address basic and other health-related self-care needs. The target population included people ages 18 and older, English speaking, living in 1 of 16 ZIP codes on Chicago's south and west sides (106 mi^2) who received care at ≥1 of 28 CommunityRx partner sites and had a diagnosis of asthma or COPD. Between December 2015 and December 2016, information about pulmonary research participation opportunities was included on the HealtheRxs of eligible patients contemporaneously with usual registry recruitment methods. Usual methods, used since 2010 by the PRR group, included public advertisements requiring the patient to call the research team for more information and education of eligible patients identified during routine clinical care with a Pulmonary/Critical Care clinician or when enrolling in a pulmonary clinical trial. We hypothesized that, compared with usual recruitment practices, the HealtheRx recruitment strategy would increase the rate and decrease the per subject cost of patient recruitment to a prospective registry. Total annual recruitment costs for each method were calculated and divided by the number of consented PRR enrollees per method. RESULTS/ANTICIPATED RESULTS: Between December 22, 2015 and December 15, 2016 13,437 HealtheRxs (8762 for asthma, 3842 for COPD, and 833 for both asthma and COPD) were generated with the recruitment advertisement. In total, 41 patients responded to the ad and participated in the phone survey. In which 15 (36.5%) participants self-reported a diagnosis of asthma only (65% of all HealtheRxs with advertisement were for asthma only), 9 (22%) reported a diagnosis of COPD only (28.5% of all HealtheRxs with advertisement were for COPD only), and 17 (41.5%) reported diagnoses of both asthma and COPD (6.2% of all HealtheRxs with advertisement were for asthma and COPD). Most participants were female (n = 28), non-Hispanic black (n = 37), and not employed (n = 39). The median age was 57. The majority (n = 31) had never participated in health or medical research and was not aware of current opportunities to participate in research (n = 25). All 41 participants expressed interest in joining PRR and were mailed a blank PRR consent form and a prepaid return envelope with their incentive check for the telephone survey. To date, 5 participants returned a signed consent form via mail and were enrolled in PRR. During the same period, 4 patients enrolled in PRR via usual recruitment methods. The cost per subject to enroll in PRR was $364.40 using the HealtheRx recruitment and $4590 using usual practice. DISCUSSION/SIGNIFICANCE OF IMPACT: NIH has called for innovation in research recruitment methodologies to increase enrollment especially of people who are under-represented in clinical trials research. This study demonstrates the feasibility and efficiency of using an EMR-integrated recruitment method to enroll people of under-represented minority groups to a research registry.

2302

Downregulation of miR-1207-3p is correlated to upregulation of FNDC1, FN1, AR, and c-MYC in aggressive prostate cancer in men of African ancestry

Dibash K. Das, Akintunde T. Orunmuyi, Gabriel Olabiyi Ogun, S. Adekola Adebayo, A. Ayo Salako, Adeodat Ilboudo, E. O. Olapade-Olaopa and Olorunseun O. Ogunwobi

Department of Biological Sciences, Hunter College, New York, NY, USA

OBJECTIVES/SPECIFIC AIMS: Prostate cancer is the second most common cancer in the world for men. For reasons still unclear, aggressive PCa disproportionately affects males of African ancestry (MoAA). Incidence and mortality rates are highest in MoAA as they have consistently shown a 2.3–3.0-fold higher risk of mortality compared with Caucasian men. This aggressiveness of PCa may be due to specific biological factors. Studies have established that microRNAs (miRNAs), essential in post-transcriptional gene regulation, are dysregulated in PCa. miR-1207-3p is encoded at the PVT1 gene locus, which is located downstream of MYC on the 8q24 PCa susceptibility locus. The chromosomal region 8q24 is associated with aggressive PCa. Yet miR-1207-3p in PCa in MoAA has never been investigated. Moreover, studies have shown that PVT1/MYC co-operation is a fundamental feature in all cancers with 8q24 amplification and 98% of the 8q24 amplicons contained concurrent amplification of the MYC and PVT1 loci. Moreover, MYC has been linked to PCa aggressiveness, and has been reported to be downstream of androgen receptor (AR) in some PCa. However, the mechanisms regulating c-MYC have never been studied in MoAA. We have recently demonstrated that miR-1207-3p has prognostic value in PCa, and directly binds to the 3′ UTR of Fibronectin type III domain containing 1 (FNDC1) to regulate a novel FNDC1/fibronectin (FN1)/AR pathway upregulated in metastatic PCa. However, the relevance of this novel and clinically significant miR-1207-3p molecular pathway in PCa in MoAA is unknown. Therefore, we hypothesized that miRNA 1207-3p, encoded at the 8q24 PCa susceptibility locus, is a PCa biomarker with clinical applications in MoAA. Our specific aim was to assess the clinical relevance of miR-1207-3p, FNDC1, FN1, AR, and MYC expression in aggressive PCa in a cohort of West African Black males. METHODS/STUDY POPULATION: Consequently, we aimed to determine the expression profile of miRNA-1207-3p, FNDC1, FN1, AR, and MYC in histologically confirmed normal prostate (n = 24), benign prostate (n = 44) and malignant prostate tissue (n = 29) from prostatectomy or transrectal ultrasound-guided biopsies in patients recruited at the University College Hospital, Ibadan, Nigeria, a sub-Saharan Black African population. In total, 17 patients had tumor tissues with Gleason score ≥8. Tissues were collected in compliance with Institutional Ethics Board approved protocols. RNA extraction, cDNA synthesis, and quantitative-PCR were performed to analyze mRNA expression of miRNA-1207-3p, FNDC1, FN1, AR, and MYC. Statistical analysis were performed using SPSS software. All data were analyzed by the 1-way ANOVA test. Tukey post-hoc test was performed to determine the differences in mean expression between normal and tumor prostate tissues. $p < 0.05$ were considered significant. RESULTS/ANTICIPATED RESULTS: We discovered that miR-1207-3p is significantly underexpressed in prostate tumor tissues [0.09 ± 0.02, 95% CI (0.04, 0.136), $p = 0.000$] in comparison with normal prostate tissue in MoAA [0.92 ± 0.15, 95% CI (0.60, 1.244), $p = 0.000$]. This is the first description of miR-1207-3p differential expression in human clinical PCa in MoAA. In contrast, FNDC1 was significantly overexpressed in prostate tumor tissues [21.93 ± 8.21, 95% CI (4.97, 38.89), $p = 0.003$] in comparison with normal prostate tissues in MoAA [1.57 ± 0.45, 95% CI (0.625, 2.51), $p = 0.003$]. The same positive correlation with advanced disease held true for FN1 [tumor: 13.66 ± 3.53, 95% CI (6.38, 20.93), $p = 0.000$; normal: 1.07 ± 0.235, 95% CI (0.58, 1.56), $p = 0.000$], AR [tumor: 20.49 ± 6.74, 95% CI (6.50, 34.48), $p = 0.000$; normal: 0.94 ± 0.20, 95% CI (0.52, 1.36), $p = 0.000$], and c-MYC [tumor: 33.93 ± 8.43, 95% CI (16.53, 51.33), $p = 0.000$; normal: 1.94 ± 0.36, 95% CI (1.18, 2.70)]. The significantly increased mean expression for FNDC1, FN1, AR, and c-MYC in prostate tumor tissues in comparison with normal prostate tissues indicate that their overexpression is associated with increased risk of cancer progression in MoAA. DISCUSSION/SIGNIFICANCE OF IMPACT: These results show that the underexpression of miR-1207-3p and the overexpression of FNDC1, FN1, AR and MYC is associated with aggressive PCa in MoAA. miR-1207-3p, and it molecular target FNDC1, may be useful biomarkers for prognostication of PCa in MoAA.

2307

Resting state network profiles of Alzheimer disease and frontotemporal dementia: A preliminary examination

Joey Annette Contreras, Shannon L.. Risacher, Mario Dzemidzic, John D. West, Brenna C. McDonald, Martin R. Farlow, Brandy R. Matthews, Liana G. Apostolova, Jared Brosch, Bernard Ghetti and Joaquin GoÑi

Indiana University School of Medicine, Indianapolis, IN, USA

OBJECTIVES/SPECIFIC AIMS: Recent evidence from resting-state fMRI studies have shown that brain network connectivity is altered in patients with neurodegenerative disorders. However, few studies have examined the complete connectivity patterns of these well-reported RSNs using a whole brain approach and how they compare between dementias. Here, we used advanced connectomic approaches to examine the connectivity of RSNs in Alzheimer disease (AD), Frontotemporal dementia (FTD), and age-matched control participants. METHODS/STUDY POPULATION: In total, 44 participants [27 controls (66.4 ± 7.6 years), 13 AD ($68.5.63 \pm 13.9$ years), 4 FTD (59.575 ± 12.2 years)] from an ongoing study at Indiana University School of Medicine were used. Resting-state fMRI data was processed using an in-house pipeline modeled after Power *et al.* (2014). Images were parcellated into 278 regions of interest (ROI) based on Shen *et al.* (2013). Connectivity between each ROI pair was described by Pearson correlation coefficient. Brain regions were grouped into 7 canonical RSNs as described by Yeo *et al.* (2015). Pearson correlation values were then averaged across pairs of ROIs in each network and averaged across individuals in each group. These values were used to determine relative expression of FC in each RSN (intranetwork) and create RSN profiles for each group. RESULTS/ANTICIPATED RESULTS: Our findings support previous literature which shows that limbic networks are disrupted in FTLD participants compared with AD and age-matched controls. In addition, interactions between different RSNs was also examined and a significant difference between controls and AD subjects was found between FP and DMN RSNs. Similarly, previous literature has reported a disruption between executive (frontoparietal) network and default mode network in AD compared with controls. DISCUSSION/SIGNIFICANCE OF IMPACT: Our approach allows us to create profiles that could help compare intranetwork FC in different neurodegenerative diseases. Future work with expanded samples will help us to draw more substantial conclusions regarding differences, if any, in the connectivity patterns between RSNs in various neurodegenerative diseases.

https://doi.org/10.1017/cts.2017.38 Published online by Cambridge University Press