Despite the decline in cigarette smoking in the last few years as reported by the Centers for Disease Control and Prevention, and the well known health risks in cardiovascular and pulmonary diseases, millions of Americans continue to smoke cigarettes. It has been proven by both experimental and clinical observation that cigarettes impair bone and wound healing. The purpose of this article is to review the chemical components of cigarette smoke and its relationship with plantar callus formation.

Increased plantar callus formation with patients who smoke cigarettes seems to be a common problem. There are approximately 46.6 million smokers in the US. There was a decline during 1997-2003 in the youth population but during the last years the rates are stable (1). Approximately 3,450 young people between 12 and 17 years of age smoke for the first time (2). Approximately eight billion cigarettes are smoked annually in the US alone. Per capita, cigarette consumption stands at 3,900 cigarettes per adult smoker per year (3, 4).

Smoking a single cigarette can produce cutaneous vasoconstriction for up to 90 minutes (5). Depending on the amount of inhalation, 85-98% of the nicotine in the smoke can remain in the body (6). The tobacco smoke components absorbed from the lungs reach the heart immediately. Smoking increases the heart rate, arterial blood pressure, and cardiac output. There is a 42% reduction in the digital blood flow after a single cigarette (7, 8). Nicotine has a direct cutaneous vasoconstrictive effect and is the principle vasoactive component in the gas phase of cigarette smoke. It is an odorless, colorless, and poisonous alkaloid that when inhaled or injected, can activate the adrenal catecholamines from the adrenergic nerve endings and from the adrenal medulla, which cause vasoconstriction of vessels especially in the extremities. Nicotine also induces the sympathetic nervous system, which results in the release of epinephrine and therefore peripheral vasoconstriction and decreases blood flow (9, 10).

The authors believe that vasoconstriction of the peripheral vessels can lead to atrophy of subcutaneous tissue, which in turn can lead to increased bone to skin contact and callus formation. These same catecholamines act as cofactors in the formation of chalones, which are wound hormones that inhibit epithelization (Figures 1-5).

Whole fresh cigarette smoke has more than 4,000...
different compounds either in the gaseous or particulate phase with the two most common gases in smoke being carbon monoxide and hydrogen cyanide. Two to three milligrams of nicotine and twenty to thirty milliliters of carbon monoxide are present in each cigarette (3, 4). It takes a minimum of twelve hours in order to clear the carbon monoxide from the blood and return the carboxyhemoglobin level to normal (4). The affinity of carbon monoxide hemoglobin binding is 200 times that of oxygen so the carbon monoxide inhibits the binding of oxygen and this in return leads to decreased oxygen carrying capacity of hemoglobin. This further reduced amount of oxygen to tissue leads to cellular hypoxia and cutaneous vasoconstriction (4). Hydrogen cyanide blocks cellular oxygen metabolism therefore decreasing tissue oxygen consumption (9, 10). Smoking a cigarette for ten minutes decreases the tissue oxygen tension for almost an hour.

Also, the odorless, colorless, poisonous alkaloid nicotine has been shown to increase platelet adhesiveness and platelet turnover, which in turn leads to increased blood viscosity. This increased blood viscosity may cause microclots and decrease microperfusion (9-14). The rates of collagen deposition, epithelization, and angiogenesis are related to arterial and tissue oxygen content. Jensen et al showed that after smoking for ten minutes, the tissue oxygen content decreased to 60 +/- 6mmHg and continued to decrease to 44 +/- 3mmHg after thirty minutes (15). Compare this to tissue oxygen tension, which decreases to 0mmHg only after 30 minutes of tourniquet induced ischemia. Hunt TK et al, showed that a constant reduction of tissue oxygen tension in animals by 30% reduced collagen deposition by about 30-50% (16).

Smoking increases the risk for facial wrinkling two- to three-fold due to the decreased capillary and arterial blood flow in the skin. This may lead to ischemia of the dermis and leads to collagen and elastin damage. These are the connective tissue elements that are important to maintain the integrity of the skin. It has also been hypothesized that smoking decreases vitamin A levels, which provide protection against oxygen radicals that damage DNA and connective tissue (17).

In 1983, Ludwing and Hoidal (18) showed that neutrophils of smokers release excessive amounts of oxidants, which damage tissue and antiproteases. Fibroblasts can survive but cannot synthesize collagen without oxygen (16). Strength is provided by members of the collagen family. The ability of skin to recoil after transient stretching is provided by elastic fibers. Elastin, a 70Kd glycoprotein forms an interlacing network of fibers in the papillary dermis and long thick fibers in the reticular layer of the dermis. Elastin is rich in glycine and proline, but unlike collagen, it contains almost no hydroxylated amino acids. Elastin molecules are
cross-linked and form an extensive network. This network
determines the capacity of stretching and recoil (12). It has
been found that smokers have a higher blood concentration
of neutrophils and lymphocytes (14). Neutrophils contain
elastase, the enzyme that breaks up elastin. The authors
believe that the increase in neutrophil levels will result in an
increase in elastase levels and this in return will cause
damage to the tissue elastin. The damaged elastin will cause
the plantar skin to become less pliable and this will result in
increased callus formation.

Cigarette smoke has been shown to increase plasma
neutrophils and elastase activity, which may cause abnormal
elastin formation (5). In vitro, cigarette smoke blocks the
cross-linking that normally occurs with elastin (5). Elastin in
smokers has shown to thicken and become more fragmented
than in nonsmokers (5). Damage to elastin causes wrinkling
of the skin and the wrinkles become coarse and the skin is
thickened (11). This thickening of the skin can lead to
increased plantar callus formation due to the loss of
elasticity and pliability of the skin and increased sensitivity to
friction (Figures 6-8).

Chronic ischemia of the dermis due to cigarette smoke
also plays a role in damaged elastic fibers as well as the
decrease in collagen synthesis (5). The typical pack-a-day
smoker will remain hypoxic for most of the day (9, 11-13).
The combination of the above effects will increase plantar
callus formation.

There are other factors that are involved in the
development of callus formation that were not discussed;
however, our focus was to establish and determine the
correlation between cigarette smoking and callus formation.
The increase in elastase levels will result in the damage of
eLASTIN in the tissue and therefore lead to an increased callus
formation. The vasoconstriction effect of nicotine
contributes to increased callus formation by causing a
decrease in blood supply and perfusion to the tissue, which
ends in connective tissue degradation, atrophy, and more
bone to skin contact.

Despite the known health hazard seen with cigarette
smoking, millions of Americans will continue to smoke. Our
duty as podiatrists is to provide the best care, attention, and
education to our patients. Prevention should be our number
one priority. Patients should be educated and aware of the
relationship between smoking and plantar callus formation
not only to benefit the health of their feet, but also to
realize the extent of this habit. Sometimes we are only aware
of the damage within when we can see it in the outside.

The clinical guidelines for tobacco induced
hyperkeratosis are 1. multiple (more than three) plantar
lesions; 2. plantar heel lesions identifies tobacco use;
3. multiple porokeratosis with plantar hyperkeratosis.
REFERENCES

1. Substance Abuse and Mental Health Services Administration. Results from the 2009 National Survey on Drug Use and Health: Detailed Tables.


