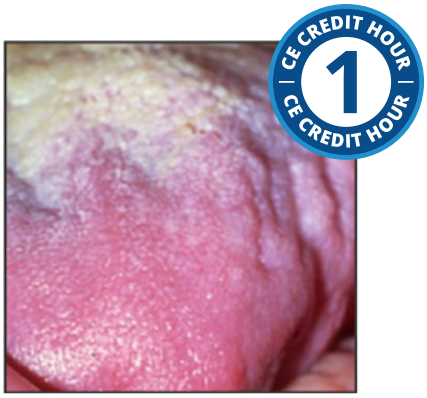
Biological Effects of Radiation



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- Dr. Geha reports no conflicts of interest associated with this course. He has no relevant financial relationships to disclose.

Introduction – Biological Effects of Radiation

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Course Contents

Overview

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Syllabus: Dental Radiology

The information in this 10-module syllabus is intended (1) to meet elements of initial educational/training requirements for Dental Students, Dental Hygiene Students, and Dental Assistant Students related to dental radiography; (2) to provide a framework for an in-service training program in oral healthcare settings to meet annual educational/training requirements as mandated by federal, state, local and professional organizations; and (3) to serve as a resource for oral healthcare personnel wishing to review evidence-based information on specific topics related to dental radiography. A PDF is available for each module that may serve as a convenient resource. *Learn More*

Learning Objectives

Upon completion of this course, the dental professional should be able to:

- Explain the difference between nonionizing and ionizing radiation.
- Discuss the mechanisms of free radical generation.
- Discuss potential cell damage related to direct and indirect ionization.
- Discuss, in general terms, the effect of free radicals on cellular proteins as a function of the various phases of the cell cycle.
- Discuss the effect of radiation dose on cell dynamics.
- Discuss the effects of high-dose ionizing radiation on tumor cells.
- Discuss the deterministic effects (short-term and long-term) of high dose radiation normal tissues in the head and neck.
- Discuss stochastic effects and mutations associated with ionizing radiation.

Introduction

Radiation biology examines how radiation affects biological systems. When radiating energy interacts with living cells, energy transfer results in either excitation or ionization. Low-energy nonionizing radiation, such as ultraviolet radiation, microwaves, and extra-low-frequency electromagnetic radiation, induces molecular excitation. This excitation happens when an electron shifts its energy level without leaving the atom's orbit.[1-3]

Ionizing radiation has enough energy to remove an electron from an atom's orbit, creating an ion pair (the ejected electron and the remaining atom), thereby causing ionization.[1,4,5] Both particulate radiation and electromagnetic radiation can lead to ionization. Particulate radiation consists of streams of atomic or subatomic particles, such as α-particles and neutrons, generated by nuclear disintegration, while high-energy electromagnetic radiation includes δ-rays and x-rays (photons).[1,5-8]

Radioactive substances emit gamma-rays. X-ray photons are man-made. They are produced by x-ray units when fast-moving electrons interact with tungsten atoms.[9] When photons with

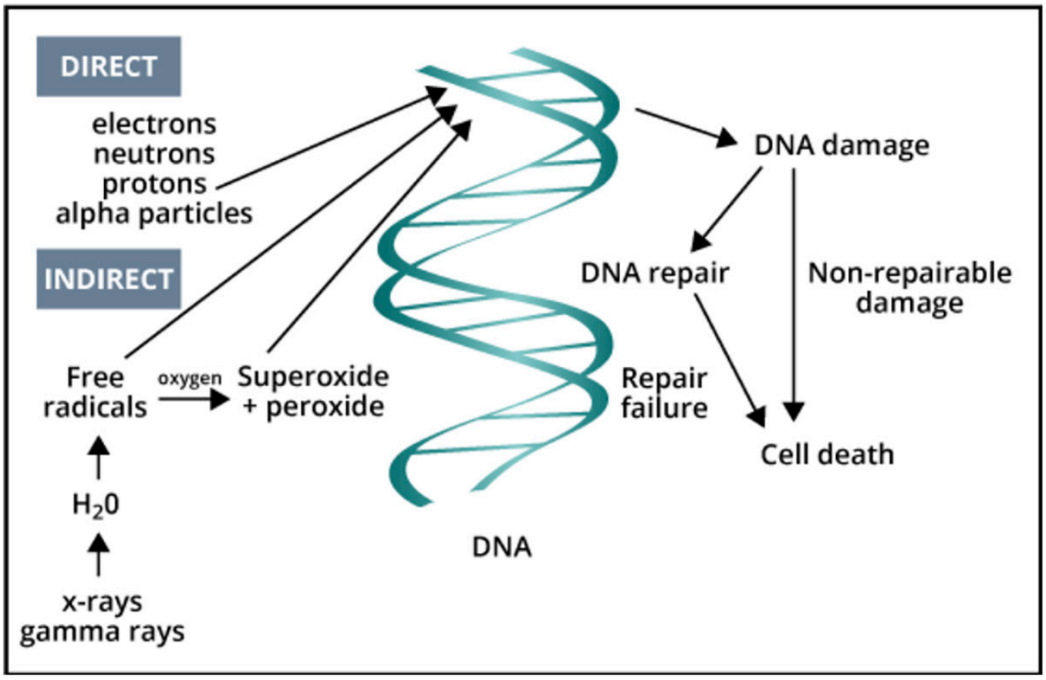

Figure 1. Cell damage can be caused by direct or indirect ionization.

sufficient energy liberate orbital electrons from atoms and their corresponding molecules, the creation of unpaired electrons results in the formation of highly reactive free radicals. Free radicals in a biological system can damage cellular proteins by altering their chemical structure.[1,5-8,10,11]

Direct versus Indirect Ionization

Cell damage can result from either direct or indirect ionization (Figure 1). Direct ionization takes place when charged particles, such as electrons with sufficient kinetic energy, collide with cellular atoms or molecules, generating free radicals.[4,6,10,11] This process is termed "direct" because the interaction happens immediately between the particle and a cellular component, without an intermediate step. The charged particle may continue to interact with other molecules until it has expended all its kinetic energy.

Indirect ionization occurs when non-charged particles, e.g., photons, interact with cellular water. The energy absorbed by an H_2O molecule will form ion pairs and reactive oxygen metabolites such as hydroxyl radicals.[4,6,11] These free radicals interact with cellular atoms and molecules, damaging cellular proteins, and may form additional free radicals.[2,4,6,10,11] The process is indirect because of the intermediate step of H2O-based free radical formation.

Free Radicals

Free radicals exhibit a high degree of chemical reactivity.[4,10] When they interact with cellular macromolecules, such as proteins, and modify their chemical structures, they can cause either repairable or irreversible damage, leading to significant downstream effects like altered cell function or cell death.[5,6,8] The impact of free radicals on deoxyribonucleic acid (DNA), which carries the genetic code, is particularly critical and varies across different phases of the cell cycle.[2,4,6,8,11,12]

Normal cells progress through five physiological phases: G0, G1, S, G2, and M (Figure 2).[13] The G0 phase represents the latent or resting state. Cells recruited from G0 transition into the G1 phase, the first active stage of the reproductive cycle. During G1, cells synthesize ribonucleic acid (RNA), enzymes, and proteins in preparation for subsequent phases of the reproductive cycle.

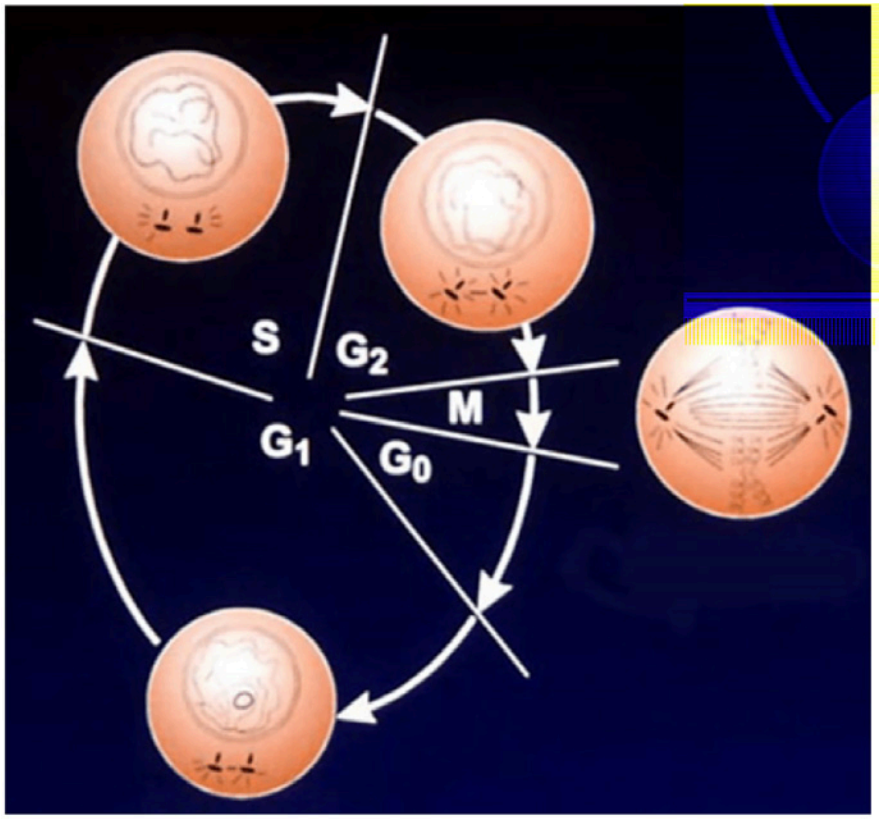

Figure 2. Five physiological phases of the cell cycle: G_0, G_1, S, G_2, and M.

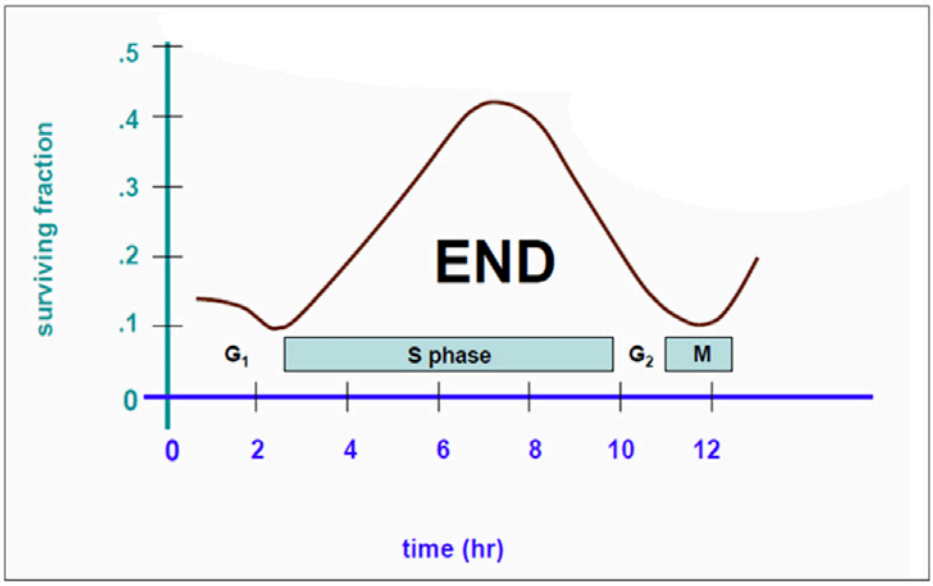

Figure 3. Cells are most radiosensitive in the G_1, G_2, and M phases, respectively; and most radioresistant in the S phase.

The S phase follows the G1 phase. The predominant event in the S phase is the synthesis of DNA. At the end of the S phase, the cells contain twice the original amount of DNA. The G2 phase follows the S phase. During the G2 phase, the mitotic spindle is created, which is essential for cell divisionIn the M or mitotic phase, cell division occurs. Cells are most radiosensitive in the G1, G2, and M phases, respectively. During the S phase, the cells are the most radioresistant. (Figure 3).[8,11]

There is a wide variation in radiosensitivity among different cell types. For example, rapidly dividing cells or cells with a potential for rapid division are more radiosensitive than those that do not divide. In addition, undifferentiated cells are more radiosensitive than highly specialized cells. Finally, within the same cell families, the immature forms rapidly dividing are more radiosensitive than the mature cells that have specialized in function and have ceased to divide.[6,8,10]

In summary, cell radiosensitivity is directly proportional to the rate of cell division and inversely proportional to the degree of cell differentiation, i.e., actively dividing cells or those not fully mature are most at risk from radiation. Highly sensitive cells include germ cells, immature red blood cells, and lymphocytes (an exception to the above). Epithelial cells are moderate to highly radiosensitive. Cells of low radiosensitivity include muscle and nerve.

Effect of Radiation Dose on Cell Dynamics

When cells with high mitotic activity are exposed to high-dose radiation, mitotic activity experiences a severe delay for an extended period before gradually returning to normal. In contrast, exposure to moderate doses of radiation causes a moderate delay in mitotic activity for a limited duration, followed by a temporary moderate increase before the mitotic rate returns to its normal level. (Figure 4).[2,6,8]

Exposure of cells with high mitotic activity to low dose radiation would result in an initial mild delay in mitotic activity followed by a short period of increased mitosis before the mitotic rate would return to normal.[2,6,8] In association with dental radiography, neither short-term nor long-term effects of radiation on mitotic activity are discernable.[2,5-7,11] However, to minimize exposure and maximize diagnostic yield, follow the dictum of ALARA - As Low As Reasonably Achievable.

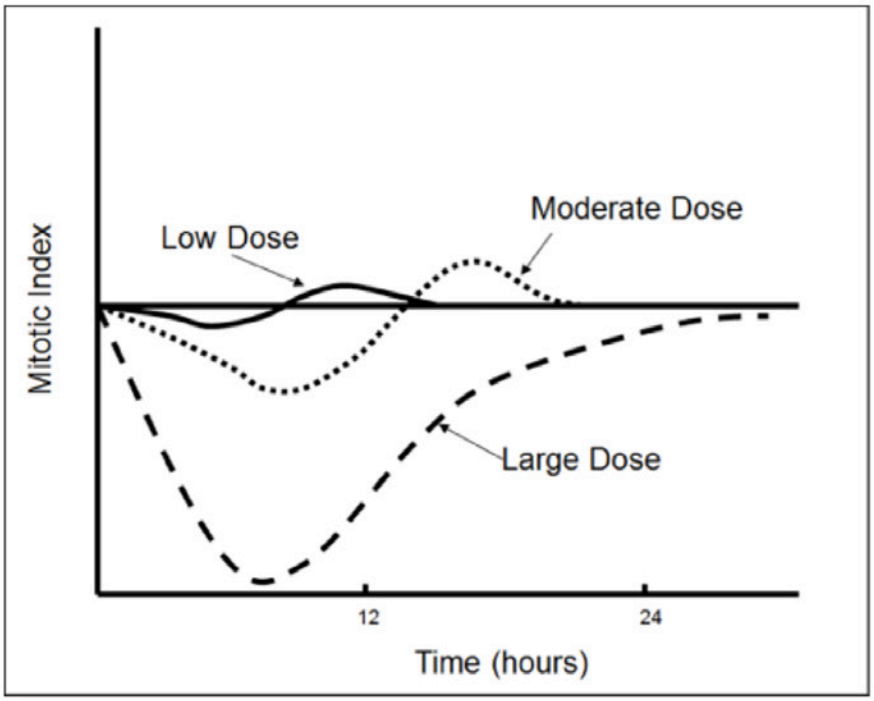

Figure 4. Change of mitotic rate as a function of dose and time.

Effects of High-dose Ionizing Radiation

High-dose ionizing radiation serves as an effective alternative to surgery or a valuable complement to surgery and/or chemotherapy in the locoregional treatment of head and neck malignancies. Oral healthcare providers should anticipate caring for head and neck cancer patients undergoing radiotherapy or those who have previously completed treatment. Therefore, understanding the deterministic biological changes caused by high-dose ionizing radiation is essential.[5,6,11]

The therapeutic effectiveness of ionizing radiation is linked to its impact on tumor cell DNA. Radiation can damage DNA directly by inducing breaks or indirectly by interacting with water or oxygen molecules, generating ion pairs and reactive oxygen species such as H_2O_2 and hydroxyl radicals (Figure 5).13 If unrepaired, DNA damage is likely to result in cell death, thereby eliminating malignant activity.[13]

Malignant cells have a decreased capacity to repair radiation damage. They can fall into one of three categories: (1) lethal damage, which occurs when no DNA repair is possible and leads to cell death, (2) sublethal damage, which is repairable as a function of time provided no further radiation damage is incurred prior to the

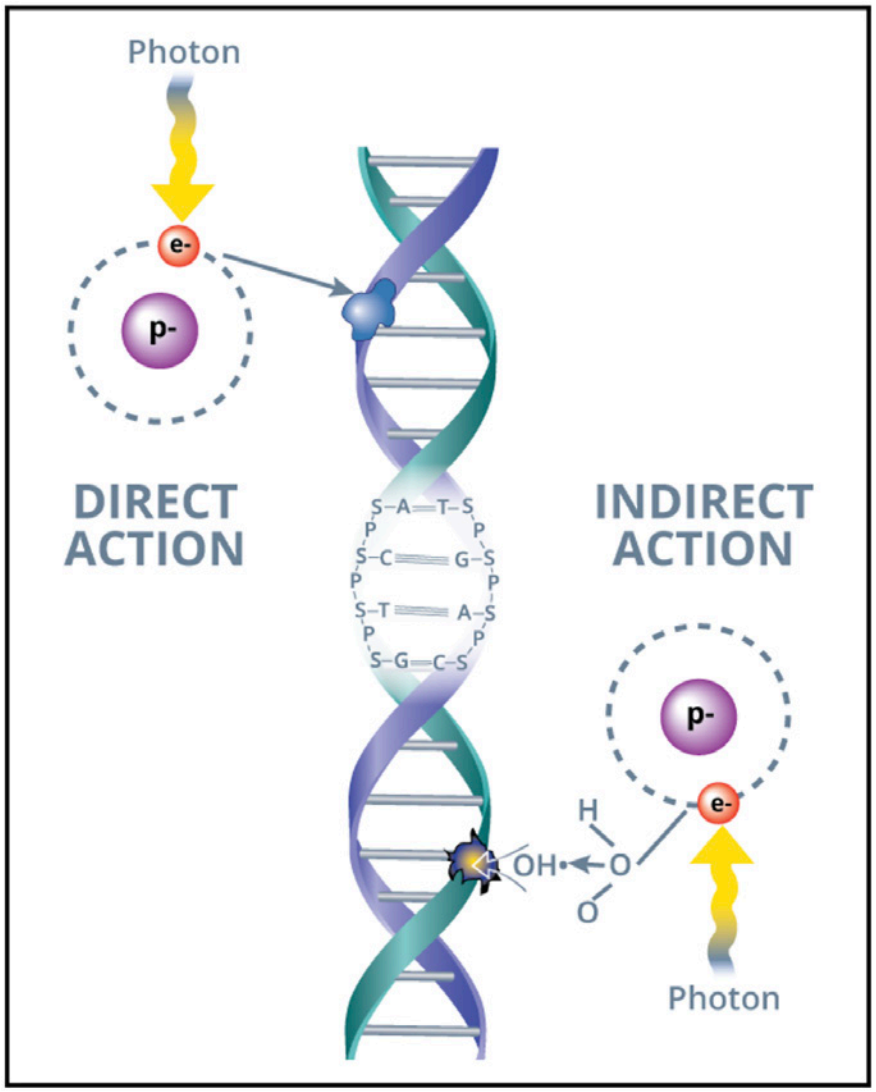

Figure 5. It is estimated that two-thirds of radiotherapy-induced damage to DNA is caused by hydroxyl radicals.

repair, and (3) potentially lethal damage, which is a condition in which cells may survive without repair, depending on post-radiation conditions.[13]

The radiation dose is defined as the amount of energy absorbed by the irradiated tissue. The unit of absorbed dose is the Gray (Gy = 1 J/kg). A standard radiotherapy regimen for head and neck neoplasms involves administering 60–70 Gy, fractionated at a rate of 2.0 Gy per day, 5 days per week, over 6 to 7 weeks. However, newer and often more aggressive protocols and techniques may be utilized to enhance tumor control and/or minimize side effects.[13]

In theory, any malignant tumor can be eradicated by ionizing radiation if a sufficiently high dose is delivered. The primary limiting factor is the tolerance level of the surrounding normal tissues.13 These adverse effects can be classified as either direct, meaning radiation-induced destruction or damage to vulnerable cells resulting in tissue function loss or disruption, or indirect, meaning radiation-induced reductions in vascularity and related tissue alterations.[5,6,8]

Short-term effects - Undifferentiated cells within irradiated tissues or organs will sustain severe damage, leading to an acute but temporary disruption of their integrity and function. If radiation doses are relatively low, stem cells may eventually differentiate, facilitating healing and at least partial restoration of tissue or organ function.[5,6,8]

Long-term effects - In some cases, radiation therapy delivers doses high enough to compromise the microvasculature of tissues and organs without directly harming differentiated cells. This damage typically becomes evident 6–8 weeks after irradiation. The resulting vascular impairment leads to reduced nutrient supply to affected organs, ultimately causing degeneration and necrosis. These effects continue to progress throughout the remainder of the patient's life.[5,6,8]

Skin and Mucosa

A biological model proposed to explain radiotherapy-induced dermal and mucosal changes identifies four phases: inflammatory, epithelial, ulcerative, and healing. During the inflammatory phase, free radicals and cytokines, such as interleukin-1β, prostaglandins, and tumor necrosis factor-α (TNF-α), are released in response to irradiation. These inflammatory mediators increase vascular permeability and contribute to tissue damage.[13]

In the epithelial phase, cellular reproduction declines, leading to desquamative pseudomembranous degeneration. The ulcerative phase marks the period when ulcerated tissue is most painful and highly vulnerable to infection. Additionally, an increase in gram-negative bacteria may further amplify the inflammatory response. The final healing phase begins when epithelial regeneration restores tissue integrity.[13]

Radiodermatitis and oral mucositis arise due to the depletion of the basal cell layer, which is responsible for rapid epithelial renewal. Skin reactions to radiotherapy occur in approximately 25% of patients and depend on the radiation dose and the volume of tissue exposed. With conventional dosing, these reactions typically appear within the first three weeks of treatment and may manifest as erythematous, desquamative, or necrotic lesions.[13]

Radiotherapy-induced mucositis primarily affects non-keratinized tissues, including the labial and buccal mucosa, soft palate, pharynx, floor of the mouth, and tongue. It is characterized by edema and erythema, followed by desquamation. As desquamative lesions advance, they develop into painful ulcerations, which may become colonized by Candida species, ultimately leading to acute or chronic candidiasis (Figure 6).[13]

During conventional radiotherapy protocols (i.e., 2 Gy/day, 5 days/week, for 5–7 weeks), cellular repopulation of the epithelium can counteract the destructive effects of radiotherapy dosing of up to 1.8 Gy/day is unlikely. After the first week of therapy, there will be hyperemia and epithelial atrophy followed by edema and erythema due to hyperemia. Painful desquamative pseudomembranous lesions and ulcerations mark the second and third weeks of therapy (Figure 7).[13]

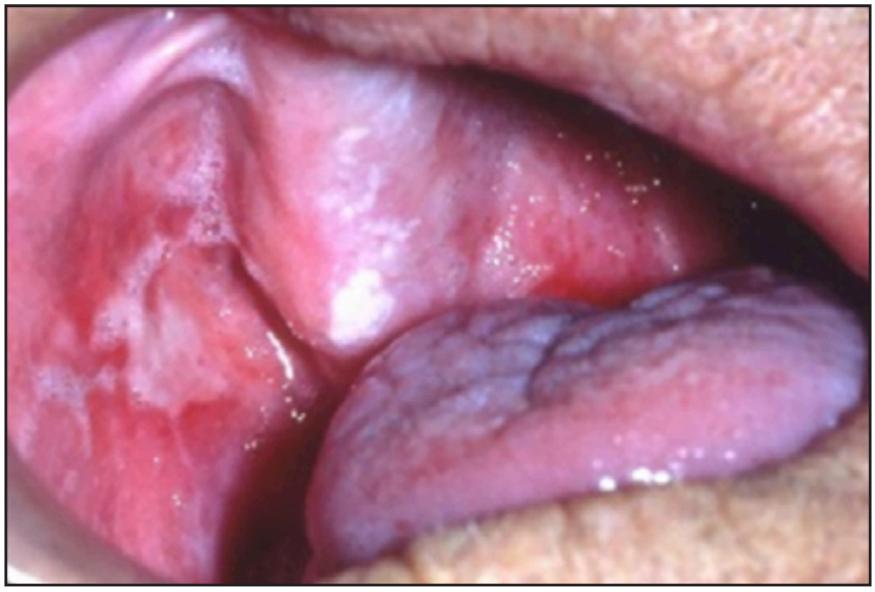

Figure 6. Acute radiotherapy-induced mucositis and candidal infection.

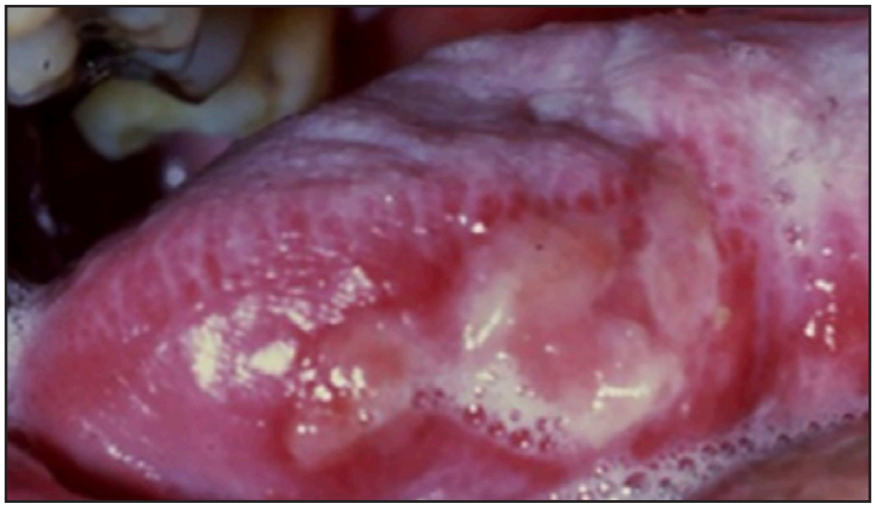

Figure 7. Acute radiotherapy-induced ulceration during the 2nd and 3rd week of therapy, also note the thick, viscous and frothy character of saliva.

Pharyngeal mucositis may impair the patient's ability to swallow and speak. An estimated 60% of patients undergoing conventional radiotherapy for head and neck cancer develop severe mucositis and require pain control and nutritional supplementation. With more intense radiotherapy, the incidence of mucositis may exceed 90%. However, complete healing in most patients occurs within four weeks after the completion of radiotherapy.[13]

Long-term effects associated with irradiated oral mucosa include tissue atrophy, telangiectasias, and increased risk of chronic ulceration (Figure 8). Cytokines and late-responding endothelial cells in the connective tissue underlie progressive fibrosis and thrombosis of small vessels in the dermis and submucosa. Radiodermatitis may result in hyperpigmentation, permanent loss of hair, and increased risk of skin cancers, typically basal cell carcinomas.[13]

Salivary Glands

A healthy individual produces up to 1.5L of saliva per day, with the major salivary glands

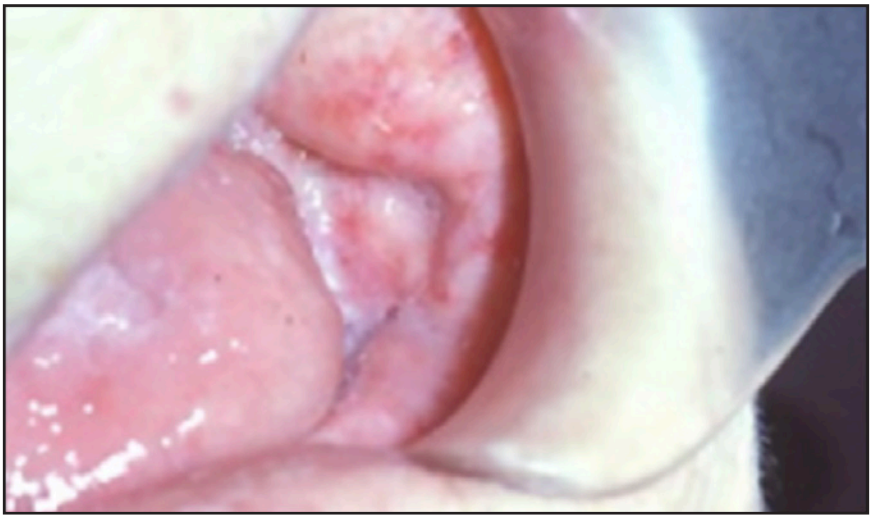

Figure 8. Long-term effects associated with irradiated oral mucosa include tissues atrophy and telangiectasias.

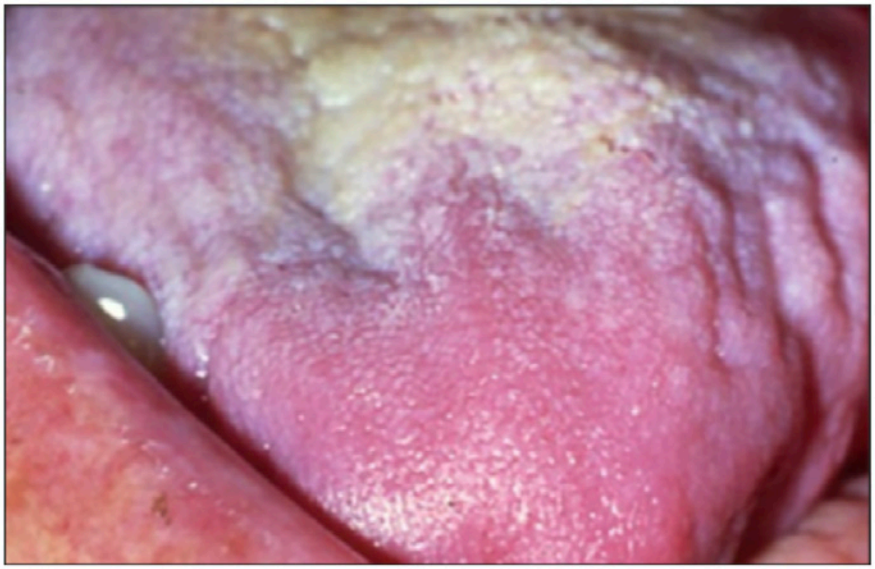

Figure 9. Xerostomia and chronic candidal infection are the most frequently reported long-term side effect of head and neck radiotherapy.

contributing 80–90% of the total volume. The parotid glands secrete serous saliva, the submandibular glands produce mixed seromucous saliva, and the sublingual and minor salivary glands secrete mucous saliva. Minor salivary glands account for 70% of mucin production. The parotid glands generate the highest volume of stimulated saliva, whereas the submandibular glands produce the most unstimulated saliva.[13]

Salivary gland cells, despite being well-differentiated and slow-replicating, exhibit high radiosensitivity for reasons that remain unclear.1, Radiotherapy induces acute degeneration and necrosis of acinar cells, leading to an 80% reduction in salivary output within the first two weeks of conventional treatment. Consequently, saliva becomes scant, thick, viscous, and either ropy or frothy. If any acinar recovery occurs, it may take 12 to 18 months following therapy completion.[13]

Both qualitative and quantitative alterations in saliva contribute to reduced lubrication, impaired lavage and cleansing of oral tissues,

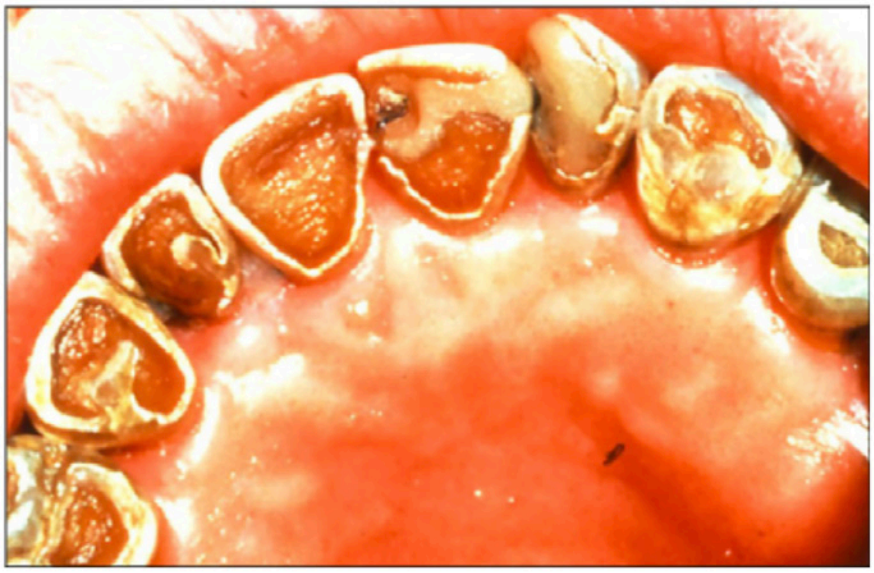

Figure 10. A distinctive form of rampant caries, termed "radiation caries", is frequently observed following head and neck radiotherapy.

and decreased immunoglobulin levels, leading to diminished antibacterial, antiviral, and antifungal activity. These changes further compromise mucosal integrity, causing patients to experience difficulty with swallowing, chewing, speaking, and wearing prostheses. Additionally, hypogeusia or ageusia may develop, potentially resulting in poor nutrition and weight loss.[13]

If radiation exposure is sufficiently high, acinar cell regeneration fails, leading to fibrosis and atrophy of glandular tissue. While the generally accepted threshold for irreversible damage is a mean dose of 60 Gy, some cases have implicated mean doses as low as 26 Gy. In most instances, patients undergoing head and neck radiotherapy will experience a lifelong significant reduction in salivary flow, resulting in xerostomia (Figure 9).[13]

The loss of saliva's buffering capacity and the decrease in the salivary immunoglobulin levels will increase cariogenic oral microflora, leading to "radiation caries". Radiation caries begin on plaque-forming surfaces and areas of exposed dentin resulting in circumferential lesions at the cementoenamel junction and smooth surface caries on cusp tips and incisal edges (Figure 10).[13]

Taste Buds

Taste buds are primarily located on the circumvallate and fungiform papillae of the tongue, with smaller distributions on the tonsillar pillars, base of the tongue, soft palate, pharynx, and larynx. Hypogeusia, a reduced ability to taste, or ageusia, a complete loss of taste perception for sweet, sour, bitter, or salty substances, is an early and common complaint among patients undergoing head and neck radiotherapy. Histological changes in taste buds appear at 10 Gy, with higher doses leading to their complete obliteration.[13]

Taste acuity may decline by a factor of 1,000 to 10,000 due to direct radiation-induced damage to the taste buds or secondary effects such as impaired salivary function and mucositis. Perception of acidic and bitter tastes is affected early, while sweet and salty sensations diminish as treatment progresses. Although taste sensation generally recovers within 2–4 months following radiotherapy, some patients may experience permanent alterations or a lifelong loss of taste perception.[13]

Teeth

Radiotherapy targeting the head and neck can significantly impact the developing dentition, with effects depending on the stage of tooth

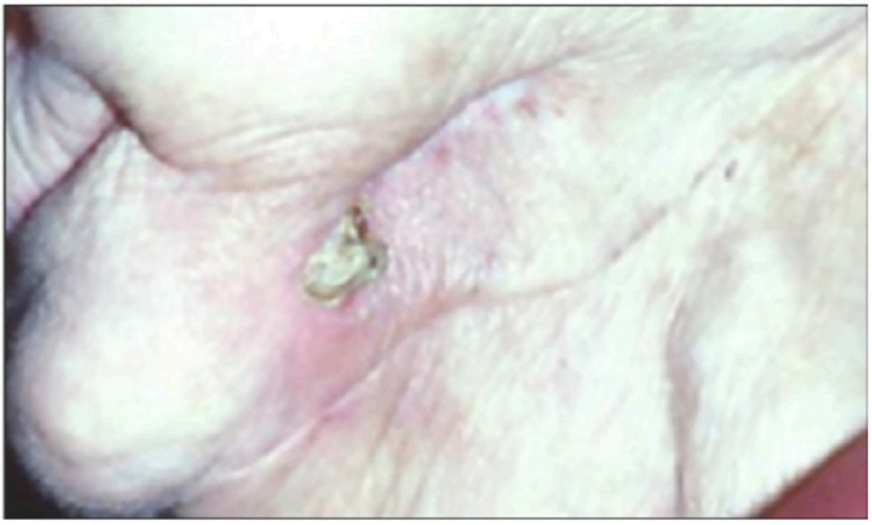

Figure 11. Chronic radiodermatitis and ulceration and osteoradionecrosis of the mandible.

development and the absorbed radiation dose. Exposure to 10 Gy can permanently damage maturing ameloblasts, while doses of 30 Gy completely halt ameloblastic activity. Radiation-induced abnormalities may include partial or complete anodontia, tooth dwarfism, incomplete root formation, premature apex closure, and localized enamel defects.[13]

Bone

Radiation doses exceeding 60 Gy result in endothelial cell death, leading to obliterative endarteritis and periarteritis. Additionally, the bone's ability to repair itself becomes compromised, causing osteoblast and osteocyte necrosis along with fibrosis of the periosteum and marrow spaces. Consequently, irradiated bone becomes hypocellular, hypovascular, and hypoxic, increasing the risk of osteoradionecrosis (Figure 11).[13]

The impact of radiotherapy on a child's developing bone depends on several factors, including the child's age at the time of treatment, the quality and quantity of radiation administered, and the location and extent of the bone within the treatment field. Doses above 20 Gy may significantly impair bone growth, leading to maxillary and mandibular hypoplasia. Additionally, asymmetrical radiation exposure may result in hemifacial hypoplasia.[13]

Temporomandibular Joint (TMJ) and Muscles of Mastication

When radiation portals include the temporomandibular joint or masticatory muscles, cellular damage and subsequent tissue fibrosis may compromise the integrity of the joint capsule or musculature. These effects, which typically appear 3 to 6 months post-radiotherapy, may cause muscle spasms, trismus, and restricted function, with threshold doses ranging between 15 Gy and 50 Gy.[13]

Dental Radiography Post-Radiotherapy

A common question gets asked about the consequences of exposing a patient to a dental x-ray after radiation therapy. The radiation doses from intraoral, panoramic radiographs, Cone beam, CT, or even medical CT are shallow compared to the doses of radiotherapy. The only concern would be obtaining intraoral radiographs within six months of radiotherapy, i.e., before mucosal healing, and further traumatizing the mucosa in the process.[6]

Late Somatic Effects of Radiation

Regardless of the radiation dose, there is always a risk of stochastic effects. Radiation exposure increases the likelihood of developing cancer by modifying DNA. Most radiation-induced malignancies emerge approximately ten years post-exposure. However, radiation does not

create entirely new cancers but rather elevates the probability of additional malignancies within the exposed population. Notably, children have twice the risk of experiencing late somatic effects compared to adults.[2,5,6,8,11]

Hereditary Effects of Radiation

Radiation-induced damage to germ cell DNA in the gonads can result in gene mutations, with mutation rates increasing alongside radiation dose. Similar to late somatic effects, radiation does not generate novel mutations but heightens the frequency of spontaneous ones. The mutation rate in male germ cells is higher than in female germ cells. However, the likelihood of mutation transmission decreases if conception occurs further from the time of exposure.[5,6,8,11]

Conclusion

Ionizing radiation generates free radicals. Free radicals interact with biological tissues, most importantly DNA. The severity of radiation-induced damage depends on the cell's sensitivity and the type and dose of radiation. There may be a DNA damage repair, but there are deterministic effects visible over a short-term period and a long-term period. The late somatic effects may manifest as malignancy and other genetic mutations.

Course Test Preview

To receive Continuing Education credit for this course, you must complete the online test. Please go to: *www.dentalcare.com/en-us/ce-courses/ce572/test*

1. Which of the following statements related to radiating energy is correct?

A. When radiating energy and living cells interact, energy transfer always leads to ionization.

B. Ultraviolet radiation, microwaves, and extra-low-frequency electromagnetic radiation are non-ionizing forms of radiation.

C. Non-Ionizing radiation is sufficiently energetic to eject an electron from an atom's orbit resulting in an ion pair, i.e., the electron and the rest of the atom.

D. Photons with sufficient energy cannot liberate orbital electrons from atoms.

2. Which of the following statements related to direct and indirect ionization is correct?

A. Indirect ionization occurs when charged particles, e.g., electrons, with sufficient kinetic energy interact with a water molecule to create free radicals.

B. Direct ionization occurs when non-charged particles, e.g., photons, interact with the DNA.

C. Ionizing radiation does not affect H2O molecules.

D. Direct ionization and indirect ionization are only the result of x-ray interacting with water molecules.

3. Which of the following statements related to free radicals and the cell cycle is correct?

A. Free radicals are chemical stable.

B. When free radicals interact with cellular macromolecules they do not cause damage.

C. In general, cells are most radiosensitive in the M phase of the cell cycle and most radioresistant in the G2, G1, and S phases, respectively.

D. Cell radiosensitivity is not affected by the differentiation level of the cell.

4. With dental radiography neither short-term nor long-term effects of radiation on mitotic activity are discernable. What dictum one should follow to minimize exposure and maximize diagnostic yield?

A. ALARP – As low as reasonably practical.

B. ALARA – As low as reasonably achievable.

C. ALRAA – As low reasonably as acceptable.

D. ALART – As low as radiographically attainable.

5. Which of the following statements related to the effects of high-dose radiation on malignant cells is correct?

A. The therapeutic benefit of ionizing radiation on tumor cells is related to its effects of tumor cell cytomplasm.

B. Radiation can interact with and damage the nuclear DNA directly by causing DNA single and double strand breaks.

C. Radiation doses of about 2Gy cause severe damage to the cytomplasm.

D. In comparison to normal cells, malignant are always radioresistant.

6. Which of the following statements related to radiotherapy is correct?

A. Absorbed dose is expressed as the absorbed energy by the irradiated tissue and the unit of the absorbed dose is the Sivert (Sv)

B. A conventional radiotherapy regimen for head and neck neoplasms consists of delivering a total of 6–7 Gy, fractionated at a rate of 0.2 Gy/day, 5 days/week, for 6 to 7 weeks.

C. Theoretically, any malignant tumor can be destroyed by ionizing radiation if the dose delivered is sufficient, the limiting factor is the amount of radiation the adjacent normal tissues will tolerate.

D. Short-term effects of ionizing radiation, which are related to damage to the microvasculature will be noticed within 6 months post irradiation.

7. Which of the following statements related to the biological effects of radiation on skin and mucosa is correct?

A. Radiodermatitis and oral mucositis develop secondary to the depletion of the rapidly dividing squamous cell layer of the epithelium.

B. Radiotherapy-induced mucositis generally appears on keratinized oral tissues, e.g., the oral mucoa and gingivae.

C. Edema and erythema due to hyperemia and epithelial atrophy of the oral mucosa appear after 6 months after radiotherapy.

D. Short-term effects associated with irradiated oral mucosa include tissues atrophy, telangiectasias, and increased risk of chronic ulceration.

8. Which of the following statements related to the biological effects of radiation on salivary glands is correct?

A. The well-differentiated and slow replicating salivary gland cells are highly radio-sensitive.

B. Salivary output may be decreased by as much as 80% within the first 2 weeks of conventional radiotherapy.

C. As a general rule, following head and neck radiotherapy, patients will experience no significant reduction in salivary flow.

D. Radiation caries begin on cusp tips and incisal edges.

9. Which of the following statements related to the biological effect of radiation on taste buds is correct?

A. Hypogeusia or ageusia is a frequent complaint associated with head and neck radiotherapy.

B. Histologically, taste buds show signs of atrophy at 0.1 Gy.

C. Perception of sweet and salt acuity are not affected early.

D. Restoration of taste sensation usually occurs within a few days following radiotherapy.

10. Which of the following statements related to biological effect of radiation on teeth is correct?

A. Radiotherapy to the head and neck area cannot delay eruption.

B. Maturing ameloblasts may be permanently damaged with as little as 0.2 Gy.

C. Radiation-induced defects may include partial or complete anodontia, tooth dwarfism, incomplete root development, premature closure of apices, and localized enamel defects.

D. A single intra-oral x-ray can cause permanent damage to a developing tooth.

11. Which of the following statements related to biological effect of radiation on bone is correct?

A. Radiation doses in excess of 5 Gy kill endothelial cells, causing obliterative endarteritis and periarteritis, and overwhelm the reparative capacity of bone.

B. Osteoblasts and osteocytes undergo necrosis and the periosteum and marrow spaces undergo regeneration.

C. Irradiated bone becomes hypocellular, hypovascular, and hypoxic; the potential clinical outcome of these effects is osteoradionecrosis.

D. After a dose of 60 Gy, there is a full repair and regeneration of endothelial cells and micorvasulature.

12. Which of the following statements related to biological effect of radiation on developing bone is incorrect?

A. The effect of radiation on developing bone does not depend on the child's age, the radiation dose delivered, and the extent of the bone in the treatment field.

B. Doses in excess of 20 Gy may significantly impair bone growth and development, resulting in maxillary and mandibular hypoplasia.

C. The use of asymmetrical radiation portals may lead to hemifacial hyperplasia.

D. The dose level is not a contributing factor to the damage to the bone in a child.

13. Which of the following is true about the radiation portals?

A. The threshold is about 5 Gy.

B. It may cause radiation related trismus.

C. It does not cause muscular atrophy.

D. The effects are observed within few days of initiation of the radiation therapy.

14. Which of the following statements related to the late somatic effects of radiation is correct?

A. Regardless of the radiation dose to which a patient is exposed, there is always the risk of stochastic, i.e., random, effects.

B. Radiation cannot cause DNA mutation.

C. Most of radiation-related cancers are unique, i.e., new cancers not normally seen in the population.

D. The risk of stochastic effects in children is half that noted in adults.

15. Which of the following statements related to biological effect of radiation on mutations is correct?

A. Radiation cannot damage the DNA of the gonads.

B. Gene mutations increase with dose and represent an increase in the rate of unique mutations that are different than those attributed to spontaneous mutations.

C. Mutations in the male germ cells are lower than in the female germ cells.

D. The rate of radiation-induced mutations is increased as the time between exposure and conception increases.

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Additional Resources

- No Additional Resources Available

About the Author

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Hassem Geha is a Professor in the Department of Comprehensive Dentistry, and the director of the Advance Oral and Maxillofacial Radiology program at the University of Texas Health Sciences Center. He received his dental degree from Saint Joseph University, School of Dental Medicine – Beirut in 1997, and two specialty degrees in oral biology and Maxillofacial radiology from the Lebanese University, School of Dentistry in Beirut in 2001. In 2002, he relocated to the United States. He became a Diplomate of the American Board of Oral and Maxillofacial Radiology in 2004. He received a Master of Dental Sciences (MDS) degree from the University of Connecticut Graduate School in 2005. Dr. Geha was appointed Assistant Professor at New York University College of Dentistry. In 2010 he joined UTHSCSA where he is Oral Radiology course director for the DS3 and he is heavily involved in the post-graduate program at the dental school. He also is a Clinical Associate in Otolaryngology and Head and Neck Surgery at the American University of Beirut Medical Center. Dr. Geha was the recipient of Albert G. Richards Award in 2003 and the Radiology Centennial Scholarship Award in 2004 given by the American Academy of Oral and Maxillofacial Radiology. He has given many presentations and continuing education courses at national and international meetings and authored many scientific manuscripts and abstracts in national and international journals. Hassem's main research focuses on enhancing digital imaging based on mathematical models. He chaired and served in several academic committees including many MS theses supervising committees in Oral and Maxillofacial Radiology.

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