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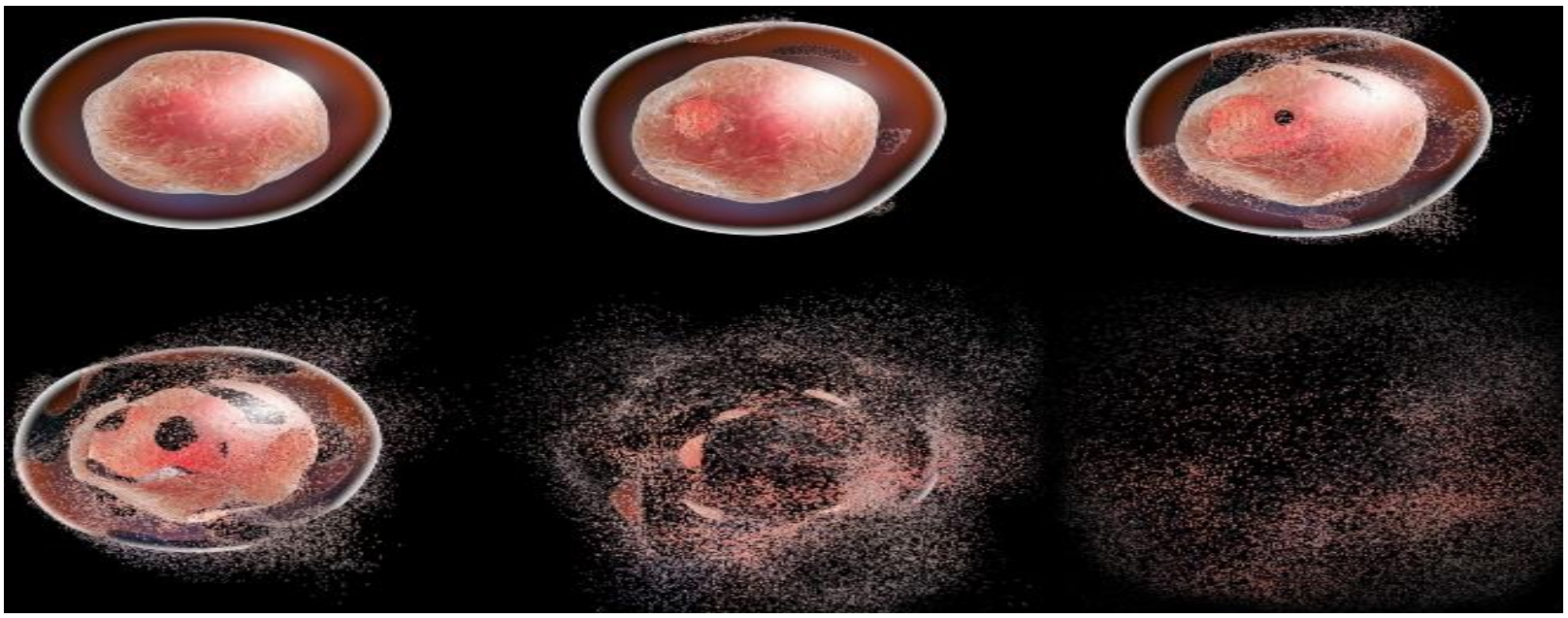
Platelet- Rich Plasma:

Asst. Prof. Dr. Bahir Abdul Razaq

Platelet-rich plasma (PRP) is a concentrate of platelet-rich plasma protein derived from whole blood, centrifuged to remove red blood cells. It has a greater concentration of growth factors than whole blood, and has been used to encourage a brisk healing response across several specialties, in particular plastic surgery, dentistry, orthopedics and dermatology. As a concentrated source of blood plasma and autologous conditioned plasma, PRP contains several different growth factors and other cytokines that can

Main indication in sports medicine and orthopedics are acute muscle strains, tendinopathy and muscle-fascial injuries and osteoarthritis. Main indications in dermatology for PRP are androgenic alopecia, wound healing, and skin rejuvenation. For preparation of PRP, various protocols are used, with an underlying principle of concentrating platelets to 3–5 times physiological levels, then injecting this concentrate in the tissue where healing is desired.



Necroptosis

Assist. Prof. Dr. Muthanna I. Al-Ezzi

As early as the mid 19th century, Virchow taught that necrosis is a recognizable form of cell death, and since then, pathologists have identified necrosis as a cause or consequence of disease. A century later, another form of cell death, apoptosis, was defined, and we now understand that this process is driven by a set of molecular mechanisms that "programs" the cell to die. It has often been assumed that necrosis is distinct from apoptosis; in part, because the former is not programmed by molecular events.

In recent years, however, it has become clear that in some settings, necrotic cell death can also be driven by defined molecular pathways. Here, we discuss one such process, a type of necrotic cell death called "necroptosis" and its role in disease. While some investigators have used this term to indicate any form of active necrosis, we will follow recent recommendations and use necroptosis to mean "necrotic cell death dependent on receptor-interacting protein kinase-3 (RIPK3)" (Figure).

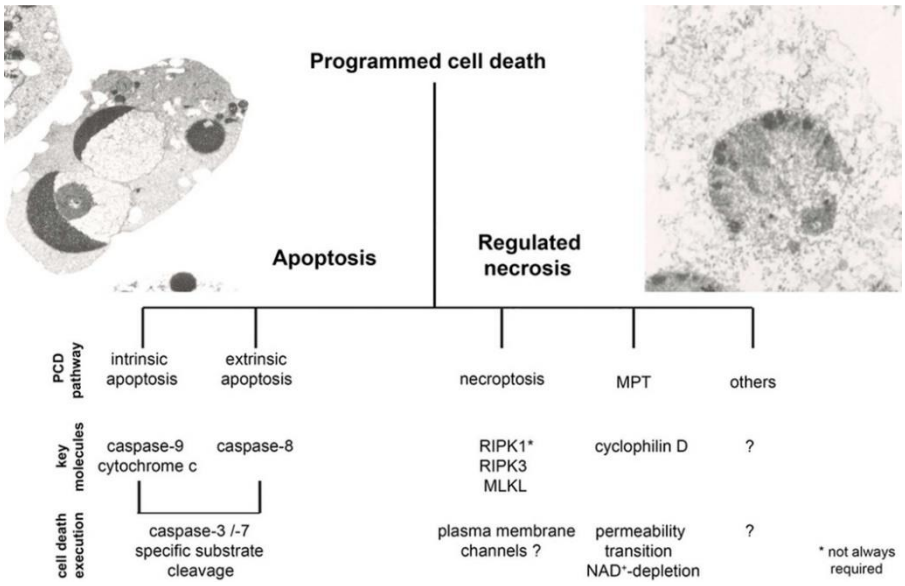


Figure: Programmed cell death is mediated by apoptosis or regulated necrosis

With our understanding of the molecular basis of necroptosis and other forms of regulated necrosis, and the availability of inhibitors, a neglected therapeutic option emerges: It is possible to therapeutically interfere with necrosis.

Necroptosis is well defined as a viral defense mechanism, allowing the cell to undergo "cellular suicide" in a caspase-independent fashion in the presence of viral caspase inhibitors. In addition to being a response to disease, necroptosis has also been characterized as a component of inflammatory diseases such as Crohn's disease, pancreatitis, and myocardial infarction.

Therapeutic strategies for the prevention of necroptotic diseases:
Theoretically, interference with necroptosis is possible at the levels of the receptor, RIPK1, RIPK3, MLKL, the assembly of the necrosome and undefined intermediate and downstream mechanisms that may ultimately lead to cellular swelling and plasma membrane rupture. With these targets only recently in hand, the major focus has been on Nec-1. Due to the outstanding specificity of Nec-1 to the RIP1 kinase, some authors have referred to the inhibition by Nec-1 as a definition of necroptosis. As the structural interaction of Nec-1 with the RIP1 kinase domain has been unraveled, novel interpretations of the inhibition of necroptosis by Nec-1 have emerged. RIPK1-deficient mice die perinatally, and it remains formally possible that RIPK1 acts as an inhibitor of necroptosis unless its kinase activity is engaged, and Nec-1 might stabilize RIPK1 in its inhibitory state.

How Scorpion Venom Could Yield New Cancer Treatment?

Asst. Prof. Dr. Yassir Mustafa Kamal

In the development of new drugs, taking something from nature and modifying it has been a successful tactic employed by medicinal chemists for years. Now, with the help of nanotechnology, researchers are turning once-discarded drug candidates into usable drugs.

An estimated 40% of clinically approved drugs fall into the category where either the natural compound itself or a modified version is the approved drug. These include statins (found in bacterial secretions) used to lower cholesterol, quinines (found in cinchona trees) as anti-malarials and paclitaxel (found in yew trees) as anti-cancer medication.

Many of these natural products are toxins produced by plants or animals as a form of defence. And scorpion venom has been gaining interest as a

source of new drugs. It contains a mixture of biological chemicals called peptides, some of which are known to trigger cell death by forming pores in biological membranes. Cell death can be useful if we are able to target, say, tumour cells to auto-destruct. These toxins can have very potent effects. For instance, one particular small peptide, known as TsAP-1, isolated from the Brazilian yellow scorpion (Tityus serrulatus), has both anti-microbial and anti-cancer properties.

However, harnessing this kind of power for clinical good has so far been challenging because these toxins kill both tumours and healthy cells. One method to control such toxicity is through using nanotechnology to build specially made drug-delivery vehicles. If successful, the toxic drug is released to kill only unwanted tissues in a body.

One such attempt has been made by Dipanjan Pan at the University of Illinois at Urbana-Champagne. In a study published in the journal Chemical Communications, scientists claim to have created spherical capsules to trap scorpion venom toxin TsAP-1. This encapsulated toxin, named NanoVenin, increases the drugs effectiveness at killing breast cancer cells by ten times.

This is an interesting development for two reasons. Firstly, the venom toxin in its natural form could not be used due to the lack of specificity and, secondly, the incorporation of the venom toxin in the nanoparticle caused a large increase in the drug's potency, making it more clinically useful.

This form of the drug works on breast cancer cells, but it is not disease-specific yet. Researchers can modify its outer shell by, for example, attaching proteins that can make it selective towards certain types of cancers. It may also be possible to coat the nanoparticle in a biodegradable layer so as to trap its toxicity until it reaches the diseased area, where the layer degrades to reveal the toxin.

Such precise delivery can work on a "lock-and-key system" of highly precise biological structures. For instance, different types of cancer cells have characteristic secretions or outer proteins – the biodegradable layer of the drug can be built to recognise these specific secretions or proteins and then trigger the degradation process, allowing precise delivery of the drug.

Often effective drugs have been discovered but not commercialised due to delivery issues. Yet the latest developments in nanotechnology illustrate how once discarded drugs sourced from natural compounds can be brought off the shelf to fight disease.

The Danger of Bad Office Chair

Lec. Dr. Raghad Abdul Mahdi

It is only logical that the human body was not created to sit still for many hours during the day. Research has proven that how much we sit has serious consequences for our weight, our posture, and even on our lifespan. Human beings did not start out with the lifestyle that most people in the western world now have. We lived on our feet, and not on our bottoms. Now that we have desk jobs, televisions, transport, and computers, most humans spend more time sitting on our bottoms the we spend sleeping. Our bodies were not meant to sit that much. In fact, sitting for more than 6 hours a day makes you 40% likelier to die within 50 years than someone who sits less than 3 hours a day. Even if you exercise regularly.

As soon as you sit down:

- Electrical activity in your body shuts off. • Calorie burn drops to 1 calorie per minute.
- Enzymes that help break down fat drop to 90%

After 2 hours of sitting:

- Good cholesterol drops 20% After a day of sitting:
- Insulin effectiveness drops 24% and risk of diabetes rises.

Some other interesting facts about sitting:

- People with sitting jobs have twice the rate of cardiovascular diseases as people with standing jobs.
- Sitting makes you gain weight.

Obese people sit for 2.5 more hours per day than thin people. Did you know that sitting expends almost no energy?

People who sit over 3 hours a day watching TV are 64% likely to die from heart disease. Each extra hour of watching TV = 11% higher death risk. The human body was simply not built for sitting over long periods of time. A hundred years ago we were toiling out in the fields, working in factories and doing much more manual labour. Obesity was almost non-existent. Think about that next time you sit in a chair that doesn't promote steady gentle movement. It's time to change the way that you sit

How to Sit Smarter

The solution seems to be less sitting and more moving overall. You might start by simply standing rather than sitting whenever you have the chance or think about ways to walk while you work. For example:

Stand while talking on the phone or eating lunch.
If you work at a desk for long periods of time, try a standing desk — or improvise with a high table or counter.
Walk laps with your colleagues rather than gathering in a conference room for meetings.
Position your work surface above a treadmill — with a computer screen and keyboard on a stand or a specialized treadmill-ready vertical desk — so that you can be in motion throughout the day.
The impact of movement — even leisurely movement — can be profound. For starters, you'll burn more calories. This might lead to weight loss and increased energy. Even better, the muscle activity needed for standing and other movement seems to trigger important processes related to the breakdown of fats and sugars within the body. When you sit, these processes stall — and your health risks increase. When you're standing or actively moving, you kick the processes back into action.

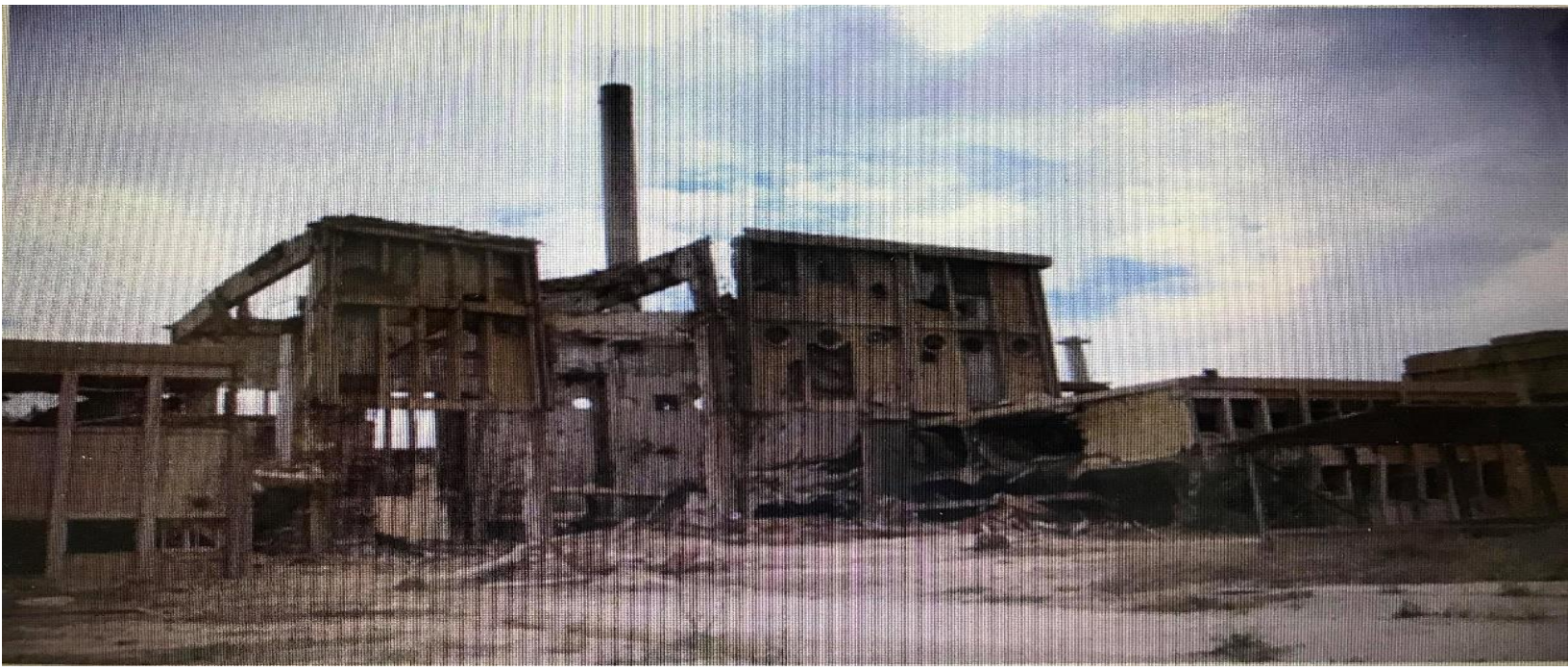


Figure: Destroyed nuclear reactor Tammuz-2 at Al-twaitha nuclear research site.

Environmental Pollution Associated with Iraqi Wars

Lec. Dr. Atheer S. Alsabah

Several wars and a 13-year embargo as well as several years of civil war with the recent war on terrorism have cumulatively damaged Iraq's land, air, water, and health infrastructure. The sand particles in Iraq contain toxic substances, which dates back to the pollution caused by military actions that disassemble the desert sands and turn it into light dust.

This dust reaches cities as dust storms that effect most Iraqi cities. The presence of depleted uranium (DU) in the Iraqi food chain is documented by measuring the uranium in animals organs in different Iraqi cities with the highest concentration in the south of Iraq.

One of the major sites of pollution in Iraq is the Al-twaitha nuclear research site. The nuclear research reactors were destroyed in the 1991 Gulf War. Barrels containing radioactive materials and sources were stolen from the site in the 2003 war. This resulted in considerable radioactive pollution at the site and in its surrounding areas. Soil sample have been found to be contaminated by Cs-137and Co-60.

Cancer and birth defects are most associated with the environmental pollution caused by the conflicts. From studying the Iraqi scientific publications, we can conclude that Basrah, Baghdad, Faluja, Mosul and Thi-Qar are the most effected cities in Iraq. This concludes that the presence of a heavily contaminated environment with war related pollutants in most of the Iraqi cities needs much attention and huge effort to reduce the related health problems.

Some Food Ingredients to be Avoided in Processed Food

Lec. Ruaa Abbas Nasir

The term processed food includes any food that has been purposely changed in some way prior to consumption. It includes food that has been cooked, canned, frozen, packaged or changed in nutritional composition with fortifying, preserving or preparing in different ways. Some of the food ingredients to be avoided in processed food are:

Artificial sweeteners: Susceptible populations for the potential deleteriouseffects of artificial sweeteners include diabetics, children,pregnant women, women of childbearing age, and breastfeedingmothers. Acesulfame-K, aspartame, sorbitol, saccharin, and sucralose are commonly used artificial sweeteners. Beverages have been identified as a major source of artificial sweeteners in the diet. Acesulfame-K contains the chemical methylene chloride, a known carcinogen.

Chronic exposure to aspartame has been reported to cause headaches, blurred vision, brain tumors, memory loss, hyperactivity, neurological problems, and behavioral disturbances. Animal studies have shown that chronic exposure to Saccharin resulted in Cancer in offspring ofbreast-fed animals, lowbirth weight, bladder cancer, and hepatotoxicity

Monosodium glutamate (MSG)is used as a flavor enhancer. It can produce a unique taste known as taste (umami) that improves the quality of food. Empirical studies have shown that prolonged consumption of MSG causes a number of toxic effects, referred to as the Chinese restaurant syndrome. Symptoms of this syndrome are sweating, nausea, headache, chest tightness and burning at the back of the neck. Moreover, the long-term intake of MSG result in an excess of appetite, obesity, asthma, poor memory, and damage to nerve cells, at the same time, researches has shown that MSG can cause brain damage in infants.

Preservatives: Butylatedhydroxyanisole (BHA) and butylatedhydrozyttoluene (BHT) are fat preservatives, and have been linked to cancerous tumor growth. Nitrate and nitrite are used as additives in processed meats to improve food quality and protect against microbial contamination. They are sources of N-nitrosocompounds (NOCs) which are known carcinogens. One of the proposed mechanisms whereby processed meat can increase colorectal cancer is the formation of NOCs.

Artificial colors:food dyes, synthesized from coal tar and now petroleum, have long been controversial. Red #40 has been linked to cancer in some studies, and it can also cause hyperactivity in children. It is banned in some European countries. Blue #2 is used in candy, and it can cause brain tumors.

7 Signs and Symptoms of Magnesium Deficiency

Asst. Lec. Deleen Abdul wahab

Magnesium deficiency, also known as hypomagnesemia, is an often-overlooked health problem. While less than 2% of Americans have been estimated to experience magnesium deficiency, one study suggests that up to 75% are not meeting their recommended intake. In some cases, deficiency may be underdiagnosed since the obvious signs commonly don't appear until your levels become severely low.

The causes of magnesium deficiency vary. They range from inadequate dietary intake to loss of magnesium from the body.

Health problems associated with magnesium loss include diabetes, poor absorption, chronic diarrhea, celiac disease and hungry bone syndrome. People with alcoholism are also at an increased risk (3, 4).

This article lists 7 symptoms of magnesium deficiency.

1. Muscle Twitches and Cramps

Common signs of magnesium deficiency include muscle twitches, tremors and cramps. However, supplements are unlikely to reduce these

symptoms in people who aren't deficient.

2. **Mental Disorders**

Magnesium deficiency may cause mental numbness, lack of emotion, delirium and even coma. Scientists have suggested that deficiency may also cause anxiety, but no strong evidence supports this idea.

3. **Osteoporosis**

Deficiency of this metal may increase the risk of osteoporosis and bone fractures, though this risk is influenced by many factors.

4. **Fatigue and Muscle Weakness**

Magnesium deficiency may cause fatigue or muscle weakness. However, these are not specific signs of a deficiency unless they are accompanied by other symptoms.

5. **High Blood Pressure**

Evidence suggests magnesium deficiency may raise blood pressure. Additionally, supplements may benefit people with high blood pressure.

6. **Asthma**

Magnesium deficiency has been associated with severe asthma. However, its role in the development of asthma is not entirely understood.

7. **Irregular Heartbeat**

One of the symptoms of magnesium deficiency is heart arrhythmia, or irregular heartbeat, which may increase the risk of more serious complications, such as a stroke or heart failure.

Seeds, nuts, cocoa, beans and whole grains are great sources of magnesium. For optimal health, make sure to eat some magnesium-rich foods every day. These foods are also high in other healthy nutrients. Including them in your diet not only lowers your risk of magnesium deficiency, but it also promotes your overall health.

The symptoms of magnesium deficiency are usually subtle unless your levels become severely low. Deficiency may cause fatigue, muscle cramps, mental problems, irregular heartbeat and osteoporosis. If you believe you may have a magnesium deficiency, your suspicions can be confirmed with a simple blood test. You should speak with your doctor to rule out other possible health problems. Whatever the outcome, try to regularly eat plenty of magnesium-rich whole foods

Painkillers in Pregnancy may Affect Fertility of Offspring

Asst. Lec. Wrood Salim

Brief exposure to over-the-counter analgesics during the first two trimesters of pregnancy may induce epigenetic changes in fetal germ cells that could reduce fertility for future generations.

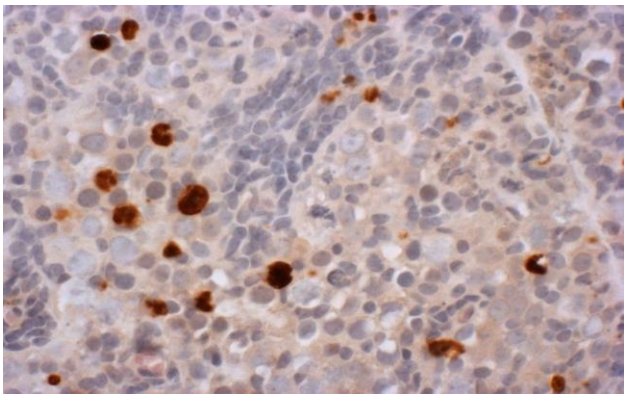
The in vitro and in vivo study of acetaminophen (paracetamol, *Tylenol*, Johnson & Johnson) and ibuprofen (*Advil*, Pfizer) exposure in human fetal testis and ovarian tissue showed, among other observations, that germ cell number was significantly reduced following exposure to either analgesic.

In tissue samples taken from fetuses during the first trimester and exposed to

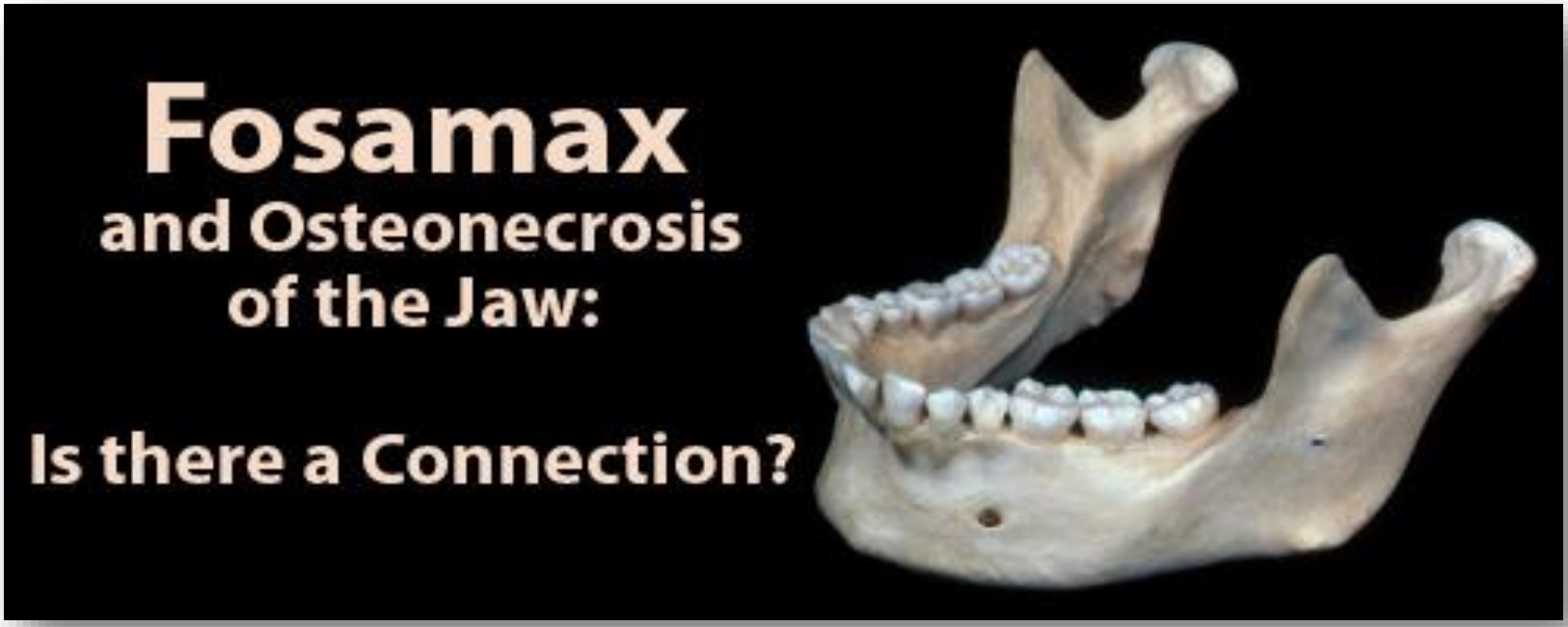
therapeutically relevant doses of acetaminophen for 7 days, the number of germ cell gonocyteswas reduced by 28% in testes and by 43% in ovaries. Similarly, exposure to therapeutic levels of ibuprofen resulted in a 22% reduction in gonocyte number in human fetal testes and a 49% reduction in human fetal ovaries.

And after xenografting human testis tissue from a fetus in the second trimester of development into pregnant mice, the researchers found that exposure to acetaminophen or ibuprofen for 7 days significantly reduced total germ cell number compared with control mice treated with vehicle (43% and 54%, respectively).

Although acetaminophen is the most commonly used over-the-counter pain medication worldwide, followed by ibuprofen, there is lack of consensus about its mechanism of action. More recently, concern has been growing about possible adverse effects on reproductive health of these analgesics.



This is ovarian tissue that has been exposed to ibuprofen for seven days. The big brown cells are dying germ cells and the smaller brown cells are also dying.



Oral Bisphosphonate Related Osteonecrosis of the Jaw

Asst. Lec. Amani Anees Abbody

Oral bisphosphonates are the most commonly prescribed drugs for the treatment of osteoporosis. However, there are several adverse effects associated with oral bisphosphonates including the bisphosphonate related osteonecrosis of the jaw (BRONJ). Reported incidences for BRONJ in oral bisphosphonate users have increased in time. Several risk factors such as dental surgery, therapy duration, and concomitant steroid usage have been linked to BRONJ. Preventative measures are of great importance for the patients at high risk.

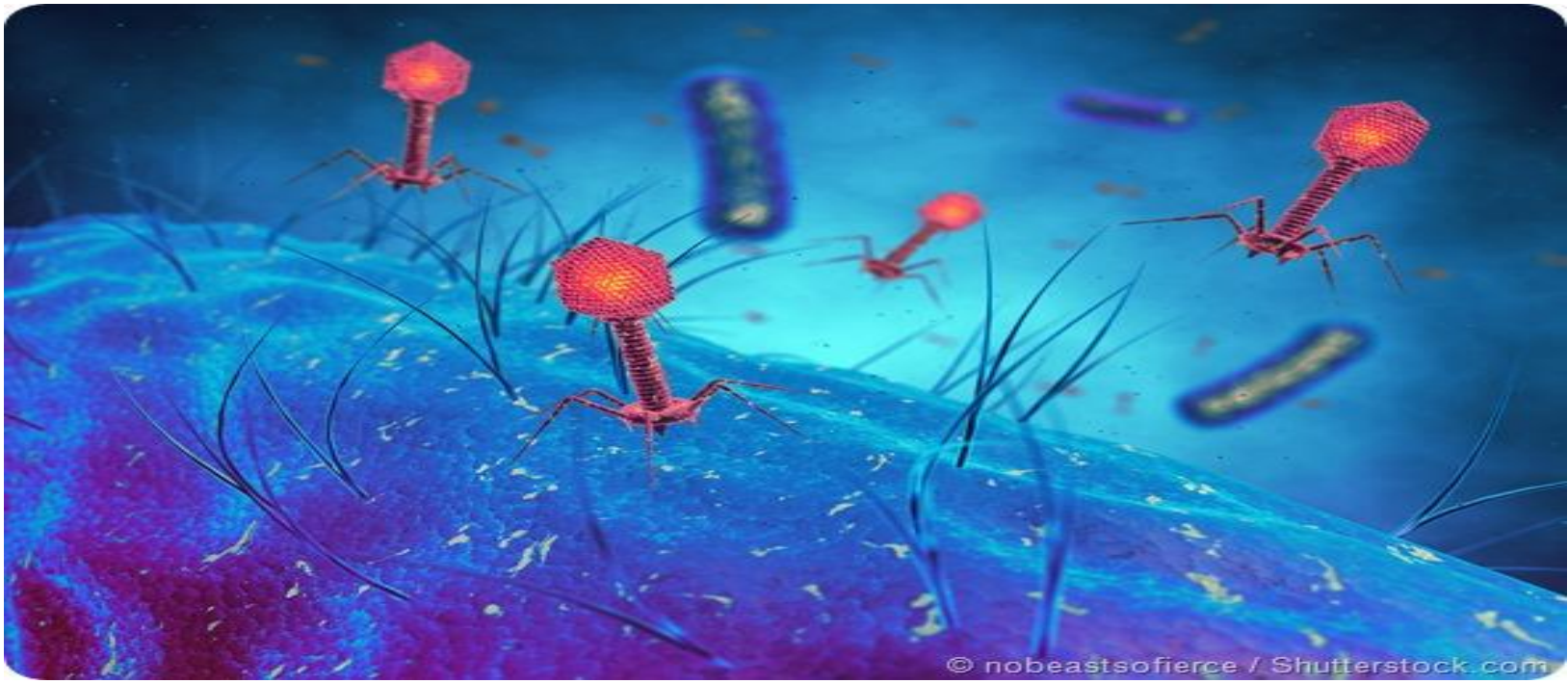
However, patients who are not receiving regular dental care or with concomitant risk factors such as cancer, corticosteroids, chemotherapy, and poor oral hygiene should undergo

a broad oral examination by a dentist either prior to or following the initiation of bisphosphonates. Stopping smoking and limiting the alcohol intake are also recommended. Patient education in terms of the symptoms and initial signs of BRONJ is essential. If diagnosed timely, outcomes of BRONJ treatments are favorable.

Therefore, detailed regular intraoral examinations are crucial for detecting the early stages of BRONJ lesions.

In cases on oral bisphosphonates and with concomitant risk factors, avoiding invasive dental surgery when possible is also recommended.

Eventually, it is of great importance for physicians dealing with osteoporosis such as physiatrists, endocrinologists, rheumatologists to be aware of the potential risk of developing BRONJ in patients on oral bisphosphonates and to work in accordance with dentists.



Bacteriophage: A Solution to our Antibiotics Problem?

Asst. Lec. Zakariya A. Mahdi

Bacteriophages or "phage" are viruses that invade bacterial cells and, in the case of lytic phages, disrupt bacterial metabolism and cause the bacterium to lyse [destruct]. Phage Therapy is the therapeutic use of lytic bacteriophages to treat pathogenic bacterial infections.

Bacterial Host Specificity

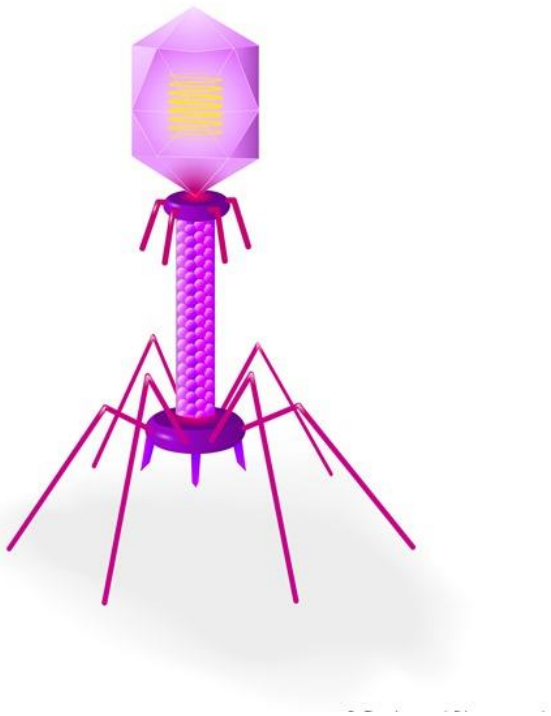
The bacterial host range of phage is generally narrower than that found in the antibiotics that have been selected for clinical applications. Most phage are specific for one species of bacteria and many are only able to lyse specific strains within a species. This limited host range can be advantageous, in principle, as phage therapy results in less harm to the normal body flora and ecology than commonly used antibiotics, which often disrupt the normal gastrointestinal flora and result in opportunistic secondary infections by

organisms such as Clostridium difficile. The potential clinical disadvantages associated with the narrow host range of most phage strains is addressed through the development of a large collection of well-characterized phage for a broad range of pathogens, and methods to rapidly determine which of the phage strains in the collection will be effective for any given infection.

Advantages over Antibiotics

Phage therapy can be very effective in certain conditions and has some unique advantages over antibiotics. Bacteria also develop resistance to phages, but it is incomparably easier to develop new phage than new antibiotic. A few weeks versus years are needed to obtain new phage for new strain of resistant bacteria. As bacteria evolve resistance, the relevant phages naturally evolve alongside. When super bacterium appears, the super phage already attacks it. We just need to derive it from the same environment. Phages have special advantage for localized use, because they penetrate deeper as long as the infection is present, rather than decrease rapidly in concentration below the surface like antibiotics. The phages stop reproducing once as the specific bacteria they target are destroyed. Phages do not develop secondary resistance, which is quite often in antibiotics. With the increasing incidence of antibiotic resistant bacteria and a deficit in the development of new classes of antibiotics to counteract them, there is a need to apply phages in a range of infections.

Lytic phages are similar to antibiotics in that they have remarkable antibacterial activity. However, therapeutic phages have some advantages over antibiotics, and phages have been reported to be more effective than antibiotics in treating certain infections especially drug-resistant and difficult to treat infections that do not respond to conventional antibiotics in humans and experimentally infected animals.



Rivaroxaban (Xarelto®)

Pharmacist Mina Ali

Venous thromboembolism, which includes deep-vein thrombosis and pulmonary embolism, is the third most common cause of vascular death after myocardial infarction and stroke. The mainstay of treatment is anticoagulation, and in patients without active cancer, guidelines suggest the use of direct oral anticoagulant agents such as rivaroxaban over vitamin K antagonists such as warfarin. Anticoagulation therapy is administered for 3 months or longer

Rivaroxaban inhibits both free Factor Xa and Factor Xa bound in the prothrombinase complex. It is a highly selective direct Factor Xa inhibitor with oral bioavailability and rapid onset of action at a dose of 20 mg once daily.

Rivaroxaban is effective for stroke prevention in patients with atrial fibrillation and for the treatment of venous thromboembolism after an initial 21-day course of higher-dose therapy. At a dose of 10 mg once daily, rivaroxaban provides effective thromboprophylaxis after elective hip or knee arthroplasty. They compared the efficacy and safety of these two doses of rivaroxaban with those of aspirin in patients with venous thromboembolism who had completed 6 to 12 months of anticoagulation therapy.

Among patients with venous thromboembolism, the risk of a recurrent event was significantly lower with rivaroxaban at either a treatment dose (20 mg) or a prophylactic dose (10 mg) than with aspirin, without a significant increase in bleeding rates.

How Your Genes Influence What Medicines are Right for You?

Pharmacist Dina Sabah

You have a headache. Do you reach for Tylenol or Advil? Most people have a preference because they have learned over time that one works better than the other at relieving their pain. This type of variability from person to person is true for nearly every medication, whether it requires a prescription or can be purchased over the counter.

Pharmacists, physicians and researchers have tried for decades to understand why the same medication, at the same dose, can work well for some people but not for others, or why some people need higher or lower doses of the same drug, or why some people have side effects, while others do not. Many factors contribute to these differences in how people respond to the same medication, including age, other medications they may be taking, kidney function and cigarette smoking, to name a few. But it's become increasingly clear that genetics can also be an important factor.

Genes influence how well drugs work

Understanding how these genetic differences work means that physicians can take a more personalized approach to selecting the right medication and dosage for each individual. This is called pharmacogenetics, and pharmacogenetic tests to guide use of

certain medications are becoming increasingly common.

Where does pharmacogenetics go from here?

Currently over 100 drugs have pharmacogenetic information in their product label from the Food and Drug Administration. Over time, more and more medications will likely be added to the list of those for which genetic data may allow for more personalized treatment decisions and the way genetic information is obtained will change in the coming years.

However, in the future it is expected that an individual's entire genome (all three billion letters of their genetic code) will be defined and stored for use throughout their lifetime. When that happens, using genetic information to inform decisions about the right drug and the right dose will likely involve computerized approaches that marry the genetic data with knowledge about drugs and genes, to lead to a personalized treatment recommendation. We are probably at least a decade or more from this reality.

In the meantime, the number of drugs whose use can be guided by genetics and the number of people who benefit are expected to grow.