Name: ____*Dr. C.*_____

OPEN-NOTES QUIZ #2

Please answer any 4 of the following 6 questions. Please be clear about which questions should be graded. You may use the front and back of this paper for your answers.

1. Describe and/or diagram the structure of a typical 12-bar blues. What patterns are used? How do the lyrics, chords, vocals, and instruments fit together?

   The 12-bar blues is typically divided into three phrases of 4 bars (4 measures) each. The chord progression for the 12 bars is: 1 4 1 1 4 4 1 1 5 4 1 1/5. Lyrically, there's an A phrase for the first 4 bars, a repeat of the A phrase for the next 4 bars, and then a B phrase for the last 4 bars. The A phrase introduces a problem, situation, feeling, etc., which the B phrase represents a response or resolution to the A phrase. Within each 4-bar section, the singing usually happens during the first two bars and the instrumental accompaniment fills out the rest of the section.

2. In what ways can Muddy Waters (McKinley Morganfield) be considered a transitional figure between the blues and early rock?

   Muddy Waters "electrified" the blues, using an electric guitar rather than an acoustic guitar, and had a band (2 electric guitars, bass, drums) resembling a rock band. His songs combined the 12-bar blues format with other formats (for example, the strophic verse-chorus structure of "Hoochie Coochie Man"). Finally, his move from the Mississippi delta to Chicago was representative of the roots of rock coming out of the south and spreading around the country.

3. In what ways does "Maybellene" by Chuck Berry resemble and not resemble a standard 12-bar blues?

   The chorus of the song is a straight 12-bar blues; lyrically, there's a repeated A phrase followed by a B phrase. The verses are also 12 bars long, but the "1" chord is used throughout, the lyrics follow an AABBCC rhyme scheme, and the words are delivered as a rapid-fire rap without a melody. Thus the verses are quite different from a 12-bar blues.

4. Which cellular structures are responsible for contraction, calcium release/uptake, and ATP production in muscle cells? How does the amount of cellular space devoted to each of these structures depend on the function of a given muscle?

   Contraction is a process powered by the myosin (thick) filaments grabbing and pulling on the actin (thin) filaments. Calcium release and uptake is handled by the endoplasmic reticulum (called the sarcoplasmic reticulum in muscle cells). ATP production is done by glycolysis (sort-term) and mitochondria (longer-term). The amount of cellular space devoted to these components depends on the function of the particular muscle. A human biceps is for short, powerful contractions, so those muscle cells will consist mostly of actin and myosin and associated proteins. If it is necessary to turn contraction on and off very rapidly, there will be more sarcoplasmic reticulum so that calcium can be shuttled in and out of the cytoplasm quickly. Endurance-oriented muscles that exercise for long periods of time will have lots of mitochondria.

5. In theory, how could you use genetic engineering to take a slow sprinter like Dr. C. and make him faster?

   Dr. C., like everyone, has the genes for both fast-twitch myosin (types IIa and IIx) and slow-twitch myosin (type I). Therefore the trick is to get his muscles to express more of the type II myosins. In theory, this could be done with a transcription factor that causes RNA polymerase to preferentially transcribe the type II myosin genes. A gene for a transcription factor could be introduced into Dr. C.'s muscle cells with a harmless virus that infects the cells but doesn't cause disease, or with liposomes (fatty spheres that fuse with the cells).

6. How might antisense nucleotides be useful someday in treating Duchenne's Muscular Dystrophy (DMD)? Could we design a single antisense nucleotide sequence to treat all DMD patients? Why or why not?

   Duchenne's Muscular Dystrophy is caused by mutations resulting in a premature stop codon and a truncated, nonfunctional dystrophin protein, which is responsible for connecting actin to the inside of the cell membrane. The unprocessed mRNA for dystrophin contains numerous exons that get spliced together; an antisense oligonucleotide could bind to the problematic exon and cause that exon to be skipped in the splicing process, ultimately resulting in a slightly shorter but partially functional protein (as in Becker's Muscular Dystrophy). A single antisense oligonucleotide sequence would only be effective for patients with a mutation in the corresponding exon; unfortunately, different patients have mutations in a variety of different exons.