

MINI-REVIEW

Ovarian cancer think tank: the use of integrated artificial intelligence and computational biology in ovarian cancer diagnosis and treatment

Eseohi Ehimiaghe^{1,*}, Hannah Dimmick², Daniel Spinosa¹, Freda Ireigbe¹, Miriam D. Post³, Rebecca J. Wolsky³, Aaron Clauset^{4,5}, Sandra Orsulic^{6,7,8}, Sarah Taylor⁹, Elena W. Y. Hsieh^{10,11}, Natalie Davidson², Marie Wood¹², Benjamin G. Bitler², Bradley R. Corr¹, Saketh R. Guntupalli¹, Lindsay W. Brubaker¹, Marisa R. Moroney¹, Kian Behbakht^{1,2}

¹Department of Obstetrics and Gynecology, Division of Gynecologic Oncology, University of Colorado Anschutz Medical Campus, Aurora, CO 80045, USA

²Department of Obstetrics and Gynecology, Division of Reproductive Sciences, University of Colorado Anschutz Medical Campus, Aurora, CO 80045, USA

³Department of Pathology, University of Colorado Anschutz Medical Campus, Aurora, CO 80045, USA

⁴Department of Computer Science, University of Colorado, Boulder, CO 80310, USA

⁵BioFrontiers Institute, University of Colorado, Boulder, CO 80303, USA

⁶Department of Obstetrics and Gynecology and Department of Pathology and Laboratory Medicine, David Geffen School of Medicine, University of California Los Angeles, Los Angeles, CA 90095, USA

⁷Jonsson Comprehensive Cancer Center, University of California Los Angeles, Los Angeles, CA 90024, USA

⁸Department of Veterans Affairs, Greater Los Angeles Healthcare System, Los Angeles, CA 90025, USA

⁹Department of Obstetrics, Gynecology and Reproductive Sciences, Magee-Womens Hospital of UPMC, University of Pittsburgh, Pittsburgh, PA 15213, USA

¹⁰Department of Pediatrics, Section of Allergy and Immunology, University of Colorado Anschutz Medical Campus, Aurora, CO 80045, USA

¹¹Department of Immunology and Microbiology, University of Colorado Anschutz Medical Campus, Aurora, CO 80045, USA

¹²Department of Medicine, Division of Medical Oncology, University of Colorado Anschutz Medical Campus, Aurora, CO 80045, USA

*Correspondence: eseohi.ehimiaghe@cuanschutz.edu (Eseohi Ehimiaghe)

Abstract

Artificial intelligence and computational biology are rapidly advancing, offering unprecedented opportunities to transform both ovarian cancer research and clinical care. However, limited understanding of how to optimally integrate the information these tools provide with existing clinical data has led to a lag in integration. This commentary emerges from a unique and focused ovarian cancer research conference that explored how these emerging tools and technologies (*i.e.*, artificial intelligence and computational biology) can be leveraged to address questions in pathology, develop new paradigms of tumor biology, and integrate precision medicine into clinical management of complex and rare subtypes of ovarian cancer. We highlight key ways in which systematic integration of Artificial intelligence (AI) and computational tools can be leveraged to improve outcomes in ovarian cancer as well as the limitations and risks of their application.

Keywords

Computational biology; Ovarian cancer; Artificial intelligence; Tumor microenvironment; Pathobiology; Machine learning; Carcinosarcoma

1. Introduction

Artificial intelligence (AI) is used across medicine for risk stratification, survival prediction, diagnosis, and multi-omics applications [1, 2]. In October 2024, the third Ovarian Cancer Innovations Group Think Tank was hosted at the University of Colorado Anschutz Medical Campus, with the theme “Exploring Unanswered Questions in Pathology and Computational Biology”. The goals were to review and discuss new computational research methods as applied to ovarian cancer biology

and to study applications of these to explore new treatments for a rare ovarian tumor, carcinosarcoma (CS).

2. Foundational concepts and research applications of big data

“AI” is a broad term for technological systems that use algorithms for problem-solving, predictive modeling, and insight generation. “Machine learning” is a subset of AI that leverages statistical algorithms to learn patterns in data and generalize

to information it has not been explicitly trained on. Some machine learning models (“supervised learning”) are trained on datasets with predefined “features” (independent variables used as inputs) and “targets” (dependent variables that correspond to features/feature sets), which seek to predict the correct target based on the input features. Other model types may be “unsupervised” or “semi-supervised”, insofar as the targets may be unknown or undefined. Deep learning is an advanced subset of machine learning that leverages multi-layered neural networks to uncover complex, non-linear relationships without the need for human intervention [3]. Clinicians and researchers are often drawn to the exciting possibilities of deep learning approaches, but these methods typically require very large datasets to be properly trained, and other methods are often more appropriate for analyzing the underlying biology of the smaller datasets typically found in ovarian cancer research.

2.1 Machine learning to deconvolve the tumor microenvironment

The tumor microenvironment (TME) plays a unique and dynamic role in ovarian cancer tumorigenesis, chemoresistance, and prognosis [4]. Hippen *et al.* [5] developed machine learning applications for defining high-grade serous carcinoma (HGSC) subtypes based on TME transcriptomic heterogeneity (*e.g.*, RNA-seq). Due to differences in sample processing and sequencing protocols, it can be difficult to integrate bulk and single cell analyses. Bulk analysis identifies patterns in RNA based on a mixture of cell populations, but risks obscuring TME heterogeneity, while single cell analysis explores all alterations in a single cell, but risks missing specific cell types (*e.g.*, macrophages, adipocytes) [6, 7]. Furthermore, single-cell experiments are much more expensive than bulk sequencing, which limits their use in capturing differences across large patient cohorts—something bulk experiments can do more feasibly. Integrating single-cell and bulk data to computationally estimate both cell type composition and cell type-specific gene expression from bulk samples combines the strengths of both approaches. This strategy provides a detailed, high-resolution view at a much lower cost, making it possible to study cell type specific variability across patients that would otherwise go undetected. This method of integration is referred to as deconvolution, as it deconvolves the original bulk sample into its cell type components. Although current deconvolution methods perform well in simulations [8], their real-world utility is limited by two key assumptions: (1) that bulk and single-cell data are generated under similar experimental conditions, and (2) that all relevant cell types are represented in the reference data. Davidson *et al.* [9] developed a machine learning model called Bulk Deconvolution with Domain Invariance (BuDDI), to better combine data from bulk and single cell samples [9] to overcome the assumption that bulk and single cell data come from similar experimental conditions. The BuDDI model learns cell-type-specific perturbation responses (*i.e.*, the difference the researcher is interested in assessing, such as sex-based or treatment status) by incorporating the variability in both bulk and single-cell RNA-seq measurements. The sources of variability used are (1) cell type proportions, (2) biological or technical variability, (3) “slack” or additional noise that is

not explicitly modeled, and (4) the selected perturbation effect. By learning each of these sources separately, BuDDI can tease apart their individual contributions and limit the effect of confounders on estimating cell type composition and cell-type specific expression, which makes it especially effective for analyzing complex and noisy data, such as heterogeneous tumor samples.

2.2 The use of multiomics to better understand gynecologic carcinosarcomas

BuDDI is just one example of an effective machine learning approach to integrate multiple data sources and types (“multi-omics”) in large, complex datasets. The use of multiomics is especially important in rarer and more complex gynecologic malignancies whose phenotypic and clinical features are distinct from the more common histotypes [7]. This is especially relevant for tumors with multiple histologic features, such as carcinosarcomas (CSs), which have previously been described as two tumors. Data suggests that uterine and tubo-ovarian CSs are dedifferentiated carcinomas arising from a single malignant clone with the ability to undergo both epithelial-mesenchymal transition and mesenchymal-epithelial transition [10]. Given their complex histological classification, patients with these tumors have often been excluded from clinical trials, which has contributed to the limited data available on current treatment options. Whether uterine and tubo-ovarian CS are considered one tumor or two, CSs are a complex tumor type requiring more study. Increased availability of big data and multiomics approaches can be leveraged to better characterize the observed heterogeneous and complex features of this malignancy, especially when incorporating spatial organization analysis and machine learning algorithms. There may be a role for deconvolution approaches to expand the available number of cell-type-specific measurements across multiple patients and compensate for the limited availability of single-cell data for investigating the cell type heterogeneity inherent in CSs. The heterogeneity of ovarian tumors poses significant challenges for traditional bulk RNA-sequencing analysis, which is particularly relevant in CS, where comparing the sarcomatous and carcinomatous components may offer insights. One single-cell RNA sequencing approach has proved particularly useful in understanding the role of epigenetic modifiers in CS proliferation and invasion [11]. Two epigenetic effector genes, histone deacetylases 2 (HDAC2) and metastasis tumor antigen 3 (MTA3), were identified via clustering analysis, and both are elevated in sarcomatous and carcinomatous components. HDAC2 and MTA3 are subunits of the nucleosome remodeling deacetylase complex [12]. By leveraging the aberrant epigenetic regulation, the HDAC2/MTA3 complex is an oncologic driver of the metaplastic differentiation that results in both tubo-ovarian and uterine CS. Thus, the HDAC2/MTA3 axis may provide a therapeutic vulnerability. A top priority for further investigation is the identification of drugs and mechanisms that can disrupt the epithelial-mesenchymal transition, as these pathways appear the most promising for inhibiting tumor growth.

2.3 Limitations of predictive modeling of patients with HGSC

Although big data and multiomic approaches promise greater insight into complex, high-dimensional tumor and TME data, the success of any analysis relies on including data sources that are meaningfully related. This was exemplified by work completed by Jordan *et al.* [13], who used a random forest approach and Nanostring transcriptome data before and after chemotherapy, to identify genes and pathways predicted to contribute to progression-free survival (PFS) in patients with HGSC. Random forests are a powerful machine learning method used widely in science that can flexibly handle the high-dimensional data produced by transcriptomic platforms (a frequent challenge in machine learning) [14]. However, in this study, the random forest method failed to identify genes that could successfully predict PFS outcomes. They also evaluated the random forest's accuracy for predicting a simple question—whether variations in biomarker expression can predict PFS of less than or greater than 8 months. This was also largely unsuccessful: the receiver operator curve for predicting PFS with an area under the curve (AUC) value of 0.56, only slightly better than a random guess [13]. This may be due to a weak relationship between transcriptome information and PFS, necessitating new hypotheses. However, alternative explanations include possible data shortcomings, *e.g.*, there may be other important biomarkers that influence PFS that are not captured by NanoString. Also, given HGSC TME heterogeneity, there may have been important differences in the pre- and post-treatment sampling location (*i.e.*, peritoneum *vs.* omentum) that introduced noise into the comparisons. While no actionable relationship was uncovered through this study, understanding the limitations of the data and analytical design may help generate further hypotheses and develop new approaches to this prediction question.

3. AI integration into pathobiology for the designing of novel therapeutics

Although this specific application of a random forest was not successful in uncovering relationships between transcriptome data and PFS, there are many tools available and unanswered questions to investigate that can continue to transform our understanding of ovarian cancer pathobiology. Computer algorithms, unlike humans, excel at quantifying both visible features conceptualizing intricate patterns. The strength of computational tools lies in their ability to handle large-scale, integrative quantitation (processing and recalling complex datasets) [15]. For instance, machine learning approaches identified correlations between stromal cell ratios and reduced platinum survival outcomes [16], which was subsequently validated through traditional methods [17]. AI models promise even deeper insights by enabling analyses of increasingly complex datasets (*e.g.*, spatial transcriptomics, multiparameter imaging) and uncovering subtle spatial patterns [18]. Work by Xu *et al.* [19] has shown how machine learning-based spatial analysis conducted can identify early recurrence patterns that were linked to changes in T cell localization, malformed tertiary lymphoid-like structures, and an increase

in podoplanin-positive cancer-associated fibroblasts that entrap plasma cells [19]. These findings highlight the potential of spatial analyses to uncover critical, but previously obscured, patterns that could inform novel drug combinations and treatment strategies [13]. Additionally, incorporating AI and computer vision tools into pathology workflows to decode patterns with clinical relevance.

4. Artificial intelligence and clinical applications

AI's benefits are not strictly limited to the laboratory, however. AI has clinical implications that are being applied in real time. Integrating AI and large language models (LLMs) as tools that help with documentation, billing, patient communication, and complex clinical decision making can decrease physician burnout and mitigate access barriers to allow for more equitable care [20–22]. Elsewhere, combinations of voice recognition software and LLMs can serve as an AI scribe and may improve patient satisfaction, with one study reporting that patients felt they received more personal attention when physicians used this type of tool [23, 24]. Several LLMs have been developed to abstract charts, saving physicians considerable time reading extensive and sometimes redundant patient files [25].

5. Discussion

AI is quickly being integrated into all aspects of medicine. The research presented at the University of Colorado's Ovarian Cancer Innovations Group Think Tank meeting focused on opportunities to integrate pathology, molecular, and mutational studies in treatment of ovarian cancers. The Think Tank gathered experts in gynecologic oncology, pathology, and computational biology to discuss advancements in machine learning, precision medicine and ways to incorporate these tools in clinical medicine and promote innovation in research on ovarian cancer.

BuDDI and other similar tools show how modeling data expression can be used to better model ovarian cancers, which have heterogeneous and complex tumor environments. Similarly, spatial transcriptome technologies enable protein and RNA analyses at a single-cell resolution, precise phenotype-genotype correlations, insights into the spatial organization of diverse cell subsets, and a deeper understanding of ligand-receptor interactions. These technologies are being implemented to define tumor remodeling and gene expression patterns induced by therapeutic interventions to inform subsequent therapies [13]. This is especially important as we investigate pathways and potential targets for complex and rare gynecologic malignancies, such as uterine and tubo-ovarian CSs.

While there are many potential roles for AI in research, challenges persist. Successful predictive transcriptomic tools rely on choosing the correct starting hypothesis and properly sorting through heterogeneous and large datasets. Similarly, AI-identified patterns may reside in the TME rather than cancer cells, limiting their ability to reliably define cancer phenotypes or predict prognosis. Without physician/scientist supervision,

AI systems can produce datasets rich in redundant or irrelevant information, necessitating rigorous curation to ensure actionable insights.

This is especially important in clinical applications. We previously described the value of LLMs in clinical practice; however, we must also acknowledge their limitations. While patients and providers appreciate AI in clinical practice, the use of AI to make or assist in clinical practice is in its nascency. Several potential limitations may arise during the adoption of AI tools. First, hallucinations, the generation of untrue or misleading information by LLMs that form due to limited or incomplete models, are relatively common. For example, one AI scribe tool recorded that a patient had received a prostate exam, when the physician had merely recommended scheduling one—a serious clinical error [24, 26]. Second, LLMs are non-deterministic, meaning they do not always provide the same answer to the same prompt, and the response may drift over time as new information is integrated into the model [27]. Therefore, these models are currently suited only to answer highly specific, context-dependent questions and should only be integrated as one component of a decision-making system. Lastly, the human element of gynecologic oncology care is critical and unlikely to be replaced by chatbots or other AI applications [24, 27]. Clinicians and researchers must validate findings and navigate ethical considerations, including Health Insurance Portability and Accountability Act (HIPAA) regulations, which can restrict the creation of publicly accessible datasets.

While our meeting centered on applying AI to precision medicine, pathology, and clinical practice, these represent only a subset of AI's potential in ovarian cancer research. Research efforts also focus on developing clinically impactful diagnostic tools for early detection, risk assessment, and treatment response. For early detection, machine learning models trained on lipidomic [28], circulating tumor DNA [29], and proteomic [30] data show early promise in predicting earlier stages of ovarian cancer in symptomatic women through minimally invasive diagnostic. For risk assessment and treatment response prediction, recent models have begun to focus on integration of several different types of measures, which may include as clinical, computer tomography (CT) scans, magnetic resonance imaging (MRI) images, hematoxylin and eosin (H&E) whole slide images, and molecular measurements to predict response to treatment [31–33]. While promising, these models remain in the early stages of development and must be validated in larger, prospective cohorts before they can be integrated into clinical practice.

6. Conclusions

AI and its use in clinical practice and research are rapidly evolving areas in gynecologic oncology that offer immense promise. This is especially encouraging given the inherent heterogeneity of ovarian cancer, which demands the use of large, diverse datasets to effectively advance precision medicine for patients. Our commentary highlights key areas where AI can be leveraged to advance clinical research and care, such as multiomic data integration, computer-aided pathobiology for novel therapeutics, and LLM use in clinical applications. We

also highlighted shortcoming of AI approaches, specifically that the success of AI relies on correct hypotheses, appropriate data inputted into the AI model, and the ability to filter through redundant and irrelevant information. New AI models are continually being developed, but often without the input of basic science researchers and clinicians. To fully realize the potential of AI in ovarian cancer clinical care, it is imperative that researchers and clinicians actively participate in the development of the protocols and databases that are used to design artificial intelligence systems to ensure they are developed to best benefit patients.

ABBREVIATIONS

AI, Artificial intelligence; CS, carcinosarcoma; TME, tumor microenvironment; HGSC, high-grade serous carcinoma; BuDDI, Bulk Deconvolution with Domain Invariance; PFS, progression-free survival; LLMs, large language models; HDAC2, histone deacetylases 2; MTA3, metastasis tumor antigen 3; AUC, area under the curve; CT, computer tomography; MRI, magnetic resonance imaging; H&E, hematoxylin and eosin; HIPAA, Health Insurance Portability and Accountability Act.

AVAILABILITY OF DATA AND MATERIALS

Not applicable.

AUTHOR CONTRIBUTIONS

EE, MDP, BRC, SO, ST—Writing—Original Draft, Writing—Review & Editing. HD—Original Draft, Writing—Review & Editing, Visualization. DS, RJW, AC, EWYH, ND, MW, MRM—Review & Editing. FI—Conceptualization, Project administration. BGB—Conceptualization, Writing—Review & Editing, Project administration. SRG—Conceptualization, Supervision. LWB—Review & Editing, Project administration. KB—Conceptualization, Writing—Original Draft, Writing—Review & Editing, Supervision. All authors have read and agreed to the published version of the manuscript.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

Not applicable

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CONFLICT OF INTEREST

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