PATIENT INFORMATION:
Sunburns: Prevention and Useful Information

Summer fun often involves getting outside and enjoying the sun. It is important to keep your skin healthy and protected from the sun, especially for children and those with fair skin.

Ultraviolet (UV) radiation can come from natural sunlight or tanning beds. UV rays are very damaging to the skin.

Avoid Sun Exposure
Prepare for being in the sun, especially if you are going to be outside during the middle of the day (10:00am-4:00pm) when the sun is highest in the sky. Don’t forget, sunburns can occur in the winter, too. Sunlight reflects off snow, and snow can wear off sunscreen. UV exposure increases at higher altitudes, so the risk of sunburn is increased for skiers and snowboarders.

Sunscreen is the best way to avoid sunburns. In addition, seeking shade and wearing a hat and clothes that shield skin from the sun are also good ways to avoid burns. Wear sunscreen on skin that isn’t covered, even in the shade because the sun’s rays can reflect off surfaces and cause UV damage. Apply sunscreen 15-30 minutes before exposure and reapply every 2 hours or after getting wet, sweaty, or toweling off. You can use the UV index to help predict the level of sun exposure on any given day (available online at: www.epa.gov/sunwise/uvindex.html). The index gives a rating from 0-11+.

⇒ 0 = very low risk of sun exposure.
⇒ 11+ = extreme risk of sun exposure

Use the index to decide what steps you should use to stay protected from the sun.

Why Bother?
There are other reasons to be “sun-smart” besides avoiding painful sunburns.

The amount of sun exposure and the number of sunburns a person gets greatly increases their risk of getting skin cancer. People also get wrinkles earlier when their skin is damaged from the sun.

Some people burn much easier than others. A substance in the skin called “melanin” gives skin its color and protects people from sunburns. People with less melanin have a lighter skin tone and less protection from UV rays and sunburns.

One in five Americans will develop skin cancer in the course of their lifetime. Melanoma is a type of skin cancer that can be deadly. On average, one person dies every hour from melanoma. Roughly 86% of melanoma cancers are related to UV rays from the sun.

Choosing a Sunscreen
Sunscreens work against two types of UV rays: UVA and UVB. The sunscreen you use should work against both types of UV rays. Use sunscreen with an SPF (Sun Protection Factor) of at least 15 or 30 if you burn easily and don’t tan.

An SPF of 15 allows a person to stay in the sun 15 times longer before getting a sunburn compared to a person with no sunscreen.

UVB is most likely to

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cause sunburns, skin cancer, and wrinkles. UVA gets deeper into the skin than UVB does. Although UVA doesn’t directly cause sunburns, it adds to the damage caused by UVB. Check to see that the sunscreen you’re using has products that protect your skin from both kinds of UV rays.

There are two types of sunscreen: physical blockers and chemical absorbers. Physical blockers scatter or reflect UV rays. Chemical absorbers absorb UV rays.

**Physical Blockers:**
- Titanium dioxide
- Zinc oxide

Physical blockers work on both types of UV rays, but they are less popular than chemical absorbers because they don’t “rub in” and remain visible.

**Chemical Absorbers:**
- Aminobenzoic acid derivatives (partially covers UVB):
  - para-aminobenzoic acid (PABA)
  - glyceryl PABA
  - Padimate
- Benzophenones (UVB & UVA):
  - Dioxbenzone
  - Oxybenzone
  - Sulisobenzone
- Cinnamates
  - Octocrylene (UVB & UVA)
  - Octinoxate (UVB)
  - Cinoxate (UVB)
- Salicylates (UVB)
  - Homosalate
  - Octisalate
  - Trolamine salicylate
- Others:
  - Avozobenzone (UVA)
  - Ecamsule (UVB & UVA)
  - Ensulizole (UVB & UVA)
  - Bemotrizinol (UVB & UVA)

◊ Bisocitrizole (UVB & UVA)

**Medicines that Increase Sunburn Risk**

Medications called “photosensitizing agents” greatly increase a person’s risk of getting sunburns or a rash from the sun. It is especially important to use sun protection when using the following medications:
- Diuretics or “water pills” such as hydrochlorothiazide
- Some antibiotics such as tetracycline, Bactrim® or Septra®, and ciprofloxacin
- Accutane®
- Acitretin
- Certain medicines for diabetes such as glipizide
- Prochlorperazine
- Chlorpromazine
- Drugs for mood disorders such as fluphenazine
- Amiodarone
- Diltaizem
- Quinine
- Quinidine
- Hydroxychloroquine
- Enalapril
- Dapsone

**This list is not all-inclusive.** Check with your doctor or pharmacist to see if your medications increase your risk for sunburns.

**By Brook Gould, PharmD Candidate**

**REFERENCES:**


2. Tewari A, Young AR. Patient information: sunburn prevention (beyond the basics). In: UpToDate, Post TW (Ed), UpToDate, Waltham, MA. (Accessed July 27, 2015.)

3. Tewari A, Young AR. Patient information: sunburn (beyond the basics). In: UpToDate, Post TW (Ed), UpToDate, Waltham, MA. (Accessed July 27, 2015.)


Cangrelor was approved by the FDA on June 22, 2015 after approximately ten years in the pipeline.\textsuperscript{1,2} Cangrelor is an intravenous antiplatelet drug that prevents the formation of blood clots.\textsuperscript{1,4} It is indicated for adults undergoing percutaneous coronary intervention (PCI) who are not currently being treated with a P2Y\textsubscript{12} inhibitor or glycoprotein IIb/IIIa inhibitor.\textsuperscript{1,4}

Cangrelor is a selective, reversible, rapid acting P2Y\textsubscript{12} platelet receptor inhibitor.\textsuperscript{2,5} It blocks ADP-induced activation and aggregation of platelets, preventing further signaling and platelet activation. Cangrelor reaches its max response 2 minutes after injection.\textsuperscript{2,3} It is 97-98% protein bound and is metabolized completely within the circulatory system. Cangrelor is excreted renally (~58%) and in the feces (~35%). The half-life of cangrelor is approximately 3-6 minutes.\textsuperscript{2,5}

**Cangrelor vials must be reconstituted prior to use.** The antiplatelet effects of cangrelor last for up to 1 hour after discontinuing infusion. No dose adjustments are necessary for use in elderly patients or those with renal or hepatic impairment.\textsuperscript{2,4}

**Dosing for PCI:**
- 30 mcg/kg IV bolus prior to PCI
- Followed by IV infusion 4 mcg/kg/min for 2 hours or duration of PCI (whichever is longer)

**Transitioning to oral medications:**
- Clopidogrel: 600 mg PO immediately following discontinuation of cangrelor infusion
- Prasugrel: 60 mg PO immediately following discontinuation of cangrelor infusion
- Ticagrelor: 180 mg PO initiated during infusion or immediately after discontinuation

Cangrelor is contraindicated in patients with active bleeding or who have hypersensitivity reactions to cangrelor or any component of the product.\textsuperscript{2,4} Hypersensitivity reactions to cangrelor include anaphylaxis, angioedema, and bronchospasm. Worsening renal function has occurred in patients with severe renal impairment. Thienopyridines, such as clopidogrel and prasugrel, interact with cangrelor and should be administered after the cangrelor infusion is discontinued. Cangrelor is a pregnancy category C medication.\textsuperscript{2,4}

**Patients on cangrelor should be monitored for signs and symptoms of bleeding.** The absence of myocardial infarction (MI), repeat coronary revascularization, and stent thrombosis may indicate efficacy of cangrelor.\textsuperscript{3}

**CHAMPION-PHOENIX trial:**
The incidence of death, MI, stent thrombosis, and revascularization were reduced with cangrelor treatment when compared to clopidogrel in patients requiring PCI.\textsuperscript{5} Cangrelor was compared to clopidogrel 300 or 600 mg in 11,145 patients. Fewer patients treated with cangrelor experienced death or the cardiac endpoints at 48 hours compared to patients treated with clopidogrel (4.7% vs. 5.9%, p=0.005). At 30 days, MI and stent thrombosis were still reduced in the cangrelor group (MI: 3.8% vs. 4.7%; stent thrombosis: 0.8% vs. 1.4%). However, mortality rates were similar at 30 days. Severe bleeding and blood transfusion were similar between the treatments. This trial included the Universal definition of MI and stent thrombosis as part of the composite endpoint, unlike the previous CHAMPION trials, and was the only CHAMPION trial to have statistically significant results for the composite endpoint. The CHAMPION-PHOENIX study results are limited by the inclusion of only clopidogrel-naïve patients, so the results may not be generalizable to patients who have used clopidogrel in the past.\textsuperscript{5}

**BRIDGE trial:**
Cangrelor reduced platelet reactivity when used to bridge patients on antiplatelet therapy prior to CABG surgery.\textsuperscript{5,7} The 210 patients discontinued thienopyridine therapy 2-7 days before surgery and received either cangrelor infusion without a bolus or placebo for at least 48 hours. The study treatments were stopped 1-6 hours before surgery. Cangrelor infusion resulted in a 98% reduction in platelet reactivity, and placebo treatment only reduced platelet reactivity by 19% (p<0.001). The rates of major bleeding were similar between the two groups (11.8% vs. 10.4%, p=0.763). The rate of minor bleeding was higher in the cangrelor group, but this was not statistically significant. The results of this study may not apply to patients undergoing non-cardiac surgery. In addition, the study was not designed to evaluate the risk of ischemic events or to evaluate clinical outcomes of platelet inhibition, so the clinical effect of bridging with cangrelor is unknown.\textsuperscript{5,7}

Cangrelor is an antiplatelet agent which reduced MI and stent thrombosis when compared to clopidogrel. Since cangrelor is an intravenous agent, it may help overcome some of the limitations of oral P2Y\textsubscript{12} inhibitors and may be especially helpful for patients who are intubated, vomiting, or unable to swallow medications. Cangrelor also has a fast onset and offset which provides flexibility during PCI and in emergency situations. Further studies are necessary to assess efficacy compared to other thienopyridine agents and to establish cost-effectiveness.

**By Nicole Kamura, PharmD Candidate**

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Most spiders in the United States are quite harmless; however, there are three venomous spiders that pose a threat to the general population. The three venomous spiders are the black widow, brown recluse, and the hobo spider. Spiders are not normally aggressive, and usually only bite when trapped in clothing or when contacted unintentionally.

**Black Widow:**
The black widow spider is found throughout the United States. The most common areas are the southwest and western regions. Black widows can be identified by their signature red “hour glass” pattern on the abdomen. Black widows are found in areas where debris has accumulated such as wood piles, barns, attics, and garages. A bite from a black widow spider contains a toxin that damages the human nervous system. The bite can be identified by two puncture marks in the skin and requires immediate medical attention. The most common symptoms of a black widow bite are:

- Immediate pain, burning, and swelling at site of bite
- Headache and dizziness, sweating, nausea/vomiting
- Cramping in the stomach, chest, and shoulder region

**Brown Recluse:**
The brown recluse is found in the Midwest and southern states. It can be identified by the violin-shaped marking on its head and brown body. Brown recluses can be found in places that are dry and secluded like wood piles, rock piles, closets, or attics. Bites from a brown recluse usually cause a stinging sensation and a blister at the site. The blister will continue to expand and the spider venom can cause skin necrosis. The brown recluse will only bite a human if it is pressed or trapped against skin surfaces. The bite requires immediate medical attention. Symptoms of a brown recluse spider bite are:

- Pain, burning, itching at site of bite (may take up to several hours to days to develop)
- Area around the bite will turn deep blue or purple and will be surrounded by a white circle that looks similar to a bull’s eye
- A blister that turns black will develop at bite site
- Headache, body aches, rash, fever, nausea/vomiting

**Hobo Spider:**
The hobo spider is found across the northwest. The hobo spider is a large, brown spider with yellow markings on the abdomen area. The distinct funnel webs of the hobo spider can be found around house foundations, window wells, and brick walls and between storage boxes in garages or attics. In general, hobo spiders do not climb, but can move quickly on the ground and will only bite if trapped or provoked. The bite can develop into a slow healing wound which may go unnoticed for several days. Symptoms of a hobo spider bite are:

- Burning pain, blood vessel damage, and cyst at the bite site
- Headache, nausea/diarrhea, dry mouth, joint pain

**Immediate Treatment:**
These venomous spider bites require immediate medical treatment. It is very helpful to medical personnel if the spider is captured or killed and brought in with the patient for correct identification. This identification ensures the right antivenom will be administered. The following action should be taken if bitten by a venomous spider:

- Wash bite with soap and water
- Ice the bite area to prevent spread of venom and reduce swelling
- Elevate the bite area if possible
- Seek medical attention immediately

**By Mandy Major, PharmD Candidate**

**REFERENCES:**
Cangrelor References (from page 3)


WNV References (from page 6)

West Nile Virus

West Nile virus (WNV) exposure results in infection in humans. WNV is transmitted by mosquitoes from birds (white pelicans, crows, etc.).\(^1\)\(^-\)\(^5\) The virus infects the host and spreads throughout the CNS.\(^1\)\(^,\)\(^2\) Meningitis, encephalitis, paralysis, and ultimately, death may result from WNV infection.\(^1\)\(^,\)\(^2\)\(^,\)\(^6\)\(^-\)\(^8\) Infection rates in animals are most prevalent during the summer months (June-September) when temperatures and mosquito activity are at their highest.\(^1\)\(^,\)\(^2\)\(^,\)\(^6\)\(^-\)\(^8\) Certain human populations are at increased risk for WNV infection, such as those >75 years of age and people immunosuppressed due to organ transplant.\(^1\)\(^,\)\(^2\)\(^,\)\(^8\)

Currently, there are no available treatments for human infection with WNV other than supportive therapy (analgesics, anti-emetics, fluid replacement, and ventilation). Disease surveillance and monitoring, pest management, and personal protective measures remain the strategies of choice for WNV control.\(^1\)\(^,\)\(^2\)\(^,\)\(^8\)

WNV was first discovered in humans in Uganda in 1937. The first outbreak of WNV occurred in New York City in 1999 and resulted in 59 cases of neuroencephalitis.\(^1\)\(^,\)\(^2\) From 1999-2014, WNV infected more than 40,000 people—18,800 with encephalitis—and caused 1641 deaths in North America. In 2002-2003, the largest outbreak of WNV neuroencephalitis infected 5812 individuals nationwide.\(^6\)\(^-\)\(^7\) The most recent outbreak in 2013 infected 2469 individuals with WNV.\(^6\)\(^-\)\(^8\)

Approximately 75-80% of WNV infections are asymptomatic.\(^1\)\(^,\)\(^2\)\(^,\)\(^9\) Symptoms present after a 2-14 day incubation period.\(^1\)\(^,\)\(^2\) Acute symptoms including fever, nausea/vomiting, lower back and peripheral pain, rash, and myalgia typically last 3-6 days. WNV can progress to the neuroinvasive form, which includes severe fever, seizures, muscle weakness, thinking abnormalities, and polio-like paralysis.\(^1\)\(^,\)\(^2\) WNV neuroinvasive disease can be up to 10% fatal in infected individuals.\(^1\)\(^,\)\(^2\)\(^,\)\(^8\)

Indications of WNV infiltration into geographic locations may be difficult to identify initially.\(^5\)\(^,\)\(^8\) Surveillance of potential risk areas and areas with high populations of migratory waterfowl help identify possible outbreaks and high risk areas for WNV infection. Large population corvid (crow) death surveillance was a primary method for indicating the possibility of WNV infiltration in western states.\(^5\)\(^,\)\(^8\) Certain species of birds may be better indicators of possible infection risk to human hosts (crows, white pelicans, barn sparrows).\(^3\)\(^,\)\(^5\) Stagnant water pools serve as optimum breeding grounds for mosquitoes which feed on birds in the same habitat.\(^4\)\(^,\)\(^5\)\(^,\)\(^8\)

Mitigation strategies for WNV management are determined by local governments. Draining of ponds and stagnant water sources are effective methods for controlling availability of breeding locations of mosquitoes, but migratory birds often use these sources of water for food and breeding themselves.\(^8\) Chemical larvicides and maturation inhibitors are also used to prevent the spread and growth of mosquitoes.\(^5\)\(^,\)\(^8\) Truck- and air-dispersed insecticides are often deployed during August and September to aid in controlling mosquitoes and WNV, but environmental and public health impact is a major concern.\(^8\)

Persons at increased risk should take appropriate measures to reduce exposure and WNV infection.\(^8\) Protective clothing (long-sleeve shirts, pants, closed-toed shoes) reduce exposed skin areas. Window screens reduce the risk for mosquitoes entering houses. Topical and environmental mosquito repellants that are recommended include DEET, picaridin, and oil of lemon eucalyptus.\(^8\)

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