



The 30th anniversary of Campylobacter, Helicobacter, and Related Organisms workshops—what have we learned in three decades?

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As we commemorate the 30th anniversary of the Campylobacter, Helicobacter, and Related Organisms (CHRO) workshops with this special Frontiers edition, we look back upon three decades of research and provide some highlights from the 16th International CHRO meeting. Although Theodor Escherich himself provided drawings of campylobacters back in the 1880s, *Campylobacter jejuni* was not identified until the 1950s. *Helicobacter pylori* was first described to be the causative agent of stomach ulcers at a CHRO meeting by Barry Marshall and Robin Warren—who later received the Nobel Prize for their findings that bacteria could cause diseases previously believed to be caused by human factors. Now, several genome sequences for campylobacters, helicobacters, and related organisms are available and we have moved into an era examining the intersection between host microbial ecology and pathogen infection. Both pioneers and new investigators in the CHRO research field continue to obtain “unexpected results” demonstrating that campylobacters and helicobacters do not follow classic paradigms of other well-characterized gastrointestinal pathogens and we are learning that there is a plethora of interesting related organisms beyond *C. jejuni* and *H. pylori*. This review summarizes recent discoveries in CHRO research and the exciting directions ahead.

Keywords: campylobacter, helicobacter, related organisms, genome diversity, control measures, fundamental biology, host responses, pathogenesis

INTRODUCTION

Campylobacter, Helicobacter, and Related Organisms (CHRO) researchers from around the world gathered in Vancouver, Canada in August, 2011 to share their history and exciting new findings on this unique group of microorganisms. Martin Skirrow and Martin Blaser regaled the audience during the opening session with historical stories of CHRO research, while Roger Feldman and James Fox provided perspectives on the future of CHRO research at the gala closing banquet. Three CHRO field leaders co-chaired the Young Investigator Award session—Skirrow, Hubert Endtz, and Thomas Meyer—providing inspiration to the future generations of CHRO researchers. And there were many highly interactive sessions describing new discoveries in between.

The calibre of science presented at CHRO 2011, in oral and poster presentations, was exceptional. For space purposes, this review covers an overview of select oral presentations that both reflect emerging general themes and provide additional details on certain focal points. Also, for confidentiality purposes, only published data and unpublished data for which we have received permission from the authors are described in detail, while general topics are noted for other areas of interest.

GENOMES AND EMERGING SPECIES

William Miller, an expert in CHRO sequencing, provided a comparative summary of 30 fully sequenced *Campylobacter*

taxa—and this number will soon reach 40—in addition to describing nine sequenced Arcobacters (also described by Sarah De Smet) and *Sulfurosprillum delayianum*. It is becoming more and more apparent that other members of this group of epsilon-proteobacteria are capable of causing disease; most have just not been accounted for due to culturing limitations. But, for *Campylobacter* species, Albert Lastovica's Cape Town protocol is becoming more and more widespread and demonstrating that organisms such as *C. concisus* (discussed by Nadeem Kaakoush and Hans Nielsen) and *C. upsaliensis* (pathogenesis described by Shauna Crowley/Christine Szymanski) are emerging species. CHRO researchers are also using non-culture-based methodologies such as PCR to detect these organisms in food and fecal samples, so the epidemiology, host range, and virulence properties of many *Campylobacter* species is yet to be determined. For example, Olivier Vandenberg described the use of 16S-PCR-DGGE to determine the role of epsilon-bacteria in children with abdominal complaints. Linda Mansfield provided a comprehensive overview of emerging *Campylobacter* species, while Lori Graham and Samuel Sheppard described new methods to differentiate and study *C. fetus* and *C. coli*, pathogens that are typically isolated from farm animals. There are also greater than 10 *Helicobacter* species sequenced. Bram Flahou described the non-*H. pylori* helicobacters (NHPh), such as *H. suis* that are also found in the stomach of both humans and pigs, and cause disease

despite lacking *H. pylori* virulence factors such as the vacuolating toxin VacA and the *cag* pathogenicity island (*cagPAI*) that encodes the type IV secretion pathway.

GENOMIC DIVERSITY

Both campylobacters and helicobacters are capable of varying the on/off status of several proteins allowing for the best adapted phenotype to predominate under a given selection pressure. One common mechanism of phase variation is through slipped-strand mispairing, where repeats of DNA (in the case of helicobacters and campylobacters, typically homopolymeric tracts of Gs or Cs) are either shortened or lengthened during DNA replication or repair resulting in premature stop codons in the translated protein. Genomic sequencing has identified many phase-variable genes in both genera—and there have been several reports in the literature about the consequences of varying bacterial surface structures. This is especially evident for *C. jejuni* where individual stocks of strain 11168 from different labs display different capsular polysaccharide (CPS) structures, and as such Brendan Wren appropriately called this organism “a moving target.” Generally speaking, there are also multiple gene products that influence motility in *C. jejuni*, thus demonstrating whether a mutation affects motility requires vigorous analysis since the random rate of this event is high—similarly, the motility of the population needs to be confirmed before doing any adherence/invasion or colonization studies since motility has been repeatedly shown to influence these phenotypes. For *H. pylori*, there are 46 genes prone to slipped-strand mispairing and many elegant studies have demonstrated the variation in O-antigen Lewis structures in the lipopolysaccharides (LPS), for instance, has a role in both host immune responses and bacterial colonization ability.

H. pylori faces multiple conditions in the gut such as penetrating the host mucous and tolerating high acid levels. The organism must also confront host inflammatory mediators and has a limited number of two-component signal transduction systems that commonly allow bacteria like *E. coli* to move between niches. Instead, evidence suggests that *H. pylori* uses genetic diversity for adaptation. Jay Solnick described how *H. pylori* can “tune” the host inflammatory response and adapt to the varying conditions in this environment in order to establish a chronic infection. Solnick talked about different mechanisms of genetic diversity and variation of BabA (ABO blood group adhesion) and other virulence factors. Sebastian Suerbaum further discussed mechanisms for *H. pylori* diversity including mutation-generating single nucleotide polymorphisms (SNPs) and recombination. Remarkably, *H. pylori* genomes are “sequence mosaics” and Suerbaum suggested they may actually be used as a method to trace human migrations. Suerbaum then demonstrated that DNA imports showed significantly increased frequency in the members of the protein family which includes the Bab proteins.

Since *C. jejuni* is naturally competent similar to *H. pylori*, Eduardo Taboada pointed out that there are multiple mechanisms for strain variation including exchange of genes and entire clusters by horizontal gene transfer, gene duplication, deletion, fusion, and contingency gene variation, while Christopher Bayliss described exciting new means to detect phase-variable

genes. Similarly, Lone Brondsted in collaboration with Szymanski described a study, published in this issue, examining the role of bacteriophage selection on *C. jejuni* CPS structure. Chickens were infected with a *C. jejuni* strain expressing phosphoramidate (MeOPN) on its CPS with or without a *C. jejuni* phage recognizing MeOPN. Six days later, pooled populations of *C. jejuni* from phage uninfected birds still expressed the MeOPN, were phage sensitive, and the MeOPN transferase gene showed 9 Gs (“ON”). In contrast, *C. jejuni* isolated from the phage-infected birds lacked MeOPN (in all cases except one where another phase variant was isolated), were phage resistant, and sequencing showed 8 or 10 Gs (“OFF”). Remarkably, the same level of *C. jejuni* colonization was observed in all chickens demonstrating that phage resistance does not always lead to an attenuated phenotype.

CONTROL MEASURES FOR *Campylobacter jejuni*

Qijing Zhang, Albert Lastovica, Hubert Endtz, and Patrick Kwan all presented results demonstrating that antibiotic resistance is increasing for *C. jejuni* and Francis Megraud showed similar trends for *H. pylori*. However, research is on-going to determine the molecular basis for *C. jejuni* resistance as Monika Keelan and Declan Bolton described. And scientists are using different approaches for antimicrobial development against *C. jejuni* including using CmeABC efflux pump inhibitors (Zhang), systems biology approaches (Mark Reuter and Arnoud van Vliet), and components of bacteriophages (Muhammad Javed/Szymanski). Birthe Hald presented a 4-year study on the effects of fly screens in poultry houses in Denmark and showed that screens significantly reduced flock prevalence of *C. jejuni*. Nigel French also demonstrated that a significant reduction in human cases was observed after introducing poultry interventions. Interestingly, although the majority of urban cases are associated with poultry, rural cases appear to be correlated with cattle and other environmental sources. Trudy Wassenaar emphasized that more work is needed to determine the numbers of infectious campylobacters in the environment and their source of contamination. Clarence Tam presented data from the infectious intestinal disease (IID) 2 study showing that the incidence of campylobacter in the UK has not changed in the last 15 years, while Jaap Wagenaar showed data from the European BIOHAZ group, and Birgitte Borck summarized the European CamCon project. In addition to providing a bigger-picture perspective, Julian Davies, an expert in the field of antibiotics and resistance, likewise echoed many of these sentiments in his talk and proposed novel, small molecule-based antimicrobial strategies in the future.

GLYCOBIOLOGY

Brendan Wren started this session with a prediction that *C. jejuni* will become better known for the birth of bacterial glycoengineering than as a notorious foodborne pathogen. His comments are based on the findings that *C. jejuni* was the first bacterium shown to possess an N-linked protein glycosylation pathway and that this pathway can be functionally transferred into *E. coli*. This resulted in the emergence of GlycoVaxyn, founded in 2004, and based on using the *C. jejuni* N-glycosylation machinery to

create novel bacterial glycoconjugate vaccines. Two talks in this session described the characterization of *N*-glycan pathways in other *Campylobacter* species (Harald Nothaft/Szymanski) and in related organisms such as *Wolinella succinogenes* (Jonathan Butler/Dennis Linton). Jos van Putten showed that *C. jejuni* *N*-glycans and some forms of lipooligosaccharides (LOS) are recognized by carbohydrate receptors on dendritic cells and may be able to modulate the host immune response. Then, Susan Logan described the process for *O*-linked protein glycosylation in both *C. jejuni* and *H. pylori* and the need for these glycan structures for proper flagellar filament assembly and subsequent motility. Due to the importance of flagella for both organisms, it is not surprising that small molecule inhibitors of the *O*-glycan pathway are being pursued. Interestingly, bacterial flagella are common pathogen-associated molecular patterns (PAMPs) that are recognized by Toll-like receptor (TLR)-5, but both *H. pylori* and *C. jejuni* evade this response. van Putten demonstrated that modification of *C. jejuni* flagella with *O*-linked glycans is not the mechanism for evasion, instead they identified a new β -hairpin structure involved in recognition. Stephen Trent described a new modification associated with the flagellar apparatus. Phosphoethanolamine is typically transferred to the LOS/LPS core of many bacteria including *C. jejuni* and *H. pylori*. The Trent group demonstrated that the *C. jejuni* phosphoethanolamine transferase not only modifies the LOS, but also the flagellar rod protein, FlgG. Mutation of the transferase results in abnormal flagellar filament assembly and reduced antimicrobial peptide resistance. Other forms of phosphorylated modifications were identified on the surface of *C. upsaliensis*: phosphoramidate on the CPS and phosphocholine on the LOS (Shauna Crowley/Szymanski), and data were presented demonstrating that both these modifications could contribute to bacterial survival and pathogenesis. Craig Parker described draft genomes of 56 *C. jejuni* and *C. coli* isolates and compared the LOS and CPS loci noting all the genetic mechanisms for diversity and some commonalities among the strains. Although *H. pylori* does not have an *N*-glycan pathway like *C. jejuni*, Mario Feldman showed that the *H. pylori* flippase was evolutionarily connected to the *C. jejuni* *N*-glycan pathway and is used for flipping LPS *O*-antigens into the periplasm. Feldman also demonstrated that using the *C. jejuni* *N*-glycan enzymes and the *H. pylori* fucosyltransferase together with enzymes from *Haemophilus influenzae*, he could engineer *H. pylori* Lewis antigens for the treatment of specific autoimmune diseases. Eleonora Altman, on the other hand, is interested in creating a vaccine against *H. pylori* and wants to avoid the Lewis antigens which mimic human Lewis structures. She has discovered a common α -1,6-glucan chain in clinical isolates of *H. pylori* which is now being explored. Jonathan Lane provided a different perspective to the glycobiology session when he discussed the use of sugars to inhibit bacterial binding. This would indeed be an inexpensive therapeutic, but various *C. jejuni* strains would need to be compared since it is known that *C. jejuni* infection in developing countries results in different disease symptoms. Also, Hubert Endtz reported that in their rural Bangladesh studies, breastfeeding (breast milk contains large amounts of potentially anti-infective oligosaccharides) did not prevent disease in children in the 0–6 month range.

Helicobacter pylori: CagA, VacA, AND HOST RESPONSES

CHRO 2011 saw a resurgence of presentations on *Helicobacter* biology and pathogenesis. In the areas of *H. pylori* pathogenesis and host interactions, a number of key advances on the Cag pathogenicity island (PAI) encoding a type IV secretion system were described. As Masanori Hatakeyama introduced, the CagA effector is injected into host cells, becomes tyrosine-phosphorylated by host cell oncoproteins, and mimics a host cell factor to activate or inactivate specific intracellular signaling pathways. In his presentation, Hatakeyama described the identification of several mammalian proteins containing CagA's critical EPIYA phosphorylation site motif, one of which was competitively inhibited by CagA for downstream events. This suggested that *H. pylori* acquired CagA to subvert one or more endogenous eukaryotic EPIYA-containing proteins to establish successful infection. Additional expert perspective on the impact of the *cagPAI* on host responses was provided by Thomas Meyer. Furthermore, three new protein components of the Cag type IV pilus that do not have homologs in other bacteria but are nonetheless required for CagA translocation into host cells and downstream events, such as IL-8 induction, were described by Carrie Shaffer/Timothy Cover, lending further insight into novel means by which *H. pylori* interacts with host cells.

A number of talks also addressed the intersection of CagA and iron. Manuel Amieva used live-cell microscopy data and other techniques to demonstrate that one of CagA's functions is to aid in iron acquisition from the host, and that this function is required both for microcolony formation on intercellular junctions and for colonization of gerbils. The model of iron-replete vs. iron-deplete gerbils described by Amieva was also used by his collaborators Jennifer Noto/Richard Peek, who showed that iron depletion significantly increased the frequency and severity of *H. pylori*-induced disease, including carcinoma, in a CagA-dependent manner. Noto and Peek's work also suggested that regulation of *H. pylori* virulence factors by iron availability contributes to these findings, a theme mirrored by D. Scott Merrell's presentation showing connections between CagA and the ferric uptake regulator Fur. Merrell's group found that Fur positively regulates CagA expression via a Fur box in the *cagA* promoter and that Fur is most important in early stages of infection. All three of these presentations clearly show that iron availability and acquisition play a critical role in *H. pylori* disease etiology, mediated at least in part by CagA.

Advances in understanding roles for the vacuolating cytotoxin VacA in pathogenesis were also presented. In addition to his CagA work, Manuel Amieva described how VacA influences the pathogen-host iron homeostasis via inducing mislocalization of the host epithelial cell transferrin receptor. VacA's striking effect on mitochondrial morphology was addressed by Steven Blanke, who presented work demonstrating VacA's activation of a host cell dynamin-related protein (Drp1) and the relationship of this activation to uncoupling of mitochondrial fission and fusion and induction of apoptosis. Blanke further hypothesized that these events aid *H. pylori* colonization by crippling cells at the epithelial barrier, rendering them less fit to respond to *H. pylori* infection.

Additional insight into mechanisms underlying host responses to *H. pylori* infection was provided by three different presentations. In one talk, James Neal/Karen Guillemin showed that transgenic zebrafish expressing CagA displayed early intestinal proliferation, adult intestinal hyperplasia, and activation of the Wnt pathway, further indicative of a key role for CagA in *H. pylori*-induced carcinogenesis. A connection between *H. pylori* chemotaxis and immune responses was demonstrated by Annah Rolig/Karen Ottemann, who showed that chemotaxis mutants were defective for early gastric recruitment of CD4⁺ T cells and induction of a Th17 immune response component implicated in apoptosis. As this is the first study showing an effect of bacterial chemotaxis on apoptosis, these findings will likely have relevance for other systems as well. Finally, Anne Mueller's talk addressed an intriguing paradox: namely, that persistent infection with *H. pylori* is linked to protection from allergic, chronic inflammatory, and autoimmune disease. Using an asthma model, Mueller showed an interconnection between *H. pylori* infection, immune cell responses, and reprogramming of dendritic cells toward a tolerance-promoting state. Mueller further hypothesized that this beneficial aspect of *H. pylori* infection may contribute to persistence of *H. pylori* among the human population.

Campylobacter jejuni: NEW ANIMAL MODELS PROVIDE INSIGHT INTO HOST AND BACTERIAL FACTORS MEDIATING DISEASE

A number of diverse topics were covered in sessions pertaining to *C. jejuni* pathogenesis, host responses, and host cell interactions. In addition to Patricia Guerry's overview of the contribution of CPS to virulence, various aspects of the *C. jejuni*-host cell interaction were described by Dennis Kopecko, Nicole Iovine, Dominic Mills/Nick Dorrell, and Lieneke Bouwman/Jos van Putten. Furthermore, a number of new connections between the LOS and/or sialylation and Guillain-Barré Syndrome were drawn by Ruth Huizinga, Arnoud van Vliet, and Astrid Heikema/Janneke Samsom.

An emerging area of interest, covered by two talks, is the utility of new or recently developed small animal models of disease in exploring *C. jejuni* disease markers and identifying new host and bacterial factors important for disease etiology. Christian Jobin used IL-10^{-/-} mice expressing GFP from the NF- κ B promoter, which develop invasive, campylobacteriosis-like colitis, to explore host signaling pathways involved in *C. jejuni*-induced inflammation. Daily injections with rapamycin allowed *C. jejuni* to colonize the intestine but prevented extra-intestinal dissemination and inflammation, implicating the mammalian mTOR pathway specifically in disease. Additional experiments suggested these effects were independent of T-cell activation, suggesting mTOR signaling components as possible targets for new treatments. Markus Heimesaat described the development, along with co-author Stefan Bereswill, of a new gnotobiotic mouse model harboring "humanized" gut flora. *C. jejuni* stably colonized these mice and elicited a pro-inflammatory immune response that was dependent on the host innate immune receptors TLR4 and TLR9, and on *C. jejuni* formate dehydrogenase utilization genes.

IN THE NICHE: INTERACTION OF CHRO WITH MICROBIAL COMMUNITIES AND SURVIVAL STRATEGIES IN DIVERSE ENVIRONMENTS

An emerging theme in CHRO research, and indeed in bacterial pathogenesis, is the interaction of CHRO and other "infecting" bacteria with resident microbial communities. This was introduced at CHRO 2011 by Brett Finlay, who described research in his laboratory dissecting the interplay between *E. coli* and *Salmonella* with gut microflora, and is also of relevance to the findings described above by Heimesaat and colleagues regarding their humanized gut flora mouse model of *C. jejuni*-mediated inflammation. Two *H. pylori* talks also addressed this topic. Richard Peek discussed this from the perspective of microbial and host diversity, and the contribution of each to stomach cancers. Peek noted that in addition to contributions of *H. pylori* intra- and inter-genomic diversity, *H. pylori* exist in a distinct gastric microbial ecosystem which may provide an additional genetic pool allowing *H. pylori* to develop traits further influencing its propensity to cause gastric cancers. Also noted were findings showing that host factors modulating immunity, and thus presumably also the microflora composition, likewise impact gastric cancer risks. Anica Wandler/Guillemin presented new results from a transgenic *Drosophila* model showing that intestinal expression of CagA alters the composition of the gut microbiome, further implicating CagA in new areas of *H. pylori*-host biology.

Survival strategies, primarily in the form of stress responses, were also discussed in the context of life "in the niche." Arnoud van Vliet, in a talk entitled "Try not to breathe . . .," provided an overview of how the microaerophilic *C. jejuni* counters various oxygen concentrations ranging from virtually anaerobic to atmospheric (aerobic) as it moves from niche to niche, as well as conditions caused by reactive oxygen species. van Vliet also noted that *C. jejuni* forms enhanced biofilms in aerobic conditions. Andrew Cameron/Erin Gaynor presented the first global characterization of the response of *C. jejuni* to hyper-osmotic conditions, some of which are encountered during host colonization. Finally, Alain Stintzi discussed the PerR regulon as a means by which *C. jejuni* counters oxidative stress, as well as how *C. jejuni* acquires iron, the importance of iron-related genes in colonization, and regulation of those genes by the regulator Fur.

FUNDAMENTAL BIOLOGY OF CHRO: BACTERIAL CELL SHAPE, METABOLISM, AND CHEMOTAXIS

Also new to the CHRO workshop was a session dedicated to research on basic bacterial processes. Well-suited to this theme were two talks exploring means by which *H. pylori* and *C. jejuni* derive their helical shape, and the ramification of shape/peptidoglycan alterations on other important processes. Nina Salama described the first characterization of shape-related genes in *H. pylori*. Deletion mutants displayed a variety of shape abnormalities ranging from "s" and "c" shapes with exaggerated curvature to straight rods. Some of the proteins involved, termed "Csd" for "cell shape determinant," are novel peptidoglycan peptidases, while others perform other cellular functions. Salama proposed two independent peptidoglycan modification networks, demonstrated roles for each of the *csd* genes in a mouse model of colonization, and described models for how each shape

change affects *H. pylori* motility. An independent screen in *C. jejuni*, described by Emilisa Frirdich/Gaynor, also identified a peptidoglycan peptidase (“Pgp”) as the first factor to impact *C. jejuni* shape. Deletion of this gene resulted in a straight rod morphology, modest motility and biofilm defects, peptidoglycan that hyper-activated the intracellular innate immune receptor Nod1, and a defect in chick colonization. This gene was a homolog of an unpublished *H. pylori* *csd* described by Salama, deletion of which also caused a straight rod phenotype, and the specific enzymatic activity of each protein on peptidoglycan was determined. The work by Frirdich/Gaynor also provided the first muopeptide map for *C. jejuni*.

Dave Kelly presented a comprehensive talk on numerous aspects of *C. jejuni* metabolism. One was the *C. jejuni* strain-dependent utilization of select amino acids as growth substrates, and his group’s recent observation that exogenous peptides can provide a source of amino acids. Kelly also described the highly complex and branched respiratory chains in *C. jejuni*, that new types of electron donors and acceptors distinct from those in other bacteria have recently been identified, and the dependence on the twin-arginine transporter (TAT) system for delivery of many important respiratory enzymes to the periplasm (and thereby dependence on the TAT system for the ability of *C. jejuni* to respire on substances requiring those enzymes). Hilde De Reuse presented data on the role of the pleiotropic *H. pylori* nickel-dependent regulator NikR to activate or repress important target genes such as *ureA* and *hydA*, which in turn encode urease and hydrogenase for which nickel is a co-factor and which are essential for *H. pylori* colonization. De Reuse showed that NikR-dependent repression occurred at higher nickel concentrations than activation, suggesting a chronological hierarchy and elaborate mechanisms for NikR target discrimination.

Chemotaxis in both *C. jejuni* and *H. pylori* was also discussed. Victoria Korolik presented findings on the first *C. jejuni* sensory receptor, Tlp1, re-named CcaA for its role as an aspartate receptor, and its role in motility, colonization, and cell invasion. As Kelly noted, aspartate is a major amino acid used as a *C. jejuni* carbon source, emphasizing the importance of CcaA to *C. jejuni* biology. Korolik further showed that for CcaA, signal transduction occurs via binding to CheV and not CheW, suggesting differences in this pathway from other enteric pathogens. Karen Ottemann described the chemotactic program in *H. pylori*, the phosphorylation-based signal transduction cascade, how the *H. pylori* system differs from that of *E. coli* and *Bacillus subtilis*, and other findings on newer components involved in *H. pylori* chemotaxis.

FUTURE PERSPECTIVES

These are exciting times in CHRO research—in the past, we have looked toward *E. coli* and Salmonella for understanding bacterial physiology—now campylobacter and helicobacter have become model systems for understanding bacterial glycobiology, survival in mucin, metabolism of microaerophiles, and the importance of cell shape and motility. There is also a growing appreciation for the importance of CHRO other than *C. jejuni* and *H. pylori*, and as such it is anticipated that we will learn much more about these organisms and their possible roles in human/animal health and disease. Although we were unable to highlight all of the oral talks or the outstanding poster presentations, we look forward to the next few years of CHRO publications. There is a bright future ahead for CHRO research, and we would particularly like to highlight the Young Investigator Award winners: Cody Buchanan, Jonathan Butler, Ilana Cohen, Shauna Crowley, Rajinder Dubb, David Hermans, Laura MacRitchie, Ana Martins, Dominic Mills, James Neal, Jennifer Noto, and Christian Penny. We look forward to many CHRO meetings to come.

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