

## Musing on Microarrays and Genomic Analysis

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The paper on molecular profiling of human chondrosarcoma cells by Desiderio *et al.* [1] in this issue confirms several important findings by other investigators, such as that cells cultured in 3-D structures approximate their natural environment more closely than monolayer cultures and, as a result, these 3-D cultures are quite suitable for drug discovery and screening using gene expression microarrays. The expression of stemness markers by chondrospheres described in this report also supports the use of 3-D cultures in regenerative medicine. This is not the first report of gene expression in chondrosarcoma, e.g., Yoshitaka *et al.* used microRNA microarrays to analyze 20 chondrosarcoma samples obtained at surgery and 2 chondrosarcoma cell lines [2]. However, there appears to be little or no overlap between these two studies though both employed microarrays from the same manufacturer. The reasons are multiple: Desiderio *et al.* used primary tumor-derived chondrospheres, a type of 3-D culture whereas Yoshitaka *et al.* analyzed microRNAs from two commercially available chondrosarcoma cell lines maintained in traditional 2-D configuration, and microRNAs from 20 surgically obtained chondrosarcomas. Authors of both studies observed changes in expression of many genes, but emphasized changes in those deemed of importance to their particular investigation, and, as a result analyzed only a very selected few changes. As Desiderio *et al.* were mostly interested in drug resistance, metastasis and “stemness” they directed their attention to genes likely to be involved in regulation of these three processes. In contrast, Yoshitaka *et al.* concentrated on genes whose changes in expression can be utilized in diagnosis. Genomic analysis is being developed for other types of malignancies, such as ovarian cancer [3], acute leukemia [4] and follicular lymphoma [5] to name just a few. All of this helps to drive an important point home: there are many many genes involved in carcinogenesis, some already known, and many more to be still discovered as pointed out by Lawrence *et al.* [6]. In their just published report the authors conclude that many new candidate cancer genes remain to be discovered and that the

number of additional cancer genes will go up as more tumors within the same (histopathological) group will be examined and as additional tumor types will undergo genomic analysis [6]. This will enhance the current capacity of current genomic collection, however, it will take quite a bit of effort to understand which mutations, be it somatic point mutations, substitutions, small insertion, and/or deletions contribute to carcinogenesis, and can be utilized as diagnostic and/or prognostic markers. Such analysis will also contribute to our knowledge of genes and their functions, such as in cell proliferation, apoptosis, genome stability, chromatin regulation, immune evasion, RNA processing and protein homeostasis [6]. Integrated genomic analyses carry the promise to revolutionize diagnosis and determination of proper treatment modalities for a variety of malignancies, and, indeed, other diseases as well. It also remains to be seen to what extent this new technology will transform pathology, esp., diagnostic surgical pathology, or whether, genomic analysis will even supersede this venerable old discipline!

### References

1. Desiderio V, Paino F, Nebbioso A, Altucci L, Pirozzi G, et al. (2013) Molecular Profiling of Human Primary Chondrosarcoma-Derived Spheres Reveals Specific and Target Genes Involved in Multidrug Resistance and Metastasis. *J Carcinogene Mutagene*
2. Yoshitaka T, Kawai A, Miyaki S, Numoto K, Kikuta K, et al. (2013) Analysis of microRNAs expressions in chondrosarcoma. *J Orthop Res* 31: 1992-1998.
3. Karst AM, Drapkin R (2011) The new face of ovarian cancer modeling: better prospects for detection and treatment. *F1000 Med Rep* 3: 22.
4. Chung SS (2014) Genetic mutations in acute myeloid leukemia that influence clinical decisions. *Curr Opin Hematol* 21: 87-94.
5. Okusun J, Bödör C, Wang J, Araf S, Yang CY, et al. (2013). Integrated genomic analysis identifies recurrent mutations and evolution patterns driving the initiation and progression of follicular lymphoma. *Nature Genetics*
6. Lawrence MS, Stojanov P, Mermel CH, Robinson JT, Garraway LA, et al. (2014) Discovery and saturation analysis of cancer genes across 21 tumour types. *Nature* 505: 495-501.

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