The Ochsner Journal

Continuing Medical Education

CME QUESTIONS VOLUME 13, NO. 1

This section provides a review. Mark each statement (circle the correct answer) according to the factual material contained in this issue and the opinions of the authors. A score of 70% per article is required to qualify for CME credit.

Enhanced Vascular Chymase-Dependent Conversion of Endothelin in the Diabetic Kidney

1. The ASCEND trial was terminated due to excessive rates of adverse events such as fluid overload and congestive heart failure.
   True or False

2. Endothelin-converting enzyme is the only pathway for the formation of ET-1 (1-21) from the precursor Big ET-1 (1-38).
   True or False

3. Chymase is a carboxypeptidase responsible for activation of many inflammatory stimuli.
   True or False

4. Angiotensin-converting enzyme inhibitors and angiotensin receptor blockers provide optimal renal protection for patients with diabetic disease.
   True or False

5. Pharmacological inhibition of chymase may provide additional renal protection in patients with diabetic disease.
   True or False

In Vitro Synergy of Telavancin and Rifampin Against *Enterococcus faecium* Resistant to Both Linezolid and Vancomycin

1. The results of this in vitro synergy study using the combination of rifampin and telavancin or daptomycin against linezolid-resistant vancomycin-resistant *E faecium* revealed
   a. 88% synergy for the combination of rifampin + daptomycin.
   b. 83% synergy for the combination of rifampin + telavancin.
   c. no antagonism between the drugs in either combination.
   d. all of the above.

2. To evaluate the effect of the combination in the Etest method, the fractional inhibitory concentration (FIC) was calculated for each antibiotic in each combination. The mean ∑FIC was used to interpret the results. Synergy was defined as ∑FIC <0.5.
   True or False

3. Telavancin, a newer lipoglycopeptide, was recently approved for the treatment of complicated skin and skin structure infections caused by *Staphylococcus aureus*, streptococci, and vancomycin-susceptible *Enterococcus faecalis*.
   True or False

4. The synergy testing method performed in this in vitro study was
   a. an agar diffusion Etest method.
   b. a checkerboard method.
   c. a minimal inhibitory concentration (MIC):MIC Etest method in which 2 Etest strips are superimposed sequentially so the respective MIC values are aligned on the agar plate.
   d. a time-kill assay.

Kaposi Sarcoma–Associated Herpesvirus G Protein–Coupled Receptor Enhances Endothelial Cell Survival in Part by Upregulation of Bcl-2

1. AIDS-associated Kaposi sarcoma (KS) is classified as
   a. classic KS.
   b. endemic KS.
   c. iatrogenic KS.
   d. epidemic KS.
   e. indolent KS.

2. The etiological agent of KS is
   a. HIV-1.
   b. HHV-6.
   c. HHV-7.
   d. HHV-8.
   e. HSV-1.

3. KS–associated herpesvirus encodes a constitutively active G protein–coupled receptor that most closely resembles
   a. human IL-8 receptor.
   b. human Bcl-2.
   c. HIV-1 Tat protein.
   d. human Akt protein.
   e. mammalian target of rapamycin.

4. The human Bcl-2 protein
   a. is deleted in many cancers.
   b. is proapoptotic.
   c. is antiapoptotic.
   d. is proinflammatory.
   e. promotes cell death.

Nelfinavir Suppresses Insulin Signaling and Nitric Oxide Production by Human Aortic Endothelial Cells: Protective Effects of Thiazolidinediones

1. Mr Morose has Kaposi sarcoma–like lesions all over his body. An ELISA test is positive for anti-HIV antibodies, and a viral load measurement shows high levels of HIV RNA in his blood. His physician prescribes a drug regimen of zidovudine (AZT), lamivudine (3TC), and atazanavir. What are the mechanisms of action of the 3 anti-HIV drugs prescribed?
   a. AZT and 3TC are nucleoside analogs, and atazanavir inhibits viral neuraminidase-associated viral release from the host cell.
   b. AZT and 3TC inhibit viral attachment and entry, and atazanavir is a nucleoside analog.
   c. AZT inhibits viral attachment and entry, 3TC inhibits viral uncoating, and atazanavir is a viral protease inhibitor.
   d. AZT and 3TC are nucleoside analogs, and atazanavir is a viral protease inhibitor.
   e. AZT and 3TC inhibit viral uncoating, and atazanavir inhibits viral neuraminidase-associated viral release from the host cell.

2. A 37-year-old man who is HIV-positive recently started on a highly active antiretroviral therapy regimen. His CD4 cell count subsequently falls below 200 mm^3. Over the course of the next 3 months, he develops diarrhea and notices a redistribution of fat on his body. Which of the following agents is most likely causing his symptoms?
   a. Fusion inhibitor
   b. Nonnucleoside reverse transcriptase inhibitor
   c. Nucleoside reverse transcriptase inhibitor
   d. Nucleotide reverse transcriptase inhibitor
   e. Protease inhibitor

3. Rifampin is known to induce several P450 enzymes, including CYP2C9, CYP2C19, and CYP3A4. What effect would concurrent administration of rifampin have on plasma levels of indinavir and enfuvirtide?
   a. Plasma levels of indinavir will be increased, but levels of enfuvirtide will be reduced.
   b. Plasma levels of both drugs will be increased.
   c. Plasma levels of both drugs will be decreased.
   d. Plasma levels of indinavir will be reduced, but levels of enfuvirtide will not change.
   e. Plasma levels of indinavir will be reduced, but levels of enfuvirtide will be increased.

4. We are starting therapy for an established HIV infection in a 28-year-old man. The drugs are zidovudine, didanosine, saquinavir, and low-dose ritonavir. Which of the following is the main purpose of using the low-dose ritonavir?
   a. Induces the metabolic activation of the nucleoside reverse transcriptase inhibitors, which are prodrugs
   b. Prevents the likely development of hypoglycemia
   c. Helps maintain adequate saquinavir levels by inhibiting its metabolism
   d. Reduces, or hopefully eliminates, squinavir-mediated host toxicity
   e. Serves as the main, most active inhibitor of viral protease in this combination

(Questions continue on opposite side)

(Questions continued from opposite side)

5. Mr Barbey was diagnosed with type 2 diabetes and was immediately started on a regimen of metformin and a thiazolidinedione (TZD). What is the primary mechanism of action for metformin and TZDs?
   a. TZDs and metformin are insulin sensitizers.
   b. Metformin primarily inhibits gluconeogenesis, while TZDs primarily act as insulin sensitizers.
   c. TZDs and metformin primarily inhibit gluconeogenesis.
   d. TZDs primarily inhibit gluconeogenesis, and metformin primarily acts as an insulin sensitizer.
   e. TZDs and metformin primarily increase peripheral glucose uptake.

6. Later, Mr Barbey developed some side effects from TZD-metformin combination therapy, including nausea, weight gain, diarrhea, increased appetite, and edema. What are the common side effects associated with TZDs?
   a. TZDs are associated with weight gain and gastrointestinal disturbances (nausea, diarrhea).
   b. TZDs are associated with gastrointestinal disturbances (nausea, diarrhea) and edema.
   c. TZDs are associated with gastrointestinal disturbances (nausea, diarrhea) and increased appetite.
   d. TZDs are associated with weight gain, edema, and increased appetite.
   e. TZDs are associated with gastrointestinal disturbances (nausea, diarrhea).

Tumor Necrosis Factor-α: Life and Death of Hepatocytes During Liver Ischemia/Reperfusion Injury

1. The initial immune response during hepatic ischemia/reperfusion injury is characterized by the activation of resident CD8+ T cells in the liver.
   True or False

2. The caspase cascade is involved in programmed necrosis.
   True or False

3. Research studies using small hairpin RNAs (shRNAs) to silence the tumor necrosis factor-α gene in a mouse liver ischemia/reperfusion model resulted in data that showed lower liver enzyme levels in shRNA-treated mice compared to untreated mice.
   True or False

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CME CREDIT APPLICATION FORM

☐ MD ☐ DO ☐ Other *(specify)* _____

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VERIFICATION OF ATTENDANCE

The maximum number of credits awarded for this activity is 5 *AMA PRA Category 1 credits™.*

_____ _____

Signature Date

EVALUATION

Your response to these questions helps us to enhance our CME offerings. Please take the time to respond and return the evaluation. Thank you.

Please use the following codes to answer items 1-7.

SA – Strongly Agree
A – Agree
U – Undecided
D – Disagree
SD – Strongly Disagree

1. The objectives of the CME activity were clearly stated.
   SA A U D SD
2. The content of the journal articles was up-to-date.
   SA A U D SD
3. The journal articles illustrated independence, objectivity, balance, and scientific rigor.
   SA A U D SD
4. The content was closely related to objectives of my clinical practice and/or teaching.
   SA A U D SD
5. The journal articles increased my knowledge of the subject.
   SA A U D SD
6. The content of the journal articles met my personal expectation and needs.
   SA A U D SD
7. I will apply the information learned from these journal articles in my clinical practice.
   SA A U D SD

Do you have any suggestions as to how to improve the content of the journal articles?

_____

_____

What topics would you like to see in future journal articles?

_____

_____

Thank you for completing this evaluation and survey.