-complex formula which will contain all of these B vitamins.

By adding a good multivitamin formula containing betaine and magnesium, as well as, other nutrients, better tissue support can be realized.

S-adenosylmethionine [SAM] one the main substances involved in methylation, can also directly be given as a supplement marketed as SAM-e. Several studies have shown SAM-e to be effective in decreasing the symptoms of osteoarthritis<sup>13</sup>. The pain relieving effect of SAM-e is comparable to that of nonsteroidal drugs [NSAID's] but without the side effects<sup>14</sup>. SAM-e comes in two forms. The original form is butanedisulfonate with a 5% oral bioavailability, while the other form sulfate-p-toluensulfonate also called tosylate has a 1% oral bioavailability<sup>15</sup>. The tosylate formulation does not seem to be a stable compound and concerns have been expressed regarding that issue<sup>16</sup>. For osteoarthritis an oral dose of 200 mg three times daily is commonly used.

In certain patients the cellular energy metabolism may be impaired. Some of the findings in chronic fatigue

syndrome include mild aerobic defects in skeletal muscles and low level activation of the immune system<sup>17, 18</sup>.

NADH [reduced nicotinamide adenine dinucleotide] is an essential intermediate in the production of energy in the cell, forming ATP from glucose. Through chemical reactions in the citric acid cycle NAD<sup>+</sup>, a derivative of the B vitamin niacin, react with hydrogen and dehydrogenase to form NADH. NADH can now be used as a supplement and in a study with chronic fatigue patients it showed reduction of pain and fatigue<sup>19</sup>.

Recommended dosage is 5 mg once or twice daily.

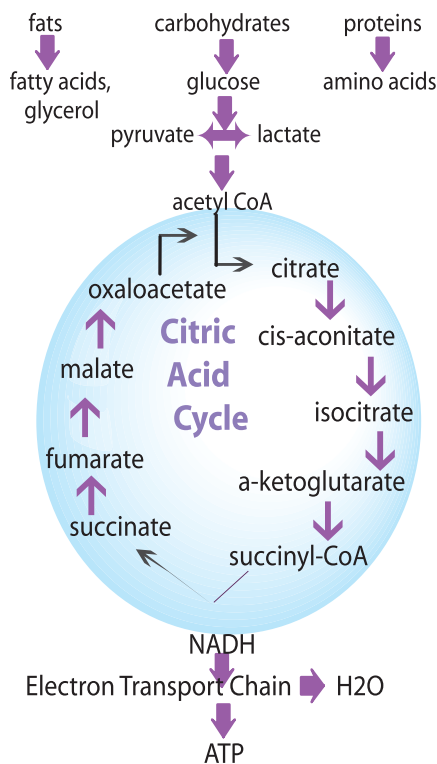
**Summary:**

Recommend a low glyce-mic index diet to promote normal biochemistry and optimal energy production. To support a normal methy-lation process recommend a well designed vitamin B-complex formula and multi-vitamin formula.

To support methylation and specifically treat osteoarthritis SAM-e can be used especially if the patient tend to be depressed. SAM-e has shown to help depression. Since SAM-e can be quite expensive, it probably should not be the first treat-ment choice for osteoarthritis. For patients suffering from chronic fatigue, fibromyalgia syndrome NADH is recommended. It is important to remember that patients treated for orthope-dic problems need support for all their connective tissue which include: bone, carti-lage, tendons and liga-ments. They also need support for neuromuscular function.

copper, manganese, vita-min D, vitamin B<sub>6</sub>, vitamin C and glucoseamine sulfate.

[references on next page]



The most important nutrients in this respect include cal-cium, magnesium, zinc,

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## Freddy Kaltenborn 80 years old. Congratulations

There are few people, if any, involved in physical therapy and manual therapy that do not recognize the name, Freddy Kaltenborn.

Professor Freddy Kaltenborn has dedicated his life to the development of manual therapy, all the way from the beginning of the concept to what it has become today.

He has spent much of his life spreading the word of this concept worldwide by writing, teaching and lecturing. His accomplishments are many. Freddy Kaltenborn is now 80 years old. While most people long before reaching his age lose interest in their profession, he is still interested in supporting further growth, progress and understanding of manual therapy. His contributions to our profession are impressive.

Last year he was awarded an honorary doctoral degree from the Ola Grimsby Institute in California, U.S.A. A title well deserved for all his hard work. We want to congratulate you not only because it is your birthday, but also for all your accomplishments and support.

Happy Birthday,

Didrik J Söpler, Ph.D., L.Ac., Editor in Chief

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### An Interview with Ola Grimsby, PT, MNFF, MNSMT, FAAOMPT

**SPT:** What is new at the OGI this year?

**Ola Grimsby:** Every thing is new, that is, we have kept the best from our part and changed our programs according to guidelines, credentialing requirements and recommendations from the APTA and the AAOMPT. This means a brand new and very exciting curriculum, updated and in compliance with recent development in our field

**SPT:** This relates to what is going on in the US, but what about internationally?

**Ola:** We are offering degree granting programs in 10 countries in Europe, 5 countries in Asia and these programs are all coordinated with our US programs. For the first time we offer certification in manual therapy in intensive weekend course over 3 ½ months at 20 US locations. Such certification serves as the first component of our new 1 year DPT program. This is followed by a 1 year fellowship and a 1 year post doctoral DMT [Doctor of Manual Therapy]. However you can better see this from the graphic presentation in this issue.

**SPT:** When does this new program go into effect?

**Ola:** We will begin January 2004. All the new changes will be posted on our website: [www.olagrimsby.com](http://www.olagrimsby.com); within the next few weeks.

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