SELEZIONE PUBBLICA FINALIZZATA AL CONFERIMENTO DI N. 1 ASSEGNO DI RICERCA NEL

PROGRAMMA N.11 **DI CUI AL D.R.5140 del 24.10.2024**

VERBALE DELLA TERZA SEDUTA

Il giorno 12.12.2024 alle ore 14,30 presso il laboratorio di Oncologia Cellulare- Sezione Biologia-DIMES, IST-NORD torre C 3° piano, Largo R. Benzi 10, Genova ha luogo la terza riunione della Commissione giudicatrice della selezione di cui al titolo per lo svolgimento del colloquio da parte dei candidati ammessi.

Il locale utilizzato è aperto al pubblico.

La Commissione ricorda che per la selezione pubblica di cui al titolo, il bando prevede la possibilità di svolgimento del colloquio in modalità telematica (videoconferenza per mezzo di SKYPE), per i candidati residenti o domiciliati fuori dal territorio italiano e per coloro che risiedono o hanno il domicilio abituale oltre i 300 Km. di distanza dalla sede della selezione, che ne abbiano fatto richiesta.

Risultano presenti i seguenti candidati di cui si accerta l'identità personale:

	Nome e cognome	Documento di identità	numero	scadenza
1	**Grasselli Sara**	Passaporto	YA8685243	27-2-2026

La Commissione al completo procede al colloquio con i candidati, singolarmente, sugli argomenti previsti dal bando per il programma di ricerca di cui al titolo.

Al termine del colloquio di ciascun candidato, usciti i presenti e prima dell'inizio della prova del candidato successivo, viene attribuito il punteggio di cui all'allegato *B* che fa parte integrante del presente verbale.

Al termine delle prove, sulla base punteggi attribuiti ai titoli e colloquio,

la Commissione indica il Dott. **Grasselli Sara** quale vincitore della selezione e redige la seguente graduatoria di merito:

CANDIDATO	PUNTEGGIO TOTALE
Grasselli Sara	**75/100**

La Commissione provvede a rendere noti i risultati del colloquio mediante pubblicazione sul sito web del Dipartimento ovvero affissione alla sede degli esami.

La seduta è tolta alle ore 15

La Commissione:

Prof. Chiara Gentili

CHIARA GENTILI
Università degli Studi di Genova
12.12.2024
16:23:13
GMT+02:00

Prof. Sara Tavella

SARA TAVELLA
Università degli Studi di Genova
12.12.2024
16:29:18
GMT+02:00

Prof. Roberta Tasso

ROBERTA TASSO
Università degli Studi di Genova
12.12.2024
16:36:11
GMT+02:00

ALLEGATO B

PUNTEGGI ATTRIBUITI AL COLLOQUIO

CANDIDATO	PUNTEGGIO
Grasselli Sara	**58/60**

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Scaffold-based 3D cellular models mimicking the heterogeneity of osteosarcoma stem cell niche

Giada Bassi, Silvia Panseri, Samuele Maria Dozio, Monica Sandri, Elisabetta Campodoni, Massimiliano Dapporto, Simone Sprio, Anna Tampieri & Monica Montesi

The failure of the osteosarcoma conventional therapies leads to the growing need for novel therapeutic strategies. The lack of specificity for the Cancer Stem Cells (CSCs) population has been recently identified as the main limitation in the current therapies. Moreover, the traditional two-dimensional (2D) in vitro models, employed in the drug testing and screening as well as in the study of cell and molecular biology, are affected by a poor in vitro-in vivo translation ability. To overcome these limitations, this work provides two tumour engineering approaches as new tools to address osteosarcoma and improve therapy outcomes. In detail, two different hydroxyapatite-based bone-mimicking scaffolds were used to recapitulate aspects of the in vivo tumour microenvironment, focusing on CSCs niche. The biological performance of human osteosarcoma cell lines (MG63 and SAOS-2) and enriched-CSCs were deeply analysed in these complex cell culture models. The results highlight the fundamental role of the tumour microenvironment proving the mimicry of osteosarcoma stem cell niche by the use of CSCs together with the biomimetic scaffolds, compared to conventional 2D culture systems. These advanced 3D cell culture in vitro tumour models could improve the predictivity of preclinical studies and strongly enhance the clinical translation.

Osteosarcoma is the most common primary malignant tumour of the bone[1], frequently presenting in young people between the ages of 10–14 years and in adults over 65 years[2]. Typically, patients with osteosarcoma are subjected to a combination of surgery, radiotherapy and chemotherapy[3], which together can lead to a survival rate improvement of another 5 years in patients with localised tumour[4–6]. However, while overall survival rate of non-metastatic tumour is about 60–70%, it remains less than 20% for patients with metastasis[7]. Moreover, tumour recurrences occur in 30–40% of non-metastatic patients[8]. Unfortunately, this scenario remained unchanged over the last 40 years, underlying the need of novel therapeutic strategies[9–11]. The therapeutic failure against osteosarcoma is mainly due to two reasons: (1) the lack of specificity for Cancer Stem Cells (CSCs) and (2) the absence of 3D microenvironment models that recapitulate the tumour complexity. Recent data have confirmed that osteosarcoma contains a distinct and defined cell population of CSCs, exhibiting stem-like phenotype with spherical colonies forming ability, called sarcospheres[12], characterized by critical properties of invasiveness, migration and drug resistance[13,14], suggesting their involvement in tumour progression, metastasis and recurrences frequently observed in osteosarcoma patients[15]. CSCs reside in an anatomically distinct and defined region inside the tumour microenvironment, called niche[16,17], which controls CSC's fate through the mutual feedback between the different cells and the extracellular matrix (ECM)[18–21].

The second cause of the therapy failure is the lack of predictive in vitro models of the in vivo physio-pathological situation. Although many new tumour drug candidates seemed to be promising in in vitro screenings, they did not show any efficacy during in vivo studies[22]. This low translational ability can be attributed to the poor reliability of in vitro 2D standard models (i.e. plastic and glass surfaces)[23]. Although they are commonly used due to their controllability, simplicity, replicability and cheapness[24,25], they do not reproduce the human disease complexity like the physico-chemical and mechanical tissue properties, the inter- and intra-tumour heterogeneity, the drug penetration through the tissue, the interaction between tumour cells and the stroma cells and the CSCs niche[26–28]. Recently, to overcome this great limitation, a significant effort has been made to clarify the dynamic interaction between the two players, cells and ECM, leading to the Tissue Engineering's evolution into "Tumour Engineering", with the aim to engineer the 3D tumour microenvironment and better elucidate several biological events and discover disruptive therapies[29,30].

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