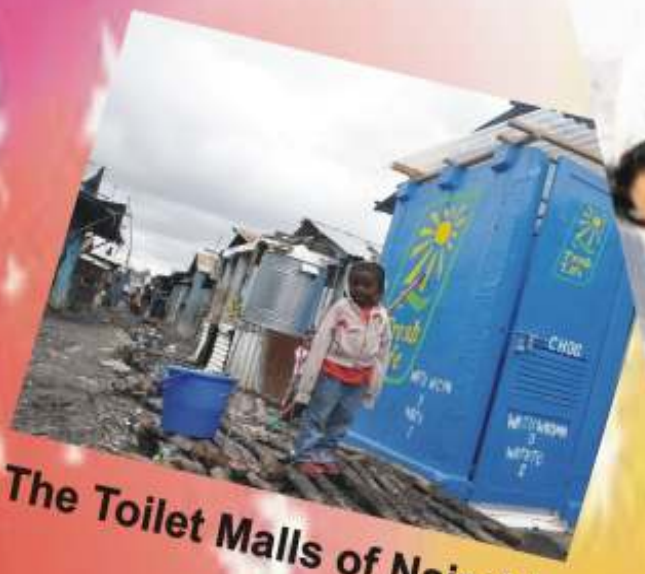
CHIEF EDITOR DR. SYED MUBIN AKHTAR

KARACHI PSYCHIATRIC HOSPITAL

BULLETIN (Psychiatric Research Articles) AUGUST-2014

Psychiatrists are receiving their gifts from Dr. Syed Mubin Akhtar



The Toilet Malls of Nairobi

Eid Mubarak

Patients are Celebrating EID with different personalities

KPH Staff singing songs with patients on EID Celebration

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Fruits and Vegetables Consumption and Risk of Stroke: A Meta-Analysis of Prospective Cohort Studies.

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Abstract

BACKGROUND AND PURPOSE:

We conducted a meta-analysis to summarize evidence from prospective cohort studies about the association of fruits and vegetables consumption with the risk of stroke.

METHODS:

Pertinent studies were identified by a search of Embase and PubMed databases to January 2014. Study-specific relative risks with 95% confidence intervals were pooled using a random-effects model. Dose-response relationship was assessed by restricted cubic spline.

RESULTS:

Twenty prospective cohort studies were included, involving 16 981 stroke events among 760 629 participants. The multivariable relative risk (95% confidence intervals) of stroke for the highest versus lowest category of total fruits and vegetables consumption was 0.79 (0.75-0.84), and the effect was 0.77 (0.71-0.84) for fruits consumption and 0.86 (0.79-0.93) for vegetables consumption. Subgroup and meta-regression showed that the inverse association of total fruits and vegetables consumption with the risk of stroke was consistent in subgroup analysis. Citrus fruits, apples/pears, and leafy vegetables might contribute to the protection. The linear dose-response relationship showed that the risk of stroke decreased by 32% (0.68 [0.56-0.82]) and 11% (0.89 [0.81-0.98]) for every 200 g per day increment in fruits consumption (P for nonlinearity=0.77) and vegetables consumption (P for nonlinearity=0.62), respectively.

CONCLUSIONS:

Fruits and vegetables consumption are inversely associated with the risk of stroke.

Serotonin syndrome

Serotonin syndrome is a potentially fatal and largely avoidable adverse drug reaction caused by serotonergic drugs. The steady increase in use of such drugs means all doctors need to be aware of what drugs increase serotonin and how to promptly recognise the syndrome and determine if it is potentially life threatening.

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What is serotonin syndrome?

Serotonin syndrome is a drug induced syndrome characterized by a cluster of dose related adverse effects that are due to increased serotonin concentrations in the central nervous system. It is also known as serotonin toxicity as it covers a spectrum from mild through to severe adverse effects depending, presumably, on the extent of increased serotonin.1 2 Severe toxicity usually occurs only with a combination of two or more serotonergic drugs (even when each is at a therapeutic dose), one of which is generally a monoamine oxidase inhibitor.1 3 Moderate toxicity has been reported with an overdose of a single drug and occasionally from increasing therapeutic doses.1 3 4 Its incidence is difficult to assess, but in large case series of overdoses, moderate serotonin toxicity occurred in 15% of poisonings with selective serotonin reuptake inhibitors (SSRIs).5 In the central nervous system, serotonin (5-hydroxytryptamine, 5-HT) is a neurotransmitter with many effects, including modification of mood, sleep, vomiting, and pain. Many drugs influence serotonergic neurotransmission, including some antidepressants, appetite suppressants, analgesics, sedatives, antipsychotics, anxiolytics, antimigraine drugs, and antiemetics.1 2

Severe or life threatening effects (rigidity and hyperthermia) seem to result only from stimulation of 5-HT2 receptors, and only drugs that generally increase serotonergic effects are expected to cause serotonin toxicity. Thus antipsychotics, anxiolytics, antimigraine drugs, and antiemetics, which are serotonin antagonists or have effects on other specific receptors (5-HT1A, 5-HT1D, 5-HT3), do not carry a significant risk of serotonin toxicity.1 4 6 Drug classes that are implicated in serotonin toxicity (see box 1) are largely restricted to serotonin precursors, serotonin agonists, drugs causing serotonin release, serotonin

reuptake inhibitors, and monoamine oxidase inhibitors.4 However, some drugs from other classes also have these effects, including some herbal medicines (box 1). A few drug interactions are clearly linked to apparently classic cases of serotonin toxicity where the mechanism remains unclear.7 These drugs generally have effects on other neurotransmitters and may have secondary effects on serotonin release or reuptake.

How does it present?

Serotonin toxicity starts within hours of ingesting drug(s) that cause an increase in serotonin. The classic triad of clinical features are neuromuscular excitation (such as clonus, hyperreflexia, myoclonus, rigidity), autonomic nervous system excitation (such as hyperthermia, tachycardia), and altered mental state (such as agitation, confusion) (fig 1□). The acute onset of these features should trigger a search for a toxic explanation (along with consideration of other conditions such as alcohol or drug withdrawal, non-convulsive seizures, and encephalitis). Although case series showed moderate serotonin toxicity occurred in 15% of SSRI overdoses, there were no severe cases.5 Serotonin toxicity did not occur in overdoses of the reversible monoamine oxidase inhibitor moclobemide alone. However, if a second serotonergic drug was ingested, serotonin toxicity was nearly always present and was severe in about half of these cases.

Box 1: Drugs that have been associated with moderate to severe serotonin toxicity*

Monoamine oxidase inhibitors

- Irreversible inhibitors—Phenelzine, tranylcypromine, iproniazid, isocarboxazid
- Reversible inhibitors of monoamine oxidase A—Moclobemide
- Non-psychotropic drugs—Linezolid, methylene blue (methylthioninium chloride)

Serotonin releasing agents

- Fenfluramine, sibutramine
- Amphetamine, methamphetamine, methylphenidate, phentermine
- Synthetic stimulants—Ecstasy, “bath salts” (cathinones, phenylethylamines)
- Serotonin reuptake inhibitors
- Selective serotonin reuptake inhibitors—Fluoxetine, fluvoxamine, paroxetine, citalopram, sertraline, escitalopram
- Serotonin-noradrenaline reuptake inhibitors—Venlafaxine, desvenlafaxine, duloxetine
- Tricyclic antidepressants—Clomipramine, imipramine
- Opioid analgesics—Pethidine, tramadol, fentanyl, dextromethorphan
- St John’s wort (*Hypericum perforatum*)

Miscellaneous

- Lithium
- Tryptophan
- Buspirone

*Severe serotonin toxicity generally involves a combination of agents from different drug classes3 4 8-12

How do we diagnose it?

The diagnosis of serotonin syndrome is clinical, and is plausible only in the setting of starting or increasing the dose (or overdose) of a potent serotonergic drug, or shortly after a second serotonergic drug is added leading to a drug interaction. Difficulties sometimes arise in identifying contributing agents because some drugs have persistent activity (irreversible monoamine oxidase inhibitors) or long half lives (fluoxetine) and may have been stopped weeks earlier. There should be a careful history of illicit drug use (stimulants such as cathinones and other synthetic stimulants, ecstasy, amphetamines, or cocaine) and of herbal medicines (such as St John's wort, ginseng, tryptophan, and pharmaceutical adulterants in appetite suppressants). Serotonergic actions of drugs that are not marketed as serotonergic (such as tramadol, fentanyl, linezolid, and methylene blue) are another trap for the unwary (see box 1). Some pathognomonic features of serotonin syndrome and combinations of clinical signs are rarely seen in other conditions, and, with a supporting drug history, these can allow a confident diagnosis. The classic features in the diagnosis are generalized clonus (inducible, spontaneous, ocular), and these form the key components of the Hunter serotonin toxicity criteria, which have been validated and can be used to confirm the diagnosis of moderate or severe toxicity (fig 2□).13 Clonus is usually most marked, and easily elicited, with ankle dorsiflexion: spontaneous clonus differs from rapid myoclonic jerks by being rhythmic, large muscle contractions, and is often triggered by minor movements or vibrations. The term ocular clonus covers a range of abnormal involuntary movements that involve fine or coarse oscillations of gaze in all directions (examples at http://curriculum.toxicology/wikispaces.net/Serotonin_video).16 These can be continuous or triggered by rapid eye movement. Other abnormal eye movements such as "ping pong gaze" (short cycle, periodic, alternating lateral gaze) may also be seen. Severe serotonin toxicity is characterised by a rapidly rising temperature and rigidity and is again diagnosed on clinical grounds. Investigations are not of diagnostic value, except to diagnose complications (such as effects of hyperthermia—disseminated intravascular coagulation, multiorgan failure, rhabdomyolysis), other drug effects in overdose (electrocardiographic changes), or to exclude other diagnoses such as encephalitis or cerebral vasculitis (most commonly head scans, electroencephalography, lumbar puncture). Among patients also

taking antipsychotic drugs it may be necessary to differentiate from neuroleptic malignant syndrome. The key differentiating features are that neuroleptic malignant syndrome is of relatively slow onset over days, and marked by extrapyramidal features and rigidity, but clonus is not a feature. A different problem relates to mild serotonin toxicity, which can be difficult to distinguish from many medical conditions or other adverse drug effects. Patients taking therapeutic SSRIs commonly have features such as lower limb hyperreflexia or a few beats of ankle clonus without toxicity. A diagnosis of mild serotonin syndrome may be tempting for any febrile, tachycardic, agitated, or confused person taking psychiatric drugs (there are many reports along these lines quoting the presence of non-specific "Sternbach criteria"14 but without the classic features of the Hunter serotonin toxicity criteria13). A diagnosis of adverse reactions to serotonergic drugs in such circumstances is largely a presumptive diagnosis after exclusion of other explanations and is possible only for drugs that are known to increase serotonin (both criteria are often ignored but explicitly specified as necessary in the Sternbach criteria14). The diagnosis is further supported by resolution on stopping serotonergic drugs, but whether the mechanism is mild serotonin toxicity or some other drug effect in such cases is moot. Mild serotonergic adverse effects in therapeutic use will not progress to severe toxicity in the absence of dose escalation or drug interactions. For some patients with a good therapeutic response, continuation of the drug at the same or a lower dose may be justifiable.

How can we treat it?

Serotonin syndrome in mild to moderate cases usually resolves in one to three days after stopping the serotonergic drugs. Severe toxicity is a medical emergency and may be complicated by severe hyperthermia, rhabdomyolysis, disseminated intravascular PRACTICE coagulation, and adult respiratory distress syndrome,17 and thus requires intensive supportive care. Supportive care largely consists of sedation as required. Ensuring adequate hydration and careful monitoring of temperature, pulse, blood pressure, and urine output are necessary. Preventing hyperthermia and subsequent multiorgan failure is a key goal in severe serotonin toxicity. In animal models lowering temperature also indirectly down regulated 5HT2A receptors in the central nervous system and reduced serotonin levels.2 Sedation to reduce muscle hyperactivity (such as midazolam infusion or oral diazepam), active cooling (fans with water sprays, ice packs, or cooling blankets), and even paralysis and ventilation may be useful in severe cases. Serotonin antagonists and in particular 5HT2A receptor antagonists reduce hyperthermia and other severe manifestations in animal studies.1 2 8 For severe serotonin toxicity, intravenous chlorpromazine is the most commonly used serotonin antagonist, but intravenous fluid loading is essential to prevent hypotension.8 Oral cyproheptadine has been used to treat moderate serotonin toxicity, with doses of 8-16 mg up to a daily maximum of 32mg. Whether its sedative or serotonin

antagonist effects are more important remains unclear. In moderate serotonin toxicity agitation is generally the most troublesome symptom, and sedation with oral diazepam may be all that is required. There are no clinical trials or other strong evidence supporting any of the above approaches to treatment,8 but recovery is usual and mortality low (<1%) when such approaches have been applied.5 15

How can we prevent it?

Several systematic reviews clarify the extent to which severe serotonin syndrome may result from drug interactions.3 4 6 9-11 However, spurious associations and cautions have proliferated elsewhere in the medical literature, and product information is a major impediment to sensible decision support in this area. Clinicians prescribing an SSRI (and their patients) can expect to be warned of up to 1000 interacting drugs (for example, on www.drugs.com/), with hundreds of these warning of “rare but serious” serotonin syndrome. Interactions of an SSRI with any monoamine oxidase inhibitor might be lethal and should be avoided at all cost. However, interactions with other serotonin reuptake inhibitors are likely to be minor (additive effect), and interactions with serotonin releasing agents (such as amphetamines) might even attenuate toxicity.18 Further, many listed interactions—such as with carbamazepine, most tricyclic and atypical antidepressants,4 12 and triptans6—have little or no evidence to support the contention that serotonergic effects are increased by coadministration. However, clomipramine and imipramine are much more serotonergic than other tricyclic antidepressants and have caused serotonin toxicity. An awareness of drugs with potent serotonergic effects is the key to preventing drug interactions. It is apparent from systematic reviews of case reports3 4 6 9-11 that nearly all severe serotonin syndromes involve a monoamine oxidase inhibitor, and the relatively small number of these can easily be committed to memory (box 1). Washout periods should be observed when switching antidepressants. If possible avoid the use of serotonergic drugs for non-psychiatric conditions (such as tramadol for analgesia). Patients also need to be aware of the potential for serious drug interactions, especially given the existence of over the counter drugs and herbal medicines with serotonergic activity (box 1). Some individuals seem to be more susceptible, but it is unclear if pharmacokinetic (such as decreased drug metabolism) or pharmacodynamic (such as serotonin receptor polymorphism) differences explain this, and strong consistent pharmacogenetic associations have not been found.19 No evidence has been found to support theories that potent dietary monoamine oxidase inhibitor compounds are a cause of serotonin toxicity in highly susceptible individuals.

Multidisciplinary Approach Lessens Tinnitus Severity

Bruce Soloway, MD, Jonathan Silver, MD reviewing Cima RFF et al. Lancet 2012 May 26.

Outcomes were better when cognitive-behavioral therapy was added to audiological therapies.

Tinnitus is common and potentially debilitating; treatment is not standardized and often is fragmented and poorly coordinated. Audiological therapies, such as habituation to an external sound generator, often are employed, and cognitive-behavioral therapy (CBT) has been proposed as a treatment. Neither approach has been evaluated rigorously.

Netherlands researchers surveyed audiological referral centers to define a "usual care" protocol that consisted primarily of audiological interventions, with an added social work component when indicated. They then randomized 492 patients with tinnitus to this usual-care protocol or to a multidisciplinary approach. In the latter approach, audiological interventions were enhanced with CBT-based educational sessions and, when indicated, supplemented by treatment that involved clinical psychologists, social workers, and movement, physical, and speech therapists — usually in a group setting.

Outcomes were measured using standard scales at 3 months (after usual or enhanced audiological treatment), 8 months (after adding social work intervention or interdisciplinary treatment as needed), and 12 months (after 4 months of no contact). Compared with patients in the usual-care group, those assigned to multidisciplinary treatment had significantly less tinnitus severity and impairment at all three time points and significantly better health-related quality of life at 8 and 12 months.

Case Managers in Primary Care: The Case for Collaborative Care

Martin T. Stein, MD reviewing Kolko DJ et al. Pediatrics 2014 Apr.

Case managers can improve the care of children with behavioral problems.

An essential component of the medical home in a primary care pediatric practice is the case manager (CM), who coordinates the care of children with chronic conditions. However, few pediatric offices have CMs on staff, and studies on CMs in primary care are limited. To assess an integrated collaborative care–CM model, researchers randomized eight pediatric practices to provide 6 months of "doctor office collaborative care" (DOCC) or enhanced usual care to children with externalizing behavioral problems.

CM-directed behavioral screening included a standardized, parent-completed checklist and clinical interviews. Of 787 referred children, 321 children (mean age, 8 years) with attention-deficit/hyperactivity disorder (64%) or disruptive behavior disorders (41%) were included; 16% had co-existing anxiety disorders. In DOCC, CMs delivered personalized, evidence-based interventions in collaboration with primary care practitioners. Usual care involved psychoeducation and a facilitated referral to specialty care.

Compared with usual care, DOCC was significantly associated with more initiation of treatment (99% vs. 54%); treatment completion (77% vs. 12%); and improvement in disruptive behaviors, hyperactivity, internalizing problems, and parents' self-reported stress. Most differences were sustained at 18-month follow-up. Perceived practice change, efficacy, and treatment skill were reported more frequently by pediatricians in the DOCC group.

COMMENT

This study demonstrates that on-site case managers working collaboratively with primary care pediatric practices improve quality of care for school-age children with behavioral conditions. When I practiced general pediatrics, I often fantasized about working collaboratively with case managers, not only for patients with behavior problems, but also for those with asthma, obesity, school underachievement, or less common conditions. Then, as now, reimbursement patterns prevented such programs, but initiatives in the Affordable Care Act may make case managers part of all primary care practices

Trimel Pharmaceuticals Announces Positive Phase II Results for Tefina™
Women treated with Tefina™ 0.6 mg reported a statistically significant increase in orgasms versus placebo

TORONTO, May 28, 2014 /PRNewswire/ -- Trimel Pharmaceuticals Corporation (TSX: TRL) announced today top-line results of its Phase II clinical trial evaluating the efficacy and safety of Tefina™, a "use-as-required" testosterone nasal gel for the treatment of Female Orgasmic Disorder (FOD). FOD, also known as anorgasmia, is characterized by a delay, absence or reduced intensity of orgasm, causing clinically significant distress.

The double-blind, placebo-controlled study enrolled 253 pre- and post-menopausal women experiencing acquired FOD in the United States, Canada and Australia. Participants were randomized to one of three dosage strengths (0.6 mg, 1.2 mg, 1.8 mg) or a placebo group and treated over the course of 84 days. The primary endpoint of the study was to compare the effects of the three dose strengths of Tefina™ nasal testosterone gel to placebo on the occurrence of orgasm. Secondary endpoints included the change from baseline in distress due to orgasmic disorder, change in sexual functioning and sexual event satisfaction. Safety and tolerability were also assessed.

Tefina™ 0.6 mg led to a statistically significant increase in the average number of orgasms during the 84-day treatment period of 2.3 versus 1.7 for the placebo arm (p=0.0015). In addition, improvements in all of the secondary endpoints were observed; however, further analysis is underway to assess statistical significance. Tefina™ was found to be well-tolerated with no reported serious adverse events.

"Female Orgasmic Disorder is the second most prevalent sexual disorder affecting women. Approximately one in five women report difficulty with orgasm and one quarter of these show marked distress, a key criterion in a clinical diagnosis," said Dr. Sheryl Kingsberg, the U.S. principal investigator for the Tefina™ Phase II clinical trial, chief of behavioral medicine at University Hospitals Case Medical Center and professor of reproductive biology and psychiatry at Case Western Reserve University in Cleveland, Ohio. "Currently, there are no approved pharmacological treatment options, leaving an unmet need that Tefina™ hopes to remedy."

"These results mark an important milestone in the development of Tefina™," said Tom Rossi, Trimel President and CEO. "They provide further evidence that Tefina™ could represent an important treatment option for the many women who suffer from this disorder. On behalf of Trimel and its various stakeholders, I am extremely excited about this positive outcome and look forward to advancing this product towards commercialization."

Investor Event

Trimel is pleased to announce that it will host an Investor Event on Thursday, June 12, 2014, from 12:30 - 3:00 p.m. (Eastern Daylight Time) to provide a more in-depth review of the Tefina™ Phase II study results. Dr. Kingsberg, U.S. principal investigator for the Tefina™ Phase II clinical trial, will be the keynote speaker at the event. The Investor Event will be held at the Toronto Region Board of Trade, First Canadian Place, Suite 350, 77 Adelaide Street West, Toronto, Ontario and by webcast. For more information or to register for the event, contact rachael.factor@fleishman.ca or visit http://trimelpharmaceuticals.com/Investors/Investor-Day. In-person space is limited.

About Female Orgasmic Disorder

Female Orgasmic Disorder, also known as anorgasmia, is characterized by a marked reduced intensity of orgasmic sensations, or marked delay in, marked infrequency of, or absence of, orgasm that has persisted for a minimum duration of approximately six months, and causes clinically significant distress in the individual. The diagnosis is further specified by whether the dysfunction has been lifelong or acquired. Currently, there are no approved treatments for Female Orgasmic Disorder.

About Trimel

Trimel is a specialty pharmaceutical company actively developing medications for male hypogonadism, female sexual dysfunction and various respiratory disorders. A New Drug Application for CompleoTRT™, a product utilizing Trimel's licensed nasal gel technology, has been filed with the United States Food and Drug Administration and is awaiting regulatory approval. For more information, please visitwww.trimelpharmaceuticals.com.

Notice regarding forward-looking statements:

Information in this press release that is not current or historical factual information may constitute forward-looking information within the meaning of securities laws. Implicit in this information are assumptions regarding our future operational results. These assumptions, although considered reasonable by the company at the time of preparation, may prove to be incorrect. Readers are cautioned that actual performance of the company is subject to a number of risks and uncertainties and could differ materially from what is currently expected as set out above. For more exhaustive information on these risks and uncertainties you should refer to our annual information form dated March 5, 2014 which is available at www.sedar.com. Forward-looking information contained in this press release is based on our current estimates, expectations and projections, which we believe are reasonable as of the current date. You should not place undue importance on forward-looking information and should not rely upon this information as of any other date. While we may elect to, we are under no obligation and do not undertake to update this information at any particular time, whether as a result of new information, future events or otherwise, except as required by applicable securities law.

United Condemnations

How they vote at the U.N.!

Below are the actual voting records of various Arabic/Islamic States which are recorded in both the US State Department and United Nations records:

Kuwait votes against the United States 67% of the time.
Qatar votes against the United States 67% of the time.
Morocco votes against the United States 70% of the time.
United Arab Emirates votes against the U. S. 70% of the time.
Jordan votes against the United States 71% of the time.
Tunisia votes against the United States 71% of the time.
Saudi Arabia votes against the United States 73% of the time.
Yemen votes against the United States 74% of the time.
Algeria votes against the United States 74% of the time.
Oman votes against the United States 74% of the time.
Sudan votes against the United States 75% of the time.
Pakistan votes against the United States 75% of the time.
Libya votes against the United States 76% of the time.
Egypt votes against the United States 79% of the time.
Lebanon votes against the United States 80% of the time.
India votes against the United States 81% of the time.
Syria votes against the United States 84% of the time.
Mauritania votes against the United States 87% of the time.

US Foreign Aid to those that hate us:

Egypt, for example, after voting 79% of the time against the United States, still receives $2 billion annually in US Foreign Aid.

Jordan votes 71% against the United States and receives $192,814,000 annually in US Foreign Aid.

Pakistan votes 75% against the United States receives $6,721,000 annually in US Foreign Aid.

India votes 81% against the United States receives $143,699,000 annually

Perhaps it is time to get out of the UN and give the tax savings back to the American workers who are having to skimp and sacrifice to pay the taxes.

Pass it along. Everyone needs to know this. Might even mention it to your congressman, who knows this anyway... what a disgrace... no wonder the world has no respect for us.

Origins: This is one of those items that seems simple enough to verify at first blush, but proves quite difficult in practice.

First of all, we have to consider what our parameters are:

- Are we measuring the voting records of the named countries across the entire six-decade history of the United Nations, or only from some subset of that period?

- Which votes are we counting — just those of the General Assembly, or also those of the Main Committees and the Security Council?

Even deciding that we're only going to consider the postions various countries took on resolutions presented to the General Assembly during a specified time period still makes compiling an accurate tally difficult, because:

- The majority of General Assembly resolutions are adopted without a vote.

- Unless a recorded vote is specifically requested before a resolution is voted upon, the U.N. makes available a voting summary which provides only a tally of the final vote, not a listing of how individual Member States voted.

Once we narrow our focus to resolutions submitted to a recorded vote, we still have some thorny issues to consider:

- Nearly every resolution ends up with some Member States either abstaining or failing to vote on it. When countries abstain from voting on a resolution which the U.S. either supports or opposes, are those countries to be regarded as voting against the U.S. (because they failed to support its vote), or are they to be considered as neutral parties neither for nor against the U.S.?

- Quite often U.N. votes address the issue of whether a single paragraph (or even just a few words) in the draft of a resolution should be changed or omitted. When the U.S.

otherwise supports a resolution but seeks to change some of its wording, are other countries to be regarded as voting against the U.S. if they do not also vote in favor of the alterations?

Since we had to start somewhere, we tallied the recorded votes for all resolutions put before the General Assembly so far during the current session, running from October 2003 to mid-April 2004. We counted all votes, whether they involved adopting resolutions as a whole or making alterations to draft resolutions. When countries abstained or otherwise failed to vote, we counted them as voting neither for nor against the U.S. Likewise, when the U.S. abstained from voting on resolutions, we did not include other countries' votes on those resolutions in our totals.

The results of this tally were even worse (from a U.S. perspective) than the message quoted above indicates, with the countries named voting contrary to the U.S. position on U.N. resolutions an aggregate 88% of the time. (Even though India is neither Arab nor particularly Islamic, we included it in our chart because the widely-circulated e-mailed list did.)

Country	**Times Voted With U.S.**	**Times Voted Against U.S.**	**% of Votes Against U.S.**
Kuwait	10	61	86%
Qatar	9	64	88%
Morocco	8	62	89%
United Arab Emirates	8	61	88%
Jordan	9	64	88%
Tunisia	8	63	89%
Saudi Arabia	7	62	90%
Yemen	9	64	88%
Algeria	9	63	88%

Oman	9	63	88%
Sudan	10	60	86%
Pakistan	9	59	87%
Libya	8	63	89%
Egypt	10	63	86%
Lebanon	7	62	90%
India	14	52	79%
Syria	7	59	89%
Mauritania	7	63	90%

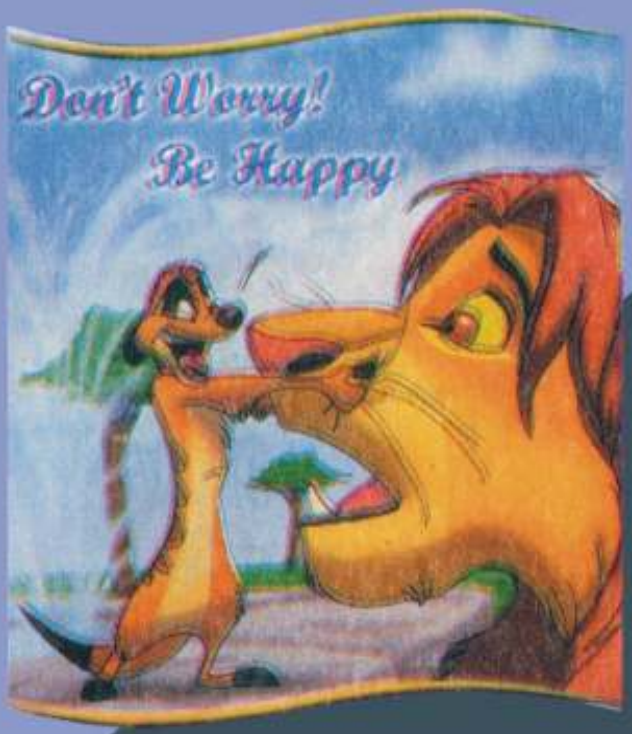
However, we also surveyed the U.N. voting records of several countries generally considered to be close allies of the U.S., and those results were none too impressive either. Only Israel consistently voted with the U.S.:

Country	**Times Voted With U.S.**	**Times Voted Against U.S.**	**% of Votes Against U.S.**
Australia	33	26	44%
Canada	31	32	51%
Israel	56	7	11%
Japan	26	36	58%
United Kingdom	40	27	40%
France	36	31	46%

How much significance one should place in these figures is problematic, because most other U.N. Member States have records of voting against the U.S. that are equally as bad as the records of the countries named in the message above. U.N. votes on resolutions are frequently lopsided, pitting a single nation or a handful of nations against all the others, and more often than not the U.S. is the one nation at odds with the rest of the world. Of the 83 resolutions we surveyed for our informal tally, in ten cases the U.S. was the only Member State to vote against them, and in five cases only one other nation joined the U.S. in voting against them. In fact, in over half the total cases (42 out of 83), the U.S. was supported by five or fewer Member States in voting against a U.N. resolution. So it isn't just the Arab/Islamic states who consistently vote against the U.S. in the United Nations — pretty much the rest of the world does, too.

Patients are playing cards.

Female Ward Unit incharge is checking patient's handicraft work.



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