

## Relationship Between Gastric *Helicobacter pylori* Infection and Colorectal Polyps: A Retrospective Cohort Study

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### Abstract

**Background:** Infection with *Helicobacter pylori* (*H. pylori*) infection is one of the most prevalent bacterial infections worldwide. It is well-established as a contributing factor to various conditions, including chronic gastritis, peptic ulcers, and gastric cancer. This study aimed to investigate the potential association between *H. pylori* infection and the presence of colorectal polyps.

**Methods:** This retrospective cohort study was conducted at the General Surgery Endoscopy Unit of İzmir Katip Çelebi University between January 2019 and December 2022. Patients who underwent both gastroscopy and colonoscopy were included in the study. The demographic data of the patients, along with their *H. pylori* status, as well as the number, size, location, and pathology of any polyps, were recorded. A logistic regression model was employed to analyze the relationship between the prevalence of *H. pylori* infection and the occurrence of colorectal polyps.

**Results:** A total of 310 patients were included in the study, of whom 122 (39.4%) were men and 188 (60.6%) were women. Colorectal polyps were identified in 133 cases, while 173 cases showed no evidence of polyps. The rate of *H. pylori* infection was significantly higher in patients with colorectal polyps compared to those without polyps ( $P < 0.001$ ). Furthermore, advanced age and the presence of multiple polyps were significantly more prevalent in the *H. pylori*-positive group ( $P = 0.002$  and  $P = 0.023$ , respectively). However, when polyps were evaluated by size, location, type, and pathological subgroup, the rates of *H. pylori* infection did not differ significantly between the groups. Similarly, when examining the type of adenoma and the degree of dysplasia, *H. pylori* positivity showed no significant difference across the groups ( $P = 0.742$  and  $P = 0.751$ , respectively).

**Conclusion:** The findings of our research imply that infection with *H. pylori* in the stomach may contribute to a heightened likelihood of developing colorectal polyps. Therefore, individuals diagnosed with *H. pylori* might gain preventive advantage from undergoing colonoscopic evaluation to detect polyps and mitigate the risk of colorectal malignancy.

**Keywords:** Polyps; Endoscopy; *Helicobacter Pylori*; Adenoma

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## Introduction

Colorectal cancer (CRC) is the third most common malignancy, accounting for 10% of all cancer cases, following breast and lung cancer. It remains one of the leading causes of cancer-related deaths. The incidence and mortality rates of CRC are closely associated with geographic variations (1, 2). Clinically, the early detection of CRC poses significant challenges, as the disease is often asymptomatic in its early stages. Most colorectal cancers develop from adenomatous polyps, which have an incidence rate of approximately 30%. The progression from polyps to malignancy is a complex process that involves the interaction of environmental and genetic factors (3, 4). Early detection of premalignant polyps significantly reduces the incidence of CRC (5, 6).

*Helicobacter pylori* (*H. pylori*) is a gram-negative microorganism found in over half of the world's population. (7). The persistent inflammation triggered by *H. pylori* infection is strongly linked to the formation of peptic ulcers and gastric cancer (7). Additionally, recent studies have suggested a potential link between *H. pylori* infection and colonic neoplasms. *H. pylori* infection induces chronic gastric inflammation, which stimulates the secretion of gastrin from the host's antral G-cells. This hypergastrinemia has been shown to promote cell proliferation within the gastrointestinal tract. Over time, this condition is believed to increase the risk of colon polyps and adenocarcinoma (8, 9). *H. pylori* presence has been linked to a higher incidence of colorectal adenomas in studies originating from East Asia (10, 11).

Globally, there is a rising trend in the occurrence of colorectal polyps and colorectal cancer. Despite this, *H. pylori* infection continues to be highly prevalent in our country. This research was conducted to investigate the association between gastric *H. pylori* infection and the formation of colonic polyps.

## Patients and Methods

### Study Design and Population

This retrospective cohort study was conducted at the Endoscopy Unit of the General Surgery Department at İzmir Katip Çelebi University from January 2019 to December 2022. Ethical approval for the study was obtained from the Katip Çelebi University Ethics Committee (İKÇÜ-GOKAEK 2024/0268). Due to the retrospective nature of the study, the ethics committee of the university waived the requirement for obtaining informed consent from the patients.

Patients who underwent gastroscopy and colonoscopy were examined retrospectively. The demographic data and presenting complaints of the patients were recorded. Polyps removed during colonoscopy and biopsies taken during gastroscopy were subsequently evaluated histopathologically.

Patients who could not tolerate gastroscopy or colonoscopy, had inadequate gastric emptying or

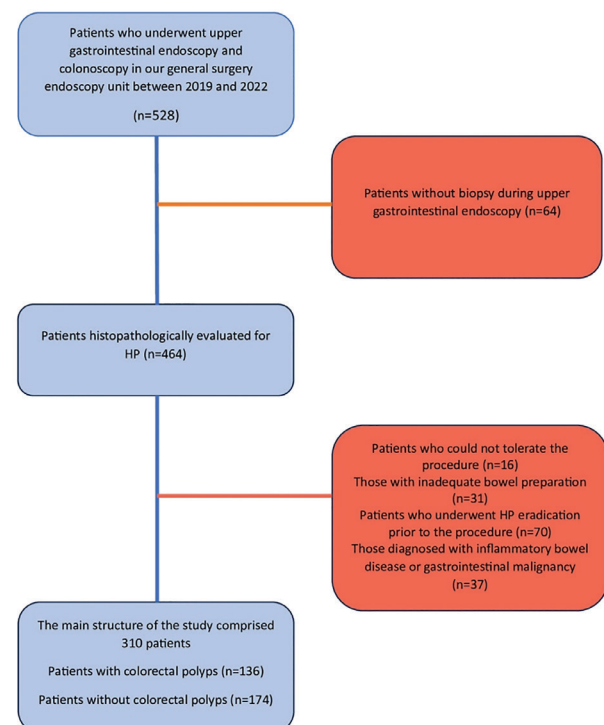
bowel preparation, had received *H. pylori* eradication therapy or proton pump inhibitors (PPIs) prior to the procedure, had inflammatory bowel disease, or were under follow-up for a previous gastrointestinal cancer were excluded from the study.

### Identification of Endoscopic Results

All patients underwent a complete colonoscopy, during which polypectomy was performed using either a hot snare or forceps. Data were collected on the location, morphology, size, and count of detected polyps. Lesions in the cecum, ascending colon, and transverse colon were defined as “proximal”, while those in the descending and sigmoid colon were categorized as “distal”. Based on size, polyps were macroscopically grouped as diminutive (<5 mm), small (5–10 mm), or large (>10 mm). Cytopathological examination identified adenomatous and other polyp subtypes. *H. pylori* detection was performed on endoscopic biopsy specimens obtained from the gastric antrum or corpus.

### Statistical Analysis

Data were analyzed using the Statistical Package for the Social Sciences (SPSS) version 25.0 (IBM Corp., Armonk, New York, USA). Descriptive statistics for categorical variables were presented as frequency and percentage, while continuous variables were presented as median and interquartile range (IQR) after assessing normality. The distribution of continuous variables was assessed using the Shapiro-Wilk test. Independent sample t-tests were applied for group comparisons. Categorical data were compared using Pearson's chi-square test or Fisher's exact test when appropriate. Risk factor analysis was conducted via binary logistic regression, with a  $P < 0.05$  considered statistically significant.



**Figure 1:** Flowchart of patients included in the study

## Results

During the study period, gastro-colonoscopy was performed on 528 patients. 64 patients were not assessable for gastrointestinal system biopsy, and 154 patients were excluded from the study due to factors such as poor procedural tolerance and inadequate bowel preparation (Figure 1). The study included 310 patients, with a median age of 58 (65-49) years. Among the patients, 122 (39.4%) were men, and 188 (60.6%) were women. The most common indication for endoscopy was chronic abdominal pain. Polyps were detected in 137 (44.2%) of the cases, and diverticular disease of the colon was present in 35 patients (11.3%). Table 1 summarizes the demographic and clinical features of the study population.

*H. pylori* was detected in 141 patients (45.5%), while 169 patients (54.5%) tested negative for *H. pylori*. A statistically significant difference was found between *H. pylori* positivity and age ( $P=0.002$ ), with the *H. pylori*-positive group being older.

Additionally, a statistically significant association was observed between *H. pylori* positivity and the presence of colorectal polyps, as well as the presence of multiple polyps ( $P<0.001$  and  $P=0.023$ , respectively). It was noted that *H. pylori* positivity was more prevalent among patients with polyps. Moreover, when comparing the number of polyps in patients with polyps, it was found that a greater number of patients with multiple polyps tested positive for *H. pylori* (Table 1).

No statistically significant differences were observed between *H. pylori* positivity and the other variables examined. In the analysis of *H. pylori* positivity in relation to demographic and clinical characteristics, the statistically significant variables—presence and number of polyps—were included in a univariate logistic regression analysis. The results of the logistic regression indicated that *H. pylori* infection was 5.8 times more prevalent in patients with polyps compared to those without, and 2.3 times more prevalent in patients with multiple polyps (Table 2). Additionally, when comparing

**Table 1:** Comparison of demographic and clinical characteristics associated with *Helicobacter pylori* positivity

Variables	Total, N (%)	Negative	Positive	P value
Age, (years, median [IQR])	58 (65-49)	55 (63.5-46)	59 (67-51)	0.002 <sup>+</sup>
Sex				
Female	188 (60.6)	108 (63.9)	80 (56.7)	0.198 <sup>*</sup>
Male	122 (39.4)	61 (36.1)	61 (43.3)	
Polyp				
Negative	173 (55.8)	127 (75.1)	46 (32.6)	<0,001 <sup>*</sup>
Positive	137 (44.2)	42 (24.9)	95 (67.4)	
Polyp Number				
Solitary	78 (56.9)	30 (71.4)	48 (50.5)	0.023 <sup>*</sup>
Multiple	59 (43.1)	12 (28.6)	47 (49.5)	
Polyp Type				
Pedunculated	107 (78.1)	33 (78.6)	74 (77.9)	0.930 <sup>*</sup>
Sessile	30 (21.9)	9 (21.4)	21 (22.1)	
Polyp Size (mm)				
Diminutive	91 (66.4)	27 (64.3)	64 (67.4)	0.463 <sup>*</sup>
Small	35 (25.5)	13 (31)	22 (23.2)	
Large	11 (8.1)	2 (4.8)	9 (9.5)	
Polyp Location				
Proximal	50 (36.5)	14 (33.3)	36 (37.9)	0.739 <sup>*</sup>
Distal	50 (36.5)	16 (38.1)	34 (35.8)	
Rectum/Anal	27 (19.7)	10 (23.8)	17 (17.9)	
Total Colon	10 (7.3)	2 (4.8)	8 (8.4)	
Diverticulosis				
Negative	275 (88.7)	154 (91.1)	121 (85.8)	0.141 <sup>*</sup>
Positive	35 (11.3)	15 (8.9)	20 (14.2)	
Polyp histology				
Adenomatous	81 (59.1)	24 (57.1)	57 (60)	0.754 <sup>*</sup>
Non-adenomatous	56 (40.9)	18 (42.9)	38 (40)	
Adenoma Type				
Tubular	76 (85.4)	20 (83.3%)	56 (86.2)	0.742 <sup>**</sup>
Villous+Tubulovillous	13 (14.6)	4 (16.7%)	9 (13.8)	
Dysplasia Degree				
Mild	74 (83.1)	21 (87.5%)	53 (81.5)	0.751 <sup>**</sup>
Moderate+Severe	15 (16.9)	3 (12.5%)	12 (18.5)	

<sup>+</sup>Independent sample t-test, <sup>\*</sup>Pearson's chi-squared test, <sup>\*\*</sup>Fisher exact test; For all analyses, a P-value<0.05 was considered statistically significant; IQR: interquartile ranges

**Table 2:** Determination of risk factors affecting *Helicobacter pylori* positivity

Variables	OR (95% CI)	P value	Variables	OR (95% CI)	P value
Constant	0.774	0.808	Constant	0.437	0.193
Age	1.020 (0.986;1.055)	0.260	Age	1.013 (0.991;1.035)	0.242
Polyp number*			Existence of polyp <sup>†</sup>		
Solitary	Ref		Negative	Ref	
Multiple	2.331 (1.060;5.125)	0.035	Positive	5.813 (3.499;9.657)	<0.001

\*Significance of the model P=0.037. Method: Enter; C.I.: Confidence Interval. OR: Odds ratio; <sup>†</sup> Significance of the model P<0.001. Method: Enter; C.I.: Confidence Interval

adenomatous polyps to other types of polyps, no significant difference was found in the frequency of *H. pylori* infection (P=0.754).

## Discussion

This comprehensive retrospective study involving patients who underwent both gastroscopy and colonoscopy provides evidence that *H. pylori* infection may contribute to an increased risk of colorectal polyps. A multivariate analysis was employed to assess the correlations between gastric *H. pylori* infection and polyp subtype, number, and various clinical characteristics.

*H. pylori* infection plays a significant role, particularly in diseases of the upper gastrointestinal tract, and is known to cause conditions such as stomach ulcers and gastritis. The possible involvement of *H. pylori* in colorectal pathology has been under investigation in recent years; however, its connection with colorectal polyp formation is still unclear. While certain studies have reported an increased risk of CRC and polyps in association with *H. pylori* infection (12-14), others have not demonstrated a significant correlation (15, 16). This discrepancy may be attributed to the fact that studies failing to find a relationship were conducted in regions with low *H. pylori* prevalence and utilized serological tests for *H. pylori* analysis.

The global prevalence of *H. pylori* infection is approximately 50% (17, 18). In our dataset, the positivity rate for *H. pylori* was 45.5%, which closely aligns with the average reported in the literature. Our study investigated the association between *H. pylori* infection, confirmed through cytopathological analysis of stomach biopsies, and the presence of colorectal polyps. Our findings support the hypothesis that *H. pylori* infection increases the incidence of colon polyps. A large multicenter study conducted in China, which included 33,439 patients, identified *H. pylori* as a risk factor for colon polyps (19). Furthermore, recent studies have demonstrated that the eradication of *H. pylori* along with PPIs therapy, reduces the occurrence of colon polyps (20, 21). In the study by Wang et al., the odds of developing colorectal polyps in patients with *H. pylori* infection were 2.19 times higher compared to those without the infection (14). In our study, we observed this odds ratio to be 5.8 times higher.

Our findings revealed that the average age of patients with *H. pylori* positivity was significantly greater. Existing data also indicate that the prevalence of colorectal polyps increases with age (22). This finding aligns with our anticipated outcome regarding the relationship between *H. pylori* infection and colorectal polyps. A significant association was observed between the number of polyps and *H. pylori* infection. Patients with multiple polyps exhibited a higher rate of *H. pylori* positivity compared to those with solitary polyps. Several studies in the literature have reported a higher prevalence of *H. pylori* infection in individuals with multiple polyps (14, 23).

Previous studies have predominantly focused on the association between adenomatous polyps and *H. pylori* infection. In our study, we found that non-adenomatous polyps also exhibited a similar correlation with *H. pylori* positivity as adenomatous polyps. There was also no significant difference in the prevalence of *H. pylori* infection with respect to polyp size or type (pedunculated versus sessile). Consistent with our findings, several studies in the literature have demonstrated a parallel increase in *H. pylori* prevalence across different polyp subtypes (13, 14, 23). Conversely, another study indicated an increased prevalence of *H. pylori* infection among individuals with adenomatous polyps (24).

The relationship between the location of colorectal polyps and *H. pylori* infection remains a topic of ongoing debate. Wang et al. reported that *H. pylori* infection increases the risk of adenomatous polyps in the proximal colon, while another study suggested a greater risk in the distal colon (11, 25). The formation of more polyps in the proximal colon is thought to be linked to increased bile acid secretion. Conversely, a hypothesis has been proposed that gastrin selectively exerts a stronger effect on the distal colon, which may explain the higher incidence of polyps in this region. Other studies have suggested that *H. pylori* increases the occurrence of polyps in both the proximal and distal colon (14, 26, 27). Our study also supports the finding that *H. pylori* infection elevates the rate of polyps across all segments of the colon without distinction. Additionally, we did not find an association between gastric *H. pylori* infection and adenoma type or degree of dysplasia. There is no consensus in the literature regarding the relationship between adenoma type and *H. pylori*. Jones et al.

reported higher *H. pylori* positivity in tubular and tubulovillous adenomas (28), while Soyly et al. found that *H. pylori* positivity was more prevalent in villous adenomas compared to other histological types (29).

Our study has several limitations due to its retrospective design and single-center nature. The retrospective approach restricted access to information regarding patients' physical conditions, dietary habits, smoking status, and the duration of *H. pylori* exposure. Furthermore, the single-center design resulted in a relatively small sample size, which may have diminished the statistical significance of our findings.

## Conclusion

The data obtained in this study indicate that individuals with *H. pylori* infection are at an increased risk for both adenomatous and non-adenomatous colorectal polyps. This risk is higher in older individuals and those with multiple polyps. Early detection is especially crucial for patients with adenomatous polyps due to their potential to

progress to cancer. Therefore, colonoscopy screening should be considered for patients diagnosed with *H. pylori* infection. Prospective studies are required to further explore the impact of *H. pylori* eradication on the development of polyps.

## Authors' Contribution

Design: F.K., H.T.; Surgical and medical practices: F.K.; Data collection and processing: H.T.; Data analysis: F.K.; Literature search: F.K.; Writing: F.K., H.T. Both authors have read and approved the final manuscript and agree to be accountable for all aspects of the work, ensuring that any questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

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## References

- Sung H, Ferlay J, Siegel RL, et al. Global cancer statistics 2020: GLOBOCAN estimates of incidence and mortality worldwide for 36 cancers in 185 countries. *CA Cancer J Clin.* 2021;71:209–49.
- Colorectal Cancer. Globocan. 2020. [(accessed on 2 July 2023)]. Available online: [https://gco.iarc.fr/today/data/factsheets/cancers/10\\_8\\_9-Colorectum-fact-sheet.pdf](https://gco.iarc.fr/today/data/factsheets/cancers/10_8_9-Colorectum-fact-sheet.pdf)
- Erdem L. Kolorektal polip ve kanser taramada türkiye verileri ve önerileri. In: Erdem L, (eds). Kolorektal Polip, Kanser Tarama, Tani ve Tedavisinde Güncel Yaklaşımlar. 1. Baskı. Ankara: Türkiye Klinikleri; 2020:9–15.
- Syed AR, Thakkar P, Horne ZD, Abdul-Baki H, Kochhar G, Farah K, Thakkar S. Old vs new: Risk factors predicting early onset colorectal cancer. *World J Gastrointest Oncol.* 2019;11:1011–1020.
- Senore C, Giovo I, Ribaldone DG, Ciancio A, Cassoni P, Arrigoni A, Fracchia M, Silvani M, Segnan N, Saracco GM. Management of P11 tumours removed by endoscopy during colorectal cancer screening: Outcome and treatment quality indicators. *Eur J Surg Oncol.* 2018;44:1873–1879.
- Changxi C, Mao Y, Du J, et al. Helicobacter pylori infection associated with an increased risk of colorectal adenomatous polyps in the Chinese population. *BMC Gastroenterol.* 2019;19:14.
- de Brito BB, da Silva FAF, Soares AS, Pereira VA, Santos MLC, Sampaio MM, Neves PHM, de Melo FF. Pathogenesis and clinical management of Helicobacter pylori gastric infection. *World J Gastroenterol.* 2019 Oct 7;25(37):5578–5589.
- Malfetheriner P, Venerito M, Schulz C. Helicobacter pylori Infection: New Facts in Clinical Management. *Curr Treat Options Gastroenterol.* 2018;16:605–615.
- Teimoorian F, Ranaei M, Hajian Tilaki K, Shokri Shirvani J, Vosough Z. Association of Helicobacter pylori Infection With Colon Cancer and Adenomatous Polyps. *Iran J Pathol.* 2018 Summer;13(3):325–332.
- Wang F, Sun MY, Shi SL, Lv ZS. Helicobacter pylori infection and normal colorectal mucosa–adenomatous polyp–adenocarcinoma sequence: a meta-analysis of 27 case–control studies. *Colorectal Disease.* 2014;16(3):246–252.
- Wang JL, Liang X, Xu J, Chen YX, Fang JY. Helicobacter pylori infection increases the risk of colorectal adenomas: An updated meta-analysis. *Clin Lab.* 2018;64(7):1163–1170.
- Lu D, Wang M, Ke X, Wang Q, Wang J, Li D, Wang M, Wang Q. Association Between H. pylori Infection and Colorectal Polyps: A Meta-Analysis of Observational Studies. *Front Med (Lausanne).* 2022 Jan 18;8:706036.
- Sonnenberg A, Genta RM. Helicobacter pylori is a risk factor for colonic neoplasms. *Am J Gastroenterol.* 2013;108:208–15.
- Wang M, Kong WJ, Zhang JZ, Lu JJ, Hui WJ, Liu WD, Kang XJ, Gao F. Association of Helicobacter pylori infection with colorectal polyps and malignancy in China. *World J Gastrointest Oncol.* 2020 May 15;12(5):582–591.
- Liou JM, Lin JW, Huang SP, et al. Helicobacter pylori infection is not associated with increased risk of colorectal polyps in Taiwanese. *Int J Cancer.* 2006;119:1999–2000.
- Luo F, Zhou P, Ran X, Gu M, Zhou S. No evident causal association between Helicobacter pylori infection and colorectal cancer: a bidirectional mendelian randomization study. *Sci Rep.* 2023;13:18544.
- Ansari S, Yamaoka Y. Current understanding and management of Helicobacter pylori infection: an updated appraisal. *F1000Res.* 2018 Jun 11;7:F1000 Faculty Rev-721.
- Peleteiro B, Bastos A, Ferro A, Lunet N. Prevalence of Helicobacter pylori infection worldwide: a systematic review of studies with national coverage. *Dig Dis Sci.* 2014;59:1698–1709.
- Feng L, Zhao K, Wang G, Dong R, Zhang M, Xia S, Zhang Y, Zhou W, Tian D, Yan W, Liao J. Relationship between endoscopic gastric abnormalities and colorectal polyps: a cross-sectional study based on 33439 Chinese patients. *Int J Med Sci.* 2023

- Jan 22;20(2):219-224.
20. Hu KC, Wu MS, Chu CH. et al. Decreased Colorectal Adenoma Risk After Helicobacter pylori Eradication: A Retrospective Cohort Study. *Clin Infect Dis.* 2019;68:2105–13.
21. Zuniga R, Bautista J, Sapra K, Westerfield K, Williams S, Sy AM. Combination of Triple Therapy and Chronic PPI Use May Decrease Risk of Colonic Adenomatous Polyps in Helicobacter pylori Infection. *Gastroenterol Res Pract.* 2015;2015:638547.
22. Sullivan BA, Noujaim M, Roper J. Cause, Epidemiology, and Histology of Polyps and Pathways to Colorectal Cancer. *Gastrointest Endosc Clin N Am.* 2022 Apr;32(2):177-194.
23. Basmaci N, Karataş A, Ergin M, Dumlu GŞ. Association between Helicobacter pylori infection and colorectal polyps. *Medicine (Baltimore).* 2023 Oct 20;102(42):e35591.
24. Zhang ZS. Predictive factors and model validation of post-colon polyp surgery Helicobacter pylori infection. *World J Gastrointest Surg.* 2024 Jan 27;16(1):173-185.
25. Nam KW, Baeg MK, Kwon JH, Cho SH, Na SJ, Choi MG. Helicobacter pylori seropositivity is positively associated with colorectal neoplasms. *Korean J Gastroenterol.* 2013;61(5):259–264.
26. Kim TJ, Kim ER, Chang DK, et al. Helicobacter pylori infection is an independent risk factor of early and advanced colorectal neoplasm. *Helicobacter.* 2017;22(3):10.1111/hel.12377.
27. Inoue I, Kato J, Tamai H, Iguchi M, Maekita T, Yoshimura N, Ichinose M. Helicobacter pylori-related chronic gastritis as a risk factor for colonic neoplasms. *World J Gastroenterol.* 2014;20:1485–1492.
28. Jones M, Helliwell P, Pritchard C, Tharakan J, Mathew J. Helicobacter pylori in colorectal neoplasm: is there an aetiological relationship? *World J Surg Oncol.* 2007;5:51.
29. Soylu A, Ozkara S, Alis H, Dolay K, Kalayci M, Yasar N, Kumbasar AB. Immunohistochemical testing for Helicobacter Pylori existence in neoplasms of the colon. *BMC Gastroenterol.* 2008 Aug 14;8:35.