# Overexpression of a splice variant of oncostatin M receptor beta in human esophageal squamous carcinoma

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- Tasneem Kausar (1)
- Rinu Sharma (2)
- Md. Raghibul Hasan (1)
- Anoop Saraya (3)
- Tushar K. Chattopadhyay (4)
- Siddartha Datta Gupta (5)
- Ranju Ralhan (1) (6) (7) (8) (9) Email author (rralhan@mtsinai.on.ca)

1. Department of Biochemistry, All India Institute of Medical Sciences, , New Delhi, India
2. School of Biotechnology, Guru Gobind Singh Indraprastha University, , Delhi, India
3. Department of Gastroentology, All India Institute of Medical Sciences, , New Delhi, India
4. Department of Gastrointestinal Surgery, All India Institute of Medical Sciences, , New Delhi, India
5. Department of Pathology, All India Institute of Medical Sciences, , New Delhi, India
6. Joseph and Mildred Sonshine Family Centre for Head & Neck Diseases, Mount Sinai Hospital, Joseph & Wolf Lebovic Health Complex, , Toronto, Canada
7. Department of Otolaryngology— Head and Neck Surgery, Mount Sinai Hospital, , Toronto, Canada
8. Department of Pathology and Laboratory Medicine, Mount Sinai Hospital, , Toronto, Canada
9. Department of Otolaryngology, University of Toronto, , Toronto, Canada


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## Abstract

### Background

Expression of oncostatin M receptor beta (OSMRβ) has been reported in human cancers, however its role in esophageal squamous cell carcinoma (ESCC) remains unknown. Using differential display, earlier we reported the identification of an alternatively spliced variant of OSMRβ in ESCC. Here in we characterized this novel variant encoding a soluble form of this receptor (sOSMRβ) and determined its clinical significance and correlation with the expression of oncostatin (OSM) and leukemia inhibitory factor receptor beta (LIFR β) in ESCC.

## Materials and Methods

*In silico* analysis was carried out to characterize the differentially expressed transcript of OSMRβ and its expression was determined in ESCCs and matched normal esophageal tissues using semiquantitative RT-PCR. The expressions of both truncated and full length OSMRβ proteins were analyzed in ESCC tissues and patients' sera using western blotting and immunoprecipitation. By immunoprecipitation we have also shown direct interaction between sOSMRB and OSM. We also explored the relationship between expression of OSM and its receptors, OSMRβ and LIFRβ, in primary human ESCCs and normal epithelia using immunohistochemistry.

## Results

Overexpression of alternatively spliced OSMR β transcript was detected by RT-PCR in 9 of 11 ESCCs. Analysis of the soluble receptor revealed absence of sOSMRβ protein in esophageal tissues, however, immunoprecipitation and western blot analysis showed its presence in sera of ESCC patients further confirming expression of the alternatively spliced OSMR β in ESCC patients. Immunohistochemical analysis in tissue microarray (TMA) format showed expression of OSMR β, LIFR and OSM in 11/50 (23%), 47/50 (94%) and 47/50 (94%) ESCCs, respectively. Strong correlation was observed between cytoplasmic expression of LIFRβ and OSM in tumor cells ($p$ = 0.000, O.R = 50, 95%CI = 8–31.9), and nuclear expression of LIFRβ and OSM ($p$ = 0.039 OR = 3.1, 95% CI = 1.1–8.2), suggesting that LIFRβ serves as the major receptor in ESCCs.

## Conclusion

An alternatively spliced variant of OSMR transcribing a soluble form of this receptor has been characterized in ESCC. We speculate that the truncated OSMR characterized here in may act as a neutralizing receptor for OSM. Our immunohistochemical study showed that OSMRβ and its pathway is not activated in ESCCs.

# Keywords

Esophageal cancer Oncostatin M receptor β Oncostatin M
Leukemia inhibitory factor β sOSMR: a soluble form of oncostatin M receptor

Tasneem Kausar and Rinu Sharma have equal contribution.



# Notes

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