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Analyzing marginal outcomes in pilot cancer clinical trials

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The analysis of small randomized cancer clinical trials poses known difficulty when the outcome measure is based on a marginal relative effects estimate. Confidence intervals for marginal relative effect estimates tend to be overly conservative and have little value in practice. In this presentation, we present a modified multinomial procedure for estimating marginal relative effect estimates and provide simulation results comparing the empirical and expected coverage of the estimated values.

Biography

Jimmy T. Efird is an Associate Member of the Leo Jenkins Cancer at Brody School of Medicine. Additionally, he holds a joint appointment as Associate Professor in the Department of Public Health and as Epidemiologist/Chief Statistician in the Center for Health Disparities. He received his Ph.D. from Stanford University. His expertise includes statistical methods for assessing gene-environment interaction, clinical trial design, computing power and sample size for correlated samples, and multiplicity adjustments for confidence intervals. He has over 100 publications in scientific journals and technical proceedings. Additionally, he serves as a Senior Consultant for The NCRR-funded RCMI Translational Research Network Data and Technology Coordinating Center.

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MicroRNA-122-based therapeutics might be a double-edged sword in the liver diseases

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MicroRNA-122-based therapeutics might be a double-edged sword in the liver diseases There is increasing evidence that miR-122 may be a potential target for the treatment of liver diseases, especially in the treatment of dyslipidemias, hepatitis C virus (HCV), and hepatocellular carcinoma (HCC). For instance, miR-122 sequestration leads to decreased cholesterol levels and low density lipoprotein and high density lipoprotein fractions both in the liver and blood, and decreases liver fat accumulation in the liver. In addition, miR-122 silencing or the functional inhibition of miR-122 leads to potent and sustained inhibition of HCV replication. On the contrary, overexpression and restoration of miR-122 may inhibit the replication of HBV and the growth and metastasis of HCC, also sensitize HCC cells to chemotherapeutic agents, but can stimulate the replication of HCV. Collectively, the above evidences show that miR-122 may play contradictory roles in liver diseases.

Miravirsen, a 15-nucleotide locked nucleic acid-modified DNA phosphorothioate antisense oligonucleotide, can sequester mature miR-122 and thus inhibit its function. It has been initiated for treatment of HCV infection. Data from the phase 2a clinical study showed that miravirsen treatment is well tolerated, and reduces HCV RNA levels to undetectable levels in five patients with HCV receiving short-term miravirsen (four weeks), validating its promising potential for CHC therapy. Unfortunately, four of these five patients had a rebound in viral levels at the end of the study, indicating that receiving short-term miravirsen was insufficient to achieve a sustained virologic response in patients. Notably, receiving miravirsen of longer duration may increase the risk of HCC in patients with chronic HCV infection, due to insufficient function of miR-122 in a long time. Because lines of evidence shown that miR-122 acts as a tumor suppressor in HCC.

Finally, we conclude that microRNA-122-based therapeutics might be a double-edged sword in the liver diseases. If such approaches are applied, the efficacy and safety in long duration should be evaluated and the status of selected patients should be detected carefully.

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