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REVIEW

Indoor air pollution and respiratory health of children in the developing world

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Abstract

Indoor air pollution (IAP) is a key contributor to the global burden of disease mainly in developing countries. The use of solid fuel for cooking and heating is the main source of IAP in developing countries, accounting for an estimated 3.5 million deaths and 4.5% of Disability-Adjusted Life Years in 2010. Other sources of IAP include indoor smoking, infiltration of pollutants from outdoor sources and substances emitted from an array of human utilities and biological materials. Children are among the most vulnerable groups for adverse effects of IAP. The respiratory system is a primary target of air pollutants resulting in a wide range of acute and chronic effects. The spectrum of respiratory adverse effects ranges from mild subclinical changes and mild symptoms to life threatening conditions and even death. However, IAP is a modifiable risk factor having potential mitigating interventions. Possible interventions range from simple behavior change to structural changes and from shifting of unclean cooking fuel to clean cooking fuel. Shifting from use of solid fuel to clean fuel invariably reduces household air pollution in developing countries, but such a change is challenging. This review aims to summarize the available information on IAP exposure during childhood and its effects on respiratory health in developing countries. It specifically discusses the common sources of IAP, susceptibility of children to air pollution, mechanisms of action, common respiratory conditions, preventive and mitigating strategies.

Key words: Indoor air pollution; Air pollution; Respiratory health; Children; Developing countries

Core tip: Indoor air pollution (IAP) is a key contributor to the burden of disease in developing countries; use of solid fuel for cooking and heating is the main source of IAP. Children are among the most vulnerable groups for adverse effects of IAP. The respiratory system is a primary target of air pollutants resulting in a wide range of acute and chronic effects. The spectrum of respiratory adverse effects ranges from mild subclinical changes and mild symptoms to life threatening conditions, and even death. This review summarizes the available information on IAP exposure during childhood and its effects on respiratory health in developing countries.

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INTRODUCTION

Indoor air pollution (IAP) is a key contributor to the

global burden of disease[1,2]. There is convincing evidence of the adverse effects of IAP on human health, children being one of the most vulnerable groups[3-5]. The respiratory system is a primary target for air pollutants resulting in a wide range of acute and chronic effects[6].

Several reasons are attributed to children's high susceptibility to the harmful effects of air pollution; the growth of airways and alveoli of the respiratory system are guided through a complex chemical pathway and air pollutants are known to interfere with these pathways[4]. The airway epithelium of growing children is more permeable to air pollutants and the lung defence system is not adequately evolved. Children have a differential ability to metabolize, detoxify, and excrete environmental agents thereby making them prone to more harm[7]. A higher resting metabolic rate of oxygen consumption per unit body weight in children due to the larger surface area per unit body weight and rapid growth, as compared to adults, makes them more vulnerable. Further, children engage in more physical activity than adults which leads to a higher intake of air relative to body size[4,8].

This article is an overview of the major sources of IAP exposure in households and associated respiratory health effects in children in developing countries; we do not discuss specific pollutants and their associated effects *per se*.

SOURCES AND CONSTITUENTS OF INDOOR AIR POLLUTION

The sources, pollutant types and extent of IAP are a result of complex interactions between structures, building systems, source strength, removal and deposition rates, indoor mixing and chemical reactions, furnishings, the outdoor environment and source strength, and practices and behaviours of the inhabitants[9-12]. Thus, variations are seen at different levels (*i.e.*, developed *vs* developing countries; between developing countries; between cities/regions of a country; and between households).

Solid fuel

Fuels used for cooking and heating purposes may be solid or non-solid. Solid fuel can be biomass or coal; biomass fuel includes wood (unprocessed and charcoal), dung or crop residues[13]. The types of solid fuels used vary by country. For example, coal is commonly used in China[14], but not in Sri Lanka[15]. Solid fuels emit a complex mixture of pollutants with more than 200 chemicals and compound groups[13]. The composition differs by type of solid fuel. Common pollutants include inorganic gases (*e.g.*, CO, NO_2, O_3 *etc.*), particulate matter (PM), hydrocarbons (*e.g.*, polycyclic aromatic, monoaromatic such as benzene *etc.*), oxygenated organic compounds (*e.g.*, aldehydes, phenols *etc.*), chlorinated organic compounds (*e.g.*, methylene chloride *etc.*) and free radicals[16, 17].

Solid fuel use for cooking and heating is the main source of IAP in developing countries[17]. PM less than 2.5 μm in diameter ($PM_{2.5}$) is one of the most hazardous pollutants emitted by solid fuels; its concentration increases to milligrams per cubic meter inside kitchens during cooking[18-20] well above the World Health Organization (WHO) guidelines of 25 $\mu g/m^3$ (24 h average)[21].

The use of solid fuel varies across WHO regions: 77%, 74%, 74% and 36% of households use solid fuels in Sub-Saharan Africa, in South-East Asia, in the Western Pacific Region and in the Eastern Mediterranean Region, respectively, as compared to 16% of households in Latin America, Caribbean, and Central and Eastern Europe[22].

Secondhand smoke

Indoor tobacco smoking is another important source of IAP which exposes non-smokers to tobacco smoke. Secondhand smoke (SHS) comprises a mixture of mainstream smoke (smoke first inhaled by an active smoker and then exhaled) and side-stream smoke (smoke emitted between puffs). Side-stream smoke accounts for about 85% of total SHS, the rest comprising mainstream smoke[23]. SHS is a complex mixture of more than 4000 chemicals, of which, more than 40 are identified carcinogens in vapor and particle phases[24]. The vapor-phase compounds include benzene, vinyl chloride, acrolein *etc.* The particulate-phase chemicals include alkaloids, nicotine and its derivatives, aromatic amines, polycyclic aromatic hydrocarbons, *etc*[25]. SHS can be more carcinogenic than mainstream smoke inhaled by active smokers[23].

Outdoor sources

With rapid urbanization and industrialization, ambient air pollutant concentrations in many cities in developing countries far exceed current WHO air quality guidelines[26]. Globally, 32% of the population live in areas exceeding WHO Level 1 interim threshold of 35 $\mu g/m^3$ largely encompassing cities in South and East Asia[27]. Most primary pollutants typically have steep decreasing gradients with distance from roads. In general, the highest exposures are found within the first 50-100 m from roadways, and exposures often fall to background levels by 300-500 m[28,29]. Interconnected high traffic roads in an area would have a high background pollution level. Pollutants with high outdoor concentrations infiltrate indoors. Infiltration and trapping of pollutants vary depending on the local topography and the configuration of buildings[29]. A study in urban and rural areas of Bangladesh found that mean concentrations of CO, CO_2, dust particles, and major volatile organic compounds (VOC) were significantly higher in urban biomass fuel using kitchens as compared to rural counterparts[30]. In Sri Lanka, indoor NO_2, SO_2 and $PM_{2.5}$ levels were high in living rooms of houses in a traffic congested urban area as compared to living rooms of houses using clean fuels in a semi-urban area[31].

Other sources

A spectrum of biological pollutants is released from dust mites, molds, fungi, bacteria, pests (cockroaches, mice, rats) and also from byproducts of men and pets.

These pollutants may release microbial products such as endotoxins, microbial fragments, peptidoglycans and various types of allergens[10,32,33]. Several common products used in households may also release pollutants. Such products include personal care products, household products such as finishes, rug and oven cleaners, paints and lacquers, paint strippers, pesticides, mosquito repellants, dry-cleaning fluids, building materials, and home furnishings *etc*. Kerosene is considered as a cleaner alternative for solid fuels, biomass and coal[34], though it still is low in the energy ladder[35]. Kerosene use for cooking and lighting remains widespread in developing countries[34] especially in urban settings where biomass is not freely accessible[15]. It has been shown that the kerosene stoves and devices emit substantial amounts of $PM_{2.5}$, CO, NO_2 and SO_2[34]. Uranium-bearing soil releases radon which may aggregate in poorly ventilated or closed indoor air environments[36,37]. Very fine asbestos fibers may be released into the air when asbestos containing material such as roof sheets, insulation for heating systems *etc*. are used.

RESPIRATORY HEALTH

Mechanisms

Air pollutants act on one or more host defence mechanisms against pathogens in the respiratory tract. Many pollutants act together in a cascade of partly interrelated biological mechanisms. The biological mechanisms include triggering oxidative stress, both local and systemic inflammation, reduction of the mucociliary clearance, increased reactivity of the respiratory epithelia, reduction of the macrophage responses to microorganisms, increased epithelial permeability and adhesion to microorganisms, bronchial irritation *etc*[38-41]. These mechanisms differ by type of pollutant and extent of exposure[17,42]. PM_{10} emitted from a mixture of fossil and biomass fuels have been reported to enhance the capacity of pneumococci to adhere to human lower airway cells *in vitro*[43]. Several animal studies provide evidence of biological mechanisms of adverse respiratory health effects of air pollution. Hsu *et al*[44] reported that guinea pigs produce broncho-constriction when they were exposed to wood smoke, and the response increased with subsequent exposures. Rats intermittently exposed to wood smoke for 75 min daily for 15 d showed signs of mononuclear bronchiolitis and mild emphysema, while more severe conditions were reported when the exposure was extended to 30 d and 45 d[45]. Exposure of mice to cigarette smoke and subsequent infection with *Streptococcus pneumonie*, increased numbers of bacteria in lung tissue and more severe clinical signs of sepsis were reported as compared to unexposed mice[46]. Instillation of fossil-fuel-derived PM into the lower airways of mice impaired phagocytosis and clearance of *Streptococcus pneumonie*[47]. Harrod *et al*[48] exposed the mice to diesel engine emissions and subsequently infected them with *Pseudomonas aeruginosa*. The number of both ciliated and non-ciliated airway epithelial cells was reduced during the infection in a concentration-dependent manner with increased lung pathogenesis. Although some of these studies were not based on pollutants originating from indoor sources, it is reasonable to believe that a similar pathology exists for IAP sources as well.

Burden of disease

IAP in developing countries is disproportionately high as compared to developed countries due to differences in sources of IAP, the main difference being the use of solid fuels for cooking and heating purposes by the majority of the population in developing countries. In fact, 76% of all global PM pollution occurs indoors in the developing world[49]. IAP from combustion of solid fuels for cooking and space heating is one of the ten most important contributors to the global burden of disease; it is estimated to have resulted in 3.5 million deaths and 4.5% of Disability-Adjusted Life Years (DALYs) in 2010[2]. It is estimated that about 3 billion of the world's population and up to 90% of rural households in developing countries use solid fuel as the main source of energy for cooking and heating[1,50].

Almost 152 million new episodes of "clinical pneumonia" are reported from developing countries while only 4 million episodes are reported from developed countries. In this estimation, "clinical pneumonia" included episodes of pneumonia, bronchiolitis and reactive air way diseases associated with respiratory tract infections[51]. The South-East Asia Region reports the highest number of episodes of clinical pneumonia (0.36 episodes per child-year). More than half of the new pneumonia episodes are reported from India (43 million), China (21 million) and Pakistan (10 million) and 6 million each from Bangladesh, Indonesia and Nigeria[51,52]

SHS is a common source of IAP in both the developed and the developing world[53]. Children are exposed more to SHS than other age-groups as they are unable to avoid the exposure specifically when their close relatives are smoking at home. About 40% of children are exposed to SHS worldwide and an estimated 165000 children under 5 years die each year from lower respiratory infections caused by exposure to SHS. Two-thirds of these deaths occur in developing countries of Africa and South Asia[54]. The largest contribution to DALYs due to SHS exposure is from children[54].

Epidemiological evidence

Acute respiratory infections: Acute respiratory infections (ARI) can be classified into acute upper respiratory infections (AURI) and acute lower respiratory infections (ALRI) depending on the area of the respiratory tract that is affected. The upper respiratory tract consists of the airways from the nostrils to the vocal cords in the larynx, including the paranasal sinuses and the middle ear. The lower respiratory tract includes the continuation of the airways from the trachea and bronchi to the bronchioles and the alveoli[55]. AURIs are usually mild in nature and often caused by viruses, though some cases of sinusitis and otitis media may be caused by virulent bacteria.

The majority of ARI deaths and severe illness episodes are due to ALRIs, consisting mainly of pneumonia[55]. ARIs do not confine themselves to the respiratory tract and may have systemic effects due to the possible extension of infection or microbial toxins to other organs, inflammation, and reduced lung function[55]. Risk factors for ALRIs include malnutrition, low birth weight (≤ 2500 g), non-exclusive breast feeding (during the first 4 mo of life), non-immunized for measles within the first 12 mo of life, IAP and crowding[55].

A large number of epidemiological studies have demonstrated the association between ARI and IAP, though the estimations of associations vary in magnitude and consistency across individual studies[56,57] and meta-analyses[58-60]. This heterogeneity may be due to differences in the definition of ARIs, duration of exposure measurements, relative distribution of varying household characteristics including ventilation, stove types, kitchen amenities, country and/or climate, age group studied and relative extent of exposure to other sources of IAP such as indoor smoking *etc*[61].

A recent meta-analysis of eight studies found that children were more than three times likely to have ARIs when exposed to solid biomass fuel smoke as compared to non-exposed children (OR = 3.52; 95%CI: 1.94-6.43)[59]. Based on 24 studies, the overall pooled odds ratio was 1.78 (95%CI: 1.45-2.18) for pneumonia among the under-five children exposed to IAP due to solid fuel use as compared to non-exposed children of the same age group[60]. Based on eight studies conducted prior to year 2000, the estimated risk of IAP due to solid biomass fuel use for ALRI of under-five children was 2.3 (95%CI: 1.9-2.7) as compared to non-exposed children[62]. Another recent review based on sixteen studies reported significantly elevated odds ratios ranging from 1.38-6.00 for ALRI due to exposure to IAP[58]. The strongest association of exposure to IAP and respiratory health is found in the youngest age groups[60]. This finding is consistent with the fact that younger the child the more susceptible they are to air pollutants due to their physical characteristics as well as their likelihood to stay indoors and stay with their mothers during cooking. Preschool children spend more time in the kitchens as compared to their school going elder siblings[63]. As children grow, they become more independent. School children spend a considerable time away from the house and in outdoors during peak concentration levels of air pollutants indoors. In rural China, younger children living in households that use solid fuel for cooking and heating purposes had higher 24-hour average exposures of $PM_{2.5}$ levels; 5-8 year old, 9-11 year old and 12-14 year old children had exposures of 70 $\mu g/m^3$ (95%CI: 60-80 $\mu g/m^3$), 46 $\mu g/m^3$ (95%CI: 40-49 $\mu g/m^3$) and 40 $\mu g/m^3$ (95%CI: 37-40 $\mu g/m^3$), respectively[64]. Among 1397 school children (aged 7-14 years), no definitive associations were observed between wheezing and wood/coal use (OR = 1.05; 95%CI: 0.27-4.05) or kerosene use (OR = 0.57; 95%CI: 0.1 -2.12) as compared to children living in households that use gas for cooking[65].

Parental smoking has a significant effect on respiratory health of children as they may smoke at home and exposure to SHS is inevitable in many scenarios[66]. In Nepal, it has been shown that respiratory health effects among children were high (adjusted OR = 1.41; 95%CI: 1.02-1.96) when mothers smoked[67]. Based on findings of 361021 rural and urban families in Indonesia, paternal smoking was associated with increased infant mortality (rural: OR = 1.30; 95%CI: 1.24-1.35. urban: OR = 1.10; 95%CI: 1.01-1.20), and under-5 child mortality (rural: OR = 1.32; 95%CI: 1.26-1.37. urban: OR = 1.14; 95%CI: 1.05 -1.23)[68].

Studies on middle ear infections (otitis media) due to solid fuel exposure in developing countries are limited. Although otitis media is rarely fatal, it may lead to complications including deafness and mastoiditis[17]. Exposure to SHS is strongly associated with the occurrence of middle ear infections in children. A recent meta-analysis based on 61 epidemiological studies reported that maternal postnatal smoking was associated with a 1.62 (95%CI: 1.33-1.97) risk and living with a smoker was associated with a 1.37 (95%CI: 1.25-1.50) risk of middle ear infections in children. Maternal postnatal smoking and paternal smoking were associated with a 1.86 (95%CI: 1.31-2.63) and 1.83 (95%CI: 1.61-2.07) risk of surgery for middle ear infection, respectively, as compared to not having a smoker in the household[69].

Although the evidence from the developing countries is scarce, European studies show that chemicals released from other sources (*i.e.*, indoor sources other than cooking fuel and smoking) are associated with respiratory health of children. Redecoration of apartments with paints *etc.* had a significant influence on the occurrence of obstructive bronchitis in the first (OR = 4.1; 95%CI: 1.4-11.9) and in the second year of life (OR = 4.2; 95%CI: 1.4-12.9) among children living in apartments in Germany[70].

Asthma: Studies on solid fuel use and asthma have revealed contradictory findings[17]. Use of wood fuel was associated with an increased risk for asthma among adolescents in rural Belgium (OR = 2.2; 95%CI: 1.1-4.4)[71]. The pooled OR of four studies on asthma among children exposed to biomass fuels was 0.50 (95%CI: 0.12-1.98)[59]. However, wheezing, a clinical expression of asthma[72], was associated with exposure to solid fuel smoke in children. Wheezing is a clinical symptom of lower respiratory tract infections, pneumonia, *etc*[72]. A study conducted in two settings with different outdoor air pollution levels in Sri Lanka reported that indoor cooking with unclean fuels was a risk factor for wheezing among children 7-10 years of age (adjusted OR = 2.02; 95%CI: 1.13-3.59) independent of the area of residence (adjusted OR = 1.57; 95%CI: 1.01-2.46)[73].

A recent meta-analysis estimated that the asthma is 1.33 (95%CI: 1.14-1.56) times higher among children exposed to SHS and that the duration of exposure is an important factor in the induction of asthma. The same study showed that "ever having asthma" is 1.48 (95%CI:

1.32-1.65) times higher among children exposed to SHS as compared to children not exposed to SHS[74].

Exposure to molds increases the susceptibility to asthma[75]. Although evidence from developing countries is limited, many epidemiological studies in developed countries suggest that asthma is associated with residential dampness and molds. The pooled OR of mold exposure and asthma in studies conducted in Russia, North America and 10 countries in Eastern and Western Europe was 1.35 (95%CI: 1.20-1.51)[76]. In Taiwan, the presence of visible mold (adjusted OR = 1.76; 95%CI: 1.18-2.62) was shown to be an independent determinant of incident asthma[77]. Residential concentrations of propylene glycol and glycol ethers, a class of VOC, were associated with a 1.5 fold greater likelihood of asthma (95%CI: 1.0-2.3) in Sweden[78]. However, evidence of the association between asthma and microbial agents is inconclusive[79].

Lung functions: Factors that affect development of lung function in children are potentially important in determining the lung functions of adults[80]. Lung function is a strong predictor of mortality in adults. A review of data from China has shown that reductions in children's forced expiratory volume in the first second (FEV1), forced vital capacity (FVC) and peak flow are associated with domestic coal use[81]. In Ecuador, children living in homes that use biomass fuel and children exposed to environmental tobacco smoke had lower FVC and lower FEV1 ($P < 0.05$)[82]. Among 200 school children in north India, FVC and FEV1 were lowest in boys whose households used biomass fuel ($P < 0.05$). The same study reported that all parameters were lower, though not statistically significant, in passive smokers irrespective of the type of fuel used[83]. In China, FEV1 and FVC growth of children living in households that used coal as fuel was 16.5 mL/year ($P < 0.001$) and 20.5 mL/year ($P < 0.001$), respectively, lower than in children living in households that used clean fuels[84]. In a prospective study of Chinese school children ($n = 1718$, aged 10.05 ± 0.86 years), lower growth rates in forced expiratory flow ($FEF_{25\%-75\%}$) ($P = 0.020$) during the 18 mo of follow up were reported among children exposed to SHS (> 5 cigarettes/d)[85].

Other respiratory conditions: SHS exposure during childhood is associated with increased lung cancer risk among never smoking adults (OR = 2.25; 95%CI: 1.04-4.90)[86]. A considerable number of studies has reported that coal and biomass are associated with hypopharyngeal, laryngeal and lung cancers in later life with long time exposure[10,87-90]. These studies suggest that these cancers in later life may be attributable to exposures in young ages.

FUTURE DIRECTIONS IN PREVENTION

It is evident that IAP is a major risk factor of respiratory ill health in children in developing countries. Multiple synergistic strategies are required to overcome the problem. Unclean cooking fuels, the major source of IAP, become cleaner, and more convenient, efficient and costly as people move up the "energy ladder" from animal dung, the lowest in the ladder to electricity at the top of the ladder[35]. People generally move up the ladder as socio-economic conditions improve[17]. Poverty is a major constraint to move up the energy ladder as clean fuels are more costly[91,92]. Poverty is associated with poor housing conditions resulting in poor ventilation, inability to partition the kitchen from other microenvironments of houses, and lack of kitchen amenities such as chimneys that increases the exposure to IAP. Poverty also affects other factors such as nutrition which increase the susceptibility of children to ARIs[92]. There may be other factors involved in shifting towards clean cooking fuels such as their relative availability, cultural practices and attitudes[15,93]. As a consequence, shifting from unclean to clean cooking fuels in many developing countries is slow and is likely to continue for many more decades[14].

Potential interventions to reduce IAP may target sources of pollution, improvements in living environments, and change of behaviors of persons who cook and households members[94]. Introduction of tailor made cook stoves is the intervention of choice to control IAP when shifting of fuel type to cleaner ones is slow. Many limited scale cook stove interventions have been successfully implemented in developing countries and many innovations are on the way[95-98]. However, penetration of the concept to a larger proportion of the population is a major challenge[99]. Much commitment is needed to design and distribute improved cook stoves with greater community acceptance, high energy efficiency and marked reduction of emissions to needy communities. A formal evaluation of successful improved stove programs will provide opportunities to understand the reasons for success[100]; this should be an important component of ongoing and future cook stove interventions. Behavior change activities to reduce the IAP can be focused on four main areas, namely (1) improving ventilation in the kitchen during cooking (*e.g.*, opening of windows and doors during the cooking operations); (2) keeping children away from fires; (3) improving stove maintenance; and (4) reducing the duration of burn[101,102]. Interventions should attempt to address all possible activities as a package. Further, the change of behavior and practices of communities is an essential element for the sustainability of improved cook stove interventions; audience targeted communication packages should be intertwined in such programs. Essentially, the "threat" of IAP to children's health should be emphasized among traditional stove users through appropriate media and language to target the most rural communities. Improving awareness of adverse health effects of IAP among health administrators, physicians and primary health care workers would complement prevention approaches.

The public health burden of tobacco use has shifted from the developed to the developing world; the tobacco industry is using innovative marketing strategies in the

developing world[103]. Legislature has been enacted in many countries including some developing countries banning smoking in public places, thus reducing exposure to SHS[104]. However, these laws are not practically important at household level, perhaps where the most important source of exposure of children to SHS exists[66]. Thus, extensive awareness campaigns targeting change of indoor smoking habits in households is important, despite the fact that such change of habits is difficult to achieve. Studies have shown that as a country's income level increases, cigarettes become more engineered with reductions in emission levels[103]. Cigarette design and their adverse effects may be more harmful resulting in a higher disease burden in many developing countries[103]. This concept needs be explored and understood at country level to implement standards for cigarettes. In addition, the implementation of the recommended smoking prevention strategies of WHO will certainly reduce exposure to SHS[53]. Studies on health effects of IAP sources other than from solid fuel and SHS are scarce in developing countries. This may reflect a lack of expertise and capacity to conduct scientifically sound epidemiological studies in such countries. However, "modernization" of households and household practices has introduced a spectrum of hitherto unknown chemical products, the adverse effects of which are yet to be elucidated. Hence, it is important to quantify the epidemiological risk associated with these substances as an initial step in designing mitigating strategies.

CONCLUSION

IAP disproportionately affects children's respiratory health due to their physiological susceptibility and spending more time indoors. Exposure of children in developing countries to IAP has contributed significantly to the global burden of disease. IAP is a modifiable risk factor having known interventions to mitigate its effects. Other than solid fuel and SHS exposure, pollutants from other sources, yet to be explored, may play an important role in impacting on the respiratory health of children in developing countries.

Successful prevention strategies need robust information pertaining to the problem generated from diverse settings. Future research is needed in several areas. Some key areas include quantification of different air pollutants, robust estimation of associations between indoor pollutants and adverse respiratory health effects, genetic susceptibility to indoor pollutants and their carcinogenic effects, impact on lung growth and development, characteristics and assessment of successful IAP reduction interventions (*e.g.*, cook stove interventions), cultural practices and behaviors that lead to a reduction or an increase in IAP and its exposure.

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Acknowledgments

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5 **Vallancien G**, Emberton M, Harving N, van Moorselaar RJ; Alf-One Study Group. Sexual dysfunction in 1, 274 European men suffering from lower urinary tract symptoms. *J Urol* 2003; **169**: 2257-2261 [PMID: 12771764 DOI:10.1097/01.ju.0000067940.76090.73]

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10 **Sherlock S**, Dooley J. Diseases of the liver and billiary system. 9th ed. Oxford: Blackwell Sci Pub, 1993: 258-296

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12 **Breedlove GK**, Schorfheide AM. Adolescent pregnancy. 2nd ed. Wieczorek RR, editor. White Plains (NY): March of Dimes Education Services, 2001: 20-34

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15 Morse SS. Factors in the emergence of infectious diseases. Emerg Infect Dis serial online, 1995-01-03, cited 1996-06-05; 1(1): 24 screens. Available from: URL: http://www.cdc.gov/ncidod/eid/index.htm

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16 **Pagedas AC**, inventor; Ancel Surgical R&D Inc., assignee. Flexible endoscopic grasping and cutting device and positioning tool assembly. United States patent US 20020103498. 2002 Aug 1

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Write as mean ± SD or mean ± SE.

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