Guide for Student Biological Data Collection by First Approval

Introduction

The Student Data Competition hosted by First Approval invites undergraduate, graduate, and PhD students to submit their datasets for evaluation. This guide consolidates the submission process, requirements, and an exemplary format to assist participants in successfully completing their submissions.

Eligibility

- Open to students enrolled in undergraduate, graduate, or PhD programs.
- Datasets must be submitted on the First Approval platform by 15 September 2025.

Key Requirements:

- 1. Datasets must include detailed annotations explaining data acquisition and experimental specifics.
- 2. Research areas:
 - Research topics: biological science, biomedical science, biotechnology.
 - Specific areas: Molecular Biology and Biochemistry, Genetics, Cell Biology and Histology, Anatomy and Physiology Biophysics, Immunology, Neuroscience, Developmental biology, Biomedical Research, Biotechnology, Omics Technologies, Aging, Zoology, Botany and Mycology, Microbiology, Ecology, Behavioral Science.
- 3. Acceptable submissions:
 - Original datasets.
 - Replications of previous experiments.
 - Negative datasets (e.g., data refuting hypotheses).

- Previously published datasets are acceptable if:
 - i. New data have been added, and/or
 - ii. Prior annotations were insufficient for reuse (cite the original publication in such cases).

Evaluation Criteria

Submissions will be judged based on:

- Annotation Completeness: Clarity and thoroughness of dataset documentation.
- Data Accuracy: Reliability of the dataset.
- Novelty and Experimental Design Quality: Originality and scientific rigor.
- Potential for Reuse: Value for future research.

Prizes

Prizes will be awarded across **three main categories**: Undergraduate, Graduate, and PhD students. The prizes in each category are as follows:

First Place: \$1,000Second Place: \$500Third Place: \$200

• Fourth to Tenth Place (Merit Award): \$100 each

Additional special prizes include:

Best Negative Dataset: \$300Best Replication Dataset: \$300

• Special prizes from partner organizations may be added to the prize pool.

All those who advance to the final stage of the competition (**top 35%**) will be awarded a "Honorable Mention" certificate.

Instructions for claiming the prize will be sent to the winners after the results are announced. The prize money will be transferred to the winners in USDT.

Step-by-Step Submission Process

I. Registration

1. **Sign up** at <u>First Approval</u>.

Sign up for free

Join the future of scientific discovery today

Email

Continue with email →

or

or

Already have an account? Log in

By clicking "Continue with Email/Google/ORCID/Facebook/Linkedin" above, you acknowledge that you have read and understood, and agree to Terms & Conditions and

2. Enter your name in the designated field.

Welcome

To start, what's your name?

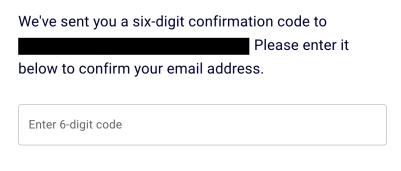


3. Create a password.



4. Verify your email address.

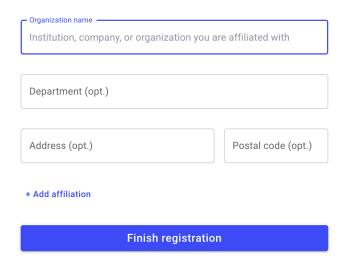
Check your email



Send code again

5. Provide **your affiliations** in the corresponding section.

Almost there! List your current affiliations:



II. Submission

- 1. Log into your First Approval account.
- On the Student Data Competition page (https://firstapproval.io/contest), press the "Apply Now" button.



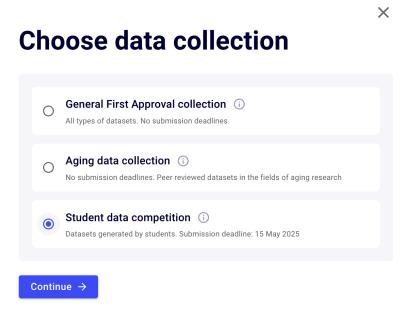
Student Biological Data Competition

First Approval is excited to announce a competition for student datasets to promote new scientific practices in publishing data within the fields of biology, biotechnology, and biomedicine. This competition aims to train students in the art of data publication, encourage the reuse of scientific data, and introduce them to decentralized solutions in science.



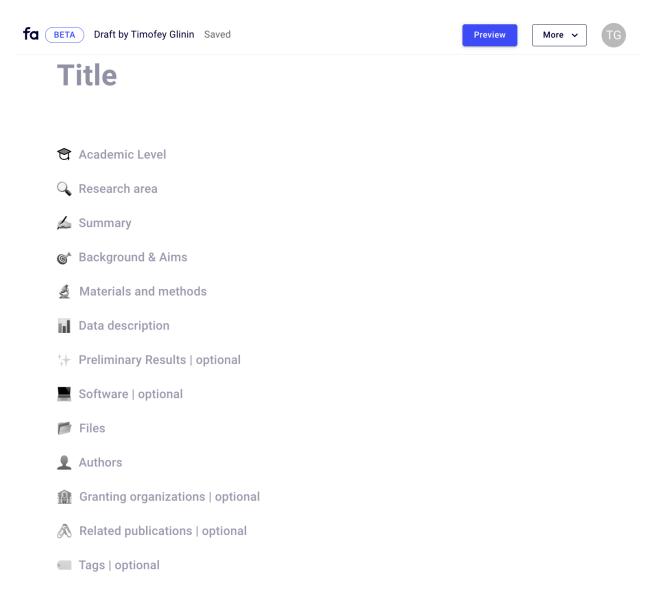
Or click the "Publish" button in the top-right corner of the homepage to start a new submission.

3. Select "Student data collection" as your submission type.



- 4. Complete all required sections.
 - Complete all sections of the submission form, including dataset annotation, detailed descriptions, and supplementary files.

- Progress will be automatically saved in the "Draft" section if needed.
- Set publication preferences in the "Publishing Conditions" section.



Make sure you have completed the annotation according to the **Dataset Annotation**Structure provided below.

Dataset Annotation Structure:

- o Title: Maximum 200 characters.
- Academic level: Make sure to indicate your participant category: Undergraduate student (or Bachelor student); Graduate student (or Master student); PhD student

- Summary: Up to 1,500 characters detailing the experiment background and aims, methods, and dataset description.
- Background & Aims: Describe the research objectives and previous relevant studies. Describe the aim of this experiment.
- Materials and Methods: Provide a comprehensive description of your study design, experimental procedures, controls, reagents, and any software used.
- Data Description: Explain dataset structure, formats, and quality assurance methods.
- **Tags**: optionally put the keywords that characterize your research
- 6. Upload all relevant data files, including:
 - o Raw and processed data.
 - Supporting materials such as images, diagrams, or supplementary files.



7. Click "Preview" to ensure all details are correct. Once you submit your dataset, no further changes can be made.



8. **Finalize your submission** by clicking "**Publish.**" Once the competition results are announced, your submission (if properly formatted) will be published on First Approval and assigned a DOI.

Additional Notes

- Each participant will receive a first-authored Open Access publication with a DOI and PDF version.
- All submissions will be reviewed and, upon acceptance, published after the competition's conclusion.
- Multiple submissions are allowed. In co-authored submissions, the prize will be awarded to the first author.

Helpful Links

• Competition Information Letter

Contact Information

For inquiries, email: competition@firstapproval.io

Example Dataset Format

Title: Evaluation of Reactive Oxygen Species (ROS) production in Endothelial cells (ECs) in response to COVID-19 patients serum.

Research Area: Medicine, Immunology and Allergy, Infectious Diseases, Cardiology and Cardiovascular Medicine

Summary: Oxidative stress and endothelial dysfunction have been shown to play crucial roles in the pathophysiology of COVID-19 (coronavirus disease 2019)(1,2,3,4). We hypothesized that oxidative stress and lipid peroxidation induced by COVID-19 in endothelial cells could be linked to the disease outcome. Thus, we collected serum from COVID-19 patients on hospital admission, and we incubated these sera with human endothelial cells, comparing the effects on the generation of reactive oxygen species (ROS) between patients who survived and patients who did not survive. We found that the serum from non-survivors significantly increased ROS production. Our data indicate that serum from patients who did not survive COVID-19 triggers ROS production in human endothelial cells.

Background & Aims: To find out if COVID-19 mortality correlates with increased ROS production in ECs. Serum from patients demised to COVID-19 will increase ROS production in ECs.

Materials and Methods:

Human umbilical vein endothelial cells.

Patients' samples

We obtained plasma samples of patients hospitalized with COVID-19 on the first day of hospital admission. Samples were divided into survivors (patients dismissed from the hospital) and non-survivors (N=22 and N=20, respectively). Mean age was 62±8.4 years and 74% were male in survivors' group and 63±14 years and 72% male in non-survivors' group. Mean time to death from blood sampling was 17.4±16.7 days in non-survival group. The study was approved by the Institutional Ethical Committee (IRB #202011756).

Cell Culture

Human umbilical vein endothelial cells (HUVECs) (Sigma, C-12205) were cultured in EGM-2 medium (Lonza, CC4147) and incubated at 37 °C and 5% CO2. Experiments on HUVECs were performed at passages 3-7. HUVECs were plated on glass bottom culture dishes (MatTek

Corporation, P35GCOL-0-10-C). When 70-80% confluent, the cells were treated with 10% patients' serum for 24h under normal condition (37 °C and 5% CO2). To prevent clot formation 10,000 U/mL Heparin (Sigma, H3393-100KU) was added to serum before the experiment.

Reactive oxygen species (ROS) assay.

ROS production was quantified 2'-7'-dichlorofluorescin diacetate (H2DCF-DA, InvitrogenTM, D399), as described previously (PMID: 20884348). Incubation for both fluorescent probes, as well as washing and imaging were done in a Krebs-Ringer solution (NaCl 115mM, KCl 5mM, NaHCO3 10mM, MgCl2 2.5mM, CaCl2 2 mM, HEPES 20 mM) supplemented with 10mM glucose. After 24h of treatment with 10% patients' serum, HUVECs were incubated with 2.5 µg/mL Hoechst 33342, trihydrochloride, trihydrate (InvitrogenTM, H21492) for 30 min, in the dark, at room temperature (RT). Then, HUVECs were washed once and incubated with 10µM H2DCF-DA for another 15 min RT, in the dark. Then HUVECs were washed 3 times and incubated without any fluorescent probes for another 15 min, RT in the dark. Immediately after this, cells were imaged by Nikon CSU-W1 Spinning Disk confocal microscope using a 40x objective (Nikon Corporation). Cells were excited with a laser at wavelengths 405 nm and 488 nm for Hoechst and H2DCF-DA respectively. Light emission was detected using 455/50 and 520/40 filters for Hoechst and H2DCF-DA respectively. The same settings (laser intensity, exposure time, pinhole width, etc) were used for imaging of both experimental groups. In order to prevent H2DCF-DA photodynamic reaction, fields of view search and focusing were performed using a Hoechst signal. Images were converted to .jpg format and quantification of H2DCF-DA fluorescence intensity was performed using ImageJ software (NIH).

Data Description:

File name consist of

"ExperimentalGroup-Probe1Name-Probe2Name-ObejectiveMagnification-PictureID.format"

Pictures with the same PictureID but different formats are the same picture.

Software:

Images were converted from .nd2 to .jpg format and quantification of H2DCF-DA fluorescence intensity was performed using ImageJ software (NIH).

Files:

Files	
▶ Download Preview sample	
NonSurv-DCF-Hoecst-40xjpg	
NonSurv-DCF-Hoecst-40xnd2	
NonSurv-DCF-Hoecst-40x-001.jpg	
NonSurv-DCF-Hoecst-40x-001.nd2	
NonSurv-DCF-Hoecst-40x-002.jpg	

Granting organizations:

The Santulli's Lab is supported in part by the National Institutes of Health (NIH): National Heart, Lung, and Blood Institute (NHLBI: R01-HL159062, R01-HL164772, R01-HL146691, T32-HL144456), National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK: R01-DK123259, R01-DK033823) (to G.S.), by the National Center for Advancing Translational Sciences (NCATS: UL1TR002556-06) (to G.S.), by the Diabetes Action Research and Education Foundation (to G.S.), and by the Monique Weill-Caulier and Irma T. Hirschl Trusts (to G.S.). S.S.J. is supported in part by a postdoctoral fellowship of the American Heart Association (AHA-21POST836407); U.K. is supported in part by a postdoctoral fellowship of the American Heart Association (AHA-22POST995561); and J.G. is supported in part by a postdoctoral fellowship of the American Heart Association (AHA-22POST995561); and J.G. is supported in part by a postdoctoral fellowship of the American Heart Association (AHA-20POST35211151).

Related publications:

Sardu C., Gambardella J., Morelli M.B., Wang X., Marfella R., Santulli G. Hypertension, Thrombosis, Kidney Failure, and Diabetes: Is COVID-19 an Endothelial Disease? A Comprehensive Evaluation of Clinical and Basic Evidence. J. Clin. Med. 2020;9:1417. doi: 10.3390/jcm9051417

Montiel V., Lobysheva I., Gérard L., Vermeersch M., Perez-Morga D., Castelein T., Mesland J.-B., Hantson P., Collienne C., Gruson D., et al. Oxidative stress-induced endothelial dysfunction and decreased vascular nitric oxide in COVID-19 patients. Ebiomedicine. 2022;77:103893. doi: 10.1016/j.ebiom.2022.103893.

Vardakas P., Skaperda Z., Tekos F., Kouretas D. ROS and COVID. Antioxidants. 2022;11:339. doi: 10.3390/antiox11020339.

Chernyak B.V., Popova E.N., Prikhodko A.S., Grebenchikov O.A., Zinovkina L.A., Zinovkin R.A. COVID-19 and Oxidative Stress. Biochem. (Moscow) 2020;85:1543–1553. doi: 10.1134/S0006297920120068

Primary article: https://doi.org/10.3390/antiox12020326

Tags:

Ros oxidative stress redox endothelial cells huvec covid19

See the sample dataset on First Approval: https://firstapproval.io/publication/BBFDWYD

Supplementary: What does the PDF of a dataset publication on First Approval look like?

first approval

Evaluation of Reactive Oxygen Species (ROS) production in Endothelial cells (ECs) in response to COVID-19 patients serum.

Jankauskas Stanislovas. Published online: 2023-10-31 https://firstapproval.io/publication/BBFDWYD https://doi.org/10.62251/fa:ds:BBFDWYD

Oxidative stress and endothelial dysfunction have been shown to play crucial roles in the pathophysiology of COVID-19 (coronavirus disease 2019)(1,2,3,4). We hypothesized that oxidative stress and lipid peroxidation induced by COVID-19 in endothelial cells could be linked to the disease outcome. Thus, we collected serum from COVID-19 patients on hospital admission, and we incubated these sera with human endothelial cells, comparing the effects on the generation of reactive oxygen species (ROS) between patients who survived and patients who did not survive. We found that the serum from non-survivors significantly increased ROS production. Our data indicate that serum from patients who did not survive COVID-19 triggers ROS production in human endothelial cells.

1 folders & 12 files - 75.64 MB

Unique archive cryptographic hash: SHA-256: 48c4129c125 507ffc555de69b1e844a42cbed780835cd457d9882ec16e5 8598a

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Authors

Jankauskas Stanislovas Albert Einstein College of Medicine Wilf Family

Granting organizations

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Related articles

Primary articles (publications based on this dataset): [https://doi.org/10.3390/antiox12020326]

- Sardu C., Gambardella J., Morelli M.B., Wang X., Marfella R., Santulli G. Hypertension, Thrombosis, Kidney Failure, and Diabetes: Is COVID-19 an Endothelial Disease? A Comprehensive Evaluation of Clinical and Basic Evidence. J. Clin. Med. 2020;9:1417. doi: 10.3390/jcm9051417
- Evidence. J. Clin. Med. 2020;9:1417. doi: 10.3390/jcm9051417

 2. Montiel V., Lobysheva I., Gérard L., Vermeersch M., Perez-Morga D., Castelein T., Mesland J.-B., Hantson P., Collienne C., Gruson D., et al. Oxidative stress-induced endothelial dysfunction and decreased vascular nitric oxide in CoVID-19 patients. Ebiomedicine. 2022;77:103893. doi: 10.1016/j.ebiom.2022.103893.
- Vardakas P., Skaperda Z., Tekos F., Kouretas D. ROS and COVID. Antioxidants. 2022;11:339. doi: 10.3390/antiox11020339.
- Chernyak B.V., Popova E.N., Prikhodko A.S., Grebenchikov O.A., Zinovkina L.A., Zinovkin R.A. COVID-19 and Oxidative Stress. Biochem. (Moscow) 2020;85:1543–1553. doi: 10.1134/S0006297920120068