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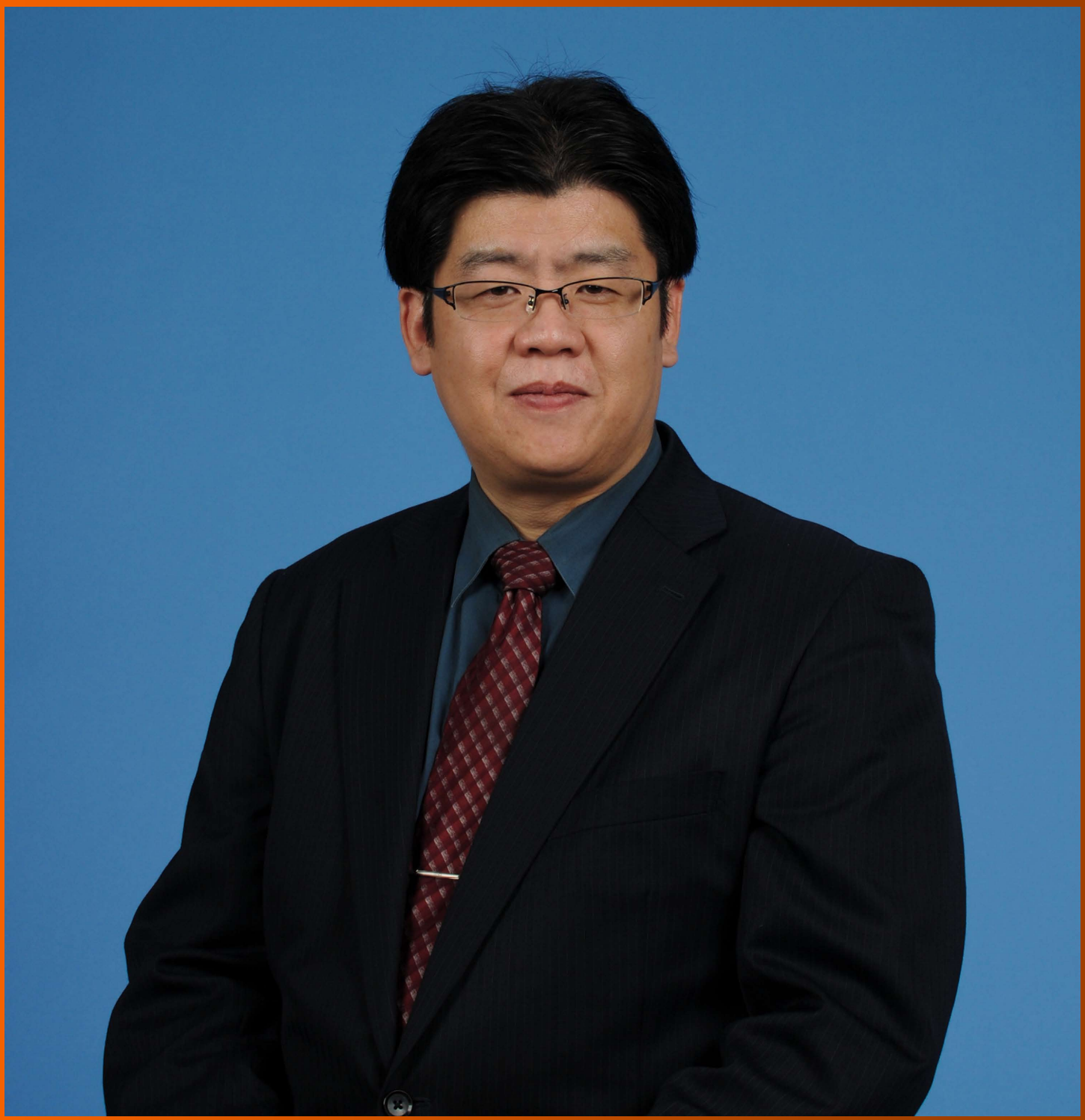

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LETTERS TO THE EDITOR

Resistance of *Helicobacter pylori* to furazolidone and levofloxacin: A viewpoint

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Abstract

In their review, Arslan *et al*[1] did not describe the status of *Helicobacter pylori* (*H. pylori*) treatment with furazolidone and the resistance to this antibiotic. We have presented different surveys showing the resistance of *H. pylori* to furazolidone from Asia and South America. The resistance rates varied but were mostly low (< 5%). There are not enough data on its efficacy and resistance in the United States and Europe. *H. pylori* mutations occurring in the *oorD* gene, including A041G, A122G, C349A(G), A78G, A112G, A335G, C156T and C165T, and in the *porD* gene, including G353A, A356G, C357T, C347T, C347G and C346A, have been indicated to be possibly related to the observed resistance. Additionally, to complete Arslan *et al*'s statement regarding levofloxacin resistance, it should be noted that compound mutations of N87A, A88N and V65I at codon Asn-87 were recently observed in the *gyrA* gene for the first time. However, the results on these topics are not sufficient, and more worldwide studies are suggested.

Key words: Susceptibility; Furazolidone; *Helicobacter pylori*; Resistance; Levofloxacin; Treatment

Core tip: We have presented different surveys showing the resistance of *Helicobacter pylori* (*H. pylori*) to furazolidone from Asia and South America. The resi-

Table 1 Studies evaluating the *Helicobacter pylori* resistance to furazolidone

Continent	Country	Study year	Strains (*n*)	Method	Resistance (%)	Author
Asia	China (Shanghai)	2000-2009	293	Agar dilution	0	Sun *et al*[6]
	China (Zhejiang)	2010-2012	21	Agar dilution	0.1	Su *et al*[7]
	China (Zhejiang)	2009-2014	9687	Agar dilution	< 0.01	Ji *et al*[8]
	India (Ghaziabad and New Delhi)	NA	68	Agar dilution	22.1	Gehlot *et al*[9]
	India (Gujarat)	2008-2011	80	Disk diffusion	13.8	Pandya *et al*[10]
	Iran (Rasht)	2012-2014	169	Disk diffusion	61.9	Maleknejad *et al*[11]
	Iran (Shiraz)	2004-2005	106	Agar dilution	9.4	Kohanteb *et al*[12]
	Iran (Sari)	2009	197	Disk diffusion	61.4	Abadi *et al*[13]
	Iran (Tehran)	2001-2004	135	Disk diffusion	0	Siavoshi *et al*[14]
	Iran (Tehran)	2002-2003	24	Disk diffusion	0	Fallahi *et al*[15]
	Iran (Tehran)	2005-2008	110	Disk diffusion	4.5	Siavoshi *et al*[16]
	Iran (Tehran)	2007-2008	104	Disk diffusion	0	Sirous *et al*[17]
	Iran	2003-2005	100	Disk diffusion	9	Rafeey *et al*[18]
	South Korea	1994-1999	220	Agar dilution	1.4	Kim *et al*[19]
	Malaysia (Malacca)	2009	90	Epsilometer test	0	Goh *et al*[20]
	Pakistan (Karachi)	2008-2013	93	disk diffusion	4.3	Siddiqui *et al*[21]
South America	Brazil (Bragança Paulista)	NA	90	Agar dilution	4	Mendonça *et al*[22]
	Brazil (Bragança Paulista)	NA	138	Agar dilution	13	Godoy *et al*[23]
	Brazil (Sao Paulo)	NA	39	Agar dilution	0	Eisig *et al*[24]
	Brazil (Sao Paulo)	2008-2009	77	Agar dilution and disk diffusion	0	Ogata *et al*[25]
	Brazil (Sao Paulo)	2008-2009	77	Agar dilution	0	Ogata *et al*[26]

stance rates varied but were mostly low (< 5%). *H. pylori* mutations occurring in the *oorD* gene, including A041G, A122G, C349A(G), A78G, A112G, A335G, C156T and C165T, and in the *porD* gene, including G353A, A356G, C357T, C347T, C347G and C346A, have been indicated to be possibly related to the observed resistance. Regarding levofloxacin resistance, compound mutations of N87A, A88N and V65I at codon Asn-87 were recently observed in the *gyrA* gene for the first time.

Zamani M, Rahbar A, Shokri-Shirvani J. Resistance of *Helicobacter pylori* to furazolidone and levofloxacin: A viewpoint. *World J Gastroenterol* 2017; 23(37): 6920-6922 Available from: URL: http://www.wjgnet.com/1007-9327/full/v23/i37/6920.htm DOI: http://dx.doi.org/10.3748/wjg.v23.i37.6920

TO THE EDITOR

We have read with great interest the valuable article by Arslan *et al*[1], titled "Importance of antimicrobial susceptibility testing for the management of eradication in *Helicobacter pylori* infection". One of the main subjects of the review was the description of the resistance rates of different antibiotics and the potential mechanisms leading to decreased in *Helicobacter pylori* (*H. pylori*) antimicrobial susceptibility. However, the authors should consider clarifying two important issues.

The authors did not allude to the status of *H. pylori* treatment with furazolidone and the resistance to this antibiotic. We have provided existing surveys reporting the resistance of *H. pylori* to furazolidone in Table 1. The resistance rates have been mostly reported to be lower than 5%; however, these rates can vary geographically. Furazolidone is not used widely in the United States and Europe; therefore, there are not enough data on its efficacy and resistance in these regions.

One of the main reasons for the emergence of resistance is related to the extensive use of furazolidone. In addition, regarding the molecular mechanisms, some genetic mutations have been identified. Mutations occurring in the *2-oxoglutarate:acceptor oxidoreductase* (*oorD*) gene, including *A041G*, *A122G*, *C349A(G)*, *A78G*, *A112G*, *A335G*, *C156T* and *C165T*, and in the *pyruvate oxidoreductase* (*porD*) gene, including *G353A*, *A356G*, *C357T*, *C347T*, *C347G* and *C346A*, are possibly related to the resistance[2,3]. *The oor and por* genes are involved in the generation of acetyl coenzyme A (acetyl-CoA) and succinyl-CoA[4]. Despite these findings, additional molecular methods are proposed to reach a better understanding of the mechanisms that were mentioned.

Arslan *et al*[1] accurately documented the mechanism of levofloxacin resistance; *i.e.*, point mutations in the *gyrA* (DNA *gyrase*) gene were stated to be potentially linked to the resistance. However, to complete their statement, it should be noted that compound mutations of N87A, A88N and V65I at codon Asn-87 were recently observed in the *gyrA* gene for the first time. L45F, A55S, A97V, D91N, R130K and G60S are other possible mutations that need to be assessed in studies with broader sample bases[5].

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