Autism is not a Disease of Definitive Cure but it is a Typical Disease of Definite Prevention

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Abstract

Aim: Demonstration of a possible role of ***Helicobacter pylori*** in the etio-pathology of autism that makes prevention of the disease possible. **Background:** ***H. pylori*** was suggested as a reason related to many medical challenges. ***H. pylori*** when forced to migrate to the colon due to the antibiotic violence; the accumulated ***H. pylori***-produced colonic ammonia in profuse amounts conforms with the elevated serum ammonia among autistic children and its toxic effect with the hypothesis of the entire brain compromise suggested in autism. Interestingly, kids develop ***H. pylori*** trans-familial during the time of weaning which is the typical timing of developing autistic features. Ammonia is the producer of nitric oxide in the gut via a shear stress effect while nitric oxide is a cure and a poison at the mean time; that is in normal residual levels it is healthy while in excess amounts it is toxic. This could explain the early brilliant behaviors in some autistic kids which are followed by loss of skills later. A striking observation has been emphasized by some study where all parents of the autistic kids of that study were having frank ***H. pylori*** dyspepsia, the kids were also positive for ***H. pylori*** fecal antigen and some kids were recovered from prodromal symptoms via early colon clear from the colonic ***H. pylori*** strains. **Conclusion:** Autism then, when established, is not a disease of definitive cure but it could be a typical disease of definite prevention via protection from transmission of parents ***H. pylori*** strains to kids.

Keywords: ammonia, autism, colon clear, Helicobacter pylori, nitric oxide.

Introduction

Autism constitutes a challenging puzzle affecting disadvantaged children at early age where they grow normal until age of 18-24 months then they begin failure to develop some skills or loose already developed skills. The exact etiology of the condition remains unclear while all promises of complete cure are unsuccessful. Autism is a brain disorder that limits a person's ability to communicate, correlate and relate to

other people. It is a series of neuro-developmental disorders that are characterized by deficits in both social and cognitive functions (Li& zhou, 2016). Autism spectrum disorders (ASD) affect about one child in 68, striking nearly five times as many boys as girls (Woolfenden et al., 2016; Baio, 2009). It was concluded that genetic and environmental factors are both responsible for the etiology of ASD. Although epidemiological studies have been conducted to clarify these factors but this conclusion remains unclear (Galichi et al., 2016; Kiely et al., 2016). Careful observers could early discover development of signs of autism in a child. Some children develop normally until 18-24 months of age and then they just stop or loose previously acquired skills. Signs of development of ASD could include repeated motions (rocking or spinning), avoiding eye contact or physical touch, delay in learning to talk, repeating words or phrases and getting upset by minor changes (Kiely et al., 2016; Allen, 1988). Young infants are very social even in the first year of age, therefore; it is possible to detect early signs of autism as early as how babies interact with their world. At this age, a child with ASD may not turn to a mother's voice, respond to his own name, look people in the eye, have no babbling or pointing and no smiling or responding to social cues from others (Emberti Gialloreti, et al., 2016; Spjut Jansson, et al., 2016; Locke, 2016). Children with autism may sometimes have physical symptoms including digestive troubles such as constipation and sleep problems. It was long believed that autism affects only those regions of the brain that control social interaction, communication and reasoning but instead it is suggested that the disorder in autism affects the entire brain (Galichi et al., 2016; Dinan& Cryan, 2016; Richter et al., 2015).

Most investigators were successful to come in agreement that various gastro-intestinal (GI) factors may contribute to behavioral disorders in children with autism (Li& zhou, 2016; Galichi et al., 2016) but they mostly missed to successfully achieve the real pathology behind these GI factors in contribution to autistic behavior. The last three decades have demonstrated prevalence of abnormal-behavior ***Helicobacter pylori*** strains and the flare up of a lot of medical challenges related to these ***H. pylori*** strains via inflammatory, toxic, immune or different unknown reasons to the extent that the medical world has believed that ***H. pylori*** eradication should be a necessary attempt (Farinha& Gascoyne, 2005; Nasrat et al., 2015a).

Aim

Demonstration of a possible role of the bacterium ***H. pylori*** in the etio-pathology of autism that makes prevention of the disease possible.

Review

Although various reports in literature refer with great concern to the role played by ***H. pylori*** in disease pathology (Farinha& Gascoyne, 2005; Nasrat et al., 2015a), research studies seldom indicated directly to the possibility that ***H. pylori*** could be hidden behind the pathogenesis of autism. Recent clinical studies have apparently revealed a high prevalence of the GI symptoms such as inflammation and dysfunction

in children with autism. Mild to moderate degrees of inflammation were almost found in both the upper and the lower intestinal tracts with an obvious decreased digestive enzyme activity in many autistic children. Treatment of these digestive problems appeared to constitute positive effects on autistic behaviors; these new observations represent only just a piece of this unsolved puzzle which is "autism" and should stimulate more researches towards the brain-gut connection (Horvath& Peman, 2002).

As ASD is often associated with different GI disturbances which may also impact behavior, therefore; alterations in autonomic nervous system functions should be also expected frequently in ASD. The relationship between these findings in autism is not clearly known. It was suggested that autonomic functions and GI problems are intertwined in children with ASD (Ferguson et al., 2017). Although the exact etiology and pathology of ASD remain unclear, a disorder of the microbiota-gut-brain axis is emerging as a prominent factor in the generation of autistic behavioral disorders. Clinical studies have shown clearly that GI symptoms and compositional changes in the gut microbiota frequently accompany cerebral disorders in patients with ASD. A disturbance in the gut microbiota which is usually induced by a bacterial infection or chronic antibiotic exposure has been implicated as a potential contributor to ASD. The bi-directional microbiota-gut-brain axis was often suggested to be acting mainly through neuro-endocrine, neuro-immune and autonomic nervous mechanisms. It was reported that application of modulators of the microbiota-gut-brain axis such as probiotics and certain special diets might be a promising strategy for the treatment of ASD. Different observations about disruption of the microbiota-gut-brain axis as concerns the pathogenesis of ASD have therefore suggested its potential therapeutic role in autistic deficits (Li& zhou, 2016).

A gut to brain interaction in ASD and the role of probiotics on clinical, biochemical and neuro-physiological parameters in autistic individuals have been emphasized and confirmed in further reports. It was furthermore adequately reported that the high prevalence of the frequent GI disturbances in patients with autism might be linked to gut dysbiosis representing a phenotype of a "gut-brain axis" disruption. Employment of strategies that could restore the normal gut microbiota and reduce the gut production and absorption of toxins such as probiotic supplements in diet might represent a non-pharmacological option in the treatment of GI disturbances in ASD. The effect of probiotic supplements in autistic children is not only specific on GI symptoms but also to improve the core deficits of the brain disorder, cognitive and language development, brain function and connectivity (Santocchi, et al., 2016). It was further reported that specific assessment of gut functions including the microbiome would be necessary to evaluate the contribution of gut physiology to functional constipation observed in autistic children (Marler, et al., 2016). As much as GI symptoms were frequently reported among autistic children, an impact of GI co-morbidity on ASD behavioral problems has been hypothesized. 'Constipated' and 'Not-Eat' were described as the most frequent GI symptoms in autistic individuals

(Fulceri, et al., 2016). Alteration in intestinal function which was often referred to as a "leaky gut" due to mucosal inflammation has been attributed to children who are on the autism spectrum; this particular symptom was even put into consideration to identify children with autism who have atypical symptoms (Kushak, et al., 2016).

The concept of gut-brain axis, its regulation by the microbiota and its role in the biological and physiological basis of neuro-developmental and neuro-degenerative disorders could thus constitute a considerable role in the pathogenesis of autism. The importance of early life gut microbiota in shaping future health outcomes should be also considered. Disturbances of this composition by way of antibiotic exposure could result in long-term effects on physiology and behavior (Dinan& Cryan, 2016; Kushak, et al., 2016). ***H. pylori*** in the stomach is leading the behavior of natural bacteria as it does not exist in the gastric lumen during presence of food and it remains settling just juxta-mucosal under the mucus layer of gastric mucosa with the ammonia at its immediate vicinity functioning to protect the gastric wall from its acid if it goes in excess (Farinha& Gascoyne, 2005; Nasrat et al., 2015a). Therefore; the antibiotic violence towards ***H. pylori*** forcing it to migrate towards the colon could definitely disturb its natural microbiotic function with its sequels on human body physiopathology.

The routes of communication between the microbiota and brain are being unraveled and could include the microbial metabolites such as ammonia (Dinan& Cryan, 2016; Farinha& Gascoyne, 2005). As ***H. pylori*** could migrate or get forced to migrate to the colon under the influence of antibiotics, it will continue producing ammonia for a reason or no reason, unopposed or buffered by any acidity, leading to accumulation of profuse toxic amounts of ammonia. Colonic ***H. pylori*** strains in their abnormal colonic habitat could lead to adverse toxic effects in the body; certainly the delicate physical structure of a child during early growth could be also severely affected by these aggressive drastic strains and the delicate integrity of the child's growing brain could further in susceptible children be a fragile target to the toxic influence of colonic ammonia (Farinha& Gascoyne, 2005; Nasrat et al., 2015a; Nasrat et al., 2015b).

The hypothesis of the entire brain involvement in autism was designed on the basis of impairment of the histology of whole areas of the brain in order to explain inability of autistic children to perform complex tasks (Donovan& Basson, 2017; Grecucci et al., 2016; Richter et al., 2015; Jou et al., 2011; Dinan& Cryan, 2016; Peeva, 2013; Casanova, 2013). In spite of the finding that many investigators have demonstrated rise of serum ammonia level among autistic children, they missed to indicate the possibility that elevated levels of serum ammonia could influence the entire functions of the whole brain of those kids (Burrus, 2012; Abu Shmais et al., 2012; Wang et al., 2012; Cohen, 2006; Corker& Tuzun, 2005; Fallon, 2005).

Discussion

H. pylori colonized the stomach since an immemorial time as if both the stomach and the bacterium used to live together in peace harmless to each other and hence ***H.***

pylori has been considered by some investigators a natural bacterium (Farinha& Gascoyne, 2005; Nasrat et al., 2015a). ***H. pylori*** when forced to migrate to the colon mainly under the influence of the antibiotic violence would lead to different dyspeptic symptoms and accumulation of profuse toxic amounts of ammonia in the colon with consequent elevated levels of serum ammonia (Farinha& Gascoyne, 2005; Nasrat et al., 2015a; Nasrat et al., 2015c; Nasrat et al., 2015d). It is common that ladies develop dyspepsia during pregnancy; abnormal ***H. pylori*** strains are responsible for most cases of functional dyspepsia but it is rarely recognized that this dyspepsia among pregnant ladies is ***H. pylori***-related (Nasrat, 2015). Accordingly; serum ammonia would be elevated in both maternal blood of those dyspeptic pregnant ladies and in the fetal blood in turn with the possibility of a toxic influence of ammonia on the delicate structure of the developing fetal brain leading also to sensitization of the fetal brain during early embryonic life to the adverse effect of ammonia. It has been reported that the neuropathology of autism starts early during embryonic life due to heterogeneity (Donovan, 2016; Chang et al., 2015; Blatt, 2012). The sustained elevated ammonia level in fetal blood caused by the colonic ***H. pylori*** strains of dyspeptic pregnant mothers could constitute a trigger of a causative pathology for neuro-development of autistic disorders confirming accordingly with the suggestion that both environmental and genetic factors are responsible for the etiology of autism (Galichi et al., 2016; Kiely et al., 2016).

The suggestion that the elevated residual serum ammonia level in fetal blood plays an early causative pathogenic factor in leading to autistic neuro-developmental disorders since embryonic life is supported by an observational finding expressed by mothers of 7 autistic children during their delivery. The mothers confirmed a frank history of ***H. pylori***-related dyspepsia during their pregnancy which had been confirmed by specific laboratory tests, they were just able to follow gastric sedatives. They were astonished that their babies did not cry immediately after delivery and suction of their secretions in spite of their good general condition (Nasrat et al., 2017). Those mothers continued having ***H. pylori*** dyspepsia symptoms after delivery because of a contraindication for eradication therapy or failure of therapies as antibiotics are seldom effective against extra-gastric ***H. pylori*** strains (Farinha& Gascoyne, 2005; Nasrat et al., 2015a; Grünberger; 2006). Later, their kids developed autism between the age of 2-3 years.

Existence of ***H. pylori*** in children occurs trans-familial via food at an early age; this matter is confirmed by the fact that ***H. pylori*** strains of children are often identical with that of their parents. Interestingly, children maintain the same strain genotype life-long even after moving to a different environment unless eradicated. ***H. pylori*** travels between parents via oral to oral route while transmission to kids occurs via meals (Nasrat et al., 2015b). The kids develop the abnormal-behavior colonic ***H. pylori*** strains at the time of their weaning when they start to share the dining table with their parents; that is typically the critical timing where children begin to develop

autistic features or loose already developed skills (Kiely et al., 2016; Allen, 1988; Farinha& Gascoyne, 2005; Nasrat et al., 2015a).

Migration of ***H. pylori*** to the colon occurs mainly under the influence of antibiotic exposure. Existence of ***H. pylori*** in the colon is typically life-long unless eradicated as antibiotics are seldom effective against extra-gastric ***H. pylori*** strains and no available measure has been proved to effectively eradicate ***H. pylori*** from the colon except the senna purge (Farinha& Gascoyne, 2005; Nasrat et al., 2015a; Grünberger; 2006; Nasrat et al., 2015e; Nasrat et al., 2015f). Accordingly; pregnant ladies who develop abnormal colonic ***H. pylori*** strains via an outside-home query meal would mostly remain dyspeptic and would become in most instances the mothers of autistic children due to persistence of a causative pathology which has triggered its early effect during pregnancy and made the fetal brain already sensitive to the toxicity of ammonia earlier throughout the embryonic life.

In addition to the toxic influence of ammonia, excess amounts of ammonia in the colon is smooth muscle spastic leading to multiple colonic spasms and a high rectal spasm which were demonstrated in ***H. pylori***-dyspeptic adults by colononoscopy. These spasms interfere with the integral colonic function of forming the motion contents, instead it squeezes the colonic contents leading to constipation and formation of small pieces of dried stool (Nasrat et al. 2015c). Existence of ***H. pylori*** in the colon was confirmed by a specific test (***H. pylori*** fecal antigen) which was found positive in all children and parents of many studies in literature. Constipation and passage of small pieces of dried stool are cardinal signs of colonic ***H. pylori***-related dyspepsia (Nasrat et al., 2015c; Nasrat et al., 2015d); these cardinal findings were found constant features in all autistic children in some studies.

The constant association of GI symptoms with autism to the extent that a gluten-free diet and probiotics were employed to improve these symptoms could further support the possibility of the role of ***H. pylori*** behind the pathogenesis of the disease. It has been further suggested that strategies of probiotic supplements that could help to restore normal gut microbiota and reduce the gut production and absorption of toxins have been advised and employed not only to improve GI symptoms in autism but also to improve the core deficits of the brain disorder (Santocchi, et al., 2016). Small bowel enteropathy has been reported in literature among patients with autism that could be attributed to embedding of ***H. pylori*** colonization towards small intestinal mucosa which is a further unrecognised unusual behavior of ***H. pylori*** (Farinha& Gascoyne, 2005; Nasrat et al., 2015a; Torrente et al., 2004; Nasrat, 2023). GI symptoms were frank and constant among patients of many studies; minute-size continuous intestinal sounds were diffusely audible over the center of abdomen that had been related to small intestinal irritation. Small intestinal enteropathy could account for the observations of “No Appetite”, “No Hunger” and “No Eat” symptoms among autistic children of these studies as kids would feel continuous abdominal discomfort that interferes with the natural desire to food. It was suggested that autonomic functions

and GI problems in autistic children are linked together (Nasrat, 2023; Ferguson et al., 2017); therefore, the quite passive peaceful attitude of some autistic children; "Non-Smiling", "Non-Reactive" was attributed in some studies to a degree of parasympathetic activation caused by the minor dull painless somatic intestinal insult. The improvement of intestinal symptoms among children of these studies upon intake of a warm mint drink, a soft caffeine drink or chocolate was explained to the improvement of this autonomic compromise. Constipation, weak appetite, passage of small pieces of dried stool or leaking small amount of soft retained/overflowing stool were encountered as constant features among those children (Nasrat et al., 2017).

Major colonization of abnormal-behavior ***H. pylori*** strains is necessary to induce symptoms and toxic complications. Spontaneous reduction below the pathologic level (50%) or even spontaneous elimination of ***H. pylori*** from the colon could occur due to variable reasons such as diarrhea or intake of foods containing bio-organic acids like lactic, formic, citric or acetic (Farinha& Gascoyne, 2005; Nasrat et al., 2015a; Zentilin et al., 2003). This could explain the wide variation in autistic features and the observation that some children develop some autistic symptoms then they skip the disease as they grow up.

A unique study has included two children newly diagnosed for autism, one was two years of age who started pronouncing some words and then he lost this skill. Immediate colon clear was employed for him and his parents within few days the clinical diagnosis was made up, that was followed by complete recovery of the child's skills. The other was three years old when diagnosed but he had lost the developed verbal skills one year earlier; he improved a little bit but did not recover completely after colon clear. That study also included a girl 11 months old; it was surprising to find a baby of that age who does not cry or even smile in response to her mother's plea, she was looking constantly to one direction and was never responsive or attentive towards her mother's voice. She was typically constipated and was crying only during passing the motion in the form of small pieces of dried hard stool. The father was having frank constipation and severe colonic dyspeptic symptoms due to frequent outside-home meals during business lunch and dinner meetings. ***H. pylori*** fecal antigen test was strongly positive for the girl and the parents; definitely the bacteria travelled from husband to his wife who gave it to her kid possibly while preparing and tasting the enfant's feeds or kissing her baby on the lips. Unfortunately, that girl was seen few months after she developed these features; immediate colon clear with a calculated dose (45 CCs) of the senna leaves extract purge was employed for her followed by vinegar-mixed fruit yoghurt twice daily. The girl improved within few days but did not recover completely because of late discovery and management of her condition; her bowel motion became easy without tragedy, the girl started to smile, look towards her mother, respond to her mother's voice and most importantly she learned to cry like any baby of her age when neglected for short time. ***H. pylori*** DNA extraction in the stool and ***H. pylori*** strain genotyping were done for the girl and parents; they were found having the same strain genotype with existence of

cytotoxin-associated gene A (cagA) positive ***H. pylori*** strains (Nasrat et al., 2017). It was emphasized that cagA of ***H. pylori*** encodes a highly immunogenic and virulence-associated protein; the presence of this virulent gene in the body could affect the clinical out-come in many children (Bulut, 2006 Jul).

Permanent compromise of some areas of the brain among autistic children such as impairments of grey or white matter, decreased cortical thickness or cortical thinning leading to dysfunction of complex interactions in disadvantaged children was confirmed in literature (Richter et al., 2015; Peeva et al., 2013; Casanova et al., 2013; Misaki et al., 2012); possibly for this reason, most researchers were just able to achieve improvement through employing different measures but never complete cure of their autistic patients. The results of various studies conform with the literature results in achieving incomplete cure of autistic features which could indicate that autism might not be a disease of definitive cure but it could be a typical disease of definite prevention via restriction of antibiotic use unless seriously indicated, extreme carefulness towards outside-home meals, natural colon care and natural colon clear on developing dyspeptic symptoms. If there is a chance for fundamental cure in autism, it might be via colon care and colon clear for both kids and parents. Early diagnosis and management are precious in this situation; recovery of the developed verbal skills for the two-years old child mentioned above with the lucky advantage of early discovery of the onset of the disease upon losing the developed verbal words is an ideal example (Nasrat et al., 2017).

Prevention is always far better than treatment; scientific research efforts could not reach until to date an adequate cure of autism while it could be greatly preventable by protecting children's brain from the bad sequels of the abnormal ***H. pylori*** strains of their dyspeptic mothers (Nasrat et al., 2015c; Debevere et al., 2001; Nasrat et al., 2017).

The reason that there are some conditions that have developed autism before the last three decades which is the particular period of the abnormal-behavior ***H. pylori*** strains prevalence, is most probably due to chronic antibiotic exposure or the frequent antibiotic abuse that would force ***H. pylori*** to migrate to the colon; the suggestion of chronic antibiotic exposure in leading to autistic disorder has been suggested by some investigators (Li& zhou, 2016). The last three decades demonstrated flare up of abnormal-behavior ***H. pylori*** strains after the the development of the strategic triple therapeutic violence against the stomach bacterium by two Australian physicians with consequent flare up of medical challenges related to these drastic abnormal-behavior ***H. pylori*** strains. It might seem that the antibiotic violence has rendered a domestic bacterium to become wild in attitude and sequels instead of getting rid of it. The challenge of autism first appeared before the lastest three decades but it has mostly dominated during these last three decades (Li& zhou, 2016; Farinha& Gascoyne, 2005; Nasrat et al., 2015a; Aksoy& Sebin, 2015; Nasrat et al., 2015g; Nasrat et al., 2017).

The literature reports indicate increased risk and rising prevalence of identified ASD among U.S. children. An investigator with his 45 collaborators reported in 2009 that the increased prevalence of identified autism among U.S. children need to be regarded as an urgent public health concern (Galichi et al., 2016). The reason that autism prevails among U.S. children could be most probably related to the fact that U.S. is a typical country of fast food dependency and the food handlers are mostly poor people migrating from poor developing countries with inadequate health care standards carrying with them abnormal-behavior ***H. pylori*** strains (Farinha& Gascoyne, 2005; Nasrat et al., 2015a). According to some personal communications; some mothers of autistic children indicated frankly that they love fast food to the extent that some particular fast food meals run in their blood while some mothers admitted that they are lazy to cook when they are pregnant and they depend on outside-home meals. Others mentioned that when they get pregnant while the previous baby is still between 2-3 years old, they depend mainly upon fast food delivery for themselves and their kids (Nasrat et al., 2017).

Summary

In Summary, realization of the real clue of a challenging illness constitutes the main success in its management; the hypothesis of the toxic influence of elevated serum ammonia in leading to the onset of an autistic behavioral disorder might remain just hypothetical until approved or disapproved but the unsolved puzzle of ASD has been considered as a "sequence" rather than a syndrome (Casanova et al, 2013). Apparently, the current available literature knowledge might seem articulating together without any little dislocation to support a concept that the spread of the abnormal-behavior/existence colonic ***H. pylori*** strains could just lie hidden behind the pathogenesis of a complex sequence of spectral events leading to the prevailing challenge known as the disorder of autistic spectrum. Therefore; research investigators should feel quite very enthusiastic towards this concept in order to support and approve or disapprove it for the sake of the possibility to alleviate misery of many kids and families of autistic children. Prevalence of the abnormal-behavior ***H. pylori*** strains followed the antibiotic violence towards this bacterium during the last three decades while flare up of medical challenges related to these ***H. pylori*** strains started also during the latest three decades; autism could be simply one member among these disease challenges. Autistic behavioral syndrome appeared earlier than the last three decades possibly also because of antibiotic abuse but it has dominated and flared up during the late decades. Antibiotic exposure was suggested as a factor leading to autism, the association of GI troubles and autism is frank and constant, development of autistic features or loss of developed skills occurs at the typical age where children could gain abnormal ***H. pylori*** strains trans-familial from their parents, ***H. pylori*** in the stomach was suggested to lead a behavior of natural bacteria while the role of microbiota and probiotics in autism is strongly suggested in literature, the elevated serum level of ammonia among autistic children is constant in most scientific reports and the toxic effect of ***H. pylori***-produced ammonia on the

whole brain conforms with the hypothesis of entire brain compromise suggested to explain inability of autistic children to perform complex interactions; all these findings seem quite articulated together to refer to the pathogenic influence of the abnormal ***H. pylori*** strains in the development of autism.

Accordingly, it seems that autism might not be a disease of definitive cure due to a permanent compromise of areas of the brain responsible for development of skills caused by a sustained toxic influence of ammonia throughout a critical period of brain growth during a child's early life. For this reason, scientific research efforts were just able to get improvement of autistic behavioral symptoms but did not achieve a real or definitive cure of autism. On the other hand, autism could be a typical disease of definite prevention via extreme carefulness towards outside-home meals, restriction of antibiotic use unless seriously indicated and natural colon care/colon clear for mothers who develop ***H. pylori***-related dyspeptic symptoms before or during pregnancy also while nursing their kid's during the early critical ages of child's growth. Early diagnosis and management of autistic features in a child could greatly improve the out-coming results of the attempts to correct the condition through colon clear for the kids themselves. Accordingly; the next kids after an autistic one could be simply at least saved from the disease as the matter of ***H. pylori*** is an environmental sanitary conflict before it is an actual medical challenge and prevention remains always far better than treatment.

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Conflict of interest

No conflict of interest is existing.

Conclusion

Autism, when established, might not be a disease of definitive cure due to permanent compromise of some areas of the brain responsible for development of skills during a critical period of a child's brain growth but it could be a typical disease of definite prevention. The value of this review study is the promising opportunity it gives for a child when discovered at the early prodromal symptoms to recover and skip the disease or at least next kids of an autistic one could be saved from developing autism via colon clear for parents.

The anti-***H. pylori*** antibiotic strategies might need to be subjected to extreme scientific revision and severe accurate re-determination owing to the possibility of having rendered an innocent biologic bacterium to become wild in attitude and sequels instead of getting rid of it.

Potent natural measures should be employed in order to control the challenge of ***H. pylori*** and functional dyspepsia instead of antibiotics.

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