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Research on the Value of G3BP1 Protein as a Biomarker in the Early Diagnosis of Gastric Cancer

Wanyue Zeng

Guangdong Experiment High School, Guangzhou, China
wanyue_z_2026@163.com

Abstract. Gastric cancer is one of the major issues for world health. Although patients have a poor five-year survival rate, early detection can significantly improve the prognosis. There are some issues with today's diagnostic techniques, such as positron emission tomography-computerized tomography (PET-CT) and endoscopic ultrasound (EUS). In this work, we investigate the G3BP1 protein as a potential biomarker for the early detection of gastric cancer. We use tumors from MKN1 xenograft mice and adjacent healthy tissues. We predicted that in cancer cells, HSU mRNA levels would decrease and G3BP1 would rise. In the experiments, we used the gastric cancer cell lines MGC-803 and AGS and the human stomach epithelial cell line GES-1. We created detecting systems like the luciferase and GFP systems. Confocal microscopy, Western blot analysis, cell transfection, and reverse transcription-polymerase chain reaction (RT-PCR) were among the experimental techniques used. There were eight combinations for the outcomes. The theory was completely supported by CR1. It showed how gastric cancer development may be influenced by both the increase in G3BP1 and the decrease in HSU mRNA. However, CR8 was entirely opposed to the notion, whereas CR2–CR7 only partially supported it. These findings indicate that there is a complicated interaction between G3BP1 and HSU mRNA in the gastric cancer development. In order to understand these interactions and clarify biomarkers for early diagnosis, we will adopt gene editing methods and multi-omics analysis in the future.

Keywords: G3BP1, Biomarker, Gastric Cancer, Early Diagnosis

1. Introduction

Gastric cancer is the fifth most common cancer in the world, and it has an extremely negative impact on public health. Around 0.8% of people all around the world have been diagnosed with stomach cancer, according to data from the World Health Organization, and the five-year survival rate for these individuals is only 20% [1]. However, there is hope because early diagnosis increases the survival percentage of patients to over 75% [1]. The early detection techniques are highlighted by this notable discrepancy.

The existing methods of diagnosing stomach cancer, like positron emission tomography (PET-CT) and endoscopic ultrasound (EUS), have some drawbacks. Using ultrasound technology, EUS entails putting an endoscope into the digestive tract to take pictures of the internal organs [2].

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However, it has trouble identifying early-stage cancers correctly, and its accuracy is almost dependent on the doctor's skill [2]. In contrast, patients have to drink the radioactive glucose markers prior to the PET-CT scan. This approach depends on the physician's interpretation abilities, even though it can assist physicians in seeing the distribution of markers in the body through CT imaging for diagnosis [3]. In addition, there may be health harm associated with the radioactivity of the markers, particularly for expectant mothers [3].

Biomarker testing appears as a definite alternative, aiming to overcome the drawbacks of traditional methods. Through a deep review of the literature, the G3BP1 protein has attracted much attention as a potential biomarker for gastric cancer. High levels of mRNA and protein of the G3BP1 gene are expressed in gastric cancer cells and highly represented in mRNA and protein compared to normal cells [4]. Besides stimulating the production of stress granules, G3BP1 binds the HSU mRNA and attracts proteins that degrade it [5]. Due to this feature, HSU mRNA can be exploited to measure the degree of G3BP1 expression to help detect stomach cancer [6].

This work examines the possibility of using G3BP1 as a biomarker for the early detection of stomach cancer. Therefore, we designed two detection systems, the GFP system and the luciferase system, to investigate the relationship between the G3BP1 expression level and gastric cancer. It is expected that our research will help in developing a more accurate and safer diagnosis of gastric cancer for improved prognosis of the patients.

Predict that G3BP1 increases and HSU mRNA levels decrease in stomach cells with cancer compared to surrounding healthy tissue using MKN1 xenograft mouse tumors as a hypothesis.

2. Materials and methods

2.1. Method 1: western blot analysis

Equipment: Protein electrophoresis equipment for molecular weight-based protein separation. It comes with gels (such as 10% SDS-PAGE gels), an electrophoresis chamber, and a power source. Proteins are moved from the gel to the membrane using transfer equipment, such as a semi-dry transfer unit.

Imaging system for detecting and analyzing protein bands: ChemiDoc XRS+ imaging system.

Materials: Tissue or cell samples, including nearby healthy tissues and stomach cancer xenografts.

Protease inhibitors (Roche) used in RIPA buffer (Beyotime) to break down cells and liberate proteins are known as lysis buffer.

Thermo Fisher Scientific's Bicinchoninic Acid (BCA) Protein Assay Kit is a protein quantification tool used to measure protein content.

Antibodies: diluted 1:1000 for western blot, rabbit anti-human G3BP1 polyclonal antibody (Abcam, ab12345). Internal reference: mouse anti-β-actin monoclonal antibody (Sigma-Aldrich, A5441), diluted 1:5000. Goat anti-rabbit IgG (ZSGB-BIO, ZB-2301) and goat anti-mouse IgG (ZSGB-BIO, ZB-2305) conjugated with horseradish peroxidase (HRP) were both diluted 1:5000 for detection.

Membranes and gels: polyvinylidene fluoride (PVDF) membranes (Millipore) for protein transfer and 10% SDS-PAGE gels for electrophoresis.

Buffers for blocking and washing: Tris-buffered saline with 5% non-fat milk and Tween 20 (TBST) for blocking and TBST for washing.

Thermo Fisher Scientific's enhanced chemiluminescence (ECL) kit is the detection reagent.

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Standard Operating Procedures: Protein extraction involves lysing cells or tissues in RIPA buffer with protease inhibitors on ice for half an hour after washing them with cold PBS. Use a high-speed centrifuge (e.g., 12,000 rpm for 15 minutes at 4°C) to collect the protein-containing supernatant and pellet detritus.

Protein quantification: As directed by the manufacturer, use the BCA kit. To find the protein concentration, combine protein samples with BCA working reagent, incubate for 30 minutes at 37°C, and measure absorbance at 562 nm.

10% SDS-PAGE gels should be prepared. Fill the wells with equal quantities of protein (30–50 μg). Until the bromophenol blue dye reaches the bottom of the gel, run the gel at a steady voltage (e.g., 80 V for the stacking gel and 120 V for the resolving gel).

Protein transfer: Use a semi-dry transfer device to move proteins from the gel onto a PVDF membrane. For one to two hours, transfer at a steady current (e.g., 0.8–1.0 mA/cm²).

Blocking: Let the membrane sit at room temperature for one to two hours in 5% non-fat milk in TBST.

Antibody incubation: G3BP1 and β-actin primary antibodies should be incubated on the membrane for a whole night at 4°C. Use TBST to wash the membrane three times for ten minutes each time. After that, incubate for one to two hours at room temperature with secondary antibodies. Use TBST three more times to wash. Use the ECL kit for detection. To find protein bands, combine the two ingredients of the ECL reagent, apply it to the membrane, then either expose it to X-ray film or utilize the ChemiDoc XRS+ imaging equipment. Both positive and negative controls. Use a cell line that is known to express G3BP1 at high levels as a positive control (for example, a cell line transfected with a G3BP1 overexpression plasmid). This guarantees the appropriate operation of the antibody detection system.

Use a sample of cells that have only been exposed to PBS as a negative control. This aids in detecting background signals and non-specific antibody binding.

Comparative Groups: Compare the G3BP1 protein bands' intensities in neighboring healthy tissue samples and gastric cancer xenograft samples. G3BP1 expression levels may be accurately compared by using the β-actin protein bands in both types of samples as an internal reference to adjust protein loading.

2.2. Method 2: Reverse Transcription-Polymerase Chain Reaction (RT-PCR)

Tools: Centrifuge To separate ingredients in reaction mixes and RNA extraction. A thermal cycler is used to carry out PCR amplification and reverse transcription. A power source, an electrophoresis tank, and a gel casting device make up the gel electrophoresis system, which is used to separate PCR results. After gel electrophoresis, a UV transilluminator is used to see the DNA bands.

Materials: Tissue or cell samples, including nearby healthy tissues and stomach cancer xenografts. TRIzol reagent (Invitrogen) is an RNA extraction reagent used to isolate total RNA. PrimeScript RT reagent kit with gDNA eraser (TaKaRa) for reverse transcription.

PCR master mix: Taq polymerase, dNTPs, and buffer are included in the SYBR Green PCR Master Mix (TaKaRa).

First things first: created for GAPDH and HSU mRNA (internal reference). 5'-[sequence1]-3' and 5'-[sequence2]-3' are the forward and reverse primers for HSU mRNA, while 5'-[sequence3]-3' and 5'-[sequence4]-3' are the primers for GAPDH.

Agarose: Used to make agarose gels (typically 1.5% for the separation of PCR products).

Ethidium bromide: for seeing DNA in gels.

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Standard Operating Procedures: For RNA extraction, mix cell or tissue samples with TRIzol reagent, then let them sit at room temperature for five minutes. Centrifuge at high speed (e.g., 12,000 rpm for 15 minutes at 4°C) after adding the chloroform and vortexing. After moving the top aqueous phase to a fresh tube, mix with isopropanol and centrifuge once more. Rinse the RNA pellet with 75% ethanol, let it air dry, and then dissolve it in water without RNase.

Quantification of RNA and evaluation of quality: To determine the absorbance of the RNA sample, use a NanoDrop 2000 spectrophotometer at 260 and 280 nm. The purity of RNA is indicated by an A260/A280 ratio of 1.8 to 2.0.

RT transcription: Put 1 μg of total RNA, 4 μL of 5× 5× PrimeScript Buffer, 1 μL of PrimeScript RT Enzyme Mix I, 1 μL of gDNA Eraser, and RNase-free water into a 20 μL reaction system. In a heat cycler, incubate for 15 minutes at 37°C and then for 5 seconds at 85°C.

12.5 μL of SYBR Green PCR Master Mix, 0.5 μL of each forward and reverse primer (10 μM), 2 μL of cDNA template, and 9.5 μL of nuclease-free water should all be added to a 25 μL PCR reaction mixture before amplification begins. The PCR procedure consists of three minutes of pre-denaturation at 95°C, thirty cycles of 95°C, 55°C, and 72°C for 30 seconds each, and a final extension at 72°C for ten minutes.

Prepare a 1.5% agarose gel in TAE solution for gel electrophoresis. Place a DNA ladder and the PCR results onto the gel. For around 30 to 60 minutes, run the gel at a steady voltage (for example, 100 V). Use ethidium bromide to stain the gel, then use a UV transilluminator to see the DNA bands. Both positive and negative controls. Use a cDNA sample from cells that have a known high level of HSU mRNA expression as a positive control. For instance, cells that were transfected with a plasmid that overexpressed HSU mRNA. Use a reaction mixture without the cDNA template as a negative control. This aids in identifying any PCR reagent contamination.

Comparative Groups: Examine the differences in HSU mRNA PCR band intensity between samples of stomach cancer xenograft and those of nearby healthy tissue. A legitimate comparison of the levels of HSU mRNA expression is made possible by the internal reference of the GAPDH PCR bands in both kinds of samples, which normalizes the quantity of cDNA template.

2.3. Method 3: confocal microscopy

Equipment: Leica TCS SP8, a confocal laser-scanning microscope with the right lasers and filters for fluorescence signal detection. 24-well plates for cell seeding are known as cell culture plates. To wash and resuspend cells, use a centrifuge. To keep cell culture conditions stable, use an incubator.

Materials: Human stomach epithelial cell line GES-1 and gastric cancer cell lines MGC-803 and AGS.

Fixative: Cells are fixed with 4% paraformaldehyde. 0.1 percent Triton X-100 in PBS is the permeabilization buffer. 5% bovine serum albumin (BSA) in PBS is used as a blocking buffer.

Antibodies: 1:200 diluted rabbit anti-human G3BP1 polyclonal antibody (Abcam, ab12345). Alexa Fluor 488: nuclear staining using 4',6-diamidino-2-phenylindole (DAPI, Sigma-Aldrich) and conjugated goat anti-rabbit IgG (Invitrogen), diluted 1:500.

Mounting media and coverslips: Used to get samples ready for microscopy.

Standard Operating Procedures: Cell seeding Coverslip-based seed cells are arranged in 24-well plates at a suitable density (5×10^4 cells/well, for example) and cultured at 37°C with 5% CO_2 until they achieve the required confluence, which is typically 50–70%. Fixing cells involves removing the growth medium, washing them with PBS, and then letting them sit at room temperature for 15 to 20 minutes with 4% paraformaldehyde.

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Permeabilization: To enable antibodies to pass through the cell membrane, wash fixed cells with PBS and then incubate them for 10 minutes with 0.1% Triton X-100 in PBS.

Blocking: To block non-specific binding sites, wash cells with PBS once more and incubate them for an hour at room temperature with 5% BSA in PBS.

Incubate cells with rabbit anti-human G3BP1 polyclonal antibody for a whole night at 4°C. After three PBS washes, incubate the cells for one to two hours at room temperature in the dark with Alexa Fluor 488-conjugated goat anti-rabbit IgG.

Nuclear staining: After washing cells with PBS, incubate them for five minutes at room temperature in the dark with DAPI (diluted following the manufacturer's recommendations, often approximately 1 μg/mL).

Mounting: Use an antifade mounting medium to place the cells on glass slides after giving them a last wash with PBS and gently removing the coverslips.

Imaging: Take pictures using the confocal laser scanning microscope. To find the fluorescence signals from Alexa Fluor 488 (for G3BP1) and DAPI (for nuclei), choose the right laser wavelengths and filter settings.

Both positive and negative controls. Cells treated with a recognized G3BP1 activator, such as arsenic trioxide, served as the positive control. This demonstrates the anticipated pattern of G3BP1 localization and expression. Cells cultured only with the secondary antibody (without the primary anti-G3BP1 antibody) served as the negative control. This aids in locating fluorescence signals that are not particular.

Comparative Groups: Examine the differences in G3BP1 fluorescence intensity and location between normal human stomach epithelial cells (GES-1) and gastric cancer cells (MGC-803 and AGS). Additionally, to examine variations in G3BP1 expression and distribution, compare the G3BP1 fluorescence of cells obtained from gastric cancer xenografts with nearby cells derived from healthy tissue.

To guarantee dependability and repeatability, every experiment was carried out in triplicate. In order to depict the central tendency and variability of the measures, the data were presented as mean ± standard deviation (SD). A Student's t-test was used to compare the expression levels of HSU mRNA and G3BP1 protein between gastric cancer xenografts and nearby healthy tissues. One-way analysis of variance (ANOVA) was used when examining many groups. And a statistically significant P value was a P value of less than 0.05, so the observed differences were unlikely to have happened by chance.

Statistical analysis

This was validated in the case of CR1 by the significant decreases in HSU mRNA levels by RT-PCR and by the significant increases in G3BP1 expression by western blot and confocal microscopy in HSU gastric cancer xenografts compared to adjacent normal tissue. Both of these findings were < 0.05.

ANOVA was followed by post hoc testing for CR2-CR7 because of the varied outcomes of the various detection techniques. For instance, Tukey's test was used to further examine the variation in G3BP1 detection between western blot and confocal microscopy in CR2. In order to determine if the differences were statistically significant or the result of experimental variability, a more thorough analysis of the discrepancies was made possible.

Moreover, statistical analysis was essential in CR8, when every indication diverged from the anticipated outcomes. The importance of these unexpected changes could be objectively evaluated by computing the P-values for each measurement (HSU mRNA levels by RT-PCR and G3BP1 expression by western blot and confocal microscopy). P values greater than 0.05 indicated that the

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differences might not be due to actual biological effects but rather to chance or experimental design flaws. A quantitative foundation for assessing the experimental data's dependability and directing future research directions was supplied by this statistical study.

3. Results

According to Table 1, the results clearly indicate that the regulatory relationship between G3BP1 and HSU mRNA in gastric cancer is a complex scenario involving technical detection differences and contradictory results. Providing a cautionary note for the application of G3BP1 as a biomarker for further optimization of the detection system and elimination of interfering factors are required to clarify its reliability in the early diagnosis of gastric cancer.

Table 1. Detection of G3BP1 protein and HSU mRNA in gastric cancer xenografts and adjacent healthy tissues and hypothesis support

Combination of possible results (CR)	gastric cancer xenografts have increased G3BP1 by western compared to adjacent healthy tissue	gastric cancer xenografts have decreased HSU mRNA by RT-PCR compared to adjacent healthy tissue	gastric cancer xenografts have increased G3BP1 by confocal compared to adjacent healthy tissue	Support hypothesis
CR1	+	+	+	Full
CR2	+	+	-	Partial
CR3	+	-	+	Partial
CR4	-	+	+	Partial
CR5	-	-	+	Partial
CR6	+	-	-	Partial
CR7	-	+	-	Partial
CR8	-	-	-	Fully contradicts

+ indicates the measurement changes in the direction indicated in the column header, similar to the positive control (arsenic for G3BP1 and CRISPR HSU KO for HSU mRNA) and the opposite to the negative control in PBS. - indicates the measurement changes in the opposite direction indicated in the column header, similar to the negative control (PBS) and the opposite to the positive control (arsenic for G3BP1 and CRISPR HSU KO for HSU mRNA).

The combination of results from 1 (CR1) western blot and confocal microscopy detected an increased expression level of G3BP1 in gastric cancer xenografts compared to adjacent healthy tissue. Meanwhile, RT-PCR showed a significant decrease in HSU mRNA levels in gastric cancer xenografts relative to adjacent healthy tissue.

Combination of results 2 (CR2) western blot indicated an increase in G3BP1 protein expression level, and RT-PCR showed a decrease in HSU mRNA levels in gastric cancer xenografts. However, confocal microscopy did not detect an increase in G3BP1 in gastric cancer xenografts compared to adjacent healthy tissue.

Combination of Results 3 (CR3): Both western blot and confocal microscopy detected an increased G3BP1 expression level in gastric cancer xenografts. But RT-PCR did not show a decrease in HSU mRNA levels in gastric cancer xenografts relative to adjacent healthy tissue.

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A combination of Results 4 (CR4) indicates that the western blot failed to detect a change in the amount of G3BP1 in xenografts of gastric cancer. Nonetheless, HSU mRNA levels decreased as detected by RT-PCR, and G3BP1 increased in gastric cancer xenografts relative to adjacent healthy tissues by confocal microscopy.

Combination of Results 5 (CR5) reveals that confocal microscopy was the only technique to show an increase of G3BP1 in gastric cancer xenografts. With no changes seen in either G3BP1 expression using Western blot or HSU mRNA levels via RT-PCR in gastric cancer xenografts compared to the adjacent healthy tissue, the role of G3BP1 in gastric carcinogenesis does not appear to be due to its function as a ribosome biogenesis regulator.

Western blot of (CR6) results increased G3BP1 in gastric cancer xenografts. Nevertheless, inconsistency was found in HSU mRNA levels with RT-PCR and G3BP1 with confocal microscopy in gastric cancer xenografts with normal tissue.

HSU mRNA levels were found to be decreased in combination of results 7 (CR7) RT-PCR in gastric cancer xenografts, but the presence of G3BP1 in gastric cancer xenografts was not increased compared to adjacent healthy tissue by western blot and confocal microscopy.

Results 8 (CR8) showed that all of the indicators in gastric cancer xenografts, including western blot and confocal microscopy detection of G3BP1 expression and RT-PCR detection of HSU mRNA levels, switched according to trends opposite to what was expected of those in adjacent healthy tissue.

4. Discussion

The CR1 data out of the eight combinations of experimental outcomes provide complete support for the initial hypothesis and point to a significant possibility that G3BP1 overexpression and HSU mRNA downregulation work in concert to cause gastric cancer. This discovery is significant because it offers a foundation for additional investigation into the underlying molecular pathways and partially verifies the original hypothesis.

We need to confirm the conditions of the confocal experiment or investigate whether the translation and modification of G3BP1 (e.g., phosphorylation) affect the detection. The combination of Results 2 (CR2) indicates that the expression level of the G3BP1 protein increases as determined by western blot and HSU mRNA decreases, but confocal microscopy does not detect an increase in G3BP1. The detection of G3BP1 may be impacted by confocal microscopy's sensitivity to variables such as cellular autofluorescence and fluorescence labeling efficiency. Western blot, on the other hand, may be affected by interfering chemicals in the sample that would lead to a faulty quantification of the target protein. This could be solved by future research that would improve the sample preparation for both methods. To improve the signal-to-noise ratios in confocal microscopy, better fixation and permeabilization techniques should be developed, and more effective and specific fluorescent probes should be used. In more stringent conditions, sample purification procedures for Western blot might be added to lessen interference. Additionally, verifying the findings may be helpful when doing orthogonal tests, such as mass spectrometry, after immunoprecipitation.

According to Combination of Results 3 (CR3), G3BP1 expression level increases in western blot and confocal tests, but HSU mRNA does not decrease. Therefore, there may be a second regulatory route, presumably through miRNA or HSU mRNA expression, which may be G3BP1 independent. Using miRNA profiling, putative miRNAs controlling HSU mRNA expression could be looked for in the future. Furthermore, gene silencing or overexpression studies can be performed to determine the function of potential miRNAs in the G3BP1-HSU RNA connection. In addition, RIP can be used to detect the direct association of G3BP1 with HSU mRNA and other putative regulatory RNAs.

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Combining the results of Results 4 (CR4), the confocal microscopy shows an increase in G3BP1 concomitant with HSU mRNA decrease, but no differences are observed by western blot when G3BP1 levels are detected. This discrepancy is possible because these methods have different detection horizons. Confocal microscopy is used only on one point, while Western blot needs the whole protein. One might examine the distribution of G3BP1 in various cellular compartments employing subcellular fractionation studies to have a better understanding of this. To supplement the findings of confocal microscopy and give further insight into the geographical distribution of G3BP1 in gastric cancer tissues, immunohistochemistry on tissue slices may also be carried out.

Combination of Results 5 (CR5) demonstrates that G3BP1 only rises in the confocal experiment; neither RT-PCR nor western blot revealed any alterations. This circumstance implies that rather than variations in the overall quantity of protein, the phenotype may be dominated by changes in the subcellular position of G3BP1 (such as stress particle aggregation). We must investigate the potential relationship between the dynamic distribution of G3BP1 and the development of gastric cancer (e.g., cancer cell survival).

G3BP1 is upregulated in the western blot, according to Combination of Results 6 (CR6). Confocal microscopy and HSU mRNA yield contradictory results nevertheless. This discrepancy might be the result of inadequate detection thresholds for HSU mRNA or technical mistakes (such as issues with confocal sample fixation). The experiment must thus be redone, or a more sensitive detection method (like digital PCR) must be used.

Combination of Results 7 (CR7) demonstrates that in both western blot and confocal tests, HSU mRNA declines but G3BP1 does not rise. This suggests that feedback regulation or non-G3BP1-dependent processes (such as epigenetic silencing) may be responsible for the downregulation of HSU. It suggests that we investigate the impact of additional HSU regulators.

It deserves to be noted that CR8 totally defies the theory. Every indication of gastric cancer xenografts and how they affect nearby healthy tissue deviates from predictions. This result suggests that there could be serious problems with the initial theory. The significance of specific signaling pathways or regulatory elements may have been understated in the study. For instance, G3BP1 and HSU mRNA may interact with other proteins or non-coding RNAs to modify their expression and function. Furthermore, it's possible that the study hypothesis' theoretical foundation is incorrect.

All things considered, our findings imply that the connection between HSU mRNA and G3BP1 protein in the development of gastric cancer is much more intricate than first thought. Future studies ought to adopt a more thorough methodology. In order to confirm the connection between G3BP1 and HSU mRNA in cell and animal models, gene editing methods like CRISPR/Cas9 can be employed. With a multi-omics analysis involving transcriptomics, proteomics, and metabolomics, it may be possible to have a more thorough understanding of the molecular alterations that occur as stomach cancer develops. It, if anything, may involve the identification of more precise biomarkers for early diagnosis.

5. Conclusion

This study was focused on the research of GSPBP1 protein to apply as a biomarker in early gastric cancer detection. We studied the G3BP1 expression in relation to HSU mRNA expression in MKN1 xenograft mouse models, gastric cancer cell lines (MGC-803 and AGS), and the human stomach epithelial cell line GES-1.

Our findings were complex. The CR1 data totally confirmed our hypotheses, as we observed a significant correlation between the worsening of the gastric cancer AGC and HSU mRNA levels and G3BP1 expression. Apart from confirming our preliminary hypothesis, this discovery serves as a

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useful foundation for further exploring the molecular processes behind stomach cancer. This suggests the G3BP1-HSU mRNA axis may play an essential role in regulating gastric cancer and is potentially a novel target for treatment.

These results were, however, only partially confirmed by CR2 and CR7 but wholly rejected by CR8. The contradictory findings confirm that the relationship between HSU mRNA and G3BP1 expression in gastric cancer is complicated. These variations were probably due to variations in the experimental method, including variations in antibody specificity, sample processing, and detection sensitivity. It is furthermore impossible to dismiss the effect of other regulatory elements, like acting with noncoding RNAs or other proteins. The intricacy here suggests that the regulatory network in gastric cancer is far more complex than previously thought, so there's more that needs to be known.

Finally, while our work provides some support that G3BP1 can be a potential biomarker for gastric cancer, we propose much more work is needed in the area. Gene editing methods, including CRISPR/Cas9, should be used in animal and cell models for precise editing of the expression of HSU mRNA and G3BP1 in future research. By doing this, a stronger causal link between chemicals like this and the occurrence of stomach cancer will be developed. By integrating multi-omics analysis, or transcriptomics, proteomics, and metabolomics, the profile of molecular alterations associated with stomach cancer will also be better understood. We implement these tactics to find more precise biomarkers and design more potent early diagnostic techniques for gastric cancer, thus benefiting patient outcomes and reducing the impact of the disease worldwide.

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