出國報告(出國類別:研究)

### 參加歐盟疾病管制局(ECDC)爲期兩 年 EPIET 訓練

服務機關:行政院衛生署疾病管制局

姓名職稱:郭宏偉 技正

派赴國家:奧地利

出國期間:96年9月至98年11月

報告日期:99年1月22日

### 摘要

職於九十六年九月奉派前往奧地利衛生暨食品安全署(Österreichische Agentur für Gesundheit und Ernährungssicherheit GmbH, AGES)參加歐盟疾病管制局(ECDC)舉辦爲期兩年的 EPIET(European Programme for Intervention Epidemiology Training)訓練計畫及實習課程。

期間與歐盟各國前後期學員約六十人參加了基礎流行病學、疫病調查應用軟體、多變項統計分析、時間序列分析、疫苗學、公共衛生事件緊急應變等專業課程,並於三場國際學術研討會中進行口頭報告及張貼海報。本訓練課程之講義與授課方式亦獲 ECDC 的同意,可提供本局做為 FETP 課程規劃安排之參考。

在 AGES 實習期間,進行奧地利結核病監視資料趨勢分析、奧地利結核病監視系統評估計畫,協助該機關進行食物中毒、麻疹、德國麻疹等疫情調查,並以第一作者身份於學術期刊發表三篇論文,完成奧地利結核病流行趨勢分析報告書,並協助完成奧地利結核病監視系統評估報告。

EPIET 首席協調員 Viviane Bremer、AGES 人類醫學處處長 Dr. Allerberger 與課程督導員 Dr. Schmid 對於本次台灣與 EPIET/ECDC 以及 AGES 的合作均表示肯定,並且同意循此模式繼續合作交流。

### 目次

一、緣起	4
二、目的	4
三、訓練過程	5
四、EPIET 簡介	6
五、奧地利衛生暨食品安全署 (AGES) 簡介	7
六、EPIET 課程	8
七、國際學術研討會	10
八、AGES 實習內容	12
九、心得與建議	13
十、附件	15

### 一、緣起

本案係外交部駐奧地利代表處與奧地利衛生暨食品安全署(AGES)治商,並經奧方同意提供一名員額予我方參與歐盟疾病管制局辦理之 EPIET 訓練課程,並於 AGES 完成該訓練計畫所要求達成之實習項目。外交部據此來函詢問本局派員之意願,本局鑒於此案有助於本局與歐盟疾病管制局連繫管道之建立,選派職參加此訓練計畫。

### 二、目的

鑒於本案係首次由本局同仁派赴歐盟參加 ECDC 舉辦爲期兩年之 EPIET 訓練計畫,擬藉由參加該訓練課程之機會收集相關訓練教材,因該計畫有機會前往歐盟其他國家參加訓練課程及開會,因此本計畫除接受流行病學相關訓練及實習外,亦可能赴疫區實地進行疫情調查,並撰寫研究報告,擬藉此受訓機會多與歐盟國家接觸,建立聯繫管道。本案亦爲本局首次與奧地利衛生暨食品安全署(AGES)進行合作交流,期望藉此機會加強雙邊合作關係,並擴展合作層面。

### 三、 訓練過程

日期	內容
96/9/11	啓程前往維也納
96/9/12-17	安頓住處與熟悉環境
96/9/18	首次參加 AGES 例行晨會,自我介紹
96/9/19-21	辦理居留證
96/9/22-10/12	前往西班牙參加基礎流行病學課程
96/10/13-16	AGES 實習
96/10/17-21	前往瑞典參加 ESCAIDE2007 國際學術研討會
96/10/21-97/1/19	AGES 實習
97/1/20-26	前往葡萄牙參加疫病調查應用軟體課程
97/1/27-4/5	AGES 實習
97/4/6-12	前往瑞典參加多變項統計分析課程
97/4/13-6/19	AGES 實習
97/6/20-27	前往挪威參加時間序列分析課程
97/6/28-8/30	AGES 實習
97/8/31-9/6	前往立陶宛進行研究計畫進度報告
97/9/7-11/17	AGES 實習
97/11/18-22	前往德國參加 ESCAIDE2008 國際學術研討會
97/11/23-98/4/18	AGES 實習
98/4/19-25	前往芬蘭參加疫苗學課程
98/4/26-6/20	AGES 實習
98/6/21-27	前往英國參加衛生事件緊急應變課程
98/6/28-8/29	AGES 實習
98/8/30-9/5	前往義大利進行研究計畫進度報告
98/9/6-10/24	AGES 實習
98/10/25-29	前往瑞典參加 ESCAIDE2009 國際學術研討會
98/10/30-11/26	業務交接及準備返台事宜
98/11/27	返台

### 四、 EPIET 簡介

歐洲流行病學訓練計畫(European Programme for Intervention Epidemiology Training, EPIET) 起始於 1996年,初期所有運作經費皆來自於歐盟議會;自 2007年起,該訓練計劃改由歐盟疾病管制局(ECDC)負責。此訓練計畫現由 ECDC 負責專業課程的規劃與授課,另由代訓機構提供受訓學員訓練與實習的機會。目前共有 42 個代訓機構,代訓機構主要是歐盟各會員國的國家級或區域級傳染病監測與防治中心,主要分布在德國、法國、西班牙、義大利等 26 個國家。此訓練計畫的受訓學員是以對實地流行病學(field epidemiology)有興趣且在公共衛生(public health)領域有實際經驗者爲招募對象。

### EPIET 的主要任務包括:

- 加強歐盟國家傳染病監測與防治
- 建立歐洲流行病學家網絡
- 提升歐盟國家對於重大衛生事件之應變能力

### 主要目標爲:

- 強化歐盟會員國傳染病監測能力
- 提升歐盟會員國面對傳染病威脅時快速有效的田野調查與防治的能力
- 建立歐洲流行病學家網絡,使用一致的研究方法分享共同成果
- 建構歐盟地區傳染病監測與防治網絡

每一屆招募受訓學員約 20-30 名,有意申請者須先經過 ECDC 以及代訓機構的學經歷表書面審查以及訪談面試,經錄取者始得參加此兩年訓練計畫。

在爲期兩年受訓期間,ECDC將定期安排受訓學員參加專業課程訓練,包括基礎流行病學、疫病調查應用軟體、多變項統計分析、疫苗學等必修課程,以及溝通、論文寫作、時間序列分析及衛生事件緊急應變等選修課程,受訓學員除了必須完成所有必修課程外,還須完成至少兩項選修課程,此外受訓學員須於國際學術期刊發表至少一篇論文,以及在國際學術研討會發表論文演說,受訓學員每三個月須回報在代訓機構的實習過程以及所進行的研究計畫的進度,並由代訓機構評量受訓學員是否具備執行疫情調查、評估或建立監測系統、資料分析以及教學等能力,完成以上目標並經EPIET認可後,才能領取EPIET結業證書。

### 五、 AGES 簡介

奥地利衛生暨食品安全署(Österreichische Agentur für Gesundheit und Ernährungssicherheit GmbH, AGES)係依據奧地利食品法規定於 2002 年成立,將 18 個不同領域的聯邦機構與部門,包括:食品檢驗、微生物學、血清學、獸醫、農業及人類醫學等部門整合併入該機構內,負責食品來源、食品製程與運送、食品保存至食品消費等食品供給與消費鏈的安全,以及負責調查、預防及控制傳染病疫情。

### AGES 下轄七個部門:

- 人類醫學
- 農業
- 食品安全
- 獸醫學
- 藥品安全
- 能力中心
- 資料收集、分析與風險評估

此次我的實習單位是在人類醫學處下的傳染流行病學中心,人類醫學處處 長 Dr. Allerberger 亦爲促成此次台奧合作案之推手,傳染流行病學中心的主要工 作任務包括:

- 疫情爆發事件調查
- 傳染病監測
- 傳染病監測資料分析
- 傳染病監測系統評估
- 建立傳染病防治工作指引
- 教育訓練
- 擔任 EPIET 代訓機構

### 六、 EPIET 課程

### 1、基礎流行病學課程

基礎流行病學課程爲期三週,課程中詳細介紹各種流行病學研究方法(case-control study、cohort study、case-cohort study、nested case-control study)、疾病的測量方法(盛行率、發生率、發生密度)常見的誤差、生物統計基本概念以及一些實務上的應用,課程是以課堂講解與互動式的個案研究等教學方式進行,課程內容包括:

- 流行病學調查方法
- 疾病的測量
- 抽樣方法
- 分析流行病學
- 統計檢定
- 環境流行病學
- 建立良好監測系統的原則
- 媒體溝通

### 2、疫病調查應用軟體課程

本課程是以介紹 EpiData 電腦軟體爲主,包括如何利用 EpiData Entry 建立適當的資料庫架構來儲存資料、設計資料輸入介面、如何處理 missing data,以及運用 EpiData Analysis 及 Stata 統計分析軟體進行統計分析和圖表製作。

### 3、多變項統計分析課程

本課程除了介紹多變項分析在流行病學研究上的應用外,亦詳細介紹多變項分析的統計法,包括:簡單線性回歸模式(linear regression model)、邏輯斯回歸模式(logistic regression model)、波以松回歸模式(poisson regression model)等,如何選擇正確的多變項分析方法,介紹 Stata 統計分析軟體指令來執行多變項分析,如何正確解釋回歸分析的結果,如何進行分層分析,如何辨別干擾因子與交互作用,如何運用多變項分析模式消除干擾因子的影響,以及如何選定最適當的模式。此外,每天下午安排個案研究,

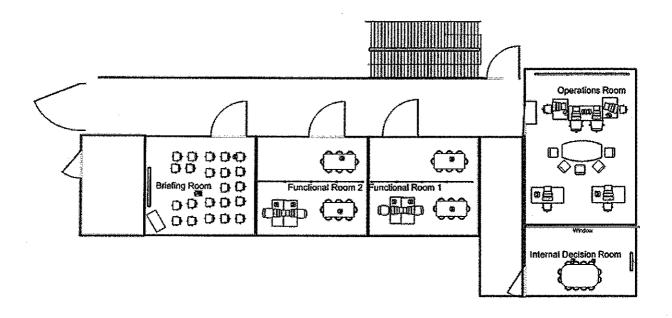
配合上午的課程內容,讓所有學員在小組中討論,並實際運用 Stata 進行多 變項分析作業。

由於本次課程在歐盟疾病管制局所在地瑞典斯德哥爾摩市舉行,因此 特地安排學員有半天參訪歐盟疾病管制局的行程,此次主要參訪歐盟疾病管 制局緊急應變中心(Emergency Operations Centre, EOC)。為因應緊急 公共衛生事件,歐盟疾病管制局在設立之初即建置了緊急應變中心,並且擬 定公共衛生事件緊急應變原則:

- ECDC 常規業務仍須繼續運作,爲優先順序可能調整
- 應變團隊的指揮官著重於專業領導及各工作小組間之協調
- 應變團隊核心成員應從日常業務中完全抽離並專注於該緊急事件
- 應變團隊的所有決策需在歐盟疾病管制局局長監督之下

該中心設置有一間作戰室,是應變中心的運作樞紐,所有公共衛生事件相關的資訊均匯集於此,各功能小組負責人亦進駐於此;一間決策室,由決策小組研判各項資訊後再此完成決策並向指揮官進行簡報;兩間幕僚室,由各功能小組人員進駐於此,進行資料彙整及分析等工作;一間簡報室,爲工作幕僚討論與交換意見,以及應變團隊對外發言場所(如圖一)。

### 圖一 緊急應變中心平面圖



### 4、 時間序列分析課程

本課程介紹時間序列分析的基本概念以及如何利用時間序列分析解釋疾病動態趨勢,剖析時間序列模式組成成分包括:長期趨勢(long-term trend)、季節性(seasonality)、週期性(periodicity)以及不規則性(white noise),應用 Stata 分析軟體進行資料處理,將資料轉化爲適合時間序列分析之格式,偵測極端值、平滑(smoothing)歷史資料及預測未來趨勢等。

### 5、疫苗學

本課程介紹疫苗發展歷史、以及疫苗可預防疾病的特性、如何運用流 行病學方法評估疫苗效益、疫苗的製作與疫苗安全性評估等議題,課程中也 簡報歐盟國家的疫苗政策,以及一些進行中的疫苗計畫。

### 6、 公共衛生事件緊急應變課程

本課程介紹何謂公共衛生事件,如何快速進行事件影響評估、如何訂定應變計畫、應變計畫應考慮到的面向、如何評估應變計畫以及如何進行媒體溝通等議題,課程中有幾位講師將他們在非洲難民營進行人道救援的實際經驗當做課程內容以及個案研究的教材,讓參訓學員進行分組討論,同時藉由當地舉辦大型音樂季的機會,讓學員們實地進行大規模人群聚集時的抽樣與調查工作。

### 七、 國際學術研討會

### 1 · ESCAIDE2007

ESCAIDE (European Scientific Conference on Applied Infectious Disease Epidemiology) 是由歐盟疾病管制局所主辦的國際學術研討會,旨在加強並擴展應用傳染流行病學網絡合作、分享知識與經驗、提供EPIET/FETP 訓練學員分享其研究成果之平台。此次研討會主要討論議題包括老年流行病學、流行性感冒、食因性傳染病、疫苗可預防疾病、疾病監測、環境流行病學、爆發流行疫情調查以及性傳染病方面等議題。

### 2 · ESCAIDE2008

在本次研討會中,職以海報(poster presentation)方式在 Food & water borne disease- Outbreak 議題發表論文- A foodbrone Norovirus outbreak likely related to the infected infant of a food handler, Austria, 2007 (如附件

一),2007年聖誕假期期間於奧地利下奧地利省爆發一起疑似參加聖誕晚宴引起的腸胃炎聚集事件,經地方衛生單位通報至奧地利衛生暨食品安全署(AGES)並請求 AGES 協助調查後,我們所進行的一項回溯性世代研究(retrospective cohort study)調查報告,在這項調查中,我們確認了致病原為 Norovirus,找出了被污染的食物,但由於與會者與餐廳負責當天餐飲的員工在該聚集事件發生前都沒有腸胃炎的症狀,於是進一步調查後發現,當晚預備該項被污染的食物的餐廳員工其六個月大的嬰兒再三天前曾因為Norovirus 感染腹瀉住院治療,因此研判此事件的感染源爲該嬰孩,並且藉由該名廚房員工將病毒帶入工作場所。

### 3 · IMED2009 (International Meeting on Emerging Disease and Surveillance)

在本次研討會中,職以海報方式發表論文- Tuberculosis in Austria: An Analysis of the past 10 years, 1997-2006 (如附件二),此論文是職在奧地利進行的研究計畫中的部份成果,旨在分析過去十年間結核病在奧地利的流行趨勢;自 1997 年至 2006 年十年間,奧地利共有 11,496 名新發生的肺結核病例,發生率呈現穩定的下降趨勢,自 1997 年每十萬人口 18.7 例,下降至 2006 年的每十萬人口 10.8 例,外來移民人口的肺結核發生率顯著高於奧地利本國國民,85.7%(72/84)多重抗藥性結核(MDR-TB)病例發生在外來移民人口,且外來移民的發生率趨勢與移民趨勢明顯相關,主要導因於 1999 年與 2004 年發生的柯索伏(Kosovo)戰爭與車臣(Chechnya)內戰,造成大量難民進入奧地利,尋求政治庇護。

### 4 · ESCAIDE2009

在本次研討會中,職以"Did increasing immigration affect the epidemiology of TB in Austria, 1997-2006"爲題進行口頭報告(如附件三);延續先前研究結果,爲探討外來移民人口對奧地利結核病流行趨勢的影響,運用相關分析法(correlational analysis)發現,外來移民對於奧地利本國國民以及長期居留在奧地利的外來移民的結核病流行趨勢並無影響,外來移民的結核病病例主要來自於高結核病發生率國家的移民,顯示主要爲潛伏結核感染病例(latent TB cases)。

### 八、 AGES 實習內容

### 1、疫情調查

由於奧地利衛生體系與台灣不同,聯邦政府未經各省政府授權或請求協助,不得逕行介入公共衛生事件調查。職在 AGES 實習期間,參加過三次急性腸胃炎聚集事件疫情調查,主要負責問卷設計與製作、研究方法設計、資料輸入與整理、資料分析、調查結果報告撰寫與投稿學術期刊(附件四、五、六)等工作。另外亦協助進行麻疹、德國麻疹群聚事件的疫情調查資料分析。

### 2、 教學經驗

AGES 每年均舉辦一次爲期三天的基礎流行病學與資料統計分析軟體 (Epilnfo)教育訓練課程,受訓學員爲各地方衛生官員,或是醫療院所醫 護人員,職在此訓練課程中擔任統計分析軟體課程的講員,負責課程內容規 劃、授課講義製作、電腦實機操作與問題解答。此外,維也納國際學校(Vienna International School)也與 AGES 合作,於 2009 年開辦基礎流行病學課程,其中亦安排了半天的統計分析軟體(Epilnfo)操作課程,亦由職擔任該課程講員。

### 3、 專題研究

在 AGES 實習期間,另外負責一項研究專案是奧地利結核病監視資料分析,我們分析了 1997 年至 2006 年奧地利結核病監測資料,結果顯示:如同一般的高收入國家像美國、加拿大、西歐等國家,奧地利公民的結核病發生率呈現穩定下降趨勢,而外來移民的結核病發生率與來自結核病高盛行率國家的移民人數呈現顯著正相關,可能是受來自結核病高盛行率國家的潛伏結核感染病例所影響,尤其在 1999 年、2000 年、2004 年以及 2005 年,外來移民人口中的結核病患增加是由於這些年的移民人口中,有非常高的比例是來自高結核病盛行率國家的難民。我們也發現,移民對於奧地利公民的結核病發生率與已長期居留在奧地利的外來移民的結核病發生率並沒有太大影響。來自高結核病盛行率國家的移民可能是低結核病盛行率國家的結核病再現的重要因素,幾乎所有高收入國家對於申請永久居留的外來移民都要求進行結核病胸部 X 光的篩檢,在奧地利,針對難民營中的居民亦是依此進行篩檢,但是並沒有進行潛伏結核感染病例的篩檢。結論建議針對高危險移民人口如難民營居民,應該進行潛伏結核感染病例的篩檢。結論建議針對高危險

防性治療。(分析報告掛載於 AGES 網站:

http://www.ages.at/ages/gesundheit/mensch/tuberkulose/tuberkulosereport/)

### 九、 心得與建議

謝謝本局長官的推薦,職有幸參加此次 EPIET 的訓練課程。由於是本局第一次派員參加歐盟疾病管制局舉辦的長期訓練計畫,也是本局第一次與奧地利進行合作交流,職抱持著戒慎恐懼的心情全力以赴,以期完成預定目標。

在這兩年訓練期間,主辦單位(EPIET coordinator team)每二至三個月就會安排一項課程,課程主要是以課堂講課與分組討論的方式進行,一般來說,上午是以課堂講授方式,下午則依據上午授課內容,設計個案研究的劇本內容,由六到八名學員分成一組,每一組均有兩位督導員協助個案研究的進行,在個案研究進行過程中,由學員依次輪流朗讀劇本內容、陳述問題以及回答問題,針對問題的回答,其餘學員再進行腦力激盪予以補充。由於個案研究內容都是課程設計小組過去所經歷的實際案例,因此所設計的問題,也是當時他們所實際碰到的問題,如此的設計讓學員們很有臨場感以及實用性。受訓學員們大部分都具有醫護、公衛或生物統計的背景,且有臨床或公共衛生的實地經驗;透過學員們的互動與腦力激盪,分享各自過去的經驗,有時所想到的解決方案,也讓督導員們有意想不到的收穫。

每天的課程結束後都會針對當天的課程設計進行問卷調查與評比,在 最後一天課程結束前,將所有回收的問卷進行統計,由授課講師、督導員與 學員齊聚一堂,公開進行課程檢討,學員們可自由發表意見,有褒有貶,不 過大家都知道,目的是爲了讓課程更趨完善,使以後的學員受惠。

由於每次的課程都安排在不同的國家城市中舉行,所以學員們都把握這難得的機會,除了分享彼此在不同代訓機構的實習生活點滴,也盡情瀏覽每個城市的風情文化,主辦單位也會特別安排一場晚宴,讓大家彼此交誼,聯絡感情。

此行另一重點是在奧地利衛生暨食品安全署的實習,由於此次是本局第一次與該署的合作交流,可以感受到奧方相當重視,對於初到維也納的我提供了許多生活上的協助。在 AGES 受訓期間主要負責結核病監測資料分析,由於該署自 2002 年正式成立以來,重點業務主要放在食品安全與畜牧衛生方面,對於傳染病監測業務也是近年才從衛生部接手部分傳染病的監視,因此從未針對傳染病監測系統與監測資料進行評估與分析,由於奧地利

的傳染病監測系統自 2007 年才轉爲網路通報方式,通報資料的檢核機制並不完善,所以首要任務是建立資料檢核的基本作業流程,將重複通報以及錯誤資料勾稽刪除與修正,資料庫整理完畢後,進行資料分析工作,完成報告撰寫。對於此項任務的完成, Dr. Allerberger 與 Dr. Schmid 均給予當肯定的評價,同時亦將此資料處理的 SOP,提供給 EPIET 作爲參考範本。此外,三次的疫情調查事件,也讓我從流行病學的研究設計到論文撰寫的各項細節,累積了許多寶貴的經驗。

在這期間,由於 AGES 意欲建立腸病毒檢驗能力,因此建議該署派員來本局進行合作交流,學習腸病毒檢驗技術。該員來台期間,接受本局研究檢驗中心相當大的協助,並共同發表了一篇學術論文,這些都讓該署覺得獲益良多,也更加深了彼此合作交流意願。

綜合以上心得,提出幾項建議:

- 1. 提供 EPIET 課程教材與本局應用流行病學專業人員訓練班, 作爲未來課 程規劃之參考。
- 2. 建議本局應用流行病學專業人員訓練班學員亦可投稿參加 ESCAIDE 國際學術研討會,藉此機會與歐洲流行病學家進行經驗分享,也可擴展本局 FETP 與 ECDC/EPIET 的交流。
- 3. AGES 未來將加強傳染病監測工作,進行監測系統評估與改善計畫,本 局可加強與該署在此方面的合作。
- 4. 邀請該署人員參訪本局或參加本局所舉辦之國際學術研討會,以加強雙 方人員互動,建立夥伴關係。

附件—

### 19.7 Reference: 20080366

Track: Food & water borne diseases - Outbreaks

### A foodborne Norovirus outbreak likely related to the infected infant of a food handler, Austria, 2007

Hung-Wei Kuo (1, 2, 4), Daniela Schmid (1), Karin Schwarz (1), Eva Magnet (3), Anna-Margaretha Pichler (1), Franz Allerberger (1)

- 1. Austrian Agency for Health and Food Safety (AGES), Vienna, Austria
- 2. European Programme for Intervention Epidemiology Training (EPIET), European Centre for Disease Prevention and Control, Stockholm, Sweden
- 3. Public Health Authority of the Province of Upper Austria, Linz, Austria
- 4. National Health Command Center, Center for Disease Control, Taipei, Taiwan

### BACKGROUND

A cluster of gastroenteritis due to norovirus occurred within two days after a Christmas party in a restaurant on December 14, 2007. Consumption of food at the party was the potential common link among all patients. We investigated the outbreak to identify the source of the outbreak and the vehicle(s) of transmission.

### METHODS

We defined a case as a person [1] who attended the party or prepared food for the party dinner[2] who fell ill with symptoms of diarrhea or vomiting after December 14. We conducted a retrospective cohort study among participants and kitchen staff of the restaurant. We calculated food-specific risk ratio (RR) and used a log-linear model for multivariable analysis. Stool samples of nine cases were tested for norovirus and diarrhoea causing bacteria by the Austrian reference laboratory for norovirus.

### RESULTS

A total of 21/63 cohort members fulfilled the definition of a case. Eight cases were female. 95% of cases fell ill within 48 hours following food exposure. Three cases tested positive for Norovirus. Consumption of "schinkenrolle" (ham roll) was associated with risk of gastroenteritis (adjusted RR: 3.9, Cl95%: 1.6-9.8). Neither food nor environmental samples were available for microbiological investigations. A kitchen assistant's infant fell sick with norovirus gastroenteritis two days prior to the party.

### CONCLUSIONS

As "schinkenrolle" is usually consumed raw, it was most likely contaminated with norovirus during preparation by the kitchen assistant. Not only food handlers but also their household contacts should be considered as a potential source of foodborne outbreaks. Food handlers should be informed of the risk of transmission of gastrointestinal illness from infected relatives and raise awareness about the hand hygiene.



### International Meeting on Emerging Diseases and Surveillance 2009

1345 (69.9%) of cases up to 2005 had a recorded treatment outcome. 1145 (85.1%) of cases had a successful treatment outcome. Spatial analysis identified one significant geographical cluster. Logistic regression showed the likelihood of successful treatment was significantly increased in those aged less than 75 years, non-Caucasian with no recorded risk factors for TB. The likelihood of successful treatment was significantly decreased in two of 14 NHS Board areas, those receiving three or fewer anti-tuberculous drugs and those with drug resistant organisms.

Conclusions: Distinct spatial and demographic patterning exists in drug therapy and treatment outcome for TB in Scotland but further investigation is required to understand the public health implications.

### Seroprevalence of Leptospirosis in São Paulo, Brazil

E.C. Romero, R.M. Blanco. Adolfo Lutz Institute, São Paulo, Brazil

Background: Leptospirosis is a zoonotic disease that has now been identified as an emerging infectious disease. It is caused by pathogenic spirochetes of the genus Leptospira. The natural hosts for Leptospira spp. come from a variety of species of which the rodent is the most important reservoir. Leptospirosis epidemics are often related to heavy rainfall and flooding favorable for rodent infestations. Because of its climate, São Paulo may be at high risk for leptospirosis. This study was undertaken to study the epidemiological profile of urban leptospirosis in the city of São Paulo, Brazil.

Methods: Retrospective study was undertaken from January 2003 to December 2007. A laboratory-confirmed case is defined as 4-fold rise in titer between acute and convalescent phase samples and presumptive when a single sample showed a minimum titer of 1:200 or when two or more samples did not show a four-fold increase in titer.

Results: A total of 520 cases were recorded. A peak was observed in 2004 and 2007 with 107 and 136 cases, respectively. The mean annual incidence rate was 0.53 per 100,000 population. Although leptospirosis occurred in persons of all ages, middle-aged adults were most frequently infected: 23.46% of the cases were in adults from 31 to 40 years of age, mostly males (85.58%) with an overall ratio of males to females of 5.9:1. Among the cases, 406 (78.08%) were considered serologically confirmed cases and 114 (21,92%) presumptive cases. Cross-agglutination with at least two serogroups occurred in 92 cases. Icterohaemorrhagiae was the predominant serogroup (297 cases, 57.11%), followed by Autumnalis (34 cases, 6.54.%) and Cynopteri (30 cases, 5.77%). The seasonal summer distribution was evident in all years, with the peak months being December to April. Several days of heavy rainfall were followed by an increase in laboratory-confirmed cases of leptospirosis.

Conclusion: This neglected disease remains as a public health problem in the country. An understanding of the relationship between leptospirosis incidence and heavy rains is indispensable for implementing appropriate preventive measures.

### 18.106 Tuberculosis in Austria: An Analysis of the Past 10 Years, 1997-2006

H.W. KUO, F. Allerberger, A. Indra, S. Pfeiffer, S. Daniela. Austrian Agency for Health and Food Safety, Vienna, Austria

Background: The objectives of this surveillance data analysis were to describe the burden of tuberculosis (TB), a mandatory notifiable disease, in Austria from 1997 to 2006 and to elucidate the relevance of immigrants for TB incidence.

Methods: The annual incidence of TB and multi-drug-resistant (MDR)-TB was calculated from the cases recorded for the years 1997-2006 at the national TB-Reference Center. Changing of TB burden over time was measured by simple linear regression or log-linear regression model. The incidence estimates were compared between immigrants to Austria and Austrian citizens by chi-square test.

Results: A total of 11,496 TB-cases including 7,009 culture-positives were recorded. During the observation period, the TB-incidence decreased significantly by 0.77/100,000 inhabitants per year (total incidence percent

age change: 41.3%). The incidence was significantly higher in immigrants: in 2000 with a 3.99 times higher incidence and in 2005 with a 6.96 times higher incidence. The TB-incidence in Austrian citizens decreased linearly by 0.91/100,000/year: their highest MDR-TB-incidences recorded were 0.03/100,000 in the years 1997, 1998, 2001 and 2003. The MDR-TB-incidence in immigrants was multiple times higher compared to Austrians (ratio range = 10.66-infinity) in all 10 years. Between 1997 and 2002 the MDR-TB-incidence in the immigrants peaked with 0.72/100,000 in 1999 and between 2002 and 2006 with 2.45/100,000 in 2004.

Conclusion: As in other European countries, the TB-incidence in Austria decreased during the past 10 years. The peaks of TB-incidence in 2000 and 2005 and of MDR-TB-incidence in 1999 and 2004 in immigrants reflect the population mobility across Europe. The highest number of immigrants to Austria between 1997 and 2006 were recorded in 1999 and in 2004, mirroring the war in Kosovo and in Chechnya-both high TBendemic areas. The influx of TB from abroad did not influence the burden of tuberculosis in Austrian citizens.

### 18.107 Invasive Pneumococcal Disease in Gran Canary Island 2004-2007

A.J. García Rojas<sup>1</sup>, P. García Castellano<sup>1</sup>, A. Bordes Benitez<sup>2</sup>, F. Troncoso Mendoza<sup>2</sup>, A. Fenoll Comes<sup>3</sup>, P. Matute Cruz<sup>1</sup>, D. Nuñez Gallo1. 1Public Health Service, Las Palmas de Gran Canaria, Spain, <sup>2</sup>Microbiology Service. Gran Canary Hospital, Las Palmas de Gran Canaria, Spain, Microbiology. Center of Majadahonda, Madrid, Spain, Public Health Service, Santa Cruz de Tenerife, Spain

We present the epidemiological decriptive characteristics and the temporary distribution in Invasive Pneumococcal Disease in general population and the mostly prevalents serotypes in children lower 5 years in Gran Canary Island, during the temporal period 2004-2007

Prospective study of the Invasive Disease confirmated and notified cases to the Gran Canary Microbiological Informative Sistem, between 2004-2007, and hospitalized in Gran Canary with laboratory diagnosis as S. Pneumococcal in blood, Cerebral Spinal Fluid, or in other usually sterile places and acute disease with a compatible clinic of Pneumococcal Disease

In the studied period, there were notified 196 cases, 40 (20%) in 2004. 63 (32%) in 2005, 43 (22%) in 2006 and 50 (26%) in 2007. Also the 29% of sickness were below 5 years old, the 39% between 5 and 65 years and the 32% older 65 years. The total number of male afected was 125 and of female was 71. Relations Male/Female: 1,76 In children lower 5 years only rested 9 processes without tipifying. Of the 48 tipified,62,5% were not prepared by the 7-valent conjugate vaccine. The serotype most identified was the 19A (27%), and the 14 (12%)

We must remark the presence of this disease in older people. The appearance of the isolated vaccinal serotypes could be conditioned by the mentioned use of the vaccine in a high percentage of lower 5 years old population. In respect to years 2004-2005, in 2006 was detected a higher numbers of serotypes not predictables for the vaccine (69%), over the predictables (31%). In 2007, 87,5% not predictables and 12,5% predictables. We have to increase the knowledge of this disease with a continued alertness, remarking the need of to confirmate the diagnosis and have isolations to identify the causer serotypes

### 18.108 Emergence of Rift Valley Fever in Mayotte, a French Indian Ocean Territory

D. Sissoko<sup>1</sup>, C. Giry<sup>2</sup>, P. Gabrié<sup>3</sup>, A. Tarantola<sup>4</sup>, F. Pettinelli<sup>5</sup>, L. Collet<sup>5</sup>. E. d'Ortenzio¹, P. Renault¹, V. Pierre¹. ¹Institut de veille sanitaire, Cellule interrégionale d'épidémiologie (Cire) Réunion-Mayotte, Drass de la Réunion, Saint-Denis de la Réunion, France, \*Laboratoire de Biologie, Centre Hospitalier, Mamoudzou, Mayotte, France, 3Cellule de veille épidémiologique, Centre Hospitalier, Mamoudzou, Mayotte, France, Institut de veille Sanitaire, Département International et Tropical, Saint-Maurice, France, Laboratoire de Biologie, Centre Hospitalier, Mamoudzou, Mayotte, France

Background: Mayotte is a French territory located in the south-west Indian Ocean. Epidemic intelligence conducted at the Institut de Veille

### Parallel Session Abstracts

20090084 Session: 14.3

Track: Tuberculosis

### Tuberculosis in children in London, 1999-2007

Aileen Kitching (1, 2), J. Carless (2), H. Maguire (2)

- European Programme for Intervention Epidemiology Training (EPIET), European Centre for Disease Prevention and Control (ECDC), Stockholm, Sweden
- 2. Health Protection Agency, Landon Region Epidemiology Unit, UK

### BACKGROUND

Tuberculosis (TB) is a particular public health concern in London, with a 1987-2007 incidence increase from 21 to 43/100,000 population. Cases of TB in young children represent recently acquired infection and serve as a surrogate marker for ongoing community transmission. The objective of the study is to describe childhood TB patterns, in order to orient TB-control activities.

### METHODS

Data on childhood (0-16 years) TB cases from the Enhanced TB surveillance system, London, 1999-2007 were analysed. We calculated incidence over time and described the childhood TB cases by country of origin and ethnicity.

### RESULTS

In 2007, almost 8% of new London TB cases were in children (n=251) and 2% in under-fives, a similar proportion since 1999. Childhood TB incidence remained stable from 1999-2001 at 11-12/100,000 population, increased to 14.6/100,000 in 2002, and remained relatively static at 16-17/100,000 from 2004-2007. Incidence in under-fives fluctuated between 9-14/100,000 since 2002 (relatively small numbers). In 2007, most (53%) London childhood TB cases were UK-born. However, 94% were from an ethnic minority - 51% Black African and 22% Indian subcontinent ethnicity. Of the non-UK born, 32% were in the UK for more than 5 years.

### CONCLUSION

In London, TB incidence in children overall is not decreasing. This is a cause for concern as it is a marker of ongoing transmission rather than reactivation of old disease. One quarter of these cases are in under-fives, with implications for household transmission. Most of these children are from ethnic minority groups. As many of these children are UK-born, or recent (less than five years) arrivals to the UK, early intervention is possible. This information should be used to target interventions and ensure no missed opportunities for prevention.

### PRESENTER: KITCHING

20090044 Session: 14.4

Track: Tuberculosis

### Does increasing immigration affect the epidemiology of TB in Austria, 1997-2006?

Hung-Wei Kuo (1, 2, 3), D. Schmid (2), S. Pfeiffer (2), A. Indra (2), F. Allerberger (2)

- European Programme for Intervention Epidemiology Training (EPIET), European Centre for Disease Prevention and Control (ECDC), Stockholm, Sweden
- 2. Austrian Agency for Health and Food Safety (AGES), Vienna, Austria
- 3. Centers for Disease Control, R.O.C. (Taiwan)

### BACKGROUND

As seen in other high-income European countries, the incidence of tuberculosis (TB) in Austria decreased over the past decade. The proportion of TB-cases in foreign-born populations settled in Europe increased during this period. Also in Austria immigration activities increased. The aim of the present study was to study the influence of immigration on the TB-burden in Austria.

### METHODS

Descriptive and correlational analyses were performed using the case data from the National TB-database and aggregated data from the Austrian population statistics and immigration statistics. Foreign-born Austrian residents (non-Austrians) were defined as long-stay non-Austrians when having lived in Austria for at least 5 years, otherwise defined as short-stay non-Austrians.

### RESULTS

There was no correlation found in the annual TB-incidences between Austrians and non-Austrians (r=0.01; p=0.98). The age distribution differed significantly between these two groups: non-Austrian TB-cases were younger. The ageadjusted province-specific incidence rates between these groups did not correlate (r=0.43, p=0.25). There was no positive correlation found between increasing immigration and TB in Austrians. A positive correlation was found between immigration from high TB incidence countries and TB in non-Austrians (r=0.87, p<0.01). A total of 94% of the TB cases in non-Austrians were diagnosed in the first 5 years after arrival. In particular, the number of TB cases in shortstay non-Austrians positively correlated with the increasing immigration from high TB-incidence countries (r=0.86, p<0.01).

### CONCLUSION

The only observed effect of immigration was that increasing immigration from high TB-incidence countries is related to increasing TB cases in short-stay non-Austrians. This indicates reactivation of remote infection. Serological studies in immigrants from high TB-incidence countries are recommended to provide evidence for decisions making on further control measures.

### PRESENTER: KUO



### Did increasing immigration affect the epidemiology of TB in Austria, 1997-2006 ?

### Hung-Wei Kuo MPH

European Programme for Intervention Epidemiology Training (EPIET), European Centre for Disease Prevention and Control (ECDC), Sweden Austrian Agency for Health and Food Safety (AGES), Vienna

ESCAIDE2009



# BACKGROUND

### 



Western Europe: downward trends in morbidity and mortality for the past decade

1997: 14/100,000

2006: 10/100,000

Migration from high to low TB-incidence countries in Europe of epidemiologic importance

# 



- Statutorily notified in all 9 provinces of Austria since 1968
- Case definitions for TB surveillance: according to EuroTB
- Notification and laboratory data consolidated in the National TB data base:
- Origin (citizenship)
- Year of immigration
- others according to required data set of EuroTB 2006

## Thigh of the North 1997 2006



Incidence decreased

-1997; 18.6/100,000

-2006: 10.9/100,000

No. of immigrants increased

-1997:56,895

-2004:108,947

-2006:85,384

73% of immigrants from high TB-incidence countries

# Aim and Objectives



### Aim

to study the influence of immigration on TB burden in Austria from 1997-2006

### Objectives

to analyse the correlation between

- TB occurrence in Austrians and non-Austrians
- immigration and TB occurrence in Austrians
- immigration and TB occurrence in non-Austrians



### METHODS

### Data sources

- National TB-database (individual data)
- Population statistics (aggregated data)
- Immigration statistics (aggregated data)

### Analyses

- Descriptive analyses with case data
- Correlational analyses with immigration and case data

### Definitions used

- Non-Austrians: citizenship other than Austrian
- Long-stay non-Austrians: in Austria for at least 5 years
- High TB-incidence country: ≥ 20/100,000



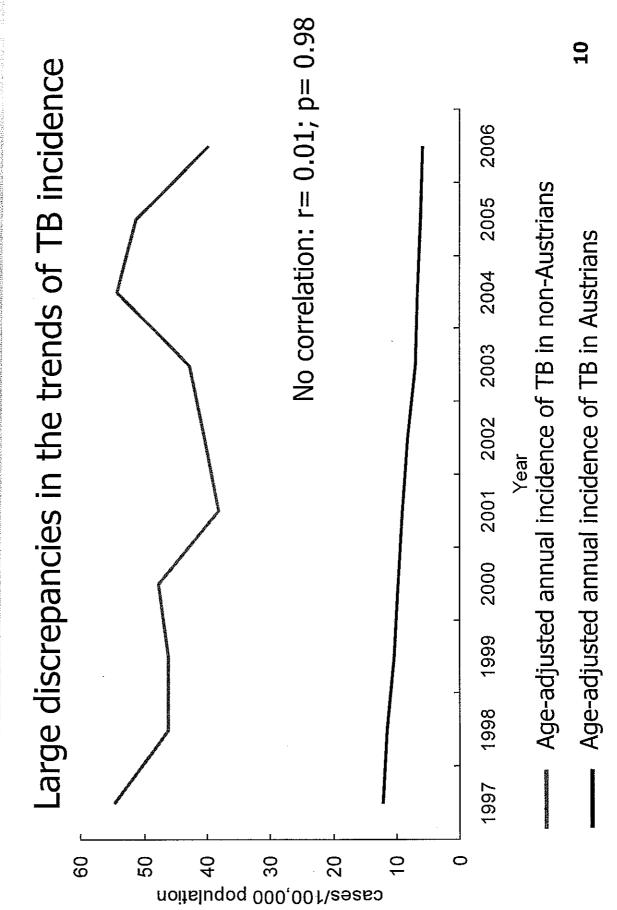


## RESULTS (I)

TB in Austrians and non-Austrians, 1997-2006

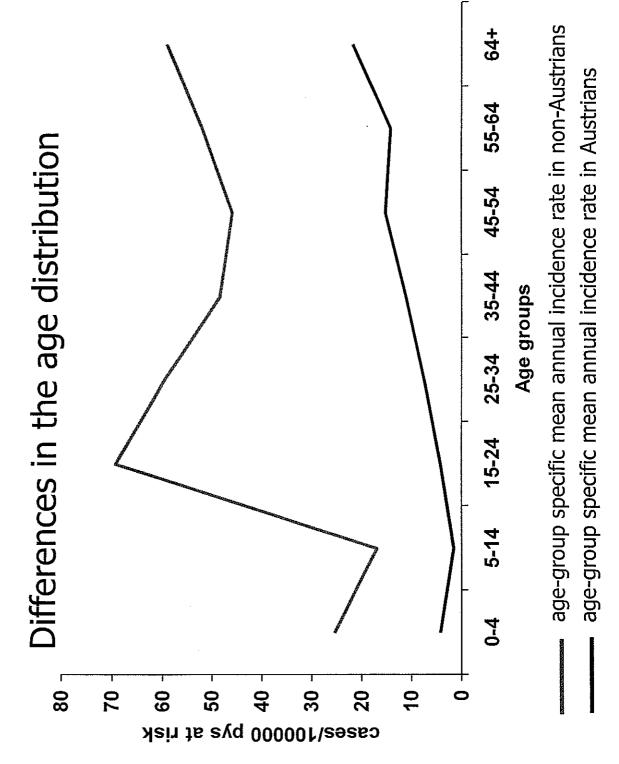
### Annual 18 incidences. Austrans and non-Austrians





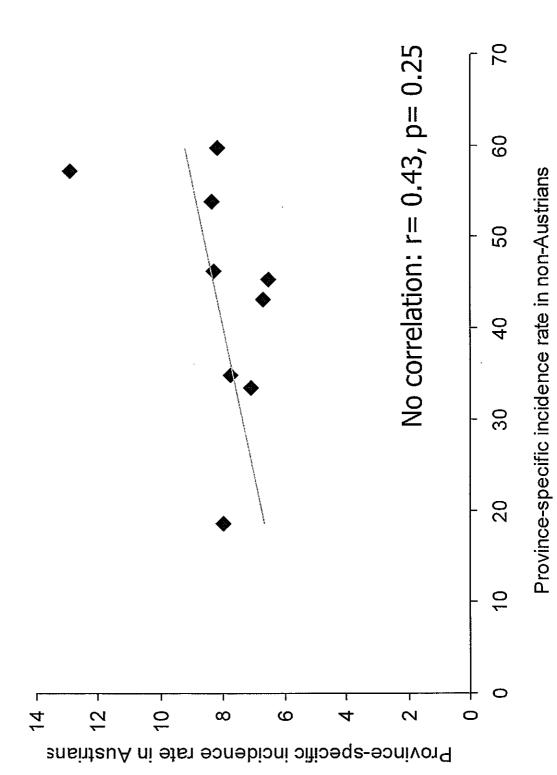
### Age-specific incidence rates: Austrians and non-Austrians





### Provinca-spacific incidence rates Austrans and non-Austrians





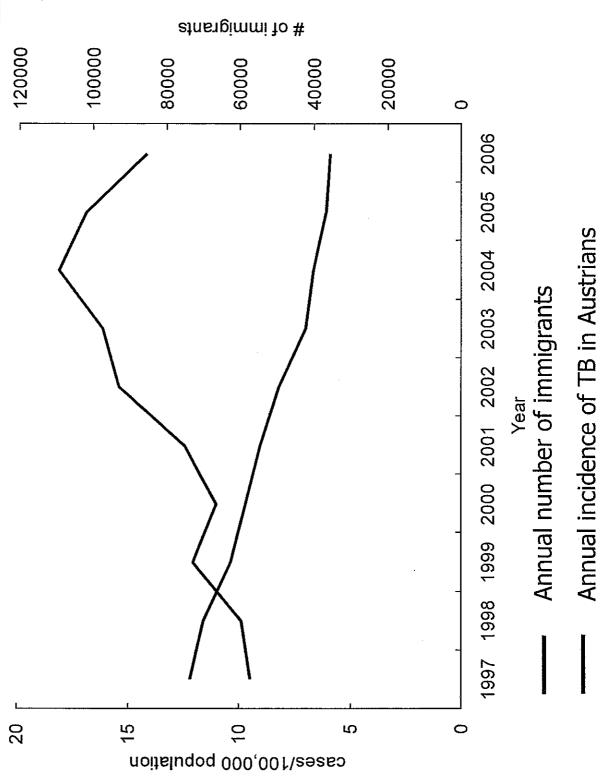


# RESULTS (II)

Immigration and TB in Austrians and non-Austrians, 1997-2006

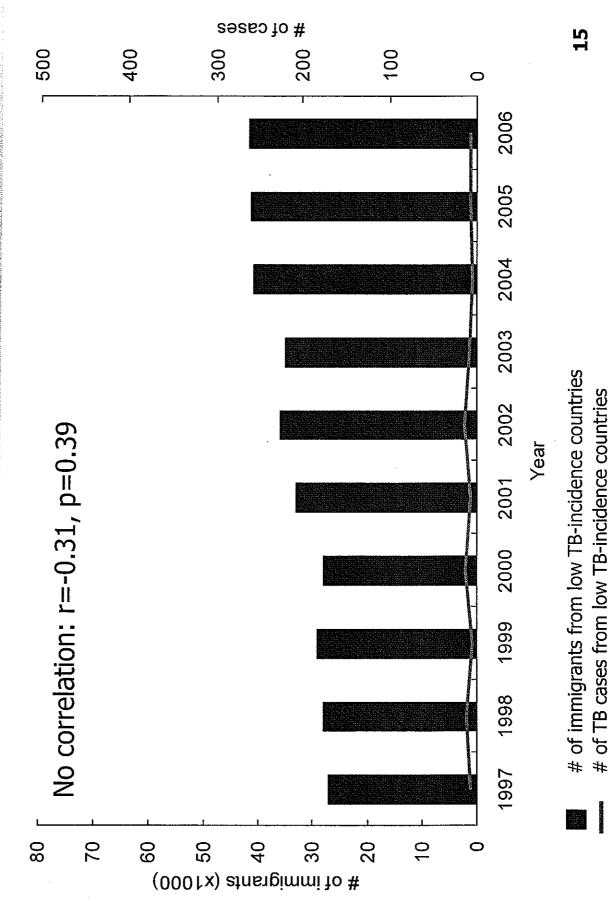
# 





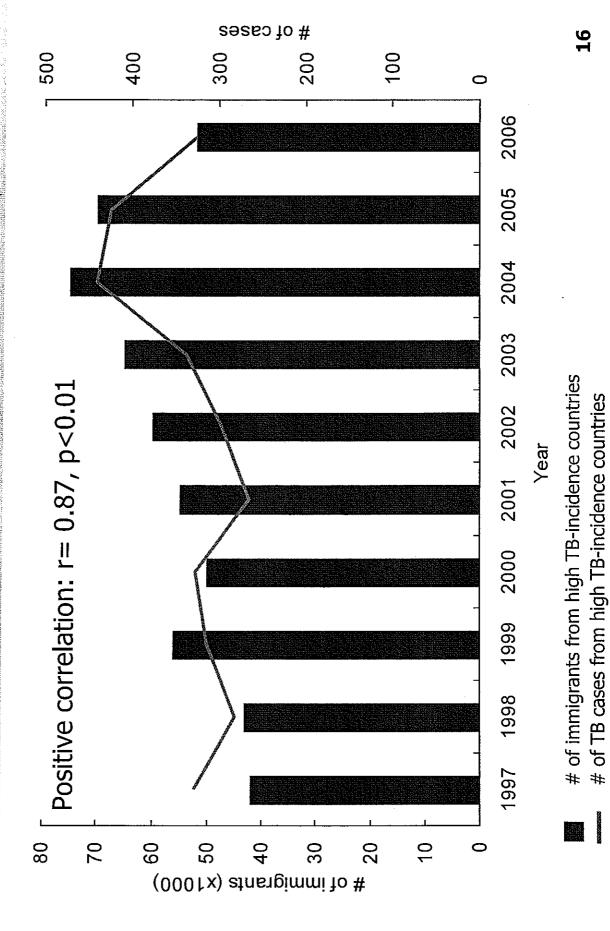
## Immigration and TB in non-Austrians from low TB-incidence countries





### Immigration and TB in non-Austrians from high TB-indidence countries





# non-Austrians by langth of residence

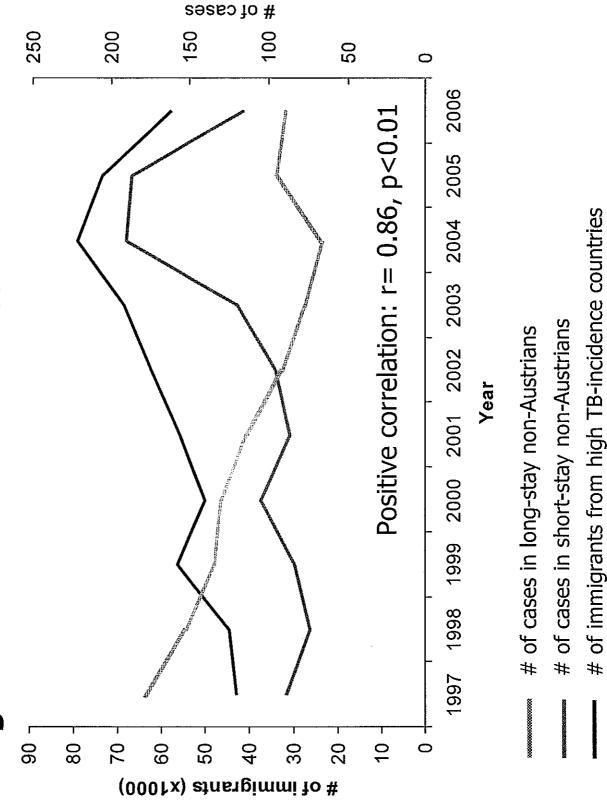


Austria did so within the first 5 years after arrival 94% of immigrants who became a case of TB in 10 11-20 > 20 တ ന 9 50 <u>4</u>0 30 20 70 20 0

% of cases

Years elapsed since entering Austria

Immigration and TB in short stay non-Austrians from high TB-incidence countries



### 



### No correlation

- between TB in non-Austrians and in Austrians
- between increasing immigration and TB in Austrians

Correlation between immigration and TB in non-Austrians

 No. of TB cases in short-stay non-Austrians increased with increasing immigration from high TB-incidence countries

# 



- No indications that increasing immigration, 1997-2006, affected the TB burden among Austrians
- 1997-2006, according to the TB burden in short-stay non-Trend of immigration from high TB-incidence countries, Austrians
- Indication for reactivation of remote infection in their countries of origin
- Serological studies in immigrants from high TB-incidence countries are recommended to provide evidence for further public health measures

## Acknowledgment



- The colleagues of AGES:
- Department for Infectious Disease Epidemiology
- National TB-Reference Laboratory

### original article

Wien Klin Wochenschr (2009) 121: 120–124 DOI 10.1007/s00508-008-1131-1 Printed in Austria © Springer-Verlag 2009

### Wiener klinische Wochenschrift

The Middle European Journal of Medicine

## A non-foodborne norovirus outbreak among school children during a skiing holiday, Austria, 2007

Hung-Wei Kuo<sup>1,2</sup>, Daniela Schmid<sup>1</sup>, Karin Schwarz<sup>1</sup>, Anna-Margaretha Pichler<sup>1</sup>, Heidelinde Klein<sup>3</sup>, Christoph König<sup>3</sup>, Alfred de Martin<sup>3</sup>, Franz Allerberger<sup>1</sup>

Ein nicht-lebensmittelbedingter Norovirus-Ausbruch unter Teilnehmern eines Schulschikurses, Österreich, 2007

Zusammenfassung. Hintergrund: Die Bedeutung von Noroviren (NV) als maßgebliche Ursache lebensmittelbedingter Krankheitsausbrüche wird zunehmend anerkannt. Wir berichten über einen Norovirus-Ausbruch mit 176 Erkrankungsfällen, der im Dezember 2007 Schüler und Lehrer von 4 Schulen betraf, die ihre Schulschiwochen in einem Schülerheim in Salzburg absolvierten.

Methodik: Durch die betroffenen Schulen wurden Fragebögen verteilt und Daten zu Erkrankungsstatus, Erkrankungsbeginn, Krankheitsdauer und Hospitalisation erhoben. Im Rahmen einer Kohortenstudie wurden die Schikursteilnehmer bezüglich Exposition gegenüber die im Rahmen des Schulschikurses angebotenen Lebensmittel befragt.

Ergebnisse: Von den 284 Teilnehmern erfüllten 176 die Definition eines Ausbruchsfalls (Befallsrate: 61,9%). Insgesamt erklärten sich 264 Teilnehmer des Schulschikurses bereit an der Kohortenstudie teilzunehmen (Rückantwortrate: 93%). Die tagesbezogene lebensmittelspezifische Analyse zeigte, dass keines der zwischen Dezember 8 und 12 im Schülerheim angebotenen Lebensmittel in einem signifikanten Zusammenhang mit den Erkrankungen stand. Die tagesbezogene Analyse belegte Montag (RR: 9,04; 95%CI: 6,02–13,6; p<0,001) und Dienstag (RR: 3,37; 95%CI: 2,56–4,43; p<0,001) als die beiden Tage mit dem höchsten Risiko einer Exposition gegenüber Norovirus.

Diskussion: Die epidemiologische Abklärung zeigte, dass eine lebensmittelbedingte Genese für den beschriebenen Norovirusausbruch auszuschließen ist. Aerogene Norovirus-Übertragung ausgehend vom Indexpatienten (wiederholtes Erbrechen im Bus und im Hotelfoyer) ist als Ausgangspunkt anzusehen. In den letzten Jahren haben sich Noroviren europaweit als die

wichtigsten Erreger von Gastroenteritis-Ausbrüchen bei Touristen etabliert. Tourismus gilt in Österreich als eine Schlüsselindustrie. Ein zeitgerechtes Hinzuziehen der Gesundheitsbehörden sollte bei jedem Norovirus-Ausbruch erfolgen, unabhängig ob lebensmittelbedingt oder nicht

Summary. Norovirus is increasingly recognized as a leading cause of outbreaks of foodborne disease. We report on an outbreak in Austria that reached a total of 176 cases, affecting pupils and teachers from four schools on a skiing holiday in a youth hostel in the province of Salzburg in December 2007. A questionnaire was sent to the four schools in order to obtain data from persons attending the school trip on disease status, clinical onset, duration of illness and hospitalization. A cohort study was undertaken to identify the sources of infection. The school trip attendees were interviewed by questionnaire or face-to-face on their exposure to food items from the menu provided by the hostel owner. Of the 284 school holiday-makers, 176 fitted the definition of an outbreak case (attack rate 61.9%). A total of 264 persons on the ski holiday participated in the cohort study (response rate 93%). The day-by-day food-specific analyses did not find any food items served on any of five days (December 8-12) of the holiday to be associated with infection risk. The day-specific risk analyses revealed Monday December 10 (RR: 9.04; 95% CI: 6.02-13.6; P<0.001) and Tuesday December 11 (RR: 3.37; 95% CI: 2.56-4.43; P<0.001) as the two most risky days for having being exposed to norovirus. According to the epidemiological investigation, airborne transmission of norovirus originating from the first vomiting case most probably initiated this outbreak; foodborne genesis was excluded. During recent years, norovirus has become increasingly established as the most important causative agent of epidemic gastroenteritis in holiday-makers all over Europe. Tourism is one of the primary industries in Austria. Timely involvement of the relevant public health authorities is essential in any outbreak of norovirus gastroenteritis, irrespective of its genesis.

Key words: Norovirus, outbreak, airborne, cohort study.

<sup>&</sup>lt;sup>1</sup>Austrian Agency for Health and Food Safety, Vienna, Austria

<sup>&</sup>lt;sup>2</sup>European Programme for Intervention Epidemiology Training (EPIET), Stockholm, Schweden

<sup>&</sup>lt;sup>3</sup>Public Health Authorities, Salzburg and St. Pölten, Austria

Correspondence: Univ.-Prof. Dr. Franz Allerberger, Österreichische Agentur für Gesundheit und Ernährungssicherheit (AGES), Spargelfeldstraße 191, 1220 Wien, Austria, E-mail: Franz. Allerberger@ages.at

### Introduction

Infection with norovirus is exceedingly widespread and common [1]. In temperate climates transmission of the virus occurs year round with a higher incidence of disease in the winter months. The average incubation period is 24-48 h, although ranges of 15-60 h have been observed. Norovirus was first recognized in association with a point-source outbreak of gastroenteritis, and this pattern has remained the most common for outbreaks in which norovirus is implicated as etiologic agent [2-4]. The virus is increasingly being recognized as a leading cause of foodborne disease in Europe and the USA [5-8], with the majority of foodborne outbreaks in which a virus is identified being caused by norovirus [9]. Most of our knowledge on norovirus transmission is gained from investigation of naturally occurring outbreaks. We describe a norovirus outbreak among pupils on a skiing holiday for which a retrospective cohort study was able to exclude food items as the source of the outbreak.

### Background

On Saturday, December 8, 2007, 44 pupils from a school in Vienna arrived at a youth hostel (4-bed rooms) in the Austrian province of Salzburg where they planned to spend a week's skiing holiday. One of the pupils had vomited several times while traveling in the bus and also immediately on arrival in the lobby of the hotel. Even though this child with gastroenteritis was given a single room to prevent further exposure of the other pupils, nine other children among the 44 pupils fell ill within the following 50 hours. The following day, December 9, an additional 240 children from three other schools (1 in Vienna, 2 in the province of Lower Austria) arrived at the same hostel. In the following five days (December 10-14) a further 159 pupils fell ill with gastroenteritis. One school group departed on Thursday, December 13 and the pupils from the other three schools on December 14. On Saturday, December 15, the Austrian Agency for Health and Food Safety (AGES) was mandated to investigate this cluster of cases.

The dominating symptom of vomiting, the rapid spread of disease, the high attack rate and the short duration of disease (<48 h) led to the suspicion of norovirus as the causative pathogen. The weekend of December 15-16, after departure of the pupils and teachers of the four schools, the hostel was cleaned and chemically disinfected. Seven more pupils fell sick with gastroenteritis on December 15, 16 and 17 (Saturday, Sunday, Monday) after returning home from the holiday. On December 19 the suspicion of norovirus as outbreak agent was confirmed when stool samples, obtained from one cluster case at the request of AGES, tested positive for norovirus by RT-PCR.

### Descriptive epidemiological investigation

### Definition of an outbreak case

An outbreak case was defined as a holiday-maker who (a) was a pupil or teacher from one of the four schools (2 in Vienna, 2 in Lower Austria), (b) had accommodation in the youth hostel between December 8 and December 14, and (c) fell ill after December 7 and not later than December 21 with symptoms of diarrhea or vomiting.

### Active case finding

A questionnaire was sent to the four schools and assigned to all participants of the skiing holiday (December 8 - December 13/14). Data were obtained on disease status, clinical onset, symptoms (nausea, fever, vomiting, diarrhea, abdominal cramps), duration of illness and hospitalization.

### Results of the descriptive epidemiological investigation

Of a total of 284 holiday-makers who stayed in hostel X in the province Salzburg during the week December 8-14, 176 fitted the definition of an outbreak case, including one case of laboratory-confirmed norovirus infection (attack rate 61.9%). Among the cases, 144 (81.8%) suffered from nausea, 152 (86.4%) from vomiting, 109 (61.9%) from abdominal cramps, 49 (27.8%) from fever, 90 (51.1%) from diarrhea and 66 (37.5%) suffered from both diarrhea and vomiting. The median duration of illness was 2 days (range 1-4). Four cases were hospitalized. The outbreak lasted from December 8 to December 17 and peaked on December 12 (Fig. 1).

The only common link identified among the cases was that they stayed in the same hostel during the week December 8-14 and consumed food prepared and served by the family who own the hostel. Questioning of hostel staff found that neither staff nor their respective household members had suffered from gastroenteritis between December 8 and December 12; that is, during the period the meals were prepared.

All cases in the school class who were first to arrive at the hostel had been exposed to the vomiting primary case on the bus or in the hostel lobby. Cases from the three schools arriving at the hostel one and two days later were exposed to pupils who were already ill and to the norovirus-contaminated environment of the hostel. There were two small dining rooms and one big restaurant in the hostel where all guests met for breakfast, lunch and dinner. Apart from the primary case of the outbreak, no other cases were isolated. During the week of the outbreak, exposed surfaces and fomites in the hostel were cleaned and disinfected using alcohol-based disinfectants.

Foodborne genesis of the outbreak was considered unlikely.

### Cohort study

### Cohort of interest

A total of 284 persons (266 pupils, 18 teachers) from four schools who stayed in the youth hostel from December 8 to December 13/14 for the one-week skiing holiday were considered as the study cohort (the at-risk group).

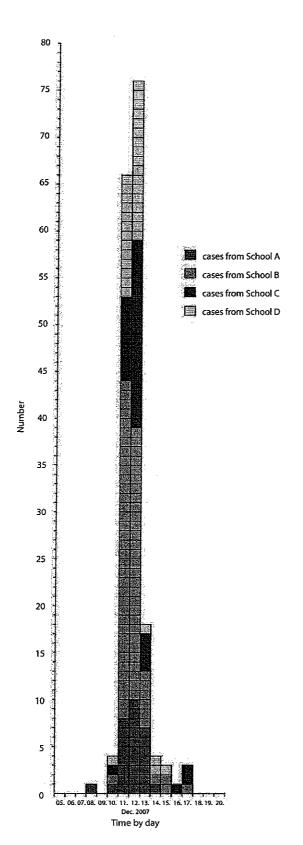


Fig. 1. Norovirus cases by date of clinical onset (n = 176) and by schools affected

### Data collection

The cohort members were interviewed by questionnaire or face-to-face on their exposure to food items from the menu provided by the hostel owner and served between December 8 and December 12.

### Study population

Of 284 persons, 265 (including the pupil who fell ill before arriving at the youth hostel) responded to the questionnaires (response rate 93%); a total of 264 persons (247 pupils, 17 teachers) free from gastroenteritis before arrival at the hostel were therefore included in the cohort analyses.

### Definition of the exposure

The hypothesis to be tested was that food items prepared in the hostel kitchen and consumed by the school groups between December 8 (Saturday) and December 12 (Wednesday) were the sources of infection. The following food items were tested as potentially risky exposure items: soup, turkey with rice, potato wedges with vegetables, broccoli and dessert buffet served Saturday evening; pasta with salad from the buffet and fruit served Sunday midday; soup, Wiener schnitzel, pudding or pizza and cheese served Sunday evening; potato soup, cut-up and sugared pancake with raisins and apple sauce or pasta with tomato sauce served Monday midday; vegetable soup, pasta Bolognese and ice cream served Monday evening; zucchini cream soup, fried meat loaf with mashed potatoes, vegetable burger and fried turkey meat loaf served Tuesday midday; soup, chicken filet with rice and potato burger served Tuesday evening; noodles with sausages and salad, stewed fruit, cheese dumplings served Wednesday midday; noodle soup, steak, potato wedges with salad and yogurt and vegetable strudel, turkey served Wednesday evening.

The five days December 8–12 (Saturday–Wednesday) were considered as the relevant days on which risky exposure to food might have occurred. For the day-by-day analyses of the food-specific attack rates and respective risk ratios, the persons who had eaten on the particular day under study were included in the day-specific cohort. The number of cases and the number of persons in the denominator therefore varied for the day-by-day food-specific cohort analyses [8, 9].

### Data analyses

Day-specific attack rates (AR %) were computed disregarding the individual food items offered on the specific day. For the day-specific risk ratios, the attack rate for the specific day was compared with the pooled attack rate of food exposure having occurred on the other four days. Taking into account that the etiological agent was norovirus and knowing its short incubation period (24–48 h), persons who had fallen ill on Sunday or Monday were included in the numerator when considering food exposure on Saturday as risky exposure. The de-

nominator was given by the number of persons who experienced food exposure on that particular day. The pooled food-specific attack rate was computed from the number of cases attributed to the remaining four days divided by the total number of food exposures experienced by the cohort members on those four days. The day-specific ratios for Tuesday, Wednesday, Thursday and Friday were similarly computed.

Food-specific attack rates and food-specific risk ratios (95% CI) were calculated day by day for all items offered between Saturday, December 8 and Wednesday, December 12. Taking into account the average incubation period of norovirus (24-48 h), food items consumed 24-48 h before onset of symptoms were considered possible sources of infection. Therefore, when computing food-specific attack rates for food items offered on Saturday, December 8, only cases who became ill on December 9 or 10 (Sunday, Monday) were included in the numerator; cases who fell ill on or after December 11 (Tuesday) were included in the denominator, as they were considered as non-ill persons in the food-specific analyses for Saturday. For food eaten on December 9 (Sunday), those who became ill on December 10 or 11 (Monday, Tuesday) were included in the numerator; those who became ill on or after December 12 were included in the denominator. For food eaten on December 10 (Monday), those who became ill on December 11 or 12 (Tuesday, Wednesday) were in the numerator. For food eaten on December 11 (Tuesday), those who became ill on December 12 or 13 (Wednesday, Thursday) were in the numerator. For food eaten on December 12 (Wednesday), those who became ill on December 13 or 14 (Thursday, Friday) were in the numerator. Persons were removed from the cohort after they were reported ill, since they were no longer at risk. Persons who fell ill after December 14 were considered as secondary cases and included in the denominator only.

We used the software package EpiInfo 3.4.1 for Windows for data entry and SAS 9.1 for Windows for analyses.

### Results

Of the 264 subjects of the cohort study, 113 were female (42.8%). A total of 168 cohort members (159 pupils, 9 teachers) fitted the definition of positive disease status possibly related to food exposure on days December 8-12; 72 of the ill persons were female (43.1%). Among

the 168 patients, 140 (83.3%) had nausea, 146 (86.9%) had vomiting, 104 (61.9%) had abdominal cramps, 48 (28.6%) had fever, 85 (50.6%) had diarrhea and 63 (37.5%) suffered from both diarrhea and vomiting.

The day-by-day food-specific analyses did not find any of the food items served on any of the five days December 8-12 to be associated with infection risk. The analyses of the day-specific risk ratios revealed that the risk of being infected was highest on Monday December 10 with a relative risk of 9.04 (95% CI 6.02-13.6; P<0.001), followed by Tuesday December 11 with a relative risk of 3.37 (95% CI 2.56-4.43; P<0.001) (Table 1).

### Discussion

We describe a non-foodborne community outbreak of norovirus in Austria that reached a total of 176 cases. The outbreak took place in a youth hostel in the province of Salzburg and affected pupils and teachers from four schools: two from Vienna and two from different public health districts of the province of Lower Austria. The local public health officer learned about the outbreak only on December 17, the day on which the last three outbreak cases occurred. She was contacted by the Agency for Health and Food Safety (AGES), which was mandated for investigation of the outbreak by the provincial public health authorities of Lower Austria. According to Austrian law, only norovirus infections that are foodborne are subject to statutory reporting, thus the clinicians who treated the hospitalized casepatients did not provide data on clinical and microbiological investigations to the local public health officer because they were convinced that the outbreak of "enteric winter-flu" had a non-foodborne genesis. All norovirus outbreaks, whether foodborne or not, should be notifiable to the public health authorities and should be investigated in order to provide information on possible hygienic shortcomings and to allow for targeted interventions such as the implementation of adequate preventive measures.

According to the descriptive epidemiological investigation of the outbreak, airborne transmission of norovirus from the first vomiting case most probably initiated the outbreak. Because of the close contact among pupils and teachers, the outbreak continued for the following nine days through direct person-to-person transmission and also through exposure to the contaminated environment, the infectious exposures most probably

Days of interest	AR % exposed	AR % unexposed	RR	95% CI	P
Saturday (December 8)	2.6 (1/39)	20.0 (167/836)	0.15	(0.02–1.05)	0.03
Sunday (December 9)	26.5 (70/264)	16.0 (98/611)	1.52	(115-2:00)	0.005
Monday (December 10)	54.6 (142/260)	4.1 (25/615)	9.04	(6.02-13.6)	<0.001
Tuesday (December 11)	48.5 (94/194)	10.7 (73/681):	-3.37	(2.56-4,43)	<0.001
Wednesday (December 12)	18.6 (22/118)	19.3 (146/757)	0.97	(0.64-1.47)	n.s.

occurring in the two small dining rooms or in the various 4-bed rooms. The two most risky days for being exposed to the virus were Monday 10 and Tuesday 11.

The analytical epidemiological investigation made it possible to exclude a foodborne genesis. Non-foodborne norovirus outbreaks are often characterized by a variety of modes of transmission: person to person via the fecal-oral route, via aerosolized vomit, or from the contaminated environment via physical contact or aerosols [9].

In the outbreak described here, the outpatient physicians and the hostel staff cooperated fully in the investigation and the application of the recommended control measures. Nevertheless, the immediate involvement of the public health officer could have facilitated a more rapid response to the outbreak through representative sample collection, case series investigation and prompt implementation of appropriate norovirus control measures such as superficial cleaning and disinfection of the affected environment and hand hygiene using appropriate disinfectants. However, following the departure of all guests, comprehensive cleaning of the environment on December 15 and 16, as well as chemical disinfection (using a peroxide-based disinfectant) and heat disinfection of items ineligible for chemical disinfection, prevented further outbreaks during the winter season in this hostel.

During recent years, norovirus has become increasingly established as the most important causative agent of epidemic gastroenteritis in holiday-makers all over Europe [10]. Although no such outbreak has previously been described in Austria, norovirus is regularly implicated in large outbreaks of vomiting and diarrhea in Austrian hotels. Tourism is one of the primary industries in Austria and prompt action in response to gastroenteritis outbreaks is highly necessary. Only the prompt isolation of patients with norovirus gastroenteritis and appropriate disinfection of contaminated hands and the contaminated environment with disinfectants appropriate for norovirus can enable rapid control of community norovirus outbreaks. Prompt involvement of the relevant public health authorities is therefore essential in any outbreak of norovirus gastroenteritis, regardless of its genesis. The authors conclude that, in addition to the existing national guidelines published in 2006 on the management of norovirus outbreaks in healthcare settings [11], common guidelines for the management of norovirus outbreaks in the hotel and tourism industry are also needed in Austria.

This non-foodborne norovirus outbreak also highlights the importance of inter-district and inter-provincial collaboration in properly investigating outbreaks involving a tourist destination, where tourists from many different areas can usually be expected. In Austria inter-district and inter-provincial collaboration is regulated only for foodborne outbreaks at present [12].

### References

- Treanor JJ, Dolin R (2005) Noroviruses and other caliciviruses. In: Mandell GL Bennett, JE Dolin R (eds) Principles and practice of infectious diseases, 6th edn. Elsevier Churchill Livingstone, Philadelphia, pp 2194–2201
- Wyatt RG, Dolin R, Blacklow NR, DuPont HL, Buscho RF, Thornhill TS, Kapikian AZ, Chanock RM (1974) Comparison of three agents of acute infectious gastroenteritis by virus challenge in volunteers. J Infect Dis 129: 709-714
- Graham DY, Jiang X, Tanaka T, Opekun AR, Madore HP, Estes MK (1994) Norwalk virus infection of volunteers: new insights based on improved assays. J Infect Dis 170: 34-43
- Gray GW, Anderson JL, Keswick BH, Johnson PC, Du Pont HL, Sine SE, Bartlett AV (1987) Norwalk virus antigen and antibody response in an adult volunteer study. J Clin Microbiol 25: 2001–2003
- Lynch M, Painter J, Woodruff R, Braden C (2006) Surveillance for foodborne-disease outbreaks-United States, 1998–2002. MMWR Surveill Summ 55: 1–42
- Marks PJ, Vipond IB, Regan FM, Wedgwood K, Fey RE, Caul EO (2003) A school outbreak of Norwalk-like virus: evidence for airborne transmission. Epidemiol Infect 131: 727-736
- Miller I, Gunson R, Carman WF (2002) Norwalk-like virus by light cycle PCR. J Clin Virol 25: 231–232
- Schmid D, Stüger HP, Lederer I, Pichler A-M, Kainz-Arnfelser G, Allerberger F (2007) A foodborne norovirus outbreak due to manually prepared salad, Austria 2006. Infection 35: 232–239
- Iturriza-Gomara M, Gallimore CI, Gray J (2007) Gastroenteric viruses. In: Simjee S (ed) Infectious disease: foodborne diseases. Humana Press Inc, Totowa, NJ, pp 215–232
- Pedalino B, Feely E, McKeown P, Foley B, Smyth B, Moren A (2003) An outbreak of Norwalk-like viral gastroenteritis in holidaymakers travelling to Andorra, January–February 2002. Euro Surveill 8 (1): 1–8
- Austrian Agency for Health and Food Safety: Vorgehen bei Gastroenteritis-Ausbrüchen durch Norovirus in Krankenhäusern und Einrichtungen der stationären Pflege. AGES
  – Österreichische Agentur für Gesundheit und Ernährungssicherheit GmbH, Vienna 2006
- Kuo HW, Schmid D, Jelovcan S, Pichler AM, Magnet E, Reichart S, Allerberger F (2009) A foodborne outbreak due to norovirus, Austria, 2007. J Food Protect 72: 193–196

Wien Klin Wochenschr (2009) 121: 157-163 DOI 10.1007/s00508-008-1141-7 Printed in Austria © Springer-Verlag 2009

### Wiener klinische Wochenschrift

The Middle European Journal of Medicine

### A food-borne outbreak of Shigella sonnei gastroenteritis, Austria, 2008

Hung-Wei Kuo<sup>1,2</sup>, Sabine Kasper<sup>1</sup>, Sandra Jelovcan<sup>1</sup>, Gerda Höger<sup>1</sup>, Ingeborg Lederer<sup>1</sup>, Christoph König<sup>3</sup>, Gerda Pridnig<sup>3</sup>, Anita Luckner-Hornischer<sup>4</sup>, Franz Allerberger<sup>1</sup>, Daniela Schmid<sup>1</sup>

- <sup>1</sup>Austrian Agency for Health and Food Safety (AGES), Vienna, Austria
- <sup>2</sup>National Health Command Center, Centers for Disease Control, Taiwan
- <sup>3</sup>Public Health Authority, Salzburg, Austria
- <sup>4</sup>Public Health Authority, Innsbruck, Austria

Lebensmittelbedingter Ausbruch von Gastroenteritis durch Shigella sonnei, Österreich, 2008

Zusammenfassung. Wir berichten, für Österreich erstmalig, über einen lebensmittelbedingten Ausbruch von Gastroenteritis durch Shigella sonnei. Am 14. Juli 2008 erlangte die AGES Kenntnis über das gehäufte Auftreten von laborbestätigten S. sonnei Infektionen in einem Bezirk des Bundeslandes Salzburg. Alle Betroffenen hatten vom 7. bis zum 9. Juli an einem (vorzeitig abgebrochenen) Jungscharlager in Tirol teilgenommen. Ein Jungschar-Ausbruchsfall wurde definiert als jemand der am Jungscharlager teilnahm und in der Zeit 8. bis 12. Juli an Durchfall erkrankte. Unter den 61 Teilnehmern erfüllten 42 Jungscharlagerteilnehmer die Definition eines Jungschar-Ausbruchsfalls; darunter fanden sich 31 kulturbestätigte Fälle und 11 Fälle mit negativen Stuhluntersuchungen. Das Alter der 42 Erkrankten betrug im Median 11 Jahre (Min: 8, Max: 60), 26 waren weiblich. Die epidemiologische Falldefinition für Sekundärfälle bei Haushaltsmitgliedern lautete: ein Haushaltsausbruchsfall war jemand, der am Jungscharlager nicht teilnahm und ab 10. Juli an Durchfall erkrankte und 1 bis 3 Tage vor Erkrankungsbeginn mit einem Jungschar-Ausbruchsfall Haushaltskontakt hatte. Von den 11 Haushaltsausbruchsfällen unter den 139 befragten Haushaltsmitgliedern wurden 10 bakteriologisch untersucht; 4 Haushaltsangehörige hatten eine kulturbestätigte S. sonnei Infektion. Das Alter dieser Sekundärfälle betrug im Median 39,5 Jahre (Min: 15 a, Max: 48 a), 6 waren weiblich. S. sonnei wurde auch bei einer der beiden Herbergsbetreiber nachgewiesen. Alle 36 Isolate waren Biotyp gl, Lysotyp 12, und mittels PFGE voneinander nicht zu unterscheiden. Eine Kohortenstudie unter den 61 Teilnehmern des Jungscharlagers erbrachte die Konsumation von Salat (an zumindest einem der drei Tage) als Risikofaktor mit einem relativen Risiko von 2,71 (95%: 95% CI: 1,38-5,32, p = 0,004). Von den 42

Correspondence: Dr. med. Daniela Schmid, Österreichische Agentur für Gesundheit, und Ernährungssicherheit (AGES), Währingerstraße 25a, 1096 Wien, Austria, E-mail: daniela. schmid@ages.at

Ausbruchsfällen des Jungscharlagers konnten 34 auf die Konsumation von Salat zurückgeführt werden. Die asymptomatisch infizierte Herbergsbetreiberin, welche die Salate mit bloßen Händen zubereitete, ist möglicherweise die Quelle der Kontamination. Ohne eine epidemiologisch-analytische Untersuchung wäre die Assoziation mit Lebensmitteln bei diesem Shigellosen-Ausbruch nicht erkannt worden.

Summary. We report on the first foodborne outbreak of Shigella sonnei described in Austria. On July 14 2008, AGES was informed of a cluster of 22 laboratory-confirmed cases of infection with S. sonnei restricted to public health district X in the province of Salzburg. All cases had attended a youth-group trip to a small village in the province of Tyrol from July 7 to July 9. An outbreak case among the trip participants was a person who (1) attended the trip and (2) fell ill with diarrhea in the period between July 8 and July 12. Among the 61 trip participants, 42 fitted the outbreak case definition, including 31 culture-confirmed cases. A household outbreak case was a person who (1) did not participate in the trip, (2) fell ill with diarrhea not before July 10 and (3) had household contact with an outbreak case between one and three days before onset of illness. Of the 11 household outbreak cases, 10 were tested by stool culture and four of these had a laboratory-confirmed S. sonnei infection. In addition, one of the two hostel staff tested positive for S. sonnei. All 36 isolates were biotype gl, lysotype 12, and were indistinguishable from each other by PFGE. A cohort study among the trip participants revealed a risk ratio of 2.71 for consumption of salad (on at least 1 of the 3 days of the trip) (95% CI: 1.38-5.32, P = 0.004). Among the 42 cases, 34 could be explained by consumption of salad. The landlady of the hostel, who prepared the salad with bare hands, was a carrier and was assumed to be the source of contamination of the salad. Without proper epidemiological analytical investigation of this shigellosis outbreak, its association with food consumption would not have been identified.

Key words: Outbreak, shigella, foodborne, salad.

### Introduction

Shigellosis is an acute bacterial disease of the large and small intestine and is caused by one of four shigella species. Shigellosis due to Shigella sonnei is more frequent in industrialized countries, whereas S. dysenteriae, S. boydii and S. flexneri account for most isolates from developing countries [1, 2]. Humans are the most relevant reservoir of shigella [1, 3, 4]. The pathogen is transmitted directly by the fecal-oral route or indirectly through contaminated food and water, with an incubation period of 1-3 days. Watery diarrhea is mainly associated with S. sonnei and bloody diarrhea with the other three species. The period of communicability lasts during the acute illness and usually until four weeks after illness; asymptomatic carriers can be another source of infection. Appropriate antimicrobial treatment reduces duration of excretion to a few days [1]. Outbreaks of shigellosis have been associated with crowded conditions and poor personal hygiene, such as in prisons, daycare centers, schools and psychiatric institutions [5-9]. Large-scale outbreaks of foodborne shigellosis have been caused by infected food handlers practicing poor standards of personal hygiene [10-13]. Information on predominant sources and transmission routes of infections is crucial for targeted prevention of foodborne illnesses, and outbreaks frequently offer the only arena for gaining knowledge. Improved knowledge, in turn, provides a basis for improved prevention recommendations. Outbreak identification and the subsequent microbiological and epidemiological investigations therefore play a central role in prevention and control of infections from foodstuffs [14]. Whereas the incidence of sporadic notifiable disease can be seen as an 'unavoidable event', the occurrence of a foodborne outbreak is often an indication of inadequate standards of hygiene [15]. If the sources of an outbreak can be identified and successfully contained, then the occurrence of further outbreaks and subsequent sporadic cases can be prevented. Outbreak investigations are thus an instrument for evaluation and improvement of existing preventive measures [16]. The incidence of shigellosis in Austria has been estimated at 1.1, 1.2, 0.96 and 1.66 per 100,000 inhabitants in 2004, 2005, 2006 and 2007, respectively [17]. We report on an outbreak of shigellosis in 2008 that could be traced to consumption of salad - to our knowledge the first foodborne outbreak of S. sonnei described

On July 14, the Agency for Health and Food Safety, AGES was informed by a public health officer about a cluster of 22 laboratory-confirmed cases of infection with S. sonnei restricted to public health district X in the Austrian province of Salzburg. All cases had attended a youth-group trip to a small village in the province of Tyrol from July 7 to July 9 and had fallen ill with diarrhea between July 8 and July 11. The one-week trip was ended on July 9 earlier than scheduled. AGES was mandated to investigate this cluster of cases. The objectives of the investigation were to confirm the cluster as an outbreak, to describe the outbreak, identify the outbreak source(s) and estimate the household attack rate.

### Descriptive epidemiology

A case in the cluster was initially defined as a person who fell ill with gastroenteritis between July 6 and July 19 (calendar weeks 28 and 29), who resided in the public health district X in the province Salzburg and who had a laboratory-confirmed infection with S. sonnei. Thirty persons fulfilled this initial case definition. Since only three cases had been notified to public health district X during the previous year, 2007, the number of cases reported in calendar weeks 28 and 29 of 2008 exceeded the expected number. The occurrence of an outbreak of shigellosis was thus verified. Questioning the initial 30 cases revealed that 25 had attended the youth-group trip and five had not participated but were household members of cases who had attended the trip. Based on these preliminary findings a specific definition of an outbreak case was established as follows. A confirmed outbreak case was a person who (1) attended the trip to village Y in the province Tyrol from July 7 to July 9 and (2) fell ill with diarrhea in the period July 8-12, 2008, and (3) had a laboratory-confirmed infection with S. sonnei. A probable outbreak case was a person who fulfilled criteria (1) and (2) but not (3) of the above definition of a confirmed outbreak case. A household outbreak case was a person who (1) did not participate in the youth-group trip from July 7 to July 9 but (2) fell ill with diarrhea not before July 10, 2008 and (3) had household contact (included sharing food or eating utensils, using the same toilet, having physical contact) with an outbreak case between one and three days before onset of the illness.

Stool samples from the trip participants, from household members of the trip participants and from the two members of staff at the hostel were cultured for Salmonella spp., Campylobacter spp., Shigella spp., Yersinia enterocolitica and enterohemorrhagic E. coli and tested for norovirus by reverse transcriptase-PCR. S. sonnei isolates were biotyped, lysotyped and genotyped by pulse-field gel electrophoresis (PFGE) as described elsewhere [18, 19].

A total of 61 persons (54 children, 7 supervisors) participated in the youth-group trip and 144 household members of the trip participants were identified. For active case finding all participants of the trip, the two staff members at the hostel hosting the youth group and 139 (96.5%) of the 144 participants' household members were interviewed on clinical signs and symptoms consistent with gastroenteritis, date of clinical onset and demographics.

Of the 61 participants of the trip, 42 (68.9%) fitted the definition of a trip outbreak case: 31 confirmed and 11 probable cases. Another 14 unaffected participants were tested and were culture negative for shigella. The median age of the outbreak cases was 11 years (min: 8, max: 60) with a male-to-female ratio of 1:1.6.

The median age of the household members was 36 years (interquartile range: 14.5–43; min: 1; max: 66); 64 were female (46% of 139). Eleven of the 139 (7.9%) household members fitted the definition of a household outbreak case; 10 of the 11 were tested by stool culture and

four had laboratory-confirmed S. sonnei infection. Another 77 unaffected household members tested were culture negative for shigella. The median age of the household cases was 39.5 years (min: 15, max: 48) with a male-to-female ratio of 1:1.2.

One of the two hostel staff tested positive for S. sonnei.

All 36 S. sonnei isolates were biotyped, lysotyped and genotyped by PFGE: all were biotype gl, lysotype 12 and were indistinguishable from each other by PFGE. All the isolates were resistant to streptomycin, sulfonamide, tetracycline and trimethoprim-sulfamethoxazole, and none were resistant to ciprofloxacin or nalidixic acid.

All 53 outbreak cases had diarrhea; in 33 (62.3%) cases this was accompanied by fever (unspecified) and in 25 (47.2%) by vomiting. One case (1.9%) was hospitalized.

The outbreak lasted from July 8 to July 19. When disentangling the cases into youth-group cases and household cases, the spatial distribution of the youthgroup cases indicated a common source with possible exposure to the infectious agent on July 7 and July 8. Figure 1 shows the outbreak curve: the youth-group cases occurred from July 8 to July 11 and peaked with 30 cases on July 9.

The hypothesis generated was that food items prepared in the kitchen of the hostel during the visit of the youth group and consumed by the trip participants on July 7, 8 and 9 were associated with risk of becoming an outbreak case. The hypothesis was tested in a retrospective cohort study as the cohort-at-risk was readily identifiable and the questioning of cohort members was feasible.

In order to assess the possibility of direct fecal-oral transmission, detailed information on participants' accommodation in the hostel was ascertained. The hostel had 15 bedrooms and was run by a staff of two persons who were also the owners of the premises. The youth group had occupied all 15 bedrooms. Among the first three cases, case 1 fell ill on July 8 (Tuesday) in the morning and the other two cases in the late afternoon. Case 1 was staying in a room (room Z) on the first floor and used only the toilet in that room. Another five cases, who were also staying in room Z, fell ill on July 9. The remaining 25 cases, occurring on July 9, stayed in the other 14 bedrooms. Of the eight cases occurring on July 10, six shared rooms with cases that had occurred on July 9. The other two cases from July 10 shared the same room with the three cases that occurred on July 8.

### Analytical epidemiology

### Materials and methods

The cohort-at-risk consisted of 61 participants of the youthgroup trip: 54 children and seven supervisors. All cohort members were interviewed by telephone using a standardized questionnaire. Data were obtained on demographics, disease status, date of clinical onset, symptoms (nausea, fever, vomiting, diarrhea, abdominal pain), duration of illness and hospitalization. Data on exposure between July 7 and July 9 were

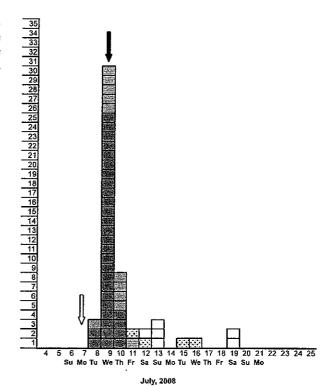


Fig. 1. Outbreak cases by date of disease onset (N = 52 of a total of 53 outbreak cases; for one non-laboratory-confirmed household outbreak case the date of disease onset was unknown); dark grey squares: youth-group trip confirmed outbreak cases (n = 31); light grey squares: youth-group trip probable outbreak cases (n = 11); white dotted squares: laboratory-confirmed household outbreak cases (n = 4); white squares: non-laboratory-confirmed household outbreak cases (n = 6); white arrow: arrival of the youth group at the hostel; black arrow: departure of the youth group

also collected: consumption of food items prepared and served in the hostel, swimming in a nearby lake and drinking water from a nearby open well.

Exposure was defined as follows: the three days from July 7 to July 9 (Monday - Wednesday) were considered as the relevant days on which risky exposure might have occurred. The following exposure factors were tested: swimming in a nearby lake; drinking water from a nearby well; consumption of food items served at breakfast; consumption of water, orange juice or cherry juice from any of three beverage dispensers on any day between July 7 and July 9; consumption of food items at lunch and dinner on July 7 and July 8, and consumption of food items at lunch on July 9. On July 7 the lunch consisted of spaghetti and as a dessert canned peaches; the dinner on that day consisted of egg soup, chicken legs, French fries, chocolate pudding, green salad, carrot salad, tomato salad, cucumber salad, cabbage, yoghurt dressing. On July 8 lunch consisted of soup with pancake, "Leberkäse" (a type of meat loaf popular in Germany and Austria), mashed potatoes and cooked vegetables. Dinner that day was turkey steak, rice, green salad, and salads of carrot, tomato, cucumber, cabbage and egg (hens eggs); dessert was vanilla ice cream. On July 9 the lunch included clear soup, noodles with sausage and green salad, tomato salad, cabbage salad and yoghurt dressing. On all three days breakfast consisted of tea, coffee, chocolate milk, milk, bread rolls, cold-cut meats, cheese, spread, butter, cereal and yoghurt.

Data were analyzed as follows. The risk of food-associated disease on a specific day between July 7 and July 9 (i.e. dayspecific food-associated attack rate, AR) was computed by defining exposure as consumption of any food item offered at breakfast, lunch or dinner on that specific day. Taking into account that the etiological agent was S. sonnei and knowing its short incubation period (24-72 h), persons who had fallen ill on July 8, 9 or 10 were included in the numerator when considering consumption of any food on July 7 as risky exposure. The denominator was given by the number of cohort members having consumed any food on July 7 (labeled as Monday-specific risk of food-associated disease). The Monday-specific risk was compared with the day-cumulative risk for the remaining days July 8 (Tuesday) and July 9 (Wednesday). The day-cumulative risk was computed from the number of cases attributed to food exposure on July 8 and July 9 divided by the cumulative number of cohort members having consumed any food on July 8 and July 9. The day-specific risks for days July 8 and 9 (Tuesday-specific risk and Wednesday-specific risk) were similarly computed. The day-specific food-associated risk ratio (RR) was calculated by dividing the day-specific risk by the daycumulative risk (Monday-specific food-associated RR, Tuesday-specific food-associated RR and Wednesday-specific foodassociated RR).

For day-by-day analyses of the food-item-specific risks of disease, the risks for Monday, Tuesday and Wednesday, the food-item-specific RRs for these days, and the persons who had eaten on the particular day were included in the "day-cohort". The number of cases and the number of persons in the denominator therefore varied for the day-by-day food-itemspecific cohort analyses. Considering the incubation period of 24-72 h, persons who had fallen ill on July 8, 9 or 10 were included in the numerator for the Monday food-item-specific cohort analyses when considering consumption of a particular food on July 7 as the risky exposure (food-item-specific RR for Monday). The food-item-specific disease risks and fooditem-specific risk ratios for Tuesday and Wednesday were computed following the same procedure. EpiInfo version 3.4.1 (CDC, Atlanta, Georgia) was used for data entry and univariable analyses. Differences in disease risk between exposed and unexposed groups were tested using the chi-squared test. The crude food-item-specific RRs were controlled for the effects of the food items on each other using a log-linear model (Poisson regression) using SAS 9.1.

### Results

All members of the cohort-at-risk responded to the telephone interviews (response rate 100%) and were included as study population (N = 61) for the retrospective cohort study. The male-to-female ratio was 1:1.44 and the median age was 11 years (interquartile range: 10–12.5 years). A total of 42 persons (35 pupils, 7 teachers) fitted the definition of a youth-group trip outbreak case: all suffered from diarrhea, 30 (71.4%) cases experienced fever (unspecified) and 22 (52.4%) vomiting.

There was no difference in the risk of becoming a case after consuming any food offered on Monday (July 7) compared with any food exposure on Tuesday (July 8) or Wednesday (July 9) (Monday-specific food-associated RR: 1.2, 95% CI: 0.9-1.6, P=0.14). Similarly, no difference was found in disease risk between food exposure on Tuesday when compared with any food exposure on Monday or Wednesday (Tuesday-specific food-associated RR: 1.1, 95% CI: 0.8-1.4, P=0.57). The risk of becoming a case associated with food exposure on Wednesday was significantly lower than any food

exposure on Monday or Tuesday (Wednesday-specific food associated RR: 0.54; 95% CI: 0.31–0.96, P=0.006). Choosing Wednesday as the reference day, the disease risk associated with consumption of any food offered on Monday was 1.9 times higher (95% CI: 1.06–3.38; P=0.008) and the disease risk associated with consumption of any food offered on Tuesday 1.8 times higher (95% CI: 1.0–3.2; P=0.02). Monday and Tuesday were identified as the days on which the risky exposures occurred. There was no difference in the day-specific food-associated disease risk between Monday and Tuesday (RR: 1.1; 95% CI: 0.8–1.4, P=0.64).

The day-by-day food-item-specific analyses for items served on Monday, Tuesday and Wednesday revealed that consumption of carrot salad, tomato salad, cucumber salad, any salad with yogurt dressing, and noodle salad on Monday were significantly associated with becoming an outbreak case. The RR of consumption of any salad served on Monday was 2.33 (95% CI: 1.32-4.11, P<0.01). On Tuesday, consumption of green salad and salads of carrot, tomato, cucumber, sweetcorn and egg (hens eggs) was associated with risk of disease: consumption of any salad on Tuesday had an RR of 2 (95% CI: 1.31–3.05, P < 0.01). Consumption of rice on Tuesday was associated with an RR of 0.66 (95% CI: 0.49-0.88; P = 0.04). Food items consumed on Wednesday were not associated with risk of disease. Cherry juice and water consumed from beverage dispensers on any of the three days (Monday, Tuesday, Wednesday) were associated with an RR of < 1 (Table 1). The RR of consumption of salad at least on one of the three days (Monday, Tuesday, Wednesday) was 2.71 (95% CI: 1.38–5.32, P = 0.004). Of the 42 outbreak cases, 34 could be explained by consumption of salad. There was no difference in the sex-specific or age-group-specific (age-group <11 years; age-group ≥11) risk of disease. The protective effects of consuming cherry juice, water from the beverage dispensers, or consuming rice indicated by the univariable analyses were further analyzed by stratified analyses and by fitting a regression model. The stratified analyses did not reveal evidence for interactions between salad and cherry juice or water consumed from the beverage dispensers. The protective effect of consumption of water and cherry juice on disease risk was indicated by the positive association of salad consumption with disease risk and the negative association of salad consumption with water or cherry juice consumption. Those who consumed water or cherry juice were significantly less likely to have consumed the risky salad (stratum consumed water: 57% exposure to salad versus stratum not consumed water: 71% exposure to salad). The stratified analysis of consumption of salad and rice on Tuesday yielded an effect modification: those who consumed rice and salad were protected from becoming a case; those who did not eat salad were no longer protected from becoming a case by consuming rice. After controlling for confounding by regression analysis, salad consumed on at least one of the three days (Monday, Tuesday, Wednesday) remained as the food item associated with risk of disease (crude RR = 2.71, 95% CI: 1.38-5.32, P = 0.004; adjusted RR: 2.11; 95 % CI: 1.22-3.66; P = 0.008).

Table 1. Food-item-specific disease risks (AR%) and risk ratios (RR) for food items significantly associated with the disease risk; 95% confidence interval (95% CI); P-value

Food items by day	AR % exposed	AR % unexposed	RR	95% CI	P
Monday-specific cohort* (N = 61)					
Carrot salad	87.5 (21/24)	541 (20/37)	1.62	(1:16-2:26)	< 0.01
Tomato saladis and a second	90.5 (19/21)	55.0 (22/40)	1.65	(1,20-2,25)	< 0.01
Cucumber salad	88.5 (23/26)	51.4 (18/35)	1.72	(1.21-2.44)	< 0.01
Noodle salad	100.0 (13/13)	58.3 (28/48)	1.71	(1.35-2.18)	< 0.01
Salad with yoghurt dressing	96.0 (24/25)	47.2 (17/36)	2.03	(1.43-2.90)	< 0.01
Any salad	84.6 (33/39)	36.4 (8/22)	2.33	(1.32-4.11)	< 0.01
Tuesday-specific cohort <sup>2</sup> (N = 58)				100	
Green salad	87.5 (14/16)	58.5 (24/41)	1.49	(1.09-2.05)	0.04
Garrot salad	92.3 (12/13)	59.1 (26/44)	1.66	(1.17-2.09)	0.02
Tomato salad	100.0 - (10/10)	59.6 (28/47)	-1.68	(1.33-2.12)	0.01
Cucumber salad	87.5 (14/16)	58.5 (24/41)	1,49	(1.09-2.05)	0.04
Sweetcom salad	88.2 (15/17)	57.5 (23/40)	1.53	(1.12-2.11)	0.02
Egg salad (hens eggs)	100.0 (7/7)	62.0 (31/50)	1.61	(1.30-2.00)	< 0.05
Salad with yoghurt dressing	94.1 (16/17)	55.0 (22/40)	1.71	(1.26-2.32)	< 0.01
Any salad	89.3 (25/28)	44.8 (13/29)	1.99	(1.30-3.04)	<0.01
Rice	60.0 (27/45)	91.7 ((1/12))	0.66	(0.49-0.88)	0.04
At least at one of the three days (N $=$ 61)			and the second		
Cherry juice:	55.9 (19/34)	85.2 (23/27)	0.66	(0,47-0.92)	0.01
Water	54.5 (18/33)	85.7 (24/28)	.0:64	(0,45-0.90)	< 0.01
Salad	85.7 (36/42)	31.6 (6/19)	2.71	(1.38–5.32)	< 0.01
<sup>1</sup> Cohort exposed to food items served or	i Monday; <sup>2</sup> Cohort exposed to 1	ood items served on Tuesc	lay.		

Sharing a room with cases that had disease onset on July 8 was not a risk factor for becoming a case after July 8 (RR: 1.24, 95% CI: 0.85-1.8). On July 9 all 14 rooms were already affected with at least one case. Sharing an affected room therefore could not be tested for association with the risk of becoming a case after July 9.

Consumption of salads on at least one of the three days Monday (July 7), Tuesday (July 8) or Wednesday (July 9) of the trip was independently associated with the risk of becoming an outbreak case. There was no salad left for microbiological testing. Thirty-four cases were explained by consumption of salad; of the remaining eight, five had disease onset on the second day of the outbreak (July 9): these may have been generated by one of the three cases who fell ill on the first outbreak day (July 7) by secondary transmission via hands or food utensils. The other three of the eight non-foodborne cases falling ill on outbreak days 3 (July 10) and 4 (July 11) may have been generated by infectious contacts on outbreaks days 1 (July 8) and 2 (July 9).

The two owners of the hostel managed hostel service and food preparation during the youth trip. Another in-depth interview with the female hostel owner, who was stool culture positive for S. sonnei, established that she had not had diarrhea, vomiting or fever during the four weeks before and during the period of the outbreak.

### Discussion

The shigellosis outbreak described is the largest one yet investigated in Austria. Epidemiological analytical investigation revealed salad as the likely source of the outbreak. Out of the 53 outbreak cases, 34 were explained by consumption of salad. Consumption of salad on at least one of the three days of the youth-group trip was highly likely to have been the source of the outbreak of shigellosis, even though there was no microbiological evidence as no salad samples were left and available for bacteriological testing. This situation is consistent with findings from other shigellosis outbreaks; whereas epidemiological methods can strongly imply a common food source, Shigella spp. are not often recovered from these foods [20]. Consumption of rice on day 2 of the youth trip proved to be a protective factor when salad was consumed on this particular day. Those who had eaten rice might have consumed a smaller portion of the risky salad.

Shigella can grow rapidly in food ingredients held at ambient temperature [21, 22] and S. sonnei has been shown to multiply in shredded lettuce and lettuce extract under certain conditions [13]. A wide range of salads have previously been involved in foodborne outbreaks of shigellosis. In October 1989, 15% of the passengers and 3% of the crew aboard a cruise ship fell ill

with shigellosis [23]. The source of this outbreak, due to S. flexneri, was German potato salad; contamination was introduced by infected food handlers in the country where the food was prepared. Contaminated carrots in salads served on airline flights caused at least 45 cases of foodborne illness due to S. sonnei in 2004 [11]. In 1990, S. sonnei was the most prevalent enteric pathogen cultured from troops engaged in the military campaign Operation Desert Shield [24]; the suspected source was contaminated fresh vegetables, notably lettuce. In 1994, 110 culture-confirmed cases of shigellosis caused by S. sonnei were reported in an outbreak in Norway, Sweden and the UK [25, 26]; based on epidemiological evidence iceberg lettuce imported from Spain was suspected as the source of this multi-country outbreak. Establishments where contaminated foods have been served include the home, restaurants, camps, picnics, schools, daycare centers, airlines, sorority houses and military mess halls, among others [20]. In a 1986 outbreak of S. sonnei in Texas, 347 patrons who had eaten at three separate restaurants fell ill; commercially distributed shredded lettuce was epidemiologically and microbiologically implicated [13].

One of the striking features of foodborne outbreaks caused by shigella is that in many situations contamination of foods may not have originated pre-harvest in the field (e.g. from fecally contaminated irrigation water) or post-harvest at the processing plant but rather the source can be traced to symptomatic or asymptomatic food handlers [27]. The spread of the pathogen may occur via fingers, flies, or inanimate objects such as utensils and cutting surfaces. Infected individuals can shed 1 x 103 to 1 x 109 colony-forming units (CFU) per gram of stool during the acute phase and 1 x 102 to 1 x 103 CFU/g in convalescence [3]. The infective dose is estimated to be as low as 10-100 bacteria, which favors high transmissibility [1, 3]. Asymptomatic carriers of shigella may exacerbate the spread of this pathogen [1]. In the Austrian outbreak, the landlady of the hostel, who prepared the salad with bare hands, was a carrier and assumed to be the source of the salad contamination. Food handling and preparation are important processes that require appropriate hygiene measures, and proper protocols for hand washing should be prominent and practiced in all food-processing facilities. Infected food handlers should be excluded from work until stool cultures are negative for shigella.

Multidrug resistance to most of the low-cost antibiotics, as exhibited by the Austrian outbreak strain, is common [1]. Antibiotics, chosen according to in vitro susceptibility testing, can be used for individual patients if warranted by the severity of illness. An additional aim of antibiotic use is to interrupt the chain of transmission in order to prevent successive cases. Appropriate antibiotic treatment usually reduces duration of pathogen excretion to a few days. In the Austrian outbreak described here at least 13 cases received antibiotic treatment (data not shown).

Nevertheless, hand washing with soap is the single most effective means of preventing secondary transmission. Outbreaks in school settings, mainly in preschool age groups, have been described with household attack rates up to 40% [28, 29]. In the Austrian outbreak, the household attack rate was 7.9%, which is low in comparison with other published shigellosis outbreaks [28, 29]. This low household transmission rate may reflect the prompt intervention of the respective public health authorities, propagating strict practice of hand hygiene.

Without proper epidemiological analytical investigation of this shigellosis outbreak the association with food would not have been identified. Patrons of affected establishments often blame inadequate hand hygiene among young guests as cause of spread of infectious gastroenteritis. Epidemiological and microbiological investigation is the only instrument for determining the cause of pathogen spread. Outbreak investigations should not be seen as a mere legal obligation according to the Zoonoses Act 2006 (Zoonosengesetz 2006) but as an opportunity to identify risky behavior in order to implement appropriate preventive measures.

### References

- Heymann DL (2004) Control of communicable diseases manual, 18th edn. American Public Health Association, Washington, DC
- Kotloff KL, Winickoff JP, Ivanoff B, Clemens JD, Swerdlow DL, Sansonetti PJ, et al (1999) Global burden of Shigella infections: implications for vaccine development and implementation of control strategies. Bull World Health Organ 77: 651–666
- Sansonetti PJ (2001) Microbes and microbial toxins: paradigms for microbial-mucosal interactions III. Shigellosis: from symptoms to molecular pathogenesis. Am J Physiol Gastrointest Liver Physiol 280: G319–323
- Lederer I, Much P, Voracek T, Vielgrader H, Allerberger F (2005) Outbreak of shigellosis in the Vienna Zoo affecting human and non-human primates. Int J Infect Dis 9: 290– 201
- Jonsson J, Álvarez-Castillo MC, Sanz JC, Ramiro R, Ballester E, Fernánez M, et al (2005) Late detection of a shigellosis outbreak in a school in Madrid. Euro Surveill 10 (10): pii=571
- Hardy A (1945) Shigellosis (bacillary dysentery) among institutional inmates. Psychiatric Quarterly 19: 377–397
- Hill PC, Hicking J, Bennett JM, Mohammed A, Stewart JM, Simmons G (2002) Geographically separate outbreaks of shigellosis in Auckland, New Zealand, linked by molecular subtyping to cases returning from Samoa. N Z Med J 115: 281–283
- Lee LA, Ostroff SM, McGee HB, Johnson DR, Downes FP, Cameron DN (1991) An outbreak of shigellosis at an outdoor music festival. Am J Epidemiol 133: 608–615
- Chao YN, Huang ASE, Chiou CS, Tsai SH, Lin SH, Lee PH (2008) A waterborne shigellosis outbreak in a primary school, Tai-Chung City, November 2007. Taiwan Epidemiology Bulletin 24: 740–751
- Lewis HC, Kirk M, Ethelberg S, Stafford R, Olsen KE, Nielsen EM, et al (2007) Outbreaks of shigellosis in Denmark and Australia associated with imported baby corn, August 2007 – final summary. Euro Surveill 12 (40): pii=3279
- Hedberg CW, Levine WC, White KE, Carlson RH, Winsor DK, Cameron DN, et al (1992) An international foodborne outbreak of shigellosis associated with a commercial airline. JAMA 268: 3208–3212
- Black RE, Craun GF, Blake PA (1978) Epidemiology of common-source outbreaks of shigellosis in the United States, 1961–1975. Am J Epidemiol 108: 47–52

- Davis H, Taylor JP, Perdue JN, Stelma GN, Humphreys JM, Rowntree R, et al (1988) A shigellosis outbreak traced to commercially distributed shredded lettuce. Am J Epidemiol 128: 1312–1321
- Ammon A (2007) Lebensmittelbedingte Infektionen: Untersuchung von Ausbrüchen der Schlüssel zur Quelle. Wien Klin Wochenschr 119: 139–141
- Krause G (2009) Investigating outbreaks: medical imperative or academic indulgence? Wien Klin Wochenschr (in this issue)
- Reingold AL (1998) Outbreak investigations a perspective. Emerg Infect Dis 4: 21–27
- Hoeger G, Kornschober C (2008) Shigellen Jahresbericht 2007 der Nationalen Referenzzentrale für Shigellen. Newsletter Gesundheit – 3. Quartal 2008. Available at: www.bmgfj.gv.at/cms/site/attachments/3/5/9/CH0951/ CMS1214392137719/shigella\_jb\_2007.pdf)
- Swaminathan B, Barrett TJ, Hunter SB, Tauxe RV, CDC PulseNet Task Force (2001) PulseNet: the molecular subtyping network for foodborne bacterial disease surveillance, United States. Emerg Infect Dis 7: 382-389
- 19. Hunter SB, Vauterin P, Lambert-Fair MA, vanDuyne MS, Kubota K, Graves L, et al (2005) Establishment of a universal size standard strain for use with the PulseNet standardized pulsed-field gel electrophoresis protocols: converting the national databases to the new size standard. J Clin Microbiol 43: 1045-1050
- Lampel KA, Maurelli AF (2007) Shigella species. In: Doyle MP, Beuchat LR (eds) Food microbiology: fundamentals and frontiers, 3rd edn. John Wiley & sons, Hoboken, NJ, pp 323-342

- Hutchinson RI (1956) Some observations on method of spread of Sonnei dysentery. Bull Ministry Health 15: 110–118
- Wu FM, Doyle MP, Beuchat LR, Wells JG, Mintz ED, Swaminathan B (2000) Fate of Shigelia sonnei on parsley and methods of disinfection. J Food Prot 63: 568-572
- Lew JF, Swerdlow DL, Dance ME, Griffin PM, Bopp CA, Gillenwater MJ, et al (1991) An outbreak of shigellosis aboard a cruise ship caused by a multiple-antibiotic-resistant strain of Shigella flexneri. Am J Epidemiol 134: 413– 420
- Hyams KC, Bourgeois AL, Merrell BR, Rozmjzl P, Escamilla J, Thornton SA, et al (1991) Diarrheal disease during Operation Desert Shield. N Engl J Med 325: 1423-1428
- Frost JA, McEvoy MB, Bentley CA, Anderson Y, Rowe B (1995)
   An outbreak of Shigella sonnei infection associated with consumption of iceberg lettuce. Emerg Infect Dis 1: 26-29
- Kapperud G, Rorvik LM, Hasseltvedt V, Hoiby EA, Iversen BG, Staveland K, et al (1995) Outbreak of Shigella sonnei infection traced to imported iceberg lettuce. J Clin Microbiol 33: 609–614
- Warren BR, Parish ME, Schneider KR (2006) Shigella as a foodborne pathogen and current methods for detection in food. Crit Rev Food Sci Nutr 46: 551-67
- Jonsson J, Álvarez-Castillo MC, Sanz JC, Ramiro R, Ballester E, Fernánez M, et al (2005) Late detection of a shigellosis outbreak in a school in Madrid. Euro Surveill 10: pii=571
- Centers for Disease Control and Prevention (2004) Day care-related outbreaks of rhamnose-negative Shigella sonnei: six states, June 2001 – March 2003. MMWR Morb Mortal Wkly Rep 53: 60-63

Journal of Food Protection, Vol. 72, No. 1, 2009, Pages 193-196 Copyright ©, International Association for Food Protection

### **Research Note**

### A Foodborne Outbreak Due to Norovirus in Austria, 2007

HUNG-WEI KUO,<sup>1,2</sup> DANIELA SCHMID,¹ SANDRA JELOVCAN,¹ ANNA-MARGARETHA PICHLER,¹ EVA MAGNET,³ SANDRA REICHART,³ AND FRANZ ALLERBERGER¹\*

<sup>1</sup>Austrian Agency for Health and Food Safety, Vienna, Austria; <sup>2</sup>National Health Command Center, Centers for Disease Control, Taipei, Taiwan; and <sup>3</sup>Public Health Authority Upper Austria, Linz, Austria

MS 08-304: Received 24 June 2008/Accepted 28 August 2008

### ABSTRACT

A foodborne norovirus outbreak occurred after a pre-Christmas celebration among a group of local foresters in Austria in December 2007. A total of 66 persons, 60 participants of the Christmas party and 6 kitchen staff members of the restaurant where the party took place, were identified as the at-risk cohort. Questioning of this cohort was performed by self-report questionnaires or telephone interviews (response rate of 95%). The outbreak attack rate was 33.3% (21 of 63 persons), including two of the kitchen staff. Three stool specimens yielded norovirus genogroup II. Univariate analysis revealed that exposure to a ham roll and pastry was possibly associated with risk of gastroenteritis (risk ratio [RR] of 4.45, 95% CI of 1.91 to 10.9; RR of 2.44, 95% CI of 0.93 to 6.39). After controlling for the effects of sex, age, and other food items with a log-linear model, ham roll remained significantly associated with disease risk (RR of 3.91, 95% CI of 1.57 to 9.76). Ham roll was most likely contaminated with norovirus during preparation by a disease-free kitchen assistant, whose infant became sick with laboratory-confirmed norovirus gastroenteritis 2 days before the party. Informing food handlers about the possible risk of food contamination with norovirus and training them in the appropriate measures of hand hygiene and environmental disinfection at the working place and at home are essential for preventing food-related norovirus outbreaks. Norovirus-infected household members of healthy food handlers must be considered a possible reservoir for foodborne norovirus outbreaks.

Infection with norovirus (NV) is exceedingly widespread and common (10). Transmission of norovirus occurs year around in temperate climates, with a higher incidence of disease in winter months. Early studies with volunteers provided our basic knowledge on infection dose, incubation period, transmission modes, and salient clinical features of NV infection (2, 3, 13). NV is easily spread via aerosolized vomit, via contaminated environmental surfaces, and directly from person to person via the fecal-oral route. NV is increasingly recognized as a leading cause of foodborne disease in the United States and Europe (6, 7). However, well-documented foodborne outbreaks due to NV are rarely found in the literature (4, 5, 9, 12). New information on patterns of NV transmission can be gained only by investigating outbreaks. We investigated an outbreak of NV gastroenteritis among a group of foresters who participated in a pre-Christmas party at a local restaurant. The objectives of the investigation were to identify the reservoir of NV and the most likely chain of transmission.

On 21 December 2007, the Austrian Agency for Health and Food Safety was informed by a local public health officer about a cluster of gastroenteritis cases among a group of foresters. The cluster occurred in the first half of the week 15 to 22 December in a small village in a northern Austrian province. The day after the foresters' Christmas party in restaurant X on Friday, 14 December, participants

fell ill with diarrhea lasting no longer than 2 days or with vomiting. Pre-Christmas celebrations are common among Austrian employees, usually consisting of dinner in a restaurant paid for by the employer. The initial case series investigation based on the epidemiological criteria defined by Kaplan et al. indicated NV as the causative pathogen (11). Because there were no indications of airborne NV spread, the consumption of food items served at the party remained the only relevant common link among the gastroenteritis patients. Environmental investigation for NV was not performed; no samples from food or kitchen surfaces were tested. A retrospective cohort study was conducted to identify the source of the NV outbreak and the reservoir of the pathogen.

### MATERIALS AND METHODS

Outbreak case definitions. A confirmed case of the outbreak was defined as a person who met all of the following criteria: 1, attended the pre-Christmas celebration held in restaurant X on the evening of 14 December 2007 or was part of the kitchen staff working in restaurant X on that evening; 2, fell ill with symptoms of diarrhea or vomiting after 14 December; 3, stool specimen tested positive for NV. A probable case of the outbreak was defined as a person who met criteria 1 and 2 of a confirmed case but did not meet criterion 3 because stool samples were either not tested or were negative for NV.

Analytical study design. A retrospective cohort study was performed because a clearly identifiable risk group for which information could be obtained was available. The risk group com-

<sup>\*</sup> Author for correspondence, Tel: 011-43-50555-35500; Fax: 011-43-50555-95-35500; B-mail: franz.allerberger@ages.at.

TABLE 1. Distribution of symptoms in the 21 outbreak cases

Symptoms	No. (%) of cases
Nausea	19 (86.4)
Diarrhea	17 (81.0)
Vomiting	15 (71.4)
Abdominal cramps	13 (61.9)
Fever	7 (33.3)

prised 66 persons including 60 foresters who participated in the celebration in restaurant X on 14 December and 6 persons of the restaurant kitchen staff who prepared, served, or tasted food items offered during this party. The hypothesis to be tested, based on the results of initial interviews of the first known cases, was that NV-contaminated food items consumed at the Christmas party were the most likely outbreak source.

Definition of exposure. Exposure was defined as consumption of at least one dish served at the party. The dishes offered were goulash soup, beef soup with *fridatten*, vegetable salad, potato salad, tomato salad, bean salad, lettuce salad, cucumber salad, tuna spread, pumpkin seed spread, paprika spread, liver spread, bread, a specific pastry called *Jourgebäck*, an assortment of cheeses, mozzarella with tomatoes, smoked salmon, roast pork, filled eggs, ham roll filled with cream cheese, and as dessert a variety of cakes, apple strudel, cheese strudel, and donuts.

Data collection and analyses. A questionnaire was developed and sent to the households of the cohort members, based on a guest list provided by the organizer of the party. When there was no response to the questionnaire, the person was contacted and interviewed by telephone. Data were obtained on demographics, clinical onset, symptoms (nausea, fever, vomiting, diarrhea, and abdominal cramps), duration of illness, hospitalization, exposure to food items listed above, and (if stool specimens were available for laboratory testing) laboratory results. Stool samples were tested for NV by reverse transcription (RT) PCR (8). For data entry and univariate analyses, the EpiInfo 3.4.1 software package (http://www.cdc.gov/epiinfo/) was used. Differences in food-specific attack rates between exposed and unexposed groups were evaluated with a chi-square test. A log-linear model (Poisson regression) was used to control for the effects of age, sex, and the relationship of food items to each other. Food items that were not considered plausible sources of infection based on the results of the univariate analysis were excluded from the multivariate analysis.

### RESULTS

Completed questionnaires were obtained from 63 of the 66 persons in the at-risk cohort (response rate, 95.5%). Twenty-one of the 63 cohort members, including 3 laboratory confirmed cases, fit the definition of an outbreak case (attack rate, 33.3%). Of these 21 cases, 19 attended the party and 2 were members of the kitchen staff. Among the 21 cases, 8 were female (38.1%), and the median age was 40 years (range, 16 to 58 years). The frequency distribution of the symptoms is given in Table 1. Nine (42.9%) of the 21 outbreak cases had stool specimens tested for NV within 7 days following the party, and three of these specimens were positive for NV genogroup II. The outbreak lasted from 15 to 17 December and peaked on 16 December (Fig. 1). Taking NV into account as the etiological agent of the

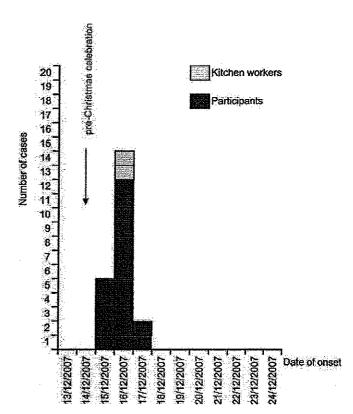


FIGURE 1. Outbreak cases by clinical onset (n = 21).

outbreak and considering its short incubation period (24 to 48 h), the pattern of the epidemic curve clearly indicates a point source outbreak.

The food-specific cohort analyses revealed that consumption of the ham roll was associated with disease risk (relative risk [RR], 4.55; 95% confidence interval [CI], 1.91 to 10.9). The association of consumption of the pastry *Jourgebäck* with NV infection risk was of borderline significance, with an RR of 2.44 (95% CI, 0.93 to 6.39; P = 0.042). After controlling for the effects of sex, age, and the association of the food items ham roll and *Jourgebäck* to each other with a log-linear model, the consumption of ham roll remained significantly associated with infection risk (adjusted RR, 3.91; 95% CI, 1.57 to 9.76) (Table 2).

Source of NV contamination. In-depth interviews of the kitchen staff on exposure to persons with gastroenteritis before the pre-Christmas celebration revealed that a kitchen assistant had spent the night of 13 to 14 December in a hospital accompanying her 6-month-old daughter, who had projectile vomiting and diarrhea since 12 December, 2 days before the party. There was no guidance given by the hospital staff to the mother on measures to prevent spread of diarrheal disease. The stool specimens obtained from the infant were positive for NV. According to information given by the kitchen assistant, she and her home environment were exposed to the infant's stool and vomiting before the infant was hospitalized. Hygienic measures appropriate for decontamination of hands, textiles, and surfaces after contamination with NV were not applied by the kitchen assistant at her home.

TABLE 2. Results of the univariate and multivariate analyses<sup>a</sup>

	124	Persons exposed	70	Per	Persons not exposed	ed	Univariate analysis	sis	Multivariate analysis	sis
	No. ill	Total	AR%	No. ill	Total	AR%	Crude RR (95% CI)	Ъ	Adjusted RR (95% CI)	Ь
Ham roll	16	26	61.5	5	37	13.5	4.55 (1.91–10.9)	<0.001	3.91 (1.57–9.76)	<0.008
Pastry Joursehäck	17	40	42.5	4	23	17.4	2,44 (0.93–6.39)	0.04	1.53 (0.61–3.89)	0.44
Salad buffet	9	24	41.7	11	39	28.2	1,48 (0.74–2.94)	0.27	NDφ	
Filled coos	<u> </u>	24	41.7	11	39	28.2	1.48 (0.74–2.94)	0.27	QZ	
Pumpkin seed spread	~	16	50	13	47	27.7	1.81 (0.92–3.55)	0.10	<del>Q</del>	
Tima spread	· oc	17	47.1	13	46	28.3	1.67 (0.84–3.30)	0.16	QN	
Paprika spread	, vc	12	50	15	51	29.4	1.70 (0.84–3.45)	0.17	Ð	
Liver spread	2 0	9	33.3	19	57	33.3	1.00 (0.30-3.29)	1.00	R	
Minced meat suread	2	4	50	19	59	32.2	1.55 (0.54-4.43)	0.47	ΩN	
Mozzarella with tomatoes	0	23	39.1	12	40	30.0	1.30 (0.65–2.62)	0.46	Ð	
Smoked salmon	. 6	21	42.9	12	42	28.6	1.50 (0.75–2.98)	0.26	ON	
Potato salad	, 01	28	35.7	=	35	31.4	1.14 (0.57–2.28)	0.72	QN	
Vecetable salad	, <b>'</b> C	16	37.5	15	47	31.9	1.18 (0.55–2.51)	89.0	ΩN	
I ettice salad	0	22	40.9	12	41	29.3	1.40 (0.70–2.79)	0.35	QN CN	
Bean calad	, σ	22	40.9	12	41	29.3	1.40 (0.70–2.79)	0.35	<del>Q</del>	
Cumber select	, (*	٧	20	18	57	31.6	1.58 (0.65–3.84)	0.36	Ð	
Tomato calad	י אי	· <del>-</del>	27.3	81	52	34.6	0.79 (0.28–2.22)	0.64	QN	
Assortment of chaeses	, \	21	28.6	15	42	35.7	0.80 (0.36-1.76)	0.57	<del>Q</del> Z	

<sup>a</sup> AR%, attack rates; RR, relative risks (crude food-specific RRs and adjusted food-specific RRs for food items significantly risk-associated in the univariate analyses); CI, confidence interval.

<sup>b</sup> ND, not done.

### DISCUSSION

The objectives of the outbreak investigation were to identify the source of the NV infection and the most likely reservoir and the pattern of spread of the pathogen. Analytical epidemiological investigation revealed that the ham roll was the most likely source of the NV in this outbreak. A kitchen assistant whose infant was sick with NV gastroenteritis 2 days before the pre-Christmas celebration did not apply measures appropriate for NV decontamination of hands and the household environment before she began food preparation for the party on 14 December. Because none of the kitchen staff was ill with NV gastroenteritis immediately before the outbreak, the kitchen assistant's infant remains the most likely reservoir of NV in this outbreak. The kitchen assistant probably acted as the vehicle by which NV was transferred from the household to the restaurant kitchen.

This mode of NV entry into a commercial kitchen was reported in an outbreak that occurred at a university cafeteria in Texas in 1998, for which case-control studies revealed that the NV infection was associated with consumption of foods from the university's main cafeteria deli (1). A gastroenteritis-free food handler who prepared sandwiches for lunch immediately before the outbreak reported that her infant had been sick with watery diarrhea since just before the outbreak. A stool sample from the infant was positive for NV by RT-PCR, and the sequence of the amplified products from deli ham specimens and the students' stool specimens. This was the first time RT-PCR and sequence analysis successfully confirmed NV spread from a food handler's household member into a commercial kitchen (1).

The findings of our investigation of the Austrian NV infection outbreak confirm that NV can enter into commercial kitchens even when none of the kitchen staff is ill from NV gastroenteritis. Although NV-infected food handlers are the most common reservoir of foodborne outbreaks, NV-infected household members of infection-free food handlers also must be considered a possible reservoir in foodborne outbreaks of NV infection. Food handlers who are caretakers of persons with gastrointestinal illness should be encouraged to pay particular attention to personal hygiene and appropriate environmental decontamination. Training of food handlers in the appropriate measures of hand hygiene and environmental disinfection at the work place and the home is essential to prevent food handler-related spread of NV.

The epidemiological investigation of the Austrian outbreak revealed a pattern of NV spread that had been documented previously only once (1). Food handlers should be informed about this possible pattern of NV spread. NV-infected household members of healthy food handlers must be considered a possible pathogen reservoir in foodborne NV infection outbreaks.

### REFERENCES

- Daniels, N. A., D. A. Bergmire-Sweat, K. J. Schwab, K. A. Hendricks, S. Reddy, S. M. Rowe, R. L. Fankhauser, S. S. Monroe, R. L. Atmar, R. I. Glass, and P. Mead. 2000. Foodborne outbreak of gastroenteritis associated with Norwalk-like viruses: first molecular trace back to deli sandwiches contaminated during preparation. J. Infect. Dis. 181:1467–1470.
- Graham, D. Y., X. Jian, T. Tanaka, A. R. Opekun, H. P. Madore, and M. K. Estes. 1994. Norwalk virus infection of volunteers: new insights based on improved assays. J. Infect. Dis. 170:34-43.
- Gray, G. W., J. L. Anderson, B. H. Keswick, P. C. Johnson, H. L. DuPont, S. E. Stine, and A. V. Bartlett. 1987. Norwalk virus antigen and antibody response in an adult volunteer study. J. Clin. Microbiol. 25:2001–2003.
- Grotto, I., M. Huerta, R. D. Balicer, T. Halperin, D. Cohen, N. Orr, and M. Gdalevich. 2004. An outbreak of Norovirus gastroenteritis on an Israeli military base. *Infection* 32:339-343.
- Hirakata, Y., K. Arisawa, O. Nishio, and O. Nakagomi. 2005. Multiprefectural spread of gastroenteritis outbreaks attributable to a single genogroup II norovirus strain from a tourist restaurant in Nagasaki, Japan. J. Clin. Microbiol. 43:1093–1098.
- Kroneman, A., H. Vennema, J. Harris, G. Reuter, C. H. von Bonsdorff, K. O. Hedlund, K. Vainio, V. Jackson, P. Pothier, J. Koch, E. Schreier, B. Böttiger, and M. Koopmans. 2006. Increase in norovirus activity reported in Europe. Euro. Surveill. 11. Available at: <a href="http://www.eurosurveillance.org/ViewArticle.aspx?PublicationType=W&Volume=11&Issue=50&OrderNumber=1">http://www.eurosurveillance.org/ViewArticle.aspx?PublicationType=W&Volume=11&Issue=50&OrderNumber=1</a>. Accessed 13 October 2008
- Lynch, M., J. Painter, R. Woodruff, and C. Braden. 2006. Surveillance for foodborne disease outbreaks—United States, 1998–2002. Morb. Mortal. Wkly. Rep. Surveill. Summ. 55:1–42.
- Miller, I., R. Gunson, and W. F. Carman. 2002. Norwalk like virus by light cycler PCR. J. Clin. Virol. 25:231–232.
- Schmid, D., H. P. Stüger, L. Lederer, A.-M. Pichler, G. Kainz-Arnfelser, E. Schreier, and F. Allerberger. 2007. A foodborne norovirus outbreak due to manually prepared salad, Austria 2006. *Infection* 35: 232-239.
- Treanor, J. J., and R. Dolin. 2005. Noroviruses and other caliciviruses, p. 2194–2201. In G. L. Mandell, J. E. Bennett, and R. Dolin (ed.), Principles and practice of infectious diseases, 6th ed. Elsevier Churchill Livingstone, Philadelphia.
- Turcios, R. M., M. A. Widdowson, A. C. Sulka, P. S. Mead, and R. I. Glass. 2006. Reevaluation of epidemiological criteria for identifying outbreaks of acute gastroenteritis due to norovirus: United States, 1998–2000. Clin. Infect. Dis. 42:964–969.
- Wu, H. M., M. Fornek, K. J. Schwab, A. R. Chapon, K. Gibson, E. Schwab, C. Spencer, and K. Henning. 2005. A norovirus outbreak at a long-term-care facility: the role of environmental surface contamination. *Infect. Control Hosp. Epidemiol.* 26:802-810.
- Wyatt, R. G., R. Dolin, N. R. Blacklow, H. L. Du Pont, R. F. Buscho, T. S. Thornhill, A. Z. Kapikian, and R. M. Chanock. 1974. Comparison of three agents of acute infectious gastroenteritis by virus challenge in volunteers. J. Infect. Dis. 129:709-714.