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Data Article

Data on differential pathogenic ability of *Helicobacter pylori* isolated from distinct gastric niches

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ABSTRACT

Helicobacter pylori infection is associated with various gastrointestinal diseases and gastric cancer. Our data shows the *H. pylori* isolates and their associated pathology, isolated from two different stomach niches: gastric epithelium and gastric juice. Gastric adenocarcinoma (AGS) cells were infected with *H. pylori* juice (HJ1, HJ10 and HJ14) and biopsy (HB1, HB10 and HB14) isolates for 6, 12 and 24 hrs. To determine the cell migration ability of the infected cells, scratch wound assay was performed. The decrease in the wound area was measured by Image J software. Status of cell proliferation accessed by counting the cell number through trypan blue exclusion method. Further assessment of pathogenic potential and carcinogenic

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ability of the isolates was done by determining the genomic instability in the cell post infection. Cells were stained with DAPI and number of micro and macro nuclei was counted in the acquired images. The data will be helpful in understanding the difference in the carcinogenic ability of *H. pylori* with respect to their physiological niche.

Specifications Table

Subject	Biological sciences
Specific subject area	Microbiology: Bacteriology, Cancer Research
Type of data	Image Graph Figure
How the data were acquired	Wound healing and cell proliferation assay was performed on *H. pylori* infected AGS cells at 0, 6, 12, 24 hrs. The wound was imaged under a light microscope and the wound area was quantified by using ImageJ software. Proliferation assay was performed by counting the cells using a Neubauer chamber with the help of an inverted light microscope. Genomic instability in infected cells was evaluated by calculating the percentage of multinuclei and micronuclei. It was estimated by counting 200 cells in 10 different fields.
Data format	Raw: https://www.ebi.ac.uk/biostudies/studies/S-BSST996 Analyzed
Description of data collection	AGS cells were infected with *Helicobacter pylori* isolated previously [1] from gastric biopsy and juice of the suspected gastritis patients. The cells were then assessed for their proliferation, migration ability 6, 12 and 24 hours post infection. We have also assessed the genomic instability in the infected cells. The collected data has been submitted to Bio Studies. data repository and can be assessed through: https://www.ebi.ac.uk/biostudies/studies/S-BSST996
Data source location	Data repository link: https://www.ebi.ac.uk/biostudies/studies/S-BSST996 Submitted by: Hem Chandra Jha and Budhadev Baral Institution: Indian Institute of Technology Indore City/Town/Region: Indore, Madhya Pradesh Country: India
Data accessibility	Repository name: BioStudies. Data identification number: S-BSST996 Direct URL to data: https://www.ebi.ac.uk/biostudies/studies/S-BSST996

Value of the Data

- The data shows the variation of *Helicobacter pylori* characteristics and pathogenic ability based on their niche.
- The *H. pylori* isolate from the gastric epithelium and juice of the same patient may vary in their physiology and pathogenicity.
- It will help the clinicians and researchers in establishing a new approach to *H. pylori* research and ignite the scientific community to consider the pathogen niche as one of the contributing factors in pathology.

1. Objective

The objective of the data set is to determine the physiological properties of gastric epithelial cells exposed to *H. pylori*. The cell proliferation, cell migration and genomic instability assays help to decipher the aggressiveness of biopsy and juice isolate infected cells. The data will help to decipher the specific role of epithelial and juice *H. pylori* in GC occurrence and progression.

2. Data Description

Gastric cancer (GC) remains a concern globally due to its high occurrence and mortality rates [2]. Persistent gastritis induced by *Helicobacter pylori* (*H. pylori*) is the strongest known risk factor for GC [3]. *H. pylori* is prevalent in about 50% world's population, while it causes cancer in less than 2 % of exposed individuals [4]. In one of our study we have isolated *H. pylori* from the gastric biopsy and juice sample of suspected gastritis patients and found that the growth pattern of these bacteria can vary widely [1]. Notably, the bacteria isolated from the biopsy and juice sample of the same subject suggest distinctly different growth patterns and drug responses [1]. The data explains the difference of biopsy and juice *H. pylori* isolates based on their oncogenic potential like abnormal cell proliferation, migration, and genomic instability (https://www.ebi.ac.uk/biostudies/submissions/S-BSST996).

Fig. 1. Shows the migration and proliferation ability of *H. pylori* infected AGS cells. The migration ability was accessed by scratch wound assay. The percentage wound area was measured in post 6, 12 and 24 hrs infection. Wound area at 0 hrs were considered 100% and the wound area in the respective time points were calculated accordingly. All the raw images from where the data was measured and the raw data can be obtained from the https://www.ebi.ac.uk/biostudies/studies/S-BSST996. The Cell proliferation post infection was determined by cell proliferation assay using trypan blue exclusion method.

Fig. 2. Describes the genomic instability induced by the *H. pylori* infection at 6, 12 and 24 hrs post infection. The genomic instability was measured by counting the no. of cells with multi and micronuclei.

Fig. 3. Shows the representative image of genomic instability in AGS cells post *H. pylori* infection at all the 3 studied time points (6, 12 and 24 Hrs).

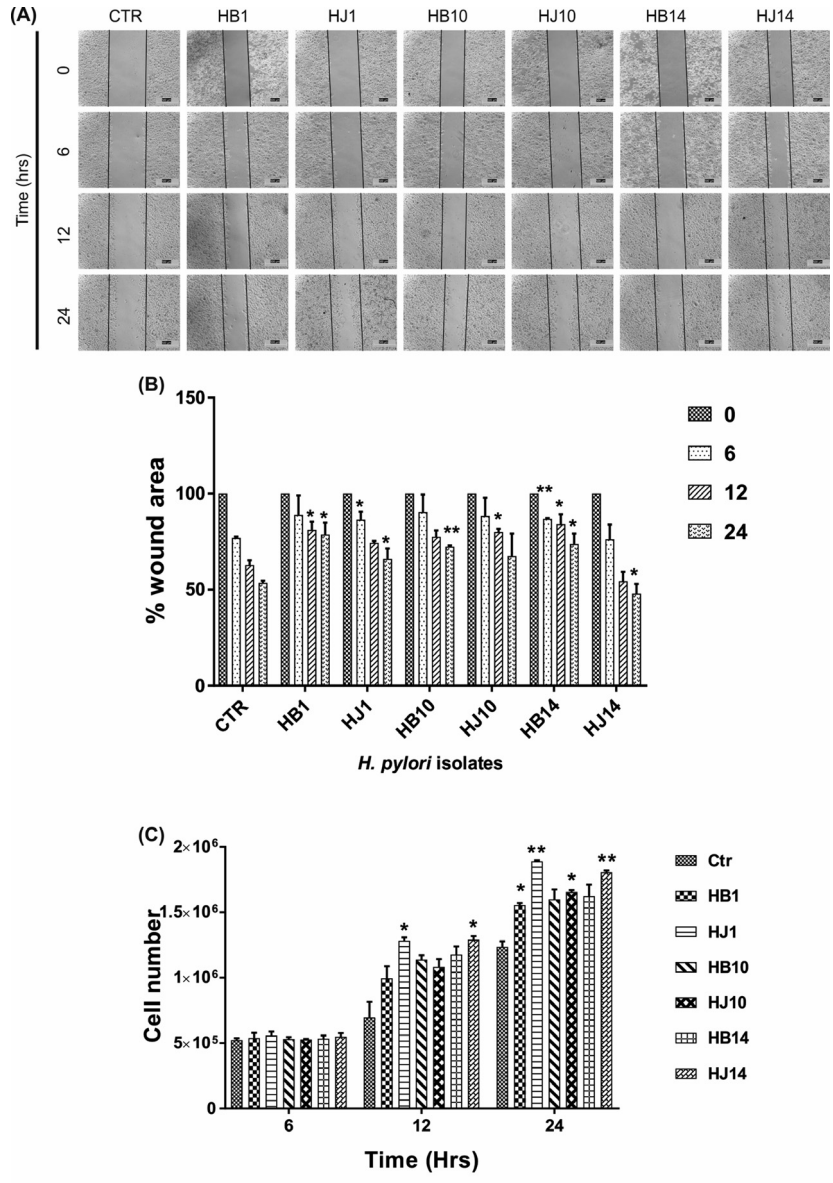

Fig. 1. Migration and proliferation ability of *H. pylori* infected AGS cells. Scratch wound assays were performed to determine the migration ability of the AGS cells after *H. pylori* infection. Images were acquired by using an inverted light microscope (Leica Ltd.) at 5X objective lens and the percentage wound closer was determined by measuring the wound area through Image J software. Scale bar: 500μm. (A) Representative image of scratch wound in *H. pylori* (HB1, HJ1, HB10, HJ10, HB14, HJ14) infected AGS cells at 0, 6, 12 and 24 Hrs post infection. (B) Graphical representation of the % wound area in *H. pylori* infected AGS cells at 0, 6, 12, 24 hrs post infection. Wound area at 0 hrs were considered 100% and the wound area in the respective time points were calculated accordingly. (C) Proliferation ability of the AGS cell was assessed by cell proliferation assay using trypan blue exclusion method. The graph represents the increase in number of cells after *H. pylori* infection at 6, 12 and 24 hrs post infection. The experiment was performed in triplicates, and the results are shown as the mean ± SD for three data points. Unpaired T tests were applied to determine the statistical significance. $p<0.05$ was considered significant in all the cases. p-values of <0.05, <0.01 and <0.0001 were represented with *, ** and *** respectively.

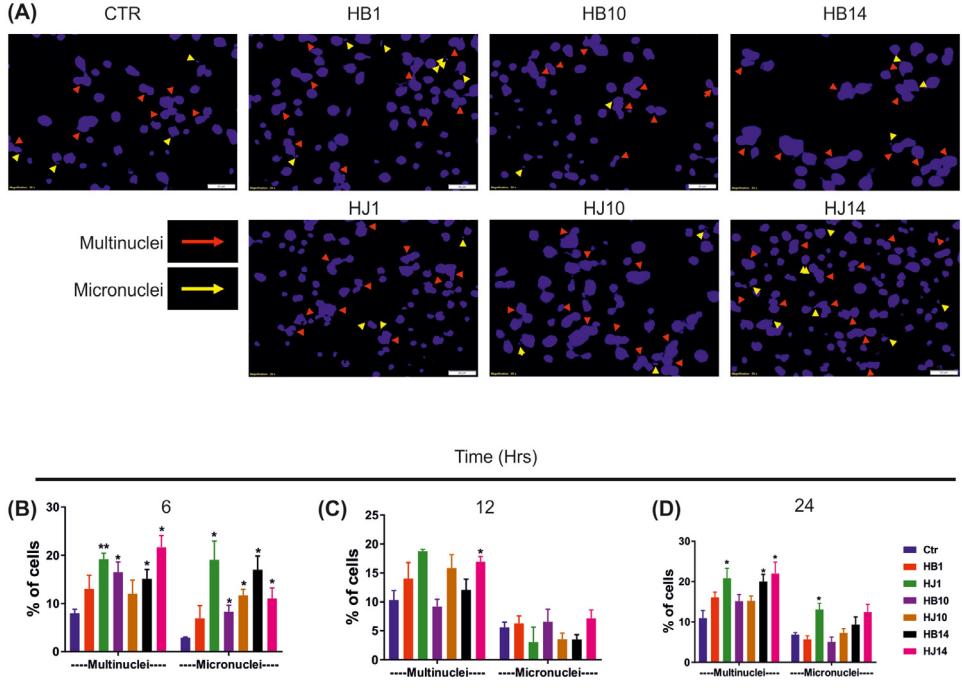

Fig. 2. Multi and micronuclei formation in AGS cell post *H. pylori* Infection. Genomic instability assay was performed to assess the formation of multi and micronuclei in AGS cell post *H. pylori* infection. AGS cells were infected with *H. pylori* isolates (HB1, HJ1, HB10, HJ10, HB14 and HJ14) for 6, 12 and 24 hrs and cells were stained with DAPI post fixation with PFA. Image was acquired at 20X objective lens magnification by using fluorescent microscope (Olympus IX83). Scale bar: 50μm. (A) Representative image of *H. pylori* infected AGS cells showing multinuclei (red arrow) and micronuclei (yellow arrow) after infection. (B) Graphical representation of multi and micronuclei formation in AGS cells, 6 hrs post infection. (C) Graphical representation of multi and micronuclei formation in AGS cells, 12 hrs post infection. (D) Graphical representation of multi and micronuclei formation in AGS cells, 24 hrs post infection. The experiment was performed in triplicates, and the results are shown as the mean ± SD. Unpaired T tests were applied to determine the statistical significance. $p<0.05$ was considered significant in all the cases. p-values of <0.05, <0.01 and <0.0001 were represented with *, ** and *** respectively.

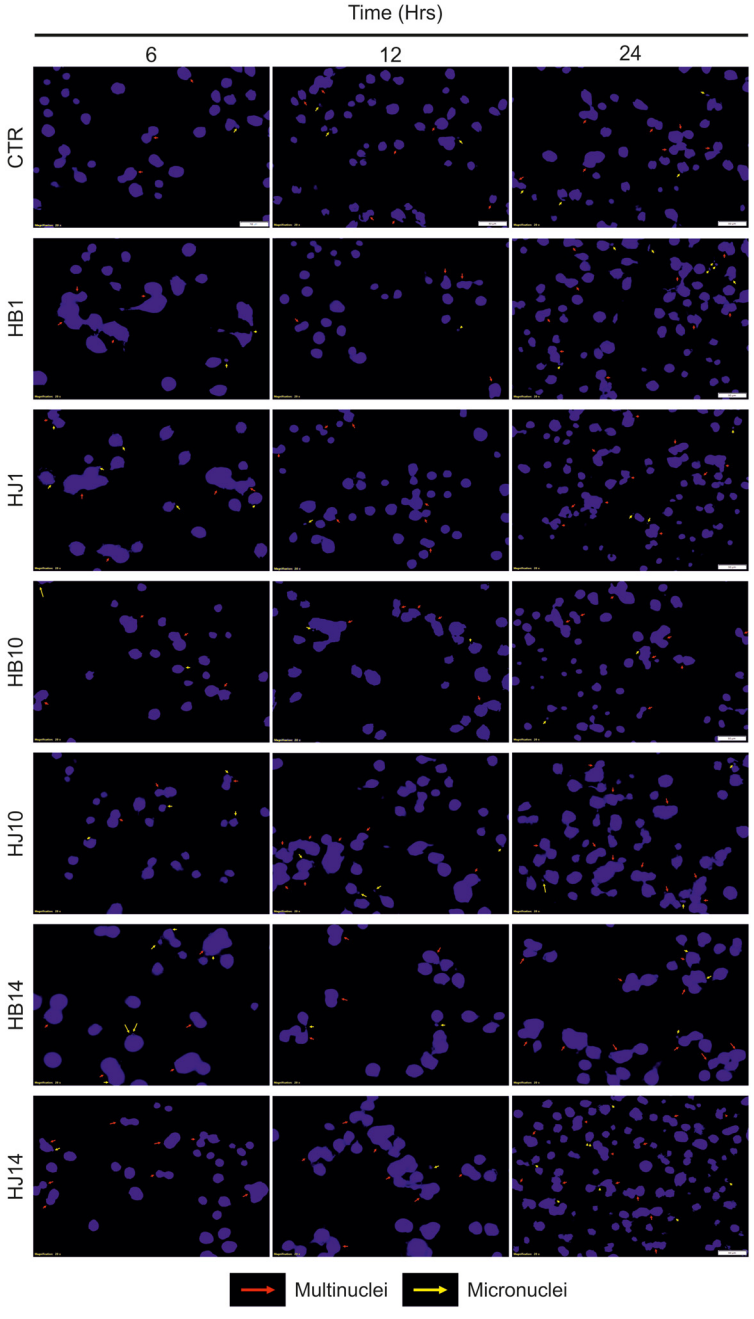

Fig. 3. Representative image of genomic instability in AGS cells post *H. pylori* infection. The AGS cells were infected with *H. pylori* (HB1, HJ1, HB10, HJ10, HB14, HJ14) *for* 6, 12 and 24 hrs followed by DAPI staining. Cells were imaged using a fluorescence microscope (Olympus IX83) at 20X objective magnification. Scale bar: 50μm Genomic instability in the cell was mapped as multi (red arrow) and micronuclei (yellow arrow) formation.

3. Experimental Design, Materials and Methods

3.1. Wound healing assay

To check the cancerous properties of AGS cells after exposure to various *H. pylori* isolates, we performed the wound healing assay as described previously [5]. The cells were seeded into a 6-well plate and continuously cultured to 100% confluency to form a monolayer. A single-line wound was formed by scratching the cell monolayer with a 1000 μl pipette tip followed by PBS wash. The cells were then infected with the *H. pylori* isolates with uninfected cells as control. Images were captured after incubation for 0, 6, 12, 24 hrs. The wound was imaged under a light microscope (DM21, Leica microsystems, Wetzlar, Germany) and the wound area was quantified by using ImageJ software.

3.2. Cell proliferation assay

An assay of cell proliferation was performed through trypan blue exclusion methods as per standard protocol. Briefly, AGS cells were seeded (0.5×10^6 cells) in 6 well plates followed by infection at MOI-100 for 6, 12, and 24 hrs. After the incubation period cells were trypsinized and counted using a Neubauer chamber with the help of an inverted light microscope (DM21, Leica microsystems, Wetzlar, Germany).

3.3. Genomic instability assay

For the evaluation of genomic instability in infected cells, cells were seeded on a coverslip placed in 6 well plates followed by infection of *H. pylori* for 6, 12, and 24 hrs at MOI-100. After the completion of incubation, coverslips were washed with PBS and the cells were fixed with a fixative (4% paraformaldehyde [PFA] containing 0.1% Triton-100) for 30 min at room temperature (RT). For visualization, the cells were stained with 4′,6-diamidino-2-phenylindole (DAPI) for 1 hr at RT. The images were acquired under fluorescence microscopy (Olympus IX83, Olympus, Tokyo, Japan) for the presence of multinuclei and micronuclei. The percentage of multinuclei and micronuclei was estimated by counting 200 cells in 10 different fields

Ethics Statements

The work did not involve human subjects or animal experiments.

CRediT Author Statement

Hem Chandra Jha, Budhadev Baral and **Dharmendra Kashyap:** Conceptualization; **Budhadev Baral, Dharmendra Kashyap, Ajay Kumar Jain** and **Debi Chatterji:** Methodology; **Budhadev Baral and Dharmendra Kashyap** and **Nidhi Varshney:** Formal analysis, Investigation, **Budhadev Baral** and **Vinod Kumar:** Data analysis and optimization; **Budhadev Baral:** Writing – original draft preparation; **Budhadev Baral, Dharmendra Kashyap, Amit Mishra, Awanish Kumar** and **Anil Kumar Singh:** Writing – review & editing; **Tarun Prakash Verma** and **Budhadev Baral:** Writing – preparation of figures; **Hem Chandra Jha:** Funding acquisition; **Hem Chandra Jha** and **Ajay Kumar Jain:** Resources; **Hem Chandra Jha:** Supervision.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Data Availability

Data on differential pathogenic ability of Helicobacter pylori isolated from distinct gastric niches (Original data)

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Data reference

Jha HC, Baral B. Data on the pathogenic ability of Helicobacter pylori isolated from distinct gastric niches. BioStudies, S-BSST996, 2023. https://www.ebi.ac.uk/biostudies/studies/S-BSST996

Supplementary Materials

Supplementary material associated with this article can be found, in the online version, at doi:10.1016/j.dib.2023.108981.

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