

Resource Summary Report

Generated by [NIF](#) on Aug 30, 2026

Louisiana State University in Shreveport Redox Molecular Signaling Core Facility

RRID:SCR_024778

Type: Tool

Proper Citation

Louisiana State University in Shreveport Redox Molecular Signaling Core Facility
(RRID:SCR_024778)

Resource Information

URL: <https://www.lsuhs.edu/centers/cardiovascular-diseases-and-sciences/cobre/cobre-core-facilities>

Proper Citation: Louisiana State University in Shreveport Redox Molecular Signaling Core Facility (RRID:SCR_024778)

Description: Facility includes Analytic Redox Biology and Molecular Signaling Sub-Cores. Provided services include measurements of reactive oxygen species, reactive nitrogen species, reactive sulfide species, protein, peptides, metabolites, and lipids in cell culture and tissue samples. High-performance liquid chromatography systems, coupled with UV-vis and fluorescence detectors to quantify cellular and mitochondrial superoxide production, hydrogen sulfide pools (free sulfide, sulfide bound to transitional metals, and sulfane sulfur), and thiols (glutathione, GSH/GSSG, cysteine, cystine, homocysteine, persulfides, and glutathionylation). Instruments include Sievers NO Analyzer (NOA 280i, GE), Agilent 8890 GC and 8335 sulfur chemiluminescence detector, Thermo Scientific Orbitrap Exploris 480 and SCIEX Triple Quad 7500 mass spectrometer. Molecular Signaling Sub-Core offers services for molecular cloning and site-directed mutagenesis, design and production of vectors for CRISPR/Cas9 modification of vascular cells, services for endothelial, smooth muscle, and cardiac myocyte cell isolation, for generation of vascular cell lines, and for lentivirus production for transient or stable modification of cardiovascular cells, access to equipment and expertise for studying cellular effects of hypoxia/reoxygenation injury (Coy Hypoxic Chamber, CLARIOstar Spectrofluorometer), for automated capillary Western blot analysis (ProteinSimple Jess Simple Western), and for high-resolution fluo-respirometry to study mitochondrial function (Oroboros O2k modular system).

Synonyms: , CoBRE Redox Molecular Signaling Core, Redox Molecular Signaling Core

Resource Type: access service resource, core facility, service resource

Keywords: ABRF, Analytic Redox Biology, Molecular Signaling,

Resource Name: Louisiana State University in Shreveport Redox Molecular Signaling Core Facility

Resource ID: SCR_024778

Alternate IDs: ABRF_2569

Alternate URLs: <https://coremarketplace.org/?FacilityID=2569&citation=1>

Record Creation Time: 20231212T050231+0000

Record Last Update: 20260829T183343+0000

Ratings and Alerts

No rating or validation information has been found for Louisiana State University in Shreveport Redox Molecular Signaling Core Facility.

No alerts have been found for Louisiana State University in Shreveport Redox Molecular Signaling Core Facility.

Data and Source Information

Source: [SciCrunch Registry](#)

Usage and Citation Metrics

We found 6 mentions in open access literature.

Listed below are recent publications. The full list is available at [NIF](#) .

Cardenas-Garza A, et al. (2025) Cardiomyocyte Glucocorticoid Receptors Exacerbate Stress Effects in Myocardial Ischemia Injury in Mice. *Cells*, 14(24).

Camarano J, et al. (2025) Hydrogen sulfide inhibits recruitment of monocyte-derived tumor associated macrophages in glioblastoma by downregulating CXCL12. *Redox biology*, 86, 103866.

Ben Dhaou C, et al. (2025) Integrin-specific signaling drives ER stress-dependent atherogenic endothelial activation. *Redox biology*, 88, 103911.

Pearson-Gallion B, et al. (2025) Fibronectin-dependent integrin signaling drives EphA2 expression in vascular smooth muscle cells. *American journal of physiology. Cell physiology*, 328(5), C1623.

Dhaou CB, et al. (2025) Integrin-Specific Signaling Drives ER Stress-Dependent Atherogenic Endothelial Activation. *bioRxiv : the preprint server for biology*.

Aishwarya R, et al. (2024) Diastolic dysfunction in Alzheimer's disease model mice is associated with A β -amyloid aggregate formation and mitochondrial dysfunction. *Scientific reports*, 14(1), 16715.