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Expert Panel
Group on
Iodine Deficiency Disorders
in Hong Kong

Consensus statement on iodine deficiency disorders in Hong Kong

香港碘質缺乏症的結論綜述

This article reviews the available data on the study of iodine deficiency disorders in Hong Kong and to discuss the approach towards preventing such disorders in Hong Kong. The importance of iodine and iodine deficiency disorders is described, and the available data on the dietary iodine intake and urinary iodine concentration in different populations of Hong Kong are summarised and discussed. Dietary iodine insufficiency among pregnant women in Hong Kong is associated with maternal goitrogenesis and hypothyroxinaemia as well as neonatal hypothyroidism. Borderline iodine deficiency exists in the expectant mothers in Hong Kong. Women of reproductive age, and pregnant and lactating women should be made aware and educated to have an adequate iodine intake, such as iodised salt, as an interim measure. A steering group involving all stakeholders should be formed to advise on the strategy of ensuring adequate iodine intake, including universal iodisation of salt in Hong Kong. Continuous surveillance of iodine status in the Hong Kong population is necessary.

本文回顧香港有關碘質缺乏症研究的現有數據，並討論在香港防止此病症的方法。描述了碘質及碘質缺乏症的重要性，同時撮要及討論香港不同人口從飲食吸收碘質及尿碘質濃度的現有數據。香港孕婦飲食碘質不足，與妊娠甲狀腺腫、甲狀腺素不足及新生兒甲狀腺功能減退有關。香港的準母親有碘質近乎不足的情況。應警惕及教育處於生育年齡、懷孕及授乳的女性吸取適量碘質，例如進食碘質化鹽以作為暫時性措施。香港應成立一個包括所有利益相關團體的督導小組，以提倡確保適量吸收碘質的政策，包括在香港推行全面碘化食鹽。香港人口的碘質狀況有需要被持續監察。

Introduction

Iodine is an important trace element and an essential substrate for the synthesis of thyroid hormones. Tri-iodothyronine (T_3) and tetra-iodothyronine (T_4) are iodinated molecules that exert diverse effects on cellular metabolism, growth, and development, especially of the brain. Throughout the world, iodine deficiency is the most important preventable cause of brain damage and mental retardation, and affects a total of one billion people.[1]

The problem of iodine deficiency disorders has been overlooked in Hong Kong because of misconceptions and wrong assumptions. To tackle this problem, a Consensus Development Conference was held in Hong Kong on 3 October 2002, attended by Prof CJ Eastman, Asia-Pacific Representative of the International Council for the Control of Iodine Deficiency Disorders (ICCIDD), World Health Organization (WHO) as the overseas expert for the development of a consensus statement. This conference was followed by an open forum on 1 December 2002, which was attended by Prof BS Hetzel, founding member of the ICCIDD.

This paper presents the scientific data collected on the study of iodine deficiency disorders in Hong Kong, and summarises the discussions and consensus made during the expert panel meeting and open forum.

Key words:
Deficiency diseases;
Iodine

關鍵詞：
缺乏疾病；
碘質

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Table 1. The spectrum of iodine deficiency disorders[2]

Population	Feature
Foetus	Abortions
	Stillbirths
	Congenital anomalies
	Increased perinatal mortality
	Increased infant mortality
	Neurological cretinism: mental deficiency, deaf mutism, spastic diplegia squint
	Myxoedematous cretinism: mental deficiency, dwarfism, hypothyroidism
	Psychomotor defects
Neonate	Neonatal hypothyroidism
Child and adolescent	Retarded mental and physical development
Adult	Goitre and its complications
	Iodine-induced hyperthyroidism
All age-groups	Goitre
	Hypothyroidism
	Impaired mental function
	Increased susceptibility to nuclear radiation

Table 2. Summary of iodine deficiency disorder prevalence, by severity of public health problem, according to indicator and target population[9]

Indicator	Target population	Prevalence of deficiency (%), by severity of public health problem		
		Mild	Moderate	Severe
Goitre grade >0	School-age children	5.0-19.9	20.0-29.9	30.0
Thyroid volume >97th centile by ultrasonography	School-age children	5.0-19.9	20.0-29.9	30.0
Median urinary iodine level (μg/L)	School-age children	50.0-99.0	20.0-49.0	<20.0
Thyroid stimulating hormone >5 mIU/L, whole blood	Neonates	3.0-19.9	20.0-39.9	40.0
Median thyroglobulin (ng/mL serum)*	Children and adults	10.0-19.9	20.0-39.9	40.0

* Different assays may have different normal ranges

The importance of iodine and iodine deficiency disorders

Iodine deficiency has deleterious effects on individuals, especially pregnant women and growing children. Table 1 shows the spectrum of clinical and subclinical manifestations of iodine deficiency, which are collectively referred to as iodine deficiency disorders.[2] In areas of endemic goitre and cretinism, the problem of iodine deficiency is not difficult to recognise, because it leads to goitre, hypothyroidism, mental deficiency, and impaired growth. Milder forms of iodine insufficiency exist in non-goitrous aspects, such as maternal goitrogenesis and hypothyroidism, increased pregnancy loss, increased perinatal and infant mortality, neonatal hyperthyrotropinaemia, neonatal hypothyroidism, and irreversible neuropsychological impairment in children.[3] The most critical period is from the second trimester of pregnancy to the third year after birth.[4-7]

Since the establishment of the ICCIDD in 1986, there has been growing awareness of iodine deficiency as a major preventable cause of mental retardation. Recognising the importance of preventing iodine deficiency disorders, the World Health Assembly in 1991 adopted the goal of eliminating iodine deficiency as a public health problem by the 2000. In 1990, the world's leaders, including those of China, endorsed this goal when they met at the World Summit for Children at the United Nations. The goal was reaffirmed at the International Conference on Nutrition in 1992. The recommended daily iodine intake is shown in the Box.[8]

Identification of the occurrence of iodine deficiency disorders

Because dietary iodine intake is difficult to ascertain, outcome indicators are used to provide a measure of iodine deficiency. These indicators can be categorised according to whether the assessments are clinical (thyroid size, preferably by ultrasound, and cretinism) or biochemical (levels of urinary iodine and thyroid-related hormones). Table 2 summarises the indicators recognised by ICCIDD for iodine deficiency and the cut-off prevalence criteria for grading the level of severity of iodine deficiency in a community.[9]

ICCIDD, UNICEF, and WHO recommendations for daily iodine intake[8]

At least:
90 μg for children aged 0-59 months;
120 μg for children aged 6-9 years;
150 μg for individuals older than 12 years and for adolescents and adults; and
200 μg for pregnant and lactating women

Iodine intake and urinary iodine concentration in Hong Kong

A study performed in 1995 found that 45.3% of children, 51.7% of adults, and 55.3% of the elderly in Hong Kong had a fasting urinary iodine concentration below the cut-off level of 100 μg/L (0.8 μmol/L), which is used by the WHO to signify iodine sufficiency.[10] The proportion of local children, adults, and elderly with a urinary iodine concentration below 50 μg/L (0.4 μmol/L)—the cut-off value for severe iodine insufficiency—was 17.0%, 16.3%, and 20.0%, respectively. These data suggested that the dietary iodine intake is just adequate among children but of borderline sufficiency among adult populations.

To evaluate the problem of dietary iodine insufficiency during pregnancy, a cross-sectional study of 253 healthy southern Chinese pregnant women and their neonates was conducted in Hong Kong in 1996.[12] The results demonstrated borderline iodine intake for high proportions of the population, with 35.8% of pregnant women having a urinary iodine concentration of less than 100 μg/L.

Another cross-sectional survey performed between 1996 and 1997 among 476 adolescent secondary-level schoolchildren, however, did not show evidence of iodine deficiency.[11] The median urinary iodine concentration was 190 μg/L. It is unclear why there is such a discrepancy between the two studies. Whether it is related to the iodine assay, sample population, or a temporal difference is unclear. The overall evidence suggests that dietary iodine intake among children and adolescents is higher than that among adults in Hong Kong. The results also stress that using the level of urinary iodine excreted as a measure of iodine deficiency should be interpreted in the context of median urinary iodine excretion of the population studied. Furthermore, the results should not be inferred to other populations, even if the median urinary iodine excretion is more than 100 μg/L.

With the exception of the prospective study on pregnant women, which was done between 1996 and 1998, all studies on urinary iodine were performed before 1997. Whether there could be a temporal change in the dietary iodine intake of the Hong Kong population or a change in the dietary fads among different age-groups during the past 5 years is uncertain.

Goitre incidence in children in Hong Kong

The prevalence of clinical goitre among schoolchildren has been widely adopted as a population-level indicator of iodine deficiency[8]: a total goitre rate of 5% or more indicates that iodine deficiency is a public health problem in that area. A study of schoolchildren in a selected region of Hong Kong noted a total goitre rate by palpation of 3.5%.[11] The specificity and sensitivity of palpation are low, however, and misclassification can be as high as 40%. The ICCIDD have suggested that low goitre rates should be confirmed by ultrasonography.[8]

Transient neonatal hypothyroidism

A pointer to possible dietary iodine insufficiency in Hong Kong is the high proportion of newborns with increased levels of cord blood thyroid-stimulating hormone (TSH; thyrotropin), and the high incidence of transient neonatal hypothyroidism. In 1982, the Queen Mary Hospital launched a neonatal thyroid-screening programme, in which cord blood TSH levels were determined.[13] Data from this programme initially revealed that the mean cord blood TSH value was 7.0 mIU/L. Half of neonates had a cord blood TSH level of more than 5.6 mIU/L, and approximately 22% of neonates had values of more than 10 mIU/L (equivalent to a whole blood TSH value of 5 mIU/L). The median TSH value for all 33 541 cord blood samples collected at the Queen Mary Hospital during 1995 and 2001 was 6.37 mIU/L, with 22% of samples exceeding 10 mIU/L (unpublished data, Dr S Tam, Clinical Biochemistry Unit, Queen Mary Hospital).

Random analysis of 8728 cord blood samples collected by the Hong Kong Department of Health in 2001 showed a median TSH value of 5.9 mIU/L, with 19% of the samples having a TSH level of more than 10.0 mIU/L (unpublished data, Dr P Hung, Department of Health). In other iodine-sufficient or iodine-replenished areas, the proportion of babies with elevated TSH values (>5 mIU/L in whole blood samples or >10 mIU/L in serum samples) as detected through the neonatal screening programme is less than 3%.[8] It is a concern that TSH levels are higher in cord blood than in samples obtained on days 2 to 5 by heel prick. Although generally true, this finding does not negate the use of cord blood TSH as a useful indicator and tool for monitoring iodine sufficiency in the population.[8] The ICCIDD also state that the cut-off value for neonatal TSH of 5 mIU/L in whole blood applies to both samples obtained from cord blood and on days 2 to 5.

In Hong Kong, approximately 23% of cases of congenital hypothyroidism detected by the neonatal thyroid screening programme were transient in nature.[13,14] The cause of the transience was not attributed to transplacental transfer of inhibitory antibodies against the TSH-receptor.[15] In iodine-sufficient areas, the incidence of transient neonatal hypothyroidism is low—for example, in the United States, only 2% of cases with congenital hypothyroidism are transient in nature.[16]

Maternal goitrogenesis, hypothyroxinaemia, and foetal hypothyroidism

Pregnancy increases the maternal requirement for iodine intake because of the increased clearance of iodine from the kidneys, as well as the transfer of iodine and iodothyronines to the foetus.[17] Previous studies conducted in Europe

revealed that a marginally low iodine intake of less than 50 mg/d was sufficient to pose a significant challenge to both the maternal and neonatal thyroid gland, resulting in goitre formation in the mother and hypothyroidism in the neonate.[18]

A cross-sectional study performed on 253 healthy pregnant women in Hong Kong[12] showed that borderline iodine intake had the following substantial effects on both maternal and foetal thyroid function:

(1) A negative correlation between maternal serum TSH concentration and urinary iodine concentration;
(2) A higher cord blood TSH level in infants whose mothers had a low urinary iodine concentration compared with infants whose mothers had normal urine iodine concentrations; and
(3) Women who had given birth to infants with a cord blood TSH level of at least 16 mIU/L had lower urinary iodine concentrations and serum free T_4 levels than did women who had given birth to infants with normal cord TSH levels, and their infants also had higher cord blood thyroglobulin levels.

To further evaluate the impact of iodine insufficiency on pregnant women and their neonates in Hong Kong, a prospective study was undertaken from 1996 to 1998.[19] A total of 230 healthy women were followed from the first trimester of pregnancy until 3 months after delivery. Urinary iodine excretion was increased from the first trimester onwards, with the median urinary iodine concentration being 0.84, 0.91, and 0.98 μmol/L in the first, second, and third trimester, respectively.[19] Circulating thyroid hormone levels declined progressively with increasing gestation; levels of free T_4 and free T_3, and the free thyroxine index (FTI) were subnormal at term in 53%, 61%, and 5% of the mothers, respectively. Furthermore, the TSH level was doubled at term. Overall, the thyroid volume, as determined by ultrasonography, was increased by 30% between the first and the third trimesters. One quarter of the pregnant women at term demonstrated goitre enlargement according to the WHO cut-off volume of 18 mL. In contrast, studies conducted in areas with adequate iodine intake because of national dietary iodine supplementation programmes, such as the United States, have shown no enlargement of the thyroid gland or, at most, enlargements of less than 5% during pregnancy.[20] In Hong Kong, the change in thyroid volume in women during pregnancy was correlated negatively with urine iodine concentrations (r= –0.15; P<0.02) at term.[19] Some 6% of these women showed excessive thyroidal stimulation, as evidenced by lower serum FTI and higher thyroglobulin levels when compared with women without evidence of thyroidal stimulation. In addition, these women with thyroidal stimulation had lower urine iodine concentrations, a lower serum FTI but higher thyroglobulin and free T_3 to free T_4 ratios, as well as a larger thyroid volume throughout pregnancy. Their neonates also had higher cord blood level of TSH and thyroglobulin, as well as a slightly larger thyroid volume. One half of neonates had subnormal free T_4 levels at birth. Women with excessive urinary iodine loss during pregnancy are more likely than others to develop new thyroid nodules during the second and third trimesters of pregnancy.[21] Thus, pregnancy in a borderline iodine-sufficient environment results in maternal hypothyroxinaemia, maternal goitrogenesis, neonatal hypothyroxinaemia, hyperthyrotropinaemia, and thyroid hyperplasia.

Elimination of iodine deficiency disorders in Hong Kong

Overall, collective evidence indicates that although the dietary iodine intake of the Hong Kong population is borderline sufficient, it is inadequate to meet extra requirements at times of thyroidal stress, such as during pregnancy, neonatal period, and possibly the first few years of child growth. It is important to recognise the existence of iodine deficiency, however mild, during pregnancy. A mild degree of maternal iodine deficiency and maternal hypothyroidism during pregnancy can result in poorer psychological and neurological performance in the child, as well as neonatal hypothyroidism.[22-24] Transient low levels of circulating T_4 at the critical stage of brain development in the newborn has been shown to result in an intelligent quotient (IQ) loss of 5 to 10 points.[25]

Strategy to increase iodine intake

Few places in this world can ensure an adequate population-level intake of dietary iodine through natural food sources. The majority of the world's population achieves adequate iodine intake through national supplementation programmes, which commonly include the addition of iodine to the salt supply. Universal salt iodisation means the iodisation of all human and livestock salt, including salt used in the food industry. The ICCIDD advocated universal salt iodisation as the ultimate approach towards eliminating iodine deficiency disorders. In 1993, WHO and UNICEF recommended this approach as the main strategy to achieve elimination of iodine deficiency.[26]

Salt iodisation involves the addition of a small amount of iodine (20-100 mg of iodine per kilogram of salt, or 20-200 parts per million [ppm]) in the form of potassium iodate rather than potassium iodide, owing to the greater stability of the former compound.[27] Supplementation by salt iodisation in populations with iodine deficiency disorders has resulted in effective clinical improvement and considerable reduction in all manifestations of iodine deficiency, including cretinism and goitre.[28,29] Currently, 75% of affected countries have legislation relating to salt iodisation, and 68% of affected populations have access to iodised salt. In China, legislation enforcing universal iodisation of salt was passed in 1996; in southern China and Guangzhou, with the government subsidy, salt has been iodised at 20-50 ppm with the aim of maintaining a level of 20 ppm at the consumer level.[30] Offering iodised

salt only to high-risk groups, such as pregnant women and young children, would be only a temporary measure, because this policy is expected to be non-sustainable. Another problem with this policy is that iodised salt is given only after women become pregnant. Iodine supplementation has to be done early, because treatment initiated in the third trimester can improve brain growth and developmental achievement, but has no influence on the neurological status of the affected child.[6] In addition, early iodine supplementation can prevent goitre development in both the pregnant mother and the foetus.[31] According to WHO recommendations, the daily iodine intake for pregnant and lactating women should be 200 μg.[8] The United States recommended in 2001 that a daily iodine intake should be 220 μg and 290 μg for pregnant and lactating women, respectively.[32]

Iodine supplementation during pregnancy is essential in Hong Kong because seafood is not commonly consumed.[10] Currently, no programme exists to eliminate iodine deficiency in Hong Kong, as a result of the assumption that such a deficiency is non-existent and the concern that a high incidence of juvenile Graves' disease may be related to a high intake of iodine in adolescent children.[33] Although high iodine intake is associated with a higher relapse rate of Graves' disease,[34,35] no published evidence demonstrates that increases in iodine intake cause this disease. In fact, the incidence of hyperthyroidism due to nodular goitre and the incidence of Graves' disease decreased in Switzerland after the gradual iodisation of salt from 7.5 ppm to 15.0 ppm.[36] A similar phenomenon was observed in Denmark and Spain, with a reduction in the incidence of subclinical hyperthyroidism both in children and adults.[37-39] The tolerable upper limit of iodine intake is set at 1100 μg/d by the WHO.[40]

There is also concern that an increase in iodine intake may result in an increased incidence of thyroid dysfunction—namely, iodine-induced hyperthyroidism[41] and iodine-induced hypothyroidism.[42] Iodine-induced hyperthyroidism is seen in certain individuals residing in low–iodine intake regions when their intake of iodine is more than what they are accustomed to, particularly in the presence of existing nodular changes in the thyroid gland. An increased incidence of hyperthyroidism following the introduction of iodised salt has been reported in severely iodine-deficient countries.[41] However, populations in these areas were exposed to iodine excesses largely because of poor monitoring of the quality of iodised salt and of the population's iodine intake. The incidence of iodine-induced hyperthyroidism during national iodine prophylaxis programmes in many countries has been low, and usually disappears several years after the initiation of the programme.[40,43] Hence, iodine-induced hyperthyroidism should not be regarded as a contra-indication to an iodisation programme, given the enormous long-term benefits of the elimination of iodine deficiency on the whole population. A systematic, closely monitored programme in which the level of iodine in salt increases in small increments can minimise the problem of iodine-induced hyperthyroidism.

Iodine-induced hypothyroidism, which can develop through a habitual intake of large amounts of iodine (1-43 mg/d, which is 10-43 times the daily requirement) has been reported in only two places in the world: Hokkaido, Japan,[42,44] and Gaojiabu Village, Shaanxi Province, China.[45]

Experience from China on universal salt iodisation

About half the world's cases of iodine insufficiency occur in China. Salt iodisation programmes in China began in 1994, and legislation on universal salt iodisation was implemented in the country 2 years later. All salt is currently iodised at 40 to 50 ppm. According to national surveillance data, the total goitre rate in 9- to 10-year-old schoolchildren decreased from 20.43% in 1995 to 10.86% in 1997, accompanied by an increase in the quality of household iodised salt from 39.9% to 81.1%,[30] and an increase in IQ by nine to 11 points in the endemic area. According to a WHO report in 1998, implementation of the national plan to eliminate iodine deficiency disorders in China is a great example to other countries of what can be done and will serve as a milestone in modern public health achievements. The key to achieving this success was the development of a strategy to involve the government and major stakeholders; training of Chinese personnel, education of the public about iodine deficiency; universal salt iodisation; and implementation of an appropriate monitoring system to collect data on salt iodine content, urinary iodine levels, and (recently) neonatal TSH levels. There has been no increase in the incidence of hyperthyroidism after iodisation in areas of iodine deficiency, mild excess, or excess.[46] These results confirm the believe that salt iodisation at appropriate levels and adequate monitoring is unlikely to give rise to iodine-induced hyperthyroidism.

Iodised salt in Hong Kong

Although most of the salt supply in Hong Kong comes from the southern provinces of China, a survey conducted by the Hong Kong Consumer Council in 1998 on local brands of salt available in supermarkets and grocery stores showed either no iodisation or low levels of iodisation.[46] According to a survey conducted by the Food and Environmental Hygiene Department in 2002, salt available in Hong Kong is predominantly not iodised. Only 15 (21%) of 72 brands of food salt contain iodine at a concentration of 3.6 to 64.0 ppm. There is currently no specific requirement for salt iodisation in Hong Kong. No data are available on whether iodised salt is being used by the food industry or the iodine content of the salt used. There is also no good quality and comprehensive survey on eating habits and use of iodised salt among different age-groups in Hong Kong.

Overall, the iodine intake of the Hong Kong population should be brought up to—but not exceed—the level at which iodine deficiency disorders can be avoided. Health care policies are urgently needed to establish intervention programmes that are aimed at achieving a relatively uniform iodine intake while avoiding deficient or excessive iodine intake in subpopulations. Until then, at-risk subpopulations, including pregnant and lactating women and young children of up to the age of 3 years, should receive a physiological dose of potassium iodate supplementation.

Suggestions

The expert panel made the following suggestions at the consensus meetings:

(1) Assessment of the situation

Although the available data suggest the existence of iodine insufficiency among pregnant women and their newborns, the situation among young children is less clear. The following assessments are urgently needed in Hong Kong:

(a) To confirm the goitre rate among schoolchildren and women of reproductive age by ultrasound assessment, and to compare thyroid volumes with age- and body-size–adjusted values obtained from iodine-repleted populations. Schoolchildren could be surveyed during their routine health screening, which is currently conducted by the Department of Health. Healthy women of childbearing age and young children could be surveyed at maternal and child health clinics or the Well Women Clinic of the Department of Health;
(b) To monitor regularly the urinary iodine concentration among high-risk groups—namely schoolchildren, women of reproductive age (18-44 years), and neonates. A standardised method of urinary iodine measurement should be adopted, and the monitoring should preferably be conducted by the Department of Health;
(c) To monitor results of the neonatal TSH screening programme to track temporal changes; and
(d) To conduct a comprehensive survey on iodine content of food items, especially food salt, in Hong Kong.

(2) Development of an action plan

A multidisciplinary and intersectoral task force group should be established to eliminate the problem of iodine deficiency disorders in Hong Kong and to address the need for legislation or regulation for universal salt iodisation. This task force group should involve all stakeholders, as follows:

(a) Government officials;
(b) Health policy–makers;
(c) Salt producers, salt importers and distributors, and food manufacturers;
(d) Concerned lay groups;
(e) The Consumer Council; and
(f) Nutrition, food, and medical professionals and scientists, and other key opinion leaders.

(3) Concerns

Some concerns regarding legislation on universal salt iodisation in Hong Kong are as follows:

(a) Matters related to public health issues;
(b) No specific group in the population should be jeopardised by this legislation; for example, people with a history of hyperthyroidism should have access to non-iodised salt; and
(c) Freedom of choice in the society.

(4) Interim measures

(a) Public education and health promotion activities are needed to improve awareness of iodine deficiency disorders, in both the public and medical profession. The first target subpopulations are schoolchildren, pregnant and lactating women, as well as women of reproductive age;
(b) While waiting for a strategy to ensure adequate iodine intake, including universal iodisation of salt, all pregnant and lactating women should be advised to increase their dietary iodine intake to not less than 200 μg/d. This level can be achieved by either prescription of potassium iodide tablets or through the intake of salt iodised at 10 to 20 ppm. However, this approach is only an interim measure and is unlikely to be sustainable; and
(c) Food labelling to increase public awareness of iodine deficiency and of the need to increase dietary iodine through natural food sources.

Consensus summary statement

Borderline iodine deficiency exists in the expectant mothers in Hong Kong. Women of reproductive age and pregnant and lactating women should be made aware and educated to have an adequate iodine intake—for example, by using iodised salt—as an interim measure. A steering group involving all stakeholders should be formed to advise on the strategy of ensuring adequate iodine intake, including universal iodisation of salt in Hong Kong. Continuous surveillance of iodine status in the Hong Kong population is necessary.

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Appendix

This consensus statement is endorsed by the following

organisations: Hong Kong College of Obstetricians and Gynaecologists; Hong Kong College of Paediatricians; Hong Kong College of Physicians; Hong Kong Dietitians Association; The Hong Kong Paediatric Society; The Hong Kong Society of Child Neurology and Development Paediatrics; The Hong Kong Society of Endocrinology, Metabolism and Reproduction; The Hong Kong Society of Paediatric, Endocrinology and Metabolism; and The Obstetrical and Gynaecological Society of Hong Kong.

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